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Impact of papillary muscles on ventricular function measurements in 3T cardiac magnetic resonance

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Kontext: Starší studie prokázaly, že se objem, hmota a funkce levé (LK) a pravé komory (PK) srdeční významně liší podle toho, zda se při výpočtu těchto parametrů zahrnou svalové trámce a papilární svaly (trabeculae papillary and papillary muscle, TPM).

Metody: V kohortě 101 pacientů bylo provedeno vyšetření srdce magnetickou rezonancí (cardiac magnetic resonance, CMR). Kohorta zahrnovala celkem 26 pacientů bez patologických nálezů na CMR (referenční skupina), pacienty s ischemickou kardiomyopatií (IKMP), s dilatační kardiomyopatií (DKMP) a pacienty s hypertrofií levé komory (vždy po 25 pacientech). Parametry LK a PK se stanovovaly pomocí dříve zavedených metod: 1. metoda zahrnující TPM a 2. metoda nezahrnující TPM do objemu příslušného srdečního oddílu.

Výsledky: Ve srovnání s výpočtem při zahrnutí TPM znamenalo vyloučení TPM z celkového objemu LK a PK významně menší objem na konci diastoly a systoly (end-diastolic and end-systolic volume, EDV, ESV) a hmotu myokardu, a vyšší hodnoty tepového objemu (stroke volume, SV) a ejekční frakce (EF) ($p = 0.001$). Podíl TPM na hmotě LK a PK byl větší u DKMP ($18,4 \pm 3,8 \%$) a IKMP ($17,8 \pm 3,2 \%$) než u referenční skupiny ($15,2 \pm 2,5 \%$; obojí $p < 0,05$); výsledkem byl statisticky významně větší rozdíl mezi oběma použitými metodami (1. metoda – 2. metoda) při výpočtu ESV, EDV, SV, EF a hmoty myokardu u nemocných s DKMP a IKMP oproti referenční skupině.

Závěr: Hodnoty parametrů LK při vyšetření srdce MR významně ovlivňuje to, zda jsou TPM považovány za součást krevního poolu LK nebo za součást hmoty LK. Aby se zabránilo chybné interpretaci výsledků vyšetření a chybným rozhodnutím, může být při dlouhodobém sledování pacientů naprosto zásadní systematické používání jedné z obou metod delineační LK.

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ABSTRACT

Background: Prior studies revealed, that left and right ventricular (LV, RV) volume, mass and function differ significantly, depending on trabeculae papillary and papillary muscles (TPM) have been included or excluded in LV and RV calculations.

Methods: A cohort of 101 patients underwent CMR. It constituted of 26 patients without pathological findings in CMR (reference group), patients with ischemic cardiomyopathy (ICM), dilated cardiomyopathy (DCM) and patients with left ventricular hypertrophy (25 per group). Left and right ventricular parameters were determined using previously established methods: Method 1 inclusion and method 2 exclusion of TPM in cavity volume.

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Results: Compared with inclusion of TPM, exclusion of TPM in the LV and RV cavity volume resulted in significantly lower end-diastolic and end-systolic volume (EDV, ESV) and myocardial mass, and larger stroke volume and ejection fraction (SV, EF) ($p = 0.001$). The fraction of the TPM on the LV and RV mass was highest in DCM ($18.4 \pm 3.8\%$) and in CM ($17.8 \pm 3.2\%$) compared to the reference group ($15.2 \pm 2.5\%$, both $p < 0.05$), which resulted in a significant larger difference between the two methods (method 1 – method 2) in calculating ESV, EDV, SV, EF and myocardial mass among DCM and ICM patients vs. reference group.

Conclusion: Global CMR LV parameters are significantly affected by whether TPM are considered as part of the LV blood pool or as part of LV mass. Therefore, a consistent method of LV cavity delineation may be crucial during longitudinal follow-up to avoid misinterpretation and erroneous clinical decision-making.

