Original Contributions

Blood Pressure After Stroke
A One-Year Follow-up Study
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Background and Purpose: Blood pressure changes in the year after acute stroke have been poorly documented.

Methods: We therefore studied blood pressure for 1 year after discharge from the hospital in 226 consecutive patients (mean age, 73 years) surviving an acute stroke.

Results: Marked increases (p<0.001) in mean systolic and mean diastolic blood pressures were seen in two thirds (69%) of the patients 1 month after discharge, and blood pressure remained stable at this level during the remainder of the follow-up year. Similar blood pressure changes were seen irrespective of sex, final stroke diagnosis, or whether the patient had a history of hypertension before the stroke. Patients with a history of hypertension had significantly higher blood pressures (p<0.001) throughout the follow-up year than previously normotensive patients. One month after discharge blood pressure was found to have decreased in 31% of the patients; these were older and had a higher mortality during the follow-up year than patients with blood pressure increases. About 20% of all patients suffered from orthostatism (defined as a decrease in systolic blood pressure of ≥20 mm Hg when rising from the supine position to standing).

Conclusions: We conclude that antihypertensive treatment should not be reduced before discharge from the hospital and that blood pressure should be checked about 1 month after discharge. We suggest that standing blood pressure also be measured to make an appropriate treatment decision. (Stroke 1993;24:195–199)

Key Words • hypertension • hypotension, orthostatic • stroke

High blood pressure (BP) declines spontaneously during the first 4 days after admission to the hospital in patients suffering an acute stroke.1,2 There is no consensus on whether previously instigated antihypertensive treatment should be maintained, reduced, or withdrawn as BP decreases during hospitalization, and the uncertainty persists at discharge. In a study of 63 patients who suffered cerebral infarction or transient ischemic attack (TIA), BP increased after discharge.3

To be able to decide what antihypertensive treatment is necessary after discharge from the hospital following acute stroke and to illustrate the frequency and degree of orthostatic BP reactions in these patients, we prospectively studied the natural BP course over 1 year.

Subjects and Methods

There were 633 consecutive patients with acute stroke admitted to the Department of Medicine, Danderyd's Hospital, Stockholm, between January of 1987 and February of 1988. Of these patients, 258 were treated in the specialized stroke unit (admission criteria: a patient, regardless of age, who without preceding trauma to the head, presented with focal neurological deficits with a duration of no more than 1 week, or patients with TIA during the week before admission). Because the availability of beds in the stroke unit was not sufficient, patients, especially those with stupor or coma, were also admitted to general medical wards. Some TIA patients were discharged directly from the emergency ward after investigation. Thus, the investigated patients constituted a selected stroke material.

To estimate whether the findings concerning our selected material are representative of the whole stroke population, patients admitted to the stroke unit were compared with those admitted to general medical wards. For further comparison, data from another Swedish stroke unit without any exclusion criteria for admission4 are also presented.

Patients were evaluated by means of a standard form including data about previous cardiovascular disease and history of hypertension, current medication, and results of physical examination. It was not possible to attain reliable and comparable figures on BP measurements before the stroke.

Antihypertensive treatment being taken on admission was maintained during the hospital stay and continued at discharge. Of the 93 treated hypertensive patients, 23% were receiving β-blockers, 17% calcium blockers, and 16% diuretics as monotherapy; 44% were taking a combination of drugs. Antihypertensive treatment was not instituted or stopped in any patient during hospitalization or follow-up. BP medication was intensified.

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during follow-up in seven patients (8% of those with treated hypertension).

Patients were seen by one physician three times during the year (at 1, 6, and 12 months) after discharge. At discharge BP was measured at various times during the day and thus was not measured in a standardized manner. Furthermore, many of the patients were anxious about being discharged and this might have affected BP levels. We therefore chose to use BP measured on the morning of the fourth day after hospital admission as the reference value. We have previously shown that BP reaches stable levels by this time. However, we also made analyses with discharge BP as the reference value. The main results were the same, but changes were less pronounced.

