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## The “athlete’s heart” features in amateur male marathon runners

Running title: The “athlete’s heart” features in amateur male marathon runners

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### Abstract

**Background:** Training on a professional level can lead to cardiac structural adaptations called the “athlete’s heart”. As marathon participation requires intense physical preparation, the question arises whether the features of “athlete’s heart” can also develop in recreational runners.

**Methods:** The study included 34 males (mean age  $40 \pm 8$  years) who underwent physical examination, a cardiopulmonary exercise test and echocardiographic examination (ECHO) before a marathon. ECHO results were compared with the sedentary control group, reference

values for an adult male population and those for highly-trained athletes. Runners with abnormalities revealed by ECHO were referred for cardiac magnetic resonance imaging (CMR).

**Results:** The mean training distance was  $56.5 \pm 19.7$  km/week, peak oxygen uptake was  $53.7 \pm 6.9$  mL/kg/min and the marathon finishing time was  $3.7 \pm 0.4$  h. Compared to sedentary controls, amateur athletes presented larger atria, increased left ventricular (LV) wall thickness, larger LV mass and basal right ventricular (RV) inflow diameter ( $p < 0.05$ ). When compared with ranges for the general adult population, 56% of participants showed increased left atrial volume, indexed to body surface area (LAVI), 56% right atrial area and interventricular septum thickness, while 47% had enlarged RV proximal outflow tract diameter. In 50% of cases, LAVI exceeded values reported for highly-trained athletes. Due to ECHO abnormalities, CMR was performed in 6 participants, which revealed hypertrophic cardiomyopathy in 1 runner.

**Conclusions:** “Athlete’s heart” features occur in amateur marathon runners. In this group, ECHO reference values for highly-trained elite athletes should be considered, rather than those for the general population and even then LAVI can exceed the upper normal value.

**Key words:** echocardiography, cardiac magnetic resonance, athlete’s heart, marathon runners; sport cardiology, hypertrophic cardiomyopathy

## **Introduction**

Regular and moderate physical activity has a positive effect in humans, however the “upper dose” of beneficial endurance exercise has not been determined [1, 2]. Participation in sport events like marathon runs has recently become very popular. The characteristics of marathon runners is evolving, with a growing percentage of non-elite amateur runners who are often middle-aged [3]. Long-term endurance training on a professional level can lead to multiple structural adaptations, called the “athlete’s heart” [4]. As marathon participation requires intense physical preparation, the question arises whether the features of “athlete’s heart” can be present in recreational runners. And if so, which echocardiographic criteria

should be applied in this group: those for the general adult population or those for highly trained elite athletes.

## **Methods**

### ***Study participants and study protocol***

Male amateur marathon runners who planned to attend the 2<sup>nd</sup> PZU Marathon in Gdansk, Poland were recruited by invitation to local running clubs. Volunteers were informed about the purpose and plan of the study and gave written consent. All participants were questioned about medical history and those with chronic diseases, or at age < 20, or > 55 years were not eligible. Two weeks before the marathon run, each of the participants underwent physical examination, treadmill cardiopulmonary exercise test (CPET) and echocardiographic examination (ECHO).

Fifteen sedentary males matched with age, body mass index (BMI) and body surface area (BSA) with marathon runners constituted the control group for ECHO. They were healthy men without any history of practicing endurance exercise. In the next step, data obtained in marathon runners were compared with reference values for cardiac chambers in male adults, provided by the American Society of Echocardiography and European Association of Cardiovascular Imaging [5]. Subsequently, results were also compared with reference values for elite athletes: 1) right chamber's dimensions with Normative Reference Values of Right Heart in Competitive Athletes [6], and 2) left chambers diameters with values reported in studies on populations of elite athletes [4, 6–8], as to our knowledge there is no single paper providing all normative reference values for the left heart in this group.

The study protocol set up that participants with abnormalities revealed by ECHO were referred for cardiac magnetic resonance imaging (CMR). These included: increased interventricular septum diameter ( $\geq 13$  mm), abnormal left ventricular (LV) contractility (ejection fraction < 52% or abnormal global longitudinal strain > -18.9%), abnormal right ventricular (RV) systolic function (tricuspid annular plane systolic excursion < 17 mm, RV strain of > -20% or spectral tissue Doppler derived tricuspid lateral annulus peak systolic velocity < 9.5 cm/s) [5, 9, 10]. The study protocol was accepted by Independent Bioethics Commission for Research of the Medical University of Gdansk (NKBBN/104/2016).

