

Maternal Race/Ethnicity, Hypertension, and Risk for Stroke During Delivery Admission

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Background—Racial disparities contribute to maternal morbidity in the United States. Hypertension is associated with poor maternal outcomes, including stroke. Disparities in hypertension might contribute to maternal strokes.

Methods and Results—Using billing data from the Healthcare Cost and Utilization Project's National Inpatient Sample, we analyzed the effect of race/ethnicity on stroke during delivery admission in women aged 18 to 54 years delivering in US hospitals from January 1, 1998, through December 31, 2014. We categorized hypertension as normotensive, chronic hypertension, or pregnancy-induced hypertension. Adjusted risk ratios (aRRs) and 95% CIs were calculated using log-linear Poisson regression models, testing for interactions between race/ethnicity and hypertensive status. A total of 65 286 425 women were admitted for delivery during the study period, of whom 7764 were diagnosed with a stroke (11.9 per 100 000 deliveries). Hypertension modified the effect of race/ethnicity ($P < 0.0001$ for interaction). Among women with pregnancy-induced hypertension, black and Hispanic women had higher stroke risk compared with non-Hispanic whites (blacks: aRR, 2.07; 95% CI, 1.86–2.30; Hispanics: aRR, 2.19; 95% CI, 1.98–2.43). Among women with chronic hypertension, all minority women had higher stroke risk (blacks: aRR, 1.71; 95% CI, 1.30–2.26; Hispanics: aRR, 1.75; 95% CI, 2.32–5.63; Asian/Pacific Islanders: aRR, 3.62; 95% CI, 2.32–5.63). Among normotensive women, only blacks had increased stroke risk (aRR, 1.17; 95% CI, 1.07–1.28).

Conclusions—Pregnant US women from minority groups had higher stroke risk during delivery admissions, compared with non-Hispanic whites. The effect of race/ethnicity was larger in women with chronic hypertension or pregnancy-induced hypertension. Targeting blood pressure management in pregnancy may help reduce maternal stroke risk in minority populations. (*J Am Heart Assoc.* 2020;9:e014775. DOI: 10.1161/JAHA.119.014775.)

Key Words: disparities • hypertension • maternal morbidity • preeclampsia • pregnancy • stroke

The incidence of stroke in the US population has declined in recent years because of better control of cardiovascular risk factors, including hypertension.^{1,2} However, the opposite trend has been seen in pregnancy-related strokes, which have shown an incremental increase of 54% during the antepartum period and up to 83% increase during the postpartum period from 1994 to 1995 to 2006 to 2007.³

This alarming trend in maternal stroke may be attributable to a variety of factors, such as advancing maternal age^{4,5} and more patients initiating their pregnancies with ≥ 1 comorbidity,^{5,6} including cardiovascular risk factors, such as obesity,^{7,8} hypertension,⁹ and diabetes mellitus.^{5,10} Maternal stroke constitutes a public health concern affecting an estimated 30 in 100 000 pregnancies, with approximately one third of strokes occurring during delivery hospitalizations.^{11–13} The effects of cerebrovascular complications in this group are potentially financially catastrophic, considering the prohibitive expenses of health care and the impact on household income attributed to years of disability in young women.¹⁴ In addition, stroke in a young woman often has devastating effects on her family, including her newborn.¹⁵

Studies have shown clear racial disparities in maternal morbidity and mortality associated with preeclampsia and other hypertensive disorders of pregnancy, adjusting for other comorbidities, with black¹⁶ and Hispanic mothers having substantially higher risk compared with their white counterparts.¹⁷ These observed effects may be attributable to genetic differences, but socioeconomic status and quality of care

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Accompanying Tables S1 through S7 are available at <https://www.ahajournals.org/doi/suppl/10.1161/JAHA.119.014775>

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Clinical Perspective

What Is New?

- This study, using administrative data from the National Inpatient Sample, is the first to show that among US pregnant women with chronic or pregnancy-induced hypertension, women in minority groups (black, Hispanic, and Asian/Pacific Islander) have up to twice the risk of stroke during delivery admissions, compared with white women.
- Among normotensive women, black women remained at 17% higher risk of stroke during delivery admission, compared with white women; however, other minority groups did not have increased risk of stroke.

What Are the Clinical Implications?

- These results confirm that there are significant disparities in maternal stroke in the United States, and they suggest that targeted management of blood pressure in pregnancy may help to reduce these disparities, improving maternal outcomes in these high-risk populations.

have also been proposed as contributing factors.¹⁸ However, the effect of maternal race on the risk of stroke is not well characterized. Of note, maternal stroke and stroke-related death may be preventable^{19,20}: detailed case reviews have found that 30% to 60% of preeclampsia-related deaths, most of which were attributable to intracerebral hemorrhage (ICH),^{21–23} are preventable, and that timely administration of antihypertensive medications is associated with decreased risk for both fatal and nonfatal strokes.^{24,25} Thus, identifying racial disparities in maternal care, and particularly in the management of hypertension, may help prevent a large proportion of maternal strokes.

We hypothesized that women from minority groups would have higher risk of stroke during delivery hospitalizations. We further hypothesized that the effect of race/ethnicity on stroke risk would be different in women with hypertensive disorders, compared with women with normal blood pressure (ie, hypertension would modify the effect of race/ethnicity on stroke risk).

Methods

All data used to support the findings of this study are publicly available from the Healthcare Cost and Utilization Project's website at <https://www.hcup-us.ahrq.gov/nisoverview.jsp>. Statistical code used for this analysis is available from the corresponding author on reasonable request.

Study Population

We conducted a cross-sectional analysis using data from the Healthcare Cost and Utilization Project's National Inpatient

Sample (NIS) from 1998 to 2014. NIS is the largest publicly available, all-payer (Medicaid, commercial, and other insurance) inpatient database in the United States and includes a sample of 20% of all hospitalizations nationally. These hospitalizations are selected to include a sample that can be weighted to provide national estimates; sampling weights were applied for this analysis. Delivery hospitalizations, strokes, and comorbidities were identified using *International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM)*, diagnostic codes. Diagnostic codes 650 and V27.x were used to identify delivery hospitalizations in women aged 15 to 54 years; these codes have been shown to ascertain >95% of delivery hospitalizations.²⁶ Because the data are deidentified and publicly available, the Columbia University Institutional Review Board deemed this analysis exempt.

