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Atrial size and function in athletes

Abstract
We wanted to explore if change in LA size would influence LA function, or increase regurgitation in the atrioventricular valves. 595 male elite football players and 47 non-athletic controls were included. End-systolic LA volume and RA area and end-diastolic LV volume and RV area were measured by two-dimensional (2D) echo. Pulsed and colour Doppler were used to estimate tricuspid and mitral regurgitations. 2D longitudinal strain of the 50 football players with the largest LA volumes were compared with the 50 players with the smallest LA volumes. The LA volumes in some athletes were more than tripled, compared to athletes with small atria. 2D strain however, could not reveal any impairment of LA function in the players with the largest atria, compared to those with the smallest LA. Tricuspid valve regurgitation was found in 343 (58%) of the athletes, compared to 17 (36%) of the controls ($p<0.01$), while mitral regurgitation was found in 116 (20%) football players and seven (15%) controls (NS). Furthermore, the RA area was significantly larger in athletes with tricuspid regurgitation compared to athletes without. The present study demonstrated a huge variation in atrial size between the athletes. This variation, however, had no impact on LA function. Tricuspid regurgitation was significantly more prevalent among the athletes, than among the controls.

Keywords: Athlete's heart, elite football players, right atrium, left atrium

Introduction

The term "athlete's heart" denotes the physiological adaptations that take place in athletes to meet the demand for higher cardiac output during exercise.[3,10,11,19,29,34] These adaptations include i.a. augmented left ventricle (LV) mass, dilatation of the heart chambers, and a larger stroke volume (SV). Different types of sports also require different types of cardiac remodelling and function.[10] Elite football players, like those in our study, need to be able to perform fast sprints, but also need to compete for 90 minute matches, and are therefore classified as low static/high dynamic or endurance athletes.[4,5,33]

While LV has been the focus of most studies among athletes, the atrias' reservoir, conduit and pump function phase have attracted more and more attention.[1,7,26] It has been shown that large left atrial (LA) diameter and volume are quite common, particularly among athletes in endurance sports.[6,12,27] However, there is little or no data on the variance of LA size and on right atrium (RA) chamber size in athletes.[18,20] Definitions of normal upper limits for the size of RA and LA in athletes are also lacking.

Echocardiography is normally the tool of choice when it comes to assessment of heart chambers. Over the past few years there has been a rapid development in image reproduction of standard methods as two-dimensional (2D) echo and pulsed Doppler as in novel echo methods such as tissue Doppler imaging and not least 2D strain, which have proved to be more sensitive to changes in heart function than standard methods.[32,37] To our knowledge, no studies have been performed on LA function using these novel methods. In a previous study of the same healthy athletes, ECG and P-wave were found to be unaffected by atrial size.[28]

The main aim of this study was therefore to investigate the remodelling of both LA and RA in athletes in high dynamic, low static sports like football, and to suggest upper normal size limits for both atria. Since the enlargement of atrial size might affect valvular adaptation, we also explored whether it could lead to more regurgitation in both atrioventricular valves, or to changes in its reservoir, conduit, or pump function. Finally, we wanted to cast light on the possible mechanisms behind the enlargement of LA and RA size in athletes.

Methods

Inclusion. The inclusion and examination were done during a pre-season training camp in La Manga (Spain), February-April 2008. The cardiac screening was performed by 11 experienced cardiologists, one scientific fellow, and one cardiology fellow, who were all trained in echo. All came from hospitals in the Oslo region. This cross-sectional study comprised 28 of a total of 30 football clubs in the Norwegian elite and 1st division leagues. 604 athletes, aged 18 to 38, were screened for inclusion. Two players were excluded due to identification problems, and nine were excluded because of lack of written consent. Finally, 595 football players were included in the study. Most of the athletes were Caucasian (504, 85%); however, a significant number were of African origin (49, 8%), and some were of mixed or other origin (42, 7%).[12] 47 male sedentary controls (training < 3 hours per week), matched for age and body size, were also included during the same period (Figure 1). The control participants were either included from the support teams, or were volunteers from the Oslo region. The control participants underwent the same examinations as the players. All study subjects gave written informed consent before enrolment in the study, which was approved by the local Regional Ethics Committee. The study was also performed in accordance with ethical standards for sports and exercise science research.[14]

Echochardiographic recordings. All echocardiographic recordings were performed with a 2.5 MHz transducer (Vivid 7 and Vivid i; GE Vingmed Ultrasound AS, Horten, Norway). The digital data were transferred to a computer (Dell Optiplex 755) for off-line analysis (EchoPAC PC'08 (BT08); GE Vingmed Ultrasound AS, Horten, Norway). One investigator (GFG) performed the off-line analyses blinded for group allocation.

