The Unchanging Incidence and Case-Fatality of Stroke in the 1990s
A Population-Based Study

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Background and Purpose—Many advances were made in stroke prevention strategies during the 1990s, and yet temporal trends in stroke incidence and case-fatality have not been reported in the United States. Blacks have a 2-fold higher risk of stroke; however, there are no data over time showing if any progress has been made in reducing racial disparity in stroke incidence. The objective of this study was to examine temporal trends in stroke incidence and case-fatality within a large, biracial population during the 1990s.

Methods—Within a biracial population of 1.3 million, all strokes were ascertained at all local hospitals using International Classification of Diseases, 9th Revision codes during July 1993 to June 1994 and again in 1999. A sampling scheme was used to ascertain cases in the out-of-hospital setting. Race-specific incidence and case-fatality rates were calculated and standardized to the 2000 US Census population. A population-based telephone survey regarding stroke risk factor prevalence and medication use was performed in 1995 and 2000.

Results—There were 1954 first-ever strokes in 1993–1994 and 2063 first-ever strokes in 1999. The annual incidence of first-ever hospitalized stroke did not significantly change between study periods: 158 per 100 000 in both 1993–1994 and 1999 (P=0.97). Blacks continue to have higher stroke incidence than whites, especially in the young; however, case-fatality rates continue to be similar between races and are not changing over time. Medication use for treatment of stroke risk factors significantly increased in the general population between study periods.

Conclusions—Despite advances in stroke prevention treatments during the 1990s, the incidence of hospitalized stroke did not decrease within our population. Case-fatality also did not change between study periods. Excess stroke mortality rates seen in blacks nationally are likely the result of excess stroke incidence and not case-fatality, and the racial disparity in stroke incidence did not change over time. (Stroke. 2006;37:2473-2478.)

Key Words: epidemiology ■ incidence ■ stroke

Stroke is the third leading cause of death and a leading cause of major adult disability in the United States.1,2 The incidence of stroke in the United States remained stable in the 1980s according to population-based studies performed in Rochester, Minn.3,4 Stroke mortality in the United States decreased in the 1980s and stabilized in the 1990s in other studies.5,6 However, trends in stroke incidence in the 1990s in the United States, a time period in which several advances were made in stroke prevention and treatment, remain unknown.

Many population-based studies of trends in stroke incidence have been obtained from populations without a significant proportion of minorities.5,7-9 Yet, stroke incidence and mortality are very different in minority populations when compared with whites both in the United States and in other countries.1,10-12 For example, blacks have a higher stroke mortality rate than whites. This can be attributed to a greater incidence of stroke in blacks, because case-fatality compared between the two races is similar.13 Trends in stroke incidence and case-fatality over time have not been previously reported within a US population that includes a significant minority proportion.

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 of 1.3 million is similar to the United States with regard to median age, percent black, median household income, and education level.14 We present data regarding the incidence and case-fatality of stroke in the years 1993–1994 and 1999.

Methods
The Greater Cincinnati/Northern Kentucky region includes two southern Ohio counties and three contiguous northern Ky counties.
that border the Ohio River. In this area, 19 hospitals were active in 1993–1994 and 18 were active in 1999. Although residents of surrounding counties also seek care at these hospitals, only residents of the five study counties, as determined by zip code of residence, are considered for case ascertainment. Previous studies have documented that residents of the five counties who have a stroke exclusively seek care at these 18 hospitals rather than at more distant hospitals in the outlying region.14 Because the study population of the GCNK region has <3% Hispanic and other minorities, we considered events occurring only in blacks and whites in our analyses. This study was approved by the Institutional Review Board at all participating hospitals for each study period.

The GCNKSS involved ascertainment of all stroke events that occurred in the population between July 1, 1993, and June 30, 1994, and again in the calendar year of 1999. In 1993–1994, study nurses screened the medical records of all inpatients with primary or secondary stroke-related International Classification of Diseases, 9th Revision codes (ICD-9) discharge diagnosis 010–019, 330–334, 343, 430–436 from the 19 acute-care hospitals in the study region. In addition, strokes not found by inpatient screening were ascertained by monitoring all stroke-related visits to hospital emergency departments (with the exception of Cincinnati Children’s Hospital), 16 public health clinics, and 14 hospital-based outpatient clinics and family practice centers. Cases for which stroke was listed as the primary or secondary cause of death by one of the five county coroners’ offices were also included. Further monitoring was performed by examining the records of potential stroke cases in a random sample of 30 of the 878 primary care physicians’ offices and 25 of the 193 nursing homes in the GCNK region. This sampling was necessary given the large number of physician offices and nursing homes in the region. Events found by out-of-hospital monitoring were crosschecked against inpatient records to prevent double counting.