Introduction

Studies have shown that ventricular ejection fraction, volumes and masses are strong therapeutic and prognostic parameters which play decisive roles in the classification of cardiac diseases [1–3]. Echocardiography is a wide-spread tool for the assessment of ventricular function in clinical practice due to its quick and economical availability. However, disadvantages lie in the high inter-observer variability, cases of insufficient acoustic window and error-prone calculations of functional parameters due to difficulty in visualizing the endocardial border and delineation. In echocardiography, essential myocardial parts such as ventricular lumen protruding trabeculae and papillary muscles (TPM) were excluded from ventricular measurements and regarded as additional blood volume [4,5]. Cardiovascular magnetic resonance (CMR) provides high temporal and spatial resolution and has emerged as a diagnostic and clinical standard for the assessment of ventricular function being more superior to echocardiography [6–8]. CMR is capable of distinguishing reliably between myocardium, trabeculae and papillary muscles, raising the question how these structures are rated for the calculation of cardiac function parameters [9,10]. The influence of the incorrect definition to include them into blood volume is discussed controversially and in particular concerning clinical consequences. Literature remains controversial. On the one hand, significant differences between in- and exclusion of papillary muscles were described. On the other hand only small differences were found, thereby leading to the recommendation for exclusion these structures in order to conserve time for CMR analysis [11,12]. Nevertheless, the correct determination of cardiac volumes and ejection fractions remains a time consuming procedure. Semi-automatic evaluation algorithms which allow an exact contour detection of epi- and endocardial borders with consideration to trabeculae and papillary muscles are still unknown [13]. Commercial evaluation software must be corrected manually [10]. In clinical TPM are not consequently in- or excluded in analysis of cardiac function, neither in CMR nor in echocardiography by hand and semi-automatic. Even normal values of cardiac function parameters were created without uniform handling of the inclusion of trabeculae and papillary muscles into ventricular volume [14–20]. Standardized recommendations are missing even though CMR is declared being golden standard of cardiac volumetry [21–28]. Therefore, the present study aims to analyze the importance of trabeculae and papillary muscles for the calculation of ventricular function parameters in 3T CMR with results

leading to recommendations on a uniform approach for a daily routine.

Methods

Study population

101 consecutive patients were prospectively enrolled. All were referred to our institution for cardiac CMR with question of myocarditis or relevant coronary artery disease to perform perfusion CMR. Patients were divided into four groups: 1) subjects without pathological findings in CMR, who constituted the reference group (RF), 2) ischemic cardiomyopathy (ICM, patients with known coronary artery disease assessed by coronary angiography and impaired left ventricular ejection fraction [LVEF] $< 50\%$), 3) dilated cardiomyopathy (DCM, missing relevant coronary artery disease, LVEF $< 50\%$ and dilated cardiac cavities with an end-diastolic volume/body surface area of $57\text{--}105\text{ ml/m}^2$ in men and $56\text{--}96\text{ ml/m}^2$ in women), and 4) patients with left ventricular hypertrophy (LVH, normal LVEF and myocardial mass $> 85\text{ g/m}^2$ in men and 81 g/m^2 in women) based on the TPM inclusion method [14,17]. The exclusion criteria included the following contraindications for CMR (claustrophobia, intracranial clips, pacemaker, defibrillator, ferromagnetic prosthesis, clips, foreign particles, cochlear implants, subcutaneous metalliferous injection systems, pregnancy in first trimester and permanent makeup) and intolerance to lying supine, non rate-controlled atrial fibrillation with a heart frequency of > 100 beats per minute. Before undergoing CMR, all subjects gave written informed consent and the study was approved by the institutional ethical board.

Cardiac magnetic resonance

CMR was performed on a 3.0 Tesla magnetic resonance system (Signa HDxt 3.0T, General Electrics, USA) using an 8-channel cardiac coil. Real time scout images in sagittal, axial and coronal planes were obtained to localize cardiac position in the thorax. Balanced steady-state-free-precession sequences from the ventricular apex to the base were assessed in the short axis view triggered by electrocardiogram during breath-hold. Parameters were as follows: echocardiography time: 1.5 ms, repeat time = 3.8 ms, slice thickness: 8 mm, slice distance: 2 mm, acceleration factor: 2, bandwidth 125 kHz, field of view: $38 \times 38\text{ cm}$, matrix (frequency \times phase): 224×224 , frames: 16 (typical temporal resolution of 46 ms), flip angle: 45° , phase encode grouping: 6–10, 8–12 short-axis slices were needed to encompass the left right ventricle. Manual tracing of epi- and

