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Effects on Cardiac Dimensions and Peak Oxygen Uptake After Long-Term Deconditioning in Elite Athletes

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ABSTRACT

This longitudinal observational study aimed to determine if ventricular dimensions of the athlete's heart remain balanced and proportional to peak oxygen uptake (VO_2peak) following long-term deconditioning in elite athletes. Fourteen mixed-type male athletes (7 soccer, 7 handball players) were prospectively evaluated with cardiac magnetic resonance imaging and cardiopulmonary exercise testing while active at elite level and after retirement. Athletes were cross-sectionally compared to 14 age-matched controls at baseline and follow-up. Statistical analysis was performed using nonparametric tests. Descriptive statistics are presented as median [Q1, Q3]. Since baseline, athletes reported continued elite sports for 5 [2, 9] years followed by retirement for 12 [7, 14] years. Left ventricular end-diastolic volume (LVEDV) decreased by 17% (261 mL to 222 mL, $p < 0.001$). Right ventricular end-diastolic volume (RVEDV) decreased by 14% (266 mL to 232 mL, $p < 0.001$). Left atrial end-systolic volume decreased by 16% (94 mL to 82 mL, $p < 0.05$). Peak oxygen uptake (VO_2peak) decreased by 17% (3.96 L/min to 3.37 L/min, $p < 0.001$). There were no differences between athletes after retirement compared to controls. LVEDV and RVEDV were balanced in athletes at baseline ($r_s = 0.92$, $p < 0.001$) and follow-up ($r_s = 0.92$, $p < 0.001$). LVEDV and RVEDV indexed to VO_2peak remained unchanged after deconditioning. Exercise-induced cardiac remodeling was reversible after long-term deconditioning in this cohort of elite athletes. LVEDV and RVEDV decreased and remained balanced and proportional to VO_2peak. This study indicates that cardiac adaptations to sports are physiological. However, more research is needed to investigate the reversibility of exercise-induced cardiac remodeling in disciplines with higher demands on endurance performance.

1 | Introduction

Endurance exercise induces cardiac remodeling, known as "the athlete's heart" [1]. This cardiac remodeling is characterized by a balanced dilation of all four chambers of the heart [2–4], and is proportional to peak oxygen uptake (VO_2peak) [2, 5–9]. Cardiac remodeling after deconditioning is, however, less studied. Short-term detraining may reverse cardiac remodeling in athletes, but not always fully normalize mass and volume [10–17]. Long-term studies on deconditioning in athletes are scarce but indicate that left ventricular (LV) hypertrophy and dilation may regress to normal values in most athletes, but not in all [18]. Furthermore, studies of the right ventricle (RV) are few and have only assessed short-term deconditioning [11, 14, 17]. Effects of long-term deconditioning of the atria have, to our knowledge, not previously been studied longitudinally.

Although the heart adapts in a balanced way to endurance exercise [2, 3], it is not known if this dimensional relationship of the ventricles is preserved long-term when the volume of training is decreased. Furthermore, the relationship between cardiac dimensions and VO_2peak has been suggested as a tool to discriminate between physiological remodeling and pathology [7, 8, 19, 20]. However, longitudinal studies to assess this relationship after long-term deconditioning are lacking.

Cardiac magnetic resonance imaging (CMR) is considered the gold standard for assessing cardiac volumes and mass of both ventricles [21], and may together with cardiopulmonary exercise testing (CPET) provide novel information on the effects of long-term deconditioning on cardiac remodeling. Therefore, the aim of this study was to investigate the effects of long-term deconditioning following retirement from elite sports; specific aims were (1) to assess the reversibility of cardiac remodeling, (2) to assess if the physiologically balanced athlete's heart remains balanced, and (3) to assess the relationship between ventricular volumes and VO_2peak.

We hypothesized that exercise-induced cardiac remodeling would be reversible following long-term deconditioning after athletic retirement, with a balance between left and right ventricular volumes. We further hypothesized that ventricular volumes would remain proportional to VO_2peak.

2 | Materials and Methods

2.1 | Study Sample and Design

This longitudinal observational study was approved by the Swedish Ethical Review Authority (registration number 2022-00954-01), conducted according to the Declaration of Helsinki, and complies with the STROBE guidelines for cohort studies [22]. All participants gave a written informed consent. The study was a long-term follow-up of athletes included in a prior study [2]. To be eligible for the follow-up study, the athletes had to be retired from elite sports.

Twenty-nine male athletes (18 soccer and 11 handball players) from local elite teams were included in the baseline study [2]. At follow-up, the aim was to include all 29 participants from the baseline study. However, 14 of the 29 athletes were excluded from the follow-up due to currently living too far away from the including hospital or lack of available contact information ($n = 10$), did not respond to the invitation to participate in the study ($n = 3$), or were deceased (sudden cardiac death, $n = 1$). Thus, 15 athletes were included for follow-up CMR and cardiopulmonary exercise testing (CPET). After examinations, one athlete was excluded due to continued exercise at the same level as during his professional career and did therefore not fit inclusion criteria. Thus, 14 athletes (seven soccer and seven handball players) were included in the analysis. Baseline investigations were performed between February 25, 2007 and November 24, 2007, and follow-up investigations between January 10, 2023 and March 10, 2024. Baseline and follow-up examinations were performed at the Department of Clinical Physiology, Skåne University Hospital Lund, Sweden. All participants underwent CMR followed by CPET on the same day. Participants were asked to abstain from exercise 48 h prior to CMR and from caffeine, tobacco, and food 2 h prior to CMR.

