Racial Disparities in Severity of Cerebrovascular Events

K.V. Kuhlemeier, PhD, MPH; S.A. Stiens, MD

Background and Purpose This study was conducted to determine if blacks hospitalized for cerebrovascular events had more severe cerebrovascular events than whites similarly hospitalized.

Methods Data from the Maryland Health Services Cost Review Commission were used to determine incidence of death, death rates, age at death of those who died, and length of stay for acute hemorrhagic and occlusive stroke in hospitalized blacks and whites after adjusting for sex and, if appropriate, age.

Results With a single exception (number of patients with hemorrhagic stroke who died during short-term hospitalization), all indices indicated that blacks incurred more severe cerebrovascular events than whites ($P<.05$ or less).

Conclusions Maryland state data from hospitalized patients indicate that blacks had more severe strokes than whites. (Stroke. 1994;25:2126-2131.)

Key Words • cerebral hemorrhage • racial differences • stroke assessment • blacks

Stroke remains the third leading cause of death for blacks in the United States. Hypertension has been identified as the predominant risk factor for strokes in all varieties. It has long been recognized that blacks in the United States suffer from a higher prevalence of hypertension and cerebrovascular disease than whites and that the death rate from stroke is higher in blacks than in whites. The first National Health and Nutrition Examination Survey showed that stroke is the single largest contributor to the black-white mortality difference. Stroke mortality is highest in the southeastern central United States (118.6 age-adjusted deaths per 100,000 population) and lowest (81.2 age-adjusted deaths per 100,000 population) in the mountain states. With the exception of a few years after World War II, death rates from stroke have been declining in white men, white women, and black women since the 1920s. The death rates in black men finally started decreasing in the mid 1960s. There is recent evidence to suggest that this rate of decline may be abating.

Although it seems clear that blacks experience a higher incidence of lethal strokes than whites, the specifics of differences in stroke severity between the races have not been reported in large studies. A survey of stroke symptoms in Copiah County, Mississippi, demonstrated that the age-adjusted prevalence of stroke is higher in blacks. Physical impairments resulting from ischemic strokes in blacks have been found to be more severe than those in whites, but the rate and extent of functional recovery do not differ significantly between races. Longitudinal study of stroke patients who return home reveals that white patients are more often functionally independent. In a recent study that looked for racial or gender disparities for admission to a formal inpatient rehabilitation program after acute stroke, we found that the fraction of black acute stroke patients admitted to rehabilitation was equal to the fraction of white stroke patients admitted to rehabilitation. However, blacks were significantly more likely than whites to die during hospitalization for acute stroke, and the short-term length of stay (LOS) for blacks was significantly longer than for whites. These findings suggested that blacks had more severe strokes than whites. In the present study, we looked for more detailed evidence for disparities in stroke severity between blacks and whites.

Methods Data for this study were collected by the Maryland Health Services Cost Review Commission for 1990. This commission collects a wide variety of data on every patient in every hospital in the state of Maryland that admits Medicare patients. The data collected included demographics, costs, diagnoses, medical procedures, and LOS. Individual identifiers were not included.

To be included in this study, patients had to identify themselves as either black or white at the time of admission, be at least 46 but less than 106 years of age, and have a primary admission diagnosis of International Classification of Diseases, Revision 9 (ICD-9) codes 430 through 436 for acute cerebrovascular disease. We attempted to embrace all acute cerebrovascular disease by including the following diagnosis codes: 430, subarachnoid hemorrhage; 431, intracerebral hemorrhage; 432, other intracranial nontraumatic hemorrhage; 433, occlusive preocerebral arterial disease; 434, occlusive cerebral arterial disease; 435, transient cerebral ischemic disease; and 436, acute but ill-defined cerebrovascular disease. Angiographic findings were not included in the data set. Ages used were those at the day of admission. Hemorrhagic strokes were those with ICD-9 codes of 430 through 432.9 for their primary diagnosis. Patients with
TABLE 1. Breakdown of Patient Population by Race, Sex, and Type of Cerebrovascular Event

