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# CME Detrimental effect of blood pressure reduction in the first 24 hours of acute stroke onset

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**Abstract**—*Background:* High blood pressure, although the main modifiable risk factor for stroke, may have a beneficial effect in maintaining brain perfusion after acute ischemia. The authors assessed the effects of blood pressure variation in the first 24 hours of stroke onset. *Methods:* The authors prospectively studied consecutive patients admitted in the first 24 hours after stroke onset. Patients were classified according to the NIH Stroke Scale (NIHSS) and Oxfordshire Stroke Classification Scale (OSCS). Stroke etiology was defined according to Trial of ORG 10172 in Acute Stroke Treatment classification. After 3 months, outcome was assessed using Rankin and Barthel scales, with poor outcome defined as Rankin score > 2 or Barthel score < 70. *Results:* A total of 115 patients were admitted between January 2001 and October 2002. Median NIHSS was 4.5; main stroke etiology was cardioembolism (30%). After 3 months, 44 (39%) patients had a poor outcome. Predictors of poor outcome in univariable analyses ( $p < 0.05$ ) were as follows: total anterior circulation classification on OSCS, nonlacunar stroke etiology, older age, higher NIHSS score, lack of antiplatelet use, higher body temperature, lower diastolic blood pressure on admission, and a larger degree of systolic blood pressure reduction. In the multivariable analysis, remaining predictors of poor outcome included the following: NIHSS score (OR = 1.55 per 1 point increase; 95% CI = 1.28 to 1.87;  $p < 0.001$ ) and degree of systolic blood pressure reduction in the first 24 hours (OR = 1.89 per 10% decrease; 95% CI = 1.02 to 3.52;  $p = 0.047$ ). *Conclusion:* Blood pressure reduction in the first 24 hours of stroke onset is independently associated with poor outcome after 3 months.

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Hypertension is the main modifiable risk factor for stroke. It is well established that blood pressure (BP) control decreases the risk of stroke recurrence.<sup>1–3</sup> However, in the acute phase, BP reduction may worsen an already compromised perfusion in brain tissue that is still viable. Based on this concern, some have recommended not lowering BP in the first few days after a stroke.<sup>4</sup> Others have questioned this approach based on recent data favoring BP lowering.<sup>5</sup> Still others have tried pharmacologically increasing BP in the acute setting in carefully selected patients, especially those with multiple intracranial or extracranial arterial stenoses.<sup>6</sup>

In the first 24 hours, two additional factors weigh in favor of leaving BP untreated. On one side, risk of stroke recurrence is known to be low (3 to 4% in the first 2 weeks),<sup>7</sup> decreasing any possible beneficial effect of acutely lowering BP. On the other side, risk of stroke progression is high (20 to 30%) and has been shown to increase with lower admission BP.<sup>8</sup> Despite these considerations, it remains common in clinical practice to use antihypertensive medications acutely.

We studied the contribution of BP variation in the first 24 hours of stroke onset in predicting patient outcome.

**Patients and methods.** We prospectively studied consecutive patients admitted to Hospital Sao Rafael, a major urban hospital in Salvador, Brazil, between January 2001 and October 2002 with a clinical diagnosis of stroke. Only patients with symptom onset within 24 hours of admission were included in this study. One of four authors (J.O.-F., S.C.S., C.C.T., or B.B.P.), all board-certified neurologists with training in application of stroke scales, evaluated each patient on admission and initiated data collection. All patients underwent brain imaging (CT in all cases, MRI in most), 12-lead EKG, chest radiographic study, and laboratory tests and were admitted into a general semi-intensive or intensive care unit for at least 24 hours. During this period, vital signs were measured at least every 2 hours, including noninvasive cuff BP, pulse rate, and body temperature. Medications were given at the discretion of physicians not involved in the study, who in general followed the recommendations of a written set of emergency room protocols that recommended captopril as first-line therapy for BP above 220/120 mm Hg.

After informed consent, we used a standardized questionnaire to record the following data on admission: time and mode of symptom onset (classified as maximum deficit at onset, progressive deficit, or fluctuating deficit), NIH Stroke Scale (NIHSS) score, premorbid and admission Rankin scale score, Oxfordshire Stroke

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Classification Scale (OSCS), and previous cerebrovascular risk factors (hypertension, diabetes mellitus, atrial fibrillation, coronary heart disease, hypercholesterolemia, peripheral vascular disease, TIA, tobacco use in the previous 5 years, and stroke). In the first 24 hours, we recorded medications used, maximum body temperature, maximum blood glucose, and values of systolic BP. Degree of systolic BP reduction was calculated using the formula  $100 \times (\text{hSBP} - \text{lSBP}) / \text{hSBP}$ , where hSBP was the highest value of systolic BP and lSBP was the lowest value of systolic BP during the first 24 hours. Diastolic BP variation was not recorded prospectively. During the hospital stay, we recorded diagnostic test results and Rankin scale on discharge, and classified stroke etiology according to the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) scale.

