**Diverging Trends in Cerebrovascular Disease and Ischaemic Heart Disease Mortality**


**SUMMARY** The trends in age adjusted and age and sex specific mortality rates for the period 1968–1980 are compared for ischaemic heart disease and cerebrovascular disease. For both sexes and at all ages over 45 the mortality rates for cerebrovascular disease have fallen significantly. However, the mortality rates for ischaemic heart disease are rising significantly for males aged 55–64 and females aged 45–64. The divergence in trend is particularly noticeable for females aged 45–64. Possible hypotheses are discussed explaining this divergence in trend between two cardiovascular diseases of assumed similar aetiology.

IN RECENT PAPERS we have analyzed and compared changes in age adjusted mortality rates due to, inter alia, cerebral thrombosis (rubric 433*) and ischaemic heart disease (rubrics 410–414). An unexpected negative correlation between the trends since 1978 for these two conditions was demonstrated. Among reasons for this phenomenon was the possibility that the risk factors involved in the pathogenesis of the two conditions are different; an alternative hypothesis was that “stroke-prone” persons are dying of ischaemic heart disease before they reach the age at which they would have suffered thrombotic strokes — thus, ischaemic heart disease is prematurely eliminating those susceptible to stroke.

A further analysis of the mortality rates for cerebrovascular disease (rubrics 430–438), cerebral thrombosis and ischaemic heart disease has been made in order to provide more evidence for the applicability of these hypotheses.

**Materials and Method**

The main source of data in this review is the official, published mortality statistics for England and Wales, for the years 1968 to 1980. The cause of death statistics are based on the underlying cause appearing on the death certificate. Numbers of deaths and mortality rates by age and sex are published for each year for the main disease groups, for example, cerebrovascular disease (rubics 430–438), and for the constituent pathological subdivisions, for example, cerebral thrombosis (rubric 433).

For each year and cause of death under consideration, mortality rates adjusted directly for age, rather than crude rates, are calculated, using as the standard the England and Wales population for 1973 (the male population being used to standardize male rates, and the female population to standardize female rates). For measuring the significance of time trends in age- and sex-adjusted and specific mortality rates a straight line of the following form was fitted by a least squares method:

\[ \log_e (mortality\ rate) = \alpha + \beta t \]

where \( t \) = calendar year

Tests may be performed to ascertain whether the estimate of a particular slope, \( \beta \), is significantly different from zero. In addition, the equality of the slopes of two such straight lines may be tested statistically by consideration of the difference between the slopes.

**Results**

In figure 1, the curves are in order of decreasing age-adjusted mortality. Thus curve a) refers to male ischaemic heart disease death rates and curve b) refers to female ischaemic heart disease death rates. Curve c) refers to female cerebrovascular disease death rates and curve d) refers to male cerebrovascular disease death rates. Curves e) and f) similarly refer to cerebral thrombosis. The same code has been used in the other figures.

Figure 1 presents the trends in age-adjusted mortality rates on a semi-logarithmic scale, for ischaemic heart disease, cerebrovascular disease and cerebral thrombosis. These trends are compared in Table 1, which shows the percentage change in the rates over the period 1968–80, the value of \( \beta \) and its significance level (a negative \( \beta \) indicates a downward slope). Figure 1 and table 1 indicate that the age-adjusted mortality rates for cerebrovascular disease and cerebral thrombosis have fallen significantly, with males experiencing the larger fall. For both cerebrovascular disease and cerebral thrombosis there is a positive, significant correlation between the trends for the two sexes. The trend for ischaemic heart disease is marginally downward for females and virtually level for males. Neither of the \( \beta \) values for the ischaemic heart disease rates is

*The use of the logarithm of the mortality rate (q) in the above regression equation is justified on the following grounds. From the viewpoint of rigorous statistical theory it would be unsatisfactory to use as an independent variable in a regression analysis a probability or rate which is constrained to lie between 0 and 1, whereas the estimated value from the regression equation could fall outside these bounds (and in particular be negative). It would be more satisfactory to use the logit function, i.e. \( \log_e (q) \), as an independent variable. This asymptotes to \( -\infty \) as q approaches zero and to \( +\infty \) as q approaches unity. Use of the logit transformation in a regression equation ensures that the estimated values of q will all lie between 0 and 1. In this case q is much smaller than 1 so it is reasonable to replace the logit function by \( \log_e (q) \).
Age adjusted death rates (per 1000 per annum) for Ischaemic Heart Disease and Cerebrovascular Disease, 1968–1980.

