High Blood Viscosity Syndrome in Cerebral Infarction

BY ERWIN O. OTT, M.D., HELMUT LECHNER, M.D., AND ALBERTO ARANIBAR, M.D.

Abstract:
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Determinations of whole blood viscosity by means of a cone plate viscometer at 37°C and at shear rates of 212, 42, 21 and 11 sec⁻¹ were done in 50 patients with recent cerebral infarction of the carotid system, and the values compared to a control group of 50 patients of the same age. In stroke patients a statistically significant elevation of blood viscosity over the whole range of shear rates was demonstrated, more obviously significant at low shear rates (P < 0.0001) such as occur in small vessels. Since the mean hematocrit levels of both groups were in the normal range, it was considered that hematocrit values estimated from peripheral blood do not necessarily give accurate information about viscosity levels existing at the same time.

In cases of severe cerebral infarction without angiographically demonstrable stenotic or obstructive lesions, it was suggested that high blood viscosity impairs hemodynamic conditions in the cerebral microvasculature in addition to narrow arteriosclerotic vessels, changes in flow velocity gradients and insufficient collateral circulation.

Additional Key Words
flow velocity gradient
angiography
cone plate viscometer
hemodynamic
microvasculature
hematocrit

Introduction

The clinical symptoms of patients with recent cerebral infarction indicate a breakdown of blood flow and metabolism in the cerebral microvasculature, frequently not discovered angiographically. Many factors are suggested to be responsible, among which blood viscosity is of considerable importance.

"High blood viscosity syndrome" describes a condition in which viscosity of the whole blood is increased as well as resistance to blood flow. The condition can be caused, moreover, by the aggregation of red blood cells and platelets as well as by an increased concentration of plasma components.1,2 The presence of either thrombotic or arteriosclerotic vessels will affect the blood flow velocity, the shear rates, the dynamics of blood coagulation and thrombosis formation.3 Hematocrits ranging from 36% to 52% have linear relationships to blood viscosity at various shear rates and no considerable effect on cerebral blood flow, if varied between 30% and 60%.4,5 The non-Newtonian behavior of whole blood with normal hematocrit was shown resulting primarily from cell-protein interactions mainly due to the presence of fibrinogen, especially at low shear rates.6

In cases of infarction and thrombosis, high viscosity was attributed to an excessive aggregation of red cells due to an elevation of lipids or fibrinogen concentrations.5,7 Likewise, slowing of flow and stasis due to increased viscosity and poor collateral circulation following experimental cerebrovascular occlusion have been observed.6,9 Large fat meals or the addition of dietary lipids in vitro and in vivo have profound effects on the rheological properties of blood and the mechanisms of coagulation producing extreme aggregation of red blood cells, increased viscosity and sedimentation rate, and a tendency toward hypercoagulability.10-14 Since the influence of high viscosity on cerebral blood flow is small, as long as fairly high flow is maintained,16 essential hemodynamic considerations must be made when blood flow disturbances in the microvasculature occur, because blood flow depends not only on viscosity but also on perfusion pressure and vascular cross section.18 It was the purpose of this study to prove the existence of blood viscosity changes in patients with completed stroke and to show that hematocrit values cannot necessarily be related to viscosity levels existing at the same time.

Methods

For the determinations we used a cone plate viscometer, which permits the rapid quantitative analysis of small samples of blood at various shear rates.17 Blood was drawn from a cubital vein and heparinized (500 E/10 ml blood). Samples were estimated at 37°C and at four different shear rates (212, 42, 21 and 11 sec⁻¹) described in detail elsewhere.4,18 The viscometer was calibrated against distilled water at 37°C. Estimations of hematocrits were done from the same samples by the Wintrobe technique.19

Viscosity and hematocrit measurements were carried out on 50 patients who suffered from recent cerebral infarction of the carotid system before treatment was started but no later than 24 hours after the acute event. The age of this
group ranged from 60 to 82 years with a mean age of 70. The results were compared with those obtained in a control group of 50 patients whose histories did not show any evidence of cerebrovascular disturbances nor of any diseases or therapy which could influence blood viscosity. The age of this group ranged from 63 to 75 years with a mean age of 68. All patients of the stroke group underwent angiographical examinations, which were performed at least two weeks after the acute event. Irregularities of the vessel walls were interpreted as arteriosclerotic changes and listed only in cases without stenotic or obstructive lesions. The ratio of males to females in both groups was 1:1. Statistical significance was evaluated by the Student t-test.

**Results**

The mean viscosity values and one standard deviation, in centipoises, are presented in table 1. The elevation of viscosity in the stroke patients is statistically significant over the whole range of shear rates, but more obviously significant at low shear rates (P < 0.0001).

