Blood Pressure Decrease During the Acute Phase of Ischemic Stroke Is Associated With Brain Injury and Poor Stroke Outcome

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Background and Purpose—Studies on the relation between blood pressure (BP) and stroke outcome have shown contradictory results. We explored the association of systolic (SBP) and diastolic (DBP) BP during acute stroke with early neurological deterioration, infarct volume, neurological outcome, and mortality at 3 months.

Methods—We included 304 patients with acute ischemic stroke. SBP and DBP on admission and on the first day were the average values of all readings obtained in the emergency department and during a 24-hour period after patient allocation in the stroke unit.

Results—A U-shaped effect was observed: for every 10 mm Hg ≤180 mm Hg of SBP, the risk of early neurological deterioration, poor outcome, and mortality increased by 6%, 25%, and 7%, respectively, whereas for every 10 mm Hg >180 mm Hg, the risk of early neurological deterioration increased by 40% and the risk of poor outcome increased by 23%, with no effect on mortality. Mean infarct volume increased 7.3 and 5.5 cm^3 for every 10 mm Hg ≤180 and >180 mm Hg. A similar pattern was found in patients with DBP ≤100 or >100 mm Hg. These effects disappeared after adjustment for the use of antihypertensive drugs and BP drop >20 mm Hg within the first day, with the latter being the more important prognostic factor of poor outcome.

Conclusions—High and low SBP and DBP, as well as a relevant drop in BP, are associated with poor prognosis in patients with ischemic stroke. **(*Stroke*. 2004;35:520-527.)**

Key Words: blood pressure ■ cerebral infarction ■ hypertension ■ prognosis ■ stroke

There is no general agreement regarding how blood pressure (BP) should be managed in the acute phase of ischemic stroke.[1,2] Current opinions vary from not to treat[1] to treat if systolic BP (SBP) is >220 mm Hg or diastolic BP (DBP) is >120 mm Hg, although the recommended cutoff values for treatment are lower in patients receiving tissue plasminogen activator.[3] Antihypertensive drugs may reduce the pressure-dependent cerebral blood flow to the ischemic penumbra, or conversely, poststroke hypertension may be deleterious and facilitate edema formation in the ischemic tissue.[4] According to these opposite effects in experimental works, recent clinical studies have obtained contradictory results regarding the influence of BP on stroke prognosis.[5,6]

In this observational study, we explored the association of arterial BP during the acute phase of cerebral ischemia with early neurological deterioration, ultimate infarct volume, neurological outcome, and mortality at 3 months.

Subjects and Methods

We studied 304 patients of a total of 352 admitted consecutively for a first episode of hemispheric ischemic stroke within 24 hours from symptom onset. Patients without a confirmed diagnosis of cerebral infarct (n=13), treated in an acute clinical trial (n=32), or with vasoactive amines (n=3) were excluded. The study was approved by an ethics committee, and informed consent was obtained from all patients or relatives.

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At admission, blood samples were taken, and a cranial CT was immediately performed to evaluate the presence of early signs of cerebral infarction or focal edema (obscuration of the lenticular nucleus, cortical or subcortical hypodensity, and mass effect on the midline structures).

BP on admission was considered the average of all readings (median, 2; range, 1 to 6) obtained in the emergency department (ED) before the administration of any antihypertensive drug. A single reading was taken in only 6 patients. BP on the first day was considered the average of the values obtained every 4 hours during a period of 24 hours after admission in the acute stroke unit or after treatment of BP if it was started in the ED. The absolute difference between SBP or DBP on admission and on the first day was calculated for each patient. In all cases, BP was determined with the patient recumbent and with a mercury sphygmomanometer.

Sixty-seven patients were treated with angiotensin-converting enzyme inhibitors (n=16), β-blockers (n=9), diuretics (n=18),

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nitroprusside (n=7), or a combination of 2 drugs (n=17) by the physicians in the ED without following specific guidelines for management of BP. After admission in the acute stroke unit, BP was treated in 31 patients with intravenous labetalol or angiotensin-converting enzyme inhibitors because of SBP >220 mm Hg or DBP >120 mm Hg, in accordance with published guidelines.[3,7] No patients received thrombolytic drugs. Intravenous heparin was administered mainly in patients with a major cardioembolic source, and activated partial thromboplastin time was maintained at <2.0 times the control value.

