Death Statistics for Cerebrovascular Disease: A Review of Recent Findings

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Abstract:

There is a large margin of error in death statistics. Mortality statistics for cerebrovascular disease show, however, the well-known rise in frequency with age, the higher frequency in men than women which is likely to affect all countries in the coming years, and the greater involvement of nonwhite Americans and Japanese than of white Americans and Japanese Americans. I believe that incidence figures, when they become available in future years, will confirm these findings. Environmental factors, such as a "water factor," also relate to cerebrovascular death rates. Possibly the biggest factor in the medical environment, causing artificial swings in both mortality and incidence figures, will be shown to be the changing diagnostic habits of physicians.

From the viewpoint of etiology, the coexistence of hypertension and cerebrovascular disease dominates the epidemiological picture. The geographic distribution of cerebrovascular mortality in the U.S.A., and the higher mortality in Negroes and in the elderly, particularly relate to underlying hypertension. Ongoing and future population studies, focusing on morbidity as well as mortality, are likely to contribute much in clarifying the relative importance of other causal factors whose harmful effects can be controlled.

ADDITIONAL KEY WORDS cerebral infarction cerebral hemorrhage hypertension etiology of stroke death certificates epidemiology

In recent years epidemiologists have tended to emphasize the defects inherent in mortality rates for cerebrovascular disease; this emphasis has discouraged the extensive analysis of routine death statistics, lest the failings of the data lead to wrong conclusions. For some years to come, however, we shall not have enough morbidity information to guide adequately the development of community programs for patients with cerebrovascular disease. It still seems appropriate, therefore, at periodic intervals to review mortality findings for this disease group, provided one emphasizes that the interpretations must be tentative, to be confirmed or refuted by future and more expensive morbidity studies which will be based on data of higher quality. It is with this philosophy of caution that I present the following review of recent death statistics, coming both from the U.S.A. and abroad, for this important disease group.

Introduction

Death rates are proportional to the frequency with which new cases occur in a defined population, usually termed the incidence rate of the disease, and to the frequency with which such cases die to be certified as having this disease, usually termed the case-fatality ratio. Less tangible factors also affect the size of death rates, such as the likelihood with which physicians will select cerebrovascular disease as the underlying cause of death in patients with multiple disease problems.

Coding procedures have also affected the death statistics for cerebrovascular disease. In 1968, for example, the Eighth Revision of the International Statistical Classification of Diseases came into use;¹ for the first time it places cerebrovascular disease among diseases of the circulatory system, and uses a more detailed
classification to show the association with hypertension. The total death rates for cerebrovascular disease will fall by about 2% with this change in coding, but rates for more specific components, such as cerebral hemorrhage, may change much more extensively. Having a class for occlusion of the precerebral arteries, the Eighth Revision will also clarify the part played by lesions of the carotid and vertebral arteries, a picture which we cannot obtain from previous revisions.

This article will focus on figures during 1949 through 1967, when most countries used the Sixth and Seventh Revisions of the International Statistical Classification of Diseases. Coding procedures, being fairly uniform during this period, played little part in changes which occurred in death statistics for cerebrovascular disease.

In most of the advanced countries, cerebrovascular disease was the third most numerous cause of death during this time. Using the death rates prevailing in the U.S.A. during 1959-1961, we can calculate that the probability at birth of eventually dying from cerebrovascular disease was about one in eight; the likelihood of developing this disease group during one's lifetime, though not necessarily dying of it, was known with less certainty, but probably approached one in three.

Cerebrovascular disease affects persons at older ages than do most other disease groups. Because of this fact, its elimination as a cause of death during 1959-1961 would have extended life in the U.S.A. an average of 1.3 years only; this compared with a 5.9 years' extension by eliminating diseases of the heart, which affect a larger group at younger ages.

**Time Trends for England and Wales**

Let us first review the recent trends in mortality for England and Wales. Table 1 shows the average annual death rates for all cerebrovascular disease and for its more specific components during 1949 through 1967. At a time when its population was slowly aging, England and Wales changed little in its death rates for all cerebrovascular disease, averaging about 1,600 per million population.

