SINCE 1938, CEREBROVASCULAR DISEASE has been the third leading cause of death in the U.S. Recent studies examining cerebrovascular disease mortality in the United States reported a downward trend in the mortality rates, with an acceleration in the trend after 1971.2,3 The data displayed in figure 1 show a significant difference in slope for the period 1950-1971 versus 1972-1977 (p < .001). These previous studies treated cerebrovascular disease as one medical entity, when in fact distinct pathologic subcategories of cerebrovascular disease exist. As a consequence, it has been unclear whether the downward trend in cerebrovascular disease mortality is general or type specific. Investigators have been hesitant to explore this question using U.S. data because of reservations about the quality of cerebrovascular disease mortality data and the “softness” of the accompanying data on the death certificate, e.g., type of stroke or mention of hypertension.

In addition to the general difficulties of basing conclusions upon mortality data,4 two other problems have a major impact on these data. The first involves the manner in which the data are coded and released. The National Center for Health Sciences (NCHS) releases data pertaining only to the underlying cause of death, e.g., pneumonia or renal failure. The last time multiple cause of death data were available was 1955, and these data showed that 82 percent of all death certificates with cerebrovascular disease as the underlying cause were considered secondary or “nonmajor” causes. However, these should not be lethal, and many of these, e.g., embolic events, tend to be more directly related biologically and temporally to the event of death than thrombotic-embolic events,5 and may result in the death rate for thrombotic-embolic events being more underreported than that for hemorrhagic events. Any difference in the degree of underreporting by diagnostic subgroup will affect comparisons between these subgroups.

The second problem concerns the accuracy of the diagnosis entered on the death certificate. The certifying official cannot always correctly specify the type of event that occurred unless an autopsy is performed, and in many cases autopsies are not performed. In addition, the possibility exists that a death is attributed to stroke, but in fact, there was another cause of death.

The problems with underlying cause versus multiple cause have not changed over the study period (1968-1977), nor is there any reason to believe that the clinical ability to specify the type of event has changed significantly during this period. Since any errors should be consistent over time, the mortality data can be examined for informational purposes and as a basis for identifying issues requiring further investigation.

Methods

The analysis utilizes mortality data gathered by the NCHS. To minimize the effects of changes in coding conventions and diagnostic procedures, the study was restricted to data which relied upon the Eighth Revision of the International Classification of Diseases, Adapted (ICDA) (1968–1978). At the time of the analysis, data for 1978 were unavailable; therefore, only data for the decade, 1968–1977, were examined. We have no reason to believe that the inclusion of the 1978 data would have altered any of the results or conclusions.

Cerebrovascular disease (stroke) is any of the ICDA codes 430–438. Due to the small number of deaths attributed to some categories as well as the diagnostic uncertainties which exist, the type specific analyses were subdivided into three broad categories: hemorrhages, infarcts and poorly-defined. Data pertaining to deaths from transient ischemic attack (ICDA code 435) were excluded; these should not be lethal, and many authorities would question transient ischemic attack as
a cause of death. Since few deaths were attributed to transient ischemic attacks, the effect of including or excluding this category is marginal.

Previous studies have shown the need to examine stroke data by specific demographic subgroups, our analysis used aggregate and age-race-sex-specific rates. The population data for our study were from the U.S. Bureau of the Census\(^1\),\(^2\) and refer to the U.S. civilian population. To enhance the clarity of presentation, attention was focused on individuals aged 45 years and older. Age groups were constructed for ten-year intervals starting with age 45 and proceeding through age 84. An open-ended interval containing data on individuals aged 85 and over was also constructed. For some of the analyses, the cell sizes were deemed too small to work with, reflecting our belief that this type of analysis should not be attempted on subgroups which consistently contain less than 100 cases per annum.

The analysis uses percentage change merely as a convenient way of quantifying the observed trends. We realize that the results of this calculation are sensitive to casual variation, but the major emphasis is on the direction and the relative magnitude rather than on the absolute magnitude of the change.

