Adaptation of left ventricular twist mechanics in exercise-trained children is only evident after the adolescent growth spurt.

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Endurance trained children have lower left ventricular twist mechanics but only after the adolescent growth spurt, where athletic structural and functional remodelling are related @CardiffMetSES @MikeStembridge

Abstract

Background: The extent of structural cardiac remodelling 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. Whilst structural cardiac remodelling has been shown to be more prominent post-peak height velocity (PHV), it remains to be determined how this maturation-dependent structural remodelling influences LV twist. Therefore, we aimed to (I) compare LV-twist mechanics between trained *vs*. untrained children pre- and post-PHV, and (ii) investigate how LV structural variables relate to LV-twist mechanics pre- and post-PHV. Methods: LV 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 categorised 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 *vs.* untrained boys (twist: P=0.630; torsion: P=0.382) or girls (twist: P=0.502; torsion: P=0.316), and LV-twist mechanics were not related with any LV structural variables (P>0.05). Post-PHV, LV twist was lower in trained *vs*. untrained boys (P=0.004), with torsion lower in trained groups, irrespective of sex (boys: P<0.001; girls: P=0.017). Moreover, LV torsion was inversely related to LV mass (boys: r=-0.55, P=0.001; girls: r=-0.46, P=0.003) and end-diastolic volume (boys: r=-0.64, P<0.001; girls: r=-0.36, P=0.025) 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 remodelling were related.

Keywords: echocardiography, myocardium, twist, youth, exercise.

Abbreviations:

A – Late ventricular filling velocity

A′ – Late ventricular tissue velocity

E – Early ventricular filling velocity

e′ – Early ventricular tissue velocity

EDV – End-diastolic volume

ESV – End-systolic volume

HR_{max} – Maximum heart rate

LV – Left ventricle.

MWT – Mean wall thickness

PHV – Peak height velocity

S′ – Systolic ventricular tissue velocity

$\dot{V}O_{2max}$ – Maximal oxygen consumption

Introduction

Endurance training results in an enlarged left ventricular (LV) end-diastolic volume (EDV) and proportional LV hypertrophy in adults [1]. LV structural adaptation to training can influence functional remodelling, 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 energy efficiency and redistribution of fibre 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 remodelling [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 (~three months) of intensified training results in an acute enlargement of EDV and an increase in LV-twist mechanics [3]. When training is continued for ~three 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 remodelling 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 with an increase in LV twist [10]. However, when LV twist is normalised 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 remodelling evident pre-adolescence, whilst a greater proportional increase in LV mass and blood-volume expansion is observed post-puberty [11,12]. LV twist data in trained pre-adolescent children are sparse, yet twist appears to be increased [9,13] and coinciding 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 remodelling is essential [14], especially throughout maturation due to the vast physiological changes. When determining whether an athlete presentation is pathological, all possible diagnostic tools should be considered. LV 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 paediatric athletic patients.

We speculate that greater LV remodelling post-PHV could decrease twist, superseding the aforementioned maturational LV growth which can otherwise increase twist. We therefore aimed to compare LV twist and torsion between endurance trained and untrained children pre- and post-PHV. We hypothesised (1) LV twist and torsion would be elevated in trained *vs.* untrained pre-PHV children and torsion would be positively related to EDV, but (2) LV twist and torsion would be lower in trained *vs.* 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 categorised 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 datasets (*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 previously been described in detail [12]. Trained children were undertaking at least three 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 by maturity-status and sex, therefore permitting the 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 h/week; post-PHV, 0.9 ± 1.1 h/week; untrained girls: pre-PHV, 1.0 ± 0.9 h/week; post-PHV, 0.5 ± 0.8 h/week). Exclusion criteria included hypertension,

smoking, cardiovascular disease, and obesity according to age- and sex-specific body mass index cut-offs of the International Obesity Task Force criteria [17].

