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Atrial Adaptations in Athletes Heart

Philipp Stadter[1] | Karsten Keller[1,2,3]

[1]Medical Clinic VII, Department of Sports Medicine, University Hospital Heidelberg, Heidelberg, Germany | [2]Department of Cardiology, University Medical Center of the Johannes Gutenberg-University Mainz, Mainz, Germany | [3]Center for Thrombosis and Hemostasis (CTH), University Medical Center of the Johannes Gutenberg-University Mainz, Mainz, Germany

Correspondence: Karsten Keller (Karsten.Keller@unimedizin-mainz.de)

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ABSTRACT

Background: Intensive training efforts are associated with hemodynamic changes accompanied by increases in cardiac output and stroke volume related to higher peak oxygen consumption and better athletic performance during exercise. These hemodynamic changes induce an enlargement of cardiac chambers, but also of the atria and may result in an athletes' heart (AH). Data from large studies about atrial enlargement in AH are sparse.

Methods: Competitive athletes aged ≥18 years, who presented for pre-participation screening 04/2020–10/2021 were included in this study and stratified for AH (defined as physiologically increased heart volume >13.0 in males and >12.0 mL/kg in females).

Results: Overall, 646 athletes aged ≥18 years (median age 24.0 [20.0/31.0] years; 206 [31.9%] females) were included in our study 04/2020–10/2021; among these, 118 (18.3%) had an AH. The computed absolute heart volume was 969.4 (853.1/1083.0) mL in athletes with AH and 841.3 (707.4/966.3) mL in those without AH ($p < 0.001$). AH was associated with larger left ventricular mass (206.6 ± 39.0 vs. 182.7 ± 44.2 g, $p < 0.001$). LA area (15.4 [13.7/18.2] vs. 14.3 [12.0/16.3] cm^2, $p < 0.001$) and RA area (15.8 [13.8/18.6] vs. 14.5 [12.3/17.0] cm^2, $p < 0.001$) were enlarged in AH versus those athletes without AH. The logistic regressions confirmed an independent association of AH on LV mass (OR 1.05 [95% CI 1.04–1.06], $p < 0.001$). LA area (OR 1.29 [95% CI 1.19–1.39], $p < 0.001$) as well as RA area (OR 1.28 [95% CI 1.19–1.38], $p < 0.001$) were afflicted by AH.

Conclusion: An AH is accompanied by significant enlargement of the atria as well as increased cardiac muscle mass.

1 | Introduction

The heart of the athlete has fascinated clinicians as well as sports scientists for more than a century [1]. Cardiac adaptations driven by physical activity were first recognized short before the beginning of the 19th century [2–4]. In the year 1899, Henschen reported, for the first time, enlarged and thickened hearts in cross country skiers and interpreted these findings as structural and functional adaptations triggered by physical activity [2–4]. During the 19th century and also at the begin of the 20th century, outstanding research regarding athletes' heart (AH) contributed to the current understanding in this field [2, 4–6]. While the research in former times based mainly on radiographic measurements of the heart volume, with introduction and implementation of the echocardiography, echocardiographic methods became the standard method in sports medicine and is still the method of choice to examine an AH [2, 4].

An AH is generally defined as a benign increase in cardiac size and mass due to cardiac remodeling caused by increased volume loading driven by the physical activity [4, 7]. The AH is observed as a harmonic eccentric enlargement of the heart [7–9]. Intensive training efforts result in hemodynamic changes accompanied by increases in cardiac output and stroke volume related to the

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ability of higher maximal oxygen consumption and better athletic performance during exercise [10]. These hemodynamic changes primarily induce an enlargement of the cardiac chambers and secondly also of the atria [10]. Besides these benign adaptations, the enlargement of the heart cavities can also be caused by cardiomyopathy [9–11]. Atrial dilatation is an independent marker for cardiovascular events and is associated with arterial as well as pulmonary hypertension, heart disease, and poor survival [11–14]. Therefore, it is of outstanding interest to distinguish between benign and pathological atrial enlargement and a clear characterization of the benign atrial adaptations in highly trained athletes investigating a huge number of athletes is necessary for the understanding of these benign adaptations.

2 | Methods

For this aim, we conducted a retrospective analysis of standard data of adult athletes (aged ≥18 years) who presented for their preparticipation screening examination at the Department of Sports Medicine (Medical Clinic VII) of the University Hospital Heidelberg (Germany) between April 2020 and October 2021. Although the study was primarily designed to investigate blood pressure adaptations in athletes, the echocardiographic measurements could be used adequately for this present study. The study design was already described in detail in two previously published papers [15, 16].

2.1 | Enrolled Subjects

Athletes were eligible to be included in this study, if they were aged at least 18 years old, in regular training for competition and presented for pre-participation screening examination at our department [15–18].

2.2 | Ethical Aspects

The requirement for an informed consent for this present study was waived as we used only anonymized retrospective data, which was routinely collected during the health screening process. Studies in Germany involving only a retrospective analysis of diagnostic standard data of anonymized patients do not require an ethics statement, in accordance with German law.

