Prehospital systolic blood pressure is higher in acute stroke compared with stroke mimics

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ABSTRACT

Objective: To assess the natural history of prehospital blood pressure (BP) during emergency medical services (EMS) transport of suspected stroke and determine whether prehospital BP differs among types of patients with suspected stroke (ischemic stroke, TIA, intracerebral hemorrhage [ICH], or stroke mimic).

Methods: A retrospective, cross-sectional, observational analysis of a centralized EMS database containing electronic records of patients transported by EMS to the emergency department (ED) with suspected stroke during an 18-month period was conducted. Hospital charts and neuroimaging were utilized to determine the final diagnosis (ischemic stroke, TIA, ICH, or stroke mimic).

Results: A total of 960 patients were transported by EMS to ED with suspected stroke. Stroke was diagnosed in 544 patients (56.7%) (38.2% ischemic stroke, 12.2% TIA, 5.3% ICH) and 416 (43.2%) were considered mimics. Age-adjusted mean prehospital systolic BP (SBP) was higher in acute stroke patients (155.6 mm Hg; 95% confidence interval [CI]: 153.4–157.9 mm Hg) compared to mimics (146.1 mm Hg; 95% CI: 142.5–148.6 mm Hg; $p < 0.001$). Age-adjusted mean prehospital SBP was higher in ICH (172.3 mm Hg; 95% CI: 165.1–179.7 mm Hg) than in either ischemic stroke or TIA (154.7 mm Hg; 95% CI: 152.3–157.0 mm Hg; $p < 0.001$). Median (interquartile range) SBP drop from initial prehospital SBP to ED SBP was 4 mm Hg (−6 to 17 mm Hg). Mean prehospital SBP was strongly correlated with ED SBP ($r = 0.82$, $p < 0.001$).

Conclusions: Prehospital SBP is higher in acute stroke relative to stroke mimics and highest in ICH. Given the stability of BP between initial EMS and ED measurements, it may be reasonable to test the feasibility and safety of prehospital antihypertensive therapy in patients with suspected acute stroke. *Neurology*® 2016;86:2146–2153

GLOSSARY

BP = blood pressure; **ED** = emergency department; **EMS** = emergency medical services; **ICH** = intracerebral hemorrhage; **SBP** = systolic blood pressure; **TOAST** = Trial of Org 10172 in Acute Stroke Treatment.

Elevated hospital admission blood pressure (BP) in patients with acute stroke is common[1] and is associated with poor outcomes.[2–5] There is limited randomized clinical trial evidence that acute BP reduction improves outcomes after intracerebral hemorrhage (ICH).[6] One of the hypothesized reasons for the modest treatment effect is that hematoma enlargement and clinical deterioration have already begun by the time antihypertensive therapy is administered. It has been suggested that initiation of BP reduction in the prehospital setting may improve the efficacy of this approach.[7]

Recent pilot studies have assessed the feasibility of a prehospital BP-lowering strategy in patients with acute stroke symptoms using topical glyceryl trinitrate[8] and sublingual lisinopril.[9] At this point, however, the natural history of BP in the prehospital setting in patients with acute stroke symptoms is poorly understood. In contrast, the course of BP after hospital admission has been well described. Pressures are highest at admission, begin to spontaneously decline almost immediately,[10] and this trend continues for several days.[11–14] It is possible that admission

pressures are similar to those taken closer to symptom onset. Alternatively, BP may acutely rise after arrival to the emergency department (ED), or the spontaneous decline seen after admission may actually begin shortly after symptom onset.

Admission BP and natural history over the first week after hospital admission have both been shown to differ with stroke type. Specifically, pressures are higher in patients with ICH, relative to those with ischemic stroke.[11,15] It is unknown whether these associations are also present in the prehospital setting.

The objectives of this study were to (1) assess the natural history of prehospital BP during emergency medical services (EMS) transport of consecutive patients with suspected stroke, and (2) determine whether prehospital BP differed among types of patients with suspected stroke (ischemic stroke, TIA, ICH, or stroke mimic). We tested the hypotheses that (1) prehospital systolic BP (SBP) in patients with suspected stroke is not significantly different from that measured at admission to the ED, and (2) prehospital BP in ICH is higher than in patients with ischemic stroke.

METHODS **Study design.** We conducted a retrospective, cross-sectional, observational study of a prospectively maintained centralized EMS database of all consecutive patients with suspected stroke transported by EMS to the ED of the University of Alberta Hospital during an 18-month period (January 1, 2012, to July 31, 2013).

