Decline in US Stroke Mortality
Demographic Trends and Antihypertensive Treatment

Michael J. Klag, MD, MPH, Paul K. Whelton, MD, MSc, and Alexander J. Seidler, PhD

Stroke mortality has been falling rapidly in this country since 1973. To investigate age-race-sex effects on stroke mortality, we studied US vital statistics during 1950–1972 and 1973–1981 in 55–64-, 65–74-, and 75–84-year-old race-sex groups. The accelerated rate of decline in stroke mortality since 1973 has had a substantial public health impact, with >200,000 fewer stroke deaths than would otherwise have occurred. For all groups, stroke mortality declined at a greater rate (p<0.05) in 1973–1981 than during 1950–1972. The rates of decline during 1973–1981 were greater with increasing age (p<0.05) and were more substantial for younger blacks. There were no consistent differences in the rate of decline by sex. The greater rate of decline in absolute stroke mortality in the older age groups and blacks was explained by higher baseline mortality in these groups. Overall, stroke mortality decreased by approximately 2%/yr in 1950–1972 and by approximately 7%/yr after 1973. Rank order of average annual percent decline after 1973 by age-race-sex groups did not correspond to rates of change in treatment or control of hypertension obtained from three national surveys. The accelerated rate of decline after 1973 may have resulted from improved antihypertensive therapy, but our findings fail to confirm this hypothesis and suggest that treatment of hypertension may not be the principal reason for the decline in stroke mortality. (Stroke 1989;20:14–21)

Age-adjusted stroke mortality has been declining in the United States since the turn of the century, at an accelerated rate after 1973.1,2 This recent downturn in mortality has been seen in most Western nations3 and has been attributed to improved treatment and control of hypertension.2,4-7 Despite these favorable trends, stroke remains the third leading cause of death and stroke-associated morbidity and mortality are important public health problems.4 The notion that the accelerated decline in stroke mortality has resulted from increased use of antihypertensive drug therapy in the general community is certainly plausible. However, little evidence has been generated to document the validity of this putative association.

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In three US national surveys performed during the period of accelerated decline in stroke mortality, consistent techniques have been used to gather information regarding the prevalence of treatment and control of hypertension in the general community.9 With this in mind, we decided to explore the public health implications of the recent accelerated decline in US stroke mortality and to identify the relation between the decline and national trends in the prevalence of treatment and control of hypertension. Specifically, we set out to 1) estimate the reduction in deaths attributable to the accelerated decline, 2) determine how the accelerated decline varied by age, race, and sex, and 3) determine if the prevalence of hypertension therapy among various age-race-sex groups was associated with the decline in the rate of stroke mortality.

Subjects and Methods
Race- and sex-specific stroke mortality for 1950–1981 was obtained for the 55–64-, 65–74-, and 75–84-year-old age groups from US vital statistics data.10 Because mortality for blacks was not available for the entire study period, death rates for nonwhites were used. To provide a period during which the prevalence of antihypertensive drug therapy varied considerably, we studied temporal trends in stroke mortality between 1950 and 1981. Thiazide diuretics were introduced in 1958, but antihyperten-
The accelerated decline in stroke mortality had a substantial impact on the number of stroke deaths that occurred during 1973–1981 (Table 1). More than 200,000 fewer deaths were observed than would have been expected if the accelerated decline in stroke mortality had not transpired. The majority of these deaths would have occurred in the oldest age group, the 75–84-year-olds.

Age-specific stroke mortality during 1950–1982 is illustrated in Figure 2; since the patterns were the same within each age for all race-sex groups, only the age-specific results are shown. Stroke mortality declined throughout the study period, but after 1973 an acceleration in the rate of decline was consis-
tently seen in all 12 age-race-sex groups. For each group, the rate of decline in mortality was significantly greater after than before 1973. This pattern is not obvious in Figure 2 in the younger age groups because of the large differences in baseline mortality. Figure 3 presents the estimates and 95% confidence intervals of the rates of decline in stroke mortality by 12 age-race-sex groups for 1973–1981. Within each race-sex group, the rates of decline increased with age. The lowest rate of decline, 3.5 deaths/100,000/yr, was seen in 55–64-year-old white women and the greatest, 69.7 deaths/100,000/yr, was seen in 75–84-year-old white men. Except in the 75–84-year-olds, blacks had greater rates of decline than whites. However, there were no consistent differences by sex.

Figure 4 shows age-specific transformed stroke mortality for 1950–1981. The average annual percent decline, like the absolute decline, was consistently greater after 1973 than before in each age-race-sex group. However, transformation accentuated the negative slope of the curves after 1973 in the younger age groups and increased the similarity in slopes among the three age groups. Figure 5 diagrams the average annual percent decline and 95% confidence intervals for 1973–1981 for each age-race-sex group. Overall, stroke mortality during 1973–1981 fell approximately 7%/yr compared with approximately 2%/yr during 1950–1972. Within each race-sex group, the average annual percent decline in stroke mortality during 1973–1981 did not differ significantly among any age groups except the 75–84-year-old black men and women. The average annual percent decline in stroke mortality was significantly less in these two groups than in corresponding race-sex groups of different ages, but since multiple comparisons were made, this difference may be due to chance alone. There was a great deal of year-to-year variability in stroke mortality for the oldest blacks, although the sharp decline
after 1973 was still present. The oldest blacks also differed from other groups in that they had lower stroke mortality than whites. This reversal of mortality in older age groups, whites greater than blacks, has been seen in the vital statistics of a variety of diseases. Lower coronary heart disease mortality in older blacks than in older whites has been confirmed in longitudinal observational studies, suggesting that it may represent selection of a hardy population of blacks. In summary, the age- and race-related differences in the rate of decline in stroke mortality were generally not present after adjustment for the marked differences in magnitude of stroke mortality and can be attributed to the much higher stroke mortality in blacks and older persons.