2.3 | Definitions and Assessed Parameters

All athletes included in this study had a transthoracic echocardiography. Echocardiographic parameters were defined according to current guidelines [18, 19]. LA and LV measurements were measured in a standardized method and all of these measurements were critically reviewed by one of the authors (K.K.). The cardiac volume in the established echocardiographic approach was estimated with the combined one- and two-dimensional method for determination of the cardiac volume, which is based on the modified Simpson rule [20–23]. The method first calculates the end-diastolic left ventricular (LV) total volume taking into account the measured end-diastolic LV diameter in the parasternal M-mode at the papillary muscle as well as at mitral valve level and the measurement of the maximum longitudinal diameter from the apex of the left ventricle to mitral annular level [20–22]. The cardiac volume is calculated based on correlation of the echocardiographic measurements to established radiographic total heart volume determinations using a regression equation method [20–23]. The underlying formula for the heart volume calculation is established in sports medicine and could be observed in previously published studies [20, 22, 23]. It has been reported that these echocardiographic method to estimate the cardiac volume is valid and offers comparable results to x-ray and magnetic resonance imaging measurements [20–23]. However, it is important to mention that this method is only reliable assessable if the heart cavity conditions are harmonious enlarged, but not pathological and unequally dilated [20, 21]. To compute the relative cardiac volume, the absolute calculated cardiac volume was related to the athlete's body weight. An AH was defined as a physiologically increased heart volume >13.0 in males and >12.0 mL/kg in females [2, 9, 21, 24].

As previously described [15, 16], LV mass was computed according to the established 2D echocardiography area-length method: LV mass = 0.80 * (1.04 * [(septal LV wall thickness + LV end-diastolic diameter + posterior LV wall thickness)^3 − (LV end-diastolic diameter)^3]) + 0.6 g [19]. Left ventricular hypertrophy (LVH) was defined as (I) septal or posterior LV wall diameter ≥13 mm [18, 25] or (II) LV mass >162 g in female or >224 g in male individuals [15, 16, 19].

Exercise hypertension was defined on basis of the systolic blood pressure/MET slope-method [26–29]: The Δ regarding systolic blood pressure was calculated as maximum systolic blood pressure during exercise—systolic BP at rest and was indexed by the increase in MET from rest (Δ regarding MET was calculated as peak MET—1) to obtain the systolic blood pressure/MET slope [28]. In accordance with previous studies, a cut-off value >6.2 mmHg/MET was used to define an exercise hypertension [15, 26, 28]. The MET value was calculated based on the athletes' VO_2 peak value during exercise testing as recommended by the ACSM guideline (MET = VO_2 peak/3.5 mL × kg × min) [30].

Cardio-pulmonary exercise testing (CPET) was performed adapted to athlete's sports specifically (bicycle, treadmill, rowing, or canoeing ergometer) efforts and according to current guidelines with electrocardiogram (ECG) and blood pressure measurements at the end of every load level. The CPET followed a level protocol with each level/step lasting 3 min. The exercise test was stopped if the athlete reached his maximum capacity or stopping criteria according to current guidelines enforced the stop of the exercise test [17, 18].

Obesity was defined as body mass index (BMI) ≥30 kg/m^2 according to the World Health Organization (WHO) [15, 16, 31]. Body fat was measured by caliper method. Calipometry is an easy and well-established method to measure subcutaneous fat tissue depth through the skinfold thickness to determine total body fat applying a specific skinfold equation with calculation of total body fat on the basis of body density manifested through regression analysis [32].

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2.4 | Statistics

The included athletes aged ≥18 years who presented for pre-participation screening examination at our department were stratified for AH in the two groups of athletes with and without AH. For descriptive analysis of baseline characteristics, dichotomous variables were presented by absolute and relative frequencies and differences assessed with Fisher's Exact Test. Continuous variables were described using the mean and standard deviation (SD), and in case of a markedly non-normal distribution, by the median and the 25th and 75th percentiles (inter-quartile range, IQR). Statistical comparisons for continuous variables were made using the Mann–Whitney U-test in case of non-normal distributions; otherwise, an unpaired *t*-test was applied, as appropriate.

We performed univariate and multivariate logistic regression models to investigate the association between different factors such as age, sex, body composition parameters, cardiovascular risk factors, and metabolic parameters on the one hand and development of AH on the other hand as well as the association between AH and different echocardiographic parameters. Multivariate regression models were adjusted for age, sex, and BMI in order to proof the independence of the statistical results of these parameters. Results of the logistic regressions are presented as odds ratio (OR) and 95% confidence interval (CI).

All of the statistical analyses were computed with the use of SPSS software (IBM Corp. Released 2017, IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY, USA). Only the p values < 0.05 (two-sided) were considered to be of statistical significance. If, we adjust the p value for multiple testing with the Bonferroni correction method, an adjusted p-value for significance of 0.0125 was computed for the present analysis.

3 | Results

Overall, 646 athletes aged ≥18 years (median age 24.0 [20.0/31.0] years; 206 [31.9%] females) were included in our study during the observational period between April 2020 and October 2021; among them, 118 (18.3%) had an AH according the above mentioned echocardiographic criteria. Overall, 425 (65.8%) were leading athletes of regional and national level. The median duration of training on a high level was 11.0 (7.0/15.0) years.

AH was most prevalent in the 3rd and 4th decade of life, whereby AH in male athletes was more often detected in younger age, whereas in female athletes the peak proportion was identified for the 4th decade of life. As illustrated in Figure 1, we detected that AH was more common in endurance sports and less frequent in game sports. Interestingly, the proportion of leading athletes of a regional and national level was comparably in athletes with and without an AH (65.3% vs. 65.9%, $p = 0.892$) (Table 1). The mean durations of training on a high level standard were similar between athletes with and without AH (12.6 vs. 11.9 years) and the proportion of the duration of high level training in relation to the life-time was also not substantially different (0.46% vs. 0.45%) between the groups.

