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# Effect Of Size Of The Blood Vassal On The Apparent Viscoscity Of Blood

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**Abstract:** In general blood has higher viscosity than plasma, and when the hematocrit rises, the viscosity of the suspension increases and the non-Newtonian behaviour of blood becomes more relevant, in particular at very low shear rates. The apparent viscosity increases slowly until a shear rate less than 1 s −1, where it rises markedly. The reason for this is that at low shear rates the erythrocytes have the ability to form a primary aggregate structure of rod shaped stacks of individual cells called rouleaux, that align to each other and form a secondary structure consisting of branched three-dimensional (3D) aggregates. It has been experimentally observed that rouleaux will not form if the erythrocytes have been hardened or in the absence of fibrinogen and globulins (plasma proteins). In fact, suspensions of erythrocytes in plasma demonstrate a strong non-Newtonian behaviour whereas when they are in suspension in physiological saline (with no fibrinogen or globulins) the behaviour of the fluid is Newtonian. For standing blood subjected to a shear stress lower than a critical value, these 3D structures can form and blood exhibits yield stress and resists to flow until a certain force is applied. This can happen only if the hematocrit is high enough.
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## 1. Introduction:

Vascular stenosis is a term that is commonly used to describe narrowing of blood vessels. When the stenosis occurs in a large artery, for example, aorta, coronary and carotid arteries, the disease is referred to as atherosclerosis, or large vessel disease.[1] Research over the past several decades has established the important role of fluid flow in large vessel disease. In recent years, however, there is an apparent paradigm shift in our understanding of vascular stenosis.[2] It has been established now that stenosis can and often occur in smaller arteries (arterioles) with internal diameters ranging up to a few hundred microns.[3] The condition, known as microvascular disease, could have severe consequences. For example, in coronary microvascular disease or CMVD, a blockage occurs in the microvessels supplying blood to heart muscles. In lacunar infarct, a blockage occurs in the smaller arteries supplying blood to the interior of the brain. Microvascular stenosis can also occur in retinal and renal microcirculation. Flow blockage in small vessels can occur also by adherent leukocytes, gas emboli, etc. Apart from physiological examples, *in vitro* microstenosis geometry are utilized in biomedical devices for various purposes, e.g., for cell separation.[4,5,6]

It is well known that blood possesses significant nonlinear Theological properties. Studies in large scale viscometers have shown a disproportionate increase in apparent blood viscosity with decrease of shear rate, especially in the range below 50 s'''.[7] Relatively few studies have been undertaken under flow conditions that might be more similar to those existing in the vascular system. Published data on tube flow appear to confirm the general trend of an increase in the apparent viscosity of blood with decreasing shear rate. Little data on the effects of shear rate on apparent viscosity are available for tubes in the diameter range of the arterioles and venules that represent the primary sites of pre- and post-capillary resistance in the vascular system. Predictions of in vivo viscosity changes with alteration of perfusion conditions are therefore largely based on data obtained from viscometric studies in large viscometers; such data, however, may not adequately reflect the Theological behavior of blood in the peripheral circulation.[8,9]

The apparent viscosity of blood flowing through narrow glass tubes decreases strongly with decreasing tube diameter over the range from about 300 $\mu$m to about 10 $\mu$m. This phenomenon, known as the Fåhraeus-Lindqvist effect, occurs because blood is a concentrated suspension of deformable red blood cells with a typical dimension of about 8 $\mu$m. Most of the

resistance to blood flow through the circulatory system resides in microvessels with diameters in this range. Apparent viscosity of blood in microvessels *in vivo* has been found to be significantly higher than in glass tubes with corresponding diameters. Here we review experimental observations of blood's apparent viscosity *in vitro* and *in vivo*, and progress towards a quantitative theoretical understanding of the mechanisms involved. [10,11,12]

The relationship between the pressure generated by the heart and the resulting flow in the circulatory system has long been a subject of study. In the mid-nineteenth century, J.L.M. Poiseuille addressed this topic, and established the fourth-power dependence of flow rate $Q$ on diameter $D$, for a tube with given length $L$ and for given driving pressure $\Delta p$ . This relationship is expressed in terms of 'Poiseuille's law'[13,14]

$$Q=\pi 128\Delta pD4L\mu \quad \ldots\ldots 1$$

where $\mu$ is the fluid viscosity. Blood, however, is a concentrated suspension of deformable cells, mainly red blood cells (RBCs), and does not exhibit a unique well-defined viscosity when it flows in narrow tubes. The resistance to flow is determined by the mechanical interactions between each RBC and the suspending medium, the tube walls, and other RBCs. [15,16]

