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Beaumont, A, Oxborough, D, George, KP, Rowland, TW, Sculthorpe, N, Lord, R and Unnithan, VB (2019) Superior cardiac mechanics without structural adaptations in pre-adolescent soccer players. European Journal of Preventive Cardiology, pp. 1-8. ISSN 2047 4873

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Superior cardiac mechanics without structural adaptations in pre-adolescent soccer players

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Word count: 4245

Abstract

Aims*:* This study aimed to evaluate left ventricular (LV) structure, function and mechanics, in highly-trained, pre-adolescent soccer players (SP) compared to age- and sex-matched controls (CON).

Design: The study design was a prospective, cross-sectional comparison of LV structure, function and mechanics.

Methods*:* Twenty-two male SP from two professional youth soccer academies (age: 12.0±0.3 years) and twenty-two recreationally active CON (age: 11.7±0.3 years) were recruited. Two-dimensional conventional and speckle tracking echocardiography were used to quantify LV structure, function and peak/temporal values for LV strain and twist, respectively.

Results*:* End-diastolic volume index was larger in SP (51 ± 8 mm/$(m^2)^{1.5}$ vs. 45 ± 6 mm/$(m^2)^{1.5}$; p=0.007) and concentricity was lower in SP (4.3 ± 0.7 g/$(mL)^{0.667}$ vs. 4.9 ± 1.0 g/$(mL)^{0.667}$; p=0.017), without differences in mean wall thickness between groups (6.0 ± 0.4 mm vs. 6.1 ± 0.5 mm; p=0.754). Peak circumferential strain at the base (-22.2 ± 2.5% vs. -20.5 ± 2.5%; p=0.029) and papillary muscle levels (-20.1 ± 1.5% vs. -18.3 ± 2.5%; p=0.007) were greater in SP. Peak LV twist was larger in SP (16.92 ± 7.55º vs. 12.34 ± 4.99 º; p=0.035) and longitudinal early diastolic strain rate was greater in SP (2.22 ± 0.40 s^{-1} vs. 2.02 ± 0.46 s^{-1}; p=0.025).

Conclusions*:* Highly-trained SP demonstrated augmented cardiac mechanics with greater circumferential strains, twist and faster diastolic lengthening in the absence of differences in wall thickness between SP and CON.

Keywords*:* Strain; Echocardiography; Speckle; Youth; Function; Left Ventricle

Abstract Word count: 234

Introduction

The match-play and training characteristics of soccer[1,2] presents an ecologically valid model to study the effects of systematic high intensity intermittent exercise training on cardiac structure and function in young, athletes.

A recent meta-analysis, has reported larger left ventricular (LV) diameter and wall thickness yet similar conventionally derived indices of systolic and diastolic cardiac function in adolescent athletes compared with non-athletes.[3] Additionally, cardiac enlargement increased with chronological age suggesting a potential role of hormones in pubertal adaptation. Importantly, the influence of exercise training on cardiac structure and function in pre-adolescent athletes may be ascertained without the confounding factors of growth and maturation. However, the effects of soccer training in these younger athletes are less clear, with some studies reporting similar absolute and scaled wall thicknesses between pre-adolescent athletes and controls.[4–6]

Our group recently documented LV structure and function in highly-trained pre-adolescent athletes, with a particular focus on conventional indices of LV function.[7] Speckle tracking echocardiography (STE) comprehensively assesses LV mechanics by quantifying deformation in the longitudinal, circumferential and radial planes, as well as rotation and twist.[8,9] Studies using STE to compare young athletes with age-matched controls have been conflicting,[10–12] likely due to variations in maturity status, the sex of the athletes, and disparate sports being studied. Some however, have reported lower longitudinal strain[12] and augmented twist.[13] Thus, there is a paucity of data detailing LV mechanics in pre-adolescent SP which warrants further investigation. Assessment of myocardial strains using STE will further our understanding on the coupling between LV structure and function in this population. Additionally, STE can facilitate temporal mechanical data that will extend our understanding of strains and rotations throughout the cardiac cycle.

