Audit of longterm mortality and morbidity outcomes for carotid endarterectomy

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Abstract

There have been no Australian studies of longterm mortality status and cause of death after carotid endarterectomy (CEA) or, for survivors, quality-of-life. We first determined rate and cause of death for a cohort of patients four years after CEA. Surviving patients were surveyed to ascertain health status, using MOS SF-36. Of 238 patients who underwent CEA in our health service in 1995, 44 (18.5%) had died within four years. The majority of deaths (61.4%) were attributable to vascular causes. Of the surviving 162 patients (survey response fraction 90%), 10 (6.2%) subsequently had suffered a non-fatal stroke in the four years following their CEA. With respect to health status, Physical Functioning scores differed significantly by age (t=2.65, df=149, P=0.01) as did Role Physical scores (t=2.10, df=142, P=0.04). We conclude that patients undergoing CEA are at high risk of dying from vascular causes, inviting concerted efforts in discharge planning to co-ordinate optimal vascular risk factor management.

Literature review

Randomised controlled trials have established the efficacy of carotid endarterectomy (CEA) to reduce stroke risk (North American Symptomatic Carotid Endarterectomy Trial Collaborators [NASCET] 1991; European Carotid Surgery Trialists' Collaborative Group [ECST] 1998; Executive Committee for the Asymptomatic Carotid Atherosclerosis Study [ACAS] 1995; Hobson, Weiss, Fields, Goldstone, Moore, Towne and Wright 1993; Mayo Asymptomatic Carotid Endarterectomy Study Group 1992; Carotid artery stenosis with asymptomatic narrowing: Operation versus aspirin Study Group [CASANOVA] 1991).

National audits (Wennberg, Lucas, Birkmeyer, Bredenberg and Fisher 1998; Troëng, Bergqvist, Norrving and Ahari 1999; Estes, Guadagnoli, Wolf, LoGerfo and Whittemore 1998), state audits (Middleton and Donnelly 2000; Rodgers, Oliver, Dobson and Thonson 2000; Kresowik, Hemann, Grund, Hendel, Brenton, Wiblin, Adams and Ellerbeck 2000) and local audits (Frawley, Hicks, Horton, Gray, Niesche and Matheson 1994; Appleberg, Cotter, Crozier, Graham and Lane 1995; Besser and Parkinson 1999) subsequently have been undertaken to determine short-term outcomes under non-trial conditions.

As an outcome indicator, mortality following CEA is most typically measured 30 days after surgery (NASCET 1991; ECST 1998; ACAS 1995; Hobson et al. 1993; Mayo Asymptomatic Carotid Endarterectomy Study Group 1992; CASANOVA 1991; Wennberg et al. 1998; Troëng et al. 1999; Estes et al. 1998; Middleton and Donnelly 2002; Rodgers et al. 2000; Kresowik et al. 2000; Frawley et al. 1994; Appleberg et al. 1995; Besser and Parkinson 1999). Longer-term mortality is reported far less commonly (Coyle, Smith, Gray, Salam, Dodson, Chaikof and Lumsden 1995; Lord 1984). Not all studies report cause of death.

Studies examining disease-specific morbidity following CEA typically report the incidence of non-fatal stroke (NASCET 1991; ECST 1998; ACAS 1995; Hobson et al. 1993; Mayo Asymptomatic Carotid Endarterectomy Study Group 1992; CASANOVA 1991). Functional status is less frequently reported. Five studies have measured patients' neurophysiological performance after CEA, using objective measures of cognitive ability following CEA (Diener, Hamster and Seboldt 1984; Hemmingsen, Mejsholm, Vorstrup, Lester, Engell and Boysen 1986; Jacobs, Ganji, Shirley, Morrell and Brinkman 1983; van den Burg, Saan, van Zomeren, Boontje, Haaxma and Wichmann 1985; King, Gideon, Haynes, Dempsey and Jenkins 1997). For some, demonstrable intellectual improvement has been reported (Diener et al. 1984; Hemmingsen et al. 1986; Jacobs et al. 1983).

