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Changes in stroke care at Auckland Hospital between 1996 and 2001

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Abstract

Aims In 1996, we performed a descriptive audit of stroke care in Auckland Hospital. Since then, a mobile stroke team has been established. We have repeated the 1996 audit to assess changes in stroke management.

Methods From 1 June to 30 September 2001, information was prospectively recorded for all patients with stroke.

Results There were 177 strokes in 175 patients (92 men, mean age 70.9, standard deviation [SD] 14.9 years). Ninety-seven percent of patients had cerebral imaging (median 4.5 hours; interquartile range [IQR] 2.7–11.6). Acute aspirin was given to 78% of patients in 2001 and 40% in 1996 ($p < 0.001$). Twenty-four percent of patients were kept 'nil by mouth' for at least 24 hours (46% in 1996, $p < 0.001$). At discharge, 73% of patients were taking antiplatelet or anticoagulant therapy (61% in 1996, $p < 0.001$). Only 50% of the patients with elevated discharge blood pressures were taking antihypertensives. There had been a reduction in the mean length of hospital stay to 16 days (21 days in 1996) but no significant change in mortality (14% compared with 17% in 1996).

Conclusion A stroke service may increase the attention to the 'processes' of stroke care and use of therapies, which are shown to be of benefit in randomised controlled trials.

In 1996, we published an audit of stroke care in Auckland Hospital.[1] At that time there was no organised inpatient stroke care in our hospital. However, there has since been increasing evidence that organised stroke care results in improved outcome compared with conventional care.[2] In 2000, a mobile stroke team was established at Auckland Hospital. The stroke team includes neurologists, geriatricians, and general physicians; a stroke nurse coordinator; and members from each of the allied health disciplines.

The majority of stroke patients are admitted to general medical wards where they are managed by a general physician. A stroke team physician reviews the stroke patients and makes recommendations about acute management and investigation, and secondary stroke prevention. The specialist stroke nurse and allied health staff have regular rounds and coordinate the multidisciplinary care of patients. Early assessment and referral for inpatient rehabilitation (as well as early discharge planning) are encouraged. There is a weekly multidisciplinary meeting where all aspects of patient care (and the service as a whole) are reviewed. The stroke nurse provides patient/family and staff education. Some of the more complex patients are referred to the Stroke Clinic for follow-up.

There have been a number of other developments in stroke management since 1996. Recombinant tissue plasminogen activator (rt-PA) within 3 hours,[3,4] and aspirin 150–300 mg within 48 hours, have been shown to reduce death and dependency following ischaemic stroke.[5] Furthermore, combined angiotensin converting enzyme (ACE) inhibitor and diuretic therapy,[6] and the 3-hydroxy-3 methylgluturyl coenzyme A reductase inhibitors, or 'statins',[7,8] have been shown to be beneficial in secondary stroke prevention.

We have repeated the 1996 stroke audit to assess the changes in stroke care at Auckland Hospital over a 5-year period. The aim was to determine the uptake of recent developments in stroke care and the impact of the stroke service on patient management.

Methods

Auckland Hospital has the Auckland isthmus as its catchment area. It also provides the neurology service for Auckland and Northland and, as such, accepts complex and often younger patients from these regions. From 1 June to 30 September 2001, all patients over the age of 15 years presenting to Auckland Hospital with symptoms and signs consistent with an acute stroke were identified by daily checks of emergency department (ED), acute admitting ward, intensive care unit (ICU), geriatric, and vascular surgery databases, as well as attendance at the medical handover meetings. A specialist stroke nurse with one of the stroke team physicians prospectively recorded information from patients, their family, or hospital notes.

The timing of the audit (June to September) and the method of identification of patients were the same as those used in 1996. The form used to record data was the same as that used in 1996 with the addition of a more comprehensive checklist of admission medications, location of inpatient rehabilitation, and differentiation between current and past cigarette smoking.

We used the World Health Organisation definition of stroke,[9] but did not include patients with subarachnoid haemorrhage, subdural or extradural haematoma, haemorrhage into a cerebral tumour, or post-traumatic intracerebral haemorrhage (ICH). We also excluded patients with a transient ischaemic attack (TIA), or patients transferred from other hospitals for tertiary neurological or neurosurgical care. Strokes were classified into three subtypes; ischaemic stroke, ICH, and unspecified stroke type in those patients who did not have cerebral imaging or an autopsy.