Experimental design

Participants attended the laboratory on two 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, whilst 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 categorised as either pre- or post-PHV based on a maturity offset value below or above zero, respectively, as originally recommended [16]. Following 10 minutes of supine rest, blood pressure was measured using an automated sphygmomanometer (Omron Healthcare, Hoofddorp, Netherlands). 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 an incremental cardiopulmonary exercise testing on an upright cycle ergometer (Lode, Excalibur, Groningen, Netherlands). During the second visit, following 10 min 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 previously [20]. Participants cycled at a continuous pace of 75-85 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 previously described [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, Horten, Norway) using a 1.5-4 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, Horton, Norway). 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. LV mass was calculated using the area-length method, derived from measures of LV length and mean wall thickness (MWT; [22]). LV 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 four-chamber view. MWT was calculated from cross-sectional areas of the epicardium and the endocardium in a short-axis view at the papillary level [22]. EDV, end-systolic volume (ESV) and ejection fraction were assessed using the Simpsons 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 analysing 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 four chamber view [7]. Intra-

observer coefficient 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 four-chamber view, with the sample volume at the valve tips. Tissue Doppler imaging (TDI) 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, Horton, Norway). 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, Stuttgart, Germany), as described previously [5]. LV twist was calculated as the time-aligned difference between basal and apical rotation. To normalise 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 ANOVA with two 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 analysed 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 (version 24, Chicago, IL) and GraphPad (Prism Version 8.1.1, GraphPad Software, San Diego, CA).

LV twist has not previously been investigated in relation to both training and maturation. However, Beaumont *et al*. [9] found a difference of 4.58º in twist between pre-adolescent soccer players *vs*. controls with SD=6.27º. 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 α=0.05.

Results

Anthropometric, haemodynamic, 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, whilst 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 *vs.* untrained post-PHV groups (P<0.001), 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 trained and untrained groups post- *vs.* 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- *vs.* pre-PHV boys, whereas LV length index was only greater in trained post- *vs.* 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- *vs.* pre-PHV.

Pre-PHV, absolute LV length, LV mass, EDV and ESV were greater in trained *vs.* untrained boys (Table 2). In pre-PHV girls, absolute LV mass was greater between trained *vs.* 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 *vs.* 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 *vs* untrained boys and girls. Post-PHV, absolute measures of LV length, LV mass, EDV and ESV were greater in trained *vs.* untrained groups for both boys and girls (Table 2). All measured relative LV structure variables were greater in post-PHV trained *vs.* 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 *vs.* 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=0.067 and P=0.708, respectively) or girls (P=0.094 and P=0.067, 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 (Table3).

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 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. LV torsion was lower in both trained boys and girls post-PHV compared with untrained groups (Table 3; boys: $P<0.001$; girls: $P=0.017$). Similarly, LV twist velocity was lower in trained boys and girls post-PHV compared with untrained counterparts (Table 3; boys: $P=0.003$; girls: $P=0.003$). 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). Whilst the only training-related difference in diastolic function between pre-PHV girls was a significantly lower late filling velocity in trained *vs.* untrained groups, which led to a greater E/A (Table 3).

Post-PHV, a significantly slower untwisting velocity was identified for trained *vs.* untrained girls (Table 3). Despite this, early diastolic filling velocities were not significantly different between post-PHV trained *vs*. untrained groups. In post-PHV trained *vs*. 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 *vs*. untrained post-PHV girls, whilst 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=0.010) and torsion (P<0.001). Additionally, post-PHV maturity-status was identified as a positive predictor of LV twist (P=0.031), whereas male sex was identified as a negative predictor of LV torsion (P=0.023).

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 two hypotheses, the main findings were: (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. (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 remodelling.

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 *vs.* untrained groups, and with previous work in pre-adolescent 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 with training status pre-PHV. However, it has been shown that pre-adolescent soccer players had a more elongated LV [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 remodelling between the current study and previous [9], may be due to differences in training intensities between endurance athletes and soccer players. Both elicit a high haemodynamic load, the intensity distribution in soccer is predominantly >80% HR_{max} [26], whereas endurance athletes train primarily <80% HR_{max} [27], a disparity which may influence the nature of cardiac remodelling. 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 remodelling does not occur prior to the growth spurt [12].

