Determinants of Handicap After Stroke
The North East Melbourne Stroke Incidence Study (NEMESIS)

Jonathan W. Sturm, PhD; Geoffrey A. Donnan, MD; Helen M. Dewey, PhD; Richard A.L. Macdonell, MD; Amanda K. Gilligan, MBBS; Amanda G. Thrift, PhD

Background and Purpose—Handicap, although more relevant to the patient than impairment or disability, has received little attention in people with stroke. The aim of this study was to identify, in an unselected population, factors determining handicap at 2 years after stroke.

Methods—All first-ever cases of stroke in a population of 306 631 over a 1-year period were assessed. Stroke severity, comorbidity, and demographic information was recorded. Among survivors, 2-year poststroke handicap was assessed with the London Handicap Scale. Disability, physical impairment, depression, anxiety, living arrangements, and recurrent stroke at 2 years were documented. If necessary, proxy assessments were obtained, except for mood. Linear regression analyses were performed to identify factors independently associated with handicap. First, all assessments (proxy and nonproxy) were examined; then, the nonproxy assessments were used to examine the effects of mood.

Results—Of 266 patients with incident stroke who were alive at 2 years, 226 (85%) were assessed. Significant determinants of handicap on univariable analysis were age, female sex, socioeconomic status, alcohol intake, stroke subtype, initial stroke severity; 2-year physical impairment, disability, depression and anxiety scores; institutionalization; and recurrent stroke. On multivariable analysis, the independent determinants of handicap were age and 2-year physical impairment and disability. In analysis restricted to nonproxy data, depression and anxiety were also independently associated with handicap.

Conclusions—Age, concurrent disability, and physical impairment were more important determinants of handicap than other demographic factors or initial stroke severity. Because depression and anxiety were independently associated with handicap, their treatment may potentially reduce handicap in stroke patients. (Stroke. 2004;35:715-720.)

Key Words: cerebrovascular disorders ■ health status ■ outcome assessment ■ quality of life

Handicap is the disadvantage resulting from ill health that limits fulfillment of societal roles; disability is the lack of ability to perform tasks.1 Handicap is common after stroke even in nondisabled patients,2 and reduction of handicap is a key aim of rehabilitation. The determinants of poststroke handicap have been examined in few studies,3–6 and data are lacking from unselected populations. Better understanding of poststroke handicap may result in improved stroke care. The study aims were, in an unselected population at 2 years after first-ever stroke, to determine patterns of handicap and factors that explain handicap.

Methods

Case Ascertainment
This was a substudy within the community-based North East Melbourne Stroke Incidence Study (NEMESIS).7 NEMESIS was conducted in northeast Melbourne, Australia, between May 1, 1996, and April 30, 1999. In the first year (the NEMESIS pilot study), residents were surveyed in an 8-postcode region containing 133 816 people. NEMESIS was expanded on May 1, 1997, to a 22-postcode region containing 306 631 people. Case-finding methodology met criteria for “ideal” stroke incidence studies.8 Cases were recruited prospectively from multiple overlapping sources, ensuring that nonhospitalized cases were obtained. All potential cases were reviewed by an expert stroke panel before inclusion. In the present study, all cases of first-ever stroke (excluding subarachnoid hemorrhage) that occurred between May 1, 1998, and April 30, 1999, were eligible (ie, the second year of accrual for the expanded NEMESIS study). Ethics committees at each participating institution approved the study, and informed consent was obtained from all participants.

Baseline Data
Cases were interviewed and examined by a trained research nurse as soon after stroke as possible. Additional clinical details were obtained from medical records and, if required, from treating doctors. Pathological stroke subtype (ischemic stroke [IS], intracerebral hemorrhage [ICH]) was determined through neuroimaging or autopsy findings.9 Patients without neuroimaging or autopsy were classified as undetermined. IS cases were further categorized by use
Handicap at 2 years after first-ever stroke: proportion of patients in each category of disadvantage (n=226). Level 1 denotes no disadvantage; level 6, extreme disadvantage.

