Nutrition in the surgical patient

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Malnutrition is a frequent concomitant of surgical illness. Indeed, several surveys have shown that protein-calorie malnutrition is present in about 50 per cent of surgical patients.^{1,2} Furthermore, in those patients identified as severely malnourished preoperatively, the incidence of post-surgical complications has been identified as being up to six times that of well-nourished patients.³ If one adds to this group those patients presenting with normal nutritional status who, because of trauma or sepsis become hypermetabolic and hypercatabolic, the magnitude of the challenge of surgical nutrition becomes clear.

Effects of malnutrition on the surgical patient

The impact of malnutrition is widespread. Many aspects of immune function are adversely affected, including intracellular bacterial killing by neutrophils, absolute T₄ helper cell numbers and function, complement levels and reticulo-endothelial cell activity. Wound healing is impaired and this is reflected in an increased incidence of wound dehiscence and anastomotic breakdown.⁴ In addition to these general effects of malnutrition, there are also striking changes in cardiorespiratory function. Myocardial mass is reduced, reflected histologically in muscle fibre atrophy and even frank myofibrillar degeneration. As a result stroke volume and cardiac output are decreased. Since starvation results in decreased oxygen consump-

tion some of these changes may simply be adaptive. However, there is the potential for cardiac dysfunction in the malnourished patient with superimposed surgical stress or sepsis. The implications of cardiac cachexia for patients undergoing cardiac surgery as well as the problems of refeeding have recently been well reviewed.^{5,6}

In terms of the respiratory system, malnutrition also has a multifactorial impact.⁷ Hypoxic ventilatory drive is diminished, as is vital capacity. Surfactant production is decreased and hence maintenance of functional residual capacity is impaired. In addition to the general effects of starvation on immune function, secretory IgA concentration and pulmonary alveolar macrophage functions are diminished. Also, diaphragm muscle mass and contractility are reduced in the underweight patient.⁸ Not surprisingly, therefore, pulmonary complications such as pneumonia and respiratory failure are common.

Indications for nutritional support

Commonly accepted indications for nutritional support are:

- 1 Ten per cent or greater reduction in lean body mass,
- 2 An anticipated period of nothing per mouth $\geq 4-5$ days,
- 3 Stress state resulting in hypermetabolism and hypercatabolism.

Nutritional assessment

Although a wide variety of objective nutritional assessment tools is available (Table I), a careful history of weight loss, changes in appetite, and GI tract disturbances combined with physical examination have been shown to correlate well with more objective tests of nutritional status.⁹ In this study, patients graded on clinical grounds as having severe malnutrition had a significant increase in infections as compared to those who were deemed to have normal nutrition. There is one caveat, however, to the use of clinical assessment alone and this concerns the morbidly obese patient. Shizgal *et al.*,¹⁰ using body consumption studies have shown an 18 per cent incidence of protein malnutrition in this population. Therefore, the possibility of underlying protein malnutri-

TABLE I Objective tests of nutritional status

Anthropometrics

- Creatinine-height index
- Triceps skin fold
- Mid-arm circumference

Biochemistry

- Serum albumin
- Transferrin
- Pre-albumin
- Retinol-binding system

Immune status

- Total lymphocyte count
- Delayed cutaneous hypersensitivity

Body composition

- Total body nitrogen
- Exchangeable potassium

Muscle function tests

- Nerve stimulation studies

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TABLE II Enteral feeding products - Vancouver General Hospital ICU 1986

