Systolic Blood Pressure and Mortality After Stroke
Too Low, No Go?
Michelle P. Lin, MD, MPH; Bruce Ovbiagele, MD, MSc, MAS; Daniela Markovic, MS; Amytis Towfighi, MD

Background and Purpose—Recent studies suggest a J-shaped association between systolic blood pressure (SBP) and cardiovascular events. The optimal SBP target after stroke remains unknown. We assessed the link between SBP and mortality after stroke.

Methods—We included adults (≥20 years) with self-reported stroke who participated in the National Health and Nutrition Examination Surveys 1998 to 2004, with mortality assessment in 2006. Baseline SBP was categorized as low to normal (<120 mm Hg), normal (120–140 mm Hg), and high (≥140 mm Hg). Independent relationships between baseline SBP and all-cause and vascular mortality were assessed using Cox proportional hazards.

Results—Of 31,126 adult participants, 455 had self-reported stroke and baseline BP readings: 19% had low to normal, 31% had normal, and 50% had high SBP. Two years after assessment, the low to normal SBP group tended to have the highest cumulative all-cause mortality (11.5%), compared with mortality rates of 8.5% and 7.5% in the normal and high SBP groups, respectively. Similar patterns were seen with vascular mortality. After adjusting for covariates, compared with the high SBP group, the low to normal group had higher all-cause mortality (adjusted hazard ratio, 1.96; 95% confidence interval, 1.13–3.39; P=0.017) and trended toward higher vascular mortality (adjusted hazard ratio, 2.08; 95% confidence interval, 0.93–4.68; P=0.075). Compared with the normal BP group, the risk of all-cause and vascular mortality trended higher in low to normal BP group but did not achieve statistical significance.

Conclusions—After stroke, compared with SBP in the high range, low to normal SBP is associated with poorer mortality outcomes. (Stroke. 2015;46:1307-1313. DOI: 10.1161/STROKEAHA.115.008821.)

Key Words: blood pressure ■ hypertension ■ mortality ■ Nutrition Surveys ■ secondary prevention ■ stroke

Hypertension is the most important modifiable risk factor for stroke, accounting for 35% to 52% of the population attributable risk of stroke. Benefit has been associated with an average reduction of 10/5 mm Hg,2,4 and observational data suggest that there is a linear association between lowering blood pressure (BP) and primary stroke prevention until 115/75 mm Hg when the cerebrovascular protective effects of lowering BP turn to harm.5–7 Optimal BP targets after stroke, however, remain uncertain. A post hoc analysis of the Perindopril Protection against Recurrent Stroke Study (PROGRESS), which randomized 6105 patients with a history of transient ischemic attack or stroke to a perindopril-based regimen or placebo revealed that the effectiveness of hypertension therapy for secondary stroke prevention diminished as the baseline BP declined. Specifically, the relative risk reduction of mortality for those with a baseline SBP of ≥160, 140 to 159, 120 to 139, or <120 mm Hg was 39%, 31%, 14%, and 0%, respectively.8 Studies in the acute stroke setting have shown no benefit of lowering BP and a suggestion of harm. First, 1 analysis found that low to normal SBP was associated with a higher risk of early recurrence by 2 weeks and poor functional outcome at 6 months compared with high and normal SBP.9 Second, results of a randomized trial in patients with acute stroke and raised BP levels (SBP ≥140 mm Hg) suggested a trend toward greater risk of poor functional outcome at 180 days after careful BP-lowering treatment was initiated within 30 hours of the index stroke.10 Third, the recent China Antihypertensive Trial in Acute Ischemic Stroke (CATIS) trial showed that immediate BP reduction by 10% to 25% within 24 hours of acute ischemic stroke or achieving BP <140/90 mm Hg within 7 days of acute ischemic stroke had no mortality or morbidity benefits.11

Received January 21, 2015; accepted February 18, 2015.

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Guest Editor for this article was Natan M. Bornstein, MD.

The online-only Data Supplement is available with this article at http://stroke.ahajournals.org/lookup/suppl/doi:10.1161/STROKEAHA.115.008821/-/DC1.

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Stroke is available at http://stroke.ahajournals.org DOI: 10.1161/STROKEAHA.115.008821
Beyond the acute stroke setting, 2 post hoc analyses of randomized controlled trials implied that there may be a J-shaped relationship between BP and recurrent vascular events within 2 years of an index stroke; poor outcomes were observed in both extremes of BPs.12,13 Both studies found that the association was more pronounced in the first 6 months after stroke.

