# The patho-physiological sensitivity of cardiac MR elastography: Preliminary results.

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**Introduction:** Elastography is capable to directly measure tissue stiffness and therewith to provide a quantitative method for 'palpating' internal organs [1]. This is particularly interesting for cardiac applications since function is here based on the alteration of the stiffness, i.e. the shear modulus of the myocardium. Recently, cardiac MR elastography (MRE) was demonstrated by measuring elasticity changes during the cardiac cycle in pigs and healthy volunteers [2-4].
**Problem:** So far cardiac MRE was only tested on animals and healthy volunteers. Up to now the amplitudes of shear waves inside the left ventricle (LV) have been evaluated with respect to their systolic values. Amplitude-based elastography requires normalization in order to deduce a generalized metric independent of experimental conditions and individual variations. No such metric has been proposed for diagnostic applications of cardiac MRE.
**Objective:** Normalized LV-shear wave amplitudes are measured in healthy volunteers (n = 11) and in patients with cardiac pathology (n = 11) to test the hypothesis that an increase in myocardial stiffness causes a decrease in wave amplitudes detectable by MRE.

**Methods:** Cardiac MRE was performed on a 1.5 T Scanner (Siemens Avanto) as described previously [2]. Oscillating shear waves of 24.3 Hz frequency were introduced through the anterior chest wall using a remote oscillator. A steady-state GRE-MRE sequence synchronized to the vibration with 8 TR = one wave cycle was used to encode three Cartesian wave components in a short cardiac axis view. Shear wave amplitudes were calculated by temporal Fourier-transformation, followed by a phase correction to maximize the real part of the 24.3-Hz signal intensity and summation of all three real-part signals corresponding to the three Cartesian vector components. Normalization of LV-amplitudes was performed by a second region-of-interest (ROI) located at the interface between heart and anterior chest wall. ROI-positions were manually traced on the magnitude images of the MRE scans.
**Patients and volunteers:** 11 patients (2 females; mean age 57 years, mean BMI 28.9) of the cardiac outpatient department with impaired relaxation diagnosed by transthoracic echocardiography were compared to 11 healthy male volunteers (mean age 37 years, mean BMI 23.8.) with no cardiac history and normal findings in echocardiography.

**Results:** Cardiac MRE could successfully be performed in all volunteers and patients. Averaged LV-amplitudes were 0.628 ± 0.181 mm in volunteers and 0.377 ± 0.088 mm in patients. Amplitudes near the interface were determined with 0.972 ± 0.233 in volunteers and 1.004 ± 0.232 in patients. LV-amplitudes are significantly lower in patients ($p < 0.001$) while there is no significant correlation between the subgroup and their amplitudes at the anterior heart boundary ($p = 0.753$). The diagnostic performance is even improved by consideration of normalized amplitudes given by the ratios between LV-amps / interface-amps. These ratios are 0.643 ± 0.077 and 0.384 ± 0.086 for volunteers and patients, respectively. A cutoff value of 0.53 resulted in a perfect separation of patients and volunteers by means of normalized wave amplitudes.

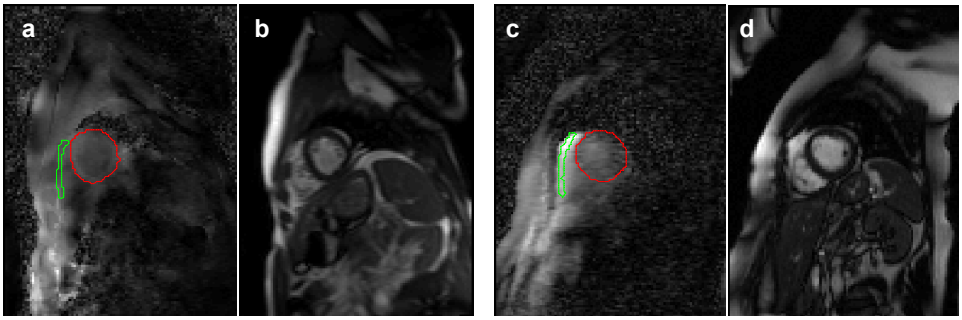


**Fig. 1:** (a) image showing the wave amplitudes resulting from 24.3-Hz vibration on top of the thorax and (b) corresponding cine-MRI in short-cardiac axis of a healthy volunteer. LV- and interface-ROI are demarcated by red and green lines, respectively. (c), (d) same as in (a) and (b) but for a patient with history of hypertension and impaired relaxation (VE/VA=0.63).

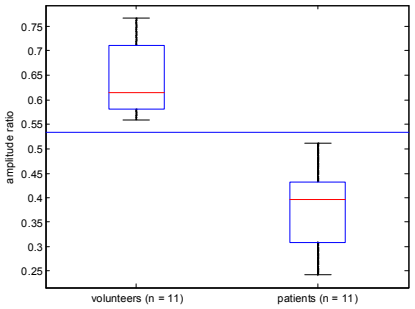


**Fig. 2:** Boxplots of averaged ratios LV-amps / interface-amps. The patient group displays LV-relaxation abnormalities which apparently show an effect on the externally induced shear wave amplitudes. The plots depict the lower and upper quartiles as well as the 50th percentile (median). Full data range is presented by the whiskers.

**Discussion and conclusion:** Our results clearly indicate the patho-physiological sensitivity of amplitude-based cardiac MRE. It was shown in [2] that wave amplitudes vary corresponding to the stiffness in the wave-transmitting medium. A model of constant energy flux predicts that increasing elasticity causes decreasing wave amplitudes. From this relation our hypothesis was derived by assuming that impaired myocardial relaxation associated to increased left ventricular stiffness causes the wave amplitudes to decrease. Our results confirm this hypothesis. However, in our preliminary study the influence of age to myocardial stiffening [5] was not accounted for. Therefore, the final threshold for the diagnosis of cardiac relaxational dysfunction and its diagnostic significance remains still to be determined. Ongoing studies are conducted to address this issue. In summary, cardiac MRE is a very promising modality for the identification of increased myocardial stiffness as a marker for cardiac pathology.

**References:**
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