

# Detection of Cardiovascular Disease in Elite Athletes Using Cardiac Magnetic Resonance Imaging

## Kardiale Magnetresonanztomografie zur Risikostratifizierung in Leistungssportlern

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### Key words

- heart
- cardiac
- MR functional imaging
- MR angiography
- ischemia/infarction
- normal variants

### Zusammenfassung



**Ziel:** In der Genese von plötzlichem Herztod bei Leistungssportlern spielen neben der hypertrophen und dilatativen Kardiomyopathie [HCM/DCM] auch die Myokarditis, Koronaranomalien und ischämische Herzerkrankungen eine wichtige Rolle. Zur Abklärung dieser potentiellen Risikofaktoren erfolgte die Durchführung einer kardialen Magnetresonanztomografie [MRT].

**Material und Methoden:** 73 männliche [M] und 22 weibliche [F] Athleten (Durchschnittsalter 35,2 ± 11,4 Jahre) wurden einer kardialen MRT zugeführt. EKG-getriggerte cine SSFP Sequenzen wurden verwendet um Wandbewegungsstörungen und Myokardhypertrophien zu diagnostizieren und um quantitative Auswertungen durchzuführen (links- und rechtsventrikuläres [LV, RV] enddiastolisches und endsystolisches Volumen [EDV, ESV], Schlagvolumen [SV], Ejektionsfraktion [EF] und die Myokardmasse [MM]). Zusätzlich wurden die Vorhofgrößen planimetrisch erfasst und Kontrastmittel injiziert, um fibrotische Veränderungen des Myokards nachzuweisen. Die Darstellung der Koronararterien erfolgte mittels einer Flash-3-D-MR-Angiografie.

**Ergebnisse:** Die quantitativen Analysen zeigten eine exzentrische LV-Hypertrophie (remodeling index [MM/LV-EDV]: männlich 0,75, weiblich 0,665), erhöhte RV-Volumina (RV-EDV: M 122,6 ± 19,0 ml/m<sup>2</sup>, F 99,9 ± 7,2 ml/m<sup>2</sup>) sowie erhöhte SV (LV-SV: M 64,7 ± 10,0 ml/m<sup>2</sup>, F 56,5 ± 5,7 ml/m<sup>2</sup>; RV-SV: M 66,7 ± 10,4 ml/m<sup>2</sup>, F 54,2 ± 7,1 ml/m<sup>2</sup>). Pathologische Befunde fanden sich in 6 Athleten (6,3%), darunter eine benigne Variante einer Koronaranomalie sowie fibrotische Myokardveränderungen in 2 Fällen. Wandbewegungsstörungen oder postischämische Veränderungen fanden sich hingegen nicht.

**Schlussfolgerung:** Die durchgeführte kardiale MRT in Leistungssportlern zeigte pathologische Befunde in über 5% der Athleten, wobei die prog-

### Abstract



**Purpose:** Sudden cardiac death [SCD] in competitive athletes is caused by a diverse set of cardiovascular diseases such as hypertrophic and dilated cardiomyopathy [HCM/DCM], myocarditis, coronary anomalies or even coronary artery disease. In order to identify potential risk factors responsible for SCD, elite athletes underwent cardiac magnetic resonance [CMR] imaging.

**Materials and Methods:** 73 male [M] and 22 female [F] athletes (mean age 35.2 ± 11.4 years) underwent CMR imaging. ECG-gated breath-hold cine SSFP sequences were used for the evaluation of wall motion abnormalities and myocardial hypertrophy as well as for quantitative analysis (left and right ventricular [LV, RV] end-diastolic and end-systolic volume [EDV, ESV], stroke volume [SV], ejection fraction [EF] and myocardial mass [MM]). Furthermore, left and right atrial sizes were assessed by planimetry and delayed enhancement imaging was performed 10 minutes after the application of contrast agent. Coronary arteries were depicted using free-breathing Flash-3 D MR angiography.

**Results:** The quantitative analyses showed eccentric hypertrophy of the left ventricle (remodeling index [MM/LV-EDV]: M 0.75, F 0.665), enlargement of the RV volumes (RV-EDV: M 122.6 ± 19.0 ml/m<sup>2</sup>, F 99.9 ± 7.2 ml/m<sup>2</sup>) and an increased SV (LV-SV: M 64.7 ± 10.0 ml/m<sup>2</sup>, F 56.5 ± 5.7 ml/m<sup>2</sup>; RV-SV: M 66.7 ± 10.4 ml/m<sup>2</sup>, F 54.2 ± 7.1 ml/m<sup>2</sup>). Abnormal findings were detected in 6 athletes (6.3%) including one benign variant of coronary anomaly and abnormal late gadolinium enhancement in 2 cases. None of the athletes showed wall motion abnormalities or signs of myocardial ischemia.

**Conclusion:** CMR imaging of endurance athletes revealed abnormal findings in more than 5% of the athletes. However, the prognostic significance remains unclear. Thus, cardiac MRI cannot

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### Bibliography

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nostische Relevanz solcher Befunde zunächst nicht abschließend zu beurteilen ist. Daher ist die routinemäßige Durchführung einer kardialen MRT in Leistungssportlern primär nicht zu empfehlen.

