

Phase-Contrast Measurements Of Aortic Wall Strain *In Vivo*

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Introduction

We expect *in vivo* studies of the relative strain in the wall of the aorta to help us understand why the abdominal aorta is more prone to aneurysms than the thoracic aorta. To measure vessel wall strain, we used a previously validated, cine phase-contrast technique [1]. The advantage of using MR over ultrasound is the ability to image the full cross-section of the vessel, taking the entire wall motion into account. Tagging [2] is not appropriate in this instance because there are very few pixels across the wall and signal is low. Using velocity data also enables us to detect displacements smaller than a pixel width, which is the limitation of magnitude image based techniques [3]. The purpose of this study was to assess the feasibility of our strain calculation technique in healthy volunteers.

Methods

The thoracic (n=4) or abdominal (n=4) region of the aorta was scanned in eight healthy volunteers using a 1.5T GE CV scanner (40 mT/m, 150 mT/m/ms). Spoiled gradient echo images were used to localize cross-sectional planes of the descending aorta either at a level near the aortic valve or half-way between the renal arteries and the iliac bifurcation. Through-plane blood flow was measured with cine phase contrast (TR/TE/flip = 18ms/6ms/20, 16kHz BW, 200mm FOV, 256x256, 5mm slice, VENC = 200cm/s). The in-plane motion of the wall was measured with 3-directional cine phase contrast (TR/TE/flip = 30ms/9ms/20, 16kHz BW, 200mm FOV, 256x256, 5mm slice, VENC = 5cm/s in-plane, 200cm/s through-plane, NSA = 2, and RF spoiling). Slabs outside the slice were pre-saturated to create a dark lumen. Respiratory compensation was used during free breathing for all velocity measurements.

Calculations of change in vessel radius and relative strain were done within Matlab (Natick, MA). Points manually placed in the vessel wall on one time frame were connected using a cubic spline. The velocities along the spline were used to use to calculate the change in radius over time. Only once was the radius of the spline fit itself used. The smallest spline radius was used as the starting point for the forward-backward interpolation scheme [4] that calculated all other radii from the measured velocities. The use of velocity data allows displacements less than a pixel in magnitude to be measured.

The changes in radius were used to calculate circumferential, or hoop, strain $\epsilon_{\theta\theta}$. Given that deformations *in vivo* are on the order of 5%, a large strain formulation was used

$$\epsilon_{\theta\theta} = \frac{1}{2} \left[\left(\frac{r_n}{r_o} \right)^2 - 1 \right] \quad [1]$$

The variance of the velocity data,

$$\sigma_v^2 = \frac{2 * v_{enc}^2}{\pi^2 * SNR^2} \quad [2]$$

was propagated through the calculations of radius and strain.

The relative strain was plotted as a function of time. It is expected that the changes in strain follow changes in pressure. While we were not able to measure local pressure, we do have the local through plane flow, which is known to lead the pressure. Therefore, strain should lag the flow waveform.

Results

All eight cases yielded reasonable flow and strain curves, with the strain lagging the flow as expected. The maximum and mean strains were 4-8% and 1-4%, respectively, which are in the

expected range. Figure 1 summarizes the data. The errors in the strain measurements were 5-10% of the calculated values.

Typical flow and strain waveforms for both the thoracic and abdominal regions are also shown alongside magnitude images of the region of interest. Velocity vectors are superimposed on the magnitude images. Rigid body motion has been subtracted.

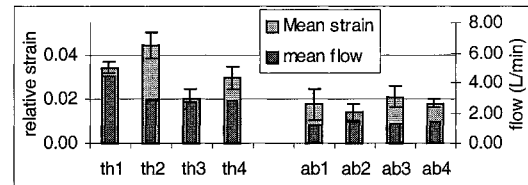


Figure 1: Mean strain and flow in the thoracic (th) and abdominal (ab) aorta.

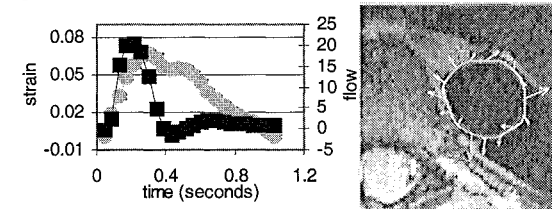


Figure 2: Thoracic Aorta flow in L/min (dark) and strain waveform (light) beside magnitude image of time point 4 with velocity vectors superimposed.

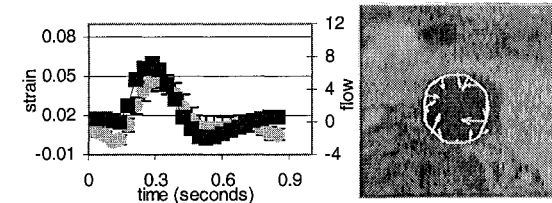


Figure 3: Abdominal aorta flow in L/min (dark) and strain waveform (light) beside magnitude image of time point 12 with velocity vectors superimposed.

Discussion

One hypothesis is that the stiffer the vessel wall is, the more prone the vessel is to vascular disease. In the normal volunteers, the abdominal strains were lower than thoracic strains. The shape of the strain waveform was also reproducibly different between the abdominal and thoracic levels. Further bio-mechanical research will have to be done to determine the significance of this.

The plots of the velocity vectors at each time point revealed that the aorta did not, at either level, undergo uniform motion. A more general analysis formalism is being developed to handle this.

Conclusions

The technique was successful in all eight cases, and differences were noticed between thoracic and abdominal strains. The most prominent difference was the shape of the strain waveform over time. More research is needed to determine the prevalence and significance of this finding.

References

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Acknowledgments

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