

Declining Rates of Fatal and Nonfatal Intracerebral Hemorrhage: Epidemiological Trends in Australia

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Background—A recent systematic review of epidemiological studies reported intracerebral hemorrhage (ICH) incidence and mortality as unchanged over time; however, comparisons between studies conducted in different health services obscure assessment of trends. We explored trends in ICH rates in a large, representative population in New South Wales, Australia's most populous state (≈ 7.3 million).

Methods and Results—Adult hospitalizations with a principal ICH diagnosis from 2001 to 2009 were linked to death registrations through to June 30, 2010. Trends for overall, fatal, and nonfatal ICH rates within 30 days and fatal rates for 30-day survivors at 365 days were calculated. There were 11 332 ICH patient admissions meeting eligibility criteria, yielding a crude hospitalization rate of 25.2 per 100 000 (age-standardized rate: 17.2). Age- and sex-adjusted overall rates significantly declined by an average of 1.6% per year ($P=0.03$). Fatal ICH declined by an average of 2.6% per year ($P=0.004$). For 30-day survivors, a nonsignificant decline of 2.3% per year in fatal ICH at 365 days was estimated ($P=0.17$). Male sex and birth in the Oceania region and Asia were associated with an increased ICH risk, although this depended on age. Approximately 12% of ICH admissions would be prevented if the socioeconomic circumstances of the population equated with those of the least disadvantaged.

Conclusions—Overall and fatal ICH rates have fallen in this large Australian population. Improvements in cardiovascular prevention and acute care may explain declining rates. There was no evidence of an increase in devastated survivors because the longer term mortality of 30-day survivors has not increased over time. (*J Am Heart Assoc.* 2014;3:e001161 doi: 10.1161/JAHA.114.001161)

Key Words: epidemiology • intracerebral hemorrhage • trends

Intracerebral hemorrhage (ICH) results in high case mortality and significant disability in survivors.¹ Although ischemic stroke incidence appears to be declining,^{2–4} a recent systematic review of 36 epidemiological studies found no decrease in ICH incidence or mortality over time.¹ Pooling epidemiological studies is problematic, given the heterogeneity of eligibility criteria, health services, and characteristics and risk factors of the underlying populations. One Australian

population-based study reported declining ICH incidence based on 32, 22, and 19 ICH cases during 3 time periods from 1989 to 2001.² Another epidemiological study from Oxfordshire, United Kingdom, reported declining ICH rates in a total of 107 cases across 2 time periods (1981–1986 and 2002–2006).³ The small numbers in these studies preclude definitive conclusions.

It has been suggested that hospitalization data sets may usefully monitor epidemiological trends in ICH.⁵ Resource-intensive methods for gold standard case ascertainment require identifying a small population base to ensure complete capture of cases typically yielding low numbers, which limit generalizability. Analysis of subgroups is also hampered. In contrast, as argued elsewhere,⁵ hospitalization administrative data sets yield very large numbers of cases that are prospectively and continuously accrued over several years and are representative of populations across a large geographic area. Hospital-based case selection has been shown to provide reliable data on ICH epidemiology when compared with a population-based gold standard registry.⁶ Using data linkage to identify individual patient admissions linked to mortality information offers a cost-effective opportunity to study patient-level trends of attack rates and fatality for ICH

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that often is not possible with traditional epidemiological studies.

ICH management is evolving with emerging medical⁷ and neurosurgical interventions.⁸ Health services require estimates of the expected number of ICH cases and their outcomes to inform service delivery. Consequently, we undertook this analysis in a large population-based cohort to identify trends in ICH rates and fatality in Australia.

Methods

Cohort Selection

Hospital separations for patients aged ≥ 20 years with a principal diagnosis of ICH (International Classification of Diseases, 10th revision [ICD-10],⁹ code I61.x) from January 1, 2001, to December 31, 2009, within New South Wales (population ≈ 7.3 million) were selected from the Admitted Patient Data Collection, a census of all hospitalizations. According to coding standards, principal stroke diagnoses refer to acute admissions reflecting the main reason for the patient's hospitalization. As a retrospective observational study using routinely collected hospitalization data, we could not prespecify the clinical and cerebral imaging criteria. These criteria are based on standardized coding by trained coders reviewing medical records including imaging to determine the main reason for the patient's hospitalization. The coding criteria were that of nontraumatic stroke-like symptoms with imaging showing intracerebral hemorrhaging and a clinician-reported diagnosis of ICH. Administrative data are the basis for hospital remuneration and are subject to rigorous quality-assurance processes.⁹ Using gold standard methods for highly accurate linkage,¹⁰ separations were linked to create patient-level data and further linked to the state death registry through June 30, 2010, the most recently available information at the time of data extraction.

We excluded admissions due to improbable age (> 105 years), admission prior to the study period, residence outside New South Wales, or likely misclassification of acute ICH indicated by early discharge within 48 hours or a concomitantly recorded diagnosis of traumatic head or stroke sequelae. We did not apply further exclusion criteria because our aim was to determine the burden of spontaneous ICH, regardless of cause, in this population. Secondary causes of ICH, such as arteriovenous malformations (AVMs) and cerebral neoplasms, were not excluded because their identification in the data set relies on recorded comorbidities, which tend to be underenumerated in routinely collected data.¹¹ In addition, ascertainment of secondary causes of ICH such as AVMs and cerebral neoplasms using magnetic resonance imaging and angiograms instead of or in addition to more conventional and widely available computed tomography

scanning may have varied across the health service and may have varied over time; therefore, exclusions may have biased results.

Statistical Analysis

Admission rates

We calculated crude admission rates per 100 000 person-years using population data for each year of our study period as denominators (<http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3201.0Jun%202009?OpenDocument>). Age-standardized rates (ASRs) were directly standardized to the world population.¹² We computed rates for fatal and nonfatal stroke at 30 days to provide respective measures of acute fatality or survival. Rates of fatal stroke at 365 days for 30-day survivors provided a measure of mortality in devastated survivors. Because 365-day follow-up was unavailable for admissions in the second half of 2009, fatal rates at 365 days for 30-day survivors for that year were adjusted using standard methods by dividing population estimates by 2.

Time trends in admission rates

The average annual percentage change was estimated to measure time trends in ICH rates using negative binomial regression models with the outcome equaling the number of ICH admissions, adjusting for age and sex and offset against the natural logarithm of the age- and sex-specific population. Overall trends in rates and trends by sex and age group were calculated. Interaction terms tested whether time trends varied according to sex, age, and year of admission and are reported as significant if the *P* value was < 0.05 .

All admissions were included in analyses including *index* (ie, the first occurrence of an event for a patient during the study period) and *subsequent* events arising in the same patient, consistent with standard epidemiological definitions of *attack rates* and previous research.^{13–16} The study of all admissions avoids bias in the assessment of time trends that would occur if we considered only index admissions, removing subsequent events that occurred for the same patient. Because index admissions would be more likely to appear in the earlier part of our study period, analysis of index admissions only would inflate the number of ICH admissions in the earlier years relative to the later years, biasing results toward a decreasing trend in admission rates.

Variability in admission rates

Negative binomial models calculating incidence rate ratios (IRRs) determined variability in ICH rates according to age, sex, socioeconomic status (SES), and region of birth. For overall, fatal, and nonfatal ICH at 30 days, we tested all possible 2-way interaction terms among age, sex, year of

admission, SES, and region of birth. A 3-way interaction term among age, sex, and year was also tested.

SES was defined using the Index of Relative Socio-Economic Disadvantage, a standardized measure of socioeconomic deprivation based on location of residence within geographic confines known as *local government areas*.¹⁷ Key variables are used to assign scores denoting the relative concentration of disadvantage in these geographically defined areas. These variables include employment status, occupation, income, educational attainment, and ownership of assets. These data are collected from a government census of all Australian households and persons occupying nonprivate dwellings and are carried out once every 5 years. Participation is a legal requirement and approaches 100% (eg, 95.8% in the 2006 census; <http://www.abs.gov.au/websitedbs/censushome.nsf/home/nonresponserates>). Scores are conventionally ranked in ascending order and categorized using quintile cutoffs.¹⁷ Patient residence within local government areas is recorded in the Admitted Patient Data Collection, and these areas were assigned the corresponding quintile score for analysis. Region-of-birth groupings were assigned using a standard classification (<http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/1269.0main+features102011>).

ICH risk factors by sex, age, SES, and region of birth

To explore reasons for variability in ICH rates, we estimated the prevalence of ICH risk factors according to sex, age, region of birth, and SES. We determined the prevalence of the following risk factors, selecting both common and rarer causes of ICH: hypertension, diabetes, cerebral malignancies, smoking, alcohol or drug use, chronic kidney disease,

anticoagulant use current at time of the hemorrhage, and AVMs and other venous malformations (ie, cerebral cavernous hemangioma). These risk factors were identified from secondary diagnoses in the hospital admission data set (up to 54 diagnoses in addition to the principal diagnosis are recorded) and 8 external cause-of-injury codes (enhancing ascertainment of smoking, drug and alcohol use, and current anticoagulant use) using ICD-10 coding⁹ (Table 1). Because comorbidities tend to be underascertained,¹¹ we applied a *look-back period* identifying risk factors coded during the ICH admission and in any admission occurring in the 6 months prior to the patient's ICH hospitalization to maximize ascertainment. Applying a look-back period maximizes ascertainment in Australian administrative data sets.^{18–20} Our selection of a 6-month look-back period was pragmatic because admissions are available to researchers only from July 1, 2000, providing 6 months of clearance for all patients admitted with ICH from 2001 onward. A look-back period was not applied when identifying anticoagulant use because we wished to determine anticoagulant use current at the time of the ICH. We also applied procedure codes for chronic kidney disease to capture patients receiving renal dialysis. These procedure codes are based on the Australian government's Medicare Benefits Schedule coding that hospital coders apply to all diagnostic and treatment procedures listed on the schedule to ensure financial reimbursement. Procedure coding is defined according to the standardized Australian Classification of Health Interventions,⁹ and procedures are coded with a high level of accuracy.²¹ Mean and median ages were calculated for sex, SES, and region of birth.

