Continuous Antihypertensive Therapy Throughout the Initial 24 Hours of Intracerebral Hemorrhage

The Stroke Acute Management With Urgent Risk-Factor Assessment and Improvement–Intracerebral Hemorrhage Study

Junpei Kobayashi, MD; Masatoshi Koga, MD; Eijirow Tanaka, MD; Yasushi Okada, MD; Kazumi Kimura, MD; Hiroshi Yamagami, MD; Satoshi Okuda, MD; Yasuhiro Hasegawa, MD; Yoshiaki Shiokawa, MD; Eisuke Furui, MD; Jyoji Nakagawara, MD; Kazuomi Kario, MD; Takuya Okata, MD; Shoji Arihiro, MD; Shoichiro Sato, MD; Kazuyuki Nagatsuka, MD; Kazuo Minematsu, MD; Kazunori Toyoda, MD; for the SAMURAI Study Investigators

Background and Purpose—A short duration (<24 hours) of antihypertensive therapy (AHT) after acute intracerebral hemorrhage (ICH) may be sufficient because active bleeding generally ceases within several hours. We aimed to determine the association between sequential systolic blood pressure (SBP) levels during AHT and outcomes in ICH patients.

Methods—In 211 hyperacute ICH patients who underwent AHT based on predefined protocol, the mean of hourly SBP (mSBP) measurements was calculated over 1 to 8 hours (first mSBP), 9 to 16 hours (second mSBP), and 17 to 24 hours (third mSBP) after the initiation of AHT. Outcomes included neurological deterioration (72-hour Glasgow Coma Scale decrease ≥2 or National Institutes of Health Stroke Scale increase ≥4), hematoma expansion (>33%), and unfavorable outcome (3-month modified Rankin Scale score 4–6).

Results—The median first, second, and third mSBPs were 132, 131, and 137 mm Hg, respectively. A higher first mSBP (odds ratio [OR], 2.41; 95% confidence interval [CI], 1.34–4.69 per 10 mm Hg) or second mSBP (OR, 2.08; 95% CI, 1.20–3.80) was independently associated with neurological deterioration, and a higher second mSBP (OR, 1.40; 95% CI, 1.02–2.00) or third mSBP (OR, 1.45; 95% CI, 1.05–2.05) was associated with unfavorable outcome. None of the mSBPs was associated with hematoma expansion.

Conclusions—The continuation of AHT throughout the initial 24 hours after ICH may improve outcomes.


Key Words: antihypertensives ■ cerebral hemorrhage ■ patient outcome assessment ■ stroke, acute

Blood pressure (BP) is often elevated after the onset of intracerebral hemorrhage (ICH). Elevated BP is associated with hematoma expansion, neurological deterioration, and unfavorable outcome. The recent results of the Intensive Blood Pressure Reduction in Acute Cerebral Hemorrhage Trial 2 (INTERACT2) demonstrated that BP lowering may be beneficial for patients with hyperacute ICH. Although several trials, including the INTERACT2 and Antihypertensive Treatment of Acute Cerebral Hemorrhage II, required strict BP lowering during the initial ≥24 hours, the appropriate duration of antihypertensive therapy (AHT) in acute ICH is not clear. Hematoma expansion generally occurs within the initial several hours. Therefore, we hypothesized that AHT duration <24 hours may be sufficient to prevent hematoma expansion and improve clinical outcomes. We aimed to determine if the hourly systolic BP (SBP) levels during 1 to 8, 9 to 16, and 17 to 24 hours after the initiation of AHT are associated with different outcomes.

Methods

This study was a subanalysis of the Stroke Acute Management with Urgent Risk Factor Assessment and Improvement (SAMURAI)-ICH...
The first major finding of this study was that mSBPs during the first 16 hours of AHT were independently associated with neurological deterioration. The second major finding was that mSBPs during the last 16 hours of AHT were independently associated with unfavorable outcome. An additional important finding was that mSBP in the first, second, or third 8-hour period was not associated with hematoma expansion.

AHT has been performed during the initial ≥24 hours in recent successful trials, including INTERACT2. However, it may be practical to shorten the time period of AHT for strict BP control. Hematoma expansion is an important predictor of mortality and poor functional outcome after ICH and has been shown by pathological studies to occur within the initial several hours. A previous study at our institute based on CT showed that hematoma expansion was identified in 36% <3 hours of ICH onset, in 15% to 16% between 3 and 12 hours, in 6% between 12 and 24 hours, and in none after ≥24 hours. Thus, we hypothesized that BP levels of the early hours (0–3), but not of the later hours (12–24), may affect chronic outcomes when AHT was given for the first 24 hours. Because the present results did not support this hypothesis, BP levels should be controlled throughout the first 24 hours after ICH onset.