### ***Cardiopulmonary exercise test***

Cardiopulmonary exercise test was performed on the treadmill (H/P/Cosmos Saturn treadmill) using the Bruce protocol. First stage started at 2.7 km/h and at 10% gradient, then the speed and incline were increased in 3 min intervals. Jaeger OxyconPro equipment with Jlab Manager V5.32.0 software was used to measure the oxygen intake ( $\text{VO}_2$ ), carbon dioxide output ( $\text{VCO}_2$ ), minute ventilation (VE), expiratory gas concentrations throughout the respiratory cycle on a breath-by-breath basis. The peak oxygen intake ( $\text{VO}_{2\text{peak}}$ ) was calculated as the highest volume averaged over 10 s at maximal endurance. The anaerobic threshold (AT) was calculated with the V-slope method and was corrected by the ventilator equivalent method.

### ***Echocardiography***

Transthoracic ECHO was performed using Vivid E9 (General Electric Medical Health) in marathon runners and sedentary controls. ECHO measurements were carried out according to the recommendations of the American Society of Echocardiography and European Association of Cardiovascular Imaging [5]. Left ventricular measurements: LV end-diastolic dimension (LV ED), LV end-systolic dimension (LV ES), diastolic interventricular septum (IVS) and posterior wall (LV PW) thickness, left atrial (LA) anteroposterior (AP) dimension and proximal RV outflow diameter (RVOT prox) were performed in the parasternal long-axis view. The LV end-diastolic (LV EDV) and LV end-systolic (LV ESV) volumes were measured with the biplane method of discs summation (the modified Simpson's rule) and then LV ejection fraction (LV EF) was calculated. The 2-dimensional (2D) speckle-tracking LV global longitudinal peak strain (LV GLS) measurements were obtained from 2-, 3-, and 4-chamber apical views and were averaged. The LV mass was assessed by the area-length method and was then indexed to BSA. In the end systole, the LA volume was indexed to BSA (LAVI) and was calculated by the area-length technique from apical 2- and 4-chamber views, whereas the right atrial (RA) area was measured in the apical 4-chamber view. The basal RV inflow diameter (RVd) and the 2D speckle-tracking-derived RV strain were obtained in the RV-focused apical 4-chamber view. The RV systolic function was assessed by measuring the tricuspid annular plane systolic excursion (TAPSE) in the M-mode

and spectral tissue Doppler-derived tricuspid lateral annulus systolic peak velocity (S'RV). The offline analyses of data were carried out using commercially available software — EchoPack 201 (General Electric).

### ***Cardiac magnetic resonance***

Cardiac magnetic resonance examinations were performed with a 1.5 Tesla scanner (Magnetom Aera, Siemens Healthcare) with an 18-channel phased-array receiver coil with repeated breath-holds, according to protocol [11]. Segmented steady-state free-precession sequence was used to acquire cine images of the heart in 2-, 3-, and 4-chamber views, as well as in short-axis views to obtain a stack of contiguous short-axis slices which include the entire LV and RV having a slice thickness of 8 mm with 2 mm gaps. In the majority of cases the parallel acquisition technique with acceleration factor of 2 was used. Late gadolinium enhancement (LGE) was assessed 7–15 min post administration of gadolinium-based contrast agent at a dose of 0.1 mmol/kg body mass, with an inversion recovery spoiled gradient echo sequence (single slice per breath hold). Inversion time was repeatedly optimized to null normal myocardium. A short-axis stack identical to that performed in cine steady-state free precession as well as 2-, 3-, and 4-chamber long axis images (slice thickness of 8 mm with in-plane resolution typically ca  $1.5 \times 1.5$  mm) were acquired in each individual. Data was analyzed using commercially available software by an experienced observer.

### ***Statistical analysis***

Continuous variables are presented as mean  $\pm$  standard deviations (SD) or median and range. The Shapiro-Wilk test was used to estimate the distribution. A comparison of the amateur marathon runners and controls was performed by the Student t-test for independent samples or the Mann–Whitney U test where appropriate. A p value  $< 0.05$  was considered statistically significant. The data were analyzed using Statistica 13 software (Statsoft Poland).

