

Article

The Effect of Physical Activity on Heart Structure and Function in African University Students: A Comparative Cross-Sectional Study

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Abstract

Background: Regular physical activity induces physiological cardiac remodeling (“athlete’s heart”), which may overlap with pathological hypertrophy. Regional echocardiographic and electrocardiographic data among young African adults are limited. This study evaluated how graded physical activity relates to cardiac structure and function among university students in Ghana. **Methods:** In this comparative cross-sectional study, 174 apparently healthy students aged 18–30 years were categorized into four physical activity groups in the preceding six months: level 1, no regular exercise (n = 29, 16.7%); level 2, <30 min/day of exercise (n = 41, 23.6%); level 3, 30 to 60 min/day of moderate exercise (n = 29, 16.7%); and level 4, >1 h/day of vigorous exercise (n = 75, 43.1%). Anthropometry, blood pressure, 12-lead electrocardiography, and comprehensive transthoracic echocardiography were obtained. Cardiac indices were compared across activity levels using the Kruskal–Wallis or Welch’s ANOVA test, with post hoc comparisons and regression analyses performed where appropriate. **Results:** Participants were predominantly male (56.3%), with a mean age of 22.3 ± 3.50 years, BMI of 23.0 ± 4.39 kg/m², systolic blood pressure of 118 ± 13.0 mmHg, diastolic blood pressure of 71.3 ± 9.11 mmHg, and heart rate of 66.9 ± 10.9 bpm. Compared with sedentary participants, those in level 4 had a higher IVSd (9.87 ± 1.61 vs. 8.17 ± 1.47 mm, $p < 0.001$), LVIDd (43.6 ± 6.96 vs. 40.2 ± 3.58 mm, $p = 0.002$), LVPWd (10.1 ± 1.95 vs. 8.91 ± 1.60 mm, $p = 0.003$), and LVM (54.6 ± 7.45 vs. 47.1 ± 6.57 g, $p < 0.001$). EDV and ESV also increased with activity (90.1 ± 24.8 vs. 69.7 ± 17.8 mL, $p < 0.001$; 32.4 ± 12.8 vs. 25.6 ± 6.52 mL, $p = 0.023$). Systolic function was preserved across groups, with an EF of $59.3 \pm 4.86\%$ in level 4 vs. $58.3 \pm 5.34\%$ in level 1 ($p = 0.707$). Level 4 participants had a higher SV (57.6 ± 16.7 vs. 46.3 ± 10.4 mL, $p = 0.003$), CO (3.83 ± 1.17 vs. 3.05 ± 0.70 L/min, $p = 0.022$), and CI (2.19 ± 0.66 vs. 1.77 ± 0.37 L/min/m², $p = 0.015$). Bradycardia was most frequent in level 4 (35.8% vs. 18.2% in level 1, $p = 0.041$), and PR interval was longer in participants exercising ≥ 30 min/day than in those exercising <30 min/day (166 ± 23.2 vs. 162 ± 21.8 ms, $p = 0.031$). **Conclusions:** In young African university students, greater physical activity was associated with mild physiological remodeling, including a higher left ventricular wall thickness, cavity size, and mass, while systolic and diastolic indices remained preserved. The mean values in the most active group were 9.87 mm IVSd and 10.1 mm LVPWd with preserved EF, supporting activity-related adaptation rather than overt pathological hypertrophy and highlighting the need for population-specific cardiovascular interpretation.



Academic Editors: Song-Young Park and Gwenael Layec

Received: 24 April 2026

Revised: 9 June 2026

Accepted: 9 June 2026

Published: 17 June 2026

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Keywords: physical activity; cardiac remodeling; echocardiography; athlete's heart; African university students; cardiovascular adaptation

1. Introduction

Regular physical activity induces beneficial cardiovascular adaptations. It improves myocardial efficiency and reduces morbidity associated with hypertension and cardiovascular disease [1]. In athletes, long-term training produces characteristic structural and functional cardiac changes collectively referred to as the athlete's heart [2]. These changes are physiological and reversible, distinguishing them from the pathological hypertrophy seen in hypertension and cardiomyopathies [3,4].

However, the distinction between physiologic and pathologic cardiac remodeling remains a diagnostic challenge, particularly in populations where hypertension and structural heart disease are prevalent [4,5]. For instance, physiological and pathological remodeling may coexist, producing hybrid phenotypes that are difficult to classify [6]. Ethnic variation remains a major area of controversy. Black/African athletes demonstrate a higher prevalence of increased LV wall thickness and repolarization changes, which may mimic cardiomyopathy. While ethnicity-specific interpretation is recommended, defining appropriate reference limits without risking misclassification remains challenging [7,8]. Again, despite general agreement on diagnostic overlap, there is no universally accepted cut-off for LV wall thickness that definitively distinguishes physiological from pathological hypertrophy. Wall thickness values between 12–15 mm may be observed in both athlete's heart and early hypertrophic cardiomyopathy (HCM), limiting the diagnostic utility of fixed thresholds [6].

Currently, screening athletes for HCM and other conditions uses “ethnic-adjusted” norms, based on the assumption that Black athletes exhibit greater left ventricular wall thickness [9,10]. However, the existing data on “Black athletes” comes from elite or collegiate cohorts in Europe and North America [10,11], with young amateur Black athletes in Africa remaining severely underrepresented in sports cardiology research [10].

Furthermore, limited access to advanced imaging modalities, such as echocardiography and cardiac magnetic resonance imaging (MRI), in many African sports medicine facilities restricts accurate characterization of these adaptations. This gap poses both diagnostic and public health concerns, as misinterpretation of physiologic hypertrophy as pathological may lead to unnecessary sports disqualification, while underdiagnosis of true pathology increases the risk of exercise-related cardiac events. Moreover, given that hypertension prevalence is disproportionately higher in African populations [11], there is a compelling need to understand how these hemodynamic factors affect cardiac structure among young amateur African athletes.

Therefore, this study aimed at using transthoracic echocardiography and electrocardiography (ECG) to assess the structure and function of the heart among university students in Ghana, including amateur athletes. The main objective was to ascertain the impact of physical activity on the structure and function of their hearts and to determine average cardiac dimensions in different physical activity levels.

2. Materials and Methods

2.1. Study Design and Setting

A comparative cross-sectional study was conducted among young adult student athletes and non-athletes at an African university to assess the impact of varying levels of physical activity on cardiac structure and function. The study adhered to the principles

of the Declaration of Helsinki and obtained ethical approval from the institutional review board prior to participant recruitment.

Setting and dates: The study was conducted at Kwame Nkrumah University of Science and Technology (KNUST), Kumasi, Ghana. Recruitment and data collection occurred between July 2024 and September 2024.

2.2. Participants

Participants were recruited during a university athletic competition event. Student athletes participating in the competition and student spectators attending the event were approached by the study team and informed about the ongoing study. Those who expressed interest in participating were screened for eligibility and were subsequently given scheduled appointments to attend the University laboratory for further assessment. At the laboratory visit, participants completed the study questionnaire and underwent anthropometric measurements, blood pressure assessment, resting 12-lead electrocardiography, and transthoracic echocardiography. Participants were then categorized into four physical activity groups based on their self-reported daily duration and intensity of physical activity in the preceding six months:

- **Level 1:** Individuals who did not regularly engage in physical activity.
- **Level 2:** Individuals who engaged in less than 30 min of moderate exercise daily.
- **Level 3:** Individuals who engaged in 30 min to 1 h of moderate exercise daily.
- **Level 4:** Individuals who engaged in vigorous exercise for more than 1 h daily.

Levels 3 and 4 included participants with higher habitual activity levels, including student athletes.

All participants were apparently healthy. For the purpose of this study, “apparently healthy” was operationally defined using a pre-enrolment screening assessment that included self-reported medical history, a basic physical examination, and resting blood pressure. Participants were eligible if they had no known physician-diagnosed cardiovascular or metabolic disease; no history of smoking, congenital or structural heart disease, cardiomyopathy, arrhythmia, diabetes mellitus, or chronic hypertension; no current cardiorespiratory symptoms; and no long-term medication use likely to affect cardiac structure or function. Blood pressure and ECG findings obtained during the study were recorded and classified for analysis; clinically relevant incidental abnormalities were reviewed by a cardiologist and considered in eligibility and interpretation of findings.

2.3. Variables

Exposure included self-reported daily physical activity duration, categorized into four levels. Outcomes included echocardiographic measures of cardiac structure and function and electrocardiographic parameters. Potential confounders considered included age, sex, body size (e.g., BMI/BSA), and blood pressure.

2.4. Bias

To minimize measurement bias, standardized protocols were used for anthropometry and blood pressure. ECG interpretation was independently reviewed by a cardiologist, and echocardiography was performed by a certified cardiac sonographer following ASE/EACVI guidance [12].

2.5. Study Size

The study size was estimated using the G*Power software version 3.1.9.7. Using F tests as the test family, test variables included effect size (f), alpha value, desired statistical power, and number of groups. The effect size used was 0.25, the alpha value was 0.05,

the statistical power was 0.80, and the number of groups was 4, representing the levels of physical activity. The estimated sample size obtained was 180. Hence, a minimum of 180 participants were required. However, due to the dropout rate, a total of 174 participants (aged 18–30 years) were enrolled using stratified random sampling.

2.6. Quantitative Variables

Continuous variables were summarized as means $\pm$ standard deviation (SD) and categorical variables as counts and percentages. Physical activity was analyzed as an ordinal exposure using the predefined four activity levels.

