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Altered regional myocardial velocities by tissue phase mapping and feature tracking in pediatric patients with hypertrophic cardiomyopathy

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Abstract

Background—Hypertrophic cardiomyopathy (HCM) is associated with heart failure, atrial fibrillation and sudden death. Reduced myocardial function has been reported in HCM despite normal left ventricular (LV) ejection fraction. Additionally, LV fibrosis is associated with elevated T1 and might be an outcome predictor.

Objective—To systematically compare tissue phase mapping and feature tracking for assessing regional LV function in children and young adults with HCM and pediatric controls, and to evaluate structure-function relationships among myocardial velocities, LV wall thickness and myocardial T1.

Materials and methods—Seventeen pediatric patients with HCM and 21 age-matched controls underwent cardiac MRI including standard cine imaging, tissue phase mapping (two-dimensional cine phase contrast with three-directional velocity encoding), and modified Look-Locker inversion

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Compliance with ethical standards

Conflicts of interest None

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recovery to calculate native global LV T1. Maximum LV wall thickness was measured on cine images. LV radial, circumferential and long-axis myocardial velocity time courses, as well as global and segmental systolic and diastolic peak velocities, were quantified from tissue phase mapping and feature tracking.

Results—Both tissue phase mapping and feature tracking detected significantly decreased global and segmental diastolic radial and long-axis peak velocities (by 12–51%, $P<0.001$ – 0.05) in pediatric patients with HCM vs. controls. Feature tracking peak velocities were lower than directly measured tissue phase mapping velocities (mean bias = 0.3 – 2.9 cm/s). Diastolic global peak velocities correlated moderately with global T1 ($r = -0.57$ to -0.72 , $P<0.01$) and maximum wall thickness ($r = -0.37$ to -0.61 , $P<0.05$).

Conclusion—Both tissue phase mapping and feature tracking detected myocardial velocity changes in children and young adults with HCM vs. controls. Associations between impaired diastolic LV velocities and elevated T1 indicate structure–function relationships in HCM.

Keywords

Children; Feature tracking; Heart; Hypertrophic cardiomyopathy; Left ventricle; Magnetic resonance imaging; Myocardial velocity; Myocardium; Tissue phase mapping

Introduction

Hypertrophic cardiomyopathy (HCM) is a common genetic condition that affects 1 in 500 people. HCM is characterized by otherwise unexplained increased left ventricular (LV) wall thickness and is associated with increased risk of heart failure, atrial fibrillation and sudden death [1]. MRI techniques assessing regional LV function [2] can provide valuable information on regional myocardial dysfunction in HCM [3], which might precede the decline of established global function parameters such as ejection fraction. In addition, altered native T1 relaxation times [4] associated with structural tissue changes such as fibrosis might be important outcome predictors for HCM [5–7].

Regional myocardial strain and velocities can be obtained using various MRI techniques, including tissue tagging [8], displacement encoding with stimulated echoes (DENSE) [9], strain encoding (SENC) [10] and tissue phase mapping [11–13]. Whereas tagging, DENSE and SENC derive strain based on tissue displacement, tissue phase mapping directly measures myocardial velocities using phase-contrast MRI. More recently, feature tracking algorithms have enabled the retrospective quantification of strain and velocities from standard-of-care balanced steady-state free precession (bSSFP) cine images [14], leading to a broad applicability [15, 16].

Myocardial strain in adults with HCM has been assessed with tagging [17] and in adults and children using feature tracking [6, 18–22]. These techniques have shown altered LV function despite normal ejection fraction. Moreover, tissue phase mapping and tissue Doppler echocardiography have proved to be valuable tools for directly assessing myocardial velocities in HCM and other non-ischemic cardiomyopathies [23, 24]. However, no direct comparison between myocardial velocities measured by tissue phase mapping and derived from feature tracking has been reported to systematically assess the performance of these

techniques for detecting regional changes in LV velocities in HCM. In addition, correlations with myocardial T1 relaxation time relating to tissue abnormalities have not been evaluated in children.

