Factors Influencing Admission Blood Pressure Levels in Patients With Acute Stroke

Bo Carlberg, MD; Kjell Asplund, MD; and Erik Hagg, MD

In clinical practice, patients with acute stroke often have high blood pressure. The aim of this study was to investigate factors correlated with blood pressure elevation in 843 consecutive stroke patients on hospital admission to a nonintensive stroke unit. Using a multivariate analysis model, we analyzed the influence on admission blood pressure of sex, age, previous hypertension, cardiac failure, diabetes, type of stroke, impaired consciousness, and latency between onset of symptoms and admission. Previous hypertension was the strongest predictor ($p<0.001$) of elevated blood pressure on admission, followed by the presence of intracerebral hemorrhage ($p<0.001$). The latency between onset of symptoms and admission showed no correlation with blood pressure levels at hospitalization. Previously, high blood pressure levels on hospital admission have been shown to decline within a few days in hospital. We therefore hypothesize that mental stress on hospital admission may be a major factor in the blood pressure elevation seen in acute stroke. (Stroke 1991;22:527–530)

Patients with acute stroke often have high blood pressure levels on hospital admission that decline spontaneously soon afterward. The reasons for the high blood pressure are not known. Many investigators have found elevated levels of plasma catecholamines in the acute phase of stroke, which may be secondary to the brain lesion. Others suggest that elevated blood pressure may be a response to decreased perfusion in the ischemic border zone. However, it is clear that previous hypertension influences the blood pressure level on admission. In addition, in an autopsy study, patients with hemorrhages and infarctions localized in the pons, mainly in the rostral tegmentum, were found to have the most prominent acute blood pressure elevations.

Immediate antihypertensive treatment in acute ischemic stroke seems to involve a considerable risk. There is a great deal of evidence that such blood pressure reduction in ischemic stroke harmfully decreases blood perfusion in the ischemic border zone. On the other hand, some researchers have emphasized the risks of increasing brain edema if an elevated blood pressure is not treated. The situation in intracerebral hemorrhage is even more complicated, but the same principle of avoiding rapid blood pressure reduction seems to be the least risky choice. Our study investigates the factors correlated with blood pressure on hospital admission in patients with acute stroke.

Subjects and Methods

We included all patients admitted to the stroke unit in the medical clinic, University Hospital of Umeå, between 1978 and 1986. The six-bed stroke unit receives patients from the city of Umeå and surrounding areas who are admitted directly from the emergency room in an unselective manner. The criteria for admission consisted of a focal neurologic deficit of presumed cerebrovascular origin, including transient ischemic attack (TIA) during the last week. Patients with only vertigo or altered consciousness without focal neurologic symptoms were not admitted to the stroke unit. With few exceptions, patients with subarachnoid hemorrhage were admitted directly to the department of neurosurgery.

Patients were investigated in a standardized manner with repeated neurologic examinations, laboratory tests, and brain computed tomography (CT) scan. Blood pressure measured in the emergency room and other data were recorded together in a prospective data record. Blood pressures were always measured with the patient in a supine position, using a calibrated sphygmomanometer, with disappearance of the Korotkoff phase 5 sound defined as diastolic blood pressure.

During the study period, a total of 843 patients (477 men, 366 women; mean age 72.2 years, range 30–102 years) fulfilled the stroke criteria. Mean age did not differ significantly between the sexes (71.2
years in men, 73.6 years in women). The criteria for the different diagnoses have been described elsewhere. 17 Seventy-seven patients (9.1%) had intracerebral hemorrhage, 440 (52.2%) had nonembolic infarction, 204 (24.2%) had embolic infarction, and 97 (11.5%) had TIA. In the remaining 25 patients (3%), the diagnosis could not be specified either because of a missing CT, because patients died before CT scans and relatives were unwilling to allow autopsy, or because of lack of cooperation. In this group, there were also five patients with subarachnoid bleeding. The fatality rate in the stroke unit was 14.6%.

In this study, previous hypertension was defined as pharmacologic treatment of hypertension before admission. Four hundred and eighteen of the patients (49.6%) had a history of hypertension. In 12 patients, no information about previous hypertension was available. In 828 patients, data on previous treatment for cardiac failure were available; 295 of these patients (31.3%) had been treated for cardiac failure. One hundred forty-seven (17.4%) patients had a history of diabetes mellitus. In four patients, diabetes data were not available. The mean age did not differ between diabetic and nondiabetic patients. The most important prognostic factor in acute stroke is the level of consciousness on admission. 18-21 We therefore separated our patients into two groups according to the level of consciousness. Eight patients could not be classified. Six hundred forty-two patients (76.9%) had no impaired consciousness, and 193 (23.1%) had impaired consciousness on admission (from somnolence to coma).