In 1999, the ICD-9 codes screened were changed to 430–436 for inpatient ascertainment, because the codes 437 and 438 produced an extremely low yield for the 1993–1994 data.14 The Children’s Hospital screening for 1999 did include 437 and 438, however, because these codes yielded a substantial number of childhood strokes in 1993–1994.15 Screening of out-of-hospital clinical sites in 1999 was nearly identical to 1993–1994. Monitoring included all visits to emergency departments (except for Children’s Hospital), the 15 area public health clinics, the 10 hospital-based outpatient clinics and family practice centers, the 5 county coroners’ offices, a random sample of 37 of 849 primary care physicians’ offices, and 23 of 171 nursing homes. These sites were selected randomly by the study statistician from a list generated from a combination of the local yellow pages and the American Medical Association listing of physicians in the region.

In addition to retrospective ascertainment by ICD-9 codes, for 1999, we also implemented a prospective case ascertainment technique. Admission logs of all local emergency departments were screened by study nurses for stroke-like symptoms (eg weakness, numbness, vision change, problems speaking). Charts for patients suspected of having a stroke were abstracted regardless of ICD-9 code assignment. Nine additional cases ascertainment only by prospective monitoring were not included in calculation of incidence rates in 1999 for this report to keep methodology the same for the two time periods. In addition, only those cases from 1993–1994 with ICD-9 codes 430–436 were included in this analysis to ensure valid comparisons between study periods.

To qualify as a GCNKSS incident case, a patient must have met the criteria for one of the five stroke categories adapted from the Classification for Cerebrovascular Diseases III16 and from epidemiologic studies of stroke in Rochester, Minn: cerebral ischemia, intracerebral hemorrhage, subarachnoid hemorrhage, or stroke of uncertain cause. Only first-ever-in-a-lifetime events were included. Transient ischemic attacks (TIA), defined as symptoms lasting less than 24 hours regardless of imaging results, were not included in this analysis. The onset of stroke symptoms must have occurred within the study time periods of July 1, 1993, to June 30, 1994, or January 1, 1999, to December 31, 1999. Charts were screened for an additional 60 days beyond the end of the both study period to capture patients who experienced a stroke during the study period but had not yet been discharged (see part 1 of the online supplement for expanded section, available at http://stroke.ahajournals.org).

A study physician reviewed every abstract and all available neuroimaging studies to verify whether a stroke or TIA had occurred. The physician assigned stroke category and mechanism to each event based on all available information using definitions listed previously and previously reported (see part 2 of the online supplement, available at http://stroke.ahajournals.org).

Calculation of Incidence and Case-Fatality

Incidence rates for first-ever stroke were calculated using two measures: strokes ascertained in hospital settings only and “all” strokes (ascertained in either hospital or out-of-hospital settings such as nursing homes, clinics, and physician offices). The numerator for calculation of the hospitalized incidence rate was the number of first-ever strokes confirmed by physician review ascertained through inpatient records or emergency departments. The numerator for the incidence rate for “all strokes” also included the number of first-ever stroke ascertained through outpatient clinics and family practice centers, and coroners’ offices plus a weighted estimate of the number of strokes ascertained only in the physician’s office or nursing home. Events ascertained in physicians’ offices and nursing homes were multiplied by 18- and 8-fold for 1993–1994 events and 23- and 7-fold for 1999 events, respectively, to account for the sampling methodology. Cases ascertained in the hospital-based clinics were not weighted, because all the clinics in the region were screened. Events were considered to be noncases if medical records could not be located.

The denominator for the calculation of incidence rates was extracted from the US Census Bureau web site (www.census.gov). The estimates are based on extrapolation and/or interpolation of county population between enumerated census years accounting for births, deaths, and migration. The at-risk population included 197 541 blacks and 1 114 092 whites for 1993–1994 and included 204 296 blacks and 1 106 934 whites for 1999. The 95% CI for the incidence rates were calculated assuming a Poisson distribution. Age-, race-, and gender-specific rates were also determined. All adjusted rates were standardized to the 2000 US population.

Case-fatality was defined as death from any cause within 30 days of stroke. To verify vital status for case-fatality calculations, death certificate data from the Ohio and Ky Department of Vital Statistics were reviewed first. The national Social Security Death Index was queried for any patient who did not have an Ohio or Ky death record. Death during hospitalization, as recorded in the medical record abstract, was confirmed in at least one of the mortality sources. Patients for whom a death record could not be found were presumed to be alive.

