Stroke in a Biracial Population
The Excess Burden of Stroke Among Blacks

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Background and Purpose—Excess mortality resulting from stroke is an important reason why blacks have higher age-adjusted mortality rates than whites. This observation has 2 possible explanations: Strokes occur more commonly among blacks or blacks have higher mortality rates after stroke. Our population-based epidemiological study is set in the Greater Cincinnati/Northern Kentucky region of 1.31 million people, which is representative of the US white and black populations with regard to many demographic and socioeconomic characteristics.

Methods—Hospitalized cases were ascertained by International Classification of Diseases (ninth revision) discharge codes, prospective screening of emergency department admission logs, and review of coroner’s cases. A sampling scheme was used to ascertain cases in the out-of-hospital setting. All potential cases underwent detailed chart abstraction by study nurses, followed by physician review. Race-specific incidence and case fatality rates were calculated.

Results—We identified 3136 strokes during the study period (January 1, 1993, to June 30, 1994). Stroke incidence rates were higher for blacks at every age, with the greatest risk (2- to 5-fold) seen in young and middle-aged blacks (<65 years of age). Case fatality rates did not differ significantly in blacks compared with whites. Applying the resulting age- and race-specific rates to the US population in 2002, we estimate that 705 000 to 740 000 strokes have occurred in the United States, with a minimum of 616 000 cerebral infarctions, 67 000 intracerebral hemorrhages, and 22 000 subarachnoid hemorrhages.

Conclusions—Excess stroke-related mortality in blacks is due to higher stroke incidence rates, particularly in the young and middle-aged. This excess burden of stroke incidence among blacks represents one of the most serious public health problems facing the United States. (Stroke. 2004;35:426-431.)

Key Words: blacks ■ cerebral infarction ■ epidemiology ■ intracerebral hemorrhage ■ stroke ■ subarachnoid hemorrhage

Stroke is a major cause of excess mortality among blacks compared with whites. In the National Health and Nutrition Survey I Epidemiologic Follow-Up Study, stroke-related mortality accounted for 28% of the difference in total mortality rates between blacks and whites.1 Although blacks have higher stroke-related mortality overall, the racial differences vary greatly with the age of the population. For example, US stroke-related mortality rates in 1998 among those 25 to 64 years of age were 3-fold higher in blacks than whites, whereas among blacks >65 years of age, rates are similar to those among whites.2 The excess stroke-related mortality among blacks has at least 2 possible explanations: Strokes occur more commonly among blacks, particularly among the young and middle-aged, or blacks have a higher likelihood of case fatality following stroke.

The Greater Cincinnati/Northern Kentucky Stroke Study (GCNKSS) is designed to investigate the differences in stroke incidence rates and case fatality in the biracial population of the greater Cincinnati metropolitan area. Our study population is similar to that of the entire United States with regard to median age, percent black, median household income, education level, and percent of population below the poverty level.3 In addition, the age-adjusted stroke mortality rates in Ohio are similar to those from the entire US population.4 Thus, our study provides a reasonable comparison of stroke incidence rates between blacks and whites, which can be generalized to the US population and can provide an estimate of the total number of strokes occurring in the United States each year. We report here the stroke incidence rates and case fatality from our biracial population in 1993–1994.
Materials and Methods

The methodology of the GCNKSS has been previously described.3 The GCNKSS study population is defined as all residents of the greater Cincinnati metropolitan region, which includes 2 southern Ohio counties and 3 contiguous northern Kentucky counties that abut the Ohio River. Included in this area are 19 hospitals. Although residents of surrounding counties also seek care at these hospitals, only residents of the 5 study area counties, as determined by ZIP code of residence, are included as cases. Previous studies have documented that residents of the 5 counties who have strokes exclusively seek care at these 19 hospitals rather than at more distant hospitals in the outlying region.6 This study was approved by the institutional review boards at all participating hospitals.

Study nurses screened the medical records of all inpatients with primary or secondary stroke-related International Classification of Diseases, ninth revision, discharge diagnoses (codes 430 through 438) from the 19 acute-care hospitals in the study region. The study nurses also reviewed all coroners’ cases in which stroke was listed as the primary or secondary cause of death.

