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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References

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±17/78±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±17/85±9 mm Hg, respectively). 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±11.3; 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.3,4 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.1-3 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.1 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.2 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
Blood pressure changes after stroke: abolishing the white-coat effect.
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