Athlete's Heart

Right and Left Ventricular Mass and Function in Male Endurance Athletes and Untrained Individuals Determined by Magnetic Resonance Imaging

Jürgen Scharhag, MD,* Günther Schneider, MD,† Axel Urhausen, MD,* Veneta Rochette,*
Bernhard Kramann, MD,† Wilfried Kindermann, MD*

Saarbrücken/Homburg, Germany

OBJECTIVES Athlete's heart represents a structural and functional adaptation to regular endurance exercise.

BACKGROUND While left ventricular (LV) hypertrophy of the athlete's heart has been examined in many studies, the extent of right ventricular (RV) hypertrophy is still uncertain because of its complex shape and trabecular structure. To examine RV hypertrophy, we used magnetic resonance imaging (MRI) and hypothesized that athlete's heart is characterized by similar LV and RV hypertrophy.

METHODS The LV and RV mass, volume, and function in 21 male endurance athletes (A) (27 ± 4 years; 70 ± 8 kg; 178 ± 7 cm; maximal oxygen uptake [Vo_2max]: 68 ± 5 ml/min per kg) and 21 pair-matched untrained control subjects (C) (26 ± 3 years; 71 ± 9 kg; 178 ± 6 cm; Vo_2max: 42 ± 6 ml/min per kg) were analyzed by MRI (Magnetom Vision 1.5T, Siemens, Erlangen, Germany).

RESULTS Left ventricular masses (A: 200 ± 20 g; C: 148 ± 17 g) and RV masses (A: 77 ± 10 g; C: 56 ± 8 g) differed significantly between the groups ($p < 0.001$). The LV and RV end-diastolic volumes (EDV) (LV-EDV 167 ± 28 ml [A]; 125 ± 16 ml [C]; RV-EDV 160 ± 26 ml [A]; 128 ± 10 ml [C]), and stroke volumes (SV) (LV-SV: 99 ± 18 ml [A], 74 ± 11 ml [C]; RV-SV: 102 ± 18 ml [A], 79 ± 8 ml [C]) were significantly different between the athletes and control subjects ($p < 0.001$), whereas ejection fractions (EF) (LV-EF: $59 \pm 3\%$ [A]; $59 \pm 6\%$ [C]; RV-EF: $63 \pm 3\%$ [A], $62 \pm 3\%$ [C]) and LV-to-RV ratios were similar for both groups (LV-to-RV mass: 2.6 ± 0.2 [A], 2.6 ± 0.3 [C]; LV-to-RV EDV: 1.05 ± 0.14 [A], 0.99 ± 0.14 [C]; LV-to-RV SV: 0.98 ± 0.17 [A], 0.95 ± 0.17 [C]; LV-to-RV EF: 0.93 ± 0.07 [A], 0.96 ± 0.10 [C]).

CONCLUSIONS Regular and extensive endurance training results in similar changes in LV and RV mass, volume, and function in endurance athletes. This leads to the conclusion that the athlete's heart is a balanced enlarged heart. (J Am Coll Cardiol 2002;40:1856–63) © 2002 by the American College of Cardiology Foundation

Regular and extensive endurance training leads to a cardiac hypertrophy called athlete's heart. It is assumed that the dimensional changes affect all cardiac cavities to the same extent, resulting in a balanced cardiac hypertrophy (1–3).

On the electrocardiogram (ECG), signs of left ventricular (LV) or right ventricular (RV) hypertrophy, such as increased R waves in the left precordial leads or an incomplete right bundle branch block, can be found without evidence of an athlete's heart (1,2,4). In the past two decades, echocardiographic studies documented typical morphologic changes in the LV, such as the increase in the inner end-diastolic diameter (EDD), myocardial wall thickness, and myocardial mass which correspond to eccentric hypertrophy (3,5–8). In contrast, only limited data are available on the RV because of its complex shape and pronounced trabecular structure, which only allows an orientating echocardiographic analysis. Maron (6) reported in a meta-analysis of 28 echocardiographic studies of athletes' hearts that the RV inner end-diastolic diameter was determined in only 8 studies and that

it was 24% greater than that in normal individuals, whereas the training-induced difference in the size of the LV averaged only 10%.

By magnetic resonance imaging (MRI), it is now possible to perform reliable determinations of both LV and RV mass and volume (9–14). However, only a few MRI studies examined the RV (9–16). A systematic study on the extent of RV hypertrophy of the athlete's heart has not been performed so far. Therefore, it was the aim of this study to compare the extent of LV and RV hypertrophy in male endurance athletes with an athlete's heart. It was hypothesized that the ratios between LV and RV mass and volume of the athlete's heart and hearts of normal size would be similar. This would prove the assumption that the athlete's heart represents a balanced cardiac hypertrophy.

METHODS

Study population. Twenty-one healthy, well-trained male endurance athletes (10 tri-athletes, 6 cyclists, and 5 runners) with a training history of many years and a control group of 21 healthy, untrained males pair-matched for height (maximal tolerated deviation ±5 cm), weight (maximal tolerated deviation ±5 kg), and body surface area (BSA; maximal

Abbreviations and Acronyms

BSA = body surface area
EDD = end-diastolic diameter
EDV = end-diastolic volume
EF = ejection fraction
ESV = end-systolic volume
LV = left ventricle/ventricular
MRI = magnetic resonance imaging
RV = right ventricle/ventricular
SV = stroke volume
VO_2max = maximal oxygen uptake

tolerated deviation ±0.06 kg/m^2) volunteered for the study (Table 1). To meet the definition of athlete's heart, endurance athletes had to have a relative heart volume of at least 13 ml/kg, as determined by echocardiography (5) at the time of the examination. To exclude cardiovascular and other relevant diseases, each participant underwent a physical examination, including a determination of usual blood parameters, blood pressure at rest, ECG at rest, and color Doppler echocardiography. The study was approved by the institutional Review Committee, and all participants gave their written, informed consent.

