Adaptation of Left Ventricular Twist Mechanics in Exercise-Trained Children Is Only Evident after the Adolescent Growth Spurt

Dean R. Perkins, PhD, Jack S. Talbot, PhD, Rachel N. Lord, PhD, Tony G. Dawkins, PhD, Aaron L. Baggish, MD, Abbas Zaidi, MD, Orhan Uzun, MD, Kelly A. Mackintosh, PhD, Melitta A. McNarry, PhD, Stephen-Mark Cooper, PhD, Rhodri S. Lloyd, PhD, Jon L. Oliver, PhD, Rob E. Shave, PhD, and Mike Stembridge, PhD, *Cardiff and Swansea, United Kingdom; Kelowna, British Columbia, Canada; Lausanne, Switzerland; and Auckland and Waikato, New Zealand*

Background: The extent of structural cardiac remodeling in response to endurance training is maturity dependent. In adults, this structural adaptation is often associated with the adaptation of left ventricular (LV) twist mechanics. For example, an increase in LV twist often follows an expansion in end-diastolic volume, whereas a reduction in twist may follow a thickening of the LV walls. While structural cardiac remodeling has been shown to be more prominent post–peak height velocity (PHV), it remains to be determined how this maturation-dependent structural remodeling influences LV twist. Therefore, we aimed to (1) compare LV twist mechanics between trained and untrained children pre- and post-PHV and (2) investigate how LV structural variables relate to LV twist mechanics pre- and post-PHV.

Methods: Left ventricular function and morphology were assessed (echocardiography) in endurance-trained and untrained boys (n = 38 and n = 28, respectively) and girls (n = 39 and n = 34, respectively). Participants were categorized as either pre- or post-PHV using maturity offset to estimate somatic maturation.

Results: Pre-PHV, there were no differences in LV twist or torsion between trained and untrained boys (twist: P = .630; torsion: P = .382) or girls (twist: P = .502; torsion: P = .316), and LV twist mechanics were not related with any LV structural variables (P > .05). Post-PHV, LV twist was lower in trained versus untrained boys (P = .004), with torsion lower in trained groups, irrespective of sex (boys: P < .001; girls: P = .017). Moreover, LV torsion was inversely related to LV mass (boys: r = −0.55, P = .001; girls: r = −0.46, P = .003) and end-diastolic volume (boys: r = −0.64, P < .001; girls: r = −0.36, P = .025) in both sexes.

Conclusions: A difference in LV twist mechanics between endurance-trained and untrained cohorts is only apparent post-PHV, where structural and functional remodeling were related. (J Am Soc Echocardiogr 2024;37:538-49.)

Keywords: Echocardiography, Myocardium, Twist, Youth, Exercise

INTRODUCTION

Endurance training results in an enlarged left ventricular (LV) end-diastolic volume (EDV) and proportional LV hypertrophy in adults.[1] Left ventricular structural adaptation to training can influence functional remodeling, which is particularly the case for LV twist — a key component to overall ventricular mechanics.[2,3] Twist is quantified as the apex-to-base difference in LV rotation and facilitates optimal

From the Cardiff School of Sport and Health Sciences, Cardiff Metropolitan University, Cardiff, United Kingdom (D.R.P., J.S.T., R.N.L., T.G.D., S.M.C., M.S.); Centre for Heart, Lung and Vascular Health, School of Health and Exercise Sciences, University of British Columbia Okanagan, Kelowna, British Columbia, Canada (T.G.D., R.E.S.); Institute of Sports Science, University of Lausanne, Lausanne, Switzerland (A.L.B.); Department of Cardiology, University Hospital of Wales, Cardiff, United Kingdom (A.Z., O.U.); Applied Sports, Technology, Exercise and Medicine (A-STEM) Research Centre, Swansea University, Swansea, United Kingdom (K.A.M., M.A.M.); Youth Physical Development Centre, Cardiff Metropolitan University, Cardiff, United Kingdom (R.S.L., J.L.O.); Sports Performance Research Institute New Zealand, AUT University, Auckland, New Zealand (R.S.L., J.L.O.); and Centre for Sport Science and Human Performance, Waikato Institute of Technology, Waikato, New Zealand (R.S.L.).

Reprint requests: Mike Stembridge, PhD, Cardiff School of Sport and Health Sciences, Cardiff Metropolitan University, Cyncoed Campus, Cyncoed Road, Cardiff, United Kingdom (E-mail: *mstembridge@cardiffmet.ac.uk*).

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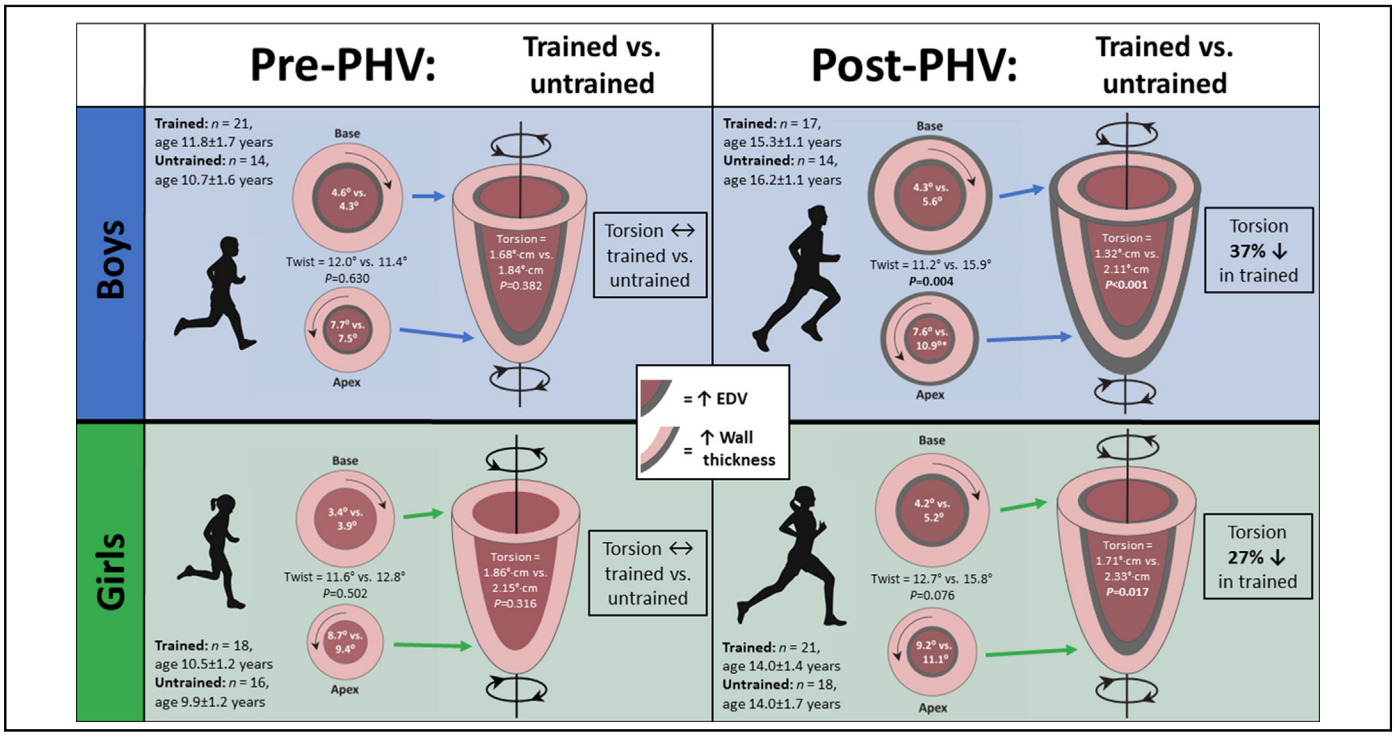

