Nutritional Support of Obese Critically Ill Patients

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Obesity: A Common Disease

Obesity is a common medical condition affecting more than 1 in 10 adults in Western European countries [1]. Its prevalence varies considerably in different countries. In Europe, it amounts to about 10–15% of the middle-aged population. It is highest in Eastern European countries, in North America, high in Africa and Eastern Asian countries, where it is strongly associated with poverty, but lower in Japan and China. There has been a progressive rise in the overall prevalence of obesity during the last decade, both in adults and children. The medical and economical consequences are enormous.

The medical spectrum of obesity is wide, ranging from simple overweight without associated medical risk, to morbid obesity with severe associated comorbidities [1]. Various diagnostic criteria have been used; the most useful and simplest relies on the body mass index (BMI) scale. According to the International Obesity Task Force of the World Health Organization, the severity of obesity is classified into 3 main categories: (1) overweight: BMI 25–30; (2) obesity: BMI 30–40, and (3) morbid obesity: BMI over 40 kg/m\textsuperscript{2}. In addition to the absolute amount of body fat, as reflected by the BMI, body fat distribution is important: centralization of body fat to the abdominal visceral stores is associated with the development of systemic and metabolic complications [2]. Body fat distribution can easily be assessed in clinical practice using simple anthropometric measurements, such as waist circumference: a circumference over 102 cm in European men and 88 cm in women is an independent risk factor for a cluster of medical and metabolic
complications, such as insulin resistance, glucose intolerance or noninsulin-dependent diabetes, arterial hypertension, cardiovascular diseases, stroke, high very low-density lipoprotein, low high-density lipoprotein, microalbuminuria, hyperuricemia [2, 3]. Previously designated as the X syndrome or the insulin-resistance syndrome, it is now called the dysmetabolic syndrome. This syndrome is important in clinical nutrition, since it may influence both the nutritional requirements and the tolerance to artificial feeding, as well as the metabolic response to stress. Grossly obese patients also have an increased incidence of respiratory diseases, particularly the sleep apnea syndrome and the restrictive lung disease, venous disease, musculoskeletal degenerative disorders, liver disease (fatty liver) and metabolic disorders consecutive to bariatric surgery.

**Critically Ill Obese: The Clinical Picture**

There is a rise in the number of obese patients requiring intensive care unit (ICU) management parallel to the currently increasing prevalence of obesity. Despite this epidemiological reality and the well-known technical difficulties related to vascular and airway management in grossly obese patients, it is surprising to note the relative paucity of medical literature devoted to this topic. Performing a Medline search using obesity and critically ill as key words furnishes only 35 English references, while the combination ‘obesity and critically ill and nutrition’ limits the list to 11 references.

Although gross obesity seems to markedly affect the survival in severe blunt trauma, this seems not to be the case in patients requiring planned surgery. In a retrospective study performed in 184 patients with severe blunt trauma, mortality was markedly increased in obese patients (BMI >31, n = 19, mortality 42%), compared to overweight (BMI 27–31, n = 25, mortality 8%) and nonobese patients (n = 140, mortality 5%), despite similar severity of injury [4]. Complications were also more frequent in the severely obese group. Multiple regression analysis showed that BMI was an independent predictor of outcome. In a prospective study performed in 24,157 consecutive patients requiring general anesthesia, gross obesity (body weight, BW, >120 kg in males, >100 kg in females) was found to be a strong risk factor for postoperative critical respiratory events (relative risk 2.2) [5]. A retrospective study including 849 patients receiving various types of surgery showed that obesity was associated with higher rates of nosocomial infections, but mortality was not affected [6]. In a retrospective study performed in 5,168 cardiac surgery patients, the incidence of deep sternal wound infections was increased in obese patients, but mortality was not influenced by the presence of obesity [7]. This contrasted with malnutrition (BMI <20, serum albumin <25 g/l), in which these variables were each independently associated with increased mortality. Altogether, these data
suggest that obesity is associated with increased morbidity after major surgery, particularly septic complications, but has no major impact on mortality. In case of more severe stress, like multiple injuries, the presence of obesity seems to markedly affect the outcome.

Table 1 summarizes our data on 131 consecutive obese patients admitted over a 2-year period to our surgical ICU. The latter represented only 5.4% of total admissions. Surprisingly mortality tended to be lower in the obese (3.8 vs. 7.5%, p < 0.08), while the ICU stay tended to be longer. Among obese patients, the longest ICU stays were related to abdominal complications of bariatric surgery. The latter patients required prolonged nutritional support (fig. 1, 2): both patients had suffered acute weight loss over a short period of time before their admission in the ICU, and were actually acutely malnourished, requiring artificial nutrition support.

<table>
<thead>
<tr>
<th>Obese (BMI ≥ 30)</th>
<th>Obese (BMI ≥ 30)</th>
<th>Other patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n = 131; 5.4%)</td>
<td>(n = 2,416)</td>
<td></td>
</tr>
<tr>
<td>Age, years</td>
<td></td>
<td></td>
</tr>
<tr>
<td>16–59</td>
<td>53 (40.5%)</td>
<td>44.5%</td>
</tr>
<tr>
<td>60–69</td>
<td>42 (32.1%)</td>
<td>24.5%</td>
</tr>
<tr>
<td>70–79</td>
<td>32 (24.4%)</td>
<td>26.0%</td>
</tr>
<tr>
<td>≥80</td>
<td>4 (3%)</td>
<td>5.0%</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30–39</td>
<td>117 (89%)</td>
<td>4.5</td>
</tr>
<tr>
<td>&gt;40</td>
<td>14 (11%)</td>
<td></td>
</tr>
<tr>
<td>Length of ICU stay, days</td>
<td>7.3</td>
<td>4.5</td>
</tr>
<tr>
<td>ICU mortality</td>
<td>5 (3.8%)</td>
<td>192 (7.5%)</td>
</tr>
</tbody>
</table>

There was a trend to a lower mortality in obese patients (p = 0.08).