## **Results**

Thirty-four amateur marathon runners were included in the study. Results of electrocardiographic examination in these subjects have recently been published [12]. Table 1 shows data on training habits and CPET. Table 2 presents features of marathon runners in

comparison with sedentary controls. There were no significant differences in age, weight, height, BSA and BMI between amateur runners and controls ( $p > 0.05$ ). All participants were healthy men of Caucasian race.

Data on ECHO parameters obtained in the amateur marathon runners studied and sedentary controls are presented in Table 2. Compared to controls, amateur athletes had larger atria, increased LV wall thickness, larger LV mass and RVd ( $p < 0.05$ ). There were no differences regarding other ECHO parameters. A comparison of parameters obtained from amateur marathon runners with reference values for the general male adult population and for professional athletes is presented in Table 3. It shows the percentage of amateur athletes exceeding the upper reference value for the adult population (URP) and the upper value range for highly-trained athletes (URA). The IVS population norm of 10 mm was exceeded in 19 (56%) runners and in 3 (9%) participants it was  $\geq 13$  mm (13 mm, 14.7 mm and 17 mm). The LV PW was  $\geq 13$  mm in 2 runners (13.6 mm and 14 mm). One subject was diagnosed with hypertrophic cardiomyopathy (HCM). All participants with LV enlargement (as indicated by LV EDV) showed IVS  $> 10$  mm, but only 3 runners with IVS  $> 10$  mm presented with an enlarged LV. One runner had mildly abnormal LV EF of 51%. The LV GLS was abnormal in 4 (12%) runners ( $> -18.9\%$ ) whereas the RV strain was altered in 6 (18%) amateurs ( $> -20\%$ ). The median S'RV was 14 cm/s (range 9–19 cm/s). In 1 participant the abnormal S'RV below 9.5 cm/s was found, whereas TAPSE was within normal ranges.

There was a negative correlation between the achieved marathon times and training distance ( $r = -0.4$ ,  $p < 0.05$ ) or oxygen uptake at the anaerobic threshold ( $\text{VO}_2\text{AT}$ ) ( $r = -0.38$ ,  $p < 0.05$ ). The training distance [km/week] correlated with LAVI ( $r = 0.44$ ,  $p < 0.05$ ). The RA area correlated with LAVI ( $r = 0.46$ ,  $p < 0.05$ ) and RVd ( $r = 0.49$ ,  $p < 0.05$ ).

The CMR was performed in 6 (18%) amateur marathon runners. The reasons for the CMR referral are presented in Table 4; all showed several abnormalities in ECHO and the most frequent was increased IVS. Results from CMR imaging are presented in Tables 4 and 5. The major abnormality was enlarged volume and depressed RV function. The RV ESV was increased in all runners and RV EDV in 1 individual. All those 6 participants presented reduced RV EF with a median of 46%. The LV was enlarged in 3 subjects (LV ESV was increased in all of them, while LV EDV in 1). In 3 participants LV EF was slightly below the lower reference limit. In 1 participant CMR imaging confirmed HCM with asymmetric hypertrophy (LVH) of LV segments: basal infero-septum and basal antero-septum with

maximum wall thickness of 17 mm. In addition, the LGE revealed myocardial fibrosis within hypertrophic ventricular segments. LGE was present only in this participant. In addition, in 1 individual CMR raised suspicion of atrial septum defect of 6 mm in diameter.

## **Discussion**

The study group represented a non-elite runner population. However, the reported finishing times vary between studies, the average time of the marathon run among amateur participants oscillates around 3.5 h, similar to the present group [13]. Professional athletes cover this distance within 2.3 h [14]. Regarding training volumes, the weekly distance in highly-trained elite and national-class runners is  $145.3 \pm 25.6$  km [14], whereas in the current group it was  $56.5 \pm 19.7$  km, comparable to other studies on amateurs [15]. The mean  $VO_{2peak}$  was similar to those previously reported among runners with comparable running performance [13]. The more time subjects spent on training the better marathon time they achieved. The  $VO_{2AT}$  appeared to be prognostic for obtained outcome at the finishing-line.

