ORIGINAL ARTICLE

SPORT CARDIOLOGY

Volumetric and functional assessment of the left atrium in young competitive athletes without left ventricular hypertrophy: the MAGYAR-Sport Study

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ABSTRACT

BACKGROUND: Left atrial (LA) remodeling may be regarded as a physiologic adaptation to exercise conditioning. Three-dimensional speckle tracking echocardiography (3DSTE) is a new promising tool for volumetric and functional characterization of the LA. The present study was undertaken to assess adaptive changes in LA volumes and functional properties respecting cardiac cycle in young competitive athletes without left ventricular hypertrophy (LVH) by detailed 3DSTE assessment.
METHODS: The study group consisted of 20 young elite basketball and handball players (mean age: 28.1±10.1 years, 8 men) without LVH, their results were compared to 23 age- and gender-matched non-sportive healthy controls (mean age: 31.7±8.5 years, 11 men. All subjects had undergone standard transthoracic two-dimensional Doppler echocardiographic study with 3DSTE.
RESULTS: Increased systolic maximum (66.5±13.6 mL *vs.* 38.5±8.6 mL, P<0.0001) and diastolic minimum (36.7±8.1 mL *vs.* 17.5±5.8 mL, P<0.0001) and preatrial contraction (46.2±10.1 mL *vs.* 26.2±7.8 mL, P<0.0001) LA volumes could be demonstrated in athletes. Total (29.7±9.0 mL *vs.* 20.7±5.0 mL, P=0.0002) and passive LA stroke volumes (19.8±8.7 mL *vs.* 12.4±4.6 mL, P=0.0009) were increased, while total (44.2±9.1 mL *vs.* 54.2±9.4 mL, P=0.001) and active LA emptying fractions (20.6±11.8% *vs.* 31.9±8.7%, P=0.0008) proved to be decreased in athletes as compared to controls. Active LA stroke volume (9.9±5.8 mL *vs.* 8.3±3.3 mL, P=0.29) and passive LA emptying fraction (29.1±10.6 mL *vs.* 32.6±11.2 mL, P=0.31) did not differ between the groups. Only circumferential global (21.1±7.7% *vs.* 27.6±9.9%, P=0.02) and mean segmental (26.1±7.1% *vs.* 35.7±12.0%, P=0.003) peak LA strains proved to be significantly reduced in athletes as compared to controls.
CONCLUSIONS: 3DSTE-derived increased cyclic LA volumes and specific alterations in LA functional properties could be demonstrated in young competing athletes which is most likely a physiologic consequence of a global cardiac adaptation to intensive and chronic training.

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Key words: Heart atria - Heart function tests - Imaging, three-dimensional - Echocardiography - Sports.

Intensive athletic conditioning is associated with physiological adoptation of the heart of an athlete.[1] Changes in left ventricular (LV) size and mass are well-known recognized features in highly trained athletes.[2] Moreover, left atrial (LA) remodeling may be regarded as a physiologic adaptation to exercise conditioning, as

well.[3-5] Three-dimensional (3D) speckle-tracking echocardiography (3DSTE) represents a promising, non-invasive, simple and reproducible technique for detailed assessment of LA morphology and function including volumetric and strain assessments at the same time from the same acquired 3D dataset.[6] Due to well-known interaction between intensive training-induced LV hypertrophy (LVH) and LA function,[1] the present study was undertaken to assess adaptive changes in LA volumes and functional properties in elite competitive athletes without LVH by detailed 3DSTE assessment and to compare their results to age- and gender-matched non-athletic healthy volunteers.

