Weight Loss After Stroke
A Population-Based Study From the Lund Stroke Register
Ann-Cathrin Jönsson, RN, MSc, PhD; Ingrid Lindgren, RPT, MSc; Bo Norrving, MD, PhD; Arne Lindgren, MD, PhD

Background and Purpose—Data on the prevalence and indicators of weight loss in population-based groups of stroke survivors are scarce. We aimed to find the predictors and indicators of weight loss >3 kg as a possible marker of malnutrition after stroke.

Methods—We registered weight at baseline, after 4 months, and 1 year later in 305 survivors from a population-based cohort of first-ever stroke patients. Characteristics of the patients were registered at baseline and follow-ups, including glycosylated hemoglobin at baseline and follow-up II, eating difficulties at both follow-ups, and screening for depression at follow-up II. We used univariate and multivariate analyses to find baseline predictors and follow-up indicators related to weight loss >3 kg from baseline.

Results—Among the 305 patients, 60% were male, the mean age was 72.5 years, and mean body mass index was 25.8 kg/m². The main stroke types were cerebral infarction (89%), intracerebral hemorrhage (7%), and subarachnoid hemorrhage (4%). Weight loss >3 kg was found in 74 (24%) patients (mean, −6.6 kg) after 4 months and in 79 patients (26%; mean, −8.3 kg) 1 year later. Severe stroke and elevated glycosylated hemoglobin levels were baseline predictors of weight loss >3 kg. Indicators associated with short-term weight loss (at follow-up I) were eating difficulties, low prealbumin value, and dependence (Barthel Index), whereas indicators associated with long-term weight loss (follow-up II) were eating difficulties, hemorrhagic stroke, and low prealbumin value.

Conclusions—Weight loss >3 kg after stroke indicates the need for closer observation regarding nutritional status. Monitoring of body weight may be useful, particularly among patients with severe stroke, eating difficulties, low prealbumin values, and impaired glucose metabolism. (Stroke. 2008;39:918-923.)

Key Words: albumin • glycosylated hemoglobin • nursing care • malnutrition • outcome • prealbumin • stroke care • weight loss

Loss of weight in stroke patients may be related to dysphagia,1 depression,2 aspiration,3 decreased dietary intake,4 and other neurologic deficits that contribute to eating difficulties.5 The definition of malnutrition varies between studies. Unintentional weight loss or a low body mass index (BMI <18 kg/m² for persons <65 years and <22 kg/m² for persons ≥65 years) has been defined as an indicator of malnutrition in stroke patients.5 Another study used a BMI <20.5 kg/m² as a risk cutoff for malnutrition.6 Low serum albumin and prealbumin levels have also been associated with malnutrition, impaired functional status, poor outcome, and mortality, especially among the elderly.7–9 Frailty10 and unawareness of weight changes may further add to the risk of weight loss and poor outcome in stroke patients.11,12 Most studies of weight loss and malnutrition after stroke have examined selected subjects, such as patients in rehabilitation units5,5 or nursing homes11,13 or patients selected by different exclusion criteria.8,12 In previous studies, malnutrition, often combined with low albumin and prealbumin values, has been reported as an indicator of poor outcome after stroke.7,8,12 However, data on the prevalence of weight loss after stroke in population-based groups of stroke survivors are scarce. Our aim was to examine the prevalence of weight loss in a population-based group of stroke survivors and to find (1) baseline predictors and (2) follow-up indicators of weight loss >3 kg as possible markers of malnutrition in a short-term as well as a long-term perspective.

Subjects and Methods
A total of 416 consecutive first-ever stroke patients with stroke onset between March 1, 2001 and February 28, 2002 were included in the Lund Stroke Register. The Lund Stroke Register covers the catchment area of Lund University Hospital, including 8 municipalities with 234 505 inhabitants (as of December 31, 2001). The methods for detecting all first-ever stroke patients and the low rate of dropouts during the defined period have been described previously.14 All patients but 1 with a final diagnosis of first-ever stroke underwent computed tomography of the brain.