After 3 months, one of the authors (E.U.S.) not involved in patient care during hospitalization and blinded to all collected data applied Rankin and Barthel scales through telephone interviews. We defined poor outcome as either a Rankin score  $> 2$  or Barthel index  $< 70$ . For patients for whom telephone interview or other access was not possible, we considered discharge Rankin scores as the final outcome. The study was approved by the local institutional review board.

For univariable analysis, continuous variables were compared through Student *t*-test and categorical variables through Fisher exact test. Variables with a significant trend in univariable analysis ( $p < 0.1$ ) were included in a stepwise logistic regression model having patient outcome as the dependent variable, dichotomized into good or poor outcome. In a post hoc analysis, factors involved in the degree of SBP reduction were also examined through simple linear regression. We used STATISTICA for Windows (StatSoft, Inc.) for all data analysis. Unless otherwise specified, continuous variables were expressed as mean  $\pm$  SD. No formal sample size calculations were performed.

**Results.** From January 2001 to October 2002, 150 patients were admitted with acute ischemic stroke. The 115 patients who arrived within 24 hours of stroke onset were included in this study. Mean time from stroke onset to hospital admission was 10 hours and 8 minutes ( $\pm 7$  hours and 36 minutes). Baseline characteristics, distribution of arterial territory, and stroke etiology are presented in table 1. Most patients were elderly men, with a median NIHSS score of 4.5 (range 1 to 30).

Systolic/diastolic BP on admission was  $160/94 \pm 34/17$  mm Hg, range 100/60 to 260/170 mm Hg. Over the first 24 hours, the systolic BP decreased by  $28 \pm 11\%$ . All patients had a BP drop in the first 24 hours, either spontaneously or by antihypertensive medication use. Overall, 66 (59%) patients received antihypertensive medication during the first 24 hours, which was either captopril (66 patients) or clonidine (5 patients). Although BP was slightly higher in patients receiving antihypertensive medications (166/97 vs 154/93 mm Hg), this difference was not significant ( $p > 0.1$  for both systolic and diastolic BP). The degree of SBP reduction was unrelated to the use of antihypertensive medications, but was fairly correlated to higher admission SBP ( $r = 0.29$ ,  $p = 0.003$ ).

By the end of 3 months, 93 (81%) patients were available for telephone interviews, the remainder being estimated based on discharge Rankin scores. Three patients who were transferred from the emergency room to outside hospitals were excluded from the analysis due to lack of follow-up data. Forty-four (39%) patients had a poor outcome. Results of univariable analysis are presented in table 2. Variables with a significant ( $p < 0.05$ ) association to poor outcome included the following: older age, higher NIHSS score, higher body temperature over the first 24 hours, total anterior circulation strokes (OSCS classification), nonlacunar strokes (TOAST classification), lack of antiplatelet use in the first 24 hours, lower diastolic BP on

**Table 1** Baseline characteristics of 115 patients with acute stroke ( $< 24$  h)

Characteristics	Values
<b>Demographics</b>	
Age, y, mean $\pm$ SD	64 $\pm$ 14
Male/female	70/45
NIHSS score, median (range)	4.5 (1–30)
<b>Oxfordshire Stroke Classification Scale, n (%)</b>	
Total anterior circulation	15 (13)
Partial anterior circulation	54 (47)
Lacunar	22 (19)
Posterior circulation	24 (21)
<b>TOAST Scale, n (%)</b>	
Cardioembolic	34 (30)
Small-artery atherosclerosis	30 (26)
Large-artery atherosclerosis	17 (15)
Other known etiologies	5 (4)
Unknown etiology	29 (25)
<b>Clinical course over first 24 h, n (%)</b>	
Maximum deficit at onset	87 (76)
Progressive deficit	25 (22)
Fluctuating deficit	3 (2)

NIHSS = NIH Stroke Scale; TOAST = Trial of Org 10172 in Acute Stroke Treatment.

admission, and a larger decrease in systolic BP over the first 24 hours. Other stroke etiologies (e.g., cardioembolic, large artery atherosclerosis), other arterial territories, and antihypertensive medication use in the first 24 hours were not significantly associated with outcome. A comparison between the 93 patients available for the 3-month follow-up and the remaining 22 patients showed a similar distribution of variables (i.e.,  $p > 0.1$  for age, NIHSS score, admission BP, degree of SBP reduction, body temperature, OSCS classification, TOAST classification, and antiplatelet use).