Materials and methods

Patients population

The study group consisted of 20 young elite basketball and handball players (mean age: 28.1±10.1 years, 8 men) without LVH. Training volume and intensity were similar to that one previously described.[7] Echocardiographic examination was performed at the beginning of the low-training period in most of cases (16 subjects). During the low-training period, athletes trained for about 18 h/week including sprinting conditioning and high-volume/low-intensity running. Training also consisted of 2 resistance-training sessions/week at high-moderate workload. During the peak training period, athletes trained for 12 h/week and had one or two matches a week. Low-volume/high-intensity running, sprinting and tactical-technical drilling were also performed at this period. Athletes trained 1-2 resistance-training sessions per week at moderate workload as well. The mean previous years of training proved to be 16±8 years. Athletes with LVH were excluded from the present study. LVH was considered if LV mass index >134 g/m^2 in men and 110 g/m^2 in women.[8] Their results were compared to 23 age- and gender-matched non-athletic healthy controls (mean age: 31.7±8.5 years, 11 men). All athletes and matched healthy controls were examined by two-dimensional Doppler echocardiography and 3DSTE. Regarding to the task force classification of the American College of Cardiology considering dynamic and static component of their training, our athletes proved to be high dynamic/moderate static (Group CII) (>70% VO_{2max}, 20-50% voluntary contraction).[9]

A special study has been organized at our department to examine diagnostic and prognostic significance of 3DSTE-derived variables in elite athletes named MAGYAR-Sport Study (Motion Analysis of the heart and Great vessels bY three-dimensionAl speckle-tRacking echocardiography in Sportmen, magyar means Hungarian in Hungarian language). All subjects provided written informed consent to participate, and ethics approval was granted by the local institutional human research committee approved the study which complied with the 1975 Declaration of Helsinki.

Two-dimensional, Doppler and tissue Doppler echocardiography

All echocardiographic studies including M-mode, 2-dimensional, Doppler and tissue Doppler studies were performed with a commercially available echocardiography equipment (Toshiba Artida™; Toshiba Medical Systems, Tokyo, Japan). A PST-30SBP phased-array transducer was used for routine measuments. LV dimensions were calculated from the standard M-mode images obtained at the parasternal long-axis views.[10] Visual grading of valvular regurgitations were confirmed by colour Doppler echocardiography. Early diastolic (E) and atrial (A) wave velocities and their ratio (E/A) were measured by pulsed-wave Doppler-derived transmitral velocity assessments.[10]

3DSTE-derived data acquisition and quantitative analysis

A special matrix phased-array transducer (2,5 MHz PST-25SX; Toshiba Medical Systems, Tokyo, Japan) was used for the acquisition of 3D echocardiographic datasets from the apical view.[6] Six R-wave-triggered subvolumes acquired for 6 consecutive cardiac cycles during one breath-hold were combined to form the pyramid-shape 3D full volume including LA. The 3D Wall Motion Tracking software version 2.7 (Toshiba Medical Systems, Tokyo, Japan) was used for quantifications. Firstly, apical two- (AP2CH) and four-chamber (AP4CH) views and three short-axis views at different levels of the LA at end-diastole were automatically selected from the full volume 3D dataset by the software. After finding optimal nonforeshortened views, the reader set markers on orthogonal AP2CH and AP4CH views.
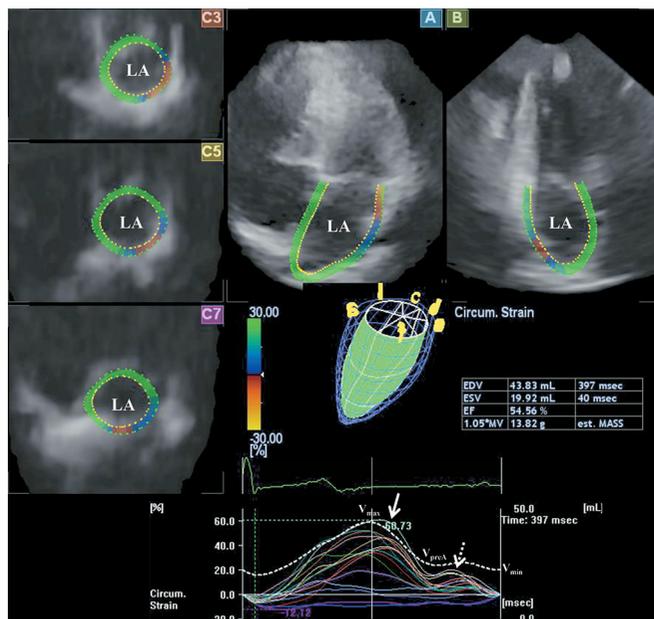
Figure 1.—Three-dimensional (3D) speckle-tracking echocardiographic assessment of the left atrium (LA) in an athlete is displayed. Apical four-chamber view (A), apical two-chamber view (B), short-axis view at basal (C3), midatrial (C5) and superior left atrial level (C7) are presented together with a 3D model of the LA and calculated volumetric data. Time — global LA volume (dashed line) — and time — LA circumferential segmental strain curves (continuous lines) — could be demonstrated. Maximum (V_{max}), minimum (V_{min}) LA volumes and LA volume before atrial contraction (V_{preA}) could be measured together with peak strain (white arrow) and strain at atrial contraction (dashed arrow).
EDV: end-diastolic volume; ESV: end-systolic volume; EF: ejection fraction; LA: left atrium.

