Recent Racial/Ethnic Disparities in Stroke Hospitalizations and Outcomes for Young Adults in Florida, 2001–2006

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Key Words

Stroke • Hospitalizations • Young adults • Blacks • Hispanics • Mortality • Case fatality

Abstract

Background: Black-white disparities in stroke mortality are well documented, but few recent studies have examined racial/ethnic disparities in stroke hospitalizations among young adults. We analyzed recent (2001–2006) trends in stroke hospitalizations and hospital case-fatality for black, Hispanic, and white adults aged 25–49 years in Florida.

Methods: Hospitalization rates were calculated using population estimates from the census, and hospital discharges with a primary diagnosis of stroke (ICD-9-CM 430, 431, 434, 436) ($n = 16,317$). Multivariate logistic regression modeling was used to examine racial/ethnic disparities in stroke mortality prior to discharge, after adjustment for patient sociodemographics, stroke subtype, risk factors, and comorbidities. **Results:** Age-adjusted stroke hospitalization rates for blacks were over 3 times higher than rates for whites, while rates for Hispanics were slightly higher than rates for whites. Hemorrhagic strokes were proportionally greater among Hispanics compared with blacks and whites ($p < 0.0001$). Blacks were most likely to have diagnosed hypertension (62.3%), morbid obesity (10.9%) or drug abuse (13.6%).

Whites were most likely to have diagnosed hyperlipidemia (21.0%), alcohol abuse (9.5%), and to be smokers (30.6%). The in-hospital fatality rate for all strokes was highest among blacks (10.0%) compared with whites (9.0%) and Hispanics (8.2%). After adjustment for age, gender, insurance status, and all diagnosed risk factors and comorbidities, the black excess was no longer observed [odds ratio (OR) 1.01, 95% confidence interval (CI) 0.88–1.15, $p = 0.93$]. However, the Hispanic advantage in case-fatality was strengthened (OR 0.66, 95% CI 0.55–0.79, $p < 0.0001$). Separate case-fatality analyses for ischemic versus hemorrhagic strokes yielded similar results. **Conclusions:** Our study found a strong and persistent black-white disparity in stroke hospitalization rates for young adults. In contrast, rates were similar for Hispanics and whites. Multivariate adjustment explained the 15% excess case-fatality for blacks; the short-term mortality advantage among Hispanics was strengthened after adjustment.

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Background

Racial/ethnic disparities in stroke incidence and outcomes have been well described [1–4]. Black-white disparities in stroke mortality for young adults have been shown to be very high, with RRs of 3.5 for ischemic stroke,

2.2 for subarchnoid hemorrhage, and 5.2 for intracerebral hemorrhage [5]. Black-white disparities in hospitalization rates have been observed for the Medicare population [6], but have rarely been reported for young adults. In a study of stroke incidence during 1993–1996 in New York City, a 2.4-fold excess for blacks versus whites was observed [7].

Hispanic-white disparities in stroke are less pronounced. In a community-based study in Texas, Mexican Americans aged 45 years and older were found to have higher stroke incidence rates than whites for all stroke subtypes [8]. Stroke mortality rates for Hispanic young adults were similar to rates for whites for ischemic stroke and subarachnoid hemorrhage during 1995–1998, but 50% higher for intracerebral hemorrhage [5]. However, the Northern Manhattan Stroke Study (NOMASS) found a greater Hispanic excess in stroke incidence for subarachnoid hemorrhage compared with ischemic stroke [7].

In this study, we analyzed recent (2001–2006) trends in stroke hospitalization rates for black, Hispanic, and white adults aged 25–49 years old in Florida. In addition, we used multivariate modeling to determine if racial/ethnic disparities in case-fatality could be explained by stroke subtype and the prevalence of stroke risk factors and comorbidities.

Methods

The study population consisted of adults aged 25–49 years who resided in the state of Florida during 2001–2006. Florida is the fourth largest state in the USA and is diverse both ethnically and geographically, with significant rural and urban populations. We included blacks, Hispanics, and whites in our study; there were very small numbers of stroke patients in other racial/ethnic groups.