Results of the multivariable analysis are presented in table 3. The only variables that remained significant predictors of poor outcome were NIHSS score (OR = 1.55 per point increase; 95% CI = 1.28 to 1.87;  $p < 0.001$ ) and the degree of SBP reduction (OR = 1.89 per 10% decrease; 95% CI = 1.02 to 3.52;  $p = 0.047$ ). No interactions were detected between a lower baseline DBP and outcome. The relationship between BP variables and outcome is presented in the figure.

**Discussion.** Previous studies have shown that hypertension is a powerful, modifiable, and independent risk factor for stroke, and that lowering BP decreases the risk of recurrence.<sup>1-3,9-12</sup> Results from a recent study provided solid evidence that reducing BP by only 12/5 mm Hg reduced stroke risk by 43%.<sup>10</sup> However, because patients were not treated within the first week of stroke onset in these studies, whether to reduce BP in the acute phase remains a controversy.

**Table 2** Univariable predictors of outcome in the first 24 h after stroke onset

Characteristics	Good outcome, n = 68	Poor outcome, n = 44*	p Value
Age, y, mean ± SD	61 ± 13	70 ± 13	<0.001
Admission NIHSS score, median	4	14	<0.001
Admission SBP, mm Hg, mean ± SD	160 ± 33	159 ± 35	NS
Admission DBP, mm Hg, mean ± SD	96 ± 19	89 ± 13	0.04
Maximum temperature, °C, mean ± SD†	36.7 ± 0.5	37.2 ± 0.8	<0.001
Maximum blood glucose, mg/dL, mean ± SD†	181 ± 86	183 ± 89	NS
SBP variation, %, mean ± SD†	26 ± 10	31 ± 12	0.04
Male sex, n (%)	45 (66)	22 (50)	NS
Antihypertensive medication use, n (%)†	44 (65)	22 (50)	NS
Total anterior circulation stroke, n (%)†	1 (1)	14 (32)	<0.001
Heparin/low-molecular weight heparin, n (%)†	5 (7)	4 (9)	NS
Aspirin, n (%)†	39 (57)	16 (36)	0.048
Small-artery atherosclerosis, n (%)	25 (37)	4 (9)	0.002
Previous risk factors, n (%)			
Hypertension	49 (72)	38 (86)	NS
Diabetes	26 (38)	19 (43)	NS
Smoking	12 (18)	3 (7)	NS
Atrial fibrillation	5 (7)	7 (16)	NS
Coronary heart disease	8 (12)	7 (16)	NS
Cholesterol >200 mg/dL	16 (24)	11 (25)	NS
Peripheral vascular disease	4 (6)	3 (7)	NS
Previous stroke	14 (21)	12 (27)	NS

\* Poor outcome was defined by Rankin scale score >2 or Barthel index <70 at 3 months after stroke onset.

† Data collected over the first 24 h.

NIHSS = NIH Stroke Scale; SBP = systolic blood pressure; DBP = diastolic blood pressure.

The main result of our study was a strong, independent relationship of BP course to adverse outcome, with an almost twofold increased risk of poor outcome for every 10% decrease in the systolic BP over the first 24 hours. The actual admission DBP was lower in patients with a poor outcome; however, this did not remain significant in the multivariable analysis. Although most patients (59%) received antihypertensive medications, this factor did not affect outcome. Moreover, the degree of SBP reduction was unrelated to antihypertensive medication use, but related to higher admission BP. It seems, therefore, that even a sponta-

