Impact of Premorbid Undernutrition on Outcome in Stroke Patients

James P. Davis, BComm, BNurs(Hons); Andrew A. Wong, MBBS; Philip J. Schluter, BSc(Hons), MSc, PhD; Robert D. Henderson, MBBS, FRACP; John D. O’Sullivan, MBBS, MD, FRACP; Stephen J. Read, MBBS, PhD, FRACP

Background and Purpose—To assess the prevalence of premorbid undernutrition and its impact on outcomes 1 month after stroke.

Methods—The study recruited from consecutive stroke admissions during a 10-month period. Premorbid nutritional status (using the subjective global assessment [SGA]), premorbid functioning (modified Rankin scale [MRS]), and stroke severity (National Institutes of Health Stroke Scale [NIHSS] score) were assessed at admission. The associations between premorbid nutritional status, poor outcome (defined as MRS ≥3), and mortality were examined before and after adjustment for confounding variables, including age, gender, stroke risk factors, stroke severity, and admission serum albumin.

Results—Thirty of 185 patients were assessed as having undernutrition at admission. Significant unadjusted associations were observed between undernutrition and poor outcome (odds ratio [OR], 3.4; 95% CI, 1.3 to 8.7; \(P=0.01\)), and mortality (OR, 3.1; 95% CI, 1.3 to 7.7; \(P=0.02\)) at 1 month. NIHSS, age, and premorbid MRS were also significantly associated with poor outcomes. After adjustment for these factors, the effect size of associations remained important but not significant (poor outcome: OR, 2.4; 95% CI, 0.7 to 9.0, \(P=0.18\); mortality: OR, 3.2; 95% CI, 1.0 to 10.4, \(P=0.05\)).

Conclusions—Premorbid undernutrition, as assessed using the SGA, appears to be an independent predictor of poor stroke outcome. Stroke prevention strategies should target undernutrition in the population at risk for stroke to improve outcomes. (Stroke. 2004;35:1930-1934.)

Key Words: stroke ■ outcome ■ diet

Undernutrition is common in hospitalized patients. Undernutrition rates of ≤50% have been reported in studies involving surgical, medical, geriatric, and stroke patients. Despite the recognized frequency of undernutrition, the impact it has on clinical outcome has not been studied widely, particularly in stroke.

Recent studies provide some indication that poor nutrition may influence outcome after stroke. Davalos et al and Gariballa et al found nutrition assessed after admission to be associated with mortality and dependence at 1 month after stroke. More recently, the FOOD Trial investigators found postadmission nutrition to be associated with death and dependence at 6 months after stroke.

Several of these studies relied on serum albumin as a marker for nutritional status. This can be a useful measure where acute changes in nutrition need to be assessed during time frames of <1 month. However, it is sometimes difficult to distinguish between changes in serum albumin as a result of nutrition versus underlying disease processes. This problem was reported in acute stroke by Davalos et al, in which stroke severity influenced serum albumin measures. Additionally, there may be a delayed change in serum albumin after a change in nutritional status attributable to the long half life of albumin.

The FOOD Trial investigators recently reported on a study that used a combination of subjective assessment, weight/height measurement, blood tests, and anthropometry. This might be expected to give a more complete picture of nutritional status. However, these methods were not applied consistently across the study population.

The aim of the present study was to assess the impact of premorbid undernutrition on stroke outcome via the consistent application of a validated nutritional assessment methodology, the subjective global assessment (SGA). SGA gives a subjective assessment of nutrition on the basis of a detailed structured nutritional history and physical examination and is independent of acute physiological measurements. This article reports our finding of premorbid undernutrition prevalence in a cohort of stroke patients and its impact on outcomes at 1 month after stroke.
Materials and Methods

Participants
All patients admitted between June 1, 2002, and March 31, 2003, with a diagnosis consistent with the World Health Organization definition of stroke were eligible for inclusion. Patients with a diagnosis of transient ischemic attack, subarachnoid hemorrhage, subdural or extradural hemorrhage, or brain injury resulting from trauma or neoplastic causes were excluded.

