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Racial Disparities in Blood Pressure at Time of Acute Ischemic Stroke Presentation: A Population Study

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Journal of the American Heart Association

ORIGINAL RESEARCH

Racial Disparities in Blood Pressure at Time of Acute Ischemic Stroke Presentation: A Population Study

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BACKGROUND: Hypertension is a stroke risk factor with known disparities in prevalence and management between Black and White patients. We sought to identify if racial differences in presenting blood pressure (BP) during acute ischemic stroke exist.

METHODS AND RESULTS: Adults with acute ischemic stroke presenting to an emergency department within 24 hours of last known normal during study epochs 2005, 2010, and 2015 within the Greater Cincinnati/Northern Kentucky Stroke Study were included. Demographics, histories, arrival BP, National Institutes of Health Stroke Scale score, and time from last known normal were collected. Multivariable linear regression was used to determine differences in mean BP between Black and White patients, adjusting for age, sex, National Institutes of Health Stroke Scale score, history of hypertension, hyperlipidemia, smoking, stroke, body mass index, and study epoch. Of 4048 patients, 853 Black and 3195 White patients were included. In adjusted analysis, Black patients had higher presenting systolic BP (161 mm Hg [95% CI, 159–164] versus 158 mm Hg [95% CI, 157–159], $P<0.01$), diastolic BP (86 mm Hg [95% CI, 85–88] versus 83 mm Hg [95% CI, 82–84], $P<0.01$), and mean arterial pressure (111 mm Hg [95% CI, 110–113] versus 108 mm Hg [95% CI, 107–109], $P<0.01$) compared with White patients. In adjusted subanalysis of patients <4.5 hours from last known normal, diastolic BP (88 mm Hg [95% CI, 86–90] versus 83 mm Hg [95% CI, 82–84], $P<0.01$) and mean arterial pressure (112 mm Hg [95% CI, 110–114] versus 108 mm Hg [95% CI, 107–109], $P<0.01$) were also higher in Black patients.

CONCLUSIONS: This population-based study suggests differences in presenting BP between Black and White patients during acute ischemic stroke. Further study is needed to determine whether these differences influence clinical decision-making, outcome, or clinical trial eligibility.

Key Words: acute stroke ■ blood pressure ■ epidemiology ■ ischemic stroke ■ race ■ thrombolysis

Race is a social construct with important implications for the treatment and health outcomes of Black patients.[1] Black patients tend to have higher hypertension prevalence, lower treatment rates, and higher hypertension-associated mortality due to multifaceted interplay between biological, behavioral, environmental, and sociocultural causes of structural racism.[2,3] Such systemic inequity contributes to higher ischemic stroke incidence and mortality in Black patients.[4,5] Although racial disparities for blood pressure

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CLINICAL PERSPECTIVE

What Is New?

- We used the Greater Cincinnati Northern Kentucky Stroke Study to determine that Black patients had consistently higher systolic, diastolic, and mean arterial blood pressures compared with White patients when presenting to an emergency department for ischemic stroke within 24 hours from last known normal time.

What Are the Clinical Implications?

- Our findings highlight an opportunity for clinicians to pursue more aggressive antihypertensive treatment in Black patients to allow for safe thrombolytic administration or acute stroke clinical trial enrollment.
- More research is needed to determine how much these differences are due to longstanding blood pressure control disparities and how elevated blood pressure in Black patients affects clinical decision-making and outcomes.

Nonstandard Abbreviations and Acronyms

AIS	acute ischemic stroke
GCNKSS	Greater Cincinnati Northern Kentucky Stroke Study
LKN	last known normal
NIHSS	National Institutes of Health Stroke Scale

(BP) management have been well defined in the outpatient setting, no study to date has evaluated whether Black patients have higher presenting BPs during acute ischemic stroke (AIS).[6,7]

Prior studies have shown systolic BP>185 mm Hg or diastolic BP >110 mm Hg is a leading exclusionary criteria for thrombolytic treatment, with antihypertensive treatment of Black patients trending toward higher rates than White patients.[8,9] Understanding racial differences in BP during AIS presentation would provide insight into another potential mechanism for lower acute treatment rates of Black patients with thrombolytics and provide valuable information for clinical trialists seeking to overcome historical underrepresentation of Black patients.[10,11]

Using the GCNKSS (Greater Cincinnati/Northern Kentucky Stroke Study), we selected adult patients with a diagnosis of AIS presenting within 24 hours of symptom onset to determine if racial differences in BP during AIS presentation exist. We hypothesized that Black patients would have higher presenting systolic and diastolic BPs than White patients.