Central Illustration Schematic summary outlining basal and apical rotation and twist and torsion differences between trained and untrained boys and girls pre- and post-PHV. We aimed to identify the influence of somatic maturation on LV twist and torsion in endurance-trained and untrained boys and girls. We found no differences in LV twist mechanics pre-PHV, whereas post-PHV endurance-trained boys and girls had a lower LV torsion and boys also had a lower twist compared with untrained groups. The lower LV twist mechanics in trained groups post-PHV also coincided with greater structural remodeling. Collectively, this highlights that the influence of endurance training on LV twist mechanics is only apparent post-PHV.

Abbreviations

ANOVA = Analysis of variance
BSA = Body surface area
EDV = End-diastolic volume
ESV = End-systolic volume
HR_{max} = Maximum heart rate
LBM = Lean body mass
LV = Left ventricular
MWT = Mean wall thickness
PHV = Peak height velocity
$\dot{V}O_{2max}$ = Maximal oxygen consumption

energy efficiency and redistribution of fiber stress across the myocardium.[4] A lower apical rotation has been observed in individuals with a high aerobic fitness in the absence of LV structural remodeling.[5] However, LV twist appears to be largely influenced by the volume and wall thickness of the ventricle,[6-9] which are typically adapted with endurance training. The relationship between LV form and function can be observed with long-term endurance training. A short period (~3 months) of intensified training results in an acute enlargement of EDV and an increase in LV twist mechanics.[3] When training is continued for ~3 years and eccentric hypertrophy of the LV walls is present, a reduction in LV twist mechanics is also observed.[3] Therefore, it appears that an enlargement of EDV with training may result in enhanced twist mechanics at rest, but in the presence of comprehensive LV remodeling associated with chronic training, resting twist mechanics are reduced.

The pubertal growth spurt, encompassing peak height velocity (PHV), is a period of time associated with significant cardiac growth and an increase in LV twist.[10] However, when LV twist is normalized for LV length (i.e., torsion), there is no significant difference from childhood to early adulthood,[10] indicating that the growth-related increase in twist is dependent on ventricular structure. When exercise training is undertaken during maturation, there is a small degree of cardiac remodeling evident in preadolescence, while a greater proportional increase in LV mass and blood-volume expansion is observed postpuberty.[11,12] Left ventricular twist data in trained preadolescent children are sparse, yet twist appears to be increased[9,13] and coincides with an elevated EDV.[9] This is similar to the acute adaptation observed with intensified training in adults.[3] Around maturation, the growth in LV size is associated with an increase in twist.[10] However, training post-PHV also leads to prominent thickening of the LV walls, which, as shown in young adults, drives a decrease in twist.[3,6]

In the clinical context, it is important for sports cardiologists to understand how the child athlete's heart structure and function are likely to adapt. An ability to differentiate between physiological and pathological cardiac remodeling is essential,[14] especially throughout maturation due to the vast physiological changes. When determining whether an athlete's presentation is pathological, all possible diagnostic tools should be considered. Left ventricular twist has shown promise as a measure to quantify dysfunction, beyond traditional markers of LV function, within various disease conditions.[15] Hence, LV twist mechanics may be a useful tool to distinguish between physiology and pathology in pediatric athletic patients.

We speculate that greater LV remodeling post-PHV could decrease twist, superseding the aforementioned maturational LV growth that can otherwise increase twist. We therefore aimed to compare LV twist

HIGHLIGHTS

- Pre-PHV training status does not alter LV twist mechanics.
- Post-PHV, LV twist mechanics are lower in endurance-trained versus untrained children.
- LV twist mechanics are related to structural remodeling but only post-PHV.

and torsion between endurance-trained and untrained children pre- and post-PHV. We hypothesized that (1) LV twist and torsion would be elevated in trained versus untrained pre-PHV children and torsion would be positively related to EDV but that (2) LV twist and torsion would be lower in trained versus untrained adolescents following PHV and torsion would be negatively related to LV mass.

METHODS

Study Participants

All procedures were approved by the Cardiff Metropolitan University Natural Sciences Research Ethics Sub-committee (PGR-1339) and conformed to the ethical standards of the Declaration of Helsinki, except for registration in a database. Written informed consent and assent were obtained from parents/guardians and participating youth, respectively. Recruitment was undertaken from the age range of 8-17 to encompass the range of maturity.[16] Participants were categorized to pre- or post-PHV as described in the "Experimental Design" section. Initially, 163 participants were recruited. Descriptive structural data have been reported previously,[12] but the present study addresses a unique a priori research question in a subset population using novel metrics of cardiac function. Participants were excluded due to incomplete data sets ($n = 4$), failing to meet our physical activity criteria as reported by participants and corroborated with parents ($n = 6$), or due to inadequate speckle-tracking analysis ($n = 15$). Endurance-trained youth were recruited from cycling, swimming, long-distance running, or triathlon clubs (boys: $n = 38$, 9.0-17.1 years; girls: $n = 39$, 8.2-17.0 years). The percentage distribution within each trained group for these respective sports was (percentage from cycling/swimming/running/triathlon) pre-PHV boys (57/19/19/5), post-PHV boys (65/0/6/29), pre-PHV girls (28/33/11/28), and post-PHV girls (38/24/24/14). Physical activity and training criteria for inclusion have been described in detail elsewhere.[12] Trained children were undertaking at least 3 hours of structured endurance training per week (boys: pre-PHV: 9.0 ± 2.4 hours/week^{-1}, post-PHV: 10.0 ± 2.6 hours/week^{-1}; girls: pre-PHV: 5.4 ± 2.0 hours/week^{-1}, post-PHV: 8.7 ± 3.7 hours/week^{-1}) for ≥12 months (boys: pre-PHV: 3.9 ± 1.5 years, post-PHV: 5.9 ± 2.4 years; girls: pre-PHV: 2.2 ± 1.3 years, post-PHV: 4.0 ± 1.7 years) and competing in their respective sport. Untrained children were recruited to form a comparative group from local schools (boys: $n = 28$, 8.0-17.7 years; girls: $n = 34$, 8.0-17.6 years). Healthy trained and untrained cohorts were categorized by maturity status and sex, thereby permitting assessment for the influence of training status on the key outcome variables between groups. Physical activity levels for untrained participants were low (untrained boys: pre-PHV, 1.1 ± 1.0 hours/week; post-PHV, 0.9 ± 1.1 hours/week; untrained girls: pre-PHV, 1.0 ± 0.9 hours/week; post-PHV, 0.5 ± 0.8 hours/week). Exclusion criteria included hypertension, smoking, cardiovascular disease, and obesity according to age- and sex-specific body mass index cutoffs of the International Obesity Task Force criteria.[17]

