Complexity of Coagulation Phenomena in Intensive Blood Flows

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Abstract

Human blood can change its aggregate state under certain hydrodynamic conditions *in vitro* and *in vivo*. Intravascular thrombus formation in intensive blood flows is initiated by an interplay of platelet activation and biochemical chain reactions known as the intrinsic and extrinsic pathways of coagulation. As a result, a central catalytic substance - thrombin is generated. Under the action of thrombin, fibrinogen is converted to fibrin and subsequent polymerisation of fibrin takes place. Recently it was found that in intensive blood flows the threshold of shear-induced platelet activation depends on the multimeric of von Willebrand factor (VWF) molecules circulating in the blood. A complex platelet activation risk index (PARI) accounting not only the value of shear stress but the degree of VWF multimerisation was introduced. Numerical calculation of the index for personalized vascular geometries opens additional facilities for the estimation of the intravascular thrombosis risk. The results of numerical simulation are compared with some *ex vivo* experimental observations. Several movies reflecting the complexity of real coagulation and fibrinolytic phenomena in intensive blood flows will be presented.
References

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