Phase 1 of the GCNKSS involved ascertainment of hospitalized and autopsied strokes in blacks between January 1, 1993, and June 30, 1993; results for this period have been previously reported.3 Phase 2 involved collection of all strokes in the study population between July 1, 1993, and June 30, 1994, to allow direct racial comparisons of incidence and case fatality rates within the same population. In addition to ascertaining inpatient strokes with the methodology described above, a sampling scheme for out-of-hospital events was used. In Phase 2, strokes not found by inpatient monitoring were ascertained by monitoring all visits to 18 of the hospital’s emergency departments (Cincinnati Children’s Hospital was excluded), 5 county coroner offices, 16 public health clinics, and 14 hospital-based outpatient clinics and family practice centers. In addition, monitoring was performed in a random sample of 50 of the 878 primary care physicians’ offices and 25 of the 193 nursing homes in the greater Cincinnati metropolitan area. Events found by out-of-hospital monitoring were checked against inpatient records to prevent double counting. For the purpose of this report, cases from both Phases 1 and 2 were used to increase statistical power for comparisons involving the black population.

To qualify as an incidence case, a patient must have met the criteria for 1 of the 5 stroke categories adapted from the Classification for Cerebrovascular Diseases III and from epidemiological studies of stroke in Rochester (Minn); cerebral ischemia, intracerebral hemorrhage (ICH), subarachnoid hemorrhage (SAH), stroke of uncertain cause, or transient ischemic attack (TIA).7,8,9 The onset of stroke symptoms must have occurred within the study period of January 1, 1993, to June 30, 1994, for blacks and July 1, 1993, to June 30, 1994, for whites and other races.

Cases were excluded if they (1) had discharge/autopsy diagnosis or neuroimaging consistent with stroke but no clinical history of stroke or (2) had a clinical diagnosis of stroke and died within 24 hours of symptom onset but had no focal neurological deficit and no confirmatory neuroimaging or autopsy. All excluded cases and the reasons for exclusion were recorded.

Once potential cases were identified, the study nurse gathered all relevant clinical data. Clinical data collected included stroke symptoms, physical examination findings, past medical/surgical history, medication use before stroke, social history/habits, prehospital evaluation, vital signs and emergency room evaluation, neurological evaluation, diagnostic test results (including laboratory testing, ECG and cardiac testing, and neuroimaging of any type), treatments, outpatient care, type of insurance, and current address. Classification of race-ethnicity was as self-reported in the medical administrative record. The study nurse abstracted all information and then determined whether a stroke or TIA had occurred. All borderline or possible cases were abstracted for physician review.

A study physician reviewed every abstract and all available neuroimaging studies to determine whether a stroke or TIA had occurred. All imaging findings were characterized. The physician assigned stroke category and mechanism to each event on the basis of all available information using the definitions listed above and previously reported.3

An analysis of agreement regarding case ascertainment was performed on 18 medical record abstracts for 3 study physicians and the 2 study nurse coordinators (who trained other study nurses). Intraclass correlation calculation indicated excellent agreement. In this quality assurance study (agreement on case versus not a case), intraclass correlation was 0.91 and \( \kappa = 0.64 \) to 0.88 between individuals, indicating excellent agreement. It should be noted that study nurses were instructed to call study physicians for any questionable case and to be overinclusive (ie, even borderline events were abstracted). In this way, it should be expected that agreement would not be 100% because study nurses were expected to include probable noncases if not sure. Nurses and physicians also determined stroke subtype (ie, ischemic stroke versus ICH versus SAH). Intraclass correlation for stroke subtype was 0.87 and \( \kappa = 0.71 \) to 0.93 between individuals.

Statistical Analysis

SAS version 8.2 (SAS Institute Inc) was used for data analysis. The numerator for incidence rate calculation was the number of incidence cases confirmed by physician review. Given our out-of-hospital ascertainment sampling scheme, the number of physician-confirmed outpatient cases was weighted to estimate the total number of out-of-hospital events in the study population. In this manner, events ascertained in the physician offices and nursing homes were multiplied by 17.56 (878 possible sites/50 sampled) and 7.72 (193 possible sites/25 sampled), respectively.

The denominator was based on linear extrapolations of county populations in race, sex, and age subcategories for the years 1993 and 1994. All population information was obtained from the US Census Bureau Website (http://www.census.gov/). The at-risk population for 1993 to 1994 included 197,541 blacks and 1,141,092 whites. The 95% confidence intervals (CIs) for incidence rates were calculated assuming a Poisson distribution. Age-, race-, and sex-specific rates were also determined. Race-specific, adjusted rates were standardized to the 1990 US population.

