Center for Fluid Mechanics, Division of Applied Mathematics
Fluids, Thermal and Chemical Processes Group, School of Engineering
Joint Seminar Series

TUESDAY – DECEMBER 14, 2010, BARUS & HOLLEY, ROOM 190, 3:00pm

Dr. Anne Robertson
University of Pittsburgh
Department of Mechanical Engineering and Materials Science
Pittsburgh, PA

On the Biomechanics of Damage in Cerebral Vessels

The mechanical integrity of the arterial wall is vital to the health of the individual. This integrity is in turn dependent on the state of the central load bearing components of the wall: collagen, elastin and smooth muscle. Elastin, found as fibers and fenestrated sheets of elastin, is viewed as responsible for the highly elastic nature of the wall at low loads. The nonrandom orientation of the collagen fibers is largely responsible for the anisotropic behavior of the wall. Collagen in the outermost laster of the wall is believed to serve largely as a protective sheath, preventing over distension of the artery.

It is now understood that the healthy arterial wall is an active structure- undergoing growth and remodeling in response to hemodynamic derived chemical and mechanical stimuli. Degradation of arterial elastin has been intimately tied to a number of pathological conditions including cerebral aneurysms, dissection aneurysms, arteriosclerosis, and complications from balloon angioplasty. Further, arterial stiffening with age has been attributed to gradual elastin fatigue damage under hemodynamic loading. The objective of this work is to develop a microstructurally based constitutive model for cerebral arteries which can ultimately be used as a tool to understanding these pathologies and as a tool for improving treatment strategies.

In this talk, we will discuss theoretical models we have developed to model the mechanical response of the arterial wall including progressive damage [1]. The contributions of elastin and collagen are modeled as distinct entities (mechanisms) with different mechanical responses, unloaded configurations and damage behavior [2]. Two types of mechanically induced damage are considered- (i) “low level fatigue” or “acute damage” will be understood as damage arising from a low number of cycles at large loads and (ii) “high cycle fatigue”– arising from the gradual degradation of the tissue under a large number of loading cycles to lower maximum loads. In addition, we will discuss enzymatic damage to the elastin as a result of the body’s response to hemodynamic loads outside a preferred range.

Multi-photon microscopy (MPM) can be utilized to visualize both elastin and collagen in arteries by exploiting auto-fluorescence and second harmonic generation, respectively. The traditional approach to visualizing these proteins involves formaldehyde fixation followed by histological staining techniques, which is destructive to the tissue, from a mechanics point of view. MPM techniques are non-destructive, so images may be obtained from a single specimen at various time points and levels of strain. At the end of this talk, we discuss recent results from our group using a new device and protocol combining uniaxial mechanical testing and MPM imaging to analyze acute rupture of elastin in arteries. Images of elastin and collagen provided insights into the damage mechanisms by allowing visualization of the initialization and progression of elastin rupture. These results are being used to further develop the theoretical damage models for cerebral vessels.

1. D. Li and A. M. Robertson, *A Structural Multi-Mechanism Damage Model for Cerebral Arterial Tissue*, J Biomech Eng-T ASME, vol. 131(10), DOI: 10.1115/1.3202559, 2009.
2. D. Li and A.M. Robertson, *A Structural Multi-Mechanism Constitutive Equation for Cerebral Arterial Tissue*, Int J Solids Struct, 46(14-15), pp. 2920-2928. DOI:10.1016/j.ijsolstr.2009.03.017, 2009.