Measuring Longitudinally the Metabolic Demands of Stroke Patients

Resting Energy Expenditure Is Not Elevated

Hillel M. Finestone, MD, FRCPC; Linda S. Greene-Finestone, RD, MSc; Norine C. Foley, RD; M. Gail Woodbury, PhD

Background and Purpose—Little is known of the acute, subacute, and longer-term energy demands of stroke, information essential to appropriate clinical and nutritional management. The goals of this study were to (1) determine the resting energy expenditure (REE) of stroke patients from stroke onset to 3 months, (2) examine relations between stroke size, type, location, severity, and REE, and (3) evaluate whether estimation of REE from the Harris-Benedict equation (HB) requires the addition of a “stress factor” to capture the possible additional REE imposed by stroke.

Methods—The REE of new stroke patients was measured prospectively at hospital admission and on days 7, 11, 14, 21, and 90 by indirect calorimetry. Stroke patients’ REEs (Kcal/d) over time and REEs as a percentage of HB were compared with control subjects’ single measurements.

Results—Mean REE and %HB of stroke patients ranged from 1521±290 to 1663±268 Kcal/d and from 107±14.9 to 114±12.9 %HB, respectively. Mean measurements of control subjects were 1665±265 Kcal/d and 112.9±11.4 %HB (NS). REE was not associated with stroke characteristics (NS). Changes in REE measured longitudinally were not clinically meaningful (4 to 62 Kcal/d) though statistically significant (P=0.004).

Conclusions—The REEs of stroke patients and controls were both ≈10% higher than those predicted by HB. No hypermetabolic response pattern of energy expenditure was evident after stroke. REE did not vary with stroke characteristics, although confirmation with larger subgroups is required. (Stroke. 2003;34:502-507.)

Key Words: cerebrovascular circulation ■ stroke ■ diet ■ energy metabolism ■ rehabilitation

Elevated metabolic rates of 130% to >200% of predicted values have been well described for some disease states, including burns,1 sepsis,2 trauma,2 and head injury3 and are reflective of increased oxygen consumption associated with injury severity. Hypermetabolism has been defined as an increase in metabolic rate above that which is predicted from equations accounting for age, sex, height, and weight.4 Metabolic rate typically peaks within several days after injury and gradually declines to noninjury levels in the absence of infections or complication. However, it is unclear whether stroke results in the same metabolic perturbations found after other injuries. This information is important both to the understanding of the metabolic demands of stroke and to the guiding of nutritional interventions in stroke patients, particularly those who are enterally fed. There is evidence that those receiving enteral feedings in acute long-term-care facilities are routinely underfed or overfed.5 Determining the energy expenditure of stroke patients more precisely will facilitate the provision of appropriate energy (caloric) levels, thus avoiding the dangers of underfeeding or overfeeding and ultimately helping to improve stroke outcome.

There is a paucity of literature describing the energy expenditure patterns of patients after stroke. Only 1 small study, comparing energy expenditure in 11 subjects twice during a 14-day period, found that patients were not hypermetabolic in the acute recovery period after stroke.6 There are no longer-term data on energy expenditure after stroke.

The Harris-Benedict equations (HBs) are widely used in clinical practice and research to estimate resting energy expenditure (REE), defined as the amount of energy (Kcal/d) needed to sustain all life processes under resting, thermoneutral conditions.7 REE typically represents 75% to 90% of total daily energy expenditure. The remainder of the total energy expenditure is accounted for by thermogenesis from nutritional intake, shivering/nonshivering thermogenesis, and physical activity.8–10 Significant deviations from the estimated REE, as predicted by the HBs, are assumed to result from the effects of a particular injury. The additional energy

Received June 27, 2002; final revision received August 27, 2002; accepted September 9, 2002.

From the Department of Physical Medicine and Rehabilitation (H.M.F.), Elizabeth Bruyère Health Centre, University of Ottawa, Ottawa; the Department of Epidemiology and Biostatistics (L.S.G.-F., M.G.W.), University of Western Ontario, London; the Department of Physical Medicine and Rehabilitation (N.C.F.), St. Joseph’s Health Care London, Parkwood Hospital, London; and the Program in Rehabilitation and Geriatric Care (M.G.W.), Lawson Health Research Institute, London, Canada.

Correspondence to Hillel Finestone, MD, Department of Physical Medicine and Rehabilitation, Elizabeth Bruyère Health Centre, 43 Bruyère St, Ottawa ON K1N 5C8, Canada. E-mail hfinesto@scohls.on.ca

© 2003 American Heart Association, Inc.