All surviving patients were contacted a median of 4 (follow-up I) and 16 (follow-up II) months after stroke onset and offered a
personal appointment with a nurse specialist (A.J.) and a physical therapist (I.L.). Data were collected for 327 patients at follow-up I and for 305 patients at follow-up II. Among the 22 patients unable to be followed up twice, 20 were deceased and 2 declined to participate at follow-up II. Thus, 305 patients were followed up twice and constitute the basis for the analyses in this report. Approximately 70% of the patients examined were able to come to the outpatient clinic, whereas the remainder were examined in primary care centers (≈10%), nursing homes (≈10%), or their own homes (≈10%). Abnormal findings were reported to the physician responsible for the care of the patient. The data were not analyzed until after the follow-up had been finished to avoid investigator bias.

Informed consent was obtained from each participant, or, if the patients were confused or had sensory dysphasia, their spouses or significant others. The study was approved by the ethics committee of the Faculty of Medicine, Lund University.

Baseline Assessments

We registered the following baseline variables: age, sex, main stroke type (cerebral infarction, subarachnoid hemorrhage, and intracerebral hemorrhage), living situation (living in own home or nursing home), family situation (living alone or with someone), social participation (0 to 9 different activities),15 and diabetes mellitus (fasting blood glucose ≥6.1 mmol/L or plasma glucose ≥7.0 mmol/L at repeated measurements or an earlier diagnosis). Each patient’s functional status was assessed with the National Institutes of Health Stroke Scale (NIHSS). We used a version of the NIHSS that included an item for right- and left-hand motor function (maximum 2 points for each hand).16 In cases where the NIHSS score had not been assessed at the clinic, the score was assigned from medical reports, a method found to have good reliability and validity.17 Weight and height were measured to calculate BMI. BMI was classified as underweight (UW), <18.5 kg/m²; normal weight (NW), a BMI of 18.5 to 24.9 kg/m²; overweight (OW)/obese as 25 kg/m².18 A BMI >20.5 kg/m² was used as a cutoff for risk of malnutrition.6 Impaired glucose tolerance is a common cause of metabolic changes that may influence body weight. Therefore, we examined glycosylated hemoglobin (HbA1c) to screen for abnormal glucose tolerance.19 Functional status before stroke onset was assessed by interview regarding ambulation indoors and outdoors, dressing, and toileting.20 If the patients could manage these activities of daily living by themselves, they were considered independent before stroke onset. The frequency of physical activities (walking, gardening, swimming, running, and other sports) before stroke and at follow-up was registered on a scale from 0 (no physical activity) to 4 (almost every day).21 Nutritional status was also examined by measuring plasma albumin and serum prealbumin concentrations.

Follow-Up Assessments

At both follow-ups, functional status was assessed with the Barthel Index (BI)22 and divided into 3 grades of dependence.23 We repeated the baseline measurements at both follow-ups, but HbA1c was classified as underweight (UW), <18.5 kg/m²; normal weight (NW), a BMI of 18.5 to 24.9 kg/m²; overweight (OW)/obese as 25 kg/m².23 A BMI >20.5 kg/m² was used as a cutoff for risk of malnutrition.6 Impaired glucose tolerance is a common cause of metabolic changes that may influence body weight. Therefore, we examined glycosylated hemoglobin (HbA1c) to screen for abnormal glucose tolerance.19 Functional status before stroke onset was assessed by interview regarding ambulation indoors and outdoors, dressing, and toileting.20 If the patients could manage these activities of daily living by themselves, they were considered independent before stroke onset. The frequency of physical activities (walking, gardening, swimming, running, and other sports) before stroke and at follow-up was registered on a scale from 0 (no physical activity) to 4 (almost every day).21 Nutritional status was also examined by measuring plasma albumin and serum prealbumin concentrations.