To compare the athletes to individuals that had not completed a full elite sports career with subsequent deconditioning, athletes were cross-sectionally compared to age-matched control groups at baseline and follow-up, $n = 14$ for each control group. Control subjects were recreationally active persons. Mean age difference between athletes at baseline and controls, and between athletes at follow-up and controls was 0.7 ± 0.7 years and 2.0 ± 0.9 years, respectively. All control subjects were free of known cardiovascular disease, were nonsmokers, and did not use medications with known cardiovascular effects.

2.1.1 | Questionnaire on Sports Participation and Recent Physical Activity

At follow-up, athletes filled out a questionnaire regarding general health, sports participation, and training level during their athletic career, and their physical activity during the past 3 months (Data S1 and S2).

2.2 | Cardiac Magnetic Resonance Imaging

Baseline CMR images were acquired using a 1.5-T Philips Intera CV scanner (Philips, Best, The Netherlands). Since baseline measurements, the scanner hardware was updated and follow-up CMR was performed on a Siemens Aera 1.5-T scanner (Siemens Healthineers, Forchheim, Germany). All images were acquired using a cardiac synergy coil with participants in a supine position. At baseline and follow-up, standard steady-state free precession breath-hold sequences with retrospective ECG-triggering were used to acquire cine images in short-axis view covering the ventricles, as well as in 2-, 3- and 4-chamber long-axis views. Typical baseline image parameters were spatial resolution 1.4×1.4 mm, temporal resolution 30 ms, flip angle 60°, echo time 1.4 ms, repetition time 2.8 ms, and 8 mm slice thickness with no slice gap [2]. Typical follow-up image parameters were spatial resolution 1.0×1.0 mm, temporal resolution 41 ms, flip angle 80°, echo time 1.1 ms, repetition time 2.6 ms, and 8 mm slice thickness with no slice gap. Resting heart rate was obtained by ECG during CMR examination.

To assess the prevalence of scar tissue, a dose of 0.15 mmol/kg gadolinium contrast agent (Clariscan, GE Healthcare) was administered during CMR. A blood sample of 2 mL was obtained prior to CMR for determination of hemoglobin, hematocrit, and creatinine levels. Hemoglobin was assessed since low values reduce maximal work capacity and VO_2peak. Hematocrit was used to calculate extracellular volume, and creatinine was analyzed to assess kidney function prior to administration of the contrast agent.

2.2.1 | Volumetric Measures

Cardiac dimensions were measured in cine images by planimetry using the software Segment v4.0 R11044c (Medviso AB, Lund, Sweden) [23]. Ventricular dimensions were measured

in short axis view. Left ventricular mass (LVM), LV end-diastolic volume (LVEDV), LV end-systolic volume (LVESV), RV end-diastolic volume (RVEDV) and RV end-systolic volume (RVESV) were measured according to guidelines [24]. Papillary muscles were excluded from the LVM. Left atrial end-systolic volume (LAESV) was computed by the area-length method from 4- and 2-chamber long axis views [25]. Right atrial end-systolic (RAES) area was delineated in the 4-chamber long axis view. Delineations of the ventricles were done by AB and confirmed in consensus by a second observer with 17 years of experience (KSE). Delineations of the atriums were done by KSE. Cardiac dimensions were normalized to body surface area with the DuBois formula [26]. Further, LVEDV and RVEDV were indexed to VO_2peak (L/min). To assess the balance between the ventricles, the LVEDV/RVEDV ratio was calculated. To further characterize the pattern of deconditioning-induced cardiac remodeling, the LVM/LVEDV ratio was calculated. To classify cardiac dimensions of the athletes as normal or enlarged, baseline and follow-up values were compared to reference ranges [27, 28].

2.3 | Cardiopulmonary Exercise Testing

Incremental exercise testing to exhaustion was performed using an electronically braked cycle ergometer and breath-by-breath gas analysis equipment. Baseline CPET was performed using an Ergomed 940 bike (Siemens, Upplands Väsby, Sweden) and Oxycon Champion (Jaeger, Hochberg, Germany) [2]. Since baseline, the cycle ergometer and gas analysis equipment had been updated, and follow-up CPET was performed using a Lode Corival CPET bike (Lode BV, Groningen, The Netherlands) and Vyntus CPX Metabolic Cart (Vyaire Medical, Mettawa, IL, Unites States).

At baseline CPET, athletes started at workloads of 70–100 Watts (W) depending on their own preferences and need for warm-up period, with an individually determined ramp of 20–30 W per minute [2]. At follow-up CPET for athletes, the test protocol consisted of a 2-min rest on the bicycle, 2–3 min constant load cycling at 30–50 W, an individually determined ramp between 15 and 30 W per minute, and a rest period of 2–3 min sitting on the bicycle after exercise. The control groups started at workloads of 70–100 W with the exception of one individual in the baseline group starting at 130 W and one individual in the follow-up group starting at 120 W. All controls had an individually determined ramp of 15–20 W per minute. The test protocol for the control group was based on age, weight, and self-rated fitness level according to clinical practice. All protocols for athletes and controls were chosen to yield exercise durations of approximately 8–12 min and continued to exhaustion, or when participants could not keep a steady pace > 60 revolutions per minute.

Resting blood pressure was measured in supine rest prior to exercise using a manual sphygmanometer. A 12 lead ECG was acquired before, during, and after exercise. Peak heart rate was defined as the highest 10 s average during the test.