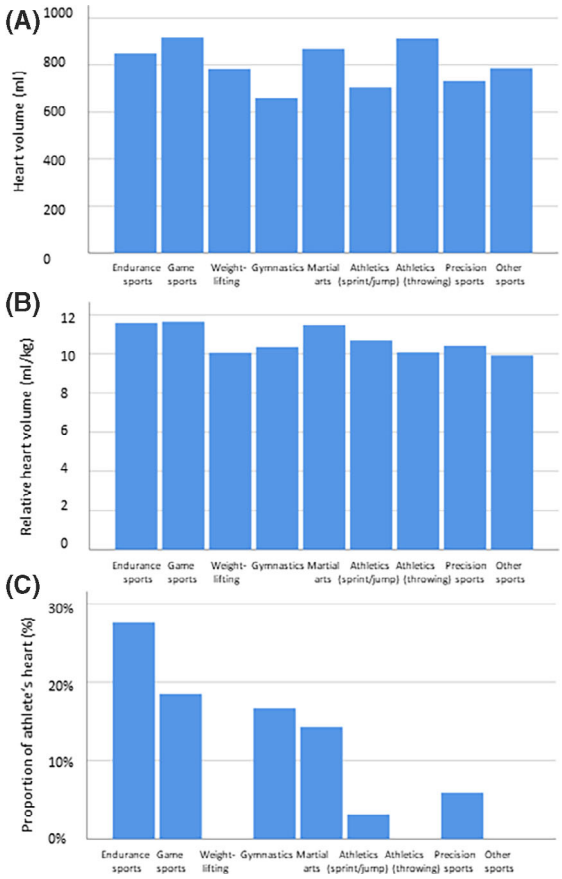

FIGURE 1 | Impact of different sports on absolute heart volume (A), heart volume related to body weight (B), and proportion of athletes with an athletes' heart (C).

3.1 | Athletes With AH Were Leaner and Had a Better Athletic Performance

Athletes with an AH were more often female (39.8% vs. 30.1%, $p = 0.041$) and revealed lower body weight, BMI, and body fat in comparison to athletes without AH (Table 1). Consecutively, obesity was more prevalent in athletes without AH (3.8% vs. 0.0%, $p = 0.034$). Cardiovascular diseases (including, e.g., arterial hypertension and coronary artery disease) were uncommon in both groups, but slightly more frequently found in athletes without AH compared to those with AH (8.7% vs. 3.4%, $p = 0.050$) (Table 1).

Blood pressure values in rest and during exercise testing did not differ between athletes with and without AH and also exercise hypertension was similar prevalent in both groups. In contrast, athletic performance detected by VO_2 peak was significantly better in athletes with AH than in those without (49.1 [45.2/53.2] vs. 42.4 [36.9/48.3] mL/min/kg, $p < 0.001$) in the light of similar exhaustion levels (RER and lactate values ware comparable between both groups) (Table 1).

The logistic regression confirmed that AH was independently associated with lower body weight (OR 0.96 [95% CI 0.94–0.98], $p < 0.001$), lower BMI (OR 0.77 [95% CI 0.70–0.84], $p < 0.001$), and lower body fat (OR 0.76 [95% CI 0.68–0.84], $p < 0.001$), but not with female sex after adjustment for age and BMI (Table 2). Remarkably, AH was associated with higher maximum

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TABLE 1 | Patients' characteristics of the 646 examined athletes stratified for athlete's heart.

Parameters	Athletes without an athlete's heart ($n = 528$; 81.7%)	Athletes with an athlete's heart ($n = 118$; 18.3%)	p-value
Age (in years)	28.5 ± 12.9	27.4 ± 11.0	0.360
Female sex	159 (30.1%)	47 (39.8%)	**0.041**
Body height (cm)	188.5 ± 9.5	177.1 ± 8.0	0.600
Body weight (kg)	77.5 (67.0/86.1)	71.9 (63.2/77.8)	**<0.001**
Body mass index (kg/m^2)	24.2 ± 2.9	22.5 ± 2.1	**<0.001**
Body fat (%)	13.4 ± 5.1	12.1 ± 4.2	**0.005**
Leading athletes of a regional and national level	348 (65.9%)	77 (65.3%)	0.892
Cardiovascular risk factors and cardiovascular diseases			
Nicotine abuse	31 (5.9%)	9 (7.6%)	0.474
Obesity	20 (3.8%)	0 (0.0%)	**0.034**
Cardiovascular diseases	46 (8.7%)	4 (3.4%)	0.050
Iron deficiency	59 (11.3%)	21 (17.8%)	0.056
SARS-CoV2 in medical history	105 (19.9%)	24 (20.3%)	0.911
Blood pressure values			
Systolic blood pressure (mmHg)	119.6 ± 13.4	117.5 ± 10.9	0.078
Diastolic blood pressure (mmHg)	71.8 ± 8.5	71.5 ± 7.4	0.711
Maximum systolic blood pressure during exercise (mmHg)	192.5 (175.0/210.0)	200.0 (180.0/212.5)	0.058
Maximum diastolic blood pressure during exercise (mmHg)	76.2 ± 9.8	74.4 ± 9.5	0.062
Exercise hypertension (defined by systolic blood pressure (BP)/MET slope-method with cut-off value > 6.2 mmHg/MET)	184 (52.1%)	31 (41.9%)	0.109
Exercise parameters			
VO_2 peak (mL/min/kg)	42.4 (36.9/48.3)	49.1 (45.2/53.2)	**<0.001**
Maximal respiratory exchange ratio (RER)	1.17 ± 0.07	1.16 ± 0.07	0.554
Maximum lactate value	9.9 ± 2.4	9.7 ± 2.5	0.592
Echocardiographic parameters			
Left ventricular hypertrophy	138 (26.1%)	66 (55.9%)	**<0.001**
Left ventricular mass (g)	182.7 ± 44.2	206.6 ± 39.0	**<0.001**
Aortic valve regurgitation	46 (8.7%)	17 (14.4%)	0.059
Mitral valve regurgitation	342 (64.8%)	80 (67.8%)	**0.009**
Tricuspid valve regurgitation	86 (16.3%)	24 (20.3%)	0.485
Pulmonary valve regurgitation	36 (6.8%)	14 (11.9%)	0.064
Absolute heart volume (mL)	841.3 (707.4/966.3)	969.4 (853.1/1083.0)	**<0.001**
Heart volume related to body weight (mL/kg)	11.0 (10.0/11.9)	13.5 (13.0/14.0)	**<0.001**
Left ventricular ejection fraction measured by Simpson method (%)	65.4 ± 4.7	66.1 ± 5.1	0.162
Left ventricular end-diastolic diameter (mm)	50.0 (47.0/54.0)	53.0 (50.8/56.0)	**<0.001**
Left atrial area (cm^2)	14.3 (12.0/16.3)	15.4 (13.7/18.2)	**<0.001**
LA Septal-lateral diameter (mm)	32.0 (28.0/35.0)	34.0 (31.0/39.0)	**<0.001**
LA Apico-basal diameter (mm)	49.0 (45.0-54.0)	51.0 (47.0/56.0)	**<0.001**
Right atrial area (cm^2)	14.5 (12.3/17.0)	15.8 (13.8/18.6)	**<0.001**
RA Septal-lateral diameter (mm)	35.0 (31.0/40.0)	38.0 (33.0/42.0)	**0.001**