Exposures of Interest

The primary exposure of interest was race/ethnicity. The NIS categorizes race and ethnicity as Hispanic, non-Hispanic white, non-Hispanic black, Asian or Pacific Islander, Native American, other, or unknown. For this analysis, other and Native American were combined into one category ("other") given that small numerators would preclude meaningful statistical comparisons. We categorized women into 3 hypertensive groups: no hypertension (reference), chronic hypertension (cHTN) (*ICD-9-CM* codes 401.x-405.x and 642.0x-642.2x), and pregnancy-induced hypertension (PIH), including gestational hypertension (*ICD-9-CM* code 642.3), preeclampsia (*ICD-9-CM* codes 642.4x, 642.5x, and 642.7x), and eclampsia (*ICD-9-CM* code 642.6x). If a woman had a diagnosis of preeclampsia superimposed on cHTN, we considered her to be in the PIH category. We also performed a post hoc sensitivity analysis, excluding women with superimposed preeclampsia.

As covariates, we included demographic factors, including maternal age, year of delivery, insurance status (Medicaid, private, Medicare, other, or uninsured), and ZIP code income quartile. Hospital characteristics included bed size (small, medium, or large), location and teaching status (urban teaching, urban nonteaching, and rural), and region (North-east, Midwest, South, or West). To determine underlying comorbid risk, an obstetric comorbidity index was used.²⁷ This validated index provides weighted scores for comorbidity for individual patients on the basis of the presence of specific diagnosis codes and demographic factors present in administrative data. Higher scores are associated with increased risk for severe morbidity.²⁸ The variables included in this comorbidity index include age and the following conditions: pulmonary hypertension, sickle cell disease, chronic renal disease, preexisting hypertension, chronic

ischemic heart disease, congenital heart disease, cardiac valvular disease, chronic congestive heart failure, asthma, preexisting diabetes mellitus, obesity, systemic lupus erythematosus, HIV, and drug or alcohol use. Obstetric complications, such as placenta previa, multiple gestation, previous cesarean delivery, gestational hypertension, mild or unspecified preeclampsia, severe preeclampsia or eclampsia, and gestational diabetes mellitus, were also included. We categorized women on the basis of comorbidity index scores: 0 (lowest risk), 1 or 2, and >2 (highest). As we were specifically investigating the interaction effects of hypertensive disorders, in calculating the comorbidity index score, we excluded hypertensive disorders from the comorbidity index and adjusted for these variables separately in the models to avoid multicollinearity. Age was similarly excluded in the comorbidity index as this factor was controlled for independently in the model.

Primary Outcome

The primary outcome was maternal acute stroke diagnosis, identified during delivery hospitalization. “Stroke” was defined as acute ischemic stroke (*ICD-9-CM* codes 433.01, 433.11, 433.21, 433.31, 433.81, 433.91, 434.01, 434.11, 434.91, and 436); ICH (*ICD-9-CM* code 431); subarachnoid hemorrhage (*ICD-9-CM* code 430); cerebral venous thrombosis (CVT) (*ICD-9-CM* code 325); transient ischemic attack (TIA) (*ICD-9-CM* code 435); or acute cerebrovascular disease in the puerperium (*ICD-9-CM* codes 674.00, 674.01, 674.02, 674.03, and 674.04). We also investigated prespecified secondary outcomes of ischemic stroke alone (excluding TIA, CVT, and acute cerebrovascular disease in the puerperium) and hemorrhagic stroke alone (including only ICH [*ICD-9-CM* code 431] and subarachnoid hemorrhage [*ICD-9-CM* code 430]). In post hoc sensitivity analyses, we excluded CVT, TIA, and acute cerebrovascular disease in the puerperium. We also performed a separate post hoc sensitivity analysis including only ICH in the outcome. We did not differentiate by ICH mechanism (arteriovenous malformation rupture versus hypertensive hemorrhage) as the reliability of arteriovenous malformation diagnostic codes in administrative data is poor.²⁹ Furthermore, hypertension can contribute to risk of rupture of arteriovenous malformations.³⁰

Statistical Analysis

Demographic comparisons were evaluated using Rao-Scott χ^2 tests. Adjusted risk ratios (aRRs) for any stroke with 95% CIs as measures of effect, accounting for national weight, age, and comorbidities, were derived from fitting a log-linear Poisson regression model. To assess for effect modification between race/ethnicity and hypertensive status, we tested

the significance of interactions using the likelihood ratio test for model comparison.

Missing Data

Within the NIS, the proportion of obstetric patients with missing data on race varies by year³¹ but is lowest from 2012 to 2014. If outcomes based on race differed significantly for patients with unknown compared with known race, reported differentials may be biased. The Healthcare Cost and Utilization Project provides several recommendations for addressing missing data, including imputation.³² We elected not to perform imputation because it was unlikely that missing data on race was (i) random and (ii) nondifferential across the years of the NIS included in this analysis.³³ Instead, a sensitivity analysis was performed, restricted to 2012 to 2014, evaluating risk for any stroke by race. Given that the Healthcare Cost and Utilization Project’s data use agreement precludes reporting cell sizes ≤ 10 , rare outcomes are not reported. All analyses were performed with SAS 9.4 (SAS Institute, Cary, NC).

Results

A total of 65 286 425 women, aged 15 to 54 years, were admitted for delivery from 1998 to 2014. Of these women, 7764 were diagnosed with a new stroke during the delivery admission (11.9 strokes per 100 000 delivery hospitalizations). Demographic characteristics for the study population are detailed in Table 1. There was a higher proportion of black women in the stroke group, compared with the nonstroke group (16.4% in stroke group versus 10.8% in nonstroke group; $P < 0.0001$). Compared with women who did not have a stroke, women with strokes were older, had more obstetric comorbidities, and had higher proportion of hypertensive disorders, including cHTN (4.1% versus 1.2%) and PIH (34.5% versus 6.9%) ($P < 0.0001$). There were no significant between-group differences in income levels or US regional differences.

Primary Outcome

For the outcome of any maternal stroke, there was strong evidence for effect modification of hypertension on race/ethnicity, with a significant interaction between maternal race/ethnicity and maternal hypertensive status ($P < 0.0001$ for interaction). In stratified analyses, among women with no hypertension, black women had increased risk of stroke (aRR, 1.17; 95% CI, 1.07–1.28); there were no other significant differences in stroke risk between racial groups. However, among women with cHTN, black, Hispanic, and Asian/Pacific Islander women had higher risk of stroke, with the highest risk

Table 1. Characteristics of US Women, Aged 15 to 54 Years, Admitted for Delivery From 1998 to 2014