Age-, race-, and sex-specific case fatality rates were calculated at 30 days, 90 days, and 1 year. Case fatality was defined as death resulting from any cause within the defined time periods. Vital status information for each patient was ascertained from the Ohio and Kentucky departments of vital statistics death certificate database and from the national Social Security Death Index. Confirmation of death during hospitalization, as recorded in the medical record abstract, was achieved by use of the databases. Those patients not listed as dead in either regional or national database within the specified time periods were presumed to be alive. Cause of death was not extracted; for the purposes of this report, deaths cannot be classified as stroke-related except for the temporal proximity to the incident stroke. To allow direct racial comparisons, black case fatality rates were age and sex adjusted to our white cases.

Results

Study nurses screened 17,318 potential events, and 4637 events were fully abstracted. Of the 17,318 events screened, 12,564 did not meet criteria after nurse review: 3916 were of the study region, 2942 occurred outside the study time period, and 5706 did not meet case criteria for stroke. There were 117 records that could not be obtained (0.6% of all potential events). Thus, 4637 events were fully abstracted by study nurses. After physician review, 4251 events were determined to meet case criteria (92% of the abstracted events).

The Greater Cincinnati/Northern Kentucky region is essentially a biracial population, and <1% of all stroke cases (n = 34) occurred in other race-ethnic groups such as Hispanic/Latino or Asian. The data presented hereafter include only
TABLE 1. Age-Specific Incidence Rates for First-Ever Stroke, Excluding TIAs, per 100,000 (for 1993–1994, Sex-Adjusted to 1990 US Population)

<table>
<thead>
<tr>
<th>Age Category, y</th>
<th>Blacks (95% CI)</th>
<th>Whites (95% CI)</th>
<th>Risk Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;35</td>
<td>12 (5–19)</td>
<td>5 (1–10)</td>
<td>2.2 (1.3–3.1)</td>
</tr>
<tr>
<td>35-44</td>
<td>96 (76–115)</td>
<td>19 (11–28)</td>
<td>5.0 (3.9–6.1)</td>
</tr>
<tr>
<td>45-54</td>
<td>308 (274–342)</td>
<td>121 (99–142)</td>
<td>2.6 (2.0–3.2)</td>
</tr>
<tr>
<td>55-64</td>
<td>526 (482–571)</td>
<td>239 (209–269)</td>
<td>2.2 (1.6–2.8)</td>
</tr>
<tr>
<td>65-74</td>
<td>1068 (1004–1132)</td>
<td>584 (537–631)</td>
<td>1.8 (1.3–2.3)</td>
</tr>
<tr>
<td>75-84</td>
<td>1829 (1745–1912)</td>
<td>1218 (1150–1287)</td>
<td>1.5 (1.4–1.8)</td>
</tr>
<tr>
<td>≥85</td>
<td>2374 (2279–2470)</td>
<td>1786 (1703–1869)</td>
<td>1.3 (1.0–1.6)</td>
</tr>
</tbody>
</table>

Table 2 displays the race-specific, age- and sex-adjusted 30-day, 90-day, and 1-year case fatality in our study region after ICH and SAH was not significantly different in 1993 to 1994 compared with data collected with similar methodology in 1988 (data not shown).6

To verify that our sampling methodology for out-of-hospital strokes did not bias our results, we recalculated incidence rates and risk ratios for inpatients only; the risk ratios were essentially unchanged, except in the 45- to 54-year-old category where white stroke patients were ascertained in the out-of-hospital setting. When only inpatient strokes are considered, the risk ratios are essentially unchanged, except in the 45- to 54-year-old category incidence rates per 100,000 were 291 (95% CI, 257 to 324) for blacks and 76 (95% CI, 58 to 93) for whites, with a risk ratio of 3.8 (95% CI, 3.2 to 4.5) (see Table 1). The inpatient-only incidence rates for blacks is almost the same, whereas the rate for whites in this age category was much lower. This demonstrates that if only inpatient strokes are considered, the risk ratio obtained would overestimate the racial disparity for this age group. It is not clear why only this age group is affected.

Table 2 displays the race-specific, age- and sex-adjusted incidence rates per 100,000 adjusted to the 1990 US population for each stroke category. Adjusted incidence rates are statistically significantly higher for blacks for all stroke categories. Sex-adjusted, age- and race-specific rates for each stroke category are presented graphically in Figure 2.

The race-specific, age- and sex-adjusted incidence rates per 100,000 for first-ever stroke were 278 (95% CI, 253 to 304) in blacks and 153 (95% CI, 146 to 160) in whites. Incidence rates for each race were calculated for each 6-month period of case ascertainment, and there was no significant variation between periods (data not shown). Table 1 displays the sex-adjusted, age-specific incidence rates and risk ratios for first-ever stroke by race. Figure 1 displays age-, race-, and sex-specific incidence rates for first-ever stroke. Stroke incidence rates are higher for blacks at every age, although the disparity in rates is highest in the younger age groups, especially those 35 to 44 years of age.