#### Kernaussagen:

- ▶ Die MRT liefert einen wichtigen Beitrag zur Diagnostik von kardiovaskulären Erkrankungen in Athleten.
- ▶ Die Differenzierung von physiologischen Anpassungsreaktionen und einer DCM/HCM stellt eine große Herausforderung dar.
- ▶ Eine routinemäßige Durchführung einer kardialen MRT in Leistungssportlern kann nicht empfohlen werden.

## Introduction

As competitive athletes symbolize vitality and health, they have always been granted a status of special appreciation and prestige. This may explain the sense of tragedy that comes with sudden cardiac death (SCD) in athletes, caused by a diverse set of cardiovascular diseases such as hypertrophic and dilated cardiomyopathy (HCM/DCM), myocarditis, arrhythmogenic right ventricular disease (ARVD), coronary anomalies or even coronary artery disease (CAD) [1, 2]. An overall incidence of 2.1/100,000/year [2] has fuelled interest in pre-participation screening and the identification of the risk factors commonly responsible for SCD in elite athletes [3–5]. Of note, morphological and functional adaptations of the cardiovascular system related to endurance training [6–10] must be distinguished from pathological findings of the myocardium. Congenital or acquired anomalies should be excluded in order to minimize the risk for cardiac events during physical activity. Previous approaches to pre-participation testing included personal and family history as well as physical examination [3, 4, 11–13]. Due to a high percentage of abnormal electrocardiographic (ECG) findings in HCM [14] and ARVD [5, 11, 12], an additional 12-lead ECG is recommended [15] since it has the potential to increase the sensitivity of the screening progress [5, 15–17]. However, these tests and even performing an additional echocardiography may fail to detect risk factors especially with regard to coronary anomalies and subtle ischemic and non-ischemic myocardial fibrosis. In this context, CMR imaging has turned out to be the method of choice using delayed enhancement and high-resolution 3D imaging techniques [18–21]. Furthermore, CMR imaging allows the assessment of ventricular chamber volumes and functional parameters with good inter- and intraobserver reliability [21, 22].

The purpose of this study was to quantify the prevalence of potential risk factors and determine ventricular functional parameters using CMR in elite athletes with unremarkable pre-participation screening.

## Methods

### Study population

The study protocol was approved by the institutional ethics committee and all participants gave written informed consent prior to the study.

We prospectively enrolled 100 healthy athletes (mean age 35.2 ± 11.4 years, range from 18 to 62 years; 77 males, mean age 37.4 ±

11.4 years, range from 19 to 62 years; 23 females, mean age 28.4 ± 8.5 years, range from 18 to 50 years). The athletes were highly trained long-distance runners (n = 39), cyclists (n = 8), triathletes (n = 34), handball players (n = 13) and one speed skater with a training history of at least two years and a weekly training workload of 13.1 ± 4.2 hours (males: 13.1 ± 4.5 hours/week, range from 5 to 30 hours/week; females: 12.8 ± 3 hours/week, range from 7 to 20 hours/week). The physical characteristics of the athletes are given in detail in **Table 1**.

#### Key points:

- ▶ CMR imaging helps clinicians to detect cardiovascular diseases in elite athletes.
- ▶ Differentiation between physiological adaptations and DCM/HCM can be a challenging task.
- ▶ Routine cardiac MRI examinations of endurance athletes cannot be recommended.

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In standard pre-participation screening which was performed prior to the CMR examination, none of the participants showed any pathologies prohibiting their sports activities. The BNP values were within normal ranges (< 100 ng/l) in all athletes and none of the participants reported previous cardiovascular disease. Furthermore, all athletes denied taking prohibited substances.

### Cardiac magnetic resonance imaging protocol

All examinations (n = 95) were performed on a 1.5 Tesla MR system (Magnetom Avanto, Siemens Medical Solutions, Forchheim, Germany) using surface coils for signal reception. In 5 cases no CMR imaging data sets could be generated because of the withdrawal of informed consent. For the evaluation of wall motion abnormalities and myocardial hypertrophy, 2D cine steady-state free precession (SSFP) sequences were acquired in a 4-chamber view, left ventricular long axis view (ECG-retro-gating, repetition time (TR) 39.75, echo time (TE) 1.12 ms, angle of excitation (FA) 70°, field of view (FOV) adapted to each athlete, pixel spacing 1.8 × 1.8, matrix 156 × 192, slice thickness (SL) 6 mm, bandwidth (BW) 930 Hz/pixel) as well as in short axis angulation (ECG-retro-gating, TR 58.96 ms, TE 1.13 ms, FA 70°, pixel spacing 2.4 × 1.8, matrix 113 × 192, SL 5 mm, BW 930 Hz/pixel).

For coronary MR angiography (MRA), an ECG-gated 3D GRE/SSFP sequence was combined with respiratory navigator gating and

**Table 1** Physical characteristics.

**Tab. 1** Körperliche Eigenschaften.

	total	male	female
age [years]	35.2 ± 11.4	37.4 ± 11.4	28.4 ± 8.5
height [m]	1.78 ± 7.6	1.81 ± 6.1	1.7 ± 6.2
weight [kg]	71 ± 9.7	72.9 ± 8.4	65.2 ± 11.4
BSA [kg/m <sup>2</sup> ]	1.87 ± 0.16	1.91 ± 0.13	1.7 ± 0.2
heart rate [bpm]	55 ± 6.9	53 ± 6.7	58 ± 7.3
VO <sub>2</sub> max. [ml/min]	55.5 ± 7.9	57.3 ± 6.9	50.1 ± 8.7
training hours/week	13.1 ± 4.2	13.1 ± 4.5	12.8 ± 3

tracking. The respiratory navigator was localized at the lung-liver interface of the right hemidiaphragm with a 2 mm gating window. The acquisition window and TR were adapted to the duration of the end-diastolic period of rest and an individual trigger delay was determined in order to minimize myocardial motion artifacts during the acquisition interval. The following settings were used: spatial resolution (voxel size)  $1.0 \times 1.0 \times 1.1$  mm, TE 1.75 ms, FA  $90^\circ$ , matrix  $216 \times 320$ , BW 601 Hz/pixel.