EMS service and suspected stroke. The unified EMS service for the city of Edmonton attends to more than 157,000 events annually and transports patients to one of 13 provincially operated Edmonton Zone EDs. Since 2009, all land ambulance services have transitioned into a unified provincial system under Alberta Health Services.[16] The University of Alberta Hospital is a tertiary health care center and one of 2 designated stroke centers within the Edmonton Zone, receiving the majority of patients transported by EMS with suspected stroke and admitting 1,000 to 1,200 stroke patients annually.

During a 9-1-1 call for suspected stroke, the emergency dispatcher used a stroke-specific interview algorithm to assign a Medical Priority Dispatch System event code for suspected stroke. This event code was subsequently communicated to EMS personnel who responded to the emergency call. The prehospital Cincinnati Stroke Screen[17] in conjunction with an in-house stroke screen developed by provincial stroke neurologists was used by EMS personnel to determine a primary EMS impression of acute stroke.

Upon first EMS assessment and during transport to the ED, individual patient data (including serial vital sign measurements such as BP, heart rate, oxygen saturation, temperature, glucose, and Glasgow Coma scale score) were recorded on electronic patient health care reports. Prehospital BP recordings were measured according to the Standard Approach and Ongoing Assessment Medical Control Protocol, where at least 2 sets of BP readings are measured by EMS personnel every 15 to 30 minutes. The initial BP was measured by auscultation with subsequent measurements using a noninvasive BP cuff. Dispatch and individual electronic patient health care reports data were then prospectively uploaded to a centralized provincial EMS database at the Alberta Health Services.

Standard protocol approvals, registrations, and patient consents. The research protocol of this study was approved for waiver of consent by the local institutional Human Research Ethics Review Board (University of Alberta).

Study population and data collection. Eligible study patients were identified within the centralized Alberta Health Services database by dispatch event and/or EMS primary impression of acute stroke. Once identified, data from individual patient electronic patient health care reports were extracted and linked to ED records through the Emergency Department Information Systems to obtain vital sign measurements on ED arrival. Individual patient electronic patient health care reports data were also linked to in-hospital patient charts and neuroimaging to determine the final diagnosis of ischemic stroke, TIA, or ICH, as per the treating physician's discharge diagnosis. Ischemic stroke etiology was classified according to TOAST (Trial of Org 10172 in Acute Stroke Treatment) criteria.[18] For the purpose of the study, stroke mimics were defined as any discharge diagnosis (other than acute stroke) for symptoms that prompted initial EMS activation for suspected stroke.

Statistical analyses. All statistical analyses were performed using SPSS 22.0 (IBM SPSS Statistics 2014, Armonk, NY). The rate of missing data (notably in baseline characteristics) was low ($<4.3\%$), and analyses were performed using pairwise deletion of missing data. Continuous variables are reported as means and SDs or medians and interquartile ranges, as appropriate. Dichotomous variables are reported as proportions. Differences in baseline characteristics in the 4 types of patients with suspected stroke were analyzed using χ^2 test. Differences in groups of continuous variables (prehospital SBP) between acute stroke and stroke mimics and between the 4 groups were analyzed by 1-way analysis of variance followed by Tukey post hoc test for independent comparisons. Analysis of covariance was used to analyze the effects of age on the differences of mean prehospital SBP between groups. The degree of correlation between prehospital SBP and ED SBP was analyzed using Pearson correlation coefficient. Prehospital SBP variability was defined as the SD of mean prehospital SBP.

RESULTS **Study population.** A total of 1,060 eligible patients were identified within the study period. A total of 100 patients were excluded from analyses because of incomplete data (inability to link EMS data with in-hospital charts, n = 62; absence of EMS vital sign recordings during EMS transport, n = 13; and in-hospital chart unavailability for review, n = 25), leaving a total study population of 960 patients.

The mean ± SD age of the entire cohort was 70.4 ± 15.6 years, and 47.8% of patients were men. Acute stroke pathology was diagnosed in 544 patients (56.7%) and 416 (43.3%) were considered stroke mimics. Among acute stroke patients, 367 (38.2%) were diagnosed with acute ischemic stroke,

117 (12.2%) with TIA, and 51 (5.3%) with acute ICH. Nine patients were diagnosed with subarachnoid hemorrhage (0.9%) and were excluded a priori in subsequent analyses.