Stroke severity was measured at the time of inclusion, at 48±6 hours, and at 90±15 days with the use of the Canadian Stroke Scale (CSS)[8] administered by an appropriately trained neurologist. We equalized CSS score to zero in those patients who were dead at the time of a particular evaluation.

Infarct volume was calculated on a CT scan performed between days 4 and 7 by using the formula $0.5\times a\times b\times c$, where a and b=larger perpendicular diameters of the zone of hypodensity and c=number of sections in which the infarct could be seen. All CT evaluations were made by the same neuroradiologist, who was blind to the clinical results.

Three clinical outcome measures were evaluated: (1) early neurological deterioration, (2) neurological deficit at 3 months, and (3) mortality at 90 days. Early neurological deterioration was diagnosed when the CSS score dropped ≥1 point within the first 48 hours of hospitalization. Patients in whom the CSS was ≤7 points at 3 months were classified in the poor outcome group.

Statistical Analyses

The results are expressed as percentages for categorical variables and as mean±SD or median (quartiles) for continuous variables depending on whether or not they were normally distributed. Proportions were compared with the χ^2 test. The t test or Mann-Whitney and ANOVA/Kruskal-Wallis tests were used to compare 2 groups or ≥3 groups, respectively.

A U-shaped relationship was found between SBP and DBP and the 4 outcome variables of the study. For the purpose of this study, the cutoff values of SBP and DBP on admission were chosen according to the minimum of the quadratic polynomials fitted to the U-shaped relationship. In all outcome variables the minimum was approximately 180 mm Hg for SBP (from 178.4 to 184.9) and 100 mm Hg for DBP (from 96.6 to 104.4). Therefore, the cutoff levels were set at 180 and 100 mm Hg for SBP and DBP, respectively. The adjusted quadratic polynomials were all significant, and their adjusted R values ranged from 0.12 to 0.32.

The differences between SBP or DBP on admission and on the first day were categorized in 2 different ways: (1) reductions >20 mm Hg, between 0 and 20 mm Hg, or any increase in SBP or DBP; and (2) reductions >20 mm Hg, or any increase or reduction between 0 and 20 mm Hg. The former was used to assess the effect of BP reductions in people who had SBP or DBP on admission below the cutoff levels, and the latter was used to assess the same effect in people who had admission SBP or DBP above the cutoff levels, since only 1 patient among the group with admission SBP >180 mm Hg and 3 patients among the group with admission DBP >100 mm Hg showed an increase in SBP or DBP on the first day.

The influence of SBP and DBP on admission above or below the cutoff points, as well as the influence of use of antihypertensive drugs on admission and differences in SBP and DBP between admission and day 1 on early neurological deterioration, poor neurological outcome, and mortality were evaluated by logistic regression analysis. Likewise, to assess the influence of SBP and DBP over or under the cutoff levels on infarct volume, general factorial linear models were used. We used 3 different models to illustrate the changes in the odds of BP on admission for the 4 outcomes: model 1 was adjusted for factors and covariates that were related to prognostic variables in the univariate analyses, model 2 was further adjusted for antihypertensive treatment, and model 3 was further adjusted for difference in BP. We fitted the models in a customized way by means of the Enter method. Values of $P<0.05$ were considered statistically significant in all tests.