The objective of this study was to (1) systematically evaluate the performance of tissue phase mapping and feature tracking for the assessment of regional LV myocardial velocities in pediatric patients with HCM compared to age-matched healthy controls, and (2) to assess structure-function relationships for myocardial velocities derived from tissue phase mapping and feature tracking with maximum LV wall thickness and myocardial T1.

Materials and methods

Study population

We retrospectively recruited 17 pediatric patients with HCM (age = 15 ± 4 years, 8 male) and 21 age-matched controls (age = 16 ± 4 years, 11 male) from 2013 to 2017 for this study. Demographic data are summarized in Table 1. All subjects underwent a physician-ordered standard-of-care cardiac MRI at 1.5 Tesla (Magnetom Aera; Siemens Medical Solutions, Malvern, PA). After written informed consent was obtained, tissue phase mapping was performed as part of this study, which was approved by our institutional review board and complied with the Health Insurance Portability and Accountability Act. Children and young adults were recruited based on a clinical diagnosis of HCM with confirmation by MRI interpretation. Healthy controls underwent cardiac MRI for evaluation of cardiac disease because of syncope, chest pain, palpitations or family history of arrhythmogenic right ventricular cardiomyopathy, but these diagnoses were ruled out by negative MRI studies and negative clinical workup. We excluded pediatric patients with other congenital heart diseases, genetic syndromes, neuromuscular diseases, coronary artery disease, or history of septal myectomy. Four patients and two controls had MRI under general anesthesia with suspended respirations for breath-hold imaging per the clinical standard.

Left ventricular global function analysis

In this retrospective study, electrocardiography (ECG)-gated cine bSSFP images in long- and short-axis orientations were acquired during breath-holds of 6–14 s. Short-axis images were used to obtain LV global function parameters, including LV indexed end-diastolic and end-systolic volumes, LV indexed stroke volume, LV ejection fraction, maximum LV wall thickness and LV end-diastolic mass index (QMass; Medis Medical Imaging Systems, Leiden, The Netherlands). Left ventricular global function parameters were analyzed at the time of image acquisition per the clinical standard. Papillary muscles and prominent myocardial trabeculations were excluded in endocardial contouring. Sequence parameters for bSSFP imaging are summarized in Table 2.

Native T1 relaxation time and extracellular volume fraction

Global native T1 relaxation time was calculated from modified Look-Locker inversion recovery (MOLLI) [25] short-axis images obtained as part of the standard clinical protocol. The number of recovery heartbeats was adjusted based on the patient's heart rate, which allowed for adequate T1 recovery between inversion pulses [26]. Pixel-wise curve fitting

was then used to produce an in-line T1 pixel-map. Segmentation of myocardial LV T1 pixel-maps was performed on a Siemens MMWP multimodality workstation (Siemens Medical Solutions, Malvern, PA) using the 16-segment American Heart Association model [27]. Global native T1 times were then determined by the mean T1 value of the 16 segments combined. Pre-contrast T1 relaxation times were available for all 17 HCM patients and 14 controls. Post-contrast T1 mapping was performed beginning 12 min following contrast administration. Extracellular volume fraction (ECV) was calculated using pre- and post-contrast T1 mapping data and patient hematocrit obtained on the same day as the MRI study [28] and was available for 12 HCM patients and 12 controls.

Tissue phase mapping

Tissue phase mapping was performed using a prospectively ECG-triggered black-blood prepared phase-contrast gradient echo sequence with three-directional velocity encoding employing k-t acceleration with an undersampling factor of $R=5$, as previously described [11, 12, 29]. Three short-axis slices at basal, mid-ventricular and apical locations were acquired with in-plane resolution of 1.6–2.5 mm² and temporal resolution of 21–25 ms, each during one breath-hold with durations of 11–26 s. Further imaging parameters are summarized in Table 2.