Furthermore, delayed enhancement imaging was performed 10 minutes after administration of 0.15 mmol/kg body weight Gadobutrol (Gadovist, Bayer Healthcare, Leverkusen, Germany) at a flow rate of 2.0 ml/s using an inversion recovery [IR] turbo FLASH 2D sequence (TR 700 ms, TE 4.91 ms, FA  $30^\circ$ , matrix  $154 \times 256$ , pixel spacing  $1.5 \times 1.12$  mm, SL 8 mm, BW 140 Hz/Pixel). Optimal inversion time (TI) was determined using a TI scout sequence (TR 23.49 ms, TE 1.12 ms, FA  $60^\circ$ , pixel spacing  $3.5 \times 1.8$  mm, matrix  $78 \times 192$ , SL 8 mm, BW 965 Hz/Pixel) to minimize the signal intensity of normal myocardium.

### Imaging analyses

Two experienced readers independently reviewed the image loops of each subject in a random fashion. Readers were blinded for subject details including age and training hours/week. Quantitative analysis was performed off-line using dedicated software (Leonardo VD30B, Siemens Medical Solutions). The left ventricular (LV) and right ventricular (RV) wall mass and volumes were measured by tracing the endocardial and epicardial borders on the short-axis views. At the base of the LV, the aorta was included in the LV volume below the aortic valve. Blood volumes above the aortic valve, as well as volumes surrounded by a thin myocardial wall on the mitral valve plane (left atrial blood volume), were excluded from the LV volume and the basal slice was selected for the left ventricle when at least 50% of the blood volume was surrounded by myocardium in both end-diastole and end-systole. By inspection of the cine loops, end-systole was defined as the frame with the smallest ventricular cavity. Papillary muscles and trabeculae were included in the ventricular volumes (and excluded from the wall mass) for efficiency and reproducibility. Epicardial fat and the pericardium were excluded from the RV and LV mass [17]. End-diastolic volumes (EDV) and end-systolic volumes (ESV) of the LV and RV were calculated according to a

modified Simpson's rule in short axis views [17–19] and were used to determine stroke volume (SV) and ejection fraction (EF). The LV mass was determined by summation of EDVs within the epicardial and endocardial borders of the short-axis slices and by multiplying the myocardial tissue volume by its specific density of 1.05 g/cm<sup>3</sup>. Atrial sizes were assessed by planimetry of the maximum right atrial (RA) and left atrial (LA) areas in a standard 4-chamber view just before the opening of the mitral valve. Pulmonary veins were excluded from the LA area [18]. Additionally, all parameters were indexed to the body surface area (BSA) for comparative analysis in order to minimize differences of cardiac parameters related to height and weight. The LV remodeling index (MM/LV-EDV) was calculated to determine the pattern of ventricular remodeling.

Delayed enhancement images, the MR coronary angiography and the Cine SSFP images were evaluated visually regarding wall motion abnormalities, coronary anomalies and ischemic or non-ischemic patterns of delayed myocardial enhancement. In the event of a deviation between both readers, the images were re-evaluated by both radiologists in consensus.

### Statistics

The data were statistically analyzed using SPSS software, version 11.5 (SPSS, Inc, Chicago, USA). Continuous data are presented as mean  $\pm$  standard deviation. Normality was tested using the Kolmogorov-Smirnov test. The inter-reader agreement regarding chamber volumes, functional parameters and MM were evaluated using the Cohen's Kappa test. Pearson correlation coefficients were calculated for selected variables. A p-value of less than 0.05 was considered statistically significant.

