Some Epidemiological Aspects of Stroke: Mortality/Morbidity Trends, Age, Sex, Race, Socioeconomic Status

Baruch Modan, MD, and Diane K. Wagener, PhD

Background and Purpose: The reasons for decreasing stroke mortality could be related to either decreasing incidence and/or improved prognosis. Thus far, secular trends of stroke have been analyzed either through mortality or morbidity data. This report examines both aspects simultaneously, on a nationwide basis, for the period 1968–1988.

Methods: Mortality statistics were based on the Compressed Mortality File. Estimates of morbidity were based on the National Hospital Discharge Survey. The Area Resource File was used to obtain county-specific socioeconomic statistics.

Results: The decline in stroke mortality continued through the 1970s and 1980s, whereas morbidity remained constant and possibly even increased. Mortality and morbidity rates were similar in both sexes, higher in blacks, and lower in other (primarily Asian) Americans. There was an inverse correlation between death rates and socioeconomic status, which was particularly marked in blacks. Temporal decline occurred in all strata.

Conclusions: The observed decrease in stroke mortality rates results most probably from an improved survival rather than from a decline in incidence. The abundance of new drugs and screening programs may not have affected the overall morbidity of stroke, possibly because of inefficient treatment regimens. (Stroke 1992;23:1230–1236)

KEY WORDS • cerebrovascular disorders • epidemiology • morbidity • mortality

The rapid decline in total mortality in the United States and other western countries may be ascribed primarily to two entities: ischemic heart disease and stroke. Many investigators relate the decline in cardiovascular mortality to decreased incidence resulting from health promotion efforts aimed at changing lifestyle, particularly cessation of smoking, prudent diet, and early treatment of hypertension. However, improved survival and/or a change in diagnostic machinery and the coding practice of specific disease categories must also be considered as contributory factors.

This article attempts to delineate cause and effect in secular trends of stroke by simultaneous assessment of national mortality and morbidity data.

Subjects and Methods

United States mortality statistics, based on death certificate information available from the National Center for Health Statistics' (NCHS) Compressed Mortality File for all 50 states and the District of Columbia, were used for the period 1968–1988. This is the most recent data year currently available. Age, sex, race, and International Classification of Diseases (ICD)-specific death rates were calculated. Population numbers were estimated for the intercensal years. Data on race are only available as whites versus nonwhites for 1968–1978. For the socioeconomic analyses (see below) of the pericensal years, 1969–1972, the black population numbers were estimated using the 1970 census data. Death records of the black population were determined from the detailed mortality files. Age-adjustment was computed using the direct method to 1980 US population age distribution.

Because of the shift from ICD version 8 to ICD version 9 in the midst of the study period, different ICD categories had to be selected. For the earlier period (1968–1978), cerebrovascular accident, i.e., stroke, codes selected included the following: 430.0, 430.9, 431.0, 431.9, 432.0, 432.9, 433.0, 433.9, 434.0, 434.9, 435.0, 435.9, 436.0, 436.9, 437.0, 437.9, 438.0, 438.9. For the later period (1979–1988), the selected codes included: 430, 431, 432.0, 432.1, 432.9, 433.0, 434.1, 434.9, 435, 436, 437.0, 437.1, 437.2, 437.3, 437.4, 437.5, 437.6, 437.8, 437.9, 438. Separate calculations were performed for hemorrhagic stroke (430–431 in ICD-8 and 430–432 in ICD-9), for thromboembolism (432–434 in ICD-8 and 433–434 in ICD-9), and ill-defined stroke (435–438 in ICD-8; 435–437 in ICD-9).

Estimates of morbidity were based on the NCHS National Hospital Discharge Survey. These data have one major drawback: multiple admissions cannot be linked and therefore yield overestimates of the incidence. However, no chronic or rehabilitative hospitals are included in the survey. To further limit this bias, we...

FIGURE 2. Graph showing age-adjusted death rates (to 1980 US age distribution) from cerebrovascular accidents in the United States between 1968 and 1988, by pathological subentity. ICD-8 and ICD-9, International Classification of Diseases versions 8 and 9, respectively.
rates of death due to thromboembolic disease continued to decline (Figure 2). Furthermore, despite continuous efforts for a more concrete categorization of specific stroke entities, the "ill-defined" category, which includes the three general categories of "transient cerebral ischemia," "acute but ill-defined disease," and "other ill-defined cerebral vascular disease," still comprises close to 60% of all stroke mortality. The proportion of all stroke mortality that has been classified as ill-defined has, in fact, increased from 49.3% in 1968 to 62.8% in 1988. The trends were similar in both sexes, as well as in whites and nonwhites.