For tissue phase mapping post-processing and data analysis, we used a custom MATLAB (MathWorks, Natick, MA) tool (Fig. 1) [29]. Epicardial and endocardial LV borders for all time frames were manually traced onto the short-axis tissue phase mapping images by a single observer (A.L., a medical student who received dedicated training to perform the post-processing). After eddy current correction, the measured Cartesian velocities were transformed to radial (v_r), circumferential (v_ϕ) and long-axis (v_z) velocity components to resemble LV contraction, rotation and shortening, respectively. Pixelwise velocities were averaged within slices and regionally, using the American Heart Association 16-segment model to obtain velocity time courses for all slices and segments (Fig. 1). Systolic and diastolic peak velocities were extracted from the radial (v_r) and long-axis (v_z) velocity time courses for all 16 segments. Subsequently, global LV peak velocities were obtained by averaging the segmental values over the entire left ventricle.

Using prospective ECG-triggering, time-resolved images are acquired for approximately 80% of the cardiac cycle, which is usually sufficient to cover the diastolic peak. However, diastolic coverage was insufficient for two patients with HCM and one control subject, so we excluded these subjects from tissue phase mapping analysis (Fig. 2).

Feature tracking analysis

Feature tracking analysis based on cine bSSFP images was performed with a commercial cardiac analysis software package (2D Cardiac Performance Analysis MR; TomTec, Unterschleissheim, Germany) by the same single observer (A.L.). Short-axis images at the LV base, mid-ventricle and apex (matching the tissue phase mapping slice locations) together with two-, three- and four-chamber long-axis views were used to obtain full three-directional motion information. After initial manual segmentation of epicardial and endocardial LV contours at end-systole and end-diastole by the same single observer as for

tissue phase mapping, the feature tracking algorithm calculated radial and circumferential velocities (v_r and v_ϕ) from short-axis views as well as long-axis velocities (v_z) from long-axis views. The software provided segmental velocity time courses for the 16 American Heart Association segments (Fig. 1), which were then averaged within slices to also obtain slice-averaged velocities for base, mid-ventricle and apex. Analogous to tissue phase mapping analysis, segmental and global velocities at peak systole and diastole were obtained in radial (v_r) and long-axis (v_z) velocity components.

For five controls and three patients with HCM, not all long-axis views were adequate for analysis, resulting in incomplete feature tracking data in long-axis direction. Thus, we excluded these subjects from analysis of the long-axis velocity component in feature tracking (Fig. 2).

Statistical analysis

All continuous data are presented as means $\pm$ standard deviations. Global LV function parameters as well as global and segmental systolic and diastolic peak velocities for both tissue phase mapping and feature tracking were compared between pediatric patients with HCM and controls. All data were tested for normality with a Lilliefors test followed by unpaired *t*-tests for normally distributed data or Wilcoxon rank sum tests for non-normally distributed data. We assessed differences between tissue phase mapping and feature tracking for the combined cohort of controls and patients with HCM using a Bland-Altman comparison of segmental peak velocities in systole and diastole. To investigate changes in the difference between velocities with respect to their mean, we performed linear regression on the data in the Bland-Altman plots. For a second comparison utilizing data from the whole cardiac cycle, slice-averaged velocity time courses for the basal, mid-ventricular and apical short-axis views from tissue phase mapping and feature tracking were interpolated to the same time steps using piecewise polynomials (splines). To assess correlations between tissue phase mapping and feature tracking data, we performed linear regression and calculated Pearson correlation coefficients *r* for all three velocity components (v_r , v_ϕ , v_z). In addition, global peak systolic and diastolic velocities from all subjects (patients with HCM and controls) were correlated with maximum LV wall thickness and native T1 relaxation time using Pearson correlation coefficients. These correlations were computed using absolute peak velocities so that positive correlations are always associated with increases in velocities independent of sign. Results were considered significant for $P < 0.05$. In addition, we performed post hoc power analysis for detecting a difference in global radial and long-axis diastolic peak velocities between controls and patients with HCM, for both tissue phase mapping and feature tracking.