<table>
<thead>
<tr>
<th>Patient Age, y</th>
<th>White</th>
<th>White</th>
<th>Black</th>
<th>Black</th>
</tr>
</thead>
<tbody>
<tr>
<td>n=12,791</td>
<td>Men</td>
<td>Women</td>
<td>Men</td>
<td>Women</td>
</tr>
<tr>
<td>Hemorrhagic stroke</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>46-55</td>
<td>72</td>
<td>30</td>
<td>50</td>
<td>63</td>
</tr>
<tr>
<td>56-65</td>
<td>96</td>
<td>85</td>
<td>31</td>
<td>51</td>
</tr>
<tr>
<td>66-75</td>
<td>142</td>
<td>121</td>
<td>47</td>
<td>46</td>
</tr>
<tr>
<td>76-85</td>
<td>115</td>
<td>170</td>
<td>40</td>
<td>24</td>
</tr>
<tr>
<td>86-95</td>
<td>23</td>
<td>70</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>96-105</td>
<td>0</td>
<td>4</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Occlusive stroke</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n=11,498</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>46-55</td>
<td>348</td>
<td>223</td>
<td>157</td>
<td>174</td>
</tr>
<tr>
<td>56-65</td>
<td>869</td>
<td>649</td>
<td>340</td>
<td>282</td>
</tr>
<tr>
<td>66-75</td>
<td>1564</td>
<td>1513</td>
<td>454</td>
<td>303</td>
</tr>
<tr>
<td>76-85</td>
<td>1089</td>
<td>1797</td>
<td>386</td>
<td>166</td>
</tr>
<tr>
<td>86-95</td>
<td>245</td>
<td>704</td>
<td>128</td>
<td>47</td>
</tr>
<tr>
<td>96-105</td>
<td>5</td>
<td>43</td>
<td>10</td>
<td>2</td>
</tr>
</tbody>
</table>

primary diagnoses of ICD-9 codes of 433 through 436 were categorized as occlusive cerebrovascular events. The resulting reduced data set included 12,791 admissions.

Outcome Measures

The outcome measures used in this study were the fraction of patients in coma during short-term hospitalization, the fraction of patients who died during short-term hospitalization, age at death of patients who died during short-term hospitalization, and short-term hospitalization LOS. The fraction of patients in coma has been reported to be a good predictor of mortality among hospitalized stroke patients.12

Statistical Analyses

Statistical analyses used in this report to determine the effects of race on stroke severity included multiple linear regression for outcomes expressed in continuous variables (age at death, LOS) and logistic regression for outcomes expressed in dichotomous outcomes (living versus dead, coma versus no coma). The initial model tested included all the main effects (age, race, sex) and all their possible interactions. If no interactions were statistically significant (P>.05), a new model was tested with only the main effects. Probability levels reported here are those from the model including all statistically significant (P<.05) interaction terms or, if none of the interactions were significant, from the model including only main effects.

Results

The total number of cerebrovascular events are distributed into age groups and divided by race, sex, and type of stroke in Table 1. Occlusive events outnumbered hemorrhagic events by a ratio of more than 9 to 1. Most patients were in the age range of 66 to 85 years, with very few patients aged 96 years or over at the time of admission.

Of the total ischemic infarctions that were coded as defined vessel occlusions, cerebral occlusions were significantly (P<.001) more frequent in blacks (of all occlusions: black men, 0.91; black women, 0.91; white men, 0.66; and white women, 0.74). Transient ischemic attacks (TIAs) represented a similar proportion of occlusive disease when comparing blacks (0.27) and whites (0.28). The proportion of ischemic events composed of TIA was significantly (P<.001, χ²) higher in women than men (white men, 0.26; white women, 0.31; black men, 0.26; and black women, 0.27).

The fractions of patients with coma are shown in Fig 1 for intracranial hemorrhagic strokes and in Fig 2 for occlusive events. Race was significantly related to the incidence of coma for both hemorrhagic events (P<.003) and occlusive events (P<.042). There was no significant relationship between sex and coma in either hemorrhagic (P>.94) or occlusive (P>.95) events. Age was not significantly related to the fraction of patients with coma for hemorrhagic stroke (P>.26) but was for occlusive stroke (P<.002). In general, coma rates were much higher in cases of hemorrhage than occlusion.

Death rates during short-term hospitalization for hemorrhagic events were generally in the range of 0.2 to 0.4 (Fig 3). Death rate data were unreliable in the 96- to 105-year age group because of the small number of cases. Logistic regression showed that neither age (P>.24), race (P>.98), nor sex (P>.38) were signifi-
Significantly associated with the probability of dying during short-term hospitalization with intracranial hemorrhage. However, for occlusive events (Fig 4), age \((P<.001)\) and race \((P<.003)\) both had significant effects on the likelihood of dying. Sex was not a significant factor in contributing to the probability of dying during hospitalization after acute ischemic events \((P>.22)\).