2.7. Data Sources and Measurements

Anthropometric and Hemodynamic Measurements

Participants' height and weight were measured using a calibrated stadiometer and digital weighing scale, respectively, with participants barefoot and in light clothing. Body mass index (BMI) was calculated as weight (kg) divided by height (m^2). BMI categorization was as follows: underweight ($<18.5 \text{ kg/m}^2$), normal ($18.5\text{--}24.99 \text{ kg/m}^2$), overweight ($25\text{--}29.99 \text{ kg/m}^2$) and obese ($>30 \text{ kg/m}^2$). Body surface area (BSA) was estimated using the DuBois formula. Blood pressure was measured using a validated automated sphygmomanometer (Omron[®] HEM series, Omron Healthcare Co. Ltd., Kyoto, Japan), following a 5 min rest period, and classified according to the American Heart Association (AHA) guidelines [13]. Systolic BP categorization was as follows: normal ($<140 \text{ mmHg}$), stage 1 ($140\text{--}159 \text{ mmHg}$) and stage 2 ($160\text{--}179 \text{ mmHg}$). Diastolic BP was categorized as normal ($<90 \text{ mmHg}$), stage 1 ($90\text{--}99 \text{ mmHg}$) and stage 2 ($100\text{--}109 \text{ mmHg}$). Heart rate (HR) was measured from the sphygmomanometer.

2.8. Electrocardiography Assessment

Resting electrocardiography (ECG) was performed on all study participants using a standard 12-lead ECG machine following established international guidelines [14]. Participants included athletes and non-athletes and were assessed under similar conditions. Prior to ECG acquisition, participants were instructed to abstain from vigorous physical activity, caffeine, and stimulant use for at least 24 h. Recordings were obtained with participants in the supine position after a minimum of 5 min of rest in a quiet environment.

Standard electrode placement was used, and ECGs were recorded at a paper speed of 25 mm/s and a calibration of 10 mm/mV . The ECG tracings were analyzed for heart rate, rhythm, frontal QRS axis, PR interval, QRS duration, and ST-T wave morphology. Cardiac chamber enlargement and hypertrophy were assessed using established voltage criteria, including Sokolow–Lyon, Cornell, and R wave amplitude in aVL for left ventricular hypertrophy, as well as criteria for atrial enlargement and right ventricular hypertrophy.

All ECGs were independently reviewed by a cardiologist to identify conduction abnormalities, arrhythmias, repolarization patterns, and other electrocardiographic variants. Findings such as sinus bradycardia, early repolarization, and first-degree atrioventricular block were interpreted with consideration of physiological adaptations to physical activity. ECG parameters were categorized and analyzed according to participants' level of physical activity to compare athletes and non-athletes.

2.9. Echocardiographic Assessment

Comprehensive transthoracic echocardiography (TTE) was performed using a 2.5–3.5 MHz phased array transducer on a Siemens Acuson 700 ultrasound system (Siemens Medical Solutions, Malvern, PA 19355, USA). Echocardiographic imaging and measurements were initially obtained by a certified cardiac sonographer and independently verified by a cardiologist before final measurements were recorded. This process was intended to mini-

mize measurement error and improve the accuracy of the recorded values. Measurements and analyses adhered to the American Society of Echocardiography (ASE) and European Association of Cardiovascular Imaging (EACVI) guidelines [12].

2.10. Left Ventricular Structure

The following two-dimensional parameters were measured from the parasternal long-axis view:

- Interventricular septal thickness in diastole and systole (IVSd and IVSs).
- Left ventricular internal diameter in diastole and systole (LVIDd and LVIDs).
- Left ventricular posterior wall thickness in diastole and systole (LVPWd and LVPWs).

Left ventricular mass (LVM) was calculated using the ASE-corrected cube formula, and relative wall thickness (RWT) was derived as follows: $[(\text{PWTd} + \text{IVSd})/\text{LVIDd}]$.

2.11. Left Ventricular Function

Systolic function was evaluated using fractional shortening (FS), Simpson's biplane ejection fraction (EF), stroke volume (SV), cardiac output (CO), and cardiac index (CI). Normal LV systolic function was defined as preserved Simpson's biplane EF within accepted sex-specific reference limits, with $\text{EF} \geq 52\%$ in males and $\geq 54\%$ in females.

Diastolic function was assessed using pulsed-wave Doppler at the mitral valve leaflet tips to record peak early (E) and late (A) filling velocities, E/A ratio, and deceleration time (Dec T). Tissue Doppler imaging (TDI) at the lateral mitral annulus measured early (E') and late (A') myocardial velocities, from which E/E' and E'/A' ratios were derived.

2.12. Right Ventricular Structure and Function

The right ventricular internal diameter in diastole (RVIDd) and right ventricular out-flow tract diameter (RVOTd) were measured in the parasternal long-axis and subcostal views. Tricuspid annular plane systolic excursion (TAPSE) was used to assess right ventricular systolic performance. Normal RV systolic function was defined as $\text{TAPSE} > 17 \text{ mm}$. Peak early (TV E) and late (TV A) tricuspid inflow velocities, as well as the TV E/A ratio, were obtained using pulsed-wave Doppler.

2.13. Atrial Measurements

The left atrial diameter (LAsd) was obtained from the parasternal long-axis view at end-systole, while the left atrial area (LAA) was measured using planimetry from the apical four-chamber view.

2.14. Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 25.0. Descriptive statistics are expressed as means $\pm$ standard deviation (SD) for continuous variables and as frequencies and percentages for categorical variables. Normality of distribution was tested using the Kolmogorov–Smirnov test, with a p -value of less than 0.05 demonstrating non-parametric data and vice versa.

Comparisons among the four physical activity groups were conducted using:

- The Kruskal–Wallis test for non-parametric variables.
- Welch's ANOVA for parametric variables.
- The Dwass–Steel–Critchlow–Fligner and Games–Howell post hoc tests for pairwise comparisons.

Associations between participants' characteristics and cardiac parameters were explored using Spearman's correlation for non-parametric variables or Pearson's correlation coefficient for parametric variables. Univariate and multivariate linear regression analyses were performed to identify independent predictors of cardiac dimensions and function. Sta-

tistical significance was set at $p < 0.05$. Covariates were included in the regression analysis based on a prior correlation analysis. Only statistically significant variables ($p < 0.05$) were included. In a multicollinearity analysis, variance inflation factor (VIF) diagnostics were run on all predictors. Initially, anthropometric parameters including height (VIF = 35.51), weight (VIF = 215.60), BMI (VIF = 24.65) and BSA (VIF = 253.30) showed severe multicollinearity. To address this, height and weight were later excluded from the final model, leading to a VIF of less than 1.70 for all predictors.

2.15. Sensitivity Analyses

No formal sensitivity analyses were performed. The primary analyses were conducted as prespecified.

2.16. Missing Data

Missing data were minimal and included the tricuspid valve E and A velocities of 12 participants. No imputation was performed; analyses were based on complete observations for the variables included in each model.

2.17. Sampling Strategy

Participants were enrolled using stratified random sampling. Analyses compared outcomes across the four physical activity levels.

3. Results

3.1. Participants' Characteristics

A total of 174 young adults were included in the study. Of these participants, 56.3% were males. The mean age was 22.3 ± 3.50 years, with 70.1% having a normal body mass index (BMI). The mean body surface area (BSA) was 1.74 ± 0.16 , and the mean systolic and diastolic blood pressures were 118 ± 13 mmHg and 71.3 ± 9.11 mmHg respectively. About 94.3% had a normal systolic blood pressure, while 5.7% had stage 1 hypertension. Also, 95.4% had a normal diastolic blood pressure, while 4.0% had stage 1 and 0.6% had stage 2 hypertension. The mean heart rate was 66.9 ± 10.9 , with 70.9% having a normal sinus rhythm. Additionally, 43.1% engaged in vigorous exercise for more than an hour, while 16.7% did not really engage in any physical activity (see Table 1).

Table 1. Characteristics of study participants.

Characteristics	Frequency (n = 174)	Percentage (%)	Mean $\pm$ SD
Gender			
Male	98	56.3	
Female	76	43.7	
Age (years)			22.3 ± 3.50
Height (cm)			169.0 ± 9.34
Weight (kg)			65.0 ± 11.0
BMI (kg/m ²)			23.0 ± 4.39
Underweight	15	8.6	
Normal	122	70.1	
Overweight	26	14.9	
Obese	11	6.3	

Table 1. *Cont.*

Characteristics	Frequency (n = 174)	Percentage (%)	Mean $\pm$ SD
BSA (m ²)			1.74 $\pm$ 0.16
Systolic blood pressure (mmHg)			118 $\pm$ 13.0
Normal	164	94.3	
Stage 1	10	5.7	
Diastolic blood pressure (mmHg)			71.3 $\pm$ 9.11
Normal	166	95.4	
Stage 1	7	4.0	
Stage 2	1	0.6	
Heart rate (bpm)			66.9 $\pm$ 10.9
Bradycardia	42	28.4	
Normal	105	70.9	
Tachycardia	1	0.7	
Level of physical activity			
1	29	16.7	
2	41	23.6	
3	29	16.7	
4	75	43.1	

SD: Standard deviation; BMI: Body mass index; BSA: Body surface area; 4: Vigorous exercise for more than an hour; 3: 30 min to an hour of moderate exercise; 2: Less than 30 min of exercise; 1: Do not really engage in exercise.

3.2. Left Ventricular Structure

Echocardiographic parameters for assessing left ventricular structure are described in Table 2, with Figure 1 depicting how images and measurements were obtained. The mean interventricular septum diameter in diastole and systole was 9.13 mm $\pm$ 1.72 and 13.8 mm $\pm$ 3.64 respectively. The mean left ventricular internal diameter in systole and diastole were 42.1 mm $\pm$ 6.41 and 27.1 $\pm$ 5.31 mm respectively. The mean left ventricular mass was 51.5 g $\pm$ 7.76, ranging from 36.70 g to 78.10 g. The mean end-diastolic and -systolic volumes were 82.3 mL $\pm$ 23.8 and 29.7 mL $\pm$ 12.5 respectively. All the parameters assessing left ventricular structure were non-parametric, except for end-diastolic volume (see Table 2).