Results

Left ventricular global function, native T1 and extracellular volume fraction analysis

Global LV function parameters for controls and pediatric patients with HCM are summarized in Table 1. Left ventricular indexed end-systolic volume was significantly reduced in patients with HCM compared to controls (29 ± 10 mL/m² vs. 38 ± 6 mL/m², $P = 0.002$), whereas LV indexed end-diastolic volume (85 ± 16 mL/m² vs. 89 ± 13 mL/m²,

$P=0.5$) and LV indexed stroke volume (56 ± 11 mL/m² vs. 51 ± 9 mL/m², $P=0.09$) were similar between the groups. Left ventricular ejection fraction was significantly increased in patients with HCM compared to controls ($66\pm 9\%$ vs. $57\pm 3\%$, $P<0.001$). Patients with HCM had significantly higher LV mass index than controls (91 ± 65 g/m² vs. 45 ± 10 g/m², $P<0.001$). Maximum LV wall thickness was also greater in patients with HCM than in controls (23.5 ± 9.1 mm vs. 7.9 ± 1.5 mm, $P<0.001$). Left ventricular native T1 was higher in patients with HCM than controls ($1,049\pm 59$ ms vs. 1000 ± 33 ms, $P=0.01$), whereas no difference was detected for ECV.

Left ventricular myocardial velocities by tissue phase mapping and feature tracking: patients with hypertrophic cardiomyopathy versus controls

Systolic and diastolic global LV peak velocities obtained by tissue phase mapping and feature tracking are summarized in Table 3. Significantly reduced diastolic radial and long-axis peak velocities for patients with HCM compared to controls were detected by both tissue phase mapping ($P<0.01$) and feature tracking ($P<0.05$). In addition, systolic long-axis peak velocities by both tissue phase mapping and feature tracking were reduced in patients with HCM ($P<0.05$).

On the segmental level, systolic radial LV peak velocities obtained from both tissue phase mapping and feature tracking appeared similar for patients with HCM and controls (Fig. 3). Tissue phase mapping detected significant differences in systolic radial peak velocities in four segments and feature tracking in two segments. In the long-axis direction, systolic peak velocities by tissue phase mapping were significantly reduced for patients with HCM in six LV segments including the whole lateral wall, whereas feature tracking detected a significant reduction compared to controls only in two segments.

In contrast, diastolic LV peak velocities demonstrated more pronounced segmental differences between patients with HCM and controls (Fig. 4). Radial diastolic peak velocities obtained from tissue phase mapping were significantly reduced for patients with HCM compared to controls in 11 segments, whereas feature tracking detected a significant decrease only in four segments. In the long-axis direction, tissue phase mapping velocities for patients with HCM were significantly reduced in 12 segments compared to controls, while feature tracking detected a significant decrease in long-axis velocities in seven segments.

Post hoc power analysis resulted in a power of 0.92 and 0.99 to detect a statistically significant difference in global radial and long-axis diastolic peak velocities, respectively, using tissue phase mapping. For feature tracking, we detected a power of 0.32 and 0.85 for global radial and long-axis diastolic peak velocities, respectively.

Left ventricular myocardial velocities: tissue phase mapping versus feature tracking

Bland-Altman analysis comparing segmental peak velocities between tissue phase mapping and feature tracking (Fig. 5) demonstrated a systematic underestimation by feature tracking compared to tissue phase mapping, with an increasing difference for higher peak velocities. The mean difference was lower for radial than for long-axis velocities, with the lowest mean

difference associated with systolic radial velocities (0.3 cm/s) and the highest with diastolic long-axis velocities (2.9 cm/s).

Comparison of slice-averaged velocity time courses revealed significant correlations between tissue phase mapping and feature tracking for all three velocity components (Fig. 6). Individual correlation coefficients were $r=0.88$, $r=0.60$ and $r=0.78$ (all $P<0.001$) for radial (v_r), circumferential (v_ϕ) and long-axis (v_z) velocities, respectively.

Relationships among myocardial velocities, maximum left ventricular wall thickness and left ventricular native T1

Among all subjects, global diastolic velocities inversely correlated with LV native T1 using both tissue phase mapping and feature tracking ($r = -0.57$ to -0.72 , $P<0.01$). Global diastolic velocities also inversely correlated with maximum LV wall thickness ($r = -0.37$ to -0.61 , $P<0.05$). Correlations between diastolic velocities with both native T1 time and maximum LV wall thickness were stronger for tissue phase mapping compared to feature tracking. Individual correlation coefficients for diastolic velocities are shown in Table 4, with corresponding correlation plots for tissue phase mapping velocities in Fig. 7.

