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ORIGINAL ARTICLE

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The effect of long-term soccer training on left ventricular structure and function in elite male youth soccer players

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Abstract

Aims: Cardiac adaptations in elite, male adolescent youth soccer players have been demonstrated in relation to training status. The time course of these adaptations and the delineation of the influence of volatile growth phases from the training effect on these adaptations remain unclear. Consequently, the aims of the study were to evaluate the impact of 3 years of elite-level soccer training on changes in left ventricular (LV) structure and function in a group of highly trained elite youth male soccer players (SP) as they transitioned through the pre-to-adolescent phase of their growth.

Methods: Twenty-two male youth SP from the highest Level of English Premier League Academy U-12 teams were evaluated once a year for three soccer seasons as the players progressed from the U-12 to U-14 teams. Fifteen recreationally active control participants (CON) were also evaluated over the same 3-year period. Two-dimensional transthoracic echocardiography was used to quantify LV structure and function.

Results: After adjusting for the influence of growth and maturation, training-induced increases in Years 2 and 3 were noted for: LV end diastolic volume (LVEDV; $p = 0.02$) and LV end systolic volume (LVESV; $p = 0.02$) in the SP compared to CON. Training-induced decrements were noted for LV ejection fraction (LVEF; $p = 0.006$) and TDI-S′ ($p < 0.001$).

Conclusions: An increase in training volume (Years 2 and 3) were aligned with LV volumetric adaptations and decrements in systolic function in the SP that were independent from the influence of rapid somatic growth. Decrements in systolic function were suggestive of a functional reserve for exercise.

KEYWORDS

cardiac adaptations, elite youth soccer, left ventricle

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1 | INTRODUCTION

The athlete's heart represents the normal physiologic adaptation to a training stimulus, with no associated clinical consequences in young athletes.[1] These assertions are primarily based on an array of cross-sectional studies.[2–6] This evidence has provided valuable insights into the structural and functional cardiac adaptations in relation to the training status of the young athlete. Cross-sectional studies are, however, unable to fully delineate the influence of growth and maturation from the training stimulus on cardiac structural and functional adaptation in the young athlete. Consequently, the efficacy of the training stimulus cannot truly be identified with these study designs, as growth and maturation per se exerts training-like cardiac structural and functional adaptations.[7] This has taken the form of exponential increases in left ventricular (LV) mass and stroke volume (SV) around the adolescent growth spurt (peak height velocity-PHV) in the absence of any training stimulus. These adaptations are potentially triggered by the presence of androgenic hormones at this time of rapid growth.[7]

Evidence from cross-sectional studies in pre-adolescent athletes, however, challenges the theory that training-induced cardiac adaptations require the presence of sex- and growth-related hormones.[8,9] These studies Zdravkovic et al.[5] and Pela et al.[3] have demonstrated the presence of the athlete's heart in pre-adolescent athletes, at a time when the androgenic stimulus is low. In order to understand the interaction between the training stimulus and the influence of growth and maturation as the athlete approaches their peak height velocity (PHV) the need for well-designed, longitudinal studies is required.[10,11]

The lack of a definitive growth and maturation time point, where cardiac adaptation occurs in pre-and adolescent athletes suggests that training intensity and volume may be the key stimuli for LV adaptation in youth athletes.[12,13] Based on this assumption, one of the most potent sports to trigger LV structural and functional adaptation in both the short- and long-term is soccer,[14] where match-play and training exposure is at 85% of maximal heart rate in youth soccer players.[15] A constellation of cross-sectional studies in both elite male and female pre- and adolescent soccer players have demonstrated greater chamber size, wall thickness, and larger LV twist and greater diastolic strain rate in the soccer players compared to their recreationally active peers.[3–6,16] These studies have provided useful information on the potential influence of training status on LV structural and functional adaptation in elite youth soccer players, but they are unable to provide any insight on the time course of evolution of these adaptations across the critical pubertal growth stage.[1]

Our research group have previously demonstrated RV and LV structural and functional adaptations through a series of cross-sectional athlete's heart studies, with respect to the training status of the pre-adolescent[2,16,17] and adolescent elite youth soccer players[4,6] compared to age-matched recreationally active control participants (CON). It was unclear, however, the time course over which these adaptations occurred and as with any cross-sectional study design, we were unable to delineate fully the influence of growth and maturation from the training stimulus on the cardiac adaptations noted in the elite youth soccer players. Consequently, the aims of this study were to evaluate the impact of 3 years of elite-level soccer training on changes in LV structure and function using two-dimensional transthoracic echocardiography in a group of highly trained elite male youth SP as they transitioned through the pre-to-adolescent phase of their growth and development. It was hypothesized that the SP would demonstrate training-related adaptations in morphologic and functional characteristics of the LV that would be greater than that seen from the influence of growth and maturation alone.

2 | MATERIALS AND METHODS

2.1 | Participants

Twenty-two elite male youth SP from two Category 1 (highest Level) English Premier League Academy U-12 teams were evaluated at rest, once a year for three consecutive soccer seasons as the players progressed from the U-12 to U-14 teams. At the same time, a group of 15 recreationally active, but not systematically trained CON were evaluated using the same protocol over the same 3-year time period. Stature, sitting height, and body mass were measured. In order to obtain a somatic marker of growth and maturation, maturity status was subsequently quantified using the Sherar et al., maturity offset method.[18] Body surface area was calculated using the Dubois and Dubois formula[19]: $\text{Mass (kg)}^{0.425} \times \text{Stature (cm)}^{0.725} \times 0.07184$.

Written assent was provided by all participants and written informed consent was provided by all parents/legal guardians. All procedures performed in this study were in accordance with the ethical standards of the Declaration of Helsinki and the study was approved by Staffordshire University Research Ethics Committee.[20]

2.2 | Study design and pre-participant requirements

The study employed a 3-year observational assessment of LV structure and function in highly trained,

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male pre-adolescent SP and CON, as they transitioned through the pre-adolescent to adolescent growth stage. All testing took place at the training grounds of the two soccer clubs and at a local school for the CON participants. Participants were instructed to avoid exercise on the day preceding the test. Also, all SP were tested prior to the start of the training session and with a minimum of 24 h after any training or match-play. For the CON, they were tested prior to any physical education classes and if they engaged in any recreational sport activity, they were tested a minimum of 24 h after these activities. Furthermore, all participants were also informed to refrain from consuming any drinks containing sugar or caffeine as well as the consumption of any food in the 2 h preceding the testing session. They were permitted to drink water ad libitum in the 2 h preceding the testing. Physical activity and training questionnaires[6] were completed prior to the testing each year.[20] The players were evaluated approximately halfway through their competitive season each year and no players had sustained any long-term injuries that required them to spend prolonged periods of time away from training.

