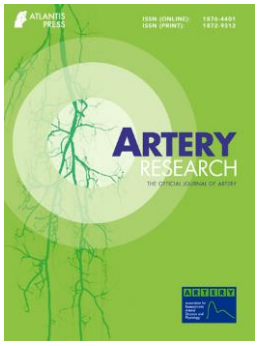

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7.6: TISSUE FACTOR PATHWAY INHIBITOR: A NEW LINK BETWEEN ARTERIAL STIFFNESS, PULSE PRESSURE AND COAGULATION IN POSTMENOPAUSAL WOMEN

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demonstrate the feasibility of this ultrasound-based technique to characterize rupture prone plaques. A parametric analysis was performed by varying tissue and acoustic beam characteristics for different vasculature and plaque component geometries, based on published images of *in vivo* carotid artery plaques obtained using MRI by Li et al.[2]
ARF induced displacements within the modelled artery were higher in the lipid (0 to 2 μm) compared to the surrounding vessel and fibrous cap (0 to 1 μm) and are consistent with *in vivo* measurements. The maximum equivalent stress increased with an increase in the fibrous cap stiffness and for the geometry shown in Figure 1 was <406 Pa for all parametric cases. A more detailed analysis of the FEM model parameters and results for other geometries will be presented along with *in vivo* ARFI images.

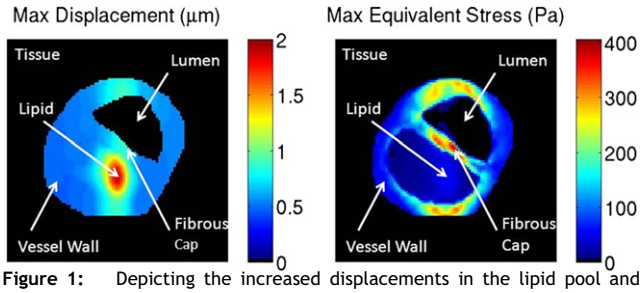

Figure 1: Depicting the increased displacements in the lipid pool and maximum equivalent stress concentration in the fibrous cap.
[1]Dahl et al. Ultrasound Med Biol. 35(5). 2009, 707-716.
[2]Li et al. J. Biomech. 39(14), 2006, 2611-2622.

7.6
TISSUE FACTOR PATHWAY INHIBITOR: A NEW LINK BETWEEN ARTERIAL STIFFNESS, PULSE PRESSURE AND COAGULATION IN POSTMENOPAUSAL WOMEN

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Objective: To investigate in women over 60 whether aortic stiffness and/or pulse pressure (PP) are associated with selected procoagulant and/or anticoagulant factors, and to examine whether pulsatile stretch influences these factors in human vascular smooth muscle cells (VSMCs) *in vitro*.
Methods and results: Aortic pulse wave velocity (PWV) and carotid PP were studied in 123 apparently healthy postmenopausal women. PWV, PP, von Willebrand factor (vWF) and free tissue factor pathway inhibitor (f-TFPI), but not mean arterial pressure, increased with age. f-TFPI and PWV were positively correlated, even after adjustment for age and PP and other confounding parameters. *In vitro*, 5 or 10% pulsatile stretch (at 1 Hz) enhanced TFPI synthesis and secretion by VSMCs in a time-independent manner (1 h to 48 h) without changes in protein level of smooth muscle myosin heavy chain. Application of 5% static stretch had no effect.
Conclusions: In postmenopausal women, f-TFPI increases as vascular wall function deteriorates and PP increases. These findings are supported by the increase in TFPI synthesized by VSMCs in response to cyclic stress *in vitro*. They suggest that VSMCs require pulsatility to interfere with the coagulation process and highlight the relevance of plasma f-TFPI levels to cardiovascular diseases.