reasons: (1) Hemorrhagic infarctions may sometimes form a massive hematoma; (2) The patient had no other apparent predisposing factors responsible for the lobar ICH; (3) The ICH in the parietal lobe was within the vascular territory of the affected artery; and (4) The coexisting right temporal lesion was also a hemorrhagic infarction within the same vascular territory. We suggest that cervical internal carotid dissection be recognized as a rare cause of lobar ICH.

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References

Red Blood Cell Deformability Related to Perfusion Pressure in Cerebral Infarction With and Without Hypertension

Patients with acute stroke are often hypertensive; however, some authorities do not recommend lowering blood pressure because of the risk of cerebral ischemia.1 The lowering of blood pressure leads to a decrease in perfusion pressure in the cerebral microvascular bed, with disturbance of the passage of red blood cells (RBCs) through capillaries. It has been suggested that RBC passage may be disturbed more when there is a decrease in RBC deformability.2 We have studied RBC deformability modeling different levels of perfusion pressure in patients with acute brain infarction.

Forty-eight patients (27 men aged 45 to 60 years) with hemispheric infarction were observed during 3 days after the onset of symptoms. Patients were excluded if there was a major life-threatening illness that might interfere with survival (eg, diabetes, myocardial infarction, renal failure, or malignant hypertension) or evidence of cerebral hemorrhage or brain tumor. All patients had atherosclerosis, and 30 had had arterial hypertension for 5 to 15 years. Systolic arterial pressure ranged from 160 to 180 mm Hg and diastolic pressure from 90 to 115 mm Hg.

Red cell deformability was determined by the method of Reid et al.3 Washed red cells suspended in phosphate buffer saline (hematocrit, 0.08) were passed through a 5-µm filter under a perfusion pressure of 1 to 60 cm H2O at a temperature of 37°C. The results were expressed as a filtration rate (FR), calculated as the volume (in milliliters) of RCBs passing through the filter in 1 minute. Normal FR levels were measured in a control group of 30 healthy volunteers (17 men aged 42 to 60 years) without a history of diseases associated with increased risk for cerebrovascular events.

In healthy subjects, the FR measurement at different pressures showed that above 8 cm H2O, FR remained practically independent of the pressure. Below this value, FR decreased sharply (Figure). In hypertensive patients with brain infarction, FR decreased below 20 cm H2O; in nonhypertensive patients, the decrease in FR appeared below 12 cm H2O (Figure). These changes resulted in differences of RBC filterability at low and high levels of the perfusion pressure between healthy subjects and patients with brain infarction. At 40 cm H2O, FR in the hypertensive and nonhypertensive patients was less than that in control subjects by 18.1±1.2% and 11.7±1.1%, respectively (P<0.05).

Filtration rate (FR, ml/min) of red blood cells depending on perfusion pressure (cm H2O) in healthy subjects (1) and patients with brain infarction without (2) and with (3) hypertension.
However, at 8 cm H₂O, these differences were 68.4±2.1% and 23.4±1.6%, respectively (P<.01).

The perfusion pressure in the cerebral capillaries combined with arterial blood pressure creates shear stresses that deform RBCs as they traverse microvessels. Our results show that in cerebral infarction, especially in hypertensive patients, RBCs are more sensitive to lowered shear stresses. Hence, hypotension in acute stroke might induce microcirculatory disorders because capillaries can be wedged by RBCs. It is likely that measurement of the RBC filterability related to perfusion pressure would be helpful for choosing patients who are at risk for cerebral ischemia during lowering of blood pressure. In these patients antihypertensive therapy should be used with caution.

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Blood Pressure Changes After Stroke: Abolishing the White-Coat Effect

Carlsson and Britton¹ report that blood pressure (BP) increases in the majority of stroke patients 1 month after discharge from the hospital and suggest that this is due to increased activity out of the hospital setting. In a previous paper,² they also reported that BP falls in the days immediately after a stroke. However, it is not known to what extent these BP changes are due to an alerting reaction, or “white-coat effect,” and how much to a true change in BP. Although a large number of subjects will increase the power of a study to show a significant difference in BP between two periods, it will not necessarily attenuate the white-coat effect—a common phenomenon.³ Reduction or abolition of this effect can be achieved by recording multiple BP readings, preferably taken by a trained nurse rather than a physician, or by using 24-hour noninvasive automatic ambulatory BP monitoring.

