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ORIGINAL PAPER

Left ventricular volumes and mass in marathon runners and their association with cardiovascular risk factors

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Abstract *Background* To assess left ventricular volumes and mass by cardiac magnetic resonance imaging in relation to conventional cardiovascular risk factors and coronary atherosclerotic plaque burden in master marathon runners aged ≥50 years. *Methods* Cardiac MRI was performed in 105 clinically healthy male marathon runners (mean age 57.3 ± 5.7 years, range 50–71 years) on a 1.5 T MR system (Avanto, Siemens, Germany). Cine steady state free precession images in standard long and short axes views were acquired to assess left ventricular volumes and mass. Cardiovascular risk factors (blood pressure, HDL/LDL cholesterol, smoking, body mass index) were assessed and coronary artery calcification (CAC) was quantified by electron beam computed tomography. *Results* Left ventricular muscle mass (mean LVMM = 140 ± 27 g; 73 ± 13 g/m^2) increased with increasing left ventricular end-diastolic volume (mean LVEDV = 137 ± 32 ml; 72 ± 15ml/m^2) ($r = 0.41$, $P < 0.0001$) and with systolic ($r = 0.33$, $P = 0.005$) and diastolic ($r = 0.28$, $P = 0.005$) blood pressures. Left ventricular

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EDV increased up to the age of 55 years, but decreased thereafter. Runners with LVMM ≥150 g had significantly higher CAC scores than runners with LVMM <150 g (median CAC score 110 vs. 25, $P = 0.04$). *Conclusions* Increases in LVMM and LVEDV may not only represent a response to exercise but are dependent on age and blood pressure, also. In addition, a left ventricular hypertrophy without an increase in volume may be an indicator for early subclinical cardiac alterations in response to risk factor exposure.

Keywords Marathon · Left ventricular volumes · Left ventricular mass · Athlete's heart · MRI

Introduction

Over the past decades, the number of marathon race participants has constantly risen [1]. During this time, the median age of marathon finishers increased from 34 years in 1980 (26% males ≥40 years) to 40 years in 2005 (44% males ≥40 years) [1]. With increasing age, an increased prevalence of subclinical cardiac disease is expected, which may have implications for pre-participation cardiovascular risk stratification. Identification of marathon runners at risk is difficult, and the need for cardiovascular screening remains a matter of debate [2, 3]. The risk of cardiac events associated with marathon running has been suggested to be too low to recommend routine screening for cardiovascular disease [4]. In master athletes, pre-participation medical evaluations are nonetheless advised [3, 5].

In young athletes, criteria have been developed to distinguish a physiological adaptation of cardiac morphology and function to exercise ("athlete's heart") from early cardiovascular disease [2]. In older adults with their higher prevalence of cardiovascular risk factors and of subclinical cardiac disease, the differentiation of athlete's heart from early cardiac disease may be more challenging. However, cardiac morphology and function in a large cohort of marathon runners aged ≥50 years has not been described before.

During the past decade, cardiac MRI has become a widely used tool for the assessment of cardiac disorders in clinical routine in patients [6] and also in athletes [7]. Cardiac MRI has become the gold standard for quantification of cardiac volumes and mass [8]. In the present cross-sectional observational study, we used cardiac MRI to assess left ventricular volumes and mass in clinically healthy marathon runners aged ≥50 years and studied their association with age and cardiovascular risk factors.

Material and methods

The Marathon Study design has been reported previously [9, 10]. The study was approved by the local ethics committee and by the National Institute of Radiation Protection (Bundesamt für Strahlenschutz, Munich, Germany). All participants gave written informed consent prior to participation in the study.