Breath-by-breath gas exchange measurements were averaged in 10 s intervals. Peak oxygen uptake and peakrespiratory exchange ratio (RER) were defined as the highest 30 s average of three consecutive 10 s intervals. Percent of predicted VO_2peak was calculated according to the reference equation by Gläser et al. [29].

2.4 | Statistical Analysis

Nonparametric testing was chosen due to the small sample size and risk of outliers in the data. Descriptive statistics are presented as median and interquartile range [Q1, Q3]. The Wilcoxon matched pairs signed rank test was used to assess differences between athletes at baseline and follow-up. The Mann–Whitney *U* test was used to assess differences between athletes and age-matched controls at baseline and follow-up, and between the control groups. Spearman's rank order correlation was used to assess the relationship between LVEDV and RVEDV, and between LVEDV and VO_2peak. To provide reference values over a large physiological range for the relationship between RVEDV and LVEDV, and LVEDV and VO_2peak, 95% prediction intervals were calculated based on all subjects in the baseline investigation [2]. Statistical analysis was done using GraphPad Prism version 10.2.3 for Windows (GraphPad Software, Boston, Massachusetts, USA, www.graphpad.com). A *p*-value of less than 0.05 was considered statistically significant.

3 | Results

3.1 | General Characteristics

General characteristics for athletes and controls are presented in Table 1. Weight and body mass index (BMI) were higher in athletes at baseline compared to controls (Table 1). There was no difference in general characteristics for athletes at follow-up when compared to controls. From baseline to follow-up, resting diastolic blood pressure increased in athletes by 10 [5, 13] mmHg ($p < 0.01$) and BMI increased by 1.3 [0.7, 2.7] kg/m^2 ($p < 0.01$). Body mass index was higher in follow-up controls compared to baseline controls.

Athletes reported continued elite sports at either national or international level for 5 [2, 9] years after baseline and a subsequent retirement period of 12 [7, 14] years. Questionnaire data on exercise and physical activity are presented in Table 2. All participants reported decreased exercise habits.

3.2 | Cardiac Dimensions and VO_2peak

Left ventricular EDV, RVEDV, and LVM decreased in athletes from baseline to follow-up (Table 3, Figure 1). Furthermore, the ratio of LVM/LVEDV increased (Table 3, Figure 1). The relationship between RVEDV and LVEDV was similar in athletes at baseline and follow-up, and within reference values (Figure 2). Accordingly, there was no difference in LVEDV/RVEDV between athletes at baseline and follow-up. When comparing athletes at baseline to controls, athletes had higher LVM, LVEDV, RVEDV, and LVEDV/RVEDV, but there was no difference in LVM/LVEDV (Table 3). When comparing athletes at follow-up

TABLE 1 | General characteristics in athletes ($n = 14$) and controls at baseline ($n = 14$) and follow-up ($n = 14$).

	Controls baseline	Athletes baseline	Controls follow-up	Athletes follow-up
Age (years)	28 [23, 32]	28 [22, 32]	41 [35, 50]	43 [37, 48]
Height (m)	1.82 [1.76, 1.85]	1.82 [1.78, 1.87]	1.81 [1.79, 1.84]	1.82 [1.78, 1.87]
Weight (kg)	76.5 [67.0, 81.5]	83.0† [79.5, 86.8]	80.5 [74.8, 94.3]	86.5** [81.5, 94.3]
BMI (kg/m^2)	22.7 [21.1, 24.6]	25.0† [24.0, 26.0]	25.2ℙ [22.7, 27.8]	26.4** [24.6, 27.8]
BSA (m^2)	1.96 [1.85, 2.05]	2.05 [1.97, 2.13]	2.01 [1.96, 2.16]	2.05*** [2.03, 2.18]
Resting SBP (mmHg)	130 [120, 135]	130 [120, 130]	130 [125, 136]	128 [120, 140]
Resting DBP (mmHg)[a]	75 [70, 80]	70† [69, 75]	78 [70, 81]	80** [73, 83]
Smokers (*n*)	0	0	0	0

Note: Numbers are median and interquartile range [Q1, Q3]. **$p < 0.01$, ***$p < 0.001$ compared to athletes at baseline by Wilcoxon matched pairs signed rank test.
† < 0.05 compared to baseline controls by Mann–Whitney *U* test. ℙ < 0.05 compared to baseline controls by Mann–Whitney *U* test.
Abbreviations: BMI, body mass index; BSA, body surface area; DBP, diastolic blood pressure; SBP, systolic blood pressure.
[a]Diastolic blood pressure was missing in one athlete at follow-up.

TABLE 2 | Questionnaire data on exercise and physical activity in athletes ($n = 14$) at baseline and follow-up.

	Athletes at baseline	Athletes at follow-up
Endurance training (hours/week)	11.0 [8.2, 13.8] ($n = 13$)	1.5 [0.9, 3.0] ($n = 14$)
Strength training (hours/week)	3.3 [2.5, 4.0] ($n = 10$)	1.3 [0.3, 2.8] ($n = 13$)
Other physical activity (hours/week)	n/a	4.3 [2.6, 7.3] ($n = 14$)

Note: Numbers are median and interquartile range [Q1, Q3].
Abbreviation: *n*, numbers of participants answering the question.

to controls, there was no difference in LVM, LVEDV, RVEDV, LVM/LVEDV, or LVEDV/RVEDV (Table 3). There was no difference when comparing baseline controls to follow-up controls.

