Abstract Submitted
for the DFD13 Meeting of
The American Physical Society

Coupled Hemodynamic-Biochemical Modeling of Thrombus Formation in Infarcted Left Ventricles[1] JUNG HEE SEO, VIJAY VEDULA, RICHARD GEORGE, RAJAT MITTAL, Johns Hopkins University — Patients with heart failure (HF) and left ventricular (LV) systolic dysfunction have higher rates of thromboembolic events including embolic stroke and peripheral arterial thrombi. A common cause of arterial emboli in HF patients is myocardial infarction (MI) and subsequent left ventricular thrombus (LVT) formation. Stagnation of blood and endocardial injury are hypothesized to promote the development of LVT. The identification of high risk patients and the pharmacologic prevention of LVT formation are the keys to preventing embolic events. Stratification of patients at risk for LVT formation is currently limited, and primarily based on global assessment of ventricular function and image based assessment of ventricular wall motion. In this study, we explore a method to predict LVT risk using a multi-physics computational model. The blood flow in the left ventricle is simulated by solving the incompressible Navier-Stokes equation using an immersed boundary method and this is coupled to a convection-diffusion-reaction equation based model of platelet activation and coagulation. The results are then correlated with the other hemodynamic metrics such as wall shear stress and residence time to develop quantitative metrics for the LVT risk prediction.

[1]Supported by NSF CDI-Type II grant IOS-1124804, Computational resource by XSEDE NSF grant TG-CTS100002.

Rajat Mittal
Johns Hopkins University

Date submitted: 20 Jul 2013 Electronic form version 1.4