Most recently, an observational community-based study involving 483 participants with stroke and 10-year follow-up reported a 61% increased risk of combined vascular outcome (stroke, acute myocardial infarction, and death) among those with SBP \( \leq 120 \) mm Hg compared with reference group of SBP 131 to 141 mm Hg.14

In this study, we used a nationally representative sample of US adults (\( \geq 20 \) years) who participated in the National Health and Nutrition Examination Surveys (NHANESs) III (1998–2004) with self-reported history of stroke to explore the effect of baseline SBP (during a single outpatient NHANES evaluation) on all-cause and vascular mortality, assessed in 2006.

### Methods

#### Study Population

NHANESs are a series of cross-sectional, national, stratified, multi-stage probability surveys constituting representative samples of the civilian, noninstitutionalized US population. The surveys and examinations are conducted by the National Center for Health Statistics. Each survey participant completed a household interview and underwent a physical examination. Detailed descriptions of the plan and operation of each survey have been published.11 The study received approval from the National Center for Health Statistics Research.

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**Table 1. Survey-Weighted Demographic and Clinical Characteristics of Stroke Survivors by Mean Systolic Blood Pressure (<120, 120–139, and \( \geq 140 \) mm Hg), National Health and Nutrition Examination Survey 1998 to 2004**

<table>
<thead>
<tr>
<th>SBP &lt;120 mmHg (n=88), 19%</th>
<th>SBP 120–139 mmHg (n=141), 31%</th>
<th>SBP ( \geq 140 ) mmHg (n=226), 50%</th>
<th>( P ) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean, y</td>
<td>54.9±2.1</td>
<td>61.8±1.7</td>
<td>70.8±1.1</td>
</tr>
<tr>
<td>Sex, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>46.9</td>
<td>47.5</td>
<td>33.9</td>
</tr>
<tr>
<td>Female</td>
<td>53.1</td>
<td>52.5</td>
<td>66.1</td>
</tr>
<tr>
<td>Race, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mexican American</td>
<td>4.8</td>
<td>3.3</td>
<td>2.9</td>
</tr>
<tr>
<td>Hispanic/other</td>
<td>10.2</td>
<td>7.0</td>
<td>7.5</td>
</tr>
<tr>
<td>White non-Hispanic</td>
<td>76.4</td>
<td>76.9</td>
<td>72.2</td>
</tr>
<tr>
<td>Black non-Hispanic</td>
<td>8.6</td>
<td>12.8</td>
<td>17.3</td>
</tr>
<tr>
<td>Poverty income ratio, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;125</td>
<td>28.1</td>
<td>30.8</td>
<td>34.8</td>
</tr>
<tr>
<td>125–200</td>
<td>16.2</td>
<td>21.1</td>
<td>16.0</td>
</tr>
<tr>
<td>200–400</td>
<td>25.5</td>
<td>26.1</td>
<td>28.9</td>
</tr>
<tr>
<td>&gt;400</td>
<td>25.6</td>
<td>15.9</td>
<td>13.3</td>
</tr>
<tr>
<td>Unknown</td>
<td>4.6</td>
<td>6.1</td>
<td>7.1</td>
</tr>
<tr>
<td>Comorbidities, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>49.2</td>
<td>65.1</td>
<td>81.5</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>13.3</td>
<td>16.8</td>
<td>28.9</td>
</tr>
<tr>
<td>Hypercholesterolemia*</td>
<td>39.9</td>
<td>40.2</td>
<td>57.4</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>19.8</td>
<td>18.4</td>
<td>18.0</td>
</tr>
<tr>
<td>Angina</td>
<td>16.0</td>
<td>17.4</td>
<td>20.8</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>13.8</td>
<td>14.5</td>
<td>17.8</td>
</tr>
<tr>
<td>Using antihypertensive medication</td>
<td>29.3</td>
<td>51.3</td>
<td>68.0</td>
</tr>
<tr>
<td>Body mass index, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;25 kg/m²</td>
<td>22.7</td>
<td>17.5</td>
<td>27.4</td>
</tr>
<tr>
<td>25–29 kg/m²</td>
<td>28.7</td>
<td>35.0</td>
<td>31.8</td>
</tr>
<tr>
<td>≥30 kg/m²</td>
<td>40.4</td>
<td>39.8</td>
<td>30.8</td>
</tr>
<tr>
<td>Unknown</td>
<td>8.2</td>
<td>7.7</td>
<td>10.1</td>
</tr>
<tr>
<td>Smoking, %</td>
<td>36.3</td>
<td>25.2</td>
<td>18.6</td>
</tr>
<tr>
<td>Mean SBP, mm Hg</td>
<td>109.3±1.1</td>
<td>129.1±0.7</td>
<td>160.7±1.6</td>
</tr>
<tr>
<td>Mean DBP, mm Hg</td>
<td>68.1±1.5</td>
<td>70.9±1.0</td>
<td>74.8±1.7</td>
</tr>
</tbody>
</table>

