Evaluation of plasma viscosity has been underutilized in the clinical practice. Plasma viscosity is determined by water-content and macromolecular components. Plasma is a highly concentrated protein solution, therefore weak protein-protein interactions can play a role that is not characterized by electrophoresis. The effect of a protein on plasma viscosity depends on its molecular weight and structure. The less spheroid shape, the higher molecular weight, the higher aggregating capacity, and the higher temperature or pH sensitivity a protein has, the higher plasma viscosity results. Plasma is a Newtonian fluid, its viscosity does not depend on flow characteristics, therefore it is simple to measure, especially in capillary viscosimeters. Its normal value is 1.10-1.30 mPa s at 37 degrees C and independent of age and gender. The measurement has high stability and accuracy, thus little alterations may be pathologically important. Inflammations, tissue injuries resulting in plasma protein changes can increase its value with high sensitivity, though low specificity. It can increase in parallel with erythrocyte sedimentation rate (ESR), but it is not influenced by hematocrit (anemia, polycytemia), or time to analysis. Based on these favorable features, in 1942 plasma viscosity was recommended to substitute ESR. In hyperviscosity syndromes plasma viscosity is better in follow-up than ESR. In rheumatoid arthritis, its sensitivity and specificity are better than that of ESR or C-reactive protein. Plasma fibrinogen concentration and plasma viscosity are elevated in unstable angina pectoris and stroke and their higher values are associated with higher rate of major adverse clinical events. Elevation of plasma viscosity correlates to the progression of coronary and peripheral artery diseases. In conclusion, plasma viscosity should be measured routinely in medical practice [1)].

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Késmárky G, Kenyeres P, Rábai M, Tóth K. Plasma viscosity: a forgotten variable. Clin Hemorheol Microcirc. 2008;39(1-4):243-6. PMID: 18503132.