Patients diagnosed with stroke mimics were younger and had lower rates of hypertension and atrial fibrillation than patients with acute ischemic stroke/TIA/ICH (table). Conversely, patients with acute ischemic stroke had higher rates of coronary artery disease and dyslipidemia, while patients with TIA and ICH had higher rates of previous TIA and ICH, respectively.

Prehospital BP in acute stroke and stroke mimic patients. Initial prehospital SBP (mean ± SD) was higher in patients with acute stroke (159.7 ± 29.5 mm Hg) compared to those with stroke mimics (147.2 ± 31.2 mm Hg, $p < 0.001$). Similarly, mean prehospital SBP was higher in patients with acute stroke (156.4 ± 26.9 mm Hg) than in patients with stroke mimics (145.3 ± 25.4 mm Hg, $p < 0.001$). An age-adjusted analysis of covariance showed that mean prehospital SBP remained significantly higher in patients with acute stroke (adjusted means 155.6 mm Hg; 95% confidence interval [CI]: 153.4–157.9 mm Hg) than in patients with stroke mimics (adjusted means 146.1 mm Hg; 95% CI: 143.5–148.6 mm Hg; $p < 0.001$).

When divided into groups based on final diagnosis, initial prehospital SBP was higher in patients with ICH (175.5 ± 32.4 mm Hg, $p \leq 0.003$) than in patients

with ischemic stroke (157.8 ± 29.5 mm Hg), TIA (157.7 ± 25.7 mm Hg), and stroke mimics (147.2 ± 31.1 mm Hg). Patients with ICH also had higher mean prehospital SBP (172.3 ± 31.7 mm Hg, $p < 0.001$) when compared to ischemic stroke (154.9 ± 26.5 mm Hg), TIA (153.3 ± 23.1 mm Hg), and stroke mimics (145.4 ± 25.4 mm Hg) (figure 1A). Mean prehospital SBP remained highest in patients with ICH (adjusted means 172.3 mm Hg; 95% CI: 165.1–179.7 mm Hg) compared to patients with ischemic stroke or TIA (adjusted means 154.7 mm Hg; 95% CI: 152.3–157.0 mm Hg; $p < 0.001$) after adjusting for age. Similar results were observed for mean diastolic BP and mean arterial pressure during EMS transport (figure 1, B and C).

Prehospital BP during EMS transport. The median (interquartile range) time of patient–EMS interaction (time of EMS arrival on-scene to ED arrival) was 45.0 (36–55) minutes. Patient–EMS interaction was slightly longer in patients with stroke mimics (48 [39–60] minutes) than in patients with acute stroke (43 [36–53] minutes, $p < 0.001$) (table). During EMS transport, prehospital BP was measured a mean 4 ± 1.9 times. In acute stroke patients, the median time from symptom onset to first prehospital BP reading was 75 (24–277) minutes.

In relation to stroke onset, 74.8% of patients had their first BP measurement ≤4.5 hours from onset, 4.8% between 4.5 and 6 hours, 12.7% between

Table	Baseline characteristics				
	Ischemic stroke (n = 367)	TIA (n = 117)	Intracerebral hemorrhage (n = 51)	Stroke mimics (n = 416)	p Value
Demographics					
Age, y, mean ± SD	73.3 ± 14	72.4 ± 14.4	73.1 ± 13.5	66.9 ± 16.9	0.03
Sex, male, n (%)	186 (50.7)	47 (40.2)	27 (52.9)	192 (46.2)	0.18
Time of patient-EMS interaction, min, median (IQR)	43 (36-52)	44.5 (36-56)	40 (34-47)	48 (39-60)	<0.001
Time from stroke onset to first BP measure, min, median (IQR)	85 (24-261)	NA	63 (23-346)	NA	
Clinical characteristics, n (%)					
Hypertension	273 (77.3)	80 (70.1)	38 (79.2)	216 (53.7)	<0.001
Type 2 diabetes	90 (25.4)	26 (22.8)	11 (22.9)	111 (27.6)	0.71
Coronary artery disease	81 (23.0)	15 (13.2)	10 (20.8)	51 (12.7)	0.001
Dyslipidemia	191 (54.1)	55 (48.2)	15 (31.3)	132 (32.8)	<0.001
Congestive heart failure	31 (8.9)	5 (4.4)	5 (10.4)	27 (6.7)	0.31
Atrial fibrillation	120 (34.1)	23 (20.2)	15 (31.3)	55 (13.7)	<0.001
Prior stroke	90 (25.5)	24 (21.1)	6 (12.5)	109 (27.1)	0.12
Prior ICH	3 (0.8)	1 (0.9)	3 (6.3)	15 (3.7)	0.01
Prior TIA	40 (11.4)	34 (29.8)	6 (12.5)	58 (14.5)	<0.001