of the Oxfordshire Community Stroke Project (OCSP) classification. Stroke severity, demographic, and comorbidity information was obtained from the patient, medical or nursing home records, and treating doctors when necessary. Demographic data included age, sex, country of birth, preferred language, and whether the patient lived alone or was institutionalized (nursing home, hostel, or supported accommodation). Occupations of the patient and spouse were used to classify socioeconomic status according to Australian Bureau of Statistics guidelines.11

Stroke severity markers recorded were dysphasia, loss of consciousness, neglect, and dense hemiparesis (loss of power against gravity in at least 1 limb) as determined from medical records and the National Institutes of Health Stroke Scale (NIHSS) examination. An acute NIHSS score was recorded prospectively when the patient was seen within 7 days; otherwise, it was recorded retrospectively from medical records.12 Comorbidities recorded were the presence of dementia, prestroke disability, and stroke risk factors (hypertension, diabetes, smoking, atrial fibrillation, peripheral vascular disease, prior transient ischemic attack, and prior myocardial infarction). Dementia, hypertension, peripheral vascular disease, prior transient ischemic attack, and prior myocardial infarction were defined as a known history. Smoking status was classified as current smoker, ex-smoker, or never smoked. Prestroke disability was defined as a prestroke Barthel Index score of <20/20. Diabetes was defined as either a known history or current presentation with fasting blood glucose ≥7.0 mmol/L. Atrial fibrillation was defined as either a known history or current presentation confirmed on ECG.

Follow-Up
Participants were assessed in a standardized face-to-face interview at 2 years after their index stroke. If severe cognitive impairment or dysphasia was present, proxy interviews from reliable informants were used except for mood assessments because such assessments may be biased. Interpreters were provided when needed.

A questionnaire was used to identify any stroke-like events in the previous 2 years. If there was a possible further event, information was obtained from medical or nursing home records and, if needed, from treating doctors. An expert panel undertook verification of recurrent events and subtype classification.

Instruments
Handicap was assessed with the London Handicap Scale (LHS).13 This scale employs the handicap domains described in the World Health Organization classification of impairments, disabilities, and handicaps.1 The LHS provides a profile of disadvantages experienced in the domains of mobility, physical independence, occupation, orientation, social functioning, and economic self-sufficiency. A weighted total handicap score is produced, with scores ranging from 0 (maximum disadvantage) to 100 (no disadvantage). Scale weights were derived from interviews with a general population sample using conjoint analysis.14 A retrospective version of the LHS was used to measure prestroke handicap.

Physical impairment was assessed with the NIHSS.15 The score range is 0 to 42, with higher scores reflecting greater neurological impairment. Mood impairment was assessed with the Irritability, Depression, Anxiety scale,16 an 18-item self-assessment measure of depression, anxiety, and “inward” and “outward” irritability. Five items each are used to assess depression and anxiety. Eight items are used to assess irritability. The 4 subscales were scored using 3 ranges: normal, borderline, and morbid, as recommended.17 Disability was assessed with the Barthel Index.18 Also recorded was the presence of disability that the patient stated was not due to stroke.

Statistical Analysis
Student’s t test, 1-way analysis of variance (ANOVA), or linear trend contrast was used to determine the significance of differences in 2-year handicap scores between patients categorized according to demographic, stroke severity, comorbidity, and stroke subtype variables; 2-year impairment and disability scores; and presence of recurrent stroke.

Univariable linear regression analyses were performed to identify factors present concurrently (ie, at the time handicap was assessed) associated with handicap. Multivariable linear regression analyses were performed, including those factors significant on univariable analysis, to identify factors independently associated with handicap. First, all assessments (proxy and nonproxy) were examined, and then nonproxy assessments alone were used to examine the effects of mood. Once the most parsimonious model was obtained by backward stepwise regression, each excluded variable was entered again separately to test its contribution to the final model. NIHSS, Barthel, Irritability, Depression, Anxiety, and LHS scores, as well as age, were all entered as continuous variables.

Results
Of 516 patients with first-ever stroke, 232 were deceased at 2 years after stroke (45%). Of the 284 survivors, 18 had subarachnoid hemorrhage and were ineligible for this study. Of the 266 patients alive and eligible, 4 were notified to the study >2 years after stroke, 37 refused participation, and 226 (85%) were assessed. The mean time to interview was 737 days (median, 731; range, 646 to 898). There were no
significant differences between those assessed and those eligible but not assessed in terms of age (P = 0.7, Student’s t test) or sex, country of birth, stroke subtype, and stroke severity markers (all P ≥ 0.9, χ²).