Discussion

We used tissue phase mapping and feature tracking to assess global and regional three-directional LV myocardial velocities in pediatric patients with HCM. When comparing these patients to healthy age-matched controls, both tissue phase mapping and feature tracking detected significantly reduced diastolic peak velocities in patients with HCM on the global and segmental levels. This was supported by the results of our post hoc power analysis, which generally demonstrated excellent power to detect differences between controls and patients with HCM, although the conclusions that can be drawn for diastolic radial peak velocities in feature tracking might be limited. However, evaluation of peak velocities obtained by both techniques revealed a systematic underestimation of regional myocardial peak velocities derived from feature tracking compared to values measured by tissue phase mapping. These differences were most pronounced for long-axis velocities; increased disagreement was found for higher velocities. Nonetheless, the comparison of velocity time courses between the techniques demonstrated significant moderate to strong correlations between feature tracking and tissue phase mapping velocities. Global native T1 relaxation times were increased in patients with HCM, consistent with prior studies in both adults and children with HCM [5, 26], indicating a higher degree of structural tissue changes in HCM. These structural changes were significantly correlated with reduced diastolic velocities, revealing structure-function relationships in HCM. The mean LV native T1 time for controls corresponded with normal values published from our group previously (990 ± 34 ms) [30].

Children and young adults with HCM had significantly reduced peak velocities in diastole compared to controls, suggestive of the diastolic dysfunction with impaired ventricular filling observed in patients with HCM [31]. In addition, peak systolic long-axis velocities were reduced in patients with HCM, suggesting concurrent systolic dysfunction despite normal LV ejection fractions. Our findings indicate that both tissue phase mapping and feature tracking can provide diagnostic value for identifying impaired LV function in

children with HCM. However, the systematic underestimation of LV peak velocities by feature tracking might result in less sensitivity of feature tracking compared to tissue phase mapping for detecting regional motion abnormalities.

In comparison to previous feature tracking MRI studies in children that investigated systolic strain and strain rate [18, 20, 22], the present study assessed diastolic function in pediatric patients with HCM via radial and long-axis peak velocities. Both tissue phase mapping and feature tracking demonstrated reduced diastolic function in these pediatric patients, which is in agreement with previous studies in adults [17, 19]. For example, Ennis et al. [17] showed that adults with HCM exhibited reduced early diastolic strain rates compared to controls using tagging. In general, multiple MR studies have reported reduced strain parameters in people with HCM compared to controls [17–19, 21, 22]. For instance, Smith et al. [18] and Mazurkiewicz et al. [22] both demonstrated decreased myocardial strain in children with HCM compared to controls using feature tracking. Diastolic myocardial dysfunction in both adult and pediatric patients with HCM has also been shown in echocardiographic studies using tissue Doppler velocity imaging and other ultrasound techniques [23, 32]. Moreover, reductions in peak systolic long-axis velocities in people with HCM are supported by findings from studies in adults [33, 34].

Although we found strong correlations between tissue phase mapping and feature tracking velocity time courses, feature tracking peak velocities were systematically lower compared to tissue phase mapping peak velocities. Feature tracking also demonstrated fewer myocardial segments with significantly decreased myocardial velocities in patients with HCM, compared to tissue phase mapping. This underestimation might be explained by enhanced temporal blurring in feature tracking. First, the temporal resolution of the cine images was lower compared to tissue phase mapping (28.5 ± 5.0 ms vs. 23.6 ± 0.6 ms), and second, the feature tracking algorithm used two neighboring cardiac time frames to derive one velocity value at a single time point. In contrast, tissue phase mapping directly measured LV velocities for each cardiac time frame. Thus, the effective temporal resolution for velocities derived from feature tracking was more than two times lower than for tissue phase mapping. For future studies it might be beneficial to use higher temporal resolution cine images [35] for feature tracking applications. Recently, researchers assessed myocardial strain using feature tracking algorithms and compared them to tagging and DENSE and showed significant differences between techniques [36, 37].