Taken together, with the increased professionalization and subsequent increased training loads in elite youth soccer, at very early ages, there is a need to further interrogate global and regional markers of LV structure, function and mechanics. It was hypothesised that, (1) LV structure would not differ

between pre-adolescent SP and controls (CON); (2) LV longitudinal strain would be lower in SP compared to CON, while, (3) LV twist mechanics would be greater in SP than CON.

Methods

Participants

Twenty-two highly trained male youth soccer players (SP, age: 12.0 ± 0.3 years) and 22 recreationally active males (CON, mean age: 11.7 ± 0.3 years) were recruited to the study. Two of the SP were British African-Caribbean with the remainder Caucasian. Similarly, one of the CON was of British African-Caribbean origin and the remainder were Caucasian.

The SP training profiles were as follows: 4.5 ± 1.5 years training, 11 ± 1 months per year training, 4 ± 1 training sessions per week and 9.4 ± 2.4 hours per week of training. This volume of exercise training had been consistent for the entirety of their active training years. SP played one competitive match per week and had been engaged in competitive soccer matches for 4 ± 2 years. The SP were recruited from two Category one English Premier league youth soccer academies. For one club, 14 boys from the U12 squad and their parents were approached, of which 3 were not enrolled because of either personal circumstances (n=2) or a football related injury (n=1). At the second club, researchers provided information to 15 U12 players and their parents, of which 2 were recovering from injury, 1 was released from the club after signing up from the study, and 1 signed up and simply did not attend the testing. Accordingly, all recruitment was consecutive and included 11 participants from both clubs, with a total of 22 SP. CON participants took part in compulsory physical education of 2 hours per week (the same as SP), were all recreationally active and without engagement of systematic training. The CON self-reported 1.53 ± 1.77 hours per week of physical activity.

Written informed parental and participant consent was obtained prior to participation. All procedures performed in the study were in accordance with the Declaration of Helsinki and the study was reviewed and approved by Staffordshire University Ethics Committee.

Protocol/Measurements

Participants were asked to refrain from physical activity for 12 hours prior to the visit. Physical activity and training questionnaires[14] were completed prior to the testing. Following this, stature and body mass

were measured. Maturity status was quantified using maturity offset.[7,15] Resting arterial blood pressure was recorded in the left arm by an automated blood pressure cuff (Boso, Medicus, Jungingen, Germany) and heart rate was assessed by a 12-lead electrocardiogram (ECG) (CardioExpress SL6, Spacelabs Healthcare, Washington US). No abnormalities were detected from the ECG recordings in the participants included within the final analysis. Resting echocardiographic measurements were taken in the left lateral decubitas position. Body surface area (BSA) was calculated by Mosteller formula.[16]

Two-dimensional echocardiography

2D echocardiographic procedures were performed by two sonographers (soccer players [DO] and controls [DO + RL]) using a commercially available ultrasound system (VividQ Ultrasound System, GE Ltd, Horton, Norway) and images were analysed offline (EchoPac version 6.0, GE Ltd, Horton, Norway). Conventional measurements of resting LV dimensions and volumes (LV end-diastolic dimension [LVEDd], LV end-systolic dimension [LVESd], LV end-diastolic volume [LVEDV], LV end-systolic volume [LVESV]) and the subsequent calculations of LV mass and relative wall thickness (RWT) were made in accordance with American Society of Echocardiography (ASE) guidelines[17] and have been reported previously.[7] Linear LV dimensions were scaled to $BSA^{0.5}$, LV mass to $height^{2.7}$ and volumes to $BSA^{1.5}$.[18] Concentricity was calculated as LVM divided by allometrically scaled LVEDV ($LVEDV^{0.667}$).[19] Sphericity index was calculated as LV length divided by LVEDd.[20]

Peak mitral inflow velocities and pulsed wave tissue Doppler imaging (TDI) were assessed as previously reported.[7] E/E' was calculated as an estimate of LV filling pressure[21] from the average of septal and lateral E'.