2.3 | Training and physical activity profiles of the participants across the 3 years of the longitudinal study

Table 1 highlights the training load and physical activity profiles of both the SP and CON. The training load for the SP at both Academies and across all 3 years incorporated pitch-based, high intensity soccer training and matches. One of the clubs also included 45 min of gym-based resistance training for the U-12 age group. Both Academies introduced 1 h of gym-based resistance work at the U-13 and U-14 age groups. In addition to this, players from both clubs took part in physical education classes at school and sports club activities such as rugby union, cycling, and cross-country running. All player training load data were taken from the Academy database from the respective clubs and the physical activity participation times came from a validated self-report questionnaire.[6]

TABLE 1 Training load and physical activity characteristics of the soccer players (SP) and controls (CON) across the 3 years of the study.

SP	U12 (h/week)	U13 (h/week)	U14 (h/week)
Training load (h/week)	7.3	10.5	10.5
Additional physical activity (h/week)	3.2	2.6	2.9
Total activity load (h/week)	10.5	13.1	13.4
CON (years of age)	11.7	12.6	13.6
Physical activity (h/week)	3.3	3.9	3.4

The recreationally active, but not systematically trained CON group took part in physical education in school and other sports activities away from school such as football, track and field, cycling, and martial arts. Based on these data, there was a 3–4 times higher volume of training and activity in the SP per year compared to the CON across the 3 years of the study.[20]

2.4 | Data collection

2.4.1 | Echocardiographic measurements: Indices of LV structure

All echocardiographic procedures each year were performed by the same experienced sonographer (DO) using a commercially available ultrasound system (VividQ Ultrasound System, GE Ltd, Horton, Norway). Two-dimensional images from the apical and parasternal orientations (long and short axis) were obtained with the participants in the left lateral decubitus position. Images were subsequently stored for offline data analyses (Echopac, Version 6.0, GEMedical, Horton, Norway).[20]

Measurements of resting LV dimensions (LV end diastolic dimension [LVED], LV end systolic dimension [LVES]), and diastolic wall thicknesses (interventricular septum [IVSd], and posterior wall [PWd]) were made in accordance with American Society of Echocardiography (ASE) guidelines[21] using two-dimensional parasternal long and short axes views, respectively. LVED and LVES parameters were scaled to body surface area$^{0.5}$.[22] LVMass was derived from the following formula[22]: LV Mass = 0.8 × (1.04 × (((LVEDD + IVSd + PWd)^3 − LVEDD^3))) + 0.6 and adjusted linearly for body surface area (LVMassIndex) and relative wall thickness (RWT) was calculated according to ASE guidelines to provide a marker of LV geometry. LV end-diastolic volume (LVEDV) and LV end-systolic volume (LVESV) were determined using the Simpson's biplane method from apical 4- and 2-chamber views.[21] LVEDV and LVESV were then allometrically scaled[22] to BSA$^{1.5}$. Concentricity was calculated as the ratio between LV mass/LVEDV$^{0.667}$.

2.4.2 | Echocardiographic measurements: Indices of LV function

The apical 4-chamber view allowed for the determination of peak early diastolic filling velocity (E). Pulsed wave

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tissue-Doppler imaging determined peak longitudinal mitral and lateral annular velocities and these were averaged in both systole (S′) and early diastole (E′) and average E/E′ was also derived. Ejection fraction (LVEF) was calculated from Simpsons biplane-derived LV volumes.

Stroke volume (SV) was calculated using continuous-wave Doppler from the suprasternal notch to detect ascending aortic flow. The velocity–time integral (VTI) was calculated and multiplied by the LV outflow tract cross-sectional area (measured from a parasternal long axis view) to determine SV. Subsequently, cardiac output (Q) was determined by multiplying SV by the heart rate (HR) (as determined from the R-R interval from the same cardiac cycle on the ECG inherent to the echocardiographic machine [VividQ Ultrasound System, GE Ltd, Horton, Norway]). A subsample of five SP in the third year of observation were asked to return 7 days after the third year cardiac evaluation to establish the test–retest reliability of functional indices. Coefficients of variation (CV) were: S′ (9.1%); E (5.8%); E′ (16.2%), and E/E′ (11.9%) and for SV and Q were 3.9% and 4.1%, respectively. None of the echo measurements were blinded with respect to the SP or CON across the 3 years. Resting arterial venous oxygen difference (AVO_2) was calculated as: Resting VO_2/Q. Resting blood pressure was determined in the left arm of the participants using the auscultatory method.

2.4.3 | Speckle tracking methodology for strain variables

A focused apical 4-chamber ultrasound of the LV was obtained and optimized to improve endocardial determination using frequency and gain with a single focal zone placed mid LV cavity to reduce the impact of beam divergence. Frame rates were maintained between a range of 60–90 fps.[20]

Offline analysis was subsequently conducted using speckle tracking software (EchoPac, Version 6.0, GE Ltd, Horton, Norway) to determine: peak longitudinal ε, systolic strain rate (SSR), and early diastolic strain rate (DSR). Global values were calculated as the mean value of six myocardial segments from the basal, mid and apical septum, and lateral walls. All images were stored, and offline analyses were conducted. The average of three to five consecutive cardiac cycles were utilized for the determination of the strain variables.

2.5 | Statistical methods

All data were tested for normality using the Shapiro–Wilk test and homogeneity of variance was evaluated using Levene's test. All data were normally distributed, consequently, a parametric statistical approach was used throughout. Descriptive statistics were calculated in the form of mean, standard deviation and 95% confidence intervals (CI) of the mean. A linear mixed effect model was developed to simultaneously control for the fixed effects of Group (SP, CON), Year (1–3) and with maturity offset adjusted as a covariate on all the dependent variables between each year. The mixed effect model estimates the coefficients of the fixed effects. Coefficients for the categorical factors Group and Year indicate the average differences between the selected category and the reference category in the outcomes measurements. For the Group factor, the reference was the CON and for the Year factor, the reference was Year 1.

The unique personal ID code for each participant was considered as a random effect. This approach takes into account the variation in the number of individuals during the 3-year longitudinal analyses. This is particularly relevant in this study, as due to SP deselection and CON school pupil relocation, the participant numbers were not constant throughout the 3 years. Twenty-two SP started in year 1. Five players were deselected from year 1 to year 2 and three players were deselected from year 2 to year 3. Four players joined the clubs at the end of year 1. Consequently, in year 3, 18 players were evaluated. Of the 18, fourteen were evaluated from years 1 to 3 and four players were monitored from year 2 to the end of year 3. Fifteen CON started in year 1, with one CON dropping out from year 1 to year 2 and another dropped out between years 2 and 3. Therefore, in year 3, 13 CON were evaluated and had been in the study from years 1 to 3.

Due to the alteration in participant numbers each year, each individual player ID was considered as a random effect. Consequently, player ID was not a fixed effect and the model was run each time point with the assumption that the ID would be varying. Using this assumption, lme4 package in R enabled the model to estimate the coefficient taking into account the changes in participant number at each time point (i.e., one participant leaves or enter the study). This model estimation is defined as a partial random effect, for those participants that do not change across the 3 years, it is fixed and for those that change, it takes this variation into account for calculating the coefficients.[20]

In the mixed effect model, a post hoc linear regression analysis was conducted. The presence of an interaction between Group and Year indicates that the effect of training follows different patterns over the 3 years.[20] The statistical approach adopted in these analyses resulted in the dependent variables being adjusted for the influence of maturity status using maturity offset. The determination of maturity offset uses somatic measurements

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TABLE 2 Physical characteristics of the SP and CON across the 3 years of the study.

