The prevalence of obesity in the general population is high and it is inevitable that artificial feeding will be needed from time to time in the obese patient, particularly in the critical care setting. Against a background of generous endogenous stores of energy as adipose tissue and the ability of obese individuals to survive starvation longer than non-obese individuals, emphasis is placed on preserving lean body mass and optimizing physiological function. Insulin resistance is typical of the obese individual and is exacerbated by stress; overfeeding is dangerous, particularly if it results in hyperglycaemia. Refeeding syndrome also has to be avoided. Weight may be difficult to measure and lean body mass difficult to assess. Calculation of energy requirements is therefore problematic in practice in the obese individual and there is substantial evidence from controlled clinical trials of the safety of feeding at or below resting energy expenditure. If this approach is taken it is wise to provide a more generous than normal protein intake and to beware of patients with a very high baseline urinary N excretion.

Survival in starvation and obesity

Whereas the Northern Ireland hunger strikers died after 57–73 d, obese individuals undergoing therapeutic starvation, in which vitamins and water are supplied, survive starvation much longer; ≥100–231 d have been recorded. While no doubt the absence of vitamins from the hunger strikers’ intake had an important impact on survival time, it seems that obese individuals can survive simple starvation much longer than lean individuals because they can preserve their lean tissue mass longer.

Insulin resistance in obesity and stress

Obesity and stress are both characterized by insulin resistance. It is not the remit of the present article to review this association in detail, but the following summary is extracted from reviews on insulin resistance in obesity (Corry & Tuck, 2001) and insulin resistance in stress (Mizock, 1995), where references to the original papers can be found.

Insulin resistance increases linearly with BMI. In the unstressed particularly viscero-obese patient a syndrome is recognized that encompasses amongst other factors hypertension, insulin resistance and impaired glucose tolerance, abnormal plasma lipids and endothelial dysfunction. The hypertension relates to abnormalities of the rennin–angiotensin system, which interplays with insulin differently in different tissues. The insulin resistance presents in three ways: (1) a resistance to insulin-mediated cellular uptake of glucose; (2) increased circulating NEFA that, in contrast with the non-obese state, are poorly suppressed by an insulin infusion. NEFA decrease the uptake of glucose into skeletal muscle and increase serum levels of a prothrombotic factor, plasminogen activator inhibitor type 1. NEFA contribute to hypertension by an $\alpha_1$-adrenergic receptor-mediated mechanism; (3) a relative failure of the normal NO-mediated vasodilatation elicited by insulin.

Metabolic stress related to illness, trauma or sepsis also results in insulin resistance. TNF-α contributes to this effect, and probably also to the insulin resistance of aging and type 2 diabetes. The effects of stress are also mediated by the classic hormones cortisol, glucagon and the catecholamines. During stress carbohydrate metabolism is...
abnormal, even in the non-obese patient, and is characterized as follows: increased glucose uptake (immune cells, non-insulin-mediated glucose uptake); increased glucose utilization (glycolysis and oxidation); hyperlactataemia (normal pyruvate:lactate); hyperglycaemia, increased gluco-metabolism; increased gluconeogenesis; decreased glycogen production.

**Risks of overfeeding and the refeeding syndrome**

Overfeeding, particularly of high-energy substrates, must be avoided because it carries risks of hyperglycaemia, increased glucose uptake (immune cells, non-insulin-mediated glucose uptake); increased glucose utilization (glycolysis and oxidation); hyperlactataemia (normal pyruvate:lactate); hyperglycaemia, increased gluconeogenesis; decreased glycogen production.

**Calculating or measuring the energy requirements for obese patients**

The standard approach to estimating energy requirements in patients requiring artificial nutritional support is to estimate the BMR using either the Schofield (1985; weight, age, gender) equation or the Harris-Benedict (Harris & Benedict, 1919; height, weight, age, gender) equation. The Schofield (1985) equation is based on a much larger database (that encompasses that used by Harris & Benedict, 1919) and does not require height, and therefore has been used as standard practice by most clinical nutritionists, although its precision as an estimate of total energy expenditure is uncertain.