Patterns of Handicap
There were substantial proportions of cases disadvantaged in each domain examined. Physical independence, mobility, and occupation (including hobbies) were most severely affected (the Figure). Patients were significantly more handicapped at 2 years after stroke than before stroke (mean score, 73 versus 94; P < 0.001, Student’s t test).

Univariable Relationships Between Handicap and Demographics, Stroke Severity Variables, Comorbidities, and 2-Year Outcomes
Women were more handicapped than men (Table 1). Handicap consistently increased with increasing age. Patients institutionalized at stroke onset were more handicapped, as were those in the lowest socioeconomic category (unskilled workers). Handicap did not differ significantly according to country of birth or preferred language.

There was no significant difference in handicap between patients with IS and ICH; however, patients of undetermined subtype were significantly more handicapped than those with known subtype (Table 2). Patients with total anterior circulation infarct (TACI) were more handicapped than those with other IS subtypes. All stroke severity markers were significantly associated with greater handicap.

Patients with known dementia and with pre-existing disability were more handicapped than those without these problems (Table 3). Patients who had never smoked were most handicapped, ex-smokers were the least handicapped, and current smokers had intermediate handicap, although differences were of borderline statistical significance. There were no differences in handicap according to the presence of other stroke risk factors.

Handicap was significantly greater in patients who had (as measured at 2 years after stroke) greater physical impairment and disability and the presence of depression, anxiety, and inward irritability (Table 4). Significantly greater handicap was found in patients with disability unrelated to stroke, those with recurrent stroke, and those institutionalized at 2 years after stroke.

Independent Concurrent Determinants of Handicap
The factors present at the time of the assessment of handicap (ie, demographics, comorbidities, and 2-year outcomes) that were independently associated with handicap on multivariable regression were disability at 2 years, physical impairment at 2 years, and age (adjusted R² = 0.61, n = 205). When
analysis was restricted to nonproxy respondents (enabling assessment of the effects of mood), the independent determinants of handicap were these 3 factors plus anxiety and depression (adjusted $R^2=0.47$, $n=163$; Table 5). Including stroke severity markers in the models did not improve the explanatory power.

**Discussion**

Our first aim was to determine patterns of handicap after stroke. At 2 years after stroke, the mean total handicap score was 73, almost identical to that found in the NEMESIS pilot study in a different cohort of first-ever patients at 12 months after stroke.2 A typical patient with a score of 73 would be able to get to most (but not all) places that they wanted to, would require help with tasks such as housework and shopping, would be limited in work and leisure activities (gardening, hobbies), and would have a slightly limited social life. Although the domains of physical independence, mobility, and occupation were most severely affected, stroke survivors were handicapped over a wide range of domains. These results are similar to those of studies with shorter follow-up,2,13 providing some evidence that the domains of handicap affected are stable over time.

The second aim was to identify factors present at 2 years after stroke that explain handicap at that time. The independent determinants of handicap (ie, statistically significant on multivariable analysis) were age and 2-year disability, physical impairment, anxiety, and depression (Table 5). Concurrent physical impairment and disability have previously been identified as independent determinants of handicap3–6; however, this is the first evidence for this in an unselected population. Age3,5,6 and depression4,5 have also been identified as independent determinants of handicap. In the present study, sex (Table 1) was not independently associated with handicap; however, others have demonstrated greater handicap in men6 and women,5 whereas an interaction between sex and marital status has also been reported.4 Also, in contrast to a previous investigation, we did not demonstrate an independent effect of living arrangements (Table 4).4 Stroke recurrence (Table 4) would also influence handicap. Although stroke recurrence associated with handicap on univariate analysis, it was not an independent determinant. Presumably, the effects of recurrent stroke are encompassed in the assessment of impairment and disability.