Atrial and ventricular size. LA size was measured as LA diameter in a parasternal long-axis view, LA area from 4 chamber view, and LA volume by biplane apical 2 and 4 chamber views, using the area length method (Figure 2). All measurements were performed in end systole.[18] LV end-diastolic and end-systolic volume were measured by modified Simpson's rule from biplane 2 and 4 chamber views [18,31] and end-systolic annulus diameter for both mitral and tricuspid annulus from an apical 4 chamber view in end diastole. For the quantification of end-diastolic area of the right ventricle (RV area) and end-systolic area of the right atrium (RA area), apical 4 chamber view was used. RA end-systolic diameter (RA diameter) was measured as the RA axis perpendicular to the atrium's length axis.[16,18,30] In accordance with recommendations,[9,18] relevant data regarding chamber quantification are indexed to body surface area (BSA) and marked $_I$, i.e. LA diameter$_I$. The simplified formula by Mosteller (1987) was used to calculate BSA.[21]

Atrioventricular regurgitations, hemodynamics and LA function. Continuous wave Doppler and colour flow mapping were used to identify individuals with tricuspid regurgitations. Maximum jet velocity estimates the pressure gradient (PA) between the RV and RA, using the simplified Bernoulli equation.[30] In cases where maximum jet velocity was difficult to detect, the regurgitation area was measured by colour flow Doppler to identify individuals with tricuspid regurgitation. For mitral regurgitation, calculations of vena contracta were performed from the parasternal view, while the length and area were calculated from apical 4 chamber view. Standard definitions for the quantification of mitral regurgitation[36] were used to divide into minimal, mild, moderate and severe regurgitation.

Early diastolic (E') and atrial (A') peak velocity at the septal and lateral part of the mitral valve were calculated by tissue velocity imaging, and averaged. The ratio between early transmitral peak velocity (E) by pulsed Doppler and E' was calculated as an estimate for LV filling pressure.[22] Two dimensional (2D) strain analyses were performed on LA loops, with frame rates between 60 and 100 frames/s from 4 chamber view, to estimate LA reservoir, conduit and pump function. An average of six segments or regions of interest of these strain analyses were calculated and defined as LA global strain (Figure 2).[26] The regions of interest were identified automatically, and adjusted manually in accordance with the thinner walls of the left atrium. Regions of interest that could not be tracked were discarded and not included in the data processing. To cast light on changes associated with large LA, the 50 athletes with the largest atria were compared with the 50 athletes with the smallest atria.

Statistics. The results are presented as averages with standard deviations (SD). When comparing two different groups, a two-sided student t-test was performed, and Chi-square was used as statistical analysis for proportions. To determine the influence of independent variables, standard linear regression analysis was performed, and a multivariate regression model was constructed, using univariate p-value below 0.25. Reproducibility data were tested (with F-test, test value 0) by intra-class correlation coefficient (ICC) for LA area in 10 randomly selected athletes. The measurements were done by two of the authors (ZR and GFG). The statistical analyses were performed using the statistical software SPSS 18.0 (PASW Statistics 18, IBM Corporation 2010, NY, USA). Differences were considered significant when $p<0.05$.

Results

Demographic data. General characteristics of the test subjects are shown in table 1. The athletes' end-systolic LA $area_I$, LA $volume_I$ and end-diastolic LV $volume_I$ were 23%, 32% and 17% larger, respectively (Figure 3A). Comparable changes were observed for RA $diameter_I$ (10%), RA $area_I$ (22%) and RV $area_I$ (11%) (Table 2 and Figure 3A). Indexed for BSA, the LA volume of 79% of the athletes exceeded normal ranges (Figure 3B), while all athletes fell within the normal reference range (1.5-2.3 cm/m^2) for indexed LA diameter. There were no significant differences in LA or RA sizes between athletes of different ethnicity.