Hypertension, diabetes mellitus, coronary artery disease, angina, and congestive heart failure were defined by self-reported history. DBP indicates diastolic blood pressure; and SBP, systolic blood pressure.

*Total cholesterol ≥200 mg/dL.
SBP indicates systolic blood pressure.

Ethics Review Board, and participants were asked to sign an informed consent form. The sample included adults (≥20 years) with self-reported stroke (n=455) followed from survey participation (1998–2004) to mortality assessment in 2006. The definition of stroke used in the NHANES III survey was based on self-reported physician diagnosis.

Primary Outcome: Mortality
To calculate all-cause and vascular mortality rates, we used data from the NHANES III Linked Mortality File, in which NHANES III–eligible participants were matched, using a probabilistic matching algorithm, to the National Death Index through December 31, 2006, to determine their mortality status. The International Classification of Diseases Tenth Revision, codes I00–I99, were used to identify deaths from diseases of the vascular system. Participants not matched with a death record were considered alive through the entire follow-up period. A detailed description of the methodology is described elsewhere.16

BP Measurements
BP was obtained in accordance with the NHANES procedure manual.17 Briefly, after the patient had rested quietly in a seated position for 5 minutes, the maximum inflation level was determined, and 3 consecutive BP readings were obtained. If a BP measurement was interrupted or incomplete, a fourth attempt was made. Mean SBP of the measurements was used in the analyses.17

Statistical Analyses
Subjects were categorized by prespecified groups according to their mean of 3 SBP readings obtained at the NHANES visit: low to normal (<120 mm Hg), normal (120–140 mm Hg), and high (≥140 mm Hg). The SBP groups were chosen to reflect levels mentioned in guideline recommendations,3,4 for consistency with other studies,12,13,18,19 and practical considerations in routine clinical settings. Baseline sociodemographic and clinical characteristics were assessed by SBP group. The primary outcome was all-cause mortality and the secondary outcome was vascular mortality. Cumulative incidence curves of all-cause mortality were computed by SBP group using the Kaplan–Meier method and weighted appropriately. Cumulative incidence curves of vascular mortality were computed similarly after adjusting for the competing risk of nonvascular mortality. Models were fitted using SBP as categorical and continuous variables. To account for nonlinearity, a linear and a quadratic SBP term were included in the models.

To examine the association between SBP and all-cause and vascular mortality rates after stroke, univariate and multivariate analyses were performed using Cox regression models after adjusting for the survey design variables (ie, variables extracted from the NHANES surveys). For vascular mortality, the Cox model was expanded to a competing risk Cox model to adjust for the competing risk of nonvascular mortality. Models were fitted using SBP as categorical and continuous variables. To account for nonlinearity, a linear and a quadratic SBP term were included in the models.

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mortality were age, poverty:income ratio, diabetes mellitus, coronary artery disease, and smoking. Furthermore, the final Cox models also allowed for potential interactions of each SBP category with time (nonproportional hazards) by including the appropriate interaction terms into the models. Specifically, we tested for interactions of each SBP category with (1) log of time and (2) time categorized as <2, 2 to 4, and >4 years. The latter analysis did not impose any functional form (such as linearity) on the relationship between SBP category and the time variable in predicting mortality. The above interactions with time were evaluated at P<0.15 as the level of significance using the χ² test under the above Cox regression models. Finally, to evaluate the goodness of fit of the final regression model, we used methods by Hosmer and Lemeshaw adapted for time to event data.20 We excluded 45 participants from the analyses because of missing SBP data. This comprised 9% of the initial sample. To assess the potential influence of missing data on the results, we compared covariates between people with and without missing data to determine whether the distributions were comparable in the 2 groups. In addition, missing values for the covariates were imputed using the Markov Chain Monte Carlo method of imputation for the purpose of the multivariable analyses.21

Results

A total of 31 126 adults participated in NHANES III (1988–1994). Five hundred ninety reported history of stroke, of whom 135 were excluded for missing SBP data, nonpositive survey weights, or missing follow-up data. The final cohort consisted of 455 subjects: 88 (19%) had low to normal SBP, 141 (31%) had normal SBP, and 226 (50%) had high SBP. Compared with individuals with low to normal SBP, individuals with high SBP were older; more likely to be women, black, and poor; to have a history of hypertension, diabetes mellitus, and
hypercholesterolemia; and to use antihypertensive medications. They were less likely to be obese or to smoke (Table 1).