Left atrial ESV decreased in athletes from baseline to follow-up, but RAES area did not (Figure 1). Athletes at baseline had higher RAES area compared to controls, but there was no difference in RAESi area (Table 3). Right atrial area could not be delineated in one follow-up control due to an image artifact in the 4-chamber view and was excluded from right atrial analysis. When comparing athletes at follow-up to controls, there was no difference in LAESV or RAES area (Table 3). Further, there was no difference in LAESV or RAES area when comparing baseline controls to follow-up controls.

Cardiac dimensions at baseline and follow-up in relation to reference values are presented in Table 5. At baseline, 12 out of 14 athletes had at least one enlarged cardiac chamber. At follow-up, 3 out of 14 athletes had at least one enlarged cardiac chamber. It can be noted that one athlete had persisting RVEDVi enlargement, while the LVEDVi had returned to normal values (Table 5).

Absolute VO_2peak decreased from baseline to follow-up (Table 4). There was no relationship between LVEDV and VO_2peak in athletes at baseline or at follow-up (Figure 3). Furthermore, there was no difference in LVEDV/VO_2peak or RVEDV/VO_2peak between athletes at baseline and follow-up, or compared to controls (Table 3).

A representative image showing the changes in an athlete's heart at baseline and follow-up is shown in Figure 4.

3.3 | Cardiovascular Symptoms and Disease

Before follow-up examination, out of the 14 athletes included in the final analysis, one athlete reported a history of intermittent symptoms of diffuse chest pain during exercise, and one athlete used a low dose of beta blockers for treatment of mild arrhythmia. The athlete with previous symptoms of chest pain also had signs of myocardial ischemia on exercise-ECG and was referred for further examinations at the cardiology department. In another athlete, results of the CMR examination revealed one previously unknown myocarditis scar.

4 | Discussion

This study shows that exercise-induced cardiac remodeling in soccer and handball players was reversible with long-term deconditioning, although it can be noted that right atrial area was unchanged from baseline to follow-up. Furthermore, ventricular volumes remained balanced to each other and proportional to VO_2peak after deconditioning.

4.1 | Reversibility of Exercise-Induced Cardiac Remodeling

Although exercise-induced ventricular remodeling was reversible in the present study, it cannot be excluded that more marked remodeling may be only partly reversible. Pelliccia et al. [18] observed incomplete regression of LV cavity dimensions following retirement in a group of mainly high-level

TABLE 3 | Cardiac dimensions and function in athletes ($n = 14$) and controls at baseline ($n = 14$) and follow-up ($n = 14$).

	Controls baseline	Athletes baseline	Controls follow-up	Athletes follow-up	Δ Athletes baseline—follow-up (%)
LVM (g)	104 [100, 117]	129† [116, 133]	111 [99, 126]	117** [107, 124]	-10 [−15, −6]
LVMi (g/m^2)	54 [52, 58]	64‡ [59, 68]	55 [48, 60]	54*** [52, 57]	−12 [−17, −7]
LVEDV (mL)	206 [185, 223]	261§ [241, 279]	209 [196, 228]	222*** [200, 239]	−17 [−20, −14]
LVEDVi (mL/m^2)	105 [101, 111]	128§ [125, 134]	105 [93, 110]	102*** [100, 113]	−20 [−21, −14]
RVEDV (mL)	219 [202, 235]	266‡ [253, 282]	224 [201, 266]	232*** [212, 255]	−14 [−16, −10]
RVEDVi (mL/m^2)	112 [105, 118]	132‡ [126, 138]	115 [98, 124]	109*** [104, 116]	−15 [−18, −13]
LVEDV/VO_2peak	61.4 [57.4, 66.7]	65.0 [58.6, 74.4]	62.5 [59.7, 68.7]	65.1 [57.3, 75.9]	0.0 [−5.2, 6.2]
RVEDV/VO_2peak	66.3 [62.2, 72.7]	67.7 [59.4, 74.9]	65.6 [60.9, 73.4]	68.0 [61.8, 80.7]	−0.8 [−4.4, 12.8]
LVEDV/RVEDV	0.93 [0.91, 0.95]	0.98† [0.94, 0.99]	0.94 [0.89, 0.99]	0.95 [0.92, 0.99]	−3 [−7, 2]
LVM/LVEDV	0.52 [0.50, 0.55]	0.50 [0.47, 0.53]	0.55 [0.48, 0.56]	0.53* [0.49, 0.59]	9 [−3, 16]
LVSV (mL)	115 [101, 136]	137† [128, 153]	118 [112, 138]	123** [105, 128]	−15 [−23, −3]
LVSVi (mL/m^2)	60 [53, 63]	69‡ [63, 75]	58 [54, 67]	56*** [54, 61]	−18 [−23, −6]
LVEF (%)	56 [53, 59]	53 [51, 56]	57 [52, 61]	55 [53, 56]	1 [−4, 8]
RVEF (%)	51 [50, 54]	52 [49, 55]	53 [51, 57]	52 [49, 55]	3 [−8, 11]
LAESV (mL)	72 [65, 87]	94† [80, 105]	79 [60, 94]	82* [68, 89]	−16 [−23, −6]
LAESVi(mL/m^2)	37 [33, 44]	46† [40, 53]	40 [30, 43]	38* [34, 42]	−18 [−28, −7]
RAES area (cm^2)	24 [22, 29]	29† [25, 29]	27 [23, 30]	28 [26, 33]	0 [−7, 6]
RAESi area (cm^2/m^2)	12 [11, 13]	13 [12, 14]	13 [11, 15]	14 [12, 15]	6 [−8, 11]
CO (L/min)	6.7 [6.2, 7.8]	7.9 [6.8, 8.5]	6.8 [6.1, 8.4]	6.6 [6.0, 8.1]	−9.4 [−19, 5.1]
HR rest (1/min)	59 [55, 69]	56 [51, 58]	61 [55, 70]	58 [54, 62]	2 [−2, 11]

