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Left Ventricular Trabeculae and Papillary Muscles: Correlation With Clinical and Cardiac Characteristics and Impact on Cardiovascular Magnetic Resonance Measures of Left Ventricular Anatomy and Function

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Abstract

Objective—We sought to assess the relationship of left ventricular (LV) trabeculae and papillary muscles (TPM) with clinical characteristics in a community-based, free living adult cohort and to determine the effect of TPM on quantitative measures of LV volume, mass and ejection fraction (EF).

Background—Hypertrabeculation has been associated with adverse cardiovascular events, but the distribution and clinical correlates of the volume and mass of the TPM in a normal left ventricle have not been well characterized.

Methods—Short-axis cine cardiovascular magnetic resonance (CMR) images, obtained using a steady-state free precession sequence, from 1494 members of the Framingham Offspring cohort were analyzed using software that automatically segments TPM. Absolute TPM volume, TPM as a fraction of end-diastolic volume (TPM/EDV), and TPM mass as a fraction of LV mass (TPMm/LVM) were determined on all Offspring and in a referent group of Offspring free of clinical cardiovascular disease and hypertension.

Results—In the referent group (aged 61±9 years, with 262 men and 423 women) TPM was 23±3 % of LV EDV in both sexes (p=0.9). TPM/EDV decreased with age (p<0.02) but was not associated with body mass index (BMI). TPMm/LVM was inversely correlated with age (p<0.0001), BMI (p<0.018) and systolic blood pressure (p<0.0001). Among all 1494 participants

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(699 men) LV volumes decreased 23%, LV mass increased 28% and EF increased by 7.5 EF units ($p<0.0001$) when TPM were considered myocardial mass rather than part of the LV blood pool.

Conclusions—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. Our cross-sectional data from a healthy referent group of adults free of clinical cardiovascular disease demonstrate that TPM/EDV decreases with increasing age in both sexes, but is not related to hypertension or obesity.

Keywords

magnetic resonance imaging; population study; trabeculae; papillary muscle; left ventricular ejection fraction

INTRODUCTION

Left ventricular (LV) "hypertrabeculation" has been associated with adverse cardiovascular outcomes (1–3) and with extracardiac disease including neuromuscular disorders (4), but the normal range of trabeculation and papillary muscle mass (TPM) on cardiovascular magnetic resonance (CMR) imaging has not been fully characterized. In addition, the effect of the trabeculae and papillary muscles (TPM) on determination of LV volumes, mass and ejection fraction (EF) has not been assessed in a large, community-dwelling population. Cine steady-state free precession (SSFP) CMR provides high-resolution imaging of the left ventricle, with excellent visual contrast between myocardium and LV blood pool (5,6). These desirable attributes emphasize the papillary muscles and the trabeculae carnae, which were less well seen with other CMR sequences. There is variability in the treatment of TPM with respect to quantification of LV mass (LVM) and EF, important indices with diagnostic and prognostic value. The TPM are often considered part of LV cavity volume, i.e. bloodpool, because this simplifies analysis and has been shown to improve observer reproducibility, particularly with manual tracing of endocardial contours (7). Although this is a reasonable approach for many patients, the proportional impact of TPM may be greater in select patient groups, such as those with hypertrophic cardiomyopathy (8) or markedly impaired LV EF (9).

We sought to determine the relationship of TPM to global LV cavity size, mass and global systolic function metrics in a cohort of free-living adults and to assess the effect of treating TPM as LV bloodpool, versus as myocardial mass, on those LV metrics. We also sought to determine whether TPM varies with sex, age, body mass index (BMI), and history of hypertension or prior adverse cardiovascular events.

METHODS

Study Population

The Framingham Heart Study Offspring cohort was initiated in 1971 and comprises 5124 participants who are the children of the original cohort or the spouses of those children (10). Offspring cohort members were eligible for participation in the FHS CMR substudy if they attended the seventh Offspring cycle examination (1998–1999, N=3799), were in sinus rhythm and had no contraindications to CMR. A total of 1794 Offspring underwent CMR from 2002 to 2006. All participants provided written informed consent and the study was approved by the institutional review boards of the Beth Israel Deaconess Medical Center and the Boston University Medical Center.