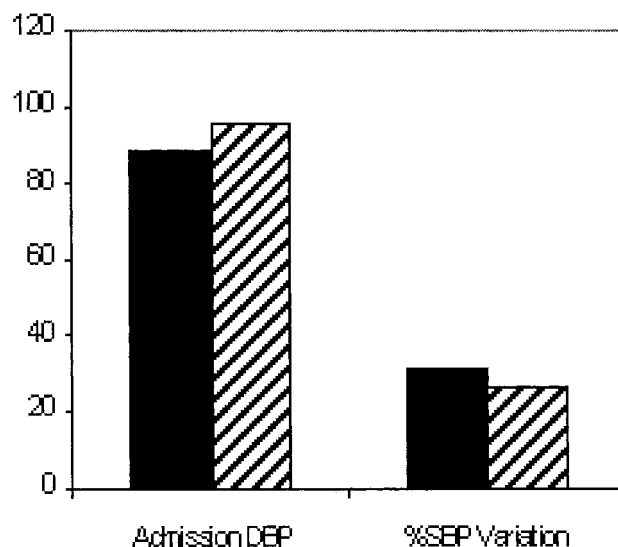
**Table 3** Multivariable predictors of stroke outcome in the first 24 h of stroke onset (stepwise logistic regression analysis)

Characteristics	OR	95% CI	p Value
Age	1.51 per 10-year increase	0.87–2.64	0.15
NIHSS score	1.55 per 1-point increase	1.28–1.87	<0.001
Degree of SBP reduction	1.89 per 10% decrease	1.02–3.52	0.047

NIHSS = NIH Stroke Scale; SBP = systolic blood pressure.

neous decrease in BP may be harmful to ischemic brain tissue. However, we cannot rule out that further decrease of BP with antihypertensive medications may be harmful in this setting. This is an important finding, as neurologists are frequently asked to opine on ideal BP levels after a stroke.

Previous larger studies examining the effects of BP in stroke have usually recorded single values of BP on admission. Some population studies have demonstrated a J-curve phenomenon, where extreme blood pressures (both high and low) are associated with adverse outcome.<sup>13</sup> A recent analysis of the International Stroke Trial cohort confirmed this relationship, suggesting an “ideal” SBP on admission of 150 mm Hg.<sup>12</sup> One study demonstrated that lower BP on admission were more frequently seen in patients who worsened over the first 24 hours, but did not measure BP course over the first 24 hours.<sup>8</sup> In a multicenter trial of IV nimodipine in acute stroke, an adverse effect of nimodipine was attributed to a decrease in BP over the first few days after stroke



**Figure.** Relationship between blood pressure variables and stroke outcome. Full box = group with poor outcome; box with grids = group with good outcome; DBP = diastolic blood pressure; %SBP variation = degree of systolic blood pressure variation (reduction) in the first 24 hours after admission. Comparisons between the two groups were significant with  $p < 0.05$ .

onset.<sup>14</sup> This study showed a similar increase in the odds of death or dependency in patients with >20% decrease in DBP, although with wide confidence intervals (OR = 10.16; 95% CI = 1.02 to 101.74).<sup>14</sup> More recently, a retrospective analysis of multicenter trial data was published in abstract form showing that higher admission BP was associated with good outcome<sup>15</sup>; however, extreme BP increases over the first 60 hours were associated with poor outcome (unpublished data presented at the 28th International Stroke Conference). In our study we included all patients presenting with an acute stroke, collected data prospectively, and had predefined endpoints, as opposed to a more restricted population in a stroke intervention trial (extreme BP was an exclusion criterion in that particular trial<sup>15</sup>), which may explain some of the discrepancy in the results.

In support of our results, several investigators have examined the effect of pharmacologically increasing BP in acute stroke. Experimentally, increasing BP improves blood flow to the ischemic area and decreases final infarct size.<sup>16-18</sup> Case reports document that carefully selected patients experience an improvement in clinical deficit after increasing their BP.<sup>19,20</sup> In one study, a subset of patients with intracranial or extracranial stenoses appeared to benefit the most after induced hypertension.<sup>6</sup> Our data do not answer the question whether to treat BP in the acute setting. However, current data set the stage for an acute intervention trial randomizing patients to predefined BP values.

Our study did not show a significant relationship between other variables and stroke outcome. This is probably due to lack of power to demonstrate such an effect. Not surprisingly, the NIHSS score was the most significant predictor of outcome and probably masked all other variables. Patients with more severe strokes on admission (higher NIHSS scores) will frequently present with total anterior circulation strokes, nonlacunar strokes, and higher body temperatures, which would explain the lack of sufficient predictive power in a multivariable model. Conversely, the degree of SBP variation over the first 24 hours was unrelated to stroke severity but robustly related to stroke outcome.

