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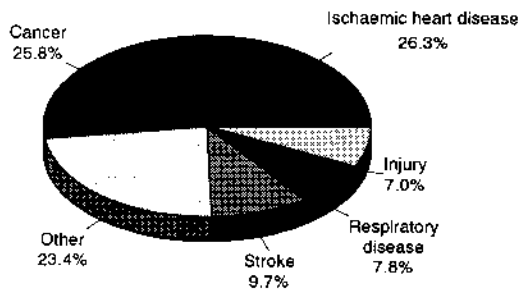
Epidemiology and Health Information Branch
CIRCULAR

STROKE MORTALITY AND IMPLICATIONS FOR PREVENTION AND TREATMENT

Stroke is the third major cause of death in Queensland (Figure1) and is also an important cause of hospitalisation and disability. Trends, current regional patterns and mortality differentials for cerebrovascular diseases(CVD) are described and the implications for primary and secondary prevention are reviewed.

Figure 1: Major causes of death, Queensland, 1992

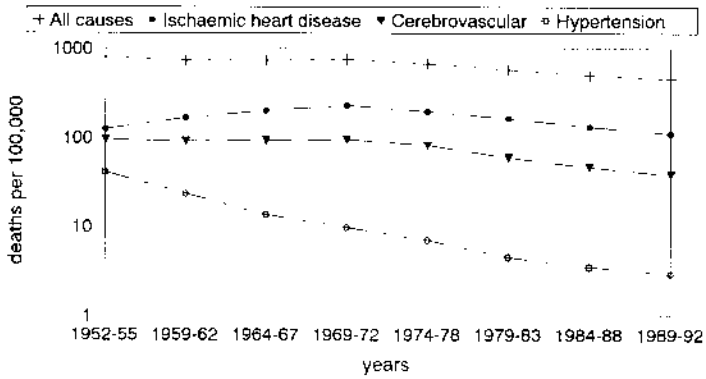


Source: Epidemiology and Health Information Branch, Queensland Health

Trends in stroke mortality and incidence

There have been substantial improvements in the mortality rates from each of the major vascular causes of death in Queensland since the late 1960s (Figure 2). In the past two decades, the relative decline has been greater for stroke (60% reduction) compared to the 51% decline in the ischaemic heart disease (IHD) mortality rates.

Figure 2: Trends in Queensland mortality*, persons, 1954 - 1992



* death rates standardised to world population
Source: Epidemiology and Health Information Branch, Queensland Health

However, in absolute terms, the health gain has been greater for ischaemic heart disease. Over the period 1981-82 to 1991-92, the reduction in IHD mortality was responsible for 45% of the overall improvement in the all cause rates, whereas the gain for cerebrovascular rates was 22%. Therefore, *these two causes together contribute to over two thirds of the recent health gains in the mortality from all causes.*

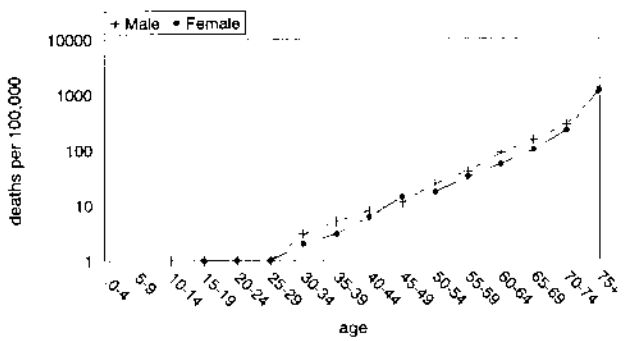
The continuing decline in mortality is explained partly by changes in the incidence of CVD, but also must be due to improvements in case fatality[1].The major determinants probably relate to declines in the risk factors leading to CVD eg. increasing treatment of hypertension, decreasing prevalence of cigarette smoking and changes in the diet[2] although more recent treatment interventions may also be playing a part.

There are recent findings from some of the population stroke registers[3,4,5,6,7,8,9] that the decline in incidence may have plateaued and may even be reversing in some subgroups of the population.

Ageing of the population and the burden of stroke

The burden of stroke is greatest in the elderly (Figure 3). There is a 130 fold increase in risk of death from stroke for males and a 190 fold increase in risk for females when comparing those aged over 75 years with people 40 years of age. Of those aged 45 years, almost one in four men and in one in five women can expect to have a stroke if they live to their 85th year[1]. The greatest number of deaths occurs in the elderly with 70% of stroke deaths occurring in people over 75 years.