Clinical covariates and medication information (blood pressure, height, weight and BMI, antihypertensive drug treatment) were collected in a structured examination by a physician during the seventh cycle examination. Hypertension was defined as a systolic blood pressure

≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg on the mean of two measurements by a physician or use of antihypertensive medication. Data regarding cardiovascular disease events, such as myocardial infarction and heart failure were collected and reviewed. All cardiovascular disease events were adjudicated by a panel of three physicians who were blinded to participant CMR data, using standardized criteria (11).

CMR Imaging

Participants underwent supine CMR scanning using a 1.5-T system with a 5-element cardiac array coil for radiofrequency signal reception (Gyroscan NT, Philips Healthcare, Best, the Netherlands). After scout imaging to determine the orientation of the heart within the thorax, a contiguous stack of short-axis 2D SSFP cine images, encompassing the left ventricle from apex to base, was obtained. Imaging parameters included: TR=3.2 ms, TE=1.6 ms, flip angle=60°. Slice thickness was 10 mm (no interslice gap) with 1.9×1.6-mm in-plane spatial resolution.

Image Analysis

Images were transferred to a dedicated workstation (Extended MR Workspace 2009, Philips Healthcare) for analysis. Epicardial and endocardial LV contours were delineated across the cardiac cycle using an automated contour detection algorithm followed by manual correction if and as needed. The automated contour detection method was applied to the LV short-axis images only, and has been previously described (12). Briefly, it models the short-axis myocardium as a ribbon of variable width, with the inner (endocardial) and outer (epicardial) contours described by interpolation of a minimal number of splines for each. An energy-minimizing criterion that favors smooth, circular shapes, combined with regional constraints favoring homogenous segmentation, results in contours which treat TPM as part of the LV bloodpool. The automated analysis (which can be manually corrected as needed) agrees well with fully manual analysis, with correlation coefficients of 0.99 for LV end-diastolic volume (EDV) and end-systolic volume (ESV), 0.96 for LVM and 0.97 for LV EF (12).

The initial segmentation, which considered TPM as part of LV bloodpool, determined EDV_{INIT}, ESV_{INIT}, EF_{INIT} and LVM_{INIT}. Next, the TPM volume was quantified using a fuzzy thresholding algorithm (12) based on the difference between bright bloodpool and darker myocardial intensities with the threshold determined based on image characteristics. (Thresholds are exam-specific; there are no pre-determined global cutpoints for myocardium versus bloodpool.) Figure 1 shows two examples of TPM segmentation. Although the sample figures are posterized (black or white) to emphasize trabecular fine details, the actual algorithm used to determine TPM volume accounts for partial volume effects. Finally, LV parameters were adjusted (ADJ) by subtracting TPM volume from LV volume at each phase, yielding EDV_{ADJ} and ESV_{ADJ}, and the calculated mass of TPM (TPMm = TPM volume × 1.05 g/ml) was added to LVM, yielding LVM_{ADJ}. LV EF_{ADJ} was computed from EDV_{ADJ} and ESV_{ADJ}. All image data were analyzed by a single experienced operator (CJS) who was unaware of participant characteristics.

Statistical Analysis

Data were analyzed using SAS v9.2 (The SAS Institute, Cary, NC). Continuous data were checked for normality and are summarized as mean±standard deviation. Pearson correlation was used to assess linear relationships between TPM and clinical characteristics. We defined a healthy referent group, free of any history of prior myocardial infarction or heart failure; any wall motion abnormality (of ≥2 anatomically contiguous segments) on CMR; and any history of hypertension to determine the amount of TPM, and its effect on LV EF and LVM, in this putatively normal group. Global LV and TPM measures were compared between the

sexes, and within-sex between referent and non-referent participants using the two-sample t test with equal or unequal variance as indicated. Pairs of unadjusted and adjusted LV parameters were compared using the paired t test. Linear correlations were assessed using Pearson correlation coefficient. Reproducibility was assessed by intraclass correlation coefficient (ICC) on a sub-sample of 48 participants randomly selected from equal strata of sex and age-tertile. A p-value of <0.05 was considered statistically significant.

RESULTS

Study Cohort

Data from 1494 consecutive Offspring undergoing CMR were analyzed for this study. In this cohort, 53 (3.6%) had a documented prior myocardial infarction or heart failure, 125 (8.4%) had a focal LV wall motion abnormality on CMR, and 785 (52.5%) had documented hypertension or use of antihypertensive medication. Application of these exclusion criteria left 262 men (37.5%) and 423 women (53.2%) in the healthy referent group. (A particular subject could have >1 criterion for exclusion from the referent group.) The clinical characteristics of the referent and non-referent groups, and the study population as a whole, are shown in Table 1. There were differences between men and women in anthropometric and blood pressure measures, as expected.

