Hematocrit and Clinical Outcome in Acute Cerebral Infarction
G. Ozaita, MD, L. Calandre, MD, E. Peinado, MD, A. Rodríguez-Antigüedad, MD, and F. Bermejo, MD

Hematocrits of 131 cerebral infarction cases were correlated with the outcome at 2 weeks. Bivariate analysis showed that cases with more intense admission deficit had lower admission hematocrit and that cases with poorer outcome had lower Day 2 and 4 hematocrits. However, multivariate analysis of several prognostic factors (including hematocrit, glycemia, blood pressure, age, and sex) showed that hematocrit was not independently related to outcome. Higher hematocrit thus is not indicative of less favorable short-term outcome. (Stroke 1987;18:1166–1168)

Hematocrit plays an important role in blood rheology and in cerebral blood flow (CBF) dynamics.1–4 It has been shown that reduction of hematocrit improves CBF so it seemed reasonable to assume that hematocrit reduction would improve circulation and subsequently ameliorate the clinical manifestations of cerebral infarction. Initially encouraging results have been reported in a recent clinical study on hemodilution therapy. However, there are few studies6–7 on the role of hematocrit in the clinical outcome of cerebral infarction cases, and the few studies tend to demonstrate the opposite: the lower the hematocrit, the less favorable the outcome. With the aim of reassessing these conflicting results, we made the following study.

Subjects and Methods
Our study was carried out in a retrospective series of 131 consecutive patients admitted to the Department of Neurology and diagnosed as cerebral infarction. All were admitted within 48 hours of the onset of symptoms. The diagnosis was based on clinical data and evidence from a computed tomography (CT) scan, which was either normal or showed an area of infarction. Neurologic status was evaluated at the time of admission and 2 weeks later. The neurologic deficit was considered to be mild when the patient did not need help to feed himself or to walk and could communicate correctly; moderate when the patient needed some help for feeding and could move his limbs against resistance, could walk with help, or could communicate in some way; and severe when the patient had to be fed, could not move his limbs against resistance or walk, or could not communicate in any way. This scale was validated by the significant relation (p<0.001, analysis of variance) found when it was compared with the Canadian Neurological Scale,8 also assessed in our series. Outcome analysis was based on the absolute deficits at the end of follow-up. Patients were treated with supportive measures and antiedema agents when needed.

Hematocrit was assessed in every patient from blood samples obtained between Days 2 and 4 after admission. In 126 cases, admission hematocrit was also assessed. Other data noted were history of hypertension and diabetes, and blood pressure and blood glucose levels, both measured on Days 1 and 3 of hospitalization. Patients with evidence of valvular heart disease or recent myocardial infarction were excluded.

We used the Mann-Whitney and Kruskal-Wallis nonparametric tests for bivariate analysis and Pearson's coefficient for correlations. For multivariate study, we used multiple regression analysis and a stepwise discriminant analysis using the statistical package BMDP.

Results
Age, sex, and other general data are listed in Table 1.

The correlation between late (Days 2–4) hematocrit and neurologic deficit assessed after 2 weeks was significant (p<0.05, Kruskal-Wallis test). Table 2 shows how hematocrit decreased as outcome worsened (except in deaths). It is clear that there is no correlation between hematocrit and admission neurologic deficit. In bivariate analysis a strong correlation was found between hematocrit and sex (men: 48.8±5.5, mean ± SD; women: 43±4.3; p<0.001, Mann-Whitney test). There was also a significant correlation between systolic and diastolic blood pressure and hematocrit (r=0.24 for both; p<0.01). No relation existed between hematocrit and age, location of the infarction, history of hypertension (although mean hematocrit was 47.7 in cases with hypertension and 45.9 in cases without), or blood glucose levels. Admission hematocrit (mean ± SD 47.2±6) was slightly greater than late hematocrit (47.1±5.4; nonsignificant, paired means difference test), and both values were strongly correlated (r=0.74; p<0.001). At variance with late hematocrit, admission hematocrit was significantly related (p<0.01, Kruskal-Wallis test) to admission deficit but not to outcome deficit.
In multiple regression analysis there was clear-cut evidence that late hematocrit was not significantly related to clinical outcome when the 2-week deficit was the dependent variable and age, sex, history of diabetes and/or hypertension, clinical deficit on admission, blood pressure, and blood glucose levels were the independent variables. Significance was reached only by history of diabetes (p<0.03) and blood glucose levels (p<0.009). Likewise, discriminant analysis showed no influence of hematocrit on neurologic outcome.