Correlations between myocardial velocities and native T1 time as well as maximum LV wall thickness were stronger for tissue phase mapping than for feature tracking in our study. This further suggests decreased sensitivity for detecting changes in regional myocardial function using feature tracking, compared to tissue phase mapping. Previous studies using MR have also correlated reduced myocardial strain with structural myocardial changes in HCM. Wu et al. [6] found that increased native T1 values were associated with reduced peak systolic and early diastolic strain rates in adults with HCM. In addition, Dass et al. [5] and Swoboda et al. [7] found significant association of segmental myocardial strain with segment thickness and native T1 in adults with HCM. Our study did not suggest significant correlation of peak systolic velocities with structural parameters among children with HCM, a finding that might be related to early stages of disease progression. Nonetheless, our results did support

significant relationships between radial as well as long-axis diastolic dysfunction and the degree of LV hypertrophy and fibrosis in children with HCM.

Limitations of our study include inadequate cine bSSFP long-axis views for eight subjects and insufficient coverage of diastole in tissue phase mapping images for three subjects. This resulted in the exclusion of these subjects from tissue phase mapping analysis and feature tracking long-axis analysis, respectively. The number of subjects in this study is also relatively small, which limits the conclusions that can be drawn. This could be improved with a larger sample size in future studies. To directly compare feature tracking with tissue phase mapping velocities, we used velocities provided by the feature tracking commercial software rather than strain. Future studies could thus include a comparison between feature tracking strain and strain calculated from tissue phase mapping velocities [38] as well as a systematic analysis of the impact of spatial and temporal resolution on myocardial velocities or strain, both of which were beyond the scope of this study.

Whereas our study detected a significant difference in native T1 between patients with HCM and controls, there were no changes in ECV. This might be caused by a lower sensitivity for ECV resulting from a smaller number of subjects who underwent post-contrast imaging.

Although tissue phase mapping provided higher peak velocities resulting in better sensitivity to detect changes than feature tracking, tissue phase mapping requires additional scans with long breath-holds, whereas feature tracking uses standard-of-care cine bSSFP images. Although this did not significantly impact image quality in this study, it is a potential limitation to more widespread use of tissue phase mapping in clinical practice. Promising developments to overcome this issue and to increase the applicability of tissue phase mapping include advanced imaging acceleration with non-Cartesian k-space trajectories and compressed sensing reconstruction [39] to reduce breath-hold duration, as well as simultaneous multislice imaging to acquire all three tissue phase mapping slices at once [40].

An additional limitation of this study is that only a single observer performed image analyses for both tissue phase mapping and feature tracking, which did not allow for inter-observer variability analysis. While tissue phase mapping has been shown to have good inter- and intra-observer reliability [13, 41], feature tracking has demonstrated more mixed results [15, 16, 18]. Moreover, with reported differences among techniques [36, 37], the quantification of myocardial velocities and strain lacks a proper ground truth.

Conclusion

Both tissue phase mapping and feature tracking yielded decreased diastolic velocities in children with HCM despite normal LV ejection fraction. Direct associations between myocardial dysfunction and structural parameters representing myocardial hypertrophy and fibrosis were also demonstrated with both tissue phase mapping and feature tracking. Feature tracking systematically underestimated myocardial velocities and correlated less well with structural parameters such as native T1 values and maximum LV wall thickness compared to tissue phase mapping velocities. This might suggest decreased sensitivity for

detecting regional motion abnormalities with feature tracking compared to tissue phase mapping. Although further investigations are needed for development of full clinical implementation, MR techniques such as tissue phase mapping and feature tracking might be useful in detecting early indicators of disease and might provide helpful data in multi-parametric analysis of pediatric HCM.

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d

Feature Tracking

long-axis cine bSSFP
images to obtain long-axis
motion information

segmental velocity time courses with arrows
marking systolic and diastolic peak velocities

Fig. 1.