endocardial borders of the left and right ventricle in the contiguous short-axis slices at the end diastole (first cine phase of the R-wave triggered acquisition) and the end systole (image phase with smallest cavity area) were carried out for calculation of the ventricular end diastolic volumes and ventricular end systolic volumes using the slice summation method, from which ventricular ejection fractions and masses were derived. Two different methods were applied. In method 1, trabeculae and papillary muscles were included to ventricular volume (Fig. 1) and excluded in method 2 (Fig. 2). Both methods were compared concerning end-diastolic and end-systolic left and right ventricular volumes, ejection fractions, stroke volumes and masses. All measurements were performed with commercially available software (Report CARD 4.0, General Electric Healthcare, Waukesha, Wisconsin, USA). Cardiac risk factors were also matched within the patient groups.

Statistical analysis

Statistical analysis was completed with Microsoft Excel 2010 and Stata 11.2. Categorical characteristics were presented in absolute and relative frequencies. Mean values and standard deviations [20] were used for description. In categorical characteristics exact Fisher-Test was used to display whether special characteristic and group membership were independent. In metrical characteristics the Kruskal-Wallis test provided information on distribution of a characteristic within the four groups were identical. Values of patient groups in pairs were compared using the

Mann-Whitney-U-Test. Wilcoxon Sign-rank test for paired samples proved whether results for method 1 and 2 were identical. A p -value of 0.05 was used as the cut-off value for statistical significance. Inter- and intra-observer variability was obtained by analysis of two independent, blinded, experienced observers and again 7 days after first analysis. For this purpose the Wilcoxon Sign-rank test for paired samples and Bland-Altman analysis were performed.

Results

Epidemiological data

101 patients (23 female) had a mean age of 54.8 ± 15.3 years. The cardiovascular risk factors of all groups are displayed in Table 1.

Method comparison

Wilcoxon Sign-rank test for paired samples proved significant differences between method 1 and method 2 ($p < 0.01$) in all parameters (EDV, ESV, SV, EF) for both, left and right ventricular measurements. A detailed comparison between the four patient groups is displayed in Tables 2 and 3.

Volumetry and masses

Left ventricle

In determining EDV, ESV, SV and EF, we observed significant differences between method 1 and 2 between all groups ($p < 0.05$) (Table 3).

Table 1 – Distribution and comorbidities of the study population.

	RF (N 26)		ICM (N 25)		DCM (N 25)		LVH (N 25)		All (N 101)	
	n	%	n	%	n	%	n	%	n	%
Hypertension	4	15.4	20	80.0	13	52.0	23	92.0	60	59.4
Diabetes	0	0.0	15	60.0	4	16.0	3	12.0	22	21.8
DLP	10	38.5	20	0	15	60.0	16	64.0	61	60.4
Smoking	2	7.7	9	36.0	7	28.0	8	32.0	26	25.7
MI	0	0.0	25	100	0	0.0	1	4.0	26	25.7
CAD	0	0.0	25	100	2	8.0	9	36.0	36	35.6
Overweight	2	7.7	8	32.0	6	24.0	7	28.0	23	22.8

CAD – coronary artery disease; DCM – dilated cardiomyopathy; DLP – dyslipoproteinemia; ICM – ischemic cardiomyopathy; LVH – left ventricular hypertrophy; MI – myocardial infarction; RF – reference group.

Table 2 – Methodologic differences in calculated indices of the left ventricle among the study population.

