

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

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25 **Abstract**

26

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

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

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

35 **Results:** End-diastolic volume index was larger in SP ( $51 \pm 8 \text{ mm}/(\text{m}^2)^{1.5}$  vs.  $45 \pm 6 \text{ mm}/(\text{m}^2)^{1.5}$ ;  
36  $p=0.007$ ) and concentricity was lower in SP ( $4.3 \pm 0.7 \text{ g}/(\text{mL})^{0.667}$  vs.  $4.9 \pm 1.0 \text{ g}/(\text{mL})^{0.667}$ ;  $p=0.017$ ),  
37 without differences in mean wall thickness between groups ( $6.0 \pm 0.4 \text{ mm}$  vs.  $6.1 \pm 0.5 \text{ mm}$ ;  $p=0.754$ ).  
38 Peak circumferential strain at the base ( $-22.2 \pm 2.5\%$  vs.  $-20.5 \pm 2.5\%$ ;  $p=0.029$ ) and papillary muscle  
39 levels ( $-20.1 \pm 1.5\%$  vs.  $-18.3 \pm 2.5\%$ ;  $p=0.007$ ) were greater in SP. Peak LV twist was larger in SP  
40 ( $16.92 \pm 7.55^\circ$  vs.  $12.34 \pm 4.99^\circ$ ;  $p=0.035$ ) and longitudinal early diastolic strain rate was greater in SP  
41 ( $2.22 \pm 0.40 \text{ s}^{-1}$  vs.  $2.02 \pm 0.46 \text{ s}^{-1}$ ;  $p=0.025$ ).

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

45

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

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## 54 **Introduction**

55 The match-play and training characteristics of soccer<sup>1,2</sup> presents an ecologically valid model to study  
56 the effects of systematic high intensity intermittent exercise training on cardiac structure and function  
57 in young, athletes.

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

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

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

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

## 82 **Methods**

### 83 *Participants*

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

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

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

104

### 105 *Protocol/Measurements*

106 Participants were asked to refrain from physical activity for 12 hours prior to the visit. Physical activity  
107 and training questionnaires<sup>14</sup> were completed prior to the testing. Following this, stature and body mass

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

114

### 115 *Two-dimensional echocardiography*

116 2D echocardiographic procedures were performed by two sonographers (soccer players [DO] and  
117 controls [DO + RL]) using a commercially available ultrasound system (VividQ Ultrasound System,  
118 GE Ltd, Horton, Norway) and images were analysed offline (EchoPac version 6.0, GE Ltd, Horton,  
119 Norway). Conventional measurements of resting LV dimensions and volumes (LV end-diastolic  
120 dimension [LVEDd], LV end-systolic dimension [LVESd], LV end-diastolic volume [LVEDV], LV  
121 end-systolic volume [LVESV]) and the subsequent calculations of LV mass and relative wall thickness  
122 (RWT) were made in accordance with American Society of Echocardiography (ASE) guidelines<sup>17</sup> and  
123 have been reported previously.<sup>7</sup> Linear LV dimensions were scaled to  $BSA^{0.5}$ , LV mass to height<sup>2,7</sup> and  
124 volumes to  $BSA^{1.5}$ .<sup>18</sup> Concentricity was calculated as LVM divided by allometrically scaled LVEDV  
125 ( $LVEDV^{0.667}$ ).<sup>19</sup> Sphericity index was calculated as LV length divided by LVEDd.<sup>20</sup>

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

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

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

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

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

149

#### 150 *Statistical analysis*

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

157

## 158 **Results**

### 159 *Missing data*

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

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

164

### 165 *Physical characteristics*

166 SP were chronologically slightly older ( $p < 0.05$ ) than CON ( $12.0 \pm 0.3$  and  $11.7 \pm 0.3$  years), but  
167 maturity offset ( $-2.1 \pm 0.6$  and  $-2.1 \pm 0.6$  years) and age at peak height velocity ( $14.0 \pm 0.5$  and  $13.9 \pm$   
168  $0.6$  years) were similar between SP and CON, (both  $p > 0.05$ ). Similarly, stature ( $1.51 \pm 0.06$  and  $1.49 \pm$   
169  $0.07$  m) and body mass ( $40.2 \pm 5.8$  and  $44.0 \pm 11.7$  kg) were similar between SP and CON, respectively  
170 (both  $p > 0.05$ ). There were no inter- group differences for systolic (SP:  $100 \pm 8$  mmHg; CON:  $105 \pm 13$   
171 mmHg) and diastolic blood pressure (SP:  $61 \pm 9$  mmHg; CON:  $61 \pm 10$  mmHg).