Table 2. Parameters measuring left ventricular structure.

Parameters	Mean $\pm$ SD	Range (Min–Max)	Kolmogorov–Smirnov <i>p</i> -Value
IVSd (mm)	9.13 $\pm$ 1.72	5.40–14.30	0.001
LVIDd (mm)	42.1 $\pm$ 6.41	4.60–55.90	<0.001
LVPWd (mm)	9.63 $\pm$ 1.80	5.20–15.60	<0.001
LVM (g)	51.5 $\pm$ 7.76	36.70–78.10	<0.001
IVSs (mm)	13.8 $\pm$ 3.64	1.40–50.80	<0.001
LVIDs (mm)	27.1 $\pm$ 5.31	2.50–48.10	<0.001
LVPWs (mm)	15.1 $\pm$ 2.55	6.00–23.40	0.027
End-diastolic volume (EDV) (mL)	82.3 $\pm$ 23.8	15.20–153.00	0.081
End-systolic volume (ESV) (mL)	29.7 $\pm$ 12.5	6.50–108.00	<0.001

SD: Standard deviation; Min: Minimum; Max: Maximum; IVSd: Interventricular septum diameter in diastole; LVIDd: Left ventricular internal diameter in diastole; LVPWd: Left ventricular posterior wall in diastole; LVM: Left ventricular mass; IVSs: Interventricular septum in systole; LVIDs: Left ventricular internal diameter in systole; LVPWs: Left ventricular posterior wall in systole; EDV: End-diastolic volume; ESV: End-systolic volume; mL: milliliters.

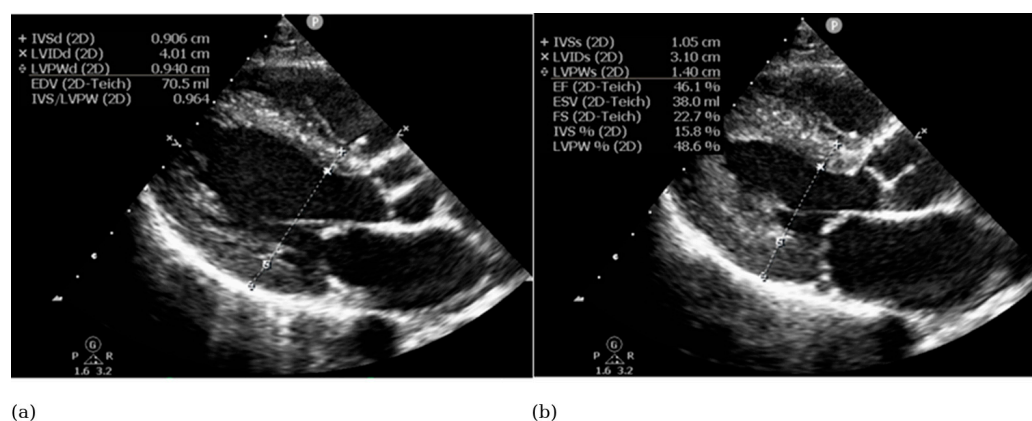


Figure 1. (a,b) Parasternal long-axis view in diastole (a) and systole (b) showing left ventricular measurements.

Table 3 presents the echocardiographic data on the left ventricular systolic and diastolic function of the study participants. The mean ejection fraction estimated using Simpson's biplane method (Figure 2) was 59.2 ± 5.31 , and the mean cardiac output (CO) was 3.57 ± 1.11 . For diastolic function parameters (Figures 3 and 4), the mean peak early left ventricular diastolic filling velocity (E) was 0.813 ± 0.1 , ranging from 0.140 to 1.20. The mean E/A ratio was 1.74 ± 0.44 , while the mean E/E' was 4.94 ± 0.94 , ranging from 0.651 to 7.86. All parameters were non-parametric except EF ($p > 0.05$).

Table 3. Left ventricular function echocardiographic parameters of study participants.

Parameters	Mean $\pm$ SD	Range (Min–Max)	Kolmogorov–Smirnov p -Value
Systolic			
EF (Simpson) (%)	59.2 ± 5.31	47.7–75.40	0.105
FS (%)	36.0 ± 7.13	24.2–85.50	<0.001
CO (L/min)	3.57 ± 1.11	1.78–8.33	<0.001
CI (L/min/m ²)	2.04 ± 0.62	0.187–4.67	0.002
Diastolic			
E (cm/s)	0.813 ± 0.16	0.140–1.20	0.010
A (cm/s)	0.491 ± 0.15	0.280–1.98	<0.001
E/A ratio	1.74 ± 0.44	0.510–3.16	<0.001
Dec T (s)	200 ± 70.80	1.96–533.00	<0.001
E' (cm/s)	0.166 ± 0.03	0.0700–0.24	0.039
A' (cm/s)	0.0989 ± 0.17	0.0400–2.23	<0.001
E/E'	4.94 ± 0.94	0.651–7.86	<0.001
E'/A'	2.01 ± 0.59	0.0783–4.00	0.003

EF—Ejection fraction (Simpson's method); FS—Fractional shortening; CO—Cardiac output; CI—Cardiac index; E—Peak early diastolic mitral inflow velocity; A—Peak late (atrial) diastolic mitral inflow velocity; E/A ratio—Ratio of early to late diastolic mitral inflow velocities; Dec T—Deceleration time of the E wave; E'—Peak early diastolic mitral annular velocity; A'—Peak late diastolic mitral annular velocity; E/E'—Ratio of early mitral inflow to annular velocity; E'/A'—Ratio of early to late diastolic mitral annular velocities.

3.3. Right Ventricular Structure and Function

For right ventricular structure, the right ventricular internal diameter in diastole (RVIDd) and right ventricular outflow tract in diastole (RVOTd) were measured. For systolic function, tricuspid annular plane excursion (TAPSE) was measured (Figure 5). Peak early diastolic tricuspid inflow velocity (TV E), peak late (atrial) diastolic tricuspid inflow velocity (TV A), and ratio of early to late diastolic tricuspid inflow velocities were used to assess diastolic function. The mean RVIDd and RVOTs were 29.3 ± 4.06 mm and 27.4 ± 4.48 mm, respectively. The mean TAPSE was 23.3 ± 4.16 mm, ranging from 12.7 to 35.5 mm. Only peak late (atrial) diastolic tricuspid inflow velocity (TV A) and ratio of early to late diastolic tricuspid inflow velocities had a non-parametric distribution (Table 4).

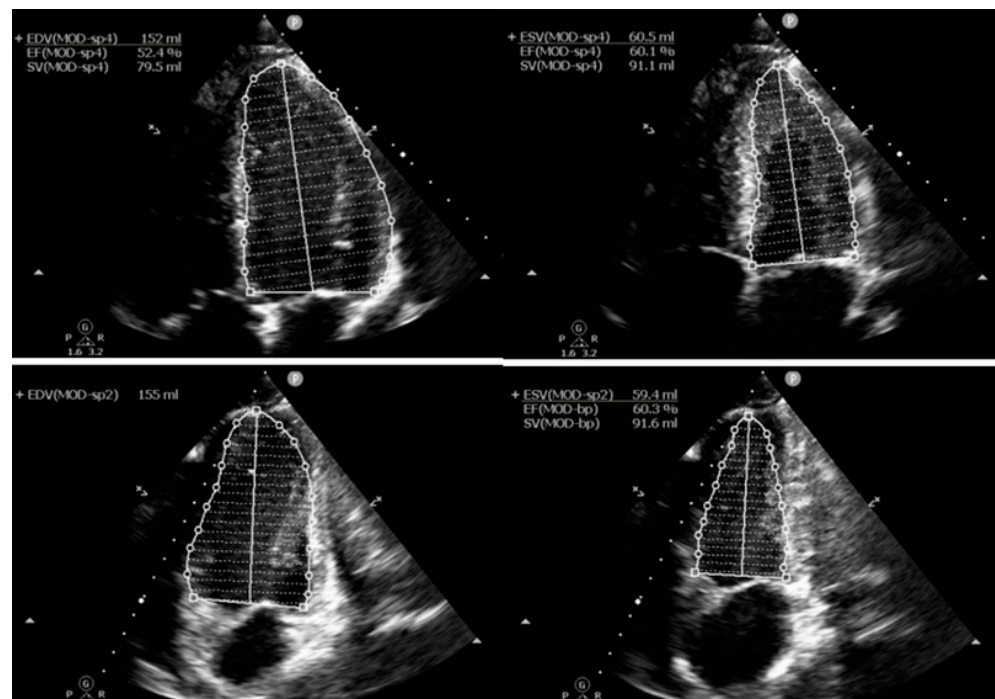


Figure 2. Echocardiographic image of the apical four- and two-chamber views demonstrating measurement of ejection fraction using Simpson's biplane method.

