427a Multiphase Hemodynamic Analysis of Cardiovascular Systems

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The initiation and progression of focal atherosclerotic lesions has been associated with regions of distributed blood flow, a concentrated suspension of formed elements in plasma, yet the correlations of local hemodynamics to vessel wall are poorly understood. Recent studies suggested that one of the earliest events is the adhesion of circulating monocytes (leukocytes) to the intact surface of endothelial cells. Data on the roles of physiologically blood particulates in hemodynamics are critically needed to better understand cardiovascular diseases. Little work has been done on the transport of blood particulates, as never predicted by single-phase computational fluid dynamics (CFD) models. In our study, the blood flow patterns including plasma, red blood cells (RBC) and leukocytes were numerically simulated using the multiphase hemodynamic CFD models, FLUENT code. First, the modeling including plasma and leukocytes was used to predict U-937 cell adhesion to the inert surface of a three-dimensional perfusion model with pulsatile flow waveform. The computational results showed higher cell deposition in the stenosis, in a good agreement with in-vitro data of cell trajectories tracked by high-speed video cameras. The model also predicted the circulation flow pattern and the cell adhesion, agreed with the experimental data in the parallel-plate flow chamber. Second, the blood flow patterns and RBC buildup were numerically simulated using the multiphase non-Newtonian theory of dense suspension hemodynamics with 45% volume RBC in an idealized coronary artery and a realistic right coronary artery having various cross sections, where the leukocytes were neglected. The rheological properties of human blood were determined from a non-Newtonian shear-thinning model as a function of shear rate and hematocrit, based on experimental data. The location of RBC buildup on the inside curvature correlated with lower wall shear stress (WSS) relative to the outside curvature. The local hemodynamic factors, such as WSS, RBC buidup, viscosity, and velocity, varied with spatially nonuniform vessel structures and temporal cardiac cycles. Most of the total WSS with somewhat higher wall shear stress gradient near the walls was due to the RBC, not to the plasma. The oscillatory flow with flow reversal as well as the vessel geometry resulted in RBC buildup due to the prolonged particulate residence time, specifically, at the end of the diastole cycle. The increase in the initial plasma viscosity caused lower WSS. Third, with the addition of the leukocytes of 0.5% volume, the RBC-monocytes interactions were very important, resulting in leukocytes buildup in specific areas on the inside radius of curvature. Low WSS was found in higher leukocytes buildup region, in particular, at the area of maximum curvature, where the rather higher plasma concentration exists. These predictions have significant implications for understanding the local hemodynamic phenomena that may contribute to the earliest stage of atherosclerosis, as clinically observed on the inside curvatures of coronary arteries.