Figure 1. Age-, race-, and sex-specific incidence rates per 100,000 for first-ever stroke (excluding TIAs).
medical administrative databases using admission data. To refine our estimates, we supplemented our data for blacks and whites with the Northern Manhattan Stroke Study (NOMASS) rates for Hispanics and used our white rates for other race-ethnic groups (for which no stroke incidence data currently exist). In doing so, we conservatively estimate that a minimum of 705,000 strokes occurred in the US during 2002. Furthermore, we conservatively estimate that a minimum of 616,000 cerebral infarcts, 67,000 ICHs, and 22,000 SAHs occurred in the US in 2002. If we use the higher adjusted incidence rates from Rochester for whites (with Cincinnati rates for blacks, NOMASS rates for Hispanics, and Rochester white rates for other race-ethnic groups), we estimate that a maximum of 736,000 strokes occurred in the United States during 2002. We estimate that a minimum of 80,000 excess strokes occur in the United States each year because of higher incidence rates in blacks. In other words, 80,000 fewer strokes would occur each year if blacks had the same age-adjusted incidence rates as whites.

**Discussion**

Our data from a biracial metropolitan population of 1.31 million demonstrate that blacks have nearly double the rates of first-ever stroke and all stroke compared with whites. Blacks who are <65 years of age have 2 to 5 times the risk of stroke compared with whites of similar age. Even in the oldest age groups (≥85 years), blacks have higher rates of all stroke, first-ever stroke, and first-ever infarct, although the difference is smaller than for younger ages. Rates of ICH are higher in blacks than whites except for persons who are ≥85 years of age. Rates of SAH are generally higher in blacks except for persons who are ≥65 years of age. This substantial excess burden of stroke incidence among blacks represents one of the most serious public health problems facing the United States. Knowledge that incidence rates are substantially increased for all stroke categories in blacks is important if measures are to be taken to reduce this disparity.

The excess stroke-related mortality among blacks can be attributed to higher stroke incidence because the age- and sex-adjusted case fatality is not significantly different between blacks and whites. Furthermore, the age-related difference in mortality rates, with higher stroke-related mortality in younger and middle-aged blacks, may be attributed to increased stroke incidence among blacks <65 years of age (Figure 1). The interaction between race and age with regard to stroke incidence is complex. Although the racial disparity in stroke incidence is greatest at younger ages (see Table 1), the absolute number of strokes that occur is greatest in the older age categories (especially for ischemic stroke; data not shown). The fact that the racial disparity persists in older age groups despite proportionally fewer blacks surviving into the older age categories has important public health implications. Interventions designed to reduce risk or increase knowledge about stroke must be targeted to young blacks and older members of both racial groups.

One possible explanation for the racial disparity in stroke incidence rates is a disparity of stroke risk factors among different racial-ethnic groups. Risk factor prevalence data from a 1995 random-digit dialing telephone survey of Greater

**TABLE 3. Adjusted** Case Fatality, 1993–1994

<table>
<thead>
<tr>
<th></th>
<th>30 Days, %</th>
<th>90 Days, %</th>
<th>1 Year, %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Blacks</td>
<td>Whites</td>
<td>Blacks</td>
</tr>
<tr>
<td>First stroke</td>
<td>13</td>
<td>16</td>
<td>20</td>
</tr>
<tr>
<td>First infarct</td>
<td>10</td>
<td>13</td>
<td>17</td>
</tr>
<tr>
<td>First ICH</td>
<td>36</td>
<td>35</td>
<td>40</td>
</tr>
<tr>
<td>First SAH†</td>
<td>32</td>
<td>43</td>
<td>32</td>
</tr>
</tbody>
</table>

*Black case fatality adjusted to the white group for that subtype by age and sex, except for SAH.
†SAH case fatality unadjusted because numbers are too small to estimate adjusted percentages.
Cincinnati and Northern Kentucky area residents matched to our stroke population by age, race, and sex have shown that there is a significantly higher prevalence of stroke risk factors such as hypertension, diabetes, smoking, and excessive alcohol use among the black at-risk population compared with whites of similar ages. The distribution of stroke risk factors varied in a race-specific pattern for the cases in this report (see Table I). This disparity of stroke risk factors has previously been documented in the NOMASS triethnic population and other studies. Further study of risk factor variation by race is necessary, including such issues as risk factor severity, duration, and treatment.