Participants

One hundred-eight master marathon runners (mean age 57.2 ± 5.7 years, range 50–71 years) were enrolled, with the following inclusion criteria: males aged ≥50 years, at least five completed full-distance marathon races (42.195 km) during the past 3 years. Exclusion criteria comprised history of established heart disease, diabetes mellitus, angina pectoris, and renal failure, musculo-skeletal disease at inclusion preventing future regular marathon running, psychiatric disease, and unwillingness to give informed consent [9]. Participants were recruited in three ways: (1) an advertisement in a German marathon journal ("Runners World"), (2) a press conference at inauguration of the study, and (3) inclusion of colleagues and friends of participants, if inclusion criteria were met [9]. Two males were excluded from the study because of prior unreported myocardial infarction in one and severe renal failure due to untreated prostate disease in the other. Additional three athletes did not undergo MRI scanning because of a cochlear implant ($n = 1$), metal splinter in a rib ($n = 1$), and claustrophobia ($n = 1$), leaving 105 runners that constitute the cohort for the present report. Marathon runners had completed 20 marathons (median value, Q1–Q3: 14–41.5), had started marathon running 9 years ago (Q1–Q3: 7–16), and trained 55 km (approx. 35 miles; Q1–Q3: 45–65) on 5 days per week throughout the year.

Cardiovascular risk factors

Conventional cardiovascular risk factors (CVRF) that are a part of the Framingham risk score were assessed

[11]. Blood pressure was measured with an automatic oscillometric blood pressure device (Omron 705, OMRON, Mannheim, Germany) and the mean value of the second and third of three measurements taken at least 3 min apart was recorded. Standard enzymatic methods were used to measure HDL and LDL cholesterol [mg/dl]. Current smoking was defined as a history of smoking during the past years. Body mass index (BMI [kg/m^2]) was calculated from standardized measurements of height and weight.

Electron beam computed tomography (EBCT)

EBCT scans were performed using a GE-Imatron C-150 scanner (General Electric Medical Systems, South San Francisco, California, USA). After determination of the heart position, 30–40 axial slices were acquired during inspiratory breath hold from the base to the apex of the heart. Imaging was done using the high resolution single slice mode with 100 ms exposure time, 3 mm slice thickness, and 3 mm table feed between consecutive slices. Image acquisition was triggered to the patient's ECG at 80% of the cardiac cycle. Coronary artery calcifications (CAC) were defined as at least four contiguous pixels with a CT density $\geq$130 Hounsfield units and were quantified using the Agatston scoring method [12].

Cardiac magnetic resonance imaging (MRI)

All examinations were performed on a 1.5 Tesla MR scanner equipped with high performance gradients (Magnetom Avanto, Siemens Medical Solutions, Erlangen, Germany). For image acquisition, two elements of the spine coil and a phased-array torso coil with six active coil elements were used. Fully coherent steady state free precession cine images (cineSSFP: TR 3 ms, TE 1.5 ms, FA 60°, slice thickness 8 mm, 1.6 mm inter-slice gap, in-plane resolution 1.75 $\times$ 1.75 mm^2, temporal resolution 45 ms, bandwidth 900 Hz/pixel) were acquired in standard long and short axes views during breath holding in end-expiration.

MRI analysis

Measurements of left ventricular volumes and muscle mass were performed based on the short axes scans covering the entire left ventricle using the ARGUS software (Siemens Medical Solutions, Erlangen, Germany) with semiautomatic contour detection. The most basal slice was defined as that slice at which at least 50% of the circumference of the left ventricular wall was seen at end-diastole and end-systole, respectively. The apical slice was defined as the last slice showing an intra-cavity blood pool. Manual correction of the software-proposed epicardial and endocardial contours at end-diastole (image with the largest left ventricular cavity) and end-systole (image with the smallest left ventricular cavity) was performed in all cases. Papillary muscles were included to the ventricular cavity and were not considered for calculation of mass.

Statistical analysis

Data are presented as mean $\pm$ SD, median [Q1 (25th percentile), Q3 (75th percentile)] or percentage. Associations were analyzed using linear regression, including analysis of residues and curve-linearity, or using Spearman correlation coefficients. A P-value less than 0.05 was regarded to indicate statistical significance. Statistical analysis was performed using SAS v.9.1 (SAS Institute, Cary, NC, USA) and SPSS 12.0 (SPSS Inc., Chicago, IL, USA).

Results

Cardiovascular risk factors

Cardiovascular risk factors in this cohort were representative of the entire cohort (Table 1). Thirteen percent of participants showed arterial hypertension (defined as systolic blood pressure $\geq$140 mmHg), 9.5% hypercholesterolemia (defined as LDL-Cholesterol $\geq$160 mg/dl), and 33.3% a BMI $\geq$25 kg/m^2. There were 5 current smokers and 55 were previous smokers. In this cohort, blood pressure did not increase with age ($r = 0.032$, $P = 0.73$).