Stroke is available at http://www.strokeaha.org

DOI: 10.1161/01.STR.0000053031.12332.FB
The demands imposed by hypermetabolism associated with various diseases and injuries has led to the adjustment of the equation with appropriate “stress factors.” The factors enable more accurate predictions of total energy requirements when the HBs are used. A “stress factor,” however, does not exist for stroke, and the assessment of stroke patients’ energy requirements usually assumes that there is an elevation of REE as a consequence of stroke.

The goals of the study were to (1) describe the energy expenditure of the stroke patient during the 3-month period after stroke; (2) examine relations between stroke size, type, location, severity, and energy expenditure; and (3) develop a “stroke stress factor” appropriate for use with the HBs.

Materials and Methods

This project received ethical approval from the Institutional Review Board for Health Sciences Research Involving Human Subjects. Informed consent was obtained from all participants or their designates. From August 1996 to August 1999, all patients presenting to the neurological/neurosurgical unit of the London Health Sciences Centre with symptoms of an acute cerebral vascular accident were screened for entrance into the study. Subjects included those who had sustained their first ischemic or nonsurgical hemorrhagic stroke, with onset of symptoms within 5 days of admission and who were normally nourished according to subjective global assessment criteria. Patients were not considered potential candidates if they (1) had a subarachnoid hemorrhage; (2) had major systemic injuries, including active malignancies; (3) were comatose; (4) had minimal neurological deficits or experienced a transient ischemic attack; or (5) were participating in another trial. Major systemic injuries were defined to be sepsis, carcinoma with or without chemotherapy, any medical illness (eg, uremia) requiring dialysis, congestive heart failure with pulmonary edema, or respiratory disorders, such as severe asthma or pneumonia, which were subjectively determined by the investigators to possibly raise REE. Coma was considered to be an unresponsive state in which the patient usually required an intensive care unit admission. Mild neurological deficits were subjectively defined as minimal cognitive and physical deficits for which staff estimated that the length of stay would not exceed 2 weeks on the acute and/or rehabilitation services. There were 688 stroke patients admitted within the study period. Of these, ~50% were ineligible to enter the study based on the exclusion criteria. Of the remaining patients, 91 agreed to participate, 21 refused to participate, and the remainder were not invited to participate because of an anticipated length of stay <14 days and/or distance from home >75 km, which would normally not allow for follow-up visits after discharge. The patients studied represented a fairly homogeneous group: those admitted for their first ischemic or hemorrhagic stroke, being normally nourished, with no major medical complications, and with an expected length of stay of at least 14 days. Ten control subjects of similar age range, volunteers from a larger study group investigating asymptomatic carotid artery disease for a 5-year period, were also recruited.

On admission, stroke location (right or left hemisphere, basal ganglia/thalamus, cerebellum or brain stem), stroke size (lesion <0.5 cm, 0.5 to <1.5 cm, 1.5 cm to <½ of the vascular territory, ½ to 2/3 of the vascular territory, and >2/3 of the vascular territory), and stroke type (ischemic or hemorrhagic) were confirmed by either MRI or CT imaging. Stroke severity was assessed on day 5 after stroke onset by use of the Canadian Neurological Scale performed by 1 of 3 Neurology Fellows. The Canadian Neurological Scale is a 10-point ordinal scale designed to assess mentation and motor function. The values were collapsed into 3 categories: 0 to 3, 3.5 to 7, and >7.5, representing severe, moderate, and mild strokes, respectively. Additional information obtained from the patient’s hospital chart included age, sex, diet type, and elevations in temperature (>38.0°C), as well as urinary tract and pulmonary infections.

Energy expenditure was measured via indirect calorimetry, a noninvasive test that uses in vivo gas (O₂ and CO₂) exchange to determine the REE. A portable metabolic cart with an open canopy system (Deltatrac, SensorMedics) was used. All measurements were performed on non–ventilator-dependent patients who were not receiving oxygen therapy. Patients received either continuous enteral nutrition or an appropriate oral diet. Measurements were performed at least 2 hours after meal consumption (enteral feedings were not discontinued for metabolic cart measurements) and after the patient had been recumbent for at least 1 hour. Measurements were discarded if they failed to produce a steady state (defined as an average minute VO₂ and CO₂ exchange of <10% during the testing period, typically 15 minutes). The O₂ and CO₂ sensors were calibrated before each test, and the accuracy of the instrument was assessed biannually. Measurements were taken while the stroke subjects were inpatients on the neurology and/or rehabilitation services and subsequently while outpatients. Control subjects were measured as outpatients.