Method	RF		DCM		ICM		LVH	
	1	2	1	2	1	2	1	2
EDV in ml	162.7 ± 38.5	137.1 ± 34.7	280.9 ± 97.4	235.5 ± 85.8	231.8 ± 75.8	194.7 ± 66.4	178.6 ± 52.6	146.0 ± 46.1
ESV in ml	62.6 ± 20	46.8 ± 15.9	209.0 ± 99.0	170.1 ± 88.2	164.1 ± 76.0	130.5 ± 66.0	67.0 ± 28.9	49.5 ± 21.7
SV in ml	100.1 ± 23	90.4 ± 23.6	71.9 ± 20	65.4 ± 19.7	67.7 ± 21.6	64.2 ± 20.1	111.6 ± 32.8	96.5 ± 31
EF in %	61.8 ± 5.7	66.0 ± 5.8	28.5 ± 11.5	31.3 ± 13.5	30.6 ± 10.3	34.5 ± 10.9	63.4 ± 10.2	66.6 ± 10.0
MM in g	122.0 ± 31.2	143.6 ± 34	196.1 ± 54.	240.3 ± 65.5	174.8 ± 53.4	212.0 ± 62.9	197.7 ± 40.2	224.0 ± 46.8

Data presented as mean ± standard deviation.

DCM – dilated cardiomyopathy; EDV – end-diastolic volume; EF – ejection fraction; ESV – end-systolic volume; ICM – ischemic cardiomyopathy; LVH – left ventricular hypertrophy; MM – myocardial mass; RF – reference group; SV – stroke volume.

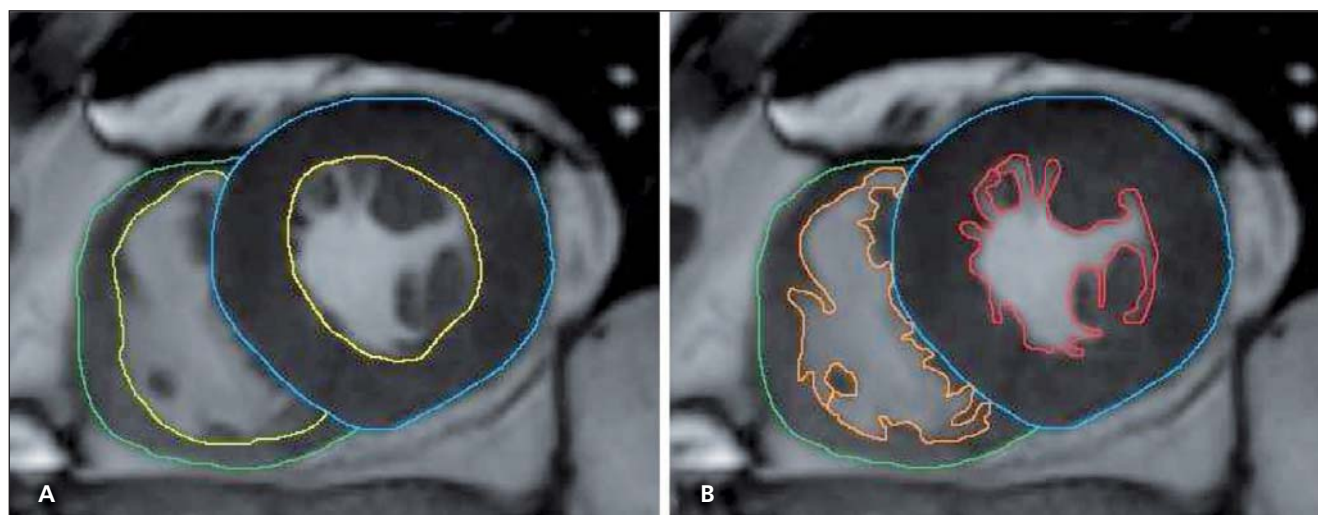


Fig. 1 – Representative short axis images (obtained at mid ventricular level) demonstrating the two methods 1 and 2 employed for determination of left ventricular volumes, ejection fraction, and myocardial mass. (A) Method 1, inclusion of trabeculae and papillary muscles (TPM), (B) method 2, exclusion of TPM from LV cavity volume.

Table 3 – Methodologic differences in calculated indices of the right ventricle among the study population.

