A 3D HISTOMECHANICAL GROWTH AND REMODELING FRAMEWORK FOR ARTERIES WITH APPLICATION TO ABDOMINAL AORTIC ANEURYSMS

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Introduction. Mechanical stress and deformation influence vascular cell physiology and play a key role in explaining the origin and progression of vascular pathologies like Abdominal Aortic Aneurysms (AAA). AAAs are progressive dilatations of the infrarenal aorta and if untreated they enlarge and eventually rupture. The recommended AAA management is ultrasound surveillance until either the aortic diameter reaches 5.5cm or increases by more than 1.0cm per year, then open or endovascular (EVAR) repair is offered. Despite growing evidence that a biomechanical assessment could potentially improve this clinical practice [2], robust and physiologically relevant constitutive models are central to reliable simulation results. Specifically, histomechanical constitutive models [3] overcome several draw-backs known from purely phenomenological approaches.

Method. A previously reported active smooth muscle cell (SMC) model [6] and a refined description of collagen turnover were incorporated in our previously developed histomechanical constitutive model for vascular tissue [4]. Specifically, collagen turnover in the medial and adventitial layers are thought to be realized by SMC and fibroblasts (FB), and a collagen fiber having a referential orientation of **M** is maintained according to the stimuli

$$\xi_{\rm med}(\mathbf{M}) = H_{\rm SMC} |\mathbf{M} \cdot \mathbf{E}_{\theta}| \frac{\overline{\rho}_{\rm med}}{\rho} \quad ; \quad \xi_{\rm adv}(\mathbf{M}) = \frac{\lambda(\mathbf{M})}{\lambda_{\rm ph}} \frac{\overline{\rho}_{\rm adv}}{\rho} \,. \tag{1}$$

Here, \mathbf{E}_{θ} and H_{SMC} denote the circumferential direction and the SMC tonus, respectively. In addition, the formulations assume that the collagen density ρ remodels towards target densities $\overline{\rho}_{\text{med}}$ and $\overline{\rho}_{\text{adv}}$, and λ_{ph} is the collagen fiber's physiological stretch. Collagen mass synthesis and degradation is given by

$$\dot{\rho}^- = \eta \rho \quad ; \quad \dot{\rho}^+(\mathbf{M}) = \eta \rho \xi_i(\mathbf{M}) \quad , \quad i = \text{med}, \text{adv} \quad ,$$
 (2)

where η introduces the time scale of the collagen turnover process. Finally, it is assumed that the produced collagen integrates into the existing structure using the weighted decomposition theorem [4].

Results. The G&R model has been implemented at the Gauss point-level of a Q1P0 mixed finite element formulation (FEAP, University of California at Berkeley) and applied to a patient-specific AAA. Abdominal aortic geometry was reconstructed from Computer Tomography images (A4clinics Research Edition, VASCOPS GmbH), which have been recorded at Karolinska University Hospital, Stockholm, Sweden. The computational time step was related to the maximum increment of synthesized collagen mass, and Fig. 1 illustrate the development of the AAA shape over time predicted by the proposed G&R model.



Figure 1: Development of AAA shape over time as predicted by the proposed G&R model.

Conclusion. A novel G&R model was introduced and successfully applied to predict the evolution of a patient-specific AAA. The example computation showed characteristic AAA features known from clinical follow-up data [5]. Despite these qualitative agreements further model refinement and calibration is required to enhance the model's predictability, where also the formation and aging of the intra-luminal thrombus should be addressed [1].

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