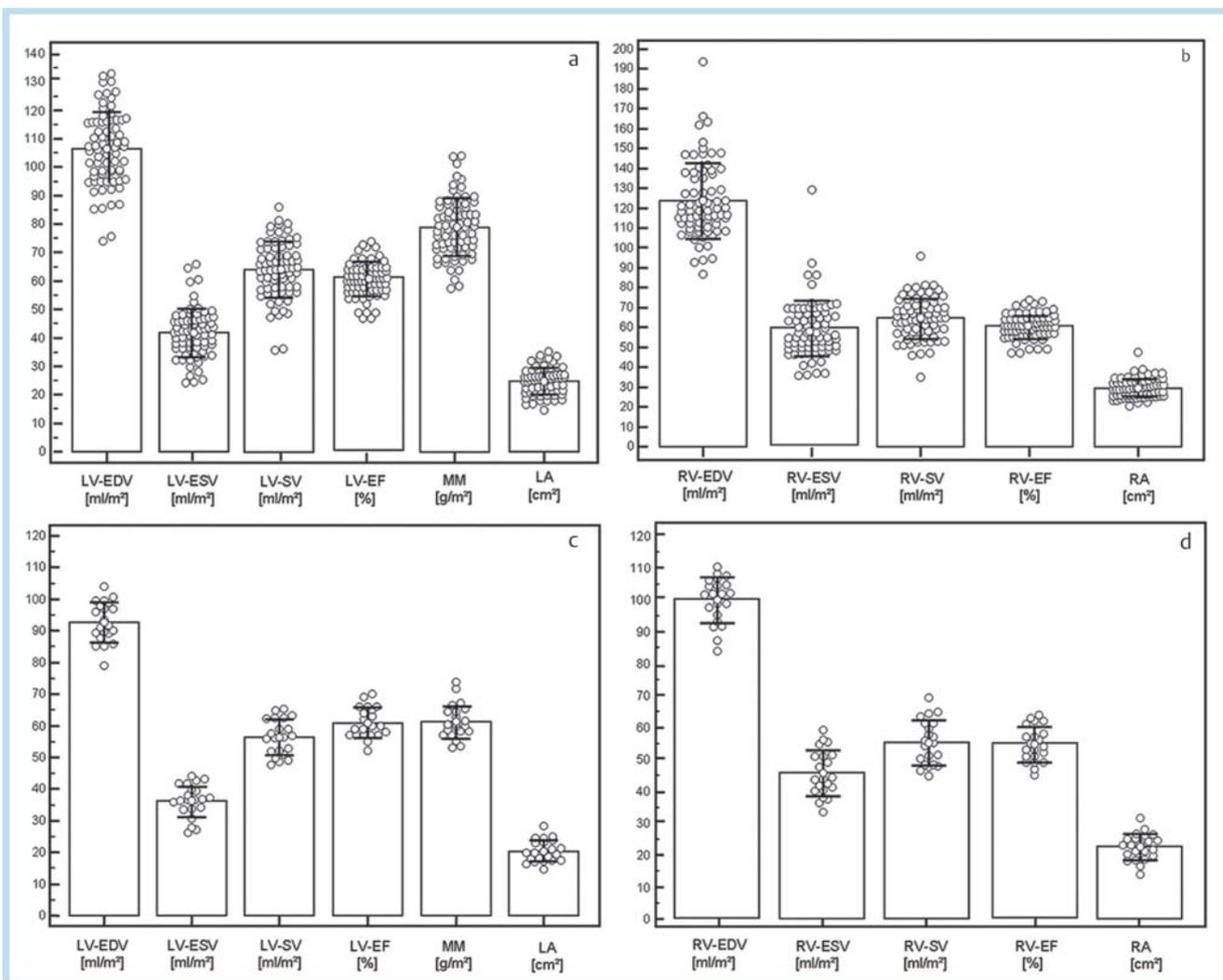
### Results

For cine SSFP and delayed enhancement images, diagnostic image quality was obtained in all of the 95 MRI examinations. The mean total imaging time was 50:42 min  $\pm$  10:12 min (range from 34 to 74 min) and the acquisition time of the MR coronary angiography was 14:33 min  $\pm$  5:25 min (range from 6:34 to 44:42 min). The results of the evaluation of the chamber volumes, atrial sizes and functional parameters are given in detail in **Table 2** and

**Table 2** Results of the evaluation of the ventricular volumes and functional parameters. A p-value of  $<0.05$  was regarded as statistically significant.  $\kappa$ -value in parentheses. [LV] left ventricular [RV] right ventricular [EDV] end-diastolic volume [ESV] end-systolic volume [SV] stroke volume [EF] ejection fraction [MM] myocardial mass [LA] left atrium [RA] right atrium.

**Tab. 2** Ergebnisse der quantitativen Auswertung von Ventrikelvolumina und Funktionsparametern. Ein p-Wert von  $<0,05$  wurde als statistisch signifikant erachtet.  $\kappa$ -Werte in Klammern. [LV] linker Ventrikel [RV] rechter Ventrikel [EDV] enddiastolisches Volumen [ESV] endsystolisches Volumen [SV] Schlagvolumen [EF] Ejektionsfraktion [MM] Myokardmasse [LA] linker Vorhof [RA] rechter Vorhof.

	total	male	female	p-values
LV-EDV [ml/m <sup>2</sup> ]	103.3 $\pm$ 13.1 (0.80)	106.5 $\pm$ 12.9	92.7 $\pm$ 6.4	$<0.001$
LV-ESV [ml/m <sup>2</sup> ]	40.4 $\pm$ 8.2 (0.78)	41.8 $\pm$ 8.5	36.3 $\pm$ 5.2	0.007
LV-SV [ml/m <sup>2</sup> ]	62.9 $\pm$ 9.7 (0.81)	64.7 $\pm$ 10.0	56.4 $\pm$ 5.7	0.001
LV-EF [%]	60.8 $\pm$ 5.7 (0.75)	60.8 $\pm$ 6.0	60.9 $\pm$ 4.8	0.916
RV-EDV [ml/m <sup>2</sup> ]	118.4 $\pm$ 19.7 (0.67)	126.6 $\pm$ 19.0	99.9 $\pm$ 7.2	$<0.001$
RV-ESV [ml/m <sup>2</sup> ]	56.1 $\pm$ 14.2 (0.65)	59.9 $\pm$ 14.5	45.7 $\pm$ 7.3	$<0.001$
RV-SV [ml/m <sup>2</sup> ]	62.3 $\pm$ 10.4	66.7 $\pm$ 10.4	54.2 $\pm$ 7.1	$<0.001$
RV-EF [%]	56.2 $\pm$ 5.4 (0.60)	52.6 $\pm$ 6.4	54.7 $\pm$ 5.6	0.191
MM [g/m <sup>2</sup> ]	74.8 $\pm$ 11.9 (0.78)	78.9 $\pm$ 10.2	61.4 $\pm$ 5.4	$<0.001$
remodeling index	0.726	0.750	0.665	0.0001
LA [cm <sup>2</sup> ]	23.7 $\pm$ 4.7	24.8 $\pm$ 4.6	20.3 $\pm$ 3.6	0.0001
RA [cm <sup>2</sup> ]	27.8 $\pm$ 5.6	29.5 $\pm$ 4.9	22.6 $\pm$ 3.3	$<0.0001$