Formula	Kcal·ml ⁻¹	Osmolality	Nitrogen g·L ⁻¹	Na mEq·L ⁻¹	K mEq·L ⁻¹	
<i>Meal-replacement</i> Osmolite HN®	1.06	310	7.1	40.4	40.0	<ul style="list-style-type: none"> - standard formula for stressed patients - isotonic - suitable for jejunal feeding
<i>Calorie-dense</i> Ensure Plus®	1.50	600	8.8	47.8	48.6	<ul style="list-style-type: none"> - more nutrition in less volume for hypermetabolic or fluid restricted patients - hyperosmolar - for volume restricted patients, lower potassium - hyperosmolar
Magnacal®	2.00	590	11.2	44.0	32.0	<ul style="list-style-type: none"> - for volume restricted patients, lower potassium - hyperosmolar
<i>Chemically defined</i> Vital HN®	1.00	460	6.7	20.0	34.0	<ul style="list-style-type: none"> - elemental formula for patients with malabsorption, maldigestion disorders (pancreatitis, SBS, severe IBD, fistulae) - more expensive - moderately hyperosmolar
<i>Added fibre</i> Enrich®	1.10	480	6.4	37.0	40.0	<ul style="list-style-type: none"> - for patients on long term tube feeding who may benefit from added fibre - slowly advance strength then volume - initially abdominal distention and flatus may occur - contains 14 g dietary fibre/litre (soy polysaccharides)

tion in the obese patient, who may appear to most observers to be more than adequately nourished, should always be considered.

Feeding routes

Once it is established that nutritional support is required, consideration should next be given to determining the route of administration. Enteral feeding should be used whenever possible. It has the advantages of being cheaper and more physiological than total parenteral nutrition (TPN). Small-bore silastic feeding tubes can be placed via the nasogastric or nasoduodenal route and are comfortable and well-tolerated by patients. Alternative routes for enteral feeding are surgically placed gastrostomy or jejunostomy tubes. Newer techniques permit the percutaneous insertion of feeding gastrostomy tubes with the use of upper GI endoscopy.¹¹ Feeding tubes placed distal to the pylorus should be used for patients in whom airway protection is a problem, to reduce the risk of reflux and aspiration. In our experience, enteral feeding is feasible even in critically ill patients and, in fact, over 80 per cent of our ICU patients are supported enterally. There is also preliminary evidence in a burn injury rat model that early enterogastric feeding results in less weight loss and better preservation of gut mass compared with intravenous

feeding.¹² It is theorized that preserving gut mucosal integrity reduces bacterial translocation across the gut wall, thereby attenuating the hypermetabolic response.

TPN should be used when the GI tract is not accessible or is functioning poorly. In patients with severe inflammatory bowel disease a period of TPN to permit "bowel rest" may be of value. TPN may take the form of peripheral intravenous nutrition in patients with modest protein-calorie requirements, particularly when only a short course of support is anticipated. For hypercatabolic, hypermetabolic patients, or patients requiring long-term TPN, central access becomes necessary. For this purpose, a percutaneously placed, silastic or polyurethane catheter is employed. For long-term use, a tunnelled silastic catheter (Broviac® or Hickman®) is preferred.

Feeding solutions, protocols and complications

The principles of enteral feeding include:

- 1 Selection of an enteral formula suitable to the patient's needs (Table II).
- 2 Commencement of formula at an isotonic strength and a slow hourly rate.
- 3 A gradual increase in the concentration or volume of the feed at 12-24 hour intervals.

- 4 Final feeding volume should provide for the estimated calorie and nitrogen requirements of the patient.
- 5 Use of continuous feeding (either by gravity or pump), to reduce risk of aspiration and dumping.

For patients with unique requirements, such as medium chain triglycerides or high nitrogen intake, a modular diet can be prepared with the assistance of an experienced nutritionist. The major problems encountered with enteral feeding are poor gastric emptying and diarrhoea. A trouble-shooting protocol is invaluable in minimizing these complications (Table III). Inadvertent malposition of the feeding tube can be avoided by obtaining x-ray confirmation of tube placement before feeding is begun.

