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Aneurysm enlargement utilizing a fiber-based growth model

*** Fred Nugen[1], Yuri Bazilevs[2], and Thomas J.R. Hughes[3]**

Institute for Computational Engineering and Sciences
The University of Texas at Austin
1 University Station, C0200
Austin, TX 78712-0227

[1]nooj@ices.utexas.edu
http://www.ices.utexas.edu/~nooj

[2]http://www.ices.utexas.edu/~bazily
bazily@ices.utexas.edu

[3]hughes@ices.utexas.edu
http://www.ices.utexas.edu/~hughes

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ABSTRACT

Aneurysms are common, life-threatening, and poorly understood. It is nearly impossible to observe the disease process which leads to aneurysms, especially in early stages. As a result, little is known about causes of the disease, and there is no strongly-correlated marker of patient prognosis. Several things are known. An aneurysm's mechanical environment has a strong influence over its behavior; weakening of the arterial wall is the main proponent of aneurysm rupture; and remodeling and turnover are chief mechanisms of weakening. We utilize these principles to build computational and analytic tools that can reconstruct the disease process and serve as a predictor of patient prognosis.

Geometry and the mechanical flow complexities caused by geometry carry an attributable risk of growth and rupture. Growth and geometry are deeply intertwined through signaling and hemodynamic stimuli: the uneven stimuli cause uneven growth, which changes the geometry, which changes the hemodynamics, and the process loops.

We have developed a theoretical formulation, a modeling pipeline, and a set of simulation tools for the prediction of enlargement of aneurysms. The modeling paradigm is multiscale in time, with one timescale on the order of seconds (the cardiac cycle) where we compute hemodynamic stimuli, and the other on the order of weeks to months (the aneurysm's enlargement) where we compute growth and remodeling. Computation will proceed using a staggered approach common in fracture mechanics, switching between the two timescales.

The simulation will build on existing technologies for modeling tissue as a solid anisotropic elastic material. The short timescale utilizes the isogeometric fluid-structure interaction simulation tools presented in [1]. The constitutive relations for this timescale describe a hyperelastic material with exponential material stiffening, and two families of supporting fibers also with exponential stiffening. The fibers are

assumed to be perfectly oriented, wrapping around the artery wall helically and represent collagen fibers which make up the main structural component of aneurysmal tissue.

The long timescale utilizes a constitutive relation describing mass growth (as a source term) and remodeling of fibers (as a history-dependent strain energy function) based on the stresses computed from the short timescale. The mechanics of remodeling and turnover used are described in [2].

The proposed implementation is verified on geometries representing healthy arteries under normal conditions. One of the requirements is a stability check: ensuring the simulation tools predict that healthy arteries remain healthy.

By using patient-specific geometries from longitudinal image data, we can quantify attributable risk of aneurysm growth and remodeling due to mechanical factors. Validation comes as follows: two image datasets are obtained from the same subject, spaced at least one year apart. A simulation is run beginning from a geometry representing the early dataset, and is used to predict the geometry of the later dataset.

A main focus of the work is to develop a state-of-the-art aneurysm modeling system from patient-specific image data to predicted outcome. This application is general: it can be extended to the study of healthy arteries anywhere in the body, and to arterial systems. The application serves as a testbed for experimental boundary conditions (on both the solid and the fluid) and ongoing research in the structure and structural changes of arteries.

The modeling system involved in this work is a necessary step to provide better conclusions as to the behavior and mechanical nature of diseased arteriesi, as well as to improve the overall predictive capability of simulation tools. Physicians who use these tools will better understand aneurysm dynamics, improve their morbidity and mortality rates, and save lives.

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