Feeding the patient who cannot eat

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The importance of nutritional support of the hospitalised patient is being increasingly recognised. Patients are frequently malnourished, with a recent study performed at Groote Schuur Hospital indicating that more than 40% of patients admitted for surgery were nutritionally depleted (C Hartman, 2000 — unpublished data). These figures are in agreement with published data from other centres where malnutrition rates approaching 50% of hospitalised patients have been reported.[1-6] Of particular concern was that nutritional status was not adequately appreciated in 40% of these patients, and they did not receive additional nutritional support while in hospital.

Although many patients are malnourished on admission to hospital, the nutrition status of patients frequently deteriorates during their stay as a consequence of poor hospital diet, the withholding of meals due to diagnostic tests or pre- and post-surgery, and inadequate recognition by hospital staff of the increased nutritional needs due to illness and injury.[7,8] Butterworth[3] commented that malnutrition may actually be induced during hospitalisation, and that hospital stays are frequently prolonged by 'neglect of the patient's nutritional health'.

An impaired nutritional status is likely to impact adversely on the patient's clinical condition. Studies have indicated a close relationship between physiological function and lean body mass. Significant physiological impairment with increased postoperative complications and a prolonged hospital stay are associated with a 20% loss of body protein.[4] A 10% body weight loss relates to a loss of between 15.2% and 20.8% body protein, 15% weight loss to between 19.2% and 24.8% protein loss, and 20% body weight loss to between 23.0% and 29.0% protein loss. Therefore, in previously healthy individuals, a weight loss of 15% would relate to a body protein loss of approximately 20%, and a significantly increased risk of postoperative complications. However, John Kinney has previously reported that physiological derangements begin to appear at between 5% and 10% body weight loss, and in the presence of disease such derangements are likely to be clinically significant.[9]

It is therefore imperative that hospitalised patients receive adequate, appropriate nutrition, and it is the responsibility of

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the hospital staff to ensure that this occurs. The average patient requires between 25 and 30 kcal/kg/day energy and 0.6 - 1.5 g/kg/day protein, while patients with severe injury or burns require substantially more. If this requirement is not provided for, deterioration in nutritional status will occur.

Difficulties encountered in attempting to re-feed patients

Difficulties in providing adequate nutrition to patients generally result from psychological, physiological, or mechanical problems.

Psychological problems

It is important to appreciate the psychological consequences of disease and malnutrition. In the classic studies of Ancel Keys and colleagues[10] published in 1950, nutritional deprivation for a period of 24 weeks resulted not only in significant deterioration in physiological functions such as muscle strength, but also in psychological derangements such as depression, anxiety, introversion and apathy.[10,11] Despite nutritional restitution following 20 weeks of re-feeding, the subjects remained significantly depressed and demotivated. When considered together with the psychological consequences of illness, as well as the generally unappetising nature of hospital diets, it is not surprising that patients are often unable to maintain an adequate nutritional intake unassisted, and it should never be presumed that they are able to do so. In the words of Florence Nightingale during the Crimean War, 1859:

'Every careful observer of the sick will agree in this. That thousands of patients are annually starved in the midst of plenty, from want of attention to the ways which alone make it possible for them to take food. ... I would say to the nurse, have a rule of thought about your patient's diet; consider, remember how much he has had, and how much he ought to have today.'

Nutritional support of patients, especially of those who have lost in excess of 10% of their body weight, has to be an active process, and often requires the use of continuous fine-bore nasogastric feeding to ensure an adequate supply of nutrients.

Physiological problems

Physiological derangement of digestive function may result from: (*i*) premucosal defects; (*ii*) mucosal defects; or (*iii*) postmucosal defects.

Premucosal

Premucosal defects result in abnormalities in the digestion of ingested nutrients. This may be a consequence of digestive enzyme deficiency, bacterial overgrowth, or abnormalities in gastro-intestinal emptying, such as following gastrectomy.