Stroke volume (SV) and ejection fraction (EF) were calculated using Simpson's biplane method with cardiac output (Q) was determined by multiplying SV by the ECG determined heart rate (HR). Both Q and SV were adjusted for BSA (Qindex and SVindex).

LV mechanics were determined from 2D images with frame rates maintained as high as possible within the range of 40 to 90 fps. The cardiac cycle with the most defined endocardial border was used for analysis. Adjustments in frequency and gain were used to optimise endocardial delineation, with a single focal zone placed mid LV cavity to reduce the impact of beam divergence.

Aortic valve closure (AVC) was identified from the pulsed wave Doppler of LVOT flow and used to signify end systole. Offline analysis using dedicated speckle tracking software (Echopac V6.0, GE Healthcare, Horton, Norway) provided assessment of LV strains, strain rate (SR), rotations and net twisting.

Longitudinal strain was determined using the apical 4-chamber view. Global values were calculated as an average of 6 myocardial segments from the basal, mid and apical septum and lateral walls. Circumferential and radial strains were determined from the segmental average at the basal and mid-papillary levels. Using the mitral valve and apical levels, LV rotations were determined and twist was calculated as the difference between clockwise basal and counter-clockwise apical rotations during systole. In all instances, peak values and temporal analyses were obtained by importing stored traces into a Spreadsheet (Microsoft Corporation, Washington, USA) with a cublic spline add-in (SRS1 software, Boston, USA). Data were normalised to 5% increments during systole and diastole. Good reliability of LV mechanics data has also been established by this research team.[9]

Statistical analysis

Normality of data was assessed using Shapiro-Wilk. For normally distributed data, a Student's independent t-test was used to compare LV structure, function and mechanics in SP and CON. For non-normally distributed data, a Mann-Whitney U test was employed. The same procedures were used for temporal analysis at each 5% increment independently. A sample size of 22 SP provided a (1-β) of 80% at an alpha level of 0.05. Statistical significance was granted at $p<0.05$. Statistical analyses were performed using jamovi (version 0.9).[22]

Results

Missing data

Data were not obtained in CON (n=1) due to poor image quality in indices derived from Simpson's Biplane. Tissue Doppler and longitudinal strain indices were absent for CON (n=1). Due to >2 segments

excluded due to poor tracking in the apical plane, LV twist mechanics were not available for SP (n=1) and CON (n= 4).

Physical characteristics

SP were chronologically slightly older ($p<0.05$) than CON (12.0 ± 0.3 and 11.7 ± 0.3 years), but maturity offset (-2.1 ± 0.6 and -2.1 ± 0.6 years) and age at peak height velocity (14.0 ± 0.5 and 13.9 ± 0.6 years) were similar between SP and CON, (both $p>0.05$). Similarly, stature (1.51 ± 0.06 and 1.49 ± 0.07 m) and body mass (40.2 ± 5.8 and 44.0 ± 11.7 kg) were similar between SP and CON, respectively (both $p>0.05$). There were no inter- group differences for systolic (SP: 100 ± 8 mmHg; CON: 105 ± 13 mmHg) and diastolic blood pressure (SP: 61 ± 9 mmHg; CON: 61 ± 10 mmHg).

Conventional LV structure and function

Absolute and scaled SV, as well as scaled LVEDV were greater in SP than CON (all $p<0.05$). Absolute LV diameters, mean wall thickness and mass were not different between SP and CON (all $p>0.05$), whereas concentricity was lower and sphericity index greater in SP than CON, respectively (all $p < 0.05$, Table 1).