<table>
<thead>
<tr>
<th>TABLE 1</th>
</tr>
</thead>
<tbody>
<tr>
<td><em><em>Trends in Average Annual Crude Death Rates</em> for Cerebrovascular Disease per Million Population in England and Wales, by Diagnosis, 1949-1967</em>*</td>
</tr>
<tr>
<td>All cerebrovascular disease</td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
</tr>
<tr>
<td>Cerebral hemorrhage</td>
</tr>
<tr>
<td>Embolism and thrombosis</td>
</tr>
<tr>
<td>Other ill-defined</td>
</tr>
</tbody>
</table>

*Figures calculated by the author from the Registrar General’s Statistical Review of England and Wales for the appropriate years.*

<table>
<thead>
<tr>
<th>TABLE 2</th>
</tr>
</thead>
<tbody>
<tr>
<td><em><em>Trends in Average Annual Standardized Mortality Ratios</em> for Cerebrovascular Disease in England and Wales, by Sex, 1950-1967</em>*</td>
</tr>
<tr>
<td>Men</td>
</tr>
<tr>
<td>Women</td>
</tr>
</tbody>
</table>

*The S.M.R. is the ratio of the actual deaths to the number of deaths expected if the age-sex-specific death rates of 1950-1952 had prevailed. When the age-sex-specific death rates diminish, the S.M.R. falls below 100.
†1963 had a severe winter producing high death rates for many causes, including cerebrovascular disease.

Figures calculated by the author from the Registrar General’s Statistical Review of England and Wales for the appropriate years.
Proceeding with caution to the more specific diagnoses, we can see that death rates for subarachnoid hemorrhage have risen, most steeply at the beginning of this period, while those for cerebral hemorrhage have fallen. Similar but less marked changes have occurred in Canada, the U.S.A., and many other developed countries. Biological and anatomical characteristics are unlikely to change so swiftly in human populations; it seems probable, therefore, that some cases diagnosed as cerebral hemorrhage some years ago are now being labeled as subarachnoid hemorrhage, both in England and Wales and in many other countries.

This change in diagnostic habits of physicians may be a major but is not the sole cause of the fall in cerebral hemorrhage figures. In a lucid review of the changing pattern of cerebrovascular disease, Yates showed that autopsy figures for three hospitals in Manchester, England, reflect a similar fall in hemorrhage deaths, with diagnoses of infarction rising to dominate the autopsy picture since 1955. Though still tentative, Yates' findings support the belief that part of the change in cerebral hemorrhage death rates involves a true fall in frequency, while the rising mortality from embolism and thrombosis may also be partly true. The increasing effectiveness and spreading use of antihypertensive therapy are mentioned most often to explain the falling rates for cerebral hemorrhage.

The argument over true or artificial change is not settled, however. Anderson and MacKay used a simple innovation in studying death certificates in Ontario, Canada. Pointing out that the time between onset and death is shorter for cerebral hemorrhage than for thrombosis, they showed that the proportion of cerebrovascular deaths with survival beyond one day after onset has not changed since 1901. They considered it still possible, therefore, that the rising proportion of cerebrovascular disease ascribed to thrombosis may primarily represent changes in medical teaching and diagnostic fashion.

The rise in table 1 for mortality from "other and ill-defined" vascular lesions affecting the central nervous system is difficult to explain, partly because this wastebasket label covers a mixed group of conditions. A

<table>
<thead>
<tr>
<th>Year</th>
<th>Japan Men</th>
<th>Japan Women</th>
<th>USA Men</th>
<th>USA Women</th>
<th>Women</th>
<th>Men</th>
<th>Canada &amp; Wales Men</th>
<th>Canada &amp; Wales Women</th>
<th>Scotland Men</th>
<th>Scotland Women</th>
<th>England &amp; Wales Men</th>
<th>England &amp; Wales Women</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>1960-1961</td>
<td>2,267</td>
<td>1,594</td>
<td>2,267</td>
<td>1,594</td>
<td>2,267</td>
<td>1,594</td>
<td>2,267</td>
<td>1,594</td>
<td>2,267</td>
<td>1,594</td>
<td>2,267</td>
<td>1,594</td>
<td>2,267</td>
<td>1,594</td>
</tr>
<tr>
<td>1950-1951</td>
<td>1,750</td>
<td>1,449</td>
<td>1,750</td>
<td>1,449</td>
<td>1,750</td>
<td>1,449</td>
<td>1,750</td>
<td>1,449</td>
<td>1,750</td>
<td>1,449</td>
<td>1,750</td>
<td>1,449</td>
<td>1,750</td>
<td>1,449</td>
</tr>
<tr>
<td>Percent Change</td>
<td>29.5</td>
<td>+10.0</td>
<td>29.5</td>
<td>+10.0</td>
<td>29.5</td>
<td>+10.0</td>
<td>29.5</td>
<td>+10.0</td>
<td>29.5</td>
<td>+10.0</td>
<td>29.5</td>
<td>+10.0</td>
<td>29.5</td>
<td>+10.0</td>
</tr>
</tbody>
</table>

