

Abstract Submitted  
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**Toward a computational model of hemostasis** KARIN LEIDERMAN, NICHOLAS DANES, ROGIER SCHOEMAN, KEITH NEEVES, Colorado School of Mines — Hemostasis is the process by which a blood clot forms to prevent bleeding at a site of injury. The formation time, size and structure of a clot depends on the local hemodynamics and the nature of the injury. Our group has previously developed computational models to study intravascular clot formation, a process confined to the interior of a single vessel. Here we present the first stage of an experimentally-validated, computational model of extravascular clot formation (hemostasis) in which blood through a single vessel initially escapes through a hole in the vessel wall and out a separate injury channel. This stage of the model consists of a system of partial differential equations that describe platelet aggregation and hemodynamics, solved via the finite element method. We also present results from the analogous, in vitro, microfluidic model. In both models, formation of a blood clot occludes the injury channel and stops flow from escaping while blood in the main vessel retains its fluidity. We discuss the different biochemical and hemodynamic effects on clot formation using distinct geometries representing intra- and extravascular injuries.

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