Unlike others, we found that cognition was not an independent determinant of handicap.4,6 However, our assessment of cognition was not ideal because no formal cognitive assessments were undertaken. Instead, impaired cognition was measured by the proxy categories of "known history of dementia" and "disability unrelated to stroke" (Table 2). Patients in NEMESIS had usual care. Although many would have received rehabilitation, these data were not systematically collected, and the influence of rehabilitation on handicap was not assessed. Interestingly, Clarke and colleagues4 found that rehabilitation was associated with a small reduction in handicap at 3 months after stroke but no change at 12 months.

No significant differences in handicap were found between IS and ICH, consistent with the findings at 3 and 12 months.

---

**TABLE 2. Handicap at 2 Years After First-Ever Stroke According to Markers of Initial Stroke Severity**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Classification</th>
<th>n</th>
<th>Mean LHS (95% CI)</th>
<th>$P^*$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathological subtype</td>
<td>IS</td>
<td>193</td>
<td>74 (71–77)</td>
<td>0.003</td>
</tr>
<tr>
<td></td>
<td>ICH</td>
<td>27</td>
<td>73 (64–82)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Undetermined</td>
<td>6</td>
<td>42 (7–77)</td>
<td></td>
</tr>
<tr>
<td>Ischaemic stroke subtype</td>
<td>TACI</td>
<td>19</td>
<td>56 (44–68)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>PACI</td>
<td>78</td>
<td>73 (68–78)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>POCI</td>
<td>45</td>
<td>78 (72–84)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LACI</td>
<td>51</td>
<td>79 (73–85)</td>
<td></td>
</tr>
<tr>
<td>Dense hemiplegia</td>
<td>Yes</td>
<td>62</td>
<td>61 (54–66)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>164</td>
<td>78 (74–81)</td>
<td></td>
</tr>
<tr>
<td>Dyshasia</td>
<td>Yes</td>
<td>52</td>
<td>67 (60–73)</td>
<td>0.024</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>174</td>
<td>75 (71–78)</td>
<td></td>
</tr>
<tr>
<td>Loss of consciousness</td>
<td>Yes</td>
<td>25</td>
<td>62 (52–72)</td>
<td>0.011</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>201</td>
<td>74 (70–76)</td>
<td></td>
</tr>
<tr>
<td>Neglect</td>
<td>Yes</td>
<td>41</td>
<td>64 (57–71)</td>
<td>0.002</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>178</td>
<td>76 (73–79)</td>
<td></td>
</tr>
<tr>
<td>NIHSS</td>
<td>0–5</td>
<td>147</td>
<td>81 (77–84)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>6–10</td>
<td>48</td>
<td>63 (56–69)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>11–15</td>
<td>15</td>
<td>56 (43–68)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&gt;16</td>
<td>9</td>
<td>45 (28–61)</td>
<td></td>
</tr>
</tbody>
</table>

PACI indicates partial anterior circulation infarct; POCI, posterior circulation infarct; and LACI, lacunar infarct.

*Student’s $t$ test was used except for subtypes and NIHSS (1-way ANOVA).
TABLE 3. Handicap at 2 Years After First-Ever Stroke According to Comorbidities and Risk Factors Defined at Onset