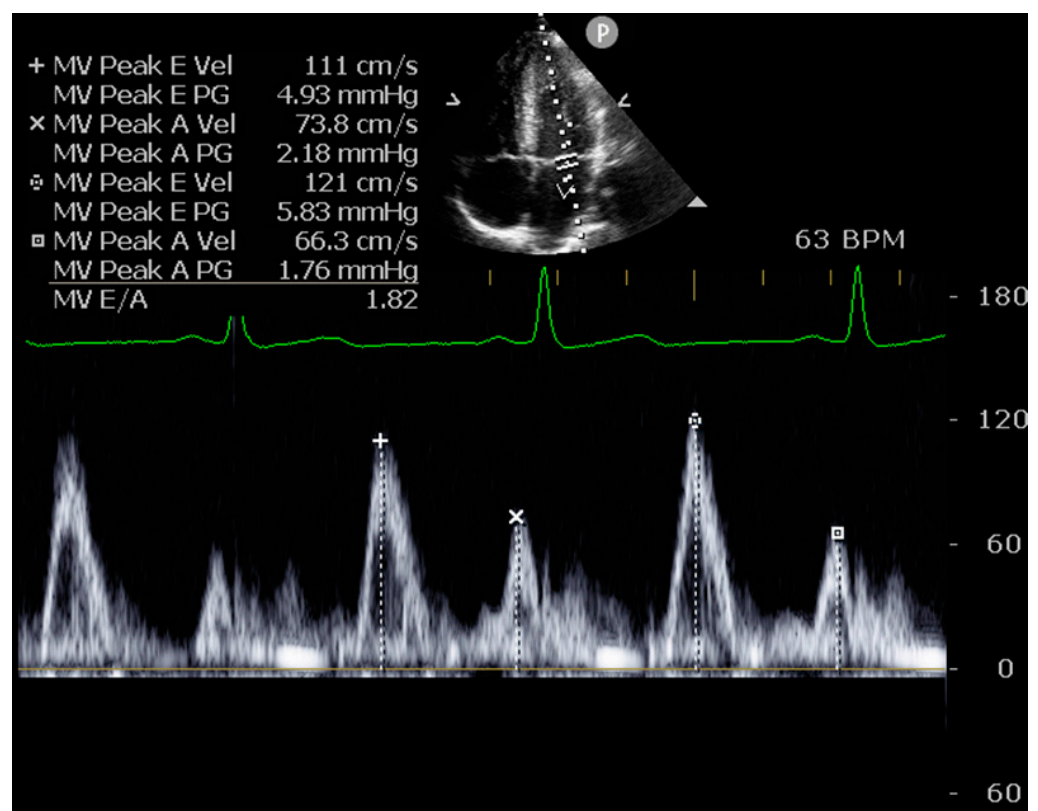


Figure 3. Trans-mitral valve pulse wave Doppler waveform, with E wave and A wave measurements.

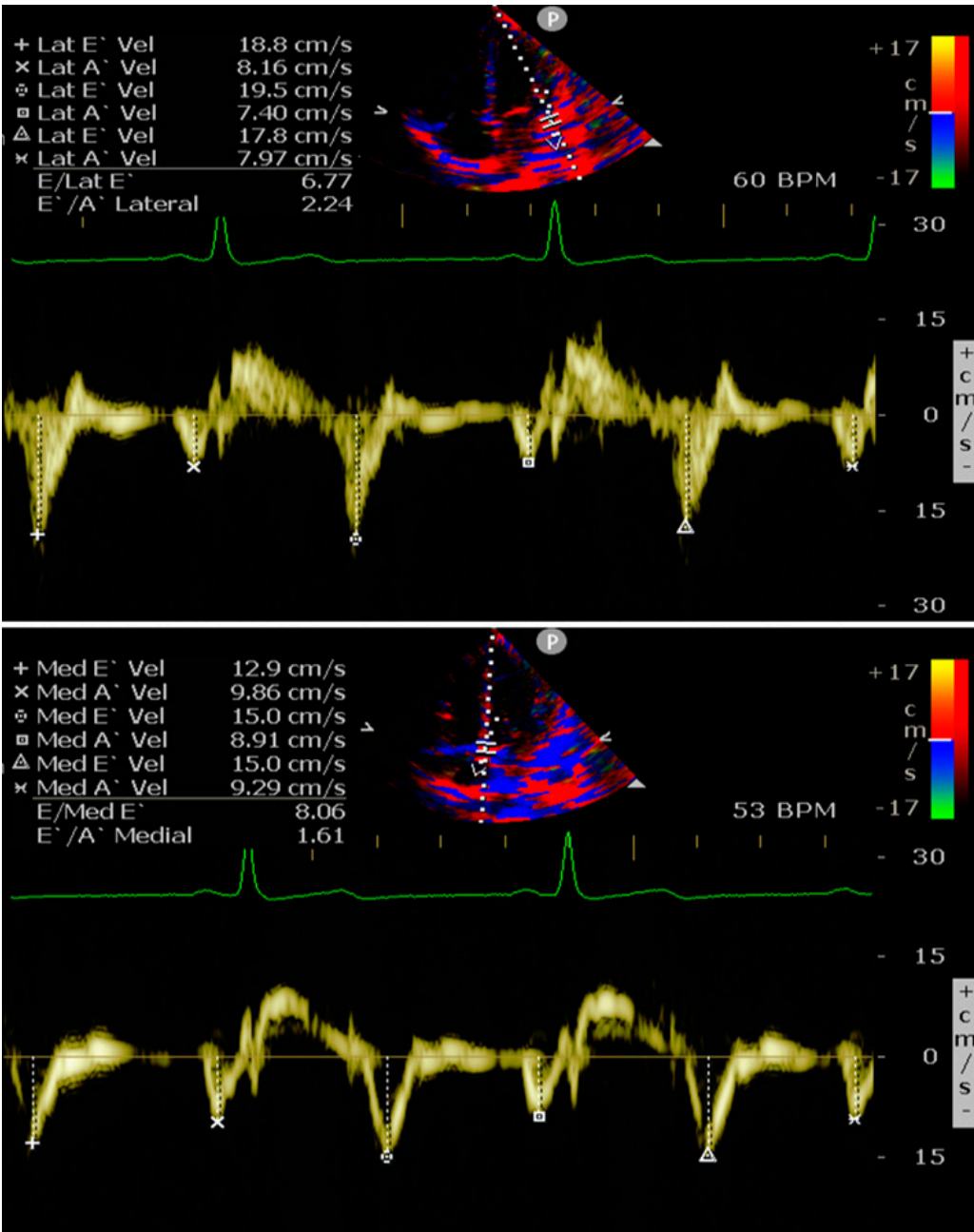


Figure 4. Medial and lateral mitral annular waveforms with measurements.

Table 4. Right ventricular parameters of study participants.

Parameters	Mean ± SD	Range (Min–Max)	Kolmogorov–Smirnov <i>p</i> -Value
Structure			
RVIDd (mm)	29.3 ± 4.06	13.4–38.8	0.056
RVOTd (mm)	27.4 ± 4.48	14.9–39.7	0.880
Systolic function			
TAPSE (mm)	23.3 ± 4.16	12.7–35.5	0.547
Diastolic function			
TV E (cm/s)	0.627 ± 0.121	0.350–0.960	0.544
TV A (cm/s)	0.370 ± 0.0814	0.180–0.660	0.010
TV E/A	1.74 ± 0.409	1.02–3.63	<0.001

RVIDd—Right ventricular internal diameter in diastole; RVOTd—Right ventricular outflow tract diameter in diastole; TAPSE—Tricuspid annular plane systolic excursion; TV E—Peak early diastolic tricuspid inflow velocity; TV A—Peak late (atrial) diastolic tricuspid inflow velocity; TV E/A—Ratio of early to late diastolic tricuspid inflow velocities.

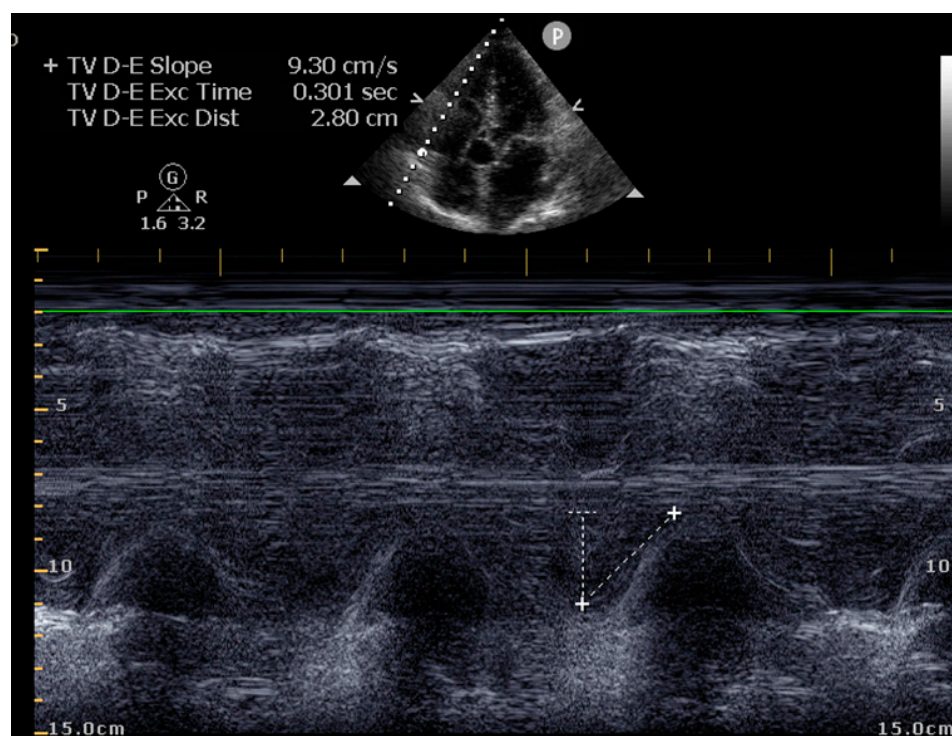


Figure 5. Echocardiographic image demonstrating measurement of tricuspid annular plane systolic excursion (TAPSE).

3.4. Comparison of Left Ventricular Structure Among Different Levels of Participants' Physical Activity

The Kruskal–Wallis and Welch's ANOVA test were used to compare left ventricular structure parameters among the different levels of physical activity. The Kruskal–Wallis test was used for non-parametric dimensions, while Welch's Anova was used for parametric dimensions (EDVs). The results reveal that there was a significant difference in IVSd, LVIDd, LVPWd, LVM, IVSs, ESV and EDV ($p < 0.05$) (see Table 5). Dwass–Steel–Critchlow–Fligner pairwise comparisons (non-parametric) and Games–Howell (parametric) post hoc tests were used to investigate the differences.

