Title: ASSESSMENT OF ENERGY EXPENDITURE AND ENERGY INTAKE IN CRITICALLY ILL PATIENTS

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Results: There was a predominance of normal (65%) and hypometabolism (23%). The measured EE of the patients ranged from 1131 to 2320 kcal.d-1, with a mean of 1515±268 kcal.d-1. There was a 53% rate of inadequate nutritional intake, with 23% of the patients being underfed and 30% being overfed. Most patients presented a negative nitrogen balance (11/14), with a mean of -5±7g.d-1 and a positive and highly significant association was detected between the energy intake/measured EE ratio and the nitrogen balance.

Conclusion: In view of the marked variations in EE estimates compared to measured EE, the recommendation is to use indirect calorimetry, with caution in the use of correction factors in order not to overestimate the energy requirements, and to monitor systematically the administration of the prescribed nutritional therapy.
ASSESSMENT OF ENERGY EXPENDITURE AND ENERGY INTAKE IN CRITICALLY ILL PATIENTS

Energy Expenditure and Intake in Critically Ill Patients

Camila Cremonezi Japur¹; Jacqueline Pontes Monteiro²; Júlio Sérgio Marchini³; Anibal Basile Filho⁴

¹Master’s degree, Department of Surgery and Anatomy; ²Assistent Professor, Department of Puericulture and Pediatrics; ³Full Professor, Department of Internal Medicine; ⁴Associate Professor, Department of Surgery and Anatomy.

Faculty of Medicine of Ribeirão Preto - University of São Paulo, Ribeirão Preto, SP, Brazil.

Number of words in the full manuscript: 5398
Number of figures: 2
Number of tables: 3

Corresponding author: Camila Cremonezi Japur
Phone: 55 16 3602-3096
Fax: 55 16 3602-3098
E-mail: camila@fmrp.usp.br; camijapur@yahoo.com.br
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Key Words: Energy expenditure, indirect calorimetry, energy intake, nitrogen balance, critically ill.
INTRODUCTION

The estimate of the energy requirements of healthy persons is a challenge for professionals since differences in real energy expenditure (EE) may occur even among persons of the same gender, age, weight and height due to various genetic and environmental characteristics (ethnic origin, body composition, diet, physical activity, environmental temperature, and altitude)\(^1\). For critically ill patients these difficulties are multiplies since, in addition to all of these factors that alter the energy metabolism, there is the influence of the disease, of the metabolic alterations associated with the critical condition, and the effects of treatment, which may elevate or reduce oxygen consumption (\(\text{Vo}_2\)) and carbon dioxide production (\(\text{Vco}_2\)), affecting EE \(^2,3\).

A method commonly employed to estimate the energy requirements is the use of predictive equations in combination with correction factors. However, these corrections may overestimate the value by as much as 50\% \(^4,5\) and cause overfeeding, which leads to respiratory and metabolic problems \(^6,7\), or may underestimate the value, leading to patient underfeeding \(^4,8\).

The objectives of the present study were to measure the EE of critically ill patients, to analyze the correction factor of the relation between real and estimated expenditure, and to evaluate the adequacy of energy intake, associating it with the nitrogen balance.
SUBJECTS AND METHODS

Subjects

A cross-sectional study was conducted in an Intensive Care Unit (ICU) of a University Teaching Hospital. We evaluated 17 critically ill adult patients who were mechanically ventilated through an orotracheal tube and who were in a hemodynamically stable condition. The local institutional review board approved the study protocol, and familial/parental written informed consent was obtained before the subjects entered the study.

Clinical data included sex, age, weight, height, diagnosis, therapy, days on mechanical ventilation and in the ICU, route of nutritional support administration, and energy intake (EI). Severity of illness was assessed by the APACHE II score [9].

Current patient weight was measured with a portable electronic scale (Slingscale 2002, Instrucom/Hill-Rom series, Hillenbrand Industries, US) and height was measured with a stadiometer to the nearest 0.1 cm.