Figure 3: Age specific stroke mortality rates by sex, Queensland, 1989 - 92



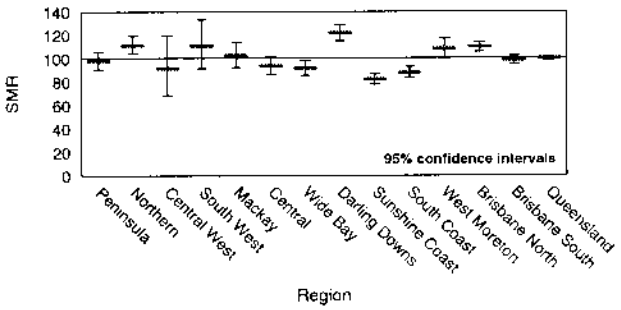
Source: Epidemiology and Health Information Branch, Queensland Health

Due to the rising incidence of stroke with age and the ageing of the population, the possibility of an increased impact on the health services needs to be considered. Some groups have tried to predict the possible impacts by investigating population projections and stroke trends. The Oxfordshire community stroke register[10] concluded that, for first ever strokes, the impact could affect the acute care services more than the disability services.

Regional patterns

The regional patterns of age standardised stroke mortality in Queensland are shown in Figure 4. There are above average rates in the Northern, Darling Downs and Brisbane North Regions and below average rates in the Wide Bay, Sunshine and South Coast Regions. These variations bear some similarity to the regional rates for ischaemic heart disease (see Circular No.8).

Figure 4: Standardised* mortality ratios (SMR) for stroke by health Region, persons, Qld 1986 - 1992



* Indirectly age standardised to 1989 Qld population
Source: Epidemiology and Health Information Branch, Queensland Health

Mortality differentials

Figure 5 shows a socioeconomic gradient in the age standardised death rates for stroke in Queensland in the most recent data period. In males, the most disadvantaged males have a 33% greater death rate than the least disadvantaged as measured by the Index of

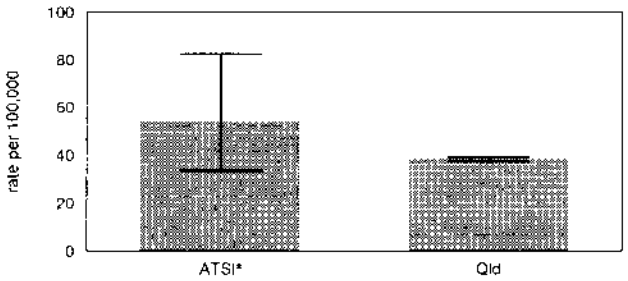
Figure 5: Standardised* death rates for stroke by socioeconomic status, Queensland, 1989 - 1992

* age standardised to world population
# SEIFA 1991 Socioeconomic Index for Areas, summarises variables relating to home ownership family income and other features of the household structure
Source: Epidemiology and Health Information Branch, Queensland Health

Economic Resources. For females, there is a 20% excess death rate for the most disadvantaged.

Figure 6 compares the standardised death rates for Aboriginal and Torres Strait Islander populations with the overall Queensland population. The Aboriginal and Torres Strait Islander rates are higher, but the difference is not statistically significant.

Figure 6: Standardised# death rates for stroke by ATSI* status, Queensland, 1989 - 1992

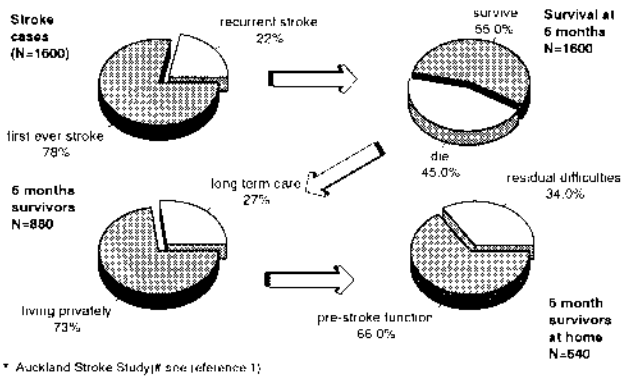


age standardised to world population
* Based on SLAs where >50% of population identifies as Aboriginal or Torres Strait Islander. Aurukun, Burke, Carpentaria, Mornington, Townsville (C) Balance
Source: Epidemiology and Health Information, Queensland Health