Characteristics	No Stroke (N=65 278 661)	Stroke (N=7764; 0.012%)	P Value From Rao-Scott χ^2 Test
Age, y			
15–17	2082 171 (3.2)	214 (2.8)	<0.0001
18–24	20 147 796 (30.9)	1880 (24.2)	
25–34	33 684 246 (51.6)	3994 (51.4)	
35–39	7 640 405 (11.7)	1316 (17)	
≥40	1 724 043 (2.6)	360 (4.6)	
Race			
White	27 787 540 (42.6)	3038 (39.1)	<0.0001
Black	7 059 614 (10.8)	1272 (16.4)	
Hispanic	11 758 578 (18)	1452 (18.7)	
Asian/Pacific Islander	2 504 843 (3.8)	263 (3.4)	
Other	2 909 044 (4.5)	301 (3.9)	
Income			
Low	13 684 742 (21)	1681 (21.7)	0.7923
Medium	15 960 897 (24.5)	1913 (24.6)	
High	16 308 428 (25)	1969 (25.4)	
Highest	18 169 393 (27.8)	2050 (26.4)	
Insurance			
Medicare	341 785 (0.5)	201 (2.6)	<0.0001
Medicaid	26 230 738(40.2)	3087 (39.8)	
Private	34 629 303 (53)	3950 (50.9)	
Self-pay	2127 613 (3.3)	304 (3.9)	
Hospital region			
Northeast	10 707 827 (16.4)	1307 (16.8)	0.4227
Midwest	14 012 304 (21.5)	1541 (19.8)	
South	24 333 021 (37.3)	3039 (39.1)	
West	16 225 508 (24.9)	1876 (24.2)	
Hospital teaching status			
Nonteaching	34 511 703 (52.9)	3113 (40.1)	<0.0001
Teaching	30 523 391(46.8)	4628 (59.6)	
No. of obstetric comorbidities			
0	51 588 639 (79)	5441 (70.1)	<0.0001
1–2	13 586 867 (20.8)	2138 (27.5)	
>2	103 154 (0.2)	185 (2.4)	
Hypertension			
Preeclampsia/eclampsia/ gestational hypertension	4484 275 (6.9)	2677 (34.5)	<0.0001
Chronic hypertension	752 162 (1.2)	321 (4.1)	

Data are given as number (percentage) and correspond to raw numbers and percentages from participants applying weighted frequencies.

Weighted percentage from missing data: race, 20.3% and 18.5%; income, 1.8% and 1.9%; and hospital teaching/no teaching, 0.4% and 0.3%, from nonstroke and stroke groups, respectively.

Table 2. Adjusted Risk of Any Stroke During Delivery Admissions in US Women, NIS 1998 to 2014, Stratified by Hypertensive Disorders

Race/ethnicity	No Hypertension			Chronic Hypertension			Pregnancy-Induced Hypertension		
	Weighted No.	Stroke Risk per 100 000	aRR (95%CI)	Weighted No.	Stroke Risk per 100 000	aRR (95%CI)	Weighted No.	Stroke Risk per 100 000	aRR (95%CI)
White	2106	8	Referent	100	32	Referent	832	42	Referent
Black	595	10	1.17 (1.07–1.28)*	98	56	1.71 (1.30–2.26)*	579	88	2.07 (1.86–2.30) [†]
Hispanic	746	7	0.85 (0.78–0.93)*	49	58	1.75 (1.24–2.47)*	657	92	2.19 (1.98–2.43) [†]
Asian/Pacific Islander	153	6	0.76 (0.64–0.89)*	25	126	3.62 (2.32–5.63) [†]	85	80	1.79 (1.43–2.24) [†]
Others	176	7	0.80 (0.69–0.94)*	NA	NA	0.63 (0.25–1.53)	120	67	1.58 (1.30–1.91) [†]

aRR adjusted for age and obstetric comorbidity index, excluding hypertension of any type. Pregnancy-induced hypertension included gestational hypertension, preeclampsia, preeclampsia superimposed on chronic hypertension, and eclampsia.

Weighted percentage from missing data across groups: no hypertension, 0.8%; chronic hypertension, 3.3%; pregnancy-induced hypertension, 4.6%. aRR indicates adjusted risk ratio; NA, number in the cell is <10 and is suppressed according to the NIS data use agreement; NIS, National Inpatient Sample.

* $P < 0.05$, [†] $P < 0.0001$.

seen in Asian/Pacific Islander women (aRR, 3.62; 95% CI, 2.32–5.63) (Table 2). Among women with PIH, all nonwhite groups were at increased risk of stroke, compared with white women, with a >2-fold risk seen in black and Hispanic women (aRR for black women: 2.07; 95% CI, 1.86–2.30; aRR for Hispanic women: 2.19; 95% CI, 1.98–2.43) (Table 2).

Secondary Outcomes

For the outcome of hemorrhagic maternal stroke, again a significant interaction was seen between maternal race/ethnicity and hypertensive status ($P = 0.002$ for interaction). Among nonhypertensive women, all minority groups were at increased risk of hemorrhagic stroke compared with white women, with the highest risk seen in black women (aRR, 1.87; 95% CI, 1.50–2.33) and Asian/Pacific Islander women (aRR, 1.93; 95% CI, 1.41–2.64). However, among women with

cHTN, black and Hispanic women were at >6-fold risk of hemorrhagic stroke, and Asian/Pacific Islander women had >17-fold risk of hemorrhagic stroke compared with whites (aRR, 17.35; 95% CI, 5.05–59.66). Finally, among women with PIH, all minority groups were at more than double risk of hemorrhagic stroke compared with whites, with the highest risk seen in Hispanic women (aRR, 2.87; 95% CI, 2.41–3.43) and Asian/Pacific Islander women (aRR, 2.84; 95% CI, 2.15–3.76) (Table 3).

Similarly, for the outcome of maternal ischemic stroke (excluding TIA), there was a significant interaction between maternal race/ethnicity and hypertensive status ($P = 0.003$ for interaction). Among women without hypertension, only black women had increased risk of ischemic stroke compared with whites (aRR, 1.41; 95% CI, 1.07–1.85). Among women with cHTN, small numbers limited the reporting of some minority groups, but black women had a nonsignificantly increased risk

Table 3. Adjusted Risk of Hemorrhagic Stroke During Delivery Admissions in US Women, NIS 1998 to 2014, Stratified by Hypertensive Disorders

Race	aRR (95% CI)		
	No Hypertension	Chronic Hypertension	Pregnancy-Induced Hypertension
White	Referent	Referent	Referent
Black	1.87 (1.50–2.33)*	6.57 (2.43–17.74) [†]	2.21 (1.83–2.69)*
Hispanic	1.25 (1.02–1.54) [†]	6.90 (2.31–20.66) [†]	2.87 (2.41–3.43)*
Asian/Pacific Islander	1.93 (1.41–2.64)*	17.35 (5.05–59.66)*	2.35 (1.64–3.38)*
Other	1.59 (1.15–2.20) [†]	NA	2.84 (2.15–3.76)*

aRR adjusted for age and obstetric comorbidity index, excluding hypertension. Hemorrhagic stroke included intracerebral hemorrhage and/or subarachnoid hemorrhage. Pregnancy-induced hypertension included gestational hypertension, preeclampsia, preeclampsia superimposed on chronic hypertension, and eclampsia. Weighted percentage from missing data across groups: no hypertension, 0.1%; chronic hypertension, 0.4%; preeclampsia/gestational hypertension, 1.3%. aRR indicates adjusted risk ratio; NA, number in the cell is <10 and is suppressed according to the NIS data use agreement; NIS, National Inpatient Sample.