Metabolic Response to Critical Illness

Extensive endocrine, metabolic and immunological changes occur in ICU patients, particularly those suffering from multiple injury, major burns, severe sepsis or severe inflammation. Resting metabolic rate increases, as well as fasting and postprandial glycemia related to insulin resistance [8]. Protein breakdown exceeds protein synthesis in both the fasting and fed state, at the cost of the muscle mass. The response to nutrition is altered: protein balance stays negative despite full nutritional support in highly stressed patients, while gluconeogenesis is not suppressed by carbohydrate administration (see Baracos, pp 1–9).
Fig. 1. Nutritional support of a 60-year-old severely obese patient (height 168 cm, admission weight 133 kg) during the first 40 days after ICU admission. The patient suffered multiple intestinal fistulae after bariatric surgery, complicated by acute respiratory and renal failure. Nutritional assessment on admission showed severe acute malnutrition, resulting from a rapid 20-kg weight loss during the 4 weeks preceding admission (BMI 54.2 → 47.1 kg/m²). Nutritional support was first exclusively provided by the parenteral route (TPN) followed by progressive reintroduction of enteral nutrition (EN). Resting energy expenditure (REE) was determined by indirect calorimetry on days 3 and 21, amounting to 3,000 and to 2,600 kcal/day, respectively. Comparison of energy delivery (shaded area) and REE (solid line) shows that moderate hypocaloric feeding (80% REE) was delivered during the first 18 days, followed by isocaloric feeding. Note that all routes of artificial feeding were used in this patient.

The metabolic response to stress in obese critically ill patients is complex, since it occurs in a population with preexistent metabolic and endocrine abnormalities. A study performed in severely traumatized obese and nonobese patients (injury severity score >18) during the early flow phase (2–4 days after ICU admission) suggests that the metabolic response to injury is influenced by obesity [9, 10]. Compared with lean controls, the obese patients (BMI >30, range 30.8–41.8) had a similar degree of hypermetabolism (140 vs. 137% of Harris Benedict prediction). Plasma glucose and insulin were increased in the 2 groups, while C peptide levels were higher in obese patients. The pattern of substrate oxidation differed in the 2 groups: in obese patients both net protein (22 vs. 15%) and net glucose (39 vs. 24%) oxidation were increased, while net fat oxidation was reduced (39 vs. 61%). Whole body protein turnover and protein synthesis were increased in obese patients. This was associated with increased daily nitrogen and 3-methylhistidine excretion, and decreased protein synthesis efficiency (synthesis/turnover). Daily muscle degradation was also higher in obese patients. Altogether, these results suggest that the metabolic and endocrine changes associated with obesity
modify the metabolic response to injury and further increased protein catabolism. This contrasts with the fair adaptation of healthy obese subjects to starvation: they have better nitrogen preservation than lean subjects and longer survival during hunger strike [11, 12].

**Nutritional Assessment**

Accurate nutritional assessment is difficult in critically ill patients [13]. This is related to the confounding effects of fluid retention on body anthropometry and to the joint effects of dilution and acute phase response on visceral protein plasma levels. BMI calculation is markedly affected by fluid retention and cannot be used to detect obesity in ICU patients [14]. Fluid therapy may induce 20–30% BW gain in patients with major burns or trauma or suffering from severe infection. In such conditions, the analysis of water balance will give more useful information than actual BMI. In patients with short-lasting illness before ICU admission, the anamnestic BMI, calculated from prehospital BW, is more accurate to assess the presence of obesity (or malnutrition).
Malnutrition and obesity may coexist [15], but the early detection of malnutrition in critically ill obese patients can be difficult, since previous malnutrition, accelerated catabolism and fluid retention may blunt nutritional assessment. In such patients, accurate nutritional assessment is not possible; it should therefore be approximate, based on history (weight loss, duration of fasting) and on simple clinical and biochemical variables.

**Nutritional Requirements**

Resting energy expenditure (REE) is increased in obese subjects, due to the concomitant increase in the fat mass and the metabolically active fat-free mass. In healthy nonobese subjects, accurate prediction equations have been developed and validated in large populations: REE predicted from the Harris-Benedict equations has a high correlation \((r > 0.80)\) with measured REE by indirect calorimetry and a fair precision (coefficient of variation of \(<10\%)\). This is not the case in healthy obese subjects with BMI of \(>30 \text{ kg/m}^2\), in whom prediction based on Harris-Benedict equations overestimates REE, particularly for BMI of \(>50 \text{ kg/m}^2\).