Firstly, the edge of septum-mitral ring was traced, then markers were set in a counterclockwise rotation around the LA to the edge of the lateral wall-mitral ring. LA appendage and pulmonary veins were excluded from the LA cavity during evaluations. Finally, the 3D endocardial surface of the LA was automatically reconstructed and tracked in 3D space throughout the entire cardiac cycle (Figure 1).

3DSTE-derived LA volumetric assessements

LA volumes were measured echocardiographically at the time of mitral valve opening (maximum LA volume, V_{max}), at the onset of atrial systole (p wave on electrocardiography = V_{preA}), and at mitral valve closure (minimum LA volume, V_{min}). The following LA functional parameters were calculated:[11]

— systolic reservoir function:
  − LA total stroke (emptying) volume (TASV)=(V_{max}-V_{min});
  − LA total emptying fraction (TAEF)=TASV/V_{max};
— early diastolic conduit function:
  − LA passive stroke (emptying) volume (PASV)=V_{max}-V_{preA};
  − LA passive emptying fraction (PAEF) = PASV/V_{max};
— late diastolic booster pump function (active contraction phase):
  − LA active stroke (emptying) volume (AASV)=V_{preA}-V_{min};
  − LA active emptying fraction (AAEF)=AASV/V_{preA}.

All measurements were averaged over 3 cardiac cycles.

3DSTE-derived LA strain measurements

3DSTE allows calculation of several unidirectional (radial [RS], longitudinal [LS] and circumferential [CS] strains) and complex (area [AS] and 3D [3DS] strains) strains the same acquired 3D dataset.[6] Peak strains (featuring LA reservoir phase) and strains at atrial contraction (featuring LA contraction phase) were calculated in each case. The software enables measurement of global (including all the data related to the heart chamber), segmental (using a 16-segment model elaborated for the LV) and mean segmental (average of calculated segmental) strains. LA regional strains (basal, midatrial and superior) were calculated from segmental strain values. In a recent study good inter- and intraboserver variability could be demonstrated in the assessment of these 3DSTE-derived uni- and multidirectional LA strains.[12]

Stastical analysis

Continuous data are expressed as mean±standard deviation, while categorical data are summarized as a count and percentage. Categorical variables were examined by Fisher's Exact Test and χ^2 test, while continuous ones were assessed with the unpaired Student t-test. Statistical significance was defined with a probability value <0.05. Data analyses were performed using Medcalc software (MedCalc, Mariakerke, Belgium).

Results

Clinical data

Neither competitive athletes, nor healthy controls had known disease or received medications. Routine two-dimensional Doppler echocardiography showed normal results without valvular alterations (Table I).

3DSTE-derived LA volumes and volume-based functional properties

Increased systolic V_{max} (66.5±13.6 mL *vs.* 38.5±8.6 mL, P<0.0001) and diastolic V_{min} (36.7±8.1 mL *vs.* 17.5±5.8 mL, P<0.0001) and V_{preA} (46.2±10.1 mL *vs.* 26.2±7.8 mL, P<0.0001) LA volumes could be demonstrated in athletes. TASV (29.7±9.0 mL *vs.* 20.7±5.0 mL, P=0.0002) and PASV (19.8±8.7 mL *vs.* 12.4±4.6

mL, P=0.0009) proved to be increased, while TAEF (44.2±9.1 mL *vs.* 54.2±9.4 mL, P=0.001) and AAEF (20.6±11.8 mL *vs.* 31.9±8.7 mL, P=0.0008) were decreased in athletes as compared to controls. AASV (9.9±5.8 mL *vs.* 8.3±3.3 mL, P=0.29) and PAEF (29.1±10.6 mL *vs.* 32.6±11.2 mL, P=0.31) did not differ between athletes and controls.