Abbreviations: BP = blood pressure; EMS = emergency medical services; ICH = intracerebral hemorrhage; IQR = interquartile range; NA = not applicable. Data were missing in 36 patients regarding time from stroke onset to first BP measure; 17 time of patient-EMS interaction; 34 hypertension, type 2 diabetes, or dyslipidemia status; 35 coronary artery disease status; 37 congestive heart failure status; 41 atrial fibrillation status; and 38 prior stroke, ICH, or TIA.
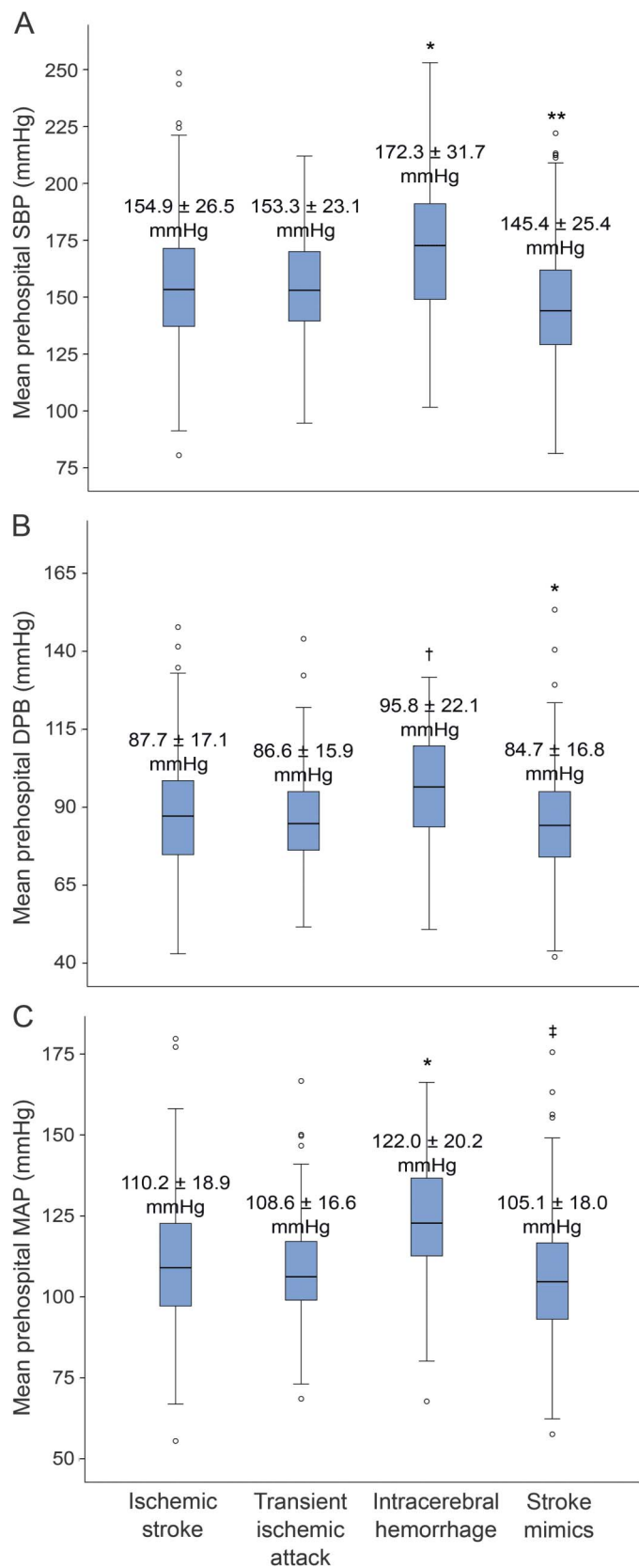
6 and 12 hours, and 7.7% >12 hours from onset. For acute stroke patients, prehospital SBP was very weakly correlated with the time from symptom onset to BP measurement ($r = 0.14$, $p < 0.001$). This did not differ in the subgroup of patients assessed within 4.5 hours of ischemic stroke onset ($r = 0.1$, $p = 0.006$) or in patients with ICH ($r = -0.06$, $p = 0.39$) (figure 2).