Table 1. ICD-10 Diagnostic and Procedure Codes Applied to Ascertaining Intracerebral Hemorrhage Risk Factors

Diabetes	E10 to E14
Hypertension	I10 to I14
Cerebral malignancies (primary or metastatic)	C70.x, C71.x, C79.3
Smoking	F17, Z72.0, Z71.6, Z86.43
Alcohol or drug use	E52, F10, K70, X45, X65, Y15, Y90, Y91, E24.4, E51.2, G31.2, G62.1, G72.1, I42.6, K29.2, K86.0, O35.4, P04.3, Q86.0, R78.0, T50.6, T51.0, T51.1, T51.9, Y57.3, Z50.2, Z71.4, Z72.1, Z78.1, F11, F12, F13, F14, F15, F16, F18, F19, T40, X42, T43.6, Z50.3
Arteriovenous malformations/other malformations	Q28.2, D18, I60.8
Chronic kidney disease	ICD-10 diagnosis codes: N00 to N08, I12, I13, N11, N12, N14, N15, N16, N18, N19, N25, N26, N27, N28, Q60, Q61, Q62, Q63, Z49, E10.2, E11.2, E13.2, E14.2, N39.1, N39.2, I15.0, I15.1, T82.4, T86.1, Z94.0, Z99.2; ICD-10 Procedure codes: 36561-00, 36503-00, 36503-01, 13100-06, 13100-07, 13100-08, 13100-00
Anticoagulant use	D68.3, T45.5, Y44.2, Z92.1

ICD-10 indicates International Classification of Diseases, 10th revision.

Ethics approval

The New South Wales Population and Health Services research ethics committee and the University of New South Wales approved analyses of ICH data as part of the Program of Research Informing Stroke Management (PRISM) study.

Results

Patient Selection and Characteristics

There were 12 304 acute admissions with a principal diagnosis of ICH identified prior to applying exclusion criteria. Overall, 7.9% of admissions were excluded because of improbable age ($n=1$), admission prior to 2001 ($n=1$), residence outside New South Wales ($n=277$), or likely misclassification indicated by early discharge within 48 hours ($n=240$) or a concomitantly recorded diagnosis of traumatic injury ($n=264$) or stroke sequelae ($n=189$), leaving 11 332 eligible admissions. Of those, 49.8% were female, with a median age of 79 years (IQR 69 to 85). Male patients were significantly younger (median age 73 years, IQR 62 to 80) ($P<0.001$). More than two thirds were born in Australia (68.3%; $n=7527$).

Time Trends in Overall Admission Rates

The crude admission rate was 25.2 per 100 000 and 17.2 when age standardized against the world population (Table 2). ASRs for men and women were 20.0 and 14.5, respectively. Age- and sex-adjusted admission rates declined during the study period by an average of 1.6% per year ($P=0.03$) (Figure 1, Table 3). Nonsignificant interaction terms indicated that the decline in rates did not differ significantly by sex or age, although declines were noted in all age groups except those aged >90 years (Table 2).

Trends in Fatal and Nonfatal ICH Rates

By 30 days, 4246 (37.5%) of ICH admissions resulted in death. Fatal ICH occurred in 9.4 patients per 100 000 population (ASR 5.9 per 100 000). The nonfatal stroke rate was 15.8 per 100 000 or 11.2 per 100 000 when standardized to the World Health Organization-reported world population.¹²

The average annual percentage decline in fatal ICH rates was 2.6% per year ($P=0.004$), and the decline was similar for men and women ($P_{\text{interaction}}=0.57$). Fatal rates declined for all ages except those aged >90 years; however, there was no significant variability in declining fatal ICH rates due to age ($P_{\text{interaction}}=0.60$) (Table 3). The 1% average percentage decline in nonfatal ICH rates was not significant ($P=0.19$).

The overall fatal ICH rate within 365 days for 30-day survivors was estimated as 2.7 per 100 000 (ASR 1.7, 95% CI

Table 2. Age-Standardised ICH Rates per 100 000 in New South Wales in 2001–2009

Age (y)	ICH Cases	Crude Rate	ASR (WHO World Population)	ASR, 95% CI
<i>Men</i>				
20 to 29	61	1.4	1.4	1.1 to 1.8
30 to 39	125	2.8	2.8	2.3 to 3.3
40 to 49	338	7.7	7.6	6.9 to 8.5
50 to 59	637	16.9	16.8	15.5 to 18.1
60 to 69	1103	42.4	42.5	40.0 to 45.0
70 to 79	1785	104.1	102.0	97.3 to 106.8
80 to 89	1438	198.4	198.8	188.7 to 209.2
≥ 90	203	238.8	233.7	202.4 to 267.1
All ages	5690	25.8	20.0	19.4 to 20.5
<i>Women</i>				
20 to 29	52	1.2	1.2	0.9 to 1.6
30 to 39	101	2.2	2.2	1.8 to 2.7
40 to 49	219	4.9	4.9	4.3 to 5.6
50 to 59	383	10.2	10.1	9.1 to 11.2
60 to 69	675	25.6	25.5	23.6 to 27.5
70 to 79	1537	77.8	74.7	70.9 to 78.5
80 to 89	2116	182.3	179.0	171.4 to 186.8
≥ 90	559	237.3	236.9	217.7 to 257.0
All ages	5642	24.6	14.5	14.1 to 15.0
<i>Both sexes</i>				
20 to 29	113	1.3	1.3	1.1 to 1.6
30 to 39	226	2.5	2.5	2.2 to 2.8
40 to 49	557	6.3	6.3	5.8 to 6.8
50 to 59	1020	13.5	13.5	12.7 to 14.3
60 to 69	1778	33.9	33.9	32.4 to 35.5
70 to 79	3322	90.0	87.4	84.4 to 90.4
80 to 89	3554	188.5	187.0	180.9 to 193.2
≥ 90	762	237.7	236.1	219.6 to 253.2
All ages	11 332	25.2	17.2	16.8 to 17.5

ASR indicates age-standardized rate; ICH, intracerebral hemorrhage; WHO, World Health Organization.

1.6 to 1.8) and declined by an average of 2.3% per year (95% CI -5.5 to 1.0); this decline was not significant ($P=0.17$).

Variability in ICH Admission Rates

Age and sex

Admission rates were significantly elevated in men and in relatively older patients ($P<0.001$ for both) (Table 2). Crude rates did not differ between the sexes (Table 2); however, age-

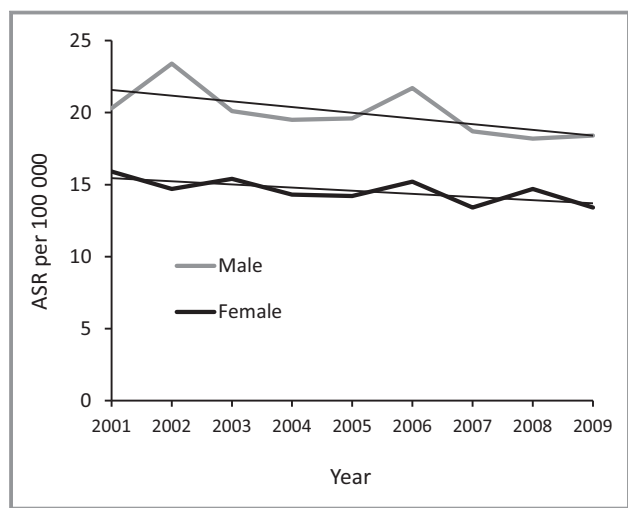


Figure 1. Declining ASR (World Health Organization world population) for ICH admissions (attack rates) from 2001 to 2009, by sex. ASR indicates age-standardized rate; ICH, intracerebral hemorrhage.

and year-adjusted ICH rates were 26% lower for women than for men (IRR 0.74, 95% CI 0.69 to 0.80). Male sex and increasing age were also associated with higher adjusted rates of both fatal and nonfatal ICH rates ($P < 0.001$ for both) (Tables 4 and 5).

Rates of ICH and fatal ICH were higher in men than in women in those aged 40 to 89 years and similar in those aged <40 years and >90 years ($P_{\text{interaction}} < 0.001$ and $P_{\text{interaction}} = 0.003$, respectively). Nonfatal ICH rates were similar for men and women aged >80 years ($P_{\text{interaction}} = 0.003$) (Tables 2, 4, and 5).

Table 3. Average APC in ICH rates in New South Wales in 2001–2009

	Overall Admission Rates (Attack Rates)			Fatal ICH Rate (30 Days)	
	APC (95% CI)	P Value		APC (95% CI)	P Value
All patients*	-1.6 (-3.0 to -0.2)	0.03	All patients*	-2.6 (-4.4 to -0.8)	0.004
Sex†			Sex†		
Male	-1.8 (-3.7 to 0.1)	—	Males	-3.0 (-5.3 to -0.6)	
Female	-1.3 (-3.2 to 0.6)	0.62‡	Females	-2.2 (-4.6 to 0.3)	0.57‡
Age§ (y)			Age§,		
20 to 39	-1.0 (-6.1 to 4.4)	—	20 to 49	-6.1 (-13.5 to 1.9)	
40 to 49	-2.6 (-6.7 to 1.6)	—	50 to 59	-3.2 (-8.4 to 2.3)	
50 to 59	-2.4 (-5.5 to 0.8)	—	60 to 69	-4.4 (-8.3 to -0.4)	
60 to 69	-3.9 (-7.1 to -0.6)	—	70 to 79	-2.8 (-6.8 to 1.4)	
70 to 79	-1.1 (-4.9 to 2.9)	—	80 to 89	-1.7 (-4.8 to 1.5)	
80 to 89	-0.3 (-2.5 to 1.9)	—	90+	0.6 (-3.0 to 4.4)	0.60‡
≥90+	0.3 (-3.2 to 3.9)	0.72‡			

APC indicates annual percentage change; ICH, intracerebral haemorrhage.

*Age and sex adjusted (using 10-year age groups).

†Age adjusted (using 10-year age groups).

‡P value for interaction with year of admission.

§Adjusted for sex.

||Age categories between 20 and 49 years collapsed when testing the interaction term because of small case numbers for those aged <50 years (n=62).

Older age and male sex were also associated with higher rates of fatal ICH by 365 days in those who survived the first 30-days after their ICH.

SES

Patients residing in the least disadvantaged areas were 27% less likely to be admitted to the hospital with an ICH than those residing in areas of greatest socioeconomic disadvantage (Figure 2, Table 6). This relationship also held for fatal and nonfatal ICH (Tables 6 and 7). Significant interactions between SES and age revealed that these associations applied to patients aged <80 years (Table 8). SES was not associated with ICH fatality at 365 days for 30-day survivors.