METHODS

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Patient Selection

The GCNKSS is a stroke epidemiological study, with full methodology previously described.[12] Briefly, the GCNKSS is a prospective population-based study of stroke incidence, risk factors, treatment, and outcomes within a 5-county region comprising approximately 1.3 million people. The study population is reflective of the percentage of Black and college-educated people, as well as the median socioeconomic distribution, in the greater United States. Cases from 16 hospitals and health care facilities located in rural and urban settings are retrospectively ascertained by discharge *International Classification of Diseases*, *Ninth* and *Tenth Revision* diagnoses (*ICD-9*: 430-438; *ICD-10*: G45-G56, H34.11, H34.12, I60-I69).

Study nurses review charts of all potential strokes and abstract pertinent demographic information and clinical data (stroke risk factors, presenting symptoms, physical examination, medications, laboratory results, and imaging results). Stroke-trained physicians review the abstracted information to determine stroke occurrence, stroke subtype (ie, ischemic or hemorrhagic), and stroke cause.[12] National Institutes of Health Stroke Scale (NIHSS) scores are retrospectively determined via extrapolation from physical examination or recorded if documented on admission.[13] The GCNKSS is approved by the institutional review boards of all participating locations with a waiver from informed consent.

The current investigation is a retrospective analysis of adult (age≥18 years) patients among Black and White residents of the Greater Cincinnati/Northern Kentucky region who presented to an emergency department (ED) for AIS during study epochs 2005, 2010, and 2015. Subjects were excluded if they were not Black or White, AIS occurred during hospitalization, patient was a direct admission, or no ED records were found. Subjects were additionally excluded with missing information, including timing of last known normal (LKN) or arrival to ED and patients arriving >24 hours from LKN time.

Study Variables

Patient demographics and presenting clinical information such as age, sex, race, body mass index (BMI), NIHSS score, medical history (hypertension, smoking,

hyperlipidemia, smoking status, prior stroke), treated hypertension, arrival systolic BP, arrival diastolic BP, arrival mean arterial pressure (MAP), time of presentation to ED, and time of LKN were collected. The number and type of antihypertensive medications was collected when available (2015 study epoch only). Race was abstracted from the electronic medical record. This method is congruent with the 1997 Office of Management and Budget standards, which has good agreement with patient self-report in prior studies.[14,15] Time of patient arrival to ED was ascertained based on the time of witnessed onset or, if onset time was unknown, the time between LKN and ED arrival.

Outcomes

The primary outcome was differences in presenting BP (mm Hg). Each subject had several BPs abstracted, but only the first documented BP upon arrival to the ED was used for this analysis. BP at time of presentation was determined in proximity to LKN time. MAP was calculated from presenting systolic and diastolic BPs.

Statistical Analysis

Demographic and clinical characteristics were described by race with median and interquartile range (IQR) for continuous variables and frequency and percentage for categorical variables. Multivariable linear regression was used to determine differences in mean BP between Black and White groups at time from LKN, adjusting for covariates. Covariates included age, sex, NIHSS score, history of hypertension, study epoch (2005, 2010, or 2015), BMI, current smoking, hyperlipidemia, and prior stroke. A final analysis including number of BP medications as an additional covariate for the 2015 study period was performed. Model assumptions were checked, and violations of homoscedasticity and normality of the model residual errors, using residual plots and model assumptions, appeared valid for each model. Model fit measures, the F test *P* value, and adjusted R^2 measures are presented for each model.

RESULTS

There were 6887 ischemic strokes of patients aged 18 years or older across all 3 study periods. Events excluded were 684 in-hospital strokes, 174 direct admissions, 22 patients with unknown/no record of ED events, and 481 due to missing times (11 missing emergency department arrival times and 470 with unknown LKN times). A total of 1441 subjects arrived >24 hours of LKN, 25 with a race other than Black or White, and 12 with missing presenting BP values.

Demographic information, initial NIHSS score, and medical history were collected for Black and White patients (Table 1). On average, Black patients were younger than White patients (median 64, IQR 54–76 versus 74, IQR 62–83), had higher NIHSS scores (median 4, IQR 2–9 versus 3, IQR 1–8), higher BMI (median 28.3, IQR 24.3–33.5 versus 26.8, IQR 23.4–31.0), were more likely to be smokers (33% versus 22%), have a prior history of stroke (36% versus 25%), but less likely to have hyperlipidemia (48% versus 54%). They were more likely to have a diagnosis of hypertension (89% versus 79%) and were more likely to be treated for hypertension before stroke presentation (81% versus 71%).