The mean age at death was somewhat lower for patients with hemorrhage than for patients with occlusion \((P<.001)\). White men who died with intracranial hemorrhage had a mean age at death of 71.79 years \((n=121, \text{SD}=10.29)\), whereas women had a mean age of 72.99 \((n=187, \text{SD}=11.01)\). Black men had a mean age of 63.51 years \((n=52, \text{SD}=9.33)\), and black women had a mean age of 67.58 \((n=57, \text{SD}=13.15)\).

The effect of race on mean age at death for intracranial hemorrhage in this study was highly significant \((P<.0001)\), and the effect of sex was marginally significant \((P=.07)\). The mean ages at admission for patients with hemorrhagic stroke who did not die during the initial hospitalization were as follows: white men, 69.32 years \((n=248, \text{SD}=11.73)\); white women, 73.17 years \((n=331, \text{SD}=12.03)\); black men, 64.51 years \((n=105, \text{SD}=10.79)\); and black women, 65.76 years \((n=110, \text{SD}=12.64)\).

The mean ages at death from occlusive events were as follows: white men, 74.95 years \((n=256, \text{SD}=9.67)\); white women, 79.27 years \((n=362, \text{SD}=9.23)\); black men, 71.35 years \((n=88, \text{SD}=9.71)\); and black women, 74.83 years \((n=101, \text{SD}=11.48)\). The effects of both race and sex on mean age at death from occlusive events were highly significant \((P<.0001)\). The mean ages at admission for those with cerebrovascular events who did not die during short-term hospitalization were as follows: white men, 70.22 years \((n=3718, \text{SD}=10.06)\); white women, 74.26 years \((n=4356, \text{SD}=10.59)\); black men, 67.37 years \((n=910, \text{SD}=10.33)\); and black women, 70.06 years \((n=1304, \text{SD}=11.37)\).

The effects of race, sex, and age on average LOS on hemorrhagic and occlusive cerebrovascular events are given in Table 2. The average overall LOS for the hemorrhagic events was 14.5 days, and for occlusive events it was 9.8 days. Race had a significant relation to LOS both for hemorrhagic \((P=.029)\) and occlusive \((P<.0001)\). After adjusting for sex and age, blacks with hemorrhagic strokes had an LOS 2.6 days longer than whites, and blacks with occlusive events had an LOS 3.3 days longer than whites. Sex was not related to LOS for either hemorrhagic or occlusive \((P=.47)\) for hemorrhagic strokes and \(P=.46\) for occlusive events). Age was not related to LOS for patients with hemorrhagic strokes \((P=.77)\), but it was related to LOS for patients with occlusive cerebrovascular events \((P<.0001)\), increasing 0.11 day per year of age.

**Discussion**

In this study we examined several indices of stroke severity and determined whether these indices differed significantly between blacks and whites after adjusting for age and sex. This was done separately for occlusive and hemorrhagic events. These indices included death rate during hospitalization for acute cerebrovascular events, age at death for those patients who died during their short-term hospitalization, fraction of patients with coma, and mean LOS. With a single exception, all indices indicated that blacks suffered more severe cerebrovascular events than whites. That single exception was that race was not a significant factor in the number of patients with hemorrhage who died during short-term hospitalization for acute cerebrovascular disease.

One explanation of these findings is that blacks do indeed have more severe strokes than whites. This finding can be explained in part by reviewing some of the stroke risk factors as they relate to race. Hyperten-
esion and diabetes contribute significantly to the risk for stroke. In the United States, hypertension and diabetes are more prevalent among blacks than whites. Indeed, the prevalence of hypertension and diabetes may be underestimated in blacks because of less frequent medical evaluation. Hypertension in diabetic patients raises stroke incidence two and one half times over incidence without hypertension, suggesting that these two risk factors act synergistically. These risk factors could lead to earlier development of atherosclerosis and progression to symptomatic disease at a greater rate. With accelerated plaque formation, black patients would be at greater risk for vessel occlusion or embolic phenomena.

Hypertension and diabetes as risk factors for stroke do not in themselves completely explain the increased incidence of stroke in blacks. Kittner et al found a higher incidence of stroke in blacks even after adjusting for the higher prevalence of hypertension and diabetes. Thus, the increased incidence and severity of stroke in blacks cannot be wholly explained by the severity of classic risk factors alone.

