Trends in Stroke Incidence in Auckland, New Zealand, During 1981 to 2003

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Background and Purpose—Long-term trends in stroke incidence in different populations have not been well characterized, largely as a result of the complexities associated with population-based stroke surveillance.

Methods—We assessed temporal trends in stroke incidence using standard diagnostic criteria and community-wide surveillance procedures in the population (~1 million) of Auckland, New Zealand, over 12-month calendar periods in 1981–1982, 1991–1992, and 2002–2003. Age-adjusted first-ever (incident) and total (attack) rates, and temporal trends, were reported with 95% confidence intervals (CIs). Rates were analyzed by sex and major age groups.

Results—From 1981 to 1982, stroke rates were stable in 1991–1992 and then declined in 2002–2003, to produce overall modest declines in standardized incidence (11%; 95% CI, 1 to 19%) and attack rates (9%; 95% CI, 0 to 16%) between the first and last study periods. Some favorable downward trends in vascular risk factors such as cigarette smoking were counterbalanced by increasing age and body mass index, and frequency of diabetes, in patients with stroke.

Conclusions—There has been a modest decline in stroke incidence in Auckland over the last 2 decades, mainly during 1991 to 2003, in association with divergent trends in major risk factors. (Stroke. 2005;36:2087-2093.)

Key Words: epidemiology ■ incidence ■ New Zealand ■ stroke ■ trends

T here is continued uncertainty about how well strategies to modify vascular risk have produced fewer and/or less severe primary and recurrent strokes. Although stroke mortality rates have fallen substantially in many Western countries over recent decades,1 stroke incidence has not declined to the same extent, and there has been a recent leveling off in some positive trends, or even an increase in age- and sex-specific rates in some populations.2 The factors responsible for the discordant secular trends in stroke incidence and mortality have not been adequately quantified, largely as a result of the difficulties in undertaking population-based stroke surveillance.3,4

The stroke incidence and outcome studies undertaken in Auckland, New Zealand (NZ), in 1981–19825 and 1991–19924,6 are among a limited number of high-quality, population-based stroke studies.2,3 Previous analyses suggested that improvements in short-term survival was the chief factor responsible for declines in stroke mortality but with divergent changes in sex-specific incidence also apparent: declining rates for younger males and increasing rates for older females.6 In this article, we expand on these investigations by including data from a third study undertaken in 2002–2003.

Subjects and Methods

Overview

The recent Auckland Regional Community Stroke (ARCOS) study used a prospective, population-based register to ascertain all cases of first-ever (incident) and recurrent stroke events that occurred among adults in the “usually resident” population (aged ≥15 years) of Auckland over the 12-month period, March 1, 2002, to February 28, 2003. The study used standard diagnostic criteria and multiple overlapping methods of case ascertainment like in the previous studies undertaken over similar calendar periods in 1981–1982 and 1991–1992.4,6 Briefly, the 1981–1982 study used a cluster sample of 50% of all registered primary care general practitioners (GPs) in Auckland to identify a representative sample of half of all stroke events in the population; a total of 703 strokes were registered in 680 patients. The 1991–1992 study used a register of all stroke events managed in hospital and a cluster sample of 25% of all GPs to estimate the total number of...
“nonhospitalized, nonfatal” stroke events; a total of 1803 strokes were registered in 1761 patients (after adjustment for sampling). For the 2002–2003 study, efforts were made to identify all new nonhospitalized cases of stroke or transient ischemic attacks (TIAs) by maintaining regular contact with all GPs in the study region. The Auckland Ethics Committee approved each of the studies and written informed consent was obtained from all patients or from a next of kin when patients were dead or severely disabled.

Study Population
Auckland is the largest city in NZ (approximately 940,000 people aged ≥15 years according to the 2001 Census). The population has been served consistently by only 4 large public acute care hospitals, 2 public medical specialist hospitals, 2 main private medical acute care hospitals, and a large number of long-stay residential care facilities over the study periods.

Definitions
In all studies, stroke was defined according to the World Health Organization (WHO) definition as “rapidly developing clinical signs of focal (or global) disturbance of cerebral function lasting more than 24 hours (unless interrupted by surgery or death) with no apparent cause other than of vascular origin.” This definition excludes cases of “silent stroke” detected by neuroimaging without appropriate clinical features and cases of TIA (ie, neurologic deficits lasting <24 hours). Any stroke that developed within 28 days of a previous event was considered as “progressing stroke” and was not recorded as a new event, but deficits that developed ≥28 days were regarded as a recurrent event. A case managed in the hospital was defined as a registered admission to hospital within 28 days of the onset of stroke. Case fatality was defined as the proportion of registered patients with stroke who died within 28 days of the onset of stroke.