ICH risk attributable to SES was 20.1%; 1 in 5 ICH admissions occurring in areas of greatest socioeconomic disadvantage can be attributed to socioeconomic deprivation. Slightly >1 in 9 ICH admissions (11.9%) in the total population would be avoided if the risk of ICH for all residents equaled that of the most affluent (Table 9).

Region of birth

Compared with people born in Australia, those born in the Oceania region or in Northeast or Southeast Asia were between 44% and 68% more likely to be admitted to the hospital with ICH and between 16% and 44% more likely to die from ICH within 30 days (Tables 6 and 10). People born in Europe were at a reduced risk of ICH and of dying from it. Residents born in the Middle East and North Africa were 10%

Table 4. ASR per 100 000 of Fatal Intracerebral Hemorrhage in New South Wales in 2001–2009 by Sex and Age

Year	ASR, WHO World Population (95% CI)
<i>Men</i>	
20 to 29	*
30 to 39	0.5 (0.3 to 0.7)
40 to 49	1.7 (1.4 to 2.1)
50 to 59	3.5 (3.0 to 4.2)
60 to 69	12.2 (10.9 to 13.5)
70 to 79	37.8 (34.9 to 40.7)
80 to 89	95.8 (88.8 to 103.1)
≥90	127.2 (104.2 to 152.4)
<i>Women</i>	
20 to 29	*
30 to 39	0.4 (0.2 to 0.6)
40 to 49	0.8 (0.5 to 1.1)
50 to 59	2.5 (2.0 to 3.0)
60 to 69	6.9 (5.9 to 7.9)
70 to 79	28.7 (26.4 to 31.1)
80 to 89	82.0 (76.9 to 87.3)
≥90	135.7 (121.2 to 151.0)
<i>Both sexes</i>	
20 to 29	*
30 to 39	0.5 (0.3 to 0.6)
40 to 49	1.2 (1.0 to 1.5)
50 to 59	3.0 (2.6 to 3.4)
60 to 69	9.5 (8.7 to 10.4)
70 to 79	32.9 (31.1 to 34.8)
80 to 89	87.4 (83.3 to 91.7)
≥90	133.3 (120.9 to 146.2)

ASR indicates age-standardized rate; WHO, World Health Organization.

*Cells sizes and calculations based on sample sizes ≤10 or cells that can be used to deduce cells with ≤10 patients have been suppressed to protect patient privacy in accordance with local standards.

more likely to experience an ICH compared with Australia-born residents, although confidence limits for the estimated effect included the null value. Differences related to region of birth depended on age ($P_{\text{interaction}}=0.02$) (Table 11). People born in the Oceania region had particularly elevated ASRs of ICH in those aged <65 years. The elevated ICH risk in Northeast Asia-born residents was evident only in those aged >45 years, whereas residents from Southeast Asia had an elevated risk regardless of age.

Among 30-day survivors, ICH fatality rates at 365 days for those born in the Oceania region, Northeast Asia, and the Middle East were between 23% and 89% higher than the rate for Australia-born residents (Table 6).

Table 5. ASR per 100 000 of Nonfatal Intracerebral Hemorrhage in New South Wales in 2001–2009 by Sex and Age

Year	ASR, WHO World Population (95% CI)
<i>Men</i>	
20 to 29	*
30 to 39	2.3 (1.9 to 2.8)
40 to 49	5.9 (5.2 to 6.7)
50 to 59	13.3 (12.1 to 14.5)
60 to 69	30.3 (28.2 to 32.5)
70 to 79	64.2 (60.5 to 68.1)
80 to 89	103.0 (95.8 to 110.6)
≥90	106.5 (85.9 to 129.2)
<i>Women</i>	
20 to 29	*
30 to 39	1.8 (1.4 to 2.2)
40 to 49	4.1 (3.5 to 4.7)
50 to 59	7.6 (6.8 to 8.5)
60 to 69	18.6 (17.0 to 20.3)
70 to 79	45.9 (43.0 to 48.9)
80 to 89	97.0 (91.5 to 102.8)
≥90	101.2 (88.8 to 114.5)
<i>Both sexes</i>	
20 to 29	*
30 to 39	2.0 (1.8 to 2.4)
40 to 49	5.0 (4.6 to 5.5)
50 to 59	10.5 (9.7 to 11.2)
60 to 69	24.4 (23.1 to 25.7)
70 to 79	54.4 (52.1 to 56.8)
80 to 89	99.6 (95.2 to 104.2)
≥90	102.9 (92.1 to 114.2)

ASR indicates age-standardized rate; WHO, World Health Organization.

*Cells sizes and calculations based on sample sizes ≤10 or cells that can be used to deduce cells with ≤10 patients have been suppressed to protect patient privacy in accordance with local standards.

ICH risk factors

Age of ICH onset was significantly lower in men; in those residing in the most disadvantaged areas; and for those born in Oceania, North Africa or the Middle East, and in Northeast and Southeast Asia (Table 12). With the exception of hypertension, all other assessed risk factors were more prevalent in men than in women, including AVMs, cerebral malignancies, and drug and alcohol use. AVMs were present in 19.2% of ICH patients aged <40 years and in 8.6% aged 40 to 49 years. Cerebral malignancies and alcohol and drug use

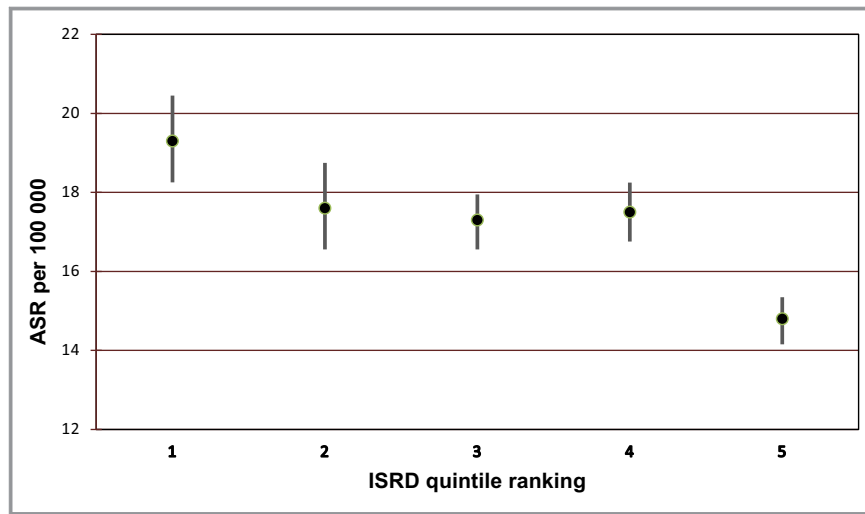


Figure 2. ASRs for intracerebral hemorrhage according to the ISRD measuring SES. 1 = low SES; 5 = high SES. ASR indicates age-standardized rate; ICH, intracerebral hemorrhage; ISRD, Index of Relative Socio-Economic Disadvantage; SES, socioeconomic status.

Table 6. IRR* in New South Wales in 2001–2009 According to Sex, Age, SES, and Region of Birth

	ICH Admission Rate IRR (95% CI)	Fatal ICH 30-Day IRR (95% CI)	Nonfatal ICH 30-Day IRR (95% CI)	Fatal ICH at 365 Days IRR (95% CI)†
<i>SES (IRSD)*</i>				
Quintile 1 (lowest SES)	Referent§	Referent	Referent§	Referent¶
Quintile 2	0.91 (0.80 to 1.05)	0.94 (0.77 to 1.14)	0.89 (0.77 to 1.04)	1.04 (0.76 to 1.41)
Quintile 3	0.89 (0.78 to 1.01)	0.94 (0.78 to 1.12)	0.87 (0.77 to 1.00)	0.88 (0.66 to 1.17)
Quintile 4	0.91 (0.80 to 1.03)	0.93 (0.78 to 1.11)	0.90 (0.79 to 1.03)	0.90 (0.68 to 1.20)
Quintile 5 (Highest SES)	0.73 (0.64 to 0.83)	0.76 (0.64 to 0.91)	0.74 (0.65 to 0.85)	0.80 (0.60 to 1.06)
<i>Region of birth</i>				
Australia	Referent§	Referent§	Referent§	Referent#
Other Oceania	1.52 (1.31 to 1.77)	1.16 (0.91 to 1.48)	1.69 (1.44 to 2.00)	1.89 (1.30 to 2.74)
Northwest Europe	0.73 (0.65 to 0.82)	0.65 (0.56 to 0.76)	0.80 (0.70 to 0.90)	0.88 (0.69 to 1.12)
Southeast Europe	0.93 (0.83 to 1.04)	0.86 (0.74 to 0.99)	0.98 (0.87 to 1.11)	0.87 (0.68 to 1.11)
North Africa/Middle East	1.10 (0.95 to 1.29)	0.99 (0.78 to 1.25)	1.17 (0.98 to 1.40)	1.23 (0.82 to 1.84)
Other Africa	0.95 (0.73 to 1.23)	0.94 (0.62 to 1.43)	0.96 (0.71 to 1.32)	0.93 (0.41 to 2.10)
Southeast Asia	1.68 (1.47 to 1.91)	1.44 (1.17 to 1.77)	1.79 (1.54 to 2.08)	0.92 (0.58 to 1.48)
Northeast Asia	1.44 (1.26 to 1.65)	1.36 (1.12 to 1.65)	1.50 (1.29 to 1.75)	1.33 (0.92 to 1.91)
Other Asia	0.80 (0.64 to 1.01)	0.75 (0.51 to 1.10)	0.83 (0.63 to 1.10)	0.30 (0.10 to 0.94)
Americas	0.95 (0.77 to 1.18)	0.64 (0.42 to 0.97)	1.13 (0.88 to 1.44)	0.53 (0.22 to 1.29)

ICH indicates intracerebral hemorrhage; IRR, incident rate ratio; IRSD, Index of Relative Socio-Economic Disadvantage; SES, socioeconomic status.

*Adjusting for age, sex, and year. IRRs can be interpreted as relative risks whereby ratios below the null value of 1 indicate a reduced risk when compared with the referent group, whereas ratios >1 indicate an increased risk relative to the referent group.

†Estimated in 30-day survivors.

‡All residential areas in New South Wales are assigned a score according to standard methods and scores are ranked according to quintiles. Patient residence within local government areas were recorded in the data set, and these geographic areas were assigned the corresponding quintile ranking. Lower quintile rankings indicate residence in geographic areas of greater relative socioeconomic disadvantage or deprivation and therefore lower SES.