Cardiopulmonary exercise test. Maximal oxygen uptake (VO_2max) was measured by indirect mixing-chamber spirometry (Cortex MetaMax I, Leipzig, Germany). An individually adjusted ramp protocol was chosen to exhaust subjects within 10 to 12 min. Tri-athletes, cyclists, and untrained control subjects were tested by cycle ergometry (Lode Excalibur, Groningen, Netherlands), and long-distance runners by treadmill ergometry (Woodway, Weil am Rhein, Germany).

Echocardiography. Echocardiography was performed on a GE System FiVe (GE, Vingmed Ultrasound, Horten, Norway) with a 2.5-MHz transducer, in accordance with the guidelines of the American Society of Echocardiography (17). M-mode echocardiography was used to determine the inner LV-EDD and RV-EDD, interventricular septal thickness (IVST), posterior wall thickness (PWT), and fractional shortening. Total LV-EDD (TEDD = LV-EDD + IVST + PWT) was determined on the mitral valve plane (TEDD$_M$) and the papillary muscle plane (TEDD$_P$). Total LV longitudinal diameter (TLD) was determined in the four-chamber-view. Parameters were measured three times, and mean values were used to calculate the LV total diastolic volume (TDV [ml]) and heart volume (HV [ml]) by the following formulae (5):

$$TDV = ([TEDD_M^2 \cdot 0.785] + [TEDD_P^2 \cdot 0.435]) \cdot TLD/2000$$

$$HV = (TDV \cdot 2.432) + 130$$

Magnetic resonance imaging. The MRI was performed on a 1.5-tesla magnet (Magnetom Vision, Siemens, Erlangen, Germany). Images were acquired with the subject in the supine position, by applying ECG-gated breath-hold sequences. To get optimal image quality of myocardial structures for evaluation of LV and RV mass, a T_1-weighted spin-echo sequence in the end-diastolic phase was used (repetition time/echo time [TR/TE] = 700/30 ms; flip angle 160°, field of view 300 to 400 mm; matrix 256 × 256). Ten to 14 oblique sagittal images perpendicular to the ventricular septum (short-axis view) (Fig. 1) with a slice thickness of 6 mm and an interslice gap of 4 mm were acquired. To evaluate functional parameters, cine images (TR/TE = 100/4.8 ms; flip angle 20°) with the same orientation as T_1-weighted imaging were used for quantification of end-diastolic and end-systolic volumes (EDV and ESV), covering the whole RR cycle in each slice with 14 to 22 images. For both sequences, the same slice positions were used. Quantitative analysis was performed off-line using dedicated software (ARGUS, Siemens). To evaluate LV and RV mass and volume, manual tracing was used to outline endocardial and epicardial borders.

The LV and RV volumes were determined as previously described (13). If the pulmonic valve was seen at the RV

Table 1. Anthropometric and Echocardiographic Data on Endurance Athletes and Untrained Control Subjects

	Endurance Athletes (n = 21)	Untrained Control Subjects (n = 21)	Difference	p Value
Age (yrs)	27 ± 5 (18–36)	26 ± 3 (21–37)	2 ± 7	NS
Weight (kg)	70 ± 8 (53–85)	71 ± 9 (55–88)	1 ± 3	NS
Height (cm)	178 ± 7 (165–190)	178 ± 6 (164–186)	0 ± 3	NS
BSA (m^2)	1.87 ± 0.14 (1.57–2.13)	1.88 ± 0.14 (1.59–2.13)	0.01 ± 0.30	NS
Body fat (%)	10.2 ± 1.8 (7.4–12.5)	17.0 ± 4.3 (9.1–26.0)	6.9 ± 4.3	<0.001
Lean body mass (kg)	63 ± 6 (49–75)	59 ± 6 (47–67)	4 ± 3	<0.001
VO$_2$max (ml/min per kg)	68 ± 5 (60–78)	42 ± 6 (28–53)	26 ± 8	<0.001
Rest heart rate (beats/min)	53 ± 6 (40–60)	66 ± 8 (52–80)	13 ± 11	<0.001
LV-EDD* (mm)	56 ± 3 (51–63)	50 ± 3 (44–57)	6 ± 3	<0.001
IVST* (mm)	11.4 ± 0.9 (10.0–13.0)	10.2 ± 0.9 (7.5–11.5)	1.1 ± 1.3	<0.001
PWT* (mm)	11.2 ± 0.9 (9.0–12.5)	9.5 ± 0.9 (7.0–11.0)	1.4 ± 1.3	<0.001
FS* (%)	35 ± 5 (25–46)	37 ± 5 (26–45)	3 ± 6	NS
RV-EDD* (mm)	26 ± 3 (20–31)	22 ± 4 (12–29)	4 ± 6	<0.05
Heart volume* (ml/kg)	14.5 ± 1.0 (13.1–16.5)	11.1 ± 0.6 (9.5–12.6)	3.5 ± 1.2	<0.001

*Parameters determined by echocardiography. Data are presented as the mean value ± SD (range).

