

Letter to the Editor

Increased blood viscosity: Disease, adaptation or treatment?

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The editorial by Forconi and Gori [1] challenges a cornerstone of cardiovascular physiology in questioning the interpretation of Hagen–Poiseuille’s law for viscous liquid flow in tubes in making the link between the fluid mechanical properties of blood and how these translate into physiological effects, which ultimately set peripheral vascular resistance, blood pressure and cardiac output. Application of this law is deceptively simple: it characterizes cardiovascular regulation through the relationship between the viscosity of blood, and the diameter of the vessels through which it flows, which determine the flow resistance to the delivery of oxygenated blood by the heart. However, this simplicity is now irremediably broken since we know that in the circulation geometry (vessel diameter) is a function of blood viscosity, or more correctly vessel wall shear stress, directly related to the product of blood viscosity and blood flow.

The relationship between vessel diameter and blood viscosity and shear stress is governed by the shear stress dependant release of vasoactive mediator such as prostacyclin and nitric oxide (NO). These effects mostly seen in endothelial cell cultures and as the reaction of the inhibition to the production of NO in intact vascular beds, have now been demonstrated in intact organisms, and even man. In essence we are presented with the counterintuitive concept that increasing blood viscosity in an intact organism may lower systemic vascular resistance. A minor acute increase in hematocrit, and therefore blood viscosity significantly reduces blood pressure and increases cardiac output, an effect not related to the improvement of oxygen carrying capacity [2].

A corollary to the prevailing view of the consequences Hagen–Poiseuille’s law is that increased blood viscosity is necessarily detrimental, since it increases vascular resistance. Again Forconi and Gori [1], challenge this concept pointing out that the opposite, namely lowering blood viscosity via for instance hemodilution, has not been demonstrated to provide a benefit. In fact the tendency to anemia, at levels that do not impact oxygen carrying capacity is shown to be associated with risk of hypertension [3], while erythrocytosis consequent to adaptation to altitude, does little to change oxygen carrying capacity, but may be involved in providing significant compensatory vasodilatation. These considerations lead to the emergence of increased viscosity as an adaptive mechanism, particularly effective on the healthy individual exposed to unusual challenges.

We refer in general to changes in blood viscosity, a process that has two components, one the changes in blood itself, and the second the changes in plasma viscosity. It is comparatively simple to lower blood viscosity by hemodilution, and blood transfusion restore blood viscosity providing beneficial effects

apparent a priori to the restoration of oxygen delivery [4]. In this context blood can be viewed as an excellent plasma expander independently of its oxygen carrying capacity, and by virtue of its viscogenic properties. It is, however, more difficult to achieve the same effect by increasing plasma viscosity, due to the lack of suitable high viscosity plasma expanders. This is also a consequence of most efforts in plasma expansion development being to the present directed at lowering blood viscosity [5]. Nonetheless, it is becoming increasingly evident that restoration of blood volume with a blood transfusion can be postponed to intrinsic blood hemoglobin levels that are significantly lower than those proposed by conventional transfusion triggers, if the viscosity of plasma is increased to the level that microvascular function is maintained [6,7].

Blood viscosity interacts with the organism via the endothelium, therefore the positive responses derived from the increased shear stress stimulus only manifest if this organ system is responsive, and presumably healthy. An alternative condition proposed by Gori et al., 2008 [8] is that the endothelium may present reduced endothelial vasomotor reactivity. The functionality of the endothelium is a critical component of the hemostatic conditions determined by blood viscosity because over or under production of shear stress dependant mediators also influence inflammation. We do not know what is the optimal blood viscosity in humans, however given their rapid and complex evolution it may be that present hematocrit levels, and therefore blood viscosity may be somewhat lower than optimal. This would account in part for the increasing incidence of pro-inflammatory conditions, and constitute a component of hypertension. In this context we should not lose sight that diuretics remain one of the most effective treatments for high blood pressure, and that one of their effects is the increase of Hct and therefore blood viscosity [9].

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