### Findings

#### All Strokes

While the general trend for stroke mortality has been amply documented,\(^2\),\(^3\),\(^4\) the findings for the aggregate data (codes 430–438) are presented at this time as a basis for interpreting the type specific findings. In addition, these data may be useful in comparisons with other published literature. During the period 1968–1977, the absolute number of stroke deaths declined by 13.9 percent (table 1). This compares to a drop in the crude stroke death rate of 20.1 percent and a drop in the age-adjusted rate of 31.5 percent. (The 1970 U.S. resident population was used as the standard for calculating the age-adjusted rate, and the direct method of standardization was used.) Over the decade, each of the age-specific rates declined by more than 25 percent, with most declining by more than 30 percent (table 2). The same percentage decline is observed in each subgroup when the data are examined separately.

### Table 1 Number and Percentage Distribution of Stroke Mortality in the U.S. by Type, for Each Year 1968–1977

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
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<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>430</td>
<td>Subarachnoid hemorrhage</td>
<td>#</td>
<td>%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>431</td>
<td>Cerebral hemorrhage</td>
<td>#</td>
<td>%</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>432</td>
<td>Occlusion</td>
<td>#</td>
<td>%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>433</td>
<td>Cerebral thrombosis</td>
<td>#</td>
<td>%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>434</td>
<td>Cerebral embolism</td>
<td>#</td>
<td>%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>435</td>
<td>Transient cerebral ischemia</td>
<td>#</td>
<td>%</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>436</td>
<td>Acute but ill-defined</td>
<td>#</td>
<td>%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>437</td>
<td>Generalized ischemic CVD</td>
<td>#</td>
<td>%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>438</td>
<td>Other and ill-defined CVD</td>
<td>#</td>
<td>%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>211322</td>
<td>207123</td>
<td>207116</td>
<td>209041</td>
<td>213314</td>
<td>214285</td>
<td>207394</td>
<td>194016</td>
<td>188605</td>
<td>181905</td>
</tr>
</tbody>
</table>

\(\) Denotes cases per annum.
TABLE 2  Percentage Change in Cerebrovascular Disease Death Rates by Sex, Race, and Time Period for Selected Age Groups; USA, 1968-1977

<table>
<thead>
<tr>
<th>Age</th>
<th>45-54</th>
<th>55-64</th>
<th>65-74</th>
<th>75-84</th>
<th>85 and over</th>
</tr>
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<tbody>
<tr>
<td>Male</td>
<td>1968-77</td>
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<td>-35.9</td>
<td>-35.7</td>
<td>-25.2</td>
</tr>
<tr>
<td></td>
<td>1968-72</td>
<td>-10.0</td>
<td>-6.4</td>
<td>-9.1</td>
<td>-4.4</td>
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<tr>
<td></td>
<td>1973-77</td>
<td>-24.9</td>
<td>-26.7</td>
<td>-26.9</td>
<td>-20.7</td>
</tr>
<tr>
<td>Female</td>
<td>1968-77</td>
<td>-35.4</td>
<td>-33.0</td>
<td>-37.1</td>
<td>-26.6</td>
</tr>
<tr>
<td></td>
<td>1968-72</td>
<td>-8.4</td>
<td>10.1</td>
<td>-11.3</td>
<td>-7.0</td>
</tr>
<tr>
<td>White</td>
<td>1968-77</td>
<td>-33.9</td>
<td>-32.2</td>
<td>-35.9</td>
<td>-27.0</td>
</tr>
<tr>
<td></td>
<td>1968-72</td>
<td>-8.3</td>
<td>5.3</td>
<td>-9.4</td>
<td>-5.8</td>
</tr>
<tr>
<td></td>
<td>1973-77</td>
<td>-24.9</td>
<td>-24.8</td>
<td>-26.4</td>
<td>-22.2</td>
</tr>
<tr>
<td>Nonwhite</td>
<td>1968-77</td>
<td>-44.9</td>
<td>-43.5</td>
<td>-41.5</td>
<td>-19.5</td>
</tr>
<tr>
<td></td>
<td>1968-72</td>
<td>-14.7</td>
<td>17.5</td>
<td>-15.2</td>
<td>-9.8</td>
</tr>
<tr>
<td></td>
<td>1973-77</td>
<td>-32.0</td>
<td>-29.6</td>
<td>-30.2</td>
<td>-11.1</td>
</tr>
<tr>
<td>Total</td>
<td>1968-77</td>
<td>-35.6</td>
<td>-34.6</td>
<td>-36.5</td>
<td>-26.3</td>
</tr>
<tr>
<td></td>
<td>1968-72</td>
<td>-9.2</td>
<td>-8.1</td>
<td>-10.3</td>
<td>-6.1</td>
</tr>
<tr>
<td></td>
<td>1973-77</td>
<td>-25.0</td>
<td>-25.7</td>
<td>-26.8</td>
<td>-21.2</td>
</tr>
</tbody>
</table>