Mortality assessment was at a mean follow-up of 4.1 years (2.6–5.7 years). At 2 years, patients in the low to normal SBP group tended to have higher cumulative all-cause mortality (11.5%) compared with those with normal SBP (8.5%) and high SBP (7.6%; Tables 2 and 3; Figure I in the online-only Data Supplement). The pattern for vascular mortality was similar: 5.0% in the low to normal SBP group, 4.5% in the normal SBP group, and 4.1% in the high SBP group. This association reversed with time from baseline evaluation. At 6 years, individuals with high baseline SBP tended to have the highest cumulative all-cause mortality (36.5%), compared with 30.0% and 19.6% in normal and low to normal SBP groups. A similar pattern was evident for vascular mortality, with mortality rates of 20.2% for high baseline SBP, 12.2% for normal baseline SBP, and 9.1% for low to normal baseline SBP (Tables 2 and 3; Figure I in the online-only Data Supplement).

We also assessed the SBP–mortality relationship using SBP as a continuous variable, and similarly found a significant correlation with all-cause mortality ($P=0.009$ for the linear term, $P=0.027$ for the quadratic term). There was a J-shaped relationship between SBP and all-cause mortality after adjusting for covariates (Figure [A]). Both low SBP and high SBP (approximately >180 mm Hg) were associated with greater risk of mortality compared with SBP in the normal or moderately high range (Figure [A]). Similar patterns were seen with cardiovascular mortality but were not as robust ($P=0.12$ for the linear, $P=0.11$ for the quadratic term). The J-shaped relationship between SBP and 5-year risk of cardiovascular mortality was similarly demonstrated using the quadratic model (Figure [B]).

In unadjusted analyses, there were no significant differences in the all-cause (low versus normal: HR, 0.86; 95% confidence interval [CI], 0.46–1.59; high versus normal: HR, 1.05; 95% CI, 0.66–1.67) or vascular (low versus normal: HR, 0.74; 95% CI, 0.30–1.85; high versus normal: HR, 1.28; 95% CI, 0.61–2.69) mortality rates across SBP groups averaging over follow-up time (Table 4). None of the interactions of SBP category with time were significant at $P<0.15$.

After adjustment for covariates, compared with the high SBP group, individuals with low to normal SBP had higher all-cause mortality (adjusted hazard ratio [AHR], 1.96; 95% CI, 1.13–3.39) and vascular mortality (AHR, 2.08; 95% CI, 0.93–4.68; Table 4). Compared with individuals with normal SBP, those with low to normal SBP had higher all-cause mortality (adjusted HR, 1.96; 95% CI, 1.13–3.39) and vascular mortality (AHR, 2.08; 95% CI, 0.93–4.68; Table 5). Compared with individuals with normal SBP, those with low to normal SBP had higher all-cause mortality (adjusted HR, 1.96; 95% CI, 1.13–3.39) and vascular mortality (AHR, 2.08; 95% CI, 0.93–4.68; Table 5).
and vascular mortality but neither of these associations were significant (AHR, 1.43; 95% CI, 0.82–2.50 and AHR, 1.71; 95% CI, 0.75–3.90). Individuals with high baseline SBP had lower all-cause and vascular mortality compared with those with normal SBP (AHR, 0.73; 95% CI, 0.45–1.18 and AHR, 0.82; 95% CI, 0.41–1.62), but these associations were not significant. To test the predictive accuracy of the final model, we applied that goodness-of-fit test to compare the predicted versus observed 5-year probabilities of all-cause mortality by decile and found that $P=0.11$ demonstrating good agreement (Table 1 in the online-only Data Supplement).