Note: Numbers are median and interquartile range [Q1, Q3]. *$p < 0.05$, **$p < 0,01$, ***$p < 0.001$ compared to athletes at baseline by Wilcoxon matched pairs signed rank test. † < 0.05, ‡ < 0.01, § < 0.001 compared to baseline controls by Mann–Whitney *U* test. Controls are age-matched to athletes at baseline and follow-up.
Abbreviations: Δ, change from baseline to follow up; CO, cardiac output; EF, ejection fraction; HR, heart rate; i, indexed to BSA, left atrial end-diastolic volume; LAESV, left atrial end-systolic volume; LVEDV, left ventricular end-diastolic volume; LVM, left ventricular mass; ns, nonsignificant; RAES, right atrial end-systolic; RVEDV, right ventricular end-diastolic volume; SV, stroke volume.

rowers, canoeists, and cyclists, possibly representing an irreversible effect of endurance training. However, the authors noted that the persisting LV dilation in these athletes might partly be explained by continued recreational activity and increased body mass [18]. Furthermore, the differences in results between the current study and the results by Pelliccia et al. may also be explained by shorter durations of detraining in the study by Pelliccia et al. [18], and that the athletes in the current study were mixed-type athletes as opposed to purely endurance-based athletes with potentially more extensive cardiac remodeling.

Due to the long follow-up in the current study, it is important to recognize the age-related effects on cardiac remodeling. For each decade of life, there is a decrease of approximately 4%–5% in LVEDVi and RVEDVi, and 2% in LVMi [27]. Since the time between baseline and follow-up in the present study was about one and a half decades, it is expected that a substantial part of the decrease in cardiac dimensions can be related to age. However, it is not clear if the age-dependent decrease in cardiac dimensions in the general population is due to lower physical activity and thus fitness, or to some other physiological effect of aging.

At baseline in the current study, there were fewer athletes with enlarged atria than enlarged ventricles (Table 5), suggesting a less pronounced remodeling of the atria compared to the ventricles. Although RAES area was larger in athletes at baseline compared to controls, there was no difference in RAESi area. After deconditioning, LAESV decreased while RAES and RAESi area remained unchanged. Because LAESV decreases slightly with age [27], a portion of the decrease could be attributed to age. The unchanged RAESi area was likely due to a lack of substantial exercise-induced remodeling of the right atrium at baseline, and thus, no deconditioning-induced remodeling could take place. Further, deconditioning-induced remodeling of the right atrium could have been masked by the small, age-dependent increase in atrial volume seen in the general population [27]. Indeed, RAES area increased in several participants. However, it should be noted that RAES area was determined from only one long-axis view and therefore was highly dependent on the acquisition