by sex; however, a difference is noted when the data are examined by race. Among nonwhites, the decline is greater than 40 percent for each age group, except the 75-84 group.

Analysis of the data for the two five-year periods within the decade indicates that the drop in mortality during the first period is approximately one-third to one-half that experienced during the second period. Each age, race, and sex group shared in the accelerated decline during the second half decade, 1973-1977. Annual percentage declines were calculated to determine when the change in rates occurred. For ages under 65, the rates decline an average of 1 percent per annum through 1972, thereafter the change is consistently over 3 percent per annum. In one instance (age 45-54 for the period 1974-1975) the drop is 9 percent per annum. For the 65 and over age groups, the change in rates occurs one year later, i.e., 1973 to 1974; prior to this, the percentage change fluctuates with no consistent pattern emerging. It is important to note that for all age groups there is a large initial percentage decline between 1968 and 1969 that ranged from 3 to 6 percent. It is not known why this occurred, but this does not appear to be related to changes in coding between the 7th and the 8th Revision of the ICDA.

The age-adjusted death rates for males is consistently higher than that for females and higher for nonwhites than for whites (fig. 2). This is in accordance with previous studies and clinical reports. The standardized incidence ratio for stroke is 44 percent greater in males than in females. The basis for the differences by race is still unknown, but may reflect differing incidence, pathology, patterns in the use of medical care, or differences in contributory factors.

Differences between the sexes in the age-adjusted rates have not narrowed and indicate that the rates declined at the same pace. In 1968 the percentage difference in the rates was 15.0, and in 1977 it was 15.4. For nonwhites and whites, the percentage differences declined erratically from 53.7 in 1968 to 41.7 in 1977 and might be related to the improved access to health care afforded minorities by the health legislation of the early 1970's in the U.S.

In summary, the data indicate higher age-adjusted death rates for males and for nonwhites. The percentage change in the age-specific death rates is smaller during the first quinquennial period, 1968-1972, than during the second period, 1973-1977.

Hemorrhage (Codes 430-431)

During 1968-1977, the total number of stroke deaths attributed to hemorrhage decreased by over 25,000, representing a 45 percent decline (table 1). Concurrently, the crude death rate for hemorrhage fell by 49 percent (from 28.31 to 14.56 per 100,000) and the age-adjusted death rate fell by 53 percent (from
28.63 to 13.53 per 100,000). These represented the largest percentage declines for any of the pathologic groups. Within the category of hemorrhage, the percentage changes for cerebral hemorrhage are approximately twice those for subarachnoid hemorrhage.

The age-adjusted data indicate that males consistently have higher rates than females and nonwhites have higher rates than whites (fig. 3). Though not shown, the age-adjusted data for subarachnoid hemorrhage indicates a consistently higher death rate for females than for males, and this is the only subcategory of stroke for which this observation can be made. Given our knowledge about the incidence of hemorrhage this observation was expected. The difference in the rates by sex have been halved; the rates for males are 15 percent greater than those for females in 1968, and by 1977 they are only 7 percent greater. The difference in the rates by race narrowed marginally. Nonwhites have rates that are 80 percent greater than those for whites, but by 1977 the difference is 73 percent.