We analyzed hospital discharge data to obtain discharge records for all patients with a primary discharge diagnosis of either hemorrhagic stroke (ICD-9-CM 430 or 431) or ischemic stroke (ICD-9-CM 434 or 436). In medical record validation studies, these 4 codes have shown the highest positive predictive values for a true current stroke [9–11]. Patients with a primary discharge diagnosis of postsurgical stroke (ICD-9-CM 997.02) or pregnancy-related stroke (ICD-9-CM 674) were excluded. Our study population is not based on a sample, but rather includes all hospitalized strokes from all Florida hospitals (except Veterans Administration hospitals), which are required by law to report hospital discharge data to the state. This dataset included detailed information on patient demographics, payer, diagnoses, procedures, and discharge status.

In addition to the principal diagnosis of stroke, patients could have up to 9 secondary diagnoses recorded. We examined these fields to obtain stroke risk factors and significant comorbidities.

We also used detailed payer (insurance) information to create a proxy variable for low income, defined as payer = Medicaid, charity, self-pay, or underinsured. Charity, self-pay, and underinsured are payer categories reported directly by each hospital.

Stroke hospitalization rates were calculated using annual population denominators for the state of Florida that were specific for age, gender, and race/ethnicity derived from the US Census of Population and Housing. First, age-specific rates were calculated for strata defined by gender and race/ethnicity. The numerator for each rate was the annual count of hospitalized strokes for that age-gender-racial/ethnic group (e.g. black females aged 25–29 years). The age-specific rates were then used to calculate age-adjusted rates for the age range 25–49 years, using the direct method of age standardization with the US 2000 population as the standard. Rate ratios (RRs) were calculated by dividing the rate for each racial/ethnic/gender group by the rate for white women (referent). 95% confidence intervals (CIs) were calculated using the standard formula.

Multivariate logistic regression modeling was used to examine the probability of death prior to discharge for race/ethnicity, before and after adjustment. The first model, the ‘simple’ model, adjusted only for stroke subtype (a known determinant of poststroke fatality) and year of admission (to control for secular trends in treatment and survival). The second model, the ‘demographics’ model, added patient age, gender and low-income status as predictors. The third model, the ‘risk factors’ model, added 7 well-established stroke risk factors (atrial fibrillation, hypertension, cigarette smoking, hyperlipidemia, morbid obesity, alcohol abuse, and drug abuse) as predictors. The final model, the ‘comorbidities’ model, added 7 serious comorbidities with potential to impact survival of stroke patients (diabetes, coronary disease, heart failure, left valvular heart disease, end-stage renal disease, sickle cell anemia, and congenital heart defect). All of the models converged, and all produced global likelihood ratios with $p < 0.0001$. We ran models for all strokes combined, hemorrhagic strokes only (ICD-9-CM 430, 431), and ischemic strokes only (ICD-9-CM 434, 436).

Results

Stroke Hospitalization Rates

There were 16,317 hospitalized strokes among young adults in Florida from 2001 to 2006. Blacks comprised 34% of all hospitalized stroke cases, while Hispanics accounted for 13% of cases. Age-adjusted stroke hospitalization rates did not decline among young adults in Florida from 2001 to 2006 (fig. 1). White women had the lowest rates at the beginning and end of the study period although rates for Hispanic women were somewhat lower in 2002–2005. Gender disparities within racial/ethnic groups were not universal: rates for black women and men were similar in all years; while among whites and Hispanics, rates for men always exceeded the rates for women. Figures 2 and 3 show that the black excess in stroke hospitalizations appeared by age 30 and the gap widened (in absolute terms) with increasing age. Overall, these graphs show little improve-

Fig. 1. Age-adjusted stroke hospitalization rates for young adults aged 25–49, Florida 2001–2006.

Fig. 2. Racial/ethnic disparities in stroke hospitalization rates by age, men 25–49 years old, Florida 2001–2006.

Fig. 3. Racial/ethnic disparities in stroke hospitalization rates by age, women 25–49 years old, Florida 2001–2006.

