

# Blood coagulation dynamics: mathematical modeling and stability results

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Blood coagulation is one of the important defense mechanisms preventing the loss of blood following a vascular injury. When the endothelium is damaged a complex physiological process called *hemostasis* is set into action: the blood vessel diameter is diminished, slowing bleeding, blood platelets get activated and a complex sequence of chemical reactions occurs, leading to the formation of a fibrin clot (thrombus) localized at the site of vessel wall damage.

The process of platelet activation and blood coagulation is quite complicated and not yet completely understood. Numerous experimental studies recognize that thrombus formation rarely occurs in regions of parallel flow, but primarily in regions of stagnation point flows, within blood vessel bifurcations, branching and curvatures. Moreover, internal cardiovascular devices such as prosthetic heart valves, ventricular assisting devices and stents, generally harbor high hemodynamic shear stresses that can cause platelet activation and result in coagulation. Thrombotic deposition encountered in these devices is a major cause of their failure. Valid models of the blood coagulation process are essential for better design of these devices and also to identify regions of the arterial tree susceptible to the formation of thrombotic plaques and possible rupture in stenosed arteries.

A number of researchers have attempted to tackle this challenging problem. In this talk we present a phenomenological model integrating biochemical, physiologic and rheological factors and show three-dimensional simulations obtained with a simplified version of this model. Regarding the biochemical process leading to clot formation, we deduce stability results for a dynamical system associated to enzymatic reactions that are assumed to follow the Michaelis-Menten kinetics.

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