Left ventricular volumes and mass and their association with cardiovascular risk factors

Left ventricular volumes and mass are detailed in Table 2. Left ventricular muscle mass increased

Table 1 Detailed characteristics of the 105 study participants with MRI scans in comparison to the entire group of 108 runners

	All ($n = 108$)	$n = 105$ with MRI
Age (years)	57.2 ± 5.7	57.3 ± 5.7
Body mass index (kg/m^2)	24.0 ± 2.3	23.9 ± 2.3
Arterial hypertension (%)	12.0	12.4
Systolic blood pressure (mmHg)	121 ± 14	121 ± 14
Diastolic blood pressure (mmHg)	75 ± 9	75 ± 9
LDL (mg/dl)	121 ± 29	121 ± 29
HDL (mg/dl)	74 ± 17	74 ± 17
Diabetes	0	0
Smoking (active/former) (%)	4.6/51.9	4.8/52.4
Ten year framingham risk (%)	7 [4–9]	7 [4–9]
CAC score (median [Q1–Q3])	36 [0–217]	34 [0–222]

Table 2 Left ventricular parameters of 105 male master marathon runners assessed by cardiac MRI: left ventricular end-diastolic volume (LVEDV), end-systolic volume (LVESV), left ventricular ejection fraction (LVEF) and left ventricular muscle mass (LVMM)

LV-parameters	Absolute	Normalized to body surface area
(a) Means ± SD for absolute and normalized LV-parameters		
LVEDV	137 ± 32 ml	72 ± 15 ml/m^2
LVESV	52 ± 17 ml	27±9 ml/m^2
LVEF	62 ± 8%	na
LVMM	140 ± 27 g	73 ± 13 g/m^2

LV-Parameters	5th		25th		Median		75th		95th	
	Abs.	Norm.	Abs.	Norm.	Abs.	Norm.	Abs.	Norm.	Abs.	Norm.
(b) Percentile distribution of absolute and normalized LV-parameters										
LVEDV (ml/ml/m^2)	82	47	117	61	136	71	160	82	187	95
LVESV (ml/ml/m^2)	29	16	39	20	51	27	63	33	84	40
LVEF (%)	49	na	56	na	63	na	68	na	73	na
LVMM (g/g/m^2)	107	57	120	64	137	71	154	81	196	100

significantly with left ventricular end-diastolic volumes (LVEDV) (Fig. 1) and with systolic and diastolic blood pressure (Fig. 2a, b), whereas no association between left ventricular muscle mass and age was observed ($r = 0.011$, $P = 0.91$). LVEDV decreased significantly with age in a linear model (Fig. 3). We also computed a curve-linear model, which showed a better fit to the data than the linear model. In the curve-linear model, LVEDV initially increased until the average age of 55, followed by a decrease in beyond this age (Fig. 3). We observed no association of LVEDV with systolic or diastolic blood pressures ($r = 0.045$, $P = 0.65$ and $r = 0.000$, $P = 0.95$, respectively).

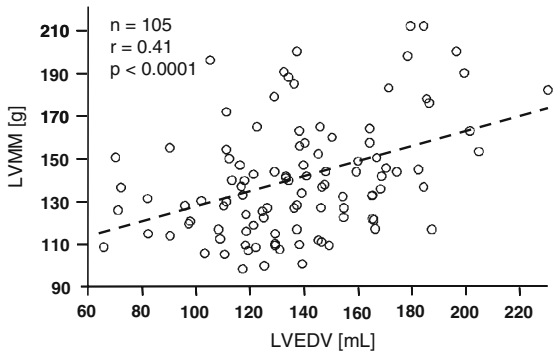

Fig. 1 Scatter plot and correlation of left ventricular end-diastolic volumes (LVEDV) and left ventricular muscle mass (LVMM) in 105 master marathon runners

