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Reproducibility and observer variability of tissue phase mapping for the quantification of regional myocardial velocities

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Abstract

To systematically investigate the reproducibility of global and segmental left ventricular (LV) velocities derived from tissue phase mapping (TPM). Breath held and ECG synchronized TPM data (spatial/temporal resolution = $2 \times 2 \text{ mm}^2/20.8 \text{ ms}$) were acquired in 18 healthy volunteers. To analyze scan–rescan variability, TPM was repeated in all subjects during a second visit separated by 16 ± 5 days. Data analysis included LV segmentation, and quantification of global and regional (AHA 16-segment modal) metrics of LV function [velocity–time curves, systolic and diastolic peak and time-to-peak (TTP) velocities] for radial (V_r), long-axis (V_z) and circumferential (V_Φ) LV velocities. Mean velocity time curves in basal, mid-ventricular, and apical locations showed highly similar LV motion patterns for all three velocity components (V_r , V_Φ , V_z) for scan and rescan. No significant differences for both systolic and diastolic peak and TTP myocardial velocities were observed. Segmental analysis revealed similar regional peak V_r and V_z during both systole and diastole except for three LV segments ($p = 0.045$, $p = 0.033$, and $p = 0.009$). Excellent ($p < 0.001$) correlations between scans and rescan for peak V_r ($R^2 = 0.92$), peak V_z ($R^2 = 0.90$), radial TTP ($R^2 = 0.91$) and long-axis TTP ($R^2 = 0.88$) confirmed good agreement. Bland–Altman analysis demonstrated excellent intra-observer and good inter-observer analysis agreement but increased variability for long axis peak velocities. TPM based analysis of global and regional myocardial velocities can be performed with good reproducibility. Robustness of regional quantification of long-axis velocities was limited but spatial velocity distributions across the LV could reliably be replicated.

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Compliance with ethical standards

Conflict of interest None.

Keywords

Tissue phase mapping; Left ventricle; Function; Velocity; Reproducibility

Introduction

Tissue phase mapping (TPM) (time resolved 2D phase contrast MRI with three-directional myocardial velocity encoding) is a tool to assess changes in left ventricular (LV) function associated with cardiac disease [1–13]. TPM acquires time-resolved (CINE) data over the cardiac cycle and permits the quantification of regional myocardial velocities over the cardiac cycles along all three principal motion directions (radial, circumferential and longitudinal) of the heart. Previous studies have shown the diagnostic value of TPM for the identification of regionally disturbed myocardial function in patients with cardiac disease such as regional mechanical dyssynchrony due to left bundle branch block [14, 15], cardiomyopathy [16, 17], or after heart transplantation [18]. Recent technical advances, such as spatio-temporal k - t imaging acceleration, permit the application of 2D TPM for the assessment of LV velocities during a single breath hold [19].

The most widely adopted method used for the analysis of myocardial motion is to calculate global and regional LV velocities from TPM data based on LV segmentation including the manual delineation of end- and epicardial contours of the myocardium. This approach has been successfully applied in studies investigating changes in global myocardial velocity dynamics as well as segmental LV peak and time-to-peak (TTP) velocities in common cardiac pathologies.

The performance of LV velocity estimation based on 2D TPM has been compared to tissue Doppler echocardiography (TDI) [20] and previous studies have demonstrated good observer variability [7, 17]. However, a detailed analysis of the scan–rescan reproducibility as well as its inter- and intra-observer dependence associated with manual segmentation of the LV is still missing, particularly regarding the quantification of segmental myocardial velocities. The aim of this study was therefore to systematically investigate the scan–rescan reproducibility and observer variability of TPM derived global and segmental LV velocities.

Materials and methods

Study cohort

Eighteen healthy subjects (12 men, 6 women, age = 48.1 ± 10.9 years) with no history of cardiovascular disease and normal cardiac function were enrolled in the study. Each subject underwent two separate MRI examinations (scan 1 and scan 2), using the same imaging protocol (time difference between first and second scan 16 ± 5 days). The study was approved by the local Institutional Review Board (IRB) and informed consent was obtained from all participants.