### Table 1. BMR in 55-year-old lean (70 kg) and obese (100 kg) male subjects

<table>
<thead>
<tr>
<th></th>
<th>Lean male</th>
<th>Obese male</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1·78 m, BMI 22 kg/m²</td>
<td>1·78 m, BMI 32 kg/m²</td>
</tr>
<tr>
<td>Schofield†</td>
<td>11·4 W + 870</td>
<td>106</td>
</tr>
<tr>
<td>Harris Benedict†</td>
<td>66 + 13·7 W + 5 H – 6·8 A</td>
<td>6·4</td>
</tr>
<tr>
<td></td>
<td>7·0</td>
<td>1668</td>
</tr>
<tr>
<td></td>
<td>6·4</td>
<td>1541</td>
</tr>
</tbody>
</table>

W, weight (kg); H, height (cm); A, age (years).

†Harris & Benedict (1919).

Avoiding refeeding syndrome requires, among other factors, avoiding excess energy input and, therefore, depends on the prescription of appropriate energy requirements; however, these requirements can be difficult to determine in the obese patient. It also depends on the adequate provision of electrolytes such as phosphate, K and Mg, and micronutrients such as thiamin and folic acid.

### Table 2. Body composition for a normal 70 kg male and an obese 100 kg male (Garrow, 2000)*

<table>
<thead>
<tr>
<th></th>
<th>Normal 70 kg male</th>
<th>Obese 100 kg male</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fat (kg)</td>
<td>12 (17%)†</td>
<td>35</td>
</tr>
<tr>
<td>FFM (kg)</td>
<td>58</td>
<td>65</td>
</tr>
<tr>
<td>Water (kg)</td>
<td>42 (60%)†</td>
<td>47</td>
</tr>
<tr>
<td>Protein (kg)</td>
<td>12 (17%)†</td>
<td>13</td>
</tr>
</tbody>
</table>

FFM, fat-free mass.

*Excess body weight comprises (kg) 750 g fat and 250 g FFM (750 g water and 250 g protein/kg).
†Percentage of total body weight.

>37, 12; severe infection or sepsis, 10–30; recent extensive operation, 10–30; fracture or trauma, 10–30; burn wounds, 50–150; respiratory distress syndrome, 20. Each stress factor is added individually to the estimated BMR to provide an estimate of total energy expenditure. For the van Lanschot et al. (1986) series of values the mean correction factor is 46 (SD 17)%, which brings the estimated total energy consumptions to approximately the same as those measured by 24 h indirect calorimetry. Similar approaches have been advocated by other researchers, e.g. Colley et al. (1985), but in the UK the nomogram of Elia (1990) has been used as standard practice by most clinical nutritionists, although its precision as an estimate of total energy expenditure is unclear. Indeed, it is unclear how important it is to clinical outcome to achieve precise energy balance in critically-ill patients with adequate stores of adipose tissue who are artificially fed in the short to medium term. Avoidance of overfeeding on the one hand and major weight loss or nutrient deficiency on the other are sensible clinical goals for most patients.

Current American Society for Parenteral and Enteral Nutrition (2002) guidelines suggest (with reservation) that BMR should be estimated by using the ideal body weight (IBW) + 25% of the difference between this value and the actual body weight in the Harris-Benedict equation. Standard stress factors are then applied. IBW in this model is calculated in pounds using the Hamwi (1994) formula: male, 106 + (height (in) – 60) x 6; female, 100 + (height (in) – 60) x 5. The result can be divided by 2·2 to convert it to kilograms. IBW calculated in this way does not give the same BMI for different heights, so the practice of calculating IBW from the patient’s weight and an assumed ideal BMI of, for example, 23 kg/m² is not strictly correct.
### Table 3. Controlled studies of hypoenergetic feeding