One important negative finding of this study was the observation that race was not significantly related \( (P > .98) \) to the risk of dying from an intracerebral hemorrhage after admission to the hospital. The findings suggest an improvement over past observations. In 1966 the vital statistics of the United States indicated an age-adjusted intracerebral-bleed death rate of 97.6 per 100,000 population for blacks and 51.6 per 100,000 for whites. This racial disparity between death rates from hemorrhagic stroke has been attributed to greater prevalence and severity of hypertension in the black population. In recent years screening and treatment has improved blood pressure control, resulting in fewer strokes overall. Improvement in the control of hypertension has produced an almost inverse linear relation with the decreasing incidence of stroke in women, but stroke incidence in men did not decrease until 10 years after improvement in their rate of hypertension began. This may be due to more frequent screening of blood pressure in women. The treatment of hypertension has reduced the frequency and severity of all hemorrhagic strokes and has significantly reduced the risk of death due to hemorrhagic stroke in blacks.

Our observed differences in ischemic stroke severity and distribution in blacks and whites may be partially explained by differences in the distribution of vessel pathology. The distribution of atheromatous involvement of the cerebrovascular tree as demonstrated by cerebral angiography is significantly different in blacks and whites. Black patients have been observed to have more severe occlusive disease of the middle cerebral artery, whereas white patients have more severe disease of internal carotid origin.

In a comparison study of data from angiographic studies of the posterior circulation, whites were found to have more high-grade lesions of the extracranial vertebral arteries, and black patients had more lesions of the intracranial distal basilar artery and branches thereof. We observed a frequency of complete cerebral artery occlusion that was much higher for blacks than whites. These differences in arterial involvement may affect the mechanisms of vessel occlusion. Whereas proximal extracranial plaque formation would be expected to increase risk for embolic phenomena, intracranial vessel narrowing may place black patients at higher risk for thrombotic occlusion.

Secondary stroke prevention often follows a symptomatic warning. TIA often bring patients to treatment for the first time. It has been suggested that TIA can be due to embolic phenomena originating on the surface of proximal arterial plaques, although Coull et al in a study of 429 stroke patients found TIA to be as frequent in patients with embolic infarcts as in those with thrombotic infarction. The specific mechanisms for TIA are as yet incompletely defined. Some investigators have found TIA to be less frequent in blacks than whites. Our findings demonstrate a similar proportion of admissions for TIA in blacks and whites. However, it is possible that minorities may ignore or not pursue medical care because of limited access to health care or socioeconomic or educational limits. To the extent that this is true, blacks may be underrepresented in the sample of hospitalized patients. The mainstays of medical and surgical stroke prevention may favor whites because of the proximal distribution of their atherosclerotic pathology. The carotid endarterectomy rate in blacks has been reported at 40% of that of whites. There is no procedure available in common use for addressing the intracranial vessel atherosclerotic involvement that predominates in black patients.

In addition to the variety of cerebrovascular pathology, black patients are more likely to be admitted with other medical comorbidities that may complicate the short-term hospital course after stroke. Prevalence studies reveal that blacks are more apt to smoke, to be overweight and/or diabetic, and to have an elevated systolic blood pressure. Age-adjusted death rates are higher for blacks for cardiovascular disease, malignant neoplasms, influenza, diabetes, chronic obstructive pulmonary disease, cirrhosis of the liver, and tuberculosis.

Baum and Goldstein (1982) used mortality statistics from the National Center for Health Statistics and found that death rates from hemorrhagic strokes were higher for blacks than for whites, unlike our findings in hospitalized patients where the number of patients who died from hemorrhagic stroke was not significantly different for blacks and whites. There are several possible explanations for this apparent discrepancy. First, the number of hemorrhagic strokes is relatively small, with hemorrhagic strokes being only about 10% of the total. Thus, there were relatively few stroke deaths in absolute numbers, particularly in blacks. Moreover, the time frame for the sample of Baum and Goldstein (1968 through 1977) was quite different from ours (1990), and at the time of their sample, mortality rates for stroke in blacks relative to whites were changing dramatically, particularly for blacks. Finally, our sample used hospitalized patients only, whereas theirs used death certificate data and may have included patients who were not hospitalized at the time of their death.