**Figure 1.** Age Adjusted Death Rates (per 1000 pa) for Ischaemic Heart Disease and Cerebrovascular Disease 1968–1980.

There is a positive correlation between the age-adjusted rates for cerebrovascular disease and ischaemic heart disease for both sexes which is significant for females only. The same is true of cerebral thrombosis and ischaemic heart disease.

An examination of the trends for the age-specific mortality rates for these two conditions will be used to gain further insight into the findings of table 1.

Table 2 presents the trends in age-specific mortality rates for cerebrovascular disease between 1968–1980. The statistics indicate that for both sexes there has been a significant downward trend, for each age-group. For each age-group, the two sexes have experienced trends which are positively correlated to a significant extent.

Detailed results for cerebral thrombosis are not tabulated here but they show similar features to cerebrovascular disease except that the declines are more rapid in each age/sex category.

Table 3 presents the trends in age-specific mortality rates for ischaemic heart disease between 1968–1980. The statistics indicate that, for both sexes, the mortality rates have increased at ages 45–64 and decreased at ages 65 and over. For males 2 of the $5\hat{\beta}$’s are significantly different from zero. For females 4 of the $5\hat{\beta}$’s significantly different from zero ($p > .05$). The trends in figure 1 appear approximately level, with a local maximum in 1972. The correlation between the ischaemic heart disease trends for the two sexes is not significant.

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are significantly different from zero, and, in particular, both upward trends at ages 45–64 are significant. For each age-group the two sexes have experienced trends which are positively correlated to a significant extent.

Figures 2–8 illustrate these trends in age/sex specific mortality rates for cerebrovascular disease, cerebral thrombosis and ischaemic heart disease. Examination of these graphs reinforces the above findings and also suggests a local maximum in year 1972 for many of the age/sex categories.

Table 4 shows the correlation coefficients between the trends in mortality rates for cerebrovascular disease and those for ischaemic heart disease. At ages under 65, the coefficients are negative (and significant for females). At ages over 65, all six coefficients are positive. Similar (unpublished) results are obtained from a comparison of trends for cerebral thrombosis and ischaemic heart disease.

A detailed examination of the age specific mortality rates for ischaemic heart disease in each of the calendar years 1968 to 1975 showed that in general the rates increased up to 1972 after which there was a levelling off or decrease (depending on the age group). Using the method suggested by Armitage we may quantify the differences between 1968 and 1972, and between 1972 and 1975. For example, the observed changes in the male age-specific death rates for ischaemic heart disease at ages 35–64 for males are pooled to form a

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**Table 3** Ischaemic Heart Disease: Trends in Age-specific Mortality Rates (per 1000 pa) (1968–80)

<table>
<thead>
<tr>
<th>Age at death</th>
<th>45–54</th>
<th>55–64</th>
<th>65–74</th>
<th>75–84</th>
<th>Over 85</th>
</tr>
</thead>
<tbody>
<tr>
<td>1980</td>
<td>2.623</td>
<td>7.123</td>
<td>15.893</td>
<td>30.751*</td>
<td>50.546*</td>
</tr>
<tr>
<td>% change</td>
<td>+7.0%</td>
<td>+2.0%</td>
<td>-0.7%</td>
<td>-2.2%</td>
<td>-14.2%</td>
</tr>
<tr>
<td>$\hat{\beta}$</td>
<td>0.0055</td>
<td>0.0022</td>
<td>0.0006</td>
<td>0.0009</td>
<td>-0.0071</td>
</tr>
<tr>
<td>Significance of $\hat{\beta}$</td>
<td>$p &gt; 0.05$</td>
<td>$p &lt; 0.05$</td>
<td>$p &gt; 0.05$</td>
<td>$p &gt; 0.05$</td>
<td>$p &lt; 0.001$</td>
</tr>
</tbody>
</table>

**Females**

| 1968         | 0.419 | 1.851 | 7.020 | 18.969 | 44.071 |
| 1980         | 0.458 | 2.033 | 6.609 | 17.577*| 36.542*|
| % change     | +9.3% | +9.8% | -5.9% | -7.3% | -17.1% |
| $\hat{\beta}$| 0.0143| 0.0070| 0.0026| 0.0030| -0.0062|
| Significance of $\hat{\beta}$ | $p < 0.01$ | $p < 0.01$ | $p < 0.05$ | $p > 0.05$ | $p < 0.05$ |