§P<0.001.

||P=0.02.

¶P=0.39.

#P=0.003.

Table 7. ASR per 100 000 of Overall, Fatal, and Nonfatal ICH by SES

IRSD Quintile*	ICH Cases	Crude Rate	ASR WHO World Population	95% CI
<i>Overall ICH (ie, admissions)</i>				
Quintile 1 (low SES)	1358	27.8	19.3	18.3 to 20.4
Quintile 2	1194	26.9	17.6	16.6 to 18.7
Quintile 3	2914	25.9	17.3	16.6 to 17.9
Quintile 4	2792	24.9	17.5	16.8 to 18.2
Quintile 5 (high SES)	2962	22.2	14.8	14.2 to 15.3
<i>Fatal ICH (30 days)</i>				
Quintile 1 (low SES)	489	10.0	6.5	5.9 to 7.1
Quintile 2	450	10.1	6.1	5.6 to 6.7
Quintile 3	1093	9.7	6.0	5.6 to 6.4
Quintile 4	1069	9.5	6.1	5.8 to 6.5
Quintile 5 (high SES)	1144	8.6	5.2	4.9 to 5.5
<i>Nonfatal ICH (30 days)</i>				
Quintile 1 (low SES)	869	17.8	12.8	12.0 to 13.7
Quintile 2	744	16.8	11.5	10.6 to 12.3
Quintile 3	1821	16.2	11.3	10.7 to 11.8
Quintile 4	1723	15.4	11.4	10.8 to 11.9
Quintile 5 (high SES)	1818	13.6	9.6	9.1 to 10.1
<i>Fatal ICH 365 days in 30-day survivors</i>				
Quintile 1 (low SES)	128	2.8	1.9	1.5 to 2.2
Quintile 2	130	3.1	1.9	1.6 to 2.3
Quintile 3	277	2.6	1.6	1.4 to 1.8
Quintile 4	281	2.7	1.6	1.4 to 1.8
Quintile 5 (high SES)	312	2.5	1.5	1.3 to 1.6

ICH indicates intracerebral hemorrhage; IRR, incident rate ratio; IRSD, Index of Relative Socio-Economic Disadvantage; SES, socioeconomic status; WHO, World Health Organization.

*Excludes 112 patients with missing data.

were also more prevalent in patients aged <50 years (≈20% and 5%, respectively). Anticoagulant use was most prevalent in patients aged between 50 and 79 years. Among patients born in regions with the highest ICH rates, diabetes, renal disease, and hypertension were more prevalent in Asia-born patients and those born in the Middle East or North Africa and Oceania. AVMs were more frequently found in patients of Southeast Asian origin (3.4%).

Discussion

PRISM Study: Declining ICH Rates

This is the first large-scale study of ICH epidemiology in Australia, and it reports falling rates of hospital admission and

Table 8. Crude and Age-Standardised Overall ICH Rates per 100 000 for SES by Age in New South Wales (2001–2009)*

SES Quintile	ICH Cases	Crude Rate	ASR, WHO World Population	
			ASR	95% CI
<i>Aged 20 to 39 years</i>				
Quintile 1 (low SES)	40	2.2	2.2	1.5 to 2.9
Quintile 2	49	3.2	3.1	2.3 to 4.0
Quintile 3	89	2.1	2.0	1.6 to 2.5
Quintile 4	97	2.1	2.1	1.7 to 2.5
Quintile 5 (high SES)	61	1.2	1.1	0.8 to 1.4
<i>Aged 40 to 59 years</i>				
Quintile 1 (low SES)	229	12.5	12.3	10.7 to 13.9
Quintile 2	179	10.7	10.5	9.0 to 12.1
Quintile 3	396	9.7	9.4	8.5 to 10.4
Quintile 4	393	10	9.8	8.9 to 10.8
Quintile 5 (high SES)	360	7.3	7.2	6.5 to 8.0
<i>Aged 60 to 79 years</i>				
Quintile 1 (low SES)	665	64.6	60.1	55.6 to 64.8
Quintile 2	557	56.2	52.1	47.8 to 56.5
Quintile 3	1460	60.8	56.6	53.7 to 59.6
Quintile 4	1181	56.2	52.4	49.4 to 55.5
Quintile 5 (high SES)	1179	48	44.7	42.2 to 47.3
<i>Aged ≥80 years</i>				
Quintile 1 (low SES)	424	190.7	190.5	172.8 to 209.1
Quintile 2	409	180.0	179.7	162.7 to 197.6
Quintile 3	969	172.6	171.3	160.7 to 182.3
Quintile 4	1121	206.4	203.8	192.0 to 216.0
Quintile 5 (high SES)	1362	202.4	197.5	187.1 to 208.2

ASR indicates age-standardized rate; ICH, intracerebral hemorrhage; SES, socioeconomic status; WHO, World Health Organization.

*To Minimize Small Cell Sizes, Age Categories were Collapsed to 20-Year Age Groups When Testing the Interaction Between Age and Index of Relative Socio-Economic Disadvantage.

fatal ICH in an unselected population of >7.3 million and 11 332 ICH patients. There has been little evidence internationally of reductions in ICH attack rates including fatal ICH.¹ The large number of ICH admissions identified over a 9-year period within the same geographically defined area is a major strength of the study.

An analysis pooling ICH cases from 36 international epidemiological studies published between 1980 and 2008 did not demonstrate declining incidence or mortality.¹ One recent study of 441 ICH patients reported no overall decline in first-ever ICH rates in Dijon, France, over 3 time periods from 1985 to 2008²² but reported increasing rates in people aged >75 years, decreasing rates in those aged <60 years, and stable rates for the population aged 60 to 74 years. The

Table 9. Calculation of Attributable Risk Percent and Population Attributable Risk Percentage

IRSD Level	Calculation	Attributable Risk Percentage
Quintile 1 (Low SES)	$[(27.8 - 22.2) / 27.8] \times 100$	20.1
Quintile 2	$[(26.9 - 22.2) / 26.9] \times 100$	17.5
Quintile 3	$[(25.9 - 22.2) / 25.9] \times 100$	14.3
Quintile 4	$[(24.9 - 22.2) / 25.9] \times 100$	10.8

Attributable risk percent: We calculated the attributable risk percent to determine the excess of ICH risk in people residing in areas of greatest socioeconomic disadvantage that can be attributed to socioeconomic deprivation. We applied the following formula, using crude rates: $[(I_e - I_o) / I_e] \times 100$. I_e =incidence in exposed per 100 000 and I_o =incidence in non-exposed.³ We used crude rates for overall ICH (ie, admission rates). Those residing in the least disadvantaged areas were considered *nonexposed* to socioeconomic deprivation (IRSD=quintile 5). Consequently, the attributable risk percent for each of the 4 quintile levels of SES indicating greater relative socioeconomic disadvantage than the least disadvantaged group (ie, the nonexposed group) is as described in the table. PAR percent: We calculated the population attributable risk to determine the excess rate of ICH in the population that can be attributed to socioeconomic deprivation. We applied the following formula, using crude ICH overall/admission rates: $PAR\ percent = [(I_r - I_o) / I_r] \times 100$. I_r =incidence of overall ICH in the population, I_o =incidence of overall ICH in the nonexposed group (ie, incidence for the population residing in the least socioeconomic disadvantaged areas [IRSD=quintile 5]). Consequently, $PAR\% = [(25.2 - 22.2) / 25.2] \times 100 = 11.9\%$. Moreover, 22.2 per 100 000 is the crude overall ICH rate for people residing in the area of the least socioeconomic disadvantage (ie, the highest SES group or quintile 5). ICH indicates intracerebral hemorrhage; IRSD, Index of Relative Socio-Economic Disadvantage; PAR, population attributable risk; SES, socioeconomic status.

authors did not assess effect modification by age, and that may limit the conclusions that can be drawn. In contrast, we found declining rates of overall and fatal ICH that were not modified by age. Another gold standard epidemiological study carried out in Texas demonstrated a 31% decrease in hospitalized ICH incidence from 2000 to 2010 in 734 ICH cases, with no decline in case fatality noted.²³ Declines in ICH incidence were seen in all ages except those aged 40 to 49 years, although effect modification by age was not formally tested. In Australia, stroke rates have been declining,^{2,24,25} although only 1 study noted time trends in ICH, reporting decreasing incidence and case fatality based on 73 ICH cases accrued over 3 discrete time periods, the latest in 2001.² Research from Oxfordshire has reported declining ICH incidence based on 107 patients recruited between 2 time periods (1981–1986 and 2002–2006).³ To our knowledge, studies using administrative data have not reported declining ICH attack rates.^{26–32} Two studies reported declines in 1 of several subgroups examined, specifically, women aged 35 to 44 years,³¹ and in men and women aged 55 to 64 years²⁷ without formally assessing effect modification. Data linkage has not been widely used.²⁸

Improved risk management, particularly of hypertension, may explain declines in ICH admissions. In Australia, the proportion of adults in the community with high blood pressure has declined significantly over time.³³ Approximately

6% and 13% of ICH can be attributed to hazardous alcohol intake and a history of ischemic stroke, respectively,³⁴ and decreasing rates of both in Australia^{2,35} may have translated into reduced ICH risk. Smoking prevalence has also decreased in our population during the study period,³⁵ although its etiological role in ICH remains uncertain.³⁶ Improvements in hypertension control in the community may also explain reductions in ICH mortality, given that prestroke antihypertensive therapy lowers overall stroke mortality.³⁷

Two key evidence-based health service strategies implemented during the study period may have affected ICH outcomes. One strategy was the coordinated implementation of New South Wales metropolitan stroke units, which commenced with 22 units from 2003 and expanded throughout the study period.³⁸ Stroke units are an innovation associated with reduced death and disability in ICH.³⁹ Another statewide network of 10 coordinated neurosurgical units was implemented. These units are colocated with tertiary intensive care and trauma services and facilitate access to these specialized services, including highly resourced and experienced neurosurgical teams that are now routinely involved in emergency ICH management. Neurosurgical care and intensive care are recommended for ICH management, although it seems difficult to show a benefit of specific treatments.⁸ Transferring patients to specialist centers may reduce mortality because outcomes for patients undergoing neurosurgery are better in hospitals with higher ICH case loads.⁴⁰

For 30-day survivors, we report a nonsignificant decline in 365-day mortality over time, suggesting that advances in acute care have not resulted in a concomitant increase in the number of devastated survivors whose deaths due to complications are delayed beyond the acute period. The nonsignificant decline suggests more could be done during the postacute period, including secondary prevention.