Six studies have examined patient health status and quality of life post-CEA (Vriens, Post van Huffelen and Eikelboom 1998; Sirkka, Salenius, Portin and Nummenmaa 1992; De Leo, Serraiotto, Pellegrini, Magni, Franceschi and Deriu 1987; Parker, Granberg, Nichols, Jones and Hewett 1983; Dardik, Minor, Watson and Hands 2001; Trudel, Fabia and Bouchard 1984) but only two followed up patients for more than eight months (Sirkka et al. 1992; Trudel et al. 1984). One of these two studies measured outcomes up to nine years post-CEA for 50 patients, using an activities-of-daily-living scale (Trudel et al. 1984). Unsurprisingly, patients with at least one non-neurological comorbidity following CEA were found to have marked dysfunction in home and outside activities as well as social interaction (Trudel et al. 1984). The second study compared patients who underwent CEA following angiography and those who did not, finding no significant differences in quality-of-life between operated and non-operated patients (Sirkka et al. 1992).

Generic 'health status' also represents a relevant quality indicator for CEA. Only one study has reported health status using the MOS Short Form 36 Health Survey Questionnaire (SF-36) (Dardik et al. 2001). Fifty symptomatic patients completed the SF-36 pre-operatively and again three months post-CEA (response rate 89%). This study found no significant differences between pre and post-operative scores for any of the eight SF-36 health domains. There was a significant improvement only in the mean 'change in health' scores (Dardik et al. 2001). The short-term nature of the three-month assessment also limits the utility of this study.

Evaluation of outcomes is receiving increased emphasis in the Australian health care system, (National Expert Advisory Group on Safety and Quality in Australian Health Care 1998). We therefore designed a comprehensive audit to determine mortality, cause of death and, for surviving patients, health status four years after CEA.

Method

Our study sample comprised all patients who had had a first or only CEA performed at any public hospital facility in Central Sydney during 1995 (ICD-9-CM procedure code 38.12: Endarterectomy of the head and neck). Patient's episode of care was categorised using the Australian National Diagnosis Related Group (AN-DRG) classification system as follows: those who underwent CEA in conjunction with cardiothoracic surgery; those who underwent a CEA, had no comorbidities and did not experience a complication ('without CCs'); and those who underwent a CEA, had a history of comorbidities (either pre- or post-operatively) and/ or experienced complications during the admission ('with CCs').

Consent for patient recruitment was first obtained from surgeons. Names and details of all patients were sent to the National Death Index (NDI) at the Australian Institute of Health and Welfare for matching for mortality status and cause of death. The NDI database contains more than 2.3 million records of all deaths occurring anywhere in Australia since 1980 (http://www.aihw.gov.au/cancer/ndi/ndi.html).

For surviving patients, surgeons signed a standardised letter inviting their participation in our study. Surviving patients were considered ineligible to participate if they or a relative notified the researchers that they were too ill to complete the questionnaire, suffered from dementia, did not speak English, or were unable to complete the questionnaire without assistance due to poor command of the English language.

With surgeon and patient consent, we mailed a 23-page questionnaire to surviving patients. It included items to determine sociodemographic information (five questions), return of vascular symptoms post-CEA (two questions) and history of second CEA (two questions). We also reproduced the MOS SF-36 (Ware 1993) to score the following eight health domains: *Physical Function* (PF), *Role - Physical* (RP), *Bodily Pain* (BP), *General Health* (GH), *Vitality* (V), *Social Functioning* (SF), *Role - Emotional* (RE) and *Mental Health* (MH). A single 'health transition rating' also was obtained where patients were asked to rate their current health compared with their health one year previously (Ware 1993).

Seven days after this mail-out, non-responders received a phone prompt. Standardised follow-up was instituted for non-responders.

Data analysis

Univariate associations between mortality status at four years by hospital, sex, AN-DRG, type of surgeon, age group, cross-boundary referrals (those patients who had their operation performed within CSAHS but were not local residents) and length of stay were performed using χ^2 tests (SPSS version 9.0) (Norusis 1999). Logistic regression then was conducted to identify independent predictors of death.

To describe morbidity among surviving patients, univariate associations between the outcome of stroke and the following variables were examined using χ^2 tests: age; sex; hospital, insurance (public, private, veteran); pre-operative symptom status (symptomatic, asymptomatic); length of stay; AN-DRG; smoking status; hypertension; employment status; return of symptoms; and / or report of one or more neurological signs or symptoms since 1995 CEA (poor vision in one eye, double vision, speech difficulties, weakness / numbness in arms or legs).