Experimental Design

Participants attended the laboratory on 2 occasions. Parents/guardians were asked to ensure that their child arrived well hydrated and having refrained from heavy exercise and caffeine consumption 12 hours before attending. The majority (~85%) of laboratory visits took place in the afternoon, while the remaining visits occurred across the day. There was no systematic bias for time of testing in any specific group. During the first laboratory visit, anthropometric measures including body mass, height, and sitting height were quantified. Lean body mass (LBM) was also quantified using skinfold measures and youth-specific equations.[18] Maturity status was estimated using the maturity offset method.[16] Participants were categorized as either pre- or post-PHV based on a maturity offset value below or above 0, respectively, as originally recommended.[16] Following 10 minutes of supine rest, blood pressure was measured using an automated sphygmomanometer (Omron Healthcare). Body surface area (BSA) was calculated using the Haycock formula.[19] Maximal oxygen consumption ($\dot{V}O_{2max}$) and maximum heart rate (HR_{max}) were assessed during incremental cardiopulmonary exercise testing on an upright cycle ergometer (Lode, Excalibur). During the second visit, following 10 minutes of supine rest, echocardiographic measurements were obtained in the left lateral decubitus position.

Experimental Measures

Cardiorespiratory Fitness. An incremental ramp protocol was used to assess $\dot{V}O_{2max}$ and HR_{max} as described elsewhere.[20] Participants cycled at a continuous pace of 75 to 85 rpm until they could no longer continue. Subsequently, participants rested for 15 minutes prior to a constant-load supramaximal verification test at 105% of their achieved peak power output to verify $\dot{V}O_{2max}$ as described elsewhere.[21] The highest 30-second average value attained from either the incremental or supramaximal test was accepted as the $\dot{V}O_{2max}$.

Resting Echocardiography. An echocardiographic examination was performed with a Vivid E9 system (GE Vingmed Ultrasound) using a 1.5 to 4 MHz phased-array transducer. Two-dimensional images were obtained from the parasternal and apical acoustic windows for the assessment of LV structure, global function, and twist mechanics as per current recommendations.[22] Images were acquired at the highest possible frame rate (70-90 frames per second) and stored digitally for offline analysis (Echopac, GE Medical). We have previously reported structural data for this cohort.[12] However, the following variables have been re-reported for preciseness given that the current study has fewer participants and to help aid the understanding of the structure and function relationships. Left ventricular mass was calculated using the area-length method, derived from measures of LV length and mean wall thickness (MWT).[22] Left ventricular length was measured at end diastole and determined as the length from the mitral valve annulus to the apical point of the apical contour from the 4-chamber view. Mean wall thickness was calculated from cross-sectional areas of the epicardium and the endocardium in a short-axis view at the papillary level.[22] End-diastolic volume, end-systolic volume (ESV), and ejection fraction were assessed using Simpson's biplane method. Linear and three-dimensional LV structural variables are scaled

height and LBM, respectively, in a dimensionally consistent manner as recommended,[23] particularly when analyzing both sexes with trained and untrained cohorts.[24] However, for clinical application and reference, LV structures were also scaled to BSA in accordance with pediatric echocardiography clinical guidelines.[25] Sphericity index was calculated as LV length/LV internal diameter measured from the 4-chamber view.[7] Intraobserver coefficients of variation for LV structural variables have been previously reported to be between 3.5% and 8.2%.[12]

Pulsed-wave Doppler measures of early (E) and late (A) peak mitral inflow velocities were obtained in the apical 4-chamber view, with the sample volume at the valve tips. Tissue Doppler imaging was quantified as an average of the peak myocardial velocities from the septal and lateral walls to determine systolic (S′), early diastolic (e′), and late diastolic (A′) velocities.

Speckle-Tracking Echocardiography. Speckle-tracking analysis of rotation parameters was completed using commercially available software (Echopac, GE Medical). Basal and apical rotation were measured from parasternal short-axis views. Raw data were time aligned and interpolated with custom-made software (2D Strain Analysis Tool), as described elsewhere.[5] Left ventricular twist was calculated as the time-aligned difference between basal and apical rotation. To normalize the LV twist for differences in LV length, torsion was calculated as LV twist/length.

Statistical Analyses and Power Calculation. Results are presented as means ± SD unless stated otherwise. Two-way analysis of variance (ANOVA) with 2 factors (training and maturity status) were used to explore differences between groups, independently for boys and girls. In the case of a significant main effect, independent-samples *t* tests were used to identify any differences. Secondary multiple linear regression analyses were also completed, with all data pooled, to identify the independent contributions of training status, maturity status, and sex on LV twist and torsion. Lastly, relationships between LV structural variables and torsion were analyzed with trained and untrained data pooled for independent groups of pre- and post-PHV, boys and girls. Linear regression analysis was used to identify relationships for LV torsion with LV mass, EDV, and wall thickness, which have been previously shown to influence twist.[6-8] Alpha was set a priori as 0.05. Statistical analyses were performed with the Statistical Package for Social Science Software (ver. 24) and GraphPad (Prism, ver. 8.1.1, GraphPad Software).

Left ventricular twist has not previously been investigated in relation to both training and maturation. However, Beaumont *et al.*[9] found a difference of 4.58° in twist between preadolescent soccer players and controls with SD = 6.27°. Using the effect size calculated from these data and accounting for the current statistical model, the calculated sample size was 40 for each sex, to provide 80% power, with $\alpha = 0.05$.

RESULTS

Anthropometric, Hemodynamic, and Cardiorespiratory Fitness Characteristics

Maturity offset, height, and body mass were significantly greater in post- compared with pre-PHV groups (Table 1). Resting HR was significantly higher, while systolic blood pressure was significantly lower in pre-PHV groups (Table 1), with no significant differences in diastolic blood pressure. No significant differences in $\dot{V}O_{2max}$ were found between pre- and post-PHV groups of the same training status (Table 1).

