Vascular MR Elastography: New Developments

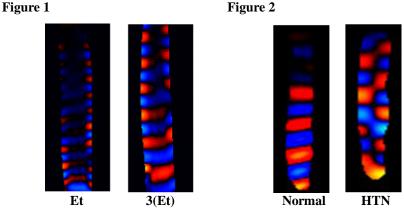
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¹Radiology, Mayo Clinic, Rochester, Minnesota, United States, ²Cardiology, Mayo Clinic, Rochester, Minnesota, United States **Background:** Hypertension affects over 140 million people in North America, many of which do not know they have it. This condition is one of the most important risk factors in the subsequent development of cardiovascular diseases leading to premature death. Hypertension and subsequent arterial stiffness are determined by vascular smooth muscle tone and by the elastin/collagen content of the vessel wall.¹ Magnetic resonance elastography (MRE) is a new technique to image tissue motion in the order of hundreds of nanometers enabling the imaging of the physical properties of tissue such as stress and strain. Our hypothesis is that MRE can be used to image early hypertensive changes enabling targeted therapy and prevention of secondary cardiovascular disease. We will demonstrate that MRE can be applied to blood vessels and can identify early hypertensive changes.

Methods: Using a vessel phantom model of thin-walled latex tubing, harmonic mechanical pressure waves were applied at frequencies of 100-500 Hz, and imaged using a modified phase contrast MR technique sensitive to cyclic motion.² Wall properties and tension were varied by changing the static pressure and wall thickness. Wavelength measurements were compared to theoretical predictions based on a mechanical model. Further experiments were performed using ex-vivo excised aortic segments of hypertensive and normal control pigs. Hypertensive pigs had a mean arterial pressure significantly higher than control pigs (115 ± 11 vs. 173 ± 12 mmHg, p=0.05). Mean duration of hypertension, induced by renal artery stenosis, was 3 months. Wavelength was measured for each frequency applied (100-500 Hz) and normalized to a stimulation frequency of 100 Hz. Initial histologic examination was performed on the ex vivo aortic segments to measure wall thickness.

Theory: The mechanical model used to predict wave propagation in our system is the thin-walled elastic tube model published in <u>Biomechanics Circulation</u> by Y.C. Fung. Using this model, the Moens-Korteweg formula predicts the wave speed to be: $C = \sqrt{(Et/2pa)}$, where c is wave speed, E is Young's modulus, t is wall thickness, ρ is filling fluid density, and a is the inner radius. Furthermore, the model predicts that tension and pressure have a direct linear relationship. From the wave speed equation, we can predict that changing the Young's modulus-wall thickness product will increase wave speed and wavelength by the square root of the change.

Results: The experimental results demonstrated that propagating mechanical waves with amplitudes as small as 1 micron in the model vessel wall can be readily visualized. The measured wavelength varied with changing wall thickness and tension in agreement with our model. Varying the wall thickness and Et product, with constant stimulation frequency, progressively increased the wavelength as demonstrated in Figure 1.



In the ex vivo aorta segments, the measured wavelength for each applied frequency was longer in the hypertensive vessels $(5.83 \pm 0.53 \text{ cm} \text{ at } 100 \text{ Hz})$ compared to normals $(4.87 \pm 0.35 \text{ cm} \text{ at } 100 \text{ Hz}, \text{ p} < 0.05)$ (Figure 2). The initial histologic analysis demonstrates no significant difference between the intima-media wall thickness in the control $(0.49 \pm 0.12 \text{ mm})$ or hypertensive $(0.54 \pm 0.31 \text{ mm})$ aortas. **Conclusions:** The results indicate that MR Elastography is a sensitive method for examining changing wall mechanical properties. Furthermore, the technique can be applied to ex vivo aorta vessels and can detect early intramural hypertensive changes that alter the elastic properties of blood vessel walls before significant changes in wall thickness. This suggests that hypertension produces changes in the elastic properties of the vessel before the wall thickness changes. **References:** 1. Van Bortel et al., AJH 15:445-452, 2002. 2. Manduca et al., MIA 5:237-254, 2001.