*1979 most recent year available.
Age specific death rates (per 1000 per annum) at ages 55-64 for Ischaemic Heart Disease and Cerebrovascular Disease, 1968-1980

Age specific death rates (per 1000 per annum) at ages 65-74 for Ischaemic Heart Disease and Cerebrovascular Disease, 1968-1980

Age specific death rates (per 1000 per annum) at ages 75-84 for Ischaemic Heart Disease, 1968-1979

Age specific death rates (per 1000 per annum) at ages 75-84 and over 85 for Ischaemic Heart Disease, 1968-1979.
Age specific death rates (per 1000 per annum) at ages over 85 for Cerebrovascular Disease, 1968-1979

FIGURE 8. Age Specific Death Rates (per 1000 pa) at ages over 85 for Cerebrovascular Disease 1968-1979.

for a single age/sex group could produce similar results, examination of figures 3, 4, 5 and 7 confirms that there was a general increase between 1968 and 1972 and a general decrease thereafter.

Discussion

The official mortality statistics for England and Wales are well presented and more comprehensive than those of other countries. There are however potential sources of bias which impede the use of mortality statistics for epidemiological purposes. These errors are well documented. If these errors are constant in magnitude between years and between the subgroups of the population under consideration, then mortality data may furnish valuable epidemiological information, providing, inter alia, an inexpensive and convenient means of obtaining clues to aetiological hypotheses and determining consistency between hypotheses.

An unexpected negative correlation between the trends for cerebral thrombosis and ischaemic heart disease has been documented. Among the possible reasons that have been suggested for this feature are the following: (a) different risk factors are involved in the pathogenesis of the two conditions, (b) persons who would have suffered a cerebrovascular accident are being killed off prematurely by ischaemic heart disease. In other words, the "stroke-prone" or susceptibles are killed off by ischaemic heart disease, (c) the trends reported may have been affected by changes in diagnostic practice, for example, for deaths with both cerebrovascular disease and ischaemic heart disease present, there is the problem that only one underlying cause of death may be chosen — changes in diagnostic practice will affect the choice.

This list, however, is by no means exhaustive. The applicability of (a) and (b) are discussed in this paper. The validity of hypothesis (c) is discussed in a further paper.

Ages under 45 are excluded from the analysis presented since the number of deaths is too small at these ages to form the basis of a reliable statistical analysis.

Figures 1-7 present the mortality rates for cerebrovascular disease, cerebral thrombosis and ischaemic heart disease (specific for age at death and sex) plotted against year of occurrence with a semi-logarithmic ordinate scale to fit in with the proposed regression model and so that the vertical difference between two curves is a measure of the ratio between the two mortality rates.

For cerebrovascular disease and cerebral thrombosis, it appears from table 1 that the female age-adjusted mortality rates are higher than the corresponding male rates. This arises because the female standard population used is more concentrated at the higher ages than the male standard population and because the age specific mortality rates increase approximately exponentially with age. Thus, in the standard populations, the percentages at the oldest ages are as follows:

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>% aged 65-74</td>
<td>7.74</td>
<td>9.90</td>
</tr>
<tr>
<td>% aged 75-84</td>
<td>2.65</td>
<td>5.18</td>
</tr>
<tr>
<td>% aged 85 and over</td>
<td>0.47</td>
<td>1.33</td>
</tr>
<tr>
<td>% aged 65 and over</td>
<td>10.86</td>
<td>16.41</td>
</tr>
</tbody>
</table>

An examination of the age-specific mortality rates reveals that the female rates are lower at ages under 85. Comparing the trends for the two sexes in each of Tables 1, 2 and 3 reveals a significant positive correlation in all of the cases, suggesting that the two sexes have experienced parallel trends from the points of view of direction and regularity.

