Left ventricular biomechanics in professional football players

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Chronic exercise induces adaptive changes of left ventricular (LV) ejection and filling capacities which may be detected by novel speckle-tracking echocardiography (STE) and tissue Doppler imaging (TDI)-based techniques. A total of 103 consecutive male elite Norwegian soccer players and 46 age-matched healthy controls underwent echocardiography at rest. STE was used to assess LV torsional mechanics and LV systolic longitudinal strain (LS). Diastolic function was evaluated by trans-mitral blood flow, mitral annular velocities by TDI, and LV inflow propagation velocity by color M-mode. Despite similar global LS, players displayed lower basal wall and higher apical wall LS values vs controls, resulting in an incremental base-to-apex gradient of LS. Color M-mode and TDI-derived data were similar in both groups. Peak systolic twist rate (TWR) was significantly lower in players (86.4±2.8 vs controls 101.9±5.2 deg/s, P<.01). Diastolic untwisting rate (UTWR) was higher in players (−124.5±4.2 vs −106.9±6.7 deg/s) and peaked earlier during the cardiac cycle (112.7±0.8 vs 117.4±2.4% of systole duration, both P<.05). Untwisting/twisting ratio (−1.48±0.05 vs −1.11±0.08; P<.001) and untwisting performance (=UTR/TW; −9.25±0.34 vs −7.38±0.40 s−1, P<.01) were increased in players. Augmented diastolic wall strain (DWS), a novel measure of LV compliance in players, was associated with improved myocardial mechanical efficiency. The described myocardial biomechanics may underlie augmented exertional cardiac function in athletes and may have a potential role to characterize athlete’s heart by itself or to distinguish it from hypertensive or hypertrophic cardiomyopathy.

KEYWORDS
athlete, diastolic function, left ventricular function, speckle-tracking, strain, tissue Doppler, torsion

INTRODUCTION

Chronic exercise training induces adaptive changes in LV wall thickness, cavity dimensions, and LV and atrial function that are commonly referred to as “athlete’s heart”. Speckle-tracking echocardiography (STE) allows comprehensive evaluation of LV mechanics including myocardial torsional function and deformation, that is, LV strains. LV twist by STE, in particular apical rotation, is an important contributor to maintain stroke volume (SV) during preload reductions. LV twist and peak diastolic untwisting rate (UTR) increase with exercise in normal subjects. UTR is important in the generation of intraventricular pressure gradient, a key component of diastolic suction and early LV filling dynamics. In healthy subjects, relatively greater enhancements of LV torsional mechanics than LV strains upon graded submaximal exercise were reported.

Most data on LV torsion and regional deformation in athletes stem from small and heterogeneous cohorts. Neilan and co-workers showed enhanced LV twist and strains after high-intensity exercise in 17 elite rowers, but did not report on UTR. Whether athletes exhibit greater torsional reserve compared to other LV functional cues upon exercise is unclear. Nottin reported lower resting LV torsion and apical radial strain in 16 male elite cyclists. In addition, the same group reported delayed and reduced torsion and untwisting...
in 23 triathletes after competition ultralong duration exercise. Some authors reported reduced torsion and rotational velocities in soccer players; others found the opposite or no differences. Thus study settings, the duration and nature of exercise, its timing to echocardiographic assessment, and as a consequence, loading conditions differ greatly between those reports. The evidence regarding not only LV deformation and torsion, but also myocardial velocities in athletes, is also divergent. Diastolic wall strain (DWS) has been recently proposed to reflect LV compliance, but no data have been reported in athletes.

Thus, this study aimed to comprehensively describe LV myocardial velocities, strains, torsional biomechanics and LV compliance in a large homogenous cohort of elite soccer players in comparison with age-matched healthy controls.