Data Collection
All patients were assessed within 24 hours of stroke. Data were obtained directly from patients via their next of kin or from medical records. Demographic data, stroke factors, serum albumin, stroke severity, premorbid functional capacity, and premorbid nutritional status were assessed at baseline. Stroke severity was assessed using the National Institutes of Health stroke score (NIHSS). Premorbid functional capacity was measured using the modified Rankin scale (MRS) and was repeated at 1 month as one of the primary outcomes. Premorbid nutritional status was assessed at admission using SGA. Baseline serum albumin was recorded as a comparative measure given its use as a marker for nutritional status in previous studies.

Primary Outcomes
The primary outcomes for this study were mortality and “poor outcome,” defined as MRS ≥3, measured at 30 days after stroke. The rating MRS ≥3 was used to maintain consistency with the baseline assessment.

Nutritional Assessment
SGA was applied in this study as described originally by Detsky et al. SGA determines nutritional status on the basis of a clinical evaluation of patient history and a physical examination. The patient history is a structured questionnaire administered by the assessor and details weight changes, dietary intake, gastrointestinal symptoms, functional capacity, and disease. The physical examination assesses subcutaneous fat, muscle wasting, edema, and ascites. For each of these subcriteria, the assessor gives a rating on a 3-point ordinal scale. An overall rating for nutritional status is then determined on the basis of subcriteria ratings assessment. Patient nutritional status is rated by the assessor as well nourished (A rating), moderately undernourished (B rating), or severely undernourished (C rating). For analytical purposes, in our study, patients with an SGA rating of B or C were considered “undernourished,” whereas patients with an A rating were considered “well nourished.” The decision to combine the B and C ratings was made when results revealed only 1 patient with a C rating.

Ethics
This study was approved by the hospital human research ethics committee. Written informed consent was obtained from patients or their next of kin in cases in which a patient could not communicate.

Statistical Analysis
Comparisons of baseline variables by outcomes and premorbid nutrition were undertaken using the Fisher exact test for categorical variables and an unpaired t test for continuous variables, respectively. Significant potential confounders (including serum albumin), the nutrition variables of interest, and all 2-way interactions were then entered into logistic regression models, and the most parsimonious models were derived using manual backward elimination methods. This analytical approach was selected because the sample size was not large (only n=185), and we had a number of potentially important independent variables, but their relationship to the dependent variable or each other was not well understood. Moreover, these independent variables were not “design,” “structural,” or “cluster” variables that necessitated their automatic inclusion in the regression model. Statistical analyses were performed using the Statistical Package for the Social Sciences version 11 (SPSS for Windows), and the significance level α=0.05 was used for all statistical comparisons.

Results
A total of 220 patients were screened at admission. The figure depicts the recruitment process. Thirty-five patients were ineligible to participate, refused consent, or failed to complete the study, leaving 185 patients for analysis.

At admission, premorbid undernutrition was found in 30 (16%) patients. At 1 month after stroke, 28 (15%) patients had died and 108 (58%) patients had a poor outcome (MRS ≥3). Table 1 shows baseline characteristics of patients partitioned by undernutrition at admission, mortality, and poor outcome at 1 month. Patients who were undernourished were significantly more likely to die (30% versus 12%; P=0.02) or have a poor outcome (80% versus 54%; P=0.01) than their well-nourished counterparts. Stroke severity measured by the mean (±SD) NIHSS was not associated with premorbid nutrition (SGA A 8 [±8] versus SGA B/C 9 [±9]; P=0.7), but it was strongly associated with both outcome variables (mortality 20 [±9] versus 7 [±6], P<0.001; and poor outcome 13 [±8] versus 3 [±2], P<0.001). Age was significantly associated with premorbid nutrition (P<0.001) and both outcomes (P<0.05). Baseline MRS was associated with premorbid nutrition (P<0.05) and poor outcome (P<0.001) but not mortality.