This detailed classification follows the information coded from the death certificates. It must be taken into consideration that before and during the early years after the introduction of computed tomography, the differentiation between hemorrhagic and occlusive cerebrovascular disorders was primarily inferential. In the later period, the use of this diagnostic tool has become more frequent; therefore, the clinical diagnosis of hemorrhagic disorder may be more reliable. However, computed tomography is still not performed in a large proportion of cases, as evidenced by the large proportion of ill-defined stroke.

Throughout the 21-year period, the age-specific death rates for total stroke were higher in nonwhites compared with whites aged <75 years (Table 1 gives the rates for selected years). This risk ratio declined with age. Thus, among the population aged 25–34 years, the ratio of death rates between nonwhites:whites was approximately 3:1; among the population aged 75–84 years the rates were equal; and among the population aged ≥85 years it reversed to 0.84:1. These ratios have not changed over the 21-year period (Figure 3).

Figures 4 and 5 examine the patterns of change within the context of socioeconomic status (SES), using two measures: median family income and percentage of adults who had completed high school. For the total population there is a clear pattern of inverse correlation between stroke mortality among individuals aged ≥25 years and each SES measure (for trend, p<0.001), with death rates in the lowest SES quintile being approximately 10–30% higher than in the highest quintile for income and 25–40% higher between quintiles of education. This gradient was practically identical in both sexes but was particularly strong in the black population (Figure 5), where the rates in the lowest SES-income quintile were about 50% higher than in the uppermost quintile for all three time periods, compared with a ratio of only about 1.3 in 1969–1972 and 1.07 in the two later periods for whites. In this context, the range of median income between quintiles was greater among nonwhites (2.7-fold versus 1.7-fold, respectively, in 1969).

The "nonwhite, nonblack" population is a heterogeneous group, consisting predominantly of Asian-Americans. The death rates for the "other" group were much lower than either white or black rates for all SES quintiles. For instance, in 1987–1988 the rates for the lowest quintile of median income through the highest quintile were 65.2 per 100,000, 54.5, 50.4, 54.7, and 70.0, respectively. This is in contrast to the death rates among whites of 96.8, 102.4, 94.2, 97.7, and 90.9 and among blacks of 166.4, 148.0, 137.6, 122.7, and 116.4. There was no clear association of SES quintiles and mortality for the "other" group.

Morbidity data show a different trend. Total stroke morbidity shows practically no decline over the 20-year study period, even when the analysis is limited to new...
TABLE 1. Age-Specific Mortality From Stroke at Three Selected Time Points Throughout the Period 1968–1988, by Age and Race

<table>
<thead>
<tr>
<th>Year/Age group (age in years)</th>
<th>Whites (per 100,000)</th>
<th>Nonwhites (per 100,000)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1968</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>105.08</td>
<td>112.12</td>
</tr>
<tr>
<td>&lt;5</td>
<td>1.42</td>
<td>2.76</td>
</tr>
<tr>
<td>5–14</td>
<td>0.58</td>
<td>0.71</td>
</tr>
<tr>
<td>15–24</td>
<td>1.25</td>
<td>2.31</td>
</tr>
<tr>
<td>25–34</td>
<td>2.98</td>
<td>9.71</td>
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<tr>
<td>35–44</td>
<td>10.13</td>
<td>37.10</td>
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<tr>
<td>45–54</td>
<td>29.24</td>
<td>99.02</td>
</tr>
<tr>
<td>55–64</td>
<td>85.31</td>
<td>235.98</td>
</tr>
<tr>
<td>65–74</td>
<td>306.13</td>
<td>584.84</td>
</tr>
<tr>
<td>75–84</td>
<td>1112.29</td>
<td>1116.92</td>
</tr>
<tr>
<td>≥85</td>
<td>3029.10</td>
<td>2176.05</td>
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</table>

1978

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<th>Year/Age group (age in years)</th>
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<th>Nonwhites (per 100,000)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>80.15</td>
<td>72.24</td>
</tr>
<tr>
<td>&lt;5</td>
<td>1.45</td>
<td>3.09</td>
</tr>
<tr>
<td>5–14</td>
<td>0.52</td>
<td>0.67</td>
</tr>
<tr>
<td>15–24</td>
<td>1.05</td>
<td>1.50</td>
</tr>
<tr>
<td>25–34</td>
<td>2.03</td>
<td>6.90</td>
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<tr>
<td>35–44</td>
<td>7.68</td>
<td>24.01</td>
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<td>45–54</td>
<td>22.04</td>
<td>68.74</td>
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<td>55–64</td>
<td>62.30</td>
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<tr>
<td>65–74</td>
<td>223.80</td>
<td>418.65</td>
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<tr>
<td>75–84</td>
<td>843.50</td>
<td>913.58</td>
</tr>
<tr>
<td>≥85</td>
<td>2447.34</td>
<td>1903.28</td>
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1988