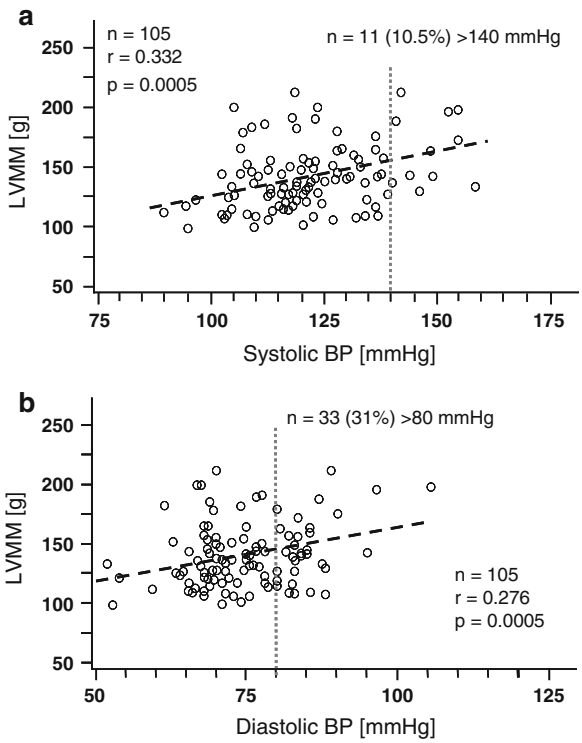

Fig. 2 Scatter plot and correlation of left ventricular muscle mass (LVMM) and systolic (**a**) and diastolic (**b**) blood pressure (BP) in 105 master marathon runners

	Linear Model	Curve-Linear model	
	(age vs. EDV)	(age*age vs. EDV)	(age vs. EDV)
ß	-1.21	-0.2	21.9
SE	0.54	0.09	11.0
r	0.216	0.295	
p-value	0.027	0.038	0.049

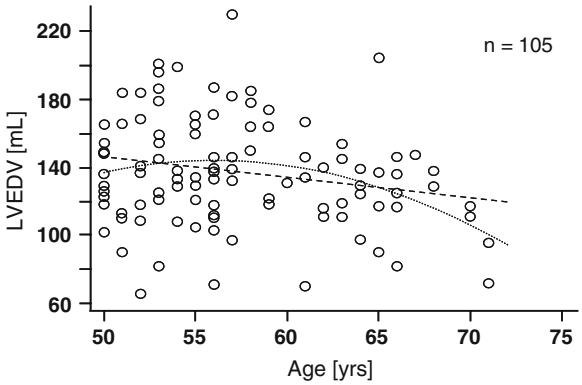

Fig. 3 Scatter plot and curve-linear correlation of left ventricular end-diastolic volume (LVEDV) and age

Physical activity and its association with left ventricular volumes and mass

We found no association between any of the indexes of physical activity with left ventricular volumes or mass (Table 3). Regression analyses revealed no hints for curve-linearity in any of these relations, and the respective 95% confidence intervals of estimated slopes all included zero. Training mileage did not change with age ($r = 0.000$, $P = 0.92$) and we found no association of training mileage with systolic or diastolic blood pressure ($r = 0.105$, $P = 0.29$, and $r = 0.077$, $P = 0.44$, respectively). In one marathon runner, finishing times were available from 1,437 marathon competitions. As expected, competition times show a wide range throughout the year. However, best finishing times increased approximately until the age of 55, but gradually decreased with further increase in age (Fig. 4).

Coronary artery calcium and its association with left ventricular volumes and mass

A CAC <100, ≥100 to <400, and ≥400 was observed in 62.9, 23.8, and 13.3% of marathon runners, respectively. The CAC score was not related to left ventricular end-diastolic volume ($r = 0.118$, $P = 0.23$), left ventricular end-systolic volume ($r = 0.044$, $P = 0.66$), or mass ($r = 0.053$, $P = 0.59$). No significant differences in LV volumes and mass were observed between marathon runners with (CAC > 0) and without (CAC = 0) coronary artery calcifications ($P > 0.05$). However, the CAC burden in marathon runners with a left ventricular muscle mass ≥150 g was higher ($n = 32$, 110 [1–319]) than in runners with a muscle mass <150 g ($n = 73$, 25 [0–144], $P = 0.04$).

