

GUIDELINES

Guidelines for enteral feeding in adult hospital patients

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1.0 FOREWORD

Patients with undernutrition to a degree that may impair immunity, wound healing, muscle strength, and psychological drive are common in UK hospital populations.¹ These individuals cope poorly with modern medical and surgical interventions and, on average, stay in hospital for approximately five days longer than the normally nourished, incurring approximately 50% greater costs.^{2,3} Hospitals should therefore aim to provide at least adequate nutrition to all patients. In the majority, this can be achieved by the catering services if they offer good food and care is taken to avoid missed meals and to provide physical help with eating, as necessary. However, even if these ideals are met, many hospital patients do not or cannot eat adequately. Some of these will benefit from oral supplements but others will need active nutritional support. This can usually be provided by enteral tube feeding (ETF).

This document contains guidelines covering the indications, benefits, administration, and problems of ETF in adult hospital practice. The guidelines were commissioned by the British Society of Gastroenterology (BSG) as part of an initiative in several areas of clinical practice. They are not rigid protocols and should be used alongside clinical judgement, taking local service provision into account.

2.0 FORMULATION OF GUIDELINES

These guidelines were compiled from the relevant literature by the authors in discussion with dietitians and specialist nutrition nurses. They were subsequently reviewed by the BSG small bowel/nutrition committee and dietetic, nursing, pharmacy, and medical representatives of the British Association of Parenteral and Enteral Nutrition (BAPEN). The strength of evidence used is as recommended by the North of England evidence based guidelines development project.⁴

Ia—Evidence obtained from meta-analysis of randomised controlled trials.

Ib—Evidence obtained from at least one randomised trial.

IIa—Evidence obtained from at least one well designed controlled study without randomisation.

IIb—Evidence obtained from at least one other type of well designed quasi experimental study.

III—Evidence obtained from well designed non-experimental descriptive studies such as comparative studies, correlation studies, and case studies.

IV—Evidence obtained from expert committee reports or opinions or clinical experiences of respected authorities.

Unfortunately, many aspects of ETF have not undergone rigorous evaluation, partly because ethical considerations make placebo controlled trials of any nutritional intervention difficult (see section 4.2). Nevertheless, recommendations based on the level of evidence are presented and graded as:

- grade A—requiring at least one randomised controlled trial of good quality addressing their topic of recommendation;
- grade B—requiring the availability of clinical studies without randomisation on the topic of recommendation;
- grade C—requiring evidence from category IV in the absence of directly applicable clinical studies.

3.0 SUMMARY OF RECOMMENDATIONS

Indications for enteral feeding

- Health care professionals should aim to provide adequate nutrition to every patient unless prolongation of life is not in the patient's best interest (grade C).
- It should be hospital policy that the results of an admission nutritional screening are recorded in the notes of all patients with serious illness or those needing major surgery (grade C).
- Artificial nutrition support is needed when oral intake is absent or likely to be absent for a period >5–7 days. Earlier instigation may be needed in malnourished patients (grade A). Support may also be needed in patients with inadequate oral intake over longer periods.
- Decisions on route, content, and management of nutritional support are best made by multidisciplinary nutrition teams (grade A).
- ETF can be used in unconscious patients, those with swallowing disorders, and those with partial intestinal failure. It may be appropriate in some cases of anorexia nervosa (grade B).
- Early post pyloric ETF is generally safe and effective in postoperative patients, even if there is apparent ileus (grade A).
- Early ETF after major gastrointestinal surgery reduces infections and shortens length of stay (grade A)
- In all post surgical patients not tolerating oral intake, ETF should be considered within 1–2 days of surgery in the severely malnourished, 3–5 days of surgery in the moderately malnourished, and within seven days of surgery in the normally or over nourished (grade C).
- If there are specific contraindications to ETF, parenteral feeding should be considered. If patients are taking >50% of estimated nutritional requirements, it may be appropriate to delay instigation of ETF (grade C).

Abbreviations: ETF, enteral tube feeding; EN, enteral nutrition; PN, parenteral nutrition; BMI, body mass index; BSG, British Society of Gastroenterology; BAPEN, British Association of Parenteral and Enteral Nutrition; NG, nasogastric; NJ, nasojejunal; PEG, percutaneous endoscopic gastrostomy; PEGJ, percutaneous endoscopic transgastric jejunostomy; LCT, long chain triglyceride; MCT, medium chain triglyceride; SCFA, short chain fatty acid

- ETF can be used for the support of patients with uncomplicated pancreatitis (grade A).

Ethical issues

- ETF should never be started without consideration of all related ethical issues and must be in a patient's best interests (grade C).
- ETF is considered to be a medical treatment in law. Starting, stopping, or withholding such treatment is therefore a medical decision which is always made taking the wishes of the patient into account.
- In cases where a patient cannot express a wish regarding ETF, the doctor must make decisions on ETF in the patient's best interest. Consulting widely with all carers and family is essential.

