Factors Affecting Changes in Blood Pressure After Acute Stroke

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**Background and Purpose** We sought to establish the pattern of blood pressure (BP) change after hospitalization for acute hemispheric stroke.

**Methods** In 292 patients from the Leicester teaching hospitals with acute hemispheric stroke within the previous 24 hours (139 men; median age, 75 years [range, 42 to 98 years]), we prospectively studied BP changes between admission, 24 hours, 1 week, and 4 to 6 weeks. Changes were assessed in relation to main stroke risk factors, stroke type and severity, and antihypertensive drug treatment. All subjects were followed up for 1 week, with 117 subjects followed up for 4 to 6 weeks. Changes were assessed by repeated-measures ANOVA, and Student’s *t* tests were used to compare group pairs.

**Results** Systolic and diastolic BP fell by 12 mm Hg (95% confidence interval [CI], 8 to 15 mm Hg) and 7 mm Hg (95% CI, 5 to 9 mm Hg), respectively, in the first 24 hours and 22 mm Hg (95% CI, 18 to 25 mm Hg) and 12 mm Hg (95% CI, 10 to 14 mm Hg), respectively, during the first week (all changes significant at *P*<.01) but no further thereafter. In those patients receiving no antihypertensive medication before or after stroke, the pattern of change was similar to that of the whole group. Previously diagnosed hypertensive subjects (n=106) had higher initial BP values than did normotensive subjects, although by 1 week the levels were not significantly different. Patients with cerebral hemorrhage confirmed by computed tomography (n=20) had higher systolic BP, but not diastolic BP, throughout the first week than those with cerebral infarction (n=89). The severity of stroke, age, and previous stroke history did not appear to alter the BP pattern. Stroke patients who were moderate to heavy alcohol consumers had lower convalescent systolic BP levels than lighter drinkers or abstainers.

**Conclusions** We have demonstrated a marked fall in systolic and diastolic BP levels during the first 7 days after acute hemispheric stroke, with little change thereafter. Higher initial systolic BP values were found in patients with cerebral hemorrhage compared with those with cerebral infarct. Moderate to heavy alcohol consumption before stroke was associated with a greater systolic BP decline in the first week after the event compared with stroke patients who were light drinkers or abstainers. (Stroke. 1994;25:1726-1729.)

**Key Words** • alcohol drinking • blood pressure • cerebral hemorrhage • cerebral infarction

Previous studies on the changes in blood pressure (BP) after acute stroke have shown relatively high early readings, which subsequently fall.1-4 The mechanisms for these changes are unclear but may be related to stroke-induced changes in sympathoadrenal activity,5,6 stress reaction to hospital admission or BP measurement, or central mechanisms.7,8 An increase in BP in the acute stage of cerebral ischemia could be a pathophysiological response to maintain or enhance perfusion of reversibly damaged cerebrum, where normal autoregulatory mechanisms are impaired.9-11 Lowering BP at this early stage, either intentionally or secondary to other therapy, could perhaps result in further cerebral damage by directly reducing perfusion to the compromised tissues.12,13 Most authorities would delay treatment of all but sustained malignant hypertension until this risk is past.9,14

Data as to when BP finally stabilizes after acute stroke are not available, and thus it is not known when measured values become representative. Wallace and Levy1 followed up subjects until day 10 after stroke, at which time BP still appeared to be falling. Other investigators have demonstrated a continued fall between day 4 and discharge, but during this period high BP levels were treated, and the actual time to discharge was not reported.2 Factors that are known to influence BP levels in nonstroke subjects, eg, alcohol consumption, have not been studied in regard to BP changes after stroke.

Therefore, the aims of the study were to assess (1) the factors influencing BP changes between admission and day 7 after acute mild or moderate hemispheric stroke and (2) BP levels on day 7 and after prolonged follow-up at 4 weeks after hospital admission.

**Subjects and Methods**

The Leicester Hospitals Stroke Register was established in July 1989.12 All patients admitted to the four Leicester hospitals who had a stroke within the previous 24 hours were seen by one investigator (G.H.), and an extensive questionnaire was completed assessing past and current medical problems, the stroke itself, and progress at fixed intervals after stroke onset. As part of this exercise the BP readings at admission, 24 hours, 1 week, and 4 to 6 weeks were recorded. If possible the mean of three or more readings was taken as the value for that day. The readings used were those recorded by the resident nursing and medical staff using a conventional mercury sphygmomanometer with the patient supine or semi-supine, with diastolic BP taken at Korotkoff phase V. No attempt was made to use a random-zero sphygmomanometer or automated device because the study was primarily an observation of routine clinical measurement. Timings of the BP recordings were kept as similar as possible during the study.
period. The 4- to 6-week follow-up BP measurement was made in all patients still resident in the hospital (ie, excluding those who had died between day 7 and follow-up and those discharged). One- and 4-week overall mortality rates were 12% and 25%, respectively, for all patients in the stroke register.