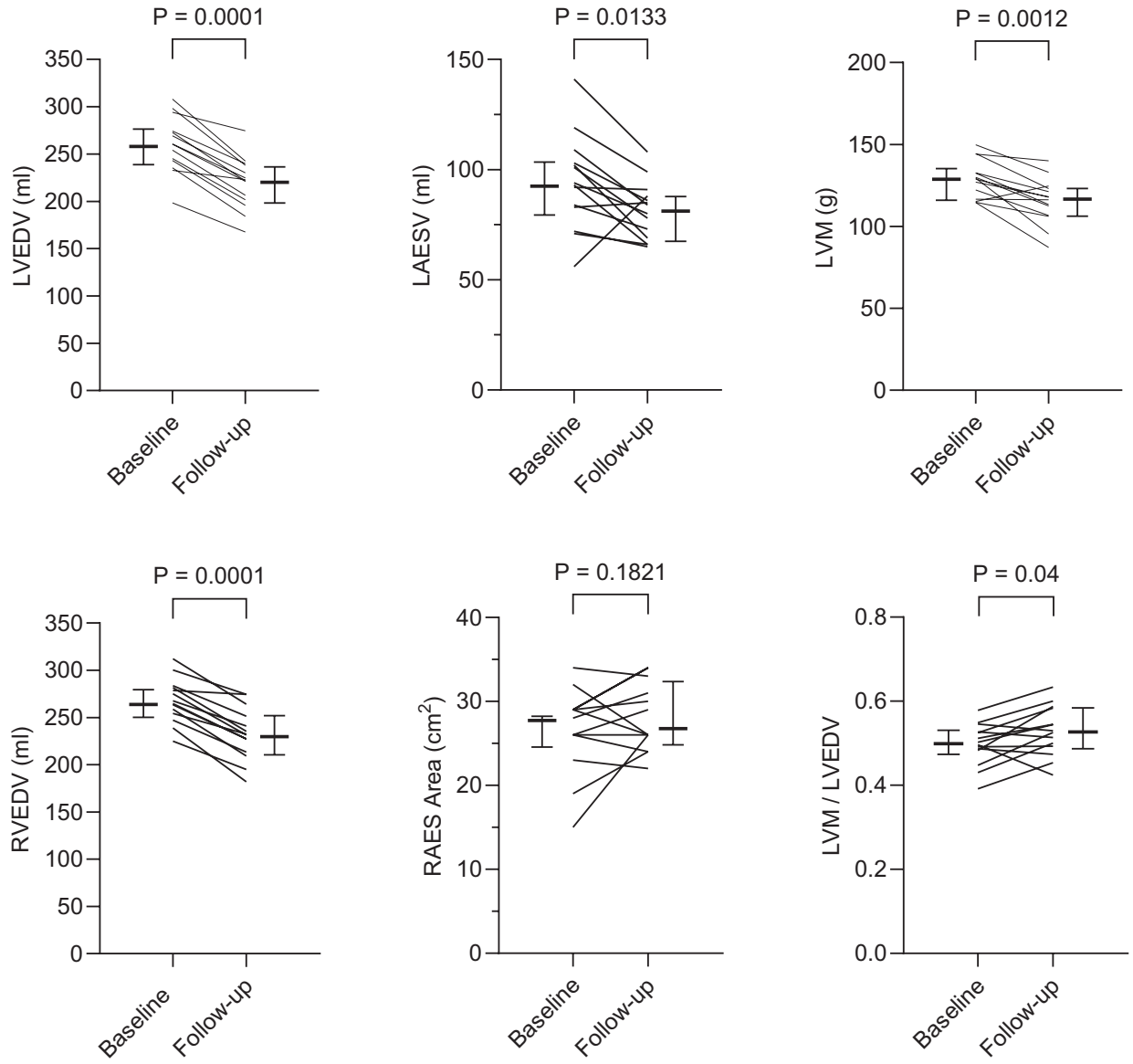

FIGURE 1 | Change in left and right ventricular end-diastolic volume (LVEDV, RVEDV), left atrial end-systolic volume (LAESV), right atrial end-systolic (RAES) area, left ventricular mass (LVM), and LVM/LVEDV in athletes from baseline to follow-up. LVEDV, RVEDV, LAESV and LVM decreased following long-term deconditioning, while LVM/LVEDV increased. Horizontal lines with brackets are median and interquartile range [Q1, Q3]. *p*-values indicate Wilcoxon matched pairs signed rank test between baseline and follow-up.

angle. Future studies should assess right atrial volumes from for example, short-axis stacks to better describe the effects of deconditioning on the right atrium.

4.2 | Changes in Left Ventricular Mass Versus Volume

In the present study, LVEDV decreased more than LVM, resulting in an increased LVM/LVEDV ratio. This can be explained by more extensive remodeling of ventricular volumes than that of mass at baseline, as seen in comparison to reference values [27]. There were larger absolute and relative decreases in reported endurance training compared to strength training from baseline to follow-up. Since the increased preload associated with endurance exercise is the primary stimulus for LV dilation [30–32], this may have contributed to a larger decrease of LVEDV than LVM in the present study. Of note, there is a slow decrease in LVMi with increasing age [27], and a small portion of the decrease in LVM in the present study might be attributed to the effect of aging. In contrast, an increase in LVM from early adulthood until midlife has been observed by echocardiography, although less pronounced when indexing for body surface area [33]. However, LVM obtained by echocardiography is not directly comparable to CMR [34, 35]. Finally, the increase in BMI from baseline to follow-up might thus have mitigated decreases in LVM [36].

4.3 | Balance Between LVEDV and RVEDV

Both LV and RV EDV decreased to a similar extent and remained balanced following long-term deconditioning. This indicates a healthy, physiological adaptation to exercise in this study sample

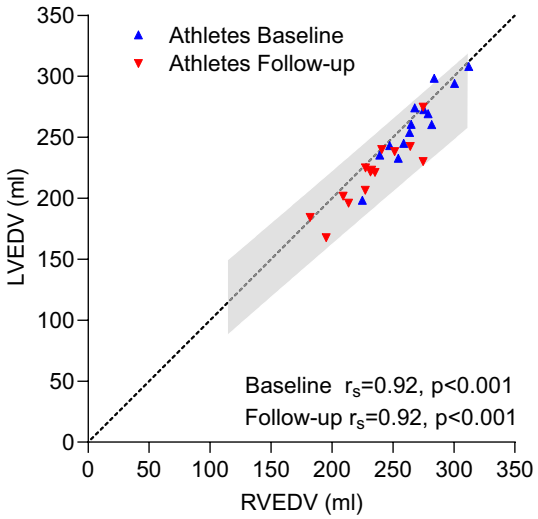

FIGURE 2 | The relationship between the left and right end-diastolic volume (LVEDV, RVEDV) in athletes at baseline and follow-up. The balanced dimensions of the left and right ventricles observed in the enlarged athlete's heart are preserved following long-term deconditioning. Blue triangles are the subset of athletes at baseline that were then included for follow-up examination. Red triangles are the same athletes at follow-up. The dashed line is the line of identity, and the shaded gray area is the 95% prediction interval provided for reference, based on all subjects in the baseline study [2]. Used with permission. r_s: Spearman's correlation coefficient.

of professional soccer and handball players, and that long-term effects on RV remodeling were similar to that of the LV in this group of athletes. However, studies of athletes with higher loads of endurance training may show different findings in the future. With increasing exercise intensity, the RV has been shown to be subjected to higher relative loads compared to the LV [37], which could make the RV susceptible to disproportional remodeling [38]. Such adverse remodeling has been proposed as a link to exercise-induced arrhythmogenic right ventricular cardiomyopathy [38]. It can be noted that in the current study sample, RVEDV decreased proportionally to LVEDV and to volumes similar to the control group in all but one athlete. The athlete, a soccer player, had persisting RVEDVi enlargement consistent with major Task Force criteria for arrhythmogenic right ventricular cardiomyopathy [39], but was otherwise healthy and with no apparent cardiovascular disease. This athlete reported 13 years of elite sports followed by a retirement of 3 years after baseline and until follow-up. He reported no endurance training during the 3 years of retirement. Whether the persisting enlargement of RVEDVi constitutes incipient pathological remodeling with predisposition for future risk remains unknown.