Influence of training status on systolic twist mechanics post-PHV

LV-twist mechanics are well known to be sensitive to changes in preload and afterload [28]. Post-PHV, systolic blood pressure 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 *vs*. 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 remodelling [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, whilst 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 three months [3], respectively, as opposed to a longer training history in the current study. It is likely that after a more chronic training period, lower torsion is the result of greater overall LV remodelling. 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 the greater overall LV eccentric remodelling, 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 *vs.* girls. Collectively, these results illustrate the intricate link between LV form and function, with reduced LV twist mechanics following structural remodelling 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 remodelling 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 which 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- vs. 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 remodelling 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 recognise 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 recognised 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, whilst 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.

Conclusions

LV 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 remodelling has occurred, LV twist and torsion are lower in endurance trained adolescents *vs*. untrained counterparts. These findings highlight that the maturational threshold for significant structural remodelling with exercise training also influences the nature and degree of functional remodelling.

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Author Contributions

DRP, RSL, RES, JLO and MS contributed to the conception and study design. All authors were involved with acquisition, or analysis and interpretation of data. DRP and MS drafted the manuscript, and all authors were involved in revising it critically for important intellectual content. All authors approved the final version of the manuscript.

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Table 1. Participant characteristics and cardiovascular measurements

		Boys					Girls				
Anthropometric characteristics		**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§	*P*=0.771	***P*<0.001**	***P*=0.007**	10.5±1.2	14.0±1.4§	*P*=0.275	***P*<0.001**	*P*=0.362
	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§	*P*=0.906	***P*<0.001**	*P*=0.053	-1.3±1.0	1.8±1.1§	*P*=0.164	***P*<0.001**	*P*=0.381
	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§	*P*=0.721	***P*<0.001**	*P*=0.517	144.0±9.7	164.3±6.8§	***P*=0.023**	***P*<0.001**	*P*=0.249
	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§	*P*=0.544	***P*<0.001**	*P*=0.810	35.1±5.6	53.6±8.5§	*P*=0.933	***P*<0.001**	*P*=0.497
	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†§	***P*<0.001**	***P*<0.001**	*P*=0.254	74±9	62±7†§	***P*=0.001**	***P*<0.001**	*P*=0.987
	UN	72±7	63±8‡				81±16	70±7‡			
Blood pressure											
Systolic BP (mm Hg)	ET	105±8	116±9§	*P*=0.599	***P*<0.001**	*P*=0.273	104±8	111±7‡	*P*=0.105	***P*<0.001**	*P*=0.833
	UN	102±8	117±7§				101±7	109±6§			
Diastolic BP (mm Hg)	ET	60±7	62±5	*P*=0.431	*P*=0.891	*P*=0.159	63±7	65±6	*P*=0.529	*P*=0.482	*P*=0.653
	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†	***P*<0.001**	*P*=0.056	*P*=0.341	50.5±5.8†	50.9±5.8†	***P*<0.001**	*P*=0.820	*P*=0.127
	UN	43.4±6.1	48.3±3.7				41.9±5.8	37.8±5.7			

BP, blood pressure; *ET*, endurance trained; *PHV*, peak height velocity; *UT*, untrained. Data from two-way ANOVAs with training and maturity status as fixed factors. Independent samples *t*-tests were used to identify differences. Data expressed as mean±SD. **P*<0.05, †*P*<0.001 trained *vs*. untrained; ‡*P*<0.05, §*P*<0.001 post-PHV *vs*. pre-PHV.

Table 2. Left ventricular structure

		Boys					**Girls**				
		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§	***P*=0.018**	***P*<0.001**	*P*=0.781	6.6±0.6	7.5±1.0‡	*P*=0.066	***P*<0.001**	*P*=0.573
	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†§	***P*<0.001**	***P*<0.001**	*P*=0.934	63±6	75±6*§	***P*=0.002**	***P*<0.001**	*P*=0.364
	UN	62±5	77±7§				60±4	69±6§			
LV mass (g)	ET	91±25*	155±28*§	***P*<0.001**	***P*<0.001**	*P*=0.219	81±16*	124±30*§	***P*<0.001**	***P*<0.001**	*P*=0.333
	UN	74±10	125±20§				68±12	102±18§			
EDV (mL)	ET	66±18*	104±16†§	***P*<0.001**	***P*<0.001**	*P*=0.659	48±10	75±14†§	***P*<0.001**	***P*<0.001**	***P*=0.030**
	UN	49±9	83±14§				43±10	58±13§			