Table 1. Adults Presenting in 2005, 2010, and 2015 With Acute Ischemic Stroke

	Black (n=853)	White (n=3195)
Age, y, median (IQR)	64 (54–76)	74 (62–83)
Female sex	475 (56%)	1738 (54%)
National Institutes of Health Stroke Scale score, median (IQR)	4 (2–9)	3 (1–8)
Known history of hypertension	755 (89%)	2536 (79%)
Treated hypertension	688 (81%)	2269 (71%)
Hyperlipidemia	411 (48%)	1711 (54%)
Current smoker	281 (33%)	699 (22%)
Body mass index, median (IQR)	28.3 (24.3–33.5) [n=777]	26.8 (23.4–31.0) [n=2964]
Prior stroke	310 (36%)	811 (25%)

Treated hypertension was defined by at least 1 antihypertensive medication prescribed in the electronic medical record at time of presentation.

Arrival BP and MAP were analyzed between Black and White patients presenting within 24 hours of LKN time (Table 2). Black patients were more likely to have higher systolic BP (mean difference [MD], 4.5 [95% CI, 2.1–6.8], $P<0.01$), diastolic BP (MD, 6.7 [95% CI, 5.2–8.2], $P<0.01$), and MAP (MD, 6.0 [95% CI, 4.4–7.5], $P<0.01$) compared with White patients. Adjusted analysis could be performed on only 777 (out of 853) Black and 2964 (out of 3195) White patients due to missing BMI data. When adjusted for age, sex, NIHSS score, history of hypertension, hyperlipidemia, smoking, prior stroke, BMI, and study period, Black patients still had significantly higher average presenting systolic BP (MD, 3.6 [95% CI, 1.1–6.2], $P<0.01$), diastolic BP (MD, 3.4 [95% CI, 1.9–5.0], $P<0.01$), and MAP (MD, 3.5 [95% CI, 1.9–5.2], $P<0.01$) than White patients. A table of presenting BP by stroke subtype can be found in Table S1.

Black and White patients were next compared in the hyperacute window, defined as time <4.5 hours from LKN (Table 3). Adjusted analysis could be performed for only 356 (out of 393) Black and 1360 (out of 1471) White patients due to missing BMI data. In adjusted analysis, Black patients had higher diastolic BP (MD, 5.7 [95% CI, 3.4–8.0], $P<0.01$) and MAP (MD, 4.6 [95% CI, 2.1–7.0], $P<0.01$) compared with White patients.

Table 2. Multivariable Analysis of Presenting Blood Pressure by Race and Time Within 24 Hours of Last Known Normal

	Black (n=853)	White (n=3195)	Mean difference (95% CI)	*P* value
Arrival BP within 24 h of last known normal				
Systolic BP, mean (95% CI)	162 (160–164)	158 (156–159)	4.5 (2.1–6.8)	<0.01
Diastolic BP, mean (95% CI)	89 (88–90)	82 (82–83)	6.7 (5.2–8.2)	<0.01
Mean arterial pressure, mean (95% CI)	113 (112–115)	107 (106–108)	6.0 (4.4–7.5)	<0.01
	(n=777)	(n=2964)		
Adjusted arrival BP within 24 h of last known normal*				
Systolic BP, mean (95% CI)	161 (159–164)	158 (157–159)	3.6 (1.1–6.2)	<0.01
Diastolic BP, mean (95% CI)	86 (85–88)	83 (82–84)	3.4 (1.9–5.0)	<0.01
Mean arterial pressure, mean (95% CI)	111 (110–113)	108 (107–109)	3.5 (1.9–5.2)	<0.01

Measures of model fit: systolic BP model F test $P<0.001$ and adjusted $R^2=0.02$; diastolic BP model F test $P<0.001$ and adjusted $R^2=0.08$; mean arterial pressure model F test $P<0.001$ and adjusted $R^2=0.04$. BP is reported in mm Hg. BP indicates blood pressure.

*Model includes age, sex, National Institutes of Health Stroke Scale score, history of hypertension, history of hyperlipidemia, current smoking, prior stroke, body mass index, and study period.

A sensitivity analysis of the 2015 study epoch containing the number of antihypertensive medications for all patients presenting with AIS within 24 hours of LKN to an ED was performed (Table 4). A total of 347 (out of 350) Black and 1164 (out of 1174) White patients were included due to missing BMI values. After adjusting for age, sex, NIHSS score, history of hypertension, hyperlipidemia, smoking, prior stroke, BMI, and number of antihypertensive medications, there was no statistical difference in systolic BP (MD, 2.7 [95% CI, −1.1 to 6.5], *P*=0.16), diastolic BP (MD, 2.2 [95% CI, −0.2 to 4.6], *P*=0.07), or MAP (MD, 2.4 [95% CI, −0.2 to 4.9], *P*=0.07) between Black and White patients. Antihypertensive type and number of drugs can be found in Table S2.

