NUMERICAL SIMULATION OF BLOOD FLOW THROUGH MICROVASCULAR CAPILLARY NETWORKS

(ARTIGO ORIGINAL)

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A numerical method is implemented for computing blood flow through a branching microvascular capillary network. The simulations follow the motion of individual red blood cells as they enter the network from an arterial entrance point with a specified tube hematocrit, while simultaneously updating the nodal capillary pressures. Poiseuille's law is used to describe flow in the capillary segments with an effective viscosity that depends on the number of cells residing inside each segment. The relative apparent viscosity is available from previous computational studies of individual red blood cell motion. Simulations are performed for a tree-like capillary network consisting of bifurcating segments. The results reveal that the probability of directional cell motion at a bifurcation (phase separation) may have an important effect on the statistical measures of the cell residence time and scattering of the tube hematocrit across the network. Blood cells act as regulators of the flow rate through the network branches by increasing the effective viscosity when the flow rate is high and decreasing the effective viscosity when the flow rate is low. Comparison with simulations based on conventional models of blood flow regarded as a continuum indicates that the latter underestimates the variance of the hematocrit across the vascular tree. **[Bull Math Biol 2009; 71(6):1520-1541]**

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PECULIAR FLOW PATTERNS OF RBCS SUSPENDED IN VISCOUS FLUIDS AND PERFUSED THROUGH A NARROW TUBE (25 MICROM)

(ARTIGO ORIGINAL)

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Red blood cells (RBCs) generally deform to adopt a parachute-like, torpedo-like, or other configuration to align and flow through a capillary that is narrower than their major axis. As described herein, even in a narrow tube (25 microm) with diameter much larger than that of a capillary, flowing RBCs at 1 mm/s align axially and deform to a paraboloid shape in a viscous Newtonian fluid (505 kDa dextran medium) with viscosity of 23.4-57.1 mPa.s. A high-speed digital camera image showed that the silhouette of the tip of RBCs fits a parabola, unlike the shape of RBCs in capillaries, because of the longer distance of the RBC-free layer between the tube wall and the RBC surface (approximately 8.8 microm). However, when RBCs are suspended in a "non-Newtonian" viscous fluid (liposome-40 kDa dextran medium) with a shear-thinning profile, they migrate toward the tube wall to avoid the axial lining, as "near-wall-excess," which is usually observed for platelets. This migration results from the presence of flocculated liposomes at the tube center. In contrast, such near-wall excess was not observed when RBCs were suspended in a nearly Newtonian liposome-albumin medium. Such unusual flow patterns of RBCs would be explainable by the principle; a larger particle tends to flow near the centerline, and a small one tends to go to the wall to flow with least resistance. However, we visualized for the first time the complete axial aligning and near-wall excess of RBCs in the non-capillary size tube in some extreme conditions. **[Am J Physiol Heart Circ Physiol 2009; 297(2):H583-589]**

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VENOUS THROMBOEMBOLIC EVENTS IN HOSPITALIZED MEDICAL PATIENTS
(ARTIGO ORIGINAL)

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The number of acutely ill hospitalised medical patients at risk for acute venous thromboembolism (VTE) has not been well defined. Therefore, we used the 2003 United States Healthcare Cost and Utilization Project (HCUP) Nationwide Inpatient Sample database to estimate VTE events among hospitalised medical patients. We then modeled the potential reduction in VTE with universal utilisation of appropriate pharmacological thromboprophylaxis. We calculated that 8,077,919 acutely ill hospitalised medical patients were at risk for VTE. Heart failure, respiratory failure, pneumonia, and cancer were the most common medical diagnoses. We estimated that 196,134 VTE-related events occurred in 2003, afflicting two out of every 100 acutely ill hospitalised medical patients. These VTE-related events were comprised of 122,235 symptomatic deep venous thromboses, 32,654 symptomatic episodes of pulmonary embolism, and 41,245 deaths due to VTE. In our model, rates of pharmacological thromboprophylaxis prescription were low for various acute medical illnesses, ranging from 15.3% to 49.2%. However, with universal thromboprophylaxis, 114,174 VTE-related events would have been prevented. In conclusion, acutely ill medical patients represent a large population vulnerable to the development of VTE during hospitalisation. The number of VTE-related events would be halved with universal thromboprophylaxis. Further efforts focused on improving VTE prevention strategies in hospitalised medical patients are warranted. **[Thromb Haemost 2009; 102(3):505-510]**

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