4.4 | Cardiac Remodeling in Relation to Peak Oxygen Uptake

Left ventricular EDV and VO_2peak remained proportional following long-term deconditioning. This is in line with previous cross-sectional research, showing cardiac size to be a strong predictor of VO_2peak from untrained individuals to elite athletes [2, 7–9]. Although LVEDV remained proportional to VO_2peak within the reference range, there was no significant relationship at baseline and follow-up, similar to results by Ekblom & Hermansen [40]. This is likely explained by the relatively narrow range of observed LVEDV values in relation to the VO_2peak variation for a given LVEDV. Hence, when investigating small or homogeneous groups, it is important to recognize that they may represent only a small part of the larger physiological range.

Previous studies have shown a strong relationship between VO_2peak and heart volume in healthy subjects [2, 7–9], while this relationship is lost in heart failure patients [7, 8]. This finding has been suggested as a tool to differentiate between physiological and pathological remodeling in athletes [19, 20], as an athlete with a given heart volume should be able to perform a

TABLE 4 | Cardiopulmonary exercise testing variables in athletes ($n = 14$) and controls at baseline ($n = 14$) and follow-up ($n = 14$).

	Controls baseline	Athletes baseline	Controls follow-up	Athletes follow-up	Δ Athletes baseline—follow-up (%)
VO_2peak (L/min)	3.49 [2.88, 3.80]	3.96‡ [3.78, 4.19]	3.35 [2.92, 3.86]	3.37*** [3.01, 3.68]	−17 [−24, −8]
VO_2peak (mL/kg/min)	44.3 [38.8, 49.2]	49.3 [45.0, 50.4]	40.6 [37.2, 44.8]	37.5*** [36.3, 39.3]	−21 [−28, −15]
VO_2peak (% predicted)	117 [103, 127]	133‡ [122, 144]	124 [110, 132]	119*** [108, 131]	−11 [−19, −4]
HR peak (1/min)	189 [184, 195]	175§ [170, 182]	181ℙ [175, 191]	181** [171, 184]	3 [0, 6]
RER peak	1.32 [1.27, 1.35]	1.33 [1.28, 1.40]	1.31 [1.25, 1.33]	1.24* [1.21, 1.29]	−6 [−9, −2]
Peak work rate (W)	298 [240, 333]	353‡ [318, 375]	288 [253, 315]	310** [272, 323]	−12 [−20, −3]
TTE (min)	10.9 [8.4, 11.9]	9.3 [8.2, 10.8]	10.7 [10.2, 12.8]	11.3* [9.3, 13.3]	21 [−2, 52]

Note: Numbers are median and interquartile range [Q1, Q3]. *$p < 0.05$, **$p < 0,01$, ***$p < 0.001$ compared to athletes at baseline by Wilcoxon matched pairs signed rank test. †< 0.05, ‡< 0.01, §< 0.001 compared to baseline controls by Mann–Whitney U test. ℙ< 0.05 compared to baseline controls by Mann–Whitney U test. Controls are age-matched to athletes at baseline and follow-up. % Predicted VO_2peak according to reference equation [29].
Abbreviations: Δ, change from baseline to follow up; HR peak, peak heart rate; RER, respiratory exchange ratio; TTE, time to exhaustion; VO_2peak, peak oxygen uptake.

corresponding VO_2peak. To further increase our understanding of the long-term effects of elite sports, future studies of deconditioning in athletes should be recommended to include VO_2peak indexed for total heart volume [2] or LVEDV indexed for VO_2peak [8].

4.5 | Limitations

Due to the small sample size and only two sport disciplines included, care should be taken when generalizing the results to a broader athletic population. Because only 15 out of 29 elite athletes from the baseline examination were examined at follow-up, there is a risk of selection bias. However, because the most common cause of exclusion from follow-up examination was missing contact information or living too far from the including hospital, it is unlikely that this resulted in a systematic bias. Although scanner hardware was updated between baseline and follow-up examinations, it is unlikely that this influenced CMR findings because standard steady state free precession breath hold sequences were used on both occasions. Furthermore, only male athletes were included, and potential sex-dependent effects of long-term detraining were not determined. Participants were heterogeneous in age and variations in effects of long-term detraining across an individual's life span were not studied. The comparison to the control group was cross-sectional and therefore the longitudinal effects of deconditioning and aging on cardiac remodeling could not be separated. Reference data indicate that LVEDVi decreases approximately 4% per decade in men [27], but these data do not separate potential effects of aging per se, to deconditioning as a result of decreased physical activity with increasing age. This underscores the importance of relating cardiac dimensions to VO_2peak when studying cardiac

