CVD Mortality, 1968–1978: Observations and Implications

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SUMMARY U.S. multiple cause of death data were examined for the period 1968–1978. Specifically, the role of cerebrovascular disease mortality as an underlying and associated cause of death was studied. The number of deaths where cerebrovascular disease was cited as the underlying cause of death declined much faster than the number of deaths where cerebrovascular disease was cited as the associated cause of death. This trend is indicative of a possible change in the role of cerebrovascular disease as an associated cause of death. Cause elimination life tables were constructed for cerebrovascular disease as the underlying cause of death and as any cause of death. In the general population, eliminating stroke as a cause of death is projected to have less impact in 1978 than in 1968, for men than for women, and for whites than for nonwhites. Tables were also constructed to examine the life expectancy gains for the group of individuals who died of cerebrovascular disease. For these individuals, the gain in life expectancy at birth ranges from 9 years for white males to 18 years for nonwhite females.

In order to comprehend mortality trends it is necessary to understand the standard terms used on a death certificate and how this information is coded. Causes of death are cited on two parts of the certificate. Part I on the death certificate is designed to be used by the physician to list the morbid conditions which define the causal sequence leading to death. Usually, but not always, the underlying cause of death will be selected as the condition on the "lowest used line" of Part I. Part II of the death certificate is to be used by the physician to list any other significant medical conditions which contributed to death but which were not part of the causal chain indicated in Part I.

In standard statistical computations all of the medical information on Parts I and II is summarized in the selection of a single condition to represent the underlying cause of death. The underlying cause of death is defined to be the cause which began a morbid process that lead to death. In this paper, any condition which is not the underlying cause of death is referred to as an "associated" cause of death. A condition which is an underlying or associated cause of death is referred to as "any" cause of death. Using the medical condition listed on the death certificate in this way is consistent with the Twentieth World Health Assembly which defined the causes of death to be entered in the cause of death statement as "all those diseases, morbid conditions or injuries which either resulted in or contributed to death."15

The improved clinical management of cerebrovascular events, coupled with an improved understanding of the mechanism of these events, has resulted in a reevaluation on how physicians view a "stroke". Strokes are now viewed as both preventable and treatable. If current methods of prevention and treatment are succeeding then survival rates should be improving. Examination of the coding of cerebrovascular disease as an underlying or an associated cause of death will enable us to determine if part of the decline in the reporting of cerebrovascular disease as an underlying cause of death is a shift to coding it as an associated cause of death. Such a shift might be interpreted as evidence that there has been an improvement in the management of these events.
Though mortality rates for cerebrovascular diseases have declined markedly, this does not necessarily mean that the lifetime significance of cerebrovascular diseases has declined to a similar degree. This results from the fact that cerebrovascular diseases, as well as many other chronic diseases, tend to contribute proportionately more to mortality at older ages. Hence, mortality reductions which increase the number of persons surviving to advanced ages will tend to increase the lifetime contributions of the major chronic disease to total mortality. Furthermore the results of Kuller et al. are instructive in that they suggest the symptoms expressed where stroke was an underlying versus non-underlying cause of death on the certificate are the same. Hence, nonunderlying cause mentions of the stroke may represent as severe and life threatening a disease process as when it is the underlying cause. In this case, for either rate or life expectancy calculations, the multiple cause data would present a more accurate perception of the implications of the disease process. To determine the implications of current declines in mortality rates for the total lifetime significance of cerebrovascular diseases, calculations on the change in life expectancy attributable to the control of stroke as a cause of death are presented.

The standard measures of the lifetime consequences of specific causes of death are calculated in ways that average the effect of the disease over all persons in the population. Since the mortality risk associated with cerebrovascular disease is, clearly, not distributed equally over each member of the population, such calculations do not tell us about the impact of the disease upon affected individuals. As a consequence life expectancy measures are calculated in a way so that they reflect the change in life span for persons directly affected by the disease. This would seem to give a more accurate picture of the actual changes in survival that could be expected clinically if the disease processes were eliminated.