Source of data: reference 9.
progressively aging population will produce more deaths for which precise diagnoses are difficult; the change in age is not sufficient, however, to explain this large rise in death rates. Moreover, the rise in England and Wales has occurred at a time when the comparable rates in the U.S.A. have fallen, confounding still further a satisfactory explanation.4 Another marked difference between these two areas is the smaller rates for subarachnoid hemorrhage and for cerebral embolism and thrombosis in the U.S.A.5

**Trends in Standardized Mortality Ratios**

To help compensate for changes in age of the population during 1950-1967, table 2 shows the trends in standardized mortality ratios (S.M.R.'s). The S.M.R. for year X is the ratio of actual deaths in year X to the number of deaths that would have occurred if the age-specific death rates of the base years had persisted. The S.M.R.'s above 100 reflect age-specific rates that are higher than those of the base years, which are 1950-1952 in table 2. This table quickly shows that most age-specific death rates for cerebrovascular disease reached a plateau during 1950-1959 in England and Wales; they have tended to fall since then, more swiftly in women than in men. A similar table for the U.S.A. would show a steeper fall since 1950, without the plateau which occurred in England and Wales.

**Further International Contrasts**

Table 3 contrasts some age-adjusted death rates for recent years in different countries. An age-adjusted rate is a summary figure which can give at a glance a comparison free from the effect of differences in age distributions in the five countries. Each figure represents the rate that would have existed if the age-specific rates of the particular year prevailed in a population whose age distribution was like that of 46 countries combined around 1950.6

The death rates for Japan remain the highest of any country in the world;7 they will be discussed later in this review. The nonwhite population of the U.S.A., a lower-income and mainly Negro group who receive inferior health services, ranks second in all countries for high death rates from cerebrovascular disease. The high prevalence of hypertension in Negroes relates to this finding, but the differential between whites and nonwhites has been rising, with the nonwhite mortality about 80 per cent higher than white rates in recent years.8 Howard has suggested that more hypertensive whites than nonwhites receive antihypertensive therapy, but this hypothesis must still be documented.9

In most countries, including those in table 3, rates are falling more or rising less for women than for men. Most countries now show higher age-adjusted death rates for men than women; in the minority of countries which still have cerebrovascular disease mortality rates which are higher for women than men at each age, women tend also to have the higher death rates for all causes combined. This situation is likely to reverse itself in the coming decades, as has recently occurred in U.S. nonwhites; only for subarachnoid hemorrhage are women likely to outrank their male contemporaries in the size of death rates. It should be emphasized, however, that most countries will continue to have higher crude
death rates for cerebrovascular disease in women, because of the greater age of the female population.

Additional Contrasts by Age and Sex

Table 4 gives death rates, specific for age and sex, for cerebrovascular disease among whites in the U.S.A. and in the total population of England and Wales. The figures show that a ten-year rise in age is accompanied by about a threefold increase in mortality in both areas. Not shown in table 4 is the additional fact that the change with age is proportionally smaller among nonwhites than among whites in the U.S.A. Among the more specific diagnoses, mortality from subarachnoid hemorrhage rises more slowly than others with age, and tends to remain level above 65 years; it thus forms a larger section of all cerebrovascular deaths in the younger ages. The other diagnoses show a steeper rise with age and less tendency to plateau at the older ages; thus “other and ill-defined” conditions form a rising proportion of all cerebrovascular deaths with increasing age.

Except at the youngest and oldest ages, death rates are 10 to 50% higher in men than among women of the same age; this difference is smaller, however, than for most other causes of death. In those countries where hospital discharge figures are available, the figure for men is rarely more than 10% above and is sometimes below that for women. It is possible, therefore, that the incidence rates for cerebrovascular disease, at present unknown, differ less by sex than do the death rates. The higher rates for men affect all diagnoses except subarachnoid hemorrhage for which the sex ratio is reversed; women show higher rates for subarachnoid hemorrhage, both for deaths and hospital discharges, than men of the same age. In the U.S.A., death rates for nonwhites differ less by sex than they do for whites; indeed until the late 1960's, women usually had the higher age-specific death rates in the nonwhite population.