Patients with at least one of the following conditions were excluded: spontaneous ventilation, absence of a bladder catheter, diuresis of <50 mL.h⁻¹, age >85 and <15 years, need for inspired air oxygen fraction (FiO₂) >60 %, mean arterial pressure <50 mmHg, heart rate <50 or >140 beats per minute, presence of a bronchopleural air fistula, irreversible circulatory shock, and brain death.
Energy Expenditure - Indirect Calorimetry

$VO_2$ and $V_{CO_2}$ were determined by indirect calorimetry (IC) over duplicate periods of 45 minutes separated by a 30 minute interval using a portable calorimeter (DELTRATAC II Metabolic Monitor - Datex Ohmeda, Finland) coupled to a microprocessed respirator. The apparatus was heated for 30 minutes before the measurements (Figure 1). Automatic pressure calibration was adjusted manually to the local atmospheric pressure measured with a Torricelli barometer installed in the ICU and gases were calibrated automatically by means of a command in the apparatus itself with gas containing 95% oxygen and 5% carbon dioxide, according to manufacturer instructions. The measurements were made between 23 and 15 h, a period during which the physiological variables are close to the daily means, permitting a reliable use of the data generated by the calorimeter for the extrapolation of the EE measured to 24 h$^{[10]}$.

All patients lay in the supine position, were afebrile and were fed by the enteral, parenteral or mixed route at the time the measurements were made. The ventilatory parameters remained constant and the diagnostic and therapeutic procedures such as bronchial hygiene, bathing, venous puncture and exams were interrupted for at least 30 minutes before the respiratory gas measurements.

The equation of Weir$^{[11]}$, modified $[(3.941*VO_2)+(1.106*V_{CO_2})]*1.44$, was used to calculate EE, considering the interval of measurement of steady-state $VO_2$, $V_{CO_2}$ and respiratory quotient (RQ) data, characterized by a 5 minute interval during which the $VO_2$ and $V_{CO_2}$ values varied less than 10% and the RQ values varied less than 5%, excluding the values outside the physiological range$^{[12,13]}$. 
For standardization, it was established that the energy requirements corresponded to the measured EE without the addition of any correction factor\[^{[5,14]}\].

**Characterization of the metabolic study**

Basal energy expenditure (BEE) was estimated by the formula of Harris-Benedict, which uses weight in kg, height in cm and age in years, and gender\[^{[15]}\].

\[
\text{BEE}_{\text{men}} = 66.4730 + (13.7516 \times \text{weight}) + (5.0033 \times \text{height}) - (6.7550 \times \text{age})
\]

\[
\text{BEE}_{\text{women}} = 655.0955 + (9.5634 \times \text{weight}) + (1.8496 \times \text{height}) - (4.6756 \times \text{age})
\]

Hypermetabolism was considered to be present when the patient presented a measured EE of more than 30% of BEE and hypometabolism to be present when this value was 10% below BEE. Normometabolic patients are situated between these two extremes\[^{[16]}\].

**Energy intake, energy balance and characterization of feeding status**

The nutritional therapy route and the total value of infused enteral and parenteral nutrition were determined for the calculation of the energy balance (EB) by the difference between the total energy value infused and the EE value measured by the calorimeter.

Feeding status was determined according to the following criterion: underfeeding was considered to be present when the energy intake was \(\leq 90\%\) compared to the measured EE, normal feeding was considered to be present when the energy intake was 90 to 100% compared to measured EE, and overfeeding was
considered to be present when energy intake was more than 110% compared to measured EE\textsuperscript{[16]}

**Degree of protein catabolism and nitrogen balance**

Moderate catabolism was considered to be present when daily nitrogen excretion was 5 to 10g, increased catabolism was considered to be present when daily nitrogen excretion was 10.1 to 15g, and hypercatabolism was considered to be present when daily nitrogen excretion was more than 15.1g\textsuperscript{[17]}

Twenty-four h urine was collected for the calculation of nitrogen balance and the determination of total urinary nitrogen by the method of Kjeldahl\textsuperscript{[18]}. Three patients were excluded from this analysis due to losses of 24 h urine collection that did not permit the determination of urinary nitrogen.