Case-Finding Procedures
A combination of “hot pursuit” (screening of hospital admissions and referrals from GPs, the coroner’s office, and community healthcare providers and organizations as stroke events occurred) and “cold pursuit” (retrospectively identifying and extracting information from medical records) were used to ascertain cases of stroke. All possible efforts were made to collect information on nonhospitalized patients with stroke. To this end, frequent regular contacts were made with GPs, residential care facilities, and community organizations, and a variety of promotional activities, including seminars and newsletters, were used to raise awareness of the study and encourage early notification to a central office. To manage the large number of community contacts in the most recent study, we maintained an electronic database of names and used e-mail and facsimile as the principal means of monthly contact with GPs and community organizations as compared with telephone in previous studies. They were asked to report all patients with stroke or TIA once they had obtained consent for disclosure of information. In addition, computerized databases for presentations, admissions, and separations in hospitals, and a centralized register of deaths, avoided the need for manual prospective searches of medical reports and death certificates as had been used in previous studies. Final checks for completeness of case ascertainment were made by conducting quarterly searches up to 6 months after the end of the study period among residents of Auckland who were listed in 2 public databases held by the New Zealand Health Information Service: the national separations database of all public and private hospitals and the mortality register for any mention of stroke as a primary or secondary diagnosis.

Assessment Procedures
Trained and supervised study nurses undertook face-to-face interviews with patients or, when the patient was dead or disabled, the partner or next of kin as soon as possible after notification of a stroke. A structured questionnaire, with item definitions consistent across the 3 studies, was used to obtain information regarding demographics, clinical features, management, and major risk factors. Patients who were not known to have died were followed up by telephone (or by interview for a minority of cases) at 1 and 6 months after the index event.

Statistical Analyses
All original data from the 1981–1982 and 1991–1992 studies were reviewed and reanalyzed, and rates recalculated using standardized methods and published Census data. Descriptive statistics were used to assess (unadjusted) trends in the characteristics of patients with stroke across the studies: significance of trends in the distribution of categorical variables was tested using the Cochrane-Armitage method and in continuous variables with Kruskal-Wallis nonparametric analysis of variance. Crude annual incidence (first-ever events) and attack (all events) rates per 100,000, together with 95% confidence intervals (CIs), were calculated using Poisson distribution, with adjustments made for the sampling procedure in each of the first 2 ARCOS studies (crude numbers were multiplied by 2 for the 1981–1982 study and nonhospitalized, nonfatal cases were weighted by 4 for the 1991–1992 study). Standardized rates were calculated by the direct method of adjusting to the age distribution of the WHO “world” population and by the indirect method with adjustment to the age and sex structure of the population of Auckland in 2001. Standardized rate ratios (2002–2003 to 1981–1982) were calculated to examine the overall difference in rates and tests of the heterogeneity in particular subgroups were performed.

Data Quality
Capture recapture was used to assess the completeness of the case ascertainment in the 3 studies using log-linear modeling assuming a Poisson distribution. The sources of notification were combined into 4 main sources of notification: (1) hospital admissions and discharges, (2) death certification or coroners’ reports, (3) general practitioner, and (4) other community notifications such as private residential care facilities, service providers, and charitable organizations. The final model with the least deviance for the 3 studies included the main effects of the 4 sources with a 3-way interaction among hospital, death certification, and GPs. In addition, we
examined the quality of the data using WHO Monitoring Trends and Determinants in Cardiovascular Disease (MONICA) criteria of†‡: (1) 28-day case fatality (<40%), (2) the proportion of fatal cases that occurred outside the hospital (>10%), (3) the proportion of nonfatal, nonhospitalized cases (≥5%), and (4) the proportion of fatal cases examined by a physician before death or subject to autopsy.

### Results

Overall, 1406 strokes among 1360 patients (after adjustment for sampling) were registered in 1981–1982, 1803 events among 1761 patients in 1991–1992, and 2001 events among 1938 patients in 2002–2003. There were significant changes in the patterns of healthcare delivery and adaptations to the complexities of surveillance research over time (Table 1). Yet, all 3 studies fulfilled MONICA quality criteria, and there was a progressive decline in both early case fatality and the proportion of missed cases estimated from capture recapture analyses.