REE measurements were performed within 5 days of onset of symptoms and on days 7, 11, 14, 21 (±1 day), and 90 (±10 days). Fourteen week data were collected in 12 patients who were transferred to the rehabilitation service at the same institution. REE measurements were compared with predicted values of energy expenditure, obtained by using the HB and expressed as a percentage. The HBs use a patient’s sex, height, weight, and age to estimate basal energy expenditure expected under normal, nonillness situations. The following are the HBs for the predicted REE of males and females: male: 66.5 + (13.8 × weight (kg)] + [6.8 × height (cm)] – [7.8 × age (years)], R² = 0.75; female: 655 + [9.6 × weight (kg)] + [1.8 × height (cm)] – [4.7 × age (years)], R² = 0.53. Patients’ weights were measured on admission to hospital and on days 14, 21, and 90 with either standing balance-beam scales or bed scales. Weights on days 7 and 11 were estimated by averaging admission and day-14 weights.

The energy expenditure of obese patients is more difficult to predict. For obese patients, defined as >125% of ideal body weight (IBW), an adjusted body weight was used to calculate the predicted energy expenditure by using the formula adjusted body weight (kg) = [(actual body weight − IBW) × 0.25] + IBW. The use of unadjusted weight in obese patients may result in an overestimation of REE, because adipose tissue is less metabolically active. Self-reported height was used. Control subjects were studied as outpatients. Information regarding recent infections was obtained. Subjects were weighed, their estimated energy requirements were calculated using the HBs equations and a single metabolic cart measurement was taken to determine REE.

To determine whether REE changed during the study period, an unbalanced repeated-measures ANOVA that allowed for missing values was used. This procedure does not require all subjects to have measurements performed at each of the testing intervals. All available data can be used, and subjects with missing data do not have to be excluded from the analysis. Confidence intervals (95%) about the REE and %HB means were calculated to denote the interval likely to contain the true value. ANOVA and t tests were used to examine differences in mean REE in relation to stroke location, type, severity, and size. The need for a “stress factor” was based on a significant deviation (>10%) of the REE results from those predicted by the HBs and from the controls, as well as a comparison of the control subjects’ actual and predicted energy expenditure. An α < 0.05 was considered statistically significant. SAS, release 8.01 and SPSS, release 10.0 were used for all statistical analyses. Results are reported as mean ± SD.

Results

Ninety-one patients entered the study, 63 men (69%) and 28 women (31%), with a mean age of 69.0 ± 11.3 years. The mean age of the control subjects was 72 ± 6 years; 7 subjects were
men and 3 were women. Admission weight and age were similar in the control and stroke subjects (NS). Seventy-six patients (84%) had sustained ischemic strokes and 15 (16%), hemorrhagic strokes. Stroke location included 22 (24%) right hemisphere, 33 (36%) left hemisphere, 15 (17%) basal ganglia/thalamus, 9 (10%) cerebellar, and 12 (13%) brain stem. Severity of stroke was estimated to be mild in 43 patients (47%), moderate in 34 (37%), and severe in 13 (14%).

Valid energy expenditure measurements were obtained for 62 stroke patients on admission; for 65 on day 7; for 54 on day 11; for 51 on day 14; and for 47 on day 90. Patients were studied a minimum of once (n/H1100511) to a maximum of six testing times (n/H1100519). Measurements were available for a minimum of 3 evaluation times for 73.3% of subjects. Failure to produce a steady-state reading occurred in 32 (5.9%) of REE measurements. Mean REE and %HB of the control subjects and the stroke group at each of the testing intervals are presented in Table 1. Mean REE of the stroke patients ranged from a low of 1521 ± 290 Kcal/d at day 21 to a high of 1663 ± 268 Kcal/d at day 90. Mean %HB was 112.9 ± 11.4% for the controls. There were no differences between mean REE or %HB measurements and control values at any of the evaluation times (Table 1). From the unbalanced repeated-measures ANOVA, differences between mean REE levels at the 6 testing times were evident but small, ranging from 4 to 62 Kcal/d between groups (P=0.004; see the Figure). No hypermetabolic response pattern of energy expenditure was evident.