172

### 173 *Conventional LV structure and function*

174 Absolute and scaled SV, as well as scaled LVEDV were greater in SP than CON (all  $p < 0.05$ ). Absolute  
175 LV diameters, mean wall thickness and mass were not different between SP and CON (all  $p > 0.05$ ),  
176 whereas concentricity was lower and sphericity index greater in SP than CON, respectively (all  $p <$   
177  $0.05$ , Table 1).

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

182

183 **[Table 1]**

184

### 185 *LV mechanics*

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

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

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

199 [Table 2]

200 [Fig. 1]

## 201 Discussion

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

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

216 Similar longitudinal strain between SP and CON disagrees with a similar study in young  
217 footballers,<sup>12</sup> yet supports the majority of existing literature in young athletes.<sup>10,11</sup> Although



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

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

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

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

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

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

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

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

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

### 279 ***Limitations***

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

296

### 297 **Conclusions**

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

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

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### 316 **Declaration of conflicting interests**

317 The Authors declare that there is no conflict of interest.

318

### 319 **Author contributions statement**

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

325

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427 **Figure legend**

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

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**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	<b>0.005</b>
LVEDd (mm)	42 ± 4	44 ± 4	0.142
LVEDd index (mm/(m <sup>2</sup> ) <sup>0.5</sup> )	37 ± 3	38 ± 3	0.343
LVESd (mm)	28 ± 3	29 ± 3	0.403
LVESd index (mm/(m <sup>2</sup> ) <sup>0.5</sup> )	25 ± 3	25 ± 2	0.725
MWT (mm)	6.0 ± 0.4	6.1 ± 0.5	0.754
MWT (mm/(m <sup>2</sup> ) <sup>0.5</sup> )	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 <sup>2</sup> )	25 ± 5	28 ± 7	0.051
LV length (mm)	76 ± 6	74 ± 5	0.316
LV length index (mm/(m <sup>2</sup> ) <sup>0.5</sup> )	67 ± 5	64 ± 5	0.140
LVEDV (mL)	75 ± 10	69 ± 15	0.106
LVEDV index (mm/(m <sup>2</sup> ) <sup>1.5</sup> )	51 ± 8	45 ± 6	<b>0.007</b>
LVESV (mL)	26 ± 4	26 ± 8	0.696
LVESV index (mL/(m <sup>2</sup> ) <sup>0.5</sup> )	18 ± 2	17 ± 3	0.625
Concentricity (g/mL) <sup>0.667</sup> )	4.3 ± 0.7	4.9 ± 1.0	<b>0.017</b>
Sphericity index	1.8 ± 0.2	1.7 ± 0.1	<b>0.034</b>
SV (mL)	49 ± 8	43 ± 10	<b>0.031</b>
SV index (mL/(m <sup>2</sup> ) <sup>1.0</sup> )	38 ± 6	32 ± 5	<b>0.002</b>
Q̇ (L/min)	3.19 ± 0.63	3.12 ± 0.67	0.741
Cardiac index (L/min/(m <sup>2</sup> ) <sup>1.0</sup> )	2.48 ± 0.46	2.36 ± 0.42	0.391



436 Data are mean  $\pm$  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
<i>Longitudinal</i>			
Strain (%)	-20.3 ± 1.6	-19.6 ± 2.5	0.283
SRS (s <sup>-1</sup> )	-1.07 ± 0.11	-1.11 ± 0.18	0.427
SRE (s <sup>-1</sup> )	2.22 ± 0.40	2.02 ± 0.46	<b>0.025</b>
SRA (s <sup>-1</sup> )	0.57 ± 0.10	0.66 ± 0.15	0.105
<i>Mitral Valve (Base)</i>			
Circumferential strain (%)	-22.2 ± 2.5	-20.5 ± 2.5	<b>0.029</b>
Circumferential SRS (s <sup>-1</sup> )	-1.29 ± 0.15	-1.29 ± 0.17	0.925
Circumferential SRE (s <sup>-1</sup> )	2.16 ± 0.37	2.08 ± 0.34	0.457
Circumferential SRA (s <sup>-1</sup> )	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 <sup>-1</sup> )	2.12 ± 0.50	2.22 ± 1.01	0.675
Radial SRE (s <sup>-1</sup> )	-2.54 ± 0.81	-2.46 ± 0.81	0.954
Radial SRA (s <sup>-1</sup> )	-0.74 ± 0.36	-1.08 ± 0.67	0.065
<i>Papillary Muscle (mid-ventricular)</i>			
Circumferential strain (%)	-20.1 ± 1.5	-18.3 ± 2.5	<b>0.007</b>
Circumferential SRS (s <sup>-1</sup> )	-1.17 ± 0.17	-1.19 ± 0.18	0.732
Circumferential SRE (s <sup>-1</sup> )	1.89 ± 0.33	1.57 ± 0.39	<b>0.001</b>
Circumferential SRA (s <sup>-1</sup> )	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 <sup>-1</sup> )	2.38 ± 0.89	2.47 ± 1.28	0.944
Radial SRE (s <sup>-1</sup> )	-3.64 ± 1.54	-3.35 ± 1.47	0.569
Radial SRA (s <sup>-1</sup> )	-1.08 ± 0.54	-1.60 ± 1.54	0.463
<i>Twist Mechanics</i>			
Apical rotation (°)	11.95 ± 5.31	7.58 ± 3.55	<b>0.005</b>