Antihypertensive medication use was unrelated to initial BP levels. This was an unexpected finding, because our written recommendation specifies only treating a BP level above 220/120 mm Hg, and probably reflects a type II error to demonstrate such a difference. An alternative explanation would be the current lack of level I evidence favoring a fixed BP threshold in the acute phase of stroke reducing compliance with local guidelines. Noncompliance with written guidelines regarding BP control in stroke was also found in over half of emergency room physicians in one local study.<sup>21</sup>

In this study we chose to record only SBP variation over the first 24 hours based on previous studies suggesting a more important relationship between SBP—as opposed to DBP—and outcome in stroke.<sup>2,11</sup>

As occurs with any single-center study, it is possible that in other populations the contribution of BP variables will vary. Because of the excess number of variables entered into the multivariable model for the number of least frequent outcomes, it will be important to confirm our results in other data sets with different patient populations. However, we believe that our population is similar to others in regards to the distribution of stroke etiologies, arterial territories, and stroke severity, which would make the results more generally applicable. BP in this study was not measured continuously and most patients arrived beyond a 3-hour time window. However, we expect that the contribution of BP variables will be even more important at earlier time points, but cannot prove this based on this study. Finally, in this study there was a proportion of patients lost to follow-up (19%) who had their discharge data carried forward to 3 months. We do not believe this has affected our results based on similar baseline characteristics of patients lost to follow-up as compared to the remaining population.

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# Prognostic value of Pulsatility Index in acute intracerebral hemorrhage

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**Abstract—Objective:** To investigate whether data obtained by transcranial Doppler (TCD) have prognostic value in patients with intracerebral hemorrhage (ICH). **Methods:** A prospective study of patients with an acute (<12 hours from onset of symptoms) spontaneous supratentorial ICH was conducted. Mortality was assessed at 30-day follow-up. TCD parameters were obtained from both middle cerebral arteries: systolic, diastolic, and mean velocities and Pulsatility Index (PI) from the affected and unaffected hemispheres. The following variables were included in a univariate analysis: age, sex, hematoma volume, hypodense volume around the hematoma, total volume, midline shift, ventricular size, Glasgow Coma Scale score, intraventricular hemorrhage, body temperature, white cell count, blood glucose, mean blood pressure, and TCD data. A multivariate analysis was performed with variables that showed significance in the univariate analysis. Receiver-operator characteristic (ROC) curves were obtained. **Results:** Forty-eight patients (age  $66.5 \pm 12.5$  years; 28 men) were studied. Mortality at 30 days was 31%. The only predictor of mortality was the Glasgow Coma Scale score (odds ratio [OR] 0.67, CI 0.53 to 0.84,  $p = 0.001$ ), whereas the PI from the unaffected hemisphere was correlated with mortality (OR 2.3, CI 0.92 to 5.72,  $p = 0.07$ ). The area under the ROC curve was 0.92. A cutoff for PI from the unaffected hemisphere of 1.75 showed a specificity of 94% and a sensitivity of 80% as a predictor of death at 30 days. **Conclusions:** The PI of the unaffected hemisphere may be a predictor of death in acute ICH. These findings suggest that intracranial hypertension is the most likely cause of death in most patients with ICH.

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The prognosis of patients with spontaneous intracerebral hemorrhage (ICH) is generally poor. Within the first 30 days, 35 to 52% of patients die, and only about 20% are independent 6 months after onset.<sup>1,2</sup> Mortality in the first hours or days after an ICH is attributable to increased intracranial pressure (ICP) and tissue shifts, whereas later death is usually caused by complications related to immobility.<sup>3–5</sup> Most studies agree with these observations and consistently show that the volume of the hematoma and the level of consciousness at admission are the main predictors of survival.<sup>1,2,6</sup> These two predictors are indirectly related to increased ICP either as a cause (volume of the hematoma) or as a consequence (level of consciousness).

Transcranial Doppler (TCD) is used increasingly as an indirect measure of ICP, because increased

ICP causes characteristic changes in the Doppler waveform, that is, decreased end-diastolic flow velocity (DFV) and increased Pulsatility Index (PI).<sup>7</sup> Several studies have confirmed that in patients with severe brain injuries, the PI correlates with the ICP.<sup>7–9</sup> However, there is no information about the prognostic value of TCD during the acute stage of ICH. Therefore, we prospectively evaluated accepted prognostic indicators of ICH, along with TCD measures, in a group of patients with acute nontraumatic ICH. Our hypothesis was that increased PI and decreased DFV are independent predictors of survival in patients with ICH.

**Methods. Patients.** We prospectively included patients of any age with a first-ever single supratentorial nontraumatic ICH, diagnosed by CT up to 12 hours after the onset of symptoms. Patients in a coma or with signs of brainstem dysfunction were

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