TABLE 1. Baseline Characteristics Recorded at Admission by Outcome Groups

	Total	Early Neurological Deterioration		Poor Outcome		Mortality	
	n=304	Yes (n=75)	No (n=225)	Yes (n=153)	No (n=105)	Yes (n=37)	No (n=221)
Age, y	71.8±9.0	71.1±9.3	72.0±8.4	72.6±8.2	71.6±7.7	72.1±8.9	72.2±7.8
Sex (M), %	52.3	45.3	54.2	51.6	56.2	51.4	53.8
History of high blood pressure, %	50.9	45.3	53.8	49.0	57.1	29.7	56.1*
History of diabetes, %	18.7	16.0	20.0	22.2	17.1	29.7	18.6
History of atrial fibrillation, %	37.8	44.0	35.6	46.4	26.7*	37.8	38.5
Admission delay, h	7 [4.5–9.5]	7 [4.5–10.5]	6.5 [4.6–9]	7 [4.5–10.2]	6.2 [5–8.3]	8 [4.5–12.2]	6.5 [4.6–9]
Body temperature, °C	36.8 [36.3–37.4]	37.5 [36.6–37.8]	36.8 [36.3–37.2]*	37.6 [37.1–37.9]	36.6 [36.3–36.9]*	37.8 [37.2–38.2]	36.8 [36.3–37.2]*
Serum glucose, mg/dL	138.5 [117–168]	143 [123–172]	136 [112–168]	143 [120–183]	133.5 [113–162]*	162 [134–201]	137 [112–168]*
Fibrinogen, mg/dL	355 [319–414]	356 [322–415]	347.5 [316–412]	354 [318–415]	349 [319–406]	362 [321–417]	349 [319–408]
SBP on admission, mm Hg	178.8±35.9	174.6±47.2	180.6±31.2	178.9±41.2	177.8±26.1	171.0±45.3	179.8±33.7
DPB on admission, mm Hg	96.9±23.4	95.7±29.9	97.6±21.2	96.8±26.0	96.4±19.1	91.8±27.7	97.5±22.6
Anticoagulant treatment, %	43.8	48	41.3	52.9	32.4*	51.4	43.4
Antihypertensive drugs, %	22.1	38.7	16.9*	30.1	9.5*	24.3	21.3
Admission CSS	5 [3.5–6]	5 [3.5–6]	5 [3.5–6]	4 [3–5]	6 [5–7]*	3.5 [3–5.2]	5 [3.5–6]*
Early CT signs, %	32.2	37.3	31.1	35.3	27.6	32.4	32.1
Diagnosis, %					*		
Atherothrombotic	35.9	37.3	36.0	39.2	35.2	37.8	37.6
Cardioembolic	37.5	46.7	34.7	47.7	26.7	35.2	39.8
Lacunar	17.4	9.3	20.4	3.3	34.3	10.8	16.7
Unknown etiology	9.2	6.7	8.9	9.8	3.8	16.2	5.9

*P value<0.05.