Very little information is available about racial disparities in stroke treatment after hospitalization. In a field study of the prevalence of stroke in Mississippi, Schoenberg et al found that whites with stroke tended to be seen by a specialist in neurology and/or neurosurgery more often than blacks with stroke. It is not known...
if care by these specialists results in superior outcome for acute stroke patients. Other investigators have documented inequalities in healthcare resource distribution on the basis of race and income. However, in a prepaid health maintenance organization, blacks were more likely than whites to be hospitalized for hemorrhagic cerebrovascular disease, cerebral thrombosis, and ischemic cerebrovascular disease. In the same study, whites were more likely to be hospitalized for extracranial occlusive disease, possibly reflecting a bias toward treating extracranial occlusive disease with carotid surgery.

We tested the hypothesis that there was a difference in hospital-based expenditures for black versus white patient care after acute stroke to determine if the advent of diagnosis-related groups (DRGs) and emphasis on early discharge may have compromised the treatment given blacks. A disproportionate share of the black stroke patients in Maryland have their hospitalization costs paid by Maryland Medicaid. In this study, 0.023 and 0.177 of the white patients with hemorrhagic and occlusive strokes, respectively, had Medicaid as their primary payer. In contrast, 0.01159 and 0.0828 of the black patients with hemorrhagic and occlusive strokes had Medicaid as their primary payer. However, the mean total hospital charge for surviving black patients with hemorrhagic events was $14,994, and for white patients it was $13,967 ($P = .61, t test with unequal variances), suggesting no significant difference. The average charge for occlusive events tended to be lower ($6,380 for surviving black patients and $5,017 for surviving white patients), but the total charges for black patients were significantly higher than for white patients ($P < .0001, t test with unequal variances). On the basis of the total hospital charges, we conclude that the observed increased severity of stroke in black patients is unlikely to be an artifact of less intensive medical treatment during the short-term hospitalization.

It should be noted that Maryland, in contrast to all other American states, does not use the DRG system for hospital-based reimbursement. The DRG waiver is dependent on the state collecting accurate charge and demographic information on all (not just Medicare or Medicaid) patients hospitalized in the state in all nonfederal acute-care hospitals. The state compares the costs to the Health Care Finance Administration (HCFA) from the current DRG-exempt system with those from the traditional DRG system. The waiver remains in effect because the state is able to show that its system is less costly to the HCFA than the DRG system. It is in the hospitals' interests to code the information accurately to maintain the DRG waiver. A proprietary firm audits a sample of records from 10 of the 52 nonfederal acute-care hospitals in the state each year. In the past year, this represented about 2,500 audits on 68,000 patient admissions.

In Maryland, unlike other states, hospital-based charges and collections are the same regardless of payer, be it Medicare, Medicaid, private insurance, or self-pay, because state law requires identical charges for all payers. From the hospitals' point of view, the nature of the payer is irrelevant, and there is no incentive to treat Medicaid or Medicare patients differently from those with private insurance.

Much of the 1990 Maryland Health Services Cost Review Commission data from this study correlates with recently published studies of stroke. The community hospital-based stroke program study reviewed 4129 hospitalized cases of stroke from North Carolina, Oregon, and New York. They found a higher proportion of hemorrhagic strokes with a ratio of 4.5 to 1 for infarction to hemorrhagic strokes. Patients with hemorrhagic stroke were most likely to be comatose (0.43) on admission and most likely to die (0.45) during the admission. These coma rates are much higher than what we observed (0.01 to 0.16). It is possible that the fraction of our patients that were in coma was underestimated because we depended on the ICD coding for indication of coma rather than individual chart review. Our observations of hemorrhagic stroke mortality were comparable (0.19 to 0.41) with similar mortality through all age groups from 45 to 95 years of age.

The indices that we used to judge severity of stroke were indirect, but we believe they all have face validity. The use of LOS as an indicator can be criticized as being somewhat subjective, since it is dictated by the attending physician and is therefore subject to manipulation. However, if the LOS was racially motivated in favor of whites (ie, white patients are allowed to stay longer than blacks), then the differences in severity are understated by the LOS reported here. The death rate, age at death of those who died, and the presence of coma are less subject to manipulation than LOS, and all of these indices suggest more severe strokes in blacks.

References


Racial disparities in severity of cerebrovascular events.
K V Kuhlemeier and S A Stiens

Stroke. 1994;25:2126-2131
doi: 10.1161/01.STR.25.11.2126
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1994 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/25/11/2126

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

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

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/