Non invasive measurement of the volume-pressure work of the human heart by cardiac MR elastography

T. Elgeti[1], J. Rump[1], D. Klatt[1], U. Hamhaber[2], S. Papazoglou[1], J. Braun[2], B. Hamm[1], and I. Sack[1]
[1]Department of Radiology, Charité Universitätsmedizin Berlin, Berlin, Germany, [2]Institute for Medical Informatics, Charité Universitätsmedizin Berlin, Berlin, Germany

PURPOSE: The pressure-volume relationship is a reliable index for assessing myocardial contractility in the intact circulation. However, its use is clinically limited as exact intracardiac pressure changes can be measured only invasively [1,2]. It is proposed that in vivo time-resolved cardiac magnetic resonance elastography (MRE)[3] enables the non invasive assessment of myocardial stiffness changes which are related to blood-pressure changes inside the ventricular cavities [4]. Therefore, an experiment is developed that identifies variations of the myocardial wall pressure during the cardiac cycle by measuring changes in externally induced shear-wave amplitudes. The MRE-pressure curves are compared with left ventricular volumes registered in the same MRI examination.

MATERIAL: Six healthy male volunteers underwent cardiac MRI on a clinical 1.5 T scanner (Siemens Magnetom Sonata). First 3D left ventricular volume data were acquired using balanced cine-SSFP-MRI. Volume time curves were obtained by 2D-segmentation of the left ventricular endo- and epicardial border in the short axis view over the entire cardiac cycle. Secondly, an ECG-gated, motion-sensitized and segmented spoiled GRE-MRE sequence was used for acquiring 360 phase-contrast wave images with a temporal resolution of 5.6 ms in the short-axis view over two heart beats. A custom-made remote oscillator was used to vibrate the thorax with 24.3 Hz synchronized to the MRE sequence. An autocorrelation analysis allowed separating induced phase-signal oscillations from the contributions of intrinsic heart motions and blood flow. The remaining wave amplitudes ($u(t)$) were attributed to the wall pressure $\mu(t)$ by

$$\mu(t) = \left(u_{sys} / u(t)\right)^4 P_{sys} \qquad \text{(Eq.1)}$$

where u_{sys} and P_{sys} denote systolic wave amplitudes and systolic pressure. Eq.1 was derived from a linear elastic material model assuming a constant flux of wave energy and a linear blood-pressure response to the myocardial contraction.

RESULTS AND DISCUSSION: The figure shows typical volume-pressure (VP)-time functions and VP-cycles acquired in six healthy volunteers. In principal, the contractile branches of the VP-cycle displayed steep slopes indicating the isovolumetric rise in myocardial wall tension. Volunteer 2 showed a mild insufficiency of the mitral valve in the MRI examination and a less steep slope. This diagnosis was confirmed by echocardiography. The mean isovolumetric contraction time of the other volunteers was measured with 33 ± 20ms ms. Isovolumetric relaxation times were estimated from the delay between the maximum negative wall-pressure rise and the left ventricular dilation. The interindividual average was found with 58.4 ± 20ms. Aside that of volunteer 2, all measured VT-cycles indicate that the decrease in wall tension coincides with small volume changes, i.e. the conservation of volume during relaxation is not as pronounced as seen during contraction. This deviation between the systolic increase of wall pressure and the diastolic relaxation is presumably attributable to the hyperelastic behavior of the myocardium. The mean VP-work of all volunteers per cardiac cycle was 6520 ± 1006 mm Hg ml and equivalent to 0.87 ± 0.1 J, similar to values in the literature [5].

CONCLUSION: The sensitivity of shear waves to myocardial tension is the first noninvasive probe of ventricular pressure evolution. The experiments demonstrate that cardiac MRE is capable of detecting shear wave amplitude variations driven by the alteration of myocardial tension during the cardiac cycle. In combination with volume data obtained by cine-MRI cardiac MRE can characterize the ventricular pressure-volume evolution in the human heart. Thus, cardiac MRE has the potential to assess diastolic dysfunction.

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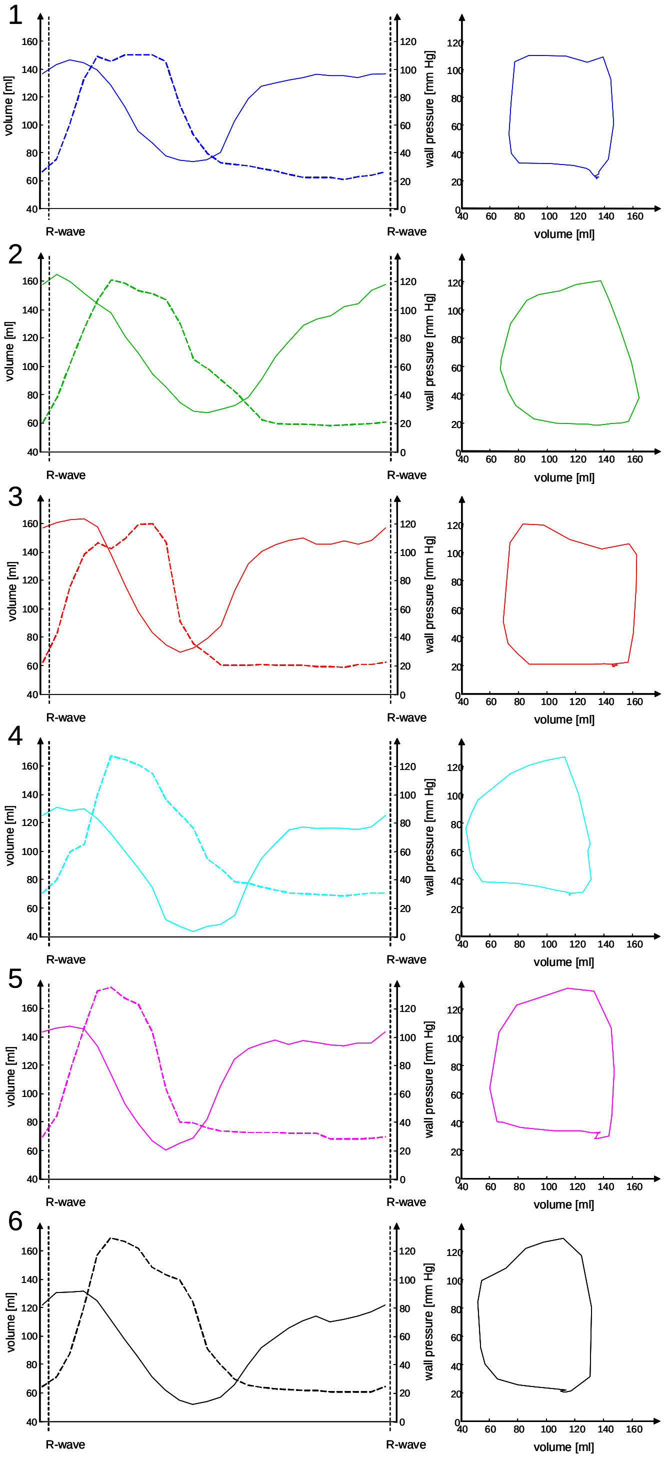

Figure 1 a) left side: Graphs showing the course of left ventricular volume (solid line, 3D-volumetry) and the wall-pressure course (dashed line, MRE) over 1 complete RR-interval in six healthy male volunteers (numbered 1-6)
Figure 1 b) right side: Resulting VP-cycles of the volunteers; the enclosed area represents external VP-work.