Incidence and Outcome of Cerebrovascular Disease in Perth, Western Australia

Gary Ward, MBBS, Konrad Jamrozik, MBBS, DPhil, and Edward Stewart-Wynne, FRACP

We estimated the event rates for stroke and transient cerebral ischemic attacks in a prospective community-based epidemiologic study in a representative segment of the city of Perth, Western Australia, during a 10-month period in 1986. Of 349 persons with an initial diagnosis of stroke or transient ischemic attack, 154 had suffered a first stroke, 75 a recurrent stroke, and 47 a transient ischemic attack; the remaining 73 persons were thought not to have had an episode of acute cerebrovascular disease. Annual event rates for first stroke (age-standardized to the “world” population) were 120 per 100,000 for males and 56 per 100,000 for females. The crude case-fatality ratio at 28 days after the index event for first stroke was 23% and varied from 0% for lacunar infarction to 57% for subarachnoid hemorrhage. (Stroke 1988;19:1501-1506)

Mortality from cerebrovascular disease in Australia has decreased over at least the last 3 decades,1 and this trend began 15 years before a decline in mortality from ischemic heart disease (IHD) became apparent. In common with several other Western countries,2-3 both the rate and the extent of the decrease in mortality from stroke has been greater than that for IHD. However, apart from single point estimates of the incidence of all strokes, both fatal and nonfatal,4 and the prevalence of survivors of stroke in Melbourne,5 there has been little study of either etiologic factors for stroke or trends in nonfatal cerebrovascular disease in Australia. Therefore, it is impossible to determine whether the decline in mortality from stroke is due to a decline in the incidence or in the severity of new strokes, either of which might reflect better control of risk factors, or to the improved care of persons with new strokes, resulting in a lower case-fatality ratio. Moreover, a change in the mixture of the diverse pathologic entities encompassed by the term “stroke” might also bring about a decline in mortality in the absence of a change in overall incidence of the condition. Clearly, all of these factors might have contributed to the downward trend in mortality from stroke that has been observed in our community, and all of the factors must be examined if we are to explain this downward trend.

Unlike IHD, the epidemiologic surveillance of stroke is complicated by the fact that a sizable proportion of persons with stroke is never admitted to a hospital.6 Hospital-based registers of stroke therefore provide a misleading reflection of the burden of disease borne by the community as a whole, particularly if the proportion of persons with stroke that is admitted to a hospital changes over time. To avoid these problems, we compiled a community-based register of acute cerebrovascular disease events in a representative segment of the population of Perth, Western Australia. Care was taken to collect all available objective evidence pointing to the underlying pathology in each episode and to follow up all persons with events to obtain a complete picture with regard to outcome.

Subjects and Methods

We conducted our study in a geographically well-defined and demographically representative segment of the Perth metropolitan area with a population of 134,690 persons according to the 1986 census. Events were registered for the 10-month period January 20, 1986, to November 17, 1986. All general practitioners and hospital medical staff within and around the study area were requested to report cases to an office equipped with a 24-hour answering machine. Residents of the study area presenting to major teaching hospitals in Perth with episodes of acute cerebrovascular disease were detected by monitoring casualty attendances and hospital admission lists. Surveillance of lists for radiologic procedures, computerized hospital separation data, cor-
oner’s records, and death registrations yielded additional cases. To ensure that ascertainment of cases over the 10-month period was complete, all general practitioners serving the study area were contacted by mail and by telephone in December 1986 and asked to recall all cases of stroke that they had seen during the preceding year. Any cases not previously registered were then assessed using the method described below.

Each patient was visited by a medical registrar as soon as possible after report of the case, and personal details, history of the index event, medical and neurologic history, family and social history (including consumption of tobacco and alcohol), and results of cardiovascular and neurologic examinations were recorded. The results of any hematologic, biochemical, or radiologic investigations performed were obtained, although the study team did not itself initiate any investigations. All cranial computed tomographic (CT) scans were reviewed by one or more members of the study team and independently by a neuroradiologist. The anatomic diagnosis and apparent pathologic basis of each event were based on all available information, using the strict clinical and radiologic criteria published by the Harvard Cooperative Stroke Registry. A specific pathologic diagnosis was not made without either a CT scan or a postmortem examination. Each patient’s description of existing disability from any cause prior to the index event was recorded, and a Barthel functional assessment score was calculated at the time of the first assessment.

Patients with an initial diagnosis of transient cerebral ischemic attack (TIA) who were seen within 24 hours of the onset of symptoms were reviewed at least 1 day later to ensure accurate categorization. All patients were assessed again 3 months after the index event to obtain information regarding survival, place of residence, change in levels of disability and dependence, and use of medical services such as physiotherapy and occupational therapy.