Trained and untrained groups were well matched for maturity offset and all anthropometric characteristics (Table 1). Resting HR was significantly lower in trained versus untrained post-PHV groups ($P < .001$) but not between pre-PHV groups. Trained groups had a higher $\dot{V}O_{2max}$ than their untrained counterparts, irrespective of maturity status (Table 1).

Left Ventricular Structure

Absolute MWT, LV length, LV mass, EDV, and ESV were greater for the trained and untrained groups post- versus pre-PHV in boys and girls (Table 2). When scaled to height or LBM for linear or three-dimensional variables, respectively, there were no maturity-related differences in any LV structural variables (Table 2). However, when scaled to BSA with clinically accepted exponents, MWT index and LV mass index were greater in both trained and untrained post- versus pre-PHV boys, whereas LV length index was only greater in trained post-versus pre-PHV boys. In contrast, the only maturity-related differences for girls were for ESV index, which was greater in trained and smaller in untrained post- versus pre-PHV.

Pre-PHV, absolute LV length, LV mass, EDV, and ESV were greater in trained versus untrained boys (Table 2). In pre-PHV girls, absolute LV mass was greater between trained and untrained groups. Training-related differences in scaled LV structures pre-PHV were a significantly greater LV length/height, EDV/LBM, and ESV/LBM in trained versus untrained boys, with no differences in pre-PHV girls (Table 2). These training-related differences pre-PHV remained when LV structural variables were scaled to BSA, with the addition of a greater LV mass index for trained versus untrained boys and girls. Post-PHV, absolute measures of LV length, LV mass, EDV, and ESV were greater in trained versus untrained groups for both boys and girls (Table 2). All measured relative LV structure variables were greater in post-PHV trained versus untrained boys (Table 2). Similarly, trained post-PHV girls had a greater LV mass/LBM, EDV/LBM, and ESV/LBM. When scaled to BSA, the same training-related differences were found post-PHV, with the addition of a greater LV length in trained versus untrained girls.

Influence of Maturity Status on Cardiac Function

Systolic Function. Apical rotation was greater in post- compared with pre-PHV untrained boys (Table 3), with no significant maturity-related differences in basal rotation between any groups. However, the main effects of maturation for LV twist and torsion did not reach significance for boys ($P = .067$ and $P = .708$, respectively) or girls ($P = .094$ and $P = .067$, respectively; Table 3).

Diastolic Function. Despite similarities in untwisting velocity between pre- and post-PHV groups, and the dependency of untwist velocity on early LV filling, maturity-related differences were observed in diastolic filling velocities (Table 3). Early and late diastolic filling velocities were lower in untrained boys post-PHV, whereas late diastolic filling was lower and E/A was greater in untrained girls post-PHV (Table 3).

Influence of Training Status on Cardiac Function Pre- and Post-PHV

Systolic Function. Pre-PHV, no significant differences in LV twist mechanics were observed with training status, aside from a lower LV twist velocity in pre-PHV trained girls (Table 3). No other

Table 1 Participant characteristics and cardiovascular measurements

	Boys		*P* value			Girls		*P* value		
	Pre-PHV	Post-PHV	Training effect	Maturity effect	Interaction	Pre-PHV	Post-PHV	Training effect	Maturity effect	Interaction
Group *n*										
ET	21	17				18	21			
UN	14	14				16	18			
Age, years										
ET	11.8 ± 1.7	15.3 ± 1.1*	.771	<.001	.007	10.5 ± 1.2	14.0 ± 1.4*	.275	<.001	.362
UN	10.7 ± 1.6	16.2 ± 1.1*				9.9 ± 1.2	14.0 ± 1.7*			
Maturity offset, years										
ET	−2.0 ± 1.2	1.5 ± 1.0*	.906	<.001	.053	−1.3 ± 1.0	1.8 ± 1.1*	.164	<.001	.381
UN	−2.6 ± 1.1	2.0 ± 1.1*				−1.9 ± 0.9	1.7 ± 1.1*			
Height, cm										
ET	149.1 ± 11.9	175.8 ± 9.0*	.721	<.001	.517	144.0 ± 9.7	164.3 ± 6.8*	.023	<.001	.249
UN	146.5 ± 10.3	176.6 ± 10.3*				137.3 ± 9.8	162.0 ± 6.3*			
Body mass, kg										
ET	39.8 ± 8.8	61.1 ± 10.1*	.544	<.001	.810	35.1 ± 5.6	53.6 ± 8.5*	.933	<.001	.497
UN	40.6 ± 9.0	63.2 ± 10.4*				33.5 ± 5.7	54.8 ± 12.3*			
Resting HR, beat/min^{-1}										
ET	65 ± 13	51 ± 4†*	<.001	<.001	.254	74 ± 9	62 ± 7†*	.001	<.001	.987
UN	72 ± 7	63 ± 8‡				81 ± 16	70 ± 7‡			
Blood pressure, mm Hg										
Systolic										
ET	105 ± 8	116 ± 9*	.599	<.001	.273	104 ± 8	111 ± 7‡	.105	<.001	.833
UN	102 ± 8	117 ± 7*				101 ± 7	109 ± 6*			
Diastolic										
ET	60 ± 7	62 ± 5	.431	.891	.159	63 ± 7	65 ± 6	.529	.482	.653
UN	64 ± 7	61 ± 7				63 ± 5	63 ± 6			
Cardiorespiratory fitness $\dot{V}O_{2max}$, mL/kg/min^{-1}										
ET	59.4 ± 5.8†	65.4 ± 7.5†	<.001	.056	.341	50.5 ± 5.8†	50.9 ± 5.8†	<.001	.820	.127
UN	43.4 ± 6.1	48.3 ± 3.7				41.9 ± 5.8	37.8 ± 5.7			

ET, Endurance trained; *UT*, untrained.
Data are from 2-way ANOVAs with training and maturity status as fixed factors. Independent-samples *t* tests were used to identify differences. Data are expressed as mean ± SD.
†*P* < .001 trained versus untrained.
‡*P* < .05 post-PHV versus pre-PHV.
**P* < .001 post-PHV versus pre-PHV.

significant differences were found in measures of systolic function based on training status pre-PHV (Table 3).

Post-PHV, LV twist was lower in trained boys compared with their untrained counterparts, but this was not found between post-PHV girls (Figure 1; Table 3). The lower twist in trained boys post-PHV was driven by apical rotation, with no significant differences found in basal rotation between any groups. Left ventricular torsion was lower in both trained boys and girls post-PHV compared with untrained groups (Table 3; boys: $P < .001$; girls: $P = .017$). Similarly, LV twist velocity was lower in trained boys and girls post-PHV compared with untrained counterparts (Table 3; boys: $P = .003$; girls: $P = .003$). Despite these observed differences in LV twist mechanics, ejection fraction and S′ were similar between trained and untrained post-PHV groups (Table 3).