Population-Based Telephone Survey

Our general population was surveyed regarding self-reported stroke risk factors and medication use in 1995 and again in 2000. The detailed methods for this telephone survey and results with regard to stroke knowledge and prevalence of risk factors have been previously published and are available in part 3 of the online supplement (available at http://stroke.ahajournals.org; Ref. 19 is cited in this supplement).17,18 The phone survey is designed to ensure that the respondents represent a randomly selected group of individuals whose demographics (age, race, and gender) closely match the expected demographics of the population of patients sustaining a stroke.

Results

There were 1954 first-ever strokes in 1993–1994 and 2063 first-ever strokes in 1999 among residents of the five-county area. In 1993–1994, 117 patient charts could not be located (0.7% of all charts reviewed by study nurses) as compared with 27 charts (0.2%) in 1999. The demographics of the stroke populations were similar between study periods: in
1993–1994, the stroke population was 18.2% black and 57.0% female; and in 1999, it was 17.7% black and 57.7% female. There were no significant differences in age, gender, or race/ethnicity between cases ascertained in inpatient and out-of-hospital settings in either study period.

The annual incidence of first-ever hospitalized stroke did not significantly change between study periods: 158 per 100 000 in both 1993–1994 and 1999 (P=0.97). Race-specific incidence rates of first-ever hospitalized stroke are presented in Table 1 and P values are presented in the supplemental Table I, available online at http://stroke.ahajournals.org. The incidence of hospitalized first-ever stroke in 1999 did not differ significantly from that of 1993–1994 for blacks or whites. The incidence of each stroke subtype, including ischemic stroke, intracerebral hemorrhage, and subarachnoid hemorrhage, also did not change between study periods.

The inclusion of cases ascertained in out-of-hospital settings (see Table 2 and P values are presented in the supplemental Table II, available online at http://stroke.ahajournals.org) did slightly increase the incidence of all stroke, from 182 to 206 (P<0.001). This increase was driven primarily by ischemic strokes ascertained in the out-of-hospital setting for whites, in which an increase from 156 to 181 was noted (P<0.001). The incidence of first-ever ischemic stroke in blacks did not change (226 in 1993–1994 and 219 in 1999, P=0.71). Other race and stroke subtype categories did not show evidence of change.

Age-specific incidence rates of ischemic stroke were also analyzed. Table 3 demonstrates the relative risk ratios for stroke in blacks compared with whites in the two study periods. As has been previously described from our 1993–1994 population,13 the relative risk of stroke is higher in younger blacks (<55 years old) when compared with whites. In the elderly, the risk of stroke is roughly equal between blacks and whites.

Thirty-day case-fatality rates were not significantly different between study periods (13.9% in 1993–1994 versus 14.7% in 1999). Table 4 and supplemental Table III, available online at http://stroke.ahajournals.org, presents the case-fatality rates stratified by race and stroke subtype in the two study periods. There were no significant differences in the 30-day all-cause case-fatality by race or by study period (P=0.36).

Use of diagnostic imaging increased in the 1999 study period when compared with the 1993–1994 study period: CT was documented in 95% in 1999 versus 91% in 1993–1994 (P<0.0001) and MRI 28% versus 18% (P<0.0001). However, the increased use of imaging did not result in an increased ascertainment of milder stroke events, because the severity of stroke was very similar between study periods with identical median stroke severity scores of 4.0.

The prevalence of stroke risk factors and medication use was surveyed in the telephone survey, and there were 1855 respondents in 1995 and in 2111 in 2000. The two surveyed groups were similar in race (27% black in 1995 versus 26% in 2000) and gender (60% female versus 61%). The group was slightly older in 1995 (mean age, 63 years in 1995 versus 61 in 2000, P=0.002). See Table 5 for the self-reported medication use of these respondents. The prevalence of stroke risk factors over time was previously published,17,18 and we reported that diabetes and hypercholesterolemia rates significantly increased (diabetes 16% in 2000 versus 14% in 1995, hypercholesterolemia 38% versus 32%) but that hypertension and current smoking rates remained stable (hypertension 45% versus 44%, smoking 20% versus 21%) within our population. Medication use for treatment of stroke risk factors significantly increased over time, including antihypertensive agents, lipid-lowering agents, aspirin, and oral medications for diabetes. Insulin use did not significantly increase between study periods. Use of other medications such as anticoagulants and other antiplatelet agents was not collected in the survey.