* $P < 0.0001$, [†] $P < 0.05$.

Table 4. Adjusted Risk of Ischemic Stroke During Delivery Admissions in US Women, NIS 1998 to 2014, Stratified by Hypertensive Disorders

Race	aRR (95% CI)		
	No Hypertension	Chronic Hypertension	Pregnancy-Induced Hypertension
White	Referent	Referent	Referent
Black	1.41 (1.07–1.85)*	1.59 (0.77–3.29)	2.44 (2.00–2.97) [†]
Hispanic	0.99 (0.76–1.28)	NA	2.00 (1.63–2.46) [†]
Asian/Pacific Islander	0.46 (0.24–0.88)*	NA	1.61 (1.01–2.56)*
Other	0.70 (0.41–1.19)	3.99 (1.45–10.99)*	1.31 (0.87–1.97)

aRR adjusted for age and obstetric comorbidity index, excluding hypertension. Ischemic stroke excluded transient ischemic attack and cerebral venous sinus thrombosis. Pregnancy-induced hypertension included gestational hypertension, preeclampsia, preeclampsia superimposed on chronic hypertension, and eclampsia. Weighted percentage from missing data across groups: no hypertension, 0.06%; chronic hypertension, 0.4%; preeclampsia/gestational hypertension, 0.9%. aRR indicates adjusted risk ratio; NA, number in the cell is <10 and is suppressed according to the NIS data use agreement; NIS, National Inpatient Sample.

* $P<0.05$, [†] $P<0.0001$.

of ischemic stroke (aRR, 1.59; 95% CI, 0.77–3.29). Among women with PIH, all minority groups had higher risk of ischemic stroke, with the highest risk seen in black women (aRR, 2.44; 95% CI, 2.00–2.97) (Table 4).

Prespecified Sensitivity Analysis

In a sensitivity analysis of years 2012 to 2014, the years with the least missing data for race and ethnicity, we found similar disparities in the primary outcome. Evidence of effect modification was found between race/ethnicity and hypertensive status ($P<0.0001$ for interaction). Among normotensive women, Hispanic women had increased risk of any stroke (aRR, 1.32; 95% CI, 1.11–1.57), with no other significant differences seen between racial groups. Among women with cHTN, a 5-fold risk of maternal stroke was seen in black women (aRR, 5.14; 95% CI, 1.87–14.17) and >7-fold risk was seen among Hispanic women (aRR, 7.56; 95% CI, 2.58–22.14). Among women with PIH, all minority groups had more than double the risk of stroke compared with whites, with the highest risk in Asian/Pacific Islanders (aRR, 3.26; 95% CI, 2.21–4.83) (Tables S1 and S2). The relatively small sample limited our ability to report secondary outcomes in the sensitivity analysis.

Post Hoc Sensitivity Analyses

In a post hoc sensitivity analysis excluding women with superimposed preeclampsia, and excluding CVT and TIA from the outcome, results were almost identical to the primary analysis (Tables S3 through S6). A second post hoc sensitivity analysis with the outcome restricted to only ICH showed significant racial disparities in both nonhypertensive and PIH groups, but effect sizes were larger in the PIH group, particularly in Hispanic and Asian/Pacific Islander women (Table S7).

Discussion

Racial disparities in maternal morbidity and mortality have been consistently reported in the literature, with risk of maternal mortality 3.5-fold higher for black women.^{18,34} Our analysis of US delivery hospitalizations from 1998 to 2014 confirms a greater risk of all types of stroke among patients from minority groups compared with non-Hispanic whites, and provides new evidence that the effect of race and ethnicity on stroke risk is modified by the hypertensive status of the patient.

Socioeconomic differences, access to health services, and differences in the prevalence of comorbidities, such as hypertension⁹ and obesity,³⁵ have been hypothesized as potential causes for racial disparities in maternal outcomes. Our study, however, did not show between-group differences in income for patients with and without stroke and showed only small differences in insurance status. Furthermore, when we adjusted our data for age and number of comorbidities, minority women continued to have an increased risk of stroke.

Our findings show that the risk of stroke differs for minority women, compared with non-Hispanic whites, depending on a woman's hypertensive status. Specifically, black women without hypertension had a 17% increased risk of any stroke, an 87% increased risk of hemorrhagic stroke, and a 41% increased risk of ischemic stroke. However, in women with cHTN or PIH, all minority groups had increased risk of stroke. These effects were most dramatic in the case of hemorrhagic stroke, where black, Hispanic, and Asian/Pacific Islander patients had more than twice the risk for intracerebral or subarachnoid hemorrhage, adjusting for age and comorbidities. In the case of women with cHTN, the risk of hemorrhagic stroke was >6-fold higher among blacks and Hispanics, and 17-fold higher in Asian/Pacific Islanders, compared with white women (although small samples

precluded precise estimates for Asian/Pacific Islanders). Previous studies have demonstrated that black women are at increased risk for maternal morbidity and mortality.^{18,36–38} Our data suggest that Hispanic and Asian/Pacific Islander women, too, are at significantly increased risk for maternal stroke if they have hypertension.

The reasons for the disparities we detected are unclear. It is possible that genetic variation between groups portends a differential response to antihypertensive treatments³⁹ used in pregnancy. However, case series have revealed that a large proportion of deaths attributable to maternal stroke would have been preventable with earlier transfer to higher level of care or more aggressive blood pressure treatment.^{24,40} Our results suggest that minority women with hypertension may not be receiving adequate blood pressure treatment. In addition, in black women, even women without hypertension remained at increased risk of stroke, suggesting that additional factors besides hypertension may contribute to increased maternal morbidity in this group.

Our results have important implications for maternal care. Blood pressure goals may need to be reviewed in pregnant women from minority groups, to avoid cerebrovascular complications. The possibility of a variable response to antihypertensive medication, depending on individual racial and ethnic background, should be explored. A personalized approach to blood pressure management in pregnancy may offer particular benefits for minority women.

Strengths of our analysis include its large size, giving us the power to detect interactions and better understand the factors contributing to racial disparities in maternal health. In addition, because the NIS is a nationally weighted estimate of hospital admissions across the country, our results are highly generalizable to the US population. We used well-validated diagnostic codes for stroke and obstetric comorbidities.