Training-induced changes in cardiac morphology, named the “athlete’s heart” are a common finding among professional athletes. Recurrent exercise-induced pressure or volume overload causes cardiac remodeling with increased chamber dimensions, LV mass and LV wall thickness [4, 7]. Physiological in elite athletes, these modifications in the general adult population are considered pathological. Type of exercise, its intensity, duration of training, age, sex, race, BSA and other unrecognized individual factors can influence the occurrence of “athlete’s heart” [4, 16]. It can appear even after 8 weeks of intense training and may disappear after sport termination [17, 18]. The question arises, whether the “athlete’s heart” features also develop in middle-aged recreational runners. In the present group of amateur marathon runners, the cardiac dimensions assessed by ECHO frequently exceeded those obtained in sedentary controls, as well as reference ranges for the general adult population. Atrial enlargement was one of the most common findings and both atria were significantly larger in comparison to sedentary controls. Due to significant hemodynamic overload and increased atrial pressure during intense exercise, larger LA in professional athletes were expected with volumes on average of  $7.0 \text{ mL/m}^2$  greater than those met in the general population [19, 20]. Noteworthy, in the present study was that LAVI in amateur runners exceeded not only upper value ranges for the general population, but in half of them upper ranges were also reported for highly trained athletes. The more time runners spent on training the more their LA was enlarged, which was demonstrated by positive correlation between



LAVI and weekly training distance. More than half of the current group had an enlarged RA area and changes in RA correlated with those of LA. Possibly, atria of amateur runners are especially prone to enlargement and this magnification may not happen without consequences — as we know that exercise-induced atrial remodeling increases the risk of atrial fibrillation [21]. The next important finding in the amateur runners studied was the LV thickening, which was significant in comparison with sedentary controls. The measurement of the wall thickness is especially important in differential diagnosis between physiological exercise-induced LVH and HCM. HCM remains one of the most common causes of sudden cardiac death in elite athletes and individuals with this diagnosis are advised to discontinue competitive sport activity [22, 23]. The LV wall of 13–14 mm is the grey zone in differential diagnosis among athletes and HCM patients, whereas  $\geq 15$  mm or evident asymmetric hypertrophy suggests pathology [16, 23]. The prevalence of LV wall thickness  $\geq 13$  mm was reported as 1.7% among athletes, however training-related IVS can (rarely) reach even 16 mm [7]. In the group studied the IVS of  $\geq 13$  mm was more frequent. Two cases raised suspicion of HCM, and was later confirmed in one individual. The recognition of HCM never relies on a single ECHO parameter and the assessment of diastolic function may also be helpful [16, 23]. The exercise-related LV thickening usually corresponds with LV enlargement, whereas in HCM patients the LV diastolic volume is rather small [16]. In the current study, LV dilation was rarely encountered and IVS thickening was not observed parallel to LV enlargement. What can be used to differentiate “athlete’s heart” with cardiomyopathies is the speckle tracking-derived LV GLS assessment, which enables detection of systolic abnormalities much earlier than the LV EF deteriorates [23, 24]. The sedentary population norms of LV GLS vary between studies, according to meta-analysis it should not be  $> -18.9\%$ . Nevertheless, one should take into account the software that was used — in EchoPAC from GE the lower limit of normal range for LV GLS is  $-18\%$  [5, 9]. Noteworthy, LV GLS normal values for athletes resemble those for the general population and abnormal LV GLS (especially when  $> -15\%$ ) in athletes should not be regarded as cardiac training adaptation, but rather as pathological and should prompt further diagnostics [24].

As RV remodeling is one of the most characteristic features of “athlete’s heart” it is necessary to apply special normative reference values for RV evaluation in elite athletes [6]. In healthy sportsmen, the size of RV is increased but its function is preserved, although according to recent meta-analysis athletes present lower RV EF in CMR than the general population (with mean of 52%) [25]. The RV enlargement is also typical for arrhythmogenic

RV cardiomyopathy (ARVC), which should be ruled out in differential diagnosis [26]. In the present study nearly half of the amateur runners showed enlarged RV (RVOT prox). Standard 2D echocardiographic evaluation of RV remains challenging, because of its complicated structure and lack of a single parameter that would precisely describe RV systolic function [27]. The assessment of RV is very important, as RV, may be “the Achilles heel” of the competing heart. In the current study 6 participants presented with slightly reduced RV systolic function, as indicated by abnormal RV strain and also decreased S’RV in one subject. It has also been shown previously in elite athletes, that adaptation for training means better RV deformation and that there is a correlation between training experience and RV strain; the more years of training — the more negative the RV strain values can be [28].