BSA = body surface area; FS = fractional shortening; IVST = interventricular septal thickness; LV-EDD = inner left ventricular end-diastolic diameter; PWT = posterior wall thickness; RV-EDD = inner right ventricular end-diastolic diameter; VO$_2$max = maximal oxygen uptake.
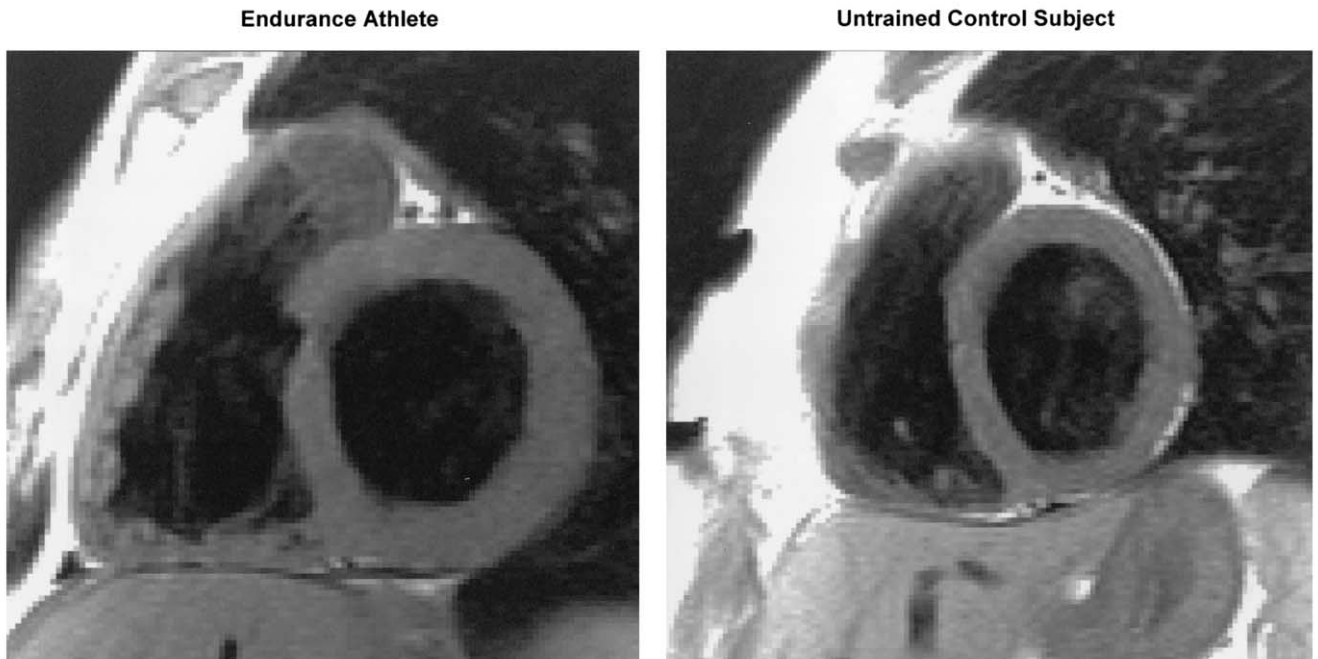
Endurance Athlete

Untrained Control Subject

Figure 1. End-diastolic T_1-weighted short-axis slice from an endurance athlete **(left)** and an untrained control subject **(right)**. Compared with the heart of the control subject, the endurance athlete's heart is characterized by an enlarged volume and a greater myocardial mass of both ventricles, while the proportions of the left and right heart are the same as in the untrained control subject.

volume, the portion above the valve as well as the right atrial volumes were excluded. At the base of the LV, the aorta was included in the LV volume below the aortic valve. Blood volumes above the aortic valve, as well as volumes surrounded by a thin myocardial wall on the mitral valve plane (left atrial blood volume), were excluded from the LV volume. By inspection of the cine loops, end-systole was defined as the frame with the smallest ventricular cavity. The LV-ESV and RV-ESV included aortic and pulmonary outflow tract volumes below the valves. Atrial portions were excluded. To differentiate papillary muscles from the wall in end-systole, cine loops were inspected, and the grayscale was adjusted so that variations near the endocardial border could be seen.

The EDV and ESV were used to determine stroke volume (SV), ejection fraction (EF), cardiac output, and cardiac index. The LV and RV mass was determined by summation of EDVs within the epicardial and endocardial borders of the short-axis slices and multiplying the myocardial tissue volume by its specific density of 1.05 g·cm^{-3}:

$$\text{Myocardial mass} = 1.05 \cdot \sum_{\text{slices}} \text{thk} \cdot (A_{\text{epi}} - A_{\text{endo}})$$

where *thk* represents layer thickness (10 mm); A_{epi} represents epicardial area; and A_{endo} = endocardial area.

Epicardial fat and the pericardium were excluded from RV and LV mass. Determination of LV mass included the septum, but in contrast to Lorenz et al. (13), the papillary muscles were excluded from LV mass analysis to avoid relevant partial volume effects and therefore were added to

the LV volume (18,19). The RV free wall was used to determine RV mass, according to the recommendations of Cutrone et al. (12).

Statistical analysis. Normality was tested by the Kolmogorov-Smirnov test, and differences between endurance athletes and control subjects were measured by the paired *t* test. Pearson correlation coefficients were calculated for selected variables. Data are expressed as the mean value ± SD. An alpha error level of $p < 0.05$ was considered as statistically significant.

