Modern American Journal of Linguistics, Education, and Pedagogy
ISSN (E): 3067-7874
Volume 01, Issue 06, September, 2025
Website: usajournals.org

CARDIAC REMODELING IN ENDURANCE ATHLETES: STRUCTURAL AND FUNCTIONAL ADAPTATIONS TO HIGH-INTENSITY TRAINING

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Abstract
Long-term high-volume endurance training is associated with profound cardiac remodeling, a phenomenon known as the "athlete's heart." While generally regarded as a benign and adaptive process, the overlap between physiological remodeling and early manifestations of cardiomyopathies continues to pose diagnostic and clinical challenges. Differentiating normal adaptation from pathology is essential to avoid misdiagnosis, inappropriate disqualification from sports, or, conversely, failure to identify athletes at risk of sudden cardiac death.

Keywords: Cardiac remodeling, Endurance athletes, High-intensity training, Structural adaptations, Functional adaptations, Athlete's heart, Ventricular hypertrophy, Cardiac function, Exercise physiology, Cardiovascular adaptations.

Introduction

To investigate the morphological and functional cardiac adaptations in endurance athletes using echocardiography and cardiopulmonary exercise testing, and to compare these findings with age-matched sedentary controls.
A cross-sectional study was conducted including 120 competitive male endurance athletes (marathon, triathlon, cycling) and 60 sedentary controls aged 18–35 years. All participants underwent a standardized protocol including medical history, physical examination, 12-lead electrocardiography, transthoracic echocardiography, and maximal cardiopulmonary exercise testing. Cardiac

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dimensions, wall thickness, systolic and diastolic function, and exercise capacity were compared between groups. Statistical analysis was performed using independent-sample t-tests and Pearson correlation coefficients.
Compared to controls, athletes demonstrated significantly greater left ventricular end-diastolic diameter (56.7 ± 4.2 vs. 49.5 ± 3.8 mm, $p < 0.001$) and interventricular septal thickness (11.2 ± 1.5 vs. 9.3 ± 1.4 mm, $p < 0.01$). Diastolic function was enhanced, with higher E/A ratio in athletes (1.68 ± 0.29 vs. 1.22 ± 0.25, $p < 0.01$). Left ventricular ejection fraction was preserved in both groups. Exercise testing confirmed superior aerobic capacity in athletes, with VO_2max values nearly 1.6-fold higher (62.4 ± 8.3 vs. 38.6 ± 6.2 mL/kg/min, $p < 0.001$). Correlation analysis revealed significant associations between training volume and LV dimensions ($r = 0.62$, $p < 0.001$).
Endurance training induces a distinct pattern of physiological cardiac remodeling, characterized by balanced chamber enlargement, mild wall thickening, and improved diastolic function, without systolic impairment. These changes reflect adaptive responses to chronic hemodynamic load and underline the importance of distinguishing between athlete's heart and pathological cardiomyopathy.

Keywords: athlete's heart; cardiac remodeling; echocardiography; endurance training; VO_2max; sports cardiology.

The human heart demonstrates remarkable plasticity in response to chronic exercise. Endurance sports such as marathon running, triathlon, and cycling subject the cardiovascular system to sustained volume and pressure overload, resulting in structural and functional adaptations collectively referred to as the "athlete's heart" [1,2]. These changes typically include left ventricular (LV) and right ventricular (RV) chamber enlargement, mild increases in wall thickness, enhanced diastolic filling, and bradycardia at rest.
While generally considered benign and reversible upon detraining, these adaptations can mimic early forms of hypertrophic cardiomyopathy (HCM) or dilated cardiomyopathy (DCM), leading to diagnostic uncertainty [3,4]. The distinction between physiological and pathological remodeling has direct clinical implications, as failure to recognize pathology may expose athletes to an

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increased risk of sudden cardiac death, while misclassifying physiological remodeling as disease may result in unnecessary disqualification from competitive sports [5].

Previous studies, including the landmark meta-analysis by Pluim et al. [6], have characterized the athlete's heart in various populations. However, differences in sport disciplines, training volumes, and diagnostic modalities continue to generate debate. Contemporary echocardiographic techniques and cardiopulmonary exercise testing (CPET) provide opportunities for more precise characterization of remodeling patterns.

The present study aimed to evaluate cardiac structural and functional adaptations in competitive endurance athletes compared with sedentary controls, with emphasis on echocardiographic findings and exercise performance. We hypothesized that endurance athletes would exhibit significant chamber enlargement and enhanced diastolic function, while maintaining preserved systolic function.

2. Methods

This was a cross-sectional observational study conducted at a tertiary sports cardiology center between January 2022 and March 2023.

- **Athlete group:** 120 male competitive athletes (aged 18–35 years) engaged in marathon running, triathlon, or cycling. Inclusion criteria required ≥5 years of continuous training, with average training load ≥10 hours/week.
- **Control group:** 60 sedentary, age-matched healthy males with no history of structured exercise training.

