Analyzing Myocardial Torsion based on Tissue Phase Mapping MRI

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Target Audience: Radiologists and researchers interested in measuring the torsion motion of the left ventricular wall.

Purpose: The torsional motion of the left ventricle (LV) is known to play a crucial role in cardiac contraction and relaxation [1]. The kinematics of the microfibers having different orientation in the myocardial wall enables rapid blood ejection and refilling during the cardiac cycle. Torsion is considered to be a potential marker for cardiovascular disease and previous studies have attempted to quantify it using tagging MRI [2,3] or Doppler tissue imaging [4]. However, the rapid and complex rotational motion pattern of the heart muscle is difficult to quantify and the low spatial resolution of the functional information in MR-Tagging data limits the assessment of endo- versus epicardial differences. MRI Tissue Phase Mapping (TPM) [5] allows the quantitative assessment of myocardial velocities along all three principal motion direction of the heart (radial, long-axis, circumferential) with high temporal resolution and spatial resolution. Previous studies have shown the potential of TPM to detect disturbed regional myocardial function in patients with hypertrophy [6], cardiomyopathy [7], after heart transplantation [8], and other cardiac diseases. Moreover, TPM was shown to be sensitive to the temporal dynamics of rotational velocity during the cardiac cycle, producing a complex twist pattern [9]. Quantification of changes in LV cardiac motion induced by structural or functional abnormalities is important for assessing the impact of these cardiovascular diseases and better understanding their implications and severity. In this work, we present a method to quantify the myocardial and transmural (epicardial and endocardial) torsion using TPM data. The feasibility of TPM based torsion quantification to detect abnormal cardiac function was evaluated in a study with 27 patients with non-ischemic cardiomyopathy and 10 normal controls. In addition, changes in LV torsion were compared with standard clinical measures of cardiac abnormalities such as ejection fraction (EF), wall motion abnormalities (WM) and delayed gadolinium enhancement (DE).

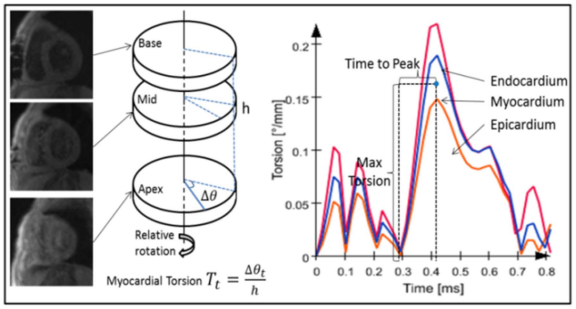

Figure 1: Left - Rotational velocity from TPM for base, mid, and apical slices determines the relative rotation angle Δθ between LV slices. The myocardial torsion is defined as Δθ normalized by the distance between slices. Right - base to apex torsion as a function of time computed for myocardium, epicardium, and endocardium independently.

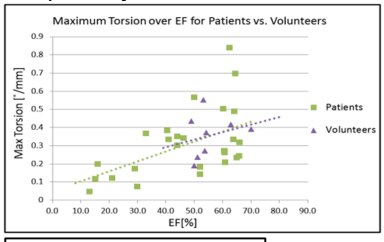

Figure 2: Maximum torsion over EF for patients with preserved and reduced EF and control subjects with normal EF.

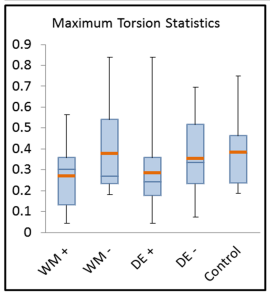

Figure 3: Maximum torsion for patient subgroups w/o wall motion abnormalities (WM+/-) and w/o delayed enhancement present (DE+/-), compared to a group of healthy control subjects.