RESULTS

Ventricular mass. The LV and RV masses are presented in Figure 2. The LV and RV masses were significantly increased in endurance athletes (A) by 36 ± 14% and 37 ± 17%, respectively, compared with untrained control subjects (C). In addition, the indexed LV and RV masses (values divided by BSA) showed similar differences. The ratio of LV-to-RV mass was equal for athletes and control subjects (Fig. 2). The LV and RV masses correlated significantly with V_{O_2}max (r = 0.89 and 0.85, respectively) (Fig. 3).

Ventricular volume. A significant correlation between LV-EDV and RV-EDV was found (r = 0.76; SEE = 21 ml [LV-EDV = 0.91 · RV-EDV + 16]; $p < 0.001$). The LV-EDV and RV-EDV values are shown in Figure 4. In athletes, LV-EDV and RV-EDV were significantly greater by 34 ± 22% and 25 ± 19%, respectively, compared with control subjects. Indexed LV-EDV and RV-EDV values showed similar differences. The LV-EDV-to-RV-EDV
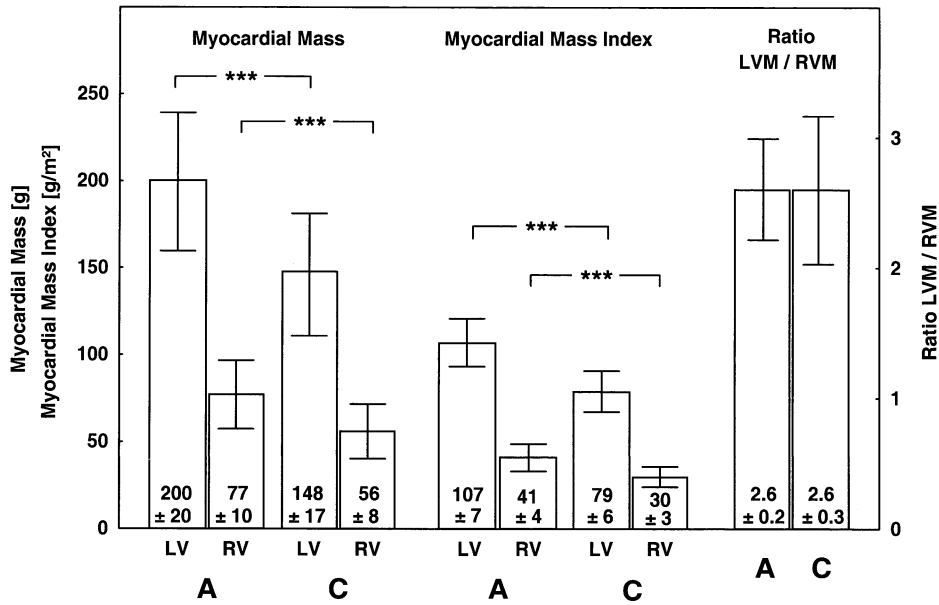
Figure 2. Myocardial mass, myocardial mass index (myocardial mass divided by body surface area) and ratio of left ventricular (LV) to right ventricular (RV) mass (ratio LVM/RVM) in endurance athletes **(A)** and untrained control subjects **(C)**. Data are expressed as the mean value $\pm$ SD. Error bars represent 95% confidence intervals. ***$p < 0.001$ (paired t test).

Figure 3. Correlation of left ventricular mass (LVM) and right ventricular mass (RVM) to maximal oxygen uptake (VO$_2$max).