Table 5. Comparison of left ventricular structure among physical activity levels.

Parameters	1	2	3	4	χ^2	df	<i>p</i>
IVSd (mm)	8.17 ± 1.47	8.97 ± 1.47	8.41 ± 1.75	9.87 ± 1.61	31.54	3	<0.001
LVIDd (mm)	40.2 ± 3.58	41.8 ± 4.46	40.6 ± 8.49	43.6 ± 6.96	14.44	3	0.002
LVPWd (mm)	8.91 ± 1.60	9.61 ± 1.54	9.28 ± 1.68	10.1 ± 1.95	13.79	3	0.003
LVM (g)	47.1 ± 6.57	51.0 ± 6.48	48.8 ± 8.00	54.6 ± 7.45	31.64	3	<0.001
IVSs (mm)	13.5 ± 3.40	13.3 ± 2.80	13.9 ± 2.13	14.8 ± 4.64	8.87	3	0.031
LVIDs (mm)	26.2 ± 2.99	27.3 ± 3.69	26.3 ± 6.22	27.7 ± 6.29	6.67	3	0.083
LVPWs (mm)	14.3 ± 2.76	15.1 ± 2.48	14.8 ± 3.19	15.4 ± 2.19	4.16	3	0.245
ESV (mL)	25.6 ± 6.52	28.9 ± 8.73	27.7 ± 18.6	32.4 ± 12.8	9.51	3	0.023
EDV (mL)	69.7 ± 17.8	79.0 ± 19.8	79.1 ± 25.3	90.1 ± 24.8	-	3	<0.001 *

*: Welch's ANOVA test *p*-value; *p*-value < 0.05 shows statistical significance; bolded *p*-values show statistical significance; IVSd: Interventricular septum diameter in diastole; LVIDd: Left ventricular internal diameter in diastole; LVPWd: Left ventricular posterior wall in diastole; LVM: Left ventricular mass; IVSs: Interventricular septum in systole; LVIDs: Left ventricular internal diameter in systole; LVPWs: Left ventricular posterior wall in systole; EDV: End-diastolic volume; ESV: End-systolic volume; mL: milliliters.

This revealed that there was a significant difference in IVSd and LVM between those who engaged in vigorous exercise for more than an hour and the other physical activity

levels ($p < 0.05$). There was a significant difference in LVIDd, LVPWd, IVSs, ESV and EDV between those who engaged in vigorous exercise for more than an hour and those who did not really engage in exercise ($p < 0.05$). Also, there was a significant difference in EDV between those who engaged in vigorous exercise for more than an hour and those who engaged in less than 30 min of exercise ($p = 0.047$) (Table 6). A comparison of left ventricular structure parameters across physical activity levels per gender revealed that there was a significant difference in IVSd between males and females in physical activity level 4. It also revealed that males with a physical activity level of 4 had a significantly higher LVM compared to females ($p < 0.05$) (Table 7).

Table 6. Post hoc tests.

Parameter			W	<i>p</i>
IVSd (mm)	1	2	3.099	0.126
	1	3	0.209	0.999
	1	4	6.078	<0.001
	2	3	−2.332	0.351
	2	4	4.628	0.006
	3	4	5.901	<0.001
LVIDd (mm)	1	2	2.167	0.418
	1	3	1.595	0.673
	1	4	5.003	0.002
	2	3	−0.194	0.999
	2	4	3.035	0.139
	3	4	2.871	0.177
LVPWd (mm)	1	2	3.18	0.110
	1	3	1.64	0.653
	1	4	4.59	0.006
	2	3	−1.79	0.586
	2	4	1.77	0.595
	3	4	3.32	0.087
LVM (g)	1	2	3.465	0.068
	1	3	0.869	0.928
	1	4	6.3	<0.001
	2	3	−2.883	0.174
	2	4	4.035	0.022
	3	4	5.838	<0.001
IVSs (mm)	1	2	0.894	0.922
	1	3	1.738	0.609
	1	4	3.636	0.050
	2	3	0.936	0.912
	2	4	3.253	0.098
	3	4	1.349	0.776
ESV (mL)	1	2	1.872	0.548
	1	3	−0.374	0.994
	1	4	3.788	0.037
	2	3	−1.906	0.533
	2	4	1.515	0.707
	3	4	3.183	0.110

Table 6. Cont.

Parameter			W	p
EDV (mL)	1	2	.	0.184 *
	1	3	.	0.368 *
	1	4	.	<0.001 *
	2	3	.	1.000 *
	2	4	.	0.047 *
	3	4	.	0.201 *

*: Games–Howell *p*-value; *p*-value < 0.05 shows statistical significance; bolded *p*-values show statistical significance; W: sum of squares; 4: Vigorous exercise for more than an hour; 3: 30 min to an hour of moderate exercise; 2: Less than 30 min of exercise; 1: Do not really engage in exercise; IVSd: Interventricular septum diameter in diastole; LVIDd: Left ventricular internal diameter in diastole; LVPWd: Left ventricular posterior wall in diastole; LVM: Left ventricular mass; IVSs: Interventricular septum in systole; EDV: End-diastolic volume; ESV: End-systolic volume.

Table 7. Comparison of left ventricular structure among physical activity levels per gender.

Parameters	1			2			3			4		
	Female	Male	<i>p</i>	Female	Male	<i>p</i>	Female	Male	<i>p</i>	Female	Male	<i>p</i>
IVSd (mm)	7.89 ± 1.42	8.69 ± 1.51	0.171	9.03 ± 1.54	8.92 ± 1.45	0.809	8.09 ± 1.10	8.80 ± 2.30	0.287	9.27 ± 1.39	10.10 ± 1.64	0.037
LVIDd (mm)	40.56 ± 2.43	39.62 ± 5.24	0.513	40.38 ± 3.77	43.06 ± 4.72	0.055	39.50 ± 4.22	41.88 ± 11.93	0.462	41.59 ± 4.74	44.50 ± 7.57	0.101
LVPWd (mm)	8.73 ± 1.45	9.25 ± 1.89	0.418	9.38 ± 1.99	9.80 ± 1.01	0.383	9.05 ± 0.86	9.56 ± 2.35	0.424	9.66 ± 2.13	10.20 ± 1.86	0.265
LVM (g)	45.99 ± 6.10	49.18 ± 47.04	0.220	50.43 ± 7.51	51.47 ± 5.58	0.616	47.18 ± 3.07	50.45 ± 11.63	0.288	51.85 ± 6.79	55.60 ± 7.60	0.047
IVSs (mm)	11.43 ± 1.02	11.91 ± 1.03	0.238	13.67 ± 2.23	12.99 ± 3.23	0.444	13.34 ± 1.76	14.57 ± 2.41	0.124	13.74 ± 1.33	15.30 ± 5.41	0.199
LVIDs (mm)	26.36 ± 1.94	25.76 ± 4.49	0.615	25.73 ± 3.51	28.74 ± 3.30	0.007	23.62 ± 4.25	29.58 ± 6.82	0.008	25.30 ± 6.16	28.70 ± 6.13	0.033
LVPWs (mm)	13.77 ± 2.65	15.37 ± 2.80	0.142	14.89 ± 2.94	15.28 ± 2.07	0.625	13.75 ± 2.52	16.03 ± 3.56	0.053	14.22 ± 1.97	16.00 ± 2.08	0.001
ESV (mL)	25.68 ± 4.69	25.38 ± 9.38	0.909	25.08 ± 7.61	32.15 ± 8.44	0.008	20.38 ± 8.59	36.66 ± 23.62	0.016	27.20 ± 9.98	34.60 ± 13.26	0.021
EDV (mL)	72.72 ± 10.35	64.09 ± 26.86	0.281	72.38 ± 15.91	84.73 ± 21.43	0.073	69.02 ± 16.75	91.61 ± 28.86	0.028	78.17 ± 19.82	95.10 ± 25.13	0.006

p-value < 0.05 shows statistical significance; bolded *p*-values show statistical significance; IVSd: Interventricular septum diameter in diastole; LVIDd: Left ventricular internal diameter in diastole; LVPWd: Left ventricular posterior wall in diastole; LVM: Left ventricular mass; IVSs: Interventricular septum in systole; LVIDs: Left ventricular internal diameter in systole; LVPWs: Left ventricular posterior wall in systole; EDV: End-diastolic volume; ESV: End-systolic volume; mL: milliliters.

3.5. Comparison of Left Ventricular Function Among Different Levels of Participants' Physical Activity

The Kruskal–Wallis and Fisher's ANOVA test were used to compare left ventricular function parameters among the different levels of physical activity. The Kruskal–Wallis test was used for non-parametric dimensions, while Welch's Anova was used for parametric dimensions. The results reveal that there was a significant difference in SV, CO, CI, and deceleration time among physical activity levels (*p* < 0.05). All other dimensions revealed no significant difference among physical activity levels (Table 8).

The Dwass–Steel–Critchlow–Fligner pairwise comparison post hoc test (non-parametric) was used to investigate differences among physical activity levels. It revealed that there was a significant difference in SV, CO, and CI between those who engaged in vigorous exercise for more than an hour and those who did not really engage in exercise (*p* < 0.05) (Table 9). After comparing parameters per gender, there was a significant difference in SV between males and females with physical activity levels of 3 and 4. There was a significant difference in E' / A' between males and females with physical activities 2 and 3 (see Table 10).

Table 8. Comparison of left ventricular function among physical activity levels.