Diastolic Function. Pre-PHV, there were no significant differences in diastolic function measurements between trained and untrained boys (Table 3), while the only training-related difference in diastolic function between pre-PHV girls was a significantly lower late filling velocity in trained versus untrained groups, which led to a greater E/A (Table 3).

Table 2 Left ventricular structure

	Boys		*P* value			Girls		*P* value		
	Pre-PHV	Post-PHV	Training effect	Maturity effect	Interaction	Pre-PHV	Post-PHV	Training effect	Maturity effect	Interaction
Group, *n*										
ET	21	17				18	21			
UN	14	14				16	18			
LV structure										
MWT, mm										
ET	6.6 ± 1.0	8.0 ± 0.9*	.018	<.001	.781	6.6 ± 0.6	7.5 ± 1.0†	.066	<.001	.573
UN	6.1 ± 0.4	7.4 ± 0.5*				6.4 ± 0.4	7.2 ± 0.5*			
LV length, mm										
ET	71 ± 8‡	86 ± 6*‡	<.001	<.001	.934	63 ± 6	75 ± 6*§	.002	<.001	.364
UN	62 ± 5	77 ± 7*				60 ± 4	69 ± 6*			
LV mass, g										
ET	91 ± 25§	155 ± 28*§	<.001	<.001	.219	81 ± 16§	124 ± 30*§	<.001	<.001	.333
UN	74 ± 10	125 ± 20*				68 ± 12	102 ± 18*			
EDV, mL										
ET	66 ± 18§	104 ± 16*‡	<.001	<.001	.659	48 ± 10	75 ± 14*‡	<.001	<.001	.030
UN	49 ± 9	83 ± 14*				43 ± 10	58 ± 13*			
ESV, mL										
ET	27 ± 7§	42 ± 7*§	<.001	<.001	.637	18 ± 4	30 ± 7*‡	<.001	<.001	.004
UN	19 ± 5	33 ± 7*				16 ± 4	21 ± 6†			
Sphericity index										
ET	1.7 ± 0.2	1.7 ± 0.2	.031	.904	.781	1.6 ± 0.2	1.6 ± 0.2	.669	.267	.517
UN	1.6 ± 0.2	1.6 ± 0.2				1.5 ± 0.1	1.6 ± 0.1			
Scaled LV structure										
MWT/height, mm/m										
ET	4.4 ± 0.5	4.5 ± 0.5§	.023	.475	.525	4.6 ± 0.4	4.6 ± 0.5	.694	.131	.191
UN	4.2 ± 0.4	4.2 ± 0.3				4.7 ± 0.4	4.4 ± 0.3			
LV length/height, mm/m										
ET	48 ± 3‡	49 ± 3‡	<.001	.323	.952	44 ± 4	45 ± 3	.075	.795	.098
UN	43 ± 3	43 ± 4				44 ± 3	43 ± 4			
LV mass/LBM, g/kg										
ET	2.7 ± 0.3	2.9 ± 0.4‡	<.001	.319	.085	2.9 ± 0.4	2.9 ± 0.4§	.003	.571	.481
UN	2.5 ± 0.4	2.4 ± 0.3				2.7 ± 0.3	2.6 ± 0.3			
EDV/LBM, mL/kg										
ET	2.0 ± 0.3§	1.9 ± 0.2‡	<.001	.716	.985	1.7 ± 0.3	1.8 ± 0.3§	.004	.312	.028
UN	1.6 ± 0.3	1.6 ± 0.2				1.6 ± 0.2	1.4 ± 0.3			
ESV/LBM, mL/kg										
ET	0.8 ± 0.1§	0.8 ± 0.1§	<.001	.761	.997	0.6 ± 0.1	0.7 ± 0.1‡	.011	.502	.004
UN	0.6 ± 0.1	0.6 ± 0.1				0.6 ± 0.1	0.5 ± 0.1			
Clinically scaled LV structure										
MWT index, $m/(m^2)^{0.4}$										
ET	5.9 ± 0.7	6.4 ± 0.6†§	.005	.004	.724	6.2 ± 0.4	6.3 ± 0.7	.091	.882	.299

(*Continued*)

Table 2 (*Continued*)

	Boys		*P* value			Girls		*P* value		
	Pre-PHV	Post-PHV	Training effect	Maturity effect	Interaction	Pre-PHV	Post-PHV	Training effect	Maturity effect	Interaction
UN	5.6 ± 0.4	6.0 ± 0.4†				6.1 ± 0.4	6.0 ± 0.3			
LV length index, mm/(m²)^0.45										
ET	64 ± 5‡	67 ± 4†‡	<.001	.005	.729	59 ± 5	61 ± 5§	.003	.295	.169
UN	56 ± 5	60 ± 5				57 ± 3	57 ± 4			
LV mass index, g/(m²)^1.25										
ET	66 ± 9‡	79 ± 11*‡	<.001	<.001	.159	66 ± 8§	71 ± 12‡	<.001	.284	.198
UN	55 ± 6	62 ± 6†				59 ± 8	58 ± 6			
EDV index. mL/(m²)^1.3										
ET	48 ± 8‡	51 ± 5‡	<.001	.030	.797	39 ± 7	43 ± 7‡	<.001	.989	.012
UN	36 ± 7	40 ± 6				36 ± 5	32 ± 6			
ESV index, mL/(m²)^1.3										
ET	20 ± 3‡	21 ± 3‡	<.001	.047	.772	14 ± 3	17 ± 3†‡	<.001	.888	.001
UN	14 ± 3	16 ± 3				14 ± 3	12 ± 3†			

ET, Endurance trained; *UN*, untrained.
Data are from 2-way ANOVAs with training and maturity status as fixed factors. Independent-samples *t* tests were used to identify differences. Data are expressed as mean ± SD.
§*P* < .05 trained versus untrained.
‡*P* < .001 trained versus untrained.
†*P* < .05 post-PHV versus pre-PHV.
**P* < .001 post-PHV versus pre-PHV.

Post-PHV, a significantly slower untwisting velocity was identified for trained versus untrained girls (Table 3). Despite this, early diastolic filling velocities were not significantly different between post-PHV trained versus untrained groups. In post-PHV trained versus untrained girls there was a significantly lower late filling velocity, resulting in a greater E/A (Table 3). The lower late filling velocity was also reflected in the tissue velocity, with a lower A′ in trained versus untrained post-PHV girls, while no significant tissue velocity differences were found in post-PHV boys (Table 3).

Multiple Linear Regression Analyses. Multiple linear regressions revealed that endurance training was a negative independent predictor of both LV twist ($P = .010$) and torsion ($P < .001$). Additionally, post-PHV maturity status was identified as a positive predictor of LV twist ($P = .031$), whereas male sex was identified as a negative predictor of LV torsion ($P = .023$).