There was great variability among individuals at each of the testing times, as indicated by the large standard deviations and wide 95% confidence intervals of both the control and stroke groups (Table 1). Variability in the stroke group was greater from admission to day 11 than from days 14 to 90. Forty-three patients (47%) displayed energy expenditure values >115%HB on at least 1 occasion. There were no differences in mean REE at any time in relation to stroke location, stroke size, or stroke severity (Tables 2 through 4; NS). In terms of complications, 13 patients developed an elevated temperature, urinary tract infection, or pulmonary infection within the first 21 days of stroke. Six of these patients (46%) experienced an elevation of their metabolic rate in excess of 15% on at least 1 occasion during the same period. The range of REE

### Table 1. Measured Resting Energy Expenditure and Percentage of Harris-Benedict Equation for Stroke Patients and Controls

<table>
<thead>
<tr>
<th>Time (n observations present/missing)</th>
<th>REE (Kcal/24 hrs±SD)</th>
<th>REE (95% CI)</th>
<th>% of HB±SD (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Admission: stroke (n=62/29)</td>
<td>1603±346*</td>
<td>1515–1705</td>
<td>109.8±15.4*</td>
</tr>
<tr>
<td>Day 7: stroke (n=65/26)</td>
<td>1618±352*</td>
<td>1531–1705</td>
<td>112.6±16.4*</td>
</tr>
<tr>
<td>Day 11: stroke (n=54/37)</td>
<td>1647±355*</td>
<td>1550–1744</td>
<td>111.3±17.7*</td>
</tr>
<tr>
<td>Day 14: stroke (n=51/40)</td>
<td>1535±286*</td>
<td>1455–1615</td>
<td>107.2±13.3*</td>
</tr>
<tr>
<td>Day 21: stroke (n=63/28)</td>
<td>1521±290*</td>
<td>1448–1594</td>
<td>106.6±14.9*</td>
</tr>
<tr>
<td>Day 90: stroke (n=47/44)</td>
<td>1663±268*</td>
<td>1584–1740</td>
<td>113.7±12.9*</td>
</tr>
<tr>
<td>Control (n=10/0)</td>
<td>1665±265</td>
<td>1497–1833</td>
<td>112.9±11.4</td>
</tr>
</tbody>
</table>

*All values compared to controls are not significant.
values for patients with infections and fevers was 90% to 150% of predicted values.

Discussion
The aim of the current study was to determine whether a pattern of metabolic stress was evident. For the hypermetabolic, hypercatabolic response associated with major stress, large variations in REE (30% to >100% increases) have been observed.1-3 This study had the statistical power to exclude a similarly substantial and consequential effect.17 The results of this study indicate that patients do not demonstrate evidence of a hypermetabolic response to stroke. The smallest clinically important difference, in terms of offering appropriate nutrition support, is estimated to be a 10% to 15% change in REE. At this level, the number of patients and controls would not be sufficiently large to avoid a type II error. However because the maximum REE difference observed between patients and controls was 8.6% (Table 1), a value below the level of clinical meaningfulness, an insufficiency of power at this level is not problematic. When longitudinal change in REE was investigated with the unbalanced repeated-measures ANOVA with all subjects, differences in REE between testing times were small. Similarly, these differences were not deemed to be clinically significant, particularly in view of the large individual variation in measurements of the controls and stroke patients (the Figure).

Weekes and Elia15 demonstrated that mean REE was 1252±192 Kcal/d (n=11) 1 to 3 days after stroke and 1219±242 Kcal/d (n=11) 10 to 14 days after stroke (NS). Although these REE measurements are lower than those of the present study, the variation may largely be explained by (1) differences in weight between the stroke subjects in the 2 studies (58.2±12.1 vs 77.7±16.2 kg, respectively), likely attributable to differences in the male/female ratio (53:47 vs 70:30, respectively) and (2) their measurement after an overnight fast vs 2-hour NPO or continuous enteral feedings in this study. However, after controlling for weight, height, age, and sex in the HBs, the initial %HB between the 2 studies was very similar (107.0±6.9% vs 109.8±15.4 in this study), thereby corroborating previous results.