(Continues)

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TABLE 1 | (Continued)

Parameters	Athletes without an athlete's heart ($n = 528$; 81.7%)	Athletes with an athlete's heart ($n = 118$; 18.3%)	*p*-value
RA Apico-basal diameter (mm)	46.0 (42.0/51.0)	48.0 (44.5–51.5)	**<0.001**
Tricuspid annular plane systolic excursion (TAPSE, cm)	2.6 ± 0.4	2.7 ± 0.4	**0.007**
Systolic pulmonary artery pulmonary pressure (mmHg)	20.2 ± 4.5	20.6 ± 4.5	0.489
E/A quotient	2.3 (1.7/3.3)	2.9 (2.0/4.1)	**<0.001**
E/E' quotient	4.9 ± 1.5	4.6 ± 1.3	**0.023**

Bold values indicate statistical significance $p < 0.05$.

TABLE 2 | Association between different factors and development of an athletes' heart (univariate and multivariate logistic regression model).

	Univariate regression model		Multivariate regression model (adjusted for age, sex, and body-mass-index)	
	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value
Age (years)	0.99 (0.98–1.01)	0.406	1.00 (0.98–1.02)	0.910
Female sex	0.65 (0.43–0.98)	**0.041**	1.11 (0.70–1.76)	0.657
Body height (cm)	1.00 (0.97–1.02)	0.638	1.01 (0.98–1.04)	0.501
Body weight (kg)	0.96 (0.95–0.98)	**<0.001**	0.96 (0.94–0.98)	**<0.001**
Body mass index (kg/m^2)	0.77 (0.71–0.85)	**<0.001**	0.77 (0.70–0.84)	**<0.001**
Body fat (%)	0.94 (0.90–0.99)	**0.012**	0.76 (0.68–0.84)	**<0.001**
Leading athletes of a regional and national level	0.97 (0.64–1.48)	0.892	1.04 (0.61–1.77)	0.895
Cardiovascular risk factors and cardiovascular diseases				
Nicotine abuse	1.32 (0.61–2.86)	0.476	1.67 (0.71–3.89)	0.238
Cardiovascular diseases	0.37 (0.13–1.05)	0.060	0.43 (0.14–1.35)	0.148
Iron deficiency	1.69 (0.98–2.91)	0.058	1.36 (0.73–2.54)	0.330
SARS-CoV2 in medical history	1.03 (0.63–1.69)	0.911	1.13 (0.67–1.90)	0.650
Exercise hypertension (defined by systolic blood pressure (BP)/MET slope-method with cut-off value > 6.2 mmHg/MET)	0.68 (0.40–1.10)	0.111	1.00 (0.56–1.78)	0.993

Bold values indicate statistical significance $p < 0.05$.

systolic blood pressure during exercise (mmHg) (OR 1.02 [95% CI 1.01–1.03], $p < 0.001$) and increased VO_2 peak (OR 1.19 (95% CI 1.12–1.25, $p < 0.001$) (Table 3).

3.2 | AH Is Associated With Increased Muscle Mass and Larger Atria

The computed absolute heart volume was 969.4 (853.1/1083.0) ml in athletes with AH and 841.3 (707.4/966.3) mL in those without AH ($p < 0.001$). AH was associated with increased left ventricular muscle mass, seen in a larger left ventricular mass (206.6 ± 39.0 vs. 182.7 ± 44.2 g, $p < 0.001$) and higher rate of LVH (55.9% vs. 26.1%, $p < 0.001$) (Table 1).

As expected, the LV end-diastolic diameter was larger in athletes with AH than in those without (53.0 [50.8–56.0] vs. 50.0 [47.0/54.0] mm, $p < 0.001$), whereas LV ejection fraction measured by Simpson method was similar between both groups ($p = 0.162$) (Table 1). Regarding heart valve insufficiencies, only mitral valve insufficiency was more prevalent in athletes with AH (67.8% vs. 64.8%, $p = 0.009$), whereby all of these detected heart valve insufficiencies were of a small extent (Table 1). Tricuspid annular plane systolic excursion (TAPSE) and E/A quotient were both larger in athletes with AH, while systolic pulmonary artery pulmonary pressure did not differ between both groups (Table 1).

