On the Mathematical Modeling of Atherosclerosis Disease

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This presentation is divided into two parts:

- The inflammatory process of atherosclerosis leads to the formation of an athero- matous plaque in the intima of the blood vessel. The plaque rupture may result from the interaction between the blood and the plaque. In each cardiac cycle, blood interacts with the vessel, considered as a compliant nonlinear hyperelastic. A three dimensional idealized fluid-structure interaction model is constructed to perform the blood-plaque and blood-vessel wall interaction studies. An absorbing boundary condition is imposed directly on the outflow in order to cope with the spurious re- flexions due to the truncation of the computational domain. The difference between the Newtonian and non-Newtonian effects is highlighted. It is shown that the von Mises and wall shear stresses are significantly affected according to the rigidity of the wall. The numerical results have shown that the risk of plaque rupture is higher in the case of a moving wall, while in the case of a fixed wall the risk of progression of the atheromatous plaque is higher.
- Monocytes play a significant role in the atherosclerosis development. During the inflammation process, monocytes that circulate in the blood stream are activated. Upon activation, they adhere to the endothelium and extravasate through the latter to migrate into the intima. In this work we are concerned with the transmigra- tion stage. In our study, the constitutive equations for Oldroyd-B fluids are used to capture the viscoelastic behavior of monocytes. We first establish and analyze a simplified mathematical model describing the coupled deformation-flow of an individual monocyte in a microchannel. Then we describe the numerical implementation of the mathematical model using the level set method and show the numerical results. Further extensions of this model are also discussed.