The range in the decline for the age-specific data is from 43 percent in the youngest age group to 68 percent in the oldest age group, and the absolute change rose monotonically with age. This observation was only for all hemorrhages and for cerebral hemorrhage. The percentage change in the age-specific rates for subarachnoid hemorrhage shows no consistent pattern of decline with age. The decline in age-specific rates for all hemorrhages is substantial during both quinquennial periods, but is greater during the second period. With just a few exceptions, the percentage declines are identical when examined by the various sex and race groups.

Cerebral Infarction (Codes 432–434)

The number of deaths attributed to cerebral infarction declined from 64,588 to 45,317 — representing a 30 percent decline (table 1). Concurrently, the crude rate fell 35 percent (from 32.39 per 100,000 to 21.11 per 100,000), and the age-adjusted rate fell 45 percent (from 33.00 per 100,000 to 18.18 per 100,000). Another indication of this decline is the fact that the percentage of cerebral infarction deaths as a function of all stroke related deaths declined from 31 percent in 1968 to 24 percent in 1977.

The age-adjusted death rates for males are consistently higher than those for females, and this is also true for nonwhites when compared to whites (fig. 4). The difference in the standardized rates for the sexes has remained at approximately 20 percent, an indication that the decline in rates for each sex occurred...
uniformly. In 1968, the age-adjusted rate for nonwhites is approximately 24 percent greater than that for whites and by 1977 the difference is 14 percent, indicating a more rapid decline among nonwhites.

The age-specific rates show declines ranging from 31 percent, for the 45–54 age group, to 49 percent, for the 65–74 age group. For each age group, the percentage change is slightly larger for females than for males, and with the exception of the 75–84 age group, the percentage change is larger among nonwhites than whites. The percentage change during 1973–1977 is much greater than that for 1968–1972, and there is no observed variation in this by sex or race group.

Poorly Defined (Codes 436–438)

The ICDA code numbers contained in this group correspond to the following categories:

- **436** — acute but ill-defined cerebrovascular disease
- **437** — generalized ischemic cerebrovascular disease
- **438** — other and ill-defined cerebrovascular disease

The number of deaths coded as poorly defined stroke rose from 90,218 in 1968 to 105,257 in 1977 — representing an increase of 16.7 percent. The increase in the number of deaths attributed to poorly defined strokes is not monotonic, but rather reflects a consistent rise from 1968 through 1973 followed by a consistent decline. The percentage of poorly defined stroke deaths in relation to the total number of stroke deaths has consistently increased from 42.7 percent in 1968 to 57.9 percent in 1977. It was unanticipated that such an increase would be present during a period that experienced improvements in diagnostic capabilities. One possible explanation for the increase is that the improvement in diagnostic capabilities has resulted in a higher standard being adopted as a basis for a definitive diagnosis.

The crude rate also rose, but the increase is smaller (8.4 percent). The smaller increase results from the changes in the population over the time period. The age-adjusted rates declined 8.8 percent, from 46.10 per 100,000 in 1968 to 42.04 per 100,000 in 1977. As with the absolute numbers, the crude and adjusted rates peaked in 1973.

The age-adjusted rates for males are uniformly 17–20 percent higher than those for females (fig. 5). In 1968 the age-adjusted rate among nonwhites was 59.7 percent higher than that among whites, and this narrowed to 43.4 percent in 1977 (fig. 5).

Contrary to the trend in the numbers, the age-specific rates declined during the decade (table 3). This is accounted for by the fact that the increase in the number of stroke deaths categorized as poorly defined is concentrated in the older age groups, and over the study period these groups increased in size. In the 75–84 age group the rates did not change over the decade. The largest percentage decline, 20.3, was experienced in the 45–54 age group. Generally, the larger percentage changes were experienced by the younger age groups. The age-specific rates were observed to increase during 1968–1972 and decrease during the next quinquennial period.