The nitrogen balance (NB) was obtained as the difference between ingested nitrogen (IN) and excreted nitrogen (EN). IN corresponded to 16% of the total protein provision by the diet and EN to urinary nitrogen (UN) plus 2 g, relative to the estimate of imperceptible losses through the skin and feces \textsuperscript{[17]}. The results were classified as positive NB when the result was more than zero and negative when the result was less than zero.

**Statistical analysis**

The results are reported as descriptive measures of sample position and variability, expressed as mean and standard deviation.
The coefficient of variation of EE was calculated as the ratio between the standard deviation of the sample and the mean, multiplied by 100. The means were compared by the nonparametric Mann-Whitney test and the mean EE values for the groups receiving amines (n=1) or not (n=16) were compared by the t test for one sample.

In addition, we conducted a Bland-Altman analysis to determine the limits of agreement between measured and calculated EE. The limits of agreement between methods were defined as the mean difference ± 2 SD\cite{19}.

The correlations between the two variables were analyzed by the Spearman coefficient. The level of significance was set at 95% (p<0.05) in all analyses.
RESULTS

Critically ill patients (8 women and 9 men) with a mean age of 51±21 years were evaluated in the present study (n=17). The diagnoses were varied, including neurological, renal, respiratory, and gastroenterological diseases, with only one patient with cranioencephalic trauma. The APACHE II obtained ranged from 7 to 38 (mean: 22±8) and the mortality rate was 35% (Table 1). The median hospitalization time was 13 days, and the median time of mechanical ventilation was 14 days.

The measured EE of the patients ranged from 1131 to 2320 kcal.d\(^{-1}\) (and from 14 to 37 kcal. kg\(^{-1}\).d\(^{-1}\)) and the mean was 1515±268 kcal.d\(^{-1}\) (Table 2). Two patients were hypermetabolic, 4 were hypometabolic and 11 normometabolic. The patients who died did not present alterations of EE compared to those who survived (p=0.73).

All patients were using medications that might reduce EE (analgesics), but only one used vasoactive amines, which seem to increase EE. However, his EE was significantly lower compared to the mean for the rest of the group (p=0.001). It should be pointed out that this patient was receiving sedatives and muscle relaxants.

Ten patients were sedated (59%), and their mean EE (1599±306kcal.d\(^{-1}\)) did not differ significantly (p=0.16) from that of patients who were not receiving sedatives (1395±145kcal.d\(^{-1}\)). Only one of the sedated patients was not receiving a muscle relaxant. When the patients were divided into two groups, i.e., patients receiving (1541±157 kcal.d\(^{-1}\)) or not (1500±319kcal.d\(^{-1}\)) muscle relaxants, again no statistically significant difference was observed (p=0.35).

The mean EE of patients with a time of hospitalization of less (1467±123kcal.d\(^{-1}\)) or more (1548±337kcal.d\(^{-1}\)) than one week did not differ significantly (p=0.81).
When patients with and without pneumonia were compared, their mean EE of 1571±331kcal.d\(^{-1}\) and 1465±202kcal.d\(^{-1}\), respectively, did not differ significantly (p=0.88).

Bland-Altman analysis showed a mean of -30±248 kcal.d\(^{-1}\) for EE and BEE. Only one patient was outside the limits of agreement between the two methods (IC an Harris-Benedict), which were -525 to 465 kcal.d\(^{-1}\).

Feeding consisted of enteral nutritional (EN) or parenteral (PN) therapy, with 11 patients being on exclusive EN, 2 on exclusive PN, and 4 on mixed nutrition (EN+PN), and mean calorie intake was 1400±600 kcal.d\(^{-1}\). Regarding feeding status, 4 patients were underfed (23%), 8 were normally fed (47%) and 5 were overfed (30%), and 4 of the overfed patients were on exclusive PN and/or mixed nutrition.