The study was designed to assess only those patients with a definite mild to moderate hemispheric stroke (World Health Organization definition) admitted within 24 hours of the event. Patients with a transient ischemic attack, subarachnoid hemorrhage, atypical presentation of neurological deficit, or in sustained coma and those discharged or dying within 1 week were excluded, as were patients with acute heart failure, acute myocardial infarction, or other serious illness. Computed tomography was not routinely used for all strokes in Leicester at the time of the study. However, the scoring systems used to distinguish hemorrhage from infarct depend on BP values and hypertension history and would not be suitable for this study. Thus, this distinction has been made only on those strokes with definite computed tomographic diagnosis.

Subjects labeled hypertensive on admission had a medical history of elevated BP levels and/or were receiving or had received antihypertensive medication. Antihypertensive therapy given during the study period included all recognized agents even when used for other reasons, eg, β-blockers for rhythm disorders. Individual clinicians were allowed to institute antihypertensive treatment at any time after stroke, as clinically appropriate.

Alcohol consumption was recorded as moderate for a weekly consumption of 14 or more units for men or 7 or more units for women. Severity of stroke was judged by the power in the affected limb 1 week after stroke. A Medical Research Council power grade of 3 or less in limb flexion at 1 week constituted a moderate to severe stroke.

The results were analyzed with MINITAB and SAS statistical software on a desktop computer. The overall change in BP over time and between groups was assessed with repeated-measures ANOVA. If an overall difference was found, the changes between time points and groups were assessed by Student’s t tests with Bonferroni correction for multiple comparisons. Univariate analysis was performed with Pearson’s correlation and multivariate comparisons with multiple linear regression. Data are presented as mean±SD; 95% confidence intervals (CI) are provided where indicated. Significance was set at the 5% level.

Results

Of the 292 patients admitted within 24 hours of their stroke and followed up for 1 week, 117 had BP measurements repeated 4 to 6 weeks later. The age, sex, BP levels, and other characteristics of the study group are shown in the Table. There was no significant difference in any of these parameters between those followed up for 1 week and those seen at 4 to 6 weeks. Thirty percent of the whole group were smokers. Male stroke subjects were younger and more likely to be heavy alcohol consumers (Table). Those diagnosed as hypertensive before admission were older than normotensive subjects (age difference, 4.0 years; 95% CI, 2 to 7 years; P<.002).

For the total group systolic BP fell by 12 mm Hg (95% CI, 8 to 15 mm Hg) and diastolic BP by 7 mm Hg (95% CI, 4 to 10 mm Hg) from admission to 24 hours and by 22 mm Hg (95% CI, 18 to 25 mm Hg) and 12 mm Hg (95% CI, 10 to 14 mm Hg), respectively, from admission to 1 week (P<.01) (Figure). These changes were not influenced by age, severity of stroke, or history of stroke, but men at day 7 had a lower systolic BP than women (difference, 6 mm Hg; 95% CI, 1 to 11 mm Hg; P<.05), although on admission there was no sex difference. There was no change in BP between day 7 and day 28 (Figure) in the 117 patients studied. In those with a definite cerebral hemorrhage, systolic BP but not diastolic BP on admission was higher (difference, 19 mm Hg; 95% CI, 4 to 45 mm Hg; P<.02) and showed a greater fall over time (P<.01, repeated-measures ANOVA) than those with cerebral infarction. At the 1-week and 1-month follow-ups there was no difference in BP levels between stroke types. The diastolic BP differences showed a similar trend but did not reach statistical significance (P=.07).

There was no difference in BP levels between moderate to heavy alcohol consumers (169/99 mm Hg) and...
light drinkers or nondrinkers (168/96 mm Hg) on admission, although the fall in systolic BP during the 7 days was greater in the heavy drinkers than in the light drinkers and nondrinkers (P<.01, repeated-measures ANOVA). The difference between the light and heavy alcohol consumers at day 7 was 7 mm Hg (95% CI, 0 to 13 mm Hg; P<.05), but the differences in diastolic BP at this time were not significant. The mean age of patients in the high alcohol intake group was 7 years younger (P<.005).

