

BIOMEDICAL MODELING AND COMPUTATION FOR COMPLEX INTERFACE PROBLEMS

Kun Gou*, **Simona Hodis***

* Department of Mathematics, Texas A&M University-Kingsville, Texas, 78363, US.
kun.gou@tamuk.edu, simona.hodis@tamuk.edu

MINI-SYMPOSIUM PROPOSAL

Keywords: *Complex interface, Soft tissue, Stiffness, Flow pattern, Continuum mechanics*

1 INTRODUCTION

Complex-interface problems are commonly encountered in biomedical studies. Many human organs bear several layers for stable structural functions providing challenging and interesting complex interface problems. For example, the trachea has an inner soft submucous layer and a harder outer cartilaginous layer. The interface between the two layers are irregular causing the interface to be a local attractor of stress concentration under tissue swelling [1]. Another example is the blood vessel. There are two interfaces in it including the interface between the inner intima layer and the middle media layer, and the one between the media and the outer adventitia layers. Each layer has different material properties. The two interfaces provide a sharp change for many vital physical properties [6]. Other complex interfaces come from fluid-solid interaction or the difference between an organ and its environment [3, 7]. Inside the blood vessel, the flowing blood and the vessel wall form a significant boundary value problem. Studying this interface is key for understanding how plaques are formed along the wall from deposits of the blood. Based on the importance of the complex-interface problems, this minisymposium is held to present research work of modeling various diseases and fulfilling its computation involving complex interfaces. The research work pioneered novel methodologies in the related areas, and established great foundations for more profound future studies.

2 RESEARCH AREAS

Damage of the brain may be caused by mechanical loads and chemical imbalances under various parts inside the brain with complex interfaces. A unified variational framework was proposed for the modeling of neuronal electromechanics. A constrained Lagrangian formulation was developed that takes into account Newton's law of motion of a linear viscoelastic KelvinVoigt solid-state neuron and the classic HodgkinHuxley equations of the electronic neuron [3]. The system of differential equations describing neuronal electromechanics was obtained by applying Hamilton's principle.

When a human organ involves composite or multiple elastic materials, elasticity interface problems come into play. This work introduces the matched interface and boundary (MIB) method for solving 3D elasticity interface problems [2]. The proposed MIB elasticity interface scheme utilizes fictitious values on irregular grid points near the material interface to replace function values in the discretization so that the elasticity equation can be discretized using the standard finite difference schemes as if there were no material interface.

Tracheal angioedema refers to the swelling of the airway system. A rapid occurrence of this disease may rapidly block the airflow and cause an emergency. Hyperelasticity is employed in the modeling where the more conventional anisotropic and incompressible soft tissue framework is generalized to incorporate the swelling effect in the form of a specified volume constraint [1]. Finite element stress analysis then proceeds with the aid of compressible versions of the soft tissue models for the three predominant tissue types in the trachea.

Blebs are pressure protrusions that play an important role in cell migration. A bleb is initiated when the cytoskeleton detaches from the cell membrane, resulting in the pressure-driven flow of cytosol towards the area of detachment and local expansion of the cell membrane. A dynamical computational model of the cell was developed that includes mechanics of and interactions between the cytoplasm, the acting cortex, the cell membrane and the cytoskeleton [7]. The model results quantify the relationship between cytoplasm rheology, pressure, and bleb expansion dynamics, and provide a more detailed picture of intracellular pressure dynamics.

The blood vessel plaque growth, identification and rupture analysis are important for studying the aneurysms or atherosclerosis along the vessel wall. The formation of plaque is a very complicated process under the effect of various complex interfaces. We are trying to quantify plaque growth in order to aid in the detection and diagnosis of plaque [6]. Our research aims to answer some important basic questions concerning plaque growth such as how shear stresses affect the leukocyte adhesion cascade (LAC) and, subsequently, how the LAC affects plaque formation. Ascending thoracic aortic aneurysms (ATAAs) are focal, asymmetric dilatations of the aortic wall which are prone to rupture. To identify potential rupture locations in advance, it is necessary to consider the inhomogeneity of the ATAA at the millimeter scale [4]. Towards this end, a combined experimental and computational approach was developed using bulge inflation tests and digital image correlation to characterize the pointwise stress, strain, and hyperelastic properties of the ATAA. To prevent a spontaneous rupture, an aneurysm is usually recommended for treatment based on its size and location. The mechanism of rupture is not completely understood, but recent studies showed that ruptured cerebral aneurysms were characterized by complex flow patterns to determine a threshold value of the flow complexity parameter [5].

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