There was an increase in the average age of patients, and a decline in the proportion of self-identified NZ/European people, with correspondingly increasing proportions of patients of other ethnic origins, in particular Pacific and “other” peoples, in which the proportions increased nearly 4- and 8-fold, respectively, over the study periods (Table 2). Although the proportion of current smokers declined from 27.7% in 1981 to 1982 to 14.0% in 2002 to 2003, average body mass index (BMI) and proportions of people with a history of high blood pressure and diabetes increased markedly. Histories of myocardial infarction or stroke were stable over the 3 studies. Significant changes in the patterns of management of patients with stroke were clearly evident, with the proportions of patients admitted and managed in hospital and having neuroimaging increasing over the study periods.

Adjusted incidence and attack rates were essentially stable from 1981–1982 to 1991–1992 and then declined in 2002–2003 (Table 3). Indirect standardization of rates, which takes into account the age and sex structure of the Auckland population, and places greater weight on older age groups compared with direct standardization, produced slightly higher overall rates and showed a near significant, nonlinear, downward trend across the studies. The figure shows direct standardized rate ratios, 2002–2003 to 1981–1982, by sex, age group, and sequence of stroke. Overall, there were modest decreases in standardized incidence of 11% (95% CI, 1 to 19%) and attack rates 9% (95% CI, 0 to 16%), respectively, over the study periods. There was no significant heterogeneity in rate ratios for the major age groups, indicating that the decline in rates for the age group 75 to 84 years was most likely related to chance (Table 3; Figure).

The increasing stroke rates with increasing age, and the higher rates in males compared with females, were consistent across all 3 studies (Table 4). There were sex-specific differences in temporal trends in rates: for males, there was a
significant 16% (95% CI, 2 to 27%) decline in incidence and 14% (95% CI, 2 to 24%) decline in attack rates across the study periods; but for females, these rates increased in 1991–1992 and then declined in 2002–2003, to produce nonsignificant differences in rates between the beginning and end of the study period (Figure).

**Discussion**

This study supports accumulating evidence of heterogeneity in temporal trends in stroke rates in different populations and geographic areas. Declines in stroke incidence, although not to the same extent as stroke mortality, have been reported from the United States, Asia, Europe, and Australia, with some studies also showing a subsequent deceleration of these trends during recent decades.\(^1\)\(^2\) The declines in rates in Auckland is consistent with most studies undertaken in the more homogeneous but older populations of Northern Europe\(^15\)–\(^17\) and are within the estimates surrounding the recently reported large (29%) decline in stroke incidence in Oxfordshire, England, over 20 years.\(^16\)

In showing modest declines in overall stroke incidence and attack rates in Auckland over 2 decades, this unique population-based series provides some feedback on the success of strategies to modify the risk of primary and secondary stroke on a background of structural and other changes in the population. However, the divergent trends in the frequencies of exposures provide mixed views as to scenarios regarding the future burden of stroke, both locally and regionally.

Although routine mortality statistics are useful for making national and international comparisons of trends in the epidemiology of stroke,\(^3\) it is important that stroke is also studied in a population-wide context because many patients survive the acute phase with residual disability and a large proportion of the burden of care occurs outside of the hospital sector.\(^3\)\(^4\) Changes in diagnostic coding practices and referral patterns can significantly distort trends derived solely from hospital-based data. Yet, there are few population-based studies when compared with the number of studies that use mortality or hospital-based register data, and the limited number of stroke incidence studies that fulfill certain “ideal” criteria further reflects the complexities to such investigations.\(^2\)\(^–\)\(^4\)

To what extent can the trends in stroke rates be accounted for by changes in prevalence of known risk factors in Auckland? On the basis of epidemiologic data of a strong, direct, and near-continuous association between stroke incidence and level of blood pressure, which can be reversed within a few years of blood pressure-lowering, nonoptimal control of blood pressure levels is considered the most important risk factor for stroke, accounting for almost two thirds of the global burden of stroke.\(^18\)