	Soccer players year 1	Soccer players year 2	Soccer players year 3	Control year 1	Control year 2	Control year 3
Maturity offset (years)	−2.1 ± 0.58	−1.1 ± 0.56	−0.5 ± 0.69	−2.4 ± 0.45	−2.5 ± 0.48	−0.5 ± 0.72
Age (years)	12.0 ± 0.3	13.0 ± 0.3	13.9 ± 0.3	11.7 ± 0.2	12.6 ± 0.1	13.6 ± 0.2
Stature (cm)	151.3 ± 6.3	156.1 ± 7.8	164.3 ± 8.8	146.8 ± 6.4	152.4 ± 5.9	161.9 ± 6.9
Mass (kg)	40.2 ± 5.8	45.4 ± 6.6	50.7 ± 7.6	43.3 ± 12.1	48.2 ± 12.4	57.8 ± 14.7
Body surface area (m^2)	1.29 ± 0.13	1.41 ± 0.13	1.54 ± 0.15	1.32 ± 0.18	1.43 ± 0.18	1.62 ± 0.19

Note: All values are mean ± SD.

of growth (stature and body mass). Consequently, SV and Q were not adjusted for body size, as this was already accounted for by the individual maturity offset value of each participant. Descriptive statistics (mean and SD) were derived for all strain variables for the SP and CON. A linear mixed effect model was developed to simultaneously control for the fixed effects of Group (SP and CON), Year (1–3) and with maturity offset adjusted as a covariate for the three dependent variables (peak longitudinal ε, SSR and DSR). A sample size of 22 SP resulted in a (1−β) of 80% at an alpha level of 0.05 at the start of the study and all statistical analyses were programmed in R.

3 | RESULTS

Table 2 highlights the physical characteristics of the SP and CON. There were no significant intergroup differences in maturity (Table 2), determined through the lack of difference in maturity offset at the start of year 1. Tables 3 and 4 highlight the descriptive characteristics of all the cardiac structure and function variables across all 3 years.

3.1 | LV Structure after controlling for maturity offset

Table 5 outlines the estimated coefficients for Group, Year and Group and Year interactions after controlling for maturity offset. Significant interactions were noted (Table 5) for: LVES, IVSd, PWd, RWT, LVEDV, LVESV, and LVMassIndex, and these are visualized in the box plots (Figure 1). Subsequently, the effect of Group was looked at separately for each year. The SP presented with greater LVES in Y1 and Y3 compared to CON after adjusting for maturity offset. IVSd and LVMassIndex were higher in Year 1 in the SP compared to CON. Subsequently, a similar training-induced decrease was noted for IVSd, PWd, RWT, and LVMi across all 3 years in the SP compared to the CON after adjusting for maturity offset; this was particularly evident in Year 3. LVEDV was higher in Year 1 in the SP compared to CON and then a similar pattern of a training-induced manifestation of greater LVEDV and LVESV were noted in Year 3 in the SP compared to CON. A Group effect was noted for Concentricity, with SP lower than CON in Year 3 only.

3.2 | LV Function after controlling for maturity offset

Significant year by group interactions were noted for: LVEF, HR, AVO_2 difference and S′. Consequently, the effect of Group was evaluated on a year-by-year basis after adjusting for maturity offset. A significant reduction in HR was noted in the SP across all 3 years, but this was particularly so in Year 3. AVO_2 difference was significantly greater in the SP in Year 3 compared to the CON. With respect to markers of LV contractile function, LVEF was significantly lower in the SP compared to the CON in Year 3 and this pattern of reduced contractile function was supported by the lower S′ noted in the SP compared to the CON in Year 3. The interactions are also visualized by the box plots in Figure 1. Group effects were noted for SV, with SP greater than CON in Year 3 and for E and E′ with SP greater than CON in Year 1. No Interaction or Group effects were noted for Q, E/E′, SBP, and DBP.

3.2.1 | Group effects for cardiac structure after controlling for maturity offset

There was no intergroup difference in LVED in Years 1 and 2, but a significantly greater influence of training on LVED in the SP in Year 3; this resulted in a greater LVED in Year 3 in the SP compared to CON. Concentricity ($LVMass/LVEDV^{0.667}$) demonstrated no significant intergroup differences in Years 1 and 2, but was significantly lower in the SP compared to the CON in Year 3 (Table 5, Figure 1).

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TABLE 3 Cardiovascular and tissue-Doppler Measurements at rest across the 3 years of the study.

	Soccer players year 1	Soccer players year 2	Soccer players year 3	Control year 1	Control year 2	Control year 3
LVED (mm)	45 ± 3	46 ± 3	49 ± 3	45 ± 2	43 ± 3	46 ± 4
LVED (mm.BSA$^{-0.5}$)	39 ± 3	39 ± 2	40 ± 2	39 ± 2	37 ± 2	37 ± 2
LVES (mm)	30 ± 2	26 ± 3	31 ± 3	29 ± 2	26 ± 2	28 ± 3
LVES (mm.BSA$^{-0.5}$)	27 ± 2	22 ± 3	25 ± 2	25 ± 2	22 ± 2	22 ± 2
IVSd (mm)	8 ± 1	8 ± 1	8 ± 1	7 ± 1	8 ± 1	8 ± 1
PWd (mm)	6 ± 1	7 ± 1	6 ± 1	6 ± 1	7 ± 1	7 ± 1
RWT	0.32 ± 0.05	0.36 ± 0.05	0.30 ± 0.05	0.30 ± 0.03	0.36 ± 0.05	0.39 ± 0.04
Concentricity (g/mL$^{0.667}$)	6.0 ± 0.7	7.1 ± 1.0	5.8 ± 0.7	5.7 ± 0.6	6.1 ± 1.1	7.5 ± 1.0
LVEDV (mL)	75 ± 10	76 ± 12	98 ± 16	67 ± 13	69 ± 12	80 ± 17
LVEDV (mL.BSA$^{-1.5}$)	51 ± 9	46 ± 5	51 ± 6	45 ± 6	42 ± 7	40 ± 6
LVESV (mL)	26 ± 4	24 ± 6	38 ± 9	24 ± 6	23 ± 3	28 ± 7
LVESV (mL BSA$^{1.5}$)	18 ± 3	14 ± 3	20 ± 4	16 ± 3	14 ± 2	14 ± 2
LVMass (g)	105 ± 14	128 ± 22	124 ± 22	93 ± 14	104 ± 23	140 ± 30
LVMassIndex (g/m^2)	81 ± 10	90 ± 9	80 ± 10	71 ± 10	74 ± 12	87 ± 13
LVEF (%)	66 ± 4	68 ± 4	61 ± 5	64 ± 5	67 ± 4	65 ± 3
Heart rate (bpm)	66 ± 9	68 ± 10	60 ± 8	75 ± 12	79 ± 12	86 ± 18
Q (L/min)	3.9 ± 0.8	4.5 ± 1.3	4.3 ± 1.0	4.2 ± 0.8	4.0 ± 0.9	4.8 ± 1.2
SV (mL)	61 ± 11	67 ± 18	71 ± 14	56 ± 9	50 ± 8	56 ± 9
AVO_2 difference (mL.100 mL^{-1})	8.0 ± 2.3	4. 9 ± 1.5	7.4 ± 2.0	6.8 ± 1.9	7.0 ± 1.2	4.7 ± 1.3
VO_2 (L/min)	0.31 ± 0.09	0.21 ± 0.04	0.31 ± 0.06	0.27 ± 0.04	0.27 ± 0.05	0.22 ± 0.05
S′ (cm/s)	10 ± 1	9 ± 1	8 ± 1	8 ± 1	10 ± 1	10 ± 2
S′adj (cm/s/mm)	1.25 ± 0.15	1.22 ± 0.19	1.00 ± 0.20	1.13 ± 0.30	1.30 ± 0.23	1.30 ± 0.28
E (cm/s)	90 ± 15	91. ± 9	85 ± 13	81 ± 7	85 ± 9	83 ± 9
E′ (cm/s)	15 ± 3	14 ± 3	12 ± 3	12 ± 3	13 ± 3	12 ± 2
E′adj (cm/s/mm)	0.20 ± 0.04	0.18 ± 0.04	0.15 ± 0.04	0.17 ± 0.05	0.17 ± 0.04	0.15 ± 0.04
E/E′	6 ± 1	7 ± 1	7 ± 2	7 ± 2	7 ± 1	8 ± 2
SBP (mmHg)	100 ± 8	107 ± 10	113 ± 13	105 ± 13	98 ± 11	111 ± 12
DBP (mmHg)	61 ± 9	62 ± 8	64 ± 3	61 ± 10	55 ± 7	60 ± 7

