Of Mice and Men: A quantitative comparison of 3D cardiac motion in mice and humans

E. Dall'Armellina[1], H. Barnes[1], S. Neubauer[1], S. E. Petersen[1], M. Markl[2], J. E. Schneider[1], and B. A. Jung[2]

[1]Department of Cardiovascular Medicine, University of Oxford, Oxford, Oxon, United Kingdom, [2]University Hospital Freiburg, Freiburg, Germany

Introduction: Tissue Phase Mapping (TPM) is a well-established technique to assess regional cardiac function in humans [e.g. 1,2]. Only two studies reported on the application of this technique in mice [3,4], none of which analysed the time course of the radial, rotational (i.e. tangential) and longitudinal velocity components of the myocardium. The aim of our study was to perform a detailed analysis of transmural wall motion in normal mice, and to quantitatively compare the murine velocity patterns to those found in human hearts.

Methods: TPM on humans was performed on a 1.5T Sonata MR-system (Siemens, Germany) and murine measurements on a 9.4T VNMRS DirectDrive MR-system (Varian Inc, USA). In both experiments, a black blood multi-frame gradient echo sequence with prospective ECG-gating combined with navigator gating during free breathing (humans) or respiratory gating (mice) was applied. Three slices in short axis view (basal, mid, apical) were acquired in 12 volunteers (30 ± 4 yrs) and 5 healthy C57/Bl6 mice (22.9 ± 0.6 g).

	Mice	Humans
Matrix size	128^2 (256^2 interp.)	256 x 96 (192 interp.)
Spatial resolution [mm]	0.1 x 0.1	1.3 x 1.3
Slice thickness [mm]	1	8
Temporal resolution [ms]	4.6	13.8
Venc in-plane [cm/s]	6	15
Venc through-plane [cm/s]	8	25

Table 1: Parameters for mice and human measurements.

Data post-processing was performed using customized software programmed in Matlab. After contour segmentation and a correction for translational motion components, the measured in-plane velocities were transformed into an internal polar coordinate system positioned at the center of mass of the left ventricle. As a result, motion parameters are decribed in terms of radial, rotational and longitudinal velocities. To avoid temporal jitter, the time axis was normalized to the end-systolic time as defined by the first minimum peak of the global radial velocities during diastole (see Fig. 2). This minimum could be observed in all measurements (isovolumetric relaxation). Global velocity time courses were calculated as the mean of all myocardial pixels within the segmentation mask.

Results: Fig. 1 shows velocity vector field plots and color-coded maps of radial velocities in a basal slice for four characteristic time frames during the cardiac cycle for mice (top rows) and humans (bottom rows). The counterclockwise rotation during early diastole (isovolumetric contraction) is less pronounced in mice. The radial velocities show a similar behavior in all cardiac frames whereas the rotation during systole in mice does not change the direction compared to humans as indicated by the arrow plot. Fig. 2 illustrates that the velocity twist in humans during systole between basal and more apical slices is inverted in mice. Furthermore, radial velocities demonstrate a slight discrepancy between different slices during diastole (later expansion of more basal slices) as well as an additional slight expansion during early systole. Time courses of longitudinal velocities demonstrate agreement in all slices. Note that the peak velocities in mice are a factor of ~3 lower for all velocity components compared to humans.

Discussion: Although fewer mice compared to human subjects were studied so far, this work illustrates that the motion pattern of normal mouse hearts is similar to the human heart for radial and longitudinal, but inverted for tangential velocities. About 20-30% of the murine cardiac cycle (corresponding to the mid-diastolic phase) was not covered due to the black-blood module at the end of the cine train, and to allow for variations in heart rate. Therefore, together with the 8-10-fold higher heart rate in the mouse, the sampling density in murine experiments was only about 50% compared to humans – despite the 3x higher temporal resolution. While the motion pattern of mouse hearts is in agreement with previously published work (4), absolute peak velocities were higher in our study. Work is in progress to perform a segment-by-segment comparison between the two species.

Acknowledgements: This work was funded by the British Heart Foundation and supported by the British Council.

References:

[1] Hennig et al. JMRI 1998;868. [2] Jung et al. JMRI 2006;1033.
[3] Streif et al, MRM 2003; 49:315. [4] Herold et al. MRM 2006; 55:1058-64.

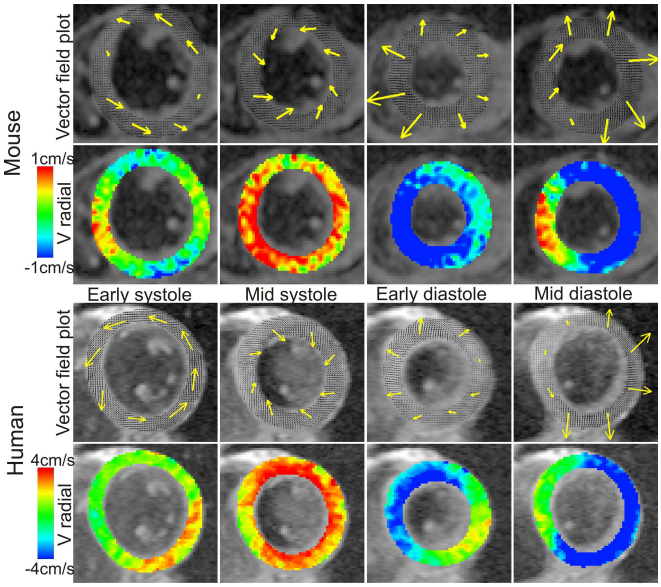

Fig.1: Pixel-by-pixel vector field plots and color-coded maps of radial velocities of in-plane velocities in a basal slice for four characteristic cardiac frames. Note the different rotational behavior during mid systole in the vector field plots for the human subject and the mouse.

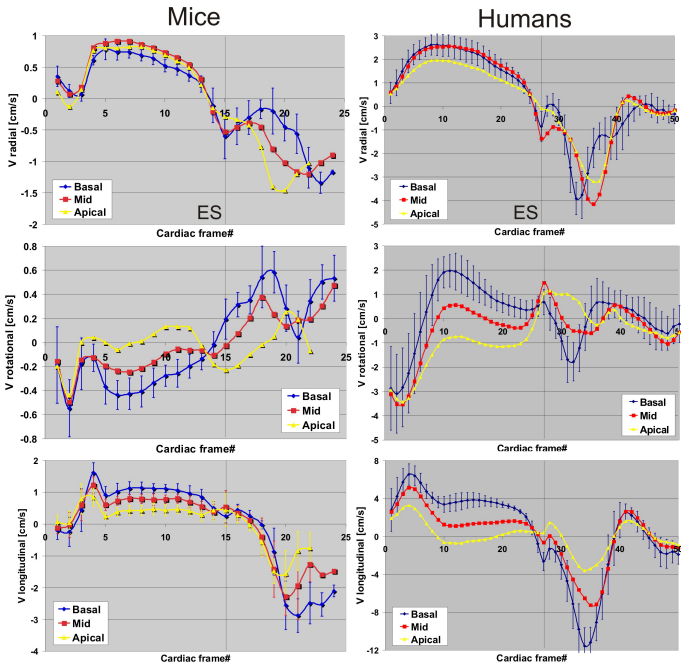

Fig.2: Global velocity time courses for all velocity components averaged over 5 mice (panels in left column) and 12 humans (panels in right column). The systole of the time courses was normalized as indicated by the end-systole or early diastole (ES). Standard deviations are only shown for the basal slice for better visualization purposes but were comparable for mid and apical slices.