Overview of the workflow for tissue phase mapping (**a** and **b**) and feature tracking (**c** and **d**).

a Three-directional tissue phase mapping velocities of the segmented myocardium are visualized by color-coding in through-plane direction and in-plane velocity vectors for short-axis views at base, mid and apex. We performed analysis with a custom MATLAB tool, which transforms the measured Cartesian velocities in long-axis, radial and circumferential components. **b** Pixelwise velocities were averaged within the American Heart Association (AHA) 16-segment model to obtain segmental velocity time courses (*colored curves* for basal segments together with *black* for slice-averaged velocity). We performed feature tracking analysis with commercial software (TomTec, Unterschleissheim, Germany). **c, d** From short-axis (**c**) and long-axis (**d**) cine balanced steady-state free precession (bSSFP)

images, we obtained corresponding segmental velocity time courses for radial/circumferential and long-axis components, respectively (*colored curves* together with *white* for slice-averaged velocity). In a final step, we extracted systolic and diastolic peak velocities from segmental long-axis and radial velocity time courses for both tissue phase mapping and feature tracking (*arrows*). *RV* right ventricle

Fig. 2.

Diagram represents the exclusions applied to the total number of subjects for tissue phase mapping and feature tracking analysis, resulting in reduced subject numbers, which for feature tracking differed between radial/circumferential (rad/circ) and long-axis velocity components. For the direct comparison between the modalities, only the common data were used, as shown in the bottom table. *FT* feature tracking, *TPM* tissue phase mapping

Fig. 3.

Systolic peak velocities for radial (v_r) and long-axis (v_z) components in the 16-segment American Heart Association model obtained by tissue phase mapping (*left*) and feature tracking (*right*) for controls and patients with hypertrophic cardiomyopathy (HCM). Asterisks mark significant differences (* $P < 0.05$, ** $P < 0.01$) between controls and patients with HCM

Fig. 4.

Diastolic peak velocities for radial (v_r) and long-axis (v_z) components in the 16-segment American Heart Association model obtained by tissue phase mapping (*left*) and feature tracking (*right*) for controls and patients with hypertrophic cardiomyopathy (HCM). Asterisks mark significant differences (* $P < 0.05$, ** $P < 0.01$) between controls and patients with HCM

Fig. 5.

Bland-Altman analysis of segmental radial (v_r ; **a**) and long-axis (v_z ; **b**) peak velocities in systole and diastole for the combined cohort of controls (*blue circles*) and patients with hypertrophic cardiomyopathy (HCM) (*red diamonds*). *Thick black lines* show the mean difference between tissue phase mapping and feature tracking, and *dashed lines* show corresponding limits of agreement (± 2 standard deviations [SD]). *Oblique lines* represent least-square fits indicating differences between lower and higher velocities

Fig. 6.

Correlation analysis between tissue phase mapping (TPM) and feature tracking (FT) of slice-averaged velocity time courses for radial (v_r , *left*), circumferential (v_ϕ , *middle*) and long-axis (v_z , *right*) components. Shown in the top two rows are velocity time courses for base, mid and apex averaged over controls and patients with hypertrophic cardiomyopathy (HCM) (mean $\pm$ standard deviation) comparing tissue phase mapping (*black*) and feature tracking (*blue/red*). Shown in the bottom row are correlation plots of tissue phase mapping vs. feature tracking for the individual velocities from all slices and all subjects (*blue circles* for controls and *red diamonds* for patients with HCM). For each velocity component, Pearson correlation coefficient r is calculated. In addition, the equation obtained from linear regression is shown. Note that feature tracking circumferential velocities are displayed in units of degrees/second (deg/s) as calculated by the feature tracking software, whereas all other velocities are measured in cm/s

a

b

c

d

Fig. 7.