Note: All values are mean ± SD. E/E′ surrogate marker of preload.

Abbreviations: AVO_2, Arterial venous oxygen difference; DBP, diastolic blood pressure; E, Peak early diastolic filling velocity; E′, early peak longitudinal mitral annular velocity in diastole; E′adj, adjusted for LV length; IVSd, interventricular septum; LVED, Left ventricular end diastolic dimension; LVEDV, Concentricity; left ventricular end diastolic volume; LVEF, left ventricular ejection fraction; LVES, left ventricular end systolic dimension; LVESV, left ventricular end-systolic volume; LVMass, left ventricular mass unadjusted; LVMassIndex, adjusted for body surface area; PWd, posterior wall thickness; Q, Cardiac output; RWT, relative wall thickness; S′, Peak longitudinal mitral annular velocity in systole; S′adj, adjusted for LV length; SBP, Systolic blood pressure; SV, Stroke volume; VO_2, Oxygen uptake.

3.2.2 | Group-by-year interactions for cardiac function after controlling for maturity offset

Resting HR was significantly lower in the SP compared to CON in Years 1 and 2, but this was accentuated in Year 3. No intergroup LVEF differences were noted in Years 1 and 2, but lower LVEF was noted in the SP in Year 3. This decrement in systolic function was also supported by the pattern of change in S′ across all 3 years. SP were greater than CON in Year 1, demonstrated no intergroup differences in Year 2, and were significantly lower than CON in Year 3 (Table 5, Figure 1).

3.2.3 | Group effects for cardiac function after controlling for maturity offset

SV was significantly greater in the SP compared to CON in Year 3, with no intergroup differences in Years 1 and 2. Diastolic function (E and E′) were superior in Year 1 in the SP compared to CON, but there was no subsequent influence

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TABLE 4 Illustrates the observed mean ± SD for SP and CON from years 1 to 3 for all strain data.

	SP year 1	SP year 2	SP year 3	CON year 1	CON year 2	CON year 3
Peak ε (%)	−16.67 ± 1.72	−15.15 ± 1.71	−15.56 ± 2.13	−15.78 ± 2.49	−15.93 ± 1.51	−13.41 ± 2.41
SSR (1/s)	−0.98 ± 0.12	−0.99 ± 0.17	−0.96 ± 0.16	−1.00 ± 0.13	−0.98 ± 0.06	−0.94 ± 0.11
DSR (1/s)	1.56 ± 0.21	1.55 ± 0.33	1.48 ± 0.30	1.69 ± 0.44	1.83 ± 0.58	1.50 ± 0.43

Abbreviations: DSR, strain rate during diastole; SSR, strain rate during systole; ε, Peak longitudinal strain.

TABLE 5 Influence of training at each year for all structural and functional variables.

	Year 1 group (Soccer player)	Year 2 group (Soccer player)	Year 3 group (Soccer player)	Interaction
LVED (mm $BSA^{-0.5}$)	0.4 ($p = 0.680$)	2.4 ($p = 0.100$)	2.7 ($p = 0.010$)*	$p = 0.254$
LVES (mm $BSA^{-0.5}$)	1.9 ($p = 0.010$)*	−0.6 ($p = 0.710$)	3.0 ($p < 0.001$)*	$p = 0.001$*
IVSd (mm)	0.6 ($p = 0.030$)*	−1.0 ($p = 0.120$)	−0.8 ($p < 0.001$)*	$p = 0.003$*
PWd (mm)	0.5 ($p = 0.100$)	−1.1 ($p = 0.050$)	−1.6 ($p < 0.001$)*	$p < 0.001$*
RWT	0.03 ($p = 0.090$)	−0.03 ($p = 0.260$)	−0.09 ($p < 0.001$)*	$p < 0.001$*
Concentricity ($g/mL^{0.667}$)	0.3 ($p = 0.187$)	−0.9 ($p = 0.0562$)	−1.7 ($p < 0.001$)*	$p = 0.220$
LVEDV (mL $BSA^{-1.5}$)	6.8 ($p = 0.020$)*	7.0 ($p = 0.070$)	11.2 ($p < 0.001$)*	$p = 0.020$*
LVESV (mL $BSA^{-1.5}$)	1.6 ($p = 0.160$)	2.2 ($p = 0.180$)	6.4 ($p < 0.001$)*	$p = 0.020$*
LVMassIndex (g/m^2)	9.6 ($p = 0.010$)*	−2.5 ($p = 0.610$)	−4.5 ($p = 0.270$)	$p = 0.004$*
LVEF (%)	1.6 ($p = 0.290$)	−0.2 ($p = 0.950$)	−4.6 ($p = 0.010$)*	$p = 0.006$*
Heart rate (bpm)	−9 ($p = 0.010$)*	−17 ($p = 0.020$)*	−25 ($p < 0.001$)*	$p = 0.002$*
Q (L/min)	−0.42 ($p = 0.120$)	−0.69 ($p = 0.310$)	−0.56 ($p = 0.180$)	$p = 0.599$
SV (mL)	2 ($p = 0.540$)	4 ($p = 0.690$)	14 ($p < 0.001$)*	$p = 0.184$
AVO_2 difference ($mL.100\,mL^{-1}$)	1.4 ($p = 0.060$)	−1.1 ($p = 0.240$)	2.6 ($p < 0.001$)*	$p < 0.001$*
S′ (cm/s)	1.44 ($p = 0.010$)*	−0.95 ($p = 0.270$)	−1.58 ($p = 0.020$)*	$p < 0.001$*
E (cm/s)	10 ($p = 0.020$)*	5 ($p = 0.360$)	2 ($p = 0.670$)	$p = 0.284$
E′ (cm/s)	2.37 ($p = 0.040$)*	2.73 ($p = 0.130$)	0.8 ($p = 0.420$)	$p = 0.354$
E/E′	−0.6 ($p = 0.300$)	−0.9 ($p = 0.300$)	−0.3 ($p = 0.720$)	$p = 0.372$
SBP (mmHg)	−6 ($p = 0.110$)	−4 ($p = 0.480$)	3 ($p = 0.650$)	$p = 0.143$
DBP (mmHg)	−2 ($p = 0.560$)	1 ($p = 0.790$)	4 ($p = 0.240$)	$p = 0.412$
Peak ε(%)	−0.88 (0.347)	−0.73 (0.542)	−1.99 (0.052)*	0.548
SSR (1/s)	0.02 (0.756)	0.01 (0.913)	−0.01 (0.863)	0.8640
DSR (1/s)	−0.17 (0.250)	0.36 (0.136)	−0.06 (0.568)	0.933