Statistical analyses were limited to those events for which a final diagnosis of acute cerebrovascular disease was made by the study team. Stroke was defined as the rapid onset of “clinical signs of a focal or global disturbance of cerebral function, lasting more than 24 hours or until death, with no apparent non-vascular cause.” A TIA was defined as an event for which all symptoms and clinical signs had resolved completely within 24 hours of the onset of the first symptoms. First and recurrent events were distinguished on the basis of the
Results

The study area was triangular and consisted of the central business district of Perth, where the Royal Perth Hospital is sited, and the suburbs lying immediately to the north (Figure 1). Effectively, its geographic boundaries were the Swan River to the south and east, the northern limit of the Perth metropolitan area to the north, and a major road, the nominal boundary between the drawing areas of two major teaching hospitals, to the west. The study area covered eight complete postal code districts and part of a ninth. Data from the 1981 census indicated that the study population was somewhat older (Figure 2) and less likely to have changed address between 1976 and 1981 than the remainder of Perth, but otherwise the study population was quite representative of the Perth metropolitan area as a whole. In addition, patterns of admission to and length of stay in a hospital with stroke in 1984–1985 were typical of the entire city (Table 1).

Over the 10 months of our study we registered a total of 369 events in 349 persons. Half the events were detected through surveillance of admissions to hospitals, 16% were first reported by general practitioners, 11% were reported by hospital doctors, 10% were discovered by review of death certificates, and the remainder were from various other sources. The median delay between onset of symptoms and assessment by a member of the study team was 5.2 days.

A final diagnosis of acute cerebrovascular disease was made in 276 of the 369 events. Among the 93 events in which the final diagnosis was not acute cerebrovascular disease, nine were believed to be attacks of migraine, 10 were seizures, and eight were vasovagal episodes; there were numerous other conditions that occurred less frequently. Of the 276 events with a final diagnosis of acute cerebrovascular disease, 57% were a first stroke, 27% a recurrent stroke, 14% a TIA, and 3% amaurosis fugax. Annual age-standardized event rates per 100,000 population for first stroke were 120 (95% confidence limit [CL] 95–145) for males and 56 (95% CL 40–72) for females. For the population aged 35 years or older the event rates were 306 (95% CL 241–371) for men and 147 (95% CL 106–188) for
women; 40% of the events occurred in persons younger than 65 years. Age-specific annual incidence rates are presented in Table 2.

In 28% of the 229 events with a final clinical diagnosis of stroke, the pathologic basis could not be ascertained because either the appropriate investigations were not performed or because they were unhelpful; 70% of the patients with a completed stroke underwent a cranial CT scan at the instigation of the doctor in charge of their care. As indicated in Table 3, lacunar infarction accounted for 28% of all strokes, large-artery thromboses 20%, embolization 12%, and hemorrhagic strokes including subarachnoid hemorrhage 12%. There were no significant differences in the apparent pathologic bases of first and recurrent strokes.

Of the 276 events in which a final diagnosis of acute cerebrovascular disease was made, 19% were managed at home or by relatives (55% of the 47 persons with a final diagnosis of TIA were managed in this way), 76% resulted in admission to an acute-care hospital, 4% were managed in nursing homes, and one case of subarachnoid hemorrhage was medically unattended and came to light through coroner's records.

Of the 154 persons in whom a final diagnosis of first stroke was made, 23% died within 28 days of the index event. Case-fatality ratios at 28 days varied from 57% for subarachnoid hemorrhage to 0% for lacunar infarction (Table 3).

**Discussion**

Our study has confirmed that it is necessary to compile a community-based register if one is to obtain an accurate picture of the burden of cerebrovascular disease borne by the community. Surveillance only of admissions to hospital with stroke would seriously underestimate the true incidence and event rates for this condition, partly because a sizable proportion of persons, both surviving and dying of stroke, is never admitted to a hospital, and partly because there is often considerable cross-boundary flow to other hospitals. Even if the policies of individual medical practitioners concerning admission of persons with stroke to a hospital remain static, changes in the incidence of stroke in the entire community might not be reflected in hospital statistics if the early case-fatality ratio changed simultaneously. Since stroke remains a major cause of severe and chronic disability in our community, it is extremely important that those responsible for the planning of health services for this condition have accurate and complete data on which to base their calculations.

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**Table 1. Hospital Admissions for Stroke, Perth Statistical Division and Study Area, 1984–1985, Age Groups 35 Years or Older**

<table>
<thead>
<tr>
<th></th>
<th>Admissions for stroke</th>
<th>Total bed-days</th>
<th>Mean stay (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Males</td>
<td>Females</td>
<td>Males</td>
</tr>
<tr>
<td>Perth</td>
<td>682</td>
<td>410</td>
<td>14,417</td>
</tr>
<tr>
<td>Study area</td>
<td>644</td>
<td>430</td>
<td>16,478</td>
</tr>
</tbody>
</table>

*Annual age-standardized rates per 100,000 (‘‘world’’ population).