Tricuspid regurgitation. Traces of mild tricuspid regurgitation was found in 343 (58%) of the athletes, compared to 17 (36%) among the controls ($p < 0.01$). There was no difference between these two groups in maximum tricuspid regurgitation velocity, 2.15 ± 0.2 vs. 2.15 ± 0.2 m/s.

Football players with tricuspid regurgitation had significantly larger tricuspid annulus diameter, 17.0 ± 2.4 vs. 16.6 ± 2.2 mm ($p < 0.05$), compared to athletes without tricuspid regurgitation. There was no difference in demographic data between these two groups of athletes. Athletes with tricuspid regurgitation also displayed enlarged RA $diameter_I$, 22.8 ± 3.0 vs. 22.0 ± 3.0 mm/m^2 ($p < 0.05$), RA $area_I$, 11.0 ± 1.9 vs. 10.4 ± 1.9 cm^2/m^2 ($p < 0.05$), and RV $area_I$, 13.8 ± 2.3 vs. 13.3 ± 2.4 cm^2/m^2 ($p < 0.05$), when compared to controls. Tricuspid regurgitation, maximum velocity above 2.5 m/s and below 3 m/s, was found in 19 athletes and one control.

Mitral regurgitation. All in all, mitral regurgitation was found in 116 (20%) football players and 7 (15%) controls (NS). There was no difference in demographic data, vena contracta, 1.8 ± 0.5 vs 1.9 ± 0.8 mm (NS), or mitral regurgitation area, 2.0 ± 1.2 vs. 1.3 ± 0.6 cm^2 (NS), in athletes and controls with mitral regurgitation. Athletes with regurgitations had a significantly larger mitral annulus $diameter_I$, 18.6 ± 2.0 vs. 17.9 ± 2.0 mm/m^2 ($p < 0.001$), compared to athletes without. Similarly, LA $diameter_I$ was larger in athletes with mitral regurgitation, 17.8 ± 1.7 vs. 17.3 ± 1.8 mm/m^2 ($p < 0.05$), but there was no difference in LA $area_I$, LA $volume_I$, or LV $volume_I$. Three athletes had clinically mild mitral regurgitation, and 113 athletes had traces of mitral regurgitation.

Multilinear regression analysis. In a model of linear regression analysis with LA volume and RA area as dependent variables, and age, BSA, systolic BP, HR and SV as independent variables ($p < 0.05$), univariate analysis showed that SV could explain 21% ($R^2 = 0.21$) of the variation in LA volume ($p<0.001$), and only 6.9% of the variation in RA area ($p < 0.05$). The other variables accounted for five to seven percent of the changes in LA volume and RA area. In multiple regression models including all of the above-mentioned variables, 26% of change in LA volume and 17% of change in RA area could be explained by the model ($p<0.01$). Age, BSA, HR and SV were all highly significant. None of the independent variables had inter-correlations above 0.7. E/E' did not have a significant impact on LA volume (NS).

LA function. Demographic, LA size and function of the 50 athletes with the smallest and largest atria are presented in table 3.

Variability. The intra-class coefficient of inter-observer variability for LA area were tested in 10 subjects and compared to an experienced cardiologist (ZR). The results with confidence interval (CI) were respectively 0.95 (0.78-0.99) and was significant ($P<0.001$).

Discussion

The study demonstrated a significant variation in the size of both atria among the football players, varying from low normal size to more than twice the upper normal reference values. Moreover, compared to controls, the football players' atria were disproportionally enlarged compared to the corresponding ventricles (Figure 3). Although the athletes presented markedly more tricuspid regurgitation, we could not document any major pathological changes regarding atrial function.