Mean daily urinary nitrogen excretion was 13±5 g (range: 6 and 22 g. d\(^{-1}\)). Five patients presented moderate catabolism, five increased catabolism and four were hypercatabolic.

Most patients (11/14) presented a negative NB, with a mean of -5±7g.d\(^{-1}\) (range: -16 to 10 g.d\(^{-1}\)), as a consequence of the elevated urinary nitrogen excretion and/or low energy and protein intake. Mean protein intake was 0.9±0.4g.kg\(^{-1}\). Comparison of mean ingested (10±5g.d\(^{-1}\)) and excreted (15±5g.d\(^{-1}\)) nitrogen showed a significant difference (p=0.02).

There was no significant difference in mean EE measured by calorimetry between patients with a positive or negative NB (p= 0.45), nor were there any differences in energy intake (p=0.36), EB (p=0.45), or in the ratio between provision and EE (p=0.45) (Table 3).
There was a low correlation between NB and measured EE (kcal. kg$^{-1}$) ($r=0.079$; $p=0.78$), and between NB and energy intake (kcal. kg$^{-1}$) ($r=0.27$; $p=0.34$) even when the two patients (patients 9 and 10) with a low energy intake and a high and negative NB were excluded from the analysis. However, there was a highly significant positive association of the ratio between energy intake and measured EE with NB (Figure 2).
DISCUSSION

In the present study, the method used to determine the real energy requirements, indirect calorimetry, provided data about EE plus the thermal effect of food, since the patients were being fed at the time of measurement. The patients were bedridden and the effect of physical activity was limited to the activities of respiratory physiotherapy, changes in position in bed, weighing, and bathing provided by the professionals of the unit. Swinamer et al. reported that, among others, these activities influence the total expenditure by 1 to 4% during the 24 h\cite{20}. However, for the sake of standardization, in the present study we opted to consider the measured EE as the daily total.

Important variations in measured EE were detected among the patients, probably due to the different phases of disease, to the presence and magnitude of infection and inflammation, to the medication administered and to the biological variation itself (weight, height, age and sex) of the patients.

Two recent studies agreed in reporting the lack of influence of the type of injury on the measured EE. The first evaluated 18 critically ill afebrile patients who required mechanical ventilation, stratified by type of injury (clinical, surgical or traumatic) and showed no significant differences in the EE/BEE ratio\cite{21}, and the second evaluated 27 patients and detected mean EE/BEE ratios of 1.23, 1.26 and 1.18 for clinical, surgical and traumatized patients, respectively, with no significant difference between them (p=0.43)\cite{4}.

In the present study there was a predominance of normal metabolism and hypometabolism, in contrast to the classical statement that critically ill patients in
most cases are hypermetabolic. Modern mechanical ventilation instruments and drug therapies widely used in intensive care units such as sedatives, analgesics and muscle blockers seem to induce a reduction of metabolic and systemic stress, with a consequent reduction of energy requirements\textsuperscript{[2,4,22]}. However, statistical analysis of the present data did not reveal an influence of the use of medications on energy requirements, probably due to the fact that all patients were receiving many medications simultaneously, possibly contributing to a confusion of hypo- or hypermetabolic effects, and also due to the small sample size. Brandi et al. discussed the results of several studies in which 30 to 50\% of critically ill patients were normometabolic, 35 to 65\% were hypermetabolic and 15 to 20\% were hypometabolic\textsuperscript{[14]}. In the present study, 65\% (11/17) of the patients were normometabolic, 12\% (2/17) were hypermetabolic and 23\% (4/17) hypometabolic.

Hoffer stated that many critically ill patients have a measured EE that characterizes them as normometabolic\textsuperscript{[23]}. He pointed out the study by Zauner et al.\textsuperscript{[24]} who detected a mean of 23kcal. kg\textsuperscript{-1}. d\textsuperscript{-1} before and during the administration of parenteral nutrition, indicating that these patients, who are inactive, under moderate stress and continuously fed, have a mean measured EE close to the total daily EE.