In a final analysis, Black and White patient BPs were compared over 2005, 2010, and 2015 study years to assess for temporal change. No significant change in presenting systolic BP was observed comparing years 2005, 2010, and 2015 in Black patients (mean 164, SD 35; 162, SD 33; 160, SD 33, respectively) or in White patients (158, SD 31; 157, SD 30; 157, SD 29, respectively). Similarly, no significant change was observed in presenting diastolic BP between 2005, 2010, and 2015 in Black patients (mean 90, SD 22; 90, SD 21; 87, SD 21, respectively) or in White patients (83, SD 19; 82, SD 19; 83, SD 19, respectively).

DISCUSSION

In our population-based stroke epidemiology study, we found significantly higher systolic and diastolic BPs in Black patients compared with White patients, a difference that remained consistent when adjusted by age, sex, NIHSS score, history of hypertension, hyperlipidemia, smoking, prior stroke, BMI, and study period. Moreover, this finding was statistically unchanged across all 3 study periods spanning 10 years. To our knowledge, these are the first prospectively collected data examining racial differences in BP during AIS.

Black patients in our population were ≈10 years younger on average than White patients presenting with AIS, a finding that replicates other studies.[4,16] We also found a significantly higher presenting NIHSS score in Black compared with White patients. This is somewhat atypical, as other epidemiological studies have found Black patients to have similar or lower NIHSS scores than White patients.[17,18] However, a large study of

Table 3. Emergency Department Recordings of Presenting Blood Pressure by Race and Time for Patients Presenting in <4.5 Hours of Last Known Normal

	Black (n=356)	White (n=1360)	Mean difference (95% CI)	*P* value
Adjusted arrival BP within 4.5 h of last known normal*				
Systolic BP, mean (95% CI)	160 (157–163)	158 (156–160)	2.2 (−1.5–6.0)	0.24
Diastolic BP, mean (95% CI)	88 (86–90)	83 (82–84)	5.7 (3.4–8.0)	<0.01
Mean arterial pressure, mean (95% CI)	112 (110–114)	108 (107–109)	4.6 (2.1–7.0)	<0.01

Measures of model fit: systolic BP model F test $P<0.001$ and adjusted $R^2=0.02$; diastolic BP model F test $P<0.001$ and adjusted $R^2=0.11$; mean arterial pressure model F test $P<0.001$ and adjusted $R^2=0.05$. BP is reported in mm Hg. BP indicates blood pressure.

*Model includes age, sex, National Institutes of Health Stroke Scale score, history of hypertension, history of hyperlipidemia, current smoking, prior stroke, body mass index, and study period.

Table 4. Emergency Department Recordings of Presenting Blood Pressure by Race and Time for Patients Presenting Within 24 Hours of Last Known Normal in 2015 Only

	Black (n=347)	White (n=1164)	Mean difference (95% CI)	*P* value
Adjusted arrival BP within 24 h of last known normal*				
Systolic BP, mean (95% CI)	161 (158–164)	158 (157–160)	2.7 (−1.1–6.5)	0.16
Diastolic BP, mean (95% CI)	86 (84–88)	84 (83–85)	2.2 (−0.2–4.6)	0.07
Mean arterial pressure, mean (95% CI)	111 (109–113)	109 (108–110)	2.4 (−0.2–4.9)	0.07

Measures of model fit: systolic BP model F test $P<0.001$ and adjusted $R^2=0.04$; diastolic BP model F test $P<0.001$ and adjusted $R^2=0.08$; mean arterial pressure model F test $P<0.001$ and adjusted $R^2=0.05$. BP is reported in mm Hg. BP indicates blood pressure.

*Model includes age, sex, National Institutes of Health Stroke Scale score, history of hypertension, history of hyperlipidemia, current smoking, prior stroke, body mass index, and number of antihypertensive medications.

veterans did show that Black patients presented with worse stroke severity than White patients, even after controlling for prior stroke and stroke subtype.[19]

Black patients were also more likely to have hypertension and were more likely than White patients to have been prescribed at least 1 antihypertensive medication. Of note, "treated hypertension" in our study does not imply appropriately controlled outpatient BP. For this reason, it is difficult to determine whether our results are congruent with other studies, in which Black patients are more likely to have uncontrolled hypertension in the outpatient setting despite treatment.[20] However, the sensitivity analysis of the 2015 study epoch suggests that number of BP medications prescribed is relevant. When adjusted for the number of antihypertensive medications, in addition to other significant variables such as age and NIHSS score, BP differences between races were no longer significant.

