11th World Congress on Computational Mechanics (WCCM XI)
5th European Conference on Computational Mechanics (ECCM V)
6th European Conference on Computational Fluid Dynamics (ECFD VI)
July 20–25, 2014, Barcelona, Spain

MULTI-PHYSICS MULTI-SCALE MODELS FOR RETINAL BLOOD FLOW AND ITS RELATION TO GLAUCOMA

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Key words: *Coupled Problems, Blood Flow, Fluid-Structure Interaction, Retina.*

Glaucoma is an optic neuropathy characterized by progressive retinal ganglion cell death and structural changes to the retina and optic nerve head ultimately leading to irreversible visual field loss. To date, elevated intraocular pressure (IOP) is the only treatable glaucoma risk factor, although there is overwhelming evidence that other factors might be involved in the disease. Many individuals with elevated IOP never develop glaucoma, and many patients develop glaucoma despite reduced IOP [1].

The definition of glaucoma has now expanded to include vascular, genetic, anatomical, and other systemic factors. In particular, the scientific community continues to accrue evidence suggesting that alterations in ocular hemodynamics play a significant role in the pathophysiology of glaucoma. Significant correlations have been found between impaired vascular function and both glaucoma incidence and progression; however, the mechanisms are still unclear. Clinical and animal studies are the most popular approaches currently utilized by the scientific and medical communities to shed light on the hemodynamic contribution to the pathophysiology of glaucoma. Many factors influence ocular hemodynamics, including IOP, arterial blood pressure and blood flow autoregulation, and it is extremely challenging to single out their individual contributions during clinical and animal studies [2].

In the recent years, we have been developing mathematical models to be used in conjunction with clinical studies to unravel the mechanisms behind the observed correlations in the data [3]. In this talk, we will present mathematical models that describe (i) the blood flow in the central retinal artery and vein, accounting for the IOP-induced deformation of the vessel walls; (ii) the blood flow in the retinal microvasculature, accounting for the interaction between the IOP mechanics on the blood vessels and the fluid-dynamics of the blood inside the vessels; (iii) the autoregulation of blood flow in the retina, accounting for the myogenic, shear- stress, CO_2 and O_2 responses; (iv) O_2 transport, diffusion and

consumption in the retinal vasculature and tissue.

Results will show how the synergy between mathematical modeling and clinical data has allowed us to unravel the relative importance of various mechanisms contributing to the autoregulation of retinal blood flow [4], the relative contribution of IOP, arterial blood pressure and blood flow autoregulation on retinal perfusion, and the sensitivity of retinal oxygenation to changes in blood pressure and hematocrit.

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