Method	RF		DCM		ICM		LVH	
	1	2	1	2	1	2	1	2
EDV in ml	154.1 ± 41.7	136.1 ± 37.4	187.2 ± 44.8	160.8 ± 41.5	143.9 ± 48.8	120.8 ± 40.0	167.2 ± 51.8	143.7 ± 47.8
ESV in ml	75.0 ± 26.4	62.3 ± 23.8.9	127.9 ± 48.1	160.8 ± 41.5	77.2 ± 42.1	61.9 ± 35.1	78.5 ± 26	63.6 ± 23.0
SV in ml	79.1 ± 25.9	74.0 ± 23.5	59.4 ± 20.2	55.3 ± 19.0	66.7 ± 20	58.9 ± 19.3	88.7 ± 32.2	80.0 ± 31.4
EF in %	51.5 ± 9.3	54.6 ± 9.3	33.4 ± 12.9.5	36.1 ± 13.3	40.6 ± 14.4	49.7 ± 12.9	53.1 ± 8.1	55.6 ± 9.0
MM in g	38.0 ± 9.4	53.3 ± 12.2	52.6 ± 13.5	77.0 ± 18.75	41.0 ± 15.7	60.2 ± 24.1	45.7 ± 12.6	64.9 ± 15.7

Data presented as mean ± standard deviation.

DCM – dilated cardiomyopathy; EDV – end-diastolic volume; EF – ejection fraction; ESV – end-systolic volume; ICM – ischemic cardiomyopathy; LVH – left ventricular hypertrophy; MM – myocardial mass; RF – reference group; SV – stroke volume.

Table 4 – Pair-by-pair comparison of the difference of both methods (method 1 and method 2).

	EDV	ESV	SV	EF	MM
LV					
RF vs. ICM	0.000	0.000	0.000	0.250	0.000
RF vs. DCM	0.000	0.000	0.154	0.014	0.000
RF vs. LVH	0.007	0.580	0.000	0.067	0.057
ICM vs. DCM	0.034	0.105	0.077	0.056	0.055
ICM vs. LVH	0.220	0.000	0.000	0.271	0.001
DCM vs. LVH	0.002	0.000	0.000	0.475	0.000
RV					
RF vs. ICM	0.098	0.239	0.048	0.115	0.127
RF vs. DCM	0.001	0.000	0.965	0.142	0.000
RF vs. LVH	0.005	0.151	0.010	0.493	0.013
ICM vs. DCM	0.104	0.004	0.100	0.840	0.009
ICM vs. LVH	0.246	0.775	0.644	0.564	0.271
DCM vs. LVH	0.264	0.003	0.047	0.530	0.063

Data presented as p value.

DCM – dilated cardiomyopathy; EDV – end-diastolic volume; EF – ejection fraction; ESV – end-systolic volume; ICM – ischemic cardiomyopathy; LV – left ventricle; LVH – left ventricular hypertrophy; MM – myocardial mass; RF – reference group; RV – right ventricle; SV – stroke volume.

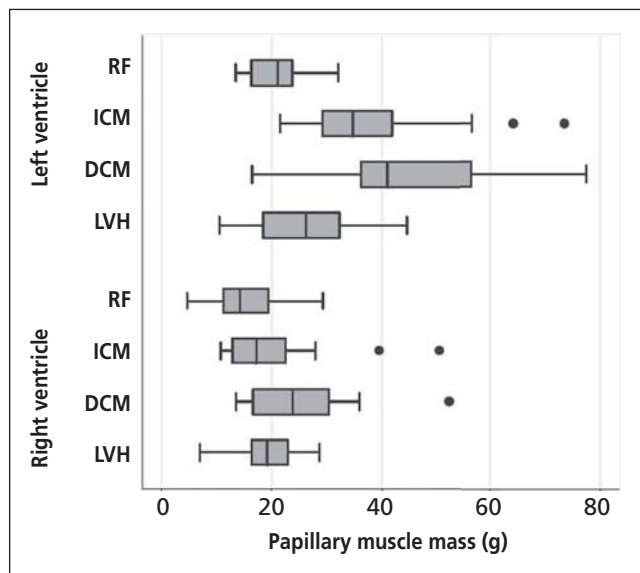


Fig. 2 – Boxplots: Trabeculae and papillary muscles mass in the right and left ventricle. DCM – dilated cardiomyopathy; ICM – ischemic cardiomyopathy; LVH – left ventricular hypertrophy; RF – reference group.