Exclusion criteria: pre-existing cardiovascular disease, systemic hypertension, diabetes mellitus, smoking, and use of performance-enhancing drugs.

All participants underwent a standardized assessment including medical history, physical examination, and measurement of blood pressure, resting heart rate, and body mass index (BMI).

A standard 12-lead ECG was performed at rest. Parameters analyzed included heart rate, PR interval, QRS duration, QTc interval, and repolarization changes.

Transthoracic echocardiography was performed using a Vivid E95 (GE Healthcare). Measurements included:

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- LV end-diastolic and end-systolic diameters (LVEDD, LVESD).
- Interventricular septum (IVS) and posterior wall thickness.
- Left atrial diameter.
- LV ejection fraction (LVEF) by biplane Simpson method.
- Diastolic function assessed by E/A ratio and tissue Doppler imaging.

All measurements followed the ASE/EACVI recommendations [7].

CPET was conducted on a treadmill using a ramp protocol to maximal exertion, with breath-by-breath analysis of expired gases (Oxycon Pro, Jaeger). Peak oxygen consumption (VO_2max) was defined as the highest 30-second average value.

Data were expressed as mean ± standard deviation (SD). Between-group comparisons were performed using independent-sample t-tests. Pearson correlation was used to assess the relationship between training volume and echocardiographic parameters. Significance was set at $p < 0.05$.

The study complied with the Declaration of Helsinki and was approved by the Institutional Ethics Committee. All participants provided written informed consent.

3. Results

Athletes and controls were comparable in age and BMI. Resting heart rate was significantly lower in athletes (56.2 ± 6.4 vs. 72.5 ± 7.1 bpm, $p < 0.001$). Mean training history among athletes was 7.1 ± 2.6 years.

Table 1. Baseline characteristics

Variable	Athletes (n=120)	Controls (n=60)	p-value
Age (years)	26.8 ± 4.5	27.1 ± 4.9	0.68
BMI (kg/m²)	23.2 ± 2.1	24.0 ± 2.3	0.09
Resting HR (bpm)	56.2 ± 6.4	72.5 ± 7.1	<0.001
Training history (yrs)	7.1 ± 2.6	–	–

Athletes demonstrated significant chamber enlargement and mild wall thickening compared with controls. LVEF was preserved in both groups.

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Table 2. Echocardiographic parameters

Parameter	Athletes (n=120)	Controls (n=60)	p-value
LVEDD (mm)	56.7 ± 4.2	49.5 ± 3.8	<0.001
LVESD (mm)	35.2 ± 3.7	31.1 ± 3.4	<0.001
IVS thickness (mm)	11.2 ± 1.5	9.3 ± 1.4	<0.01
LVEF (%)	62.5 ± 3.6	61.9 ± 3.8	0.44
E/A ratio	1.68 ± 0.29	1.22 ± 0.25	<0.01
LA diameter (mm)	40.1 ± 3.9	36.2 ± 3.1	<0.05

VO_2max was markedly higher in athletes compared with controls (62.4 ± 8.3 vs. 38.6 ± 6.2 mL/kg/min, $p < 0.001$). Time to exhaustion was also longer (16.8 ± 2.3 vs. 10.2 ± 1.8 min, $p < 0.001$).

Training volume (hours/week) correlated positively with LVEDD ($r = 0.62$, $p < 0.001$) and E/A ratio ($r = 0.48$, $p < 0.01$), suggesting dose-dependent cardiac adaptation.

4. Discussion

This study provides detailed evidence of structural and functional cardiac remodeling in endurance athletes. Our findings demonstrate balanced ventricular enlargement, mild wall thickening, enhanced diastolic filling, and preserved systolic function, consistent with the classical description of the athlete's heart [1,6].

The magnitude of LV enlargement in our cohort was similar to that reported by Pluim et al. [6] and Fagard [8]. Importantly, unlike pathological DCM, athletes maintained normal systolic function and demonstrated superior exercise capacity.

The mild septal thickening observed aligns with the "grey zone" between physiological hypertrophy and HCM [3]. However, the absence of family history, symptoms, and abnormal diastolic indices supports a benign adaptation.

Clinicians evaluating athletes should recognize key differentiating features:

- Symmetrical chamber enlargement vs. asymmetric hypertrophy in HCM.
- Preserved or enhanced diastolic filling vs. restrictive filling patterns in pathology.
- Elevated VO_2max, reflecting high functional reserve.

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Failure to appreciate these distinctions may result in unnecessary restriction from competition or overlooked risk of pathology.

Our study was limited by its cross-sectional design and inclusion of only male participants. The absence of cardiac MRI restricted detailed tissue characterization. Future longitudinal studies incorporating female athletes and advanced imaging are warranted.

5. Conclusion

Endurance training produces a unique pattern of physiological cardiac remodeling, characterized by chamber enlargement, mild hypertrophy, and improved diastolic function without systolic impairment. Recognition of this adaptive phenotype is essential for accurate differentiation from cardiomyopathy in sports cardiology practice.

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