Table 1. Functional Status of 305 Stroke Patients Followed Up Twice

<table>
<thead>
<tr>
<th>Grade of Dependence</th>
<th>Before Stroke, %</th>
<th>Follow-Up I, %</th>
<th>Follow-Up II, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Independence (BI 95–100)</td>
<td>98</td>
<td>71</td>
<td>69</td>
</tr>
<tr>
<td>Moderate dependence (BI 60–90)</td>
<td>1</td>
<td>19</td>
<td>19</td>
</tr>
<tr>
<td>Major dependence (BI 0–55)</td>
<td>1</td>
<td>10</td>
<td>12</td>
</tr>
</tbody>
</table>

*Assessed by interview regarding indoor and outdoor ambulation, dressing, and toileting.

indicators assessed at follow-up II that were correlated with weight loss >3 kg between baseline and follow-up II.

Statistical Analysis

The variables HbA1c, albumin, and prealbumin were dichotomized into 2 groups, ie, above/below the lower reference value for albumin <34 g/L according to our clinical chemical laboratory, for prealbumin <0.20 g/L,9 and for HbA1c >5.3%.26 Other dichotomized variables were GDS-20 (≥6 or not), main stroke type (infarction or hemorrhage), diabetes mellitus (yes/no), and independence (BI 95 to 100) or dependence (BI 0 to 90).25 First we analyzed the variables assessed at each measuring point separately by using χ² tests for dichotomous variables and the Mann-Whitney test for continuous variables by making comparisons with the dichotomous variable, weight loss >3 kg or not, as the dependent variable. Afterward, we performed a forward logistic-regression analysis with the same variables as in the univariate analyses and also with the dichotomous variable, weight loss >3 kg or not, as the dependent variable. Probability values <0.05 were considered significant.

Results

Patient Characteristics

Among the 305 patients, 183 (60%) were male and the mean age at stroke onset was 72.5 years, with a quarter of the patients below retirement age (65 years). Stroke main type was cerebral infarction in 89%, intracerebral hemorrhage in 7%, and subarachnoid hemorrhage in 4%. At baseline, 42 patients had diabetes mellitus and 54 patients had HbA1c levels above the upper reference value; 18 of these 54 patients had not been diagnosed with diabetes mellitus previously. Albumin and prealbumin values below lower reference values were found in 51 of 293 (17%) and 55 of 262 (21%) patients at baseline. The mean/median NIHSS score at stroke onset was 5/3, and the mean/median BMI was 25.8/25.4. Functional status before and after stroke is presented in Table 1, which shows that the patients had good functional status before stroke, with 98% being independent. At both follow-ups, ≈70% were independent (Table 1). The majority (53% to 55%) of the patients were OW/obese, and only ≈5% were UW on the 3 assessment occasions when the total group of 305 patients was classified (Table 2).

Baseline Predictors

At follow-up I, weight loss from stroke onset of >3 kg (range, −3.1 to −30.2 kg; mean/median, −6.6/−5.2 kg) was present in 74 (24%) of the 305 patients. Among the other 231 patients, the range of weight differences was −2.9 kg to 9.1 kg, and the mean/median weight difference was 0.6/0.1 kg. For the total group of 305 patients, the mean/median weight
loss was $-1.1\pm 0.6$ kg. The mean/median NIHSS score was 8/5 in the patients with weight loss $>3$ kg and 4/3 in the other patients. Baseline predictors of weight loss $>3$ kg at follow-up I in the univariate analyses were hemorrhagic stroke type, higher (worse) NIHSS score, diabetes mellitus, and HbA1c above the upper reference value. Remaining baseline predictors in the multivariate regression analysis were higher (worse) NIHSS score and HbA1c above the upper reference value (Table 3).