Global LV and TPM Measures

Table 2 shows the global LV parameters in referent-group men and women. In the referent group, EDV_{INIT}, ESV_{INIT}, and LVM_{INIT} were greater in men than women, while EF_{INIT} was greater in women. LVM_{INIT} was linearly correlated with systolic blood pressure (SBP) in referent men ($r=0.26$) and women ($r=0.26$), $p<0.0001$ for both, and across all participants (men: $r=0.21$, women: $r=0.31$, $p<0.0001$ for both). TPM was greater in men than women, but TPM volume as a fraction of EDV_{INIT} (TPM/EDV) did not differ between sexes. TPM mass as a fraction of LVM_{INIT} (TPMm/LVM) was slightly but significantly greater in women than men.

After subtraction of TPM volume (ADJ) we expected EDV to decrease, and LVM to increase with the addition of TPM mass. In the referent group EDV decreased by an average of 23% in both sexes, while LVM increased 30% and LV EF increased by 7.5 EF units after ADJ. Changes of the same direction and similar magnitude were seen post-ADJ across the study group as a whole (Table 2). Post-ADJ LV volumes and mass remained greater in men versus women. LV EF increased after ADJ in both sexes, and EF_{ADJ} remained greater in women than men. EF_{INIT} and EF_{ADJ} were highly correlated with one another among men ($r=0.95$, $p<0.0001$) and among women ($r=0.92$, $p<0.0001$).

Reproducibility of TPM Measures

Intraobserver ICCs were 0.99, 0.99, 0.99 and 0.97 for EDV_{ADJ}, ESV_{ADJ}, LVM_{ADJ}, and EF_{ADJ} respectively. Interobserver ICCs were 0.99 for EDV_{ADJ}, 0.98 for ESV_{ADJ}, 0.99 for LVM_{ADJ}, and 0.96 for EF_{ADJ}.

Variation in Global LV and TPM Measures With Hypertension and Focal LV Dysfunction

Sex-specific comparisons of referent participants to those with history of hypertension (+HTN) and those with wall motion abnormalities (+WMA) are presented in Table 3. In each sex the referent-group participants were younger than those in the +HTN and +WMA groups. As expected, referent participants also had lower BMI and had lower systolic blood pressure than the +HTN or the +WMA groups. EDV_{INIT}, ESV_{INIT} and LVM_{INIT} were greater in the +WMA versus referent groups for both men and women, while EF_{INIT} was lower in the +WMA group. The +HTN group had greater LVM_{INIT} than the referent group;

EF_{INIT} was slightly higher in referent women versus +HTN women, but did not differ among men.

Absolute TPM volume was significantly greater in +WMA versus referent men (p=0.0002); a similar pattern was seen among women, but this was only of borderline significance (p=0.03). TPM/EDV was similar in magnitude among both men and women, with at most borderline decreases associated with +HTN or +WMA. TPMm/LVM was significantly lower in men (p=0.0003) and women (p<0.0001) with +HTN versus their referent counterparts. TPMm/LVM was also lower in +WMA versus referent women (p=0.0046), but not in men. EF_{ADJ} was consistently greater than EF_{INIT} in the referent group as well as in the +HTN and +WMA groups.

Correlation of Clinical Characteristics and LV Indices With TPM

Linear correlations between TPM measures and age, SBP, BMI and global LV parameters are presented in Table 4. Absolute TPM volume was inversely and significantly correlated with age in both sexes for referent participants and for the Offspring as a whole, but the correlation coefficient was attenuated in the referent group as compared with all participants. TPM/EDV was also inversely correlated with age in both sexes, but this was attenuated in the referent group as compared with all participants. TPM/EDV was not associated with BMI in either sex. There was an inverse correlation between TPM/EDV and SBP in men but not in women.

With respect to LV parameters, absolute TPM increased with greater EDV_{INIT} and ESV_{INIT} in both sexes. TPM/EDV was weakly and inversely correlated with EDV_{INIT} and ESV_{INIT} in referent men, but not in men as a whole, nor at all in women. Absolute TPM mass increased with LVM_{INIT} in both sexes, but TPMm/LVM was significantly *inversely* correlated with LVM_{INIT} in both men and women. Finally TPM was inversely correlated with EF_{INIT} in both sexes, with r=−0.30 in men and r=−0.31 in women, p<0.0001 for both, but TPM/EDV was not correlated with EF_{INIT} in either men or women.