ESV (mL)	ET	27±7*	42±7*§	***P*<0.001**	***P*<0.001**	*P*=0.637	18±4	30±7†§	***P*<0.001**	***P*<0.001**	***P*=0.004**
	UN	19±5	33±7§				16±4	21±6‡			
Sphericity index	ET	1.7±0.2	1.7±0.2	***P*=0.031**	*P*=0.904	*P*=0.781	1.6±0.2	1.6±0.2	*P*=0.669	*P*=0.267	*P*=0.517
	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*	***P*=0.023**	*P*=0.475	*P*=0.525	4.6±0.4	4.6±0.5	*P*=0.694	*P*=0.131	*P*=0.191
	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†	***P*<0.001**	*P*=0.323	*P*=0.952	44±4	45±3	*P*=0.075	*P*=0.795	*P*=0.098
	UN	43±3	43±4				44±3	43±4			
LV mass/LBM (g/kg)	ET	2.7±0.3	2.9±0.4†	***P*<0.001**	*P*=0.319	*P*=0.085	2.9±0.4	2.9±0.4*	***P*=0.003**	*P*=0.571	*P*=0.481
	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†	***P*<0.001**	*P*=0.716	*P*=0.985	1.7±0.3	1.8±0.3*	***P*=0.004**	*P*=0.312	***P*=0.028**
	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*	***P*<0.001**	*P*=0.761	*P*=0.997	0.6±0.1	0.7±0.1†	***P*=0.011**	*P*=0.502	***P*=0.004**
	UN	0.6±0.1	0.6±0.1				0.6±0.1	0.5±0.1			
Clinically scaled LV structure											
MWT index ($mm/(m^2)^{0.4}$)	ET	5.9±0.7	6.4±0.6*‡	***P*=0.005**	***P*=0.004**	*P*=0.724	6.2±0.4	6.3±0.7	*P*=0.091	*P*=0.882	*P*=0.299
	UN	5.6±0.4	6.0±0.4‡				6.1±0.4	6.0±0.3			
LV length index ($mm/(m^2)^{0.45}$)	ET	64±5†	67±4†‡	***P*<0.001**	***P*=0.005**	*P*=0.729	59±5	61±5*	***P*=0.003**	*P*=0.295	*P*=0.169
	UN	56±5	60±5				57±3	57±4			
LV mass index ($g/(m^2)^{1.25}$)	ET	66±9†	79±11†§	***P*<0.001**	***P*<0.001**	*P*=0.159	66±8*	71±12†	***P*<0.001**	*P*=0.284	*P*=0.198
	UN	55±6	62±6‡				59±8	58±6			
EDV index	ET	48±8†	51±5†	***P*<0.001**	***P*=0.030**	*P*=0.797	39±7	43±7†	***P*<0.001**	*P*=0.989	***P*=0.012**

($mL/(m^2)^{1.3}$)	UN	36±7	40±6				36±5	32±6			
ESV index	ET	20±3†	21±3†	***P*<0.001**	***P*=0.047**	*P*=0.772	14±3	17±3†‡	***P*<0.001**	*P*=0.888	***P*=0.001**
($mL/(m^2)^{1.3}$)	UN	14±3	16±3				14±3	12±3‡			