All results are presented as the medians and interquartile range (IQR) unless stated. Comparisons of categorical variables between the two audits were made using the chi-square ($?^2$) test. Other comparisons were made using the Wilcoxon rank-sum test. Results were considered significant at the 5% level.

Results

There were 177 stroke events in 175 patients (92 men, mean age 70.9 ± standard deviation 14.9 years) during the 122 days of the audit. Two patients had two admissions for stroke during the period of the study. Four patients had strokes while in hospital. Patients were admitted under the general medicine (83%), neurology (6%), geriatric (3%), neurosurgical (2%) or vascular surgical (1%) service. The stroke or neurology services reviewed 155 of 177 (88%) patients.

There has been an increase in the proportion of patients taking aspirin or warfarin at the time of admission with stroke—from 34% in 1996 to 61% in 2001 (Table 1). There has also been a increase in the proportion of patients with atrial fibrillation taking warfarin, from 12% in 1996 to 32% in 2001 ($?^2 = 4.203$, $p = 0.040$).

Table 1. Comparison of stroke patient characteristics and management: 2001 and 1996

	2001 n/total (%)	1996 n/total (%)	c^2 / p =
Stroke type			
Ischaemic	146/177 (82)	135/184 (73)	0.006
Intracerebral haemorrhage	25/177 (14)	26/184 (14)	
Unspecified	6/177 (3)	23/184 (13)	
Admission medications			
Aspirin	98/177 (55)	49/167 (29)	<0.001
Warfarin	11/177 (6)	8/167 (5)	
Neither	68/177 (38)	110/167 (66)	
Onset to arrival			
Within 3 hours	35/177 (20)	57/184 (31)	<0.001
Within 6 hours	50/177 (28)	70/184 (38)	
Neither	92/177 (52)	57/184 (31)	
CT scan			
	167/177 (94)	161/184 (88)	0.024
Nutrition			
Patients 'nil by mouth'	42/177 (24)	85/184 (46)	<0.001
NG feeding	21/177 (12)	23/184 (13)	
PEG insertion	5/177 (3)	6/184 (3)	
Neither	109/177 (61)	70/177 (38)	
Acute medications			
Aspirin	107/138 (78)	54/135 (40)	<0.001
Discharge medications			
Aspirin	44/177 (25)	81/184 (44)	<0.001
Warfarin	27/177 (15)	29/184 (16)	
Aspirin & Warfarin	12/177 (7)	0/184 (0)	
Aspirin & Dipyridamole	46/177 (26)	2/184 (1)	
Neither	48/177 (27)	72/184 (39)	
Mortality			
Ischaemic stroke	14/177 (8)	13/184 (7)	0.9488
Intracerebral haemorrhage	10/177 (6)	10/184 (5)	
Neither	153/177 (86)	161/184 (88)	

NG = nasogastric; PEG = percuataneous endoscopic gastrostomy; CT = computerised tomography

Time to arrival and medical assessment Compared with 1996, there has been a reduction in the number of patients arriving in hospital within the first 3 to 6 hours of symptom onset (Table 1). The time of symptom onset was available for 93 of 177 (53%) stroke events. In the remaining 84 events, a patient had woken with symptoms or was unable to give a time of symptom onset and there was no reliable witness. Of all 177 patients, only 20% had a known time of onset and had arrived at hospital within 3 hours.

General practitioners (GPs) assessed 63 (36%) patients prior to hospital arrival. Patients assessed by a GP arrived at hospital later (median 18.5 hours from symptom onset, IQR 6.2–24) than those not assessed by a GP (3.9 hours, IQR 1.1–11.1; Wilcoxon rank-sum test, p <0.0001).

Emergency department (ED) physicians assessed 79 (45%) patients (median time from arrival to assessment 50 minutes, IQR 20–75). The remaining patients were first assessed by the general medicine, neurology, neurosurgical or geriatric services (2.8

hours, IQR 1.4–4.3). The median time from arrival in hospital to first medical assessment was 68 minutes (IQR 35– 115).

Investigation Almost all patients (97%) had some form of cerebral imaging (Table 1). Computerised tomography (CT) scans were performed in 167 (94%) patients compared with 88% in 1996 ($?^2 = 5.097$, $p = 0.024$). Magnetic resonance imaging (MRI) was obtained in 25 (14%) patients; of whom 21 had also had CT scans. The median time from arrival of the patient in hospital to cerebral imaging was 4.5 hours (IQR 2.7–11.6); down from a median of 10 hours in 1996.