3DSTE-derived LA strain parameters

Only peak global (21.1±7.7% *vs.* 27.6±9.9%, P=0.02) and mean segmental (26.1±7.1% *vs.* 35.7±12.0%, P=0.003) peak LA-CS proved to be significantly reduced in athletes as compared to controls. All peak global, mean segmental and segmental LA strains and LA strains at atrial contraction are presented in Tables II-V.

Discussion

To the best of authors' knowledge this is the first time to demonstrate alterations in 3DSTE-derived LA

TABLE I.—*Two-dimensional echocardiographic data of athletes and controls.*

	Athletes (N.=20)	Controls (N.=18)	P value
Left atrium (mm)	37.8±4.0	33.9±4.1	0.006
LV end-diastolic diameter (mm)	49.2±3.5	48.4±6.3	0.60
LV end-diastolic volume (ml)	110.4±22.3	104.2±21.1	0.36
LV end-systolic diameter (mm)	29.3±2.5	29.9±3.9	0.58
LV end-systolic volume (mL)	32.8±6.9	34.4±10.3	0.54
Interventricular septum (mm)	7.8±1.3	8.8±2.2	0.07
LV posterior wall (mm)	8.0±1.2	8.9±2.4	0.13
LV ejection fraction (%)	71.4±5.3	67.1±6.9	0.03
E/A	1.88±031	1.30±0.17	0.05

TABLE II.—*Comparison of 3DSTE-derived peak global and mean segmental left atrial strain parameters between athletes and controls.*

	Athletes (N.=20)	Controls (N.=23)	P value
Peak global strains			
Radial strain (%)	-17.9±4.8	-19.8±10.9	0.48
Circumferential strain (%)	21.1±7.7	27.6±9.9	0.02
Longitudinal strain (%)	26.2±7.8	26.0±7.1	0.92
3D strain (%)	-10.0±4.1	-12.0±10.0	0.42
Area strain (%)	53.5±16.7	58.7±17.6	0.33
Peak mean segmental strains			
Radial strain (%)	-19.6±4.4	-21.8±8.4	0.29
Circumferential strain (%)	26.1±7.1	35.7±12.0	0.003
Longitudinal strain (%)	28.2±7.2	31.7±6.7	0.11
3D strain (%)	-13.9±3.7	-15.1±6.6	0.45
Area strain (%)	59.0±15.7	73.2±21.5	0.06

3DS: three-dimensional, 3DSTE: three-dimensional speckle-tracking echocardiography

TABLE III.—*Comparison of 3DSTE-derived peak regional left atrial strain parameters between athletes and controls.*

	Athletes (N.=20)	Controls (N.=18)	P value
Peak regional radial strains (RS)			
Basal RS (%)	-19.5±4.4	-23.1±13.0	0.24
Midatrial RS (%)	-17.6±4.9	-22.7±10.2	0.05
Apical RS (%)	-22.7±9.9	-21.2±13.1	0.67
Peak regional circumferential strains (CS)			
Basal CS (%)	30.4±8.4	39.7±10.7	0.003
Midatrial CS (%)	21.7±5.9	30.9±10.2	0.001
Apical CS (%)	26.2±21.5	29.8±19.4	0.57
Peak regional longitudinal strains (LS)			
Basal LS (%)	29.1±11.8	17.5±8.4	0.0006
Midatrial LS (%)	36.5±13.8	41.6±13.6	0.22
Apical LS (%)	14.6±7.1	23.1±9.6	0.002
Peak regional 3D strains (3DS)			
Basal 3DS (%)	-14.4±4.1	-17.6±12.0	0.26
Midatrial 3DS (%)	-11.6±4.1	-15.1±8.6	0.10
Apical 3DS (%)	-16.4±8.6	-15.4±9.9	0.73
Peak regional area strains (AS)			
Basal AS (%)	61.1±19.8	55.6±17.7	0.34
Midatrial AS (%)	60.8±22.7	80.3±23.2	0.008
Apical AS (%)	53.2±46.0	63.9±39.9	0.42

3D: three-dimensional; 3DS: three-dimensional strain; 3DSTE: three-dimensional speckle-tracking echocardiography; AS: area strain; CS: circumferential strain; LS: longitudinal strain; RS: radial strain.

