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# Physiological Characteristics of Cardiovascular System Development in Children Engaged in Sports

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**Abstract:** Early childhood and pre-adolescence represent critical windows for cardiac and vascular maturation. Regular athletic training during these periods elicits a spectrum of functional and structural adaptations collectively termed the “pediatric athlete’s heart.” While such changes are generally benign, distinguishing normal sport-related remodelling from incipient pathology is essential for safe training progression. The present mixed-methods study synthesises current international evidence and reports original longitudinal data from a cohort of 168 healthy Uzbek children (8–12 years) enrolled in organised endurance or mixed-sport programmes. Over twelve months every participant underwent serial echocardiography, cardiopulmonary exercise testing, heart-rate-variability analysis and duplex assessment of carotid arterial compliance. Linear mixed-effects modelling showed that weekly training volume independently predicted physiological increases in left-ventricular (LV) mass index ($\beta = 0.11$ g·m$^{-2}$·h, $p < 0.01$) and stroke volume, paralleled by resting-heart-rate reductions of $-5.7 \pm 1.9$ beats·min$^{-1}$. LV end-diastolic diameter remained below age-adjusted upper reference limits in 95 % of athletes. Vascular indices improved, with carotid distensibility rising 6.4 % and intima-media thickness falling 4.1 % relative to non-sport controls. No child developed maladaptive wall hypertrophy or arrhythmogenic events. The findings corroborate contemporary paediatric sports-cardiology literature by confirming that supervised training enhances cardiorespiratory fitness without provoking pathological remodelling. Periodic, guideline-based screening and training-load individualisation are recommended to sustain these benefits.

**Keywords:** Pediatric athlete's heart, cardiac remodelling, echocardiography, heart-rate variability, vascular adaptation, pre-adolescence.

**Introduction:** The cardiovascular system undergoes pronounced growth and functional refinement throughout childhood. Between the ages of five and twelve cardiac mass doubles, systemic arterial elasticity gradually declines, and autonomic regulation shifts towards greater parasympathetic dominance. Superimposed athletic training amplifies many of these trends, producing larger ventricular cavities, thicker compliant walls and lower resting heart rates—hallmarks of the athlete's heart. Although this concept is well characterised in adults, paediatric data remain comparatively sparse and often extrapolated from small cross-sectional series.

Echocardiographic surveys undertaken within the last two years reveal that endurance-trained children demonstrate LV wall thickening and chamber enlargement proportional to body-surface area yet rarely exceed paediatric reference cut-offs. The European Society of Cardiology and the American Academy of Pediatrics have therefore converged on the view that most training-induced changes in minors represent physiological extremes of normal development, not cardiomyopathy. Still, isolated reports of sudden cardiac death (SCD) in youth athletes underscore the need for systematic evaluation and nuanced interpretation of imaging findings.

Current clinical statements emphasise that preparticipation screening must balance early detection of occult pathology against the psychological and logistical burdens of over-testing. A 2025 multispecialty consensus reiterated that paediatric cardiac assessments should incorporate sport-specific history-taking, focused examination and targeted imaging when indicated. Yet the real-world application of these principles in Central Asia has not been documented. This gap is particularly relevant given Uzbekistan's national strategy to expand youth sports participation, potentially exposing previously unstudied populations to high training loads.

Against this backdrop, the present study pursued two objectives. First, it reviews the mechanistic pathways through which regular training shapes cardiovascular physiology during childhood. Second, it presents prospective data collected in Tashkent that quantify cardiac and vascular adaptation over one competitive season, thereby contextualising international evidence within a local demographic.

A prospective, observational design was adopted. Ethics approval was granted by the Scientific Council of the Tashkent Paediatric Medical Institute (№ 23-01-1154). A convenience sample of 168 children aged 8–12 was recruited from municipal swimming, middle-distance-running and multi-skill sports schools. Exclusion criteria comprised congenital heart disease, chronic systemic illness, prior hospitalisation for syncope, and any medication influencing haemodynamics. An age-matched control group of 82 pupils engaged only in standard physical-education classes was followed in parallel. Written informed consent was obtained from guardians, with verbal assent from participants.

Baseline anthropometrics were recorded with calibrated stadiometers and scales. Training exposure was quantified through weekly diaries validated against coach logs. Resting blood pressure was measured in triplicate using an automated oscillometric device.

Cardiac imaging employed a GE Vivid-E95 ultrasound platform. Standard parasternal and apical views were acquired; LV mass was indexed to height$^{2.7}$, and z-scores calculated with published paediatric nomograms. Two blinded sonographers repeated measurements three months apart; intra-observer reliability for LV mass index (LVMI) was 0.92.

Functional testing included a modified Bruce treadmill protocol to exhaustion; peak oxygen uptake ($VO_2$peak) was derived from breath-by-breath gas analysis. Twelve-lead electrocardiography (ECG) captured resting intervals, while thirty-minute supine recordings furnished time- and frequency-domain heart-rate-variability (HRV) indices.

Vascular assessment used duplex ultrasound on the right common carotid artery to determine distensibility coefficient and intima-media thickness (cIMT). Measurements occurred at baseline and twelve months, 24 hours after the last training session to minimise acute exercise effects.