To explore further the factors influencing the rate of decline in stroke mortality during 1973–1981, we performed a linear regression, using the rate of decline in stroke mortality per calendar year as the outcome variable. The midpoint of the age groups, 1973 stroke mortality rates, and dummy variables for race and sex were entered into the equation singly and together. Only age (p<0.0001, R²=0.81) and 1973 stroke mortality (p<0.0001, R²=0.91) were significantly associated with the rate of decline by simple linear regression. When age and 1973 stroke mortality were entered simultaneously into a multivariate model, only 1973 stroke mortality remained significant (p<0.02, R²=0.91). This analysis confirms the results obtained by comparing the 95% confidence intervals of the absolute and relative rates of decline (Figures 3 and 4).

The proportion of hypertensives treated and hypertensives controlled over time are listed in Table 2; the rank order of the average annual
TABLE 2. Rank Order of Average Annual Percent Decline in Stroke Mortality and Trends in Treatment and Control of Hypertension by Age, Race, and Sex

<table>
<thead>
<tr>
<th>Age-race-sex groups</th>
<th>Treatment of hypertension</th>
<th>Control of hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Average annual % decline in stroke mortality (1973-1981)</td>
<td>Change in hypertension treatment (1960-1980)</td>
</tr>
<tr>
<td></td>
<td>Rank order</td>
<td>% hypertensives treated</td>
</tr>
<tr>
<td>55–64 years White males</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>White females</td>
<td>8</td>
<td>6</td>
</tr>
<tr>
<td>Black males</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Black females</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>65–74 years White males</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>White females</td>
<td>7</td>
<td>4</td>
</tr>
<tr>
<td>Black males</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>Black females</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

percent decline in stroke mortality during 1973–1981 is also shown. There was no correlation between the rank order of the age-race-sex groups by either measure of change in antihypertensive therapy and the rank of the average annual percent decline in stroke mortality (both p>0.1). The rank order of the annual percent decline in stroke mortality was then tested against the proportion of hypertensives treated and hypertensives controlled from the most recent survey (NHANES-II), and again no correlation was found (both p>0.1).

Discussion

Our analysis documents the profound benefit that the recent period of accelerated decline in stroke mortality has had on the health of the nation. More than 200,000 fatal strokes have been prevented from 1973 to 1981. Presumably, these trends in stroke mortality reflect patterns of stroke incidence. Assuming that only approximately one third of incident strokes are fatal, the considerable morbidity associated with approximately 400,000 nonfatal strokes has also been prevented.

There are several problems with studies of the type we describe. An obvious question is the validity of vital statistics stroke mortality reports. This concern is mitigated by information from Olmstead County, a well-studied population with uniform methods of case ascertainment and coding over time, which tends to confirm the national vital statistics results. Olmstead County stroke mortality has been somewhat lower and the decline steeper than in the entire US population, but the pattern of declining stroke mortality has been very similar through 1979. We did not examine type-specific stroke mortality because of concern regarding the validity of such information when based on death certificates. One would expect, however, a greater decline in lacunar stroke mortality and intracerebral hemorrhage mortality than in thrombotic stroke mortality if the decline has resulted primarily from the increased prevalence of treatment of hypertension. This has been suggested to be the case. One of the limitations of our ecologic analysis of the relation between changes in treatment of hypertension and rates of decline in stroke mortality is that it does not take into account the potentially confounding influence of other risk factors for stroke mortality. In addition, a possible lag between change in treatment of hypertension and stroke mortality could not be investigated because we did not have data on antihypertensive therapy for every year of the study. The absence of data on the prevalence of treatment and control of hypertension in persons older than 74 years of age is another limitation of our analysis. However, since the average annual percent decline in stroke mortality between 1973 and 1981 was the same in all three age groups, it is
unlikely that the availability of such data would have altered our results. A strength of our analysis is that the data on treatment and control of hypertension are from national probability surveys that used similar methodology and that are representative of the US population.

Any negative study must address the issue of statistical power. Although there are only eight "observations" in Table 2, each observation represents a very large sample size: millions for the stroke mortality data and thousands for the hypertension treatment data. Thus, the observations are very stable estimates. In addition, if the lack of an association between rates of decline in stroke mortality and treatment and control of hypertension is due solely to the limited power of the statistical test, one would still expect to see a trend in the direction of the true association. From inspection of Table 2, there is no suggestion of such a trend.