<table>
<thead>
<tr>
<th>Variable</th>
<th>Classification</th>
<th>n</th>
<th>Mean LHS (95% CI)</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prestroke disability</td>
<td>Yes</td>
<td>25</td>
<td>60 (51–70)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>176</td>
<td>77 (74–80)</td>
<td></td>
</tr>
<tr>
<td>Dementia†</td>
<td>Yes</td>
<td>11</td>
<td>39 (23–55)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>214</td>
<td>75 (72–77)</td>
<td></td>
</tr>
<tr>
<td>Diabetes‡</td>
<td>Yes</td>
<td>43</td>
<td>68 (62–74)</td>
<td>0.138</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>182</td>
<td>74 (70–77)</td>
<td></td>
</tr>
<tr>
<td>Atrial fibrillation‡</td>
<td>Yes</td>
<td>53</td>
<td>72 (65–78)</td>
<td>0.618</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>173</td>
<td>73 (70–77)</td>
<td></td>
</tr>
<tr>
<td>PVD†</td>
<td>Yes</td>
<td>14</td>
<td>69 (57–81)</td>
<td>0.522</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>212</td>
<td>73 (70–76)</td>
<td></td>
</tr>
<tr>
<td>Hypertension†</td>
<td>Yes</td>
<td>133</td>
<td>72 (68–76)</td>
<td>0.479</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>93</td>
<td>74 (69–79)</td>
<td></td>
</tr>
<tr>
<td>Alcohol intake†</td>
<td>Never</td>
<td>80</td>
<td>68 (63–73)</td>
<td>0.097</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>122</td>
<td>75 (71–79)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Heavy</td>
<td>13</td>
<td>80 (70–90)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ex-heavy</td>
<td>7</td>
<td>81 (56–100)</td>
<td></td>
</tr>
<tr>
<td>Smoking†</td>
<td>Never</td>
<td>101</td>
<td>69 (64–74)</td>
<td>0.047</td>
</tr>
<tr>
<td></td>
<td>Ex</td>
<td>80</td>
<td>78 (73–83)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Current</td>
<td>42</td>
<td>73 (66–79)</td>
<td></td>
</tr>
<tr>
<td>Prior MI†</td>
<td>Yes</td>
<td>20</td>
<td>76 (67–85)</td>
<td>0.554</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>206</td>
<td>73 (69–76)</td>
<td></td>
</tr>
<tr>
<td>Prior TIA†</td>
<td>Yes</td>
<td>21</td>
<td>79 (69–90)</td>
<td>0.181</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>205</td>
<td>72 (69–75)</td>
<td></td>
</tr>
</tbody>
</table>

PVD indicates peripheral vascular disease; MI, myocardial infarction; and TIA, transient ischaemic attack.

*Student's t test was used except for alcohol and smoking (1-way ANOVA).
†Defined as a known past history.
‡Defined as a known past history or current presentation.

TABLE 4. Handicap at 2 Years After First-Ever Stroke According to Impairment, Disability, Recurrence, and Institutionalization Status at 2 Years After Stroke

<table>
<thead>
<tr>
<th>Variable</th>
<th>Classification</th>
<th>n</th>
<th>Mean LHS (95% CI)</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>NIHSS</td>
<td>0–5</td>
<td>170</td>
<td>80 (78–83)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>6–10</td>
<td>20</td>
<td>52 (44–59)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>11–15</td>
<td>10</td>
<td>39 (21–56)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&gt;15–20</td>
<td>5</td>
<td>33 (28–38)</td>
<td></td>
</tr>
<tr>
<td>Barthel</td>
<td>0–4</td>
<td>24</td>
<td>32 (27–38)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>5–9</td>
<td>15</td>
<td>53 (43–63)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>10–14</td>
<td>16</td>
<td>56 (48–63)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>15–19</td>
<td>69</td>
<td>71 (68–75)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>101</td>
<td>89 (87–92)</td>
<td></td>
</tr>
<tr>
<td>Nonstroke disability</td>
<td>Yes</td>
<td>47</td>
<td>64 (58–70)</td>
<td>0.002</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>178</td>
<td>75 (72–79)</td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>Yes</td>
<td>36</td>
<td>71 (65–76)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>139</td>
<td>84 (81–86)</td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td>Yes</td>
<td>17</td>
<td>67 (58–76)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>158</td>
<td>82 (80–85)</td>
<td></td>
</tr>
<tr>
<td>Inward irritability</td>
<td>Yes</td>
<td>8</td>
<td>67 (63–71)</td>
<td>0.021</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>167</td>
<td>82 (81–83)</td>
<td></td>
</tr>
<tr>
<td>Outward irritability</td>
<td>Yes</td>
<td>6</td>
<td>72 (64–80)</td>
<td>0.191</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>169</td>
<td>81 (80–82)</td>
<td></td>
</tr>
<tr>
<td>Stroke recurrence</td>
<td>Yes</td>
<td>15</td>
<td>59 (49–70)</td>
<td>0.018</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>211</td>
<td>74 (71–77)</td>
<td></td>
</tr>
<tr>
<td>Institutionalized†</td>
<td>Yes</td>
<td>51</td>
<td>51 (45–57)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>175</td>
<td>79 (76–82)</td>
<td></td>
</tr>
</tbody>
</table>

*pStudent’s t test was used except for Barthel and NIHSS (linear trend contrast).
†in an institution at 2 years after stroke.