Impact of stroke in the community

Data from the Auckland Stroke Study[1] gives a perspective on the burden of stroke in a population of 1 million (see Figure 7). Each year, 1250 people will experience their first ever stroke with another 350 people suffering a recurrent episode. Of these 1600 people, only 880 will survive six months. Of these 880 survivors, 27% will be in long term care and 73% will be living in a private residence. Of the 640 living in the home, only two thirds regard themselves as back to their pre-stroke functional capacity.

Figure 7: Predicted* stroke numbers in a population of 1 million in one year



* Auckland Stroke Study(# see reference 1)

Risk factors and primary prevention strategies

Primary prevention is important because risk factors such as elevated blood pressure, smoking and high cholesterol levels are important determinants of incident cases Epidemiological studies have demonstrated a reduction in the number of cases following intervention.

High blood pressure has been the most extensively researched risk factor and is associated with increased risk in both ischaemic and haemorrhagic stroke[11,12,13,14]. Other reviews have cautioned that it does not explain all the risk reduction[15,16,17].

When considering the options for primary prevention strategies, there is debate regarding the relative merits of adopting a strategy which targets those known to be at high risk versus a strategy targeting the general population[11]. Using blood pressure as an example, it is argued by some that greater reductions in stroke would be achieved by mass strategies because targeting the larger proportions of the community with blood pressures in the middle of the population distribution would bring larger absolute gains[18,19].

However, such strategies depend on significant changes in dietary intakes, eg. of salt, which may not be easily achievable in practice. Therefore, the treatment of the higher risk individuals in the community with conventional antihypertensive agents is still essential even though smaller numbers of people are involved. Public health programs need to embrace the control of hypertension as well as the reduction of blood pressure in the whole population.

A New Zealand review[20] has recommended that decisions to treat raised blood pressure should be based on the estimated absolute risk of cardiovascular disease rather than on blood pressure alone. The risk of stroke associated with raised blood pressure is affected by the presence of other risk factors. Such a strategy may lead to an increased proportion of older people (older than 60 years) being treated with antihypertensives and a reduction in the proportion of younger people, especially women, receiving treatment.

The reduction of smoking, the reduction of excess alcohol intake and dietary measures are also likely to be beneficial.[11]

A meta-analysis of the smoking studies[21] showed the overall risk elevation from smoking for ischaemic stroke was 1.9 times the risk in non-smokers. A dose response relation has been noted with increasing smoking levels[21,22] and some investigators[23] have noted that the

excess risk declines slowly over some years when smokers quit.

The cholesterol relation raises the importance of considering the different subtypes of stroke (see Appendix) when assessing its role as a risk factor in stroke. Serum cholesterol appears to be related to death from ischaemic stroke, but a more complex relation exists for haemorrhagic stroke.[11,22,24] In one prospective study[24], total cholesterol was positively associated with risk of non-haemorrhagic events , but only for persons with cholesterol levels >8mmol/L.

Other risk factors for stroke are discussed in references 11 and 25.

Secondary prevention

Secondary prevention of stroke is of increasing importance because of the possible greater absolute benefits that may accrue in populations of symptomatic patients[26]. General measures such as lifestyle modification (eg. smoking reduction) continue to be relevant, but specific interventions (medical and surgical) are also important.

Recent evidence suggests that cardioembolic strokes are among the most preventable causes of brain ischaemia[27]. Echocardiography has served to identify the importance of this group and clinical trials have shown nonvalvular atrial fibrillation provides the best opportunities for prevention.

15% of ischaemic strokes may be due to this cause (P Glasziou pers. comm.) and the trial data suggests that a decrease of 50% to 75% in stroke or all cause mortality can be achieved by warfarin prophylaxis.[28] Warfarin is strongly recommended for persons with nonvalvular atrial fibrillation who are over 60 years old, especially those with other associated disease risk factors (eg. hypertension, history of transient ischemic attack [TIA] or stroke, evidence of left ventricular dysfunction, etc), but its use may need to be tempered in those over 75 years old.