All investigated atrial dimensions were larger in athletes with AH than in those without. In the left atrium (LA), LA area (15.4 [13.7/18.2] vs. 14.3 [12.0/16.3] cm^2, $p < 0.001$), LA septal-lateral diameter (34.0 [31.0/39.0] vs. 32.0 [28.0/35.0] mm, $p < 0.001$) and LA apico-basal diameter (51.0 [47.0/56.0] vs. 49.0 [45.0–54.0] mm, $p < 0.001$) were larger in athletes with AH than in those without (Table 1). Regarding the right atrium (RA),

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TABLE 3 | Association between athletes' heart and echocardiographic parameters (univariate and multivariate logistic regression model).

	Univariate regression model		Multivariate regression model (adjusted for age, sex, and body-mass-index)	
	OR (95% CI)	***p*-value**	**OR (95% CI)**	***p*-value**
Blood pressure values				
Systolic blood pressure (mmHg)	0.99 (0.97–1.00)	0.119	1.00 (0.98–1.02)	0.942
Diastolic blood pressure (mmHg)	1.00 (0.97–1.02)	0.733	1.01 (0.99–1.04)	0.381
Maximum systolic blood pressure during exercise (mmHg)	1.01 (1.00–1.02)	0.077	1.02 (1.01–1.03)	**<0.001**
Maximum diastolic blood pressure during exercise (mmHg)	0.98 (0.96–1.00)	0.066	1.00 (0.98–1.01)	0.339
Exercise parameters				
VO_2 peak (mL/min/kg)	1.14 (1.09–1.18)	**<0.001**	1.19 (1.12–1.25)	**<0.001**
Maximal respiratory exchange ratio (RER)	0.36 (0.01–11.28)	0.562	0.29 (0.01–10.62)	0.498
Maximum lactate value	0.97 (0.85–1.09)	0.573	0.99 (0.86–1.13)	0.843
Echocardiographic parameters				
Left ventricular hypertrophy	3.59 (2.38–5.42)	**<0.001**	7.58 (4.68–12.29)	**<0.001**
Left ventricular mass (g)	1.01 (1.01–1.02)	**<0.001**	1.05 (1.04–1.06)	**<0.001**
Aortic valve regurgitation	1.76 (0.97–3.20)	0.062	1.85 (0.98–3.51)	0.060
Mitral valve regurgitation	1.15 (0.75–1.75)	0.533	1.25 (0.81–1.95)	0.318
Tricuspid valve regurgitation	1.31 (0.79–2.17)	0.291	1.39 (0.81–2.39)	0.229
Pulmonary valve regurgitation	1.84 (0.96–3.53)	0.067	2.05 (1.04–4.07)	**0.040**
Left ventricular ejection fraction measured by Simpson method (%)	1.03 (0.99–1.08)	0.139	1.03 (0.98–1.07)	0.250
Left ventricular end-diastolic diameter (mm)	1.17 (1.11–1.24)	**<0.001**	1.45 (1.34–1.57)	**<0.001**
Left atrial area (cm^2)	1.14 (1.07–1.21)	**<0.001**	1.29 (1.19–1.39)	**<0.001**
LA Septal-lateral diameter (mm)	1.10 (1.05–1.15)	**<0.001**	1.17 (1.12–1.23)	**<0.001**
LA Apico-basal diameter (mm)	1.06 (1.03–1.09)	**<0.001**	1.09 (1.05–1.12)	**<0.001**
Right atrial area (cm^2)	1.11 (1.05–1.17)	**<0.001**	1.28 (1.19–1.38)	**<0.001**
RA Septal-lateral diameter (mm)	1.06 (1.02–1.09)	**<0.001**	1.11 (1.07–1.16)	**<0.001**
RA Apico-basal diameter (mm)	1.06 (1.03–1.10)	**<0.001**	1.13 (1.08–1.17)	**<0.001**
Tricuspid annular plane systolic excursion (TAPSE, cm)	1.96 (1.21–3.19)	**0.007**	3.33 (1.94–5.73)	**<0.001**
Systolic pulmonary artery pulmonary pressure (mmHg)	1.02 (0.97–1.08)	0.491	1.05 (0.99–1.12)	0.109
E/A quotient	1.33 (1.14–1.55)	**<0.001**	1.32 (1.11–1.56)	**0.002**
E/E' quotient	0.86 (0.74–0.99)	**0.040**	0.80 (0.68–0.95)	**0.011**

Bold values indicate statistical significance $p < 0.05$.

RA area (15.8 [13.8/18.6] vs. 14.5 [12.3/17.0] cm^2, $p < 0.001$), but also RA septal-lateral diameter (38.0 [33.0/42.0] vs. 35.0 [31.0/40.0] mm, $p = 0.001$) and RA apico-basal diameter (48.0 [44.5–51.5] vs. 46.0 [42.0/51.0] mm, $p < 0.001$) were enlarged in AH versus those athletes without AH (Table 1). These comparisons remained significant after adjustment with the Bonferroni correction method.

In the sex-specific analysis, the absolute heart volume was lower in female in comparison to male athletes. However, the impact of AH on echocardiographic parameters was similar to the non-sex-specific analysis. Especially the impact of AH on atrial dimensions enlargements were similar and therefore, not sex-specific (Table 4).