Conventionally derived systolic and diastolic function in SP and CON are presented in Appendix Table A.1. LV EF was greater in SP than CON, whereas absolute and scaled lateral S' were lower in SP (all $p<0.05$). E wave deceleration time was longer in SP than CON ($p<0.05$). No other functional differences were observed between groups.

[Table 1]

LV mechanics

Peak longitudinal strain was not different between groups ($p>0.05$). Circumferential strain at the basal and papillary muscle levels were greater in SP than CON (both $p<0.05$). Also, peak circumferential and longitudinal diastolic SR were greater in SP than CON (both $p<0.05$). Apical rotation was higher in SP than CON ($p<0.05$), without differences at the basal level ($p>0.05$) and thus, peak LV twist was greater

in SP ($p<0.05$). No further differences in peak LV mechanics were observed between groups (all $p>0.05$, Table 2).

In SP, longitudinal SR was greater than CON during early diastole ($p<0.05$) and showed a leftward shift in the descending arm during mid diastole (Fig. 1 A, B). Circumferential strain at basal and papillary muscle levels were greater in SP than CON throughout systole ($p<0.05$). SR at the papillary muscle level was greater in SP ($p<0.05$), corresponding to early diastole (Appendix Fig. B.1). Similarly, apical rotation and twist were greater in SP than CON through the majority of systole ($p<0.05$), while temporal analysis of basal rotation did not differ between groups ($p>0.05$, Appendix Fig. B.2).

[Table 2]

[Fig. 1]

Discussion

The major findings from the study were that SP had augmented peak LV mechanics (circumferential strain, apical rotation and twist) and faster early diastolic lengthening. Additionally, SP had lower concentricity than CON, without concomitant differences in wall thickness or longitudinal strain between SP and CON.

Lower concentricity was evident in the presence of larger scaled LVEDV, indicative of heightened preload, in SP compared to CON. It is possible that this is the consequence of plasma volume expansion common in well trained adolescents.[23] The similar LV structure between groups contradicts previous echocardiographic work that has reported greater LV wall thickness or chamber diameter in pre-adolescent SP.[12,24] These contrasting findings may be a product of differences in maturation status of the studied populations. Indeed, a recent meta-analysis found pre-adolescent athletes (<14 years) presented cardiac enlargement to a lesser extent than those within the pubertal growth stage.[3] Further, Nottin et al[4] found similar wall thicknesses in pre-pubertal cyclists and sedentary children. Taken together, these data support the contention that sufficient maturity is necessary to promote exercise induced increases in LV structure.

Similar longitudinal strain between SP and CON disagrees with a similar study in young footballers,[12] yet supports the majority of existing literature in young athletes.[10,11] Although

documentation of longitudinal strain in paediatric athletes is currently in its infancy, these observations corroborate with the adult athlete's heart that longitudinal strain remains largely unaltered in chronically trained athletes.[25] Reduced longitudinal strain has been suggested as an uncommon feature of the adult athlete's heart[26] and this may hold true also for paediatric athletes given the accumulation of evidence to suggest unaltered longitudinal strain in healthy athletic children.

Circumferential strain was greater in SP at both the base and papillary muscle levels and highlights a more notable difference in systolic functioning in SP compared to the untrained state. Although the greater circumferential strain in SP contrasts recent work,[11] the increase observed here is likely indicative of greater overall systolic function in SP. Yet, the purpose of these adaptations and responsible mechanistic underpinning requires clarification.