Pancreatic enzyme deficiency. This may be due to chronic pancreatitis or cystic fibrosis (children). Therapy involves the use of adequate doses of pancreatic enzyme supplements. Severe malnutrition has also been associated with significant impairment of pancreatic enzyme secretion.[12] Re-feeding may initially require the use of semi-elemental (predigested) formulas, and results in a reasonably rapid restitution of pancreatic function.[12]

Small-bowel bacterial overgrowth. The small bowel is usually 'sterile' of bacteria. However, disorders of small-bowel motility due to diseases such as diabetes and scleroderma, as well as in the case of strictures and obstruction (such as in Crohn's disease), the small bowel may become inhabited by a variety of bacterial strains. Small-bowel bacterial overgrowth is defined as a colony growth of greater than 1 x 105/ml. Bacteria most commonly isolated include *Escherichia coli, Bacteroides* spp. and *Enterobacter* spp. The presence of the bacteria may affect digestion as a result of the deconjugation of bile salts, as well as affecting secretion and motility as a consequence of toxins produced resulting in diarrhoea. The bacteria also ferment carbohydrates generating short-chain fatty acids, which may exert a cathartic effect in the small bowel.[13] Management of the condition involves correction of the underlying disorder (such as adequate control of diabetes, and treatment of strictures and obstruction). Intermittent courses of antibiotics may also be required.

Postgastrectomy 'dumping' syndromes. Fortunately, with the advent of *Helicobacter pylori* eradication regimens for peptic ulcer, and the subsequent marked reduction in recurrent, complicated peptic ulcer disease, most of the previous indications for gastrectomy have now been avoided, and complications of the surgery are becoming increasingly uncommon. The main indication for gastrectomy now is malignant disease. The symptoms of the postgastrectomy syndrome relate to the presence of a small stomach, and the rapid entry of hypertonic gastric contents into the small bowel following a meal. The syndrome is classically divided into an 'early phase', characterised by dizziness consequent to the rapid fluid shifts into the bowel as a result of the hyperosmotic load, and a 'late phase' characterised by hypoglycaemia consequent to the hyperinsulinaemic response to the absorbed carbohydrate in the meal. Management involves frequent small meals, the avoidance of fluids with meals, and also consideration for correctional surgery.

Mucosal

Mucosal defects relate to abnormalities in gut mucosal structure resulting in malabsorption. These may be consequent to villous atrophy, inflammation or insufficient gut length due to surgical resection (short bowel syndrome).

Villous atrophy. Villous atrophy may be as a result of conditions such as coeliac sprue, tropical sprue, immunoproliferative small-intestinal disease and Whipple's disease, as well as a consequence of malnutrition. Following gastro-enteritis the villi may also be damaged, with decreased secretion of the disaccaridases and secondary lactose intolerance. If severe, malabsorption and diarrhoea may occur as a result of the decreased mucosal surface area. Therapy is directed at management of the underlying condition.

Inflammatory bowel disease. Inflammatory bowel disease has been associated with increased permeability and decreased absorption.[14] Enteric mucosal disease may result in diminished brush border enzyme activity, which is most likely to affect lactose, the disaccharide with the lowest mucosal hydrolase activity.[15] Approximately 10% of Crohn's disease patients in the UK are intolerant of milk products.[16] Crohn's disease has also been associated with excessive protein loss from the gut, mainly due to the loss of plasma proteins, rather than impaired protein digestion and absorption,[17] as well as with increased fat excretion. Disease of the distal ileum may interrupt bile salt recirculation, with impairment of duodenal fat emulsion and miscelle formation. Furthermore, bacterial overgrowth, frequently seen in patients with Crohn's disease, may derange fat digestion both by deconjugating bile acids and by toxic effects of bacterial products on the enteric mucosa.

Management strategies for patients presenting with acute, active inflammatory bowel disease previously involved keeping the patient nil per mouth for fairly extensive periods of time in order to 'rest the bowel'. Realisation of the frequent presence of significant malnutrition in these patients, as well as the catabolic nature of the disease and its therapy with steroids and possibly surgery, has led to the adoption of policies of early nutritional support. If the disease is colonic or non-obstructive in nature this can usually be achieved by the enteral route, usually with continuous fine-bore nasogastric feeding.

Short-bowel syndrome. The average length of small bowel in adult humans, measured at laparotomy, has been reported to be 500 cm (range 302 - 777 cm).[18] Excessive resection of the small bowel, particularly if the ileum is removed, may result in the 'short-bowel syndrome' characterised by malabsorption, diarrhoea, and excessive fluid and electrolyte loss. The minimum length of small bowel required in order to maintain nutrition is variable between individuals, but is in the region of 100 cm. The importance of the colon in 'salvaging' not only fluid and electrolytes, but also energy in the form of short-chain fatty acids, cannot be overemphasised. It has been calculated that the presence of at least 50% of the colon is equivalent to about an additional 50 cm of small bowel.[19] Patients with less than approximately 150 cm small bowel may require fluid and electrolyte supplements, as well as supplementation with vitamins, particularly vitamin B_{12} and folate. Frequent small meals, which are low in fat and high in protein, are advised. Patients with short bowel, particularly if they have ileo/jejunostomies, should be advised to avoid drinking tap water owing to the loss of body electrolytes in the stomal effluent. An adequate fluid intake with isotonic 'sipping solutions' should be encouraged. With less than 100 cm small

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bowel (or 50 cm with an intact colon), patients may not be able to absorb sufficient calories to sustain life, and parenteral nutritional support may become necessary.