In this paper, the mortality consequences of cerebrovascular disease are evaluated in three ways: 1) the role of stroke as an associated cause of death, 2) the impact of recent stroke mortality declines on the entire life span of the general population, and 3) the consequences of changes in the mortality rates for cerebrovascular diseases on those who would have died from a cerebrovascular event. It is hoped that this treatment of the problem will be a more realistic portrayal of the population implications of the trends.

Methods

These analyses employ cause-specific mortality data for the United States, provided by the National Center for Health Statistics for the period 1968–1978. They differ from the usual mortality data in that all medical conditions reported on the death certificate are coded. Previous analyses have used only the ACME (Automated Classification of Medical Entities) coded underlying cause of death. Throughout the study period, data were coded according to the 8th Revision of the International Classification of Diseases, Adapted (ICDA). This prevents artifacts being engendered by revisions in the coding scheme.

A code of 430–438 is considered to be a cerebrovascular event. If these codes are not present anywhere on the death certificate, the death is considered to be non-stroke related. The reason for using the total range 430–438 is that there are questions concerning the precision of specific subdiagnoses. Furthermore, for many national health policy decisions it is a trend in the overall category that is of most significance.

Stroke is considered to be the underlying cause of death if one of the aforementioned codes is so designated. If one of these codes is present, but not as an underlying cause, stroke is considered to be an associated cause of death.

The results will examine: a) basic frequencies and ratio measures for stroke as an underlying cause of death or as any cause of death, b) life expectancy gains attributable to the elimination of stroke as an underlying or as any cause of death in the general population, and c) life expectancy gains attributable to the elimination of stroke both as an underlying or any cause of death among individuals who actually died of stroke. The analyses are conducted using race/sex specific data.

Life expectancy (e_x) is the expected number of years to be lived, on average, after a given age (x). Life expectancy is one of many estimates derived from a life table. The life table is a statistical model which combines mortality data for various age groups. Using current mortality patterns, age specific mortality risks and expectations can be derived from the life table. There are various standard methodologies for calculating life table functions, and in this paper those of Chiang will be used. Additional details concerning these methodologies are presented in the appendix.

The difference between the life expectancy under observed conditions (e_x), and the life expectancy if the set of deaths did not occur (e_x), represents the increase in life expectancy that could be achieved by eliminating condition "A" as a cause of death. As an example, during 1969–1971 life expectancy was 70.8 years. If malignant neoplasms were eliminated as a cause of death, life expectancy would be 73.2 years. The gain in life expectancy, 2.4 years, represents the difference between these life expectancies. The basic assumption of the cause elimination technique is that mortality due to cause "A" is independent of the remaining causes of death. In the present context it means that the individual who would have died of a stroke, but did not, has an equal chance of dying from the other remaining causes of death. An implicit assumption is that cerebrovascular events would still occur but would no longer be fatal.

When calculating the expected impact of eliminating a specific cause of death, it is necessary to recognize that the increases in life expectancy are averaged over the entire population. This can hide benefits which accrue only to those individuals who have a given disease or condition. Therefore, additional calculations were done to reflect the gain in life expectan-
When reviewing the increases in life expectancy, it is important to place them in perspective. This can be done by examining age-specific data and relating them to table 1. The life table functions were calculated from single years of age data, but only reported for selected ages. A gain of 6.5 years for a white male aged 60 in 1978 means that the individual could expect to live an additional 23.7 years rather than the 17.3 years shown in table 1.

Results
During the period 1968–1977, the age/race/sex standardized mortality rates for stroke (cerebrovascular disease) declined by 32 percent (from 103.4 per 100,000 to 70.0 per 100,000), while total mortality declined by only 18 percent (from 944.3 per 100,000 to 776.9 per 100,000). This can now be related to how stroke was coded as a cause of death.

During the 1968–1978 period, the number of deaths where stroke was an underlying cause declined by 16.8 percent (from 211,321 to 175,817), while the number of deaths where stroke was any cause decreased by 11.0 percent (from 325,533 to 289,885). The ratio of the number of deaths where stroke was coded as the underlying cause of death was 1.54. By 1978 this had risen to 1.64. The ratio can be interpreted as meaning that for every ten deaths where stroke was the underlying cause, there were an additional six deaths where stroke was an associated cause. The aforementioned ratio varies slightly by race/sex groups. In 1977 the ratio for white males was the highest (1.72) with all other race/sex groups having a ratio of approximately 1.61. Thus, the rate of decline of stroke mentioned as underlying cause of death was 52.7 percent faster than its decline mentioned anywhere on the death certificate.