LV twist was higher in SP mediated through greater apical rotation, without changes in basal rotation, with temporal analysis indicating higher apical rotation and twist through the majority of systole. To our knowledge, this is the first echocardiographic documentation of LV twist mechanics in pre-adolescent athletes, yet concurs with a recent MRI study.[13] These data begin to define the twisting profiles of chronically trained pre-adolescents, which appear to contrast their elite level adult counterparts of reduced net twist compared to untrained controls.[25,27] A phasic response in LV twist has been reported in adults with twist increased initially before returning to baseline following structural (true eccentric) remodelling during a more extended period of exercise training.[28] Indeed, larger wall thickness appears to drive the reduction in apical rotation and net twist in adults.[27] The absence of differences in MWT between SP and CON, could explain why twist was higher in SP, owing to heightened scaled LVEDV, considering the preload dependence of LV apical rotation and twist.[29] Thus, the adaptations in chronically trained pre-adolescents may be more reflective of the acute phase of exercise training in adults.[28]

Alternatively, in adults LV twist is influenced by muscle fibre orientation and both apical rotation and twist are independently associated, in a parabolic manner, with sphericity index.[20] In this study, sphericity index was slightly higher in SP and may therefore, be placed higher on the ascending arm of the parabolic curve. Irrespective of the mechanistic underpinning, taking these observations

together we propose that the heart of paediatric athletes presents a useful model to study the influence of exercise training on LV twist mechanics prior to structural remodelling.

Greater circumferential strains and LV twist mechanics could explain the greater EF in pre-adolescent SP. Subsequently, the likely combination of a larger LVEDV and increased EF led to a greater SV. The higher EF observed is not in agreement with other similar work, although the dataset presented within this recent meta-analysis was highly heterogeneous.[3] The reasons for discrepancies between studies is unclear. However, EF is considered a surrogate marker of LV pump function,[30] and others have reported pre-adolescent athletes have similar EF with comparable or lower LV strain mechanics than CON.[10–12] Whereas, in this study SP had a greater EF which was accompanied with augmented circumferential strain and twist, and thus the altered mechanics may explain the greater EF. Together, the combination of LV mechanics and EF in this study suggests augmented systolic function, yet the functional capacity and potential reserve during exercise warrants further investigation.

Mitral inflow and early diastolic tissue velocities were similar between SP and CON, whereas assessment of temporal SR indicated SP had faster diastolic lengthening during the early phases of diastole. SR was lower in SP at 35-40% diastole during the cardiac cycle signifying superior lengthening velocities leading to a longer period of diastasis. These data are supported by the greater peak longitudinal and circumferential (papillary muscle level) SRE in SP than CON. Accordingly, it is plausible that novel assessment using STE may facilitate the detection of subtle differences that are not apparent using TDI, with the former being advantageous in being relatively angle independent and less affected by tethering from adjacent segments.[8] Collectively, these data suggest improved relaxation may contribute to the enhanced preload/LVEDV and the larger ensuing SV. The functional importance of these observations is yet to be fully realised, however, it is possible that this becomes pertinent to support LV filling especially when diastole shortens during exercise.

Considering that youth SP perform at high intensities,[1,11] the differentiation between pathological and physiological adaptation is of paramount importance and is of current interest.[31] The present study highlights that elite-level soccer training may be a strong enough stimulus to induce LV mechanical adaptations, even in the absence of morphological difference. We report normal values of LV mechanics in asymptomatic pre-adolescents, therefore, the clinical inference is challenging and may

warrant further investigation in those with and without known cardiovascular diseases. Additionally, given the small sample sizes in this study, the clinical implications of this work require further study in larger groups, in association with exercise training status. Future longitudinal data is needed to track these players throughout the volatile growth periods, and as a result, these insights may be helpful to diagnose early stages of cardiomyopathies, such as in HCMP phenotype negative but genotype positive individual, for example.