ECHO remains the main tool in the recognition of the “athlete’s heart” and in differential diagnosis with cardiomyopathies. Nevertheless, CMR provides the most accurate estimation of both ventricles including the prevalence of myocardial fibrosis [29]. The presence of LGE in hypertrophic segments may suggest HCM, but it does not always mean a certain diagnosis [23, 30]. Generally, in elite athletes, CMR mainly demonstrates the biventricular enlargement of volumes: EDV and ESV [29, 31]. Usually these changes are symmetrical and those in the RV reflect those in the LV [25, 32]. In the present study nearly half of participants presented enlarged RV but it was not accompanied by an increase in LV diameters or volumes. These observations were previously explained as RV sensitiveness and an expected response to increased overload [25]. Nevertheless, current results concerning the RV and LV systolic function suggest difficulties of RV for amateur marathon runners to adapt to exercise and can support a thesis that RV as an “Achilles heel” of the competing heart. Not only RV but also RA may limit the heart function, as in the present group, both right heart chambers were dilated and the RA area and RVd correlated positively. Probably, the right heart of predisposed individuals, when exposed to repetitive episodes of overload, may be prone to irreversible damage. The recurrent extreme effort can lead to so-called Phidippides cardiomyopathy, in which the focal areas of cardiac fibrosis develop and become the substrate for ventricular arrhythmias and a reason for sudden death [33].

## **Conclusions**

The results of the present study demonstrate that “athlete’s heart” features do develop in amateur marathon runners. One of the most important findings was increased LAVI, which exceeded even the upper reference limit for highly-trained athletes in half of the study

participants. It may reflect abnormal atrial response to pressure overload in recreational marathon runners not sufficiently adapted to endurance exercise. Another important issue was the high prevalence of IVS thickening among amateur athletes and a confirmed diagnosis of HCM in one participant. Echocardiography should play a pivotal role in the medical assessment of this population. In individuals with the history of marathon attendance ECHO reference values for highly trained elite athletes may be more helpful than those applied for the general adult population. CMR imaging is indicated when it is difficult to differentiate between physiological “athlete’s heart” remodeling and conditions like HCM.

**Conflict of interest:** None declared

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**Table 1.** Characteristics of amateur marathon runners studied (n = 34).

<b>Parameter</b>		<b>Marathon runners</b>
Training distance [km/week]		56.5 ± 19.7
Training time [h/week]		6.5 ± 2.3
Marathon finishing time [h]		3.7 ± 0.4
Cardiopulmonary exercise test:		
	VO <sub>2</sub> peak [mL/kg/min]	53.7 ± 6.9
	VO <sub>2</sub> 60sec [mL/kg/min]	19.9 ± 3.7
	VO <sub>2</sub> AT [mL/kg/min]	39.7 ± 6.9
	Respiratory exchange ratio	1.2 ± 0.1
	Time of effort [min]	12:46 ± 1:24
	HR max [bpm]	178 ± 12
	HR in 180 s of recovery [bpm]	111 ± 16

Data are shown as mean ± standard deviation; VO<sub>2</sub>peak — peak oxygen intake; VO<sub>2</sub>60sec — oxygen intake at 60 s of recovery; VO<sub>2</sub>AT — oxygen intake at anaerobic threshold; HR — heart rate

**Table 2.** Results of echocardiographic examination performed in amateur marathon runners and subjects from the control group.

<b>Parameter</b>	<b>Marathon runners (n = 34)</b>	<b>Control group (n = 15)</b>	<b>P</b>
Age [years]	41 (24–55)	42 (24–55)	> 0.6709 <sup>^</sup>
Weight [kg]	80 (67–97)	80 (64–100)	> 0.3878 <sup>^</sup>