Standardised formulas were used to calculate MOS-SF-36 scores (Ware 1993; Medical Outcomes Trust 1994). High mean scores for each of the eight health domains reflect better states of health and wellbeing (Ware 1993). Differences in mean values for all of the eight SF-36 health domains were examined by sex and age using t-tests. Population norms by sex for MOS SF-36 scores were obtained from the Australian National Health Survey 1995 (Australian Bureau of Statistics 1995). This publication presents profiles for various demographic subgroups and also for patients experiencing various types of illness.

We compared mean scores for the eight SF-36 health domains in our sample with these Australian population norms for healthy males and females of similar ages for patients in our study (Australian Bureau of Statistics 1995). Furthermore, as CEA is a stroke prevention strategy, we sought also to compare mean scores for surviving patients aged 55 years and over with Australian population norms for those of the same age who had experienced a stroke (Australian Bureau of Statistics 1995). As the published Australian Bureau of Statistics norms did not provide specific Design Effects (DEFFs) for each of the health domains, it was not possible to conduct statistical comparisons between our sample and the National Health Survey sample (Australian Bureau of Statistics 1995).

Results

In total, 238 patients were identified as having had a first or only CEA in 1995 at a facility in Central Sydney. Twenty-eight patients (11.8%) had had a cardiothoracic procedure (either a valve replacement or coronary artery bypass surgery) performed as a staged operation at the same time as their CEA. Two hundred and ten (88.2%) patients had CEA performed without simultaneous cardiothoracic surgery. Table 1 summarises patient characteristics by hospital.

These first or only CEAs had been performed by 16 different surgeons: four (25.0%) at Concord Repatriation General Hospital (CRGH) and 12 (75.0%) at Royal Prince Alfred Hospital (RPAH). Eight (50.0%) surgeons specialised in vascular surgery; five (31.3%) in cardiothoracic surgery (all from RPAH); two (12.5%) in neurosurgery; and one (6.3%) in liver transplantation. All surgeons agreed to participate in the study.

As expected, we found a significant univariate association between AN-DRGs and length of stay. Patients who underwent cardiothoracic surgery simultaneously with their CEA (n=28, 100%) had a length of stay greater than or equal to seven days while 68.4% (n=39) of the 57 patients who underwent CEA without cardiothoracic surgery but who experienced comorbidities and complications (according to AN-DRG coding) had a length of stay greater than or equal to seven days. By contrast, only 30.1% (n=46) of the 153 patients who underwent CEA without cardiothoracic surgery and had no comorbidities or complications stayed seven days or more (χ^2= 59.6, df = 2, P<0.001). These differences remained significant when the cardiothoracic patients were excluded from the analysis (χ^2 = 25.4, df = 1, P<0.001). In contrast to previous findings, female sex was not significantly associated with length of stay (z=-0.423, P=0.67) (Roddy, Estes, Kwoun, O'Donnell and Mackey 2000).

Mortality

Four years after CEA, 44 patients had died (overall mortality rate 18.5%) (95% CI: 13.9% - 23.8%). Four of these 44 patients had died within 30 days of their CEA operation giving a 30-day mortality rate of 1.7%. There were no significant differences in characteristics of those four patients who died within 30 days of their CEA and all other patients (n=234) by age (≤75 or > 75 years) (χ^2 =1.25, df=1, P=0.26), sex (χ^2 =0.05, df=1, P=0.82), or length of hospital stay (< six days or ≥ seven days) (χ^2 =1.23, df=1, P=0.27) and insurance status (χ^2 =3.31, df=2, P=0.20).

Mortality at four years further was unrelated to patient sex (χ^2 =0.70, df=1, P=0.40), surgeon specialty (χ^2 = 0.00, df=3, P=0.98), age (OR 1.00, 95% CI: 0.96 - 1.04), AN-DRG (χ^2 =1.20, df=2, P=0.50), length of stay (χ^2 =3.20, df=3, P=0.35) and place of residence (χ^2 =1.27, df=1, P=0.26). However, univariate analysis demonstrated two significant unadjusted associations with mortality at four years, namely hospital and insurance type (public, private or veteran). Specifically, 23.9% of patients at CRGH had died compared with 13.6% of patients at RPAH (χ^2 =4.17, df=1, P=0.04). Further, 35.3% of veterans died compared with 17.6% of public patients and 12.7% of private patients (χ^2 =8.22, df=2, P=0.02). This apparent univariate effect of hospital on mortality was confounded because all veterans had been operated on at CRGH. Yet war veterans were no more likely to have had their episode of care coded into the AN-DRG code with CCs than the AN-DRG without CCs (χ^2 =0.12, df=1, P=0.75). After adjusting for age and sex but excluding hospital from the logistic regression model, insurance status remained a significant predictor of death. Specifically, war veterans were independently more likely to have died post-CEA when compared with public (OR 2.63, 95% CI: 1.05 - 6.67) or privately insured patients (OR 3.85, 95%CI: 1.43 - 11.11).