Limitations

We acknowledge the limitations of cross-sectional studies including for example, the ability to clearly attribute the cardiac functional changes to soccer training, or the genetic predisposition for preadolescents to perform soccer at a high-level. Plasma volume was not assessed in this study due to ethical restrictions for blood sampling in the studied population. Two-dimensional echocardiography is inherently limited by out-of-plane motion such that, apical and basal imaging planes may not be the same through the entire cardiac cycle.[8] Limitations in current echocardiographic techniques may explain why LV twist was not obtained in 5 participants (n=1 SP and n=4 CON), since optimal acquisition and speckle-tracking are required at both the base and apex. Accordingly, this resulting in slightly unequal samples sizes between SP and CON. While this presents challenges for clinical utility, these parameters are increasing acknowledge for their potential use in characterising the athlete's heart, although work is still needed for normative values in LV twist and circumferential strain.[32] In this study, however, we applied stringent criteria to both image acquisition and analysis to facilitate confidence in obtaining physiologically meaningful data. Finally, if age-associated increases in LV twist during childhood are related to maturational adaptive modulation[33,34], it would be unlikely that the small differences in chronological age between SP and CON would impact results, since biological age (maturity offset) and LV length were similar (i.e. twisting occurs along the same length LV).

Conclusions

Augmented resting LV mechanics (twist, apical rotation and circumferential strain) were observed in highly trained SP, including increased circumferential strains, apical rotation and LV twist, with supportive temporal analysis demonstrating faster early diastolic lengthening than untrained, matched

controls. These data highlight superior function derived by STE LV mechanics in the absence of LV wall thickness changes yet with lower concentricity.

Acknowledgments

The authors would like to thank Mr. Marc Campbell (Wolverhampton Wanders FC) and Dr. Russ Wrigley (Blackburn Rovers FC) in the organisation of the SP data collection sessions. Ms. Katie Davis and Ms. Leanne Brittle for their help with the organisation of the testing session of CON (Staffordshire University Academy).

Funding

This research did not receive any specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Declaration of conflicting interests

The Authors declare that there is no conflict of interest.

Author contributions statement

V.B.U. conceived and designed the research study. V.B.U., T.W.R., R.L., and D.O. conducted the experiments and collected the data. D.O., and A.B. analysed data. A.B., D.O., N.S., and V.B.U. interpreted the data. A.B. prepared figures and wrote the manuscript. D.O., K.G., T.W.R., N.S., R.L., and V.B.U. edited and revised the manuscript. All authors read and approved the final version of the manuscript.

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Figure legend

Fig. 1. Temporal analyses of left ventricle longitudinal strain (**A**) and strain rate (**B**) during the cardiac cycle in soccer players (SP) and controls (CON). Data are means ± standard deviation. AVC, aortic valve closure. Shaded areas indicate statistical significance at $p < 0.05$.

433 **Tables**

Table 1 Left ventricular structure and volumes in soccer players (SP) and controls (CON).

	SP	CON	p-value
HR (beats/min)	65 ± 8	74 ± 10	**0.005**
LVEDd (mm)	42 ± 4	44 ± 4	0.142
LVEDd index (mm/$(m^2)^{0.5}$)	37 ± 3	38 ± 3	0.343
LVESd (mm)	28 ± 3	29 ± 3	0.403
LVESd index (mm/$(m^2)^{0.5}$)	25 ± 3	25 ± 2	0.725
MWT (mm)	6.0 ± 0.4	6.1 ± 0.5	0.754
MWT (mm/$(m^2)^{0.5}$)	5.3 ± 0.4	5.3 ± 0.5	0.769
RWT	0.29 ± 0.04	0.28 ± 0.04	0.387
LVM (g)	75 ± 14	82 ± 18	0.204
LVM index (g/m^2)	25 ± 5	28 ± 7	0.051
LV length (mm)	76 ± 6	74 ± 5	0.316
LV length index (mm/$(m^2)^{0.5}$)	67 ± 5	64 ± 5	0.140
LVEDV (mL)	75 ± 10	69 ± 15	0.106
LVEDV index (mm/$(m^2)^{1.5}$)	51 ± 8	45 ± 6	**0.007**
LVESV (mL)	26 ± 4	26 ± 8	0.696
LVESV index (mL/$(m^2)^{0.5}$)	18 ± 2	17 ± 3	0.625
Concentricity (g/mL)$^{0.667}$)	4.3 ± 0.7	4.9 ± 1.0	**0.017**
Sphericity index	1.8 ± 0.2	1.7 ± 0.1	**0.034**
SV (mL)	49 ± 8	43 ± 10	**0.031**
SV index (mL/$(m^2)^{1.0}$)	38 ± 6	32 ± 5	**0.002**
$\dot{Q}$ (L/min)	3.19 ± 0.63	3.12 ± 0.67	0.741
Cardiac index (L/min/$(m^2)^{1.0}$)	2.48 ± 0.46	2.36 ± 0.42	0.391