Discussion and Conclusions

The cerebrovascular disorders, including stroke, continue to be a major cause of death and disability in all societies in which they have been studied. In developing countries, as death due to infections and nutritional disorders decrease, the morbidity and mortality due to stroke appear to be on the increase; some developing countries such as Egypt, Nigeria and the Peoples Republic of China already report stroke as a major public health problem. Whether this is a function of increased longevity or of biological or environmental changes remains to be identified. Paradoxically, in the

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**Figure 5.** Age-adjusted death rates for poorly defined strokes, USA, 1968–1977, by sex and race.

Other than for the 85 and over age group, the age-specific rates for females declined slightly more than for males (table 3). The age-specific rates for nonwhites show a larger percentage change than those for whites, e.g., the percentage change in the rate for the 45–54 age group among whites is −12.7 percent, and the comparable data for nonwhites is −38.4 percent. Contrary to the observed pattern for whites, the age-specific rates, with the exception of the 75–84 age group, decreased for nonwhites during the first half of the decade. Over the decade, the death rate for nonwhites in the 75–84 age group increased by 7.6 percent.
TABLE 3 Percentage Change in Poorly Defined Cerebrovascular Disease Death Rates by Sex, Race, and Time Period for Selected Age Groups: USA, 1968–1977

<table>
<thead>
<tr>
<th>Age</th>
<th>Male</th>
<th>Female</th>
<th>White</th>
<th>Nonwhite</th>
</tr>
</thead>
<tbody>
<tr>
<td>45–54</td>
<td>−18.6</td>
<td>−22.2</td>
<td>−12.7</td>
<td>−38.4</td>
</tr>
<tr>
<td>55–64</td>
<td>−14.2</td>
<td>−20.2</td>
<td>−11.3</td>
<td>−31.7</td>
</tr>
<tr>
<td>65–74</td>
<td>−12.3</td>
<td>−17.4</td>
<td>−13.8</td>
<td>−22.8</td>
</tr>
<tr>
<td>75–84</td>
<td>+1.5</td>
<td>+0.9</td>
<td>−1.2</td>
<td>+7.6</td>
</tr>
<tr>
<td>85 and over</td>
<td>−13.3</td>
<td>−9.1</td>
<td>−10.8</td>
<td>−21.6</td>
</tr>
</tbody>
</table>

The issue before us is: what are the factors that have led to this welcome decrease in stroke incidence and mortality? Some authors claim that important changes in coding definitions and in diagnostic methodology were important contributing factors and that the decrease may be more apparent than real. By examining data only for the 8th ICDA, we have controlled for the effects of changes in coding definitions. Diagnostic methodologies are constantly changing; however, one of the newest and more important diagnostic tools — the CAT Scan — was not commonly available during this period. Therefore, radical changes in coding definitions and diagnostic methodologies should not explain the observed decline in mortality.

However, several factors do need to be considered. As we have learned more about both the unique physiology and specific pathophysiology of the circulation of the brain and about neuronal response to ischemia and hypoxia, the clinical approach to stroke and to the stroke prone patient has undergone important changes. Perhaps the single most important change is the acceptance of stroke as a class of diseases, many of which are preventable and/or treatable. This change in the attitude of physicians has led to intervention and to aggressive treatment. Stroke patients are now rarely turned away as untreatables. The second change has been the development of improved methods of differential diagnosis and of specific therapies both for the cerebrovascular event and for the complications often associated with it. As a result, the person who has suffered a cerebrovascular event now usually survives that event and the immediate complications following it; this is an important contribution to the decrease in stroke mortality.13, 15, 16

A critical class of contributions to the decrease in stroke incidence and mortality is the identification of stroke risk factors. The recognition of transient ischemic attacks as medical emergencies demanding immediate attention and the development of methods of intervention are surely of major importance. As methods of anatomic and physiologic diagnosis becomes more precise, and with the continuing improvement in pharmacologic and surgical tools for intervention, we can look forward to the additional control of stroke risk factors and a resulting decrease in stroke incidence and mortality.