### TABLE 2. Baseline Characteristics, Medical History, and Management of Patients With Stroke in Each Study

<table>
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<tbody>
<tr>
<td></td>
<td>(n = 1360)</td>
<td>(n = 1761)</td>
<td>(n = 1938)</td>
<td>P Value*</td>
</tr>
<tr>
<td>Male</td>
<td>662 (48.7)</td>
<td>817 (46.4)</td>
<td>892 (46.0)</td>
<td>0.15</td>
</tr>
<tr>
<td>Age, mean (±SD), y</td>
<td>71.2 (13.3)</td>
<td>71.6 (13.5)</td>
<td>73.0 (13.8)</td>
<td>&lt;0.001†</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
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<tr>
<td>NZ/European</td>
<td>1248 (91.8)</td>
<td>1532 (87.0)</td>
<td>1431 (73.8)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Maori</td>
<td>60 (4.4)</td>
<td>82 (4.7)</td>
<td>102 (5.3)</td>
<td></td>
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<tr>
<td>Pacific Island</td>
<td>32 (2.4)</td>
<td>111 (6.3)</td>
<td>197 (10.2)</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>20 (1.5)</td>
<td>36 (2.0)</td>
<td>162 (8.4)</td>
<td></td>
</tr>
<tr>
<td>Married/partnered</td>
<td>680 (50.0)</td>
<td>857 (48.7)</td>
<td>963 (51.7)</td>
<td>0.28</td>
</tr>
<tr>
<td>Medical history</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>High blood pressure</td>
<td>700 (51.5)</td>
<td>910 (52.1)</td>
<td>1079 (59.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>156 (11.5)</td>
<td>288 (16.5)</td>
<td>240 (12.7)</td>
<td>0.55</td>
</tr>
<tr>
<td>Stroke</td>
<td>330 (24.3)</td>
<td>456 (25.9)</td>
<td>477 (24.6)</td>
<td>0.91</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>134 (10.0)</td>
<td>236 (13.6)</td>
<td>329 (17.4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Current smoker</td>
<td>374 (27.7)</td>
<td>411 (23.5)</td>
<td>241 (14.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Body mass index, mean (±SD)‡</td>
<td>23.7 (4.6)</td>
<td>24.1 (4.9)</td>
<td>25.6 (5.9)</td>
<td>&lt;0.001†</td>
</tr>
<tr>
<td>Management</td>
<td></td>
<td></td>
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<tr>
<td>Admission to hospital</td>
<td>850 (62.5)</td>
<td>1276 (72.5)</td>
<td>1757 (91.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Neuroimaging, CT/MRI</td>
<td>162 (21.0)</td>
<td>541 (41.9)</td>
<td>1694 (87.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Necropsy</td>
<td>142 (10.4)</td>
<td>41 (2.3)</td>
<td>24 (1.2)</td>
<td>&lt;0.001</td>
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*P value calculated using Cochran–Armitage trend test.
†P value calculated using analysis of variance.
SD indicates standard deviation; CT, computed tomography; MRI, magnetic resonance imaging.
though there was an increase in the proportion of stroke patients with a history of high blood pressure across the 3 ARCONS studies, these data are confounded by changes in diagnostic criteria for hypertension and a lowering in the threshold to commence treatment over time. There is some evidence of a decline in blood pressure levels, albeit within selected subsets of the NZ/European population of Auckland during the 1980s, whereas the Oxfordshire study has shown that declines in stroke rates were associated with favorable trends in premorbid blood pressure levels and use of blood pressure-lowering medication. However, many other studies have documented suboptimal levels of awareness and control of high blood pressure levels within communities, and that control of blood pressure levels in the years following stroke was linked to long-term survival.

### TABLE 3. Age-Specific Annual Rates per 100 000 Population of First-Ever and Total Stroke Events in Auckland, New Zealand, 1981 to 2003