Data on current life expectancy (eₓ), i.e., how long a person could be expected to live if there was no change in the current mortality conditions, for the various age and race/sex groups are presented in table 1. As noted in the methods section, these life expectancies are the comparison levels from which the gains in life expectancy are then measured. The data indicate that life expectancy at birth is greater for whites than nonwhites and greater for females than males.

Under the hypothetical situation that both the force of mortality due to stroke could be completely eliminated and that the force of mortality due to stroke is independent of the forces of mortality for other diseases, the estimated gains in life expectancy (\(e_{x,A} - e_{x}\)) can be calculated. Though both these events are unlikely, the figures serve to illustrate the total lifetime consequences of stroke mortality given current conditions. The elimination of stroke as an underlying cause of death, in 1968, results in a gain in life expectancy at birth of 0.90 years for white males. This means that, if in 1968, stroke were eliminated as an underlying cause of death, life expectancy at birth for white males would increase from 67.5 years to 68.4 years. For ages 45, 60 and 75 the increase in life expectancy remains constant at approximately 0.9 years. At age 75 this means that life expectancy would increase from 8.1 years to 9.0 years. If stroke were eliminated as any cause of death, life expectancy at birth would increase an additional 0.52 years (to 68.9 years), and life expectancy at age 75 would increase an additional 0.56 years (to 9.6 years).

The increase in life expectancy (at birth and ages 45, 60, and 75) for white females is approximately 1.4 years for eliminating stroke as an underlying cause of death and 2.3 years for eliminating stroke as any cause of death. Similar data for nonwhite males is about 1.5 years and 2.4 years. Nonwhite females showed the largest increase, 2.6 years for underlying cause and 4.3 years for any cause. A comparison across the race/sex groups indicates that the gains were smaller for men than for women and for whites than for nonwhites.

Increases in life expectancy are smaller in 1978 than in 1968; reflecting the decline in mortality. If stroke were eliminated as a cause of death in 1978, the increase in life expectancy among white males would be approximately 0.7 years for underlying cause and 1.3 years for any cause. Among white females the increase would be 1.2 years and 2.0 years. Among nonwhite males the increase would be 1.1 and 1.9 years, respectively. Nonwhite females would still experience the largest increase, 1.9 years and 3.3 years.

The aforementioned calculations are useful for reflecting on the aggregate total effect of stroke mortality. However, the results are somewhat artificial, since the effects of eliminating stroke mortality are averaged over the entire population. In order to understand the impact of mortality for cerebrovascular disease, it is necessary to examine a life table which represents the mortality conditions only among individuals who died of stroke (table 2).

To aid in comprehending this table another life table function, \(l_{x}\), is introduced. The function \(l_{x}\), represents the number of deaths, attributable to a specific cause, that would occur at and after age \(x\), if the current mortality conditions did not change. Thus for white males in 1968, there would be 9,640 deaths where