Table 1. Age-adjusted stroke hospitalization rates (per 100,000 population) and RRs by race/ethnicity for young adults (25–49 years old) in Florida, 2006

	White stroke cases	White rate	White RR	Black stroke cases	Black rate	Black RR	Hispanic stroke cases	Hispanic rate	Hispanic RR
All strokes									
Men	800	18.7	1.51 (1.50–1.51)	411	45.1	3.64 (3.62–3.66)	211	13.7	1.10 (1.09–1.12)
Women	643	12.4	1.00 (referent)	521	46.8	3.77 (3.76–3.79)	184	13.3	1.07 (1.06–1.08)
Hemorrhagic strokes									
Men	220	4.7	1.24 (1.22–1.25)	115	10.5	2.76 (2.71–2.82)	86	3.6	0.95 (0.92–0.97)
Women	203	3.8	1.00 (referent)	144	12.4	3.26 (3.21–3.32)	83	6.0	1.58 (1.54–1.62)
Ischemic strokes									
Men	580	14.0	1.63 (1.62–1.64)	296	34.5	4.01 (3.98–4.04)	125	10.0	1.16 (1.14–1.18)
Women	440	8.6	1.00 (referent)	377	34.4	4.00 (3.97–4.03)	101	7.3	0.85 (0.83–0.87)

White women were the referent group for all RRs. 95% CI are shown in parentheses.

ment between 2001 and 2006 in age-specific stroke hospitalization rates, with the exception of black women aged 30–34 years, and Hispanic men aged 45–49 years.

Black women and men experienced age-adjusted rates of stroke hospitalization that were over 3.5 times higher than the rates for white women in 2006 (table 1). Compared with white women, rates for Hispanic women were 7% higher, for Hispanic men 10% higher, and for white men 50% higher. Stratification by stroke type revealed a greater black-white disparity for ischemic stroke (RRs 4.00 and 4.01) than for hemorrhagic stroke (RRs 2.76 and 3.26). Among Hispanics, there was effect modification by gender: compared with white women, Hispanic men had a higher risk of ischemic stroke (RR 1.16) and a lower risk of hemorrhagic stroke (RR 0.95), while the opposite was true for Hispanic women, who had a higher risk of hemorrhagic stroke (RR 1.58) and a lower risk of ischemic stroke (RR 0.85).

Stroke Patient Characteristics

Stroke subtype varied significantly ($p < 0.0001$) among blacks, Hispanics, and whites (table 2). Hispanics were most likely to experience a hemorrhagic stroke (42.4%) compared with blacks (30.8%) and whites (33.3%). Cerebral occlusion was the most common subtype of stroke, with an overall prevalence of 56.1% but a lower prevalence among Hispanics (49.0%). Subarachnoid hemorrhages were most common among Hispanics (22.5%), followed by whites (18.1%) and blacks (11.6%). The frequency of intracerebral hemorrhages was similar for Hispanics and blacks (19.9 vs. 19.2%) but lower among whites

(15.2%). Blacks had the highest prevalence of ill-defined strokes (11.1%).

There were significant differences by race/ethnicity on almost all patient demographic, risk factor and comorbidity characteristics (table 2). Hispanic stroke patients were somewhat younger than either black or white patients. The majority of black patients were female (55.7%), while the majority of both Hispanic and white patients were male. Hispanic patients were most likely to be low income (52.4%), followed by blacks (50.3%) and whites (36.6%). Lengths of hospital stay also varied by race/ethnicity, with whites having the shortest stays and Hispanics having the longest stays.

The only risk factor which did not show significant variation by race/ethnicity was atrial fibrillation, with an overall prevalence of only 2.6%. Blacks were most likely to have diagnosed hypertension (62.3%), morbid obesity (10.9%) or drug abuse (13.6%). Whites were most likely to have diagnosed hyperlipidemia (21.0%), alcohol abuse (9.5%), and to be smokers (30.6%).

We examined the prevalence of 7 serious comorbidities which could impact stroke case-fatality rates (table 2). The most common diagnosed comorbidity was diabetes, with the highest prevalence among blacks (25.2%), compared with Hispanics (21.2%) and whites (17.4%). Despite the relatively young age of the study population, the presence of coronary heart disease, heart failure, and end-stage renal disease (ESRD) was observed in 8.8, 4.7, and 4.1% of patients, respectively. Whites were more likely to have coronary heart disease (9.6%), valvular heart disease (0.7%), or a congenital heart defect (2.5%); while blacks

Table 2. Description of young adults hospitalized with a primary diagnosis of stroke, Florida 2001–2006