Assessment of the covariates revealed that older age, poverty, and diabetes mellitus were associated with higher all-cause mortality and older age, poverty, and coronary artery disease were associated with higher vascular mortality (Table 5). The AHR of aging per year was 1.06 (95% CI, 1.03–1.09) for all-cause mortality and 1.10 (95% CI, 1.06–1.13) for vascular mortality. Individuals with income:poverty ratio >400% had 60% lower all-cause mortality (AHR, 0.40; 95% CI, 0.18–0.90) and 86% lower vascular mortality (AHR, 0.14; 95% CI, 0.03–0.72) compared with those with a ratio <125%. Diabetes mellitus was associated with higher all-cause mortality (AHR, 1.77; 95% CI, 1.01–3.08), but not vascular mortality (AHR, 1.43; 95% CI, 0.55–3.73). Coronary artery disease was associated with higher vascular mortality (AHR, 2.16; 95% CI, 1.08–4.32), but not all-cause mortality (AHR, 1.55; 95% CI, 0.84–2.85; Table 5). Current smokers tended to have higher all-cause and vascular mortality than their counterparts who did not smoke, but this association was not significant beyond chance (Table 5). Comparison of covariates between people with and without missing SBP data revealed a similar distribution of covariates. Furthermore, results using Markov Chain Monte Carlo imputation and additional sensitivity analyses closely agreed with results using complete cases on the covariates.

Discussion

In this community-based study of participants from the NHANES 1998 to 2004, we found that among individuals with a self-reported history of stroke, those with baseline SBP in the low to normal range had higher all-cause mortality and trended toward higher vascular mortality than individuals with normal SBP and high SBP, after adjusting for major sociodemographic and vascular risk factors during a mean follow-up of 4.1 years (2.6–5.7 years).

Our results support emerging data suggesting that there may indeed be a threshold beyond which further harm occurs with BP lowering.\textsuperscript{12,13,18,19,22} Two recent post hoc analyses of randomized controlled trials of secondary stroke prevention revealed that risk of recurrent stroke was higher in those with SBP <120 compared with those with SBP 120 to 140 mm Hg.\textsuperscript{12,13} A systematic review and meta-analysis of 11 published randomized controlled trials\textsuperscript{22} revealed that achieving SBP <130 mm Hg seemed to provide additional stroke protection only among people with risk factors but no established cardiovascular disease. Those with established cardiovascular disease at entry did not experience stroke risk reduction with tight BP control. BP control was associated with hypotension (relative risk, 3.43; 95% CI, 2.46–4.79) and occurrence of adverse events (relative risk, 1.18; 95% CI, 1.11–1.25).\textsuperscript{22}

Our study has several strengths: we used a large sample of individuals in the United States, with rigorous, validated methods for BP measurements, and information on a variety of comorbidities. This study also has several limitations. First, because stroke was assessed by self-report, we lacked information on stroke type, duration since stroke, stroke severity, and functional status; these factors, which may have had an effect on mortality, were not controlled for. Second, although BP was measured at the time of the NHANES examination, there were no subsequent BP measurements (because NHANES is a cross-sectional survey/examination). Third, NHANES only captures noninstitutionalized individuals and those who can comprehend and respond to surveys, resulting in a possible bias toward a healthier population. Fourth, missing data could introduce unaccounted selection bias; however, missing data analyses suggested that covariates were similar in the groups with versus without BP data and the Markov Chain Monte Carlo imputation demonstrated close agreement with study results. Fifth, patients with low to normal BP were younger, hence may have had a different stroke risk profile than older patients. This may have introduced confounding even after adjustment for age.

BP goals after stroke remain uncertain, given the limited data on outcomes. Forthcoming trials, including Secondary Stroke Prevention by Uniting Community and Chronic Care Model Teams Early to End Disparities (SUCCEED),\textsuperscript{23} Enhanced Control of Hypertension and Thrombolysis Stroke Study (ENCHANTED),\textsuperscript{24} Efficacy and Nitric Oxide in Stroke (ENOS)\textsuperscript{25} and Optimal Blood Pressure and Cholesterol Targets for Preventing Recurrent Stroke in Hypertensives (ESH-CHL-SHOT)\textsuperscript{26} may help further elucidate BP goals and the timing to achieve target BP. Meanwhile, new secondary stroke prevention guidelines support a target BP of <140/90 mm Hg and suggest individual tailoring of BP therapy combined with lifestyle modifications.\textsuperscript{27,28} Probability of success will likely increase if interventions are culturally tailored and take into account race/ethnic, sex-specific, educational, and socioeconomic factors.\textsuperscript{27}

Disclosures

None.