TABLE 4 Correlations Between Trends in Age-specific Mortality Rates (1968-80)

<table>
<thead>
<tr>
<th>Cerebrovascular disease and ischaemic heart disease</th>
<th>All ages (age-adjusted)</th>
<th>Age at death</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>45-54</td>
<td>55-64</td>
</tr>
<tr>
<td>Males</td>
<td>Correlation coefficient</td>
<td>+0.142</td>
</tr>
<tr>
<td></td>
<td>Significance of correlation coefficient</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Females</td>
<td>Correlation coefficient</td>
<td>+0.572</td>
</tr>
<tr>
<td></td>
<td>Significance of correlation coefficient</td>
<td>&lt; 0.05</td>
</tr>
</tbody>
</table>

*1979 most recent year available.
TABLE 5  Changes in Mortality Rates for Ischaemic Heart Disease: Standard Normal Deviates and Level of Significance

<table>
<thead>
<tr>
<th></th>
<th>1968–72 comparison</th>
<th>1972–75 comparison</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age groups</td>
<td></td>
<td></td>
</tr>
<tr>
<td>35–64</td>
<td>10.3†</td>
<td>-2.1*</td>
</tr>
<tr>
<td>Over 65</td>
<td>8.3†</td>
<td>-6.3†</td>
</tr>
<tr>
<td>Over 35</td>
<td>12.8†</td>
<td>-6.4†</td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age groups</td>
<td></td>
<td></td>
</tr>
<tr>
<td>35–64</td>
<td>6.9†</td>
<td>-0.4</td>
</tr>
<tr>
<td>Over 65</td>
<td>4.0†</td>
<td>-7.6†</td>
</tr>
<tr>
<td>Over 35</td>
<td>6.2†</td>
<td>-7.3†</td>
</tr>
</tbody>
</table>

* < 0.05; † p < 0.001.

The significant downward trends for cerebral thrombosis that have been reported are to some extent compounded by the significantly upward trend in mortality ascribed to rubric 436 (acute but ill-defined cerebrovascular disease) which has been reported elsewhere.¹ ² It is possible that the trends for cerebral thrombosis reported above mask a tendency for medi­cations to allocate such deaths increasingly to rubric 436. In view of this effect, which is discussed more fully elsewhere,³ we shall focus attention on mortality trends for cerebrovascular disease, rather than on cerebral thrombosis.

For cerebrovascular disease the age specific mortality rates have fallen most rapidly for both sexes at ages 55–64 as evidenced by the value of the regression coefficients in table 1. At the high ages (i.e. over 75), the slower fall for females can be attributed partially to the demographic ageing of the female population relative to the male population. However, for ischaemic heart disease the age specific mortality rates have risen more rapidly for females than for males at ages under 65 and fallen more rapidly for females at ages 65–84 with little difference at ages 85 and over.

The results in Tables 1–3 suggest significant downward trends in the age and sex specific mortality rates for cerebrovascular disease, and upward trends for ischaemic heart disease which are significant at ages 55–64 for both sexes and 45–54 for females. Comparing the trends for cerebrovascular disease and ischaemic heart disease in tables 2 and 3 reveals that the slopes of the trends for corresponding age- and sex-groups are significantly different for both sexes and all five age groups. Comparison may be taken further by the calculation of correlation coefficients. Table 4 shows that the trends are positively correlated for ages 65 and over and are negatively correlated for ages under 65 — these negative correlations are significant (p < .05) for females aged 45–64.

Different risk factors may be involved in the pathogenesis of cerebrovascular disease and ischaemic heart disease,⁴ and the significant factors for ischaemic heart disease may have become more potent over the period of investigation. The most important risk factors for cerebrovascular disease are ageing and hypertension.

There is evidence to suggest that, apart from these, the risk factors for ischaemic heart disease have little effect on the risk of cerebrovascular disease, for example, hypercholesterolaemia (possibly linked to a diet high in saturated fat or sugar intake) obesity, physical inactivity and cigarette smoking.⁵ ⁶ ⁷ ⁸ Regarding the potency of risk factors, the continued decline in cerebrovascular disease mortality may be related to either effective and efficient treatment of hypertension, to reductions in the risk of stroke among persons with hypertension and/or a decline in hypertensive disease in the population, secondary to changes in the risk factors for elevated blood pressure.¹ ²

Further evidence for the likelihood of a different set of risk factors operating for the two conditions comes from an examination of the age specific mortality rates for a recent year.⁹ ¹⁰ ¹¹ ¹² At each age, there are striking differences in the sex ratio of the mortality rates (i.e. male rate divided by female rate) — for example, in 1980, at ages 45–54, the ratio for cerebrovascular disease was 1.1 and for ischaemic heart disease 5.7.⁹

The statistics published by the Tobacco Research Council¹³ on recent trends in cigarette smoking indicate that, since 1968, the percentage of males who smoke has fallen at all ages, but for females the percentage has risen at ages over 60. The trend in the annual consumption of cigarettes per adult by five year age groups shows that for males aged over 45 the consumption has risen but the trends are not significant (p > .05). For females aged over 45 the trend is upward and significant in all five of the age groups. This feature may be one of the reasons explaining the above mentioned upward trend in ischaemic heart disease mortality for females at ages under 65, illustrated in tables 3–5.