The mean hematocrit values and one standard deviation also are presented in table 1. Since the ratio of males to females in both groups was 1:1, the hematocrit values of both males and females were calculated together. It should be noted that the hematocrit level of the patients with cerebral infarction remains within normal range although there is a statistically significant elevation if compared to the control group (P < 0.001).

Figure 1 displays the viscosity data, in centipoises, plotted against the rate of shear, in sec^{-1}, on a log-log scale to emphasize the effect of low shear rates on blood viscosity in patients with cerebral infarction.

Table 2 shows the angiographical findings in the stroke group. All patients of this group suffered from nonhemorrhagic infarction with severe neurological deficit.

**Discussion**

The results of our investigations show a highly significant elevation of blood viscosity in patients with recent cerebral infarction. Since the viscosity changes were obvious at low shear rates as occur in small vessels, these data suggest the important role of disturbances in the rheological properties of blood in this disease, symptoms of which indicate a breakdown of blood flow and metabolism in the cerebral microvasculature. The most important factors which can affect blood viscosity are aggregation of corpuscular elements, changes in the concentration of plasma components, flow velocity gradients and vessel diameters.1,2,7,20,21

Hematocrit was shown to have no considerable effect on cerebral blood flow if varied between 30% and 60%.8 Since the mean hematocrit value of the stroke patients was within normal range but statistically significantly elevated if compared to the control group, it might be suggested that even a mean value of 45% was too high for this group. However, we think that the increase in blood viscosity was due to other factors than changes in the red cell concentra-

![Figure 1](http://stroke.ahajournals.org/)

**Figure 1**  
Viscosity, in centipoises, plotted against shear rate, in sec^{-1}, on a log-log scale, in patients with cerebral infarction and a control group of the same age.

<table>
<thead>
<tr>
<th>Shear rate (sec^{-1})</th>
<th>10</th>
<th>20</th>
<th>40</th>
<th>80</th>
<th>100</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal control group (N = 50)</td>
<td>4.1 ± 0.3</td>
<td>5.3 ± 0.4</td>
<td>5.9 ± 0.6</td>
<td>7.2 ± 0.8</td>
<td></td>
</tr>
<tr>
<td>HTC = 40 ± 2%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cerebral infarction (N = 50)</td>
<td>4.9 ± 0.3*</td>
<td>6.7 ± 0.4†</td>
<td>8.3 ± 0.7†</td>
<td>10.3 ± 1.2†</td>
<td></td>
</tr>
<tr>
<td>HTC = 45 ± 3%*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values = mean ± standard deviation.  
N = number of cases.  
= Statistically significant (P < 0.001).  
† = Highly statistically significant (P < 0.0001).
tion, a conclusion which might be supported by results observed in patients with transient ischemic attacks.\textsuperscript{21} Observations of regional hemodynamic and metabolic changes in experimental cerebral infarction\textsuperscript{8, 9} have led us to consider that a hematocrit level estimated from peripheral blood cannot be related to regional alterations in the hematocrit of blood perfusing an ischemic zone in the microvasculature.

The pronounced elevation in blood viscosity in patients with cerebral infarction, therefore, must be accounted for by an excessive aggregation of red blood cells, attributed to an elevation of lipids and/or fibrinogen concentrations.\textsuperscript{8, 10-14} This conclusion is supported by the evidence of hyperlipoproteinemia, an elevation of fibrinogen concentration, and an increase in several coagulation parameters in stroke patients.\textsuperscript{20-22} Certain factors sometimes associated with stroke have been shown to influence blood viscosity,\textsuperscript{26-28} and a relationship between hyperviscosity and these factors have been observed.\textsuperscript{29}

The viscosity changes observed in our population sample might be argued to be due to cerebral infarction, shock, or systemic dehydration. However, the influence of high viscosity on cerebral blood flow is small as long as fairly high flow is maintained.\textsuperscript{15, 29} As a matter of physical facts, slowing of flow or stasis in the microvasculature must occur if high blood viscosity, probably due to risk factors, impairs the hemodynamic conditions together with narrow, arteriosclerotic vessels, changes in flow velocity gradients and poor collateral circulation.\textsuperscript{20-22} This could be an explanation for cases of cerebral infarction without angiographically demonstrable stenotic or obstructive lesions.

Acknowledgment

We are indebted to Mrs. H. Taus, Medical Technical Assistant, for her assistance in carrying out determinations of the blood samples.

References

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Stroke. 1974;5:330-333
doi: 10.1161/01.STR.5.3.330

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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