<table>
<thead>
<tr>
<th>Reference</th>
<th>Patient population</th>
<th>Protocol</th>
<th>Outcome</th>
<th>Reference</th>
<th>Patient population</th>
<th>Protocol</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Burge et al. (1994)</td>
<td>Obese TPN</td>
<td>7 100% REE, 2g protein/kg IBW</td>
<td>Mean N balance + 2.8 g</td>
<td>9 50% REE, 2g protein/kg IBW</td>
<td>Mean N balance + 1.3 g (NS)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Choban et al. (1997)</td>
<td>Obese: mean BMI 35 (range 26–47) kg/m² TPN</td>
<td>14 81 MJ (1909 kcal)/d, 108 g a/d</td>
<td>Mean N balance + 3.6 g Period on insulin 8 d</td>
<td>16 5.4 MJ (1290 kcal)/d, 120 g a/d</td>
<td>Mean N balance + 4.0 g Period on insulin 3 d</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dickerson et al. (2002)</td>
<td>Obese Multiple trauma Not randomized Enteral</td>
<td>12 0.10–0.13 MJ (range 0.08 MJ (20 kcal)/kg)</td>
<td>Mean N balance + 4.0 g Antibiotics 27.4 d*</td>
<td>28 &lt; 0.06 MJ (20 kcal)/kg adjusted wt 2 g protein/kg IBW</td>
<td>Mean N balance + 4.0 g Antibiotics 16.6 d*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>McCowen et al. (2000)</td>
<td>Non-obese Enteral</td>
<td>23 5.0 ± 0.9 MJ (1192 ± 212 kcal)/d, 89 ± 13 g a/d</td>
<td>LOS 17 (sd 15) d Deaths 1</td>
<td>25 3.8 ± 0.4 MJ (913 ± 90 kcal)/d, 70 ± 2 g a/d</td>
<td>LOS 19 (sd 14) d Deaths 2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

TPN, total parenteral nutrition; REE, resting energy expenditure; IBW, ideal body weight; aa, amino acids; ICU, intensive care unit; LOS length of stay. Outcomes for control groups were significantly different from those for groups receiving hypoenergetic feeding: *P < 0.03.

†Number analysed after exclusions.

### Hypoenergetic feeding: the evidence

An important study (Jeevanandam et al. 1991) in patients with multiple trauma soon after admission and given crystallloid infusions has compared the metabolic responses of obese patients (n 7, mean weight 80 kg) with non-obese (n 10, mean weight 80 kg) patients. As expected, both groups were similar in terms of initial physiologic response. In those patients who were ventilated, the measured energy expenditure (mean ± SD 10.63 MJ (2550 kcal)/d) of the obese patients was found to be remarkably similar to that of the non-obese being studied (10.35 MJ (2538 kcal)/d). Although they were heavier, the measured energy expenditure in these patients was also lower in terms of percentage of ideal body weight than in non-obese patients (10.63 MJ (2550 kcal)/d). The question arises, therefore, as to whether calorimetry is necessary for artificially-fed obese patients. The study of Glynn et al. (1999), in which short-term (12–15 min) indirect calorimetry was used in eighty-five obese patients compared the indirect calorimetry data with the Harris-Benedict equations when consideration is made for whether or not the patient is ventilated. It is suggested that indirect calorimetry is not an accurate method for assessing energy intake, and therefore, has no place in the assessment of energy intake in these patients. Additionally, the results of the study by McCowen et al. (2000) in which short-term (12–15 min) indirect calorimetry was used in eighty-five obese patients. It was found that the 88 kJ (21 kcal)/kg reported for both groups of patients was 10.63 MJ (2550 kcal)/d, whereas the mean value for the non-obese group was 10.35 MJ (2538 kcal)/d. The difference in BMR between the two groups was significant for both groups of patients. However, the measured energy expenditure (mean ± SD 10.63 MJ (2550 kcal)/d) of the obese patients was found to be remarkably similar to that of the non-obese being studied (10.35 MJ (2538 kcal)/d). As expected, both groups were similar in terms of initial physiologic response. In those patients who were ventilated, the measured energy expenditure in these patients was also lower in terms of percentage of ideal body weight than in non-obese patients (10.63 MJ (2550 kcal)/d). The question arises, therefore, as to whether calorimetry is necessary for artificially-fed obese patients. The study of Glynn et al. (1999), in which short-term (12–15 min) indirect calorimetry was used in eighty-five obese patients compared the indirect calorimetry data with the Harris-Benedict equations when consideration is made for whether or not the patient is ventilated. It is suggested that indirect calorimetry is not an accurate method for assessing energy intake, and therefore, has no place in the assessment of energy intake in these patients. Additionally, the results of the study by McCowen et al. (2000) in which short-term (12–15 min) indirect calorimetry was used in eighty-five obese patients.
responses were found to be lower (non-significantly) in the obese patients than in the non-obese patients. However, despite the RQ suggesting combustion of a similar metabolic mix, glycerol and whole-body protein turnover measurements indicate markedly less fat oxidation and more carbohydrate and protein oxidation in the obese patients, which is reflected in a much larger urinary N loss (22 g v. 14 g). This finding raises concerns about the ability of obese patients to metabolize their adipose tissue effectively during metabolic stress. However, an initial stimulus for trying a hypoenergetic approach was provided by an uncontrolled study (Dickerson et al. 1986) in which thirteen seriously-ill obese (208% IBW) post-operative patients were managed for periods ranging from 12 to 190 d with feeds providing 3.7 MJ (881 kcal)/d and 2–13 g amino acids/IBW per d with excellent results. Subsequent controlled studies (Table 3) that have compared hypoenergetic artificial feeds with feeds providing approximately the resting energy expenditure have given further support for the use of hypoenergetic feeding in critically-ill obese patients, and the large negative N balances reported by Jeevanandam et al. (1991) have not been observed in patients fed relatively large protein intakes of approximately 1.5–2.0 g protein equivalent/kg IBW per d. N balance seems to be similar whether less or more energy is infused (Burge et al. 1994; Choban et al. 1997; McCowen et al. 2000; Dickerson et al. 2002). RQ indicates adipose tissue metabolism on hypoenergetic feeds (Burge et al. 1994). Clinical outcome in all these studies tends to be similar between groups, although the hypoenergetic group does better in one non-randomized study (Dickerson et al. 2002). In most cases it is safe, therefore, to feed at energy levels below resting energy expenditure in the short to medium term. No controlled studies compare the outcome of patients fed at estimated resting energy expenditure with that of patients fed at estimated or measured total energy expenditure.