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436 Data are mean ± standard deviation. HR, heart rate; LVEDd, left ventricular end-diastolic diameter;
437 LVESd, left ventricular end-systolic diameter MWT, mean wall thickness; RWT, relative wall
438 thickness; LVM, left ventricular mass; LVEDV, left ventricular end-diastolic volume; LVESV, left
439 ventricular end-systolic volume; SV, stroke volume; $\dot{Q}$, cardiac output; SP, soccer players; CON,
440 controls.

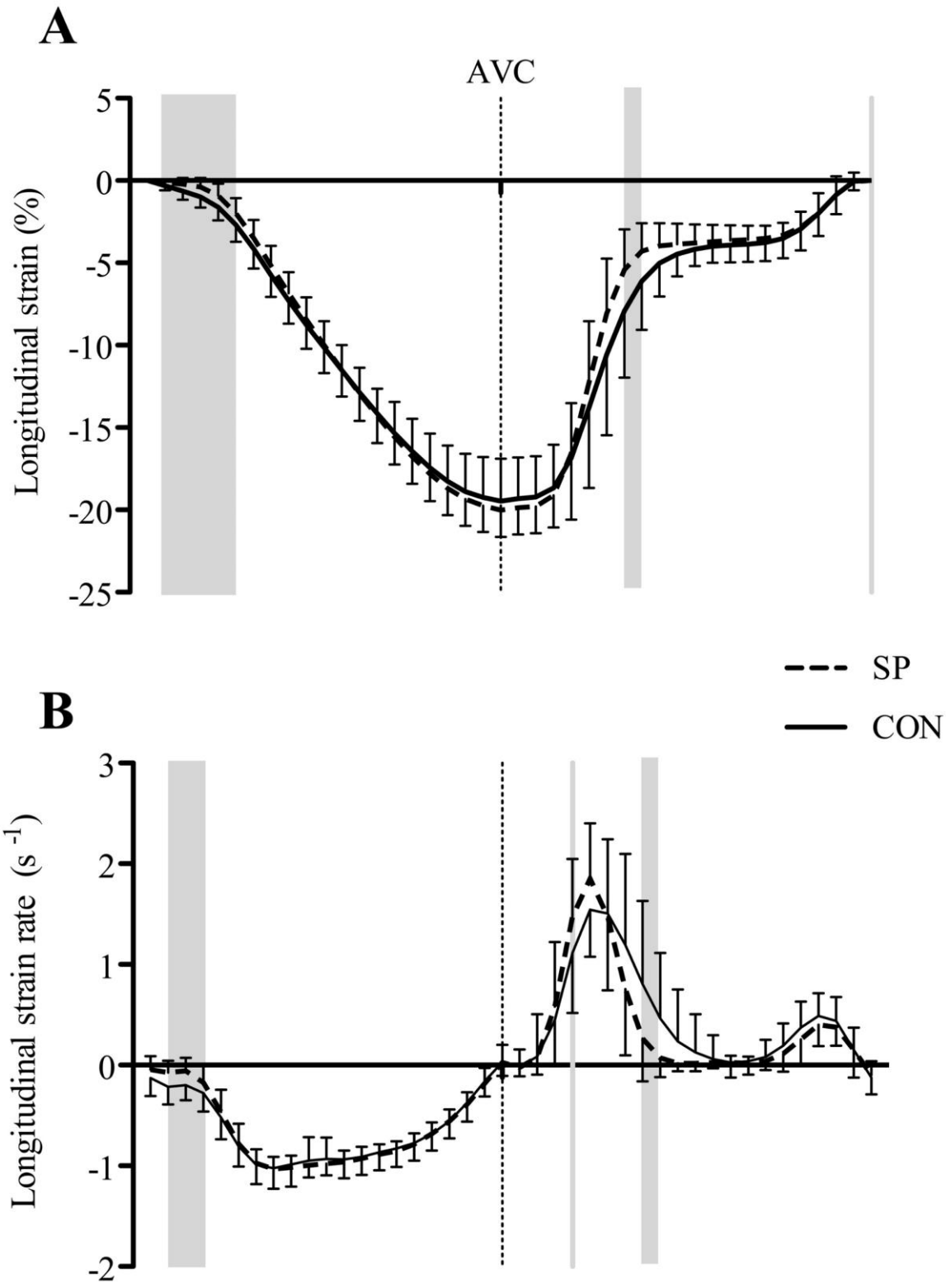
Table 2. Peak left ventricular mechanics in soccer players (SP) and controls (CON).