Means and 95% confidence limits were used to describe the blood pressure levels. To evaluate differences between the subgroups, the \( \chi^2 \) test with Yates' correction was used. For univariate regression, Pearson correlation coefficients were calculated. Multiple regression analyses were done with standardized coefficients, and values of \( p < 0.01 \) were considered statistically significant. All statistical work was performed with SYSTAT, a computerized statistical program (SYSTAT Inc., Evanston, Ill.).

**Results**

Hospital admission blood pressures of all stroke patients were averaged to yield both a mean systolic and a mean diastolic figure, which we determined to be 176/95 mm Hg (95% confidence limits, 174–178/94–96 mm Hg). One hundred and ten patients (13.0%) had a systolic blood pressure of 220 mm Hg or higher, and 67 patients (7.9%) had a diastolic blood pressure of 120 mm Hg or higher on admission.

The distribution of stroke types was the same in both sexes. Two hundred five (43.0%) of the men and 213 (58.2%) of the women had been treated for hypertension previously (\( \chi^2 = 17.5, p < 0.001 \)). On admission, mean blood pressure was 171/95 mm Hg in men and 181/95 mm Hg in women, the systolic blood pressure being significantly higher in women (95% confidence limits, 178–185 in women and 169–174 in men).

On admission, previously hypertensive patients had a mean blood pressure of 185/99 mm Hg (95% confidence limits, 182–188/98–101), whereas patients without a history of antihypertensive treatment had a mean blood pressure of 166/91 mm Hg (95% confidence limits, 163–169/90–92). The relationships between hypertension and different stroke subtypes were also considered. Significant differences in admission blood pressure levels between patients with and without previous hypertension were noted when the diagnosis was nonembolic infarction, embolic infarction, or TIA (data not shown). In patients with intracerebral hemorrhage, the corresponding difference in admission blood pressures was small, and the 95% confidence limits overlapped.

Patients with previously treated cardiac failure had a mean blood pressure on admission of 174/93 mm Hg compared with 176/96 mm Hg in patients without heart failure. The 95% confidence limits overlapped.

Of the diabetics, 61.5% had a history of hypertension, whereas 48.1% of the nondiabetics had been treated for hypertension (\( \chi^2 = 7.09, p = 0.008 \)). Diabetics was least common among patients with TIA (7.3%) and most common in patients with embolic infarction (23.6%). The blood pressure on admission in diabetics was 177/96 mm Hg and 175/95 mm Hg in nondiabetics; there was no significant difference.

Blood pressure levels on admission varied considerably by stroke subtype. Patients with intracerebral hemorrhage had the highest mean blood pressure on admission (190/99 mm Hg). Patients with nonembolic infarction had a mean blood pressure of 175/96 mm Hg; those with embolic infarction, 171/94 mm Hg; and those with TIA, 179/93 mm Hg.

A trend toward higher blood pressures existed in patients who were alert on admission when compared with those having impaired consciousness (177/96 mm Hg versus 172/94 mm Hg). However, the confidence limits overlapped. The results were the same when the four diagnostic subgroups were analyzed separately.

In 801 patients, information was available on the time interval between onset of symptoms and hospital admission. The median delay was 5 hours (90% were admitted within 41 hours) and the maximum delay was 168 hours (after that time the patients were not accepted to the stroke unit). There was no correlation between blood pressure levels on admission and time of delay between onset of symptoms and admission (systolic blood pressure, \( r = -0.04 \); diastolic blood pressure, \( r = -0.001 \)). The patients were separated into three groups according to the delay between onset of symptoms and admission to the hospital (Figure 1). The confidence limits of the admission blood pressures overlapped.

Multiple regression analyses were done to estimate independent predictors of blood pressure on admission. Items included in the analysis were type of
stroke, age, sex, history of hypertension, cardiac failure, diabetes, impaired consciousness on admission, and delay between symptom onset and admission (Table 1). Valid data on the location of the infarct lesions were not available as most CT scans were done immediately after admission and did not always visualize an infarct. Admission blood pressure was best predicted by a history of hypertension followed by intracerebral hemorrhage. In 99 patients, one of the investigated variables was missing, and these patients therefore were not included in the analyses. These individuals had no significant difference in admission blood pressure, age, or other variables in the model compared with the rest of the stroke patients.

As the admission blood pressure seemed to have a nonlinear correlation to age, separate analyses for patients older than 65 years were done. This maneuver decreased the predictability of the model somewhat but resulted in only minor changes in the standardized coefficients, except for age (data not shown).