The logistic regressions confirmed an independent association of AH on LVH (OR 7.58 [95% CI 4.68–12.29], $p < 0.001$) and LV mass (OR 1.05 [95% CI 1.04–1.06], $p < 0.001$) (Table 3). AH was also independently associated with pulmonary valve regurgitation (OR 2.05 [95% CI 1.04–4.07], $p = 0.040$) and larger LV end-diastolic diameter (OR 1.45 [95% CI 1.34–1.57], $p < 0.001$) as well as TAPSE (OR 3.33 [95% CI 1.94–5.73], $p < 0.001$), higher E/A quotient (OR 1.32 [95% CI 1.11–1.56], $p = 0.002$), and smaller E/E' quotient (OR 0.80 [95% CI 0.68–0.95), $p = 0.011$ (Table 3).

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TABLE 4 | Median echocardiographic measurements of the female and male athletes stratified for athlete's heart.

	Female athletes ($n = 206$)			Male athletes ($n = 440$)		
	Female athletes without an athlete's heart ($n = 159$; 77.2%)	**Female athletes with an athlete's heart ($n = 47$; 22.8%)**	***p*-value**	**Male athletes without an athlete's heart ($n = 369$; 83.9%)**	**Male athletes with an athlete's heart ($n = 71$; 16.1%)**	***p*-value**
Absolute heart volume (mL)	673.6 (600.9/736.6)	813.8 (742.2/925.2)	**<0.001**	915.2 (810.9/1010.3)	1052.0 (972.0/1157.0)	**<0.001**
Heart volume related to body weight (mL/kg)	10.5 (9.6/11.1)	12.7 (12.3/13.5)	**<0.001**	11.4 (10.3/12.2)	13.7 (13.4/14.3)	**<0.001**
Left ventricular ejection fraction measured by Simpson method (%)	66.0 (63.0/69.0)	66.5 (63.0/69.3)	0.917	65.0 (62.0/68.0)	66.0 (61.0/70.0)	0.312
Left ventricular end-diastolic diameter (mm)	47.0 (44.0/49.0)	51.0 (49.0/53.0)	**<0.001**	52.0 (49.0/55.0)	55.0 (52.0/56.0)	**<0.001**
Left atrial area (cm^2)	13.0 (11.0/14.9)	14.8 (12.8/16.6)	**<0.001**	14.9 (12.8/16.9)	16.5 (14.5/18.3)	**0.001**
LA Septal-lateral diameter (mm)	30.0 (27.0/33.0)	33.0 (30.0/36.0)	**<0.001**	33.0 (30.0/36.0)	35.0 (32.0/39.0)	**<0.001**
LA Apico-basal diameter (mm)	48.0 (43.0/52.0)	49.0 (47.0/55.0)	**0.013**	49.0 (45.0/54.0)	52.0 (48.0/57.0)	**0.003**
Right atrial area (cm^2)	12.9 (10.6/14.8)	14.5 (12.8/16.6)	**<0.001**	15.1 (13.3/17.7)	17.0 (14.5/18.3)	**0.001**
RA Septal-lateral diameter (mm)	32.0 (29.0/36.0)	35.0 (31.0/38.0)	**0.008**	37.0 (33.0/41.0)	41.0 (36.0/44.0)	**<0.001**
RA Apico-basal diameter (mm)	43.0 (39.0/48.0)	48.0 (44.0/51.0)	**<0.001**	47.0 (43.0/51.0)	49.0 (45.0/53.0)	**0.040**
Tricuspid annular plane systolic excursion (TAPSE, cm)	2.5 (2.2/2.7)	2.6 (2.4/2.9)	**<0.001**	2.6 (2.3/2.9)	2.7 (2.4/3.0)	0.248
Systolic pulmonary artery pulmonary pressure (mmHg)	20.0 (16.8/23.0)	19.0 (16.0/23.5)	0.616	20.0 (17.0/23.0)	21.0 (18.0/23.8)	0.096
E/A quotient	2.5 (1.8/3.5)	3.1 (2.0/4.3)	**0.020**	2.3 (1.7/3.2)	2.8 (1.9/3.8)	**0.005**
E/E' quotient	4.7 (4.0/6.0)	4.9 (4.1/5.6)	0.735	4.6 (3.8/5.6)	4.2 (3.4/5.5)	0.057

Bold values indicate statistical significance $p < 0.05$.

LA area (OR 1.29 [95% CI 1.19–1.39], $p < 0.001$) as well as RA area (OR 1.28 [95% CI 1.19–1.38], $p < 0.001$) were afflicted by AH (Table 3). AH was independently associated with enlarged LA septal-lateral diameter (OR 1.17 [95% CI 1.12–1.23], $p < 0.001$)

LA apico-basal diameter (OR 1.09 [95% CI 1.05–1.12], $p < 0.001$), RA septal-lateral diameter (OR 1.11 [95% CI 1.07–1.16], $p < 0.001$), and RA apico-basal diameter (OR 1.13 [95% CI 1.08–1.17], $p < 0.001$) (Table 3).