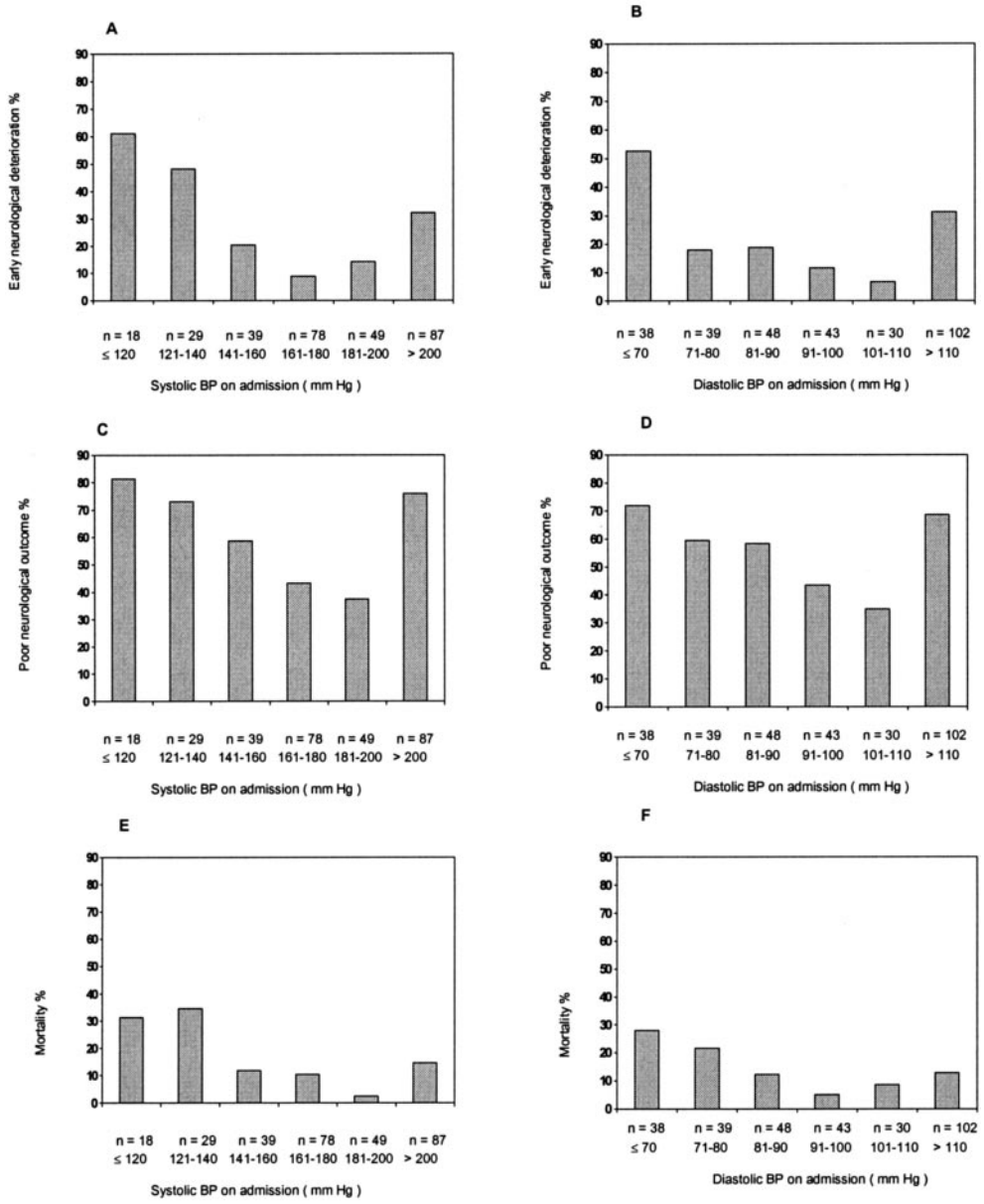

Figure 1. Proportions of early neurological deterioration (A and B), poor neurological outcome (C and D), and mortality (E and F) by SBP and DBP levels on admission.

Results

Early neurological deterioration was observed in 75 (25%) of the 300 patients in whom CSS score was evaluated at 48 hours (4 patients died within 48 hours after admission). After hospital discharge, 46 patients were lost to follow-up, and therefore 258 patients completed the study period. Mortality was recorded in 37 patients (14.3%) (8 patients had an early death, before the second CT was performed), and poor neurological outcome was recorded in 153 (59%). Baseline characteristics in each group are shown in Table 1.

SPB and DBP on admission were not related to the early and late outcome variables (Table 1). However, this fact was due to a U-shaped effect, since the group of patients with the highest or lowest values of SBP and DBP had a higher frequency of early neurological deterioration, poor outcome, and mortality at 3 months ($P<0.0001$) (Figure 1). Furthermore, infarct volume was greater in patients with the lowest and the highest SBP and DBP levels ($P<0.0001$) (Figure 2).

SBP during the first day was lower than SBP on admission in 233 patients. The mean absolute decrease was 8 ± 7 mm Hg in patients who did not receive antihypertensive treatment after admission (n=166) and 36 ± 22 mm Hg in those who were treated with hypotensive drugs (n=67) ($P<0.0001$). Similarly, DBP decreased in 249 patients during the first 24 hours, with the mean value being higher in those who received treatment (23 ± 12 versus 8 ± 7 mm Hg; $P<0.0001$). Reductions in SBP and DBP >20 mm Hg were associated with a higher frequency of early neurological deterioration, increased infarct volume, and poorer outcome at 3 months ($P<0.0001$) (Table 2). We did not record whether early