References

Lin et al  Blood Pressure and Mortality After Stroke 1313


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Stroke. 2015;46:1307-1313; originally published online March 12, 2015;
doi: 10.1161/STROKEAHA.115.008821
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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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/46/5/1307

Data Supplement (unedited) at:
http://stroke.ahajournals.org/content/suppl/2015/03/12/STROKEAHA.115.008821.DC1
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SUPPLEMENTAL MATERIAL
Supplemental Figure I. Cumulative incidence of (A) all-cause death, (B) vascular death by SBP group for persons with self-reported history of stroke, NHANES 1999-2004.
**Supplemental Table I.** Goodness of fit test using predicted vs. observed 5 year probabilities of all-cause mortality by decile in the final model, P=0.11

<table>
<thead>
<tr>
<th>Predicted risk in decile</th>
<th>Model predicted risk</th>
<th>Observed risk*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>73.6%</td>
<td>73.2%</td>
</tr>
<tr>
<td>2</td>
<td>55.9%</td>
<td>52.3%</td>
</tr>
<tr>
<td>3</td>
<td>47.7%</td>
<td>41.4%</td>
</tr>
<tr>
<td>4</td>
<td>40.5%</td>
<td>40.0%</td>
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<tr>
<td>5</td>
<td>35.2%</td>
<td>30.2%</td>
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<td>6</td>
<td>30.0%</td>
<td>39.9%</td>
</tr>
<tr>
<td>7</td>
<td>24.0%</td>
<td>18.9%</td>
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<tr>
<td>8</td>
<td>18.9%</td>
<td>8.3%</td>
</tr>
<tr>
<td>9</td>
<td>13.1%</td>
<td>1.0%</td>
</tr>
<tr>
<td>10</td>
<td>5.8%</td>
<td>6.6%</td>
</tr>
</tbody>
</table>

* based on Kaplan-Meier observed risk
수축기 혈압과 뇌졸중 후 사망률

 너무 낮게는 가지 말아야 하나?

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**Too Low, No Go?**

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*(Stroke. 2015;46:1307-1313.)*

**Key Words:** blood pressure ■ hypertension ■ mortality ■ Nutrition Surveys ■ secondary prevention ■ stroke

## 개요

가보고한 20세 이상의 성인을 분석하였다. 기저 수축기 혈압은 저-정상(<120 mmHg), 정상(120–140 mmHg), 고(≥140 mmHg)로 분류되었다. 기저 수측기 혈압과 총 사망률, 혈관성 사망률의 독립적인 연관성을 코크비례위험(Cox proportional hazards)을 사용하여 분석하였다.

## 결과

3126명의 성인참가자들 중 455명이 뇌졸중 자가보고를 하였다.

<p>| Table 4. Crude and Adjusted Hazard Ratios of All-Cause Mortality or Vascular Mortality by SBP |
|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|</p>
<table>
<thead>
<tr>
<th>Crude &amp; Adjusted SBP</th>
<th>All-Cause Mortality (n=130 Events)</th>
<th>Vascular Mortality (n=61 Events)</th>
<th>All-Cause Mortality (n=130 Events)</th>
<th>Vascular Mortality (n=61 Events)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (95% CI)</td>
<td>P Value</td>
<td>HR (95% CI)</td>
<td>P Value</td>
<td>HR (95% CI)</td>
</tr>
<tr>
<td>SBP 120–139 mmHg</td>
<td>Ref</td>
<td>Ref</td>
<td>0.86 (0.66–1.13)</td>
<td>0.521</td>
</tr>
<tr>
<td>SBP &lt;120 mmHg</td>
<td>1.05 (0.66–1.67)</td>
<td>0.834</td>
<td>1.29 (0.61–2.63)</td>
<td>0.514</td>
</tr>
<tr>
<td>Adjusted*</td>
<td>0.627</td>
<td>0.514</td>
<td>0.30–1.85</td>
<td>0.521</td>
</tr>
</tbody>
</table>

* Adjusting for age, sex, race/ethnicity, poverty income ratio, hypertension, total serum cholesterol >200 mg/dL, coronary artery disease, angina, congestive heart failure, body mass index, use of antihypertensive medication(s), smoking.

**결론**

뇌졸중 후 정상 및 낮은 범주의 수축기 혈압은 높은 범주의 수측
기 혈압과 비교할 때 더 높고한 사망률 결과를 보인다.

**Figure.** Distribution of outcomes on the modified Rankin Scale (mRS) in percentages in patients who received general anesthesia (GA; n=70) or no GA (non-GA; n=278). mRS 0 to 1 indicate excellent outcome; mRS 2 to 3, moderate disability; mRS 4 to 5, severe disability; and mRS 6, dead.