REE is difficult to predict in all critically ill patients since it is influenced by multiple and changing factors related to the acute illness, level of stress and treatments [8]. Prediction equations have been developed for specific medical conditions, although their utility in clinical practice is controversial. Measurement with indirect calorimetry is the only validated method to accurately determine the metabolic rate of obese and nonobese critically ill patients, although prediction of 24-hour REE derived from short-time measurements (30–45 min) has a lower accuracy. Prediction equations specifically devoted to obese patients have been published, although they have not been validated on a large scale [16–19]. In obese patients with BMI of \(<50 \text{ kg/m}^2\), estimated energy requirements amount to about 20 kcal/kg ABW, although variability is substantial [19, 20]. In a study performed in 57 obese critically ill patients, the Harris-Benedict equation with an adjusted BW (mean value of actual and IBW) and a stress factor of 1.3 was the most accurate predictor of measured REE, both in mechanically or spontaneously breathing patients (bias \(<200 \text{ kcal}, \text{ precision } \pm 120 \text{ ml}, 67\% \text{ of patients with predicted REE } \pm 10\% \text{ of measured REE}) [19]. This prediction performed better than the Ireton-Jones equations, and the simple rule of 21 kcal/kg ABW/day (bias 270 kcal, 24% of predictions +10% measured REE). Prediction was poor in patients with BMI \(>50\). In another study, the Harris-Benedict equation with an adjusted BW was less accurate than a kilojoules per kilogram-adjusted BW strategy to predict REE [18]. Altogether, these studies suggest that simple rules can be used to have a rough estimate of REE in critically ill obese patients with a BMI of \(<50 \text{ kg/m}^2\). In more severely obese patients, or in those with major stress (severe sepsis or trauma, multiple
organ failure), or with poor response to nutritional support, the use of indirect calorimetry is recommended, since the low accuracy of any prediction equation prevents their use to determine energy requirements.

The current literature suggests that hypocaloric nutrition should be preferred to isocaloric feeding in obese critically ill patients. If such a strategy is used, nonprotein energy requirements are calculated to cover about 50–60% of REE, amounting to about 20–25 kcal/kg ideal BW (IBW) or 13–16 kcal/actual BW (ABW) [21, 22]. It is worth reminding that estimations based on IBW tend to underestimate REE, while those based on ABW tend to overestimate it [18]. When isocaloric feeding is the goal, these figures are increased to about 35–45 kcal/kg IBW or 20–25 kcal/kg ABW [21, 22]. IBW can easily be calculated with the Hamwi equations: (1) IBWmales (kg) = 48 + (height (cm) − 152) × 1.06, and (2) IBWfemales (kg) = 45.4 + (height (cm) − 152) × 0.89 [23].

Current literature suggests that protein requirements are proportionally higher in obese than in nonobese ICU patients, amounting to about 2 g/kg IBW or 1.2–1.3 g/kg ABW, although the level of evidence is rather low. Such levels have been shown to be associated with positive or near zero nitrogen balance [21, 22, 24]. In hypocaloric feeding, the largest part of nonprotein energy should be covered by carbohydrates, keeping in mind that obese patients are often intolerant to glucose. The current literature suggests that hypocaloric carbohydrates are well tolerated in acutely ill obese patients. Most experts recommend reducing the administration of conventional fat solutions to a small part of total energy or to the amount necessary to cover the essential fatty acid requirements [14, 15]. This recommendation does not hold when fat is administered for a nonenergetic goal, i.e. to modulate body functions or responses, such as immunity and inflammation [25]. There is no indication in the literature that micronutrient requirements differ in obese and nonobese patients.

Nutritional Support in Obese Patients

Two reviews have recently been published on nutritional support of critically ill obese patients [14, 15]. Critically ill obese patients require artificial nutrition when it is not possible to feed them adequately by the oral route, as any other ICU patient [26]. Since their metabolic adaptation to fasting is altered during severe stress, they may require early nutritional support. During starvation, the enormous amount of energy stored in their adipose tissue does not protect them from rapid protein catabolism and accelerated malnutrition [9, 15]. Most experts recommend that indications and timing of artificial nutrition in obese patients submitted to major stress should not differ from the current recommendations applied in nonobese patients [14, 15, 26].
Most of the general aspects of nutritional support do not differ in obese and nonobese ICU patients: this is true for the indications to artificial feeding, techniques of feeding, routes and timing of nutrition. The only differences concern energy and nutrient requirements. Several conceptual and clinical arguments suggest that hypocaloric feeding could be an appropriate strategy in obese patients, even during acute illness [14, 15]. The concept is simple: protein sparing is the main objective, while energy requirements are partly covered by the large amount of fat stores [27]. Several studies suggest that such a strategy is effective in acutely ill obese patients (table 2) [21, 28–31]. Practically, 1.5–2.0 g/kg total BW protein is provided daily, while nonprotein energy supply is limited to about 50–60% of REE or 20–25 kcal/kg IBW.

Providing amino acids as the sole energy nutrient in nonstressed obese patients makes it possible to achieve nitrogen equilibrium in a situation of energy deficit. During moderate stress, such a mechanism of nitrogen sparing seems to be effective. This concept was first assessed in 1979 by Greenberg and Jeejeebhoy [27] in a nonrandomized study performed in 2 groups of 6 surgical patients described as ‘having sufficient fat stores to justify intravenous hypocaloric feeding during 7 days’, although no data were provided about BW. The patients received 2 different amino acid mixtures as the sole source of energy: 0.83 vs. 1.83 g/kg IBW/day. The high nitrogen solution made it possible to achieve nitrogen equilibrium (cumulated nitrogen balance + 16 ± 6.7 g after 7 days), but not the low nitrogen solution. Dickerson et al. [31] administered hypocaloric (51.5% measured resting metabolic rate), high-protein (2.1 g/kg IBW/day) total parenteral nutrition (TPN) for a prolonged period (48 ± 31 days) in 13 obese surgical patients. Such feeding was well tolerated: all patients had full tissue healing of wounds and abscess cavities, and minor metabolic complications were observed in only 1 patient. Nitrogen balance was at equilibrium or slightly positive, while serum albumin increased throughout the study. Such beneficial results occurred despite a progressive weight loss (−8.6%).