Cerebral angiography was performed in three patients. Thirty-two of 146 (22%) ischaemic stroke patients had a carotid duplex ultrasound (US) study (median time to US study 31 days, IQR 6–42). One patient had a carotid duplex US study more than 6 months after their stroke. A further six patients were still awaiting carotid studies more than 6 months after their stroke. Transthoracic echocardiography (TTE) was performed in 56 (34%) patients, transoesophageal echocardiography (TOE) in nine (5%) patients, and Holter monitoring in seven (4%) patients.

Acute management Aspirin was given (within 48 hours) to 107 of 138 (78%) ischaemic stroke patients (where this information was available), compared to 40% of patients in 1996 ($?^2 = 39.741$, $p < 0.001$). Two patients were treated with rt-PA. Only 5 of 146 (3%) ischaemic stroke patients had a known time of symptom onset and cerebral imaging within 3 hours, and could therefore be considered for rt-PA.

There has been a reduction in the number of patients kept ‘nil by mouth’ for more than 24 hours—from 46% in 1996 to 24% in 2001. Of the 31 ischaemic stroke patients who did not receive early aspirin, 15 had been kept ‘nil by mouth’ for at least 24 hours.

Twenty of 146 (14%) ischaemic stroke patients were treated with heparin, either intravenously (12) or subcutaneously (8). The indication for heparin (where it could be determined) was arterial dissection in four patients, posterior circulation stroke in two patients, and myocardial infarction in one patient. Heparin is not routinely used for deep vein thrombosis (DVT) prophylaxis at our institution—where aspirin (ischaemic stroke patients only), full-length compression stockings, and early mobilisation are recommended.

Complications Thirteen of 177 (7%) patients developed pneumonia during their admission (13% in 1996). Seven of 177 (4%) patients had neurological deterioration (4% in 1996), one patient (0.6%) had a gastrointestinal bleed (5% in 1996), myocardial infarction (2% in 1996), or DVT.

Inpatient rehabilitation Inpatient rehabilitation was carried out in 66 of 177 (37%) patients compared with 54 of 184 patients (29%) in 1996 ($?^2 = 2.56$, $p = 0.109$). Thirty-six of 115 (31%) patients aged 65 years or older were transferred for inpatient rehabilitation at a median of 9 days (IQR 5–13) from admission. Rehabilitation for patients less than 65 years is carried out at a separate inpatient facility. Fourteen of 62 (23%) patients less than 65 years-of-age were transferred for ‘younger-patient’ rehabilitation at a median of 20 days (IQR 13–28) from admission. Sixteen of 177 (9%) patients received rehabilitation in other hospitals.

Secondary stroke prevention Compared with 1996, there has been an increase in the number of patients discharged on either anti-platelet (aspirin with or without

dipyridamole) or anti-coagulant (warfarin with or without aspirin) therapy (Table 1). Almost all surviving ischaemic stroke patients were discharged on antiplatelet or anticoagulant therapy. Only six of 30 (20%) ischaemic stroke patients with atrial fibrillation on admission ECG were prescribed warfarin. The reasons for the low use of warfarin as secondary stroke prevention in patients with atrial fibrillation were not specifically identified.

The median admission systolic blood pressure (BP) was 160 mmHg (IQR 145–188) and the diastolic BP was 90 mmHg (IQR 80–100). At discharge, the systolic BP was 138 mmHg (IQR 122–150) and the diastolic BP was 76 mmHg (IQR 70–85). Sixty-six of 144 (46%) surviving stroke patients were taking antihypertensive therapy at discharge. Eleven of 20 patients with discharge systolic BPs equal to or greater than 165 mmHg, and five of 10 patients with discharge diastolic BPs equal or greater than 95 mmHg had not been prescribed antihypertensive therapy.

At admission, 46 of 177 patients (26%) gave a history of hyperlipidaemia, and 23 of 177 (13%) patients were using lipid-lowering medication. At discharge, lipid-lowering medication had been prescribed to 30 of 152 (20%) surviving patients.

Stroke outcome and length of stay There has been a 5-day reduction in the mean length of stay at Auckland Hospital to 16.3 days (SD 17.9, median 9 days; IQR 4–22) since 1996. There has been no significant change in hospital mortality between 1996 (17% in hospital mortality) and 2001 (14%).

Discussion

This study provides a description of stroke care in a New Zealand hospital. Previous studies of stroke in New Zealand hospitals have been reported.[10–14] However, this audit has enabled an examination of changes in stroke management at the same institution over a 5-year period.

