Post-Stroke Hypertension Correlates with Neurologic Recovery in Patients with Acute Ischemic Stroke

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To examine the clinical implications of post-stroke hypertension, defined as the rise in blood pressure on admission after the onset of ischemic stroke as compared with the blood pressure before stroke, and to assess the relationship between the value of post-stroke hypertension and neurologic recovery, we retrospectively studied 28 patients admitted to the hospital within 24 h (mean ± SD, 6.7 ± 7.0 h) after a first-ever, acute non-embolic ischemic stroke, whose blood pressure had been recorded at the outpatient clinic within 3 mo before stroke. The Canadian Neurological Scale was used to assess stroke severity, and neurologic recovery during the acute phase was calculated. The average duration of hospitalization was 18 ± 9 d. The value of post-stroke hypertension and stroke severity on admission independently and significantly correlated with neurologic recovery (odds ratio, 1.06; 95% confidence interval, 1.00-1.12 and odd ratio, 0.20; 95% confidence interval, 0.06-0.72, respectively). There was also a significant linear correlation between the value of post-stroke hypertension and neurologic recovery ($r = 0.50$, $p < 0.01$). Furthermore, blood pressure after the onset of ischemic stroke was quite independent of blood pressure before stroke. We conclude that the value of post-stroke hypertension correlates with neurologic recovery in patients with acute non-embolic ischemic stroke. These results suggest that blood pressure control mechanisms change after the onset of acute ischemic stroke. (*Hypertens Res* 1998; 21: 169-173)

***Key Words*: post-stroke hypertension, ischemic stroke, stroke assessment, prognosis**

Acute hypertension after stroke is widely recognized, and the blood pressure (BP) falls spontaneously in the days after acute ischemic stroke without specific antihypertensive therapy (*1-4*). It has been widely accepted "not to treat hypertension in acute ischemic stroke" (*5*). Recently, we postulated that the surge in BP did not precede but followed acute ischemic stroke (*6*). The mechanisms for these BP changes after stroke are still unclear, but may be related to stroke-induced changes in sympathoadrenergic activity (*7, 8*), stress reaction to hospital admission or BP measurement (*9*), or central mechanisms (*10*). We studied the correlation of the change in BP after the onset of stroke, as compared with the value before stroke, with stroke severity on admission and neurologic recovery during the acute phase.

Patients and Methods

Patients

From January 1995 through December 1996, a total of 127 patients were admitted to either Chikamori Hospital or Kochi Medical School Hospital, which are local referral centers, within 24 h after onset of a first-ever non-embolic ischemic stroke. Among these patients, we studied 28 consecutive patients (16 men and 12 women; mean age, 69.5 ± 10.9 yr) whose records included medical history, BP measurement(s) within the past 3 mo and medication(s). Patients who had a history of confirmed neurologic disorders or who received thrombolytic therapy were excluded. Twenty-two of the 28 patients had a history of hypertension. Among these patients, 18 were receiving antihypertensive drug(s) before admission, and 4 were not, despite the presence of hypertension. The other 6 patients were normotensive. The mean estimated time after stroke onset was 6.7 ± 7.0 h, and the average duration of hospitalization was 18 ± 9 d.

Stroke subtype was categorized as lacunar stroke ($N = 22$) or atherothrombotic stroke ($N = 6$). Our diagnostic criteria were based on the Classification of Cerebrovascular Disease III by the National Institute of Neurological Disorder and Stroke (*11*). Infact size was calculated as longest diameter × shortest diameter of the lesion on computed tomographic (CT) or magnetic resonance imaging (MRI) scans.

