The Prognostic Value of Admission Blood Pressure in Patients With Acute Stroke

Bo Carlberg, MD; Kjell Asplund, MD; Erik Hägg, MD

**Background and Purpose:** Patients with acute stroke are often found to have high blood pressures at hospital admission. Previous studies have shown variable results regarding the prognostic value of high blood pressure in acute stroke. The aim of this study was to investigate the prognostic value of admission blood pressure in a population-based sample of patients with acute stroke.

**Methods:** Eighty-five patients with intracerebral hemorrhage and 831 with ischemic disease were included in the study. The relations between admission blood pressure and 30-day mortality were studied by logistic regression analyses.

**Results:** High blood pressure in patients with impaired consciousness on hospital admission was significantly related to 30-day mortality in patients with intracerebral hemorrhage ($P=0.037$) and in patients with ischemic disease ($P<0.001$). In patients without impaired consciousness, high blood pressure at time of admission was not related to increased mortality at 30 days.

**Conclusions:** High admission blood pressure in alert stroke patients was not related to increased mortality. Stroke patients with impaired consciousness showed higher mortality rates with increasing blood pressure. However, this does not provide a basis for recommending antihypertensive therapy for such patients. (Stroke. 1993;24:1372-1375.)

**Key Words** • blood pressure • cerebrovascular disorders • hospitalization • prognosis

Patients with acute stroke often present with high blood pressure levels on hospital admission.1-3 Because high blood pressure is an important risk factor for stroke and acute severe blood pressure elevation is a potentially life-threatening condition, acute antihypertensive treatment may seem logical. On the other hand, stroke may be initiated by spontaneous or drug-induced hypotension,4-8 and acute antihypertensive therapy may be deleterious in some patients with acute stroke.9,10 This has resulted in a more careful approach toward acute antihypertensive therapy in the acute stroke situation.7,11,12 In some patients with acute cerebral ischemia, antihypertensive therapy may harmfully decrease blood flow in the ischemic areas.13 Elevated blood pressures in acute stroke patients often decrease spontaneously, making treatment unnecessary.1-2,14

In cases of intracerebral hemorrhage, the situation may be more complicated. Intracranial pressure is often raised, and blood pressure reduction may result in hypoperfusion and infarction of the surrounding edematous tissue.7 On the other hand, high blood pressure may increase edema in areas surrounding a cerebral infarct or hemorrhage.7,15 In some stroke patients (eg, in patients with aortic dissection, acute severe left ventricular failure, severe angina pectoris, and hypertensive encephalopathy), acute blood pressure treatment may be mandatory.

The question of optimal treatment of high blood pressure in patients with acute stroke can only be answered by appropriate prospective randomized intervention studies. In the absence of such studies, observational studies may provide information of the prognostic value. Several such studies have reported a relation between high blood pressure levels and poor outcome, including death in acute stroke,16-21 but this relation has not been shown consistently.22-24

The aim of this study was to determine whether initial blood pressure levels in acute stroke patients provide any prognostic value related to acute mortality. Compared with previous studies, we have studied a larger number of unselected stroke patients with verified stroke subtypes.

**Materials and Methods**

All patients admitted to the stroke unit at the Department of Medicine in the Umeå University Hospital between January 1978 and February 1988 were included in the study. All patients had focal neurological deficits of suspected cerebrovascular origin (including ischemic attacks) within the last week. The medical clinic was the only clinic for stroke patients in the hospital catchment area, and the patients were admitted directly to the stroke unit from the hospital emergency room. Home care of stroke patients is unusual in this area.25 The stroke patients in the unit are representative of all stroke patients in the hospital catchment area.26 The patients were investigated in a standardized manner with repeated neurological examinations, laboratory tests, and brain computed tomographic (CT) scan. Blood pressure, measured in the emergency room, was recorded together with other data in a prospective data...
TABLE 1. Thirty-Day Case Fatality Rate in Groups With Different Admission Mean Arterial Pressures and in Patients With Mean Arterial Pressures of 140 mm Hg or Higher

<table>
<thead>
<tr>
<th>MAP (mm Hg)</th>
<th>Patients with ischemic stroke</th>
<th>Patients with intracerebral hemorrhage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All</td>
<td>Deceased</td>
</tr>
<tr>
<td>≤99</td>
<td>91</td>
<td>12 (13%)</td>
</tr>
<tr>
<td>100–109</td>
<td>130</td>
<td>15 (12%)</td>
</tr>
<tr>
<td>110–119</td>
<td>176</td>
<td>24 (14%)</td>
</tr>
<tr>
<td>120–129</td>
<td>171</td>
<td>21 (12%)</td>
</tr>
<tr>
<td>130–139</td>
<td>125</td>
<td>10 (8%)</td>
</tr>
<tr>
<td>≥140</td>
<td>138</td>
<td>17 (12%)</td>
</tr>
</tbody>
</table>

MAP, mean arterial blood pressure.