Hypocaloric versus isocaloric parenteral nutrition was compared in 2 consecutive randomized studies performed in obese patients with severe stress by the same group of authors [21, 29]. In the first study, 16 obese patients (BW >130% IBW) were randomized to receive isonitrogenous hypocaloric or isocaloric nutrition. Nonprotein energy amounted to 50 or 100% of measured resting metabolic rate. TPN was provided for 9.6 ± 3 days. The 2 regimens were well tolerated. Cumulated net nitrogen balances in both groups were positive. Changes in BW and serum albumin did not differ between the groups. In the subsequent study, protein supply was increased to 2 g/kg IBW/day, while energy supply was calculated using a fixed total energy:nitrogen ratio (75:1 vs. 150:1 kcal/g N) [29]. This eliminated the necessity to perform indirect calorimetry. Total daily energy amounted to 94 ± 21 kJ/day (22 kcal/kg/day) in the control group and to 57 ± 12 kJ in the hypoenergetic group. Mean net nitrogen balance and weight changes were similar in the 2 groups.
**Table 2.** Studies on hypocaloric nutrition in obese patients

<table>
<thead>
<tr>
<th>Trial</th>
<th>Type of study</th>
<th>Number of patients</th>
<th>Patient category/ level of stress</th>
<th>Patient BW kg</th>
<th>Route</th>
<th>Energy supply kcal·kg⁻¹·day⁻¹</th>
<th>Protein supply g·kg⁻¹·day⁻¹</th>
<th>Length of artificial feeding, days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Greenberg et al. [27], 1979</td>
<td>Nonrandomized comparison trial 2 groups of patients</td>
<td>12</td>
<td>Patients with 'sufficient fat stores' Moderate stress</td>
<td>Not mentioned</td>
<td>PN</td>
<td>No nonprotein energy</td>
<td>0.83 vs. 1.83</td>
<td>7</td>
</tr>
<tr>
<td>Dickerson et al. [31], 1986</td>
<td>Prospective observational study</td>
<td>13</td>
<td>Postoperative Moderate stress</td>
<td>127 ± 60</td>
<td>PN</td>
<td>51.5% MREE</td>
<td>2.1/kg IBW</td>
<td>48 ± 31</td>
</tr>
<tr>
<td>Burge et al. [21], 1994</td>
<td>RCT Hypo- vs. isocaloric</td>
<td>16</td>
<td>Surgical patients Moderate/severe stress</td>
<td>90 ± 12.5 vs. 102 ± 19.9</td>
<td>PN</td>
<td>22 vs. 42/kg IBW</td>
<td>2.0–2.2/kg IBW</td>
<td>9.6 ± 3.0</td>
</tr>
<tr>
<td>Choban et al. [22], 1997</td>
<td>RCT Hypo- vs. isocaloric</td>
<td>30</td>
<td>Surgical ICU patients Severe/moderate stress</td>
<td>97 ± 19 vs. 90 ± 17</td>
<td>PN</td>
<td>22 vs. 36/kg IBW</td>
<td>2.0 vs. 2.0/kg IBW vs. 10 ± 3 vs.11 ± 2</td>
<td></td>
</tr>
<tr>
<td>Liu et al. [28], 2000</td>
<td>Retrospective study 2 groups of patients aged &lt;60 vs. ≥60 years</td>
<td>30</td>
<td>Hospital patients Light/moderate stress</td>
<td>97 ± 16 vs. 84 ± 20</td>
<td>PN</td>
<td>18.2 ± 3.7 vs. 18.3 ± 2.6/kg ABW</td>
<td>1.5/kg ABW vs. 13 ± 13 vs.13 ± 8</td>
<td></td>
</tr>
<tr>
<td>Dickerson et al. [30], 2002</td>
<td>Retrospective study Hypo- vs. isocaloric</td>
<td>40</td>
<td>Critically ill Severe stress</td>
<td>118 ± 41 vs. 102 ± 36</td>
<td>EN</td>
<td>16.2–22.2 vs. 21.5–29.9/kg IBW</td>
<td>1.14–1.53 vs. 1.29–1.85/kg IBW vs. 26 ± 15</td>
<td></td>
</tr>
</tbody>
</table>