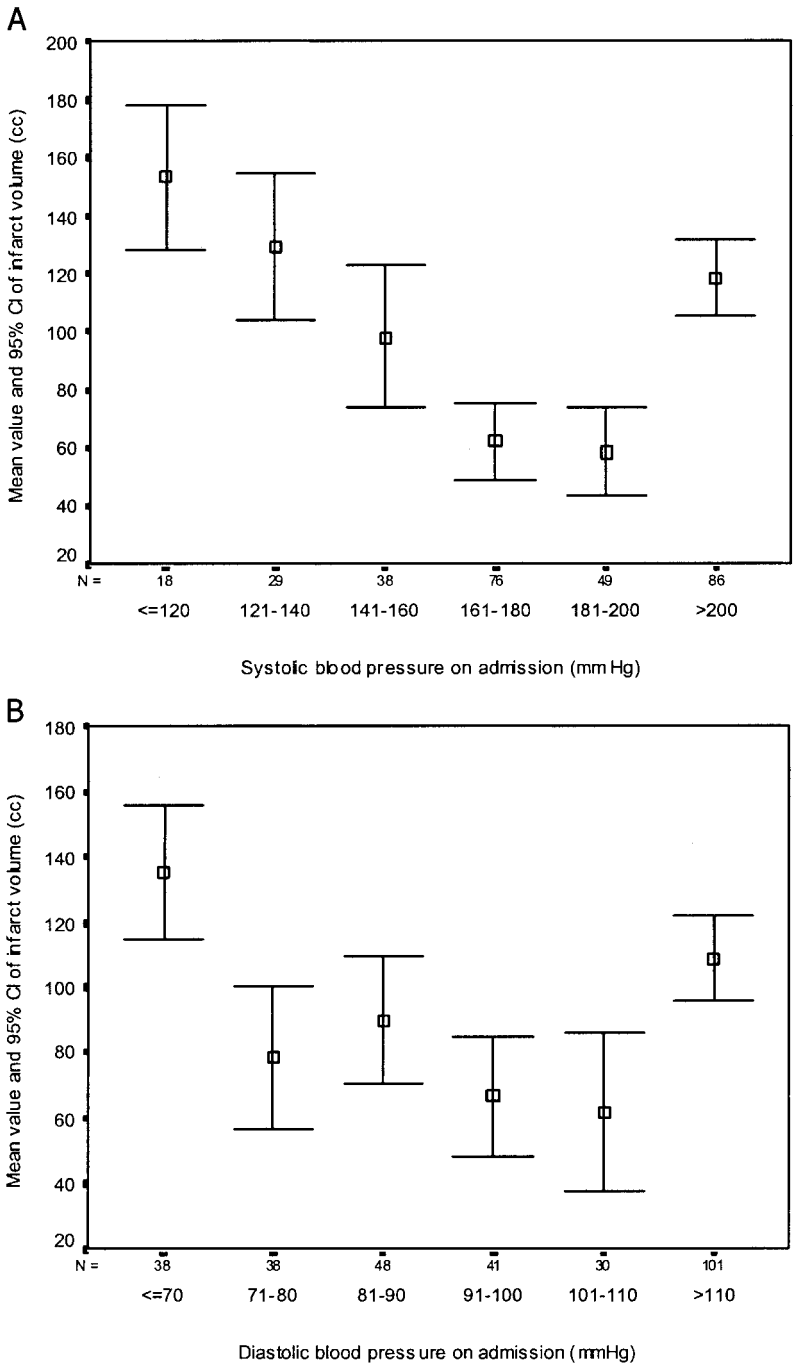

Figure 2. Mean value and 95% CIs of infarct volume on days 4 to 7 by SBP (A) and DBP (B) levels on admission.

neurological deterioration and BP drop occurred at the same time and whether there were other potential factors directly related to the BP decrease.

Logistic regression analyses showed that patients with SBP on admission ≤180 mm Hg had, for every 10 mm Hg below this cutoff value, a 6% (95% CI, 3% to 10%) increase in the risk of early neurological deterioration, a 25% (95% CI, 1% to 56%) increase in the risk of poor outcome, and a 7% (95% CI, −4% to 20%) increase in the risk of mortality (Table 3, model 1). No patient received antihypertensive drugs. When the change in SBP during the first day was taken into account (model 3), patients with a decrease in SBP >20 mm Hg showed a significantly increased odds of early neurological deterioration, poor neurological outcome, and mortality, while SBP values on admission lost significance. A similar pattern was found in patients with DBP ≤100 mm Hg (data not shown).

At SBP levels >180 mm Hg on admission, for every 10 mm Hg over this cutoff value the risk of early neurological deterioration increased by 40% (95% CI, 11% to 77%) and the risk of poor outcome increased by 23% (95% CI, 7% to 41%), while there was no influence on mortality (Table 3, model 1). Antihypertensive treatment increased the odds of early neurological deterioration by 5.3 (model 2), but this effect disappeared after adjustment for a fall in SBP >20 mm Hg during the first day; this variable was the most important factor associated with early neurological deteriora-

TABLE 2. Changes in Blood Pressure Between Admission and the First Day and Stroke Outcome*