2 | MATERIALS AND METHODS

2.1 | Study population

This work is a substudy to a cohort of 594 male professional soccer players from the top two Norwegian Soccer Leagues examined during off-season training camps in La Manga, Spain. The first 103 consecutive players and 46 age- and weight-matched male control subjects were examined by experienced cardiologists (KS and AH) with special expertise in analysis of strain and torsion. Controls were eligible if they exercised on a recreational basis of no more than 3 h/wk, excluding vigorous or competition-level exercise. Investigations were performed approximately 2 months into the new season. Players had an average weekly exercise burden of 15-20 hours and competed in 40-70 soccer matches (45 minutes×2) per year. The protocol was approved by the institutional ethics committee.

2.2 | Baseline data and studies

All study subjects were asked to abstain from training on the day prior to examination, as well as caffeine, alcohol, and tobacco products. After written informed consent, study subjects completed a questionnaire assessing demographics, including previous medical history and family history of sudden cardiac death. Supine blood pressure, heart rate, and an electrocardiogram were taken after a rest of at least 15 minutes. The rate-pressure-product (RPP) was used as a surrogate of myocardial oxygen consumption:

\[ RPP = \text{systolic blood pressure} [\text{mm Hg}] \times \text{heart rate} [\text{beats per minute}] \]

Myocardial mechanical efficiency was estimated as previously described: Myocardial mechanical efficiency = Stroke work/RPP, where stroke work is defined as systolic blood pressure×stroke volume×0.014.

Subsequently, two-dimensional and Doppler echocardiography was performed as described. Conventional echocardiographic and Doppler data were measured as recommended by the American Society of Echocardiography.

2.3 | Speckle-tracking (STE), color M-mode, and Tissue Doppler echocardiography

Peak global longitudinal strain was derived using STE of standard two-dimensional (2D) apical long-axis, four-chamber, and two-chamber projections as described.

For rotation and global twist analysis, standard basal 2D short-axis images were recorded at the level of the mitral annulus and at the apical level just proximal to the level that showed luminal closure at end-systole. After manually tracing the endocardial border in end-systole, the software automatically detected myocardial motion subdivided into six standard equidistant segments. The width of the region of interest included the entire myocardium and was adjusted to provide optimal tracking. The STE algorithm estimated the average rotation of the six segments predefined by the software where peak systolic twist (TW) could be extracted at each short-axis level. Naturally, current 2D STE only allows for sequential, not simultaneous recording of apical and basal rotation. Twist was calculated as the instantaneous difference of basal and apical rotation. Rotation rates were calculated from the first derivative of the rotation data over time; peak systolic twist rate (TWR) and peak diastolic untwisting rate (UTWR) were defined as the peak positive and negative slopes of the twist curve. Untwisting/twisting ratio was defined as UTWR divided by TWR. Peak untwisting performance, a novel age-independent diastolic function index, was defined as peak untwisting rate divided by peak twist.

In order to assess temporal distribution of events, we normalized time and twist data to systole duration where the onset of the QRS was defined as t=0% and aortic valve closure defining end-systole (=100%).

LV flow propagation \( V_p \), an index of LV suction during diastole, was determined from color M-mode Doppler images with the cursor placed through the mitral leaflet tips in parallel with LV inflow and sample volume from basal level to the apex. \( V_p \) was determined by tracking the slope of the first clearly demarcated isovelocity line during early filling from the mitral valve plane to 4 cm distally into the left ventricular cavity. Peak systolic \( S_m \), early \( E_m \) and late \( A_m \) diastolic mitral annulus velocities were obtained by TDI, and the \( E/E_m \) ratio employed as surrogate for LV filling pressures. Diastolic wall strain, an index of myocardial stiffness, was calculated using the formula: DWS=posterior wall thickness at end-systole (PWs) - posterior wall at end-diastole (PWd)/PWs.
All recordings were analyzed blinded and off-line by an experienced cardiologist using commercially available customized software within a personal computer workstation (EchoPac vers108.1.0, GE Vingmed).