In the sex-specific analysis, these associations were confirmed: In female athletes, AH was associated with larger LA area (univariate: OR 1.23 [95% CI 1.10–1.38], $p < 0.001$; multivariate [adjusted for age and BMI]: OR 1.36 [95% CI 1.19–1.55], $p < 0.001$), LA septal-lateral diameter (univariate: OR 1.12 [95% CI 1.05–1.21], $p = 0.001$; multivariate [adjusted for age and BMI]: OR 1.16 [95% CI 1.07–1.25], $p < 0.001$), LA apico-basal diameter (univariate: OR 1.07 [95% CI 1.02–1.12], $p = 0.009$; multivariate [adjusted for age and BMI]: OR 1.10 [95% CI 1.04–1.16], $p = 0{,}001$) as well as associated with larger RA area (univariate: OR 1.22 [95% CI 1.09–1.35], $p < 0.001$; multivariate [adjusted for age and BMI]: OR 1.34 [95% CI 1.18–1.53], $p < 0.001$), RA septal-lateral diameter (univariate: OR 1.08 [95% CI 1.02–1.16], $p = 0.012$; multivariate [adjusted for age and BMI]: OR 1.10 [95% CI 1.03–1.17], $p = 0.005$), and RA apico-basal diameter (univariate: OR 1.13 [95% CI 1.06–1.20], $p < 0.001$; multivariate [adjusted for age and BMI]: OR 1.19 [95% CI 1.11–1.27], $p < 0.001$) and LV mass (univariate: OR 1.04 [95% CI 1.03–1.05], $p < 0.001$; multivariate [adjusted for age and BMI]: OR 1.06 [95% CI 1.04–1.07], $p < 0.001$).

In male athletes, AH was associated with larger LA area (univariate: OR 1.13 [95% CI 1.04–1.23], $p = 0.003$; multivariate [adjusted for age and BMI]: OR 1.26 [95% CI 1.14–1.38], $p < 0.001$), LA septal-lateral diameter (univariate: OR 1.11 [95% CI 1.06–1.18], $p < 0.001$; multivariate [adjusted for age and BMI]: OR 1.19 [95% CI 1.12–1.27], $p < 0.001$), LA apico-basal diameter (univariate:

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OR 1.06 [95% CI 1.02–1.10], $p = 0.005$; multivariate [adjusted for age and BMI]: OR 1.08 [95% CI 1.04–1.12], $p < 0.001$) as well as associated with larger RA area (univariate: OR 1.11 [95% CI 1.03–1.20], $p = 0.005$; multivariate [adjusted for age and BMI]: OR 1.25 [95% CI 1.13–1.37], $p < 0.001$), RA septal-lateral diameter (univariate: OR 1.08 [95% CI 1.03–1.13], $p = 0.001$; multivariate [adjusted for age and BMI]: OR 1.12 [95% CI 1.06–1.18], $p < 0.001$), and RA apico-basal diameter (univariate: OR 1.05 [95% CI 1.00–1.08], $p = 0.042$; multivariate [adjusted for age and BMI]: OR 1.10 [95% CI 1.05–1.15], $p < 0.001$) and LV mass (univariate: OR 1.02 [95% CI 1.01–1.03], $p < 0.001$; multivariate [adjusted for age and BMI]: OR 1.04 [95% CI 1.03–1.05], $p < 0.001$).

LA area (β 0.073 [95% CI 0.041–0.105], $p < 0.001$) as well as RA area (β 0.098 [95% CI 0.069–0.128], $p < 0.001$) increased with athletes' decade of life. Relative heart volume was associated with larger LA area (β 0.57 [95% CI 0.41–0.73], $p < 0.001$) as well as larger RA area (β 0.67 [95% CI 0.50–0.85], $p < 0.001$).

4 | Discussion

The key findings of our study could be summarized as follows:

I. Only 18.3% of the screened athletes had an AH.

II. AH occurs more often in endurance sports and in part also in game sports.

III. Athletes with AH were leaner and had a better athletic performance mirrored by higher relative VO_2 peak.

IV. AH is associated with increased LV muscle mass.

V. All investigated atrial dimensions were larger in athletes with AH than in those without.

VI. The impact of AH on atrial enlargement was similar in both sexes.

High-level and high-intensity training is often related to morphological changes in the heart, including increases in LV chamber size, LV wall thickness, and muscle mass, which is termed as AH [4, 6, 8, 24, 33]. In this context, the AH is defined as a harmonic eccentric enlargement of the heart [7–9]. It is well known that these cardiac adaptations caused by intensive training efforts result in hemodynamic changes accompanied by increases in cardiac output and stroke volume associated with higher maximal oxygen consumption and better athletic performance during exercise [10, 34]. The better athletic performance was markedly demonstrated in our study with a 1.19-fold (OR) higher relative VO_2 peak value in athletes with AH compared to those without. This finding is in accordance with previously published papers demonstrating that total heart volume is a strong and independent predictor of maximal work capacity in both sexes [8, 9, 34]. In addition, our study results once again confirmed that AH develops more often in endurance sports and in part also in game sports compared to other sports entities [2, 8, 9, 35, 36]. Especially, long term intensive endurance training is related to physiologically enlarged heart cavities with a balance in enlargement between the left and right ventricular dimensions in both sexes [34]. However, only 18.3% of the screened athletes in our study presented with an AH. Of course, this frequency differs substantially driven by the examined athletes (more endurance athletes or athletes with predominantly weight and sprint training properties) and due to the used definition for an AH [2, 9, 21, 24, 37].

Previously published studies have shown that depending on the type of exercise performed, two different morphological forms of an AH could be distinguished: First, a strength-trained heart and second, an endurance-trained heart [33]. Adaptations in dynamic and static sports are different. Overall, AH demonstrated normal systolic and diastolic cardiac functions [33]. In accordance with other studies, AH was associated with increased LV muscle mass [33, 38]. Nevertheless, LV mass was in our study only moderately increased by AH (1.05-fold), supporting the assumption of different adaptation forms in AH, since this chosen definition regarding an AH of our study was primarily focused on cardiac enlargement and only subordinately on wall-thickening [2, 6, 8, 21]. It is important that LV ejection fraction was not impaired by AH. These hemodynamic changes due to high-level long-term training primarily induce an enlargement of the cardiac chambers and secondary also of the atria [10, 37]. Although the AH is observed as a harmonic eccentric enlargement of the heart [7–9], the atrial enlargement due to AH is given less attention than the enlargement of the chambers and in particular of the LV [4, 36, 37].