TABLE IV.—*Comparison of 3DSTE-derived global and mean segmental left atrial strain at atrial contraction parameters between athletes and controls.*

	Athletes (N.=20)	Controls (N.=23)	P value
Global strains at atrial contraction			
Radial strain (%)	-5.7±4.0	-7.1±7.4	0.48
Circumferential strain (%)	11.3±7.0	11.2±10.2	0.95
Longitudinal strain (%)	6.4±4.7	9.9±8.5	0.11
3D strain (%)	-4.7±4.3	-5.2±5.1	0.71
Area strain (%)	18.0±12.3	18.1±15.3	0.98
Mean segmental strains at atrial contraction			
Radial strain (%)	-8.3±3.2	-8.3±4.9	0.98
Circumferential strain (%)	12.1±5.4	13.5±9.1	0.55
Longitudinal strain (%)	7.5±3.9	9.7±5.6	0.15
3D strain (%)	-6.8±2.2	-6.6±4.4	0.88
Area strain (%)	22.4±10.2	22.0±13.7	0.92

3DS: three-dimensional; 3DSTE: three-dimensional speckle-tracking echocardiography.

TABLE V.—*Comparison of 3DSTE-derived regional left atrial strain at atrial contraction parameters between athletes and controls.*

	Athletes (N.=20)	Controls (N.=23)	P value
Regional radial strains (RS) at atrial contraction			
Basal RS (%)	-7.0±2.0	-8.3±6.3	0.39
Midatrial RS (%)	-7.5±4.4	-8.3±5.4	0.62
Apical RS (%)	-11.4±5.6	-9.7±7.4	0.39
Regional circumferential strains (CS) at atrial contraction			
Basal CS (%)	12.6±5.6	16.2±9.0	0.14
Midatrial CS (%)	9.6±5.5	12.0±8.2	0.28
Apical CS (%)	15.1±14.7	10.0±13.2	0.24
Regional longitudinal strains (LS) at atrial contraction			
Basal LS (%)	7.8±3.5	6.3±5.1	0.28
Midatrial LS (%)	7.3±5.8	11.0±7.2	0.07
Apical LS (%)	7.5±6.5	11.1±9.8	0.18
Regional 3D strains (3DS) at atrial contraction			
Basal 3DS (%)	-6.7±2.5	-6.5±5.8	0.89
Midatrial 3DS (%)	-6.1±3.0	-5.5±4.2	0.63
Apical 3DS (%)	-8.0±5.4	-8.3±6.6	0.89
Regional area strains (AS) at atrial contraction			
Basal AS (%)	21.3±8.8	19.8±10.2	0.60
Midatrial AS (%)	17.4±12.1	25.3±13.3	0.05
Apical AS (%)	21.5±29.1	20.7±30.1	0.93

3D: three-dimensional; 3DS: three-dimensional strain; 3DSTE: three-dimensional speckle-tracking echocardiography; AS: area strain; CS: circumferential strain; LS: longitudinal strain; RS: radial strain.

volumetric and functional properties in young competitive athletes. All cyclic LA volumes and LA stroke volumes in LA reservoir and conduit phases proved to be increased in athletes. Moreover, reduced LA emptying fractions in LA reservoir and active contraction phases could be demonstrated. In respect with strains, only peak global and mean segmental CSs, features of LA reservoir function, were found to be decreased in athletes, all other strains did not differ between the groups examined. However, some segmental strains showed significant differences between athletes and controls which could highlight our attentation on the importance of 3DSTE-derived detailed analysis. From LA strains at active contraction phase, only midatrial segmental LA-AS strain at atrial contraction differed significantly.