BP on day 4 was measured in the morning by the stroke nurse in charge before the patient got out of bed. After discharge all BP recordings were made by one specially trained nurse using a single mercury sphygmomanometer after the patient had rested for 5 minutes. BP was measured in both arms while the patient was lying down. BP was then measured with the patient standing, or if bedridden, sitting up. To counteract regression toward the mean, all BP readings were made twice and the mean value was used in the analyses. Disappearance of the Korotkoff sound (phase V) defined diastolic pressure. Patients admitted to a geriatric hospital or who could not come to the hospital for other reasons were checked at their place of residence. Mean arterial BP is defined as DBP+(SBP−DBP)/3, where DBP is diastolic BP and SBP is systolic BP.

Statistical probability was determined by Student’s paired and unpaired t tests and χ² analysis. A result is considered significant if p<0.05.

Results

Nineteen patients (7.4%) died before discharge, leaving 239 who were discharged alive. Thirteen patients (seven men and six women, 5.4%) were excluded; four had moved abroad, one had moved to another hospital, and eight refused to take part in the study. There were thus 226 survivors (mean age, 73 years) who took part in the study (128 men, mean age 71 years; 98 women, mean age 75 years). Forty-one percent of the patients had a history of hypertension, 20% had suffered a previous TIA or stroke, 16% had atrial fibrillation, 23% had coronary artery disease, and 13% had diabetes mellitus. History, symptoms, examination findings, course of the disease, and final stroke diagnosis were discussed and established by the stroke team at weekly meetings. The distribution of diagnoses is shown in Figure 1.

As expected, the frequency of patients with TIA was lower in the stroke unit than in the whole stroke population (Figure 1) because some patients with TIA were discharged directly from the emergency ward. The admission of fewer patients with stupor and coma caused the mean age of our subjects to be lower and resulted in more men than women and fewer patients with cerebral hemorrhage being admitted; mortality was also lower. More aggressive investigation resulted in more cerebral thromboses or cerebral emboli and fewer undefined strokes being diagnosed in the stroke unit than in the general medical wards. Only minor differences were seen in the mean age and distribution of diagnoses compared with another Swedish stroke unit (Figure 1). However, mortality in our stroke unit was lower (p<0.001).

During follow-up of the 226 study subjects, BP increased (p<0.001) in two thirds. Mean SBP increased from 139 to 160 mm Hg, and mean DBP increased from 78 to 88 mm Hg (Figure 2) from the fourth hospital day to the first checkup at 1 month. (Compared with discharge BP, the corresponding increases were 149 to 160 mm Hg for SBP and 82 to 88 mm Hg for mean DBP.) The rise was seen in both previously normotensive and hypertensive patients, but there was a constant significant difference between the mean values in the two groups (p<0.001) that was seen at all four checkup examinations (Figure 2).

Patients who were older than the mean age had a higher SBP and tendency toward a lower DBP than did patients who were younger than the mean age. Thus the older patients had a significantly higher (p<0.001) pulse pressure. Women had a higher SBP than men (p<0.01). However, when age and history of hypertension were considered there was no difference between the sexes. Nor were there any significant differences in mean BP or the increase in BP at 1 month between patients with various stroke diagnoses or patients suffering their first or a recurrent stroke. There were no significant differences in mean BP at day 4 between patients who died or had a nonfatal recurrence during follow-up and the remainder of the patients. The BP level reached at 1 month remained stable, with no significant change at 6 or 12 months (Figure 3). At 3 months, when only the nurse saw the patients, BP was somewhat lower than the previous and subsequent measurements in all groups.

SBP decreased by >5 mm Hg between day 4 and the 1-month checkup in 31% of the 226 patients. These patients were older and had a higher mortality during follow-up than those with an SBP rise (Figure 4). The
prevalence of a history of hypertension was similar in the two groups.

During the 1 year of follow-up 29 patients (13%) died and 36 suffered a further stroke. There was no significant difference concerning stroke recurrence between previously normotensive and hypertensive patients (14% versus 17%).

Orthostatic reactions, defined as a decrease in SBP of \( \geq 20 \) mm Hg when rising from the supine position to standing, were noted in about 20% of the patients. The mean decrease in SBP in this group was 27 mm Hg (Table 1). The prevalence of orthostatic reactions was constant at all follow-up examinations but tended to be greater in patients whose SBP decreased between day 4 and the first checkup (Table 1). We failed to find any characteristic in the history, BP levels, laboratory findings, or medical treatment that differed between patients with and without orthostatic reactions. Nor were there any differences in mortality or stroke recurrence.