Mean prehospital SBP remained stable during EMS transport in all 4 patient types (figure 3). Patients with ICH maintained the highest mean prehospital SBP while patients with stroke mimics maintained the lowest prehospital SBP throughout EMS transport ($p < 0.02$). The overall mean variability of prehospital SBP was 11.1 ± 7.7 mm Hg, and this did not differ among groups ($p = 0.46$).
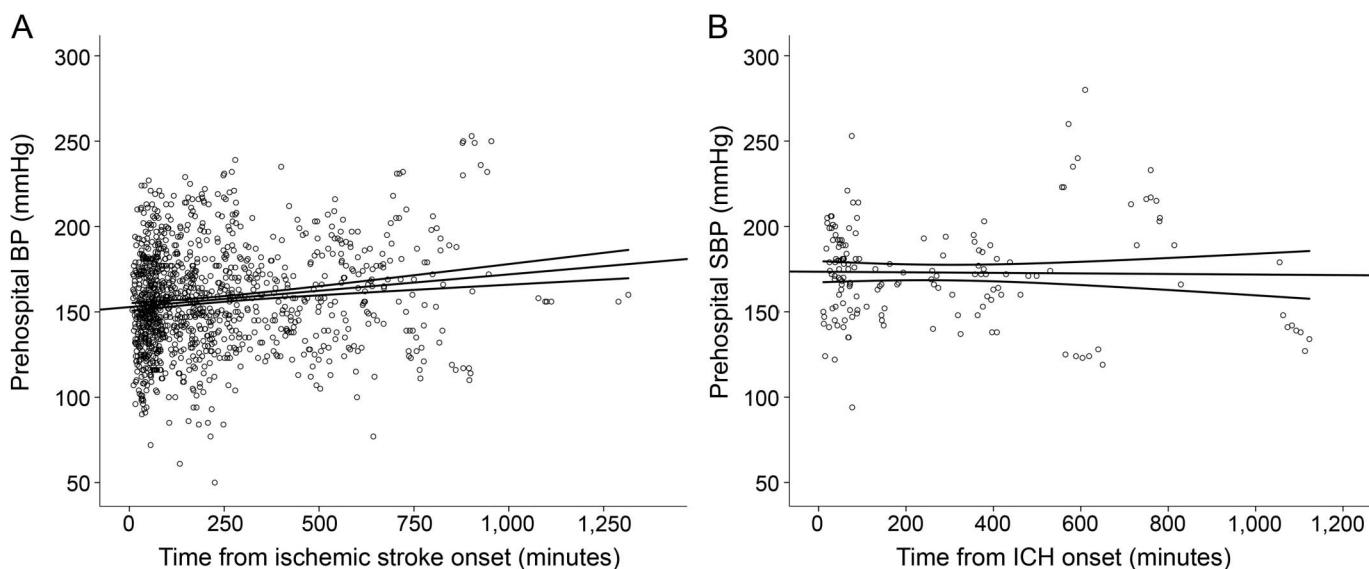
Prehospital BP among ischemic stroke subtypes. Among the 367 ischemic stroke patients, stroke etiology by TOAST classification was as follows: large artery atherosclerosis in 51 patients (13.9%), cardioembolic in 140 (38.1%), lacunar in 44 (12.0%), other determined etiology in 22 (6.0%), and cryptogenic in 110 (30.0%). Mean prehospital SBP in lacunar stroke was higher (169.8 ± 29.8 mm Hg, $p < 0.001$) than in cardioembolic stroke (150.9 ± 25.5 mm Hg) and stroke of other determined etiology (133.2 ± 19.7 mm Hg) (figure 4). There was a trend toward higher mean prehospital SBP in lacunar stroke when compared to large artery atherosclerosis (156.4 ± 26.0 mm Hg, $p = 0.08$) and cryptogenic stroke/stroke of undetermined etiology (158.0 ± 24.3 mm Hg, $p = 0.06$).

Relationship of prehospital BP and SBP on ED arrival. Mean admission SBP on ED arrival was higher in patients with ICH (168.3 ± 34.0 mm Hg, $p < 0.003$) than in patients with ischemic stroke (153.2 ± 28.3 mm Hg), TIA (151.9 ± 25.2 mm Hg), and stroke mimics (142.2 ± 26.5 mm Hg). Initial prehospital SBP was strongly correlated to ED SBP ($r = 0.74$, $p < 0.001$), as was the mean prehospital SBP to ED SBP ($r = 0.82$, $p < 0.001$).