<table>
<thead>
<tr>
<th>Year/Age group (age in years)</th>
<th>Whites (per 100,000)</th>
<th>Nonwhites (per 100,000)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>62.75</td>
<td>53.03</td>
</tr>
<tr>
<td>&lt;5</td>
<td>0.89</td>
<td>2.03</td>
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<td>5–14</td>
<td>0.20</td>
<td>0.25</td>
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<tr>
<td>15–24</td>
<td>0.68</td>
<td>0.86</td>
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<td>25–34</td>
<td>1.67</td>
<td>5.04</td>
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<td>65–74</td>
<td>142.34</td>
<td>258.98</td>
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<tr>
<td>75–84</td>
<td>542.11</td>
<td>666.68</td>
</tr>
<tr>
<td>≥85</td>
<td>1739.44</td>
<td>1390.08</td>
</tr>
</tbody>
</table>

Discussion

By study of national trends of stroke in the United States, we have shown that the decline in stroke mortality continued, whereas morbidity at best remained constant and possibly even increased. In other words, most probably the observed decrease in stroke mortality rates results from an improved survival rather than from a decline in morbidity. This contention is in line with some indications that for ischemic heart disease, the decline in mortality also may be due mainly to a better prognosis.8

Another interesting facet resulting from the above observations is that the ratio of hospitalized events to mortality increased from approximately 1.6 in 1968 to 5.2 currently, leading to an increased burden of disability. Consequently, it may be expected that the economic load of stroke on our society will increase even further in the future.9

Before embarking on such a far-reaching conclusion, certain questions must be answered. First, is the observed morbidity trend genuine? Second, to what degree do the hospitalization discharge data reflect true incidence? And finally, considering the above-mentioned constraints, what are the implications of the observed morbidity trend over the study period?
Figure 6 demonstrates that the morbidity rates were quite stable until 1978. Then they jumped by approximately 10%, continued to increase gradually through 1985, and decreased sharply by 1988. There was no overall increase in hospitalizations over the parallel period; in fact, both total hospitalizations and those restricted to those aged ≥65 declined after 1979.

There are a number of possibilities that could explain these phenomena. These include changes in ICD classification, increased stroke prevalence, new diagnostic technology such as computed tomography or magnetic resonance imaging, and changes in the payment system. The fact that the increase started abruptly in 1979, when ICD changed from the eighth to the ninth version, suggests that this may be a major underlying factor. Indeed, the “conversion factor” from ICD-8 to ICD-9, for the 430-438 categories, has a comparability ratio of 0.9318 (±3.4%). This means that recording of 1979 records with a 430-438 category diagnosis by the ICD-8 nomenclature (rather than the ICD-9 version) would have decreased the rate by 6.82%. This would still be compatible with the ratio of 1978:1979 hospitalization rates (297.2/334.4) obtained by us.

The dip in morbidity after 1987 could be ascribed to a change in sampling frame taken by NCHS in 1988. The last such change was made before 1970. The further increase during the 1980s could possibly, but not necessarily, be ascribed to the introduction of the Diagnostic Related Groups payment system.

Two other factors to consider are the dramatic increase in instruments of diagnostic technology and almost a fourfold increase in the number of board-certified neurologists over the respective time period. It is possible that these changes could affect morbidity trends only.

As noted above, the National Hospital Discharge Survey counts admissions, not persons. Because the data are obtained without personal identification, one cannot collate several admissions belonging to the same patient. Therefore, the extent to which the discharge summary data reflect incidence depends on the number of recurrent stroke attacks and the patients’ survival. Evidently with an improved prognosis, the overall prevalence of patients with old stroke will increase. Part of the bias was corrected by limiting our data to first-listed diagnosis. The hospitalization rate estimated using the full listing was about twice as high. Since secondary stroke events in the same calendar year are rare, the hospitalization rate may approximate overall increase in hospitalizations over the parallel period; in fact, both total hospitalizations and those restricted to those aged ≥65 declined after 1979.