### Follow-Up I

Among the 74 patients with weight loss $>3$ kg at follow-up I, 49 (66%) stated that they had reduced their food intake due to eating difficulties, whereas among the remaining 231 patients, only 21 (9%) reported a reduced food intake. Albumin and prealbumin values below the lower reference value was present in 19% versus 5% and 15% versus 6%, respectively, in the groups of 74 versus 231 patients. The proportions of the different BMI classes differed when we compared the 74 who had lost $>3$ kg (67.5% were OW at baseline and 61% at follow-up II) with the other 226 patients (52% were OW at baseline and 61% at follow-up II; Table 4). Indicators of weight loss at follow-up II in the univariate analyses were hemorrhagic stroke type, GDS-20 score indicative of depression, eating difficulties, and albumin/prealbumin values below the lower reference value. In the regression analysis, weight loss $>3$ kg was associated with hemorrhagic stroke, eating difficulties, and prealbumin value below the lower reference value (Table 3).

### Characteristics of the 20 Patients Who Died Between Follow-Up I and II

The 20 patients (from the original 327 patients examined at follow-up I) who died before follow-up II were older at stroke onset (mean age, 82.3 years; $P<0.001$) and had a higher (worse) mean/median NIHSS score (11.2/9.5) than the 305 survivors (5/3, $P<0.001$). Among the 20 patients, no fewer than 11 had lost $>3$ kg between baseline and follow-up I ($P=0.003$) compared with survivors (weight could not be measured for 1 person due to severe illness), and 11 had albumin ($P<0.001$) and prealbumin ($P>0.001$) values below the lower reference value at follow-up I.

### Discussion

This is the first study to report data on weight loss and its relation to predictors and indicators of weight loss over time in a population-based group of first-ever stroke patients. Previous studies of nutritional problems in stroke patients were performed in selected groups of patients discharged to rehabilitation facilities or nursing homes, who had moderate to severe stroke, and who often had prestroke disabilities and nutritional problems.\textsuperscript{2,3,12,27} Even though our patients were in good physical condition before stroke onset (98% were independent), no less than a quarter of them had a weight loss $>3$ kg in the short term as well as the long term. The strongest baseline predictor of weight loss was stroke severity (measured as NIHSS score). On the other hand, obesity, particularly abdominal obesity, has been reported to be a risk factor for stroke, and weight reduction has been emphasized in stroke prevention programs.\textsuperscript{28} However, a recent Cochrane review concluded that this hypothesis is not based on strong scientific evidence resulting from randomized, controlled clinical trials, at least not for primary prevention.\textsuperscript{29} Our results indicate a need to follow up weight after stroke to detect unintentional weight loss $>3$ kg related to possible malnutrition. Otherwise, the large proportion of patients with eating difficulties related to weight loss $>3$ kg and other factors related to nutritional status might not be detected.
It has been pointed out that there is no “gold standard” for determining nutritional status because there is no universally accepted definition of undernutrition. Despite this problem, we agree with the European Society for Clinical Nutrition and Metabolism guidelines for nutrition screening that it is important to monitor the patients’ nutritional status. In our study, we used weight loss >3 kg and albumin/prealbumin levels below lower reference values to assess possible malnutrition. Our results of an association between weight loss >3 kg and albumin/prealbumin values below the lower reference value in postacute stroke care strengthens the view that weight loss >3 kg can be regarded as a possible marker of malnutrition, especially because weight loss >3 kg was also a marker of poor outcome. A low serum albumin level has been associated with advanced atherosclerosis, adding to the prognostic information of other inflammatory markers, and can therefore be considered an acute-phase reactant. This is in line with our finding that the prevalence of albumin and prealbumin values below the lower reference value was higher at baseline than at both follow-ups. The observation that a larger proportion of patients who died between the 2 follow-ups had lower albumin values than did survivors is corroborated by other reports that low albumin concentrations predict poor outcome and mortality after stroke. Our conclusion is therefore that albumin/prealbumin levels indicate nutritional status in a population-based cohort of first-ever stroke patients in a longer perspective when the patients are more stable (but not in the acute phase).