2.4 | Statistical analysis
Statistical analysis was performed using GraphPad prism 5.0 (GraphPad Inc., La Jolla, CA, USA) and MedCalc, MedCalc Software, Ostend, Belgium). Data are presented as mean±SEM. Intra- and between-group comparison was made by two-tailed paired and unpaired Student’s t test or ANOVA where appropriate. To assess inter- and intraobserver variability, five variables in seven randomly selected study subjects (players or controls) were analyzed by two different observers, using the intraclass correlation coefficient, two-way model for estimation of reliability of single ratings, and Pearson’s correlation. P values <.05 were considered statistically significant.

3 | RESULTS
3.1 | Study subject characteristics and conventional echocardiography
Baseline characteristics are shown in Table 1. Echocardiography revealed greater LV wall thickness, greater LV end-diastolic and stroke volumes, and greater computed LV mass. Left atrial volume was approximately 50% higher in players. LV diastolic wall strain was 25% higher in the athletes compared to the controls consistent with augmented LV compliance. DWS correlated inversely with RPP (R=−.35) and positively with myocardial mechanical efficiency (R=.39; both <.00001).

3.2 | LV strains and torsional function
Peak longitudinal strain (LS) in each of the three standard apical projections and computed peak global LS were not different between players and controls (Table 2). Players exhibited significantly lower basal (−17.0±0.3 vs −18.1±0.4%; P<.05) and higher apical (−21.6±0.3 vs −19.7±0.7%; P<.01) peak LS than controls, whereas midwall LS was similar between groups (Figure 1A). Paired analysis revealed that midwall LS was significantly higher compared to basal LS within both groups. However, only players showed higher apical vs basal and vs midwall LS. This is consistent with incremental base-to-apex gradient of LS in the players vs controls (mean difference of apical minus basal LS −4.6±0.4 vs −1.5±0.8%; P<.001; Figure 1B).

Timing and magnitude of LV apical and basal rotation and LV twist were comparable in both groups (Table 3).

Apical rotation accounted for 66.3±2.2 and 67.9±3.7% of LV twist in players and controls, respectively (ns). This relatively greater contribution to systolic torsional function of apical myocardium was further illustrated by a stronger correlation between apical rotation and LV twist (Figure 2A) as well as twisting rate (Figure 2B) than basal rotation. Peak basal rotation rate was lower in players, contributing to a significantly lower peak systolic twisting rate (Table 3).

Importantly, peak diastolic untwisting rate was not only higher in players but also peaked significantly earlier (Table 3). Normalization to LV length did not change the significant intergroup differences of peak LV twist, peak twisting

| Parameter                           | Players (n=103) | Controls (n=46) |
|-------------------------------------|----------------|-----------------|
| Age (y)                             | 26.1±0.4       | 26.4±1.0        |
| Height (cm)                         | 184±1          | 182±1           |
| Body weight (kg)                    | 80.3±0.6       | 78.0±1.1        |
| BSA, body surface area (m²)         | 2.02±0.01      | 1.98±0.02       |
| SBP, systolic blood pressure (mm Hg)| 123±1          | 124±2           |
| DBP, diastolic blood pressure (mm Hg)| 70±1           | 71±1            |
| HR, heart rate (beats/min)          | 50±1***        | 63±2            |
| Rate-pressure product (mm Hg/min)   | 6361±125***    | 7567±225        |
| Myocardial mechanical efficiency (mL/s)| 110±3***    | 70±3            |
| Echocardiography                    |                |                 |
| AWT, end-diastolic anterior wall thickness (mm) | 8.6±0.1**     | 8.0±0.1         |
| PWTd, end-diastolic posterior wall thickness (mm) | 8.6±0.1**     | 8.0±0.1         |
| PWTs, systolic posterior wall thickness (mm) | 14.8±0.2**    | 11.9±0.3        |
| LVEDD, LV end-diastolic diameter (mm) | 54.4±0.4*      | 52.5±0.8        |
| LVSD, LV end-systolic diameter (mm) | 35.4±0.4       | 36.7±0.7        |
| RWT, relative wall thickness        | 0.32±0.01      | 0.31±0.01       |
| DWS, diastolic wall strain          | 0.41±0.01***   | 0.32±0.01       |
| LVM, LV mass (g)                    | 173±3***       | 150±5           |
| LVEF, LV ejection fraction (%)      | 57.9±0.5**     | 55.5±0.6        |
| LVEDV, LV end-diastolic volume (mL) | 158±3***       | 125±4           |
| LVSV, LV stroke volume (mL)         | 91.3±2.1***    | 69.3±2.3        |
| Left atrial volume (mL)             | 87.9±2.1***    | 55.1±3.0        |