The median absolute difference in SBP from the initial prehospital BP measurement to admission BP measurement on ED arrival in all patients was 4 (-6 to 18) mm Hg. When broken down by type, the median absolute difference in initial prehospital SBP and ED SBP was 4 (-6 to 17) mm Hg in ischemic

Box-and-whisker plots of (A) mean prehospital SBP by diagnosis classification, (B) mean prehospital DBP by diagnostic classification, and (C) mean prehospital MAP by diagnostic classification. The *p* values were obtained by analysis of variance followed by Tukey post hoc test for independent comparisons between categories. *$p <$ 0.001, †$p <$ 0.01 for ICH compared to ischemic stroke, TIA, and stroke mimics. **$p <$ 0.02 for stroke mimics compared to ischemic stroke, TIA, and ICH. ‡$p \leq$ 0.001 for stroke mimics compared to ischemic stroke and ICH. DBP = diastolic blood pressure; ICH = intracerebral hemorrhage; MAP = mean arterial pressure; SBP = systolic blood pressure.

Scatterplots of the relationship between time from ischemic stroke onset (A) and time from ICH onset (B) and prehospital SBP measurements. BP = blood pressure; ICH = intracerebral hemorrhage; SBP = systolic blood pressure.

stroke, 6 (−7 to 18.5) mm Hg in TIA, 9 (−3.5 to 27.5) mm Hg in ICH, and 2 (−6 to 18) in stroke mimics, and this did not differ between groups ($p = 0.36$). The median %SBP reduction from initial prehospital measurement to ED was similar in all 4 groups (3% [−3.8% to 10%] in ischemic stroke, 4.4% [−4.7% to 11%] in TIA, 6.5% [−2.4% to 13.5%] in ICH, and 3% [−4% to 11.8%] in stroke mimics, $p = 0.59$).

DISCUSSION Using robust prospectively collected regional EMS data and linkage to in-patient records, our findings suggest that prehospital SBP remains stable during EMS transport in patients with acute stroke symptoms. In addition, our study demonstrates that prehospital SBP is higher in acute stroke compared to stroke mimics, particularly in ICH.

There are very few published reports of prehospital BP in patients with suspected stroke. An observational analysis of 69 patients with acute stroke enrolled in a phase I recombinant tissue plasminogen activator study showed that elevated BP spontaneously declined up to a mean −29 ± 22 mm Hg within the first 90 minutes from stroke onset (first BP measurement taken at a mean 19 ± 13 minutes after symptom onset).[10] In contrast, we found only a 4–mm Hg median SBP decrease from the first prehospital SBP to ED SBP, and elevated prehospital SBP appeared to be relatively independent of the time from stroke onset to assessment. Possible explanations for the discrepancy between studies may be attributable to sample size. Although BP does spontaneously decline shortly after hospital admission, prehospital BP measurements were relatively stable in our study. This may reflect the relatively brief transport time.

A 10-year retrospective population-based Japanese study of >106,000 patients with

Figure 3 Natural history of prehospital SBP of patients with suspected stroke by diagnostic classification
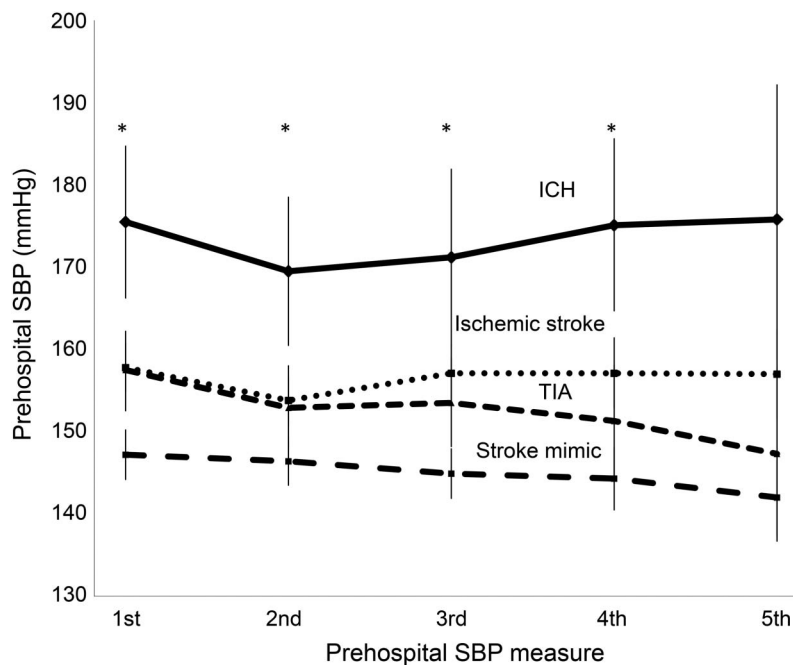
Linear plot of the natural history of prehospital SBP during emergency medical services transport by diagnostic classification. Error bars represent the 95% confidence interval of the mean. *$p < 0.02$ for ICH compared to ischemic stroke/TIA/mimics, except for the fifth prehospital measure between ICH and ischemic stroke ($p = 0.06$). ICH = intracerebral hemorrhage; SBP = systolic blood pressure.