The Canadian Neurological Scale (CNS) (*12, 13*)

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Table 1. Clinical Characteristics of Patients with Good Recovery and Poor Recovery

Variable	Poor recovery ($N=13$)	Good recovery ($N=15$)	p
Age	67.0±11.6	71.6±10.2	NS
Male sex (%)	8 (62)	7 (47)	NS
Antihypertensive treatment(s) (%)	9 (69)	9 (60)	NS
Diabetes mellitus (%)	3 (23)	4 (27)	NS
History of MI (%)	1 (8)	4 (27)	NS
Serum cholesterol (mmol/l)	4.70±1.10	5.28±0.75	NS
MAP before stroke onset (mmHg)	106±14	96±15	NS
MAP on admission after stroke onset (mmHg)	107±18	119±20	NS
Value of PSH (mmHg)	1±20	23±24	<0.05
Lacunar infarction (%)	10 (77)	12 (80)	NS
Infarct size (D×L)	139±239	137±201	NS
Stroke severity on admission (CNS score)	9.0±1.1	7.6±1.0	<0.01
Stroke severity at discharge (CNS score)	9.3±1.1	9.6±0.8	NS
Neurologic recovery (CNS score)	0.3±0.3	2.0±0.5	<0.01

NS, not significant; MI, myocardial infarction; MAP, mean arterial blood pressure; PSH, post-stroke hypertension; CNS, Canadian Neurological Scale.

was used to assess stroke severity. The CNS focuses on the level of consciousness, speech, and strength and scores neurologic severity from 1.5 to 10.0, with lower scores indicating severer disability. The CNS score of each patient was assessed by a single neurologist (YO) both on admission and discharge. Regardless of the changes during the observation period, neurologic recovery was defined as (CNS score at discharge) − (CNS score on admission).

BP Measurements

At the outpatient clinic, within 3 mo before admission, BP was measured with a manual sphygmomanometer in the doctor's office. BP was measured two consecutive times while the subject was in a sitting position after at least 2 min of rest. The average of these two values was considered baseline BP before stroke onset. Mean arterial blood pressure (MAP) was calculated for each patient according to the following formula: MAP = diastolic BP + (systolic BP − diastolic BP)/3. Any antihypertensive drugs were withdrawn during the observation period. On admission, BP was measured with a manual sphygmomanometer while patient was in a lying position in the emergency room. "Post-stroke hypertension (PSH) in MAP" was defined as (MAP on admission after onset of stroke) − (MAP at outpatient clinic before stroke).

Image Analysis

All patients underwent head CT scans, with a SCT-3000TC (Shimadzu, Japan) scanner, within an hour after admission. Contrast medium was not given routinely. Each case was reevaluated by CT or MRI scans during the following days. No patient had a evidence of hemorrhagic transformation.

Statistical Methods

Results are expressed as means ± 1SD. Differences between two groups were compared with Student's t-test. The correlation coefficient between two variables was determined by Pearson's simple regression analysis. We evaluated the relative contribution of confounding variables to good neurologic recovery by multiple logistic regression analysis. Neurologic recovery was divided into two categories (< 1 point = 0, poor recovery; ≥ 1 point = 1, good recovery). Differences were considered significant when $p < 0.05$.

Results

Of the 28 patients, 15 (54%) had good neurologic recovery. Table 1 shows the baseline characteristics of the patients, including average MAPs before and after stroke onset, average PSH, antihypertensive treatment(s), stroke severity according to the CNS score, and neurologic recovery, in the good and poor neurologic recovery groups. Age, gender, a previous history of diabetes mellitus or myocardial infarction, MAP after stroke onset, stroke subtype, infarct size, and stroke severity at discharge did not significantly differ between the two groups. Antihypertensive treatment(s) did not affect the value of PSH, stroke severity, or neurologic recovery (data not shown). Patients with good neurologic recovery had a significantly higher value of PSH and severer stroke severity on admission. Mean arterial blood pressure before stroke onset was slightly but not significantly lower in the good recovery group ($p = 0.07$).