Our study has limitations. These are observational data from an administrative database, limiting our ability to confirm the clinical diagnoses of PIH, cHTN, and strokes. In addition, administrative data have limited granularity and are unable to confirm the cause of stroke, limiting our ability to explore mechanistic hypotheses. The inclusion of TIA and CVT in the primary outcome may have overestimated the incidence of true stroke; however, our post hoc sensitivity analyses showed similar effects. In addition, although we used codes that have been well validated for acute stroke,⁴¹ we are unable to confirm with certainty whether this was a new diagnosis during the delivery admission, versus a stroke diagnosed before admission. However, our overall stroke incidence of 11.9 per 100 000 delivery hospitalizations is consistent with maternal stroke rates reported in prior studies of strokes occurring during delivery admissions.¹³ Furthermore, our secondary outcomes, which included only highly specific codes for acute intracerebral and subarachnoid

hemorrhage and acute ischemic stroke, still showed highly significant interactions and even larger effect sizes. Race and ethnicity were self-reported, and misclassification may have occurred. In addition, small numbers precluded our statistical power to examine other high-risk groups, such as Native American women. Missing data on race and ethnicity may have affected our results; however, our sensitivity analysis for the years with the least missing data showed similar findings. Because our primary interest was in the interaction of race and ethnicity with hypertension on stroke risk, to reduce the risk of type 1 error we adjusted only for age and number of comorbidities, rather than analyzing the effects of multiple individual covariates on the primary outcome; this may have increased our type 2 error. Some specific risk factors for peripartum stroke, such as infections,^{42,43} are not included in the obstetric comorbidity index, and this could have influenced our estimates of comorbidity risk. Similar to prior studies, our data showed a higher proportion of stroke in older age groups. Although we adjusted for this possible confounder, we did not evaluate for 3-way interactions between age, race, and hypertensive status; this could be specifically investigated in future studies. Our study looked only at strokes occurring during delivery hospitalizations, which constitute approximately one third of total pregnancy-associated strokes¹³; for this analysis, we did not investigate maternal readmissions for strokes in the postpartum period. Administrative data have limited ability to ascertain timing of the stroke during the delivery admission (antepartum, intrapartum, or postpartum) because only the nonspecific “cerebrovascular disorders in the puerperium” codes include modifiers for delivery timing. In addition, the NIS data set cannot address if strokes were attributable to undertreatment or if they occurred at lower blood pressures than strokes seen in the referent group. How cHTN was managed during pregnancy is also unknown; thus, we were unable to evaluate whether the severity of hypertension and status of controlled versus uncontrolled hypertension contributed to the observed differences in stroke rates on the basis of race. Access and quality of blood pressure treatment may differ in different racial and ethnic groups, contributing to differences in overall stroke risk. Given these important limitations, our results should be interpreted with caution and should be considered hypothesis generating.

Conclusions

In our sample of 65 million delivery admissions in the United States, black, Hispanic, and Asian/Pacific Islander women with a diagnosis of cHTN or PIH had higher risk of maternal stroke, compared with non-Hispanic whites. Among women without hypertension, only black women had increased risk of

maternal stroke. Prospective research is needed to develop more effective management strategies for hypertension in pregnancy, particularly in minority women who have outsized risk of maternal stroke.