Distribution of TPM Measures

Table 5 shows the median, 90^{th} and 95^{th} percentile cut-points, by sex, for TPM, TPM/EDV and TPMm/LVM among referent-group Offspring. The 90^{th} and 95^{th} percentile cut-points of TPM/EDV ratios were similar across the sexes, while TPMm/LVM was slightly greater in women than men for both thresholds. There was minimal difference between referent-group participants and the study group as a whole (data not shown). We elected not to present age-specific percentiles due to limited sample size in several of the age-specific and sex-specific categories.

DISCUSSION

In this CMR study of nearly 1500 community-dwelling members of the Framingham Heart Study Offspring cohort we found that LVM and EF increased significantly when the trabeculations and papillary muscles were considered as myocardial mass, while EDV and ESV significantly decreased, as compared to the corresponding quantities obtained when the TPM were considered as part of the LV bloodpool. We further found that TPM decreased with advancing age, and TPMm/LVM decreased with increasing BMI and systolic blood pressure, in both sexes, in a healthy referent cohort strictly free of obesity, hypertension, and any history of myocardial infarction or heart failure.

In the context of the current literature

Our average TPM volume of 23% of LV EDV is consistent with the literature. However, the majority of prior reports have considered either papillary muscle volume or trabecular volume in isolation, and have not combined them. A canine CMR study by Francois et al (13) found papillary muscle volume represented 7.7% of LVM. In a 100-participant subset of the MESA CMR study, Vogel-Claussen et al (14) found that papillary muscles comprised 8.9±0.1% of LVM. Regarding LV trabeculae, in a study of 20 healthy controls and 20 patients with decreased LV EF, Papavassiliu and colleagues (7) found that trabeculae comprised ~10% of LVM in healthy subjects and 14% in patients with systolic dysfunction. Jacquier et al (15) compared the percentage of trabecular mass to global LVM in 16 controls (12±5%) and the same number of patients, in each group, with hypertrophic cardiomyopathy (12±4%), with dilated cardiomyopathy (11±4%) and those with LV noncompaction (32±10%). Combining the published 8–9% for papillary muscles and ~12% for trabeculations results in findings similar to our data.

As expected, exclusion of TPM from the LV bloodpool and their inclusion with LVM resulted in lower LV volumes and greater mass. These findings are similar to the results of Weinsaft (9) and Papavassiliu (7), who found that EF was significantly greater when TPM were considered myocardial mass, but the magnitude of increase was larger in the present study, at ~7 EF units versus prior results of 3 to 4 EF units. However, the prior studies included subjects with advanced systolic dysfunction, a group not represented in the present study, so direct comparisons of change in EF may not be warranted. Han et al (8) found a 17% increase in indexed LVM, a 20% decrease in indexed EDV and an average 9-unit increase in EF among a cohort of 30 patients with hypertrophic cardiomyopathy. These results are broadly comparable to those of the current study. Also consistent with prior work, we found that TPM increased with greater LVM, and now extend this finding by considering TPM mass as a proportion of total LVM, which actually *decreased* with greater overall mass.

Areas for Additional Study

As this is a cross-sectional study, it is not possible to determine whether changes in total LVM are accompanied by proportional changes in TPM. For example, although it is possible that TPM increases more slowly in response to a pressure stimulus such as hypertension or aortic stenosis, than the remainder of myocardium, it is also possible that those with an initially lower proportion of TPM are more responsive to that stimulus. Although causality cannot be inferred from our cross-sectional study, we note that TPM volume (and thus TPM mass) did not correlate with systolic blood pressure either in the referent group or in with the study group as a whole, although overall LVM was significantly correlated with systolic blood pressure. This suggests that TPM mass may be less responsive to hypertension than overall myocardial mass. The lack of association between TPM mass and systolic blood pressure in our study is consistent with the MESA results (14).