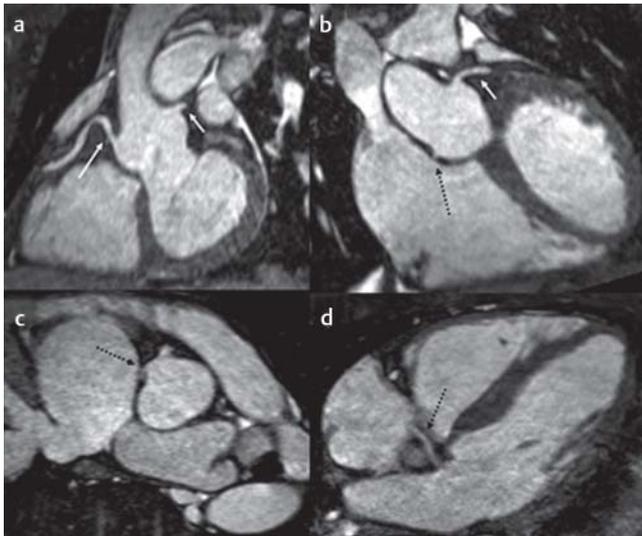
**Fig. 1** Results of the quantitative analysis of chamber volumes and functional parameters. Left ventricular [LV] end-diastolic volume [EDV], end-systolic volume [ESV], stroke volume [SV], ejection fraction [EF], myocardial mass [MM] as well as the results of the left atrial [LA] and right atrial [RA] planimetry of male athletes are shown in **a, b**; **c, d** show the results of the evaluation of female athletes.

**Abb. 1** Ergebnisse der quantitativen Auswertung der Ventrikelvolumina und funktionellen Parametern. **a, b** zeigen das enddiastolische und end-systolische Volumen [EDV, ESV], das Schlagvolumen [SV], die Ejektionsfraktion [EF] und die Myokardmasse [MM] des linken Ventrikels sowie die Ergebnisse der Planimetrie von linkem und rechtem Vorhof [LA, RA] in männlichen Athleten, **c, d** die Ergebnisse der Athletinnen.

• **Fig. 1.** The quantitative analyses showed a simultaneous increase of left ventricular chamber size and MM, called eccentric hypertrophy of the left ventricle (LV-EDV: male  $106.5 \pm 12.9$  ml/m<sup>2</sup>, female  $92.7 \pm 6.4$  ml/m<sup>2</sup>; MM: male  $78.9 \pm 10.2$  g/m<sup>2</sup>, female  $61.4 \pm 5.4$  g/m<sup>2</sup>; remodeling index: male 0.75, female 0.665). Furthermore, an enlargement of the right chamber volumes (RV-EDV: male  $122.6 \pm 19.0$  ml/m<sup>2</sup>, female  $99.9 \pm 7.2$  ml/m<sup>2</sup>) and an increased LV-SV (male:  $64.7 \pm 10.0$  ml/m<sup>2</sup>, female  $56.5 \pm 5.7$  ml/m<sup>2</sup>) as well as RV-SV (male:  $66.7 \pm 10.4$  ml/m<sup>2</sup>, female  $54.2 \pm 7.1$  ml/m<sup>2</sup>) were found. The evaluation of the chamber and stroke volumes as well as the MM revealed significantly elevated values for male athletes in comparison to the female study population (• **Table 2**). The Cohen's kappa test showed good results regarding the interobserver variability ( $\kappa$ -values are also shown in • **Table 2**). There was a significant correlation between weekly training volume and LV-EDV ( $r^2 = 0.2375$ ,  $p = 0.0430$ ) and LV-ESV ( $r^2 = 0.2465$ ,  $p = 0.0355$ ) in male athletes whereas in female athletes no significant correlations between chamber volumes and training load were found.

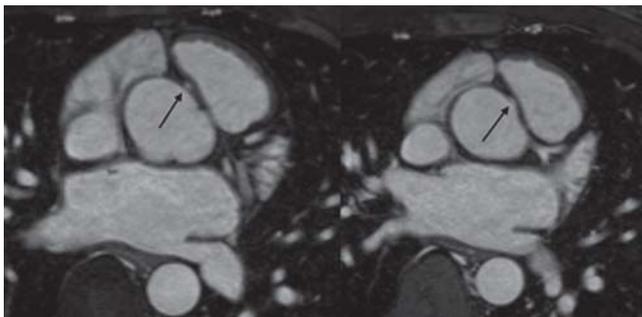
However, pathological findings were detected in 6 athletes (6.3%).