Note: All values are Coefficient (*p*-value). The model is adjusted for individual maturity offset values in each year. E/E′ was calculated as an estimate of LV filling pressure and thus preload.

Abbreviations: DSR, strain rate during diastole; SSR, strain rate during systole; ε, Peak longitudinal strain; AVO_2, Arterial venous oxygen difference; Q, cardiac output; LVEDV, Concentricity; left ventricular end diastolic volume; DBP, diastolic blood pressure; E′, early peak longitudinal mitral annular velocity in diastole; IVSd, interventricular septum; LVEF, left ventricular ejection fraction; LVED, Left ventricular end diastolic dimension; LVES, left ventricular end systolic dimension; LVESV, left ventricular end-systolic volume; LVMassIndex, left ventricular mass adjusted for body surface area; E, Peak early diastolic filling velocity; S′, Peak longitudinal mitral annular velocity in systole; PWd, posterior wall thickness; RWT, relative wall thickness; SV, Stroke volume; SBP, Systolic blood pressure.

*$p < 0.05$.

of training on these parameters in Years 2 and 3 (Table 5, Figure 1), There were no interaction or group effects for Q, E/E′, SBP, or DBP (Table 4, Figure 1). Furthermore, there was a group effect for peak longitudinal ε in year 3, with the SP presenting with lower ε compared to CON. There was no interaction or group effects for SSR or DSR (Table 5).

4 | DISCUSSION

The major finding from this novel, 3-year observational investigation was that after controlling for the influence of a period of volatile growth, there was more evidence of eccentric remodeling (LV chamber enlargement) rather

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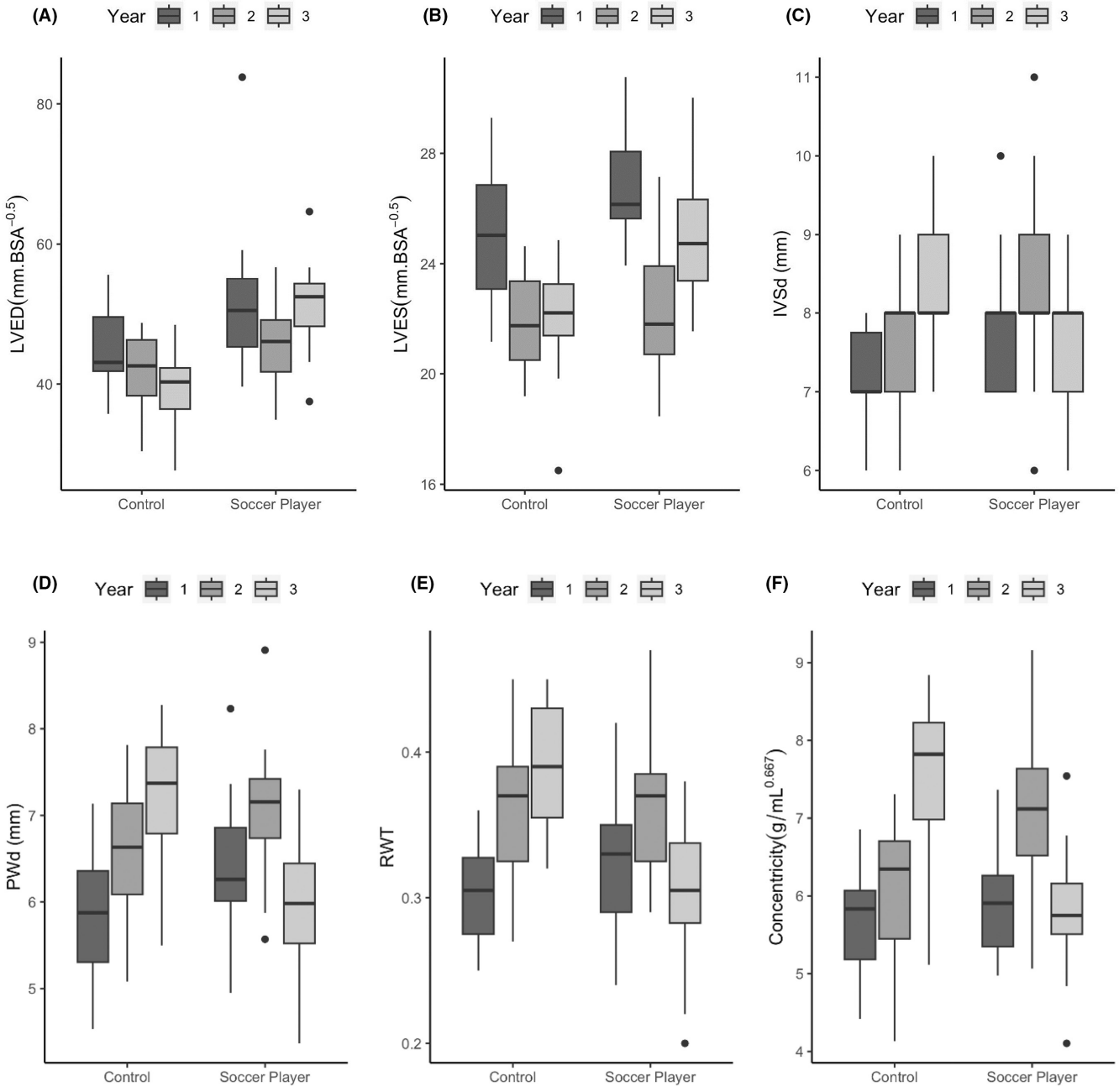