Macronutrient calorie sources for TPN are hypertonic glucose and lipid. Lipid solutions available are soy-bean and/or safflower oil based and are marketed as 10 or 20 per cent mixtures. These contain $1.1 \text{ Kcal} \cdot \text{ml}^{-1}$ and $2.0 \text{ Kcal} \cdot \text{ml}^{-1}$ respectively and are nearly isotonic compared with 50 per cent glucose which has an osmolality of $2526 \text{ mosm} \cdot \text{kg}^{-1}$. Intravenous glucose solutions provide $3.4 \text{ Kcal} \cdot \text{g}^{-1}$ of glucose. The complications of TPN can be divided into technical, septic, and metabolic categories. Technical complications include all the potential complications of achieving central venous access, with the additional risks of thrombosis of the great veins, catheter dislodgement and embolization. Infectious complications are most commonly due to gram positive organisms, *S. aureus* and *S. epidermis*, and fungi such as *Candida*. In the neutropenic host, a variety of less common pathogens can also be responsible for catheter sepsis. Metabolic complications include hyper- and hypoglycaemia and electrolyte imbalance, cholestatic jaundice, essential fatty acid deficiency and, if micronutrient needs are not met, a variety of vitamin and trace mineral deficiency states. This topic has been reviewed in detail.^{4,13,14}

Prescribing macronutrients for the starvation-adapted and the stressed patient

Determining optimal calorie-nitrogen intake requires an understanding of the differences between the "starvation-adapted" and the stressed, critically ill patient. The chronically malnourished individual is "starvation adapted" in that nitrogen losses are minimized and energy expenditures are reduced to below normal values. By contrast, the critically ill individual has increased nitrogen losses and an increased energy expenditure. Catecholamine, glucagon and cortisol levels are high. Hepatic gluconeogenesis proceeds at an accelerated rate, despite peripheral hyperglycemia and undesirable muscle catabolism takes place to meet the demand for increased amounts of gluconeogenic amino acids. The "starvation-adapted" patients responds predictably to exogenous substrate administration with a gratifying improvement in nitrogen and energy

TABLE III Enteral feeding trouble-shooting protocol - Vancouver General Hospital ICU/Burn Unit

Poor gastric emptying (high residuals)

- If no contraindications, start on metoclopramide (Maxeran®) 10 mg IV q 6 hours.
- Consider changing to a naso-jejunal tube (Cartmill®). Administer 10 mg IV Maxeran® 30 minutes before tube placement. In most patients the tube will pass through the pylorus within 24-36 hours. If it does not, endoscopic or fluoroscopic placement may be required.
- Rule out severe hypomagnesaemia or hypoproteinaemia as a cause for gastric ileus.

Diarrhoea

- Check equipment is clean and formula is not contaminated.
- Rule out impaction causing overflow incontinence.
- Check that patient is not receiving metoclopramide.
- Consider other medications or antibiotics that may cause diarrhoea. Also, hypoalbuminaemia ($<2.5 \text{ gm\%}$ albumin) may contribute to poor absorption of formula.
- If diarrhoea continues, reduce feed concentration (i.e., full strength to $\frac{1}{2}$ strength).
- Persistent diarrhoea requires re-assessment to rule out subacute bowel obstruction, gut ischaemia, pseudomembranous colitis (obtain abdominal x-rays, stool for C&S, *C. difficile* culture and toxins, consider sigmoidoscopy).
- If no other cause for diarrhoea is found consider adding codeine, Imodium® or Lomotil®. Addition of a bulk laxative or a high fibre formula may also be tried. Discontinue feed only as a last resort.

balance. Such is not the case with a septic or traumatized patient. Burke *et al.*¹³ have shown that increasing glucose infusion rates above $5 \text{ mg} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ in burn patients does not increase glucose oxidation or protein synthesis. Rather, the respiratory quotient increases and fatty liver infiltrates are observed. The increased CO_2 production shown in stressed patients with hypercaloric glucose infusion may also contribute to the development of respiratory failure or difficulty in weaning from mechanical ventilation.¹⁶⁻¹⁸ In advanced degrees of stress, particularly sepsis, lipid clearance and utilization is also impaired¹⁹ and indeed, ultimately branched chain amino acids become utilized as an energy source.²⁰ This phenomenon, referred to as "sequential fuel failure" has led to an interest in other energy sources in severe sepsis.²¹