In accordance with the patient perspective in our study, we registered eating difficulties related to loss of appetite and digestive, chewing, or swallowing problems from the patients’ self-reports. All of these problems may be caused directly or indirectly by stroke. In our study, eating difficulties were registered as present for patients who indicated that their food intake had declined over the past 3 months due to loss of appetite, digestive problems, or chewing or swallowing difficulties.

### Table 3. Stroke Patients With/Without Weight Loss >3 kg

<table>
<thead>
<tr>
<th>Variable</th>
<th>Weight Loss &gt;3 kg, n (%)</th>
<th>All Other Patients, n (%)</th>
<th>Univariate Analyses P Value*</th>
<th>Forward Logistic-Regression P Value*</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Baseline predictors†</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of patients</td>
<td>74</td>
<td>231</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroke type: ICH or SAH</td>
<td>13 (18)</td>
<td>19 (8)</td>
<td>0.05</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NIHSS score, mean/median</td>
<td>8/5</td>
<td>4/3</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>1.128</td>
<td>1.070–1.189</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>17 (23)</td>
<td>25 (11)</td>
<td>0.01</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HbA1c &gt;5.3%</td>
<td>19 (26)</td>
<td>35 (15)</td>
<td>0.05</td>
<td>0.02</td>
<td>2.278</td>
<td>1.115–4.653</td>
</tr>
<tr>
<td><strong>Indicators at follow-up I ‡</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of patients</td>
<td>74</td>
<td>231</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroke type: ICH or SAH</td>
<td>13 (18)</td>
<td>19 (8)</td>
<td>0.05</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BI 0–90</td>
<td>37 (50)</td>
<td>51 (22)</td>
<td>&lt;0.001</td>
<td>0.01</td>
<td>3.360</td>
<td>1.653–6.828</td>
</tr>
<tr>
<td>Living in nursing home</td>
<td>20 (27)</td>
<td>18 (8)</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Living alone</td>
<td>39 (53)</td>
<td>83 (36)</td>
<td>0.01</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical activity, mean/median</td>
<td>2/3</td>
<td>2.7/3.0</td>
<td>0.001</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social participation, mean/median</td>
<td>2.4/2.0</td>
<td>3.4/3.0</td>
<td>0.001</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eating difficulties§</td>
<td>49 (66)</td>
<td>21 (9)</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>0.048</td>
<td>0.24–0.99</td>
</tr>
<tr>
<td>Albumin &lt;34 g/L</td>
<td>14 (19)</td>
<td>12 (5)</td>
<td>0.001</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prealbumin &lt;0.20 g/L</td>
<td>11 (15)</td>
<td>14 (6)</td>
<td>0.03</td>
<td>0.04</td>
<td>0.325</td>
<td>0.114–0.929</td>
</tr>
<tr>
<td><strong>Indicators at follow-up II ¶</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of patients</td>
<td>79</td>
<td>226</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroke type: ICH or SAH</td>
<td>16 (20)</td>
<td>16 (7)</td>
<td>0.003</td>
<td>0.04</td>
<td>2.466</td>
<td>1.033–5.890</td>
</tr>
<tr>
<td>GDS-20 ≥6</td>
<td>42 (53)</td>
<td>76 (34)</td>
<td>0.004</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eating difficulties§</td>
<td>36 (46)</td>
<td>22 (10)</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>0.129</td>
<td>0.065–0.255</td>
</tr>
<tr>
<td>Albumin &lt;34 g/L</td>
<td>19 (24)</td>
<td>9 (4)</td>
<td>0.01</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prealbumin &lt;0.20 g/L</td>
<td>22 (28)</td>
<td>19 (8)</td>
<td>0.02</td>
<td>0.03</td>
<td>0.377</td>
<td>0.155–0.919</td>
</tr>
</tbody>
</table>

*OR indicates odds ratio; ICH, intracerebral hemorrhage; and SAH, subarachnoid hemorrhage.

†Analysis to find baseline predictors of weight loss >3 kg at follow-up I. Nonsignificant variables at baseline were sex, living alone, albumin value, prealbumin value, living situation, physical activity, social participation, BMI <20.5 kg/m², and age.