FIGURE 1 Changes in cardiac structure (Panel A- LVED, Panel B- LVES, Panel C- IVSd, Panel D- PWd, Panel E- RWT, Panel F- Concentricity, Panel G- LVEDV, Panel H- LVESV, and Panel I- LVMassIndex), global cardiac function (Panel J- LVEF, Panel K- HR, Panel L- Q, Panel M- SV, Panel N- AVO_2 Difference), TDI derived markers of Panel O- systolic (S′), and diastolic function Panels P and Q and R (E, E′ and E/E′), Panel S- SBP and Panel T- DBP at rest in the control participants and soccer players over the course of the 3-year observational study. AVO_2 difference, Arterial venous oxygen difference; DBP, diastolic blood pressure; E, Peak early diastolic filling velocity; E′, early peak longitudinal mitral annular velocity in diastole; IVSd, interventricular septum; LVED, Left ventricular end diastolic dimension; LVEDV, Concentricity index- LVMassIndex/LVEDV; left ventricular end diastolic volume; LVEF, left ventricular ejection fraction; LVES, left ventricular end systolic dimension; LVESV, left ventricular end-systolic volume; LVMassIndex, left ventricular mass adjusted for body surface area; PWd, posterior wall thickness; Q, cardiac output; RWT, relative wall thickness; S′, Peak longitudinal mitral annular velocity in systole; SBP, Systolic blood pressure; SV, Stroke volume. E/E′ was calculated as an estimate of LV filling pressure and thus preload. All values are median and interquartile range.

than eccentric hypertrophy (concomitant LV chamber dilatation and increase in LV wall thickness) in SP compared to CON. Furthermore, there was also evidence of functional adaptations in the form of decrements in LVEF and TDI-S′ over time in SP compared to CON after adjusting for a period of rapid growth.

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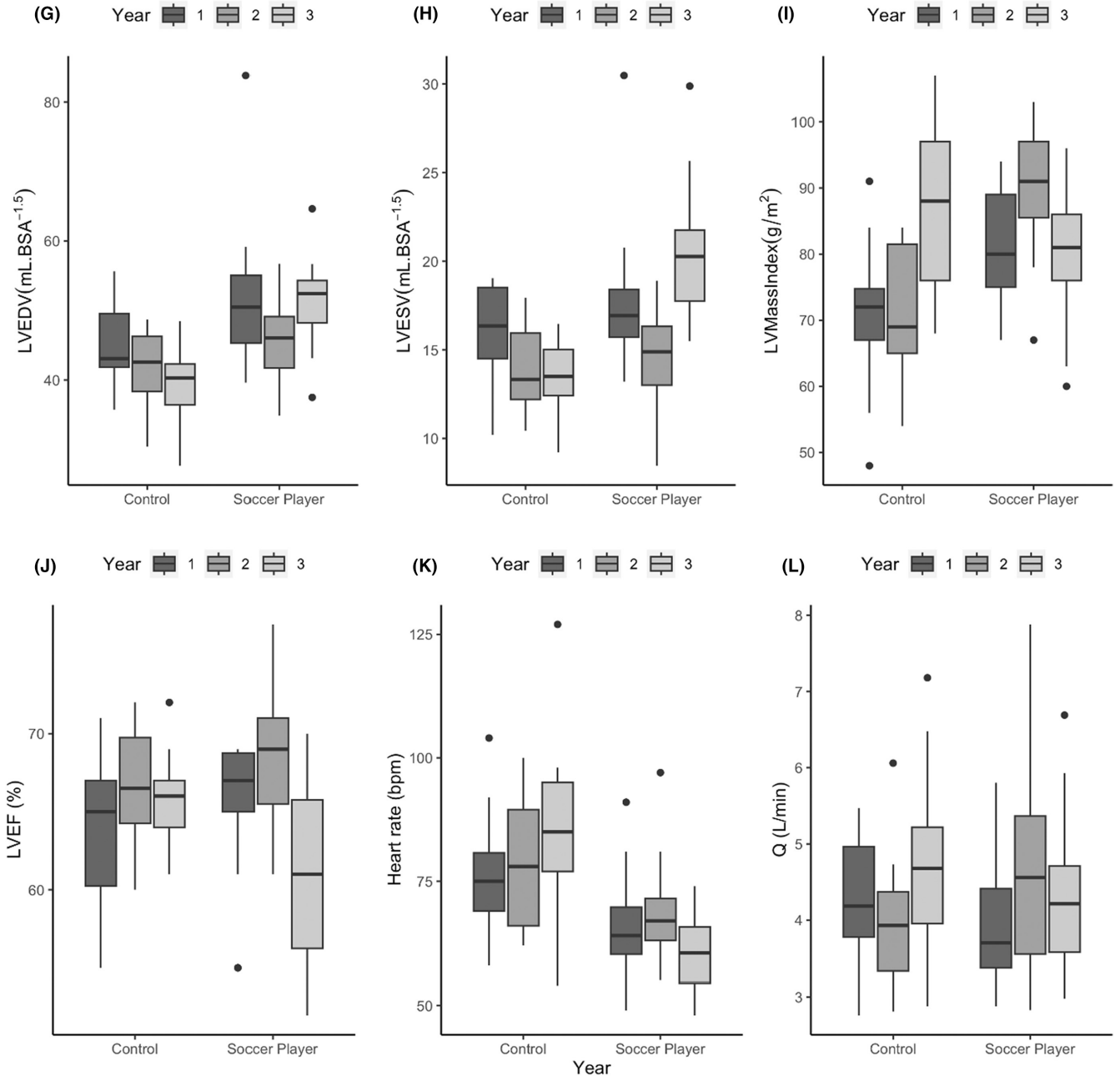

FIGURE 1 (Continued)

The SP were already engaged in systematic training for 4 years prior to the initiation of this study and this training exposure would have influenced the Year 1 cardiac adaptations noted in the SP in the Year 1 evaluation. After adjusting for the influence of a period of rapid growth, greater septal wall thickness (IVSd) was noted in the SP compared to CON with minimal evidence of systematic eccentric remodeling. There is evidence from both cross-sectional[5,23,24] and longitudinal data[12,25,26] to support these Year 1 findings and the pattern of data over Years 2 and 3. Krustrup et al.,[27] demonstrated that 10 weeks of small-sided games soccer training led to increases in PWd and IVSd in recreationally active children (10.4 years) compared to their peers that engaged in regular school physical activity classes only. Furthermore, Bjerring et al.[28] in the first year of a longitudinal tracking study of cross-country skiers demonstrated more evidence of greater wall thickness and less evidence of chamber dilatation in their athletes at 12 years of age compared to age-matched recreationally active controls. The pattern of eccentric remodeling seen from years 1 to 3 in this study, was also noted by Bjerring et al.[28] These authors identified that by 15 years of age, those cross-country skiers still engaged in endurance training demonstrated no significant difference in wall thickness compared to those adolescent athletes that no