	SP	CON	p-value
Longitudinal			
Strain (%)	-20.3 ± 1.6	-19.6 ± 2.5	0.283
SRS (s^{-1})	-1.07 ± 0.11	-1.11 ± 0.18	0.427
SRE (s^{-1})	2.22 ± 0.40	2.02 ± 0.46	**0.025**
SRA (s^{-1})	0.57 ± 0.10	0.66 ± 0.15	0.105
Mitral Valve (Base)			
Circumferential strain (%)	-22.2 ± 2.5	-20.5 ± 2.5	**0.029**
Circumferential SRS (s^{-1})	-1.29 ± 0.15	-1.29 ± 0.17	0.925
Circumferential SRE (s^{-1})	2.16 ± 0.37	2.08 ± 0.34	0.457
Circumferential SRA (s^{-1})	0.36 ± 0.13	0.38 ± 0.09	0.587
Radial strain (%)	38.3 ± 13.7	33.7 ± 15.4	0.304
Radial SRS (s^{-1})	2.12 ± 0.50	2.22 ± 1.01	0.675
Radial SRE (s^{-1})	-2.54 ± 0.81	-2.46 ± 0.81	0.954
Radial SRA (s^{-1})	-0.74 ± 0.36	-1.08 ± 0.67	0.065
Papillary Muscle (mid-ventricular)			
Circumferential strain (%)	-20.1 ± 1.5	-18.3 ± 2.5	**0.007**
Circumferential SRS (s^{-1})	-1.17 ± 0.17	-1.19 ± 0.18	0.732
Circumferential SRE (s^{-1})	1.89 ± 0.33	1.57 ± 0.39	**0.001**
Circumferential SRA (s^{-1})	0.37 ± 0.14	0.42 ± 0.12	0.215
Radial strain (%)	61.6 ± 15.8	62.2 ± 20.9	0.918
Radial SRS (s^{-1})	2.38 ± 0.89	2.47 ± 1.28	0.944
Radial SRE (s^{-1})	-3.64 ± 1.54	-3.35 ± 1.47	0.569
Radial SRA (s^{-1})	-1.08 ± 0.54	-1.60 ± 1.54	0.463
Twist Mechanics			
Apical rotation (º)	11.95 ± 5.31	7.58 ± 3.55	**0.005**

Basal rotation (º)	-5.69 ± 3.14	-5.64 ± 2.65	0.959
Twist (º)	16.92 ± 7.55	12.34 ± 4.99	**0.035**

Data are presented as means ± standard deviation. SRS, peak systolic strain rate; SRE, peak early diastolic strain rate; SRA, peak late diastolic strain rate.

446 **Figure 1**



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