Regarding the coronary anomalies, we found one case with the circumflex artery originating from the right coronary sinus and course posterior to the ascending aorta (• **Fig. 2**). Furthermore, one athlete showed an atypical, anterior origin of the right coronary artery (RCA), formally not according to a coronary anomaly but which might cause symptoms of ischemia during exercise due to the long-distance course between the ascending aorta and pulmonary trunk (• **Fig. 3**). In addition, one athlete was suffering from ectasia of the ascending aorta (• **Fig. 4**) but had normal blood pressure values (120/80 mmHg) and in 2 athletes we found pathological signal alterations in the delayed enhancement imaging without corresponding wall motion abnormalities, due to the spot-shaped pattern consistent with a non-ischemic, post-inflammatory genesis (• **Fig. 5**). In addition, one of the athletes showed pleural effusions and pericardial effusion, but no wall motion abnormalities or delayed enhancement of the myocardium (• **Fig. 6**). On presentation, the athlete previously experienced a



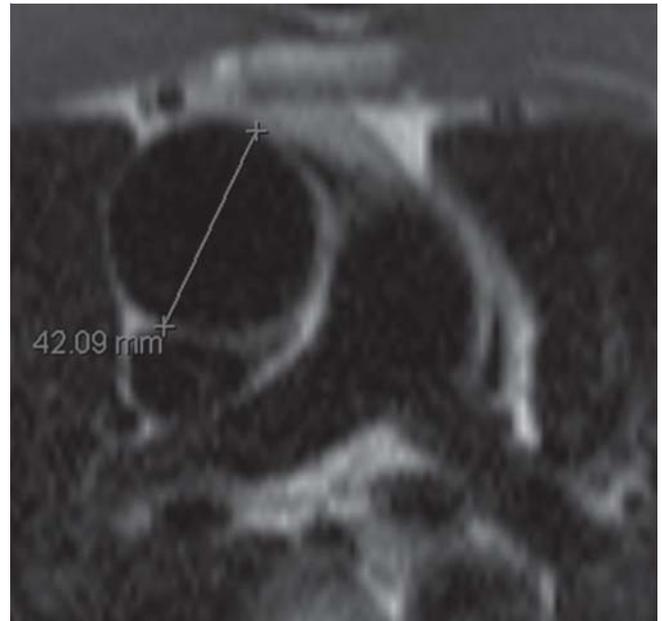
**Fig. 2** ECG-gated 3-D MR angiography of a 46-year-old male triathlete with a training load of 18 hours per week. **a** Origin of the left anterior descending artery from the left sinus (short arrow) and of the right coronary artery from the right sinus (long arrow). **b+c** show the left circumflex artery (LCX), also originating from the right sinus (dotted arrow) and taking course dorsal to the ascending aorta (dotted arrow in **d**), which represents the most common but benign variant of coronary anomaly.

**Abb. 2** EKG-getriggerte 3-D-MR-Angiografie eines 46-jährigen männlichen Triathleten mit einem Trainingspensum von 18 Stunden pro Woche. **a** Abgang des Ramus interventricularis anterior aus dem links-koronaren Sinus (kurzer Pfeil) und der rechten Koronararterie aus dem rechts-koronaren Sinus (langer Pfeil). **b+c** zeigen den Ramus circumflexus, welcher ebenso dem rechts-koronaren Sinus entspringt (gepunkteter Pfeil) und dorsal der aufsteigenden Aorta (gepunkteter Pfeil in **d**) verläuft, was der häufigsten und als benigne einzustufenden Variante einer Koronaranomalie entspricht.



**Fig. 3** ECG-gated 3-D MR angiography of a 39-year-old male marathon runner with a training load of 10 hours per week reveals a right coronary artery [RCA] originating from the left corner of the right sinus which is formally not considered as a coronary anomaly but might cause recurrent ischemic alterations due to the long course of the RCA between the aorta and pulmonary trunk.

**Abb. 3** EKG-getriggerte 3-D-MR-Angiografie eines 39-jährigen männlichen Marathonläufers mit einem Trainingspensum von 10 Stunden pro Woche. Hier entspringt die rechte Koronararterie [RCA] weit links im Bereich des rechts-koronaren Sinus, was formal nicht als Koronaranomalie erachtet wird, aufgrund des langstreckigen Verlaufs der RCA zwischen Aorta und Truncus pulmonalis jedoch zu rezidivierenden ischämischen Ereignissen führen kann.



**Fig. 4** 43-year-old marathon runner with a training load of 10 hours per week. HASTE sequenz shows an ectasia of the ascending aorta of 4.2 cm. This athlete will be closely monitored.

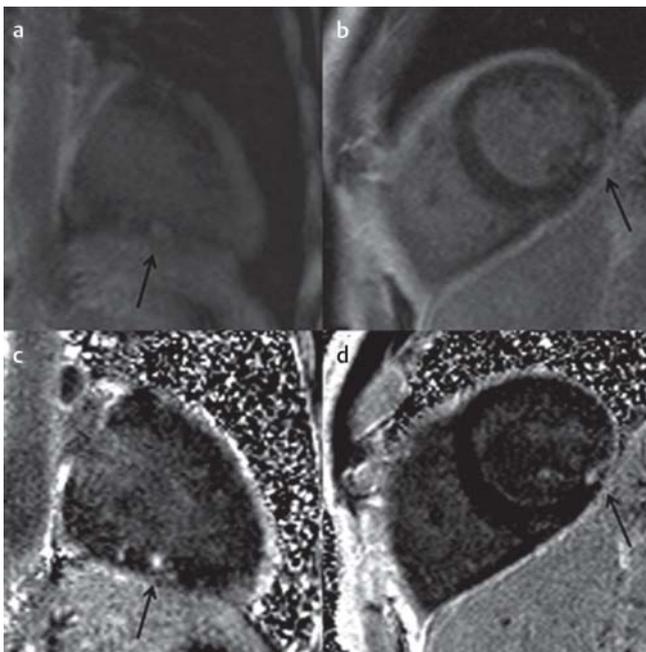
**Abb. 4** Die HASTE-Sequenz eines 43-jährigen Marathonläufers mit einem Trainingspensum von 10 Stunden pro Woche zeigte eine Ektasie der Aorta ascendens auf 4,2 cm. Dieser Befund wird engmaschig verlaufskontrolliert.