Relationships Between LV Structure and Torsion. Relationships for LV torsion with LV mass, EDV, and MWT are shown in Figure 2. No significant relationships were identified pre-PHV for either sex. Inverse relationships were identified for LV mass and EDV with torsion in post-PHV boys. The same structural variables, with the addition of MWT, were inversely related to torsion in post-PHV girls.

DISCUSSION

The aim of this study was to identify the influence of maturity status on endurance training–related adaptations in LV twist mechanics. In relation to our 2 hypotheses, the main findings were that (1) LV twist mechanics are not significantly different between pre-PHV trained and untrained boys or girls, nor are there relationships between LV structure and torsion pre-PHV; and (2) post-PHV, LV torsion was lower in trained boys and girls alongside significant inverse relationships between LV torsion with LV mass and volumes in post-PHV boys and girls. Collectively, these findings indicate that changes in LV twist mechanics with extensive exercise training are more evident post-PHV and relate to greater structural remodeling.

Influence of Training Status on Systolic Twist Mechanics Pre-PHV

Pre-PHV, LV twist and torsion were not significantly different between trained and untrained groups. This was in contrast to our hypothesis that LV twist mechanics would be greater in pre-PHV trained versus untrained groups and with previous work in preadolescent soccer players.[9,13] The differing results may be explained by ventricular shape because LV sphericity index, a measure of LV elongation, holds a parabolic relationship with LV twist.[7] In the current study, LV sphericity was not significantly different compared with training status pre-PHV. However, it has been shown that preadolescent soccer players had a more elongated left ventricle[9] alongside elevated twist, and the authors speculated that this placed them higher on the ascending arm of the parabolic relationship between LV twist and sphericity index. The disparity in LV remodeling between the current study and the previous study[9] may be due to differences in training intensities between endurance athletes and soccer players. Both elicit a high hemodynamic load; the intensity distribution in

Table 3 Left ventricular function and twist mechanics

	Boys		*P* value			Girls		*P* value		
	Pre-PHV	Post-PHV	Training effect	Maturity effect	Interaction	Pre-PHV	Post-PHV	Training effect	Maturity effect	Interaction
Group *n*										
ET	21	17				18	21			
UN	14	14				16	18			
LV systolic function										
Apical rotation, degrees										
ET	7.7 ± 2.6	7.6 ± 3.2*	.036	.029	.018	8.7 ± 1.9	9.5 ± 4.2	.163	.238	.533
UN	7.5 ± 2.1	10.9 ± 3.7†				9.4 ± 4.3	10.9 ± 5.2			
Basal rotation, degrees										
ET	−4.6 ± 2.0	−4.3 ± 3.0	.455	.505	.256	−3.4 ± 2.9	−4.7 ± 3.6	.350	.173	.747
UN	−4.3 ± 3.1	−5.6 ± 3.1				−3.9 ± 3.4	−4.6 ± 2.8			
Twist, degrees										
ET	12.0 ± 4.1	11.2 ± 3.4‡	.040	.067	.008	11.6 ± 3.7	12.7 ± 4.1	.083	.094	.425
UN	11.4 ± 3.0	15.9 ± 4.9†				12.8 ± 6.0	15.8 ± 6.3			
Torsion, degrees (cm)										
ET	1.7 ± 0.5	1.3 ± 0.4*†	.001	.708	.021	1.9 ± 0.6	1.7 ± 0.5‡	.018	.952	.377
UN	1.8 ± 0.5	2.1 ± 0.7				2.2 ± 1.0	2.3 ± 1.0			
Twist velocity, degrees/sec										
ET	85 ± 21	80 ± 17‡	.004	.187	.037	84 ± 25‡	76 ± 19‡	<.001	.592	.588
UN	90 ± 24	111 ± 35				107 ± 40	107 ± 40			
LV ejection fraction, %										
ET	59 ± 3	59 ± 4	.146	.995	.834	63 ± 5	61 ± 4	.356	.766	.017
UN	61 ± 5	60 ± 5				61 ± 3	64 ± 4			
S′, cm/sec										
ET	0.09 ± 0.01	0.10 ± 0.01†	.612	.011	.413	0.10 ± 0.01	0.10 ± 0.01	.837	.089	.100
UN	0.10 ± 0.01	0.10 ± 0.01				0.09 ± 0.01	0.10 ± 0.01			
LV diastolic function										
Untwist velocity, degrees/sec										
ET	−107 ± 30	−99 ± 25	.391	.673	.104	−129 ± 39	−111 ± 36‡	.133	.610	.009
UN	−102 ± 38	−117 ± 20				−117 ± 50	−146 ± 53			
E, cm/sec										
ET	0.96 ± 0.14	0.91 ± 0.11	.577	.009	.180	1.01 ± 0.18	1.01 ± 0.18	.763	.517	.491
UN	0.99 ± 0.17	0.84 ± 0.15†				0.97 ± 0.13	1.02 ± 0.19			
A, cm/sec										
ET	0.42 ± 0.08	0.40 ± 0.09	.395	.039	.167	0.35 ± 0.06*	0.32 ± 0.08‡	<.001	.005	.335
UN	0.46 ± 0.08	0.39 ± 0.07†				0.48 ± 0.11	0.40 ± 0.08†			
E/A										
ET	2.40 ± 0.65	2.36 ± 0.52	.174	.970	.703	2.96 ± 0.73‡	3.36 ± 0.85‡	<.001	.010	.779
UN	2.16 ± 0.38	2.22 ± 0.54				2.15 ± 0.57	2.65 ± 0.65†			
e′, cm/sec										
ET	0.17 ± 0.02	0.18 ± 0.02	.514	.978	.645	0.19 ± 0.03	0.19 ± 0.02	.088	.415	.837

(*Continued*)

Table 3 (*Continued*)

	Boys		*P* value			Girls		*P* value		
	Pre-PHV	Post-PHV	Training effect	Maturity effect	Interaction	Pre-PHV	Post-PHV	Training effect	Maturity effect	Interaction
UN	0.17 ± 0.02	0.17 ± 0.02				0.18 ± 0.03	0.18 ± 0.03			
A′, cm/sec										
ET	0.05 ± 0.01	0.06 ± 0.01	.010	.606	.977	0.07 ± 0.01	0.07 ± 0.01‡	.009	.344	.322
UN	0.06 ± 0.01	0.06 ± 0.01				0.08 ± 0.02	0.08 ± 0.01			

ET, Endurance trained; *UN*, untrained.
Data are from 2-way ANOVAs with training and maturity status as fixed factors. Independent-samples *t* tests were used to identify differences. Data are expressed as mean ± SD.
‡*P* < .05 trained versus untrained.
**P* < .001 trained versus untrained.
†*P* < .05 post-PHV versus pre-PHV.