Figure 4. End-diastolic volume, end-diastolic volume index (end-diastolic volume divided by body surface area) and ratio of left ventricular (LV) to right ventricular (RV) end-diastolic volume (EDV) (ratio LV-EDV/RV-EDV) in endurance athletes **(A)** and untrained control subjects **(C)**. Data are expressed as the mean value ± SD. Error bars represent 95% confidence intervals. ***p < 0.001 (paired *t* test).
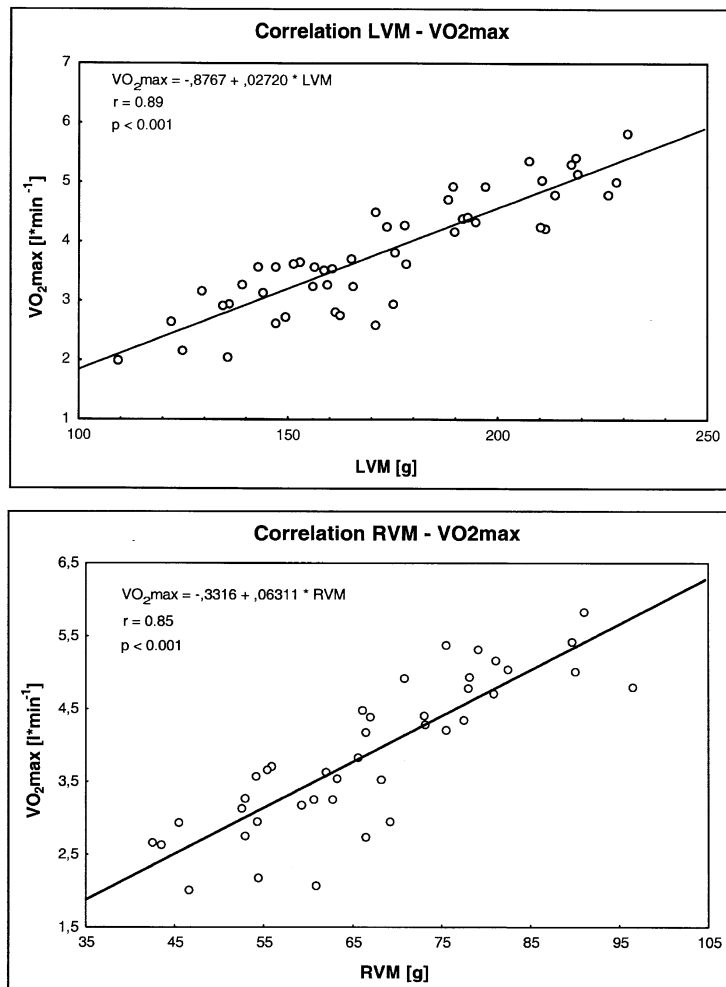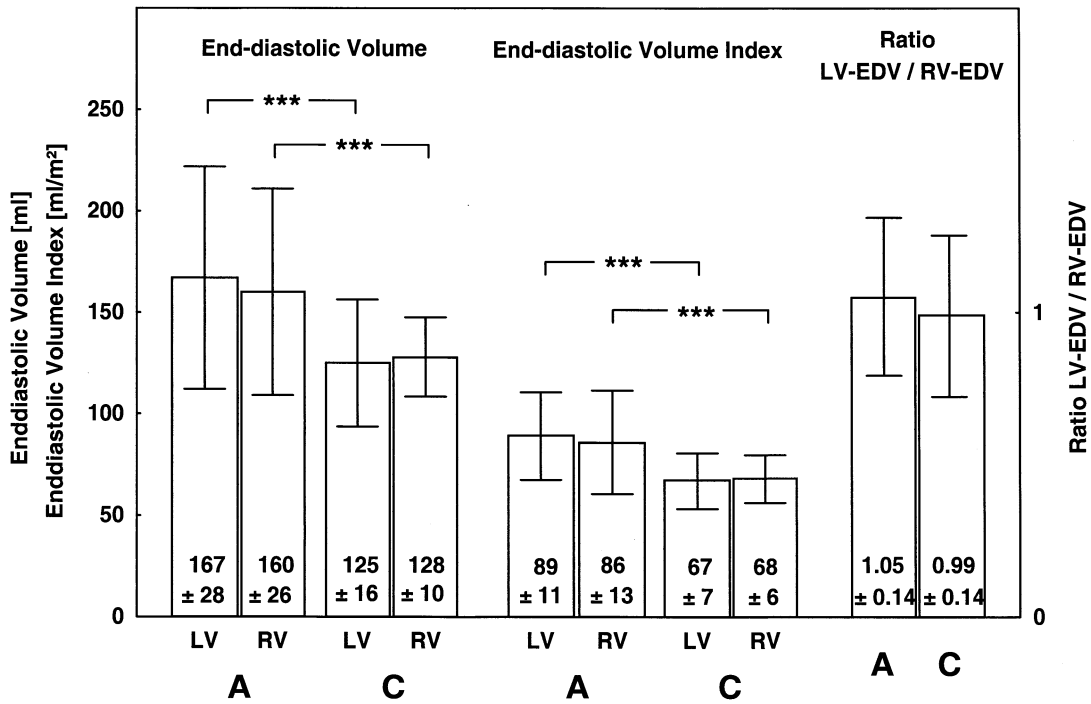
ratio did not differ between athletes and control subjects (Fig. 4).

The LV-ESV and RV-ESV and indexed ESVs were also

significantly different between athletes and control subjects (p < 0.001): LV-ESV 68 ± 11 ml (A) and 51 ± 10 ml (C); indexed LV-ESV 36.3 ± 4.5 ml/m² (A) and 27.1 ± 4.6
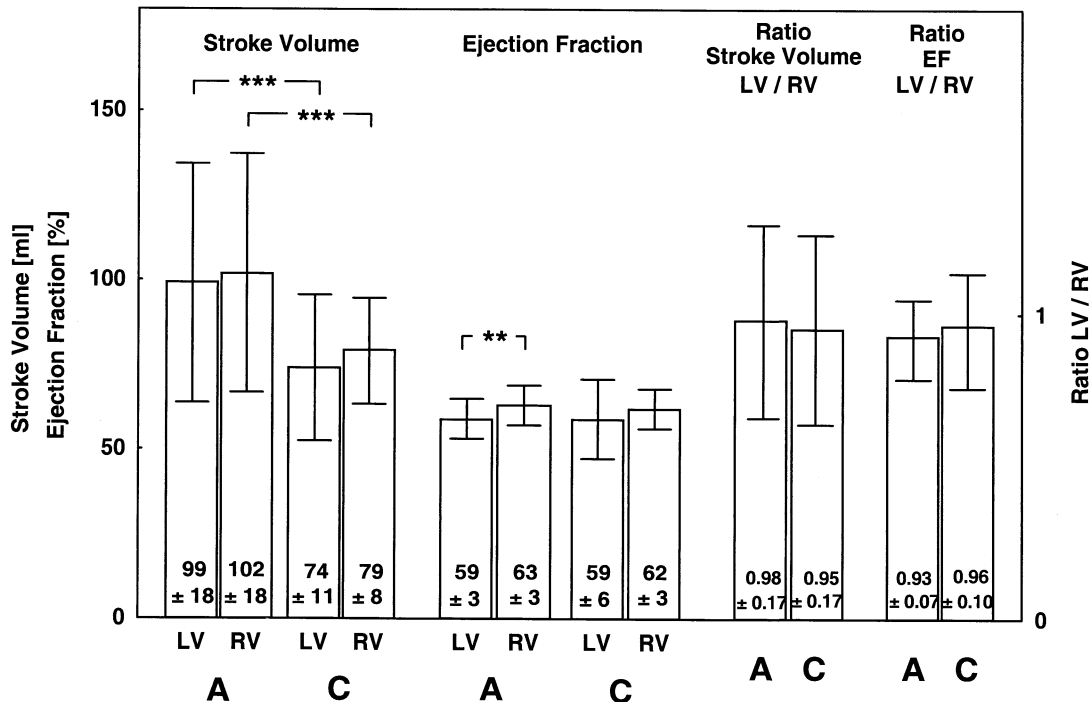