Postmucosal

Lymphangiectasia. Obstruction of the intestinal lymphatics may be a result of a congenital abnormality (primary lymphangiectasia), or due to involvement of either infection (tuberculosis) or neoplasm (lymphoma) (secondary lymphangiectasia). Lymphatic obstruction leads to leakage of fat and protein back into the gut, together with lymphocytes. This may result in a protein-losing enteropathic state with a low serum albumin. If the condition is localised, resection of the involved bowel may be curative. However, most cases are generalised and therapy is directed at reducing the enteric lymphatic flow by means of a low-fat diet, supplemented with medium-chain triglycerides, which are absorbed directly into the bloodstream.

Mechanical problems

Difficulty in swallowing

Difficulty in swallowing (dysphagia) may be a result of neurological abnormalities of the swallowing apparatus, or obstruction in the oesophagus. Painful swallowing (odynophagia) usually indicates an inflammatory/ infective process.

Neurological abnormalities. Bulbar palsy is usually consequent to cerebrovascular accident (stroke), or motor neuron disease. The neurological impairment, with inability to swallow adequately, results in a substantial risk of pulmonary aspiration of food, and in this situation normal oral intake is not advised. Nutritional intake is maintained, initially by the use of fine-bore nasogastric feeding. In the case of stroke, the impairment may improve over time, and the patient should be monitored carefully. Our policy is that if insufficient improvement has occurred over a period of approximately 1 month and the risk of aspiration remains high, then the patient must be considered for insertion of a percutaneous endoscopic gastrostomy (PEG). Motor neuron disease is a progressive condition, and once patients are experiencing difficulties with swallowing, PEG insertion should be considered.

Obstruction. Patients experiencing dysphagia should have a barium swallow performed at the initial investigation as obstructions due to oesophageal webs at the level of the crico-pharyngeus are frequently missed at endoscopy, and endoscopy may be hazardous in patients with pharyngeal pouches. Other causes of oesophageal obstruction include peptic strictures due to oesophageal reflux disease as well as due to malignant lesions.

Gut hypomotility

A period of 'ileus' is invariable following abdominal surgery, but usually resolves over the course of a few days. Prolonged ileus, however, frequently poses a problem for feeding. Motility in the small bowel generally resolves before that of the stomach, and feeding in these patients can often be achieved by the passage of a nasojejunal feeding tube. This should be considered before resorting to parenteral nutrition. Pseudo-obstruction or 'Ogilvie's syndrome' may occur, particularly in ill patients. The cause of this condition is frequently unknown; however, metabolic, fluid and electrolyte imbalances should always be considered and corrected.

Enteral feeding

Wherever possible the enteral route should be used, the advantages of this being reduced cost, avoidance of complications associated with central line placement, and prevention of gut mucosal atrophy, pancreatic atrophy, bacterial translocation and cholecystitis.

If the patient is unable to swallow sufficiently, other possible techniques include fine-bore nasogastric feeding and PEG insertion.

Fine-bore nasogastric/nasojejunal feeding

Passage of a fine-bore (8FG) nasogastric tube is generally well tolerated, and allows reliable provision of enteral feeding formulas, usually as a continuous infusion.

Percutaneous endoscopic gastrostomy (PEG)

If tube feeding is required for a prolonged period of time (generally more than 1 month), insertion of a PEG should be considered. This technique has the advantages of not only being more aesthetic in appearance, but also in the long term it is likely to offer an economic advantage. The larger diameter tube (24FG) allows use of liquidised normal foods given as boluses, rather than the considerably more expensive commercially produced liquid formulations frequently used with fine-bore feeding.

There are a variety of feeding formulas available, including polymeric diets, semi-elemental/elemental formulas, lactose-free and fibre-enriched feeds. Also making headway into the market are the so-called organ-specific formulas, such as branched-chain amino acid-containing feeds for liver dysfunction, and glutamine for small-bowel disease.