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<tr>
<td></td>
<td>N</td>
<td>n</td>
<td>Rate (95% CI)</td>
<td>N</td>
</tr>
<tr>
<td>15–64</td>
<td>518 112</td>
<td>286</td>
<td>55 (46–64)</td>
<td>624 828</td>
</tr>
<tr>
<td>65–74</td>
<td>49 812</td>
<td>260</td>
<td>522 (432–612)</td>
<td>56 388</td>
</tr>
<tr>
<td>75–84</td>
<td>22 965</td>
<td>350</td>
<td>1524 (1298–1750)</td>
<td>31 701</td>
</tr>
<tr>
<td>85+</td>
<td>5691</td>
<td>134</td>
<td>2355 (1791–2918)</td>
<td>8541</td>
</tr>
<tr>
<td>Total</td>
<td>596 580</td>
<td>1030</td>
<td>173 (158–188)</td>
<td>721 458</td>
</tr>
<tr>
<td>Adjusted (Auckland*)</td>
<td>177</td>
<td>(162–192)</td>
<td>176</td>
<td>(164–189)</td>
</tr>
<tr>
<td>Adjusted (World†)</td>
<td>156</td>
<td>(143–170)</td>
<td>156</td>
<td>(145–167)</td>
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<td>49 812</td>
<td>380</td>
<td>763 (654–871)</td>
<td>56 388</td>
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<tr>
<td>75–84</td>
<td>22 965</td>
<td>498</td>
<td>2169 (1899–2438)</td>
<td>31 701</td>
</tr>
<tr>
<td>85+</td>
<td>5691</td>
<td>178</td>
<td>3128 (2478–3778)</td>
<td>8541</td>
</tr>
<tr>
<td>Total</td>
<td>596 580</td>
<td>1406</td>
<td>236 (218–253)</td>
<td>721 458</td>
</tr>
<tr>
<td>Adjusted (Auckland*)</td>
<td>244</td>
<td>(226–262)</td>
<td>244</td>
<td>(229–259)</td>
</tr>
<tr>
<td>Adjusted (World†)</td>
<td>211</td>
<td>(196–228)</td>
<td>213</td>
<td>(201–227)</td>
</tr>
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</table>

*Indirect age standardized to the 2001 Auckland population.†Direct age-standardized to the WHO world population.

Sex-, age-, and sequence-specific stroke attack rate ratios (2002 to 2003 compared with 1981 to 1982). Rates were age-standardized to the WHO world population and shown with 95% CI.
high-risk individuals explains only a small fraction of
trends in stroke rates.\textsuperscript{21,22}

Trends in the other risk factors in the ARCOS studies are
more reliable, but they are also more complex. For example,
the halving in the proportion of patients with stroke who were
current smokers was offset by considerable increases in
obesity and history of diabetes among patients with stroke
from 1981 to 1982 to 2002 to 2003. These data reinforce
the burgeoning global impact of obesity and diabetes on cardio-
vascular disease and other health problems. The aging of
populations and improved outcomes for people with vascular
disease in many Western countries has raised concerns about
future increases in the population at risk of stroke. We found,
however, that the proportion of patients with a history of
myocardial infarction was stable over the study period, which
supports other studies indicating that improvements in sur-

TABLE 4. Sex-Specific Annual Stroke Rates (per 100 000) Age-Adjusted to the
WHO World Population

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<tbody>
<tr>
<td></td>
<td>No.</td>
<td>Rate (95% CI)</td>
<td>No.</td>
<td>Rate (95% CI)</td>
<td>No.</td>
<td>Rate (95% CI)</td>
</tr>
<tr>
<td>Male</td>
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<tr>
<td>First-ever</td>
<td>510</td>
<td>184 (163–209)</td>
<td>587</td>
<td>167 (150–185)</td>
<td>667</td>
<td>156 (144–168)</td>
</tr>
<tr>
<td>Total</td>
<td>690</td>
<td>248 (223–276)</td>
<td>835</td>
<td>236 (216–258)</td>
<td>918</td>
<td>214 (200–228)</td>
</tr>
<tr>
<td>Female</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>First-ever</td>
<td>520</td>
<td>133 (118–151)</td>
<td>718</td>
<td>143 (116–241)</td>
<td>756</td>
<td>124 (115–134)</td>
</tr>
<tr>
<td>Total</td>
<td>716</td>
<td>181 (163–202)</td>
<td>968</td>
<td>190 (188–341)</td>
<td>1083</td>
<td>173 (162–184)</td>
</tr>
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</table>

counterbalanced by increases in the frequency of patients
with diabetes and obesity. Taken together with other evidence
that indicates that the best predictors of stroke risk are factors
that cannot be altered such as age and sex, it would appear
that efforts to control the growing epidemic of stroke and
vascular disease through the modification of blood pressure levels and other risk factors is being greatly attenu-
ated by aging and other structural changes, and the emergence
of other risk factors such as obesity and diabetes, in
populations.

Appendix

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References


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