We examined LV mass (and TPM volume) only at end-diastole, as is common practice. Some readers may consider that since TPM is the difference between EDV_{INIT} and EDV_{ADJ}, a similar relationship should hold between TPM, ESV_{INIT} and ESV_{ADJ} due to incompressibility of myocardium, but this not the case, as seen in Table 2. The numerical difference and apparent discrepancy is resolved when one considers that at end-systole a substantial proportion of the trabeculae are already tightly apposed against the compacted myocardium, so that the initial putative endocardial contours already classify some portion of the trabeculae as myocardium. Thus the adjustment for TPM at end-systole captures only a portion of the TPM identified at end-diastole.

Presently LV non-compaction is diagnosed by CMR based on maximal ratio of non-compacted-to-compacted layer thickness. The results of this study do not provide insight as to whether global measures of trabecular burden, such as the TPM measures presented here, or regional measures (such as the aforementioned ratio of non-compacted-to-compacted thicknesses, or perhaps the maximal thickness of the trabeculated layer, or number/location of "hypertrabeculated" segments in the standard 17-segment model, to name some possible examples) will have greater value for diagnosis and prognosis of hypertrabecular disorders of the myocardium.

Clinical Implications

The greater temporal and spatial resolution of current noninvasive imaging techniques, be they SSFP in CMR or second-harmonic imaging in echocardiography (16,17), generally result in better visualization of LV trabeculae as compared with older techniques, e.g. segmented k-space gradient echo sequences in the case of CMR. This can lead to concerns regarding possible LV non-compaction, particularly when first adopting the newer techniques. Thus it is important to determine what constitutes a "normal range" of trabeculation.

Petersen et al (18) have shown that the majority (~60%) of healthy individuals, as well as fit athletes, have marked trabeculations, principally in the apical and select mid-LV segments on SSFP CMR. Interestingly, they noted a somewhat lower proportion of patients with hypertensive heart disease who had segmental hypertrabeculation, an observation which appears broadly in accord with our finding that TPMm/LVM is inversely associated with SBP. Dawson et al (19) used SSFP CMR to study a cohort of 120 healthy subjects 20 to 80 years of age. A trabeculated endocardial layer was present in at least one segment in all subjects, and they found that the end-diastolic thickness of the trabeculated layer decreased with advancing age, broadly consistent with our finding of decreased TPM with greater age. These investigators also found that the epicardial compacted layer was thinner in women versus men, but that there was no sex difference in the thickness of the trabeculated layer. This finding is consistent with the greater TPMm/LVM ratio in women in the present study.

Although our study was based on a large community sample without any participants suspected of having LV non-compaction, we present 90^{th} and 95^{th} percentile upper limits of TPM/EDV and TPMm/LVM in a healthy referent group which may be useful in future studies of LV non-compaction or other disorders associated with abnormal trabecular or papillary muscle volume.

Limitations

The FHS Offspring cohort is overwhelmingly white, and the majority of subjects were middle-aged or older. Thus while our results are valid in whites in the community, they may not be generalizable to other age ranges or ethnic groups. Also, our study population did not include subjects with advanced systolic dysfunction. Finally, our CMR findings may not apply to results obtained with non-SSFP CMR sequences and are unlikely to apply directly to echocardiographic data (20).

Conclusions

Among adults free of clinical cardiovascular disease, LV trabeculations and papillary muscles occupy 23% of LV EDV in both men and women. Considering TPM as myocardial mass (as opposed to LV blood pool) increases LV EF significantly across the entire study cohort. Reference standards for "normal values" and comparisons across serial CMR examinations must take into account not only the CMR imaging sequence used (6), but also the image analysis protocol and its treatment of trabeculations and papillary muscles.

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ABBREVIATIONS

BMI	body mass index
CMR	cardiovascular magnetic resonance
EDV	end-diastolic volume
EF	ejection fraction
ESV	end-systolic volume
LV	left ventricular
LVM	left ventricular mass
SSFP	steady-state free precession
TPM	trabeculae and papillary muscles