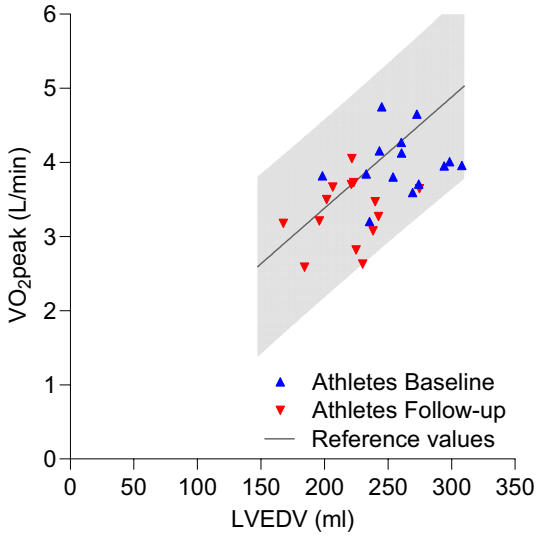

FIGURE 3 | Visualization of the relationship between left ventricular end-diastolic volume (LVEDV) and peak oxygen uptake (VO_2peak) in athletes at baseline and follow-up. Athletes shift downward within the physiological range from baseline to follow-up and VO_2peak remains proportional to LVEDV following long-term deconditioning. Blue triangles are the subset of athletes at baseline that were then included for follow-up examination. Red triangles are the same athletes at follow-up. The gray area is the 95% prediction interval provided for reference, based on all subjects in the baseline study [2], used with permission, and the solid line is the regression line.

TABLE 5 | Cardiac dimensions in relation to reference ranges [27, 28] in athletes ($n = 14$) at baseline and follow-up.

	Baseline (enlarged/normal)	Follow-up (enlarged/normal)
LVMi (g/m^2)	3/11	1/13
LVEDVi (mL/m^2)	12/2	2/12
RVEDVi (mL/m^2)	10/4	1/13
LAESVi (mL/m^2)	2/12	0/14
RAESi area (cm^2/m^2)	1/13	1/13

Abbreviations: i, indexed to body surface area; LAESV, left atrial end-systolic volume; LVEDV, left ventricular end-diastolic volume; LVM, left ventricular mass; RAES, right atrial end-systolic; RVEDV, right ventricular end-diastolic volume.

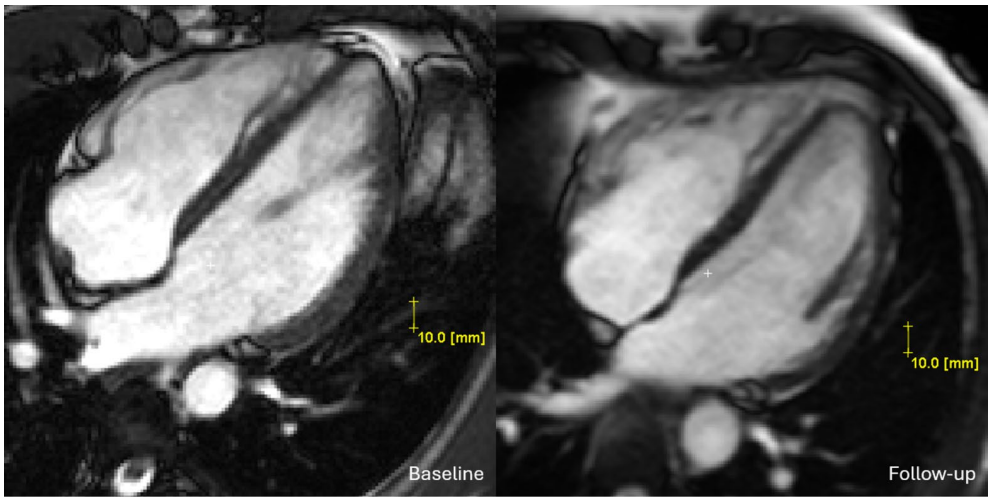

FIGURE 4 | Long-axis images of the heart in an athlete at baseline and follow-up. This figure illustrates the decrease in cardiac dimensions with deconditioning.

remodeling in both long- and short-term studies. Nonetheless, it cannot be excluded that some of the decrease in LV volumes in the present study could be attributed to aging, and the longitudinal effect of factors other than detraining on cardiac dimension and VO_2peak could not be fully appreciated. Finally, it is known that retrospective self-reported physical activity has relatively low accuracy. It is therefore not possible to draw conclusions of dose–response relationships between specific decreases in exercise load and deconditioning-induced cardiac remodeling.

5 | Perspective

Exercise-induced cardiac remodeling was reversible in this cohort of elite soccer- and handball players. Furthermore, the left and right ventricles remained balanced after long-term deconditioning, and ventricular end-diastolic volumes remained proportional to peak oxygen uptake. These findings may aid in determining types of elite sport associated with reversible cardiac remodeling, even after many years of elite sports. Although this study supports the current notion that cardiac adaptations to sports are physiological, more research is needed to investigate the upper limit of elite sports participation associated with the reversibility of exercise-induced cardiac remodeling, specifically in disciplines with higher demands on endurance performance.

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Ethics Statement

This longitudinal observational study was approved by the Swedish Ethical Review Authority (registration number 2022-00954-01) and conducted according to the Declaration of Helsinki.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Full data cannot be shared publicly because of the small sample size and the risk of identifying individual subjects. Data are available from the Cardiac MR Group in Lund, Lund University, Sweden, for researchers who meet the criteria for access to confidential data and after additional consent from the research participants. For data access requests, please contact the Cardiac MR Group in Lund via email: cmrlund@med.lu.se.

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Supporting Information

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