Height [cm]	180 (165–188)	177 (169–195)	> 0.7643 <sup>^</sup>
BSA [m <sup>2</sup> ]	2.0 (1.8–2.2)	2.0 (1.7–2.2)	> 0.5206 <sup>^</sup>
BMI [kg/m <sup>2</sup> ]	25 (19–29)	25 (22–31)	> 0.2069 <sup>^</sup>
LAVI [mL/m <sup>2</sup> ]	36 (21–51)	27 (17–35)	< <b>0.0001</b> <sup>^</sup>
RA area [cm <sup>2</sup> ]	19 (14–25)	16 (11–20)	< <b>0.0005</b> <sup>^</sup>
LV EDV [ml]	122 (78–176)	105 (66–164)	> 0.0732 <sup>^</sup>
LV ED [mm]	52 (45–58)	50 (39–59)	> 0.0729 <sup>^</sup>
IVS [mm]	11 (7–17)	10 (7–10)	< <b>0.0001</b> <sup>#</sup>
LV PW [mm]	11 (7–14)	10 (7–11)	< <b>0.0206</b> <sup>^</sup>
LV mass [g/m <sup>2</sup> ]	97 (61–117)	77 (62–108)	< <b>0.00001</b> <sup>^</sup>
LV EF [%]	66 (51–86)	62 (56–74)	> 0.1896 <sup>^</sup>
LV GLS [%]	-20 [-17 – (-25)]	-20 [-17 – (-23)]	> 0.4363 <sup>^</sup>
RVOT prox [mm]	30 (21–38)	30 (25–36)	> 0.6764 <sup>^</sup>
RVd [mm]	37 (25–47)	30 (27–40)	< <b>0.0179</b> <sup>^</sup>
TAPSE [mm]	24 (19–32)	23 (20–27)	> 0.4550 <sup>^</sup>
RV strain [%]	-22 [-27 – (-18)]	-24 [-26 – (-19)]	> 0.2978 <sup>^</sup>

Data are shown as median (range); <sup>^</sup>The Student t-test; <sup>#</sup>The Mann–Whitney U test; BSA — body surface area; BMI — body mass index; LAVI — left atrial volume indexed to body surface area; RA — right atrial; LV — left ventricular; EDV — end-diastolic volume; ED — end-diastolic dimension; IVS — interventricular septum diastolic diameter; PW — posterior wall diameter; EF — ejection fraction; GLS — global longitudinal peak strain; RVd — right ventricular diameter; RVOT prox — proximal right ventricular outflow tract diameter; TAPSE — tricuspid annular plane systolic excursion

**Table 3.** Results of echocardiographic examination performed in amateur marathon runners (n = 34) in comparison with reference ranges for the general male adult population and with reference ranges for professional athletes.

<b>Parameter</b>	<b>Reference ranges for adults (range)</b>	<b>Runners with values exceeding the upper reference value for the adult population (URP) N (%)</b>	<b>Reference ranges for highly trained athletes (range)</b>	<b>Runners with values exceeding the upper reference value for highly trained athletes (URA) N (%)</b>
LA AP [mm]	30–40 <sup>[5]</sup>	13 (38)	24–48 <sup>[8]</sup>	0 (0)
LAVI [mL/m <sup>2</sup> ]	16–34 <sup>[5]</sup>	19 (56)	26–36 <sup>[8]</sup>	17 (50)
RA area [cm <sup>2</sup> ]	10–18 <sup>[10]</sup>	19 (56)	14–23 <sup>[6]</sup>	3 (9)
LV EDV [mL]	62–150 <sup>[5]</sup>	3 (9)	180–340 <sup>[4]</sup>	0 (0)
LV ED [mm]	42–58 <sup>[5]</sup>	0 (0)	44–66 <sup>[7]</sup>	0 (0)
IVS [mm]	6–10 <sup>[5]</sup>	19 (56)	7–16 <sup>[7]</sup>	1 (3)
LV PW [mm]	6–10 <sup>[5]</sup>	14 (41)	7–13 <sup>[7]</sup>	2 (6)
LV mass [g/m <sup>2</sup> ]	50–102 <sup>[5]</sup>	10 (29)	62–176 <sup>[7]</sup>	0 (0)
RVd [mm]	25–41 <sup>[5]</sup>	5 (15)	38–42 <sup>[6]</sup>	3 (9)
RVOT prox [mm]	20–30 <sup>[5]</sup>	16 (47)	26–33 <sup>[6]</sup>	6 (18)

LA AP — left atrial anteroposterior dimension. For other abbreviations see Table 2

**Table 4.** Results of cardiac magnetic resonance imaging (CMR) performed in amateur marathon runners with abnormalities revealed in echocardiographic (ECHO) examination.