In addition to the rising frequency of cerebrovascular disease with age, the case-fatality ratio also increases with age. Taking the hospital figures for England and Wales as an example, table 5 shows that the proportion dead on discharge rises from about 20% at ages 15 to 44 to more than 50% of patients dead at age 65 and older. If one's primary concern is with the incidence of cerebrovascular disease, it thus becomes obvious that death rates approach more closely the incidence rates at the older ages; moreover, the rise in frequency with age will be steeper for mortality figures than it would be for true incidence figures. Within each age-sex group, table 5 shows that case-fatality ratios have fallen, permitting more patients to be discharged alive and in need of long-term care. However, changing hospital admission practices may have partly caused the case-fatality ratios to improve. In both Britain and North America in recent decades, general hospitals have admitted a rising fraction of all patients with cerebrovascular disease. The additional recent admissions may be less seriously disabled and less likely to die than those admitted in the past.

Differences by Marital Status

It is said that a bachelor's life is a splendid

<table>
<thead>
<tr>
<th>Year and sex</th>
<th>All ages</th>
<th>15-44</th>
<th>45-64</th>
<th>65+</th>
</tr>
</thead>
<tbody>
<tr>
<td>1966 Men</td>
<td>45</td>
<td>22</td>
<td>34</td>
<td>53</td>
</tr>
<tr>
<td>Women</td>
<td>50</td>
<td>19</td>
<td>35</td>
<td>56</td>
</tr>
<tr>
<td>1960 Men</td>
<td>45</td>
<td>24</td>
<td>33</td>
<td>55</td>
</tr>
<tr>
<td>Women</td>
<td>51</td>
<td>27</td>
<td>40</td>
<td>56</td>
</tr>
<tr>
<td>1955 Men</td>
<td>49</td>
<td>17</td>
<td>39</td>
<td>58</td>
</tr>
<tr>
<td>Women</td>
<td>53</td>
<td>36</td>
<td>42</td>
<td>60</td>
</tr>
</tbody>
</table>

breakfast, a tolerably flat dinner, and a most miserable supper. An intriguing problem for analysis is that the “most miserable supper” includes high age-specific death rates for cerebrovascular disease in the U.S.A., higher in the unmarried than the married population from the middle of adult life. This finding applies to most disease groups, and is far from unique to cerebrovascular disease.\textsuperscript{15}

Many hypotheses have been used to explain the lower death rates in the married population. Perhaps most widely held is that marriage is a self-selection process by which already diseased or disease-prone individuals are likely to remain single, or are more likely to become widowed or divorced when they do marry. Self-selection is not the sole factor involved, however; this is suggested by the finding that age-adjusted death rates for cerebrovascular disease are higher among widowed and divorced persons than among single persons. Another commonly suggested mechanism is that many unmarried individuals may falsely report to census takers that they are married; provided that marital status is more accurately reported on death certificates, this process would cause artificially high denominators for the married, with artificially low death rates. This explanation seems less likely when one finds that death rates for all disease groups are not raised by the same amount.

**Further Differences by Race**

Table 6\textsuperscript{14} compares age-adjusted death rates for whites and nonwhites in the U.S.A. during 1949 through 1967. Each figure represents the rate that would have existed if the age-specific rates of the particular period had prevailed in a population whose age distribution was like that of the U.S.A. in 1940. The figure thus adjusts for the fact that the male population is younger than the females, and that the nonwhite population is younger than the white; it is a summary figure which also adjusts for the changes in age distribution which occur from year to year.

The figures show the expected fall in death rates mentioned earlier in this article; rates for nonwhite men show the least fall in recent years. Among men the death rates for nonwhites are 60 to 80\% higher than for whites; among women the nonwhite death rates are 90 to 100\% higher. When we compare time trends in both sexes, the white death rates have fallen more than the nonwhite rates, widening still further the gap between the two groups.\textsuperscript{11}

The age-adjusted death rates have been lower for women than men since before 1940 in whites. However, they remained higher for women than men among nonwhites through the 1950's; only since 1962 have the nonwhite rates been consistently lower for women.