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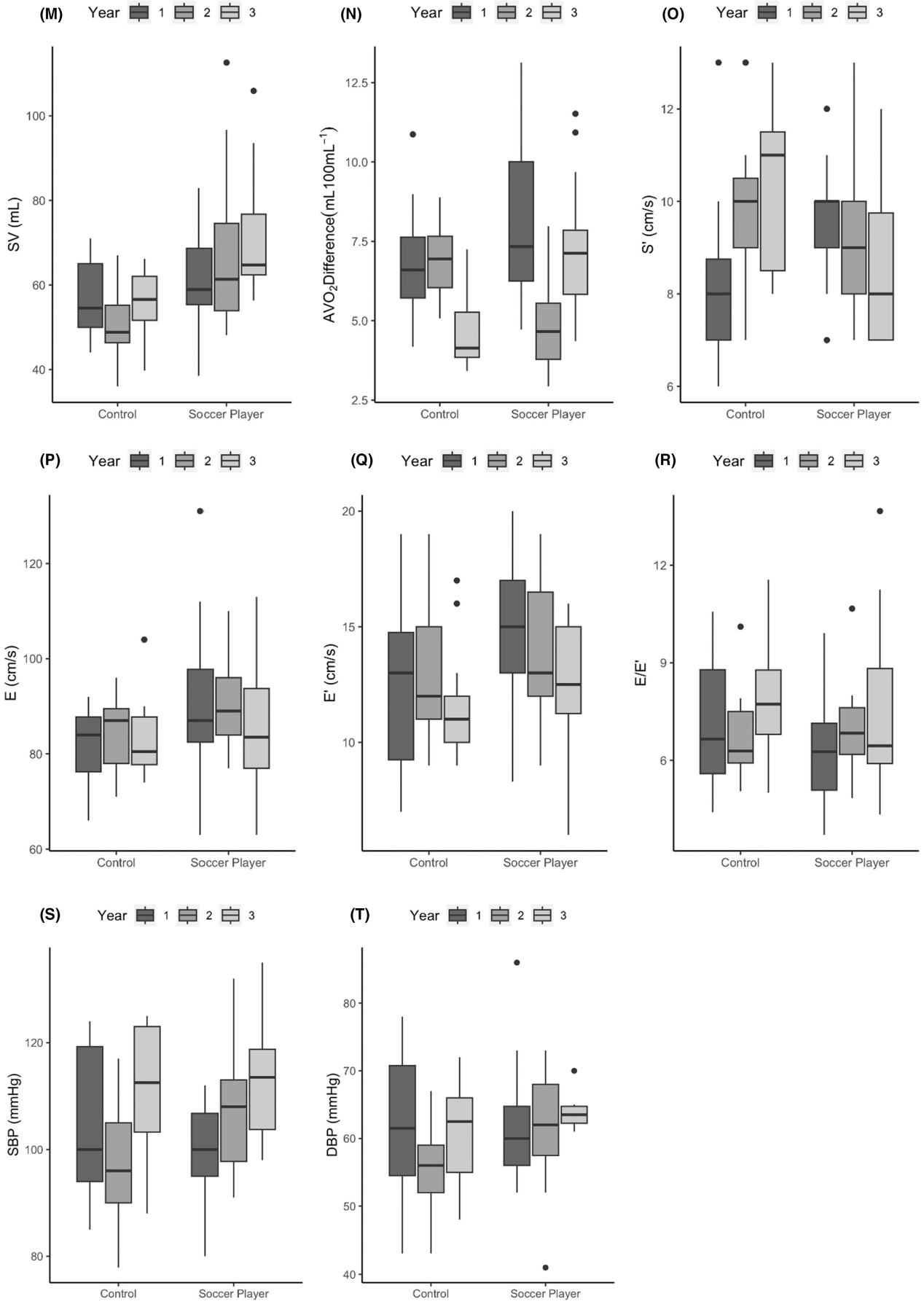

FIGURE 1 (Continued)

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longer competed. Chamber size was still greater, however, in those athletes that were still training and competing. This normalization/reduction of wall thickness over the 3 years in the presence of increased chamber size (LVED, LVES, LVEDV, and LVESV) in adolescent SP in this study has also been noted in the acute cardiac adaptations seen in untrained pre-adolescents (10–11 years) exposed to a 13-week run training program.[29] A similar pattern of adaptations were noted in exercise naive adults, with concentric wall thickening during the first 6–9 months of training followed by chamber dilatation.[30] These changes mimic the temporal cardiac adaptations seen in other adult training studies.[31]

After adjusting for the influence of this volatile growth period, the primary mechanism underpinning these temporal adaptations across the 3 years could be based on training duration[31] and volume.[32] Soccer-specific training volumes were relatively high (5–7 h per week) prior to the inception of the study and in Year 1 (8 h per week) in this study, but were significantly increased in Years 2 and 3 (10.5 h per week) in the SP. These increments in training volume were temporally aligned with greater IVSd in the SP compared to CON and were a precursor to an increase in chamber size seen in Years 2 and 3 in the SP. There is evidence from the adult and pediatric literature to support this theory. Arbab-Zadeh et al.[30] (2014) demonstrated that lower training volumes and intensities lead to increased wall thicknesses, whereas high intensity and volume exercise resulted in chamber dilatation. Pre-pubertal training studies have also demonstrated this with isolated wall thickness adaptation when a lower training load was initiated,[26] compared with LV dilation alone when sessions were longer and completed at greater than 80% maximal heart rate.[33] This intensity threshold mimics the common heart rate intensity of training and match-play in highly trained youth soccer players.[15]

The increased IVSd seen in the pre-adolescent SP in Year 1 at a chronologic age of 11.8 years and a maturity offset of −2.1 (years from PHV) in this study is unlikely to be driven by an androgenic stimulus, such as testosterone. There is evidence to support this contention in a low androgen model such as in female pre- and adolescent athletes. Evidence from Perkins et al.,[34] demonstrated increased LV wall thickness pre-PHV and an elevated LV volume post-PHV in a cross-sectional study with pre- and adolescent girls. Across the interface between pre- and post-PHV, it is possible to speculate that the increases in wall stress over time are not enough to require a concomitant increase in wall thickness. There is a need for future research to confirm this hypothesis. After adjusting for the influence of this rapid growth trajectory, increases in LVMassIndex were not aligned with increases in LVEDV in the SP over the 3 years. The rationale for this lack of change could be an artifact of the LVMass formula, which is very heavily weighted for LV wall thickness. Consequently, if there is a reduction/normalization of wall thickness over time in our SP, it is unsurprising that LVMass tracks the wall thickness findings. It is possible to speculate, therefore, that an initial concentric remodeling in response to low intensity training is then followed by chamber dilatation (eccentric remodeling) and is potentially the normal pattern in the early development of the athlete's heart in the elite youth soccer player.