MR imaging

All measurements were performed on a routine 1.5T MR system (Magnetom Aera, Siemens, Germany). For each participant, a three-plane fast localization sequence was used for anatomic orientation of all images. Four-chamber, two-chamber, and short-axis views were then acquired with a black-blood half-Fourier rapid acquisition with relaxation enhancement sequence to identify anatomy for cardiac images. TPM data were acquired at “base”, “mid” and “apex” of the LV in short-axis orientation using a black-blood prepared 2D gradient echo (GRE) cine phase-contrast MRI sequence with three-directional myocardial velocity encoding as described previously. Spatio-temporal imaging acceleration (k - t parallel imaging PEAK GRAPPA) [19] with a net acceleration factor of 3.6 was employed to permit TPM data acquisition during breath-holding (breath-hold time = 25 heart beats per short axis slice). Other imaging parameters were as follows: Slice thickness = 8 mm, in-plane spatial resolution = $2.0 \times 2.0 \text{ mm}^2$, TR/TE/Flip angle = 5.2 ms/3.4 ms/10°, velocity sensitivity (venc) = 25 cm/s, bandwidth = 650 Hz/pixel, temporal resolution = 20.8 ms/frame.

TPM data analysis

TPM data were processed using an in-house software package programmed in MATLAB (The Mathworks, USA). To segment the LV, epicardial and endocardial LV contours were manually traced in basal, midventricular, and apical short-axis slices for all acquired cardiac time frames. The measured time-resolved velocities in three directions ($V_x(t)$, $V_y(t)$, $V_z(t)$) with the LV segmentation mask were then transformed into radial velocities ($V_r(t)$), circumferential velocities ($V_\Phi(t)$), and long-axis velocities ($V_z(t)$). The resulting velocities were thus adapted to the principal motion directions of the heart and represented LV contraction/expansion (V_r), rotation (V_Φ) in the short-axis of the LV as well as through-plane and lengthening/shortening (V_z). Positive velocities were defined for systolic contraction/shortening/clockwise rotation while negative velocities indicated diastolic expansion/lengthening/anticlockwise rotation.

To investigate the dynamics of LV velocities over the cardiac cycle on a slice-by-slice basis, the temporal evolution of V_r , V_Φ , and V_z was analyzed for basal, midventricular, and apical locations. Mean velocity time-curves for each slice were calculated by averaging over all LV velocities inside the segmented LV boundaries for each time frame in the cardiac cycle.

For segmental analysis, all velocity data were mapped on the standard American Heart Association (AHA) 16-segment LV model by averaging velocities of all pixels within each of the 16 myocardial segments. For radial and long-axis velocity components, segmental systolic and diastolic peak velocities and time-to peak (TTP) velocities were determined for each segment. TTP was defined as the time interval between the R-wave and peak systolic and diastolic velocity, respectively. Mean basal, midventricular, and apical peak and TTP velocities were calculated as averaging over all LV segments in each slice.

Inter- and intra-observer variability

To assess inter-observer agreement data was analyzed by two independent readers, blinded to each other's results for all 18 volunteers. In addition, one reader re-analyzed images with a 1-month interval to evaluate the intra-observer variability of the TPM analysis.

Statistical analysis

All continuous variables were represented by mean $\pm$ one standard deviation (SD). The difference in segmental peak and TTP velocities between scan 1 and scan 2 were compared side-by-side using paired t tests. Linear regressions were applied to investigate the correlations of segmental peak LV velocities between scan 1 and scan 2. Bland–Altman plots were used to assess the intra-, inter-observer and scan–rescan mean differences and limits of agreement. Statistical analysis was performed using SPSS statistical software (Version 13.0, SPSS, Inc, Chicago, IL). A p value <0.05 was considered statistically significant.

Results

The demographics of the study cohort are summarized in Table 1. Heart rate and blood pressures were similar for all subjects between scan and rescan. TPM was successfully acquired in all 18 volunteers twice. All TPM images were eligible for quantitative analysis and resulted in 288 myocardial segments (54 LV slices) for the comparison of velocity data from two TPM scans. Figure 1 shows a typical case of peak systolic myocardial velocities at basal, mid-ventricular, and apical locations for scan and rescan in the same subject.

Average myocardial velocities—base, mid, apex

As shown in Fig. 2, mean velocity time curves on a slice-by-slice basis (representing velocity components at base, mid, and apex) showed excellent agreement between scan and rescan. Highly similar LV motion patterns for all three velocity components (V_r , V_Φ , V_z) can clearly be appreciated. A direct comparison of global peak and TTP velocities for basal, midventricular and apical locations is provided in Table 2 and confirms good agreement for average myocardial velocity parameters: No significant differences for both peak or TTP myocardial velocities between scan and rescan were observed for all short axis location, velocity components (V_r , V_Φ , V_z), or time during the cardiac cycle (systole, diastole).

