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Letter to the Editor

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I read with interest the manuscript of Lee et al. [1] in the last issue of *Clinical Hemorheology and Microcirculation*. In this study the authors found continuous progress in alternations of red blood cell (RBC) aggregation, blood viscosity adjusted to a hematocrit of 40%, plasma viscosity and yield shear stress values from stable angina (SA) to unstable angina (UA) and acute myocardial infarction (AMI). In the same issue of *Clinical Hemorheology and Microcirculation* Zorino et al. [2] noted that erythrocyte hyperaggregability is associated with a increased risk of AMI in young patients. In reference to this original observations, continuity of hemorheological abnormalities can be extended on patients with lack of tissue reperfusion following successful recanalisation of the infarct-related epicardial artery defined as the achievement of TIMI grade-3 flow. Closely related to fibrinogen concentration, RBC aggregation, plasma viscosity and yield shear stress are the major contributors that cause an increase in vascular resistance especially in the presence of endothelial cell blebbing, white cell infiltration and extravascular edema cased by ischemia and reperfusion injury [3]. The combination of reduced capillary lumen diameter and concomitant hemorheological abnormalities lead to increases in vascular resistance and inadequate microvascular reperfusion. To address those question we measured hemorheological parameters in patients with ST-segment elevation AMI. Our studies demonstrated that plasma viscosity and RBC, aggregability and baseline fibrinogen concentration were significantly higher in patients without tissue reperfusion compared to those with restoration of myocardial flow [4–6]. Those findings are in line with recent reports of Cecchi et al. [7] who noticed that alterations of hemorheological variables are in relation to the achievement of a final TIMI flow less than 3, which in turn is associated with an increased infarct size. Those observations suggest that hyperviscosity can worsen myocardial perfusion leading to an increased infarct size. In the era of interventional cardiology the order of the intensity of hemorheological abnormalities suggested by Lee et al. [1] can be specified as follows: no-reperfusion AMI $>$ reperfusion AMI $>$ UA $>$ SA. The stratification of hemorheological abnormalities especially in patients with acute coronary syndromes are of importance because improvement of blood fluidity and hence microcirculatory flow through normalizing blood rheology may be obtained using isovolemic haemodilution, apheresis or by fibrynolysis after primary percutaneous coronary intervention [8,9].

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