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References

- Vangen-Lonne AM, Wilsgaard T, Johnsen SH, Lochan ML, Njolstad I, Mathiesen EB. Declining incidence of ischemic stroke: what is the impact of changing risk factors? The Tromso study 1995 to 2012. *Stroke*. 2017;48:544–550.
- Thiele I, Linseisen J, Heier M, Holle R, Kirchberger I, Peters A, Thorand B, Meisinger C. Time trends in stroke incidence and in prevalence of risk factors in Southern Germany, 1989 to 2008/09. *Sci Rep*. 2018;8:11981.
- Kuklina EV, Tong X, Bansil P, George MG, Callaghan WM. Trends in pregnancy hospitalizations that included a stroke in the United States from 1994 to 2007: reasons for concern? *Stroke*. 2011;42:2564–2570.
- James AH, Bushnell CD, Jamison MG, Myers ER. Incidence and risk factors for stroke in pregnancy and the puerperium. *Obstet Gynecol*. 2005;106:509–516.
- Sheen J-J, Wright JD, Goffman D, Kern-Goldberger AR, Booker W, Siddiq Z, D'Alton ME, Friedman AM. Maternal age and risk for adverse outcomes. *Am J Obstet Gynecol*. 2018;219:390.e1–390.e15.
- Arany Z, Elkayam U. Peripartum cardiomyopathy. *Circulation*. 2016;133:1397–1409.
- Fisher SC, Kim SY, Sharma AJ, Rochat R, Morrow B. Is obesity still increasing among pregnant women? Prepregnancy obesity trends in 20 states, 2003–2009. *Prev Med*. 2013;56:372–378.
- Heslehurst N, Ellis L, Simpson H, Batterham A, Wilkinson J, Summerbell C. Trends in maternal obesity incidence rates, demographic predictors, and health inequalities in 36 821 women over a 15-year period. *BJOG*. 2007;114:187–194.
- Leffert LR, Clancy CR, Bateman BT, Bryant AS, Kuklina EV. Hypertensive disorders and pregnancy-related stroke: frequency, trends, risk factors, and outcomes. *Obstet Gynecol*. 2015;125:124–131.
- Scott CA, Bewley S, Rudd A, Spark P, Kurinczuk JJ, Brocklehurst P, Knight M. Incidence, risk factors, management, and outcomes of stroke in pregnancy. *Obstet Gynecol*. 2012;120:318–324.
- Miller EC, Gatollari HJ, Too G, Boehme AK, Leffert L, Elkind MS, Willey JZ. Risk of pregnancy-associated stroke across age groups in New York state. *JAMA Neurol*. 2016;73:1461–1467.
- Yoshida K, Takahashi JC, Takenobu Y, Suzuki N, Ogawa A, Miyamoto S. Strokes associated with pregnancy and puerperium: a nationwide study by the Japan stroke society. *Stroke*. 2017;48:276–282.
- Swartz RH, Cayley ML, Foley N, Ladhani NNN, Leffert L, Bushnell C, McClure JA, Lindsay MP. The incidence of pregnancy-related stroke: a systematic review and meta-analysis. *Int J Stroke*. 2017;12:687–697.
- Synhaeve NE, Arntz RM, van Alebeek ME, van Pamelan J, Maaijwee NA, Rutten-Jacobs LC, Schoonderwaldt HC, de Kort PL, van Dijk EJ, de Leeuw FE. Women have a poorer very long-term functional outcome after stroke among adults aged 18–50 years: the FUTURE study. *J Neurol*. 2016;263:1099–1105.
- Aarnio K, Gissler M, Grittner U, Siegerink B, Kaste M, Tatlisumak T, Tikkanen M, Putaala J. Outcome of pregnancies and deliveries before and after ischaemic stroke. *Eur Stroke J*. 2017;2:346–355.
- Creanga AA, Berg CJ, Syverson C, Seed K, Bruce FC, Callaghan WM. Race, ethnicity, and nativity differentials in pregnancy-related mortality in the United States: 1993–2006. *Obstet Gynecol*. 2012;120:261–268.
- Persky RW, Turtzo LC, McCullough LD. Stroke in women: disparities and outcomes. *Curr Cardiol Rep*. 2010;12:6–13.
- Petersen EE, Davis NL, Goodman D, Cox S, Mayes N, Johnston E, Syverson C, Seed K, Shapiro-Mendoza CK, Callaghan WM, Barfield W. Vital signs: pregnancy-related deaths, United States, 2011–2015, and strategies for prevention, 13 states, 2013–2017. *MMWR Morb Mortal Wkly Rep*. 2019;18:423–429.
- Berg CJ, Harper MA, Atkinson SM, Bell EA, Brown HL, Hage ML, Mitra AG, Moise KJ Jr, Callaghan WM. Preventability of pregnancy-related deaths: results of a state-wide review. *Obstet Gynecol*. 2005;106:1228–1234.
- Clark SL, Belfort MA, Dildy GA, Herbst MA, Meyers JA, Hankins GD. Maternal death in the 21st century: causes, prevention, and relationship to cesarean delivery. *Am J Obstet Gynecol*. 2008;199:36.e1–36.e5; discussion 91-2.e7-e11.
- Bateman BT, Schumacher HC, Bushnell CD, Pile-Spellman J, Simpson LL, Sacco RL, Berman MF. Intracerebral hemorrhage in pregnancy: frequency, risk factors, and outcome. *Neurology*. 2006;67:424–429.
- Liang Z-W, Lin L, Gao W-L, Feng L-M. A clinical characteristic analysis of pregnancy-associated intracranial haemorrhage in China. *Sci Rep*. 2015;5:9509.
- Foo L, Bewley S, Rudd A. Maternal death from stroke: a thirty year national retrospective review. *Eur J Obstet Gynecol Reprod Biol*. 2013;171:266–270.
- Martin JN Jr, Thigpen BD, Moore RC, Rose CH, Cushman J, May W. Stroke and severe preeclampsia and eclampsia: a paradigm shift focusing on systolic blood pressure. *Obstet Gynecol*. 2005;105:246–254.
- Cleary KL, Siddiq Z, Ananth CV, Wright JD, Too G, D'Alton ME, Friedman AM. Use of antihypertensive medications during delivery hospitalizations complicated by preeclampsia. *Obstet Gynecol*. 2018;131:441–450.
- Kuklina EV, Whiteman MK, Hillis SD, Jamieson DJ, Meikle SF, Posner SF, Marchbanks PA. An enhanced method for identifying obstetric deliveries: implications for estimating maternal morbidity. *Matern Child Health J*. 2008;12:469–477.
- Bateman BT, Mhyre JM, Hernandez-Diaz S, Huybrechts KF, Fischer MA, Creanga AA, Callaghan WM, Gagne JJ. Development of a comorbidity index for use in obstetric patients. *Obstet Gynecol*. 2013;122:957–965.
- Metcalfe A, Lix LM, Johnson JA, Currie G, Lyon AW, Bernier F, Tough SC. Validation of an obstetric comorbidity index in an external population. *BJOG*. 2015;122:1748–1755.
- Berman MF, Stapf C, Sciacca RR, Young WL. Use of ICD-9 coding for estimating the occurrence of cerebrovascular malformations. *AJNR Am J Neuroradiol*. 2002;23:700–705.
- van Beijnum J, Wilkinson T, Whitaker HJ, van der Bom JG, Algra A, Vandertop WP, van den Berg R, Brouwer PA, Rinkel GJ, Kappelle LJ, Al-Shahi Salman R, Scottish Audit of Intracranial Vascular Malformations collaborators, Klijn CJ. Relative risk of hemorrhage during pregnancy in patients with brain arteriovenous malformations. *Int J Stroke*. 2017;12:741–747.
- Andrews RM. Statewide hospital discharge data: collection, use, limitations, and improvements. *Health Serv Res*. 2015;50:1273–1299.
- Ma Y, Zhang W, Lyman S, Huang Y. The HCUP SID imputation project: improving statistical inferences for health disparities research by imputing missing race data. *Health Serv Res*. 2018;53:1870–1889.

33. Agency for Healthcare Research and Quality (AHRQ). Healthcare Cost and Utilization Project NIS description of data elements. Available at: <http://www.hcup-us.ahrq.gov/db/vars/race/nisnote.jsp#general>. Accessed December 20, 2019.
34. Louis JM, Menard MK, Gee RE. Racial and ethnic disparities in maternal morbidity and mortality. *Obstet Gynecol*. 2015;125:690–694.
35. Agyemang P, Powell-Wiley TM. Obesity and black women: special considerations related to genesis and therapeutic approaches. *Curr Cardiovasc Risk Rep*. 2013;7:378–386.
36. Gyamfi-Bannerman C, Pandita A, Miller EC, Boehme AK, Wright JD, Siddiq Z, D'Alton ME, Friedman AM. Preeclampsia outcomes at delivery and race. *J Matern Fetal Neonatal Med*. 2019;1–8. DOI: 10.1080/14767058.2019.1581522.
37. Creanga AA, Bateman BT, Kuklina EV, Callaghan WM. Racial and ethnic disparities in severe maternal morbidity: a multistate analysis, 2008–2010. *Am J Obstet Gynecol*. 2014;210:435e431–435e438.
38. Shahul S, Tung A, Minhaj M, Nizamuddin J, Wenger J, Mahmood E, Mueller A, Shaefi S, Scavone B, Kociol RD, Talmor D, Rana S. Racial disparities in comorbidities, complications, and maternal and fetal outcomes in women with preeclampsia/eclampsia. *Hypertens Pregnancy*. 2015;34:506–515.
39. Spence JD, Rayner BL. Hypertension in blacks. *Hypertension*. 2018;72:263–269.
40. Katsuragi S, Tanaka H, Hasegawa J, Nakamura M, Kanayama N, Nakata M, Murakoshi T, Yoshimatsu J, Osato K, Tanaka K, Sekizawa A, Ishiwata I, Ikeda T; on behalf of the Maternal Death Exploratory Committee in Japan and Japan Association of Obstetricians and Gynecologists. Analysis of preventability of stroke-related maternal death from the nationwide registration system of maternal deaths in Japan. *J Matern Fetal Neonatal Med*. 2018;31:2097–2104.
41. Tirschwell DL, Longstreth WT Jr. Validating administrative data in stroke research. *Stroke*. 2002;33:2465–2470.
42. Miller EC, Wen T, Elkind MSV, Friedman AM, Boehme AK. Infection during delivery hospitalization and risk of readmission for postpartum stroke. *Stroke*. 2019;50:2685–2691.
43. Miller EC, Gallo M, Kulick ER, Friedman AM, Elkind MSV, Boehme AK. Infections and risk of peripartum stroke during delivery admissions. *Stroke*. 2018;49:1129–1134.