The number of studies in which LA was assessed in athletes is increasing due to the more ready new echocardiographic methodologies and the increased scientific interest on this topic trying to understand its physiologic/pathophysiologic background.[13] In a very early study it was indicated that former endurance athletes have an enlarged LA but a normal-sized LV.[14] In a large population of highly trained athletes, a mild enlargement of LA volume index was relatively common and may be regarded as a physiologic adaptation to exercise conditioning.[15] Enlarged LA dimension ≥40 mm was found in 20% of highly trained athletes in another study, with the upper limits of 45 mm in women and 50 mm in men distinguishing physiologic cardiac remodeling

("athlete's heart") from pathologic cardiac conditions.[3] Regarding to D'Ascenzi et al. top-level athletes exhibit a dynamic morphological and functional LA remodeling, induced by training, with an increase in reservoir and conduit stroke volumes, but stable active stroke volume.[16] Kasikcioglu et al. found that athletes (male endurance runners) had higher LA volume and volume index (except the conduit volume), PAEF and TAEF compared to control subjects.[17] In contrast, increased LV-SV was associated with an increase in both PASV and AASV, and with unchanged LA emptying fractions in another study. These changes were thought to be normal physiologic adaptations to prolonged training.[1] Our results are only partially in agreement with these findings demonstrating alterations in TASV, PASV, TAEF and AAEF with unchanged AASV and PAEF.

STE was used to measure peak atrial longitudinal strain (PALS) and peak atrial contraction strain (PACS) in studies by D'Ascenzi et al.[4, 18] They found reduced

PALS and PACS in a mixed population of competitive athletes (volleyball, basketball, soccer),[18] while when only elite soccer players were examined, significant reduction could be demonstrated only in PACS as compared to controls.[4] In agreement with these findings, Gabrielli *et al.* have demonstrated decreased LA strain during active atrial contraction in professional handball players.[19] However, in contrast, no differences in intrinsic atrial strain in any phases of LA function could be demonstrated between the following groups: high dynamic/high static and low-dynamic/high static elite male athletes and sedentary controls in another recent myocardial strain tracking imaging study.[20] Our results confirmed reduction in strains featuring both LA reservoir and active contraction phases, but alterations in features of LA reservoir function proved to be more pronounced.

The peculiar pattern of LA dysfunction could be demonstrated in elite athletes in several studies. Regarding to the literature LA remodeling deemed to be a physiologic consequence of a global cardiac adaptation to the increased preload associated with intensive and chronic training.[21] The role of LV (dys)function could also not be excluded. D'Ascenzi *et al.* concluded in a recent study, that a shift in the pattern of ventricular filling period toward early diastole could be demonstrated in elite soccer players leading to a more rapid passive atrial emptying.[4] Moreover, the associated lower global LA active contraction may presumably be related to an increased myocardial distensibility at the end of diastole.[4, 22] Differences in volumetric and functional LA features between sports could be explained by different training volume, intensity, duration and type, but further studies are warranted to examine other sport-specific alterations.

3DSTE is a new promising tool based on block-matching algorithm of the myocardial speckles,[4] which just has been validated.[23, 24] One of the major advantage of 3DSTE, that all LA volumetric, volume-based and strain parameters could be calculated from the same 3D acquired dataset making the whole procedure very easy to perform.[11] Recently, increased LA volumes and specific alterations in functional properties could be demonstrated by 3DSTE in atrial fibrillation,[25, 26] hypertrophic [11] and noncompaction cardiomyopathies,[27] type 1 diabetes mellitus [12] and hypereosinophilic syndrome.[28] Our results confirm feasibility of this new non-invasive

3DSTE methodology in competitive athletes and its usefulness to assess LA volumes, function and deformation and to understand the phsysiology of 'athlete's heart.

Limitations of the study

The present study should be considered as a preliminary observational study, not a validation study.[23, 24] 3DSTE is a relatively new methodology with its specific limitations (for instance low spatial and temporal resolution, etc.), which facts should be considered when interpreting results. Another limitation is the relatively small population of athletes and matched controls. Finally, functional characterization of ventricles and right atrium was not aimed in this study.

Conclusions

3DSTE-derived increased cyclic LA volumes and specific alterations in LA functional properties could be demonstrated in young competing athletes which is most likely a physiologic consequence of a global cardiac adaptation to intensive and chronic training.

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