We performed a pair-by-pair comparison on the difference between both methods (method 1 - method 2) for each parameter (EDV, ESV, SV, EF and MM) and patient group (DCM, LVH, ICM and RF). For example, the EDV in DCM (difference of method 1 and method 2) vs. EDV in ICM (difference of method 1 and method 2). This pair-by-pair comparison showed non uniform data (Table 4). For EDV we observed significant differences among all groups except for ICM vs. LVH (p -value 0.22). Conversely it looks for the comparison of EF, whereby a significant difference was obvious only for RF vs. DCM (p -value 0.014). The difference between method 1 and method 2 according to myocardial mass was significantly lower in RF vs. patients with ICM or DCM and LVH vs. ICM and DCM ($p < 0.01$).

Right ventricle

The Wilcoxon Sign-rank test for paired samples proved significant differences between method 1 and method 2 for EDV among RF vs. LVH and DCM patients ($p = 0.001$, 0.005, respectively), for ESV in DCM vs. RF, LVH and ICM patients, for SV in RF vs. DCM and LVH patients and DCM vs. LVH patients. A detailed comparison between the four patient groups are displayed in Tables 2 and 3.

Papillary muscle mass

The data of the papillary muscle mass (PPM) are shown in Fig. 2. The lowest value was seen in the RF (32.2 ± 14.1 g), whereby patients with DCM showed the highest PPM among all groups (44.2 ± 15.1 g) and which showed a significant difference compared to the RF (p -value 0.01) and LVH (p -value 0.01). Patients with ICM did not show significant difference in their PPM compared to those in DCM patients (p -value 0.051).

The average volume of TPM /LV EDV was 23%. In LVH an RF patients this quotient was lowest with $11.6 \pm 2.7\%$, $15.2 \pm 2.5\%$, respectively. A significant higher proportion of TPM to the LV EDV was seen in DCM ($18.4 \pm 3.8\%$, $p < 0.05$) and in ICM ($17.8 \pm 3.2\%$, $p < 0.05$).

Time requirement

The overall time required for left ventricular exclusion of TPM (method 1) was 458.3 ± 72.2 s, for TPM inclusion (method 2) 674.8 ± 148.6 s ($p < 0.05$). A detailed overview is provided in the supplemental data. A subgroup analysis revealed that the time required for TPM inclusion (method 2) was significantly less in the RF group compared to ICM, LVH and DCM patients ($p < 0.05$). Among ICM, LVH and DCM patients no significant difference was evident.

The time difference between method 2 and 1 was significant less for the right ventricle (135.5 ± 41.5 s) compared with marking of the left ventricle (216.5 ± 88.3 s, $p < 0.05$).

Reproducibility of TPM measures

Assessment of the intra- and inter-observer variability showed acceptable levels of agreement for both methods, using LVEDV and EF as example calculations. The Bland Altman graphs and coefficients of repeatability are shown in the supplemental data.

Discussion

A substantial portion of the myocardial mass consists of papillary muscles and trabecular tissue. Normally, the tissue is not included in the quantification of LV function, diameter and mass with most of the in vivo cardiac imaging methods used to evaluate cardiac function. In this study, we demonstrate that the choice of including or excluding papillary muscles and trabeculations in LV mass measures can lead to large differences in the quantification of LV function, diameter and mass. Global CMR LV parameters are significantly affected by whether TPM are considered as part of the LV blood pool or as part of LV mass.

Animal studies have demonstrated that CMR quantification of TPM-inclusive myocardium provides highly accurate LV mass measurements of true necropsy-validated measurements. Fieno et al. reported in a study on dogs undergoing CMR prior to sacrifice that inclusion of papillary muscles within myocardial contours yielded CMR measurements of LV mass that were within 2.1% of necropsy weights [29]. In a similar animal study in which CMR was performed immediately prior to sacrifice, François et al. reported that inclusion of papillary muscle volumes within myocardial contours yielded LV mass calculations that were within 1.2% of necropsy weights [30]. In this study, exclusion of papillary muscles produced mass calculations that were 7.7% lower than necropsy verified values.