feverish infection, which turned out to be an Epstein-Barr virus infection. 6 weeks later the athlete underwent a control MRI examination which showed complete recovery.

## Discussion

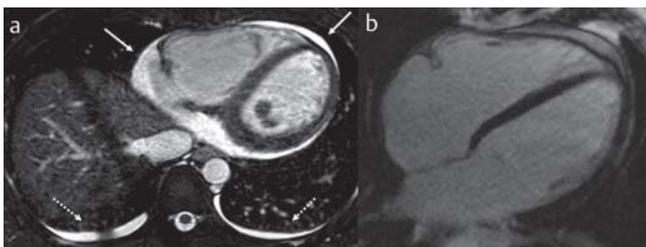
In the last decades tragic reports of SCD in highly trained athletes have received more and more attention. In most of the cases, cardiovascular diseases such as HCM, myocarditis, ARVD, coronary anomalies and even coronary artery disease are either retrospectively identified or have been ignored by doctors or the athletes [1, 2]. This has evoked increasing interest in the determination of such acquired or congenital cardiovascular diseases in athletes. In this context, the role of CMR examinations in the screening process is increasingly discussed, especially since routine testing, even in combination with echocardiography may fail in the detection of cardiovascular diseases such as coronary anomalies and subtle myocardial fibrosis that can be found in approximately 30% of patients with DCM [23]. This is of major importance as the differentiation between “athlete’s heart” – characterized by cavity dilatation and eccentric myocardial hypertrophy – and DCM or mild forms of HCM can be difficult. In fact, MRI and especially delayed enhancement imaging may be particularly helpful in making that distinction.

As expected, ventricular volumes and functional parameters as assessed in our cohort are increased for the left and right ventricular EDV, ESV and SV as well as MM in highly-trained athletes as opposed to a non-athlete population [6–8, 24–26]. The remodeling indexes were within normal ranges [8], which is consistent with eccentric hypertrophy. Of note, the remodeling index is reduced in cases of isolated cavity dilatation and increased in



**Fig. 5** Delayed enhancement imaging 10 minutes after application of contrast medium of a 47-year-old female cyclist (**a, b**) and a 50-year-old male cyclist (**c, d**), both with a training load of 15 hours per week. We found disseminated and intramural myocardial hyperenhancement representative for a non-ischemic type pattern and therefore indicative of a previous myocarditis, in both cases without any wall motion abnormalities.

**Abb. 5** Delayed-Enhancement-Bildgebung 10 Minuten nach der Injektion von Kontrastmittel, in **a, b** von einer 47-jährigen Radfahrerin und in **c, d** von einem 50 Jahre alten, männlichen Radfahrer, jeweils mit einem Trainingspensum von 15 Stunden pro Woche. Hier fanden sich in beiden Athleten disseminierte, intramyokardiale Signalalterationen vom nicht-ischämischen Verteilungsmuster als Zeichen einer stattgehabten Myokarditis, in beiden Fällen ohne Nachweis von entsprechenden regionalen Wandbewegungsstörungen.



**Fig. 6** **a** ECG-gated 3-D MR angiography of a 29-year-old triathlete reveals pericardial effusion (arrows) and pleural effusions (dotted arrows), but no wall motion abnormalities or **b** delayed enhancement of the myocardium. On presentation, the athlete previously experienced a feverish infection, which turned out to be an Epstein-Barr virus infection. 6 weeks later the athlete underwent a control MRI examination which showed complete recovery.

**Abb. 6** **a** EKG-getriggerte 3-D-MR-Angiografie eines 29-jährigen Triathleten mit Nachweis eines Perikardergusses (Pfeile) sowie von Pleuraergüsse (gepunktete Pfeile). Regionale Wandbewegungsstörungen oder **b** pathologische Signalalterationen in der delayed enhancement-Bildgebung fanden sich nicht. Anamnestisch gab dieser Athlet einen kürzlich durchgemachten, gripptalen Infekt an, welcher sich als Epstein-Barr-Virus Infektion erwies. Eine im Abstand von 6 Wochen durchgeführte Verlaufskontrolle zeigte eine vollständige Rückläufigkeit der vorbeschriebenen Befunde.

cases of concentric LV hypertrophy, which is pathognomonic for HCM [19]. Furthermore, the thickness of the interventricular septum was within normal range and there was no asymmetric thickening of the myocardial wall or typical delayed enhancement pattern indicative of HCM [27]. This suggests that the enlargement of the ventricles and the eccentric hypertrophy is a function of physiological changes caused by endurance training. In this context, Petersen et al. [10] showed that physiological LV hypertrophy in athletes can reliably be distinguished from pathological LV hypertrophy such as in HCM. However, the diagnosis of mild forms of HCM is still challenging. Therefore, athletes with a positive family history of HCM, which often shows an autosomal dominant inheritance, should be closely monitored.

