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Blood rheology and hemodynamics.

Baskurt OK[1], Meiselman HJ.

Author information

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Department of Physiology, Akdeniz University Faculty of Medicine, Antalya, Turkey.

Abstract

Blood is a two-phase suspension of formed elements (i.e., red blood cells [RBCs], white blood cells [WBCs], platelets) suspended in an aqueous solution of organic molecules, proteins, and salts called plasma. The apparent viscosity of blood depends on the existing shear forces (i.e., blood behaves as a non-Newtonian fluid) and is determined by hematocrit, plasma viscosity, RBC aggregation, and the mechanical properties of RBCs. RBCs are highly deformable, and this physical property significantly contributes to aiding blood flow both under bulk flow conditions and in the microcirculation. The tendency of RBCs to undergo reversible aggregation is an important determinant of apparent viscosity because the size of RBC aggregates is inversely proportional to the magnitude of shear forces; the aggregates are dispersed with increasing shear forces, then reform under low-flow or static conditions. RBC aggregation also affects the in vivo fluidity of blood, especially in the low-shear regions of the circulatory system. Blood rheology has been reported to be altered in various physiopathological processes: (1) Alterations of hematocrit significantly contribute to hemorheological variations in diseases and in certain extreme physiological conditions; (2) RBC deformability is sensitive to local and general homeostasis, with RBC deformability affected by alterations of the properties and associations of membrane skeletal proteins, the ratio of RBC membrane surface area to cell volume, cell morphology, and cytoplasmic viscosity. Such alterations may result from genetic disorders or may be induced by such factors as abnormal local tissue metabolism, oxidant stress, and activated leukocytes; and (3) RBC aggregation is mainly determined by plasma protein composition and surface properties of RBCs, with increased plasma concentrations of acute phase reactants in inflammatory disorders a common cause of increased RBC aggregation. In addition, RBC aggregation tendency can be modified by alterations of RBC surface properties because of RBC in vivo aging, oxygen-free radicals, or proteolytic enzymes. Impairment of blood fluidity may significantly affect tissue perfusion and result in functional deteriorations, especially if disease processes also disturb vascular properties.