The large number of normometabolic and hypometabolic patients may be explained by the predominance of a diagnosis of chronic neurological diseases that involve muscle paralysis, by the absence of fever and by the long time of hospitalization in the ICU, which probably did not reflect the acute phase of the disease. Raurich et al. studied the EE of patients with 48 to 96 h of mechanical
ventilation and observed that 63% of them were hypermetabolic\cite{21} but, in contrast to the present study, they used an EE of more than 115% of the BEE as the parameter for hypermetabolism.

The mean EE/BEE ratio was very close to 1.00, with a coefficient of variation of 17%, and with a variation of ± 10% in 10 of the 17 patients evaluated (59%). Schoeller pointed out that EE measurement is preferable because predictive equations show errors of more than 10% in one third of all patients\cite{25}.

Recent studies have recommended the use of EE with no correction factor \cite{5,26,27} or complemented with 10 to 20% at most in order to prevent metabolic overload due to excess nutrients \cite{20,28,29} and the use of 1.0 to 1.2 for BEE \cite{16,22,30}, of 1.3 \cite{4} and of up to 1.6 for a short period of nutritional therapy\cite{31}. This characterizes a wide variation of correction of EE measured by indirect calorimetry or estimated by predictive equations such as the Harris-Benedict one, with a consequent considerable difficulty in choosing the best factor to be used for this specific population.

In addition to the wide variation of the recommended correction factors, there is variation in the results of the studies that evaluated the EE/BEE ratio. In a study on hospitalized patients, Miles detected a variation of 0.94 to 1.30 in the EE/BEE ratio\cite{30} and in our study this variation was even greater, from 0.80 to 1.39. However, the mean ratio detected in most recent studies ranges from 1.0 to 1.2 \cite{5,21,30}, emphasizing the importance of caution about the use of higher correction factors such as those proposed by Long et al. in 1979\cite{32}, which are still used at some centers. Coletto et al. evaluated the use of these factors and observed overestimates in more than 50% of the patients studied\cite{5} and Miles also pointed out the importance of carefully
considering these factors in order to avoid inadequate energy intake, since in his study he detected a correction of BEE of only 13% on average\[^{30}\].

Bland-Altman analysis showed that all the plotted data, except for those referring to one patient, were within the limits of agreement, but we believe that the wide variation observed between the minimum and maximum values of these limits is clinically important. On this basis, we propose that there may be individual under- or overestimates when EE measurement by indirect calorimetry is replaced with prediction by the Harris-Benedict equation.

Four patients were found to be underfed (23%) and 5 to be overfed (30%). Among the latter, 4 were being fed by PN, a fact that highlights the need to prescribe this modality of nutritional therapy with caution.

In a study on 263 patients, McClave et al. observed that 42% of them were overfed, while 34% were underfed\[^{33}\]. These authors, however, used a different methodology since they considered EE with a correction factor of 1.1 (EI/EE x 1.1). Alberda et al. observed that 35% of 55 critically ill patients studied were underfed and 25% were overfed\[^{34}\]. When the percentages of under- and overfeeding of each study are summed, the rate of nutritional inadequacy obtained is 53% for our study and 76% and 60% for the other two, respectively.

This evidence highlights the difficulty in establishing an adequate energy intake due to both the variations existing in daily EE and the common occurrence of intolerance of nutritional therapy, metabolic complications associated with feeding and with the disease, fluid restriction, and the interruption of feeding for the
execution of procedures\textsuperscript{35}. The ideal would be to perform calorimetry measurements in all patients and to adapt and monitor the energy intake on a daily basis.

In a study on 180 ICU patients, Burzstein et al.\textsuperscript{36} detected a mean daily urinary nitrogen excretion of 13±8 g (range: 2 to 30 g), while Porter & Cohen suggested an excretion of 12 g. d\textsuperscript{-1} in most clinical cases\textsuperscript{37}. The urinary nitrogen values measured in the patients of the present study were similar to those reported in the literature, with a mean of 13±5 g. d\textsuperscript{-1} and a range of 6 to 22 g. d\textsuperscript{-1}.

