

Numerical Prediction of Blood Damage in Biomedical Devices

M. Behbahani¹, M. Probst¹, A. Mai¹, C. Waluga¹, M. Behr¹, L. Tran², K. Vonderstein², and K. Mottaghy²

Chair for Computational Analysis of Technical Systems, RWTH Aachen University and Physiology, RWTH University Hospital, Aachen, Germany
mb@cats.rwth-aachen.de

Aims: Hemolysis and thrombotic complications due to activation of platelets and plasmatic clotting factors belong still to the most investigated topics in the field of study of biomedical devices. Mathematical modeling of thrombotic reactions is established and validated in test cases. Aim of this study is to experimentally evaluate and computationally simulate red blood cells and platelets under the influence of well-defined shear flow conditions. Platelet behaviour and reactions are experimentally reproduced, measured and used for validation of the numerical simulation.

Methods: Red blood cells are modelled in analogy to the deformation behaviour of water droplets under the influence of shear. Individual cells and their morphological change is tracked along streamlines. By integration of the deformation-based hemoglobin release, the overall hemoglobin release can be computed. A mathematical model of platelet activation, adhesion and aggregation has been implemented into a finite element CFD (Computational Fluid Dynamics) code. The approach is based on the advective and diffusive transport equations for resting platelets, activated platelets and platelet released agonists. Adhesion rates for the reactive surfaces depend on the hemocompatibility properties of the bio-surface and the local shear rate. Experiments with citrate-anticoagulated freshly-drawn whole blood are performed in a perfusion flow chamber as well as in a system of rotating cylinders for Couette and Taylor-vortex flow. Different biomaterials are used. The activation, drop of platelet concentration, adhesion and aggregation are quantified using scanning electron microscopy (SEM) and flow cytometry.

Results: Regions and flow conditions with a high potential for thrombus growth could be identified. The experiments clearly show the influence of the blood contacting material and flow properties. By means of SEM diverse platelet adhesion patterns are observed. Numerical analysis can explain the patterns and the degree of thrombus formation. Quantitative agreement with experiments could be achieved.

Conclusion: Hemolysis results are in close agreement with experimental data. The numerical thrombosis model shows good agreement with experimental data indicating a possible prediction of initiation of activation and detection of the local adhesion areas. The methods presented here can be used for modeling of blood damage in biomedical devices.