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On the other hand, some of the stroke events represent recurrence in patients stricken years ago. Therefore, it is possible that a potential trend of decreased incidence is superimposed by a large number of repeated stroke events.

Our knowledge of the epidemiology of stroke is based mainly on analysis of secular trends and follow-up studies of defined populations exposed to known risk factors. Although there is little doubt that hypertension constitutes a major risk factor for stroke development, the magnitude of effective antihypertensive treatment in eliminating the threat of stroke is still questioned. It is also plausible that, thanks to either preventive treatment or another as-yet-undefined risk factor, current attacks of stroke are milder than those that occurred in the past.

In this context, it is of interest to note that Klag et al failed to find a correlation between the observed decrease in stroke mortality rates in the United States and the extent of antihypertensive treatment. Their findings are in marked contrast to the widely accepted notion that the decline of stroke mortality accelerated in the mid-1960s, simultaneously with the introduction of new, more effective antihypertensive therapy.

Broderick et al, who studied the incidence of stroke in Rochester, Minn., showed a decline in incidence that started in 1950 and stabilized in the 1970s. Their findings are thus supportive of our nationwide analysis, despite the fact that we were hampered by the lack of true incidence data. Kuller, who reviewed the Rochester data, considered several factors that could be operative in producing the stabilization, other than a change in diagnosis or better case ascertainment. These included an increased prevalence of hypertension, a less effective treatment regimen, decline in ischemic heart disease mortality, and increased complications of cardiovascular surgery. None of these seemed to Kuller to be strong enough to substantiate an increase that would counterbalance the previous trend of decline in incidence.

The notion that stroke has been controlled by screening and early treatment of hypertension is also not borne out by our findings. There is indeed a better survival in stroke patients, possibly due to earlier ambulation and better medical and supportive treatment. However, our data suggest that the abundance of new drugs and screening programs has not affected the overall morbidity of this disease, possibly because of inefficient treatment regimens. Early treat-
ment may be more effective in reducing mortality, but long-term measures appear less effective in reducing morbidity.

One other way to approach the problem of stable stroke morbidity is to assume that despite the fact that hypertension constitutes a major risk factor for stroke, it is only one component in the complex array of risk factors for this condition. Other risk factors to be considered are clotting factor abnormalities, lipid metabolism, or smoking. To further study this hypothesis, we need better data on the internal distribution of stroke entities. Current information indicates different patterns of the hemorrhagic and thromboembolic stroke categories.37-39 By the same token, mortality due to subarachnoid hemorrhage has remained constant, possibly because of a combination of increased incidence and lower case fatality.40 As long as >50% of the cases continue to be coded under the very general ill-defined stroke category, this problem will be hard to solve.

The fact that stroke incidence and mortality are higher in blacks than in any other ethnic group in the United States is well known.41 It is of interest, however, that the socioeconomic gradient in stroke mortality is also highest in the black population, suggesting that the disease is related to a strong environmental or social factor amenable to change, e.g., better treatment for hypertension, rather than to a genetic component.

The rates of the balance of the nonwhite population are more puzzling. First, this subgroup presents no inverse gradient between mortality and socioeconomic status but rather a U-shaped rate pattern. Further, the mortality rates were up to 50% lower than the rates in the black US population and 30–40% lower than in the US white population. Such low rates are incompatible with the findings in the original Asian populations, which are among the highest in the world.37-39 One could anticipate that stroke rates of Japanese and Chinese subjects in the United States would be intermediate between the rates of their parental population and the absorbing US population; it may even be plausible that they would be similar to the rates of the US population, but it does not seem reasonable that they would be lower. On the other hand, data from Los Angeles42 show rates and ethnic differentials in the same order of magnitude. One possible explanation for this discrepancy is that the way race is recorded on the death certificate and on the census may be different. This methodological inconsistency results in either inflation of the denominator or a lower race-specific reporting on the death certificate.

The interracial and socioeconomic differentials provide some potential clues for future forecasts. The inverse relation between mortality rates and socioeconomic level is consistent with observations of Kitagawa and Hauser43 using 1960 data and geographic analyses of Wing et al44 indicating a higher mortality in the southern United States. The steeper decline across SES standards of living in the United States and elsewhere in poverty. If so, the projected overall improvement in standard of living in the United States and elsewhere may propagate further reduction in mortality, in line with the dramatic decline in mortality from gastric45 and cervical46 cancer. Since such trends are integrally re-

lated to lower exposure to risk factors, we may expect a decline in the incidence of cerebrovascular accidents as well.

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