soccer is predominantly >80% HR_{max},[26] whereas endurance athletes train primarily <80% HR_{max},[27] a disparity that may influence the nature of cardiac remodeling. The greater LV length without a proportional increase in LV diameter in the soccer players could impact myofiber angle, as ventricular shape and myofiber orientation are closely related.[7] A change in myofiber angle would place those athletes higher on the ascending arm of the parabolic relationship between LV twist and sphericity index as previously speculated.[9] In contrast to previous findings,[9] ventricular shape differences were not evident with training pre-PHV in the current study, which may explain the lack of training-related differences in twist at this stage. Similar to the study by Beaumont *et al.*,[9] we found minimal differences in LV structure pre-PHV, which also explains why there were no relationships found between torsion and structural variables pre-PHV. As stated previously, it is likely that profound cardiac remodeling does not occur prior to the growth spurt.[12]

Influence of Training Status on Systolic Twist Mechanics Post-PHV

Left ventricular twist mechanics are well known to be sensitive to changes in preload and afterload.[28] Post-PHV, systolic blood pressure

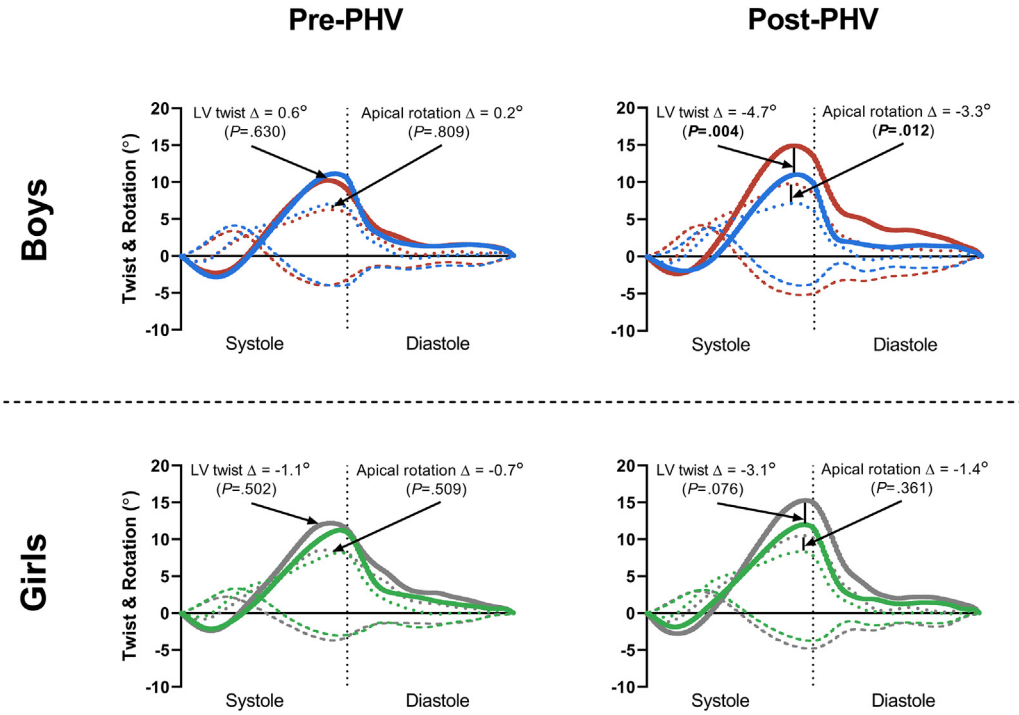

Figure 1 Temporal representation of LV twist mechanics pre- and post-PHV between trained and untrained children. *Blue* (—) and *red* (—) *lines* represent trained and untrained boys, respectively, and *green* (—) and *gray* (—) *lines* represent trained and untrained girls, respectively. *Solid* (—), *dotted* (····), and *dashed* (---) *lines* represent LV twist, apical and basal LV rotations, respectively. *Vertical dotted lines* represent aortic valve closure. *P* values represent between-group differences for 2-way ANOVA, with *t* test post hoc analysis.

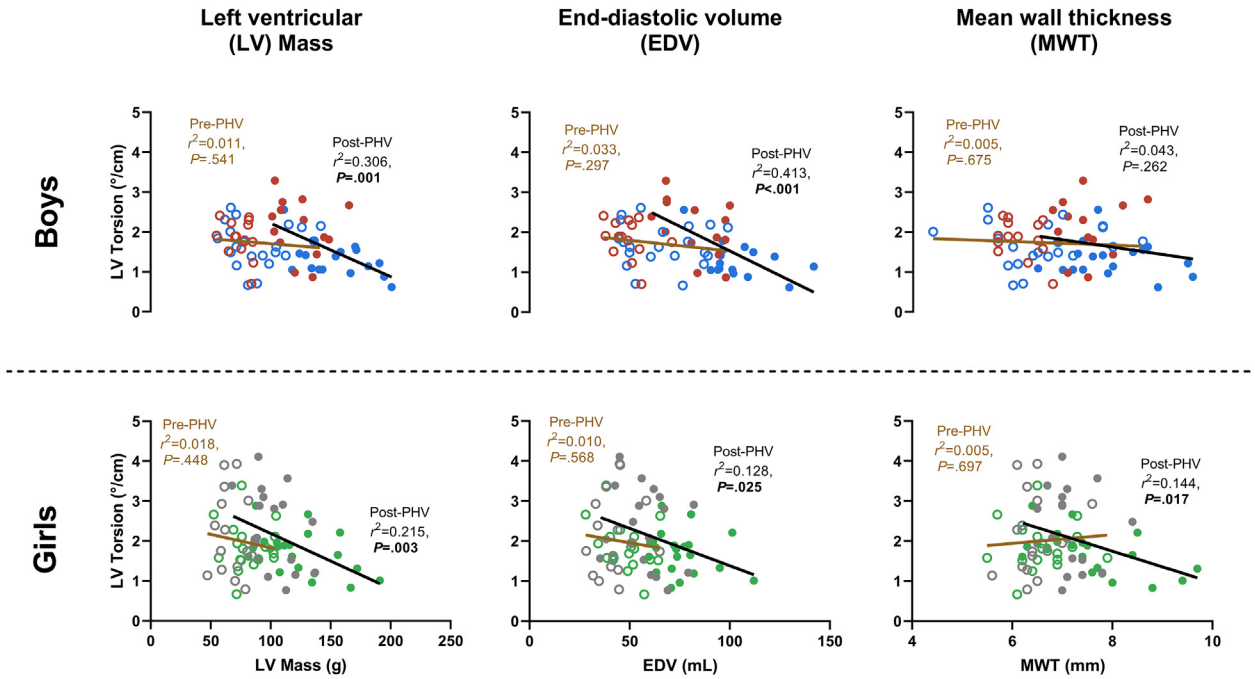

Figure 2 Linear regression analysis between LV torsion and LV mass; LV torsion and LV EDV; and LV torsion and MWT. Trained (●) and untrained (●) data for boys and trained (●) and untrained (●) data for girls are pooled for analysis at both pre-PHV *(open circles)* and post-PHV *(closed circles)*. *P* values are reported for the linear regression slope significance, and r^2 is reported to indicate the relationship's strength.