The most frequently recorded cause of death was acute myocardial infarction (10 patients, 22.7% of all deaths) (Table 2). In total, 27 (61.4%) deaths within four years of CEA were attributable to vascular disease of any type (Table 2).

Disease-specific morbidity

Of 194 surviving patients, three patients were ineligible to complete the questionnaire due to poor health (not due to non-fatal stroke however); another four were ineligible due to language difficulties and a further six were uncontactable. From 181 eligible patients, we received 162 completed questionnaires (response rate 90%). There was no difference between sex (χ^2=0.15, df=1, P=0.70,), age group (≤ 75 years or > 75 years) (χ^2=1.03, df=1, P=0.31), hospital where CEA was performed (χ^2= 2.21, df=1, P=0.14) or insurance status (χ^2=4.80, df=2, P=0.09) of responders compared with non-responders.

Ten (6.2%) (95% CI: 3.2 - 11.4) of the 162 surviving patients had suffered a non-fatal stroke in the four years following their CEA. Nine of these (8.2% of 110 symptomatic patients) had been symptomatic at the time of their CEA while only one (2.0% of 51 asymptomatic patients) was asymptomatic. There was no significant difference between pre-operative symptom status and stroke four years post-CEA (χ^2= 2.50, df=1, P=0.12). There were no other significant univariate predictors of non-fatal stroke.

Fifty-one patients (31.5%) had been asymptomatic at the time of their CEA in 1995. Nineteen (37.3%) of these asymptomatic patients reported experiencing one or more neurological sign(s) or symptom(s) since their 1995 CEA. Of 110 patients who reported being symptomatic at the time of their CEA, 37 (33.6%) stated they had experienced a return of symptoms (symptom status missing for one patient). Twenty-three (62.2%) of these 37 patients reported experiencing one or more neurological sign(s) or symptom(s) since their 1995 CEA. Twenty-three patients (14.2%) had had another CEA operation since their 1995 operation.

Health status

Data completion for the MOS SF-36 ranged from 92.6% to 100% for the eight domains. Table 3 provides estimates of mean and median values, ranges, standard deviations (SD), and 25th and 75th percentiles for each of the eight health domains.

With respect to the single-item rating of health transition, the majority of patients rated their health as 'about the same' when compared with one year ago (n=92, 56.7%). Twenty-five patients (15.4%) stated that their health was either 'somewhat worse' or 'much worse' than one year ago. There was no significant difference between males and females for this health transition rating (χ^2 = 8.96, df=4, P=0.06) or age (age ≤ 75 or age > 75) (χ^2 = 4.58, df=4, P=0.33).

In our sample, there were no significant differences in mean values for any of the eight health domains by sex. However, there were significant differences in mean values for two of the eight domains by age (age ≤ 75 or age > 75). Specifically, the older age group demonstrated lower mean scores for *Physical Functioning* (t=2.65, df=149, P=0.01) and *Role Physical* (t=2.10, df=142, P=0.04). Further, there were no significant differences in any of the eight domains for those in our sample who had had a non-fatal stroke since their CEA and those who had not (Table 4).

Mean scores by sex for the eight SF-36 health domains for our sample were compared with Australian population norms for males and females (Table 4). For males, mean scores for each age group appeared consistently lower than Australian population norms for three domains - *Physical Functioning, Vitality and Mental Health*.

For females, mean scores for each age group appeared consistently lower for five health domains: *Physical Functioning, Role Physical, General Health, Vitality and Mental Health* (Table 4).

Mean scores for all patients in our sample for each of the eight health domains appeared higher than Australian population norms for those aged 55 years and over who had experienced a stroke (Table 4). Sex-disaggregated data were not available, however, precluding further analysis.