In addition, hypothesis (b) (that the stroke susceptibles are being killed off by ischaemic heart disease) might explain the above findings, and in particular the significant rises in mortality rates for ischaemic heart disease experienced by females at age under 65. However, there is little direct evidence supporting the hypothesis that “stroke-prone” persons are dying prematurely of ischaemic heart disease before they reach the age at which they would have suffered a thrombotic stroke. However, assuming the validity of this hypothesis, the decline in cerebrovascular disease mortality may be regarded as a secondary effect to the decreased prevalence of cardiovascular complications following a myocardial infarction (e.g. congestive heart failure, left ventricular hypertrophy).

Using a linear regression approach to fit a curve to the time trend in the logarithms of the ischaemic heart disease mortality rates is not altogether satisfactory, since analysis reveals that these trends are curvilinear with the peak rates occurring in the middle of the period under investigation. Below we tabulate the peak years for each of the age and sex specific trends.

<table>
<thead>
<tr>
<th>Year</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>1975</td>
<td>1975</td>
<td>1979</td>
</tr>
<tr>
<td>1976</td>
<td>1972</td>
<td>1976</td>
</tr>
<tr>
<td>1977</td>
<td>1972</td>
<td>1972</td>
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<tr>
<td>1978</td>
<td>1968</td>
<td>1968</td>
</tr>
<tr>
<td>1979</td>
<td>1972</td>
<td>1972</td>
</tr>
</tbody>
</table>

These peak years (reinforced by the results of table 5)
suggest that, the increase in ischaemic heart mortality, which has continued unabated since at least 1950, may have been halted, for males and for females aged over 65: but falling trends since 1972 should not necessarily be taken at their face value since they can be attributed to a succession of mild winters and the absence of a severe influenza epidemic: both these factors are associated with reduced mortality from many causes. As more statistics become available, the persistence of these downward trends will be tested with more confidence.

The fall in ischaemic heart disease mortality rates since 1972 could be attributed to the widespread reduction in the smoking of cigarettes with a high tar content. The American Cancer Society Study has reported significantly lower mortality rates for both sexes for smokers of low and medium tar and nicotine cigarettes relative to smokers of high tar and nicotine cigarettes. An indirect verification of this hypothesis may be obtained by examining the mortality trend from peptic ulcer, a condition which is also closely affected by cigarette smoking. Peptic ulcer, rather than lung cancer, has been chosen because, as with ischaemic heart disease, the increased risk from smoking is promptly eliminated with the cessation of smoking. Coggin et al. provide a full discussion of the downward trend in morbidity and mortality for peptic ulcer in England and Wales. Over the period 1968–79 there is a downward trend in age-adjusted mortality rates from peptic ulcer for England and Wales for both sexes. The highest rates occurred in 1969 for males and 1972 for females. These findings give a rough guide and provide some supporting evidence for linking the 1972 peak in ischaemic heart disease mortality rates to the decreasing importance of high tar cigarettes.

The apparent peak in 1972 and subsequent fall in ischaemic heart disease mortality rates may also be attributed to the general decline in the household use of animal fats and refined sugar and an increase in the use of vegetable fats and margarine. This association has been fully discussed by Florey et al.

It is interesting to compare the mortality trends discussed above with those experienced in other western countries. In Australia, mortality rates for ischaemic heart disease for both sexes at ages 30–64 have fallen since 1965–1967.