The reduction of protein stores in these patients deserves special attention since a total protein mass loss of more than 25\% may cause patient death, whereas fluctuations in lipid and glucose stores do not have such relevant consequences\textsuperscript{6}. In critically ill patients protein catabolism persists regardless of the amount of protein in the diet until the etiologic factor is removed or the repair of the structure reaches an advanced phase of resolution\textsuperscript{38}.

There is no established treatment for the prevention of protein loss, but an appropriate nutritional therapy can attenuate it. It is important to point out that excessive protein administration does not reverse hypercatabolism and the protein surplus is oxidized and utilized as a source of energy\textsuperscript{6}.

The discussion of protein hypercatabolism in critically ill patients is based on the frequent neuroendocrine and metabolic alterations that affect them due to infection, trauma, or burns. The catabolic pattern of the patients studied demonstrated this alteration since none of them presented a urinary nitrogen excretion considered to be normal, with five of them presenting moderate catabolism, five increased catabolism, and four hypercatabolism. The inclusion of 2
patients with acute renal failure (ARF) (patients 3 and 11) and of 1 with chronic renal failure (patient 5) may have caused an underestimate of the data since these patients usually present a pattern typical of their disease of urinary nitrogen excretion altered by nitrogen retention in the organism. However, these three patients with ARF were receiving diuretic medication and presented diuresis of 2190, 4800 and 4240 ml. d⁻¹ and a nitrogen excretion of 9, 15 and 14 g. d⁻¹, respectively.

An excessive energy intake or a low EE are unable to maintain a positive or neutral NB, as observed in the present study on the basis of the absence of a correlation between NB and EI or NB and EE. However, a positive and highly significant association of the energy intake/measured EE ratio with NB was observed, supporting the importance of measuring EE and of the attempt to adapt the energy intake to the energy requirements in order to achieve a neutral or positive NB. This had also been stated by Hoffer [23], who emphasized that energy deficiency in relation to the requirements detected has a negative influence on NB. However, it is interesting to point out that, even when there is an adequate energy and protein provision, a negative NB is not always reversed, as observed in some patients studied here.

When the patients were divided into a group with a positive NB and a group with a negative NB, no significant difference was observed between groups regarding measured EE, energy balance, or energy intake/EE ratio. This lack of statistical significance should be considered with caution in view of the small number of patient in each group (n=3 for a positive NB and n=11 for a negative NB). However, when analyzing the mean results from a biological viewpoint, it can be seen that the group
with a positive NB presented a positive energy balance, 350 kcal more per day on average, and a 25% higher energy intake/EE ratio than observed in the group with a negative NB, with these differences between the two groups possibly determining the magnitude of the NB. In a study evaluating critically ill children, a significantly higher ratio between energy supply and expenditure was detected in patients with a positive NB than in patients with a negative NB\cite{39}, supporting the idea that an adequate energy intake may play a protective role against exacerbated protein catabolism.

The mean values of ingested (9±5g.d\(^{-1}\)) and excreted (15±5g.d\(^{-1}\)) nitrogen for the group differed significantly, suggesting that nitrogen intake in the form of protein was insufficient to compensate for the elevated excretion of body nitrogen. In a study on critically ill patients, Young\cite{36} detected a constantly negative nitrogen balance even with a high protein provision (1.5 g.kg\(^{-1}\).d\(^{-1}\)), indicating that protein intake by the patients was possibly insufficient (0.9±0.4 g.kg\(^{-1}\).d\(^{-1}\)) and contributed to the occurrence of a negative nitrogen balance in most of the patients studied (11/14), with a mean value of -5±7g.d\(^{-1}\).