Basal rotation (°)	-5.69 ± 3.14	-5.64 ± 2.65	0.959
Twist (°)	16.92 ± 7.55	12.34 ± 4.99	<b>0.035</b>

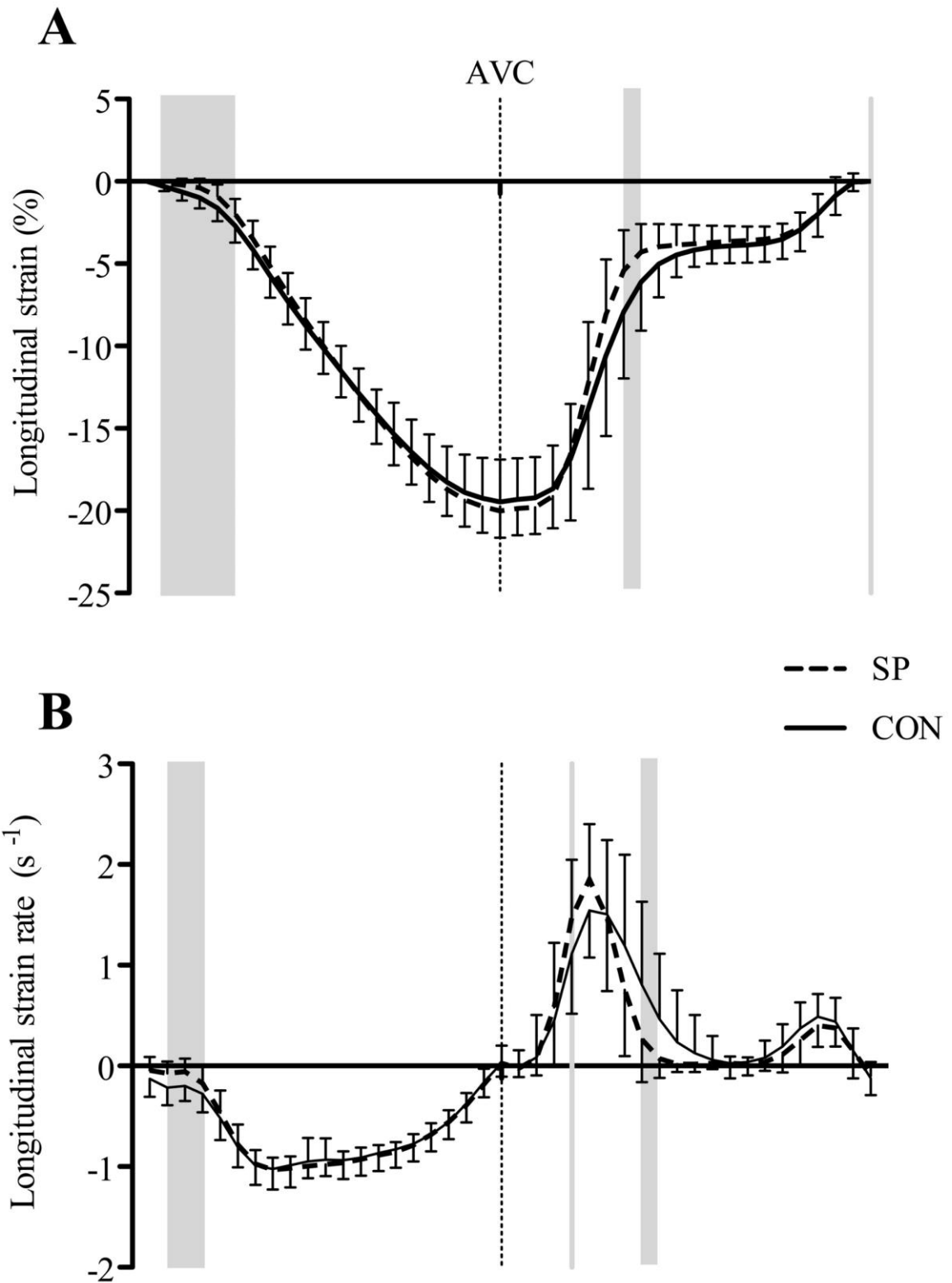
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442 Data are presented as means ± standard deviation. SRS, peak systolic strain rate; SRE, peak early  
443 diastolic strain rate; SRA, peak late diastolic strain rate.

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