More significantly, in the US for the period 1963–73, age-adjusted mortality rates for all cerebrovascular disease have fallen continuously, by 13.1% for males and by 18.1% for females. Over the same period the age-adjusted mortality rates for ischaemic heart disease have also fallen. Examination of the long-term trend for ischaemic heart disease in the US reveals that 1963 is the peak year overall. At ages 35–74 the peak year for white males is 1968, for white females it is 1958 and for non-whites it is 1968 for both sexes. This fall in USA ischaemic heart disease mortality has followed almost two decades of constant rise. Comparison of the relative falls in the age-specific mortality rates for cerebrovascular disease and for ischaemic heart disease over the period 1963–1975 reveals a faster rate of decline for cerebrovascular disease at ages over 45 and for ischaemic heart disease at ages 35–44. These findings disagree with those reported for England and Wales. In particular the following significant points are noted: a) the US shows parallel mortality trends between the two conditions since 1963; b) the peak year of ischaemic heart disease mortality is 1963 in the US, but is 1972 or later in England and Wales (for all persons aged over 35).

Further research is needed to explain these differences between the two countries, for example, by cohort mortality studies. It is likely that they may reflect differences in the incidence rates of the two conditions (caused possibly by variations in the prevalence of the major risk factors) or even in case fatality rates (caused possibly by the lethality of the major risk factors and by variations in treatment methods and their efficiency). The fall in ischaemic heart disease mortality in the US has been discussed extensively and it seems likely that there has been both a fall in incidence and in case fatality, linked to more effective treatment of the acute stages of the disease. Study of the differences between the experience of the United States and England and Wales might offer real clues to the understanding of the epidemiology of these cardiovascular diseases.

It is possible that the overall decline in heart disease mortality that has been observed in Australia and the US has begun in England and Wales. As more official statistics become available we shall be able to test the persistence of this recent downward trend in mortality.

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Computer Based Classification of Carotid Arterial Disease: A Prospective Assessment

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SUMMARY A minicomputer based pattern recognition method has been used to prospectively classify the category of disease involvement of 105 carotid arteries. The system utilized spectral patterns obtained from a combined B-mode/pulsed Doppler unit. All decisions are based upon comparison of an unknown, averaged waveform with a series of vessels with known severity of disease. The variability in the computer decision as compared to arteriography is discussed.

DOPPLER ULTRASOUND TECHNIQUES are widely used in the evaluation of arteriosclerosis of the carotid bifurcation. The ability of the technique to detect minor lesions has been further enhanced by spectral analysis of the velocity-time waveform. The severity of disease may be estimated by a visual interpretation of the spectra which involves a qualitative assessment of the peak systolic frequency, late diastolic frequency and the amount of spectral broadening in the deceleration phase of systole.1 There have been reports in the literature describing the use of velocity parameters for the detection of disease of the carotid arteries.2-5 Rutherford et al.2 used a discriminant analysis of hand-measured waveform parameters to show that velocity waveform analysis was useful in the identification of normal and diseased arteries. We have developed similar techniques using computer selected waveform parameters which are used in a three step decision process to classify disease into four categories.5 This report describes these methods, our experience with the technique, and its performance with a set of prospectively analyzed patients.

Materials and Methods

The subjects in this study consisted of 11 presumed normal volunteers and 49 patients with suspected extracranial arterial disease. In the patient group, there were a total of 83 sides suitable for study. The remaining 15 sides were excluded from the analysis for the following reasons: (1) nine sides had occlusion of the internal carotid artery; (2) four had an endarterectomy; and (3) two were not recorded on tape for unknown reasons.

Biplanar contrast arteriography was performed at the discretion of the referring physician. The arteriograms were all read by an experienced radiologist who was unaware of the result of the noninvasive study. The arteriographic classification of disease was based on measurements of the diameter of the internal carotid artery made from the unsubtracted film. The use of unsubtracted films allowed small flecks of calcification within the vessel walls to be seen, thereby permitting an estimate of the position of the vessel wall to be made. The readings were made from both the anteroposterior and lateral projections. The degree of stenosis reported was derived from the minimum residual lumen diameter measured from one or other of the projections. The degree of stenosis was calculated from these measurements using the equation:

\[
\text{% stenosis} = 100 \times \left(1 - \frac{\text{diameter of diseased vessel lumen}}{\text{diameter of this vessel if normal}}\right)
\]

Neither the length nor the surface characteristics of the lesions were assessed in this study. While these would have an impact on the velocity pattern across the lesion, we had no way of either quantitating these factors or including them into our approach to the problem.

All of the sides were studied with a pulsed Doppler duplex scanning device. The scanhead contains piezoelectric crystals which operate at 5 MHz for both the B-
Diverging trends in cerebrovascular disease and ischaemic heart disease mortality.
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