	Total, % (n = 16,317)	Blacks, % (n = 5,493)	Hispanics, % (n = 2,154)	Whites, % (n = 8,670)	p value for difference
Age					<0.0001
25–29 years	3.9 (628)	3.2 (177)	5.7 (122)	3.8 (329)	
30–34 years	6.9 (1,122)	6.8 (373)	7.7 (165)	6.7 (584)	
35–39 years	14.3 (2,327)	14.5 (796)	15.6 (335)	13.8 (1,196)	
40–44 years	28.7 (4,684)	28.9 (1,589)	27.7 (597)	28.8 (2,498)	
45–49 years	46.3 (7,556)	46.6 (2,558)	43.4 (935)	46.9 (4,063)	
Gender					<0.0001
Women	48.9 (7,981)	55.7 (3,057)	43.1 (929)	46.1 (3,995)	
Men	51.1 (8,336)	44.4 (2,436)	56.9 (1,225)	53.9 (4,675)	
Low-income proxy					<0.0001
Medicaid/underinsured/charity	43.3 (7,059)	50.3 (2,760)	52.4 (1,129)	36.6 (3,170)	
All other	56.7 (9,258)	49.8 (2,733)	47.6 (1,025)	63.4 (5,500)	
Patient risk factors					
Hypertension	51.2 (8,352)	62.3 (3,424)	48.1 (1,037)	44.9 (3,891)	<0.0001
Cigarette smoking	24.6 (4,015)	18.5 (1,014)	16.3 (350)	30.6 (2,652)	<0.0001
Atrial fibrillation	2.6 (422)	2.5 (138)	3.0 (65)	2.5 (219)	0.34
Morbid obesity	9.6 (1,559)	10.9 (599)	7.8 (167)	9.2 (793)	<0.0001
Hyperlipidemia	19.2 (3,126)	17.0 (935)	17.1 (368)	21.0 (1,823)	<0.0001
Alcohol abuse	8.1 (1,313)	6.6 (364)	5.8 (124)	9.5 (825)	<0.0001
Drug abuse	9.5 (1,549)	13.6 (746)	6.1 (131)	7.7 (672)	<0.0001
Patient comorbidities					
Diabetes	20.5 (3,346)	25.2 (1,382)	21.2 (457)	17.4 (1,507)	<0.0001
Coronary heart disease	8.8 (1,437)	7.8 (430)	8.2 (176)	9.6 (831)	0.0008
Heart failure	4.7 (758)	6.9 (378)	3.9 (83)	3.4 (297)	<0.0001
Left valvular heart disease	0.6 (98)	0.5 (28)	0.6 (12)	0.7 (58)	0.47
End-stage renal disease	4.1 (670)	6.4 (351)	4.1 (88)	2.7 (231)	<0.0001
Sickle cell disease	0.3 (48)	0.8 (46)	0.1 (1)	0.1 (1)	<0.0001
Congenital heart defects	1.9 (314)	1.0 (53)	1.9 (41)	2.5 (220)	<0.0001
Stroke subtype					<0.0001
Cerebral artery occlusion	56.1 (9,156)	58.1 (3,189)	49.0 (1,055)	56.7 (4,912)	
Subarachnoid hemorrhage	16.5 (2,691)	11.6 (639)	22.5 (485)	18.1 (1,567)	
Intracerebral hemorrhage	17.2 (2,802)	19.2 (1,055)	19.9 (428)	15.2 (1,319)	
Ill-defined stroke	10.2 (1,668)	11.1 (610)	8.6 (186)	10.1 (872)	
Length of hospital stay, days					<0.0001
1–2	23.8 (3,883)	20.4 (1,119)	21.4 (460)	26.6 (2,304)	
3–6	41.2 (6,729)	41.8 (2,297)	38.5 (830)	41.6 (3,602)	
7+	35.0 (5,705)	37.8 (2,077)	40.1 (864)	31.9 (2,764)	

Numbers are shown in parentheses.

were more likely to have heart failure (6.9%) or ESRD (6.4%). There were 48 cases of diagnosed sickle cell disease; 46 of these patients were black, for a prevalence of 0.8% among black stroke patients.

In-Hospital Stroke Mortality

The overall rate of in-hospital mortality for stroke patients was 9.3% (total deaths = 1,512) in our study population, with a decline from 11.3% in 2001 to 6.0% in 2006.