Figure 5. Stroke volume, ejection fraction (EF), ratio of left ventricular (LV) to right ventricular (RV) stroke volume (ratio stroke volume LV/RV), and ratio of LV-EF to RV-EF (ratio EF LV/RV) in endurance athletes **(A)** and untrained control subjects **(C)**. Data are expressed as the mean value ± SD. Error bars represent 95% confidence intervals. **p < 0.01 and ***p < 0.001 (paired *t* test).

ml/m^2 (C); RV-ESV 58 ± 10 ml (A) and 49 ± 4 ml (C); indexed RV-ESV 31.1 ± 4.5 ml/m^2 (A) and 26.1 ± 2.6 ml/m^2 (C).

Functional parameters. Stroke volumes, EFs, and ratios are presented in Figure 5. In athletes, the LV-SV and RV-SV were significantly higher by 34 ± 22% and 29 ± 19%, respectively, compared with control subjects. Indexed LV-SV and RV-SV showed similar differences. The LV-EF and RV-EF did not show a significant difference between athletes and control subjects. However, in athletes, a small but significantly higher RV-EF than LV-EF was noted (p < 0.01). The LV-SV–to–RV-SV ratio, as well as the LV-EF–to–RV-EF ratio, did not differ between athletes and control subjects.

There were no significant differences between athletes and control subjects for cardiac output at rest (A: 5.2 ± 0.9 and C: 4.9 ± 0.9 l/min) and cardiac index at rest (A: 2.8 ± 0.4 and C: 2.6 ± 0.5 l/min per m^2).

DISCUSSION

Although some previous MRI studies analyzed RV mass and function (9–16), studies on endurance athletes with an athlete's heart have not been performed so far. Previous studies were either used to evaluate the method's precision and reproducibility (9,11,12,14) or to examine pathologic alterations of the right heart (10,16). None of these studies differentiated between healthy, untrained subjects and endurance-trained athletes. To our knowledge, this is the first MRI study to evaluate changes in the RV in endurance athletes with an athlete's heart.

Ventricular mass. An LV mass of 115 to 189 g, and normalized to a BSA, an LV mass of 69 to 96 g/m^2, have been reported in healthy subjects in previous MRI studies (18,20–22). In the present study, the LV mass was 148 g and 79 g/m^2 in healthy, untrained control subjects, which is between the reported extreme values. The wide range of extreme values determined in previous MRI studies can be explained by: 1) gender differences and study populations with indiscernible quotas of female subjects; 2) different fitness levels and heart volumes of studied subjects; and 3) methodologic differences in the off-line image analysis. In the present study, papillary muscles were excluded from LV mass analysis, as in earlier studies (18,19), in order to avoid relevant partial volume effects and to allow a better comparison with echocardiographic studies.

For LV hypertrophy, M-mode echocardiographic studies determined an upper clinical limit for LV mass and indexed mass from 215 to 259 g and 125 to 131 g/m^2, respectively, which causes an increase in cardiovascular mortality if exceeded (23–26). In autopsy studies, upper limits from 184 to 204 g were determined (27–30). In a recent MRI study, 238 g and 113 g/m^2 were reported (13). In the present study, the LV mass of endurance athletes averaged 200 g and 107 g/m^2, which was in the upper range of limit values determined by autopsy and MRI studies. Some individual

values exceeded predetermined limit values; however, this extent of hypertrophy is still physiologic and a well-documented phenomenon (2,3,5,7,8). In addition, the LV mass of endurance athletes was very similar to that reported earlier in cyclists (200 g and 102 g/m^2) (18). Higher values reported in other studies (20,31) may be due to aforementioned methodologic differences.

The present RV mass of 56 g in untrained, healthy males was in the upper normal range compared with those values found in previous studies. In other MRI studies, RV mass ranging from 42 to 53 g has been observed in healthy subjects (10–13,15,16). The MRI study with the greatest number of subjects averaged 50 g for RV mass (13) in healthy males. In two computed tomographic studies, RV mass was 54 and 55 g (12,32). In autopsy studies, RV mass ranged from 46 to 58 g (12,27–30), and it was 56 g in males of "average weight" (28,30), which is similar to the present results.

In only two studies, RV mass was normalized to BSA (10,13). In the present study, the indexed RV mass of healthy, untrained males, was slightly higher than that previously reported by Lorenz et al. (13) (26 g/m^2). This small difference may be attributed to methodologic differences. In contrast to Lorenz et al. (13), instead of a cine sequence, we used a T$_1$-weighted sequence to more precisely depict RV trabeculae, as described by Cutrone et al. (12) for RV mass determination. The lower indexed RV mass found by Katz et al. (10) (26 g/m^2), as well as the lower absolute RV masses found in other studies, are most easily attributed to an unknown number of female subjects evaluated (11,12,15,27).

The upper clinical limit of RV mass and indexed mass determined by MRI has been reported to be 70 g and 36 g/m^2, respectively (13). In autopsy studies, the upper clinical limit of RV mass was between 65 and 79 g (27–30). The mean values of 77 g and 41 g/m^2 of the endurance athletes in this study were within the range of the upper clinical limits and, analogous to LV mass, exceeded these limits in individual cases. Comparable MRI data on the RV in athletes do not exist. The present results suggest that endurance training induces not only LV hypertrophy but also RV hypertrophy and therefore also represents a physiologic adaptation.