Correlations of global diastolic radial (v_r) and long-axis (v_z) peak velocities from tissue phase mapping with global native T1 time (**a**, **b**) and maximum left ventricular (LV) wall thickness (**c**, **d**) for the combined cohort of controls (*blue circles*) and pediatric patients with hypertrophic cardiomyopathy (HCM) (*red diamonds*) show significant inverse relationships

Table 1

Demographic data and left ventricular global function parameters

	Controls	HCM patients	<i>P-value</i> ^a
Number of subjects	21	17	
Male/female	11/10	8/9	1.0 ^b
Age (range) [years]	16±4 (1–19)	15±4 (9–21)	0.5
Height [cm]	165±23	159±19	0.1
Weight [kg]	61±25	63±29	0.8
BSA [m ²]	1.7±0.4	1.6±0.5	0.9
Heart rate [bpm]	75±16	72±10	0.5
Maximum LV wall thickness [mm]	7.9±1.5	23.5±9.1	<0.001
EDVI [mL/m ²]	89±13	85±16	0.5
ESVI [mL/m ²]	38±6	29±10	0.002
Stroke volume index [mL/m ²]	51±9	56±11	0.09
Ejection fraction [%]	57±3	66±9	<0.001
End-diastolic mass index [g/m ²]	45±10	91±65	<0.001
Native T1 time [ms]	1,000±33 (n=14)	1,049±59 (n=17)	0.01
Extracellular volume fraction [%]	26±2 (n=12)	27±4 (n=12)	0.3

^aSignificant differences ($P<0.05$) are marked in bold^bUsing Fisher exact test

BSA body surface area calculated with the Mosteller formula, *EDVI* end-diastolic volume index, *ESVI* end-systolic volume index, *HCM* hypertrophic cardiomyopathy, *LV* left ventricular

Table 2

Sequence parameters for tissue phase mapping and cine balanced steady-state free precession (bSSFP) imaging

	Tissue phase mapping	Cine bSSFP
In-plane resolution [mm ²]	(1.6–2.5) ²	(0.7–1.1) ²
Slice thickness [mm]	5–8	4–8
Temporal resolution (range) [ms]	23.6±0.6 (21–25)	28.5±5.0 (15–38)
Electrocardiogram gating method	Prospective	Retrospective
Imaging acceleration	kt-GRAPPA, R=5	GRAPPA, R=2
Breath-hold time (range) [heart beats/slice]	26±2 (20–32)	13±3 (9–27)
Breath-hold time (range) [seconds/slice]	21.0±3.4 (11–26)	10.6±1.4 (6–14)
Repetition time [ms]	5.2–6.2	2.6–3.3
Echo time [ms]	3.4–3.8	1.1–1.4
Flip angle	10°/15°	67–90°
Bandwidth [Hz/pixel]	455/650	920–945
Velocity encoding [cm/s]	25	N/A

N/A not available, *GRAPPA* generalized autocalibrating partial parallel acquisition

Table 3Global systolic and diastolic radial (v_r) and long-axis (v_z) peak velocities

		Tissue phase mapping ^a		Feature tracking ^a	
		Controls	HCM patients	Controls	HCM patients
Peak v_r [cm/s]	Systole	3.1±0.6	3.3±0.6	2.9±0.5	3.0±0.5
	Diastole	-4.9±0.8	-3.6±1.1^b	-3.3±0.4	-2.9±0.8^c
Peak v_z [cm/s]	Systole	6.2±1.8	4.9±1.9^c	3.3±0.7	2.8±0.7^c
	Diastole	-7.9±1.5	-4.8±2.1^b	-4.0±0.9	-3.0±1.0^c

^aSignificant differences ($P<0.05$) are marked in bold^b $P<0.01$ ^c $P<0.05$

HCM hypertrophic cardiomyopathy

Table 4

Correlations of diastolic global peak velocities with native T1 time and maximum left ventricular (LV) wall thickness

		Tissue phase mapping		Feature tracking	
		Radial	Long-axis	Radial	Long-axis
Native T1 time ^a	<i>r</i>	−0.68	−0.72	−0.58	−0.57
	<i>P</i> ^b	<0.001	<0.001	<0.001	0.005
Maximum LV wall thickness	<i>r</i>	−0.55	−0.61	−0.37	−0.41
	<i>P</i> ^b	<0.001	<0.001	0.03	0.02

^a Native T1 times available for all pediatric patients with hypertrophic cardiomyopathy and 14 controls (with 5 of these controls additionally excluded for feature tracking long-axis analysis)

^b *P* < 0.05 is considered significant. Correlation plots for tissue phase mapping velocities are shown in Fig. 6

r Pearson correlation coefficient