Discussion

Vascular disease is the leading cause of death in Australia, accounting for 40% of all deaths in 1998 (Australian Institute of Health and Welfare 2001). US data shows that pre-operative comorbid conditions of acute myocardial infarction, congestive heart failure and diabetes mellitus pose the greatest threat to the long-term survival rates of patients following CEA (Estes et al. 1998). In this Australian study, 61.4% (n=27) of patients who died within four years of a CEA did so from a vascular cause, confirming that patients who undergo CEA remain at high risk of death from vascular disease.

Curiously, the only independent predictor of death from any cause in our sample was insurance status. War veterans were more likely to die within four years of their CEA than either privately insured patients or those with no health insurance. It is possible that war veterans had a greater number of comorbidities than other patients. Yet, from AN-DRG data, veterans were no more likely to have experienced a complication or comorbidity when admitted for CEA than were public or privately insured patients. While collection of more sensitive comorbidity data would have provided an opportunity for further analyses, this was beyond the scope of the resources allocated to the study.

Our determination of health status among survivors revealed further useful clinical information. Results showed no significant difference by sex for any health domain scores. However, NSW population norms have been shown to be gender-sensitive (Australian Bureau of Statistics 1995).

Our patients reported health status scores that appeared higher than Australian population norms for people aged over 55 years who had experienced a stroke. Publication of gender-specific norms would have enabled further analyses. Our findings are consistent with the premise that long-term survivors did benefit from surgical stroke prevention.

Methodologically, we were pleased to have obtained 100% surgeon agreement and 90% survey return from patients. Furthermore, we obtained a high percentage of data completeness per SF-36 domain, ranging from 92.6% - 100% (Ward, Lin, and Heron 1997). Our findings have high internal validity although generalisability remains unclear.

In conclusion, it has been suggested that Australia is falling behind the UK in terms of public reporting of clinical quality in surgical services (Eno and Spigelman 2000). There is a paucity of rigorous outcome evaluation in the area of vascular disease within Australia, despite the heavy burden these patients place on our health system. Our method represents a valid and practical way for clinical services to examine not only long-term clinical benefits of CEA but also missed opportunities for better health. The value of using patient functional status to aid health services planning has previously been reported (Snow, Walker, Ahearn, O'Brien and Saltman 1999).

It has become clear that patients having CEA are at high risk of dying from vascular events within four years of their surgery, ameliorating the benefits otherwise of surgery to address atherosclerotic narrowing of their carotid arteries. When discharged from hospital after their CEA, patients may benefit from interventions that aim to increase their awareness and adoption of coronary heart disease and stroke risk factor management strategies. Specifically, co-ordination of their case management after discharge could optimise GP preventive care and patient self-management. Continued audit in vascular surgery would benefit patients, clinicians and the health system.

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Table 1: Patient characteristics by hospital for first or only CEA in 1995 (n=238)

Variable	RPAH [n=125]		CRGH [n=113]		Significance
	n	%	n	%	
Age					
<60 (n=34)	18	14.4	16	14.2	
60-69 (n=85)	57	45.6	28	24.7	
70-79 (n=101)	43	34.4	58	51.3	
80+ (n=18)	7	5.6	11	9.7	χ^2 = 12.56, df=3, P=0.005
Age Group					
< 75 years (n=182)	103	82.4	79	69.9	
≥ 75 years (n=56)	22	17.6	34	30.0	χ^2 = 5.14, df=1, P=0.02
Sex					
Male (n=166)	91	72.8	75	66.4	
Female (n=72)	34	27.2	38	33.6	χ^2 = 1.16, df=1. P=0.28
Residence					
In CSAHS (n=75)	44	35.2	31	27.4	
Out of CSAHS (n=163)	81	64.8	82	72.6	χ^2 = 1.66, df=1, P=0.20
AN-DRG					
CEA patients who underwent simultaneous cardiothoracic surgery with or without CCs (n=28)	28	22.4	0	0	
CEAs without CC (n=153)	73	58.4	80	70.8	
CEA with CC including AN-DRG 3, [Tracheostomy]) (n=57)	24	19.2	33	29.2	χ^2 = 29.2, df=2, P<0.001
Insurance Type					
Public patients (n=125)	67	53.6	58	51.3	
Private patients (n=79)	58	46.4	21	18.5	
Veterans (n=34)	0	0	34	30.1	χ^2 = 51.5, df=2, P<0.001
LOS					
1-4 days (n=47)	25	20.0	22	19.5	
5-6 days (n=78)	28	22.4	50	44.2	
7-10 days (n=51)	30	24.0	21	18.6	
11 days or greater (n=62)	42	33.6	20	17.7	χ^2 = 15.2, df=3, P=0.001