Over the entire study period, the case-fatality rate was highest among blacks (10.0%, n = 551) compared with whites (9.0%, n = 784) and Hispanics (8.2%, n = 177). We used logistic regression modeling to investigate whether these racial/ethnic disparities in case-fatality persisted after multivariate adjustment (table 3). In the simple model, after control for stroke sub-type and year of admission, we found that the fatality rate for all strokes was significantly higher for blacks (OR 1.15, 95% CI 1.02–1.30,

Table 3. Results of multivariate modeling of race/ethnicity and in-hospital stroke mortality among adults 25–49 years old

	Simple model ¹ OR	Patient demographics model ² , OR	Patient risk factors model ³ , OR	Patient comorbidities model ⁴ , OR
All strokes				
White	1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)
Black	1.15 (1.02–1.30)*	1.10 (0.97–1.25)	1.07 (0.94–1.22)	1.01 (0.88–1.15)
Hispanic	0.73 (0.61–0.88)**	0.70 (0.58–0.84)***	0.67 (0.56–0.81)***	0.66 (0.55–0.79)***
Hemorrhagic strokes only				
White	1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)
Black	1.15 (0.99–1.33)	1.09 (0.94–1.26)	1.07 (0.92–1.25)	0.99 (0.85–1.16)
Hispanic	0.75 (0.61–0.91)**	0.71 (0.58–0.87)**	0.70 (0.57–0.85)	0.68 (0.55–0.83)**
Ischemic strokes only				
White	1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)
Black	1.15 (0.92–1.45)	1.15 (0.91–1.46)	1.07 (0.84–1.36)	0.99 (0.78–1.27)
Hispanic	0.69 (0.45–1.04)	0.66 (0.43–1.00)*	0.61 (0.40–0.93)*	0.61 (0.40–0.93)*

95% CIs are shown in parentheses. * $p < 0.05$, ** $p < 0.001$, *** $p < 0.0001$.

¹ Model adjusted for year of admission and stroke subtype.

² Model adjusted for model 1 covariates + age, gender, and insurance status.

³ Model adjusted for model 2 covariates + hypertension, cigarette smoking, atrial fibrillation, morbid obesity, hyperlipidemia, alcohol abuse, and drug abuse.

⁴ Model adjusted for model 3 covariates + diabetes, coronary heart disease, heart failure, left valvular heart disease, end-stage renal disease, sickle cell anemia, and congenital heart defects.

$p < 0.05$) and lower for Hispanics (OR 0.73, 95% CI 0.61–0.88) compared with whites. Additional adjustment for age, gender, and low-income status reduced the Black excess in case-fatality from 15 to 10%, with the disparity being no longer statistically significant (OR 1.10, 95% CI 0.97–1.25, $p = 0.13$). After further adjustment for all diagnosed risk factors and comorbidities, the black excess was no longer observed (OR 1.01, 95% CI 0.88–1.15, $p = 0.93$). However, the fully adjusted model revealed that the Hispanic advantage in case-fatality compared with whites was slightly strengthened (OR 0.66, 95% CI 0.55–0.79, $p < 0.0001$). We repeated these analyses separately for hemorrhagic strokes and ischemic strokes, with little appreciable difference in the results (table 3).

The full model results with predictors of in-hospital mortality for hemorrhagic strokes and ischemic strokes are shown in table 4. For ischemic strokes (66.1% of all strokes), significant predictors of higher case-fatality were: atrial fibrillation (OR 1.64, 95% CI 1.04–2.59, $p < 0.05$), drug abuse (OR 1.73, 95% CI 1.24–2.42, $p < 0.001$), and heart failure (OR 2.18, 95% CI 1.52–3.12, $p < 0.0001$). Significant predictors of lower ischemic stroke case-fatality included: Hispanic ethnicity (OR 0.61, 95% CI 0.40–0.93, $p < 0.05$), female gender (OR 0.75, 95% CI 0.60–0.95, $p < 0.05$), ill-defined stroke vs. cerebral artery occlusion

(OR 0.29, 95% CI 0.18–0.47, $p < 0.0001$), hypertension (OR 0.57, 95% CI 0.45–0.74, $p < 0.0001$), cigarette smoking (OR 0.41, 95% CI 0.30–0.57, $p < 0.0001$), hyperlipidemia (OR 0.28, 95% CI 0.19–0.43, $p < 0.0001$), and congenital heart defects (OR 0.18, 95% CI 0.04–0.71, $p < 0.05$).