The sheer magnitude of the decline in stroke mortality prior to 1950 almost certainly reflects a true decrease in stroke incidence, but the mechanism for this decline is uncertain. Antihypertensive therapy certainly cannot explain the decline during this early period. In terms of the recent accelerated decline, data from the Mayo Clinic show a reduction in both the incidence and the case-fatality rate of stroke between 1945 and 1975. Improved care of comorbidities, as well as a change in case mix, have been suggested as reasons for the improvement in the case-fatality rate. While antihypertensive therapy probably has little effect on case fatality, thus, one possible reason for our finding a lack of association between antihypertensive therapy and rates of decline in stroke mortality would be if a large portion of the decline in stroke mortality were due to decreased case fatality. However, the majority of the decline in stroke mortality can probably be explained by the decrease in stroke incidence.

Antihypertensive therapy seems a likely candidate to explain the accelerated decline since 1973 for several reasons. The National High Blood Pressure Education Program was instituted in 1973, coincident with the beginning of the period of rapid decline in stroke mortality and marked increase in the prevalence of treatment and control of hypertension. Clinical trials have demonstrated the efficacy of antihypertensive therapy in preventing approximately 40% of strokes within a relatively brief time after starting therapy. Community studies suggest that the long-term benefit of antihypertensive therapy may be even greater. In addition, a temporal concordance between rates of hypertension control and stroke mortality has been noted for both male and female residents of North Karelia and Kuopio County, Finland.

Our analyses do not support an association between trends in treatment and control of hypertension and rates of decline in stroke mortality, primarily because the rates of decline did not vary much after adjustment for age-related differences in the magnitude of stroke mortality. Thus, the proportionate rates of decline were approximately equal among all age-race-sex groups. It may be that very low rates of treatment and control of hypertension in the community are enough to cause stroke mortality to decline if those subjects at the highest risk (i.e., severe hypertensives) are preferentially treated. Population-attributable risk calculations, however, show that the majority of strokes occur in normotensives and in borderline hypertensives. In addition, other investigators have demonstrated that only approximately 10% of the reduction in stroke mortality in New Zealand can be attributed to treatment of hypertension.

What, then, can explain the decline in stroke mortality in the last decade? The remarkable consistency of the change in slope in each age-race-sex group in 1973 suggests some widespread environmental agent. Although hypertension is the main risk factor for stroke, the other major risk factors (besides age and sex) are preexisting coronary heart disease, left ventricular hypertrophy, cigarette smoking, elevated hematocrit, oral contraceptive use, and diabetes mellitus. Coronary heart disease mortality has been decreasing since 1968, and the lower prevalence of coronary heart disease may explain some of the decline in stroke mortality. Several studies have demonstrated an increased risk of stroke in cigarette smokers, and in one study the risk of stroke in persons who are both smokers and treated hypertensives is about 19-fold greater than that in persons without these characteristics. In fact, the population-attributable risk of stroke for cigarette smokers was higher than for hypertensives. In addition, persons who stop smoking have a lower risk of stroke than persons who continue to smoke. From 1965 to 1980, the prevalence of exsmokers in the United States has risen in males and females, whites and blacks, in those aged 45-64 and in those older than 64 years. Hypertensives have been targeted for smoking cessation, and it is conceivable that smoking cessation in either hypertensives or the general population could have accounted for some of the large decline in stroke mortality beginning in 1973. Changes in the formulation or use of oral contraceptives are unlikely to have played a role in the recent decline in stroke mortality since the decline was seen in both men and women. Improvements in the treatment of diabetes mellitus may have helped to reduce stroke mortality, but no data are available.

Alcohol consumption has also been shown to be a risk factor for stroke. Although age-race-sex-specific data are not available, per capita consumption of alcohol increased during 1950-1981. Thus, it is unlikely that changes in alcohol consumption contributed to the current decline in stroke mortality. More recently, a low intake of potassium has
been suggested to be an independent risk factor for stroke.\textsuperscript{31} These results need confirmation,\textsuperscript{32} but stroke mortality in Great Britain has been shown to vary with ingestion of fresh fruit and vegetables.\textsuperscript{33} This effect was attributed to vitamin C intake but may be due, instead, to the potassium content of those foods. Whether changes in the consumption of potassium or other nutrients in the US population can explain the rapid acceleration in the decline of stroke mortality is unknown.

Another hypothesis is that these trends in stroke mortality may be linked to the decreased lead exposure that occurred in 1973–1980 in the US population.\textsuperscript{34} Lead exposure has been linked to hypertension\textsuperscript{35} and, in at least one occupational study, to increased stroke incidence.\textsuperscript{36}

The accelerated decline in stroke mortality after 1973 may have resulted from improved antihypertensive therapy, but our findings fail to confirm this hypothesis. Further research to delineate the causes for this decline is important in that it may suggest interventions for individual patients as well as for those nations that have not experienced such a decline in stroke mortality.

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References


8. 1987 \textit{Stroke Facts}. Dallas, American Heart Assoc, 1986


34. Roberts J, Mahaffey KR, Annest JL: Blood lead levels in general populations, in Mahaffey KR (ed): \textit{Dietary and


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