The second most common cardiovascular cause of SCD in highly trained athletes is a coronary anomaly [18]. In our study we were able to identify one athlete suffering from the most frequent [28] but benign variant of coronary anomaly, notably a circumflex artery originating from the right sinus (◀ Fig. 2). Another athlete showed an RCA originating from the left corner of the right sinus which is formally not considered as a coronary anomaly but might be affected during exercise due to the long course of the RCA between the aorta and pulmonary trunk (◀ Fig. 3). However, both athletes had no signs of myocardial ischemia during exercise testing. Also a perfusion defect of the myocardium in an additionally performed adenosine-induced stress perfusion MRI was ruled out in the athlete with the abnormal course of the circumflex artery.

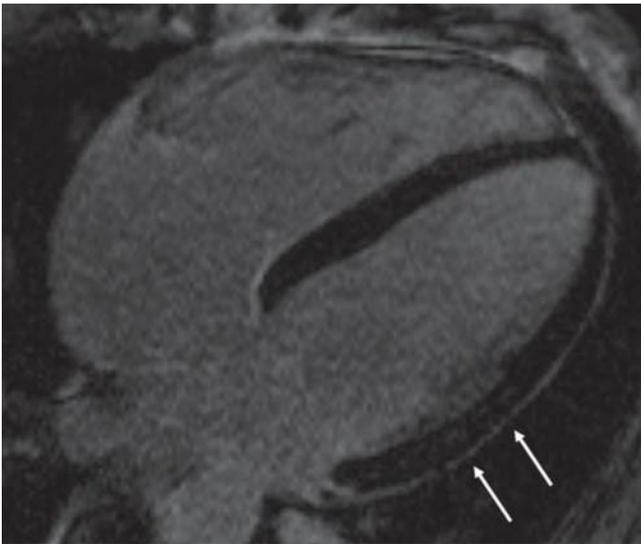
Myocarditis is known to be another common etiology for SCD in young athletes. We found disseminated and intramural myocardial hyperenhancement in two cases. While both athletes denied having a history of cardiovascular disease, this pattern is characteristic of a non-ischemic type and indicates remote myocarditis [19] (◀ Fig. 5).

As the typical patterns of hyperenhancement in acute myocarditis may decrease during healing and can be almost invisible after recovery [19], false-negative examinations cannot be excluded. With this problem in mind, we identified a restrained, non-ischemic enhancement in a further three athletes (◀ Fig. 7). However, as these observations could not be confirmed in a second plane and none of these athletes showed wall motion abnormalities, the findings were judged to be non-significant. The follow-up MRI examinations performed in the athlete with Epstein-Barr infection revealed complete recovery of the pericardial effusion and no ventricular function impairment.

### Prognostic significance of abnormal MRI findings in athletes

Although the presence of delayed enhancement has prognostic significance in the context of cardiomyopathy [29] and viral myocarditis [30], it remains unclear whether the concept may be transferred to asymptomatic athletes with delayed enhancement of non-ischemic origin. More than two years after the MRI, both athletes are still performing their activity (mountain biking) without any symptoms. Holter-ECG was performed in one of these athletes twice (the other athlete refused additional testing) and no relevant heart rhythm disorders could be detected.

The relevance of coronary anomalies in asymptomatic persons is still under debate. Coronary anomalies are disproportionately common in young athletes suffering from SCD. However, the athlete in our study was 46-years-old and had been performing high-intensity training for several years. Whether this athlete has a higher risk for SCD even in the absence of symptoms like



**Fig. 7** Delayed enhancement imaging of a 36-year-old runner with a training load of 20 hours per week shows disseminated and intramural myocardial hyperenhancement of the lateral wall (arrows). These findings could not be confirmed in a second plane and were therefore judged to be non-significant.

**Abb. 7** In der Delayed-Enhancement-Bildgebung eines 36-jährigen Läu-fers mit einem Trainingspensum von 20 Stunden pro Woche fanden sich disseminierte, intramyokardiale Signalalterationen im Bereich der lateralen Wandsegmente (Pfeile), welche sich jedoch nicht zuverlässig in einer 2. Ebene bestätigen ließen und somit als nicht-signifikant eingestuft wurden.

fainting or proven myocardial ischemia remains to be seen. Ultimately, none of the above-mentioned athletes was restricted from sports competition.

### Study Limitations

Certainly, this study has some major limitations. A control collec-tive consistent of non-athletes was not examined and the quanti-tative analyses of the RV were performed using short axis slices. No additional axial slices were acquired. Moreover, a work-up (adenosine-stress MRI, Holter-ECG, repeated MRI) was only per-formed in the case of pathological findings. Thus, it remains un-clear whether athletes with an initially unremarkable MRI would present abnormal findings during further examinations.

### Conclusion

Routine implementation of CMR imaging in the context of pre-participation screening of highly-trained athletes revealed ab-normal findings in more than 5 percent of all athletes and is a useful tool in the detection of cardiovascular disease. However, the prognostic significance of these findings remains unclear. Thus, cardiac MRI cannot be recommended as a routine examina-tion in the care of athletes.

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