When compared with whites, death rates for nonwhites are particularly high at the younger ages, and the excess mortality mainly involves subarachnoid and cerebral hemorrhage. Case-fatality ratios in the hospital are also higher for nonwhites than whites of the same age.\textsuperscript{17} It seems likely, therefore, that incidence rates will be truly higher among nonwhites, but that the racial difference will be smaller than that reflected by the death rates.

A clear assessment of the white-nonwhite

### Table 6

*Trends in Average Annual Age-Adjusted Death Rates* for Cerebrovascular Disease per Million Population in the U.S.A., by Sex and Race, 1949 Through 1967

<table>
<thead>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>White Men</td>
<td>868</td>
<td>823</td>
<td>819</td>
<td>776</td>
<td>726</td>
</tr>
<tr>
<td>White Women</td>
<td>799</td>
<td>734</td>
<td>713</td>
<td>662</td>
<td>610</td>
</tr>
<tr>
<td>Nonwhite Men</td>
<td>1,413</td>
<td>1,361</td>
<td>1,380</td>
<td>1,345</td>
<td>1,380</td>
</tr>
<tr>
<td>Nonwhite Women</td>
<td>1,533</td>
<td>1,406</td>
<td>1,379</td>
<td>1,317</td>
<td>1,172</td>
</tr>
</tbody>
</table>

*Computed by the direct method, using as the standard population the age distribution of the total population of the U.S.A. as enumerated in 1940. Each figure represents the rate that would have existed if the age-specific rates of the particular year prevailed in a population whose age distribution was like that of the U.S.A. in 1940.

contrasts becomes more difficult when we also consider the problem of inaccurate denominators. Sirken has estimated that the population undercount among Negros in the 1960 census was as great as 46% for women 55 to 64 years of age, compared with a maximum undercount of 5% for any age group among whites. The low figures for the Negro population cause artificially high death rates; moreover, an improvement in the proportion of Negros who are enumerated in the 1970 census will cause an artificial fall in nonwhite death rates for cerebrovascular disease. Nevertheless, the current rates for nonwhites probably show a true excess of deaths, over and above that caused by population underestimates.

The higher frequency of hypertension in Negros is probably the major reason for their excess in death rates; the death certificates for cerebrovascular disease also mention hypertensive disease significantly more often for nonwhites than whites.18 However, socioeconomic conditions, life stresses, and many other factors differ between whites and nonwhites in the U.S.A.; the difference in death rates may relate to a number of these factors. The greater availability of health services, including anti-hypertensive treatment, to whites and to higher-income groups may also play a part in this phenomenon.

Japanese Mortality

This article has already touched on an equally interesting contrast by race, the high mortality from cerebrovascular disease found among the Japanese. Studies among residents of Japan and among Japanese Americans are being carried out to clarify this problem. In 1950 Japanese Americans had death rates for cerebrovascular disease higher than white Americans, but lower than those of the Japanese in Japan.19 By 1960 Japanese Americans had developed rates similar to or lower than those of white Americans of the same age, and much lower than those of the Japanese in Japan.20 There has been no obvious explanation for this change. The findings suggest, however, that factors in the environment of Japan, including death certification habits, cause the high death rates from cerebrovascular disease.21

A somewhat analogous situation is shown in recent contrasts between Puerto Rico's health statistics and those for Puerto Ricans who migrate to New York City.22 The age-adjusted death rates for cerebrovascular disease in the Commonwealth are about 1¼ times those of Puerto Ricans living in the city. Self-selection by migration and differences in socioeconomic status reduce the comparability of these statistics. It seems likely, however, that environmental factors are important in causing high death rates for cerebrovascular disease, both among the Japanese and other races, and that certification habits may exaggerate but not entirely cause the differences. Some of the more important environmental factors will now be discussed.

Environmental Factors

Among whites in the U.S.A., age-adjusted death rates for cerebrovascular disease are highest in the southeastern states and lowest in the less-populated southwestern and mountain states;23 the distinct geographic pattern of mortality in the U.S.A. closely follows that for hypertension. Within Canada, the Eastern Provinces tend to have the highest death rates.3 At every age in North America, death rates for cerebrovascular disease are lower in Canada than in the warmer and more prosperous U.S.A.; at every age in Britain, however, death rates are higher in Scotland than in warmer and more prosperous England and Wales.24 Such variations and inconsistencies have stimulated the search for environmental factors which may affect mortality from cerebrovascular disease. So far these studies have made only limited inroads on the problem.