Segmental myocardial velocities

Side-by-side comparisons of segmental systolic and diastolic peak radial and long-axis velocities for scan and rescan are shown in Fig. 3. Segmental peak velocities were highly reproducible with similar peak V_r and V_z during both systole and diastole except for three segments (peak systolic V_r in apical inferior segment: 2.6 cm/s vs. 2.2 cm/s, $p = 0.045$; peak diastolic V_r in apical septal segment: -3.7 cm/s vs. -3.2 cm/s, $p = 0.033$; peak diastolic V_r in basal anteroseptal segment: -2.5 cm/s vs. -2.1 cm/s, $p = 0.009$). Similar agreement was found for TTP for radial and long-axis velocities as shown in Fig. 4.

On a per-segment basis, there were excellent and significant ($p < 0.001$) correlations between the two TPM scans for peak radial velocities ($R^2 = 0.92$) and peak long axis velocities ($R^2 = 0.90$) between test (scan 1) and retest (scan 2) (Fig. 5). Similar relationships were found for TTP for both radial and long-axis motion ($R^2 = 0.91$ and $R^2 = 0.88$, both $p < 0.001$).

Inter- and intra-observer variability

The results of Bland–Altman analysis for the assessment of intra- and inter-observer variability of segmental analysis of LV peak velocities are summarized in Fig. 6. As shown in Fig. 6a, excellent intra-observer agreement with minimal mean differences and small limits of agreement was found for all myocardial motion direction (radial, long-axis, circumferential). In addition, Fig. 6b demonstrates good inter-observer analysis agreement but increased variability for long axis peak velocities (mean difference = 0.7 cm/s).

Discussion

In the present study, we demonstrated good agreements between segmental myocardial velocities acquired with two conductive TPM exams in a cohort of healthy volunteers. Our data suggest that TPM is reliable tool for the global and regional quantification of regional myocardial velocity in three major motion directions (radial, circumferential and long-axial).

Previous studies investigating the reproducibility or observer variability of TPM are in good agreement with our findings. A TPM study by Petersen et al. [7] investigated the reproducibility of mean LV velocities on a slice-by-slice basis. In agreement with our findings, results from this study demonstrated low intra- and inter-observer variability high inter-study reproducibility for radial and circumferential velocity components at apical, midventricular, and basal levels. A study by Foell et al. [21] showed good inter-observer agreement for segmental peak velocities and TTP in ten subjects with low mean differences (ranging from -0.02 to 0.06 cm/s for peak velocities and 4.1 to 0.8 ms for TTP) and moderate limits of agreement. Similar to findings in our study, intra-observer agreement was better than inter-observer agreement. A previous study in healthy controls in heart transplant patients [22] used Bland–Altman analysis comparing peak velocities in 16 segments and 10 patients and revealed good inter-observer agreement with low mean difference (0.01 and 0.02 cm/s for peak radial and peak long-axis velocities) and moderate limits of agreement (1.1 and 0.87 cm/s). Taken together, these results confirm the reliability of TPM for the analysis of myocardial motion.

Compared to these prior studies, findings from our study add important information on the scan–rescan reproducibility of segmental LV velocities and the reliability of the assessment of the dynamics of myocardial velocities over the cardiac cycle which is necessary for the accurate description of regional myocardial velocity in the context of various cardiovascular diseases. In addition, the application of new multi-dimensional image acquisition acceleration techniques, such as k – t acceleration [19] or compressed sensing [23] or efficient non-Cartesian k-space sampling such as spiral data readouts [9] have helped to significantly reduce TPM acquisition time. In comparison to previously reported results, all data in this study was based on k – t accelerated TPM which was performed during a single breath-hold for each slice. Findings of our study thus demonstrate that the application of advanced imaging acceleration can maintain the reproducibility and observer agreement reported in previous studies which were based on TPM implementations with no or low acceleration factors.