Figure 4   Relationship between mean prehospital SBP and ischemic stroke etiology
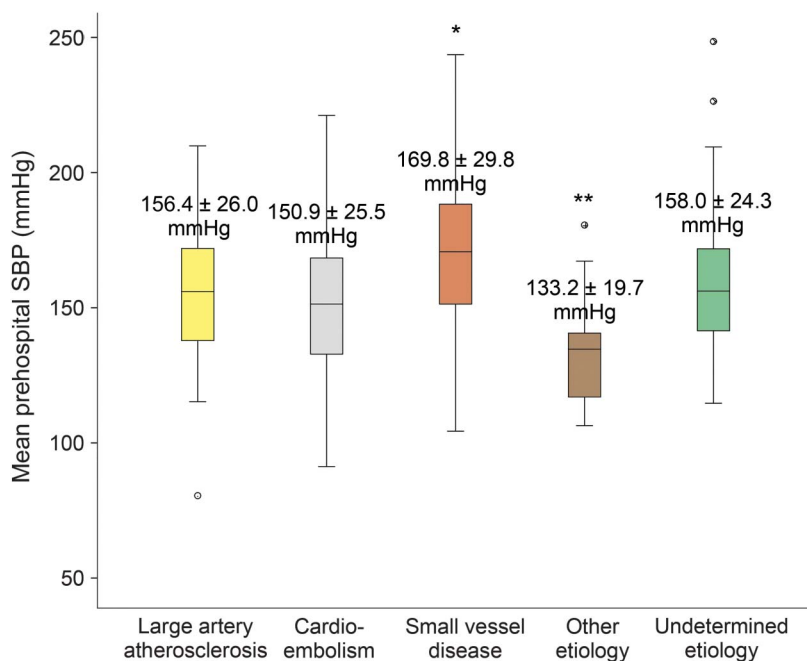
Box-and-whisker plots of mean prehospital SBP by stroke etiology, defined according to TOAST classification. The p values were obtained by analysis of variance followed by Tukey post hoc test for independent comparisons between categories. *p < 0.001 for small vessel disease compared to cardioembolic and other determined etiology groups. **p < 0.02 for other determined etiology against all other groups. SBP = systolic blood pressure; TOAST = Trial of Org 10172 in Acute Stroke Treatment.

impaired consciousness showed that stroke diagnoses increased with the presence of elevated prehospital BP, particularly >160 mm Hg, relative to patients with other diagnoses.[19] These data are consistent with our observation that elevated prehospital BP is higher in patients with acute stroke relative to other diagnoses.

To date, >10,000 patients have been enrolled in acute stroke BP treatment trials, yet optimal BP management in acute stroke remains controversial.[6,20–24] Enrolling patients in the prehospital phase may help to elucidate whether early BP management is beneficial in acute stroke. This is of particular interest in acute ICH, where the phenomenon of hematoma expansion, a well-known predictor of poor prognosis and mortality,[25] occurs most often within the first 3 hours from ICH onset.[26] In BP-lowering trials conducted to date, very few patients with ICH have been enrolled this early after onset.[6,27] Prehospital BP trials may therefore be the ideal setting to assess whether early BP treatment reduces hematoma expansion rates and improves clinical outcomes.

Nevertheless, there exists a theoretical concern regarding acute BP reduction in the EMS setting of undifferentiated stroke because of the fear of exacerbating hypoperfusion within the ischemic penumbra.

Our findings suggest that higher prehospital SBP may be associated with lacunar infarcts due to small vessel disease, which, by definition, have smaller infarct volumes and thus may be at lower risk of exacerbated hypoperfusion.