Values are means±SEM.
*P<.05, **P<.01, ***P<.001, players vs controls (unpaired t test).
or peak untwisting rates (data not shown). In addition, players exhibited higher untwisting-to-twisting-rate ratio and higher peak untwisting performance than controls. We also found a modest but significant correlation between apical and basal rotation and untwisting rate (Figure 2C). Simple correlation analysis revealed modest, significant correlations between LV untwisting rate and indices of diastolic function $E/A$, $A_m$, diastolic wall strain and diastolic BP (all $P<0.05$).

### 3.3 | Myocardial velocities and LV filling patterns

$E/V_p$ and $E/E_m$ ratios, indices of LV filling pressures, were similar between both groups (Table 4). In contrast, $A_m$ but not $E_m$ was lower in players, and the difference in the ratio of early-to-late diastolic myocardial velocities ($E_m/A_m$) was close to reach statistical significance.

### 3.4 | Reproducibility of measurements

For interobserver reliability, intraclass correlation coefficients (95% CI) were as follows: apical rotation, 0.87 (0.42-0.98); basal rotation, 0.94 (0.70-0.99); twist 0.85 (0.35-0.97); %systole duration at peak untwisting rate, 0.79 (0.19-0.96).
and untwisting rate, 0.94 (0.69-0.99); and for intraobserver reliability, apical rotation, 0.95 (0.76-0.99); basal rotation, 0.81 (0.25-0.96); twist, 0.92 (0.62-0.99); untwisting rate, 0.61 (−0.18 to 0.92) and %systole duration at peak untwisting rate, 0.97 (0.84-0.99). Pearson's correlations were as follows: for interobserver variability, apical rotation, \( r = 0.88 \); basal rotation, \( r = 0.94 \); twist, \( r = 0.86 \); twisting rate, \( r = 0.85 \) and untwisting rate, \( r = 0.95 \) (all \( P < 0.0001 \)). For intraobserver variability, Pearson's correlations were as follows: apical rotation, \( r = 0.87 \); basal rotation, \( r = 0.96 \); twist \( r = 0.87 \); twisting rate, \( r = 0.86 \) and untwisting rate, \( r = 0.95 \) (all \( P < 0.0001 \)).

4 | DISCUSSION

We herein present novel data on myocardial torsional function and regional deformation in male professional soccer players with predominantly eccentric LV remodeling.

First, we demonstrated a unique pattern of lower basal and higher apical LS consistent with an increasing base-to-apex

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**TABLE 4** Doppler, tissue Doppler, and color M-mode-derived left ventricular functional parameters

| Parameter                          | Players (n=103) | Controls (n=46) |
|------------------------------------|----------------|-----------------|
| \( E \) (m/s)                      | 0.78±0.01*     | 0.84±0.03       |
| DT, \( E \) deceleration time (ms) | 172±3          | 179±4           |
| \( A \) (m/s)                      | 0.39±0.01**    | 0.44±0.02       |
| \( E/A \)                          | 2.11±0.05      | 1.95±0.07       |
| \( E_m \) septal (cm/s)            | 10.8±0.2       | 11.1±0.4        |
| \( E_m \) lateral (cm/s)           | 16.7±0.3       | 17.0±0.5        |
| \( E/E_m \) septal                 | 6.34±0.10      | 6.61±0.26       |
| \( A_m \) (cm/s)                   | 6.0±0.2**      | 6.8±0.3         |
| \( E/E_m \) septal                 | 3.00±0.10      | 2.68±0.13       |
| \( V_p \) early diastolic propagation velocity (cm/s) | 56.8±1.6 | 56.5±2.1 |
| \( E/V_p \)                        | 1.46±0.04      | 1.54±0.08       |
| \( S_m \), peak systolic velocity (cm/s) | 10.0±0.2 | 10.7±0.3 |