To determine the predictors of good neurologic recovery, we performed univariate analyses as shown in Table 2. The value of PSH, a value of PSH ≥ 0 mmHg, and stroke severity on admission were the covariates for which p values reached significance ($p < 0.05$); they were used for multiple logistic regression analyses. Table 3 shows the results of multiple logistic regression analyses considering neurologic recovery as a dependent variable. As expected, stroke severity on admission significantly contributed to neurologic recovery. Nota-

Table 2. Univariate Analysis of Good Recovery in Patients with Acute Ischemic Stroke

Variable	Unadjusted odds ratio	95% Confidence Interval	*p*
Age	1.04	0.98-1.11	NS
Male sex (%)	1.83	0.41-8.23	NS
Antihypertensive treatment(s) (%)	1.50	0.57-3.97	NS
Diabetes mellitus (%)	0.83	0.16-4.25	NS
History of MI (%)	0.19	0.02-1.71	NS
Serum cholesterol (mmol/l)	1.02	0.99-1.05	NS
MAP before stroke onset (mmHg)	0.95	0.90-1.01	NS
MAP on admission after stroke onset (mmHg)	1.03	0.99-1.09	NS
Value of PSH (mmHg)	1.05	1.01-1.10	<0.05
Value of PSH ≥ 0 mmHg	6.40	1.27-32.35	<0.05
Lacunar stroke (%)	0.83	0.47-1.08	NS
Infarct size (D×L)	1.00	0.99-1.00	NS
Stroke severity on admission (CNS score)	0.24	0.08-0.72	<0.05
Stroke severity at discharge (CNS score)	1.53	0.63-3.74	NS

NS, not significant; MI, myocardial infarction; MAP, mean arterial blood pressure; PSH, post-stroke hypertension; CNS, Canadian Neurological Scale.

Table 3. Multiple Logistic Regression Analysis of Good Neurologic Recovery in Patients with Acute Ischemic Stroke

Covariate	Adjusted odds ratio	95% CI	*p*
Model 1			
Value of PSH (mmHg)	1.06	1.00-1.12	<0.05
Stroke severity on admission	0.20	0.06-0.72	<0.05
Model 2			
Value of PSH ≥ 0 mmHg	10.65	1.09-104.2	<0.05
Stroke severity on admission	0.21	0.06-0.72	<0.05

MAP, mean arterial blood pressure; PSH, post-stroke hypertension.

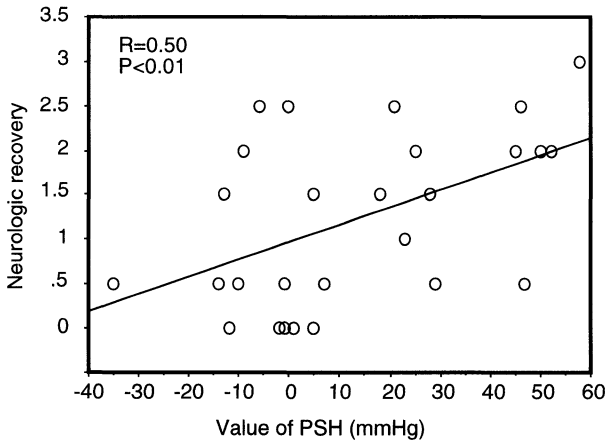

Fig. 1. *The value of PSH positively correlated with neurologic recovery.*

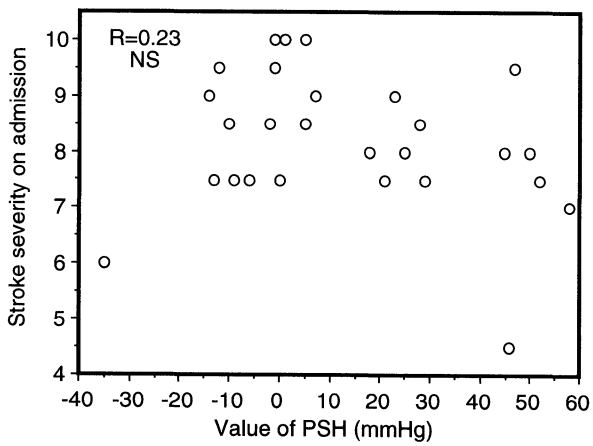

Fig. 2. *There was no significant correlation between the value of PSH and stroke severity on admission.*

bly, the value of PSH also significantly contributed to neurologic recovery independently of stroke severity on admission in both models used for multiple logistic regression analysis (Table 3). For patients with a PSH value of ≥ 0 mmHg, the odds ratio for good recovery was 10.65 (odds ratio, 10.65; 95% confidential interval, 1.09-104.2). Simple regression analysis showed a significant positive correlation between the value of PSH and neurologic recovery (Fig. 1). There was no significant correlation between stroke severity on admission and the value of PSH (Fig. 2). Mean arterial blood