Other embolic causes of stroke include a history of a TIA or mild stroke, particularly those associated with a high grade carotid artery stenosis, and embolic stroke following myocardial infarction. For persons with TIA or minor stroke, aspirin reduces recurrence and reduces mortality. For patients post myocardial infarction, aspirin is recommended for the prevention of recurrent myocardial infarction not for stroke[28,37].

The role of surgical intervention (ie. carotid endarterectomy) is a more uncertain one. There is evidence of benefits for high grade lesions in symptomatic patients when surgery is done in low risk settings. The trials are still under way for mid-range stenosis.[26,28] The role of surgery in asymptomatic stenosis is less clear.

Stroke units also save lives

A meta-analysis of clinical trials which compared the management of stroke in special acute units with care in general wards showed a reduction in both short and longer term mortality of 28% (at four months) and 21% (at 12 months) for those treated in the special units[29,30]. Preliminary indications are that disability may also be reduced in survivors treated initially in a unit.

Implications for policy and program development

1. Several reviewers have commented on the attitude of nihilism which has prevailed in the recent past with respect to the prospects for achieving better stroke outcomes either by treatment approaches or by preventive programs. This review points to the scope of potential health gains that could be realised by primary and secondary prevention strategies.

2. Queensland had a 40% excess in stroke mortality over the lowest OECD country (Canada) in 1988[31]. In 1991/92 data, Western Australia (WA) had the lowest mortality from cerebrovascular disease among the Australian states. The Queensland death rates were 11% higher than the WA rates[32]. These **benchmark figures** suggest there still is **potential for further health gains**.

3. *In Queensland Progress in Achieving National Health Goals & Targets for Preventable Mortality and Morbidity*[36]**, trends in Queensland mortality** for people aged 30 to 64 years from 1981-1991 showed our rates place us on target for the Year 2000 national targets. The potential health gains referred to in item 2 indicate the potential gains that may be attained in older population groups.

4. This report has emphasised the **importance of primary prevention in the control of stroke in the population**. Historically, medical treatment of hypertension has been vital to the achievement of declines in stroke incidence. More recent work is emphasising that other risk factors such as smoking and excessive alcohol intake be included in prevention programs. Such a strategy may be more cost effective at the population level.

5.Evidence is accumulating about the importance **of secondary prevention strategies in high risk subgroups** (eg. warfarin prophylaxis in nonvalvular atrial fibrillation,

especially in elderly people). Other strategies such as aspirin prophylaxis and consideration of surgical intervention in selected high risk groups following the presentation of symptomatic disease need to be considered.

6. The organisation of hospital acute care for stroke into specialised units has been shown to provide benefits in terms of mortality.

7. The NHMRC[33] recommends that **blood pressure be measured** in all adults aged 16 years and over. Screening should be repeated every two years if diastolic pressure>85 mm Hg and systolic>140. More frequent measurement is indicated for those with higher diastolic readings. Other examinations such as clinical auscultation of the carotid arteries or ultrasound scanning were also considered by the NHMRC Working Party. Community screening of people for asymptomatic carotid artery disease is not recommended.

8. The Royal Australasian College of Physicians in conjunction with the Australian Council of Health Care Standards Care Evaluation Program[34] have recently released **clinical indicators** that have been endorsed for the evaluation of hospital investigation and care of stroke. These indicators measure the rate of CAT scanning, the case fatality rate and the rate of discharge to rehabilitation units and nursing homes.

Appendix

The WHO definition of stroke is rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than of vascular origin[1].

It is important to be aware of which stroke subtypes have been included in the definitions when comparing incidence and mortality rates from the literature. The data presented in the figures refers to all categories of stroke ICD 430-438 (includes subarachnoid hemorrhage SAH & transient ischemic attack TIA).

Cerebral infarction (ischaemic stroke) due to thromboembolism is responsible for 80% of stroke[35]. It is important to distinguish ischaemic stroke from hemorrhagic stroke because many of the secondary prevention strategies are contraindicated if hemorrhage is the cause.

The specific treatments indicated depend on the underlying mechanisms responsible for the predisposition to ischaemic stroke. 30% of ischaemic stroke result from atherothrombosis in the extracranial and larger intracranial arteries, 20% to 25% are of cardioembolic origin and 15% to 20% (lacunar strokes) are related to small intracranial vessel changes associated with longstanding hypertension[37].

A good overview of most aspects of stroke is provided in the Octet series of articles in the *1992 Lancet*.

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