Crude data analysis (Table 1) revealed associations between premorbid nutrition, premorbid residential status, and stroke history (P<0.05) but no association with stroke risk factors such as atrial fibrillation, diabetes mellitus, hypertension, or hyperlipidemia. None of these factors were associated with mortality, and only premorbid residential status and atrial fibrillation were associated with poor outcome (P<0.05). Low serum albumin at admission (≤34g/L) was associated with premorbid nutrition (P<0.001) and mortality at 1 month (P<0.05) but not poor outcome at 1 month.

Investigating the collinearities and relationships among significant independent variables, age, premorbid MRS, premorbid SGA, and the outcome variables appeared to produce unstable estimates with large associated CIs. It emerged that only 3 of 93 (3%) patients aged ≤74 years had a premorbid...
MRS ≥3, whereas 21 of 92 (23%) patients aged ≥75 years had premorbid MRS ≥3. To ensure stability within the regression model, these 2 variables were combined and recategorized into 3 groups, namely: (1) <75 years; (2) ≥75 years and premorbid MRS ≥2; and (3) ≥75 years and premorbid MRS ≥3. The final adjusted model for mortality at 1 month included NIHSS (P < 0.001) only, whereas the adjusted model for poor outcome (MRS ≥3) included NIHSS (P < 0.001) and the combined age and premorbid MRS (P = 0.02), as shown in Table 2.

A variation to this model excluded patients with premorbid MRS ≥3 (n = 161) on the basis that the excluded patients had already achieved a poor outcome before the stroke. For mortality, this model was adjusted for NIHSS (odds ratio [OR], 2.9; 95% CI, 0.7 to 11.6). For poor outcome (MRS ≥3), the model was adjusted for NIHSS and age dichotomized at <75 or ≥75 years (OR, 3.7; 95% CI, 0.9 to 14.7).

### Discussion

This study found a significant crude association among premorbid nutritional status and mortality (OR, 3.1; P = 0.02) and poor outcome (OR, 3.4; P = 0.01) at 1 month. After adjustment for factors such as age, premorbid dependence, and stroke severity, an important effect size was seen between these variables, but this was not statistically significant (mortality: OR, 3.2, P = 0.05; poor outcome: OR, 2.4, P = 0.18).

### Table 1. Baseline Characteristics of Study Population (N = 185) Partitioned by Premorbid Undernutrition, Mortality, and Poor Outcome at 1 Month

<table>
<thead>
<tr>
<th></th>
<th>Undernutrition (SGA A or B)</th>
<th>Mortality</th>
<th>Poor Outcome (MRS ≥3)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>n (%)</td>
<td>P value†</td>
</tr>
<tr>
<td><strong>Age, y</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;75</td>
<td>93</td>
<td>3 (3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>≥75</td>
<td>92</td>
<td>27 (29)</td>
<td></td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>98</td>
<td>15 (15)</td>
<td>0.80</td>
</tr>
<tr>
<td>Female</td>
<td>87</td>
<td>15 (17)</td>
<td></td>
</tr>
<tr>
<td><strong>Nutrition status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Well-nourished</td>
<td>155</td>
<td>n/a</td>
<td>n/a</td>
</tr>
<tr>
<td>Undernourished</td>
<td>30</td>
<td>n/a</td>
<td></td>
</tr>
<tr>
<td><strong>Serum albumin</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;34</td>
<td>155</td>
<td>17 (11)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>≤34</td>
<td>30</td>
<td>13 (43)</td>
<td></td>
</tr>
<tr>
<td><strong>Residential status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aged care*</td>
<td>19</td>
<td>8 (42)</td>
<td>0.004</td>
</tr>
<tr>
<td>Home</td>
<td>166</td>
<td>22 (13)</td>
<td></td>
</tr>
<tr>
<td><strong>Baseline MRS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;3</td>
<td>161</td>
<td>21 (13)</td>
<td>0.006</td>
</tr>
<tr>
<td>≥3</td>
<td>24</td>
<td>9 (38)</td>
<td></td>
</tr>
<tr>
<td><strong>Atrial fibrillation</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>49</td>
<td>10 (20)</td>
<td>0.80</td>
</tr>
<tr>
<td>No</td>
<td>136</td>
<td>20 (15)</td>
<td></td>
</tr>
<tr>
<td><strong>Diabetes mellitus</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>32</td>
<td>6 (19)</td>
<td>0.60</td>
</tr>
<tr>
<td>No</td>
<td>153</td>
<td>24 (16)</td>
<td></td>
</tr>
<tr>
<td><strong>Hypertension</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>113</td>
<td>20 (18)</td>
<td>0.60</td>
</tr>
<tr>
<td>No</td>
<td>72</td>
<td>10 (14)</td>
<td></td>
</tr>
<tr>
<td><strong>Hyperlipidemia</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>56</td>
<td>10 (18)</td>
<td>0.70</td>
</tr>
<tr>
<td>No</td>
<td>129</td>
<td>20 (16)</td>
<td></td>
</tr>
<tr>
<td><strong>History of stroke</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>38</td>
<td>11 (29)</td>
<td>0.03</td>
</tr>
<tr>
<td>No</td>
<td>147</td>
<td>19 (13)</td>
<td></td>
</tr>
</tbody>
</table>

