Single-shot cardiac MR Elastography

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Background: In vivo assessment of the mechanical properties of the myocardium in different stages of the heart cycle may help to improve an early detection of myocardial dysfunction. MR elastography (MRE) provides a means to obtain information about the elasticity of an organ through the coupling of mechanical shear waves into the body and their detection by MR imaging [1]. Although MRE has unique capabilities for the measurement of 3D wave fields, in cardiac applications it still suffers from relatively slow and repetitive data read-out [2]. Therefore, a single-shot echo planar imaging (EPI) cardiac MRE technique is introduced which provides harmonic displacement images of the heart within 20 to 70 ms acquisition time depending on the desired motion sensitivity and image resolution.

Theory: The amplitude of an incident harmonic shear wave is inversely proportional to the stiffness of the medium. The heart cycle comprises myocardial contraction (systole) and relaxation (diastole) - physiological states which influence the amplitudes of shear waves propagating through the heart. Evaluation of the change of wave amplitudes over the heart cycle may thus provide a means for the early detection of myocardial dysfunction [3].

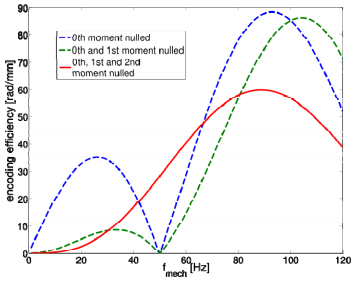

Fig. 1: Encoding efficiency of the motion-encoding gradient (MEG) versus shear wave frequency. In cardiac EPI-MRE, a MEG corresponding to the red graph was used. The dashed graphs correspond to gradients with lower order gradient moment nulling.

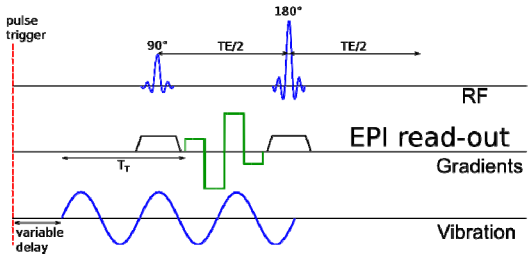

Fig. 2: Schematic representation of the imaging sequence in cardiac EPI-MRE (not to scale). The diagram shows the acquisition of one single image. All gradients are shown on the same axis, although the MEG (green waveform) was consecutively applied on each of the three gradient axes for acquiring the vector wave field in one slice.

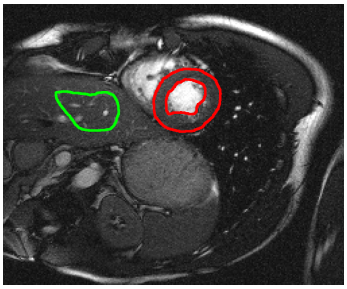

Fig. 3: The regions of interest (ROI) for the myocardium of the left ventricle (enclosed by red lines), blood pool (inner red line) and liver (green line) superimposed onto standard cine MRI.

Methods: Experiments were performed on a clinical 1.5 T scanner (Siemens, Erlangen, Germany). The mechanical vibration was generated by a modified loudspeaker at the end of the patient table and induced into the heart from the anterior chest wall by a wooden transducer piston. A single-shot spin-echo EPI sequence equipped with a motion-encoding gradient (MEG) of 20 ms duration and a periodicity of 100 Hz was used. The 0th, 1st and 2nd moment of the MEG were nulled for two reasons: i) to minimize low-frequency components of tissue motion due to blood flow and intrinsic heart motion and ii) to avoid nulls of the spectral encoding sensitivity around 50 Hz as illustrated in Fig. 1. The onset of the sequence was initiated by the pulse oxygenation trigger. After a variable delay time of 0, 100, ...1600 ms the mechanical vibration of either 50 or 60 Hz was started. After 100 ms of vibration the imaging sequence was started. In each of these measurements, 12 images were acquired (4 phases of the mechanical vibration x 3 orthogonal MEG directions) during a single breath-hold in expiration. The spatial resolution was 3.1x3.1x6.2 mm^3 with a 128x96 matrix. Echo time was 49 ms and acquisition time for a single image was 70 ms (excluding the preceding vibration). Phase data were unwrapped using a spatial gradient of the complex phase and then temporally Fourier-transformed. The amplitude of the vibration was calculated from all Cartesian directions and averaged over the ROI demarcated in Fig. 3.

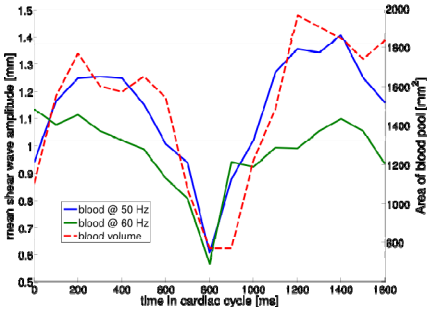

Fig. 4

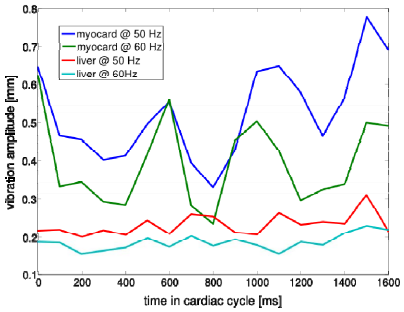

Fig. 5

Fig. 4: Mean shear wave amplitude in the blood at different time points in the heart cycle. The dashed red line shows the area of the blood pool in the image slice. The R-R interval was 1000-1100 ms. A pulse trigger on the subject's finger was used so that the time axis is delayed about 200-300 ms with respect to the R-wave.

Fig. 5: Mean shear wave amplitude in the myocardium and in the liver for reference.

Results: Mean vibration amplitudes versus the time in the cardiac cycle for myocardium and blood are shown in Figs. 4 and 5, respectively. The area of the blood pool enclosed by the myocardium is overlaid as the red dashed line in Fig 4. In animated phase images, small waves were discovered circulating around the myocardium.

Discussion: Due to boundary effects shear wave properties of the heart wall are carried into the confined blood pool resulting in significant wave amplitudes inside the ventricular cavity. Therefore, high wave amplitudes were observed during the diastolic state within the blood (Fig. 4) corresponding to myocardial relaxation. At around 100-200 ms, isovolumetric contraction begins; with the blood volume essentially unchanged while wave amplitudes decrease reciprocally to the increasing stiffness of the myocardium. Systole begins at about 400 ms with rapid decrease of the blood volume. The rise of amplitude at 600 ms in the myocardium is not fully understood and is likely caused by heart motion during contraction or amplitude pile-up due to geometrical resonances of circulating waves within the wall. During transition from systole to diastole (~800 ms) the myocardium relaxes, giving rise to higher wave amplitudes. At about 1000-1100 ms the initial state is restored and the next heart cycle starts. As expected, wave amplitudes in the liver are not affected by heart motion.

Conclusion: Cardiac single-shot EPI-MRE allows one to study multiple effects of wave dynamics in confined media with time-varying elastic and geometrical properties. Wave image acquisition can be accelerated to up to 20 ms through parallel imaging and reduced matrix size. Such high-speed cardiac EPI-MRE facilitates the time-resolved evaluation of wave amplitudes as a possible marker for myocardial dysfunction.

Literature: [1] Muthupillai and Ehman. Nature Med 1996;2(5):601-603. [2] Rump et al. Magn Reson Med 2007;57(2):388-395. [3] Elgeti et al. J. Cardiovasc. Magn Reson 2009;11(1):44.