‡Weight loss between baseline and follow-up I. Nonsignificant variables were sex, BMI <20.5 kg/m², and age.

§Eating difficulties were registered as present for patients who indicated that their food intake had declined over the past 3 months due to loss of appetite, digestive problems, or chewing or swallowing difficulties.

¶Weight loss between baseline and follow-up II. Nonsignificant variables were sex, living alone, living in a nursing home, BI, HbA1c, diabetes, age, BMI <20.5 kg/m², physical activity, and social participation.
ties showed a highly significant relation with weight loss >3 kg at both follow-ups, which further strengthens the assumption that weight loss >3 kg is associated with malnutrition.

We found an association between possible depression (GDS-20 score) and weight loss. Depression has not been diagnosed with diabetes. A lack of attention to nutritional status was linked to reduced appetite and depression,

In our study, elevated HbA1c was an independent baseline predictor of weight loss at follow-up I. There may be a lack of attention to impaired glucose tolerance in stroke patients with the risk of adverse outcomes has been reported in other studies. In another study, hyperglycemia plus a raised HbA1c concentration on admission predicted unrecognized diabetes mellitus with a sensitivity of 86% and a specificity of 94%. Impaired glucose tolerance affects metabolism, which may be related to nutritional problems, and has been found in 50% of 72 nondiabetic patients with a recent transient ischemic attack or nondisabling ischemic stroke. It could therefore be suggested that simple measurement of HbA1c and glucose levels should be used as screening tests in acute as well as in postacute stroke care.

Our results at follow-up I seem to confirm the findings of another study that eating, feeding, and nutritional status of stroke patients may be inadequately treated and cared for in nursing homes. However, at follow-up II, we did not find any evidence that weight loss was related to living in a nursing home, indicating that the situation may improve with time. We agree with recently published reports that clinical nutrition must be considered a part of medical treatment and nursing care and not only a service offered to patients in hospitals or residents in nursing homes. An intervention program has been associated with meaningful changes in nutrition support practices and patient outcomes. A systematic observation program regarding eating and nutritional status has also highlighted the complexity of eating problems. These programs could be regarded as good examples of methods in secondary prevention of weight loss and subsequent malnutrition in poststroke care.

**Conclusion**

Weight loss >3 kg after stroke is common and appears to be an indicator of the need for closer observation of nutritional status. Monitoring of body weight may be useful, particularly among patients with severe stroke, eating difficulties, low prealbumin values, and impaired glucose metabolism.

**Acknowledgment**

We thank Håkan Lövkvist, statistician, for advice.

**Sources of Funding**

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**Disclosures**

None.

**References**


**Table 4. BMI of Stroke Patients With/Without Weight Loss >3 kg**

<table>
<thead>
<tr>
<th>Weight loss &gt;3 kg, n=74</th>
<th>Baseline %</th>
<th>Follow-Up I %</th>
<th>Follow-Up II %</th>
</tr>
</thead>
<tbody>
<tr>
<td>UW + NW</td>
<td>32.5</td>
<td>57</td>
<td>46</td>
</tr>
<tr>
<td>OW</td>
<td>67.5</td>
<td>43</td>
<td>54</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Weight loss &lt;3 kg or weight gain, n=231</th>
<th>Baseline %</th>
<th>Follow-Up I %</th>
<th>Follow-Up II %</th>
</tr>
</thead>
<tbody>
<tr>
<td>UW + NW</td>
<td>48.5</td>
<td>43</td>
<td>45.5</td>
</tr>
<tr>
<td>OW</td>
<td>51.5</td>
<td>57</td>
<td>54.5</td>
</tr>
</tbody>
</table>

**Conclusion**

Weight loss >3 kg after stroke is common and appears to be an indicator of the need for closer observation of nutritional status. Monitoring of body weight may be useful, particularly among patients with severe stroke, eating difficulties, low prealbumin values, and impaired glucose metabolism.

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None.

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