*Including nursing home or hostel.
†P values calculated using Fisher exact test.
The most important finding of our study is that there appears to be an association between premorbid nutrition and stroke outcomes. The importance of this finding is indicated by the effect size of this association. Although the adjusted data were not statistically significant, the modifiable nature of nutrition status suggests that this is an important finding for clinical practice and stroke prevention strategies.

The results of this study are strengthened by the methodology used to assess nutritional status. SGA methodology provides an assessment of nutritional status using a medical history of the preceding weeks and months and a physical examination at the time of assessment. On the basis of these data, SGA is a sound estimate of premorbid nutrition because it accounts for the nutritional impact of a broad range of factors such as poor premorbid functioning, types and quantities of nutrition, and gastrointestinal system functioning. Deficiencies in these types of nutritional factors are often prevalent in the population at risk for stroke.

Another strength of this study is independence of the nutritional assessment by SGA from poststroke biochemical data. On the basis of crude data, nutrition by SGA was found to be associated with both outcomes, whereas nutrition by baseline serum albumin was associated only with mortality (Table 1). Detailed comparative studies with objective nutrition measures such as serum albumin have been reported previously. Detsky et al found SGA to have better predictive ability than objective measures for predicting postoperative complications and a high degree of inter-rater reliability (91% nurse–physician agreement; $\kappa = 0.78$). The sensitivity (0.82) and specificity (0.72) of SGA were reported as superior to other nutrition measures such as body fat percentage, delayed cutaneous hypersensitivity, serum albumin, serum transferrin, creatine height index, and prognostic nutritional index. This body of evidence suggests that SGA may be an improvement on these more traditional nutritional assessment methods.

Previous studies have tended to rely on serum albumin as a measure of nutritional status. Gariballa et al reported a tendency for nutrition (assessed by serum albumin) to be associated with stress response, stroke severity, and swallowing difficulties. In contrast, the present study found no association between nutrition and stroke severity ($P=0.7$). For both previous studies, the reported associations may have been related to use of serum albumin as the primary measure of nutrition as evidenced by the reported association between stroke severity and serum albumin. Although baseline measurements of serum albumin (<24 hours) may not be affected by the acute stress response after stroke, serial measures introduce stress response and stroke severity as potential confounders in nutritional assessment.

Less emphasis was placed on serum albumin in the FOOD Trial, in which a composite measure of nutritional status was used. The method included an unstructured subjective bedside assessment (60% of cases), weight/BMI (20%), blood indices (11%), and anthropometry (2%). Although using a combination of nutritional measures is common, these methods were applied inconsistently throughout this study.

Potential limitations of our study relate primarily to sample size and to the intrinsic difficulties in measuring nutrition. The limitation of sample size is observable in Table 2, in which effect size of the associations remained steady after adjustment despite a decrease in statistical significance. These data suggest that a larger sample size may have produced statistically significant results.