No.	Reason for CMR	CMR results
	ECHO abnormalities	
M06	<p>LV GLS Avg -17%, with abnormal LV GLS pattern</p> <p>GLS 2C -16%, GLS 4C -17%, GLS Aplax -17% (n: &gt; -18.9%)<sup>[9]</sup></p> <p>RVd 47 mm</p>	Slightly reduced LV EF (53%) and RV EF (44%). Enlarged LV (LV ESV 86 mL) and RV (RV ESV 118 mL)
M29	<p>IVS 14.7 mm</p> <p>E'LAT 8 cm/s (n: &gt; 10 cm/s)<sup>[34]</sup></p> <p>RV strain -19% (n: &gt; -20%)<sup>[10]</sup></p>	Slightly reduced LV EF (54%) and RV EF (42%), LV hypertrophy (IVS 13 mm), enlarged LV (LV ESV 79 mL) and RV (RV ESV 119 mL)
M38	<p>IVS 17 mm</p> <p>LV EF 51%</p> <p>E'SEPT 6 cm/s (n: &gt; 7 cm/s)<sup>[34]</sup></p> <p>LV GLS Avg -18%, abnormal LV GLS pattern</p> <p>GLS 2C -17%, GLS 4C -17% (n: &gt; -18.9%)<sup>[9]</sup></p> <p>RVOT prox 31 mm</p>	<p>Hypertrophic cardiomyopathy (IVS 17 mm), LGE present</p> <p>Slightly reduced RV EF (47%) and enlarged RV (RV ESV 97 mL)</p> <p>Increased LA area (30 cm<sup>2</sup>)</p>
M39	<p>IVS 12 mm</p> <p>LV GLS Avg -17%, abnormal LV GLS pattern</p> <p>GLS 2C -17%, GLS 4C -16% (n: &gt; -18.9%)<sup>[9]</sup></p> <p>RV strain -18% (n: &gt; -20%)<sup>[10]</sup></p> <p>LV ED 52 mm</p>	Slightly reduced RV EF (42%) and enlarged RV (RV ESV 112 mL)
M40	<p>IVS 13 mm</p> <p>E'SEPT 7 cm/s (n: &gt; 7 cm/s)<sup>[34]</sup></p>	<p>Atrial septal defect</p> <p>Slightly reduced RV EF (48%) and</p>

	S'RV 9 cm/s (n: > 9.5 cm/s) <sup>[10]</sup> RV strain -19% (n: > -20%) <sup>[10]</sup> RVOT prox 32 mm LV ED 49 mm	enlarged RV (RV ESV 103 mL)
M41	IVS 12 mm Abnormal LV GLS pattern: GLS Avg -17%, GLS 2C -16% (n: > -18.9%) <sup>[9]</sup> LV EDV 176 mL	Slightly reduced LV EF (56%) and RV EF (49%). Enlarged LV (LV EDV 245 mL, LV ESV 107 mL) and RV (RV EDV 239 mL, RV ESV 123 mL)

For abbreviations see Table 2, for echocardiographic reference values see Table 3; values outside the range for adults. For cardiac magnetic resonance reference values see Table 5; No. — number of marathon runners; Avg — averaged; 2C — two chamber view; 4C — four chamber view; Aplax — apical long axis view; S'RV — spectral tissue Doppler tricuspid lateral annulus peak systolic velocity; LGE — late gadolinium enhancement; E' — spectral tissue Doppler mitral early diastolic peak velocity (SEPT — measured on IVS; LAT — measured on lateral wall)

**Table 5** Results of cardiac magnetic resonance examination (CMR) in amateur marathon runners.

Parameter	Study participants (n = 6); median (range)	Reference values for men < 60 years <sup>[35]</sup> (range)
LA area 4C [cm <sup>2</sup> ]	24 (18–30)	15–29
RA area 4C [cm <sup>2</sup> ]	22 (20–30)	14–30
LV EDV [mL]	182 (152–245)	119–203
LV ESV [mL]	76 (60–107)	33–77
LV EF [%]	59 (53–62)	57–75

LV mass [g]	165 (155–199)	107–187
RV EDV [mL]	202 (184–239)	119–219
RV ESV [mL]	115 (97–123)	32–92
RV EF [%]	46 (42–49)	50–78

For abbreviations see Table 2; LA — left atrial; RA — right atrial; 4C — four chamber view;  
RV EF — right ventricular ejection fraction; ESV — end-systolic volume