Dear Sir,

In their interesting paper, Valent et al. [1] suggest that mast cells might have functions beyond those involved in allergic and inflammatory reactions. Mast cells are the source of both tPA and heparin and are able to include fibrinolysis in vitro. This points to their novel role. The concept that mast cells are involved in endogenous thrombolysis appeals to me, and I would like to support it by the following data.

Atopic patients, who are prone to IgE-mediated mast cell activation, are apparently protected against sudden cardiac death following myocardial infarction [2]. They have a mild hemostatic imbalance [3], resembling that produced by aspirin [4]. It is reflected by a moderately prolonged bleeding time [5], depressed platelet aggregability [5, 6] and delayed generation of thrombin in clotting blood [7]. In these patients, the generation of a clot inside a critically obstructed coronary artery would be prolonged and delayed because of the late appearance of thrombin. Combined with endogenous thrombolysis linked to mast cell activation [1], this mechanism could protect against sudden cardiac death, the event consistently associated with occlusive coronary thrombi.

Of interest, increased circulating heparin levels have been reported in the blood of atopic patients [8, 9], and lack of coronary atherosclerosis was stated at autopsy in patients who died of acute asthmatic attack [10]. Finally, an increased IgE production in response to injury [11] might be associated with enhanced fibrinolysis via mast cell activation. Though some of these arguments need to be confirmed, they deserve, I think, consideration.

References