	Systolic Blood Pressure				Diastolic Blood Pressure			
	Drop >20 mm Hg	Drop 0–20 mm Hg	Increase >0 mm Hg	*P* Value	Drop >20 mm Hg	Drop 0–20 mm Hg	Increase >0 mm Hg	*P* Value
Early neurological deterioration, %	54.4 (57)	13.6 (177)	30.3 (66)	<0.001	56.1 (50)	14.2 (197)	35.8 (53)	<0.001
Poor neurological outcome, %	90.2 (51)	49.0 (151)	57.4 (54)	<0.001	77.3 (44)	53.3 (167)	62.2 (45)	0.014
Mortality at 3 months, %	23.5 (51)	10.6 (151)	13.0 (54)	0.066	25.0 (44)	9.0 (167)	20.0 (45)	0.009
Volume of infarct, mean±SD, mL	133±66 (56)	77±60 (174)	108±73 (66)	<0.001	137±60 (49)	80±64 (195)	110±70 (52)	<0.001

Number of patients evaluated in each group is shown in parentheses.

*Changes in BP during the first day were not obtained in 2 patients who died within the first 24 hours.

tion and poor outcome (model 3). A similar effect was found in patients with DBP >100 mm Hg (data not shown).

Generalized linear models showed that patients with SBP on admission ≤180 mm Hg had, for every 10 mm Hg below this cutoff value, a 7.3-cm^3 increase in mean infarct volume (Table 4, model 1). This effect remained significant after adjustment for changes in SBP during the first day, but the harmful effect of a decrease in SBP >20 mm Hg was even higher and was associated with a 61-cm^3 increase in mean infarct volume (model 3). A similar mean infarct volume increase was found for every 10 mm Hg <100 mm Hg in DBP (data not shown). At SBP levels >180 mm Hg, for every 10 mm Hg over this cutoff value, there was a 5.5-cm^3 increase in mean infarct volume (Table 4, model 1). This effect lost significance after adjustment for the use of antihypertensive drugs (model 2) and decrease in SBP >20 mm Hg during the first day, with the latter being the most important factor associated with a 32-cm^3 increase in mean infarct volume (model 3).

Discussion

The present work confirms, in a series of consecutive and nonselected patients, that both high and low SBP or DBP values within the first 24 hours after stroke onset are associated with a poor prognosis in terms of early neurological deterioration, neurological deficit at 90 days, and infarct volume. This effect is independent of prognostic factors such as stroke severity, body temperature, serum glucose, and stroke subtype.

The prognostic influence of BP during the acute phase of ischemic stroke is still a matter of controversy. Several works have associated raised BP levels with a poor prognosis,[9,10] whereas others have found a relationship with good outcome[11,12] or no influence.[13] In our opinion, these opposite findings may be partially explained by a U-shaped relationship between BP levels and outcome measures. Recently, Leonardi-Bee et al[14] observed a U-shaped relationship between a single measure of SBP within the first 48 hours after stroke and both early death and late death or dependency among the International Stroke Trial population. The lowest frequency of poor outcome was found at approximately 150 mm Hg, although there was a plateau between 140 and 179 mm Hg. Because the prognostic value of an isolated BP recording in acute stroke has been questioned on account of the variability of BP readings,[10] we used the mean value of all

TABLE 3. Adjusted Odds Ratios (95% CI) of the Outcome Variables for SBP on Admission, Use of Antihypertensive Drugs, and Changes in SBP During the First Day

	Early Neurological Deterioration		Poor Neurological Outcome		Mortality	
	SBP ≤180 mm Hg (n=164)	SBP >180 mm Hg (n=136)	SBP ≤180 mm Hg (n=143)	SBP >180 mm Hg (n=115)	SBP ≤180 mm Hg (n=143)	SBP >180 mm Hg (n=115)
Model 1						
SBP, by 10 mm Hg	1.06 (1.03, 1.10)	1.40 (1.11, 1.77)‘	1.25 (1.01, 1.56)	1.23 (1.07, 1.41)	1.07 (0.96, 1.20)	0.87 (0.76, 1.00)
Model 2						
SBP, by 10 mm Hg	. . .	0.93 (0.89, 0.97)	. . .	1.17 (1.00, 1.37)	. . .	0.87 (0.76, 1.00)
Hypotensive drugs	. . .	5.27 (1.98, 14.05)	. . .	3.10 (0.90, 10.67)	. . .	1.62 (0.30, 8.63)
Model 3						
SBP, by 10 mm Hg	1.01 (0.95, 1.06)	0.92 (0.87, 0.97)	1.10 (0.96, 1.26)	1.15 (0.98, 1.34)	1.00 (0.88, 1.14)	0.87 (0.76, 1.00)
Hypotensive drugs	. . .	1.54 (0.43, 5.50)	. . .	1.23 (0.29, 5.01)	. . .	1.12 (0.09, 14.13)
Difference in SBP:						
Decrease 0–20 mm Hg	1	1	1	1	1	1
Decrease >20 mm Hg	12.74 (1.32, 122.22)	7.84 (2.36, 26.05)	61 (1.00, 3705)	8.79 (1.89, 55.62)	38.9 (4.2, 358)	1.56 (0.15, 15.40)
Any increase in SBP	0.49 (0.22, 1.12)	. . .	1.83 (0.72, 4.67)	. . .	0.72 (0.21, 2.43)	. . .