Fatality Multiple Logistic Regression Analysis in Patients With Ischemic Stroke

<table>
<thead>
<tr>
<th>Independent variables</th>
<th>Coefficient</th>
<th>Wald χ²</th>
<th>P</th>
<th>exp(B)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>-7.355</td>
<td>4.374</td>
<td>.0001</td>
<td></td>
</tr>
<tr>
<td>Hemiplegia</td>
<td>1.834</td>
<td>17.220</td>
<td>&lt;.0001</td>
<td>6.258</td>
</tr>
<tr>
<td>MAP and impaired consciousness</td>
<td>0.014</td>
<td>26.733</td>
<td>&lt;.0001</td>
<td>1.720</td>
</tr>
<tr>
<td>Age</td>
<td>0.054</td>
<td>12.091</td>
<td>.0005</td>
<td>1.056</td>
</tr>
<tr>
<td>Diabetes</td>
<td>0.866</td>
<td>9.789</td>
<td>.018</td>
<td>2.380</td>
</tr>
<tr>
<td>Cardiac failure</td>
<td>0.542</td>
<td>4.374</td>
<td>.036</td>
<td>1.720</td>
</tr>
<tr>
<td>Impaired consciousness and hemiplegia</td>
<td>-1.093</td>
<td>3.647</td>
<td>.056</td>
<td>0.335</td>
</tr>
</tbody>
</table>

exp(B), odds change with one-unit increase of the variable; MAP, mean arterial blood pressure.

Rejected covariates were as follows: MAP, impaired consciousness, MAP and hemiplegia, and MAP and cardiac failure. Model χ², P<.0001. Fifty-one patients were deleted because of incomplete data. Association of predicted probabilities and observed responses was as follows: concordant, 89%; discordant, 11%.

Blood pressures were always measured with a calibrated sphygmomanometer and with patients in the supine position, and the disappearance of the Korotkoff phase-5 sound was defined as the diastolic blood pressure. Survival at 30 days after admission was checked by the population census registry in patients discharged from the unit before that day.

During the study period, 953 patients were treated in the stroke unit. Eight patients were excluded because their data records were lost or did not contain information about the admission blood pressure. The remaining 945 patients (529 males, 416 females) had a mean age of 72.3 years (range, 30 to 102 years). The criteria for diagnosis of stroke subtypes have been described elsewhere. Eighty-five patients (9.0%) had intracerebral hemorrhage, 498 (53%) had noncardioembolic infarction, 227 (24%) had cardioembolic infarction, and 106 (11%) had transient ischemic attacks. In the remaining 29 patients, the stroke diagnosis could not be specified (reasons included missing CT scan, death of patient before CT scan with relatives unwilling to approve autopsy, refusing collaboration, etc, and subarachnoid hemorrhage [five patients]). Mean arterial pressure (MAP) was calculated in each patient according to the following formula: MAP = diastolic blood pressure + (systolic blood pressure – diastolic blood pressure)/3. Patients treated with antihypertensive therapy before their stroke continued with their medication. High blood pressures were usually not treated in the acute stage. During the first 4 days, antihypertensive therapy was started or intensified in 16 patients.

Independent t tests were used to test differences in blood pressure between groups, and P<.05 was considered statistically significant. Multiple logistic regression was performed with spss, a computerized statistical program using forward selection followed by backward elimination of covariates, resulting in an equation where only covariates that significantly increase the predictability of the dependent variable are included. Age (years), impaired consciousness on admission (no/yes), hemiplegia on admission (incomplete/complete), history of cardiac failure (no/yes), diabetes (no/yes), and MAP (millimeters of mercury) were included as independent variables. The interactions of MAP and impaired consciousness, MAP and hemiplegia, impaired consciousness and hemiplegia, and MAP and cardiac failure were also tested in the model. The selected variables have all been shown to be associated with poor outcome after stroke.

The multiple logistic regression analyses were done separately for patients with ischemic disease (cerebral infarction or transient ischemic attack) and intracerebral hemorrhage. In the cases of intracerebral hemorrhage, the covariate factors diabetes and cardiac failure were excluded because their inclusion resulted in contingency tables with zero cells.

**Results**

Median latency between the onset of symptoms and hospital admission was 5 hours, and 85% of the patients were hospitalized within 24 hours. The 30-day case fatality rate was 14%. The case fatality rate within the first week was 8%. The first week, 90% of all deaths were ascribed directly to the stroke. The most common cause of death after the first week was pulmonary embolism, followed by stroke, cardiac disease, and bronchopneumonia, as previously reported.

The mean value of MAP on hospital admission in all patients was 122 mm Hg (175/95 mm Hg). Patients who died within 30 days had a mean admission MAP of 122 mm Hg (176/95 mm Hg); the surviving patients, 121 mm Hg (175/95 mm Hg) (P = NS). Blood pressures did not differ between surviving and deceased patients in the diagnostic subgroups. There was no significant correlation...
Plots showing 30-day case fatality rate in groups with different blood pressures at hospital admission. MAP indicates mean arterial blood pressure; •, patients with impaired consciousness; and ○, alert patients. The calculated regression lines for patients with impaired consciousness are inserted.

(Spearman’s rank correlation \( r_s \)) between MAP on admission and mortality \( r_s = 0.035 \) for intracerebral hemorrhage, and \( r_s = -0.022 \) for ischemic stroke).