Multiple regression analyses were done separately for the different stroke subtypes (Table 1). Highly significant correlations ($p<0.005$) between the blood pressure levels on admission and previous hypertension were found in all diagnostic groups except for intracerebral hemorrhage.

**Discussion**

History of hypertension bore the strongest relationship to blood pressure level on admission in our stroke patients, which is well supported by previous reports. Furthermore, the presence of intracerebral hemorrhage correlated with high blood pressure on admission. Patients with TIA had blood pressure levels that were in between those of patients with cerebral hemorrhages and infarcts, which contrasts to a previous study in which patients with TIA had the lowest pressures. In that study, many blood pressure recordings were delayed because of referral from other hospitals, making a direct comparison with our results impossible.

In patients with intracerebral hemorrhage, the admission blood pressure level difference between hypertensives and normotensives was small, and multiple regression analysis did not show any significant relationship between previous hypertension and blood pressure on admission. The high blood pressure in patients with intracerebral hemorrhage thus may involve other mechanisms than in patients with infarctions.

A weak, negative correlation was found between systolic blood pressure and impaired consciousness on admission. Patients with impaired consciousness seem to have more widespread cerebral lesions as indicated by a higher mortality in many studies. Thus, the extent of brain damage did not seem to be of

<table>
<thead>
<tr>
<th>Variable</th>
<th>All diagnoses</th>
<th>Hemorrhage</th>
<th>Nonembolic infarction</th>
<th>Embolic infarction</th>
<th>TIA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SBP</td>
<td>DBP</td>
<td>SBP</td>
<td>DBP</td>
<td>SBP</td>
</tr>
<tr>
<td>Hypertension</td>
<td>0.26*</td>
<td>0.28*</td>
<td>NS</td>
<td>NS</td>
<td>0.25*</td>
</tr>
<tr>
<td>Hemorrhage</td>
<td>0.17†</td>
<td>0.11†</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Age</td>
<td>0.14*</td>
<td>NS</td>
<td>0.38†</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Impaired consciousness</td>
<td>−0.14*</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Female sex</td>
<td>0.11†</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Total no.</td>
<td>746</td>
<td>744</td>
<td>70</td>
<td>70</td>
<td>406</td>
</tr>
<tr>
<td>Multiple regression, r value</td>
<td>0.38</td>
<td>0.33</td>
<td>0.44</td>
<td>0.27</td>
<td>0.36</td>
</tr>
<tr>
<td>p value of the model</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>0.052</td>
<td>0.691</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

The variables "cardiac failure," "diabetes mellitus," and "delay until admission" are excluded from the presentation as no significant correlations were found. Standardized coefficients are presented. TIA, transient ischemic attack; SBP, systolic blood pressure; DBP, diastolic blood pressure.

*p<0.001, †0.01, respectively.
significant importance for the high admission blood pressures. The complete lack of correlation between blood pressure on admission and delay between onset of symptoms and arrival at the hospital was remarkable when the well-documented decline of blood pressure after hospitalization is considered.\(^1\)\(^2\) It is generally believed that the early blood pressure rise in acute stroke is a direct and specific reaction to the cerebrovascular event itself, either reflecting a severe neuroendocrine stress response or being a purposeful reaction to hypoperfusion of brain tissue. Our observations challenge this view. Most importantly, we observed that blood pressure levels were similar on hospital admission whether patients entered early or late after onset of symptoms. In addition, the severity of stroke symptoms had no clear relationship to blood pressure levels, because patients with TIA had levels at least as high as those with manifest ischemic lesions and patients with impaired consciousness, indicating extensive lesions, had no higher levels than those with unimpaired consciousness.

With the present data, we propose that admission to the hospital, and the emergency room setting in particular, may cause blood pressure elevation. Thus, acute mental stress may be a major contributor to high blood pressure on hospital admission in stroke patients. Britton et al\(^3\) have demonstrated similar blood pressure responses on hospital admission in patients with acute surgical diseases. However, those elevations were not as pronounced as in the stroke patients, which may reflect the higher prevalence of hypertension in stroke patients in general or an effect of mental stress in the acute phase of stroke.

In conclusion, the high blood pressures on admission had no correlation to the latency between onset of symptoms and admission, which contrasts to the rapid blood pressure decline seen after hospitalization, and could be the result of a blood pressure elevation at admission due to mental stress.

References


Key Words • blood pressure • cerebrovascular disorders • hypertension • stress, psychological
Factors influencing admission blood pressure levels in patients with acute stroke.
B Carlberg, K Asplund and E Hägg

Stroke. 1991;22:527-530
doi: 10.1161/01.STR.22.4.527
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1991 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/22/4/527