*\( P < 0.05 \), **\( P < 0.01 \), ***\( P < 0.001 \), players vs controls (unpaired t test).
gradient of LS in players, despite similar average global LS values. Second, systolic twisting rate was lower in players compared to controls. Third, untwisting rate was higher and peaked earlier in players. Finally, this is the first report of increased diastolic wall strain in athletes consistent with greater LV compliance.

4.1 Regional deformation

Despite slightly higher LV stroke volume, LV mass and EF, peak LS in each of the three standard apical projections as well as computed peak average global LS was not different in soccer players vs controls. This is consistent with findings in healthy controls and competitive rowers showing similar peak global LS despite marked cardiac hypertrophy, greater stroke volumes, and a non-significant 5% greater EF in rowers. Others also reported similar LS in athletes vs controls.

The pattern of regional systolic LV deformation in soccer players deserves further comment: at similar midwall LS values in both groups, players displayed lower basal LS values and higher apical LS values than controls. Moreover, paired analysis revealed a significant incremental basis-to-apex gradient of LS in players, while controls did not exhibit such regional differences in systolic LV deformation. Other studies in athletes did not consistently report such regional levels of LS. Our data are in line with other reports showing that global average LS increased only modestly upon exercise, if at all, and without differences between apical and basal strain. The relevance of the distinct pattern of increasing regional systolic deformation from base to the apex in this study is unclear.

Gelderis et al. have shown correlations between global LS and $E/E_m$ in a heterogenous pool of rowers, controls, and hypertensives; however, the correlation in athletes only seemed far weaker. In our homogenous cohort, we were not able to identify a significant correlation between LS and $E/E_m$, other diastolic or systolic indices in our study population.

4.2 Torsional mechanics

Peak apical and basal rotation and consequently, systolic LV twist were similar in players and controls, even after adjustment for LV length, in line with a recent longitudinal study on the effects of exercise. Peak twist was better correlated with apical than basal rotation, which has previously been described and might be due to relatively greater through-plane movements of the LV base. Systolic twisting rate was lower in players, presumably reflecting basal myocardial properties and consistent with lower basal peak LS values and lower basal rotation rates. In support of this, systolic twisting rate coincided with peak basal rotation rate. Interestingly, peak twisting rates correlated with RRP (data not shown) which may suggest a link between systolic torsional deformation and myocardial oxygen consumption and energy efficiency.

A key finding of the current study was markedly enhanced peak untwisting rate in players. Peak LV untwisting rate was reported to best reflect LV relaxation. As a consequence, the ratio of twisting-to-untwisting rates which has been shown to increase with exercise in healthy subjects was substantially higher in players than controls. Untwisting performance was significantly greater in players. LV untwisting is a major determinant of very early diastolic function at rest and upon exercise, allowing for efficient LV filling. A study in healthy subjects found the exercise-induced increase in peak untwisting rate to be greater than that of LV strain rates.

The current study is the first to report that peak untwisting rate occurred significantly earlier in athletes. In line with this finding, Notomi et al. reported increased magnitude and a (non-significant) trend for earlier peak untwisting rate in healthy subjects during exercise. Also others reported augmented untwisting acutely during exercise in endurance athletes and following long-term structured exercise program. In contrast, delayed untwisting has been shown in ischemia, valvular heart disease, and hypertrophic obstructive cardiomyopathy (HOCM). This may suggest a link between systolic torsional deformation and myocardial mechanical efficiency indicating a link between systolic torsional deformation and myocardial mechanical efficiency.
LV compliance and myocardial energy consumption. Indices of LV relaxation, $E_m$ and $V_p$, and indices of LV filling pressures, $E/E_m$ and $E/V_p$, were similar in both groups. Lower $A$ and $A_m$ and higher $E_m/A_m$ in players also suggest lower left atrial pressures during late diastolic LV filling.