Discussion

The present study was designed to provide reference values and determinants of left ventricular volumes and mass in presumably healthy male marathon runners aged ≥50 years. Our findings may help to distinguish physiological adaptation to exercise (athletes' hearts) from alterations in response to cardiovascular risk factors and ageing. Left ventricular end-diastolic volumes decreased with increasing

Table 3 Association of indexes of physical activity with left ventricular volumes and mass

	EF	LVESV	LVEDV	LVMM
Weekly METs	$r = 0.126$	$r = 0.167$	$r = 0.118$	$r = 0.173$
	$P = 0.19$	$P = 0.09$	$P = 0.23$	$P = 0.076$
Years of running	$r = 0.024$	$r = 0.032$	$r = 0.1$	$r = 0.024$
	$P = 0.80$	$P = 0.72$	$P = 0.33$	$P = 0.81$
Number of marathon races completed[a]	$r = 0.022$	$r = 0.057$	$r = 0.077$	$r = 0.000$
	$P = 0.83$	$P = 0.56$	$P = 0.42$	$P = 0.99$
Training mileage	$r = 0.032$	$r = 0.028$	$r = 0.084$	$r = 0.142$
	$P = 0.71$	$P = 0.77$	$P = 0.41$	$P = 0.15$

[a] One runner had completed 1,437 marathon races. Associations with CMR-parameters did not change, however, when this runner was excluded from analysis

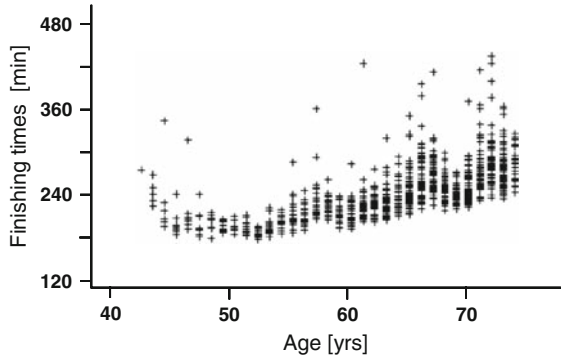

Fig. 4 Finishing times of a 71 year old marathon runner, who participated in more than 1,400 marathon races since 1974

age while LV muscle mass remained unaffected. LV muscle mass increased with systolic and diastolic blood pressure but not with age, indicating that blood pressure contributes to changes in left ventricular mass in well-trained marathon runners, even when blood pressures are normal or near normal. These findings have implications for cardiovascular risk assessment in recreational athletes.

Left ventricular end-diastolic volumes in marathon runners

The cardiac response of individual athletes to systematic training is not uniform [13]. Endurance training (e.g. marathon running or long distance swimming) leads to a ventricular enlargement without wall thickening [14, 15], while strength training (e.g. weight-lifting) generally results in left ventricular hypertrophy without an increase in ventricular size. In endurance-trained athletes left ventricular enlargement within limits represents a physiological adaptation, which in combination with greater venous return and expansion in plasma volume contributes to an increase in LV cardiac output during long lasting high-intensity exercise [15].

To our surprise, mean end-diastolic volumes (LVEDV = 137 ml) and left ventricular ejection fractions (LVEF = 62%) in our marathon runners were slightly lower compared to previously published MRI data, i.e. mean LVEDV = 152 ml and mean LVEF = 67% in non-athletic males aged 50–69 years [16], and mean LVEDV = 149 ml and mean EF = 71% in males ≥35 years [17]. These findings may be explained in part by slight methodological differences in volumetric calculations, and by the low sample sizes in the normal cohorts [$n = 10(15)$ and $n = 32(16)$, respectively].

The correlation between LV end-diastolic volumes and age suggests that end-diastolic volumes decrease by approximately 0.5% per year, assuming a linear trend. Since training mileage did not change with age in our cohort, the decrease of LV end-diastolic volumes with age could not be explained by detraining [18], but seems attributable to a physiological aging effect [16, 19], which has been suggested to be caused by an age-dependent decrease in the number of cardiomyocytes [20]. The assumption of an decrease of LV end-diastolic volumes with age due to physiological aging is supported by the observations of Scharhag et al. [7], who reported considerable higher LV end-diastolic volumes in young endurance athletes (27 ± 4 years; LVEDV = 167 ± 28 ml) compared to our data.