In some disease states, there is evidence to support a positive correlation between severity of injury and elevations of metabolic rate.18,19 Increases in both metabolic rate and catabolism after injury have largely been attributed to the effects of the acute-phase response, mediated through the effects of cytokines and counterregulatory hormones.20 Because elevations of peripheral plasma catecholamines, corti-

<table>
<thead>
<tr>
<th>Location/Type</th>
<th>Acute Period (Admission–Day 7), n</th>
<th>Subacute Period (Days 11–21), n</th>
<th>Follow-Up Period (Day 90), n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right hemisphere</td>
<td>1581±257 (20)</td>
<td>1557±256 (18)</td>
<td>1559±201 (9)</td>
</tr>
<tr>
<td>Left hemisphere</td>
<td>1546±240 (29)</td>
<td>1498±275 (28)</td>
<td>1608±325 (17)</td>
</tr>
<tr>
<td>Basal ganglia/thalamus</td>
<td>1595±441 (12)</td>
<td>1667±426 (12)</td>
<td>1730±218 (8)</td>
</tr>
<tr>
<td>Cerebellum</td>
<td>1839±483 (8)</td>
<td>1723±350 (6)</td>
<td>1772±157 (5)</td>
</tr>
<tr>
<td>Brain stem</td>
<td>1762±495 (10)</td>
<td>1587±296 (10)</td>
<td>1761±279 (8)</td>
</tr>
<tr>
<td>P value</td>
<td>0.274</td>
<td>0.585</td>
<td>0.356</td>
</tr>
<tr>
<td>Infarct</td>
<td>1625±336 (68)</td>
<td>1556±282 (63)</td>
<td>1663±273 (41)</td>
</tr>
<tr>
<td>Hemorrhage</td>
<td>1586±456 (11)</td>
<td>1651±441 (11)</td>
<td>1665±260 (6)</td>
</tr>
<tr>
<td>P value</td>
<td>0.460</td>
<td>0.078</td>
<td>0.984</td>
</tr>
<tr>
<td>Total observations present/missing</td>
<td>79/11</td>
<td>74/17</td>
<td>47/44</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Size of Lesion</th>
<th>Acute Period (Admission–Day 7), n</th>
<th>Subacute Period (Days 11–21), n</th>
<th>Follow-Up Period (Day 90), n</th>
</tr>
</thead>
<tbody>
<tr>
<td>No lesion on CT</td>
<td>1677±450 (14)</td>
<td>1581±383 (11)</td>
<td>1793±181 (9)</td>
</tr>
<tr>
<td>&lt;0.5 cm</td>
<td>1585±671 (8)</td>
<td>1496±348 (8)</td>
<td>1542±224 (6)</td>
</tr>
<tr>
<td>0.5 to &lt;1.5 cm</td>
<td>1575±219 (14)</td>
<td>1597±271 (11)</td>
<td>1727±388 (8)</td>
</tr>
<tr>
<td>1.5 to &lt;½</td>
<td>1658±323 (18)</td>
<td>1582±358 (20)</td>
<td>1646±220 (13)</td>
</tr>
<tr>
<td>½ to &lt;¾</td>
<td>1582±196 (14)</td>
<td>1497±200 (13)</td>
<td>1500±306 (7)</td>
</tr>
<tr>
<td>&gt;¾</td>
<td>1541±318 (7)</td>
<td>1582±306 (8)</td>
<td>1860 (no SD) (1)</td>
</tr>
<tr>
<td>P value</td>
<td>0.279</td>
<td>0.242</td>
<td>0.196</td>
</tr>
<tr>
<td>Total observations present/missing</td>
<td>75/16</td>
<td>71/20</td>
<td>44/47</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CNS Scores</th>
<th>Acute Period (Admission–Day 7), n</th>
<th>Subacute Period (Days 11–21), n</th>
<th>Follow-Up Period (Day 90), n</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 to 3</td>
<td>1555±332 (10)</td>
<td>1524±302 (12)</td>
<td>1632±309 (5)</td>
</tr>
<tr>
<td>3.5 to 7</td>
<td>1640±315 (30)</td>
<td>1594±321 (28)</td>
<td>1624±305 (18)</td>
</tr>
<tr>
<td>7.5 to 10</td>
<td>1615±391 (38)</td>
<td>1560±309 (33)</td>
<td>1699±236 (24)</td>
</tr>
<tr>
<td>P value</td>
<td>0.343</td>
<td>0.583</td>
<td>0.655</td>
</tr>
<tr>
<td>Total observations present/missing</td>
<td>78/13</td>
<td>73/18</td>
<td>47/44</td>
</tr>
</tbody>
</table>
sol, glucagon, interleukin-6, interleukin-IRA, and acute-phase proteins have been well described after stroke, a systemic inflammatory response resulting in elevations of metabolic rate would be expected.21–27 Although an elevation in metabolic rate associated with stroke size, severity, or type did not occur in this study, this finding should be verified in larger within-subgroup samples, because there were not enough observations in certain groups to provide sufficient power to find intergroup differences of 10% to 15%. For these analyses (Tables 2 through 4), our results should be considered exploratory.