The primary stimulus for the volumetric adaptations seen in our study in the highly trained SP may be pre-load enhancement during exercise. There is evidence to support this contention, as work from Unnithan et al.,[20] with the same cohort of players used in this study demonstrated enhanced early diastolic filling in the SP during submaximal exercise. Superior E was identified in the SP compared to the CON measured at the same relative submaximal exercise intensity across the 3 years of the study. Furthermore, superior "downstream" ventricular relaxation properties (E′) were noted in the SP in Year 2 of the study compared to CON during submaximal exercise, which were temporally aligned with a significant increase in training volume.[20]

It is possible that increased blood volume in response to high volume soccer training could be another factor that could trigger an increase in pre-load and the subsequent eccentric, volumetric adaptations seen in this study. There is evidence from the extant literature to support this hypothesis. Blood volume expansion via increases in both hemoglobin (Hb) mass and plasma volume have been identified in pre- and post-adolescent endurance athletes.[35] Furthermore, a training volume threshold of greater than 4 h per week and increases in lean body mass have also been identified as determinants of increases in Hbmass in adolescent soccer players.[36]

There were an array of functional adaptations in the SP in response to the training stimulus after adjusting for the influence of this rapid growth period. As LV chamber dilatation occurred in response to the training stimulus across the 3 years, there was a simultaneous decrease in markers of systolic function. Decrements were noted in LVEF and TDI-S′ over time; a pattern of a larger LV cavity size and no enhancement in LVEF and reduced TDI-S′ has been noted in both elite adult rugby league players compared to recreationally active CON[37] and Arab and Black adolescent athletes.[38] Similarly, LV structural adaptations were accompanied by lower LVEF at rest in adult male endurance cyclists compared to nonathletes.[39] This may suggest the potential for a functional systolic reserve that could be utilized during exercise.[40]

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There was evidence of greater E and E′ in the SP in Year 1 compared to CON.[29,41] But, over time, there was a normalization of diastolic function (E, E′, and E/E′) across the 3 years in the SP. This could be a product of LV cavity enlargement obviating the need for enhanced E, or a preservation of E for utilization during exercise. Evidence to support this latter contention was noted in elite, male endurance cyclists, whereby lower E and E′ velocities were identified compared to sub-elite and nonathletes at rest supporting the theory of a diastolic functional reserve.[39] Furthermore, decrements in E/A ratio were noted alongside increases in LV chamber size in adolescent athletes tracked over a 10-month period.[12] Unnithan et al.,[20] using the same cohort of SP seen in this study demonstrated higher E and E′ during submaximal cycle ergometer exercise compared to CON when exercising at the same relative exercise intensity. These findings support the theory of a diastolic functional reserve for exercise in the SP.

Resting HR declined over the 3 years in the SP along with an increase in SV. The influence of the increase in LV cavity size outweighed the reduction in LVEF resulting in a larger SV in Year 3 in the SP. The resultant of the decrement in HR alongside the increase in SV was no change in Q when comparing SP to CON. These resting adaptations in the SP form the bases for a functional reserve for the SP during exercise.[37] Evidence from the same cohort of SP during submaximal cycle ergometer exercise supports this cardiac reserve hypothesis. Work by Unnithan et al.,[20] demonstrated superior: QIndex, SVIndex, E, and E′ in the SP compared to CON when exercising at the same relative, submaximal exercise intensity. Deeper interrogation of LV mechanics with the derivation of twist has also been theorized to provide a functional reserve in adults, capable of being utilized during exercise.[41,42]

There was some evidence of a training-induced decrement in peak longitudinal ε in Year 3 in the SP compared to CON with this finding being aligned with the TDI-S′ decrease in Year 3. There was no evidence of any training-induced changes in SSR and DSR. Our findings were delimited to longitudinal ε. It is possible, however, to speculate that there may be other mechanisms for the myocardial contractile response noted in these SP. Indeed, evidence from adolescent soccer players during submaximal cycle ergometer exercise suggested a greater contribution from circumferential rather than longitudinal strain to myocardial contractility during submaximal exercise.[43]

The main limitation associated with this study was player deselection by the coaching staff that occurred at the football clubs and this impacted upon participant retention, but was reflective of the elite youth soccer environment that the study was conducted in. The statistical approach to normalizing the structural data in this study did not primarily use allometry, due to the fact that the maturity status adjustment already included markers of body mass and stature. There is evidence from the extant literature to support our contention that allometry does not always improve the explained variance of the effect of training on cardiac adaptation of IVSd and PWd.[44] The window of evaluation of this unique study was 11–14 years, but the SP had been training prior to this time, it is not possible to delineate whether the adaptations presented in Year 1 of this study were the resultant of genetic preselection for soccer (larger chamber size and wall thickness) or training-stimulated cardiac adaptations prior to the start of the study.[6] Furthermore, the authors' acknowledge that the incorporation of atrial and right ventricular data would have provided a more comprehensive picture of the cardiac adaptation to training in these young soccer players. Evaluation of circumferential and radial strain could also have provided deeper insight into the myocardial adaptations in these SP, and therefore, further studies should aim to elucidate holistic cardiac adaptation. It is also acknowledged that while the maturity offset method was a practical way of capturing maturity status data over the 3 years, there are limitations associated with this methodology. The use of this method to capture maturity offset has applicability for boys aged between 12 and 15 years who are on-time rather than boys who are early-or late-maturing individuals with respect to their growth trajectory.[45]

This novel 3-year observational study investigating the impact of high-volume soccer training on LV adaptations as players transitioned from the pre-to-adolescent stage demonstrated two unique trends in the SP that were independent of the influence of this rapid growth period. There was evidence of progressive eccentric remodeling of the LV cavity and functional adaptations in systolic function that gave rise to a potential cardiac reserve that the SP could prospectively exploit during exercise. Our findings indicate that a training volume threshold exists for cardiac adaptation in elite youth soccer players that occurs independently from the effect of the transition from pre-circa adolescence in the development of the athlete's heart in these young athletes.[11] The structural and functional changes seen in the SP may suggest a greater phenotype plasticity in these young athletes, as they mimic the pattern of adaptation seen in older, more experienced athletes.[39]

4.1 | Perspective

The broad clinical implications of our findings can be contextualized using LV- Z score nomograms for: LVED, LVES. IVSd and PWd generated from a sample of 2151 adolescent

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male soccer players.[44] There is no evidence of any athletes in this study presenting LV phenotypes in the pathophysiologic gray zone between the athlete's heart and cardiac abnormalities. This study represents the first of its kind to track the evolution of the athlete's heart in highly trained youth SP over a 3-year period (11–14 years of age) and to delineate the influence of this volatile growth period from the training stimulus. The evidence from this original body of work suggests that there is a training volume-based threshold that stimulates LV structural and functional adaptations—independent of the influence of growth and maturation in highly trained youth soccer players.

AUTHOR CONTRIBUTIONS

Viswanath B. Unnithan designed the study, collected data, analyzed data, and wrote the article; Alexander Beaumont analyzed data and edited the article; Thomas Rowland had input into the study design, collected data, and edited the article; Keith George had input into the study design and edited the article; Laura Stewart led the statistical analyses and edited the article; Nicholas Sculthorpe analyzed data and edited the article; Rachel N. Lord collected data and edited the article; David L. Oxborough had input into the study design, collected data, analyzed data, and edited the article.

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CONFLICT OF INTEREST STATEMENT

There was no conflict of interest for any author involved in this research project.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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