Discussion

In recent years a promising therapeutic modality for acute cerebral infarction has been modification of blood rheologic conditions. Experimental evidence suggests that high hematocrit is related to a larger area of focal ischemia and to increased metabolic damage. This finding correlates with the fact that high hematocrit leads to more extensive cerebral infarction, as shown by CT scan, in patients with carotid occlusion. In stroke patients, hematocrit has been related positively with mortality in cases <75 years of age. It has also been shown than in nonstroke cases, hematocrit >47% raises the blood viscosity and lowers CBF, which increases after hematocrit reduction by venesection. Hematocrit has been found to be increased in acute brain ischemia patients compared with controls, and reduction of hematocrit by isovolemic hemodilution is significantly related to a better outcome in cerebral infarction cases as well as to improvement of cerebral perfusion in such cases. Although theoretically hematocrit reduction by hemodilution should correlate with a decrease in oxygen-carrying capacity, according to Shapiro's nomogram, it has not been shown to be related to any significant change in oxygen partial pressure, oxygen delivery capacity, or cerebral metabolic rate for oxygen. It is possible that in stroke patients hemodilution increases oxygen supply to the infarcted area as a result of increased cardiac output or improved focal flow. Experimental evidence shows that hematocrit reduction by hemodilution improves CBF in animals with focal ischemia. These results have prompted the design of extensive therapeutic trials to assess the effect on clinical outcome of hematocrit reduction by venesection and hemodilution. Nonetheless, little attention has been paid to the role of hematocrit in the clinical outcome of untreated patients. The most relevant study to address this subject is the one recently published by Levy et al, who point out that hematocrit is inversely related to neurologic outcome. Thus, only 50% of ischemic stroke patients with hematocrit <40% regained independence, whereas 71% of cases with hematocrit >40% regained independence (p<0.01). In bivariate analysis, Levy et al showed that the influence of hematocrit on outcome is not related to age, sex, hypertension, or diabetes, although the figures given are not easy to interpret because of the different number of patients in the various groups. Another recent report shows that mean hematocrit decreases from cases with transient ischemic attacks (46%) to cases with reversible ischemic neurologic deficit (45%) to cases with completed stroke (44%). As regards this negative role

### Table 1. Clinical Characteristics of 131 Cases of Cerebral Infarction

<table>
<thead>
<tr>
<th>Age</th>
<th>60.9 years (SD 12.8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>64.1</td>
</tr>
<tr>
<td>Female</td>
<td>35.8</td>
</tr>
<tr>
<td>History of</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>47.3</td>
</tr>
<tr>
<td>Previous stroke</td>
<td>30</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>13</td>
</tr>
<tr>
<td>Infarction</td>
<td></td>
</tr>
<tr>
<td>Carotid system</td>
<td>74</td>
</tr>
<tr>
<td>Verteobasilar system</td>
<td>26</td>
</tr>
</tbody>
</table>

### Table 2. Hematocrit and Clinical Deficit

<table>
<thead>
<tr>
<th>Admission hematocrit</th>
<th>Normoglycemic patients</th>
<th>Hyperglycemic patients</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td>Mild</td>
<td>48.4 ± 4.6</td>
<td>46.7 ± 5.4</td>
<td>49.0 ± 5.2</td>
</tr>
<tr>
<td>Moderate</td>
<td>48.0 ± 6.0</td>
<td>46.4 ± 5.6</td>
<td>44.8 ± 3</td>
</tr>
<tr>
<td>Severe</td>
<td>45.3 ± 6.5</td>
<td>46.2 ± 5.4</td>
<td>47.7 ± 3</td>
</tr>
<tr>
<td>Outcome deficit</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nil</td>
<td>46.3 ± 5.1</td>
<td>46.3 ± 4.7</td>
<td>49.2 ± 1</td>
</tr>
<tr>
<td>Mild</td>
<td>48.8 ± 4.2</td>
<td>48.5 ± 5.6</td>
<td>49.1 ± 4.9</td>
</tr>
<tr>
<td>Moderate</td>
<td>48.2 ± 7.1</td>
<td>47.7 ± 6.6</td>
<td>45.7 ± 2.5</td>
</tr>
<tr>
<td>Severe</td>
<td>45.2 ± 5.1</td>
<td>42.0 ± 1.4</td>
<td>45.5 ± 2.9</td>
</tr>
<tr>
<td>Death</td>
<td>47.3 ± 5.6</td>
<td>—</td>
<td>45.0 ± 6.4</td>
</tr>
</tbody>
</table>

*Cases with 2 separate glucose values >120 mg/dl. Cases with transient hyperglycemia are excluded.*
of low hematocrit, an epidemiologic study has shown that cerebral infarction incidence is increased in women (but not in men) with low hematocrit. The authors of this study comment that one of the reasons for this relation could be that low hematocrit is related to reduced tissue oxygenation. It has also been demonstrated that hematocrit <30% has deleterious effects on brain metabolism.

In our study, the initial bivariate analysis revealed that short-term neurologic outcome was related to late hematocrit; the higher the hematocrit the better the outcome. However, a subsequent multivariate analysis including other prognostic variables did not corroborate the significance of the role of hematocrit in prognosis. Further analysis showed that hematocrit was significantly related to sex, being lower in women. Although admission hematocrit was slightly greater than late hematocrit, the difference between them was not significant, which contradicts the idea that initial hematocrit can be increased partly due to early dehydration. However, our findings show that there was only a tendency to elevated hematocrit in the first days in cases with severe admission deficit, suggesting that these cases are more liable to dehydration in the immediate postadmission period. In our study we confirm the relation between higher hematocrit and hypertension reported in other studies.

Our main conclusion is that hematocrit does not influence the short-term outcome of cerebral infarction cases.

Acknowledgment
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References

KEY WORDS • cerebral infarction outcome • hematocrit
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