PN = Parenteral nutrition; EN = enteral nutrition; IBW = ideal body weight; ABW = adjusted body weight; RCT = randomized controlled trial.
A recent retrospective study suggests that hypocaloric enteral feeding was as effective as isocaloric feeding in 40 critically ill obese patients [30]. The patients had weights of >125% IBW and received 7 days or more of enteral nutritional support. They were divided into 2 groups according to energy supply: isocaloric feeding, 20 kcal/kg adjusted BW/day, and hypocaloric feeding <20 kcal/kg adjusted BW/day, with adjusted BW = (ABW − IBW) × 0.25 + IBW. BW, BMI, severity of illness and protein supplies were similar in the 2 groups. Despite lower energy supply, the hypocaloric group had similar nitrogen balance and serum pre-albumin as the isocaloric group. Glycemia was not affected by energy supply. The hypocaloric group had a significantly shorter ICU stay (19 ± 10 vs. 29 ± 16 days, p < 0.03) and shorter antibiotic therapy (17 ± 12 vs. 27.4 ± 17 days, p < 0.03). Altogether, these data show that hypocaloric feeding is well tolerated in acutely ill obese patients, even when prolonged: it does not seem to be associated with increased protein catabolism, or with deleterious clinical consequences. These data seem to be in contradiction to those collected in nonobese ICU patients receiving prolonged enteral or parenteral nutritional support, since there is evidence that marked energy and protein deficits are associated with poor clinical outcome and with increased systemic complications like infections in the nonobese [32, 33]. Clearly, the safe level of energy deficit remains to be determined in different patient populations. This is particularly important when considering the actual predominance of exclusive enteral feeding.

Bariatric surgery generally does not require prolonged ICU support. It may however result in severe abdominal complications requiring artificial nutrition, like intestinal leakage and fistulae. They frequently occur during the period of maximal weight loss after surgery, i.e. during the period at risk of malnutrition. Such complications generally involve the gut, which is out of function for variable periods of time: TPN is required for life support [34]. Figures 1 and 2 show examples of such patients admitted to our surgical ICU, with the progressive introduction of enteral feeding. These patients were in septic shock due to peritonitis with acute respiratory failure on admission and required repeated surgical sessions. On admission they had acutely lost 20 and 45 kg BW, respectively, and had been fasted for many days.

**Technical Problems**

Vascular access can be a major technical problem in obese patients requiring anesthesia and surgery or venous access for parenteral nutrition. Peripheral veins are usually difficult to detect and consequently catheter insertion can be a worry. A more frequent use of central venous access is therefore commonly required, but internal jugular and subclavian vein catheterization may be difficult, resulting in a higher incidence of puncture complications and catheter malposition [35]. This may favor catheter-related thrombosis and infection.
Although obese patients have an increased incidence of infections, particularly postoperative wound infection, obesity per se is usually not considered as a risk factor for catheter sepsis [36]. Considering all these potential risks related to vascular access and catheter maintenance, it is rather surprising that there is no single catheter complication reported in the 6 studies of table 2 (141 patients enrolled). This may reflect the fact that many nutritionists do not insert the catheters themselves in difficult patients, such as the grossly obese, and therefore underreport the related complications.

Complications of Nutritional Support

Malnourished stressed obese patients may constitute a true challenge for the nutritionist. However, despite such difficulty there is little evidence in the literature that complications related to nutritional support are common [37].

Obese patients are prone to develop glucose intolerance or diabetes. There is, however, no indication in the literature that artificial feeding in obese ICU patients is associated with hyperglycemia requiring intensive insulin therapy or with diabetic decompensation. This may partly be explained by the administration of hypocaloric nutrition. A randomized controlled trial compared hypo- and isocaloric intravenous nutrition in obese hospitalized patients: glucose control was little affected by the amount of delivered energy and carbohydrate: there was no difference in the number of patients requiring insulin in the 2 groups [21]. However, there was a significantly higher number of days with insulin therapy in the isocaloric group and a tendency toward higher insulin requirements (daily dose $61 \pm 61$ vs. $36 \pm 47$ U/day, NS). In another study comparing the nutritional and clinical efficacy of hypo- and isocaloric enteral support in critically obese patients, plasma glucose seemed to be unaffected by the amount of nonprotein energy supply (glucose-fat mixture) [30]. Unfortunately, neither the number of patients requiring insulin therapy nor the amount of insulin was described. These data suggest that glucose control is not a major problem in obese patients with moderate or severe stress receiving either hypo- or isocaloric feeding.

Bronchopulmonary aspiration is an established risk of enteral feeding, particularly in comatose patients. Gross obesity is often mentioned as a risk factor, since gastric emptying is delayed and intra-abdominal pressure is increased compared to healthy subjects [14, 15]. However, such an assumption is not supported by the existing literature and there is presently no rationale to avoid or to delay enteral feeding in obese patients.

Perspectives

Numerous questions remain unsolved regarding the metabolic responses to critical illness and the techniques of nutritional support in obese patients.
including: the role of the metabolic syndrome; nutrient utilization; the optimal feeding route, and appropriate timing of nutritional intervention. Another important issue is the assessment of nutritional status which is complicated by the acute changes in body composition related to critical illness. There is actually no validated method to detect malnutrition early in the course of disease. Another interesting issue, which deserves further research, is the role of the adipose tissue in the systemic inflammatory responses, particularly regarding cytokine and mediator release.

**Conclusions**

Considering the increasing incidence of obesity and the paucity of the available literature dealing with the critically ill obese, solid nutritional and metabolic studies should be promoted. Obese patients submitted to severe stress are unable to adapt to prolonged starvation. Nutritional support should be provided using similar indications, routes and timing as in nonobese critically ill patients. Numerous studies suggest that hypocaloric hyperprotein feeding should be preferred in such patients.