### Table 1. Race-Specific Annual Incidence of First-Ever Stroke per 100 000 in 1993–1994 vs 1999, Inpatient Ascertainment Only

<table>
<thead>
<tr>
<th></th>
<th>1993–1994 (95% CI)</th>
<th>1999 (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All</td>
<td>Black</td>
</tr>
<tr>
<td>All stroke subtypes</td>
<td>158 (151–165)</td>
<td>238 (212–264)</td>
</tr>
<tr>
<td>Ischemic</td>
<td>139 (132–146)</td>
<td>199 (176–223)</td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>7 (5–8)</td>
<td>10 (5–15)</td>
</tr>
</tbody>
</table>

### Table 2. Race-Specific Annual Incidence of First-Ever Stroke per 100 000 in 1993–1994 vs 1999, Inpatient Plus Out-of-Hospital Ascertainment

<table>
<thead>
<tr>
<th></th>
<th>1993–1994 (95% CI)</th>
<th>1999 (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All</td>
<td>Black</td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>7 (6–9)</td>
<td>11 (5–16)</td>
</tr>
</tbody>
</table>

Age- and gender-adjusted to the 2000 US Census.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>1993–1994 (95% CI)</th>
<th>1999 (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–34</td>
<td>2.05 (1.49–2.62)</td>
<td>2.01 (1.50–2.52)</td>
</tr>
<tr>
<td>35–44</td>
<td>4.18 (3.96–4.41)</td>
<td>3.33 (3.12–3.54)</td>
</tr>
<tr>
<td>45–54</td>
<td>2.02 (1.90–2.15)</td>
<td>2.64 (2.53–2.76)</td>
</tr>
<tr>
<td>55–64</td>
<td>1.74 (1.65–1.84)</td>
<td>1.82 (1.73–1.91)</td>
</tr>
<tr>
<td>65–74</td>
<td>1.66 (1.60–1.72)</td>
<td>1.18 (1.11–1.25)</td>
</tr>
<tr>
<td>75–84</td>
<td>1.33 (1.28–1.38)</td>
<td>0.92 (0.86–0.97)</td>
</tr>
<tr>
<td>85+</td>
<td>1.11 (1.06–1.15)</td>
<td>0.77 (0.73–0.82)</td>
</tr>
</tbody>
</table>

A risk ratio of >1 implies greater risk for blacks than whites, and risk ratio <1 implies a greater risk for whites than blacks.

If 95% CI does not include 1, then the risk ratio is significantly different from 1 (P<0.05).

Discussion

Despite advances in stroke prevention strategies and treatments during the 1990s,20–26 the incidence of hospitalized stroke did not decrease within our large biracial population of 1.3 million. However, given the aging of our population, even with stable incidence rates of stroke, the total number of stroke events will continue to increase over time and will become an even larger burden on the United States.27 Our finding of stable stroke incidence in the United States is quite similar to the static stroke mortality rates seen in other studies the United States during the 1990s.5,12 Unfortunately, the racial disparity in stroke incidence is also not changing over time, with blacks continuing to have nearly twice the stroke incidence of whites. Previously, we reported that the greater stroke mortality seen in blacks is likely the result of greater stroke incidence, a finding that we confirm in this subsequent study within the same population.

Previous population-based studies in the literature have tracked stroke incidence over time with similar ascertainment methodologies such as in Rochester, Minn, and in Oxfordshire, UK.3,9 In Rochester, the incidence of stroke initially decreased during the 1970s but then stabilized and increased during the 1980s. Rothwell et al recently reported a nearly 40% decrease in stroke incidence between the 1980s and 2002 in Oxfordshire, UK. However, it should be noted that both of these studies were performed within more affluent white populations in smaller environments when compared with the GCNK population. Possible explanations for the differences in temporal trends during the 1990s between the GCNK region and Oxfordshire region may include varying prevalences in risk factors and treatment strategies for stroke prevention. Other population-based studies, including Anderson et al in New Zealand,28 used different ascertainment strategies over the study periods and make temporal trends difficult to interpret. Studies from Corpus Christi, Texas,29 and New York30 have analyzed the incidence of stroke in Hispanics, which we are unable to address within our population, and both reported a twofold greater stroke incidence in Hispanics compared with whites but have not yet reported temporal trends in stroke incidence within these same populations.