The scatter plot of age and LV end-diastolic volumes also allows for the assumption of a curvelinear relation between age and LV end-diastolic volumes, which suggest, that LV end-diastolic volumes may increase with endurance training until the age of approximately 55–60 years, whereas thereafter the physiological aging effect of the heart results in

an approximately 2% annual decrease in LV end-diastolic volumes. Interestingly, in male world-class athletes, marathon finishing times are closely related to LV end-diastolic volumes, with faster running times in athletes with larger end-diastolic volumes [15]. This finding together with our cross-sectional observations may have a role in the 2% decline in annual marathon finishing times beyond age 55 in one participant, who meticulously recorded all marathon finishing times (Fig. 4). In comparison, VO_2max decreases by approximately 0.7–1.6% per year with increasing age [21–23]. Thus, in addition to maximal oxygen consumption, exercise economy and the lactate threshold, which are believed to be key determinants of endurance exercise performance in older athletes [24], a change in LV end-diastolic volumes may also contribute to this age-related decrease in functional capacity.

Left ventricular muscle mass in marathon runners

In accordance with previously published data [14, 15], left-ventricular muscle mass increased in parallel with left ventricular end-diastolic volumes. However, median left ventricular muscle mass (LVMM = 140 g) was slightly higher in our cohort compared to previously published MRI data for non-trained persons [17]. The increase in muscle mass could not only be explained by increases in LV end-diastolic volumes, as muscle mass was related to systolic and diastolic blood pressure, even though most runners had normal resting blood pressure values. The implication of this finding is that a high LV muscle mass represents in part a long-term response to blood pressure and should not be solely interpreted as a physiological adaptation to exercise. This is important because runners with an LV muscle mass $\geq$150 g had significant higher CAC scores than runners with LVMM <150 g. Arterial hypertension does not only cause increases in myocardial mass, but is also a key determinant in the development of coronary artery calcium (CAC) [25]. A CAC score >100, which is generally accepted as a useful threshold for elevated risk in middle aged asymptomatic men, was found in as many as 37% of runners. The implication of this finding is that a muscle mass $\geq$150 g may indicate early cardiac and coronary disease, which may warrant a more detailed cardiovascular risk assessment as the runners' risk may be higher than anticipated from conventional risk factor assessment. However, this finding should be interpreted with caution, given that the association between CAC and LVMM was statistically not significant across the entire range of CAC and LVMM values.

Interestingly, we observed a considerably lower median LV mass in our cohort of marathon runners aged $\geq$50 years compared to previously published MRI data of young endurance athletes [7]. Since we observed no association between left ventricular muscle mass and age in our study, this difference may be attributable to a different training status in the younger athletes.

Limitations

There are several limitations to our study. First, we can only provide cross-sectional but no longitudinal data. Especially the changes in LV volumes with increasing age should be assessed longitudinally. Second, it was not the purpose of this study to provide reference values for maximal oxygen consumption, exercise economy, lactate threshold and other measures of endurance exercise performance. However, given the potential implications of our study, the relationship of changes in LV volumes with changes in measures of exercise performance, remains to be studied. Furthermore, our data may not be representative for all marathon runners, especially women and non-caucasian athletes. Finally, our data may be subject to recruitment bias due to the nature of our recruitment strategy. However, we have excluded diabetics and runners with known cardiac disease including angina so that our cohort is likely representative of many runners aged >50.

Conclusion

In the present study, we provide reference values for left ventricular volumes and myocardial mass, which is important for clinical care of athletes and for research purposes. Our findings may contribute to distinguish physiological adaptation to exercise from early subclinical cardiac alterations in response to risk factor exposure including arterial hypertension and age. The decrease in left ventricular end diastolic volume with age may have a role in the competitiveness marathon runners. In recreational runners aged $\geq$50 years, a high myocardial mass may not

always indicate athletes' heart but rather a higher risk than that anticipated from conventional risk factor assessment.

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