was higher in the current study compared with pre-PHV, although there was no significant effect of training. Therefore, the training-related differences in twist and torsion are unlikely to be due to afterload. Despite no effect of training on blood pressure, post-PHV trained children exhibit a greater LV mass.[12] The greater ventricular size will decrease the mechanical advantage for the subepicardial layer,[8] resulting in a lower twist due to a modified lever length.[29] The post-PHV adaptation of LV mass to training is also greater in boys compared with girls,[12,30] potentially due to an interaction between training and sex-specific hormones. These structural differences may explain why LV twist was lower in trained versus untrained post-PHV boys but did not reach significance in girls. Additionally, twist is closely related to LV length,[31] which was greater in both pre- and post-PHV trained boys but not trained girls, possibly providing further explanation for the sex disparity in LV twist. However, once twist is scaled for length, the lower torsion in trained boys and girls compared with untrained counterparts highlights that LV twist mechanics are reduced in both sexes but to a lesser extent in trained girls.

Our data support the notion that a reduction in LV twist with training occurs with prominent LV remodeling,[3] potentially as a result of an altered LV microstructure and subsequent rearrangement of the myofibers.[5] In support of this, we observed an inverse relationship for LV torsion with both LV mass and volumes in boys, while girls had the same inverse relationships, in addition to MWT, with torsion. The inverse relationship with EDV was in contrast with our hypothesis and a unique finding. Previous work has shown that with increased volume, increased twist follows.[3,32] However, these findings were observed with acute saline infusion[32] and after a shorter training period of 3 months,[3] respectively, as opposed to a longer training history as in the current study. It is likely that after a more chronic training period, lower torsion is the result of greater overall LV remodeling. This is supported by the inverse relationships we found between LV mass and torsion. It could be suggested that wall thickness is driving this inverse relationship, as found previously;[6] however, this relationship was only observed in girls in the current study. Thus, the lower torsion in trained groups does not appear to be specifically due to greater wall thickness but more likely to the greater overall LV eccentric remodeling post-PHV. This is supported by the greater training-related structural differences post-PHV in boys compared with girls given that the inverse relationships post-PHV for torsion with both LV mass and EDV were also stronger in boys versus girls. Collectively, these results illustrate the intricate link between LV form and function, with reduced LV twist mechanics following structural remodeling post-PHV, similar to the findings with chronic training in adults.[3] These results extend the understanding of cardiac adaptations in youth endurance athletes, highlighting that lower LV twist mechanics are only evident with structural remodeling post-PHV.

When all groups were pooled to identify the collective predictors of LV twist and torsion, endurance training was found to be a negative predictor for both. These were the most significant findings of our multiple linear regression analysis and primarily driven by the training-related differences post-PHV. These analyses also identified post-PHV maturity status as a positive predictor of LV twist, in agreement with previous work that showed an age-related increase in LV twist across maturation.[10] This finding is evidently driven by our untrained groups, which collectively have a greater LV twist post- versus pre-PHV. Interestingly, these findings highlight that, as previously shown, with healthy but sedentary aging, LV twist will increase. However, if children are endurance trained, LV twist will likely remain similar to pre-PHV values.

Influence of Training Status on Diastolic Function Pre- and Post-PHV

The significant post-PHV training-related differences in systolic twist could be expected to modify diastolic function. The energy developed at peak twist is stored within the extracellular collagen matrix and the cardiomyocytes due to the protein titin.[33] During early relaxation, this energy is rapidly released, helping to generate intraventricular pressure gradients, aiding passive ventricular filling.[34] A positive relationship therefore exists between untwisting velocity and LV twist.[34] Indeed, a linear increase in twist and untwisting velocity is shown from age 0 to 50 years but not with untwisting velocity expressed relative to LV twist.[10] This pattern was evident in our cohort, whereby the untwisting velocity is lower in trained post-PHV girls, alongside a lower torsion. A lower untwisting velocity at rest may be indicative of a greater capacity to augment intraventricular pressure gradients via increases in untwisting velocity during exercise. Indeed, cyclists have been shown to have a greater untwisting velocity than untrained controls during exercise.[35] The ability to augment filling during exercise is related to aerobic capacity,[36] highlighting the potential functional benefit of a lower resting untwisting velocity.

Clinical Perspective

Clinical echocardiography examination of LV mechanics in adolescents must consider training status. An endurance-trained, post-PHV adolescent who presents with significant structural remodeling will demonstrate lower systolic twist mechanics compared with normal development. This adaptation is likely to be physiological in nature and may provide the sports cardiologist with an additional tool to differentiate from pathological adaptation. Endurance training in children does not lead to a consistent adaptation in traditional measures of LV function or LV strain.[30] However, LV torsion appears to be comparatively more sensitive to training adaptations.[2] This might help clinicians to confirm physiological adaptation, especially if the suspected pathology is known to increase twist, such as hypertrophic cardiomyopathy or conditions associated with a pressure overload.[15] To further confirm the presence of normal LV mechanics, stress echocardiography may be required. If an assessment of LV function in a child undertaking endurance training is ongoing across pubertal development, these findings should be considered during clinical assessment.

Limitations

We recognize that the cross-sectional nature of our experimental design does not enable us to establish causality for training-related adaptations. A longitudinal training intervention spanning from childhood to adolescence is required to further understand the influence of maturation on training-related cardiac adaptations. However, our participants had been training for at least 12 months, which has previously been shown to lead to structural cardiac adaptations similar to those of elite adult athletes.[37] Our LV structural parameters were quantified using echocardiography rather than magnetic resonance imaging. However, echocardiography is recognized to provide a more accurate representation of LV function due to its higher imaging frame rate,[38] which was the focus of our hypotheses. Echocardiography is also frequently used for the assessment of LV structure[22] and has been validated in children.[39] Lastly, while a limited number of participants were included, our prospective power analysis indicated that the current study was sufficiently powered to identify differences in the key outcome variables.

CONCLUSION

Left ventricular twist and torsion do not differ significantly between trained and untrained pre-PHV children, where structural adaptation is limited. Post-PHV, where prominent exercised-induced LV remodeling has occurred, LV twist and torsion are lower in endurance-trained adolescents versus untrained counterparts. These findings highlight that the maturational threshold for significant structural remodeling with exercise training also influences the nature and degree of functional remodeling.

CONFLICTS OF INTEREST

None.

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