A more complex set of issues arises in relation to nutritional assessment. The subjective nature of SGA lends itself to potential bias. It may be argued that a patient history is potentially biased by the incidence of a sudden onset of symptoms such as stroke. Although this is possible, the impact of this bias on clinician assessment is no different to potential confounders in nutritional assessment. After this, potential bias may occur because of differences in clinical judgment. This source of bias has been shown to be minimal in previous reports of inter-rater reliability studies.

Another potential limitation of this study is that it may be argued that a longitudinal study of a “healthy” population at risk for stroke is a more accurate means for assessing premorbid nutrition. This may be the case; however, in designing a low-cost, clinically pragmatic study such as ours, it is suggested that the use of SGA provides an adequate estimate of nutrition in the weeks and months preceding the clinical event.

Finally, another issue is the impact of illness on nutrition before the stroke event. This was a particular issue with our study because a significant proportion of participants had achieved a poor outcome (MRS ≥3) before the stroke event (n = 24). However, for SGA, this is less of an issue than for measurements such as poststroke serum albumin. SGA accounts for previous functioning and its impact on nutrition. In addition, analysis of the subgroup with good premorbid functioning suggests that nutrition (by SGA) remains a significant predictor of poor outcome after adjustment for stroke severity and older age (OR, 3.7; 95% CI, 0.9 to 14.7; $P=0.06$; n = 161).

Our results imply that for people at risk of stroke, maintenance of good nutrition may influence their survival or their

---

**TABLE 2. Crude and Adjusted ORs Relating Characteristics of Study Population to Mortality at 1 Month and Poor Outcome at 1 Month**

<table>
<thead>
<tr>
<th>characteristic</th>
<th>mortality</th>
<th>poor outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Crude OR (95% CI)</td>
<td>Adjusted OR (95% CI)</td>
</tr>
<tr>
<td>mortality</td>
<td>n/N (%)</td>
<td></td>
</tr>
<tr>
<td>Well-nourished</td>
<td>19/155 (12)</td>
<td>1.0</td>
</tr>
<tr>
<td>Undernourished</td>
<td>9/30 (30)</td>
<td>3.1 (1.3, 7.7)</td>
</tr>
<tr>
<td>poor outcome</td>
<td>n/N (%)</td>
<td></td>
</tr>
<tr>
<td>Well-nourished</td>
<td>84/155 (54)</td>
<td>1.0</td>
</tr>
<tr>
<td>Undernourished</td>
<td>24/30 (80)</td>
<td>3.4 (1.3, 8.7)</td>
</tr>
</tbody>
</table>

*The model for mortality was adjusted for NIHSS only. Poor outcome was adjusted for NIHSS and the trichotomous age/premorbid MRS variable.
level of independence poststroke, independent of other factors. With an incidence of premorbid undernutrition of 16% in our study population, this study suggests that attention to nutrition when considering other stroke prevention strategies may improve outcomes for a significant proportion of the at-risk population.

In keeping with previous studies, we also found older people, especially those with impaired functional capacity, and those living in aged care facilities to be more susceptible to undernutrition. These groups in particular need to be targeted by nutrition improvement strategies to limit the impact undernutrition has on stroke outcomes.

In summary, this study has shown that premorbid undernutrition may increase the risk of poor outcome at 1 month after stroke. Importantly, this study measured premorbid undernutrition, which was the only modifiable risk factor to show an important effect size for both outcomes that approached statistical significance. These results suggest that strategies aimed at improving premorbid nutrition in the population at risk for stroke may improve poststroke outcomes. Such strategies might include screening for undernutrition among the stroke-age population using SGA and nutrition improvement programs aimed at older people, either living at home or in supported aged care facilities.

Acknowledgments
This project was supported by the Royal Brisbane and Women’s Hospital Foundation, Australia.

References
Impact of Premorbid Undernutrition on Outcome in Stroke Patients
James P. Davis, Andrew A. Wong, Philip J. Schluter, Robert D. Henderson, John D. O'Sullivan and Stephen J. Read

*Stroke*. 2004;35:1930-1934; originally published online June 24, 2004;
doi: 10.1161/01.STR.0000135227.10451.c9
*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/35/8/1930

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/