Burden of ICH in Australia

We report a crude hospitalization attack rate of 25.2 per 100 000 and a fatal attack rate within 30 days of 9.4 per 100 000 in an adult population. Our age-standardized attack rate was 17 per 100 000. The rates reported in this paper include both incident and recurrent hospitalized ICH admissions in adults aged >20 years. Direct comparisons between this estimate and other studies are difficult because studies vary in their case definition and ascertainment, the age groups represented (eg, adult ages versus all age groups), whether or not only ICH cases were included as the first-ever stroke, and the selection of the standard population. We also note that the cohort under study in this paper includes all nontraumatic, spontaneous causes of ICH to permit an assessment of the burden of ICH in our community. Although this approach is consistent with several previous epidemiological studies,^{1,23}

Table 10. Region of Birth and Risk of Overall ICH and Fatal and Nonfatal ICH

Region of Birth	ICH Cases	Crude Rate	ASR	95% CI
<i>Overall ICH admission rates</i>				
Australia	7491	30.8	21.8	21.3 to 22.3
Other Oceania	275	24.9	31.1	27.5 to 35.0
Northwest Europe	962	31.4	16.3	15.3 to 17.5
Southeast Europe	983	46.8	20.9	19.4 to 22.5
North Africa/Middle East	257	25.0	24.8	21.8 to 27.9
Other Africa	*	*	*	*
Southeast Asia	378	25.2	35.4	31.8 to 39.2
Northeast Asia	382	29.1	31.8	28.7 to 35.1
Other Asia	*	*	*	*
Americas	*	*	*	*
<i>Fatal ICH (30 days)</i>				
Australia	2933	12.1	8.0	7.7 to 8.3
Other Oceania	73	6.6	8.7	6.7 to 10.8
Northwest Europe	333	10.9	5.2	4.6 to 5.8
Southeast Europe	363	17.3	6.7	6.0 to 7.5
North Africa/Middle East	82	8.0	8.0	6.3 to 9.8
Other Africa	*	*	*	*
Southeast Asia	112	7.5	11.2	9.2 to 13.4
Northeast Asia	131	10.0	10.9	9.1 to 12.8
Other Asia	*	*	*	*
Americas	*	*	*	*
<i>Nonfatal ICH (30 days)</i>				
Australia	4558	18.7	13.8	13.4 to 14.3
Other Oceania	202	18.3	22.5	19.4 to 25.7
Northwest Europe	629	20.6	11.2	10.2 to 12.1
Southeast Europe	620	29.5	14.2	12.9 to 15.6
North Africa/Middle East	175	17.0	16.8	14.4 to 19.4
Other Africa	*	*	*	*
Southeast Asia	266	17.7	24.2	21.3 to 27.3
Northeast Asia	251	19.1	20.9	18.4 to 23.6
Other Asia	*	*	*	*
Americas	*	*	*	*
<i>Fatal ICH (365 days in 30 day survivors)</i>				
Australia	763	3.3	2.2	2.0 to 2.4
Other Oceania	32	3.1	4.2	2.9 to 5.8
Northwest Europe	121	4.2	1.9	1.6 to 2.3
Southeast Europe	95	4.8	2.1	1.6 to 2.6
North Africa/Middle East	27	2.8	2.8	1.8 to 3.9

Continued

Table 10. Continued

Region of Birth	ICH Cases	Crude Rate	ASR	95% CI
Other Africa	*	*	*	*
Southeast Asia	19	1.3	2.0	1.2 to 3.1
Northeast Asia	34	2.8	3.0	2.1 to 4.1
Other Asia	*	*	*	*
Americas	*	*	*	*

ASR indicates age-standardized rate; ICH, intracerebral hemorrhage.

*Cells based on sample sizes ≤ 10 or cells that can be used to deduce cells with ≤ 10 patients have been suppressed to protect patient privacy in accordance with local standards.

other researchers have elected to remove some secondary causes of ICH such as AVMs¹ and/or malignancies.^{1,23} By definition, gold standard epidemiological studies usually,¹ but not always, include out-of-hospital events,²³ whereas our study included hospitalizations to determine the burden of ICH in our statewide health service. Worldwide rates of incident ICH vary significantly from 1.8 to 129.6 per 100 000. Van Asch et al¹ pooled ICH crude incidence rates and reported a worldwide pooled rate of 25.6 per 100 000 and 24.2 per 100 000 in white populations, broadly corresponding to our estimated crude attack rate of hospitalized ICH of 25.2 per 100 000.

The burden of ICH is unequally distributed in this large Australian community. As expected, increasing age was associated with an increasing risk of ICH,¹ whereas male sex conferred a higher risk of ICH hospitalization and of fatal and nonfatal ICH. ICH risk factors were more prevalent in male patients, corroborating the association between sex and ICH. ICH risk in men and women was equivalent in adults aged <40 years and in the very elderly. Male sex has been associated with increasing ICH risk in Australia,^{2,41} a finding that is not universal.¹ Other studies have also reported varying patterns of stroke risk by sex according to age, but effect modification is rarely assessed.^{27,31,42} Nonmodifiable risk factors, such as aneurysms; cavernous angiomas; hypocholesterolemia; hematological cancers; coagulopathies; and, in women, pregnancy-related complications such as pre-eclampsia may have attenuated the heightened risk in men and in the relatively young,⁴³ whereas cerebral amyloid angiopathy, which affects men and women equally, assumes greater importance with age.⁴⁴ As observed elsewhere,⁴² sex differences in stroke risk among the elderly may be partly explained by using an open-ended age category because women are more likely to realize stroke risk during their increased life span.

The Australian population comprises a large proportion of people born overseas ($\approx 33\%$ in this study), affording a unique opportunity to examine disparities in ICH risk based on region

Table 11. Crude and Age-Standardised Overall Intracerebral Hemorrhage Rates per 100 000 for Region of Birth by Age in New South Wales (2001–2009)*

Region of Birth	ASR, WHO World Population (95% CI)
<i>Aged 25 to 44 years</i>	
Australia	3.3 (2.9 to 3.6)
Other Oceania	5.6 (3.9 to 7.6)
Northwest Europe	1.6 (0.8 to 2.5)
Southeast Europe	2.9 (1.5 to 4.8)
North Africa/Middle East	2.5 (1.3 to 4.1)
Other Africa	†
Southeast Asia	4.8 (3.4 to 6.4)
Northeast Asia	2.6 (1.5 to 3.9)
Other Asia	†
Americas	†
<i>Aged 45 to 64 years</i>	
Australia	14.6 (13.8 to 15.4)
Other Oceania	31.0 (25.5 to 37.0)
Northwest Europe	11.2 (9.5 to 13.1)
Southeast Europe	13.9 (11.6 to 16.3)
North Africa/Middle East	16.6 (12.8 to 20.8)
Other Africa	†
Southeast Asia	28.0 (23.4 to 33.0)
Northeast Asia	19.6 (15.5 to 24.2)
Other Asia	†
Americas	†
<i>Aged 65 to 74 years</i>	
Australia	60.0 (57.0 to 63.1)
Other Oceania	69.4 (50.0 to 92.0)
Northwest Europe	44.0 (38.1 to 50.3)
Southeast Europe	58.5 (51.7 to 65.9)
North Africa/Middle East	70.7 (54.5 to 89.1)
Other Africa	†
Southeast Asia	91.6 (71.0 to 114.7)
Northeast Asia	95.7 (77.6 to 115.7)
Other Asia	†
Americas	†
<i>Aged ≥75 years</i>	
Australia	180.9 (175.6 to 186.3)
Other Oceania	211.1 (168.7 to 258.3)
Northwest Europe	143.9 (132.5 to 155.8)
Southeast Europe	174.5 (160.4 to 189.1)
North Africa/Middle East	212.8 (175.7 to 253.4)
Other Africa	†

Continued

Table 11. Continued

Region of Birth	ASR, WHO World Population (95% CI)
Southeast Asia	280.7 (235.3 to 330.0)
Northeast Asia	281.3 (242.3 to 323.1)
Other Asia	†
Americas	†

ASR indicates age-standardized rate; WHO, World Health Organization.

*To minimize small cell sizes when assessing interactions between age and region of birth and age and socioeconomic status, age categories were collapsed to 20-year age groups commencing from ≥25 years. Population data (ie, denominator information) for region of birth for the years 20 to 24 was unavailable.

†Cells based on sample sizes ≥10 or cells that can be used to deduce cells with ≥10 patients have been suppressed to protect patient privacy in accordance with local standards.

of birth within the same geographically defined area and time frame. Stroke risk associated with country of birth has been explored in Australia, but that analysis excluded ICH.⁴⁵ Our analysis represented 10 regions and overcame limitations of comparing rates from different countries, for which differences in health service delivery, case selection, and time period under study may have confounded observed geographic variations. Using administrative data, we report that risks are elevated for patients born in Northeast and Southeast Asia but not in other Asian regions. Elevated incidence in Pacific Islander and Asian populations has been reported in Auckland, New Zealand, using gold standard epidemiological methods.⁴⁶ Residents born in the Middle East or North Africa also experienced an increased risk of nonfatal ICH; few studies have explored ICH risk in these populations.^{47,48} In all cases, these patients experienced ICH at younger ages than Australia-born residents, exacerbating the burden of disease in these communities. Evidence from this study and others⁴⁶ suggests that the increased prevalence of hypertension, renal disease, and diabetes likely underpins the increased risk seen in these groups. Significant effect modification between region of birth and age is a novel finding suggesting different etiological influences. The risk associated with being born in the Oceania region appeared stronger in those aged <65 years, whereas the increased risk of ICH seen in people of Northeast Asian origin was evident only in those aged >45 years. Southeast Asia-born members of the community were at a similarly elevated risk throughout adulthood, with our results suggesting that AVMs are an important etiological risk factor.