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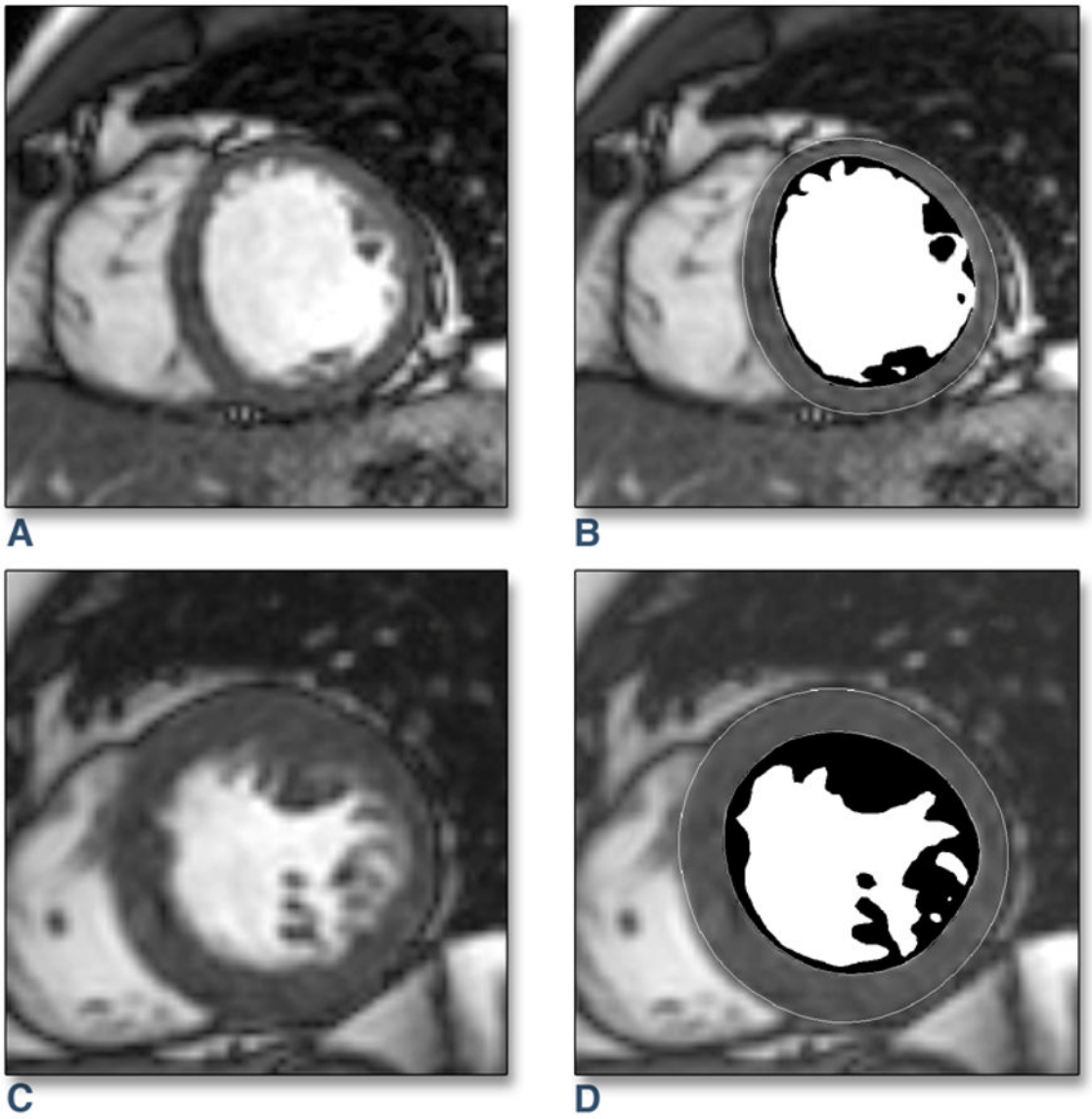

Figure 1. Examples of TPM quantitation

a) end-diastolic mid-LV slice from a relatively lightly trabeculated participant. b) the same slice after endocardial and epicardial border detection and segmentation of TPM; TPM are shown in black and the residual bloodpool (slice EDV_{ADJ}) is shown in white. For this slice EDV_{INIT} = 24.6 ml and LVM_{INIT} = 12.5 g, while slice EDV_{ADJ} =19.9 ml and LVM_{ADJ} = 17.5g. Panels (c) and (d) are from a more heavily trabeculated participant with slice EDV_{INIT} = 21.5 ml and LVM_{INIT} = 17.4 g, following TPM segmentation slice EDV_{ADJ} = 12.1 ml and LVM_{ADJ} = 27.3 g.

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Table 1

Clinical characteristics of the healthy Referent group and the entire study population.