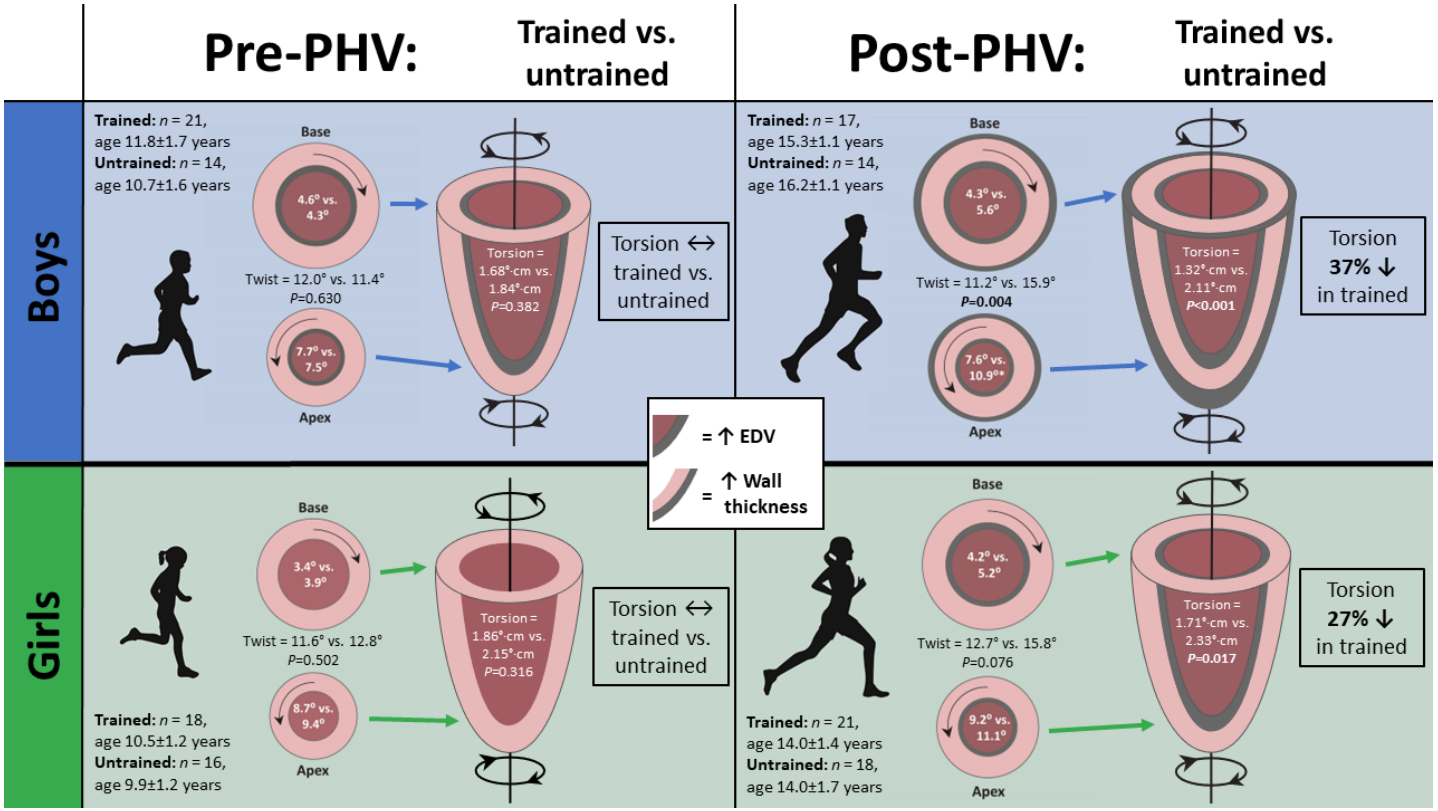
EDV, end-diastolic volume; *ESV*, end-systolic volume; *ET*, endurance trained; *LV*, left ventricular; *MWT*, mean wall thickness; *PHV*, peak height velocity; *UN*, untrained. Data from two-way ANOVAs with training and maturity status as fixed factors. Independent samples *t*-tests were used to identify differences. Data expressed as mean±SD. **P*<0.05, †*P*<0.001 trained *vs*. untrained; ‡*P*<0.05, §*P*<0.001 post-PHV *vs*. pre-PHV.