**CONCLUSION**

The assessment of EE indicated a predominance of normal and hypometabolism. The use of indirect calorimetry is recommended, with caution about employing correction factors in order not to under- or overestimate the energy requirements. Constant monitoring of the administration of prescribed nutritional therapy is indispensable for the maintenance of the production of energy by exogenous sources and in an attempt to establish a neutral or positive nitrogen balance.


40. Young B, Ott L, Rapp R, Norton J. The patient with critical neurological
Patient selection

Preparation of urine collection flasks

**Figure 1. Study design.** (IC: indirect calorimetry; T₀: Beginning of urine collection; between T₀ and T₁: IC Heating; T₁: Beginning of the 1st IC measurement; T₂: End of the 1st IC measurement; between T₂ and T₃: IC interval; T₃: Beginning of the 2nd IC measurement; T₄: End of the 2nd IC measurement CI; T₅: End of urine collection.)
Figure 2. Correlation of the ratio between energy intake and energy expenditure with the nitrogen balance ($r = 0.67$; $p = 0.007$).
Table 1. Characterization of the patients studied.

<table>
<thead>
<tr>
<th>Case</th>
<th>Diagnosis</th>
<th>Sex*</th>
<th>Age (years)</th>
<th>Apache II</th>
<th>Death risk</th>
<th>Death **</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Acute respiratory insufficiency, status epilepticus</td>
<td>F</td>
<td>52</td>
<td>20</td>
<td>48</td>
<td>N</td>
</tr>
<tr>
<td>2</td>
<td>Huntington disease, pneumonia</td>
<td>M</td>
<td>24</td>
<td>20</td>
<td>24</td>
<td>N</td>
</tr>
<tr>
<td>3</td>
<td>Acute renal failure (pre-renal)</td>
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<td>79</td>
<td>38</td>
<td>88</td>
<td>Y</td>
</tr>
<tr>
<td>4</td>
<td>Hypoxic encephalopathy, pneumonia</td>
<td>M</td>
<td>20</td>
<td>17</td>
<td>35</td>
<td>N</td>
</tr>
<tr>
<td>5</td>
<td>Respiratory insufficiency secondary to pneumonia, chronic renal failure</td>
<td>M</td>
<td>80</td>
<td>33</td>
<td>79</td>
<td>Y</td>
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<tr>
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<td>Churg-Strauss syndrome, mesenteric ischemia</td>
<td>F</td>
<td>52</td>
<td>25</td>
<td>79</td>
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<td>Neurotoxoplasmosis, status epilepticus, pneumonia</td>
<td>F</td>
<td>56</td>
<td>23</td>
<td>46</td>
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<tr>
<td>8</td>
<td>Guillain-Barré syndrome</td>
<td>M</td>
<td>38</td>
<td>7</td>
<td>4</td>
<td>N</td>
</tr>
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<td>Respiratory insufficiency secondary to Guillain-Barré syndrome, pneumonia</td>
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<td>69</td>
<td>18</td>
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<td>N</td>
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<td>Chronic respiratory insufficiency, pneumonia</td>
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<td>74</td>
<td>19</td>
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<td>11</td>
<td>Meningoencephalitis, acute renal failure (in reduction)</td>
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<td>60</td>
<td>35</td>
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<td>Severe cranioencephalic trauma</td>
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<td>16</td>
<td>15</td>
<td>18</td>
<td>N</td>
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<tr>
<td>13</td>
<td>Respiratory insufficiency secondary to malnutrition</td>
<td>F</td>
<td>29</td>
<td>23</td>
<td>56</td>
<td>N</td>
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<td>14</td>
<td>Enterectomy, fecal peritonitis</td>
<td>M</td>
<td>56</td>
<td>19</td>
<td>48</td>
<td>Y</td>
</tr>
<tr>
<td>15</td>
<td>Late postoperative (bone marrow transplantation), multiple sclerosis,</td>
<td>M</td>
<td>50</td>
<td>23</td>
<td>26</td>
<td>N</td>
</tr>
<tr>
<td></td>
<td>pneumonia</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>16</td>
<td>Postoperative abdominal aneurismectomy, pulmonary obstructive syndrome</td>
<td>M</td>
<td>78</td>
<td>22</td>
<td>61</td>
<td>Y</td>
</tr>
<tr>
<td>17</td>
<td>Systemic lupus erythematosus/systemic sclerosis, Hypercapnic respiratory</td>
<td>M</td>
<td>31</td>
<td>10</td>
<td>5</td>
<td>N</td>
</tr>
<tr>
<td></td>
<td>insufficiency, pneumonia</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Mean±SD 51±21 22±8 51±32