The remaining variability of velocity measures may origin from multiple sources at levels of image acquisition or image processing. TPM images were acquired at three locations (base, mid, apex) of the LV at short-axis view. Those imaging planes were manually selected by the MRI technicians for each of the two MRI exams (scan and rescan). Myocardial slice or angle mismatch between multiple scans may exist and thus act as a source of inter-study variations. In addition, the physiological conditions of the same subject, such as BP and heart rate, may vary between scans and rescans and can potentially alter the heart motion patterns after a nearly 2-week interval. However, in our study cohort, no significant differences in baseline physiology were observed and all subjects had similar heart rates and blood pressures for scan and rescan visits.

It should be noted that LV dynamics can be very different for rapid contraction and expansion during systole and early diastole compared to the more passive filling phased during diastole. An ideal TPM protocol would thus use higher frame rates for data acquisition during systole and early diastole compared to the remainder of the cardiac cycle (i.e. non-constant temporal resolution for systole compared to diastole). In addition, the highly anisotropic resolution of the TPM data (2 mm in-plane resolution vs. 8 mm slice thickness) may results in increased averaging of velocities along the slice direction. This could lead to the underestimation of peak long-axis velocities compared to radial or circumferential motion directions.

Another major drawback is related to the complexity of the presented data analysis strategy which involves the manual delineation of end- and epicardial boundaries which can introduce operator dependent variations in LV segmentation contours. The calculation of global myocardial velocities on a slice-by-slice basis, which showed only small variability, is relatively independent from such inaccuracies. Low or zero velocity components missed or added due to segmentation errors will lead to only small errors for parameters averaged over the entire LV wall at basal, mid-ventricular or apical locations. In contrast, the assessment of segmental LV motion and TTP was based on the calculation of regional averages of LV velocities and is this expected to be more sensitive to the definition of the segmentation contours. In addition, the manually definition of myocardial segments may affect the regional LV velocity indices. TPM analysis in this study included the manual identification of the RV insertion point at the LV to define the location of the myocardial segments in the AHA 16-segment model. A mismatch on individual RV insertion points between observers may thus results in variations in velocity measures due to the variations of motion patterns between different parts of myocardium. This observer-dependency should be eliminated in future studies by the introduction of semi- or fully automated segmentation methods into the data processing workflow.

Despite of those adverse factors, our study is the first study to advance the reproducibility of TPM to a persegment level. Regional myocardial motion indices are expected to provide important information on the progression of in cardiovascular diseases. In this context, it should be noted that the findings of this study are based on intra-modality comparisons and no gold standard was used to evaluate the accuracy of TPM parameter analysis. Actually, no such a “gold standard” exists in clinical practice that can define “normal” or “abnormal” segmental myocardial motion patterns in general populations. Additional limitations of our

study includes that all 18 subjects are healthy volunteers. Therefore, the reproducibility and accuracy of TPM needs to be further evaluated on special patients groups for various clinical purposes. Further, the investigated scan–rescan reproducibility was a best-case scenario with both scans being performed on the same type of MR system with exactly the same imaging protocol. Incorporating another scanner type or varying the spatio-temporal resolution may have resulted in altered reproducibility, which should be investigated in more detail in future studies. In addition, findings in larger numbers of patients showing a higher variability of cardiac anatomy and/or motion should be performed to further evaluate the robustness of this method for clinical use.

Further, a comparisons between TPM and TDI in a previous study demonstrated good agreement between both techniques [20]. However, a direct comparison between TPM and TDI was not performed for the current study. Future studies are thus needed to further evaluate the performance and reproducibility of TPM compared to the clinical reference standard TDI.

In conclusion, analysis of slice-by-slice and segmental myocardial using aortic TPM in normal volunteers can be performed with low variability. The reproducibility for quantification of regional peak velocity was limited but relative regional distribution along all motion directions could reliably be replicated. A more automated analysis of TPM data may have the potential to further improve robustness and may allow its wider clinical application.

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Fig. 1.

An example case for the evaluation of reproducibility. The three images represent typical short axis slice locations (base, mid, apex) for both scan 1 and scan 2. The *arrows* represent the pixel-by-pixel in-plane myocardial velocity vectors inside the segmented myocardium. The *large arrows* show the average LV velocity vectors in eight angular segments. Repeated TPM acquisition resulted in similar LV myocardial velocity patterns in basal, mid-ventricular, and apical short axis location (peak systolic velocity vectors mapped onto the segmented LV) for scan 1 (**a**) and scan 2 (**b**). It should be noted that the slice locations are based on manual positioning of TPM imaging planes based on anatomic landmarks for each scan. As a result, slice locations may not be identical for scan 1 and scan 2. *LV* left ventricle, *RV* right ventricle

Fig. 2.