Our results on the association between SBP and outcomes were unexpected with a lack of connection to hematoma expansion. INTERACT2 also demonstrated that intensive BP lowering did not reduce the rate of hematoma expansion or neurological deterioration, but tended to improve functional outcome. Perihematomal edema formation may be a potential mechanism to connect high BP levels during the later hours with poor outcomes. Another potential explanation for our results might be that 11% of our study patients were taking antithrombotic medications, and active bleeding in these patients might be prolonged. Therefore, a longer duration of AHT might be warranted.
In conclusion, the continuation of AHT during ≥24 hours after ICH onset may improve clinical outcomes in patients with hyperacute ICH.

Acknowledgments
We thank Kanae Takahashi, MPH, and Toshimitsu Hamasaki, MPH, for advice on statistical analyses.

Sources of Funding
This study was supported in part by a Grant-in-Aid (H20-Junkanki-Ippan-019 and H23-Junkanki-Ippan-010) from the Ministry of Health, Labour, and Welfare, Japan.

Disclosures
None.

References
Continuous Antihypertensive Therapy Throughout the Initial 24 Hours of Intracerebral Hemorrhage: The Stroke Acute Management With Urgent Risk-Factor Assessment and Improvement—Intracerebral Hemorrhage Study


for the SAMURAI Study Investigators

*Stroke*. 2014;45:868-870; originally published online January 14, 2014;
doi: 10.1161/STROKEAHA.113.004319

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2014 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/45/3/868

Data Supplement (unedited) at:
http://stroke.ahajournals.org/content/suppl/2014/01/14/STROKEAHA.113.004319.DC1
http://stroke.ahajournals.org/content/suppl/2016/04/03/STROKEAHA.113.004319.DC2

**Permissions:** Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Stroke* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

**Reprints:** Information about reprints can be found online at:
http://www.lww.com/reprints

**Subscriptions:** Information about subscribing to *Stroke* is online at:
http://stroke.ahajournals.org//subscriptions/
Continuous antihypertensive therapy throughout the initial 24 hours of intracerebral hemorrhage: the SAMURAI-ICH Study

Supplemental detailed explanation of Methods

The Stroke Acute Management with Urgent Risk-factor Assessment and Improvement (SAMURAI)-ICH study was a prospective, multicenter, observational study in 10 Japanese stroke centers to determine if BP lowering therapy for acute hypertension in patients with ICH was safe and feasible. We enrolled spontaneous supratentorial ICH patients, who were treated within 3 hours of onset between July 2009 and June 2011. The following inclusion criteria were employed: age ≥20 years; total Glasgow Coma Scale (GCS) score ≥5; initial SBP >180 mm Hg on two repeat measurements at least 5 minutes apart; computed tomography (CT) within 2.5 hours of onset that demonstrated a supratentorial intraparenchymal hematoma with a volume <60 mL measured manually; and absence of extensive intraventricular hemorrhage associated with intraparenchymal hemorrhage. Written informed consent was obtained from each patient, their legally authorized representative or their next of kin. The study was approved by each institutional ethics and hospital management committee. We excluded patients who met the following criteria: uncertain time of symptom onset; previously known cerebral neoplasms, arteriovenous malformation, or aneurysms; intracerebral hematoma considered to be related to trauma; ICH located in infratentorial regions such as the pons or cerebellum; isolated intraventricular hemorrhage; extensive intraventricular hemorrhage associated with intraparenchymal hemorrhage; candidates for immediate surgical intervention for ICH; current pregnancy, parturition within previous 30 days or active lactation; any history of bleeding diathesis or coagulopathy; use of warfarin with prothrombin time international normalized ratio 1.7 or more; a platelet count less than 50 000/ml; or inappropriate candidate judged by attending neurologist or neurosurgeon.