Patients with mild strokes (n=148) showed no difference in BP profile from those with more severe strokes. Although there was a tendency for BP to be higher in patients aged 75 years or older, this only became significant at 1 month. There was no difference in BP between those with first and recurrent strokes during the first week. Patients previously diagnosed as hypertensive had higher systolic and diastolic BP levels up to day 7 (P<.05 and P<.005, respectively, repeated-measures ANOVA) than normotensive subjects. The fall in diastolic BP over time in the hypertensive group was also greater (P<.01, repeated-measures ANOVA). There was a weak but significant correlation between admission systolic BP and previous diagnosis of hypertension (r=−.23, P=03) and the presence of cerebral hemorrhage (r=−.24, P=.04) on stepwise multiple regression analysis. Admission diastolic BP also correlated significantly with a history of hypertension (r=−.25, P<.001).

Patients continuing with (n=66) or started on (n=24) antihypertensive medication after their stroke showed a greater fall in systolic BP during the first 24 hours (19 versus 10 mm Hg; P<.04) and the first week (31 versus 19 mm Hg; P=.01) than those on no medication.

Discussion

We have shown that BP falls during the first 7 days after admission in a selected group with acute ischemic stroke, but it is unlikely to decrease further thereafter. Although individual variations in these BP changes were wide, the fundamental trend is for a steady fall after acute stroke. In those patients with cerebral hemorrhage, this fall may be sustained beyond the first week. The present pattern of BP change after stroke reflects the findings of others,1,4,17,18 but our study does differ quite markedly from other published work in several aspects. First, we included all patients with acute stroke and not just those selected for admission to a specialist Stroke Unit.1,4,17 With the inherent problems of bias this may impose. Second, BP readings were from patients who had sustained a stroke within the previous 24 hours, whereas other studies have included those who had had a stroke in the previous 7 to 14 days before admission.3,4,19 The timing of BP recording is vitally important; as we have shown, there is little change in BP after 7 days poststroke. If patients are included at a later time after stroke, the BP changes demonstrated are likely to be smaller and are probably more a reflection of the BP changes to hospital admission itself than to stroke.20

The prognostic value of initial elevated BP levels after acute stroke is unclear. Carlberg et al21 found admission BP to be unrelated to outcome except in those with impaired conscious levels, in whom an increased BP was associated with a worse prognosis. Britton and Carlsson22 also found that those with a very high BP on admission had greater mortality, whereas Allen19 reported that higher systolic BP levels on admission but not at 24 hours after admission indicated a good outcome. Therefore, the value of treating an elevated BP at this stage after stroke remains unclear. However, most general practitioners continue antihypertensive treatment during the initial phase of stroke.23 There is a theoretical advantage in delaying antihypertensive therapy after stroke in that it allows recovery of the damaged cerebral vessels, restoration of local autoregulation, and improvement in the collateral supply.11–15 but at this time the prevalence and duration of these changes are unknown in humans. If these changes persist indefinitely in some individuals, BP reduction after stroke may lead to a poorer outcome in such patients. This in turn may offset any benefit that may be gained in the reduction of further events and may account for the lack of a clear relation of BP control in the months after stroke and poststroke mortality.24 The dangers of overaggressive lowering of BP in the early stages are also well recognized.13 Severe hypertension after mild stroke may increase the risk of further stroke and death.25 Our study suggests that 7 days after the initial event BP levels are unlikely to change in the next 4 weeks while the patient is hospitalized.

A recent study has demonstrated a clear and sustained rise in BP following hospital discharge after acute stroke.26 This increase was probably due in part to the alerting or “white-coat” effect sustained by patients when they attended the follow-up clinics; this response can be abolished by 24-hour BP monitoring.18 The slight BP rise noted at 1 month in some subgroups in our study did not reach significance, and it remains unclear whether the lower hospital readings are more representative of final BP load than the higher clinic results.

The lower convalescent BP in the moderate/heavy alcohol group before stroke has not been previously described. Regular alcohol consumption is known to have a pressor effect in both normotensive27 and hypertensive28 subjects. The BP changes demonstrated suggest that alcohol abstention in moderate/heavy drinkers results in a substantial fall in BP over the poststroke period. The rapid effect of alcohol abstention on BP has previously been shown in hospitalized hypertensive subjects.28 Heavy alcohol consumption has been shown in some studies to be a risk factor for stroke,29 although from the way the data were gathered in this study we have no information on the relation between more recent alcohol intake (within 48 hours before stroke onset) compared with alcohol intake in the week before stroke. The actual mechanisms relating to the effect of alcohol on stroke are unknown but could be related to changes in BP, direct cerebral vasoconstrictor effects, changes in platelet function, or alteration of coagulation factors.

In conclusion, our findings show that high early BP values observed after cerebral infarction are not generally sustained beyond the first week. Patients with hypertension or cerebral hemorrhage are more likely to have higher values at 1 week, although antihypertensive treatment results in a further fall. Therapeutic reduction of BP immediately after stroke leads to a more precipitous fall in the early period, which may be a dangerous and an unnecessary intervention.

References


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