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Life Expectancy (in Years) at Selected Ages, by Race/ Sex Group, 1968 and 1978*</th>
<th>Age</th>
<th>Birth</th>
<th>45</th>
<th>60</th>
<th>75</th>
</tr>
</thead>
<tbody>
<tr>
<td>White males 1968</td>
<td>67.5</td>
<td>27.2</td>
<td>15.8</td>
<td>8.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1978</td>
<td>70.4</td>
<td>29.2</td>
<td>17.3</td>
<td>8.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White females 1968</td>
<td>74.9</td>
<td>33.0</td>
<td>20.2</td>
<td>9.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1978</td>
<td>77.8</td>
<td>35.2</td>
<td>22.3</td>
<td>11.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonwhite males 1968</td>
<td>60.1</td>
<td>23.7</td>
<td>14.5</td>
<td>9.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1978</td>
<td>65.7</td>
<td>26.8</td>
<td>16.4</td>
<td>9.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonwhite females 1968</td>
<td>67.5</td>
<td>28.6</td>
<td>14.5</td>
<td>9.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1978</td>
<td>73.9</td>
<td>33.0</td>
<td>21.4</td>
<td>12.9</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Estimates for life expectancy in 1978 are computed solely from mortality and the latest estimate of the "correct" population for 1978.
stroke was the underlying cause of death. By 1978 this would have declined to 7,820 deaths. As is indicated by the data in the table, nonwhite females had the largest number of people dying from stroke, and for each race/sex group the numbers were lower in 1978 than in 1968. The data also indicate that, for white males in 1978, 93 percent (12,338/13,278) of all the stroke related deaths occurred after age 60. Comparable percentages for white females, nonwhite males, and nonwhite females are 96, 84, and 91, respectively.

The data on strokes can be compared to data for all causes of death, using underlying cause of death data, and for each race/sex group the numbers were lower in 1978 than in 1968. The data indicate that, for white males in 1978, 93 percent (12,338/13,278) of all the stroke related deaths occurred after age 60. Comparable percentages for white females, nonwhite males, and nonwhite females are 96, 84, and 91, respectively.

The data in Table 2 represent the hypothetical situation of Table 2 applied to the population in table 2, i.e., the expected increase in life expectancy among persons who died of stroke, if stroke were eliminated as a cause of death. The gains noted in this table are 5 to 7 times greater than the gains noted for the general population. These data indicate that if stroke were eliminated as a cause of death in 1968, a white male who would have died of stroke could be expected to live an additional 10.25 years. This decreased to 10.00 years in 1978. The largest gains were noted for nonwhite females, and the gains are greater for females than males and nonwhites than whites.

### Discussion

Throughout the study period, the three leading causes of death, using underlying cause of death data, were heart disease, cancer, and stroke. These diseases, respectively, accounted for 38.6 percent, 16.5 percent and 11.0 percent of the underlying causes of death in 1968. By 1978 this had changed to 37.8 percent, 20.6 percent, and 9.1 percent, respectively. Examination of
The general decline in death rates resulted in an increase in evidence to indicate that, at least for cerebral infarction, the median age at death. The data indicate that, after the period of stroke deaths cited as underlying cause versus stroke as any cause. This was observed in each of the age groups. This increase is significant (37 and 29 percent). This increase is significant as those occurrences when it is the underlying cause of death.

A comparison of the age specific frequencies (Birth–64, 65–74, 75–84, 85+) of stroke as an underlying cause of death versus stroke as any cause of death showed slightly larger percentage declines for the underlying cause group. This was observed in each of the race-sex groups, with the largest percentage declines being observed among the Birth–64 age group. This probably implies either a decline in incidence coupled with a decline in case fatality rates, or just a large decline in case fatality rates. However, between 1968 and 1978, not all age groups displayed a decline. The 85+ age group registered an increase in the number of deaths citing stroke as the underlying cause of death and as any cause of death. This increase was observed for each race-sex group, except white males. For white and nonwhite females this increase was quite substantial (37 and 28 percent). This increase is significant from a public health viewpoint. In 1978, one-third of all stroke related deaths among white females occur to individuals aged 85 and over, and half of all the stroke related deaths occur to white females. Therefore, an increase in the number of stroke related deaths among white females aged 85 and over has a major impact upon the general stroke mortality rates.

From 1968 to 1978 there was a slight decline in the ratio of stroke deaths cited as underlying cause versus any cause. The implication is that there was a shift in coding of stroke away from an underlying cause and towards an associated cause of death. One explanation of such a shift would be that there was improved survival from stroke during the period. There is some evidence to indicate that, at least for cerebral infarctions, this may have been the case. In addition, the general decline in death rates resulted in an increase in the median age at death. The data indicate that, after age 45, the mean number of conditions reported on the death certificates goes up with age. This could be interpreted, in layman’s terms, that with increasing age there are more things that go wrong with the body. Given this, there are more causes of death to choose from as the underlying cause, and the probability of stroke being the underlying cause would decrease while the probability of stroke being an associated cause of death would increase. Thus a nonunderlying cause mention of stroke at more advanced ages could represent a morbid process equally as serious as those occurrences when it is the underlying cause of death.