Table 2: Causes of Death

Cause of Death	n	%
Vascular cause	27	61.4
AMI	10	
Chronic ischaemic heart disease	5	
Other cardiovascular disease	4	
Cerebrovascular disease	8	
Cancer	9	20.5
Renal failure	2	4.5
Injury	2	4.5
Other	4	9.1
Total	44	100

Table 3: Descriptive statistics for the MOS SF-36 (n=162)

	Physical Function	Role - Physical	Bodily Pain	General Health	Vitality	Social Functioning	Role - Emotional	Mental Health
Mean	56.6	55.2	67.2	58.2	54.5	75.0	70.6	72.7
Median	56.3	50.0	62.0	62.0	55.0	87.5	100.0	78.0
SD	27.5	42.0	28.9	23.4	21.7	29.1	41.3	20.0
25th percentile	38.9	0	41.0	40.0	40.0	50.0	33.3	56.0
75th percentile	80.8	100.0	100.0	77.0	70.0	100.0	100.0	88.0
Range	0-100	0-100	0-100	0-100	0-100	0-100	0-100	20-100

Table 4: Mean scores for the MOS SF-36 (n=162)

	Physical Function	Role - Physical	Bodily Pain	General Health	Vitality	Social Functioning	Role - Emotional	Mental Health
Central Sydney Data (n=162)								
Sex								
Male	57.0	52.9	68.0	57.5	54.1	73.4	69.4	73.3
Female	55.7	60.3	65.5	59.9	55.3	78.5	73.0	71.3
Males (n=112)								
≤ 55 yrs (n=2)	82.5	100.0	92.0	84.5	60.0	100.0	100.0	66.0
55 - 64 yrs (n=15)	64.5	83.3	75.1	58.0	52.1	85.0	82.2	75.1
65 - 74 yrs (n=37)	62.2	50.8	72.3	57.6	56.1	69.9	62.6	72.0
≥ 75 yrs (n=52)	52.0	42.2	64.2	57.7	52.6	72.6	68.4	75.5
Missing (n=6)								
Females (n=50)								
< 55 yrs (n=3)	66.7	66.7	77.0	65.7	55.0	75.0	66.7	69.3
55 - 64 yrs (n=7)	74.8	71.4	78.6	60.9	62.9	87.5	81.0	70.9
65 - 74 yrs (n=21)	60.4	59.2	62.5	59.2	53.3	78.0	68.3	67.2
≥ 75 yrs (n=18)	38.3	53.1	60.9	59.0	53.8	75.0	77.1	76.0
Missing (n=1)								
Stroke data (n=157) (persons 55 years and over)								
No stroke (n=147)	56.5	55.0	66.4	57.6	54.6	75.0	70.9	72.6
Stroke (n=10)	49.6	50.0	71.3	59.6	52.0	72.5	63.0	76.8
Australian Data (n=7673)								
Males (n=3681)								
45 - 54 yrs	84.6	82.7	77.6	70.8	67.0	87.2	85.3	76.8
55 - 64 yrs	77.0	74.2	71.4	65.5	64.1	83.5	81.2	76.8
65 - 74 yrs	67.5	58.9	68.8	61.2	61.8	81.7	76.9	78.4
≥ 75 yrs	55.7	50.7	65.9	59.2	56.7	75.6	67.6	77.6
Females (n=3992)								
45 - 54 yrs	81.8	81.0	74.8	72.9	64.5	85.7	84.0	75.5
55 - 64 yrs	75.2	72.9	70.9	68.1	63.0	84.6	80.6	75.0
65 - 74 yrs	65.2	65.8	69.0	64.1	60.0	82.2	75.9	75.3
≥ 75 yrs	51.4	56.7	63.5	63.9	58.0	77.4	75.1	76.8
Australian stroke data (n=4569) (age and sex standardised for persons 55 years and over, includes after-effects of stroke)								
No Stroke	67.9	66.1	69.2	64.7	61.6	82.0	77.5	76.7
Stroke	45.0	29.7	54.0	43.8	46.2	64.4	60.5	66.3