* F: female; M: male. ** Y: yes (death); N: no (discharged from the ICU).
Table 2. Energy expenditure and basal energy expenditure.

<table>
<thead>
<tr>
<th>Patient</th>
<th>EE (kcal. d⁻¹)</th>
<th>EE (kcal. kg⁻¹. d⁻¹)</th>
<th>BEE (kcal. d⁻¹)</th>
<th>BEE (kcal. kg⁻¹. d⁻¹)</th>
<th>EE/BEE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1601</td>
<td>20.4</td>
<td>1467</td>
<td>18.7</td>
<td>1.09</td>
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<tr>
<td>2</td>
<td>1654</td>
<td>28.8</td>
<td>1536</td>
<td>26.7</td>
<td>1.08</td>
</tr>
<tr>
<td>3</td>
<td>1393</td>
<td>16.6</td>
<td>1382</td>
<td>16.5</td>
<td>1.01</td>
</tr>
<tr>
<td>4</td>
<td>1335</td>
<td>23.5</td>
<td>1552</td>
<td>27.4</td>
<td>0.86</td>
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<tr>
<td>5</td>
<td>1488</td>
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<td>1507</td>
<td>18.6</td>
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</tr>
<tr>
<td>6</td>
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<td>29.6</td>
<td>1276</td>
<td>21.6</td>
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<tr>
<td>7</td>
<td>1279</td>
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<td>1493</td>
<td>17.9</td>
<td>0.86</td>
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<tr>
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<td>1366</td>
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<td>1604</td>
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<tr>
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<td>16.9</td>
<td>1390</td>
<td>17.4</td>
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<tr>
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<tr>
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<td>1363</td>
<td>21.5</td>
<td>1264</td>
<td>19.9</td>
<td>1.08</td>
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<tr>
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<td>1315</td>
<td>21.5</td>
<td>1639</td>
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<tr>
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<td>28.3</td>
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<tr>
<td>14</td>
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<td>20.9</td>
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<td>2320</td>
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<tr>
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<td>20.1</td>
<td>1540</td>
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<td>1.01</td>
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<tr>
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<td>1531</td>
<td>36.6</td>
<td>1242</td>
<td>29.7</td>
<td>1.23</td>
</tr>
</tbody>
</table>

Mean = 1515±268, 22.5±6.1, 1485±160, 21.9±4.6, 1.03±0.17

CV (%) = 18, 27, 11, 21, 17

EE: energy expenditure; BEE: basal energy expenditure.
Table 3. Comparison of energy expenditure, energy intake, energy balance, ratio between energy intake and energy expenditure between patients with a positive (NB>0) or negative (NB<0) nitrogen balance.

<table>
<thead>
<tr>
<th></th>
<th>NB&gt;0 (n=3)</th>
<th>NB&lt;0 (n=11)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EE (kcal. d(^{-1}))</td>
<td>1380±110</td>
<td>1480±193</td>
</tr>
<tr>
<td>Energy intake (kcal. d(^{-1}))</td>
<td>1590±190</td>
<td>1300±610</td>
</tr>
<tr>
<td>Energy balance (kcal. d(^{-1}))</td>
<td>170±170</td>
<td>-180±630</td>
</tr>
<tr>
<td>Energy intake/EE</td>
<td>1.12±0.12</td>
<td>0.89±0.42</td>
</tr>
</tbody>
</table>

EE: Measured energy expenditure, NB: Nitrogen balance