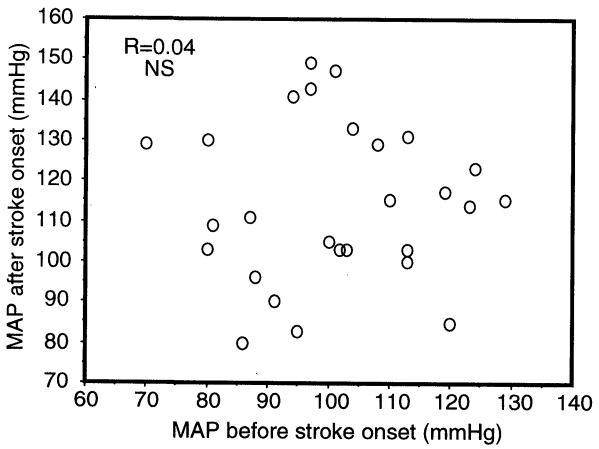

Fig. 3. *There was no significant correlation between MAP on admission after the onset of stroke and MAP before stroke.*

pressure after stroke onset was quite independent of MAP before stroke onset (Fig. 3). In addition, the maximum value of MAP on admission after stroke onset was 149 mmHg (208/119 mmHg) in a 65-yr-old man who showed good neurologic recovery. In 5 of 7 patients with PSH values ≥ 0 mmHg who received no previous antihypertensive treatments, BP was below the baseline level for 5 or more days after stroke onset.

Discussion

Based on analyses of diurnal BP variation, we previously postulated that the surge in BP did not precede but followed acute ischemic stroke (*6*). We also reported that the nocturnal dip in BP diminished in relation to stroke severity in patients with acute ischemic stroke, suggesting that compensatory mechanisms maintaining perfusion of the ischemic brain might act against acute ischemic stroke (*14*). The present study showed that the value of PSH and stroke severity on admission independently and significantly correlated with neurologic recovery. These findings suggest that patients with a higher value of PSH have greater cerebral perfusion, resulting in better neurologic recovery, and support our previous hypothesis that BP rises after the onset of acute ischemic stroke. We therefore believe that PSH is caused primarily by compensatory mechanisms against the ischemic event, rather than by simple stress reactions to hospital admission or BP measurement (*9*), or by Cushing reflex (*10*); moreover, PSH is independent of stroke severity on admission.

The reasons why patients with severer stroke severity on admission had better neurologic recovery are still unclear. Because most patients in our study had high CNS scores at discharge, many may have had mild stroke. Future studies of larger number of patients evaluated with different stroke scales are needed to arrive at firm conclusions.

Another important finding in this study was that BP after stroke onset was quite independent of the baseline BP before stroke, even in individual patients. Although several reports have mentioned acute hypertension after stroke (*1-4, 8, 9, 15*), to our knowledge no study has evaluated PSH similarly to our study. Our results suggest that BP control mechanisms change dramatically after the onset of acute ischemic stroke.

The recommendation "not to treat hypertension in acute ischemic stroke" would be justified by our findings. Hachinski proposed that antihypertensive treatment is warranted in patients with hypertensive encephalopathy, myocardial infarction, or extreme hypertension (blood pressure $>220/130$ mmHg) (*16*). As maximum BP on admission after acute ischemic stroke onset was lower than 220/130 mmHg in our study, we believe that few patients with acute ischemic stroke would benefit from antihypertensive therapy.

In conclusion, BP after the onset of stroke is independent of BP before stroke, and the value of PSH weakly but significantly correlates with neurologic recovery in patients with acute ischemic stroke during short-term hospitalization, supporting the recommendation "not to treat hypertension in acute ischemic stroke." Clarification of the relation between the value of PSH and long-term neurologic recovery would require extended follow-up of patients who have had a stroke.

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