Using the latter method, in 33 conscious subjects (mean age, 77 years) admitted to the hospital with an acute flaccid hemiparesis, we found that mean 24-hour systolic BP fell by 7 mm Hg (95% confidence interval [CI], 0 to 14 mm Hg; P<.05) and mean 24-hour diastolic BP by 3 mm Hg (95% CI, 0 to 6 mm Hg; P<.05) from day 1 to day 6, whereas there was no fall in mean 24-hour BP seen in an in-patient, nonstroke control group of 21 subjects. A subgroup of 11 stroke subjects underwent 24-hour BP monitoring at home 6±3 months after stroke. They exhibited a fall in BP while in the hospital (seen also in the whole stroke group) but there was no further change in mean 24-hour BP from day 6 to month 6 (day 1, 152±16/84±14 mm Hg; day 6, 137±17/79±13 mm Hg; month 6, 138±16/78±11 mm Hg).

Eight subjects died within 9 months of follow-up; their BP was higher at days 1 and 6 than those who survived this period (day 1, 162±23/94±13 mm Hg; day 6, 153±35/91±19 mm Hg versus day 1, 144±18/81±12 mm Hg; day 6, 137±16/78±9 mm Hg, P<.05). Furthermore, the “nonsurvivors” had higher nighttime than daytime BP readings on day 1 that increased significantly (P<.01) at day 6 (day-night BP difference: day 1, -4.2±15.4/-3.2±11; day 6, -14.0±11.1/-9.5±7.0 mm Hg). In contrast, those who survived this early period had a significant nocturnal fall in diastolic BP (P<.01) at day 6 (day-night systolic BP difference of 1.9±10.5 mm Hg and diastolic BP difference of 5.9±8.1 mm Hg).

Although the numbers reported are small, the ambulatory BP monitoring allowed an average of 50 BP readings to be obtained during each 24-hour period, reducing intrasubject BP variability compared with using the mean of only two BP readings.⁴,⁵ A further advantage of 24-hour BP monitoring is the ability to examine changes in daytime and nighttime BP. In contrast to Carlsson and Britton, who found no significant difference in mean BP at day 4 between patients who died and the remainder, we did note (albeit on a small sample) higher mean 24-hour BP levels and higher nighttime-to-daytime levels in the stroke patients who died early compared with those who survived to 6 months.

The increase in BP immediately after stroke, which settles within the first few days, appears not to be due solely to hospitalization and the white-coat effect but also to the effect of the stroke itself on BP regulation. However, much of the increase in BP recorded by two supine measurements 1 month after discharge from the hospital reported by Carlsson and Britton may be due to this alerting reaction. Patients with initially high BP levels at this time would, as usual, have to undergo repeated BP measurements over a period of weeks or months to ascertain their “usual” BP level and the possible need for antihypertensive treatment.

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References

Response
We would like to address the interesting comments by Dr Fotherby and colleagues on our studies on the BP course in stroke patients. As we understand, their main point was how to separate the white-coat effect from true blood pressure changes. In the acute stage of stroke all the BP recordings were made by specially trained nurses. The same procedure was used for the age- and sex-matched acute surgical patients. BP in patients with stroke decreased rapidly, with the greatest decline occurring in those with the highest BP. BP in control patients also decreased after admission but was lower than that in stroke patients all the time. It does not seem possible to explain the changes or the differences between the groups by a white-coat effect because this should have had a similar impact on all the patients on the various days.

When it comes to the poststroke period we tried to minimize the white-coat effect by having the same nurse measure the BP twice in the lying as well as in the standing position at all the checkups. When BP on the day of discharge was compared with that at 3 months, two checkups at which only this nurse saw the patients, there was still an increase in the BP level. However, this was
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Stroke. 1993;24:1421-1422
doi: 10.1161/01.STR.24.9.1421

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