It has been hypothesized that poor BP control in Black patients is associated with younger age of AIS. In an analysis of the REGARDS (Reasons for Geographic and Racial Differences in Stroke) study following 27 748 patients for 4.5 years, a 10 mm Hg elevation in outpatient systolic BP conferred a 24% risk of increased stroke for Black patients compared with only an 8% risk of increased stroke in White patients.[21] In our study, which is limited by the absence of prestroke BP recordings, it is difficult to deduce whether the racial difference in presenting BP is due to prestroke BP control, racial differences in the acute hypertensive response, or both.

We also examined whether BPs within 4.5 hours of LKN were different between Black and White patients. Interestingly, only diastolic BP and MAP were higher in Black patients. The biologic implication of our findings is uncertain, as the relationship between systolic BP, diastolic BP, MAP, and stroke outcome is not fully elucidated. Although MAP is known to have a direct relationship with cerebral perfusion pressure, peaks and variability in these metrics also appear to influence outcome.[22] Acute systemic hypertension is often seen during AIS, with some contribution from prestroke hypertension.[23] Racial differences in the acute hypertensive response may in part be explained by a differential sensitivity to the renin-angiotensin-aldosterone system, hypothesized to be a key driver of sympathetic tone following AIS.[23–25] It is unclear based on current evidence whether the acute hypertensive response occurs as a compensatory mechanism to increase blood flow through collateral vasculature or whether it is simply a reaction triggered by various physiologic consequences of stroke.[23] It is possible that in response to acute stroke, diastolic BP and thereby MAP are preferentially increased, perhaps to maintain perfusion pressure. Because cerebral perfusion pressure is largely regulated by MAP rather than intracranial pressure, increases in MAP disproportionately affect cerebral perfusion pressure, particularly when cerebral autoregulation is impaired.[22] Of note, this impairment has been observed in up to 32% of patients.[26] A complete review of MAP, its relationship to hemorrhage risk, and ultimately its impact on clinical outcome was beyond the scope of this study.

Practically, our findings may influence treatment decisions and acute clinical trial enrollment. BP >185/110 mm Hg resistant to acute antihypertensive treatment remains an absolute contraindication to thrombolytic administration within 4.5 hours due to risk of hemorrhagic transformation.[27] Although a difference of 4 mm Hg or 6 mm Hg in systolic or diastolic BP in Black patients may not seem profoundly disparate, this difference may act in concert with known disparities in stroke presentation time, emergency medical services use, ED triage times, treatment-resistant hypertension rates, consent time, cultural skepticism of the medical system, and overall lower treatment of Black patients with thrombolysis to work against prompt reperfusion compared with White patients.[10,28–34] Moreover, distrust of the research process and trial exclusion criteria that disproportionally affect Black patients (eg, renal function) may combine with this seemingly small disparity in presenting BP to hinder Black patient enrollment in acute stroke clinical trials.[35,36] Given the emphasis to increase equitable representation of Black patients for generalizability of results for AIS studies, trialists,

coordinators, and local coinvestigators must consider acute presenting BP differences and how this may affect enrollment of Black patients.[37] This may mean earlier preactivation with specific BP metrics or earlier use of antihypertensive medications with known advantages in Black patients (ie, calcium channel blockers over beta blockers).[38]

The strengths of this work inclide a large sample size by year and a population-based study with Black/White racial demographics mirroring that of the greater United States. The study is limited by its observational design, retrospective data collection after patient discharge, an inability to ascertain which patients had previously controlled BPs in the outpatient setting, and an inability to determine the magnitude of autoregulatory change in initial BPs obtained in the ED compared with the patient's baseline BP. As with any epidemiologic study, it is possible that our results are significant, may reflect either true association, statistically significant but not clinically significant, or are a result of unmeasured confounding.

CONCLUSIONS

Future trials are needed in order to understand whether clinical outcomes are affected by the increased BP parameters reported in Black patients and whether BP excludes some patients from expeditious treatment with thrombolysis differentially by race. Clinical trialists hoping to capture population-appropriate representation should consider the results of this study when planning future trials.

ARTICLE INFORMATION

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Disclosures

None.

Supplemental Material

Tables S1–S2

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