Approximately 12% of ICH admissions in this population could have been prevented if the socioeconomic circumstances of the population were equal with those of the least disadvantaged in the Australian population. The relationship between SES and stroke risk has been reported previously, although results appear more consistent for ischemic rather

Table 12. ICH Risk Factors[#] by Sex, Age, and SES

Variable	Median Age (IQR)	Hypertension	Diabetes	Cerebral Malignancies	Smoking	Alcohol/Drug Use	Renal Disease	Anticoagulant Use	AVMs/Other Malformations
<i>Sex</i>									
Male	73 (62 to 80)	64.1	14.5	3.7	37.5	8.2	8.1	9.0	2.7
Female	79 (69 to 85)*	64.1	10.5*	1.9*	18.4*	2.5*	5.1*	6.6*	1.9 [†]
<i>Age (y)</i>									
20 to 39	—	28.6	‡	5.3	33.0	18.9	5.3	‡	19.2
40 to 49	—	50.6	6.5	4.3	39.7	17.4	5.6	3.2	8.6
50 to 59	—	64.4	12.0	5.3	38.6	13.8	6.2	4.1	4.7
60 to 69	—	68.2	16.3	4.5	40.9	8.4	7.0	8.1	2.2
70 to 79	—	67.6	15.9	2.7	28.8	3.5	6.9	10.1	1.1
80 to 89	—	65.3	10.6	1.2	19.2	1.1	6.8	8.6	0.5
≥90	—	59.2*	8.1*	‡*	10.1*	‡*	6.0	4.9*	‡*
<i>IRSD (SES)</i>									
Quintile 1 (low)	75 (63 to 81)	66.6	14.5	3.0	28.9	6.1	8.1	5.1	2.0
Quintile 2	75 (63 to 82)	65.2	13.9	2.8	29.9	4.6	7.0	9.5	2.3
Quintile 3	75 (65 to 82)	63.6	14.1	3.0	29.6	5.7	6.1	8.0	1.9
Quintile 4	76 (65 to 83)	63.3	11.2	2.5	29.6	5.4	7.2	8.2	2.6
Quintile 5 (high)	78 (68 to 85)*	63.8	10.9*	2.8	23.7*	4.9	5.7 [§]	7.9*	2.5
<i>Region of birth</i>									
Australia	77 (66 to 84)	62.1	10.9	3.2	27.9	5.9	6.1	8.1	2.3
Oceania	63 (51 to 77)	67.4	15.2	‡	27.9	5.1	8.3	7.6	‡
Northwest Europe	78 (69 to 84)	63.2	10.1	3.2	34.7	4.8	5.1	8.5	1.5
Southeast Europe	76 (69 to 82)	69.5	19.8	1.9	29.3	3.6	10.1	7.7	1.2
North Africa/Middle East	73 (62 to 79)	76.3	28.8	‡	33.1	‡	9.3	5.4	‡
Other Africa	75.5 (62.83)	68.2	†	‡	19.7	‡	‡	‡	‡
Southeast Asia	67 (52 to 78)	74.3	16.0	‡	20.7	‡	8.4	4.7	3.4
Northeast Asia	73 (63 to 81)	71.4	14.3	‡	20.0	‡	7.3	5.7	‡
Other Asia	68 (55 to 78)	74.1	18.8	‡	17.6	‡	‡	‡	‡
Americas	65.5 (58 to 76)*	60.0*	20.0*	‡	38.0*	‡*	‡*	11.0 [§]	‡

AVM indicates arteriovenous malformation; ICH, intracerebral hemorrhage; IRSD, Index of Relative Socio-Economic Disadvantage; IQR, interquartile range; SES, socioeconomic status.

* $P < 0.001$.

[†] $P = 0.006$.

[‡]Cells based on sample sizes <10 or cells that can be used to deduce cells with <10 patients have been suppressed to protect patient privacy in accordance with local standards.

[§] $P = 0.05$.

^{||} $P = 0.008$.

[#]All risk factors ascertained using International Classification of Diseases, 10th revision-coded comorbidities recorded during the admission and in any hospital admission that occurred during the 6 months prior to the ICH admission with the exception of anticoagulant use, where recorded use in the current ICH acute admission was ascertained.

than hemorrhagic stroke.^{49–52} In this study, SES was correlated with overall, fatal, and nonfatal ICH attack rates but not with fatal ICH at 365 days for 30-day survivors. Our finding of an association with socioeconomic deprivation and fatal ICH incidence at 30 days is consistent with a recent study from France demonstrating an association between socioeconomic deprivation and stroke case mortality in the

early postacute period (up to 90 days after stroke), specifically, after the first 2 weeks.⁵³ As in France, Australia has a universal healthcare system with free access to public hospitals and thus minimal financial barriers to access acute care facilities; in the New South Wales health system, acute stroke is managed overwhelmingly in public hospitals, with only 1 private hospital offering an acute stroke unit. Access to

postacute care, rehabilitation, and community support services may be more variable, providing patients with greater resources and possibly influencing survival in the postacute period. That SES did not affect 365-day mortality for 30-day survivors in our study may reflect that ICH survivors are more homogenous in their SES compared with patients who die from ICH.

ICH risk due to socioeconomic disadvantage was not evident among people aged >80 years, suggesting that the influence of SES may wane with increasing age. Alternatively, geographically derived estimates of SES among the elderly may be subject to misclassification because retirement and aged-care facilities may skew scores in their geographic locations. Location of residence was used to determine SES rather than individually assessed indicators of affluence; therefore, our finding may be vulnerable to an ecological fallacy. However, identifying geographic pockets of stroke risk allows targeting of health services and clinicians to provide services in areas in need.

Metrics of stroke mortality include case fatality^{1,2}; mortality rates calculated using cause of death data; and, as we have done, calculation of fatal incidence or attack rates, selecting patients with an acute presentation of stroke who die within a defined period (eg, 30 days).^{49,50} Determining the burden of fatal ICH in the population requires reference to population denominators, and our results demonstrate heightened burden within subgroups. We found that fatal ICH rates are higher for men, increase with age, and vary according to region of birth and SES.

Limitations

Exhaustive case ascertainment and clinician-validated stroke diagnoses are methodological strengths of gold standard epidemiological stroke studies that cannot be matched by research using administrative health data; however, administrative data sets cost-effectively collect health information continuously over time for large jurisdictions yielding high numbers of cases.⁵

The available evidence supports high levels of coding accuracy in Australia. In general, positive predictive values of principal diagnoses in Australian data sets are high (>90%) and are often $\geq 95\%$,^{18,24,54–57} with extensive formal training of coders, routine auditing, and high levels of interrater reliability.²¹ Two Australian validation studies reported high positive predictive values of principal stroke diagnostic codes (95%²⁴ and 96%⁵⁶), with 1 study attributing all inaccuracies to clinician-validated ischemic strokes being assigned an ICD code for *unspecified stroke*.⁵⁶ Because this study determined attack rates of stroke and its subtypes, the validation analysis implies that stroke subtypes including ICH were coded accurately in all cases except for ischemic stroke. Although

the sensitivity of Australian stroke coding has not been specifically reported, researchers have validated diagnostic coding of stroke mimics, including transient ischemic attack, migraine with hemiparesis, and hypoglycemia, to determine whether strokes were misclassified as such.²⁴ None of these events were considered to be strokes on clinician review, indicating that sensitivity is likely to be high.

These Australian results concur with international reports of high levels of accuracy for stroke coding and, specifically, ICH coding. The specificity and positive predictive value of a principal ICH diagnosis was reported in a US study to be 96% and 89%, respectively.⁵⁸ In Canada and England, the positive predictive value of ICH ICD-10 stroke coding has been reported as 98% and 95.9%, respectively.^{59,60} A systematic review including the US and Canadian study cited above reports positive predictive values of 92% and 100% of a principal ICH diagnosis in an additional 2 studies.⁶¹

In France, researchers citing unpublished data reported very high levels of sensitivity and specificity of stroke coding (96% and 93%, respectively),³⁰ and in Sweden, the sensitivity of stroke hospitalization coding has been reported to be 89.3% for definite or possible strokes according to Multi-national Monitoring of Determinants and Trends in Cardiovascular Disease (MONICA).¹⁶ An Italian study noted positive predictive values of principal and secondary ICH codes between 81% and 86%; the inclusion of secondary codes would have likely resulted in some misclassification, given reports that secondary codes reduce coding accuracy.⁶² The sensitivity of principal ICH diagnoses has been reported to be 85% (ICD-9),⁵⁹ 87.9% (ICD-10),⁶³ and 78% (ICD-9),⁶⁴ with the authors of the latter study noting potential misclassification in the gold standard, which would have contributed to inaccuracies. We acknowledge that high levels of accuracy have not been universally reported. One study validated ICD-10 ischemic stroke coding, reporting high positive predictive value of 95.1% but low sensitivity of 67.3%⁶⁵; these results may not be generalizable to ICH coding because higher levels of accuracy have been reported for hemorrhagic compared with ischemic stroke coding.^{58,59,62} Although sensitivity of ICH principal diagnoses were reported to be high (87.9%) in 1 French study,⁶³ the positive predictive value and false-positive and false-negative rates were reported to be 64.8%, 16.2%, and 7.0%, respectively. However, this study also reported substantially improved positive predictive values of stroke coding over a 5-year period (from 54.3% to 81.2%), in line with increasing coder experience of a recently implemented costing model using hospital administrative data.

In Australia and elsewhere (eg, United Kingdom, United States, New Zealand, Sweden), principal diagnoses reflect the main reason for the patient admission; however, in other jurisdictions, including France until 2009 and Italy, the main condition noted in administrative data sets reflects the

condition that consumes the most resources during the hospital stay.⁶⁶ It has been suggested that such a *resource use* criteria for defining the main condition may underascertain cases.⁶⁶ We note that variability in the reported accuracy across jurisdictions may be caused by several factors including the use of different definitions of a principal diagnosis; differences in the classification system used and in defining gold standards⁶⁶; validation of principal diagnosis versus both principal and secondary diagnoses^{58,61}; the length of time administrative coding has been used to enable financial reimbursement; and stroke subtype,⁶³ with hemorrhagic stroke coding generally found to be more accurate than ischemic stroke.^{58,59,62}

We included only principal ICH diagnoses, as elsewhere,^{28–32} and thus may have underenumerated ICH if cases were not coded in the primary diagnostic position; however, the positive predictive value of principal ICH codes is high (89% to 98%),^{58–60} and selecting secondary diagnoses reduces specificity without improving the sensitivity of case selection.⁵⁸

Comorbidities tend to be underenumerated when compared with the original medical records but are coded with very low false-positive rates (<0.3%),¹¹ although 1 recent Australia study using New South Wales hospital admissions data—as we have done—found that comorbidities ascertained from administrative data correlated well with medical records.¹⁸ Furthermore, we applied the maximum available look-back period of 6 months to minimize underascertainment and capture diagnoses recorded in those previous hospital attendances. Although a longer look-back period would have further maximized capture, more proximally experienced risk factors may have had greater impact on ICH risk and patient outcomes than those that could have been identified only in the more distant medical history.²⁰