Parameter	1	2	3	4	χ^2	df	p
Systolic							
SV (mL)	46.3 ± 10.4	50.4 ± 14.1	55.6 ± 16.9	57.6 ± 16.7	14.277	3	0.003
FS (%)	35.0 ± 4.58	34.6 ± 5.55	38.7 ± 7.46	36.1 ± 8.30	6.033	3	0.110
CO (L/min)	3.05 ± 0.70	3.40 ± 1.03	3.54 ± 1.16	3.83 ± 1.17	9.674	3	0.022
CI (L/min/m ²)	1.77 ± 0.37	1.88 ± 0.66	2.09 ± 0.53	2.19 ± 0.66	10.442	3	0.015
EF (%)	58.3 ± 5.34	59.0 ± 5.39	60.0 ± 6.33	59.3 ± 4.86	-	3	0.707 *
Diastolic							
E wave (cm/s)	0.85 ± 0.11	0.80 ± 0.14	0.84 ± 0.17	0.79 ± 0.18	4.773	3	0.189
E/A ratio	1.72 ± 0.33	1.80 ± 0.39	1.79 ± 0.54	1.69 ± 0.45	2.549	3	0.467
Dec T (s)	236 ± 106	182 ± 61.5	182 ± 41.1	205 ± 66.0	8.602	3	0.035
E' (cm/s)	0.16 ± 0.02	0.17 ± 0.03	0.17 ± 0.03	0.17 ± 0.03	0.934	3	0.817
A' (cm/s)	0.08 ± 0.02	0.08 ± 0.02	0.09 ± 0.02	0.12 ± 0.26	6.237	3	0.101
E/E'	5.32 ± 0.81	4.88 ± 0.87	5.04 ± 0.97	4.80 ± 0.99	6.487	3	0.090
E'/A'	2.09 ± 0.66	2.11 ± 0.60	1.89 ± 0.56	1.98 ± 0.57	2.594	3	0.459

*: Fisher's ANOVA test *p*-value; *p*-value < 0.05 shows statistical significance; bolded *p*-values show statistical significance; SV—Stroke volume; EF—Ejection fraction (Simpson's method); FS—Fractional shortening; CO—Cardiac output; CI—Cardiac index; E—Peak early diastolic mitral inflow velocity; A—Peak late (atrial) diastolic mitral inflow velocity; E/A ratio—Ratio of early to late diastolic mitral inflow velocities; Dec T—Deceleration time of the E wave; E'—Peak early diastolic mitral annular velocity; A'—Peak late diastolic mitral annular velocity; E/E'—Ratio of early mitral inflow to annular velocity; E'/A'—Ratio of early to late diastolic mitral annular velocities.

Table 9. Dwass–Steel–Critchlow–Fligner pairwise comparisons.

Parameter			W	p
SV (mL)	1	2	1.75	0.601
	1	3	3.39	0.078
	1	4	5.01	0.002
	2	3	1.55	0.692
	2	4	3.18	0.110
	3	4	1.03	0.886
CO (L/min)	1	2	1.555	0.690
	1	3	2.019	0.482
	1	4	4.176	0.017
	2	3	0.41	0.992
	2	4	2.473	0.299
	3	4	1.937	0.519
CI (L/min/m ²)	1	2	1.17	0.843
	1	3	2.81	0.193
	1	4	4.13	0.018
	2	3	1.55	0.690
	2	4	2.93	0.163
	3	4	1.07	0.876
Dec T (s)	1	2	−2.392	0.328
	1	3	−2.815	0.192
	1	4	−0.38	0.993
	2	3	−0.779	0.946
	2	4	2.678	0.231
	3	4	3.405	0.076

p-value < 0.05 shows statistical significance; bolded *p*-values show statistical significance; W: sum of squares; SV—Stroke volume; CO—Cardiac output; CI—Cardiac index; Dec T—Deceleration time of the E wave.

Table 10. Comparison of left ventricular function among physical activity levels per gender.

Parameter	1			2			3			4		
Systolic	Female	Male	<i>p</i>	Female	Male	<i>p</i>	Female	Male	<i>p</i>	Female	Male	<i>p</i>
SV (mL)	47.13 ± 8.63	44.68 ± 13.51	0.556	47.38 ± 12.40	53.06 ± 15.22	0.202	48.54 ± 12.97	54.22 ± 17.56	0.010	50.97 ± 12.66	60.37 ± 17.50	0.026
FS (%)	34.97 ± 4.33	35.03 ± 5.28	0.976	35.71 ± 6.11	33.69 ± 4.96	0.249	40.22 ± 7.77	36.83 ± 6.88	0.230	35.97 ± 5.16	36.19 ± 9.34	0.916
CO (L/min)	3.08 ± 0.58	3.01 ± 1.04	0.826	3.34 ± 0.94	3.38 ± 1.10	0.903	3.08 ± 0.75	4.47 ± 1.52	0.006	3.61 ± 0.87	3.81 ± 1.16	0.489
CI (L/min/m ²)	1.78 ± 0.30	1.76 ± 0.55	0.920	1.88 ± 0.45	1.94 ± 0.67	0.762	1.97 ± 0.45	2.43 ± 0.69	0.055	2.17 ± 0.50	2.12 ± 0.63	0.763
EF (%)	57.09 ± 4.23	60.32 ± 6.59	0.311	59.06 ± 4.97	59.02 ± 5.82	0.890	62.52 ± 6.44	56.92 ± 4.79	0.030	58.42 ± 4.19	59.63 ± 5.12	0.277
Diastolic												
E wave (cm/s)	0.87 ± 0.10	0.82 ± 0.13	0.200	0.79 ± 0.13	0.81 ± 0.15	0.565	0.88 ± 0.18	0.79 ± 0.15	0.153	0.78 ± 0.16	0.80 ± 0.18	0.786
E/A ratio	1.73 ± 0.28	1.69 ± 0.43	0.749	1.61 ± 0.31	1.97 ± 0.38	0.002	1.97 ± 0.62	1.55 ± 0.27	0.038	1.60 ± 0.42	1.73 ± 0.46	0.257
Dec T (s)	247.57 ± 124.88	218.80 ± 73.77	0.523	194.25 ± 49.21	172.36 ± 69.57	0.295	180.81 ± 36.26	183.00 ± 48.56	0.892	200.32 ± 52.77	206.54 ± 71.34	0.714
E' (cm/s)	0.17 ± 0.02	0.16 ± 0.02	0.205	0.16 ± 0.03	0.17 ± 0.03	0.121	0.18 ± 0.03	0.16 ± 0.03	0.191	0.17 ± 0.03	0.16 ± 0.03	0.457
A' (cm/s)	0.09 ± 0.02	0.08 ± 0.02	0.628	0.09 ± 0.02	0.08 ± 0.01	0.002	0.09 ± 0.02	0.10 ± 0.02	0.004	0.09 ± 0.02	0.13 ± 0.31	0.518
E/E'	5.34 ± 0.86	5.30 ± 0.76	0.906	5.01 ± 0.99	4.76 ± 0.74	0.353	5.02 ± 0.82	5.05 ± 1.19	0.938	4.64 ± 0.79	4.87 ± 1.07	0.384
E'/A'	2.08 ± 0.66	2.09 ± 0.69	0.962	1.81 ± 0.49	2.38 ± 0.57	0.001	2.15 ± 0.54	1.58 ± 0.42	0.005	2.06 ± 0.54	1.95 ± 0.58	0.472

p-value < 0.05 shows statistical significance; bolded *p*-values show statistical significance; SV—Stroke volume; EF—Ejection fraction (Simpson's method); FS—Fractional shortening; CO—Cardiac output; CI—Cardiac index; E—Peak early diastolic mitral inflow velocity; A—Peak late (atrial) diastolic mitral inflow velocity; E/A ratio—Ratio of early to late diastolic mitral inflow velocities; Dec T—Deceleration time of the E wave; E'—Peak early diastolic mitral annular velocity; A'—Peak late diastolic mitral annular velocity; E/E'—Ratio of early mitral inflow to annular velocity; E'/A'—Ratio of early to late diastolic mitral annular velocities.

3.6. Comparison of Right Ventricular Dimensions Among Different Levels of Participants' Physical Activity

The Kruskal–Wallis and ANOVA tests were used to compare right ventricular parameters among the different levels of physical activity. The Kruskal–Wallis test was used for non-parametric dimensions, while the ANOVA test was used for parametric dimensions. There was a significant difference in RVOTd and TVA among different physical activity levels ($p < 0.05$) (see Table 11). Table 12 reveals that there is a significant difference in RVIDd, RVOTd and TV E/A between males and females with physical activity levels of 3 and 4.

Table 11. Comparison of right ventricular dimensions among physical activity levels.

	1	2	3	4	F	df1	df2	<i>p</i>
RVIDd (mm)	29.8 ± 3.89	28.3 ± 4.34	28.4 ± 3.30	29.9 ± 4.16	1.98	3	168	0.119 *
RVOTd (mm)	28.4 ± 3.36	26.3 ± 3.35	25.8 ± 5.33	28.1 ± 4.82	3.37	3	169	0.014 *
TAPSE (cm)	24.6 ± 3.74	23.5 ± 3.53	22.9 ± 3.77	22.9 ± 4.69	1.3	3	156	0.275 *
TV E (cm/s)	0.65 ± 0.09	0.60 ± 0.11	0.67 ± 0.12	0.62 ± 0.14	1.98	3	161	0.119 *
TV A (cm/s)	0.40 ± 0.08	0.36 ± 0.08	0.38 ± 0.10	0.36 ± 0.08	-	3	-	0.038
TV E/A	1.68 ± 0.45	1.73 ± 0.37	1.80 ± 0.38	1.75 ± 0.43	-	3	-	0.302

*: ANOVA test *p*-value; *p*-value < 0.05 shows statistical significance; bolded *p*-values show statistical significance; RVIDd—Right ventricular internal diameter in diastole; RVOTd—Right ventricular outflow tract diameter in diastole; TAPSE—Tricuspid annular plane systolic excursion; TV E—Peak early diastolic tricuspid inflow velocity; TV A—Peak late (atrial) diastolic tricuspid inflow velocity; TV E/A—Ratio of early to late diastolic tricuspid inflow velocities.