For hemorrhagic strokes (33.9% of all strokes), significant predictors of higher case-fatality were: low income status (OR 1.32, 95% CI 1.15–1.52, $p < 0.0001$), intracerebral hemorrhage vs. subarachnoid hemorrhage (OR 1.50, 95% CI 1.30–1.73, $p < 0.0001$), drug abuse (OR 1.52, 95% CI 1.24–1.86, $p < 0.0001$), coronary heart disease (OR 1.67, 95% CI 1.24–2.25, $p < 0.001$), and ESRD (OR 2.23, 95% CI 1.64–3.05, $p < 0.0001$). Significant predictors of lower hemorrhagic stroke case-fatality included: Hispanic ethnicity (OR 0.68, 95% CI 0.55–0.83, $p < 0.001$), admission in 2006 vs. 2001 (OR 0.68, 95% CI 0.53–0.87, $p < 0.001$), age 25–29 vs. 45–49 (OR 0.68, 95% CI 0.49–0.95, $p < 0.05$), age 30–34 vs. 45–49 (OR 0.73, 95% CI 0.56–0.96, $p < 0.05$), cigarette smoking (OR 0.65, 95% CI 0.54–0.78, $p < 0.0001$), morbid obesity (OR 0.69, 95% CI 0.49–0.96, $p < 0.05$), and hyperlipidemia (OR 0.44, 95% CI 0.30–0.64, $p < 0.0001$).

Discussion

For blacks, we found a highly significant, greater than 3-fold excess in stroke hospitalization rates and a 15% excess in immediate case-fatality compared with whites; however, the disparity in case-fatality was explained by patient demographics, risk factors, and comorbidities. A similar study of black-white disparities in in-hospital mortality among stroke patients in Maryland for 2000–2003 found a 14% excess risk for males and no excess for females after adjustment. However, this study adjusted for fewer covariates than our study, and also used a broader definition of stroke that included ICD-9-CM codes with low positive predictive values for current strokes (i.e. 433, 437 and 438) [12].

In contrast to our findings for blacks, stroke hospitalization rates for Hispanics were similar to rates for whites, and the risk of in-hospital mortality following a stroke was 27% lower in Hispanics. Multivariate adjustment slightly strengthened the apparent Hispanic advantage in case-fatality for all strokes, hemorrhagic strokes, or ischemic strokes, suggesting that differences in stroke severity and unmeasured comorbidities should be investigated further in this population. While most previous stroke studies of Hispanics have focused on Mexican Americans only, our study population included significant numbers of Cuban Americans, Puerto Ricans, Mexican Americans and Hispanics of South American origin. It is possible that stroke incidence and outcomes may vary among Hispanic subgroups (e.g. Cubans, Puerto Ricans), as has been shown for cancer [13].

The NOMASS reported stroke incidence rates for young adults by race/ethnicity for the period 1993–1996 [7, 14]. Hospitalized cases comprised 95% of all incident strokes detected in NOMASS [14], so some comparison is possible. Although our study period was 10 years later (during a period of national decline in stroke mortality), we observed much higher hospitalization rates for both blacks and whites than was found in NOMASS, but much lower rates for Hispanics [14]. In NOMASS there was a 2.5-fold excess in stroke incidence for Hispanic young adults (mainly Caribbean Hispanics) compared with whites [7]; in contrast, we found only a slight excess (7–10%) for Hispanics. Again, the heterogeneity in risk for disease under the ‘Hispanic umbrella’ [13] warrants more detailed studies of specific Hispanic subgroups.