We cannot exclude the possibility that temporal changes in coding may have influenced our findings. We considered hospital admission rates as proxies for attack rates and acknowledge that deaths occurring outside the hospital due to an acute ICH or *mild* cases not reaching the hospital would have been missed. Internationally, it has been reported that very few ICH cases are not hospitalised,^{5,23} and the percentage of out-of-hospital strokes in Australia has been declining over time. The Perth Community Stroke Study reported declines in out-of-hospital strokes from 21.5% in 1989–1990 to 7.7% in 2000–2001,² and another Australian study carried out in 2009–2010 reported that only 4% of strokes do not reach the hospital.⁶⁷ This trend would have underestimated our reported average annual decline in ICH rates. Furthermore, during the study period, health services in New South Wales experienced a growth of funding in stroke care that was conditional upon providing greater inpatient access for stroke patients. A series of state government task forces particularly addressed equitable access to stroke services and provided

additional funding and resources linked to evidence-based practices and pathways beginning in emergency departments to ensure that patients with focal neurological symptoms, syncope, and headaches were directed to specialist inpatient services⁶⁸ (<http://www.aci.health.nsw.gov.au/networks/stroke/about>). During the study period, hospitals in our health service received recurrent funding to support the implementation of stroke units and noted increases in the numbers of stroke admissions to these units.³⁸ Education of general practitioners and members of the local community was undertaken to increase community and primary care services for focal neurological symptoms and the need for their urgent referral to the hospital. Consequently, in the health service under study, a trend toward greater resources and admissions would have underestimated declining admission rates in our study.

Hospitalized cases transferred outside the region may have been lost to follow-up because state-based death registries do not record deaths of persons dying outside their state of residence unless requested by family; however, there were few transfers (1.8%), and it is likely that patients with better prognoses are transferred for treatment and/or rehabilitation, having survived the acute period. We may have underestimated variation in stroke risk according to region of birth because first-generation Australian residents may share risk factors with their immigrant parents. Furthermore, nationality at birth does not necessarily reflect the cultural and linguistic diversity within countries that may also affect stroke risk.⁶⁹

Conclusion

We report falling overall and fatal ICH rates, which have rarely been reported. These decreases may reflect the impact of cardiovascular prevention measures and innovations in health service delivery. Men, those living in areas of relatively greater socioeconomic disadvantage, and those born in some regions of the world were at heightened risk of ICH and of dying from it.

Disclosures

John Worthington is currently medical co-chair of Stroke Services, New South Wales, an honorary leadership position tasked with executive decision making regarding stroke services.

References

- van Asch CJJ, Luitse MJA, Rinkel GJE, van der Tweel I, Algra A, Klijn CJM. Incidence, case fatality, and functional outcome of intracerebral haemorrhage over time, according to age, sex, and ethnic origin: a systematic review and meta-analysis. *Lancet Neurol*. 2010;9:167–176.
- Islam MS, Anderson CS, Hankey GJ, Hardie K, Carter K, Broadhurst R, Jamrozik K. Trends in incidence and outcomes of stroke in Perth, Western

- Australia 1989–2001. The Perth Community Stroke Study. *Stroke*. 2008;39:776–782.
3. Rothwell PM, Coull AJ, Giles MF, Howard SC, Silver LE, Bull LM, Gutnikov SA, Edwards P, Mant D, Sackley CM, Farmer A, Sandercock PA, Dennis MS, Warlow CP, Bamford JM, Anslow P; Oxford Vascular Study. Change in stroke incidence, mortality, case-fatality, severity, and risk factors in Oxfordshire, UK from 1981 to 2004 (Oxford Vascular Study). *Lancet* 2004;363:1925–1933.
 4. Kleindorfer DO, Khoury J, Moomaw CJ, Alwell K, Woo D, Flaherty ML, Khatri P, Adeyoye O, Ferioli S, Broderick JP, Kissela BM. Stroke incidence is decreasing in whites but not in blacks: a population-based estimate of temporary trends in stroke incidence from the Greater Cincinnati/Northern Kentucky Stroke Study. *Stroke*. 2010;41:1326–1331.
 5. Kleindorfer D. The bad news: stroke incidence is stable. *Lancet Neurol*. 2007;6:470–471.
 6. Cordonnier C, Rutgers MP, Dumont F, Pasquini M, Lejeune JP, Garrigue D, Béjot Y, Leclerc X, Giroud M, Leys D, Hénon H. Intracerebral haemorrhages: are there any differences in baseline characteristics and intra-hospital mortality between hospital and population-based registries? *J Neurol*. 2009;256:198–202.
 7. Anderson CS, Heeley E, Huang Y, Wang J, Stapf C, Delcourt C, Lindley R, Robinson T, Lavados P, Neal B, Hata J, Arima H, Parsons M, Li Y, Wang J, Heritier S, Li Q, Woodward M, Simes J, Davis SM, Chalmers J. Rapid blood-pressure lowering in patients with acute intracerebral haemorrhage. *N Engl J Med*. 2013;368:2355–2365.
 8. Morgenstern LB, Hemphill JC, Anderson C, Becker K, Broderick JP, Connolly ES, Greenberg SM, Huang JN, Macdonald RL, Messé SR, Mitchell PH, Selim R; J on behalf of the American Heart Association Stroke Council and Council on Cardiovascular Nursing. AHA/ASA Guidelines for the management of spontaneous intracerebral hemorrhage. *Stroke*. 2010;41:2108–2129.
 9. National Centre for Classification in Health. Australian Coding Standards for ICD-10-AM and ACHI, Australian Classification of Health Interventions (ACHI). *The International Statistical Classification of Diseases and Related Health Problems. Tenth Revision, Australian Modification (ICD-10-AM)*. 6th ed. Sydney: University of Sydney; 2008.
 10. Centre for Health Record Linkage (CheReL). 2012 Quality Assurance Report. Available at: <http://www.cherel.org.au/quality-assurance>. Accessed March 16, 2014.
 11. Preen DB, Holman CDJ, Lawrence DM, Baynham NJ, Semmens JB. Hospital chart review provided more accurate comorbidity information than data from a general practitioner survey or an administrative data-base. *J Clin Epidemiol*. 2004;57:1295–1304.
 12. Ahmad OB, Boschi-Pinto C, Lopwz AD, Murray CJL, Lozano R, Inoue M. Age standardization rates: a new WHO standard. GPE Discussion Paper Series: No. 31. EIP/GPE/EDB World Health Organisation, 2001.
 13. Thorvalden P, Asplund K, Kuulasmaa K, Rajakangas AM, Schroll M. Stroke Incidence, case fatality and mortality in the WHO MONICA project. *Stroke*. 1995;26:361–367.
 14. Jamrozik K, Broadhurst RJ, Lai N, Hankey GJ, Burvill PW, Anderson CS. Trends in the incidence, severity and short-term outcome of stroke in Perth, Western Australia. *Stroke*. 1999;30:2105–2111.
 15. Thrift AG, Dewey HM, Macdonell RAL, McNeil JJ, Donnan GA. Stroke incidence on the East Coast of Australia. *Stroke*. 2000;31:2087–2092.
 16. Köster M, Asplund K, Johansson Å, Stegmayr B. Refinement of Swedish administrative registers to monitor stroke events on the national level. *Neuroepidemiology*. 2013;40:240–246.
 17. ABS. Socio-economic Indexes for Areas (SEIFA), Technical Paper 2006. Canberra: Commonwealth of Australia; 2008. Available at: <http://www.abs.gov.au/ausstats/abs@.nsf/mf/2039.0.55.001/>. Accessed March 16, 2014.
 18. Robertson J, Pearson SA, Attia JR. How well do NSW hospital data identify cases of heart failure. *Med J Aust*. 2014;200:25.
 19. Gattellari M, Goumas C, Garden F, Worthington JM. Relative survival after transient ischaemic attack: results from the Program of Research Informing Stroke Management (PRISM) study. *Stroke*. 2012;43:79–85.
 20. Chen JS, Roberts CL, Simpson JM, Ford JB. Use of hospitalisation history (lookback) to determine prevalence of chronic diseases: impact on modelling of risk factors for haemorrhage in pregnancy. *BMC Med Res Methodol*. 2011;11:68.
 21. Henderson TR, Shepherd K, Sundararajan V. Quality of diagnosis and procedure coding in ICD-10 administrative data. *Med Care*. 2006;44:1011–1019.
 22. Béjot Y, Cordonnier C, Durier J, Aboa-Eboulé C, Rouaud O, Giroud M. Intracerebral haemorrhage profiles are changing: results from the Dijon population-based study. *Brain*. 2013;136:658–664.
 23. Zahuranec DB, Lisabeth LD, Sánchez BN, Smith MA, Brown DL, Garcia NM, Skolarus LE, Meurer WJ, Burke JF, Adelman EE, Morgenstern LB. Intracerebral hemorrhage mortality is not changing despite declining incidence. *Neurology*. 2014;82:2180–2186.
 24. Marsden DL, Spratt NJ, Walker R, Barker D, Attia J, Pollack M, Parsons MW, Levi CR. Trends in stroke attack rates and case fatality in the Hunter Region, Australia 1996–2008. *Cerebrovasc Dis*. 2010;30:500–507.
 25. Nedkoff L, Briffa TG, Knuiman M, Hung J, Norman PE, Hankey GJ, Thompson PL, Geelhoed E, Sanfilippo FM, Hickling S, Bremner A, Hobbs M. Temporal trends in the incidence and recurrence of hospitalised atherothrombotic disease in an Australian population: 2000–2007: data linkage study. *Heart*. 2012;98:1449–1456.
 26. Sturgeon JD, Folsom AR. Trends in hospitalization rate, hospital case fatality, and mortality rate of stroke by sub-type in Minneapolis-St. Paul, 1980–2002. *Neuroepidemiology*. 2007;28:39–45.
 27. Towfighi A, Markovic D, Ovbiagele B. Recent patterns of sex-specific midlife stroke hospitalization rates in the United States. *Stroke*. 2011;42:3029–3033.
 28. Mayo NE, Nadeau L, Daskalopoulou DD, Côté R. The evolution of stroke in Quebec: a 15-year perspective. *Neurology*. 2007;68:1122–1127.
 29. Fang J, Alderman MH, Keenan NL, Croft JB. Declining US stroke hospitalization since 1997: National Hospital Discharge Survey, 1988–2004. *Neuroepidemiology*. 2007;29:243–249.
 30. Béjot Y, Aouba A, de Peretti C, Grimaud O, Aboa-Eboulé C, Chin F, Woimant F, Jouglé E. Time trends in hospital-referred stroke and transient ischaemic attack: results of a 7-year nationwide survey in France. *Cerebrovasc Dis* 2010; 30:346–354.
 31. George MG, Tong X, Kuklina EV, Labarthe DR. Trends in stroke hospitalizations and associated risk factors among children and young adults, 1995–2008. *Ann Neurol*. 2011;70:713–721.
 32. Rincon F, Mayer SA. The epidemiology of intracerebral hemorrhage in the United States from 1979 to 2008. *Neurocrit Care*. 2013;19:19–102.
 33. Australian Institute of Health and Welfare. Australia's Health in 2010. Australia's health series no. 12. Cat. No. AUS 122. Canberra: AIHW; 2010.
 34. Woo D, Sauerbeck LR, Kissela BM, Khoury JC, Szaflarski JP, Gebel J. Genetic and environmental risk factors for intracerebral hemorrhage: preliminary results of a population-based study. *Stroke*. 2002;33:1190–1196.
 35. NSW Government Health Statistics New South Wales. Key population health and performance indicators. Available at: <http://www.healthstats.nsw.gov.au/ContentText/Display/KeyHealthIndicators>. Accessed April 5, 2014.
 36. Arisen MJ, Claus SP, Rinkel GJE, Algra A. Risk factors for intracerebral hemorrhage in the general population: a systematic review. *Stroke*. 2003;34:2060–2066.
 37. Hertua K, Tabák AG, Martikainen P, Vahtera J, Kivimäki M. Adherence to antihypertensive therapy prior to the first presentation of stroke in hypertensive adults: population-based study. *Eur Heart J*. 2013;34:2933–2939.
 38. Gattellari M, Worthington J, Jalaludin B, Mohsin M. Stroke unit care in a real-life setting. Can results from randomized controlled trials be translated into everyday clinical practice? An observational study of hospital data in a large Australian population. *Stroke*; 40:10–17.
 39. Langhorne P, Fearon P, Ronning OM, Kaste M, Palomaki H, Vemmos K, Kalra L, Indredavik B, Blomstrand C, Rodgers H, Dennis MS, Al-Shahi Salman R; Stroke Unit Trialists' Collaboration. Stroke unit care benefits patients with intracerebral hemorrhage: systematic review and meta-analysis. *Stroke* 2013;44:3044–3049.
 40. Patil CG, Alexander AL, Hayden Gephart MG, Lad SP, Arrigo RT, Boakye M. A population-based study of inpatient outcomes after operative management of nontraumatic intracerebral hemorrhage in the United States. *World Neurosurg* 2012;78:640–645.
 41. Thrift AG, Dewey HM, Sturm JW, Srikanth VK, Gilligan AK, Gall SL, Macdonell RA, McNeil JJ, Donnan GA. Incidence of stroke subtypes in the North East Melbourne Stroke Incidence Study (NEMESIS): differences between men and women. *Neuroepidemiology*. 2009;32:11–18.
 42. Apellos P, Stegmayr B, Terént A. Sex differences in stroke epidemiology: a systematic review. *Stroke*. 2009;40:1082–1090.
 43. Ruiz-Sandoval JL, Cantú C, Barinagarrementeria F. Intracerebral hemorrhage in young people: analysis of risk factors, location, causes and prognosis. *Stroke*. 1999;30:537–541.
 44. Thanvi B, Robinson T. Sporadic cerebral amyloid angiopathy—an important cause of cerebral hemorrhage in older people. *Age Ageing* 2006;35:565–571.
 45. Dassanayake J, Gurrin L, Payne WR, Sundararajan V, Dharmage SC. Is country of birth a risk factor for acute hospitalization for cardiovascular disease in Victoria, Australia? *Asia Pac J Public Health*. 2011;23:280–287.
 46. Feigin V, Carter K, Hackett M, Barber PA, McNaughton H, Dyal L, Chen M, Anderson C. Ethnic disparities in incidence of stroke sub-types: Auckland