Another possibility is that blacks as a group have less access to medical care. Our study was not specifically designed to measure access to care, but previous studies have documented a racial disparity in access to health care both before and after stroke. If blacks obtain less care, undergo less modification of risk factors, and are less likely to take medications to reduce stroke risk, this may account for the higher stroke incidence rates seen. Although insurance status is a weak measure of access to care, our black stroke population was more likely to have Medicaid for insurance than whites. Our study is limited by having few other measures of socioeconomic status. It is important to note in this context that race-ethnicity is a sociopolitical construct that may describe socioeconomic status to greater degree than a genetic or biological subgrouping.

Our population-based study of stroke is perhaps the largest ever performed in the United States, with substantial statistical power and narrow CIs around incidence point estimates. However, despite the large numbers of cases collected, some age-, race-, and sex-specific strata have small numbers, which can limit statistical precision when comparisons are made. Another limitation is the potential for information bias (missing or erroneous data) because of the sheer volume of data collected. Our study is potentially limited by selection bias resulting from our methodology in which true strokes may be unrecognized clinically or miscoded and thus not ascertained. This may be balanced in part by inclusion of some events that mimic stroke and meet the case definition but cannot be excluded by retrospective physician review. Our study was performed with a methodology similar to that in previous epidemiologic studies (Rochester, NOMASS), which suffer from the same limitations. The similarities in methodology allow comparisons between studies.

Our results must be taken in the context of the other US population-based epidemiology studies that use similar methodology. The population-based study in Rochester has reported sex- but not race-specific incidence rates because the population of 85,800 in Rochester is 88% white and only 4% black (US census 2000). Our stroke incidence rates in whites are lower than those in Rochester, although this finding may be explained in part by the removal of prevalent cases of stroke from the Rochester at-risk population. If the entire population were considered at risk, then their rates would be approximately 7% lower (personal communication, Robert Brown, Jr, MD, April 2003). Moreover, although we are confident in our case ascertainment for hospitalized and autopsy-confirmed strokes, the logistical difficulty in ascertaining cases in the out-of-hospital setting for a population of 1.31 million requires a sampling scheme. Only 9.5% of our stroke cases were ascertained in the out-of-hospital setting compared with 15% in Rochester (4% ascertained in nursing homes, 11% in other settings; personal communication, Robert Brown, Jr, MD, April 2003). Residents of the Rochester community have medical diagnoses from every hospitalization, outpatient visit, nursing home physician interaction, and death certificate or autopsy entered into a central computer index. It should be noted that risk ratios did not change significantly when our entire ascertainment methodology was compared with inpatient-only methodology (except for the 45- to 54-year-old age group). Thus, there does not appear to be a systematic bias in our sampling methodology for out-of-hospital ascertainment.

NOMASS is a population-based study of a triethnic population of ~149,000 that is primarily Hispanic (64%), with fewer whites (22%) and blacks (13%). Our rates are higher than the rates NOMASS for both blacks and whites. For its denominator, NOMASS considers only those individuals >20 years of age at risk. If the <20-year-old age group, the largest segment of the population with the lowest stroke risk, were taken into account, then the NOMASS rates would be comparatively even lower. However, NOMASS currently provides the best estimate of stroke incidence in the Hispanic population.

Given the observational nature of our region-specific data, care must be taken in making inferences and generalizing to the United States as a whole. Our study population is similar to that of the United States with regard to many demographic and socioeconomic variables, but stroke risk factors, stroke incidence, and stroke mortality vary by region of the country. For example, the “stroke belt” has been defined as those states having the highest mortality from stroke, and it should not be assumed that similar results would be found in these regions. Yet, our estimates for the total burden of stroke in the United States during 2002 are similar to those obtained from a national administrative database and other population-based studies from other regions, suggesting that our estimates are a reasonable approximation for the United States. Ideally, population-based epidemiological studies from other regions would be performed for comparison and could be validated by other approaches such as the use of administrative databases.

In summary, our study has demonstrated that stroke incidence rates are higher for blacks in all stroke categories (cerebral infarction, ICH, and SAH). Stroke risk in blacks is higher at all ages but excessively so in the younger age categories (35 to 54 years of age). Case fatality data are similar between races in all stroke categories. Thus, the excess stroke-related mortality in blacks documented in previous studies is due to increased stroke incidence rather than a higher likelihood of dying after stroke. Further research is necessary to elucidate the reasons for the higher stroke incidence rates among blacks.

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