Table 3. Left ventricular function and twist mechanics

		Boys					**Girls**				
		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			
Left ventricular systolic function											
Apical rotation (°)	ET	7.7±2.6	7.6±3.2†	***P*=0.036**	***P*=0.029**	***P*=0.018**	8.7±1.9	9.5±4.2	*P*=0.163	*P*=0.238	*P*=0.533
	UN	7.5±2.1	10.9±3.7‡				9.4±4.3	10.9±5.2			
Basal rotation (°)	ET	-4.6±2.0	-4.3±3.0	*P*=0.455	*P*=0.505	*P*=0.256	-3.4±2.9	-4.7±3.6	*P*=0.350	*P*=0.173	*P*=0.747
	UN	-4.3±3.1	-5.6±3.1				-3.9±3.4	-4.6±2.8			
Twist (°)	ET	12.0±4.1	11.2±3.4*	***P*=0.040**	*P*=0.067	***P*=0.008**	11.6±3.7	12.7±4.1	*P*=0.083	*P*=0.094	*P*=0.425
	UN	11.4±3.0	15.9±4.9‡				12.8±6.0	15.8±6.3			
Torsion (°·cm)	ET	1.7±0.5	1.3±0.4†‡	***P*=0.001**	*P*=0.708	***P*=0.021**	1.9±0.6	1.7±0.5*	***P*=0.018**	*P*=0.952	*P*=0.377

	UN	1.8±0.5	2.1±0.7				2.2±1.0	2.3±1.0			
Twist velocity (º/sec)	ET	85±21	80±17*	***P*=0.004**	*P*=0.187	***P*=0.037**	84 ± 25*	76 ± 19*	***P*<0.001**	*P*=0.592	*P*=0.588
	UN	90±24	111±35				107 ± 40	107 ± 40			
LV ejection fraction (%)	ET	59±3	59±4	*P*=0.146	*P*=0.995	*P*=0.834	63±5	61±4	*P*=0.356	*P*=0.766	***P*=0.017**
	UN	61±5	60±5				61±3	64±4			
S′ (cm/sec)	ET	0.09±0.01	0.10±0.01‡	*P*=0.612	***P*=0.011**	*P*=0.413	0.10±0.01	0.10±0.01	*P*=0.837	*P*=0.089	*P*=0.100
	UN	0.10±0.01	0.10±0.01				0.09±0.01	0.10±0.01			
Left ventricular diastolic function											
Untwist velocity (º/sec)	ET	-107±30	-99±25	*P*=0.391	*P*=0.673	*P*=0.104	-129±39	-111±36*	*P*=0.133	*P*=0.610	***P*=0.009**
	UN	-102±38	-117±20				-117±50	-146±53			
E (cm/sec)	ET	0.96±0.14	0.91±0.11	*P*=0.577	***P*=0.009**	*P*=0.180	1.01±0.18	1.01±0.18	*P*=0.763	*P*=0.517	*P*=0.491
	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	*P*=0.395	***P*=0.039**	*P*=0.167	0.35±0.06†	0.32±0.08*	***P*<0.001**	***P*=0.005**	*P*=0.335
	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	*P*=0.174	*P*=0.970	*P*=0.703	2.96±0.73*	3.36±0.85*	***P*<0.001**	***P*=0.010**	*P*=0.779
	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	*P*=0.514	*P*=0.978	*P*=0.645	0.19±0.03	0.19±0.02	*P*=0.088	*P*=0.415	*P*=0.837
	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	***P*=0.010**	*P*=0.606	*P*=0.977	0.07±0.01	0.07±0.01*	***P*=0.009**	*P*=0.344	*P*=0.322
	UN	0.06±0.01	0.06±0.01				0.08±0.02	0.08±0.01			

A, late diastolic filling velocity; *A′*, late diastolic tissue velocity; *E*, early diastolic filling velocity; *e′*, early diastolic tissue velocity; *ET*, endurance trained; *PHV*, peak height velocity; *UN*, untrained. Data from two-way ANOVAs with training and maturity status as fixed factors. Independent samples *t*-tests were used to identify differences. Data expressed as mean±SD. **P*<0.05, †*P*<0.001 trained *vs*. untrained; ‡*P*<0.05, §*P*<0.001 post-PHV *vs*. pre-PHV.



Central Illustration. Schematic summary outlining basal and apical rotation, twist and torsion differences between trained and untrained boys and girls pre- and post-peak height velocity (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 both 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 remodelling. Collectively, this highlights the influence of endurance training on LV twist mechanics is only apparent post-PHV.