Current guidelines for BP management in acute stroke suggest an SBP target of <180 mm Hg for all patients with ICH on ED arrival.[28] Furthermore, the recommended target SBP in patients with ischemic stroke who are eligible for thrombolysis is <180 mm Hg.[29] Given the stability of prehospital BP between EMS and ED measurement, it may be reasonable to target an SBP of <180 mm Hg in all patients presenting with suspected stroke and in a thrombolysis window while en route to hospital. The safety and feasibility of this approach needs to be assessed in a prospective trial. Although not target-based, studies of prehospital BP reduction are ongoing.[30]

Although this is a large observational study of consecutive patients with suspected stroke transported by EMS personnel, it is limited by its retrospective design and lack of standardized hypertension monitoring protocols. Conversely, individual EMS patient data were acquired in a prospective manner and subsequently uploaded to a centralized database and EMS staff members were unaware of the study, which may help to limit bias. Furthermore, we were able to achieve >90% linkage of individual EMS patient data to in-hospital patient charts and neuroimaging to appropriately classify patients by final diagnosis. Although all patients with suspected stroke identified by either dispatch event codes for suspected stroke or EMS primary impression of stroke within the study period were captured in our study, prehospital data of patients with an ultimate diagnosis of acute stroke who were not identified as such by EMS personnel (false-negative patients) were not accounted for. However, this is beyond the scope of the present analyses where the focus was to assess the natural history of prehospital BP in patients whom EMS personnel deem eligible for prehospital BP studies in acute stroke. Furthermore, patients with suspected stroke transported to other hospitals within the Edmonton Zone were not accounted for, which could potentially affect the generalizability of our results. However, as the primary designated stroke center within the Edmonton Zone with endovascular capabilities, we believe the bias is minimal, as persons with more severe strokes are transported to the University of Alberta Hospital. Nine patients (0.9%) with subarachnoid hemorrhage were excluded from analyses because the pathophysiology and management of these patients is so different from acute ischemic stroke and ICH patients. We had planned a priori to assess these patients separately, but the small number precluded this. Although it is difficult to differentiate a patient

with subarachnoid hemorrhage from ischemic stroke or ICH in the prehospital setting, we believe the bias is minimal in our study. Finally, a higher than expected stroke mimic rate (43%) was observed in our study. This may be related to the previous observation that dispatch diagnosis of acute stroke has modest sensitivity and positive predictive value (approximately 45%).[31]

Prehospital BP is similar to that at hospital admission and remains stable during EMS transport in patients with acute stroke symptoms. Prehospital BP is higher in acute stroke patients compared to stroke mimic patients, particularly those with ICH. Although not reliably predictive of stroke or stroke type, elevated BP may be useful in patient selection for ongoing and future prehospital stroke treatment trials.

AUTHOR CONTRIBUTIONS

L.C. Gioia: designed and conceptualized the study, collected data, performed analysis and interpretation of the data, and drafted the manuscript. R.T. Zewude: collected data, performed analysis, and revised the manuscript. M.P. Kate: designed and conceptualized the study and revised the manuscript. K. Liss: provided data and revised the manuscript. B. Rowe: designed and conceptualized the study and revised the manuscript. B. Buck: analysis and interpretation of data and revised the manuscript. T. Jeerakathil: helped with the statistical analyses and interpretation of the data and revised the manuscript. K. Butcher: designed and conceptualized the study, analyzed and interpreted the data, and revised the manuscript.

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DISCLOSURE

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This Week's *Neurology*® Podcast

Clinical and genetic features of cervical dystonia in a large multi-center cohort (see the June 2016 issue of *Neurology*® *Genetics*)

This podcast begins and closes with Dr. Robert Gross, Editor-in-Chief, briefly discussing highlighted articles from the June 7, 2016, issue of *Neurology*. In the second segment, Dr. Michelle Fullard talks with Dr. Mark LeDoux about his *Neurology: Genetics* paper on the clinical and genetic features of cervical dystonia. Dr. Ted Burns interviews Dr. Dave Sharp about his paper on amyloid pathology and axonal injury after brain trauma for our "What's Trending" feature of the week. In the next part of the podcast, Dr. Andy Southerland focuses his interview with Dr. Andy Solomon on his abstract presentation at the AAN Annual Meeting about the spectrum of multiple sclerosis misdiagnosis in the era of the McDonald criteria.

Disclosures can be found at Neurology.org.

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No CME will be offered this week: Interviews based on articles from *Neurology*® *Clinical Practice*, *Neurology: Genetics*, and *Neurology*® *Neuroimmunology & Neuroinflammation* are excluded from the CME program.

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