From 1968 to 1978 there was a decline of over 30 percent in the rate of stroke mortality as an underlying cause of death. Since the mortality rate was declining, the gains in life expectancy in 1978 should be less than those in 1968. It was anticipated that the change in the life expectancy gains would be strongly related to the drop in mortality; however, the life expectancy gains at birth decreased only moderately (16 to 27 percent). Comparable data for stroke as any cause of death are 14 to 23 percent. The smaller decline in life expectancy gains for any cause is another reflection of the shift in coding of stroke as an associated cause of death rather than as an underlying cause of death. It is unclear as to why the decline in the gain in life expectancy is so much less than that for stroke mortality, but this is probably related to the generally old age of the survivors.

The smallest change for life expectancy was observed among white females. This possibly results from the large increase in stroke related deaths among the 85+ age group for white females (see above), and the modest increase for the 75–84 age group.

Life expectancy measures have been used to argue that the control of chronic disease will have only a modest impact on the health of the population, since there is usually only a small number of years of life expectancy gain associated with the elimination of these conditions. This statement is true when referring to life expectancy at birth. However, in 1978, nearly 85 percent of all stroke deaths were to individuals aged...
65 and over. Elimination of stroke as a cause of death has minimal impact until the later years of life, and therefore the reference point should be moved to reflect this. Since the added number of years an individual could expect to survive remains fairly constant to age 65, the percentage gain increases with age. If the reference point were moved to age 60, then elimination of stroke as a cause of death would result in an 8.3 percent gain (1.42/17.1) in life expectancy for white males. The percentage gain would be 10.5, 12.9, 18.9 for white females, nonwhite males and nonwhite females, respectively. It is difficult not to call these gains substantial.

In addition, if the effects are restricted to the population at risk, i.e., those who died of a stroke, the effect of eliminating stroke as a cause of death is even greater. These adjusted data (table 3) show a 14.6 percent gain in life expectancy at birth and a 49.8 percent gain in life expectancy at age 60, among white males. Large gains for the other race/sex groups can also be shown. These adjusted gains are greater for nonwhites than for whites, reflecting the larger mortality rates among nonwhites.

The number of deaths where stroke was cited as the underlying cause of death declined much faster than the number of deaths where stroke was cited as any cause of death. An examination of cause elimination at risk, i.e., those who died of a stroke, the effect of eliminating stroke as a cause of death is even greater. These adjusted data (table 3) show a 14.6 percent gain in life expectancy at birth and a 49.8 percent gain in life expectancy at age 60, among white males. Large gains for the other race/sex groups can also be shown. These adjusted gains are greater for nonwhites than for whites, reflecting the larger mortality rates among nonwhites.

The frequency of stroke deaths is important to study, since it defines the size of the clinical problem. The gain in life expectancy due to the theoretical elimination of stroke as a cause of death is important because: a) it represents the lifetime impact of the disease, and b) since it weights each event by the number of years of increased survival, it portrays the future effect of stroke mortality on the current population. Clearly, if more persons survive to advanced ages, proportionately more people will die of the conditions with the highest forces of mortality at those ages. Thus, the life expectancy gain measure is useful in helping put changes in the mortality risk of a given condition in context with the age specific changes in risk for other conditions.

**Conclusion**

This paper has examined various facets of mortality due to cerebrovascular disease, stroke, and the changes in this mortality during the period 1968–1978. The number of deaths where stroke was cited as the underlying cause of death declined much faster than the number of deaths where stroke was cited as any cause of death; indicating that a change in the role of cerebrovascular disease as an associated cause of death has probably occurred. An examination of cause elimination life tables revealed that the life expectancy change which could be expected from the elimination of stroke mortality had not declined as markedly as the stroke mortality rates and that at advanced ages the change was positive. Adjusting the calculations to refer to those who died of stroke showed that the life expectancy gains of eliminating stroke as a cause of death would be quite large.