Under such conditions, the effect of blood's rheological properties on resistance to flow can be represented in terms of its apparent viscosity, which is derived from Poiseuille's law: [17,18]

$$\mu app=\pi 128\Delta pD4QL \quad \ldots\ldots 2$$

For flow in a tube with given length and diameter, the resistance to blood flow (the ratio of pressure drop to flow) is directly proportional to the apparent viscosity. [19] In general, the apparent viscosity may depend on tube diameter, flow rate, hematocrit (volume fraction of RBCs), viscosity of plasma (the suspending fluid), biophysical properties of RBCs and biophysical properties of the tube or blood vessel. The purpose of this review is to present a summary of experimental and theoretical results concerning the apparent viscosity of blood and its underlying mechanisms, and to indicate some areas of current investigation. [20,21]

## 2. Blood components

Blood is a concentrated suspension of several formed cellular elements, red blood cells (RBCs or erythrocytes), white blood cells (WBCs or leukocytes) and platelets (thrombocytes), in an aqueous polymeric and ionic solution, the plasma, composed of 93% water and 3 % particles, namely, electrolytes, organic molecules, numerous proteins (albumin, globulins and fibrinogen) and waste products. [22,23] Plasma's central physiological function is to transport these dissolved substances, nutrients, wastes and the formed cellular elements throughout the circulatory system. Normal erythrocytes are biconcave discs with a mean diameter of 6 to 8 μm and a maximal thickness of 1.9 μm. The average volume of an erythrocyte is 90 μm3. [24,25,26] Their number per cubic millimetre of blood is approximately 5 to 6 x 106 and they represent approximately 40 to 45% by volume of the normal human blood and more than 99% of all blood cells. The first percentage is called hematocrit. [27] The primary function of erythrocytes is to transport oxygen and carbon dioxide. Leukocytes are roughly spherical and much larger than erythrocytes, but they exist in a smaller number in blood: their diameter ranges between 6 and 17 μm and there are approximately 7 to 11 x 103 per cubic millimetre in a normal adult. Leukocytes are subdivided into granulocytes (65%), lymphocytes (30%), monocytes (5%) and natural killer cells. Granulocytes are further subdivided into neutrophils (95%), eosinophils (4%) and basophils (1%). The leukocytes play a vital role in fighting infection and thus are able to migrate out of the blood vessels and into the tissues. Thrombocytes are small discoid non-nucleated cell fragments, much smaller than erythrocytes and leukocytes (approximately 2 to 3 μm3 in volume). Thrombocytes are a vital component of the blood clotting mechanism. The total volume concentration of leukocytes and thrombocytes is only about 1%. Blood cells are continually produced by the bone marrow over a human's life. For example, erythrocytes have an average lifetime of 120 days and the body must produce about 3 x 109 new erythrocytes for each kilogram of body weight every day. Due to ageing and rupturing they must be constantly replaced. [28,29,30]

## 3. Experimental observations of apparent viscosity

### 3.1. Observations in vitro

Around 1930, Martini et al. and Fåhraeus and Lindqvist observed a marked decrease in the apparent viscosity of blood in glass tubes with diameter decreasing below 300 $\mu$m, a phenomenon known as the Fåhraeus-Lindqvist effect. For example, the apparent viscosity in a tube of diameter 40 $\mu$m is about 60% of its value in a 300 $\mu$m tube. In subsequent years, a number of authors measured the apparent viscosity of blood in narrow glass tubes. Most of these studies used suspensions of human RBCs or whole human blood with anticoagulants. Results from several studies were assembled and reanalyzed in 1992 by Pries et al., who developed empirical equations to describe the dependence of relative apparent viscosity $\mu_{rel}$ (the ratio of apparent viscosity to suspending medium viscosity) on tube diameter and hematocrit: [31]

$$\mu_{rel} = 1 + (\mu_{0.45} - 1)\frac{(1 - H_D)^C - 1}{(1 - 0.45)^C - 1} \quad \ldots.3$$

where

$$\mu_{0.45} = 220\exp(-1.3\,D) + 3.2 - 2.44\exp(-0.06\,D^{0.645}) \quad \ldots 4$$

and

$$C = (0.8 + \exp(-0.075\,D))(-1 + (1+10^{-11}D^{12})^{-1}) + (1+10^{-11}D^{12})^{-1} \quad .5$$