The LV-to-RV mass ratio in both groups in this study was 2.6, which is almost identical to the results of the two major autopsy studies that determined a normal range from 2.3 to 3.3 (mean 2.7) and 2.1 to 3.6 (mean 2.7), respectively (27,28). However, most important was the finding that normal, healthy subjects and endurance athletes have identical LV-to-RV mass ratios, representing a balanced biventricular hypertrophy in the athlete's heart. This balanced biventricular hypertrophy is also reinforced by the close correlations of LV and RV mass to VO_2max.

Because valid data on the RV of the athlete's heart have not been available so far, the extent of RV hypertrophy has primarily been a matter of speculation. The high prevalence of

an incomplete right bundle branch block, as well as early echocardiographic studies in athletes have led to the assumption that hypertrophy of the athlete's heart might be especially related to the RV (2,4,6). Now, for the first time, the real extent of RV hypertrophy in the athlete's heart has been demonstrated.

Ventricular volume. The LV- and RV-EDV and -ESV of control subjects were similar to those values reported in the literature (9,11,13,14,18,20–22,31,33–35). According to previous results, a close matching between RV-EDV and LV-EDV was found (11,14,16). In two studies in which the RV-EDV was higher than the LV-EDV, a different methodology (9) and partial volume effects of the papillary muscles (9,13) may have led to an underestimation of the LV-EDV.

In the present study, endurance athletes showed greater EDV and ESV of both ventricles. In addition, the similar ratio of LV-EDV to RV-EDV in both groups indicates that endurance training induces both, a balanced biventricular myocardial hypertrophy and a balanced biventricular dilation.

The fact that these endurance athletes showed normal cardiac enlargement is in agreement with previous MRI studies (20,31,35). Only in one study of cyclists, there was evidence of a much larger LV-EDV (222 ml and 112 ml/m^2) (18). However, because the LV-EDV of healthy control subjects was also clearly higher in this previous study (184 ml and 93 ml/m^2) (18), it seems possible that parts of the LV outflow tract above the semilunar valves and/or parts of the left atrium were added to the LV volume.

Functional parameters. For control subjects and endurance athletes, SVs and EFs tended to be somewhat lower than those in the literature (9,11,13,21,22,31,33,35). This could be explained by a more difficult outlining of the papillary muscles in end systole, with a minor overestimation of ESV. Compared with untrained control subjects, the higher LV-SV and RV-SV of endurance athletes is due to a greater EDV of hypertrophied ventricles and not to a higher EF.

Noninvasive studies, in particular, echocardiographic studies, have shown that LV systolic function remains unchanged in the athlete's heart (2,3,6,8). These observations were confirmed in the present study, and in addition are now documented in the RV by the present MRI data. Furthermore, for both study groups, the MRI-derived cardiac output at rest of ~5 l/min also corresponds to that of earlier studies (2,13,31,35).

Study limitations. In addition to a cine sequence for volume determination, a spin-echo sequence was used to get optimal image quality for mass determination, which may have caused some overlap, albeit small, in the measurement of mass and volume. Furthermore, the findings of the present study do not apply to female endurance athletes.

Conclusions. The findings of physiologic LV hypertrophy induced by regular and extensive endurance training in previous echocardiographic and MRI studies can be con-

firmed by the present MRI study. In addition, for the first time, the extent of physiologic RV hypertrophy, including anatomic and functional parameters of the athlete's heart, was evaluated by MRI. Thus, it was possible to demonstrate that regular and extensive endurance training results in similar changes of LV and RV mass, volume, and function, as demonstrated by the constant ratios of the determined parameters. Therefore, the athlete's heart is a balanced enlarged heart.

Reprint requests and correspondence: Dr. Jürgen Scharhag, Institute of Sports and Preventive Medicine, University of Saarland, campus, Building 39.1, 66123 Saarbrücken, Germany. E-mail: j.scharhag@mx.uni-saarland.de.

