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THE COUPLED PASSIVE-ACTIVE MECHANICAL RESPONSE OF THE HUMAN ARTERY WALL – A HIGH ORDER FINITE ELEMENT STUDY

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The human artery is inherently anisotropic, slightly compressible and comprised of several thin layers that undergo large deformations under physiological conditions. It has long been suggested that a link exists between stress and strain gradients in the artery wall and the development of arterial disease. Due to the complex layered structure and anisotropy of the tissue analytical solutions for the mechanical fields are not possible and one must utilize computational methods to study this hypothesis. The mechanical response of the artery is governed by the coupling between its passive and active constituents. From an engineering view point the passive constituents of each layer can be represented by a slightly compressible hyper-elastic matrix (elastin) embedded with two families of stiff fibers (collagen) [1]. The active constituents in each layer are the Smooth Muscle Cells (SMC) which are commonly assumed to be oriented in the circumferential direction. The level of SMC contraction is a function of both chemical stimuli and SMC stretch level. Because the SMC stretch level depends also on the passive components, the passive and active response are closely coupled. A strain energy density function (SEDF) which takes into account the contribution of both the passive and active components was proposed in [2].

This study utilizes the p-version of the FEM and the SEDF proposed in [2] to investigate the stress and strain fields which develop in a healthy artery as a result of this passive-active coupling. The goals of this study were to examine how SMC initial assumed orientation, activation level and tissue compressibility affect the coupled mechanical response in a bi-layered (Media, Adventitia) slightly compressible artery. The effect of homogenous and inhomogeneous initial SMC orientation across the artery thickness was considered. The results of this study demonstrate that, due to the passive-active coupling, the stress gradients

across the artery thickness increase with internal pressure for a constant activation level. This increase is the result of inhomogeneous contractile forces generated by the SMCs across the vessel wall. It is also shown that considering initial SMC orientations which deviate from the circumferential direction yields higher stress gradients across the artery thickness in both the circumferential and longitudinal directions.

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