A commonly used method for estimating calorie requirements is the Harris-Benedict equation for determining basal energy expenditure (BEE):

$$\text{BEE (male)} = 66 + 13.7 (\text{weight in kg}) + 5 (\text{height in cm}) - 6.8 (\text{age in yr})$$

$$\text{BEE (female)} = 655 + 9.6 (\text{weight in kg}) + 1.8 (\text{height in cm}) - 4.7 (\text{age in yr})$$

In the chronically depleted patient, repletion can then be accomplished by feeding approximately 1.5 times the resting energy expenditure ($\text{REE} = \text{BEE} \times 1.1$). In the

stressed patient, if at all possible REE should be measured, using indirect calorimetry. For this purpose, systems for continuous expired gas analysis such as the Metabolic Measurement Cart (Sensor Medics®) and the Siemens-Elema® Servo Ventilator 900-series are employed. Use of these systems in ventilator-dependent patients requires great care to obtain accurate, steady-state, reproducible data.²² For example, at high inspired O₂ concentrations (FiO₂ ≥ 0.5–0.6) oxygen consumption may not be accurate. Recent experience with indirect calorimetry in critically ill patients has led to the conclusion that total energy expenditure in stress is not as greatly increased as previously thought. In fact, in patients who are sedated and at rest, energy expenditure may be lower than predicted from the Harris-Benedict equation. Alternatively, septic and traumatized patients may have energy expenditures exceeding those predicted.²³

In practical terms, critically ill patients can most safely be fed using a mixed fuel source (50:50 glucose:lipid) to meet energy needs. Ideally REE should be measured with indirect calorimetry. If the respiratory quotient exceeds 1.0, suggesting net lipogenesis or if CO₂ production increases, glucose intake should be reduced. If indirect calorimetry is not available, it is usually safe to target calorie intake at predicted Harris-Benedict REE. If the latter approach is used, minute ventilation and pCO₂ should be followed. An increase in either of these parameters may reflect "overfeeding" with an excess of glucose calories. To monitor lipid clearance, triglyceride levels or clearance studies should be performed at regular intervals.

Positive nitrogen balance can be obtained in unstressed chronically malnourished patients with 0.1 g to 0.15 g nitrogen · kg⁻¹. In stress, it is recommended that nitrogen intake be increased to 0.3 g to 0.5 g nitrogen · kg⁻¹.²⁴ Even at these high nitrogen intakes, the accelerated proteolysis seen in trauma and sepsis may make it impossible to achieve positive nitrogen balance. Use of new enteral and parenteral solutions high in branched-chain amino acid has met with some success in achieving positive nitrogen balance in stressed patients.^{25,26} The recent recognition of a circulating proteolytic factor,^{27,28} which may be interleukin-1 may lead in the future to an ability to attenuate the accelerated proteolysis seen in stress.

Nutrition in the perioperative period

In preparation for surgery, enteral feeding is usually discontinued six to eight hours preoperatively. If feeding is taking place distal to the pylorus and intra-abdominal surgery is not involved, it is theoretically possible to continue enteral feeding through the perioperative course. Difficulties in monitoring formula infusion rates intraoperatively usually makes this impractical, however.

TPN can and should be maintained intraoperatively. The specific requirements of the TPN patient intraoperatively are:

- 1 Maintenance of line patency:
 - infusion pumps
 - avoid admixture of anaesthetic drugs with TPN solutions
- 2 Prevention of hypoglycemia:
 - maintain preop infusion rates
 - do not discontinue hypercaloric glucose infusion
 - monitor with Glucometer® q 1–2 hours
- 3 Maintenance of strict aseptic technique

Other considerations

Efforts should be made to maintain normal body temperature intraoperatively, thereby minimizing shivering, CO₂ production and proteolysis postoperatively.²⁹ It has also recently been shown that TPN can alter rates of hepatic drug oxidation.^{30,31} More investigation will be required before any modifications of drug therapy are recommended.

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