Most widely studied in recent years has been the relationship between water supply and death rates. First reported in 1957, studies particularly in England and Wales have found that the harder the local drinking water and the more calcium it contained, the lower were the age-specific death rates from cerebrovascular and other cardiovascular disease.25, 26 While the association cannot be adequately explained at present, the British authors suggest that a "water factor" contributes to cerebrovascular mortality.

Several studies from different countries support these findings, although they disagree on many details. A study in Sweden, for example, found a significant association only for women 45 to 64 years.27 Other studies in Oklahoma and Ireland have produced negative findings.28, 29 and recent findings in the U.S.A.
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have borderline significance. After assessing the small amount of data published through 1962, a multidisciplinary panel concluded that no causal relationship had been established between the inorganic constituents of drinking water and mortality from cerebrovascular disease; this may still be the most realistic conclusion to draw from the data available through 1968. An epidemiological relationship seems clear, at least for England and Wales, but other factors occur in localities with soft water which may cause the rise in mortality.

Better established is the seasonal variation in mortality for cerebrovascular disease. Most countries show the greatest mortality in early winter, rising some 30 to 70% above the lowest mortality in the late summer or early autumn. This seasonal swing is characteristic of most cardiovascular deaths in the older age groups, and probably relates more to the incidence of secondary infection rather than the incidence of cerebrovascular disease.

Urban size does not relate consistently to death rates for cerebrovascular disease, judging from recent statistics for England and Wales. Table 7 shows that infant mortality rates and the S.M.R.'s for arteriosclerotic heart disease fell with decreasing urban size in 1967; in contrast, the figures for cerebrovascular disease were more erratic.

Conclusions

At a time when our epidemiological knowledge of cerebrovascular disease remains primitive, it still seems appropriate to review recent death statistics, provided we realize the large margin of error in such data. Mortality statistics show, however, the well-known rise in frequency with age, the higher frequency in men than women which is likely to affect all countries in the coming years, and the greater involvement of nonwhite Americans and Japanese than of white Americans and Japanese Americans. I believe that incidence figures, when they become available in future years, will confirm these findings.

Environmental factors also relate to cerebrovascular death rates, but today's suspects, such as a "water factor," may eventually be replaced by others which relate more directly to disease incidence. Possibly the biggest factor in the medical environment, causing artificial swings in both mortality and incidence figures, will be shown to be the changing diagnostic habits of physicians. Their rising proclivity to diagnose other cardiovascular rather than cerebrovascular disease may well be the major cause of the falling death rates for cerebrovascular disease in recent decades; a smaller, true fall has probably also occurred, and possible reasons such as the better treatment of hypertension must still be proved.

From the viewpoint of etiology, the coexistence of hypertension and cerebrovascular disease dominates the epidemiological picture. The geographic distribution of cerebrovascular mortality in the U.S.A., and the higher mortality in Negroes and in the elderly, particularly relate to underlying hypertension. Other epidemiological findings give more help to health services administrators, concerned

TABLE 7

Infant Mortality Rates* and Standardized Mortality Ratios* for Men from Arteriosclerotic Heart Disease and Cerebrovascular Disease in 1967, England and Wales, by Degree of Urbanization

<table>
<thead>
<tr>
<th></th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Infant mortality rate (1967)</td>
<td>arteriosclerotic heart disease</td>
</tr>
<tr>
<td>Conurbations</td>
<td>19.93</td>
<td>102</td>
</tr>
<tr>
<td>Urban area</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Population 100,000+</td>
<td>19.72</td>
<td>109</td>
</tr>
<tr>
<td>Population 50-100,000</td>
<td>18.51</td>
<td>103</td>
</tr>
<tr>
<td>Population below 50,000</td>
<td>17.18</td>
<td>101</td>
</tr>
<tr>
<td>Rural districts</td>
<td>15.98</td>
<td>90</td>
</tr>
</tbody>
</table>

*Infant mortality rate per 1,000 live births in 1967. The S.M.R. is above 100 when the age-specific death rates are higher than the average for England and Wales in 1967.

†Outside conurbations.

Source of information: reference 32.
with better community care for cerebrovascular disease patients, than to clinicians concerned with etiology. But ongoing and future population studies, focusing on morbidity as well as mortality, are likely to contribute much in clarifying the relative importance of other causal factors whose harmful effects can be controlled.

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