The lack of any significant change in the age-adjusted incidence rates of stroke in GCNKK region for whites and blacks during the 1990s is disappointing. This is especially true given the primary and secondary prevention efforts for cardiovascular disease within our community during the 1990s as well as the increased use of medications to modify stroke risk factors in the general population (including aspirin, antihypertensive and hypoglycemic agents, see Table 5). One possible explanation is a failure to translate primary prevention efforts into substantive decreases in the prevalence of key risk factors. In fact, within our community, we saw increasing prevalence of diabetes and reported hyperlipidemia.17 Whether this is the result of a true increase in prevalence, an increase in disease detection, or a combination of both is not clear. These findings are consistent with other large surveys of disease prevalence and treatment over time, including the National Health and Nutrition Examination Surveys in 1990 and 2000.31–34

The lack of change in stroke incidence could also be related to the medical control of stroke risk factors, which may still


<table>
<thead>
<tr>
<th>Stroke Subtype</th>
<th>1993–1994 (95% CI)</th>
<th>1999 (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All†</td>
<td>13.9%</td>
<td>14.7%</td>
</tr>
<tr>
<td>Black*</td>
<td>13.1%</td>
<td>12.8%</td>
</tr>
<tr>
<td>White*</td>
<td>14.8%</td>
<td>16.9%</td>
</tr>
<tr>
<td>Ischemic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black*</td>
<td>9.4%</td>
<td>10.2%</td>
</tr>
<tr>
<td>White*</td>
<td>7.8%</td>
<td>9.1%</td>
</tr>
<tr>
<td>Intracerebral hemorrhage</td>
<td>34.0%</td>
<td>37.6%</td>
</tr>
<tr>
<td>Black*</td>
<td>35.1%</td>
<td>36.2%</td>
</tr>
<tr>
<td>White*</td>
<td>33.0%</td>
<td>39.0%</td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>37.2%</td>
<td>31.3%</td>
</tr>
<tr>
<td>Black*</td>
<td>37.3%</td>
<td>28.2%</td>
</tr>
<tr>
<td>White*</td>
<td>37.1%</td>
<td>34.7%</td>
</tr>
</tbody>
</table>

*Adjusted for age and gender.
†Adjusted for age and gender and race.


<table>
<thead>
<tr>
<th>Medication</th>
<th>1995 Survey (n=1855)</th>
<th>2000 Survey (n=2111)</th>
<th>P Over Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antihypertensive medication</td>
<td>34.5%</td>
<td>39.7%</td>
<td>0.0008</td>
</tr>
<tr>
<td>Insulin medication for diabetes</td>
<td>4.0%</td>
<td>4.2%</td>
<td>0.77</td>
</tr>
<tr>
<td>Oral medication for diabetes</td>
<td>6.9%</td>
<td>10.8%</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Lipid-lowering agents</td>
<td>9.9%</td>
<td>20.1%</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Aspirin use</td>
<td>25.9%</td>
<td>35.6%</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>
be suboptimal even if these conditions are diagnosed and treated more frequently. We are unable to address this question, because we do not have data regarding the quality of care or medication compliance from our self-reported telephone survey. Another possible explanation is that the time elapsed between study periods was too short to be able to detect long-term decreases in stroke incidence secondary to improved treatment of stroke risk factors. Five years is a relatively short time interval to see changes in primary prevention of a chronic disease, although some of the advances of the 1990s such as warfarin use for atrial fibrillation, and carotid endarterectomy after TIA might be expected to have a more rapid effect than other behavioral changes such as diet, exercise, or hypertension control.

The potential for bias of incomplete case ascertainment is important to consider in any study that examines temporal trends in the incidence rates of a disease. Our use of passive surveillance of emergency rooms, nursing homes, physician offices, and clinics may reduce the chances of incomplete ascertainment. However, the random sampling of offices, nursing homes, etc., assumes a uniform distribution of strokes by region; this, of course, may not be the case, particularly because differences by race may impact the assumption of uniformity. For example, we suspect that the slight increase in our out-of-hospital stroke incidence in 1999 may reflect improved case ascertainment as a result of better billing record information in physician offices. However, our methodology for identification of hospitalized strokes was consistent between the two study periods, and the overall incidence rates of hospitalized stroke in the two study periods were nearly identical. Changes in clinical practice such as the increased frequency of MRI imaging may also affect the detection and diagnosis of stroke. However, our case definition for stroke was based on the presence and duration of focal clinical symptoms in both study periods, not on imaging findings. This is confirmed by the very similar stroke severity and case-fatality between study periods. Therefore, we believe that our consistent methodology and clinical case definition over the two study periods has minimized possible ascertainment biases for hospitalized strokes. In addition, any incidence study that relies on medical contact for counting of events risks missing events that were not recognized by the general public as needing medical attention. All of these considerations mean that several counterbalancing biases may influence the final incidence rates of stroke that we observed.

Clearly, there is still much work to be done to understand the temporal trends and racial disparities in stroke incidence. Population-based studies of temporal trends in stroke incidence rates are critical for providing a report card of our overall progress in primary stroke prevention as well as our efforts to shrink the continuing large disparity in incidence rates between white and black populations in the United States.

Disclosures

None.

References


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