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**Table 2. Annual Incidence Rates for Stroke During 10 Months in 1986, Study Area, Perth, Western Australia**

<table>
<thead>
<tr>
<th>Age group (yr)</th>
<th>Males Incidence rate</th>
<th>Cases</th>
<th>Females Incidence rate</th>
<th>Cases</th>
<th>Total Incidence rate</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>30–34</td>
<td>66</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>34</td>
<td>3</td>
</tr>
<tr>
<td>35–39</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>40–44</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>45–49</td>
<td>160</td>
<td>5</td>
<td>64</td>
<td>2</td>
<td>112</td>
<td>7</td>
</tr>
<tr>
<td>50–54</td>
<td>133</td>
<td>4</td>
<td>37</td>
<td>1</td>
<td>87</td>
<td>5</td>
</tr>
<tr>
<td>55–59</td>
<td>271</td>
<td>8</td>
<td>73</td>
<td>2</td>
<td>175</td>
<td>10</td>
</tr>
<tr>
<td>60–64</td>
<td>519</td>
<td>14</td>
<td>301</td>
<td>8</td>
<td>411</td>
<td>22</td>
</tr>
<tr>
<td>65–69</td>
<td>478</td>
<td>9</td>
<td>319</td>
<td>7</td>
<td>392</td>
<td>16</td>
</tr>
<tr>
<td>70–74</td>
<td>1,119</td>
<td>18</td>
<td>546</td>
<td>12</td>
<td>788</td>
<td>30</td>
</tr>
<tr>
<td>75–79</td>
<td>1,283</td>
<td>15</td>
<td>566</td>
<td>10</td>
<td>852</td>
<td>25</td>
</tr>
<tr>
<td>80–84</td>
<td>1,721</td>
<td>10</td>
<td>851</td>
<td>10</td>
<td>1,139</td>
<td>20</td>
</tr>
<tr>
<td>85+</td>
<td>2,053</td>
<td>7</td>
<td>981</td>
<td>9</td>
<td>1,272</td>
<td>16</td>
</tr>
</tbody>
</table>

Total (all ages)

Crude rate 166.6 93 108.1 61 137.2 154
Standardized rate* 120.2 55.9 84.7
95% confidence limit 95.1-145.4 40.3-71.6 70.4-98.9

Incidence rate per 100,000 person-years using data from 1986 census as denominator.

*Standardized to ‘‘world’’ population by direct method.
As Bonita et al. have pointed out, multiple sources for ascertaining cases should be used if a community-based register is to be complete. Although we had good cooperation from general practitioners serving our study area (16% of first reports came from this source), many doctors indicated that they relied on us to detect cases admitted to a hospital rather than notifying us directly. As it was, 7% of all events registered came to light only from scanning computerized records of hospital separations. However, many of these admissions were to small private hospitals or to hospitals outside Perth, institutions that would be impractical to contact on a daily basis.

Since the age and sex structure of our study population and the pattern of admissions to hospitals of persons with cerebrovascular disease from the study area over recent years have both been typical of the whole of Perth, we are confident that our results accurately reflect the incidence and case-fatality rates at 28 days for persons with cerebrovascular disease from the population and the pattern of admissions to hospitals or to hospitals outside Perth, institutions.

<table>
<thead>
<tr>
<th>Register</th>
<th>Year</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perth, Australia*</td>
<td>1986</td>
<td>122</td>
<td>93</td>
</tr>
<tr>
<td>Auckland, New Zealand</td>
<td>1981–1982</td>
<td>112</td>
<td>264</td>
</tr>
<tr>
<td>Melbourne, Australia</td>
<td>1978–1979</td>
<td>165</td>
<td>245</td>
</tr>
</tbody>
</table>

Incidence rates per 100,000 population standardized to "world" population. 
*Figures differ from those in Table 2 because different age groups were used for calculating age-standardized rates.
with first intracerebral hemorrhage and persons in whom the pathologic process of the first stroke was unknown was similar to that of those with subarachnoid hemorrhage, but the latter group almost certainly represents a mixture of pathologies, some having a worse and some a better outlook than cases of proven intracerebral hemorrhage.

Despite a long-established downward trend in mortality from stroke, it remains a common condition in our community. Some 40% of cases occur in people of working age and, overall, stroke continues to have a sizable early case-fatality ratio. Many survivors are elderly and are likely to have some degree of persistent disability causing long-term dependence on relatives, health services, or both. Hospital-based registers of stroke are likely to underestimate the true incidence of the condition and to give an incomplete picture of the outcome. If we are to understand the changing pattern of cerebrovascular disease and to plan health services for victims of stroke rationally, it is necessary as a first step to have an accurate picture of the true extent of the problem. Such a picture can be obtained only from a community-based register with complete follow-up.

Acknowledgments

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References


KEY WORDS • cerebrovascular disorders • epidemiology • Western Australia
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