Mean velocity time curves for test (scan 1) and retest (scan 2). Each velocity–time curve represents the temporal evolution of mean LV velocities (averaged over the LV for base, mid apex) for all $n = 18$ subjects. The standard deviations reflect inter-individual differences in LV velocities. *Asterisk* indicates significant differences at individual time points in the cardiac cycle (paired t test, $p < 0.05$)

Fig. 3.

Systolic and diastolic peak radial LV velocities (a) and peak long-axis velocities (b) for test (scan 1) and retest (scan 2). The individual plots show the distribution of peak systolic radial and long-axis myocardial velocities in the AHA 16-segment model. The value of each segment corresponds to the average peak velocity over 18 normal subjects

Fig. 4.

Systolic and diastolic time-to-peak (TTP) for radial LV velocities (**a**) and peak long-axis velocities (**b**) for test (scan 1) and retest (scan 2). The individual plots show the distribution of peak systolic radial and long-axis myocardial TTP in the AHA 16-segment model. The value of each segment corresponds to the average TTP over 18 normal subjects

Fig. 5.

Correlation analysis between scan 1 and rescan (scan 2) for segmental radial (**a**) and long-axis (**b**) LV peak velocities. The individual graphs show the results of the correlation of regional velocity with myocardial velocities for 18 subjects. For each subject, data from all 16 segments were included, resulting in a total of $18 \times 16 = 288$ data points for each graph

Fig. 6. Bland–Altman analysis of inter- and intra-observer variability. Each Bland–Altman comparison included 18 subjects. For each subject, data from all 16 segments were included, resulting in a total of $18 \times 16 = 288$ data points for each Bland–Altman analysis

Table 1

Demographics of the study cohort

Volunteers (N = 18)	
Male (%)	12 (67)
Age (years old)	48.1 ± 10.9
Weight (kg)	83.5 ± 11.8
Height (cm)	173.7 ± 9.5
Heart rate (beats/min)	
Scan 1	68.0 ± 11.1
Scan 2	67.1 ± 12.3
Systolic blood pressure (mmHg)	
Scan 1	121.3 ± 28.3
Scan 2	120.5 ± 30.7
Diastolic blood pressure (mmHg)	
Scan 1	79.6 ± 19.8
Scan 2	80.7 ± 20.1

Scan-rescan of mean systolic and diastolic peak and TTP radial and long-axis velocities for scan 1 and 2 for basal, midventricular, and apical short axis locations

Table 2

	Peak radial vel. (cm/s)		TTP radial vel. (ms)		Peak long-axis vel. (cm/s)		TTP long-axis vel. (ms)	
	Systole	Diastole	Systole	Diastole	Systole	Diastole	Systole	Diastole
Base								
Scan 1	2.7 ± 0.5	-2.9 ± 1.2	134 ± 30	495 ± 61	5.7 ± 2.1	-4.7 ± 2.6	99 ± 35	498 ± 76
Scan 2	2.7 ± 0.5	-2.7 ± 0.8	123 ± 28	485 ± 41	6.2 ± 1.9	-4.4 ± 2.1	91 ± 15	507 ± 58
p value	0.505	0.203	0.066	0.499	0.313	0.241	0.351	0.453
Mid								
Scan 1	2.7 ± 0.4	-2.8 ± 1.2	131 ± 27	521 ± 76	4.2 ± 2.0	-2.8 ± 1.9	88 ± 15	482 ± 84
Scan 2	2.6 ± 0.5	-2.5 ± 0.9	131 ± 19	513 ± 36	4.3 ± 1.1	-2.8 ± 1.6	93 ± 15	497 ± 76
p value	0.087	0.100	1.000	0.681	0.847	0.896	0.215	0.214
Apex								
Scan 1	2.4 ± 0.3	-3.0 ± 1.0	134 ± 31	525 ± 66	2.7 ± 1.6	-1.6 ± 1.1	95 ± 41	435 ± 72
Scan 2	2.4 ± 0.5	-2.8 ± 0.9	133 ± 20	540 ± 35	2.8 ± 1.1	-1.6 ± 1.1	97 ± 51	466 ± 89
p value	0.782	0.181	0.878	0.420	0.661	0.763	0.631	0.216

All numbers reflect mean velocities and TTP averaged over all LV segments in each slice location (base, mid, apex) and all subjects