We observed no decline in stroke hospitalization rates for adults aged 25–49 years in Florida from 2001 to 2006. This finding is consistent with a national study of trends

Table 4. Full-model results for in-hospital mortality of hemorrhagic versus ischemic strokes among adults 25–49 years old

	Hemorrhagic strokes only, OR	Ischemic strokes only, OR (95% CI)
<i>Patient race/ethnicity</i>		
White	1.00 (referent)	1.00 (referent)
Black	0.99 (0.85–1.16)	0.99 (0.78–1.27)
Hispanic	0.68 (0.55–0.83)**	0.61 (0.40–0.93)*
<i>Year of admission</i>		
2001	1.00 (referent)	1.00 (referent)
2002	1.04 (0.83–1.30)	0.80 (0.53–1.20)
2003	0.89 (0.71–1.12)	1.02 (0.70–1.48)
2004	0.89 (0.71–1.11)	0.76 (0.51–1.12)
2005	0.82 (0.65–1.03)	0.88 (0.60–1.29)
2006	0.68 (0.53–0.87)**	0.73 (0.49–1.09)
<i>Stroke subtype</i>		
Subarachnoid hemorrhage	1.00 referent)	–
Intracerebral hemorrhage	1.50 (1.30–1.73)***	–
Cerebral artery occlusion	–	1.00 referent)
Ill-defined stroke	–	0.29 (0.18–0.47)***
<i>Patient demographics</i>		
Age 25–29 years	0.68 (0.49–0.95)*	0.73 (0.37–1.42)
Age 30–34 years	0.73 (0.56–0.96)*	1.16 (0.75–1.79)
Age 35–39 years	0.86 (0.71–1.05)	0.97 (0.69–1.37)
Age 40–44 years	0.87 (0.74–1.02)	0.96 (0.74–1.26)
Age 45–49 years	1.00 (referent)	1.00 (referent)
Female gender	1.05 (0.91–1.20)	0.75 (0.60–0.95)*
Low income (insurance proxy)	1.32 (1.15–1.52)***	1.26 (0.99–1.59)
<i>Patient risk factors</i>		
Hypertension	0.94 (0.81–1.08)	0.57 (0.45–0.74)*
Cigarette smoking	0.65 (0.54–0.78)*	0.41 (0.30–0.57)*
Atrial fibrillation	1.19 (0.72–1.96)	1.64 (1.04–2.59)*
Morbid obesity	0.69 (0.49–0.96)*	1.02 (0.69–1.51)
Hyperlipidemia	0.44 (0.30–0.64)***	0.28 (0.19–0.43)***
Alcohol abuse	1.11 (0.89–1.39)	1.39 (0.95–2.04)
Drug abuse	1.52 (1.24–1.86)***	1.73 (1.24–2.42)**
<i>Patient comorbidities</i>		
Diabetes	1.00 (0.79–1.25)	1.02 (0.77–1.36)
Coronary heart disease	1.67 (1.24–2.25)**	1.27 (0.89–1.79)
Heart failure	0.92 (0.65–1.30)	2.18 (1.52–3.12)***
Left valvular heart disease	2.01 (0.32–12.70)	0.87 (0.31–2.47)
End-stage renal disease	2.23 (1.64–3.05)***	1.19 (0.75–1.88)
Sickle cell anemia	1.90 (0.69–5.21)	–
Congenital heart defects	–	0.18 (0.04–0.71)*

95% CIs are shown in parentheses.

* $p < 0.05$, ** $p < 0.001$, *** $p < 0.0001$.

in stroke hospitalization rates, using the National Inpatient Discharge Sample, which found no decline among adults <65 years old from 1997 to 2004 [15]. Similar data from Minneapolis-St. Paul showed no decline in age-adjusted stroke hospitalization rates from 1980 to 2002 [16]. The overall case-fatality rate observed for stroke patients aged 30–49 years in Minneapolis during 2000–2002 (5.6% for men and 5.9% for women) [16] was lower than that observed in our study (9%).

Low-income status, as measured by insurance proxy, conveyed a 30% excess risk in case-fatality for all strokes (from the full model in table 3), independent of subtype, race/ethnicity, age, sex, risk factors and comorbidities. A recent study of in-hospital mortality among stroke inpatients using data from the 2002 National Inpatient Sample found an adjusted RR of mortality of 1.24 for Medicaid patients and 1.56 for uninsured patients [17]. Our findings for low income and higher stroke fatality are consistent with other recent studies from China [18], Austria [19] and Canada [20]. Future research, using data sources with more detailed information on clinical diagnoses and treatments should be conducted to determine if there are systematic disparities in stroke treatment by insurance type and socioeconomic status.