Almost all stroke patients now have cerebral imaging. In 1991/1992, 40% of stroke patients had cerebral imaging,[15,16] increasing to 88% in 1996 and 97% in 2001. This is the likely reason for the reduction in the proportion of strokes classified as being of 'unspecified' type, and reflects recognition of the need for imaging to exclude stroke mimics and to differentiate between cerebral infarction and ICH. Three quarters of stroke patients now have brain imaging within 12 hours.

It is concerning that there has been a reduction (from 31% in 1996 to 20% in 2001) in the number of patients reaching hospital within three hours of stroke onset. The cause of this increased delay to hospital arrival (between 1996 and 2001 audits) has not been assessed, and any discussion as to the reason is speculative. Specifically, general practitioners and the community may perceive greater pressures on hospital beds—leading to a delay in seeking admission, greater stresses on the Ambulance Service, and worsening Auckland traffic. There continues to be a delay between hospital arrival, medical assessment, and brain imaging. These findings may account for the low use of rt-PA. It is not clear why three of five potentially eligible patients were not treated.

Compared with 1996, almost twice as many patients are now treated with aspirin within 48 hours of symptom onset. This probably reflects greater awareness of the benefits of early aspirin therapy and earlier imaging to exclude cerebral haemorrhage. Aspirin 160–300 mg daily (started within 48 hours) reduces the risk of death or

dependency from 47.0% to 45.8%.[5] Therefore, assuming 550 stroke patients are admitted to Auckland Hospital per year, the increased use of aspirin acutely has resulted in two to three fewer dead or disabled patients in 2001 compared with 1996.

Fewer patients were kept 'nil by mouth' compared to 1996. This is due to the encouragement of the use of dysphagia assessments and simple bedside swallow tests by the Stroke Service. The reduction of patients kept nil by mouth may partly explain the increased use of aspirin acutely. The number of patients managed with enteral feeding has remained constant since 1996.

There has been an increase in the use of antiplatelet and anticoagulant therapy for secondary stroke prevention. There has also been a dramatic increase in the use of dipyridamole in conjunction with aspirin. These changes reflect greater awareness of the benefits of antiplatelet and anticoagulant therapy in secondary stroke prevention. Other antiplatelet medications (such as Clopidogrel) are not subsidised by the New Zealand government so no patients were prescribed these medications during the time of the audit.

Just over half of the patients in the present study were discharged on no antihypertensive therapy. The Perindopril Protection Against Recurrent Stroke Study (PROGRESS) trial found that (in patients with stroke) combined angiotensin converting enzyme inhibitor and diuretic therapy reduced the risk of recurrent stroke or major vascular event.[6] The benefits of combined therapy were even seen in patients who were 'normotensive' at baseline. The PROGRESS results were published towards the end of recruitment. It is of concern that many patients are discharged with blood pressure measurements still in the hypertensive range.

One in five patients were taking lipid-lowering therapy at discharge. In contrast, almost no patients were taking lipid-lowering therapy in 1996. This increase reflects the dissemination of the results of studies demonstrating the benefit of statins in patients with ischaemic heart disease (IHD).[7] Since this audit was completed, the MRC/BHF heart protection study has shown that simvastatin given to patients with cerebrovascular disease, with or without a history of IHD, significantly reduces the risk of a major vascular event.[8] Furthermore, this reduction in risk did not appear to be influenced by pre-treatment cholesterol or triglyceride concentrations. It is likely that the use of such therapy will increase dramatically in response to this trial and the recent relaxation of the PHARMAC restrictions on the use of statins following stroke.

There has been a 5-day reduction in the length of stay at Auckland Hospital between 1996 and 2001. It is tempting to suggest that the reduced length of stay is due to improved stroke outcome. However, there is greater pressure on hospital beds in general, and there has been no reduction in mortality. There may also have been an apparent reduction in stay as a result of the more accurate classification of patients with mild symptoms as stroke and not TIA.

There has been an improvement in stroke care since 1996. We believe this is probably due (in part) to the direction provided by the Stroke Service, but we acknowledge that this study does not specifically examine this question. In 1996, a neurologist saw only 13% of patients. In 2001, a Stroke Service physician or neurologist had seen 88% of patients. The Stroke Service has formulated guidelines for the investigation and management of stroke patients and has ensured the more rapid and even implementation of stroke therapies across the hospital. Coordinated multidisciplinary

care and the encouragement of early discharge planning and referral for inpatient rehabilitation may have led to shorter lengths of stay. The Stroke Service, in conjunction with the Radiology Department, has encouraged the more appropriate use of magnetic resonance imaging and carotid ultrasound scanning. Furthermore, there is now formal staff and patient/family stroke education.