Methods: TPM in the short axis orientation was performed on 27 patients (15 females, age 18 to 77) with non-ischemic cardiomyopathy with average EF = 47.4+/- 17.1 (min =15, max = 66), and 10 control subjects (6 females, age 20 to 48) with average EF = 58+/-8 (min =48.9, max = 71) using 1.5T MR systems (Aera and Avanto, Siemens Medical Systems, Erlangen, Germany). All patients underwent standard cardiac MRI including late gadolinium enhancement (DE) for the assessment of myocardial scar as well as ECG gated time-resolved (CINE) cardiac MRI for the evaluation of global cardiac function. In addition, patients underwent 2D TPM at basal, midventricular (mid), and apical locations. TPM consisted of a black-blood prepared cine phase-contrast sequence with three-directional velocity encoding of myocardial motion (venc = 25cm/s, temp res = 20.8ms, spatial res between 2.0-2.4 x 2.0-2.4 x 8mm). Spatio-temporal imaging acceleration (k-t GRAPPA) with a net acceleration factor of R_{net} = 3.6 was employed which permitted data acquisition during breath-holding (breath-hold time = 25 heart beats per slice). DE and cardiac CINE MRI were qualitatively evaluated by an experienced observer based on the AHA 16-segment model. LGE analysis included the assessment of presence of delayed enhancement (DE+). Regional wall motion was classified according to the following scheme: Normal wall motion (0), mildly hypokinetic (1), moderately hypokinetic (2), severely hypokinetic (3), akinetic (4), or dyskinetic (5). Abnormal wall motion (WM+) was noted if at least one segment was had grading >1. The TPM data was pre-processed to correct for eddy currents using a first order approximation. Static tissue with low standard deviation in all three velocity directions was automatically located (user interaction sometimes required to exclude artifacts). Semi-automatic myocardium segmentation was subsequently performed. Two manual contours were drawn for each slice in the systolic and diastolic intervals, which were then propagated over the entire image series based on the velocity field. The directly measured three-directional LV velocities (v_x,v_y,v_z) were projected using cylindrical coordinates into radial, long-axis, and rotational velocities as described as described in [5]. The average rotational velocity of each myocardial slice was normalized with the average myocardium radius for every time point to obtain the angular velocity. By integrating the angular velocity over time the rotation angle θ(t) was oblations. As shown in Figure 1-left, the myocardial torsion at time t between two slices (in this case base-apex) was defined as their relative rotation angle Δθ(t) normalized by the distance h between slices. Torsion was also computed for endocardium and epicardium independently after separating the myocardium mask at its center line. The maximum and time-to-peak (TTP) of the torsion curve, as shown in Figure 1-right, were calculated.

Results: Figure 1-right shows the evolution of myocardial torsion computed between apex and base over one cardiac cycle. The torsion curve reaches a maximum during the end-systolic phase when the LV is fully contracted. When distinguishing between endo- and epicardial regions, endocardial torsion is significantly higher than the epicardial in all subjects (p=0.0014), as shown in Figure 1-right, in accordance with the fact that sub-endocardial fibers show a more dynamic motion pattern. The average maximum endocardial torsion is 0.47 +/- 0.33 °/mm compared to 0.26 +/- 0.13°/mm for the epicardial. There was a significant relationship between maximum myocardial torsion and EF (r=0.52, p = 0.002) as shown in Figure 2 indicating that reduced global cardiac function negatively impacts LV torsion. Average max torsion is 0.23 +/- 0.11°/mm for the subgroup of patients with reduced EF, compared to 0.37 +/- 0.19°/mm for patients with preserved EF and to 0.38 +/- 0.16°/mm for the controls. When comparing the maximum torsion between subgroups of patients w/o wall motion abnormalities (WM+/-) and w/o visible delayed enhancement (DE+/-) in at least one AHA segment, as shown in Figure 3, we observed a trend towards reduced torsion for groups WM+ and DE+. However, in this case differences were not significant (p=0.08 and p=0.16, respectively).

Discussion: We proposed an approach for assessing myocardial torsion based on TPM capable of imaging myocardium rotation dynamics with transmural accuracy. For healthy volunteers the torsion parameter has a higher variability than the EF, which could be explained by the different positioning of base and apical slices, and there was no correlation to the EF which was within normal range for all control subjects. Apart from apex-base torsion, we can measure base-mid and mid-apex torsion, which vary in amplitude depending on slice positioning. Although for the small groups of patients and volunteers considered in this work the difference in measured torsion has not reach the 0.05 p-value significance, torsion supplies additional information about the changes in cardiac motion induced by diseases and could be more sensitive to their early manifestation. The limitations of our method are sensitivity to noise (especially in apical slices) which may cause uncertainty in the myocardium segmentation, but also the limited temporal resolution which may not always capture the fastest temporal variations in the velocity field. This could be one reason why the maximum torsion shows high variability among healthy subjects, but further investigation is required to establish the normal limits of these variations. In the future, analyzing myocardial torsion from TPM data with even higher temporal resolution would enable us to better understand the rotation dynamics of the LV.

References: [1] Greenbaum et al. *Br Heart J*. 1981, 45:248-263; [2] Rüssel et al. *JCMR* 2008, 10:26; [3] Buchalter et al. *Circulation* 1990, 81:1236-1244; [4] Notomi et al. *Circulation* 2005, 1140:1146; [5] J Magn Reson Imaging 2006;24:1033-9; [6] Eur Radiol 2013;23:339-47; [7] J Magn Reson Imaging 2013;37:119-26; [8] Foll, et al. *Eur J Cardiothorac Surg* 2013: ezt448; [9] Jung et al. *Cardiovasc Magn Reson* 2012, 14-87.