- Regional Community Stroke Study, 2002–2003. *Lancet Neurol.* 2006;5:130–139.
47. Tran J, Mirzaei M, Anderson L, Leeder SR. The epidemiology of stroke in the Middle East and North Africa. *J Neurol Sci.* 2010;295:38–40.
 48. Azaepazhooh MR, Etemadi MM, Donnan GA, Mokhber N, Majidi MR, Ghayour-Mobarhan M, Ghandehary K, Farzadfar MT, Kiani R, Panahandeh M, Thrift AG. Excessive incidence of stroke in Iran: evidence from the Mashhad Stroke Incidence Study (MSIS), a population-based study of stroke in the Middle-East. *Stroke.* 2010;41:e3–e10.
 49. Thrift AG, Dewey HM, Sturm JW, Paul SL, Gilligan AK, Srikanth VK, Macdonell RA, McNeil JJ, Macleod MR, Donnan GA. Greater incidence of both fatal and nonfatal strokes in disadvantaged areas: the Northeast Melbourne Stroke Incidence Study. *Stroke.* 2006;37:877–882.
 50. Cesaroni G, Agabiti N, Forastiere F, Peruci CA. Socioeconomic differences in stroke incidence and prognosis under a universal healthcare system. *Stroke.* 2009;40:2812–2819.
 51. Addo J, Ayerbe L, Mohan KM, Crichton S, Sheldenkar A, Chen R, Chen R, Wolfe CD, McKeivitt C. Socioeconomic status and stroke: an updated review. *Stroke.* 2012;43:1186–1191.
 52. Heeley E, Wei J, Carter K, Islam M, Thrift A, Hankey G, Cass A, Anderson CS. Socioeconomic disparities in stroke rates and outcome: pooled analysis of stroke incidence studies in Australia and New Zealand. *Med J Aust.* 2011;195:10–14.
 53. Grimaud O, Leray E, Lalloué B, Aghzaf R, Durier J, Giroud M, Béjot Y. Mortality following stroke during and after acute care according to neighbourhood deprivation: a disease registry study. *J Neurol Neurosurg Psychiatry.* 2014;85:1313–1318.
 54. Teng TH, Finn J, Geelhoed E, Hobbs M. A validation study: how effective is the Hospital Morbidity Data as a surveillance tool for heart failure in Western Australia? *Aust N Z J Public Health.* 2008;32:405–407.
 55. Brameld KJ, Thomas MAB, Holman CDJ, Bass AJ, Rouse IL. Validation of linked administrative data on end-stage renal failure: application of record linkage to a 'clinical base population'. *Aust N Z J Public Health.* 1999;23:464–467.
 56. Wang Y, Levi CR, Attia JR, D'Este CA, Spratt N, Fisher J. Seasonal variation in stroke in the Hunter region, Australia. A five-year hospital-based study, 1995–2000. *Stroke.* 2003;34:1144–1150.
 57. Lee AH, Somerford PJ, Yau KKW. Risk factors for ischaemic stroke recurrence after hospitalisation. *Med J Aust.* 2004;181:244–246.
 58. Tirschwell DL, Longstreth WT Jr. Validating administrative data in stroke research. *Stroke.* 2002;33:2465–2470.
 59. Kokotailo RA, Hill MD. Coding of stroke and stroke risk factors using International Classification of Diseases, Revisions 9 and 10. *Stroke.* 2005;36:1776–1781.
 60. Kirkman MA, Mahattanakul W, Gregson BA, Mendelow AD. The accuracy of hospital discharge coding for hemorrhagic stroke. *Acta Neurol Belg.* 2009;109:114–119.
 61. Andrade SE, Harrold LR, Tija J, Cutrona SL, Saczynski JS, Dodd KS, Goldberg RJ, Gurwitz JH. A systematic review of validated methods for identifying cerebrovascular accident or transient ischemic attack using administrative data. *Pharmacoepidemiol Drug Saf.* 2012;21(S1):100–128.
 62. Palmieri L, Barchielli A, Cesana G, de Campora E, Goldoni CA, Spalaore P, Ugucioni M, Vancheri F, Vanuzzo D, Ciccarelli P, Giampaoli S; Research Group of the Project 'Italian National Register of Coronary and Cerebrovascular Events. The Italian register of cardiovascular diseases: attack rates and case fatality for cerebrovascular events. *Cerebrovasc Dis* 2007;24:530–539.
 63. Aboa-Eboulé C, Mengue D, Benzenine E, Hommel M, Giroud M, Béjot Y, Quantin C. How accurate is the reporting of stroke in hospital discharge data? A pilot validation study using a population-based stroke registry as control. *J Neurol.* 2013;260:605–613.
 64. Jones SA, Gottesman RF, Shahar E, Wruck L, Rosamond WD. Validity of hospital discharge codes for stroke: the Arteriosclerosis Risk in Communities Study. *Stroke.* 2014;45:3219–3225 doi:10.1161/STROKEAHA.114.006316-/DC1.
 65. Haesebeert J, Termoz A, Polazzi S, Mouchoux C, Mechtouff L, Derex L, Nighoghossian N, Schott AM. Can hospital discharge databases be used to follow ischemic stroke incidence? *Stroke.* 2013;44:1770–1774.
 66. Quan H, Moskal L, Forster AJ, Brien S, Walke R, Romano PS, Sundararajan V, Burnand B, Henriksson G, Steinum O, Drosler S, Pincus HA, Ghali WA. International variation in the definition of the "main condition" in ICD-coded health data. *Int J Qual Health Care.* 2014;26:511–515.
 67. Leyden JM, Klenig TJ, Newbury J, Castle S, Cranefield J, Anderson CS, Crotty M, Whitford D, Jannes J, Lee A, Greenhill J. Adelaide stroke incidence study: declining stroke rates but many preventable cardioembolic strokes. *Stroke.* 2013;44:1226–1231.
 68. Cadilhac DA, Pearce DC, Levi CR, Donnan GA. Improvements in the quality of care and health outcomes with new stroke care units following implementation of a clinician-led, health system redesign programme in New South Wales, Australia. *Qual Saf Health Care.* 2008;17:329–333.
 69. Katzenellenbogen JM, Vos T, Somerford P, Begg S, Semmens JB, Codde JP. Burden of stroke in indigenous Western Australians: a study using data linkage. *Stroke.* 2011;42:1515–1521.