Nitrogen intake

Greenberg & Jeejeebhoj (1979) have shown that a hypoenergetic intake of only 2·1 MJ (500 kcal)/d normal-weight individuals are in negative N balance when 1 g protein/kg per d is supplied, but could go into positive N balance on intakes of ≥1·5 g protein/kg. The controlled trials supporting hypoenergetic feeding mentioned earlier (Table 3) have all used approximately 2 g protein/kg IBW per d. While energy intakes close to the total or resting levels of energy expenditure may require lower inputs of protein, it seems wise to provide at least 1·5 g protein/kg IBW per d if energy is undersupplied.

Artificial feeding: special problems in obesity

Obesity carries an increased risk of oesophageal reflux (Nilsson et al. 2003), particularly in women, which will present an increased risk of aspiration pneumonia and may be of particular importance during enteral feeding of the obese. Patients should be nursed at 30° from the horizontal to minimize this risk. In consideration of this problem it may be wise to feed obese patients post-operatively through a needle catheter jejunostomy. Sarr (1999), for example, has presented a series of such patients with generally good results, although there have been rare reports of intestinal necrosis (Schunn & Daly, 1995) and other more minor complications, including small bowel obstruction, pneumatosis intestinalis, diarrhoea (15%) and tube occlusions.

Although diabetes may result in an autonomic neuropathy and is, therefore, a risk factor for delayed gastric emptying, the gastric emptying of obese patients in general is not slower than normal and does not present a special problem. Gastric emptying is, of course, delayed in most critically-ill patients.

Conclusions

With the high prevalence of obesity in the general population it is inevitable that artificial feeding will be needed from time to time in the obese patient, particularly in the critical care setting. Emphasis is placed on avoiding overfeeding against the background of the insulin resistance typical of the obese state and worsened by stress. Calculation of energy requirements is often difficult in practice in the obese patient and there is substantial evidence of the safety of feeding at or below resting energy expenditure. If this approach is adopted it is probably wise to provide a more generous than normal protein intake. The present paper argues for feeding obese critically-ill patients at or a little below the estimated BMR, taking care to avoid mineral and micronutrient deficiency while being cautious of reflux.

References


