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EFFECTS OF TISSUE COMPONENTS ON THE VULNERABILITY OF ATHEROSCLEROTIC PLAQUES: A COMPUTATIONAL STUDY

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ABSTRACT

Atherosclerosis is the main determinant of cardiovascular diseases, the leading cause of cardiovascular morbidity and mortality around the globe. Although lumen narrowing and exaggerated or anomalous vasoconstriction contribute to some of the clinical manifestations of arterial diseases, it is the superposition of an arterial thrombus and the underlying ruptured or eroded plaque that is responsible for the vast majority of acute ischemic syndromes. The ability to identify rupture-prone, high-risk plaques and to intervene successfully before acute plaque rupture occurs, has been an elusive goal of clinicians over the last decades.

The predominant features of plaque vulnerability are increased numbers of macrophages, increased expression of tissue factor, reduced number of smooth muscle cells, a lipid core that occupies a high proportion of the overall plaque volume, and a thin plaque cap [1]. The rapid development in the area of arterial wall imaging made it possible to characterize the plaque volume and the plaque cap. Morphological criteria, however, cannot fully explain the mechanisms of plaque rupture, and cannot reliably assess vulnerability. Stability analyses require additional information on the biomechanical stresses occurring in the plaque's structural components. Recent research attempts have used numerical tools (such as the finite element method) in combination with imaging techniques (IVUS, MRI) in order to provide deeper insights and predictors on plaque fracture. A common denominator of most studies has been the effect of tissue components' structure and material properties on the overall mechanical environment of the plaque and especially of the deciding fibrous cap. However, the majority of efforts are based on simplified (two-dimensional) modeling approaches or on the assumption of plane stress. The layered structure of diseased arteries has been more or less ignored. Additionally, the arterial tissues have been assumed to be isotropic, an argument that does not reflect the experimentally observed anisotropic behavior of diseased vascular tissues. Hence, there is a need to model the complex layer-structured arterial geometry and the related mechanics in a more realistic way.

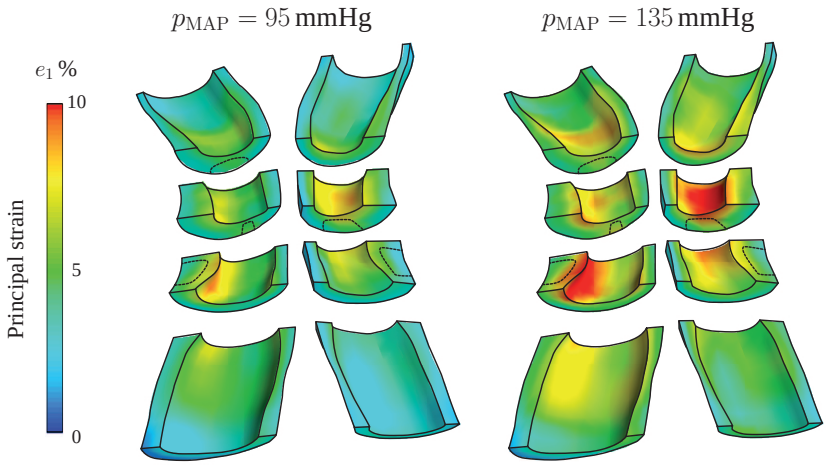

Figure 1: Comparison of the Euler-Almansi principal strain fields at the deformed configuration of the internal carotid artery: (a) at $p_{\mathrm{MAP}} = 95\,\mathrm{mmHg}$ and (b) at $p_{\mathrm{MAP}} = 135\,\mathrm{mmHg}$ mean arterial pressure.

The main objective of this work is the presentation of a computational methodology to analyze the mechanical environment of atherosclerotic plaques and to identify vulnerable plaques. In particular, a patient-specific morphology of a human carotid bifurcation based on high-resolution MRI is considered. The arterial wall is modeled as a non-homogeneous solid that consists of three different tissue components (non-diseased wall, lipid pool, calcification). The adopted constitutive model [2] takes the nonlinear and anisotropic behavior of the arterial components into account. The material parameters are obtained through mechanical tests on the isolated tissue components. The nonlinear finite element method, in particular FEAP [3], is used for the analysis of plaque mechanics.

Additionally, the proposed computational model attempts to understand and quantify the effect of three factors on the stress environment of human atherosclerotic plaques, and consequently on the plaque vulnerability, i.e.: (i) the pressure load, (ii) the plaque structure, and (iii) the properties of the tissue components. To accomplish this goal, a parametric analysis is performed. More specifically, variations of the blood pressure (see, e.g., Figure 1) and of the lipid pool's stiffness and size are studied. These cases are of particular clinical interest because the varied parameters can be controlled by drugs such as statins [4].

The proposed morpho-mechanical approach provides important information for the understanding of plaque stability. It offers a meaningful approach for the assessment of the vulnerability of atherosclerotic plaques when combined with powerful imaging modalities which may significantly contribute to the current screening and diagnosis means.

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