**Acknowledgements**

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**References**


Discussion

*Dr. Bozzetti:* The story of obese patients reminds me of the story of elderly patients. The surgical literature is full of papers stating that you can operate on patients 80, 90 years old. You can do pancreatectomy in these patients with an acceptable risk which is quite similar to that of adult subjects. In my opinion this reflects a strong selection of the patients because if you look at the morbidity and mortality of elderly patients admitted to intensive care units (ICUs) for trauma, you see a clear correlation between age, morbidity and mortality. My interpretation is not that critically obese patients have a poor tolerance of their situation but this reflects the absence of a selection, so you see that obesity is really a major aspect. The question is have you some suggestions about the water requirement? I am not speaking of patients admitted to the ICU where I expect that the water requirement follows some rules which are different from the usual, but for the usual obese patients operated, in the postoperative period, what is the rule for the water requirements?

*Dr. Chioléro:* This is an important question since severely obese patients do not tolerate water and electrolyte excess in contrast to other acutely ill surgical patients, for whom we prescribe a large quantity of water and electrolyte. Obviously this has to be taken into account when you prescribe the nutrition. I think your first comment is also important. For a lot of surgical procedures we can do what we have to do with the obese patients: the experience all over the world in cardiac surgery patients, a population with a lot of obese patients, shows that such patients do tolerate surgery quite well. But there is a limit as shown by the data collected in severe trauma or severely burned obese patients, demonstrating an increased rate of complications and even decreased survival rate. Bariatric surgery complicated by abdominal and septic postoperative problems is another field where we sometimes have a lot of difficulty in severely obese patients associated with a poor clinical outcome. Clearly, a lot of work should be done to identify the patients at risk and to delineate therapeutic strategies.

*Dr. Labadarios:* I would like to give Dr. Bozzetti's question a little bit more of a global nature. We are all concerned about this so-called pandemic of obesity and in my opinion correctly so. But we have been so preoccupied, almost in a state of panic, about addressing this emerging pandemic. Is there room for us to consider the concept of healthy obesity? I mean, you have not shown us that these people actually behave any differently from you and me. Is there room for such a concept of healthy obesity? I know our American friends would now shout me down and we can argue about the data that we have one way or the other, but have we ever spent time thinking about this concept and is there such a concept to think about?

*Dr. Chioléro:* I think this is an interesting comment which includes two aspects. As an intensivist I could answer that there is little problem. If you consider epidemiology, I think the point of view is different since clearly even simple overweight is associated with increased comorbidities over a long period. So it is both true and false that overweight is a problem. A normal body mass index (BMI) is required to have a maximal life expectation. This is not true during short periods, like the postoperative period, where this epidemiological point of view is not relevant.
Dr. Moore: Your talk identifies a major problem that is not very well studied. When we ran across that Choban study, we returned to the ICU similar to you. Unfortunately in Texas there are a lot of obese patients, and we really could not document any difference in mortality. We showed the obvious: the bigger the patient is the harder it is for us to get the patient off the ventilator, and this increased ventilator-associated pneumonias, but those ventilator-associated pneumonias don’t really contribute very much to mortality. You are an expert in indirect calorimetry, and we have a very difficult time using indirect calorimeter to identify how we should feed patients in the ICU, so we vary between using ideal body weight and adjusted body weight. Which one should we use?

Dr. Chioléro: It is a difficult question. I agree with you that indirect calorimetry is difficult, particularly in obese trauma patients in the ICU setting. For example I made the comment that net substrate calculation using indirect calorimetry in the ICU is particularly difficult since small errors on the respiratory quotient induce a large change in substrate oxidation. But if it will give you an idea of how much energy the patient is spending at that time, remember that we do 20- or 30-min measurements a day, a very short measurement time taking into account the 1,440-min length of the whole day. Concerning your question on the kind of body weight for nutrition prescription (actual or ideal), I would say that, except in obese patients with renal or acute liver failure, in whom protein supply should probably be reduced, I think the concept developed by Choban and Flancbaum based on ideal body weight is a clever convenient one and very simple. They prescribe energy and nutrient based on a rule consisting of giving 2 g protein/kg ideal body weight with an energy protein ratio of 50 kcal/g nitrogen. The concept of hypocaloric nutrition is well established but the optimal energy and protein supply has not been assessed. What we know is that hypocaloric feeding which is quite well tolerated, may facilitate glycemia control. It will decrease fluid supply and it is easy to use for the nutritionist.

Dr. De Bandt: There is convincing evidence of disimmunity associated with diabetes and obesity. Is there an improvement or some hint of improvement in immunological parameters during a hypocaloric diet in these patients?

Dr. Chioléro: This is certainly an important point but I have no information on this aspect since I am not a specialist in immunology. In the literature I have seen no mention of this important point.

Dr. Rosenfeld: You mentioned that syndrome X and intra-abdominal fat distribution in these patients clearly have more complications. Isn’t it time for critically ill obese patients to be stratified by fat distribution? In our unit we have seen patients who had more intra-abdominal fat and higher levels of reactive C protein and more complications, more organic failure. Isn’t it time to stratify patients by fat distribution?

Dr. Chioléro: I agree with your comment that fat distribution could be important in critically ill patients, remembering that waist circumference may be influenced by fluid accumulation in surgical patients or by bowel distention. Anthropometry assessment is sometimes very inaccurate in such patients, including fat distribution. Since many of these patients require CT scan for non-nutritional purposes, we could perhaps collect interesting information on fat distribution. But I am unaware of any study on this point.