High blood pressure has been established as a major risk factor for stroke.2 This has resulted in the use of anti-hypertensive medication in stroke prevention and therapy, which may help account for the greater decline in mortality for hemorrhagic strokes when compared to cerebral infarctions. Nevertheless, there are still important debates about how far to lower increased diastolic pressure, particularly in the aged, and whether it is efficacious or even safe to reduce isolated systolic hypertension. Obviously, we must balance the negative effects of increased pressure against those of decreased perfusion. The issue is clearly not yet resolved.

The apparent beneficial effect of hypertension control should not obscure the fact that in the U.S. a very large proportion of patients who die of stroke do not have a clinical history of hypertension mentioned on the death certificate. One-quarter of those aged 45–64 who died of stroke during the study period had hypertension mentioned on the death certificate. This drops to 16 percent for those aged 65–74. We recognize that the class without-mention-of-hypertension probably contains three groups of patients: non-hypertensives, labile hypertensives, and unreported hypertensives.

Figures 6–8 indicate that, in addition to age, the percentage of death certificates with mention of hypertension varies by type of stroke. Other than for cerebral infarctions there does not appear to be any important change in the percentages over time. Data gathered from 1971 through 1974 as part of the National Health and Nutrition Examination Survey (HANES) can be used to estimate the prevalence of definite hypertension in the US population.16 For the three age groups 45–54, 55–64, 65–74, the rates are: 24.2 percent, 33.2 percent, and 40.7 percent, respectively. This series of rates are in striking contrast to that displayed in the graphs. This is so for two reasons. First, the rate of...
definite hypertension in the population increases with age, but the percentage of death certificates with mention of hypertension declines with age. Second, given our knowledge about the relationship of stroke and hypertension, we expected a larger percentage of deaths to have mention of hypertension.

Based upon these differences one may speculate that the results pertaining to mention of hypertension on the death certificate are artifactual. To prove this one would have to determine whether individuals were correctly classified as either dying with- or without-mention-of-hypertension. This would require detailed medical data on each death, and unfortunately such data are unavailable on a national basis. Despite this deficiency, an estimate of the magnitude of the error can be made if one is willing to make some assumptions. Using data from the death certificates, the Census Bureau, and the HANES, age-hypertension-specific stroke mortality rates can be computed for the period 1971-1974. The death rates for the without-mention of hypertension group are lower than those for the group with-mention of hypertension. If one assumes that the death rates for the group with-mention of hypertension should be as great as the rates for the group without-mention-of-hypertension, then estimates of underreporting may be generated.

For this to occur among the 65-74 year age group, approximately one quarter of all the cerebrovascular deaths would need to be in error. Comparable figures for hemorrhages are 12 percent, cerebral infarctions — 30 percent, and poorly-defined — 27 percent. Whether one in ten cases or as many as three in ten cases could be in error is for the reader to decide. One must recall, however, that the decade of the 1970’s heralded an era of greater awareness of hypertension, both in the medical community and the lay community.

In summary, the data indicate that: 1) the decline in stroke mortality has not occurred uniformly for the three stroke types examined; 2) the decline in the age-adjusted death rates predates the start of hypertension awareness programs; and 3) the percentage of stroke deaths with hypertension was much lower than expect-
ed; and 4) contrary to expectation the percentage of stroke deaths with mention of hypertension does not increase with age. A death rate, in a small finite time frame, may be viewed as a function of two other rates: a prevalence rate and a case fatality rate. The observed decline in the death rate could be attributed to either one or both of these declining. As previously noted, there is some indication that survival following a stroke has improved. Data from Washington County, Maryland indicated that the case fatality ratios declined slightly for all types of stroke during the period 1969–1974 and 1976. More information, however, is needed before we can clearly identify the cause of the decline in the stroke mortality rate. In addition, despite the importance of hypertension as a risk factor in stroke incidence, the mortality data reveal that there may be other important risk factors that demand our attention. It is perhaps prudent for medical investigators to focus on the problem of stroke prevention in the group of patients without hypertension.

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Stroke. 1982;13:810-817
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