ORs for SBP on admission are expressed by 10 mm Hg difference under or above 180 mm Hg. END models were adjusted for stroke subtype (lacunar vs nonlacunar) and body temperature (<37.5°C vs ≥37.5°C). Poor neurological outcome models were adjusted for atrial fibrillation, stroke subtype, CSS, body temperature, serum glucose on admission, and anticoagulant treatment. Mortality models were adjusted for history of hypertension, CSS, body temperature, and serum glucose on admission.

TABLE 4. Adjusted Increase in Mean (95% CI) Infarct Volume for SBP on Admission, Use of Antihypertensive Drugs, and Differences in SBP During the First Day

	SBP ≤180 mm Hg (n=161)	R^2	SBP >180 mm Hg (n=135)	R^2
Model 1				
SBP, by 10 mm Hg	7.3 (4.0, 10.6)	0.89	5.5 (1.6, 9.4)	0.87
Model 2				
SBP, by 10 mm Hg	...		1.4 (−3.8, 6.7)	0.88
Hypotensive drugs	...		23.1 (28, 43.4)	
Model 3				
SBP, by 10 mm Hg	6.0 (2.5, 9.6)	0.89	−1.6 (−7.1, 3.7)	0.89
Hypotensive drugs	...		11.1 (−9.9, 32.1)	
Difference in SBP:				
Decrease 0–20 mm Hg	0		0	
Decrease >20 mm Hg	61 (13.3, 109.6)		32.2 (12.1, 52.3)	
Any increase in SBP	12.1 (−2.3, 26.6)		...	

Mean volumes for SBP on admission are expressed by 10 mm Hg difference under or above 180 mm Hg. All models were adjusted for stroke subtype (lacunar vs nonlacunar), time from onset to inclusion, CSS, body temperature (<37.5°C vs ≥37.5°C), and serum glucose on admission. Values are in milliliters.

the readings taken in the ED. Following this method, we found the lowest frequency of a poor prognosis at approximately 180 mm Hg for SBP.

For a clinically relevant interpretation of our results, it was crucial to differentiate whether high BP levels or subsequent treatment with antihypertensive drugs in many of these patients might have negatively influenced the outcome. This issue has not been answered in a recently published similar study.[14] Our findings indicate that the use of antihypertensive drugs and, more importantly, the fall in BP during the first day after admission are detrimental for patients with acute ischemic stroke. The key factor seems to be a fall >20 mm Hg in SBP. This was the most important variable associated with early neurological deterioration, poor outcome, and large infarct volume in patients with SBP ≤180 mm Hg who were not treated with hypotensive drugs after admission and in patients with SBP >180 mm Hg after adjustment for the use of antihypertensive therapy. This finding raises the question of whether the frequent spontaneous decrease of BP during the first day of stroke, or other potential causes of fall in BP that were not controlled in this study, contributed to the poor prognosis.

This study has a number of methodological limitations that might have influenced some of the findings. First, although most baseline variables and all outcome variables were defined a priori in the protocol, the cutoff values of SBP and DBP were chosen in a post hoc analysis. However, they were not chosen in an arbitrary way, and the selected values were clinically meaningful because they were quite similar to the values over which treatment of BP is recommended for patients treated with thrombolytic drugs.[15] Second, we have not established the optimal SBP for particular subgroups because of the small number of patients, and therefore our findings might differ depending on the presence of some vascular risk factors or the stroke subtype. For example, a decrease in BP might not have detrimental effects in lacunar infarctions to the same extent as in large territorial ischemia, in which a penumbral area is more likely.[16] Third, patients who were lost to follow-up showed a trend of being younger and having less severe strokes, particularly lacunar infarctions (data not shown). This fact might have biased the results on late outcome and mortality. Finally, this is an observational study, in which many factors were not controlled, and therefore the results should be interpreted cautiously.