From clinical perspective, this leads to some implications. Previous population based CMR studies among healthy subjects have reported normative cutoffs that differ by as much as 16% even after adjustment for sex and body habitus [18,31]. The main difference in these studies was the methodological assignment of TPM, with the exclusion of TPM from the total myocardium resulting in lower normative cutoffs.

These differences are not only evident among individuals free of cardiovascular disease. Our data demonstrate significant differences between the inclusion or exclusion of papillary muscles for patients with DCM, ICM and LVH across a range of parameters. While the impact of our analysis approach was modest in the control subjects, the magnitude of

these differences was more pronounced in the LVH cohort. These findings correspond well with postmortem observations by Estes et al. and Roberts and Cohen, who reported a proportional hypertrophy of the papillary muscles in patients with LV hypertrophy and systemic hypertension [32,33].

Similarly, Janik et al. showed that the relative impact of trabeculae and papillary muscles exclusion on calculated mass and ejection fraction was increased among patients with hypertrophy-associated left ventricular remodeling in a CMR based study [34].

To our knowledge for the first time we were able to show the linear proportional relationship between papillary muscle mass and total LV mass may not be valid in other clinical circumstances (DCM, ICM). Interestingly, the fraction of TPM on the LV mass was highest in DCM and ICM patients. One could suggest that necrosis or fibrosis of the myocardium in the event of myocardial infarction, myocarditis, or low cardiac output lead to a higher fraction of TPM on the LV mass.

Whether TPM should be considered in the calculation of ventricular mass or volume is a matter of controversy that has already been discussed in the literature. Lorenz et al. supported the inclusion of TPM, which appeared to be attached to ventricular wall [15]. Rominger et al. determined that papillary muscles and trabeculations, including the moderator band, should belong to the ventricular lumen [35]. Young et al. and Pattynama et al. subtracted the papillary muscles for the determination of left the ventricular volumes [36,37].

In the future, a re-evaluation of cardiac MRI-based LVEF cut-off values is needed, which is described in the following passages. The main eligibility criterion for primary prevention implantable cardioverter defibrillator (ICD) therapy is a left ventricular ejection fraction $\leq 35\%$ [38]. This is largely based on studies using echocardiography, however, guidelines do not recommend a method for LVEF assessment. A number of studies showed that LVEFs assessed by CMR are consistently smaller than those assessed by 2D echocardiography [39,40]. Furthermore Rinjers et al. showed, that cardiac MRI-LVEF assessment resulted in more patients eligible for ICD implantation compared with 2DE which showed a relatively low event rate during follow-up. The event rate in patients with cardiac MRI-LVEF $\leq 30\%$ was comparable with patients having a 2DE-LVEF $\leq 35\%$. Therefore we think, that there is a need for re-evaluation of cardiac MRI-based LVEF cut-off values for ICD eligibility. For the resetting of cut-off values when CMR is used, we strongly recommend the use the TPM inclusion method (method 2 in this study). Our data indicate, that this method is capable of determining LV function, mass and diameters more accurately not only in healthy subjects, particularly in ICM and DCM patients, where the influence of TPM on the above mentioned parameters is more pronounced. Furthermore, the time required for marking TPM, is only slightly increased compared to TPM exclusion.

Limitation

First, the sample size of 25 patients in each group is relatively small. However, we believe that significant differences in volume, diameter and function were observed. Pa-

pavassiliu et al. found that only 12 patients were needed to establish a power of 90% for the comparison between the inclusion and exclusion methods [41].

Conclusion

Global CMR LV parameters are significantly affected by whether TPM are considered a part of the LV blood pool or part of LV mass. Our cross-sectional data from reference group, DCM ICM and LVH patients demonstrate that TPM/EDV is significantly related to the underlying disease. Therefore, a consistent method of LV cavity delineation may be crucial during longitudinal follow-up to avoid misinterpretation and erroneous clinical decision-making.

Conflict of interest

No conflict of interest.

Funding body

No funding.

Ethical statement

All persons included in this study were recruited and examined according to ethical standards. The ethics committee approved the whole study (Number 369122009), and all participants provided their signed informed consent for the examination and all procedures.

Informed consent

All persons included in this study signed informed consent.

Appendix A – Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.crvasa.2016.06.002.

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