Parenteral nutrition

Intravenous feeding may be necessary when provision of adequate nutrition is not possible via the enteral route. Indications include disease, injuries or operations involving the face, mouth, oesophagus, stomach or small intestine, where oral feeding or the passage of a nasogastric feeding tube is not possible, or is considered undesirable. In exceptional cases undernourished patients may require parenteral nutrition in preparation for surgery, chemotherapy or radiotherapy. Parenteral nutrition may be given in addition to enteral feeding when gut function is borderline (supplemental parenteral nutrition), or may be the sole source of nutrition when gut

failure is present (total parenteral nutrition). It is usually indicated for only a short period of time to help tide the patient over a crisis, but in certain instances, such as patients with short-bowel syndrome or permanent gut failure, it may be required long term. In these circumstances the patient and family may be trained to administer the nutrition at home (home parenteral nutrition). There are, however, many potential complications associated with parenteral nutrition, with septic and mechanical problems related to the parenteral line, and physiological disturbances related to the bypassing of the gut. Parenteral feeding should therefore never be undertaken lightly, and should only be used for specific indications under the guidance of a specialised team.

Total parenteral nutrition requires the administration of a solution containing amino acids, glucose, fat, electrolytes, trace elements and vitamins. In practice, parenteral nutrition is administered as either a single 2 or 3 litre 2-in-1 solution containing amino acids and glucose, or with all three macronutrients, i.e. amino acids, glucose and fat provided in a 3-in-1 emulsion. Admixtures of electrolytes, trace elements and vitamins must be performed carefully under strict protocol, as incompatibility can occur resulting in embolism. The solution is administered to the patient via a dedicated central venous catheter inserted under full aseptic conditions. As all solutions are hypertonic, they should be given as slow continuous infusions, preferably controlled by a pump. Nutritional and electrolyte status must be monitored regularly throughout the period of treatment.

Protein

Nitrogen is given as mixtures of essential and non-essential synthetic L-amino acids (D-amino acids cannot be used for protein synthesis in man). Commercially available solutions vary in their compositions, and may contain an energy source (usually glucose) and electrolytes. Nutritional requirements vary, but the amino acid source should provide 0.1 - 0.24 g nitrogen/kg body weight/day (equivalent to 0.6 - 1.5 g protein/kg/day).

Carbohydrate

Glucose is the preferred carbohydrate, and as it is administered as a hypertonic solution, a central venous line is necessary to prevent thrombosis. Patients will often tolerate up to 400 g of glucose a day but are usually restricted to below 300 g/day to avoid the risk of fatty liver. Monitoring of blood glucose is mandatory. Blood glucose is often mildly elevated (up to 15 mmol) but the addition of insulin to the infusion is only required if higher levels are found.

Fat

Fat is usually given as an emulsion of soya bean oil. This has the advantages of high energy-to-volume ratio, iso-osmolarity with plasma and a neutral pH, and provides essential fatty acids. Fat emulsions should be restricted in patients with pre-existing hyperlipidaemia and daily checks

are necessary to ensure complete clearance from the plasma in such patients. Incomplete clearance of the fat emulsion may theoretically result in immunoparesis as well as interference with certain biochemical measurements, such as blood gases and calcium. Febrile 'hypersensitivity' reactions are rare. Additives are only mixed with fat emulsions where compatibility is known. Intralipid should not be stored frozen, and 3-in-1 mixtures should be used soon after production as the emulsion is potentially unstable. The minimum parenteral nutrition requirement is 500 ml twice weekly.

Total energy requirements

Energy requirements must be met in order that infused amino acids are preferentially utilised for protein synthesis and tissue maintenance. Most hospital patients require no more than 25 - 30 kcal/kg/day (105 - 126 kJ/kg/day), but in the case of patients with severe burns, critical illness, sepsis and over- or undernutrition it is advisable to measure energy expenditure by indirect calorimetry and balance losses. A mixture of glucose and fat (containing 30 - 50% fat) appears to be the best energy source, having the greatest nitrogen-sparing effect. However, in view of expense and stability, glucose is generally used as the major energy source.

One should be careful to avoid overzealous nutritional replacement with the subsequent development of the re-feeding syndrome, characterised by paraesthesia, weakness, seizures and cardiovascular failure. This may be a consequence of fluid overload, impaired metabolism of electrolytes and glucose, vitamin deficiency and hypophosphataemia.

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