If these results are confirmed in randomized controlled trials, they would necessitate a reconsideration of the current indications for antihypertensive treatment during the acute phase of ischemic stroke.[3,7]

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Editorial Comment

Persisting Dilemma: To Treat or Not to Treat Blood Pressure in Acute Ischemic Stroke

Treatment of blood pressure in acute stroke is controversial, whether attempts are made to reduce or to increase blood pressure. Few clinical studies are available to guide clinicians. A Cochrane review[1] on deliberately altering blood pressure within 2 weeks of stroke onset found 5 small trials, involving 218 patients randomized to nimodipine, nicardipine, captopril, clonidine, glyceryl trinitrate, or perindopril versus placebo or control treatment. The limited number of data made it impossible to assess the relationship between blood pressure and clinical outcome.

Ahmed et al[2] made a post hoc analysis on the effect of intravenous nimodipine in acute ischemic stroke within 24 hours. They found that a reduction of diastolic blood pressure of about 15 mm Hg was associated with poor outcome, whereas a spontaneous fall in the placebo group of about 8 mm Hg was associated with a better outcome.

In this issue of *Stroke*, Castillo et al found that a fall in systolic blood pressure of more than 20 mm Hg was associated with neurological deterioration and poor outcome. This was an observational study in 258 acute stroke patients, of whom many had their blood pressure lowered. Antihypertensive treatment was started already in the emergency department by other doctors than those involved in the study. The blood pressure–lowering treatment was given according to international guidelines, which, however, are based on reasoning rather than on evidence. Antihypertensive drugs were given to 38.7% of patients with early neurological deterioration versus 16.9% in those without deterioration, $P<0.05$. In patients with poor outcome, antihypertensive drugs were given to 30.1% versus 9.5% in patients with good outcome, $P<0.05$.

The findings seem to agree with those of Ahmed et al,[2] while they are at variance with those of Chamorro et al,[3] who found that a 20% to 30% drop in mean arterial blood pressure on day 2 after stroke onset almost tripled the odds of full recovery with no indication of adverse effect of hypotensive agents.

In an observational study of 759 patients with acute ischemic stroke, Christensen et al[4] found that a spontaneous fall in blood pressure within the first 4 hours after admission was associated with good outcome. A partial explanation of the divergent results might be that a spontaneous fall in blood pressure is a benign change in contrast to an induced fall in blood pressure. This notion is supported by a Cochrane review[5] on the effect of calcium antagonists for acute ischemic stroke, in which intravenous administration of calcium antagonists was associated with poor outcome compared to placebo.

Further, Castillo et al found a U-shaped association between blood pressure and outcome with a systolic blood pressure of 180 mm Hg being the optimal. A U-shape was also found in the International Stroke Trial (IST),[6] but here the optimal blood pressure was around 150 mm Hg. The IST being so much larger is likely to yield the more robust data. However, analysis of the GAIN International Trial[7] in 1455 patients with acute ischemic stroke did not find a U-shape of the relation between blood pressure and outcome. Neither was a 30% decrease in mean arterial pressure from baseline associated with outcome. If the U-shape relation is real, it may reflect cardiac failure being more important in those with low systolic blood pressure and an adverse effect on brain edema in those with very high blood pressure.

In agreement with most guidelines, antihypertensive treatment in acute stroke should probably be restricted to those in the high range of systolic blood pressure. Due to the tendency for the blood pressure to fall spontaneously within the first hours after admission, antihypertensive treatment might be delayed for 8 to 12 hours, if earlier treatment is not required as for instance in patients suitable for thrombolysis. Presumably, antihypertensive treatment needs to be tailored individually. It is unlikely that there will be one treatment for all.

There is an urgent need for a sufficiently large randomized clinical trial on antihypertensive treatment in acute stroke in order to solve the dilemma.[8]

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Blood Pressure Decrease During the Acute Phase of Ischemic Stroke Is Associated With Brain Injury and Poor Stroke Outcome

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