LA enlargement. As expected, as a consequence of the physiological adaptation to training, our football players presented much larger atria than the sedentary controls. The majority of previous studies have used diameter by recordings from the parasternal long axis view to evaluate LA size.[13,15,27] A study by Pelliccia et al. reported a prevalence of only 20% of augmented LA size, when the upper LA diameter limit was set at 40 mm.[27] Compared to previous studies, however, our study shows markedly larger LA volumes in athletes performing high dynamic and low static sports. D'Andrea et al. 2010 showed on average LA ESV of 28.2 ± 6.2 ml/m^2 in male athletes in both strength and endurance sports,[6] and D'Ascenzi et al. 2011 found indexed volumes of 26.3 ± 4.3 ml/m^2 in 23 male soccer players.[7] There are, however, several reasons that can explain this discrepancy, e.g. different recording conditions, different calculation methods,[1] disproportionate numbers of high static/low dynamic athletes,[6] and small sample sizes.[7]

We have documented that measurement of LA volume reflects enlargement of LA more precisely than LA diameter, which clearly underestimates LA size. This point of view is substantiated by three arguments; firstly, the present study has demonstrated that the correlation between LA volume and SV was markedly higher than between LA diameter and SV (Figure 4). Secondly, the enlargement in the anterior-posterior direction between sternum and column is limited, with the consequence that the expansion in the longitudinal and lateral directions is not accounted for by the LA diameter method. Thirdly, 79% of the subjects in our study showed indexed LA volume above normal range, but none of the athletes had an LA diameter that exceeded upper normal limits.[18] Based on the results from the present study and the mean + 2SD, we suggest 57 ml/m^2 as the upper normal limit for LA indexed volume in athletes, which is considerably more than we have suggested for the left ventricle.[12]

Pelliccia et al. 2005 suggest that LA enlargement in athletes is largely explained by LV cavity enlargement in dynamic sports.[27] Other studies, such as D'Andrea et al. 2010, found age, number of years of training, endurance type, LV EDV, and male gender to be important factors for LA volume enlargement.[6] We were, however, not able to detect predictors for the increased LA size in our material. Age, BSA, HR and SV were all significant, but had little impact in the model, which could indicate that genetic factors are of great significance for the documented great variation in LA size.

RA enlargement. Few studies have focused on the quantification of RA size, and guidelines are rather vague.[18] Due to its irregular shape and the impossibility of biplane recording, RA is measured as an area by using the apical 4 chamber view. Further, it is not possible to perform reliable calculations of RA volume by single plane 2D echo.[18] The minor-axis dimension, perpendicular to the long axis of the RA, is therefore often used to estimate RA size. A reference range for healthy individuals has been defined with regards to this dimension, but not for RA area.[18,35]

When comparing upper limits for RA area in athletes, as suggested by Zaidi et al.[35], 27 (4.6%) of our players exceeded the suggested limit of 28 cm^2, and 28 (4.7%) exceeded the suggested limit when indexed values (14 cm^2/m^2) were used. However, compared with guidelines for ordinary subjects, almost 50% of our athletes displayed enlarged dimensions when the RA axis was not indexed to BSA, while only 18% were considered enlarged when indexed values were used.[18] We hold this to support a routine practice of indexing length, area and volume measurements to BSA. As a consequence, guidelines should be determined for the RA area and the RA minor axis for both healthy individuals and athletes. As with LA, we recommend using mean + 2SD as the upper limit.

With regards to athletes, our study indicates upper limits of 14.5 cm^2/m^2 for RA area, and 2.9 cm/m^2 for the RA minor axis.

Atrioventricular valves. Although we have demonstrated a relatively large difference in tricuspid regurgitation between the football players and the controls, our data does not indicate this to be of clinical importance. The difference can probably be explained by the small, but significant change of RV and RA size and tricuspid annulus diameter due to the increased volume load in athletes. Enlargement of the RV will also impose a tethering effect via chordae tendinae on the annulus fibrosus that attaches the valvular system, which again leads to poorer adaptation of the valves and thus a higher prevalence of valvular regurgitations. Although these regurgitations are minor, they will result in a somewhat larger pendulating volume of blood, which again may increase the stretching force in the atria.