SUPPLEMENTAL MATERIAL

Table S1. Sensitivity analysis: characteristics of US women ages 15-54 admitted for delivery from 2012-2014.

	Delivery Cohort				P value from Rao-Scott Chi-Square Test
	Stroke No (N=11,077,094)		Stroke Yes (N=1,345; 0.012%)		
	Weighted N	Weighted %	Weighted N	Weighted %	
Race					<0.0001
White	5649533	51	540	40.1	.
Black	1522931	13.7	260	19.3	.
Hispanic	2194150	19.8	350	26	.
Asian or Pacific Islander	536345	4.8	50	3.7	.
Unknown	702245	6.3	70	5.2	.
Hypertension	.				<0.0001
None	9986029	90.2	790	58.7	
Preeclampsia/eclampsia/gestational hyper	907340	8.2	520	38.7	
Chronic hypertension	183725	1.7	35	2.6	
Comorbidity with age					<0.0001
0	45110986	69.1	4425	57	.
1 to 2	19126466	29.3	2898	37.3	.
>2	1041208	1.6	441	5.7	.
Age	<0.0001
15-17	2082171	3.2	214	2.8	.
18-24	20147796	30.9	1880	24.2	.
25-34	33684246	51.6	3994	51.4	.
35-39	7640405	11.7	1316	17	.
>39	1724043	2.6	360	4.6	.
Insurance	.			0	<0.0001
Medicare	341785	0.5	201	2.6	.
Medicaid	26230738	40.2	3087	39.8	.
Private	34629303	53	3950	50.9	.
Self pay	2127613	3.3	304	3.9	.
Other/Unknown	1949222	3	222	2.9	.
Income	.				0.7923
Low	13684742	21	1681	21.7	.

Medium	15960897	24.5	1913	24.6	.
High	16308428	25	1969	25.4	.
Highest	18169393	27.8	2050	26.4	.
Unknown	1155201	1.8	150	1.9	.
<i>Year of Delivery</i>					
2012	3748711	5.7	440	5.7	.
2013	3727113	5.7	455	5.9	.
2014	3783690	5.8	460	5.9	.
Hospital bedsize	.				0.0032
Small	1517395	13.7	100	7.4	.
Medium	3280174	29.6	335	24.9	.
Large	6279524	56.7	910	67.7	.
<i>Hospital region</i>	.				0.4227
Northeast	1797594	16.2	240	17.8	.
Midwest	2358677	21.3	265	19.7	.
South	4265497	38.5	485	36.1	.
West	2655326	24	355	26.4	.
<i>Hospital location</i>	.				<0.0001
Rural	1157588	10.5	55	4.1	.
Urban	9919506	89.5	1290	95.9	.
<i>Hospital teaching</i>	.				<0.0001
Non teaching	4974712	44.9	355	26.4	.
Teaching	6102381	55.1	990	73.6	.

Table S2. Sensitivity analysis: adjusted risk of any stroke during delivery admissions in US women, National Inpatient Sample 2012-2014, stratified by hypertensive disorders.

Race/ ethnicity	No hypertension		Chronic hypertension		Pregnancy-induced hypertension	
	Weighted N	aRR (95%CI)	Weighted N	aRR (95%CI)	Weighted N	aRR (95%CI)
White	375	Referent	5	Referent	160	Referent
Black	105	1.09 (0.88, 1.36)	15	5.14 (1.87, 14.17)*	140	2.46 (1.96, 3.09)**
Hispanic	195	1.32 (1.11, 1.57)*	10	7.56 (2.58, 22.14)*	145	2.73 (2.18, 3.41)**
Asian/ Pacific Islander	20	0.52 (0.33, 0.82)*	NA	NA	30	3.26 (2.21, 4.83)**
Others	45	0.96 (0.70, 1.31)	NA	NA	25	1.44 (0.94, 2.20)

*P<0.05, **P<0.0001

aRR (adjusted risk ratio) adjusted for age and obstetric comorbidity index, excluding hypertension of any type.

Pregnancy-induced hypertension included gestational hypertension, preeclampsia, preeclampsia superimposed on chronic hypertension, and eclampsia. NA indicates the number in the cell is less than 10 and is suppressed according to the National Inpatient Sample data use agreement.

Table S3. Characteristics of US women ages 15-54 admitted for delivery from 1998 to 2014, excluding patients with superimposed preeclampsia (preeclampsia with a history of cHTN, or 642.7x) from exposure, and CVT (325) and TIA (435) from outcome.

	No stroke (N=64,523,599)	Stroke (N=7,287; 0.013%)	P value from Rao- Scott Chi-Square Test
	No.(%)	No.(%)	
Age			<0.0001
15-17	2063926 (3.2)	206 (2.8)	
18-24	19945119 (30.9)	1800 (24.7)	
25-34	33296828 (51.6)	3806 (52.2)	
35-39	7528688 (11.7)	1173 (16.1)	
≥40	1689038 (2.6)	302 (4.1)	
Race			<0.0001
White	27679998 (42.9)	2901 (39.8)	
Black	6982394 (10.8)	1163 (16)	
Hispanic	11715445 (18.2)	1359 (18.6)	
Asian/Pacific Islander	2495726 (3.9)	249 (3.4)	
Other	2439068 (3.8)	248 (3.4)	
Income			0.8283
Low	13448532 (20.8)	1534 (21.1)	
Medium	15777478 (24.5)	1812 (24.9)	
High	16154616 (25)	1870 (25.7)	
Highest	18015210 (27.9)	1931 (26.5)	
Insurance			<0.0001
Medicare	336036 (0.5)	187 (2.6)	
Medicaid	25873958 (40.1)	2868 (39.4)	
Private	34291229 (53.1)	3754 (51.5)	
Self- pay	2103367 (3.3)	272 (3.7)	
Hospital region			0.2933
Northeast	10594361 (16.4)	1234 (16.9)	
Midwest	13884938 (21.5)	1434 (19.7)	
South	24040769 (37.3)	2880 (39.5)	
West	16003529 (24.8)	1740 (23.9)	
Hospital teaching			<0.0001
Non- teaching	34154560 (52.9)	2949 (40.5)	
Teaching	30131179 (46.7)	4315 (59.2)	
Number of obstetric			<0.0001
0	44662584 (69.2)	4231 (58.1)	
1 to 2	18842466 (29.2)	2655 (36.4)	
>2	1018548 (1.6)	401 (5.5)	
Hypertension			<0.0001
Preeclampsia/eclampsia/gestational hypertension	4155614 (6.4)	2259 (31)	
Chronic hypertension	747089 (1.2)	315 (4.3)	
N% Corresponds to raw numbers and percentages from participants applying weighted frequencies.			
^a Weighted percentage from missing data; Race 20.5%, 18.7%; Income 1.7%, 2.0%; Hospital teaching/no teaching 0.4%, 0.3% from non-stroke and stroke groups respectively.			

