

NUMERICAL MODELING OF STRESS CONCENTRATIONS IN MICRO-HETEROGENEOUS BIO-TISSUE

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ABSTRACT

Although the mechanisms that cause atherosclerotic plaque rupture are not fully understood, there has been much focus in the literature on the role of high stress concentration in the failure of plaque lesions. In general, stress concentrations are dependent on many factors, including the vessel geometry and material composition of both the lesion itself and the surrounding tissue. Tang *et al.* [1], among others, have performed finite element simulations to assess plaque cap vulnerability relative to a "local maximal stress hypothesis" assuming homogeneous material composition of the lesion.

However, due to the presence of biological fluids and various chemical species within the bio-tissue, hard deposits ("inclusions"), can be formed. Such inclusions are frequently calcifications and can form in the regions around atherosclerotic plaque lesions. Calcification has been studied in plaque tissue by, for example, Huang *et al.* [2] and Tang *et al.* [3]. For reviews, see Humphrey [4] or [5]. Recently, particulate micro-calcifications have been observed by Vengrenyuk *et al.* [6] in plaque tissue. These authors have hypothesized that if an imbalance in physiochemical conditions occurs, micro-calcifications can develop, which serve as (a) indicators that there is impending danger due to the mismatch in material properties at the micro-calcification interface and (b) catalysts, due to alteration of the stress fields, that cause the body to react with further chemical events (inflammation) and potential cap rupture, due to thinning.

Generally, regardless of the specific system, heterogeneities can dramatically alter the stress fields within bio-tissue. In an effort to account for such heterogeneities, this work will provide analytical and numerical estimates of the stress concentrations within bio-tissue containing micro-scale particulate heterogeneities which, in this specific case, are micro-calcifications. Stress concentration functions are developed in order to determine the load sharing of each phase as a function of the volume fraction and of the mechanical properties of the constituents.

In this work, the finite element method is employed to evaluate the mechanisms that can lead to stress concentrations in stenotic arteries. This is done using a multi-scale approach. First, the fluid-structure interaction (FSI) problem of blood flow in a stenotic artery is solved. Next the solution at the critical point in the plaque cap is used to drive a micro-scale problem involving heterogeneities embedded in a representative volume element (RVE) of diseased arterial tissue. A sensitivity study is conducted to

quantify the influence of perturbations on important parameters of the model, such as material properties and geometry of the lesion. The practical goal of the study is to quantify the coupling between various observable parameters in terms of their effects on the development of stress concentration at the point of failure.

REFERENCES

- [1] D. Tang, C. Yang, J. Zheng, P. K. Woodard, J. E. Saffitz, J. D. Petrucci, G. A. Sicard, and C. Yuan, Local maximal stress hypothesis and computational plaque vulnerability index for atherosclerotic plaque assessment, *Annals of Biomed. Eng.*, vol 33, pages 1789-1801, 2005.
- [2] H. Huang, R. Virmani, H. Younis, A. P. Burke, R. D. Kamm, and R. T. Lee, The impact of calcification on the biomechanical stability of atherosclerotic plaques, *Circulation*, vol 103, pages 1051-1056, 2001.
- [3] D. Tang, C. Yang, J. Zheng, P. K. Woodard, J. E. Saffitz, G. A. Sicard, T. K. Pilgram, and C. Yuan, Quantifying effects of plaque structure and material properties on stress distributions in human atherosclerotic plaques using 3D FSI models, *J. Biomech. Eng.*, vol 127, pages 1185-1194, 2005.
- [4] J. D. Humphrey, Continuum biomechanics of soft biological tissues, *Proceedings of the Royal Society*, vol 459, pages 3-46, 2002.
- [5] J. D. Humphrey, Cardiovascular Solid Mechanics. Cells, Tissues, and Organs, Springer-Verlag, New York, 2002.
- [6] Y. Vengrenyuk, S. Carlier, S. Xanthos, L. Cardoso, P. Ganatos, R. Virmani, S. Einav, L. Gilchrist, and S. Weinbaum, A hypothesis for vulnerable plaque rupture due to stress-induced debonding around cellular microcalcifications in thin fibrous caps, *Proceedings of the National Academy of Sciences of the United States*, vol 103, pages 14678-14683, 2006.