PRESENTATION ABSTRACT

It is well recognized that tissue mechanics plays an important role in regulating cellular function and dysfunction. A remaining challenge is to create new avenues that can use this understanding to impact the clinic. This talk will present two examples towards this end, a computational study of altered lens capsule mechanics after cataract surgery and an experimental study of the role of mechanics in atherosclerosis development and regression. Cataract surgery is the most commonly performed surgical procedure in the US. During the procedure, a permanent hole is placed in the anterior portion of the lens capsule and the native lens fibers are replaced with a prosthetic intraocular lens. This portion of the talk will present our efforts to characterize the dramatically altered mechanical environment of the lens capsule after cataract surgery and progress in the development of a finite element growth model that can predict the associated cell-mediated remodeling over time. Atherosclerosis is the leading cause of death in the Western world. An interesting feature is that, despite the many systemic risk factors, plaques do not form randomly in the vasculature, but at predilection sites associated with disturbed blood flow. This portion of the talk will present causal evidence for the relationship between disturbed flow and atherogenesis, followed by consideration of whether a therapeutic mechanical stimulus could promote normal endothelial cell behaviors and plaque regression.

ABOUT DR. PEDRIGI

Ryan Pedrigi received his B.S. (2003) in Mechanical Engineering from Kansas State University and Ph.D. (2008) in Biomedical Engineering from Texas A&M University. His Ph.D. thesis focused on characterizing the mechanics of the lens capsule from native to after cataract surgery in the research group of Dr. Jay Humphrey. He worked as a postdoctoral researcher in two groups at Imperial College London with an emphasis on endothelial cell mechanobiology. His first postdoctoral position (2009-2012) studied altered stiffness of Schlemm’s canal endothelial cells and its role in the development of ocular hypertension in glaucoma. His second postdoctoral position (2012-2016) examined the mechanobiology of advanced atherosclerotic plaques using isolated cell and animal (pig and mouse) models. He joined the Department of Mechanical and Materials Engineering at the University of Nebraska-Lincoln as an assistant professor in 2017. His research interests are in mechanobiology, biomechanics, regenerative medicine, ophthalmology, and cardiovascular medicine with an emphasis on endothelial cell dysfunction in atherosclerosis.