

# Spatial Heterogeneity of Carotid Artery Wall Strain Using Displacement-Encoded MRI at 1.5T and 3.0T

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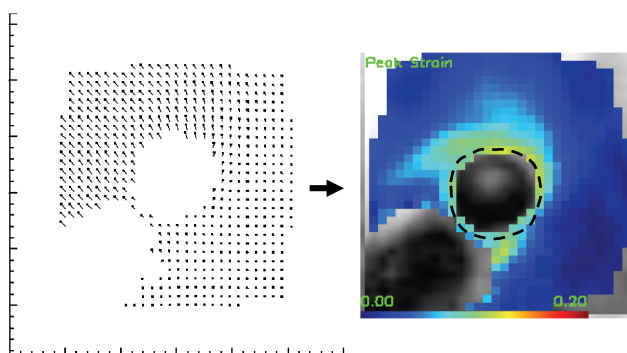
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## Introduction:

Rupture of the atherosclerotic plaques in the carotid arteries results in stroke, one of the leading causes of death and disability across the world [1]. Arterial wall strain has been shown to not only be correlated with but may also be an underlying cause of atherosclerosis [2]. Current methods of measuring carotid arterial distension utilize changes in lumen area over the cardiac cycle to provide global measures of strain. These measurements can not provide regional localization of strain, presumably the site of atherogenesis. Recent studies have shown that displacement encoding with stimulated echoes, or DENSE MRI sequence can provide a localized carotid arterial wall strain map [3]. While this technique has been validated for global strain [4], regional heterogeneity has not been explored. It is therefore the objective of this study to determine regional variation in carotid lumen wall and furthermore to determine the inter-operator variability in post-processing at both 1.5T and 3.0T.

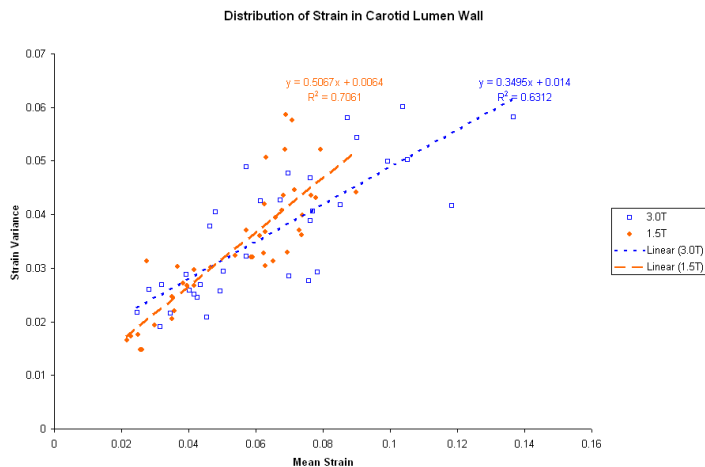
## Methods:

Normal volunteers were consented and scanned on either a 1.5T Siemens Sonata (n = 4, male, ages 28 - 37) or 3.0T Siemens Trio MRI scanner (n = 17, 7males 10 females, ages 18 - 26) under IRB approval. A total of 76 DENSE strain measurements were acquired at 1.5T (n=42) and 3.0T (n=34) using a fully-balanced SSFP readout with 0.60 mm in-plane resolution as previously described [3,4]. Slices were placed at the common carotid artery approximately 1 cm below the bifurcation. The scans were triggered by the R-wave of the ECG, and image acquisition was consistently placed at the time of maximum lumen diameter judging from the cine scans, while in two separate scans the encoding portion was placed at 40 ms and 80 ms after the R-wave to capture the maximum wall strain and intermediate strain. Displacement encoding was acquired in three oblique directions to produce a pixel-by-pixel 2D axial displacement map. Circumferential strain for each pixel was calculated by taking all quadrilateral elements of neighboring pixels in post-processing as shown in Figure 1. Once the strain map was obtained, the contour of the vessel wall was manually drawn and the average and variance of the pixels over the contour was calculated as shown by the dotted line in Figure 1. To determine the inter-operator variability in post-processing, three independent operators performed the post-processing blinded to others results.



## Results:

The average strain in the lumen wall at maximum lumen diameter for 1.5T was  $0.076 \pm 0.006$  with a variance of  $0.040 \pm 0.010$ . At 3.0T the average strain was  $0.072 \pm 0.039$  with a variance of  $0.040 \pm 0.013$ . The mean strain measurements were plotted against strain variance as shown in Figure 2. At both 1.5T and 3.0T a strong linear relationship is evident, demonstrating that the variance is not noise. The correlation between mean and variance shows that there is a real variability of strain in different regions of lumen wall that is 35% and 51% of the mean strain based on the linear fit at 1.5T and 3.0T, respectively. Qualitatively there is a noticeable increase in strain, when compared to the lumen mean strain, in the region of the lumen wall close to the jugular vein and other major blood vessels. These results were also reproducible by all operators. The percentage difference between operators was  $8\% \pm 5\%$  across all strain measurements at 1.5T and  $5\% \pm 4\%$  at 3.0T.



## Conclusion:

The strength of DENSE strain measurements is that it can provide regional measurements of strain as opposed to global measures from ultrasound and CINE MRI. Our results show that there is significant regional variation in strain in the lumen wall of the carotid artery. Further investigation should focus on any common baseline pattern of the strain distribution in healthy humans, and possible correlation between the baseline strain pattern and likely locations of plaque formation.

## References:

1. Mitchell, G.F., et al., *Circulation*, 2005. 112(2): p. 194-9. 2. Dijk, J.M., et al., *Stroke*, 2004. 35(10): p. 2258-62. 3. Wen H, et al. *Magn Reson Mater Phy* 2005;18(S1):S60-S61. 4. Lin et al. *J Cardiovasc Magn Reson* 2006. 8(1):48

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