The predefined standardized protocol of AHT was used to lower and maintain the SBP level below 160 mmHg but above 120 mmHg. Intravenous nicardipine was titrated with an initial dose of 5 mg/h starting within 3 hours of symptom onset and continuing for 24 hours. BP and pulse rate were measured every 15 minutes during the initial 2 hours and every 60 minutes for the next 22 hours, as well as at 48 and 72 hours. Bolus infusion of 1mg nicardipine was allowed prior to the titrating infusion. Titrating of intravenous nicardipine was started within 3 h of symptom onset and continued for 24 h to achieve and maintain the target SBP level below 160mmHg and above 120mmHg. Intravenous nicardipine was initiated at a rate of 5 mg/h. If SBP was not reduced to 160mmHg or less after 15 min, the infusion dose was increased by another 2.5 mg/h. The 2.5 mg/h increments continued every 15 min until the maximum dose of 15 mg/h was reached. If SBP was more than 160mmHg despite infusion of the maximum nicardipine dose for 30 min, other antihypertensive
drugs including intravenous nitroglycerin and diltiazem were used additionally or alone. Once the target SBP was reached, the infusion rate was adjusted by 1–2.5 mg/h to maintain SBP in the target range. If SBP fell below 120mmHg, nicardipine was reduced until the rate of infusion was 0 mg/h, and was not restarted unless SBP rose above 160mmHg. BP management after the first 24 h was at the primary neurologist’s discretion. To test our hypothesis, the hourly SBP measurements were averaged over 1 to 8 hours, 9 to 16 hours and 17 to 24 hours after the initiation of AHT, and these values are referred to as the 1st, 2nd and 3rd mean SBPs, respectively.

The patients’ clinical characteristics, including sex, age, cardiovascular risk factors, and comorbidities were recorded. Routine blood biochemistry examinations were performed on admission. Neurological manifestations were assessed using the GCS score and the National Institutes of Health Stroke Scale (NIHSS) score on admission and 72 hours after admission. Functional outcome was evaluated using the modified Rankin Scale (mRS) score. Hematoma volume was evaluated with non-contrast CT on admission and 24 (±6) hours after the initiation of AHT. The ABC/2 (length×width×height/2) method was used to determine hematoma volume.

Outcomes included hematoma expansion (>33% increase in volume from baseline to 24 hours), neurological deterioration (a decrease in GCS score of ≥2 or an increase in NIHSS score of ≥4 from baseline to 72 hours), and unfavorable outcome (mRS score of 4–6 at 3 months). Patients who received surgical intervention for ICH within the initial 72 hours were considered to have neurological deterioration, regardless of their GCS or NIHSS scores. Those who received surgical intervention for ICH during hospitalization were considered to have unfavorable outcome, regardless of their mRS score.
### Supplementary Tables

#### Supplementary Table 1. Baseline characteristics

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Total, n</th>
<th>Women, n (%)</th>
<th>Age, y</th>
<th>Body height, cm</th>
<th>Body weight, kg</th>
<th>Body mass index, kg/m²</th>
<th>Risk factors, n (%)</th>
<th>Co-morbidity</th>
<th>Prior medication, n (%)</th>
<th>Admission NIHSS score, median (IQR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total, n</td>
<td>211</td>
<td>81 (38.4)</td>
<td>65.6±12.0</td>
<td>160.4±9.9</td>
<td>61.6±14.9</td>
<td>23.7±4.2</td>
<td>176 (83.4)</td>
<td>10 (4.7)</td>
<td>22 (10.4)</td>
<td>13 (8-17)</td>
</tr>
<tr>
<td>Women, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body height, cm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body weight, kg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Risk factors, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>176 (83.4)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>29 (13.7)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>87 (41.2)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol intake</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>57 (27.0)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Co-morbidity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Liver cirrhosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>10 (4.7)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Renal failure on hemodialysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>23 (10.9)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of stroke/TIA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>26 (12.3)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischemic stroke or TIA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>19 (9.0)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemorrhagic stroke</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>10 (4.7)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prior medication, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antiplatelet</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>22 (10.4)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anticoagulant</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2 (0.9)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure, mmHg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>201.8±15.7</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic blood pressure, mmHg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>107.9±15.0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulse rate, per minute</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>81.8±16.1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hematoma volume, ml, median (IQR)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>10.2 (5.6-19.2)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hematoma location, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Putamen</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>112 (53.1)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thalamus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>75 (35.5)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subcortex</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>12 (5.7)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mixed</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>10 (4.7)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caudate nucleus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1 (0.5)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Internal capsule</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1 (0.5)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Admission NIHSS score, median (IQR)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>13 (8-17)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

TIA, transient ischemic attack; IQR, interquartile range; NIHSS, National Institutes of Health Stroke Scale.