Dr. Martindale: I routinely do bariatric surgery on patients over 350 kg. It is a very interesting population. As Dr. Moore mentioned, after the Choban study came out he went to the unit and looked at the complications in that population. We did the same and we very interestingly found a big dichotomy between those who came in ambulatory and those who did not. The 350-kg patient who walks into the hospital does very well. The 350-kg patient who can no longer walk because of the obesity has a tremendously long hospital stay, a tremendously long ventilator requirement and has
the complications associated with hospital stay, not so much with the surgical procedure, I mean routine PE. Any nonambulatory patient over 350 kg who comes to the hospital for a bariatric procedure gets a filter because they have PE. Virtually all of them get PEs postoperatively. So I think that is a key. Looking at their premorbid conditions in the surgery is the key, and in trauma we obviously can’t do that.

Dr. Chioléro: I think this is an interesting comment since organ function including muscle is related to nutritional status and is an important aim in nutritional support. A similar observation was made more than 20 years ago in patients requiring aggressive pulmonary resection. Those who are able to walk and perhaps even to climb some steps usually had an uncomplicated postoperative course, while those with more severe functional abnormalities usually had a bad postoperative evolution.

Dr. Allison: Concerning the protein metabolism of obese surgical patients, I guess you would accept that the higher nitrogen output simply reflects the fact that these people have a bigger lean mass. Elia reviewed prolonged fasts in very obese individuals, some up to a year. He showed with a prolonged fast that the obese individuals tend to protect their lean mass in a curious way. In other words even when you adjusted it for lean mass their nitrogen output was consistently 2 g/day less over the whole period of time. What you are saying is that this protective relationship, almost as if the lean mass knows what the adipose tissue reserve is, is abolished when you have an injury. So do you think this protective effect of obesity which is seen in prolonged starvation is lost with the stress response?

Dr. Chioléro: Yes, and this is not specific to the obese patients since it may be observed in all severely stressed ICU patients. There are few studies on starvation in ICU patients. Ketosis is nearly totally suppressed in critically ill obese and nonobese patients. This is an adaptation phenomenon related to the increased glucose requirements of the wounds and inflammatory cells. In such conditions there is a redistribution of the metabolic priorities. But I am unaware of data on ketosis in obese patients with prolonged hypocaloric feeding. In Dickerson's study in which there was prolonged hypocaloric feeding in surgical patients, no data on ketosis were given. I think there is a lot of work to be done on starvation in these patients.

Dr. Cynober: What about patients who undergo gastroplasty surgery in the short term and in the middle term? Are there some data especially with regard to lean body mass variations?

Dr. Chioléro: Yes, there are data coming from America. Dr. Martindale explained that he observed many patients weighing more than 350 kg being operated on the stomach or having bypasses. Bariatric surgery is a complex issue. Data on body composition after surgery were presented at the last ESPEN meeting. The patients lose weight but lean body mass seems to be preserved, provided adequate nutritional support is offered.

Dr. Martindale: I think you are right, they lose weight. They protect their lean body mass. Persons over 350 kg lose about 80% of their excess weight at 5 years, and by 10 years it is down to about 65%, and 15 years it stays about that.

Dr. Cynober: In our institution we have an important department looking at obesity and surgery and so on. For example we are looking at trace elements, vitamins, vitamin A and so on. We have the feeling that these patients are rapidly depleted in micronutrients. I don’t know if in your experience you are measuring lean body mass but when you are looking with Dexa, these patients clearly become malnourished very rapidly. I don’t know if you agree that there are very few studies looking very carefully at the behavior of lean body mass in these patients after surgery.

Dr. Chioléro: They need regular specialized nutritional support otherwise they will become malnourished after 1–2 years.

Dr. Labadarios: In relation to these very interesting data that Dr. Martindale mentioned, I would like to ask whether he has got any data on biomarkers between
these two classes of people, the ones that are ambulatory and the ones that are not. My question is, are these, the ambulatory patients, not the ones that have really adapted to an ‘unphysiological’ state as opposed to those that come in bedridden? You said pulmonary embolism is common in all of them, but is it really common in all of them? Do you have any data on that specific point?

*Dr. Martindale:* Before we were routinely putting in filters. We have data on that just because of the poor clinical outcomes: the bigger they are, the more obese they are, and the longer they stay in bed. I think the comment about rapid malnutrition, the problem in the US is that the reimbursements for bariatric surgery are very high. So many surgeons who have no interest in obesity and no interest in nutrition are now doing bariatric surgery. To me this is criminal because they are doing it for the reimbursement, not because they care about the problems in the patients. So by 1, 2 and 5 years these patients are left by themselves with nobody following their malnutrition problems. The true studies that have been done are really only in abstract form showing that these people are all malnourished at 1 year. It is controlled undernutrition, and that exact word, control, is not in there. There is nobody controlling these people when they are left alone except in a few centers. In a few centers where vitamin levels and protein levels are routinely followed and outputs at 6 months and 1 year are routinely looked at, patients are put on a treadmill to see what their performance levels are. But nobody in the US has got any long-term studies in this area, and the problem is that there are 65,000 bariatric surgery procedures being done in the US this year. It is estimated that in 2005 it will go to 80,000, and the reimbursement for a bariatric procedure is USD 4,200, the reimbursement for a ripple procedure in the US right now is USD 2,600.

*Dr. Bouletreau:* Would you say in your patients that the risk is more the complications of obesity than the obesity itself?

