Quantification of myocardial volume change during systole and diastole using 3D-DENSE

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Aim: To measure the cyclic intramyocardial tissue volume change in canine hearts non-invasively between systole and diastole using MRI.

Introduction: Contraction and relaxation of the heart muscle results in periodic oscillations of the intramyocardial pressure, which is reported to cause a fluctuation of the vascular volume in the rat heart (1). This amounted to between 8.1% and 3.3% of the total tissue volume from endocardial to epicardial layers. The mechanism of this change can be either local water redistribution or global transport of blood in and out of the tissue. The latter scenario would cause the tissue volume to fluctuate accordingly by as much as 8%: the cyclic movement of such a large fraction of the vascular volume may significantly alter the oxygen gradients and the degree of perfusion heterogeneity in the tissue as understood in a unidirectional flow model. However, to the authors' best knowledge, it has not yet been possible to perform myocardial volume measurements non-invasively.

The MRI pulse sequence known as DENSE (2) has been shown to measure displacement taking place in time intervals compatible with normal systole and diastole durations. Local tissue volume change is associated with the deformation of the tissue as represented by the 3D strain tensor. The strain tensor can be measured with a 3D DENSE sequence. This paper presents measurements of regional tissue volume changes in the endo-, mid- and epicardial layers in canine hearts between systole and diastole.

Methods: Seven closed-chest beagles (2 male, 5 female, weight = 9.5 – 12.5 kg) were used, six of which were successfully scanned. Animals were ventilated and paced using an intraventricular catheter, pacing and ventilation being phase-locked to allow imaging during the same phase of the respiratory and cardiac cycles.

Imaging was performed in a Siemens 1.5 T Magnetom Sonata (Siemens Medical Systems, Erlangen, Germany) using a 3D DENSE sequence. A FISP (3) like readout was used for acquisition, consisting of a 128 x 64 x 16 acquisition matrix, 16 k-space lines per breath-hold. FOV was 192 x 120 x 48 mm or 160 x 100 x 48 mm. Strain tensor measurements from end diastole to end systole and vice versa were performed, which allowed us to calculate volume changes during both the systolic period and the diastolic period. To remove instrument related errors, reference data of volume changes between the same cardiac phases were acquired. Each experiment and its corresponding reference constitute a dataset.

Once acquired, raw data were processed using IDL (Research Systems Inc., Boulder, CO, USA). DENSE encodes displacements into phase maps; these phase maps are unwrapped and converted to displacement vectors. Strain tensors are then derived from the spatial derivatives of the 3D displacement field. The left ventricular wall of each heart is divided into three concentric layers and the volume change of each layer is calculated.

Results: Figure 1 contains the results (mean and standard deviation) averaged over the six animals. The shaded columns are the decrease during the systolic period (negative values), and the un-shaded columns the increase during the diastolic period (positive values). Since both measure the difference between end systole and end diastole, the sum of the two should be zero, which is confirmed by the results.

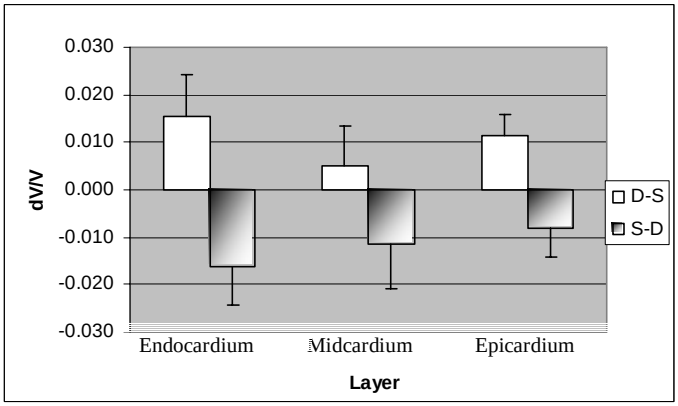

Figure 1: Volume change in the three cardiac layers.

Conclusion: The measured volume change is about 1 % of the tissue volume in all layers of the wall. To a 95 % confidence level, the volume change is statistically significant in all cases except the mid layer during systole. This result is only about 15 % of the one reported by Toyota el al. (1) in rats, and amounted to the transport of 1.2 ml/g/min of blood in and out of the tissue. This level of volume change is still significant in light of the mean perfusion level of approximately 1.0 ml/g/min in the myocardium, and is consistent with arterial and venous flow being completely out of phase. The fact that all layers of the wall experience similar volume fluctuations suggests that the pressure gradient instead of the absolute pressure may have more influence on the capillary flow dynamics. Further studies will investigate the link between capillary flow and local pressure gradient fluctuation in regions of normal and impaired contractile function.

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