The REE of stroke patients during the study period was \( \approx 10\% \) higher than the values predicted by the HBs. The lack of a clinically significant change in REE over time and the absence of differences between study subjects and controls suggest that this 10% in measured REE above the HB-predicted REE reflects population or behavioral differences rather than a “stress factor” specific to stroke. The REE of the outpatient controls was 13% higher than the values predicted by the HBs. Potentially, this may be reflective of their greater muscle mass and more strenuous physical exertion before their hour-long recumbent position before testing. Alternatively, controls with some degree of peripheral and/or cerebrovascular disease may have had somewhat higher REEs than healthy controls.

There is evidence to suggest that there is substantial individual variation in REE in both health and disease states. Roza and Shizgal28 examined the original Harris and Benedict data in addition to a larger data set published by Benedict, which incorporated normal subjects with a wider age range. The HBs estimated the REE of a normally nourished subject with a precision of 14%. Other studies examining the REE of normal volunteers also describe variations of 12.5% to 23.2% over 2 days and 2 years, respectively.29,30 In this study, there were large individual variations in REE at each of the testing times. Individual patients also exhibited a variation in REE from 1 testing time to the next. Almost half of all patients displayed energy expenditure values \( > 15\% \) above those predicted by the HBs on at least 1 occasion. No specific pattern suggesting a direct association between infection, elevated temperature, and energy expenditure was observed, but it is difficult to draw conclusions because so few were affected.

The HBs explain 53% to 75% of the variability in REE. Approximately 75% of this explained variation is accounted for by body weight.31 Whereas it has been postulated that lean body mass is the entity most predictive of basal energy expenditure, there is a covariance between total body weight, lean body mass, and basal energy expenditure.32 Potentially, some of the “risk factors” for stroke, such as diabetes, coronary artery disease, and hypertension,33 or certain sedatives or other drugs may also contribute to variation in REE. These issues should be investigated in future studies. It is possible that after accounting for potentially influential factors such as these, longitudinal differences in REE may become evident.

Some limitations are apparent. Historically, examinations of energy expenditure are confined to more controlled environments to help reduce potential sources of variability in measurement. Critically ill, sedated patients and ventilator-dependent or healthy individuals who are studied in a metabolic chamber, where continuous readings for a 24-hour period can be obtained, are most often studied. In this clinical setting, patients receiving enteral nutrition would be expected to have somewhat higher metabolic rates owing to diet-induced thermogenesis relative to those patients who were fasted. The metabolic cost of digestion and absorption of nutrients has been reported to be \( \approx 10\% \).34 This may have contributed to some of the individual variation in REE in the stroke and control groups. Missed or failed measurements of REE were related to patients’ early discharge from hospital, failure to produce a steady-state reading, or the need for oxygen therapy in a small number of patients in the acute period after stroke. Although most local patients who were discharged from hospital before day 21 returned to complete their testing, this was not always possible, and consequently, not all patients were tested at all 6 intervals. To statistically compensate for this, an ANOVA that allowed for missing values was used for the longitudinal analysis.

A strength of this study is that it prospectively followed stroke patients to determine their energy requirements and expenditures after acute stroke, during the subacute rehabilitation phase, and at the 3-month poststroke stage. To the best of our knowledge, REE determination has never before been performed beyond 2 weeks after stroke, with as frequent serial measurements during the acute period (when the metabolic stress response is most evident) and with as large a cohort.

Conclusions

This analysis of energy expenditure suggests that stroke patients are not hypermetabolic within the 90 days after their stroke event. It is possible that future studies, accounting for other influential factors, may detect differences in REE over time. Subgroup analysis of stroke patients did not detect differences in mean energy expenditure in relation to stroke characteristics, but these observations should be confirmed with larger groups. The HBs may underpredict REE by an average of 10%, but individual REE variation is high. If an accurate assessment of caloric requirements is required for an individual patient, indirect calorimetry is indicated.

Acknowledgment

This study was funded through grants NA3052 and T3602 awarded by the Heart and Stroke Foundation of Ontario.

References


Downloaded from http://stroke.ahajournals.org/ by guest on January 10, 2018


Measuring Longitudinally the Metabolic Demands of Stroke Patients: Resting Energy Expenditure Is Not Elevated
Hillel M. Finestone, Linda S. Greene-Finestone, Norine C. Foley and M. Gail Woodbury

Stroke. 2003;34:502-507; originally published online January 23, 2003;
doi: 10.1161/01.STR.000053031.12332.FB

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/34/2/502

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/