	Men (REF)	Women (REF)	Men (nonREF)	Women (nonREF)	Men_all	Women_all
N	262	423	437	372	699	795
Age, years	60.6±8.5	61.7±8.6*	67.4±8.6††	67.8±8.3††	64.9±9.1	64.6±9.0**
Height, m	1.76±0.07	1.62±0.06**	1.74±0.06†	1.60±0.06††	1.75±0.06	1.61±0.06**
Weight, kg	84.6±14.0	67.9±12.4**	88.7±14.0††	74.7±16.5††	87.2±13.4	71.1±14.8**
BMI, kg/m^2	27.2±3.5	25.8±4.4**	29.1±4.3††	29.0±6.0††	28.4±4.1	27.3±5.4**
SBP, mmHg	117±10	114±12*	132±17††	135±19††	126±17	124±19*
DBP, mmHg	73±11	70±8**	77±11††	75±11††	76±10	72±10*
+HTN, %	N/A	N/A	96.3	97.8	60.2	45.8
+WMA, %	N/A	N/A	21.7	8.1	13.6	3.8
+MIHF, %	N/A	N/A	9.8	2.7	6.2	1.3

REF = healthy referent group, nonREF = participants not in the referent group, BMI=body mass index; SBP, DBP = systolic and diastolic blood pressure; +HTN = presence of any antecedent hypertension or use of antihypertensive medications; +WMA = having a CMR focal wall motion abnormality; +MIHF = positive history of adjudicated myocardial infarction or heart failure. N/A = not applicable; the Referent group was free of HTN, WMA and MIHF by definition.

The * indicates $p<0.05$ between men and women of corresponding groups (e.g. Men (REF) vs. Women (REF)),

† indicates $p<0.05$ within-sex difference between REF and non-REF.

Doubled symbols (** or ††) indicate $p<0.001$.

Table 2

Global left ventricular (LV) parameters in men and women in the healthy referent group and among all participants.

	Referent Men (N=262)	Referent Women (N=423)	P: men vs. women	All Men (N=699)	All Women (N=795)	P: men vs. women
EDV_{INIT}, ml	143±25	107±18	<0.0001	146±29	108±20	<0.0001
EDV_{ADJ}, ml	111±19	83±15	<0.0001	113±24	84±16	<0.0001
ESV_{INIT}, ml	49±13	35±9	<0.0001	52±19	34±11	<0.0001
ESV_{ADJ}, ml	30±10	20±7	<0.0001	32±15	20±8	<0.0001
$LVEF_{INIT}$, %	66.0±5.3	68.0±5.0	<0.0001	65±7	69±6	<0.0001
$LVEF_{ADJ}$, %	73.4±6.5 *	75.6±5.9 *	<0.0001	72±9 *	76±7 *	<0.0001
LVM_{INIT}, g	123±22	81±15	<0.0001	129±26	86±17	<0.0001
LVM_{ADJ}, g	157±26	107±18	<0.0001	163±30	112±20	<0.0001
TPM, ml	33±8	24±5	<0.0001	33±9	24±6	<0.0001
TPM/EDV	0.23±0.03	0.23±0.03	0.90	0.22±0.04	0.23±0.04	0.22
TPMm/LVM	0.28±0.07	0.32±0.06	<0.0001	0.27±0.07	0.30±0.07	<0.0001
TPM/BSA, ml/m^2	16.2±3.9	13.9±2.6	<0.0001	16.7±4.3	14.4±3.1	<0.0001

TPM = trabecular and papillary muscle volume, TPMm = trabecular and papillary muscle mass, INIT = TPM considered as part of left ventricular (LV) bloodpool, ADJ = TPM considered as myocardial mass. EDV=end-diastolic volume, ESV=end-systolic volume, EF=ejection fraction, LVM=LV mass, BSA=body surface area.

* $EF_{ADJ} > EF_{INIT}$, p<0.0001.

Table 3

Global LV parameters in men and women with hypertension or CMR LV wall-motion abnormality compared with the healthy referent group.