These observations suggest that there has been progress in the clinical management of cerebrovascular disease; however, stroke still remains a major force of mortality. The various measures presented here indicate that the impact of stroke mortality is greater than that indicated by the underlying cause of death data. The relative decline in stroke mortality was greater for nonwhites, though the demographic subgroup with the largest number of stroke related deaths was white females. Overall, this study suggests that to adequately describe the population implications of chronic disease conditions it is necessary to examine a variety of aspects beyond the data relating to underlying cause of death.

**References**


**Appendix**

The basic parameter of the life table, from which all other parameters can be derived, is the age specific probability of death calculated as the ratio of the number of deaths during the year for people aged x to x + 1 divided by the average number of persons age x at the start of the year, or:

\[ q_x = \frac{D_x}{N_i} \]  

The age specific probability of death from cause A can be calculated by dividing the age specific number of deaths due to cause A by the population, \( N_i \), or...
To further explore the cause specific structure of mortality a number of derivative life table quantities can be calculated. For example, one might wish to know what the age specific probability of death might be if deaths due to a specific disease were hypothetically eliminated. This adjusted probability of death can be calculated as:

\[ q_{1A} = 1 - (1 - q_1)(D_{1B}/D_1) \]  

where \( D_{1B} \) is the number of deaths due to all causes but A, so that \( D_1 = D_{1B} + D_{1A} \).

The age specific probabilities of death are useful measures of differences in risk between populations. However, one may be interested in producing summary, life table measures of those survival differences. A life table measure that reflects differences in survival over the life span is the "life expectancy." The "life expectancy" measure reflects the average age to which people can expect to survive after birth, or the number of years a person can expect to live after age x. The life expectancy calculation requires two parameters, \( l_x \), the number of persons from the original cohort \( l_0 \) which can expect to survive to age x, and \( T_x \), the total number of years lived by the population after age x. These two parameters may be calculated as:

\[ l_x = \int_0^x d_y \]  

where \( d_y = l_{y+1} q_y \), and \( l_{x+1} = l_x - d \)  

and (using the trapezoidal rule, Chiang 1968)

\[ T_x = \int_0^x \tau_y dy \]  

The parameter \( T_x \) simply represents the sum, over all ages after age x to the end of the life table, of two components: a) the number of person years lived during an age interval by persons who survived to the end of an interval (e.g., \( l_{x+1} \)) and b) the number of person years lived by persons who died during the interval (e.g., \( \frac{1}{2} \) year). Using \( l_x \) and \( T_x \), life expectancy is calculated as

\[ e_x = T_x/l_x \]  

Life expectancy in a cohort after cause of death A is eliminated is calculated by substituting \( q_{1A} \) in (5) and proceeding with the computations in (4), (6), and (7). A measure of the impact on life expectancy of eliminating cause A is obtained by taking the difference between the regular life expectancy \( e_x \) and that calculated using \( q_{1A} \). This difference, \( G_x \), represents the average number of additional years that could be expected to be lived in the population if cause A were eliminated, and is represented as:

\[ G_x = e_x - e_x \]  

This measure, however, does not reflect the fact that only a given proportion of the population will die of cause A. In the previous calculations, the total number of person years of life gained by eliminating cause A are averaged over the entire population. To more specifically evaluate the mortality experience of persons dying of cause A a life table for only that population is calculated. This results in the following:

\[ e_{xA} = \frac{1}{l_A} \sum_{y=x}^{\omega} d_{yA} \]  

and

\[ d_{yA} = l_{y+1} q_{yA} \]  

and

\[ T_{xA} = \int_x^\omega \tau_y dy \]  

and

\[ e_{xA} = T_{xA}/l_A \]  

where \( l_{1A} \) is the number of deaths expected to occur after age x from cause A among the \( l_x \) persons alive at x. Similarly, definitions for the other functions can be derived. A further quantity of interest is the life expectancy gain to be expected among persons who died of cause A if cause A were eliminated. This represents the actual gain in life expectancy expected to occur if a clinical cure of A were achieved. This value is:

\[ G_{xA} = e_{xA} - e_x \]
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