*Dr. Martindale:* No, it is the complication of the obesity if you look at hypertension, glucose intolerance, sleep apnea and those things. Sleep apnea within 16 weeks is 100% cured, the diabetes is about 80% cured, and the national study health just made a comment on curing type-II diabetes with bariatric surgery in the obese population, and the President of the United States recently declared that obesity is a disease, an independent disease, not just a risk factor or comorbid factor, but a disease, and that allows the government to pay for much of the comorbidity associated with it. So we can cure the diabetes, the hypertension and sleep apnea almost 100% of the time with the operation. The problem is we are not treating. As you saw from those maps the only state in the United States that has under 15% obesity is Colorado and that is because of an education program from day 1 of starting elementary school. We have lost our benefit once we are treating the disease surgically, it is too late.

*Dr. McClave:* Your data would suggest that they don’t have any trouble mobilizing fat. My question revolves around the concept of cyclic feeding. Is it important to have an off period where we can allow the insulin levels to drop and mobilize that fat, and if we provide enteral feeding but infuse it over 24 h do we jeopardize that?

*Dr. Chioléro:* Yes, if we increase the glucose supply and give insulin we are going to block the mobilization of the triglyceride from the fat stores, so clearly it is an important point considering cyclic feeding to optimize energy and nutrient utilization.

*Dr. McClave:* Did most of the studies you presented provide nutrition in a cyclic manner?

*Dr. Chioléro:* No, it was noncyclic nutrition.

*Dr. Heyland:* I have a question in that context, and I am talking here about not the severely obese but rather the more mild, moderately obese which is more common in the ICU, and the question is in the context of designing and interpreting clinical trials. I heard you say that the obese patient has different outcomes compared to the
nonobese, and has different responses to stress compared to the nonobese. But specifically do they have a different response to nutritional support whether enteral or parenteral, so that they are randomized to a fixed diet one intervention, intervention A versus intervention B? Might I expect a different response in the obese versus the nonobese? And if so, how do I better characterize the obese patient, is it on the basis of the BMI, is it on the basis of the fat distribution or does it have more to do with insulin resistance, and do I need to characterize that better, and how would I do that?

Dr. Chioléro: This is a difficult question since even in nonobese patients it would be difficult to address your question. Clearly an important point to consider is the absolute value of the BMI, i.e. the fat deposition. Visceral fat deposition is another aspect to consider, as well as the relationship between the metabolic syndrome and the response to injury. Nobody has systematically assessed these points and I think they could be important. Unfortunately, there are very few nutritional and metabolic studies comparing the metabolic and nutritional response in obese and nonobese critically ill patients. To perform studies comparing the effects of a given nutritional intervention in obese and nonobese stressed patients would therefore be very interesting.

Dr. Bouletreau: Don’t you think it is important even in hypocaloric feeding to preserve a certain amount of glucose and not to go back to the first studies, the first recommendations? What minimal level of glucose would you recommend?

Dr. Chioléro: I fully agree with your point of view, underlying the importance of glucose supply during hypocaloric nutrition. The best is probably to give the largest part of nonprotein energy as glucose, and only to give fat to avoid essential fatty acid deficiency or to exert specific systemic effects such as with fish oil. Concerning the minimal amount of glucose supply, I think there is no difference between obese and nonobese stressed patients. They clearly require a minimal amount of glucose to have the best metabolic control. Stress patients are not like unstressed patients during starvation.

Dr. Nitenberg: You showed many data about surgical patients and moderately stressed patients. Do you think that we have to consider obese patients differently in more severe aggressions such as severe burns or sepsis or other types of severe situations? Do you have any data on that or do other people in the audience have that?

Dr. Chioléro: Interesting question. We had a discussion after Dr. Allison’s presentation concerning specific patient populations in the ICU, and we have presently many obese patients in our population. Unfortunately there is very little information in the literature concerning obese patients with specific critical conditions such as sepsis, trauma or burns. The only strong evidence is related to the severity of critical illness. Published studies show that obese patients tolerate a moderate level of stress well, but behave worse than nonobese patients during major stress.

Dr. Martindale: My question is on the volume of distribution of drugs. I worry very often that we are delivering nutrient based on ideal body weight in many cases, nitrogen anyway, but we don’t change the delivery of our drugs, and the volume of distribution in these patients. I don’t really have any data and I don’t know what do you do at your place.

Dr. Chioléro: It is an important question. In the usual pharmacokinetic approach we should calculate the hydrosoluble drug prescription on the fat-free mass basis. But we have a lot of factors, at least in the ICU setting, which are going to influence drug distribution. We administer more fluid, they have fluid retention, and drug elimination may be altered by various factors such as liver, renal failure and other factors. Actually, in our practice in obese patients, we usually measure the plasma level of many drugs but we find no simple relationship with actual or ideal body weight. There is
clearly no simple relationship between ideal and adjusted body weight and drug prescription.

Dr. Zazzo: Just a comment. Analogically, for prescribing antibiotics to obese patients the literature say that we calculate 40% of the difference between ideal and actual weight. I think it is perhaps a way to compare antibiotic equations or recommendations for it and energy supply.

Dr. Chioléro: In your experience, does it work?

Dr. Zazzo: Yes, in my experience it works for amino acids and bronchomycine because in clinical practice it is very difficult to get the dosage for β-lactamine.

Dr. Chioléro: Yes, but the volume of distribution of bronchomycine is not the right one, so probably it is a good example which goes with the concept.

Dr. Dechelotte: Just to add to the discussion: we should take the lipophilic severity of the drug into account. It is quite different between drugs that will accumulate in fat stores.

Dr. Chioléro: I fully agree.