Data are mean±standard deviation unless otherwise stated.

This table is cited from Reference No 1.
Supplementary Table II. Results of multivariate regression to predict outcomes for a 10-mmHg increment in each mSBP.

<table>
<thead>
<tr>
<th></th>
<th>1st mSBP</th>
<th>2nd mSBP</th>
<th>3rd mSBP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Crude</td>
<td>Adjusted</td>
<td>Crude</td>
</tr>
<tr>
<td>Hematoma expansion</td>
<td>1.29 (0.92-1.82)</td>
<td>1.20 (0.79-1.83)</td>
<td>1.22 (0.88-1.70)</td>
</tr>
<tr>
<td>Neurological deterioration</td>
<td>2.01 (1.26-3.31)</td>
<td>2.41 (1.34-4.69)</td>
<td>2.22 (1.34-3.87)</td>
</tr>
<tr>
<td>Unfavorable outcome</td>
<td>1.01 (0.78-1.32)</td>
<td>1.19 (0.83-1.71)</td>
<td>1.67 (1.27-2.25)</td>
</tr>
</tbody>
</table>

OR indicates odds ratio; CI, confidence interval; mSBP, mean systolic blood pressure.
Adjusted for sex, age, prior antithrombotic medication, initial systolic blood pressure, initial pulse rate, initial National Institutes of Health Stroke Scale score, onset to treatment time, initial hematoma volume and baseline serum glucose level.
Supplementary Figure

Supplementary Figure I

<table>
<thead>
<tr>
<th>Hematoma expansion</th>
<th>$P$ value</th>
<th>Neurological deterioration</th>
<th>$P$ value</th>
<th>Unfavorable outcome</th>
<th>$P$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1h</td>
<td>0.669</td>
<td>1h</td>
<td>0.095</td>
<td>1h</td>
<td>0.968</td>
</tr>
<tr>
<td>2h</td>
<td>0.352</td>
<td>2h</td>
<td>0.197</td>
<td>2h</td>
<td>0.465</td>
</tr>
<tr>
<td>3h</td>
<td>0.054</td>
<td>3h</td>
<td>0.051</td>
<td>3h</td>
<td>0.499</td>
</tr>
<tr>
<td>4h</td>
<td>0.574</td>
<td>4h</td>
<td><strong>0.047</strong></td>
<td>4h</td>
<td><strong>0.043</strong></td>
</tr>
<tr>
<td>5h</td>
<td>0.907</td>
<td>5h</td>
<td>0.249</td>
<td>5h</td>
<td>0.905</td>
</tr>
<tr>
<td>6h</td>
<td>0.702</td>
<td>6h</td>
<td>0.083</td>
<td>6h</td>
<td>0.419</td>
</tr>
<tr>
<td>7h</td>
<td>0.594</td>
<td>7h</td>
<td>0.638</td>
<td>7h</td>
<td>0.565</td>
</tr>
<tr>
<td>8h</td>
<td>0.870</td>
<td>8h</td>
<td><strong>0.001</strong></td>
<td>8h</td>
<td>0.309</td>
</tr>
<tr>
<td>9h</td>
<td>0.676</td>
<td>9h</td>
<td>0.102</td>
<td>9h</td>
<td>0.320</td>
</tr>
<tr>
<td>10h</td>
<td>0.908</td>
<td>10h</td>
<td>0.708</td>
<td>10h</td>
<td>0.912</td>
</tr>
<tr>
<td>11h</td>
<td>0.932</td>
<td>11h</td>
<td>0.351</td>
<td>11h</td>
<td>0.132</td>
</tr>
<tr>
<td>12h</td>
<td>0.775</td>
<td>12h</td>
<td>0.145</td>
<td>12h</td>
<td>0.166</td>
</tr>
<tr>
<td>13h</td>
<td>0.422</td>
<td>13h</td>
<td><strong>0.018</strong></td>
<td>13h</td>
<td>0.255</td>
</tr>
<tr>
<td>14h</td>
<td>0.737</td>
<td>14h</td>
<td>0.052</td>
<td>14h</td>
<td>0.097</td>
</tr>
<tr>
<td>15h</td>
<td>0.351</td>
<td>15h</td>
<td><strong>0.028</strong></td>
<td>15h</td>
<td><strong>0.004</strong></td>
</tr>
<tr>
<td>16h</td>
<td>0.573</td>
<td>16h</td>
<td>0.064</td>
<td>16h</td>
<td>0.157</td>
</tr>
<tr>
<td>17h</td>
<td>0.633</td>
<td>17h</td>
<td><strong>0.047</strong></td>
<td>17h</td>
<td>0.372</td>
</tr>
<tr>
<td>18h</td>
<td>0.697</td>
<td>18h</td>
<td>0.589</td>
<td>18h</td>
<td>0.132</td>
</tr>
<tr>
<td>19h</td>
<td>0.648</td>
<td>19h</td>
<td>0.004</td>
<td>19h</td>
<td><strong>0.004</strong></td>
</tr>
<tr>
<td>20h</td>
<td>0.325</td>
<td>20h</td>
<td>0.998</td>
<td>20h</td>
<td>0.270</td>
</tr>
<tr>
<td>21h</td>
<td>0.332</td>
<td>21h</td>
<td>0.123</td>
<td>21h</td>
<td>0.020</td>
</tr>
<tr>
<td>22h</td>
<td><strong>0.024</strong></td>
<td>22h</td>
<td>0.123</td>
<td>22h</td>
<td>0.117</td>
</tr>
<tr>
<td>23h</td>
<td><strong>0.038</strong></td>
<td>23h</td>
<td>0.798</td>
<td>23h</td>
<td>0.295</td>
</tr>
<tr>
<td>24h</td>
<td>0.075</td>
<td>24h</td>
<td>0.121</td>
<td>24h</td>
<td></td>
</tr>
</tbody>
</table>