TableS4. Adjusted risk of any stroke during delivery admissions in US women, National Inpatient Sample 1998-2014, stratified by hypertensive disorders, excluding patients with superimposed preeclampsia (preeclampsia with a history of cHTN, or 642.7x) from exposure, and CVT (325) and TIA (435) from outcome.

Race/ ethnicity	No hypertension			Chronic hypertension			Pregnancy-induced hypertension		
	Weighted N	Stroke risk per 100,000	aRR (95%CI)	Weighted N	Stroke risk per 100,000	aRR (95%CI)	Weighted N	Stroke risk per 100,000	aRR (95%CI)
White	2091	8	Referent	95	30	Referent	715	39	Referent
Black	590	9	1.17 (1.07, 1.28)*	98	56	1.80 (1.36, 2.38)**	474	82	2.13 (1.90, 2.39)**
Hispanic	746	7	0.86 (0.79, 0.93)*	49	58	1.84 (1.30, 2.60)*	564	84	2.20 (1.97, 2.46)**
Asian/ Pacific Islander	153	6	0.76 (0.65, 0.90)*	25	126	3.79 (2.43, 5.91)**	71	73	1.82 (1.43, 2.33)**
Others	152	7	0.83 (0.70, 0.98)*	NA	NA	0.82 (0.34, 2.03)	91	68	1.75 (1.41, 2.18)**

* P<0.05, **P<0.0001

aRR (adjusted risk ratio) adjusted for age and obstetric comorbidity index, excluding hypertension of any type.

Pregnancy-induced hypertension included gestational hypertension, preeclampsia, and eclampsia. NA indicates the number in the cell is less than 10 and is suppressed according to the National Inpatient Sample data use agreement.

Table S5. Adjusted risk of hemorrhagic stroke during delivery admissions in US women, National Inpatient Sample 1998-2014, stratified by hypertensive disorders, excluding patients with superimposed preeclampsia (preeclampsia with a history of cHTN, or 642.7x).

Race	No hypertension aRR (95%CI)	Chronic hypertension aRR (95%CI)	Pregnancy-induced hypertension aRR (95%CI)
White	Referent	Referent	Referent
Black	1.87 (1.50, 2.33)**	6.57 (2.43, 17.74)*	2.65 (2.15, 3.26)**
Hispanic	1.25 (1.02, 1.54)*	6.90 (2.31, 20.66)*	3.10 (2.56, 3.76)**
Asian/Pacific Islander	1.93 (1.41, 2.64)**	17.35 (5.05, 59.66)**	3.00 (2.07, 4.33)**
Other	1.68 (1.19, 2.36)*	NA	3.00 (2.16, 4.18)**

* P<0.05, **P<0.0001

aRR adjusted for age and obstetric comorbidity index, excluding hypertension. Hemorrhagic stroke included intracerebral hemorrhage and/or subarachnoid hemorrhage. Pregnancy-induced hypertension included gestational hypertension, preeclampsia, and eclampsia. NA indicates the number in the cell is less than 10 and is suppressed according to the National Inpatient Sample data use agreement.

Table S6. Adjusted risk of ischemic stroke during delivery admissions in US women, National Inpatient Sample 1998-2014, stratified by hypertensive disorders, excluding patients with superimposed preeclampsia (preeclampsia with a history of cHTN, or 642.7x).

Race	No hypertension aRR (95%CI)	Chronic hypertension aRR (95%CI)	Pregnancy-induced hypertension aRR (95%CI)
White	Referent	Referent	Referent
Black	1.41 (1.07, 1.85)*	1.59 (0.77, 3.29)	2.28 (1.83, 2.83)**
Hispanic	0.99 (0.76, 1.28)	NA	1.85 (1.48, 2.30)**
Asian/Pacific Islander	0.46 (0.24, 0.88)*	NA	1.85 (1.16, 2.96)*
Other	0.55 (0.29, 1.04)	4.99 (1.81, 13.73)*	1.48 (0.95, 2.32)

* P<0.05, **P<0.0001

aRR (adjusted risk ratio) adjusted for age and obstetric comorbidity index, excluding hypertension. Ischemic stroke excluded transient ischemic attack and cerebral venous sinus thrombosis. Pregnancy-induced hypertension included gestational hypertension, preeclampsia, and eclampsia. NA indicates the number in the cell is less than 10 and is suppressed according to the National Inpatient Sample data use agreement.

Table S7. Adjusted risk of intracerebral hemorrhage during delivery admissions in US women, National Inpatient Sample 1998-2014, stratified by hypertensive disorders.

Race	No hypertension aRR (95%CI)[#]	Chronic hypertension aRR (95%CI)	Pregnancy-induced hypertension aRR (95%CI)
White	Referent	NA	Referent
Black	2.58 (1.86, 3.58)**	NA	2.19 (1.73, 2.78)**
Hispanic	1.86 (1.37, 2.52)**	NA	3.32 (2.69, 4.09)**
Asian/Pacific Islander	2.82 (1.82, 4.38)**	NA	3.15 (2.11, 4.68)**
Other	1.50 (1.10, 2.06)*	NA	1.05 (0.80, 1.38)

* P<0.05, **P<0.0001

aRR adjusted for age and obstetric comorbidity index, excluding hypertension. Hemorrhagic stroke included intracerebral hemorrhage only (ICD-9 code 431). Pregnancy-induced hypertension included gestational hypertension, preeclampsia, preeclampsia superimposed on chronic hypertension, and eclampsia. NA indicates the number in the cell is less than 10 and is suppressed according to the National Inpatient Sample data use agreement.

Model did not converge.