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Erythrocyte Aggregation and the Fåhraeus-Lindquist-Effect

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ABSTRACT

The message provides an analysis of changes in the aggregation properties and orientation of erythrocytes in the shear flow in small vessels. It is suggested that the mechanisms under consideration do not participate in the manifestation of the Fåhraeus-Lindquist effect. The decrease in apparent viscosity and hematocrit is mediated by the transformation of erythrocytes in the shear flow and the exchange of the aqueous phase of erythrocytes and blood plasma.

Keywords: Erythrocyte, Aggregability, Fahraeus-Lindquist effect, Shear stress

INTRODUCTION

Many years have passed since the discovery of a decrease in blood viscosity and hematocrit with a decrease in the size of a vessel with a diameter of less than 150 μm (Fåhraeus-Lindquist effect), but the mechanism has not yet been clarified. Numerous attempts to computational modeling of the effect in small vessels based on the paradigm of redistribution of erythrocytes in the bloodstream and that describes the interaction of plasma with red blood cells, appear unconvincing. The real reason for the change in plasma viscosity and hematocrit of the blood flowing in small vessels remains enigmatic.

Indeed, erythrocytes slide around the plasma layer in axial cylindrical tubes. Plasma acts as a lubricant layer that reduces endothelial injury. It is believed that the movement of blood leads to the depletion of marginal zone concentration by cells, which accelerates the movement of the liquid core. Thus, red blood cell aggregation along with inward radial migration in small vessels is two significant mechanisms determining the effect. But the redistribution of particles in the bloodstream does not can change the ratio of the solid and liquid phases in the vessel, i.e., decrease of the apparent viscosity and hematocrit of the blood. By the way, the widely used term "apparent viscosity" requires clarification. It should be noted that the term “apparent” (or “effective”) viscosity means the derived value of blood viscosity and reflects the viscosity of a Newtonian fluid that would yield the same flow under otherwise identical conditions. As we suggested, increased shear stress in vessels less than 150 μm in size stimulates the transformation of erythrocytes. The pressure gradient causes the liquid phase to move from the erythrocyte to the capillary lumen, which leads to a decrease in blood viscosity and hematocrit. These transformations are reversible. When the erythrocyte emerges from the capillary, the shear stress decreases, the cellular form is restored and water returns to the cell [1]. Generally speaking, the participation of the aggregation properties of red blood cells in the rheology of blood should, in our opinion, is reconsidered. In this context, it is appropriate to realistically assess the aggregation phenomenon of red blood cells.

Erythrocytes have a tendency to aggregate and form what are known as “rouleaux”, but at low shear rates. The effects of such formations are to increase low shear viscosity. **Figure 1** shows tend of the erythrocytes to form aggregates at the shear forces for normal blood.

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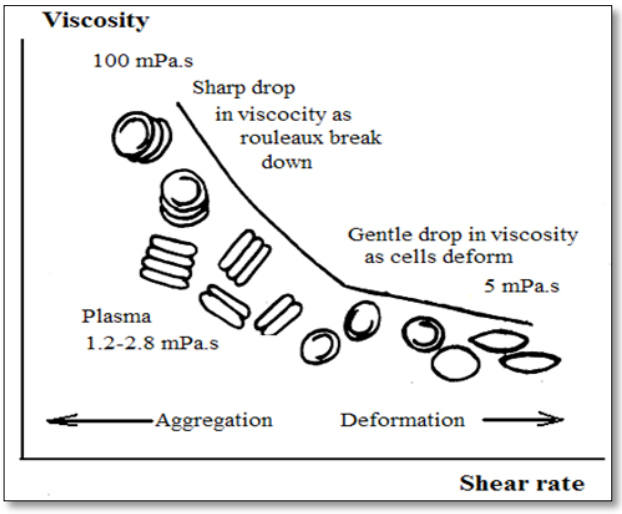

Figure 1. Log/log plot of viscosity versus shear rate in autologous plasma.

If the shear forces acting on blood are drop, the erythrocytes tend to form stacks or aggregates. These stacks are held together by weak forces that are mediated by certain of the plasma proteins. The shear rate is calculated as the speed gradient $\gamma=\partial V_x/\partial y$ in fluid flow tubes in the direction perpendicular to the movement vector Y. Considering parabolic distribution of velocities in the vessel section (Bernoulli effect for viscous fluids flowing slowly in the laminar mode), the shear rate in the vessel clearance is measured linearly, from zero value in the vessel center to the maximum at its wall. Consequently, the true ratio is $\gamma_{threshold}/\gamma_{max}=D_{threshold}/D_{max}$. Thus, for vena cava, the ratio of diameters with the threshold shear rate and the maximum reaches 50/100 or ½, that is, $D_{threshold}$=5 mm at D_{max}=10 mm. Consequently, the conditions for RBC aggregation phenomenon manifestation in vena cava are only created when the vessel clearance is occluded by 4 times. And this is true to the condition if the blood flow rate will not increase when the clearance narrows. For venules with the diameter of 0.02 mm, this ratio reaches 50/1000, that is, the clearance should decrease by 40 times. For arterioles with the diameter of 0.007 mm, the ratio makes up 50/8000, that is, aggregation is possible if the clearance decreases by more than 25 thousand times! As for capillaries whose diameter sometimes comes to one third from the RBC diameter and where all the main metabolic processes occur, there is no point in talking about them [2-4]. Thus, as aggregates are easily destroyed in the normal blood flow under the influence of slight shear efforts, the tendency for their formation in bloods under physiological conditions in vivo does not considerably influence the blood flow. Thus, the phenomenon of RBC aggregation is shown only in case on complete blood flow stoppage. In our opinion, RBC aggregation is a minor physiological phenomenon, the phenomenon of resting blood, comparable with the establishment of hydrogen bonds on the formation of the spatial structure of biological molecules and Van der Waals forces between particles [5,6]. Strengthening of aggregation capacity of a large number of oxygen carriers should be considered as the protective reaction of the organism to change in the protein pattern of plasma in case of pathology on the whole and inflammation in particular, which in fact improves blood supply to tissues. Really, it has been suggested that the effects of aggregation on hemodynamic mechanisms (e.g. plasma skimming, Fåhraeus-Lindquist effect, microvascular hematocrit) may promote rather than impede vascular blood flow [7].

Increased aggregation contributes to accumulation of RBC in central areas of vessels, formation of plasmatic lining and sliding layer near endothelial lining. The presence of rouleaux in the low shear rate central core of large vessels causes the velocity profile to become blunted. So called "plug flow" occurs where the mass of cells is lubricated by a slip film of plasma at the vessel walls. This benefit the distribution of erythrocytes, as well as promoting the margination of leucocytes and platelets towards the vessel endothelium where they may detect injury more readily. And this is observed precisely in large vessels with low shear stresses. It seems unreasonable to actively fight it. It is appropriate to support the mechanism of struggling, which was generated by the organism in the course of evolution for support of blood flow properties in case of pathological processes. For example, with means of medicines like immunoglobulins, that increases pro-aggregation effect along with raising high-density lipoprotein cholesterol level as attractive treatment strategy, by concentrating attention on deformation properties of red blood cells.

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