Blood Viscosity and Cerebral Blood Flow

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Introduction

The blood flow to the brain is largely controlled by the metabolic demand. This determines the rate of delivery of oxygen and the removal of carbon dioxide. Under normal physiological conditions, there is usually considerably circulatory reserve. In patients with occlusive vascular disease, when conditions are becoming critical, the first response is to improve cerebral blood flow by vasodilatation, then if necessary, when conditions become more extreme, by increased oxygen extraction [1].

What influence does blood viscosity have on the circulation's ability to supply the metabolic demands of the brain? Blood flow is proportional to the pressure gradient and to the fourth power of the radius of the blood vessels and is inversely proportional to the viscosity. Therefore changes in viscosity can be compensated for by relatively small changes in the radius of the vessel. Except in situations where there is already maximal vasodilatation, it is possible that any viscosity effect on limiting blood flow and oxygen supply to the brain would be compensated for by vasodilatation. However, the situation is complex, because factors influencing flow, viscosity, and oxygen carriage are all interrelated.

Blood Viscosity

The two main determinants of whole blood viscosity are the shear rate and hematocrit. The shear rate is directly proportional to the rate of blood flow and inversely proportional to the diameter of the vessels. Therefore where there is vasodilatation and slowing of blood flow, the net effect on viscosity will depend on the degree of dilatation and the amount by which blood flow has changed. In most situations, however, when blood flow drops the shear rates fall and viscosity increases.

The hematocrit depends largely on the number and size of red cells in the circulation. It varies considerably between large vessels and the microcircu-
lation, but one can assume that subjects with high hematocrit in venous blood will have a higher than normal hematocrit in the microcirculation.

Other influences on whole blood viscosity include plasma viscosity. This is mainly influenced by the large protein molecules like fibrinogen and globulin. But these plasma proteins also interact with the cellular components of the blood, producing a reversible red cell aggregation, especially at low shear rates. The size and number of these aggregates have a considerable influence on the fluidity of the blood within the microcirculation. The rigidity of the red cells also limits the blood’s ability to pass through the microcirculation, as well as influencing the rate of flow through the major vessels. The white blood cells, because of their small numbers, except in patients with leukemia, have relatively little impact on whole blood viscosity in major vessels. However, it is likely that they have an important role in determining the rate of flow through the microcirculation, particularly in disease states.

**Cerebral Blood Flow**

In view of the complexity of the interrelationships described above, it is clearly necessary to measure cerebral blood flow and blood viscosity in different situations in order to attempt to understand the influence of rheological factors on the cerebral circulation. Cerebral blood flow has been shown to be high in patients with anemia [2, 3] and therefore low hematocrit and low whole blood viscosity. Conversely, blood flow has been shown to be reduced in patients with polycythemia [4] with elevated hematocrit and whole blood viscosity. The chance of stroke is reduced in anemia [5] and greatly increased in polycythemia [6]. However, most of the patients who have stroke, do so when their hematocrit is normal, so what is the relationship between the flow and hematocrit and viscosity within the normally accepted physiological range? Do the cerebral blood vessels autoregulate to changes in hematocrit and viscosity in the physiological range in the same way as they autoregulate to changes in blood pressure? The results of blood flow studies indicate that this is not the case and that throughout the physiological range, there is an inverse relationship between hematocrit [7], viscosity [8], and blood flow.

If patients with polycythemia have hematocrit reduced by venesection, then the blood flow can be restored toward normal [4]. Similarly, in subjects with high normal hematocrit, venesection and subsequent hematocrit reduction results in an increase in cerebral blood flow [7].

Lowering hematocrit results in a reduction in the oxygen-carrying capacity of each milliliter of blood, and this observed increase in flow may be to maintain oxygen supply rather than due to a reduction in viscosity. Studies on patients with a hemoglobin variant with increased oxygen affinity [9] showed they had a much higher cerebral blood flow than patients with the same hematocrit but with normal hemoglobin. It is also known that patients with polycythemia secondary to lung disease and hypoxia have a higher cerebral blood flow than subjects with similar hematocrit and viscosity [10]. This is only in part due to