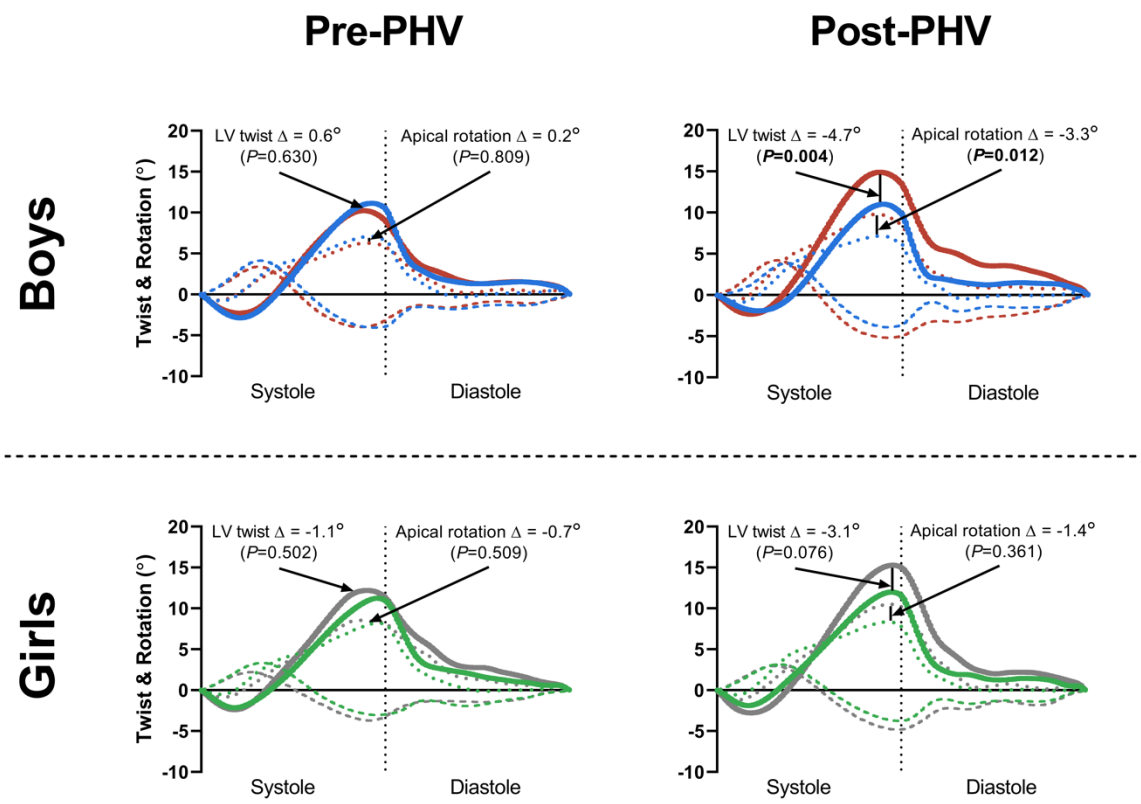


Figure 1. Temporal representation of left ventricular (LV) twist mechanics pre- and post-peak height velocity (PHV), between trained and untrained children. Blue (▬) and red (▬) lines represent trained and untrained boys, respectively, and green (▬) and grey (▬) lines represent trained and untrained 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 two-way ANOVA, with t-test post hoc analysis.

