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BAPEN Symposium 1: Malnutrition in obesity

The management of short-term intestinal failure in obese patients*

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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.

Obesity: Nutritional support: Intestinal failure: Critical illness

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 viscerally-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 α_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

Abbreviation: IBW, ideal body weight.
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Table 1. BMR in 55-year-old lean (70 kg) and obese (100 kg) male subjects

	Lean male		Obese male			
	1·78 m, BMI 22 kg/m^2		1·78 m, BMI 32 kg/m^2		1·83 m, BMI 30 kg/m^2	
	MJ	kcal	MJ	kcal	MJ	kcal
Schofield* (11·4 × W + 870)	7·0	1668	8·8	2010		
Harris Benedict† (66 + 13·7W + 5 H − 6·8A)	6·4	1541	8·1	1952	8·2	1977

W, weight (kg); H, height (cm); A, age (years).
*Schofield (1985).
†Harris & Benedict (1919).

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 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 CO_2 production (Guenst & Nelson, 1994) and refeeding syndrome, in which hypophosphataemia, hypokalaemia, hypomagnesaemia (Hearing, 2004) and acute deficiency of thiamin (Anonymous, 1997) complicate the early phases of nutritional support in the undernourished individual. Hyperglycaemia, in particular, must be prevented in critically-ill patients by precise insulin administration in order to minimize mortality (van den Berghe *et al*. 2001). 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.

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 advantages. Table 1 shows worked examples for non-obese and obese males, and shows how the Schofield equation tends to predict an energy requirement that is slightly higher than that predicted by the Harris-Benedict equation. Percentage increments over basal values are then added to allow for stress, using stress factors such as those used by Van Lanschot *et al*. (1986): elevated temperature per °C >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.

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

	Normal 70 kg male	Obese 100 kg male
Fat (kg)	12 (17%)†	35
FFM (kg)	58	65
Water (kg)	42 (60%)†	47
Protein (kg)	12 (17%)†	13

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.

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) × 6); female, 100 + ((height (in) − 60) × 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/m^2 is not strictly correct.

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Table 3. Controlled studies of hypoenergetic feeding

Reference	Patient population	Control			Hypoenergetic feeding		
		n	Protocol	Outcome	*n*	Protocol	Outcome
Burge *et al.* (1994)	Obese TPN Approximately 10 d	7	100% REE, 2 g protein/kg IBW	Mean N balance + 2·8 g	9	50% REE, 2 g protein/kg IBW	Mean N balance + 1·3 g (NS)
Choban *et al.* (1997)	Obese: mean BMI 35 (range 26–47) kg/m^2 TPN	14	8·1 MJ (1939 kcal)/d 108 g aa/d	Mean N balance + 3·6 g Period on insulin 8 d	16	5·4 MJ (1290 kcal)/d 120 g aa/d	Mean N balance + 4·0 g Period on insulin 3 d
Dickerson *et al.* (2002)	Obese Multiple trauma Not randomized Enteral	12	0·10–0·13 MJ (25–30 kcal)/kg, 25% adjusted wt 2 g protein/kg IBW	Hospital 37·2 d ICU 28·5 d* Antibiotics 27·4 d* Deaths 1	28	< 0·08 MJ (<20 kcal)/kg adjusted wt 2 g protein/kg IBW	Hospital 29·6 d ICU 18·6 d* Antibiotics 16·6 d* Deaths 0
McCowen *et al.* (2000)	Non-obese TPN	23 19†	5·0 + 0·9 MJ (1192 + 212 kcal)/d 89 + 13 g aa/d	LOS 17 (SD 15) d Deaths 3 N balance −0·6 (SD 4·8)*	25 21†	3·8 + 0·4 MJ (913 + 90 kcal)/d 70 + 0·2 g aa/d	LOS 19 (SD 14) d Deaths 2 N balance −8·3 (SD 9·2)*

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.

More recently, studies (Glynn *et al.* 1999; Barak *et al.* 2002) using indirect calorimetry have suggested that the IBW + 50% (actual body weight – IBW) reflects more appropriately the measured total energy consumption once standard stress factors are employed. 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 value + 50%, + stress factor, and with the Ireton-Jones equations (see Glynn *et al.* 1999) for (a) obesity and (b) hospitalized patients plus an allowance of 88 kJ (21 kcal)/kg. It was found that the 88 kJ (21 kcal)/kg allowance is not an accurate predictor and the Harris-Benedict value + 50% is clearly superior to the Ireton-Jones equations when consideration is made for whether or not the patient is ventilated. It is suggested that indirect calorimetry, if possible, is most appropriate for use in assessing energy requirements, while calculated BMR based on IBW + 50% of the difference between IBW and actual body weight is the most precise technique for estimation if standard stress factors are to be used to predict total energy expenditure.

In the UK very few of the hospital units have the resources of personnel or equipment to offer routine indirect calorimetry for artificially-fed obese patients. The question arises, therefore, as to whether calorimetry is necessary for excellent practice. BMR increases as a function of lean body mass and excess adipose tissue is approximately 25% lean. Thus, increments in weight above IBW result in only small increments in lean body mass (Table 2), and increments in BMR may be of little importance clinically, especially since obese patients have excess endogenous energy stores that they can afford to lose. The patients for whom the greatest care is needed are critically-ill patients for whom it may be very difficult to assess body weight or IBW accurately. One approach, based on the Horgan & Stubbs (2003) reassessment of the Schofield (1985) database would be to assume a BMR of 7·3 MJ (1750 kcal) for males >80 kg and 6·3 MJ (1500 kcal) for females >80 kg, with correction using standard stress factors. Another approach would be to intentionally under-supply energy in these patients at levels relating to the approximate BMR (no stress factors) or less.

Hypoenergetic feeding: the evidence

An important study (Jeevanandam *et al.* 1991) in patients with multiple trauma soon after admission and given crystalloid infusions has compared the metabolic responses of obese patients (*n* 7, mean weight 103 kg) with non-obese patients (*n* 10, mean weight 78 kg). Although they were heavier, the measured energy expenditure (mean 10·63 MJ (2550 kcal)/d) of the obese patients was found to be remarkably similar to that of the non-obese patients (10·58 MJ (2538 kcal)/d), with the mean RQ being similar for both groups at 0·80. As expected, both groups were reported to exhibit insulin resistance, with elevated blood levels of insulin and glucose, but with C-peptide levels for obese patients of about twice those of the non-obese patients (4·1 ng/ml *v.* 2·1 ng/ml), indicating a greater

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insulin secretion in the obese patients. Catecholamine 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 & Jeejeebhoy (1979) have shown that on 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.

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