Our large study demonstrated that all investigated atrial dimensions were larger in athletes with AH than in those without and the impact of AH on atrial enlargement was similar in both sexes. The LA area as well as RA area were both 1.3-fold larger in athletes with AH than in those without. These findings are in accordance with smaller previously published studies [1, 39–44]: Hauser et al. performed an echocardiographic study that showed in a very small group of 12 endurance athletes that their left atria was larger than those of sedentary control subjects [39]. Henriksen et al. demonstrated that right and left atrial measurements were distinctly enlarged in 127 male elite endurance athletes compared with earlier studies of normal, active subjects [40]. Another small study including 13 former competing elite sportsmen and 21 men with sedentary life-style revealed that the former athletes had at a mean age of 66 years still larger LA dimensions than controls of similar age [41]. Hoogsteen et al. suggested that longer training experience would be reflected in larger atrial dimension of the LA [42]. Thus, they compared LA dimensions in cyclists with a median age of 17 ± 0.2 years ($n = 66$) with the dimensions of older cyclist of a mean age of 29 ± 2.6 years ($n = 35$) and detected larger LA dimensions in the older athletes [42]. Pelliccia et al. examined LA dimensions in 1777 competitive athletes and identified an enlarged LA dimension ≥40 mm in 20% of these athletes [43]. Atrial fibrillation and other supraventricular tachyarrhythmias were uncommon in these athletes with a prevalence of 1% and therefore, similar to that in the general population, despite the LA enlargement [43]. D' Andrea et al. reported that LA volume index was mildly enlarged in 150 athletes (24.3%) of the 615 included athletes and moderately enlarged in 20 athletes (3.2%), who were all males [1, 44]. A large meta-analysis of 54 studies comprising 7189 elite athletes and 1375 controls confirmed that elite athletes had larger LA dimensions in comparison to controls when evaluated by either LA diameter or LA volume corrected for body surface area [37]. The largest LA diameters were found in endurance athletes [37].

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It is well established that severe mitral and tricuspid valve regurgitations were associated with atrial enlargement [45–47]. In our study only one athlete presented with moderate tricuspid valve regurgitation. All other valve regurgitations were very small and of first degree. Thus, the influence of valve regurgitations on LA and RA diameters can be widely neglected [45, 47]. However, it is important to mention that echocardiography consistently underestimates atrial volumes compared to cardiac magnetic resonance imaging [48–50].

In summary, LA remodeling with LA enlargement in asymptomatic competitive athletes are not uncommon and remodeling has to be regarded as a physiologic adaptation to exercise conditioning, which is up to a certain point widely without adverse clinical consequences [43]. Exercise and physical activity with higher fitness level have positive effects on several risk factors which are related to atherosclerosis [18]. Physical activity and better fitness have benefits for individual health irrespective of age, sex, and presence of comorbidities [18]. Remarkably, there is a dose-effect-relationship between exercise accompanied by better fitness and reduced cardiovascular as well as all-cause mortality compared with sedentary individuals [18].

However, it is well known that patients with cardiomyopathy often present with LA dilation driven by increased LV pressures and atrial myopathy [35] and atrial dilatation is an independent predictor for cardiovascular events and is associated with arterial as well as pulmonary hypertension, heart disease, and poor survival [11–14]. Thus, it is of outstanding interest to distinguish between benign and patholocial atrial enlargement and developing a clear characterization of the benign atrial adaptations in highly trained athletes investigating a large number of athletes in order to understand the nature of these benign adaptations. In this context, it was surprising and evident that even moderate and severe LA enlargement was present in 11% and 4% in a previously published study investigating international-level rowers [51]. In endurance athletes this LA enlargement is strongly correlated with exercise capacity and therefore might be interpreted as a further manifestation of the AH phenotype [35, 51]. In this circumstances, it has to be mentioned that the atrial dilatation driven by athletic training might be asymptomatic in short-term but is associated with increased risk to develop atrial fibrillation in long-term [52–55]. After the sports career the regression of the athletic heart is often incomplete [9, 56]. Especially the LV and LA dimensions due to sport specific dilatation remain enlarged for longer time [9, 57], whereas the muscle thickness of the ventricles mostly normalize after a short period [9, 57]. Studies have shown that the LV myocardium thickness, LV muscle mass and dimensions of the RA decreases substantially after 4 weeks of significant training reduction [57]. After 8 weeks of significant training reduction a substantial decrease of the RV dimension was recognized [57]. In contrast, LA dimensions were not significantly reduced after 8 weeks of significant training reduction [57], which might be one reason for higher prevalence of atrial fibrillation in athletes with AH [58, 59]. Nevertheless, echocardiographic reference values regarding LA and RA dimensions for highly-trained athletes, in addition to established values from the general population, should be formulated for both elite and amateur athletes in order to avoid misdiagnosis of pathological LA enlargement [12, 35].

5 | Conclusions

An AH is accompanied by significant enlargement of the atria as well as increased cardiac muscle mass. Echocardiographic reference values regarding LA and RA dimensions for highly-trained athletes, in addition to established values from the general population, should be defined for both elite and amateur athletes in order to avoid misdiagnosis of pathological LA enlargement.

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Conflicts of interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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