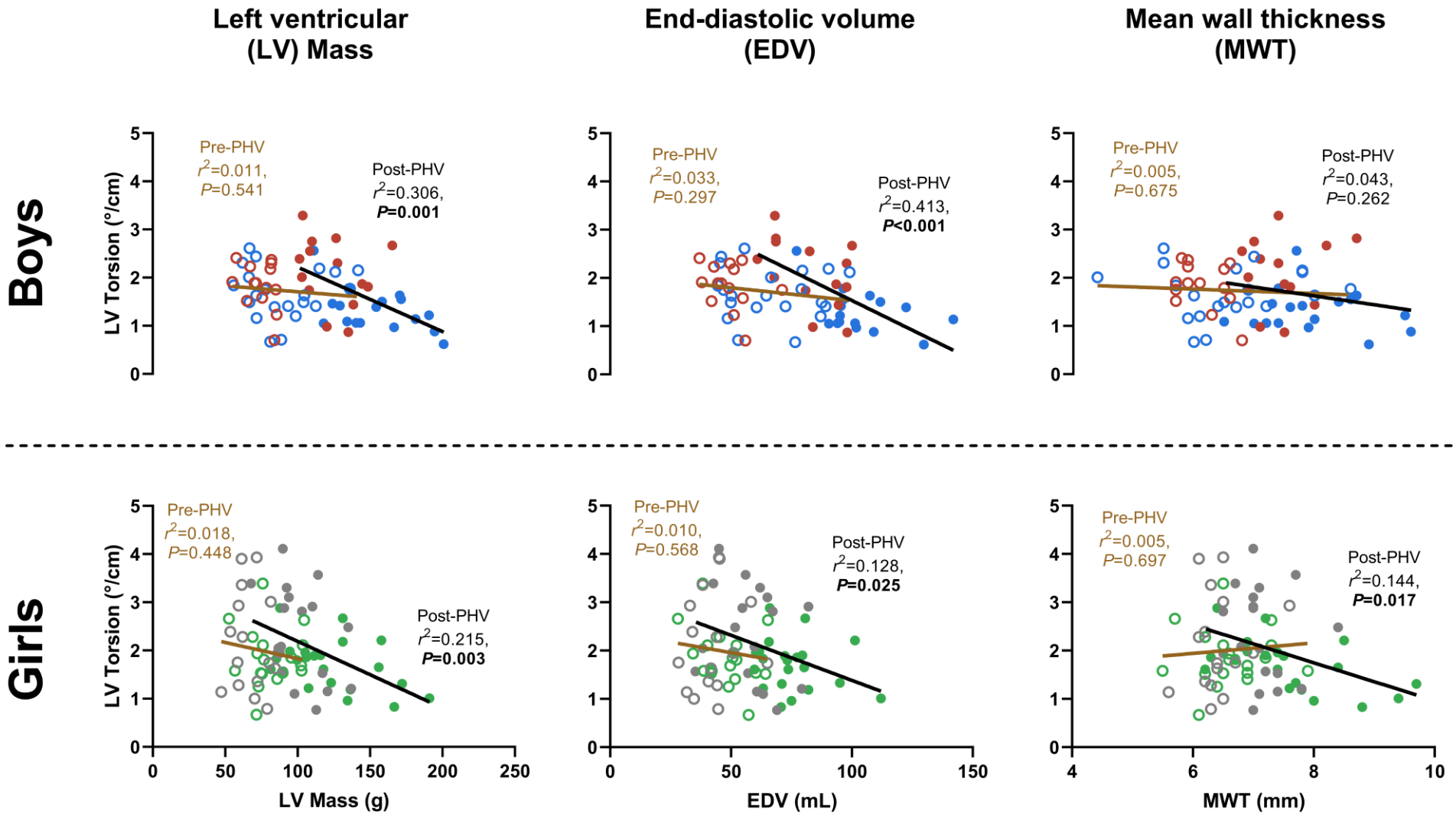

Left ventricular (LV) Mass
End-diastolic volume (EDV)
Mean wall thickness (MWT)
Boys
Girls
LV Torsion (°/cm)
Pre-PHV
r²=0.011,
P=0.541
Post-PHV
r²=0.306,
P=0.001
Pre-PHV
r²=0.033,
P=0.297
Post-PHV
r²=0.413,
P<0.001
Pre-PHV
r²=0.005,
P=0.675
Post-PHV
r²=0.043,
P=0.262
Pre-PHV
r²=0.018,
P=0.448
Post-PHV
r²=0.215,
P=0.003
Pre-PHV
r²=0.010,
P=0.568
Post-PHV
r²=0.128,
P=0.025
Pre-PHV
r²=0.005,
P=0.697
Post-PHV
r²=0.144,
P=0.017
LV Mass (g)
EDV (mL)
MWT (mm)

Figure 2. Linear regression analysis between left ventricular (LV) torsion and LV mass; LV torsion and LV end-diastolic volume (EDV); and LV torsion and mean wall thickness (MWT). Trained (●) and untrained (●) data for boys, and trained (●) and untrained (●) data for girls are pooled for analysis at both pre-peak height velocity (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.