**Discussion**

We found that BP increased considerably in two thirds of stroke patients 1 month after discharge from the hospital, and this increase was seen in patients with and without a history of hypertension. This increase was not dependent on the final stroke diagnosis and occurred equally in patients with cerebral hemorrhage, infarction, or TIA. Mean BP was significantly higher in previously hypertensive patients than in previously normotensive patients throughout the follow-up year.

Our patients were slightly younger than those in some studies. The mean SBP level might therefore be somewhat lower, but our findings should otherwise be representative. We also had fewer patients with cerebral
hemorrhage than other studies, but because BP did not differ in patients with different stroke diagnoses, our results appear to be applicable to patients with various types of acute stroke.

BP remained stable after the initial increase seen at 1 month. The BP level at the 3-month examination was not directly comparable to that at the 1-month or later examinations because the physician did not see the patient at this checkup. The lower SBPs and DBPs recorded at the 3-month examination probably reflect a reverse white-coat reaction.5,6

The increase in mean SBP and DBP up to 1 month after discharge was seen in 69% of our patients. A similar course has been reported in other studies,3,7-9 including studies of 12 patients with essential hypertension (mean age, 53.9 years)7 and 72 patients (mean age, ≤70 years) discharged after myocardial infarction and without antihypertensive treatment.8 An increase in BP after discharge from the hospital following acute stroke was seen in 63 patients with cerebral infarction or TIA reported by Janssen et al.3 In a double-blind, placebo-controlled trial of nimodipine in selected patients with acute stroke an increase in both SBP and DBP was shown in a table but not commented upon.9

The 31% of our patients in whom BP declined after day 4 were older, appeared to be more seriously ill, and had a higher mortality during the follow-up year than those with a BP increase. However, the prevalence of a history of hypertension or congestive heart disease was not higher in these patients.

Dobkin10 reported orthostatism to be a risk for the development of stroke. Thirteen of 80 consecutive stroke patients had orthostatic BP reactions defined as a decrease in SBP of >20 mm Hg when rising from the supine position to standing. There appear to be no other reports of orthostatic reactions in stroke patients. The prevalence of these reactions was about 20% in our study, and we caution that orthostatic reactions be taken into consideration when deciding antihypertensive treatment.

The marked increase in BP that occurred in the majority of our patients after discharge from the hospital suggests that antihypertensive treatment should not be reduced during hospitalization or at discharge except in obviously hypotensive patients. The BP during a hospital stay with bed rest and relative inactivity should not be considered as the usual resting BP. Treatment should therefore be adjusted at an outpatient follow-up

![Graph](https://example.com/graph.png)

**FIGURE 4.** Bar graph showing characteristics of stroke patients with decrease in systolic blood pressure (filled bars, n=69) at first checkup compared with remaining patients (shaded bars, n=157). *p<0.05.

**TABLE 1.** Frequency of Orthostatic Reaction Among All Patients and Those With Decreasing or Increasing SBP From Day 4 to 1 Month After Stroke

<table>
<thead>
<tr>
<th>Month</th>
<th>Mean±SD SBP decrease for patients with orthostatic reaction (mm Hg)</th>
<th>Patients with orthostatic reaction (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All patients (n=226)</td>
<td>Patients with SBP decrease (n=69)</td>
</tr>
<tr>
<td>1</td>
<td>26±19</td>
<td>23</td>
</tr>
<tr>
<td>3</td>
<td>27±11</td>
<td>17</td>
</tr>
<tr>
<td>6</td>
<td>27±10</td>
<td>23</td>
</tr>
<tr>
<td>12</td>
<td>33±26</td>
<td>23</td>
</tr>
</tbody>
</table>

SBP, systolic blood pressure; orthostatic reaction, SBP decline of ≥20 mm Hg when rising from supine position to standing.
examination 1 month after discharge. If the patient has
an orthostatic BP reaction, BP in the standing position
determines what treatment can be tolerated.

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