A relation between blood pressure and mortality need not be linear. Therefore, the patients were separated into six admission blood pressure groups, and the mortality in each group was calculated. Table 1 shows the 30-day case fatality in all patients with ischemic disease (cerebral infarction and transient ischemic attack) and intracerebral hemorrhage for the different blood pressure groups. The mortality rates were similar in all admission blood pressure groups. Functional outcome (independent, walking with support, or bedridden) at discharge did not differ between the different admission blood pressure groups on admission (data not shown).

When using multiple logistic regression, MAP was found to be significantly related to 30-day case fatality in patients with impaired consciousness on hospital admission in both ischemic (Table 2) and hemorrhagic stroke \( \exp(B), 1.018 \) [odds change per 1 mm Hg increase of blood pressure]; \( P = .037; \) not shown in Table 2) patients. MAP or impaired consciousness as isolated variables did not increase the predictability of the model.

As shown in the Figure, the relation between blood pressure on admission and prognosis differed in patients who had lowered consciousness as compared with alert patients. Thus, higher MAP was associated with increased mortality in patients with impaired consciousness, irrespective of whether they had ischemic or hemorrhagic stroke. No such relation was found in patients who were alert on admission to the hospital. Major medical variables in the study patients are shown in Table 3.

Alert patients had somewhat higher blood pressures than those with impaired consciousness. In all 945 patients, admission MAP was 122.3 mm Hg in alert patients and 119.5 mm Hg in patients with lower grades of consciousness \( (P = .046). \) In patients with ischemic disease and a MAP of 130 mm Hg or higher, the mortality rate was 4% \( (9 \text{ of } 219) \) in alert patients and 42% \( (18 \text{ of } 43) \) in patients with impaired consciousness. Corresponding results for patients with intracerebral hemorrhage were 0% \( (0 \text{ of } 26) \) and 59% \( (10 \text{ of } 17) \), respectively.

**Discussion**

The causes of the high blood pressure in patients with acute ischemic stroke are not completely known. Approximately 50% of the patients have a previously known hypertension.1 2 3 3 The Cushing reflex in patients with increased intracranial pressure increases blood pressure. Sympathetic activation secondary to the brain lesion may be a beneficial homeostatic response to increase blood flow in the ischemic penumbra.4 3 We have previously observed that there is no time relation between the onset of symptoms and the blood pressure

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**TABLE 3. Medical Variables on Hospital Admission in the Study Patients**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Patients with intracerebral hemorrhage</th>
<th>Patients with ischemic disease</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Impaired consciousness (n=38)</td>
<td>Alert (n=47)</td>
</tr>
<tr>
<td>Age (y, mean±SD)</td>
<td>72±10</td>
<td>70±10</td>
</tr>
<tr>
<td>Male</td>
<td>18 (47%)</td>
<td>32 (68%)</td>
</tr>
<tr>
<td>Previous hypertension</td>
<td>25 (66%)</td>
<td>20 (43%)</td>
</tr>
<tr>
<td>Blood pressure (mm Hg, mean±SD)</td>
<td>192±37/100±10</td>
<td>189±30/101±15</td>
</tr>
<tr>
<td>Previous stroke</td>
<td>11 (29%)</td>
<td>10 (21%)</td>
</tr>
<tr>
<td>Cardiac failure (treated)</td>
<td>12 (32%)</td>
<td>10 (21%)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>4 (11%)</td>
<td>6 (13%)</td>
</tr>
<tr>
<td>Deceased, 30 days</td>
<td>21 (55%)</td>
<td>2* (4%)</td>
</tr>
</tbody>
</table>

n. Number of patients in study group.
The level of consciousness was not classified in eight patients with ischemic disease.

\( *P<.0001, \) \( †P<.001, \) and \( ‡P<.05 \) vs corresponding value for patients with impaired consciousness by the Yates chi-square test.
course in a great number of stroke patients, and we have proposed that mental stress at hospitalization may be an important blood pressure-elevating factor in acute ischemic stroke.\textsuperscript{33,35} We have also found that patients with intracerebral hemorrhage have a different blood pressure course than patients with ischemic disease.\textsuperscript{35} Should high blood pressure be lowered in patients with acute stroke, as sometimes recommended?\textsuperscript{34,36,37} In our study, alert patients with ischemic disease showed no tendency to increased mortality with increasing blood pressure (Figure), suggesting that benefits of acute antihypertensive treatment were unlikely. In patients with impaired consciousness, high blood pressure was related to increased mortality, but this information does not provide a basis for recommending antihypertensive therapy for such patients.

In patients with intracerebral hematoma, there are suggestions that rebleedings in the acute stage are more common if the blood pressure is high.\textsuperscript{38,39} However, a blood pressure reduction of 20\% or more may decrease cerebral blood flow.\textsuperscript{40} There is also a risk that antihypertensive agents such as dihydralazine,\textsuperscript{41} nifedipine, chlorpromazine, reserpine,\textsuperscript{42} sodium nitroprusside,\textsuperscript{43} and nitroglycerine\textsuperscript{44} increase the intracranial pressure.

Acknowledgments

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References

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