Table 12. Comparison of right ventricular dimensions among physical activity levels per gender.

Parameters	1			2			3			4		
	Female	Male	<i>p</i>	Female	Male	<i>p</i>	Female	Male	<i>p</i>	Female	Male	<i>p</i>
RVIDd (mm)	29.80 ± 3.56	29.77 ± 4.65	0.696	28.57 ± 3.42	28.15 ± 5.08	0.875	27.26 ± 2.10	29.83 ± 4.02	0.048	28.20 ± 3.53	30.68 ± 4.21	0.015
RVOTd (mm)	27.99 ± 3.23	29.29 ± 3.61	0.613	25.60 ± 3.23	26.91 ± 3.41	0.278	24.00 ± 5.27	28.01 ± 4.68	0.033	26.30 ± 4.64	28.86 ± 4.74	0.024

Table 12. Cont.

Parameters	1			2			3			4		
	Female	Male	<i>p</i>	Female	Male	<i>p</i>	Female	Male	<i>p</i>	Female	Male	<i>p</i>
TAPSE (mm)	25.16 ± 3.87	23.30 ± 3.26	0.175	24.96 ± 3.01	22.22 ± 3.53	0.020	22.32 ± 3.18	23.42 ± 4.39	0.865	23.31 ± 3.88	22.67 ± 5.00	0.515
TV E (cm/s)	0.65 ± 0.10	0.65 ± 0.09	0.701	0.58 ± 0.10	0.62 ± 0.11	0.294	0.68 ± 0.12	0.65 ± 0.12	0.381	0.61 ± 0.13	0.62 ± 0.14	0.869
TV A (cm/s)	0.41 ± 0.09	0.39 ± 0.07	0.568	0.34 ± 0.08	0.37 ± 0.07	0.170	0.37 ± 0.11	0.40 ± 0.08	0.395	0.39 ± 0.08	0.35 ± 0.07	0.032
TV E/A	1.67 ± 0.52	1.69 ± 0.29	0.935	1.77 ± 0.35	1.71 ± 0.38	0.603	1.94 ± 0.41	1.64 ± 0.25	0.038	1.60 ± 0.29	1.82 ± 0.47	0.050

p-value < 0.05 shows statistical significance; bolded *p*-values show statistical significance; RVIDd—Right ventricular internal diameter in diastole; RVOTd—Right ventricular outflow tract diameter in diastole; TAPSE—Tricuspid annular plane systolic excursion; TV E—Peak early diastolic tricuspid inflow velocity; TV A—Peak late (atrial) diastolic tricuspid inflow velocity; TV E/A—Ratio of early to late diastolic tricuspid inflow velocities.

3.7. Association Between Participants' Characteristics and Cardiac Dimensions

A correlation matrix between participants' characteristics and cardiac dimensions was performed. There was a weak negative correlation between age and FS, CO, CI, E, E/A, E', and E'/A'. Also, there was a weak positive correlation between height and LVIDd, LVM, IVSs, LVIDs, LVPWs, ESV, SV, and CO ($p < 0.05$). Additionally, there was a weak positive correlation between weight and IVSd, LVIDd, LVPWd, LVM, LVIDs, LVPWs, ESV, SV, and CO ($p < 0.05$). There was a weak positive correlation between systolic BP and IVSd, LVIDd, LVPWd, LVM, LVIDs, LVPWs, ESV, EDV, SV, CO, and LAA ($p < 0.05$). There was also a weak positive correlation between HR and LVIDd, LVIDs, ESV, EDV, SV, CO, CI, A, E/A and RVOTd ($p < 0.05$) (see the Supplementary Material).

3.8. Factors Influencing Cardiac Dimensions of Study Participants

Univariate and multivariate linear regression was performed to assess the influence participants' characteristics had on their cardiac dimensions. Both the univariate and multivariate analysis revealed that there was a significant association between age and E/A, E' and E'/A'. Both univariate and multivariate linear regression showed a significant association between BSA and IVSd, LVIDd, LVIDs, LVPWs, ESV, SV, CO and LAA. Additionally, there was a significant association between systolic BP and LVPWd, LVM, LVIDs, LVPWs, ESV and LAsd on both the univariate and multivariate analysis (see the Supplementary Material).

3.9. Association Between Level of Physical Activity and ECG Parameters

A chi-square test of association was performed to assess the association between the various levels of physical activity and the categorized ECG parameters of all study participants. The test revealed that there was no significant association between any of the parameters and participants' level of physical activity ($p > 0.05$) (see Table 13). However, there was a significant association between bradycardia and level of physical activity, with participants who engaged in physical activity for more than an hour recording the highest number of bradycardia events.

An ANOVA and Kruskal–Wallis test was used to compare the PR interval and QRS duration among the levels of physical activity. The test revealed that there was a significant association between PR interval and level of physical activity (Table 14). A further analysis showed that participants who engaged in physical activity for 30 min or more had a longer PR interval compared to those who engaged in physical activity for less than 30 min. Additionally, participants who engaged in physical activity for 30 min or more had a longer QRS duration compared to those who engaged in physical activity for less than 30 min (see Table 15).

Table 13. Association between level of physical activity and ECG findings.

	Level of Physical Activity				<i>p</i> -Value
	1	2	3	4	
Rhythm					0.391
Bradycardia	4 (18.2)	10 (29.4)	4 (16.0)	24 (35.8)	0.041 *
Normal	18 (81.8)	24 (70.6)	21 (84.0)	42 (62.7)	
Tachycardia	0 (0.0)	0 (0.0)	0 (0.0)	1 (1.5)	
LVH (Sokolow–Lyon)					0.223
Normal	21 (95.5)	34 (100.0)	24 (96.0)	67 (100.0)	
Hypertrophy	1 (4.5)	0 (0.0)	1 (4.0)	0 (0.0)	
LVH (R in aVL)					0.175
Normal	22 (100.0)	34 (100.0)	24 (96.0)	67 (100.0)	
Hypertrophy	0 (0.0)	0 (0.0)	1 (4.0)	0 (0.0)	
LAE					0.109
No	21 (100.0)	32 (100.0)	24 (100.0)	61 (92.4)	
Yes	0 (0.0)	0 (0.0)	0 (0.0)	5 (7.6)	
RAE					0.704
No	21 (100.0)	31 (96.9)	24 (100.0)	64 (97.0)	
Yes	0 (0.0)	1 (3.1)	0 (0.0)	2 (3.0)	
Conducting defect					0.564
Right bundle branch block	0 (0.0)	0 (0.0)	1 (4.0)	1 (1.5)	
First degree AV block	0 (0.0)	2 (5.9)	0 (0.0)	6 (9.0)	
Normal	22 (100.0)	32 (94.1)	24 (96.0)	59 (88.1)	
Short PR interval bundle branch block	0 (0.0)	0 (0.0)	0 (0.0)	1 (1.5)	
S/ST changes					0.348
Benign early repolarization	0 (0.0)	5 (15.6)	2 (8.3)	13 (19.7)	
No significant changes	20 (95.2)	24 (75.0)	22 (91.7)	49 (74.2)	
Non-specific anteroseptal T wave inversions in V2, V3	0 (0.0)	1 (3.1)	0 (0.0)	0 (0.0)	
Non-specific inferior T wave abnormalities in III, aVF	0 (0.0)	2 (6.3)	0 (0.0)	1 (1.5)	
Non-specific inferolateral T wave abnormalities in I, II, III, aVL, aVF, V5, V6	0 (0.0)	0 (0.0)	0 (0.0)	1 (1.5)	
Non-specific lateral T wave abnormalities in I, aVL	1 (4.8)	0 (0.0)	0 (0.0)	0 (0.0)	
Non-specific inferolateral and anteroseptal T wave inversions in all leads	0 (0.0)	0 (0.0)	0 (0.0)	1 (1.5)	
Tall T waves in V2, V3 with benign early repolarization	0 (0.0)	0 (0.0)	0 (0.0)	1 (1.5)	

*: Kruskal–Wallis test *p*-value; *p*-value < 0.05 shows statistical significance; bolded *p*-values show statistical significance; 4: Vigorous exercise for more than an hour; 3: 30 min to an hour of moderate exercise; 2: Less than 30 min of exercise; 1: Do not really engage in exercise; LVH: left ventricular hypertrophy; LAE: Left atrial enlargement; right atrial enlargement.

Table 14. Comparison of PR interval and QRS duration among levels of physical activity.

	F	df1	df2	p
Heart rate		3		0.447
PR interval	3.05	3	144	0.031
QRS duration		3		0.218

p-value < 0.05 shows statistical significance; bolded *p*-values show statistical significance.

Table 15. Mean PR interval and QRS duration among physical activity levels.

PR Interval	Mean ± SD
1 and 2	162 ± 21.8
3 and 4	166 ± 23.2
QRS duration	
1 and 2	85.9 ± 8.41
3 and 4	89.5 ± 12.3

4: Vigorous exercise for more than an hour; 3: 30 min to an hour of moderate exercise; 2: Less than 30 min of exercise; 1: Do not really engage in exercise.