Lower $A$ and $A_m$ velocities in players may indicate that LV filling, possibly due to increased LV compliance, requires less atrial contraction and lower atrial pressure during late diastole. Another study in mixed athletes including soccer players reported enhanced early diastolic ($E_m$) myocardial velocities and $E_m/A_m$ ratios. The generalizability of that study is limited by the fact that athletes were recruited from different sports with varying exercise volumes and intensities, and further by the lack of a sedentary control group. In competitive rowers, reductions in $E/A$ ratio and $E_m$ and increase in $A_m$ have been shown after exercise. Accordingly, early relaxation-dominated LV filling at rest transitions to late atrial contraction-dominated filling upon exercise.

5 | LIMITATIONS

We should address some important issues mainly relating to study design and methodology. Due to time and logistical constraints, one entire football team consisting of 25-35 players plus controls was examined per day. Following a tight schedule, players were shuttled forth and back between the training camp and the nearby investigation facility in groups of six players every hour. This commanded a straightforward echocardiography protocol and may sometimes have limited the opportunity to obtain optimal images.

Current echocardiography-based assessment of torsion requires basal and apical short-axis images which cannot be obtained simultaneously nor allows adjusting for the distance between the two imaging planes. Through-plane motion is intrinsic to current two-dimensional STE and may attenuate myocardial tracking in particular at the basis. Real-time 3D STE may overcome these spatial limitations although temporal resolution is limited by low frame rates.

Approximately half of control subjects were recruited from support staff such as assistant coaches, physiotherapists, logistics personnel. Some of the controls are retired athletes and may therefore exhibit better cardiovascular fitness than the normal sedentary population, indicated by the relatively low resting heart rate and improved diastolic function compared to control subjects of comparable studies. This may possibly have reduced between-group differences although both cohorts were otherwise well-matched regarding age and BMI. In addition, many LV diastolic functional cues are naturally interrelated, as shown by author authors which may obscure the added value of these novel techniques in a clinical setting.

Finally, application of preload-altering maneuvers or stress testing would have enhanced the variance to which torsional and other parameters could have correlated. Logistical constraints did not allow for these. In addition, information on exercise capacity of individual players was considered of potential market value and was therefore not accessible for the investigators.

6 | CONCLUSIONS

Our findings suggest that basal-to-apical gradient of peak systolic LV strain, improved and earlier-onset peak diastolic untwisting rate and supranormal LV compliance associated with improved myocardial mechanical efficiency constitute important cardiac biomechanics in elite soccer players. Further studies are required to fully understand the potential relevance of these cardiac mechanics to exertional cardiac function and metabolic efficiency in athletes.

7 | PERSPECTIVES

Precise characterization of athlete’s heart by echocardiography helps clinicians to distinguish physiological cardiac adaption from maladaptive cardiac remodeling. We have recently described patterns of physiological cardiac remodeling in a large cohort of Norwegian professional football players in this journal. In the present paper, we focus on more profound changes of LV systolic and diastolic function in athlete’s heart by novel echo methods.

Our work is the first to show earlier-onset and enhanced diastolic untwisting rate in athletes, consistent with “supranormal” early diastolic function. In addition, we demonstrate basal-to-apex gradient of systolic LV strain, and increased LV compliance (evidenced by diastolic wall strain) in soccer players vs controls. Together, these myocardial biomechanics may underlie augmented exertional cardiac function in athletes and may have a future role to characterize athlete’s heart by itself or to distinguish it from for example hypertrophic cardiomyopathy or hypertensive left ventricle hypertrophy.

Our findings may therefore be applied to a wide range of athletes that are expected to develop exercise-induced adaptive LV remodeling. Assessment of diastolic untwisting, LV deformation, and compliance in addition to traditional echocardiographic evaluation in athletes may provide unique information on cardiac biomechanics.

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The authors have no disclosures to declare.

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