Factor XIII and Fibrinolytic Resistance
To the Editor

Reed and Houng demonstrated that factor XIII-mediated cross-linking plays a critical role in limiting fibrinolysis in experimentally formed pulmonary emboli. They suggested that factor XIII could facilitate the growth of new or forming thrombi. This concept is supported by my observations, which began 26 years ago and emphasize increased plasma factor XIII levels in hypertriglyceridemic patients who also display high plasma fibronectin concentrations and an obviously prolonged dilute blood clot lysis time. Because the in vitro inhibition of factor XIII by p-chloromercuribenzoate led to an acceleration of clot lysis, I presumed that the association of high plasma factor XIII levels with impaired fibrinolysis was not merely casual. My colleagues and I also showed that platelet factor XIII is unlikely to make any significant contribution to plasma factor XIII levels. However, the significant correlations between liver-secreted serum cholinesterase and plasma factor XIII and fibronectin, as well as data obtained in patients with severely impaired hepatic protein synthesis (decompensated liver cirrhosis) or compensatively enhanced secretion of plasma proteins (nephrotic syndrome) provide circumstantial evidence that the liver is the main contributor to plasma factor XIII and fibronectin. Presumably, overloading the liver with lipids in a patient with insulin resistance would stimulate the synthesis of factor XIII, fibronectin, and serum cholinesterase. It is also reasonable to presume that thrombi richer in factor XIII and fibronectin would not only be more resistant to fibrinolysis but also more readily attached to the vessel wall. It is hoped that the convincing results reported by Reed and Houng will revive interest in pathologically increased factor XIII.

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