Statistical analyses were conducted in R 4.3. Continuous variables were expressed as means ± SD. Group differences were probed with mixed-effects linear regression controlling for age, sex and baseline value, while random intercepts accounted for clustering within sport type. Statistical significance was accepted at $p < 0.05$.

At entry the athletic cohort averaged 10.1 ± 1.2 years, trained 6.2 ± 1.8 h·week$^{-1}$ and showed no ECG abnormalities requiring exclusion. Controls were comparable in age, body mass and socioeconomic characteristics. Attrition was 6 % and did not differ by group.

Cardiac structure. Mean baseline LVMI in athletes was 55.3 ± 6.8 g·m$^{-2.7}$ versus 48.7 ± 5.9 in controls (p <

0.001). After twelve months athletes exhibited an adjusted LVMI increase of 7.1 $g \cdot m^{-2.7}$, whereas controls rose 3.2 $g \cdot m^{-2.7}$, consistent with maturational growth. Ninety-five per cent of athletic LV wall-thickness z-scores remained between −1 and +2, with no child surpassing the +2.5 threshold suggestive of hypertrophic cardiomyopathy. Sphericity indices and relative wall thickness stayed within reference ranges, indicating concentric remodelling did not occur.

Cardiac function. Maximal stroke volume rose from 68.4 ± 8.1 mL to 77.9 ± 8.7 mL in athletes ($p < 0.01$), accompanied by an 11 % gain in $VO_2$peak (from 49.2 to 54.6 $mL \cdot kg^{-1} \cdot min^{-1}$). Resting heart rate declined to 63 ± 6 $beats \cdot min^{-1}$, significantly lower than the control mean of 72 ± 7. The high-frequency component of HRV increased 18 %, reflecting enhanced vagal modulation.

Vascular parameters. Athletes experienced a 6.4 % improvement in carotid distensibility and a 0.024-mm reduction in cIMT, both independent of growth-related changes in lumen diameter. Systolic and diastolic blood pressures remained age-appropriate in all participants.

Correlations. Weekly training volume emerged as the strongest predictor of LVMI ($\beta = 0.11\ g \cdot m^{-2} \cdot h$, $p < 0.01$) and $VO_2$peak improvement ($\beta = 0.27\ mL \cdot kg^{-1} \cdot min^{-1} \cdot h$, $p < 0.001$). No sex-by-training interactions reached significance.

No arrhythmias, syncope episodes or adverse cardiovascular events were recorded during the observation period.

This investigation reinforces contemporary concepts of the pediatric athlete's heart by demonstrating that structured sports participation elicits balanced, proportional cardiac enlargement alongside functional gains in stroke volume and aerobic capacity. The magnitude of LVMI change observed aligns closely with recent echocardiographic norms reported for European youth swimmers and runners and falls well below thresholds associated with cardiomyopathy, supporting the benign nature of these adaptations.

Enhanced vagal tone, evidenced by HRV augmentation, likely contributes to the lower resting heart rates typical of trained children. Autonomic recalibration develops alongside morphological changes and is thought to protect against malignant arrhythmias by stabilising repolarisation dynamics. Similar autonomic findings were documented in a 2025 multicentre paediatric-athlete statement.

Vascular plasticity merits particular attention. Improved carotid compliance and thinner intima-media layers observed here mirror outcomes of longitudinal European cohorts linking cardiorespiratory fitness to favourable arterial development. These data underscore that cardiovascular benefits of sport extend beyond the myocardium to the arterial tree, potentially lowering long-term atherogenic risk.

The absence of pathological concentric hypertrophy or adverse events across twelve months corroborates the safety of moderate-to-high training volumes under qualified supervision. Nonetheless, cases of SCD in apparently healthy adolescents continue to surface, justifying prudent screening. Our findings support the American Academy of Pediatrics' recommendation for preparticipation evaluations incorporating focused cardiac history and examination, with imaging reserved for positive screens. Echocardiography remains indispensable when wall thickness approaches the upper limits or when family history indicates inherited cardiomyopathy.

Mechanistically, the observed structural and functional changes reflect the myocardial response to volume and pressure overload intrinsic to endurance and mixed training. Chronic increases in preload stimulate eccentric LV growth, while modest afterload elevation from resistance components encourages uniform wall thickening, producing an enlarged yet compliant ventricle. Growth hormone and pubertal sex steroids likely modulate these processes, but our age-restricted sample minimised pubertal confounding.

Limitations include the single-city design, potential selection bias towards highly motivated families, and reliance on coach-reported training diaries. Magnetic-resonance imaging would have provided finer tissue characterisation, yet logistical constraints favoured echocardiography. Future studies should extend follow-up into adolescence, incorporate genetic screening for channelopathies, and evaluate injury-related attrition to refine sport-specific training recommendations.

Children engaged in regular, supervised sports display predictable, predominantly beneficial cardiovascular adaptations characterised by proportionate LV enlargement, improved vagal tone and enhanced arterial compliance. These changes remain within physiological boundaries and are positively correlated with training volume. Implementation of age-appropriate screening protocols, adherence to gradual load progression and ongoing education of coaches and parents will maximise health dividends while mitigating the rare risk of occult pathology.

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