REFERENCES

1. Kindermann W, Keul J, Reindell H. Grundlagen zur Bewertung leistungs-physiologischer Anpassungsvorgänge [Principles of the evaluation of achievement-physiological adaptation]. Dtsch Med Wochenschr 1974;99:1372–9.
2. Rost R, Hollmann W. Athlete's heart—a review of its historical assessment and new aspects. Int J Sports Med 1983;4:147–65.
3. Urhausen A, Kindermann W. Sports-specific adaptations and differentiation of the athlete's heart. Sports Med 1999;28:237–44.
4. Ferst JA, Chaitman BR. The electrocardiogram and the athlete. Sports Med 1984;1:390–403.
5. Dickhuth HH, Nause J, Staiger J, Bonzel T, Keul J. Two-dimensional echocardiographic measurements of left ventricular volume and stroke volume of endurance-trained athletes and untrained subjects. Int J Sports Med 1983;4:21–6.
6. Maron BJ. Structural features of the athlete heart as defined by echocardiography. J Am Coll Cardiol 1986;7:190–203.
7. Pelliccia A, Maron BJ, Spataro A, Proschan MA, Spirito P. The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. N Engl J Med 1991;324:295–301.
8. Pluim BM, Zwindermann AH, van der Laarse A, van der Wall EE. The athlete's heart: a meta-analysis of cardiac structure and function. Circulation 2000;101:336–44.
9. Sechtem U, Pflugfelder PW, Gould RG, Cassidy MM, Higgins CB. Measurement of right and left ventricular volumes in healthy individuals with cine MR imaging. Radiology 1987;163:697–702.
10. Katz J, Whang J, Boxt LM, Barst RJ. Estimation of right and left ventricular mass in normal subjects and in patients with primary pulmonary hypertension by nuclear magnetic resonance imaging. J Am Coll Cardiol 1993;21:1475–81.
11. Pattynama PM, Lamb HJ, van der Velde EA, van der Geest RJ, van der Wall EE, de Roos A. Reproducibility of MRI-derived measurements of right ventricular volumes and myocardial mass. Magn Reson Imaging 1995;13:53–63.
12. Cutrone JA, Georgiou D, Khan S, et al. Comparison of electron beam computed tomography scanning and magnetic resonance imaging quantification of right ventricular mass: validation with autopsy weights. Acad Radiol 1996;3:395–400.
13. Lorenz CH, Walker ES, Morgan VL, Klein SS, Graham TP Jr. Normal human right and left ventricular mass, systolic function, and gender differences by cine magnetic resonance imaging. J Cardiovasc Magn Reson 1999;1:7–21.
14. Rominger MB, Gachmann GF, Geuer M, et al. Accuracy of right- and left-ventricular heart volume and left ventricular muscle mass determinations by cine MRI in breath-hold technique. Rofo Fortschr Geb Rontgenstr Neuen Bildgeb Verfahr 1999;170:54–60.
15. Doherty NE 3rd, Fujita N, Caputo GR, Higgins CB. Measurement of right ventricular mass in normal and dilated cardiomyopathic ventricles using cine magnetic resonance imaging. Am J Cardiol 1992;69:1223–8.
16. Pattynama PM, Willems LN, Smith AH, van der Wall EE, de Roos A. Early diagnosis of cor pulmonale with MR imaging of the right ventricle. Radiology 1992;182:375–9.

17. Sahn DJ, DeMaria A, Kisslo J, Weyman A. Recommendations regarding quantitation in M-mode echocardiography: results of a survey of echocardiography measurements. Circulation 1978;58:1072–83.

18. Pluim BM, Lamb HJ, Kayser HW, et al. Functional and metabolic evaluation of the athlete's heart by magnetic resonance imaging and dobutamine stress magnetic resonance spectroscopy. Circulation 1998;97:666–72.

19. Geil S, Rao L, Menzel T, et al. Determination of left ventricular mass by transthoracic three-dimensional echocardiography in patients with dilated cardiomyopathy. Z Kardiol 1999;88:922–31.

20. Milliken MC, Stray-Gundersen J, Peshock RM, Katz J, Mitchell JH. Left ventricular mass as determined by magnetic resonance imaging in male endurance athletes. Am J Cardiol 1988;62:301–5.

21. Buser PT, Auffermann W, Holt WW, et al. Noninvasive evaluation of global left ventricular function with use of cine nuclear magnetic resonance. J Am Coll Cardiol 1989;13:1294–300.

22. Semelka RC, Tomei E, Wagner S. Normal left ventricular dimensions and function: interstudy reproducibility of measurements with cine MR imaging. Radiology 1990;174:763–68.

23. Casale PN, Devereux RB, Milner M, et al. Value of echocardiographic measurement of left ventricular mass in predicting cardiovascular morbid events in hypertensive men. Ann Intern Med 1986;105:173–8.

24. Devereux RB, Alonso DR, Lutas EM, et al. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. Am J Cardiol 1986;57:450–8.

25. Levy D, Savage DD, Garrison RJ, Anderson KM, Kannel WB, Castelli WP. Echocardiographic criteria for left ventricular hypertrophy: the Framingham Heart Study. Am J Cardiol 1987;59:956–60.

26. Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. N Engl J Med 1990;322:1561–6.

27. Fulton RM, Hutchinson EC, Jones AM. Ventricular weight in cardiac hypertrophy. Br Heart J 1952;14:413–20.

28. Bove KE, Rowlands DT, Scott RC. Observations on the assessment of cardiac hypertrophy utilizing a chamber partition technique. Circulation 1966;33:558–68.

29. Mitchell RS, Stanford RE, Silvers GW, Dart G. The right ventricle in chronic airway obstruction: a clinicopathologic study. Am Rev Respir Dis 1976;114:147–54.

30. Hangartner JR, Marley NJ, Whitehead A, et al. The assessment of cardiac hypertrophy at autopsy. Histopathology 1985;9:1295–306.

31. Turpeinen AK, Kuikka JT, Vanninen E. Athletic heart: a metabolic, anatomical, and functional study. Med Sci Sports Exerc 1996;28:33–40.

32. Hajduczok ZD, Weiss RM, Stanford W, Marcus ML. Determination of right ventricular mass in humans and dogs with ultrafast cardiac computed tomography. Circulation 1990;82:202–12.

33. Kennedy JW, Baxley WA, Figley MM, Doge HT, Blackmon JR. Quantitative angiography. I. The normal left ventricle in men. Circulation 1966;34:272–8.

34. Stewart GA, Foster J, Cowan M, et al. Echocardiography overestimates left ventricular mass in hemodialysis patients relative to magnetic resonance imaging. Kidney Int 1999;56:2248–53.

35. Zandrino F, Molinari G, Smeraldi A, Odaglia G, Masperone MA, Sardinelli F. Magnetic resonance imaging of the athlete's heart: myocardial mass, left ventricular function, and cross sectional area of the coronary arteries. Eur Radiol 2000;10:319–25.