4. Discussion

4.1. Summary of Principal Findings

This study examined the effect of physical activity on cardiac structure and function in 174 African university students with a mean age of 22.3 years. The main findings were that higher physical activity, especially the practice of more than one hour of active exercise daily, was associated with larger left ventricular chamber dimensions and greater ventricular wall thickness, including higher IVSd, LVIDd, LVPWd, LVM, IVSs, ESV, and EDV. However, conventional indices of left ventricular systolic and diastolic function remained largely preserved across activity categories, with no significant differences in EF or E/A ratio. Ventricular wall thickness findings in this cohort also appear modest and likely physiological. The overall mean IVSd was 9.13 mm and mean LVPWd was 9.63 mm, increasing to 9.87 mm and 10.1 mm respectively in the vigorous activity group. Right-sided adaptation was less marked. Taken together, these findings are most consistent with activity-related cardiac remodeling rather than overt pathological change in this young adult population. On ECG there were higher rates of bradycardia, repolarization and longer PR interval in the most physically active cohort.

The observed dose–response pattern, especially between lowest versus highest physical activity levels, supports the concept that training load influences cardiac remodeling.

4.2. Comparison with Other Studies

The larger ventricular wall thickness and chamber size in the most physically active group, compared with those with lower activity levels, align closely with patterns reported in previous studies. Engvall et al. [15] reported that moderately trained athletes exhibited a significantly increased LV wall thickness and chamber dimensions compared with sedentary peers yet preserved global systolic and diastolic function. The stepwise remodeling pattern with rising physical activity level supports the concept of a dose–response relationship between exercise intensity and cardiac adaptation.

Similarly, Corsi et al. [10] highlighted in their systematic review that left ventricular hypertrophy and repolarization variants are common physiologic adaptations among highly trained athletes, particularly those of African ancestry. The present findings extend this evidence by demonstrating comparable remodeling trends within a non-elite but ethnically homogeneous Ghanaian university cohort. However, the magnitude of LV wall

thickening in our population was modest (mean IVSd $\approx$ 9.87 mm), well below the upper physiologic limit (>12 mm) reported in elite athletes.

In contrast to some earlier ethnic-based interpretations of “Black athlete’s heart,” our results reinforce the growing consensus that geographic ancestry and physical-training exposure, rather than ethnicity per se, better explain observed variations in cardiac morphology.

The absence of disproportionate wall thickening in our participants underscores that physiologic remodeling in African populations follows the same adaptive continuum described in the global sports cardiology literature. To the best of our knowledge, this study is the first of its kind that compares cardiac remodeling effects on different levels of physical activity in a young adult population that includes non-professional athletes. Ngabea et al. [16], Pambo et al. [17] and Bibou et al. [18] conducted their study among professional footballers in Nigeria, Ghana, and Cameroon respectively, and they all found physiologically increased left ventricular hypertrophy among these professional athletes using electrocardiogram and echocardiogram.

The ECG finding on the prevalence of sinus bradycardia in higher levels of physical activity confirms a common finding among athletes. Some studies attribute sinus bradycardia to a change in cardiac autonomic balance, while others attribute it to a reduction in the sinus node firing frequency [19], but it is generally considered to be multifactorial [20]. The higher rate of repolarization among those who exercise more confirms previous studies in black athletes. For instance, Haïssaguerre et al. [21] reported that 13% of black athletes showed early repolarization compared to 4% of black sedentary controls. Our findings also affirm those of Rawlins et al. [22], who reported greater left ventricular hypertrophy and higher prevalence of repolarization changes in black female athletes. Again, the longer PR interval among higher-level athletes compared to the less active groups agrees with Abela et al. [23], who noted a similar finding in young athletes.

4.3. Relevance to Clinical Practice

From a clinical standpoint, these findings have several implications. First, the observed echocardiographic reference ranges among physically active African young adults can inform context-specific normal limits, reducing misclassification of physiologic hypertrophy as cardiomyopathy during pre-participation or occupational screening. Second, recognition that even moderate physical activity promotes favorable cardiac remodeling supports public health recommendations advocating regular exercise for cardiovascular health. Finally, our results highlight the importance of interpreting echocardiographic findings using fitness-level-adjusted criteria rather than rigid ethnic-based norms, consistent with the equity-focused recommendations proposed by Corsi et al. [10]. The findings of this study suggest that in highly active young adults, mild increases in wall thickness or mass should not be interpreted in isolation but alongside chamber size, functional indices, and overall training exposure. By contrast, in less active individuals, echocardiographic hypertrophy should raise concern for grey-zone or pathological remodeling, especially when chamber sizes are normal, and prompt further evaluation. Nevertheless, clinical context, including patient symptoms, family history, ECG findings and physical activity level, should be considered alongside wall-thickness thresholds when assessing possible pathological hypertrophy in African cohorts.

4.4. Strengths and Limitations

The strength of the present study is that it adds useful data from an underrepresented African population and suggests that modest increases in chamber size and wall thickness with increasing activity can occur, while conventional ventricular function remains normal. Nevertheless, some limitations merit consideration. Physical activity was primarily self-

reported, introducing possible recall bias. Underestimation or overestimation of physical activity can influence the observed associations between physical activity and cardiac remodeling. However, if more active participants systematically overreported activity, or if some participants were incorrectly classified into higher activity categories, the association between physical activity and cardiac remodeling may have been exaggerated or distorted. It is also worth stating that participants were classified by overall activity level rather than by specific exercise modality. Although exercise type may influence cardiac adaptation, many participants in higher-level activity engaged in multiple exercise types, whereas those in the lower activity groups often reported irregular or no structured activity. Another potential limitation is lifestyle differences. Although smoking was part of the exclusion criteria, some lifestyle exposures, such as dietary patterns, could not be measured and could potentially affect cardiac dimensions. Lastly, the unequal number of participants across activity categories is a limitation of this study. As this was a cross-sectional study, participants were categorized according to reported activity level rather than recruited into equal-sized strata. Level 4 contained the largest number of athlete participants, while levels 1 and 2 were largely composed of non-athletic or minimally active individuals and, when combined, were comparable in size to level 4. The level 3 category represented intermediate activity, with the highest potential risk of misclassification bias. Unequal group sizes may influence the precision of estimates and the power to detect differences between groups, particularly for smaller categories. Therefore, the findings should be interpreted in the context of these limitations.

4.5. Implications for Future Research

Future studies should employ longitudinal and experimental study designs to monitor the temporal evolution of cardiac remodeling with sustained training and detraining. Incorporation of advanced echocardiographic parameters, such as global longitudinal strain and three-dimensional LV volumes, would enhance sensitivity to subtle functional changes. Comparative analyses involving elite and recreational athletes from different African regions could further elucidate the relative influence of genetic background, training type, and environmental factors on cardiac adaptation.

5. Conclusions

Higher physical activity levels among young African university students were associated with mild physiological cardiac remodeling, including modest increases in cardiac dimensions and output, with preserved systolic and diastolic function. These findings suggest that level of physical activity should be considered when interpreting echocardiographic measurements in young African adults, particularly when mild increases in ventricular wall thickness or chamber size are observed. ECG findings such as bradycardia, repolarization changes and longer PR intervals were more common among the most active participants and may reflect exercise-related adaptation. In resource-limited settings, incorporating physical activity history into cardiovascular screening may help distinguish physiological adaptation from potential pathology and guide appropriate use of echocardiography or referral.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/hearts7020019/s1>, Table S1: Correlation matrix showing the association between participants' characteristics and cardiac dimensions; Table S2: Linear regression.

Author Contributions: Y.A.W.: Conceptualization, Data curation, Writing—original draft, and Writing—review and editing. C.K.: Resources, Validation, Writing—original draft, and Writing—review and editing. G.M.A.: Investigation, Validation, Writing—original draft, and Writing—review

and editing. P.N.A.: Formal Analysis, Writing—original draft, and Writing—review and editing. E.O.A.B.: Data curation, Writing—original draft, and Writing—review and editing. I.K.O.: Resources, Supervision, Writing—original draft, and Writing—review and editing. All authors have read and agreed to the published version of the manuscript.

Funding: No specific funding was received for this work.

Institutional Review Board Statement: The study was reviewed and approved by the Committee for Human Research and Publication Ethics of the Kwame Nkrumah University of Science and Technology (CHRPE/AP/584/24) on 8 July 2024.

Informed Consent Statement: Written informed consent was obtained from all participants. The consent form for participation was distributed to all participants and signed.

Data Availability Statement: The data presented in this study are available on request from the corresponding author.

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

LV	Left ventricle
HCM	Hypertrophic cardiomyopathy
MRI	Magnetic resonance imaging
ECG	Electrocardiography
KNUST	Kwame Nkrumah University of Science and Technology
BMI	Body mass index
BSA	Body surface area
ASE	American society of echocardiography
EACVI	European association of cardiovascular imaging
SD	Standard deviation
AHA	American heart association
HR	Heart rate
aVL	Augmented vector left
TTE	Transthoracic echocardiography
IVSd	Interventricular septum (diastole)
IVSs	Interventricular septum (systole)
LVIDd	Left ventricular internal diameter (diastole)
LVIDs	Left ventricular internal diameter (systole)
LVPWd	Left ventricular posterior wall (diastole)
LVPWs	Left ventricular posterior wall (systole)
LVM	Left ventricular mass
RWT	Relative wall thickness
EF	Ejection fraction
SV	Stroke volume
CO	Cardiac output
CI	Cardiac index
E	Early diastolic filling wave
A	Late filling velocity
Dec T	Deceleration time
TDI	Tissue doppler imaging
E'	Early diastolic mitral annular velocity
A'	Late diastolic mitral annular velocity
RVIDd	Right ventricular internal diameter (RVIDd)

RVOTd	Right ventricular outflow tract (diastole)
TAPSE	Tricuspid annular plane systolic excursion
TV E	Peak early tricuspid inflow velocity
TV A	Late tricuspid inflow velocity
LAsd	Left atrial systolic diameter
LAA	Left atrial area
IBM	International Business Machines
SPSS	Statistical Package for the Social Sciences
ANOVA	Analysis of variance
STROBE	Strengthening the Reporting of Observational studies in Epidemiology
ESV	End-systolic volume
EDV	End-diastolic volume
FS	Fractional shortening

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