Here $D$ is the diameter in $\mu$m and $H_D$ is the discharge hematocrit, defined as the volume flow rate of RBCs as a fraction of the total volume flow rate. [32,33] The dependence of viscosity on diameter for $H_D = 0.45$ (a typical value in humans) is given by $\mu_{0.45}$. The quantity $C$ describes the dependence of viscosity on hematocrit, which is approximately linear for diameters up to about 8 $\mu$m but shows a highly nonlinear dependence at large diameters. In this study, only data obtained at high shear rates ( U/D≥50s−1) were considered, where U is the mean velocity. Apparent viscosity is almost independent of flow rate in this range, but increases at lower shear rates. [34,35]

The dependence of relative apparent viscosity on diameter and discharge hematocrit, according to these equations. The trend of apparent viscosity to decrease with decreasing diameter continues down to about 7 $\mu$m. At smaller diameters, relative apparent viscosity rises rapidly as the diameter approaches a critical minimum diameter, which is about 2.7 $\mu$m. Although highly deformable, RBCs are subject to constraints of constant volume and almost constant surface area. These constraints prevent passage of intact cells through tubes narrower than this critical diameter. [36]

**3.2 Observations in vivo**

For many years, the observed reduction of blood's apparent viscosity in narrow tubes was assumed to apply also to blood flow in the microvessels of living tissues. However, direct measurements *in vivo* are technically challenging due to the difficulty of measuring pressure drops in microvessels. Lipowsky et al. performed such measurements in vessels with diameters in the range 10–60 $\mu$m and obtained estimates of apparent viscosity substantially higher than would be expected based on the *in-vitro* behavior. The data from those experiments were not sufficiently comprehensive to establish the dependence of apparent viscosity on diameter and hematocrit. [37]

Pries et al. analyzed the rheological behavior of blood in microvessels using a network-based approach. Blood flow in microvascular networks in the rat mesentery was observed experimentally and compared segment-by-segment with the predictions of theoretical models. The distributions of flow and hematocrit derived from simulations based on the parametric description of blood viscosity *in vitro* described above were found to be inconsistent with the observed behavior. However, satisfactory agreement was found when an alternative parametric description of blood viscosity was used, as follows: [38]

$$\mu_{rel} = \left[1 + (\mu^*_{0.45} - 1)\frac{(1-H_D)^C - 1}{(1-0.45)^C - 1}\left(\frac{D}{D-1.1}\right)^2\right]\left(\frac{D}{D-1.1}\right)^2 \quad \ldots\ldots\ldots 6$$

Where

$$\mu^*_{0.45} = 6\exp(-0.085\,D) + 3.2 - 2.44\exp(-0.06\,D^{0.645}) \quad \ldots 7$$

and $C$ is given by equation (7).

The dependence of relative apparent viscosity on diameter and discharge hematocrit according to these equations. The apparent viscosity decreases with decreasing diameter down to about 40 $\mu$m, and rises substantially at smaller diameters. According to this result, the apparent viscosity in microvessels with diameters in the range 5 to 20 $\mu$m is much higher than expected based on the *in-vitro* results. A substantial fraction of the flow resistance in microvascular networks resides in vessels with diameters in this range. Therefore, the overall flow resistance of the microcirculation according to this relationship is expected to be significantly higher than it would be if the *in-vitro* behavior was applicable. Pries et al. found that the pressure drop across mesenteric networks required to drive the observed flow rates was higher by a factor of almost 3 when the *in-vivo* behavior was assumed., relative to the *in-vitro* behavior, and that it agreed well with the observed pressure drop. This and subsequent experimental findings have generally supported the conclusions described here. However, due to the above mentioned technical difficulties, systematic experimental testing of this empirically derived theory for blood's apparent viscosity *in vivo* remains to be achieved. [39,40]

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