Group	Men: REF	Men: +HTN	Men: +WMA	Women: REF	Women: +HTN	Women: +WMA
N	262	421	95	423	364	30
Age	60.6±8.5	67.6±8.5††	67.6±9.1††	61.7±8.6	67.9±8.3††	69.1±8.6††
BMI	27.2±3.5	29.2±4.3††	29.0±4.3††	25.8±4.4	29.2±6.0††	28.6±6.3 †
SBP	117±10	132±17††	128±19††	114±12	136±18††	132±21††
EDV_{INIT}, ml	143±25	147±31	166±34††	107±18	110±21	127±32 †
EDV_{ADJ}, ml	111±19	115±26†	129±29††	83±15	85±18	100±26†
ESV_{INIT}, ml	49±13	52±21 †	76±28††	35±9	33±12	53±20††
ESV_{ADJ}, ml	30±10	33±17†	52±24††	20±7	19±9	35±14††
EF_{INIT}, %	66.0±5.3	65.3±8.4	55.2±9.6††	68.0±5.0	70.2±6.4††	59.7±8.4††
EF_{ADJ}, %	73.4±6.5	72.4±9.7	61.1±11.7††	75.6±5.9	77.6±7.5††	65.8±8.8††
LVM_{INIT}, g	123±22	132±27††	144±29††	81±15	92±18††	103±27††
LVM_{ADJ}, g	157±26	166±32††	182±34††	107±18	118±22††	132±32††
TPM, ml	33±8	33±9	37±10††	24±5	25±6	27±7 †
TPM/EDV	0.23±0.03	0.22±0.04 †	0.23±0.05	0.23±0.03	0.22±0.04	0.22±0.04 †
TPMm/LVM	0.28±0.07	0.26±0.07††	0.28±0.08	0.32±0.06	0.28±0.07††	0.28±0.07 †
TPM/BSA, ml/m^2	16.2±3.9	15.7±4.1	18.0±4.8††	13.9±2.6	13.5±3.2	15.0±3.8†

REF, +HTN, +WMA are as previously defined.

† p<0.05,

†† p<0.001 versus REF within each sex. (Some participants overlap in both the +HTN and +WMA groups.)

Table 4

Pearson correlation coefficients between clinical characteristics, LV parameters by CMR and TPM measures in men and women in the Referent group and the entire study sample.

	Age	BMI	SBP	EDV	ESV	LVM	EF
Referent-group Men (N=262)							
TPM	−0.33**	−0.004	−0.13**	0.80**	0.70**	0.33**	−0.30**
TPM/EDV	−0.15*	−0.08	−0.22**	0.14*	0.15*	−0.12	−0.12
TPMm/LVM	−0.23**	0.023**	−0.31**	0.39**	0.43**	−0.37**	−0.30**
Referent-group Women (N=423)							
TPM	−0.36**	0.27**	−0.017	0.75**	0.65**	0.51**	0.31**
TPM/EDV	−0.11*	0.0096	−0.090	−0.077	−0.022	−0.050	−0.054
TPMm/LVM	−0.24**	−0.12*	−0.27**	0.20**	0.28**	−0.33**	0.46**
All Men (N=699)							
TPM	−0.21**	0.087*	−0.054	0.72**	0.57**	0.34**	0.030**
TPM/EDV	−0.13**	−0.054	−0.10*	−0.024	−0.027	−0.14**	−0.001
TPMm/LVM	−0.19**	−0.17**	−0.20**	0.29**	0.26**	−0.39**	−0.20**
All Women (N=795)							
TPM	−0.28**	0.27**	−0.004	0.70**	0.58**	0.43**	−0.28**
TPM/EDV	−0.12**	0.023	−0.055	−0.07*	−0.04	−0.11*	−0.017
TPMm/LVM	−0.28**	−0.13**	−0.28**	0.17**	0.25**	−0.39**	−0.26**
All Study Participants (N=1494)							
TPM	−0.19**	0.20**	0.007	0.79**	0.68**	0.57**	−0.37**
TPM/EDV	−0.13**	−0.013	−0.079*	−0.053*	−0.043	−0.11**	−0.0001
TPMm/LVM	−0.23**	−0.17**	−0.25**	0.055*	0.10**	−0.42**	−0.15**

*p<0.05,

**p<0.001.

Table 5

Median (50^{th}) and 90^{th} and 95^{th} percentile cut-points for TPM, TPM/EDV and TPMm/LVM among Referent group participants.

	Men			Women		
	50^{th}	**90^{th}**	**95th**	**50^{th}**	**90^{th}**	**95th**
TPM (ml)	32.3	42.7	45.6	23.9	30.1	33.2
TPM/EDV	0.23	0.27	0.28	0.23	0.27	0.28
TPMm/LVM	0.28	0.38	0.40	0.31	0.40	0.42
TPM/BSA (ml/m^2)	16.7	21.8	24.2	14.3	18.4	19.5