However, in response to recent studies, many of the changes seen may have occurred anyway. We did not audit changes in allied health practices. We did not specifically determine what proportion of subjects with stroke were not identified by the Stroke Service. We accept some patients may not have been identified and included in the audit, but believe these numbers were small and of a similar proportion to those in 1996. While the Stroke Service provides support to the general medical teams, most patients are seen by a Stroke Service physician only once. This may explain why some patients with blood pressure measurements in the hypertensive range are discharged on no antihypertensive medication and why some ischaemic stroke patients in atrial fibrillation are not prescribed warfarin.

Patients in geographical stroke units have substantial improvements in management processes, fewer complications, and improved outcome (compared to patients managed on general wards with stroke team support).[17,18] In the latter half of 2002, a small acute stroke unit (with shared care between the general medical and neurology services) was opened at Auckland Hospital. This unit has close links with the geriatric and rehabilitation services, and received 32% of all stroke admissions in the first half of 2003. It is hoped that this is the forerunner of a larger, comprehensive stroke unit.

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References:

1. Charleston AJ, Barber PA, Bennett P, et al. Management of acute stroke in Auckland Hospital in 1996. NZ Med J. 1999;112:71–4.
2. Stroke Unit Trialists Collaboration. Organised inpatient (stroke unit) care for stroke (Cochrane Review). Oxford: The Cochrane Library; 2001.
3. Tissue plasminogen activator for acute ischemic stroke. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. N Engl J Med. 1995;333:1581–7.
4. Kwiatkowski TG, Libman RB, Frankel M, et al. Effects of tissue plasminogen activator for acute is chemic stroke at one year. National Institute of Neurological Disorders and Stroke Recombinant Tissue Plasminogen Activator Stroke Study Group. N Engl J Med. 1999;340:1781–7.
5. Pathansali R, Bath PM. IST and CAST: some answers but more questions. J Hum Hypertens. 1998;12:73-4.

6. PROGRESS Collaborative Group. Randomised trial of a perindopril-based blood-pressure-lowering regimen among 6,105 individuals with previous stroke or transient ischaemic attack. Lancet. 2001;358:1033–41.
7. White HD, Simes RJ, Anderson NE, et al. Pravastatin therapy and the risk of stroke. N Engl J Med. 2000;343:317–26.
8. Heart Protection Study Collaborative Group. MRC/BHF Heart Protection Study of cholesterol lowering with simvastatin in 20,536 high-risk individuals: a randomised placebo-controlled trial. Lancet. 2002;360:7–22.
9. Hatano S. Experience from a multicentre stroke register: a preliminary report. Bulletin of the World Health Organisation. 1976;54:451–553.
10. McNaughton H. Stroke audit in a New Zealand hospital. NZ Med J. 1996;109:257–60.
11. Friedman PJ. Ischaemic stroke and intracerebral haematoma in the Waikato Stroke Registry. NZ Med J. 1992;105:171–3.
12. Hanger HC. Implementing a stroke rehabilitation area: the first six months. NZ Med J. 2002;115(1158). URL: http://www.nzma.org.nz/journal/115-1158/110/
13. Barber PA, Anderson NE, Bennett P, Gommans J. Acute stroke services in New Zealand. NZ Med J. 2002;115:3–6.
14. Gommans J, Barber PA, McNaughton H, et al. Stroke rehabilitation services in New Zealand. NZ Med J. 2003;116(1174). URL: http://www.nzma.org.nz/journal/116-1174/435/
15. Anderson NE, Broad JB, Bonita R. Delays in hospital admission and investigation in acute stroke. BMJ. 1995;311:162.
16. Anderson NE, Bonita R, Broad JB. Early management and outcome of acute stroke in Auckland. Aust N Z J Med. 1997;27:561–7.
17. Kalra L, Evans A, Perez I, et al. Alternative strategies for stroke care: a prospective randomised controlled trial. Lancet. 2000;356:894–9.
18. Evans A, Perez I, Harraf F, et al. Can differences in management processes explain different outcomes between stroke unit and stroke-team care? Lancet. 2001;358:1586–92.