TABLE 5. Independent Determinants of Handicap at 2 Years After First-Ever Stroke, Including Mood Assessments

<table>
<thead>
<tr>
<th>Variable</th>
<th>Multivariable P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>2-y Disability score (Barthel)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>2-y Depression score (IDA)</td>
<td>0.002</td>
</tr>
<tr>
<td>2-y Anxiety score (IDA)</td>
<td>0.002</td>
</tr>
<tr>
<td>2-y Impairment score (NIHSS)</td>
<td>0.025</td>
</tr>
<tr>
<td>Age</td>
<td>0.023</td>
</tr>
</tbody>
</table>

IDA indicates Irritability, Depression, Anxiety scale.

*Multiple linear regression. Adjusted R² for multivariable model = 0.47.

in the NEMESIS pilot study in which there were fewer patients with ICH.² The 6 undetermined patients (ie, those without neuroimaging) who had severe handicap were highly dependent before their stroke. Similar to the NEMESIS pilot study,² TACI patients in this study were significantly more handicapped than patients with other subtypes at 2 years after stroke. The data demonstrate that the OCSP classification has utility for predicting long-term outcome; ie, TACI patients either will be dead or will be likely to have severe handicap.

The main strengths of this study were the community-based design, “ideal” methodology for case ascertainment, and high participation rate. This methodology improves the generalizability of the findings. However, factors influencing handicap such as different rates of rehabilitation for stroke patients, building design, and occupational structure may vary between countries. Our analyses were exploratory, and because of the number of comparisons made, some associations may have been significant by chance (eg, ex-smokers being less handicapped than never smokers). The results need validation in other cohorts. Furthermore, our findings are based on 2-year survivors; those who had died may have had more severe handicap.

Interventions that can reduce impairment and disability are likely to be most effective in reducing handicap. Stroke units²⁰ and thrombolysis in selected patients²¹ have been shown to reduce impairment and disability. However, most patients are ineligible for thrombolysis according to current guidelines.²¹,²² Rehabilitation has the potential to reduce impairment and disability, although work is required both to demonstrate and to improve its effectiveness.²² Our study provides evidence that mood disorders may independently contribute to handicap in stroke patients and therefore there is some rationale for the clinician to look for and treat these disorders aggressively. Treatment of depression and anxiety with a combination of patient education, psychotherapy, and
medications as appropriate could be explored as an avenue to reduce handicap. The results of small studies provide evidence that treatment of mood disorders in stroke patients can be successful, but larger randomized clinical trials are required to confirm this finding and ascertain optimal therapy.

There may be advantages for using handicap rather than disability as an outcome measure. Although disability may be less prone to biases relating to differences in societal structures between countries, handicap provides more information about the impact of stroke on patients’ lives and is a more sensitive measure. Some interventions such as rehabilitation or community support programs may prove effective in improving patient outcomes without having an impact on disability. Whichever outcome measure is used, case fatality must also be considered when outcomes between groups are compared. The effectiveness of providing physical and economic resources and social support in reducing handicap remains largely unknown and requires research.

Acknowledgments

This work was supported by grants from the National Health and Medical Research Council (NHMRC), Victorian Health Promotion Foundation, Foundation for High Blood Pressure Research, and National Stroke Foundation. Dr. Sturm was supported by an NHMRC postgraduate medical scholarship. Lichun Quang provided assistance with database management and analysis. The contribution of the following research nurses is also gratefully acknowledged: Stephen Cross, Barbara Dowell, Amanda Loth, Mary Staios, Cathy Taranto, and Dennis Young. We thank Dr. John Ludbrook, Biomedical Statistical Consulting Service, for assistance with the statistical analyses.

References

Determinants of Handicap After Stroke: The North East Melbourne Stroke Incidence Study (NEMESIS)

Stroke. 2004;35:715-720; originally published online February 12, 2004;
doi: 10.1161/01.STR.0000117573.19022.66
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2004 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/35/3/715

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/