There was a significantly lower prevalence of mitral than tricuspid regurgitation, which is consistent with other studies in healthy individuals.[6,24] The relatively low prevalence of mitral regurgitation in our study is probably a reflection of our relatively young study population.[24]

LA function. In our athletes, RA and LA will expand their sizes about twice as much as their corresponding ventricles, which reflects a remarkable augmentation of the atrial reservoir function. Considering the thinner walls of the atria, compared to the ventricles, the question arises whether enlarged atrial size would influence atrial function. The greater E/E' in football players with larger LA volumes, compared to those with smaller LA volumes, might indicate increased LA and LV filling pressures. However, the use of this non-invasive index as a surrogate for LV filling pressure is based on patients with diseased LV function.[22,25] Besides, the greater E/E' was entirely driven by E, reflecting players with the largest stroke volume, and thus improved transmitral pressure gradient with enhanced rapid early diastolic filling. These findings were not consistent with those of D'Ascenzi et al., in which a much lower number of participants were included.[8] Finally, we did not find any difference in atrial global longitudinal strain between those with large and those with small LA volumes. On the other hand, the enlarged A' in those with large LA might indicate an improvement of the late atrial kick in these athletes.[17,23]

Limitations. The difference in numbers of participants between the athlete group and the control group may have compromised statistical power. However, all measurements were normally distributed with equal variance, fulfilling the most important criteria for the use of t-test, which is considered a robust statistical test.[2] Furthermore, it should be noted that our findings are limited to male professional football players in the age group 18 to 38. Volume estimation should have been performed on RA, to better compare the true change in size compared to LA. However, a reliable algorithm based on 2D recordings is not yet available. Future studies should therefore include three dimensional echo or magnetic resonance imaging.

Conclusion

Our study demonstrated a large variation in atria size among professional football players, and the enlargement of both atria was approximately twice that of their respective ventricles. Although there were significantly more tricuspid regurgitations among the athletes than among the controls, and while those players with regurgitations at tricuspid and mitral valves had larger RA and LA, we do not consider this to be of clinical importance. 2D strain in the players with the largest LA could not reveal any impairment of LA function.

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Figure title and legends

Figure 1:
Flowchart of the included athletes and control participants

Figure 2:
Panel A displays the traditional measure of left atrial (LA) diameter from a parasternal long axis view. Panel B and C illustrate volume measurements of LA from apical 4 and 2 chamber views. Panel D: Quantification of right atrium (RA) with area and minor dimension. Panel E illustrates 2D strain of the LA during one heart cycle. Further it shows regional and global deformation and reservoir, conduit, and pump function. Panel F: Peak early (E') and late (A') diastolic mitral annular velocity by tissue velocity imaging. Large and small LA are illustrated in panel G and H.

Figure 3:
Graph A illustrates the percentage difference (enlargement) of left atrial end systolic volume (LAESV), left ventricular end diastolic volume (LVEDV), right atrial end systolic area (RAESA), and right ventricular end diastolic area (RVEDA) in athletes (filled squares) compared to sedentary control subjects (open squares), and graph B the distribution of athletes' LA ESV and LV EDV in accordance with guidelines.

Figure 4:
Panel A shows the relationship between stroke volume (SV) and left atrial volume (LA ESV), and panel B between SV and left atrial diameter (LA ESD).

Table 1 Demographic data in 595 football players and 47 control individuals.

	Football players	Controls	P value
n	595	47	
Age, years	25.1 ± 4.6	26.2 ± 6.5	NS
Height, cm	182.7 ± 6.3	181.3 ± 5.8	NS
Weight, kg	79.1 ± 7.0	77.8 ± 7.8	NS
BMI , kg/m^2	23.7 ± 1.2	23.6 ± 1.7	NS
BSA, m^2	2.0 ± 0.1	2.0 ± 0.1	NS
Systolic BP, mmHg	122 ± 11	124 ± 11	NS
Diastolic BP, mmHg	69 ± 8	71 ± 7	NS
LVEF, %	56.3 ± 3.7	55.5 ± 3.6	NS
HR, beats/min	53 ± 8	62 ± 10	< 0.001

Data expressed as mean ± standard deviation. BMI = body mass index; BSA = body surface area; BP = blood pressure (systolic- and diastolic); LVEF = left ventricle ejection fraction; HR = resting heart rate;

Table 2. Chamber quantification in 593 football players and 46 control individuals.