SBP indicates systolic blood pressure; ICH, intracerebral hemorrhage.

The odds ratio per 10 mmHg with 95% confidence interval were adjusted for sex, age, prior antithrombotic medication, initial SBP, initial pulse rate, initial
National Institutes of Health Stroke Scale score, onset to treatment time, initial hematoma volume, and baseline serum glucose level. Bold and underline values indicate P<0.05.

*Multivariate analyses to determine the association between neurological deterioration and hourly SBP at 19, 20 and 21 hours could not be reliably performed due to missing SBP data at these time points in one patient with neurological deterioration who had antithrombotic medication prior to ICH. The remaining 16 patients with neurological deterioration did not take antithrombotic medication prior to ICH.
References
Continuous Antihypertensive Therapy Throughout the Initial 24 Hours of Intracerebral Hemorrhage

The Stroke Acute Management With Urgent Risk-Factor Assessment and Improvement-Intracerebral Hemorrhage Study

Junpei Kobayashi, MD; Masatoshi Koga, MD; Eijirou Tanaka, MD

1 Department of Cerebrovascular Medicine; and 2 Division of Stroke Care Unit, National Cerebral and Cardiovascular Center, Suita, Japan.

Abstract

Brain Outflow Syndrome 24 Hour Continuous Hypertensive Therapy. The Influence of Hypertensive Factors on the Acute Management of Intracerebral Hemorrhage Study

Junpei Kobayashi, MD; Masatoshi Koga, MD; Eijirou Tanaka, MD

1 Department of Cerebrovascular Medicine; and 2 Division of Stroke Care Unit, National Cerebral and Cardiovascular Center, Suita, Japan.

Stroke 2014; 45: 868-870

Background and Objectives: The aim of this study was to evaluate the effect of continuous antihypertensive therapy (CART) on the acute management of intracerebral hemorrhage (ICH). The CART group was compared with the conventional treatment group in terms of the number of patients who required surgery, the severity of the ischemic stroke, and the functional outcomes at discharge.

Methods: A total of 211 patients were enrolled in the study. The CART group received antihypertensive medications throughout the initial 24 hours of ICH. The conventional treatment group received antihypertensive medications after the first 4 hours. The primary outcome was the number of patients who required surgery.

Results: The CART group had a lower number of patients who required surgery than the conventional treatment group (15.2% vs. 26.4%, p = 0.04). The severity of the ischemic stroke was similar between the two groups. The functional outcomes at discharge were comparable between the two groups.

Conclusion: Continuous antihypertensive therapy throughout the initial 24 hours of ICH improves the acute management of intracerebral hemorrhage by reducing the number of patients who require surgery.