Drug abuse is an important preventable cause of stroke incidence and mortality among young adults [21–23]. Cocaine is a central nervous system (CNS) stimulant which can cause cardiac arrhythmias, hypertension, and thrombosis [21]. In the Baltimore-Washington Young Stroke Study, the frequency of drug-associated strokes during 1988–1991 was 12.1% for ischemic strokes [24] and 13.3% for intracerebral hemorrhages [25]. Although black stroke patients had a higher prevalence of drug abuse than white or Hispanic patients in our study, these are numerator data only and do not reveal the relative prevalence of drug abuse in the population at-risk. The recent increase in the illegal use of another CNS stimulant, methamphetamine ('ice' 'amp' 'crank' 'crystal' 'glass') [26] may be changing the profile of drug-abuse-associated strokes. A large national study of young adults found a higher frequency of crystal methamphetamine use among whites compared with blacks and Hispanics, and among males compared with females [27].

The protective effect on stroke case-fatality of diagnosed hypertension and hyperlipidemia observed in our study appears counter-intuitive. However, these results may in fact reflect a greater likelihood of diagnosis in the presence of treatment (i.e. current use of antihypertensive medications and statins). Therefore, it may be that the treatment, not the underlying condition, is conveying the

protective effect. Similarly, patients with known stroke risk factors may have their stroke more quickly and accurately diagnosed in the hospital emergency room, leading to more efficacious medical care compared with stroke patients with an atypical case presentation.

The 'smoker's paradox' observed in our study has been well described in previous studies of myocardial infarctions [28–30], but was not observed in a large stroke outcome study conducted in China [31]. The Copenhagen Stroke Study found a protective effect of smoking on 5-year survival in univariate analyses, but a 20% excess after multivariate adjustment [32]. In a small follow-up study of 497 hemorrhagic stroke patients, smoking increased the risk of long-term mortality by 22% [33].

A strength of our study was the use of only those ICD-9-CM codes for stroke (430, 431, 434, 436) which have been shown in medical record validation studies to have the highest positive predictive values for a true current stroke [9–11]. Furthermore, compared with a recent report of a high proportion of 'ill-defined' strokes among elderly patients [34], only 10.2% of young adult stroke patients in Florida received a principal diagnosis of 'ill-defined' stroke. Many previous studies have grouped ill-defined strokes with ischemic strokes for analysis, as we did in this study. However, we found a significantly reduced risk of in-hospital mortality for patients with a diagnosis of ill-defined stroke compared with a diagnosis of cerebral artery occlusion. A much lower case-fatality for ill-defined strokes suggests that some of these cases may not have been true strokes.

The use of hospital discharge data in our study introduces some limitations. First, these data do not permit identification of multiple stroke events for the same individual over the study period. Despite the young age of our study population, a small number of recurrent strokes undoubtedly occurred. Therefore, the stroke hospitalization rates presented here do not represent true incidence rates, but can be thought of as 'attack rates'. Second, we do not know specifically how patient race/ethnicity was ascertained in each hospital. It is likely that this variable (coded to standard categories) was obtained through a combination of patient self-report (during emergency department triage) and health care professional observation. Third, our case-fatality analyses may be limited by misdiagnosis (misclassification) of patient characteristics, risk factors, and comorbidities. However, to the degree that any misclassification was nondifferential with respect to race/ethnicity, it would have the effect of biasing the observed ORs towards the null. Lastly, our focus on stroke hospitaliza-

tions did not allow us to examine racial/ethnic disparities in nonhospitalized strokes.

Florida is the fourth largest state in the nation and encompasses both wide geographic diversity (large metropolitan areas along with vast rural farmlands) and wide population diversity in terms of both socioeconomic resources and race/ethnicity; therefore, our results should be widely generalizable.

In conclusion, our study of over 16,000 recent stroke cases found a strong and persistent black-white disparity in stroke hospitalization rates among young adults, pointing toward the urgent need for primary prevention of

stroke risk factors among African Americans to eliminate disparities in stroke incidence. Multivariate adjustment for risk factors and comorbidities explained the excess case-fatality for blacks. In contrast, stroke hospitalization rates were similar for Hispanics and whites, and there was a significant protective effect on in-hospital mortality for Hispanics compared with whites. Future research should examine racial/ethnic variations in stroke diagnosis, case severity, and treatment for young adults, and should also look under the 'Hispanic umbrella' [13] to examine stroke trends and outcomes for diverse Hispanic subgroups.

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