	Football players	Controls	P value
N	593	46	
	LA data		
LA diameter, cm	3.5 ± 0.4	3.2 ± 0.4	< 0.001
LA diameter$_I$, cm/m²	1.7 ± 0.2	1.6 ± 0.2	< 0.001
LA area, cm^2	20.7 ± 4.4	16.6 ± 4.4	< 0.001
LA area$_I$, cm^2/m²	10.3 ± 2.2	8.4 ± 2.2	< 0.001
LA volume, ml	73.7 ± 20.8	55.2 ± 20.0	< 0.001
LA volume$_I$, ml/m²	36.8 ± 10.1	27.8 ± 10.1	< 0.001
	RA data		
RA diameter, cm	4.5 ± 0.6	4.1 ± 0.6	< 0.001
RA diameter$_I$, cm/m²	2.3 ± 0.3	2.1 ± 0.3	< 0.001
RA area, cm^2	21.4 ± 3.9	17.6 ± 3.9	< 0.001
RA area$_I$, cm^2/m²	10.7 ± 1.9	8.8 ± 1.8	< 0.001
	LV data		
LV volume, ml	145.8 ± 28.0	124. 8 ± 23.9	< 0.001
LV volume $_I$, ml/m²	72.8 ± 13.2	62.4 ± 11.4	< 0.001
	RV data		
RV area, cm^2	27.2 ±4.9	24.2 ± 4.0	< 0.001
RV area$_I$, cm^2/m²	13.6 ± 2.4	12.2 ± 1.8	< 0.001

Data expressed as mean ± standard deviation. LA diameter = left atrium diameter at end systole; LA area = left atrial area at end systole in an apical 4-chamber view; LA volume = left atrium end systolic volume; RA diameter = diameter of right atrium perpendicular to atriums` length-axis; RA area = right atrium end systolic area in an apical 4-chamber view; LV volume = left ventricle end diastolic volume; RA area = right ventricle end diastolic area.

Table 3. Demographic and echocardiographic data in the 50 athletes with the smallest LA volumes (LA small) and the 50 athletes with the largest LA volumes (LA large).

	LA small	LA large	P value
n	50	50	
Age, years	23.8 ± 4.1	25.7 ± 4.0	0.025
Height, cm	183.5 ± 6.3	182.8 ± 5.9	NS
Weight, kg	79.1 ± 7.7	79.8 ± 6.5	NS
BMI , kg/m^2	23.5 ± 1.4	23.9 ± 1.0	NS
BSA, m^2	2.0 ± 0.1	2.0 ±0.1	NS
Systolic BP, mmHg	120 ± 11	124 ± 12	NS
Diastolic BP, mmHg	70 ± 7	68 ± 7	NS
HR, beats/min	58 ± 9	49 ± 8	<0.001
LA $volume_I$, ml/m^2	21.1 ± 3.1	57.5 ± 5.7	<0.001
SV, ml	68.5 ± 10.5	95.2 ± 21.0	<0.001
E, cm/s	76.0 ± 13.3	85.1 ± 18.7	<0.01
A, cm/s	41.1 ± 7.9	41.9 ± 10.8	NS
E`, cm/s	13.0 ± 2.1	13.3 ± 2.7	NS
A`, cm/s	7.2 ± 1.5	8.0 ± 1.9	<0.05
E/E`	5.9 ± 1.2	6.5 ± 1.4	<0.05
LAstrain Reservoir, %	38.1 ± 9.4	33.8 ± 10.2	NS
LA strain Conduit, %	10.3 ± 3.9	9.4 ± 3.5	NS
LAstrain Pump, %	-3.3 ± 2.5	-3.0 ± 1.8	NS

Data expressed as mean ± standard deviation. BMI = body mass index; BSA = body surface area; BP = blood pressure (systolic- and diastolic); HR = resting heart rate; LA volume = left atrium end systolic volume; SV = left ventricular stroke volume; E = early transmitral peak velocity by pulsed Doppler: A = late / atrial transmitral peak velocity; E` and A` = Peak early and late (atrial) diastolic mitral annular velocity by tissue Doppler imaging, respectively; E/E` = Ratio reflecting LV filling pressure; LA strain = 2D strain was calculated as average of six segments to evaluate LA reservoir, conduit and pump function.