Access techniques

- Fine bore (5–8 French gauge) nasogastric (NG) tubes should be used for ETF unless there is a need for repeated gastric aspiration or administration of high viscosity feeds/drugs via the tube. Most fibre enriched feeds can be given via these fine bore tubes (grade A).
- NG tubes can be placed on the ward by experienced medical or nursing staff, without *x* rays to check position. Their position must be checked using pH testing prior to every use (grade A).
- The position of a nasojejunal (NJ) tube should be confirmed by *x* ray 8–12 hours after placement. Auscultation and pH aspiration techniques can be inconclusive (grade A).
- NG tube insertion should be avoided for three days after acute variceal bleeding and only fine bore tubes should be used (grade C).
- There is no evidence to support the use of weighted NG tubes, in terms of either placement or maintenance of position (grade A).
- Long term NG and NJ tubes should usually be changed every 4–6 weeks swapping them to the other nostril (grade C).
- Gastrostomy or jejunostomy feeding should be considered whenever patients are likely to require ETF for more than 4–6 weeks (grade C) and there is some evidence that these routes should be considered at 14 days (grade B).
- Suitability for gastrostomy placement should be assessed by an experienced gastroenterologist or member of a nutrition support team. Expert advice on the prognosis of swallowing difficulties may be needed (grade C).
- In patients with no risk of distal adhesions or strictures, gastrostomy tubes with rigid internal fixation devices can be removed by cutting them off close to the skin, pushing them into the stomach, and allowing them to pass spontaneously (grade A).

Feed administration

- Giving enteral feed into the stomach rather than the small intestine permits the use of hypertonic feeds, higher feeding rates, and bolus feeding (grade A).
- Starter regimens using reduced initial feed volumes are unnecessary in patients who have had reasonable nutritional intake in the last week (grade A). Diluting feeds risks infection and osmolality difficulties.
- Both inadequate or excessive feeding may be harmful. Dietitians or other experts should be consulted on feed prescription (grade C).

- If no advice is available, 30 ml/kg/day of standard 1 kcal/ml feed is often appropriate but may be excessive in undernourished or metabolically unstable patients (grade C).
- When patients are discharged to the community on continuing ETF, care must be taken to ensure all community carers are fully informed and that continuing prescription of feed and relevant equipment is in place (grade C).

Complications of enteral feeding

- Close monitoring of fluid, glucose, sodium, potassium, magnesium, calcium, and phosphate status is essential in the first few days after instigation of ETF (grade C).
- Life threatening problems due to refeeding syndrome are particularly common in the very malnourished and there are also risks from over feeding shortly after major surgery or during major sepsis and/or multiorgan failure (grade C).
- To minimise aspiration, patients should be fed propped up by 30° or more and should be kept propped up for 30 minutes after feeding. Continuous feed should not be given overnight in patients who are at risk (grade C).
- Any drugs administered via an ETF tube should be liquid and should be given separately from the feed with flushing of the tube before and after (grade C).
- Loosening and rotating a gastrostomy tube may prevent blockage through mucosal overgrowth and may reduce peristomal infections (grade C).
- In patients with doubtful gastrointestinal motility, the stomach should be aspirated every four hours. If aspirates exceed 200 ml, feeding policy should be reviewed (grade C).
- Continuous pump feeding can reduce gastrointestinal discomfort and may maximise levels of nutrition support when absorptive capacity is diminished. However, intermittent infusion should be initiated as soon as possible (grade A).
- Simultaneous use of other drugs, particularly antibiotics, is usually the cause of apparent ETF related diarrhoea (grade A).
- Fibre containing feeds sometimes help with ETF related diarrhoea, as will breaks in the feeding of 4–8 hours (grade B).
- Careful measures are needed to avoid bacterial contamination of feeds which can give rise to sepsis, pneumonia, and urinary tract infections, as well as gastrointestinal problems (grade A).
- Avoiding gastric acid suppression and allowing breaks in feeding to let gastric pH fall will help prevent bacterial overgrowth during ETF (grade A).

4.0 BACKGROUND

4.1 Malnutrition in the UK

Recent studies in a nationally representative sample showed that undernutrition is common in UK adults in both community and hospital populations.^{1 5 6} Approximately 5% of apparently "healthy" UK adults were shown to have a body mass index (BMI) <20 m/kg² and this increased to 10% or more for the chronically sick and community patients with cancer, gastrointestinal disease, respiratory problems, and neurological or psychiatric conditions. In nursing homes, 16% of elderly residents were underweight.⁶

The prevalence of vitamin deficiencies in the population is even more disturbing. In individuals over 65 years living at home, low folate levels were found in 29% and low vitamin C

levels in 14%, with figures in the institutionalised elderly rising to 35% and 40%, respectively.⁶ Furthermore, most medical and surgical problems are accompanied by declines in nutritional status due to changes in the intake, metabolism, and excretion of nutrients. By the time patients are admitted to hospital, nearly 40% are malnourished in anthropometric terms (8% severely) and their nutritional status declines further during their hospital stay.¹

4.2 Evidence of benefit

It has long been considered unethical to withhold nutritional support in the malnourished and in those likely to become so (for example, intensive care unit and burns patients). Trials of support have therefore tended to recruit patient groups with no definite need, and frequently patients in “control” groups end up switching to active intervention as soon as they run into problems. Trials are also difficult to interpret due to varied levels of nutritional support, given via different routes in heterogeneous groups, and most older trials used levels of nutritional support so high that they caused hyperglycaemia (for example, the Veterans Administration trial of perioperative parenteral nutrition⁷). Despite this, a meta-analysis of oral/enteral nutritional support trials, in more than 2000 patients of all types, showed that the pooled odds ratio for death by the end of scheduled follow up showed a reduced case fatality in treatment compared with control groups of 0.66 (0.48–0.91; 2p<0.01).⁸ An extensive semi formal review of the literature on malnutrition and nutrition support in hospital also concluded that targeted nutritional support is of benefit in reducing hospital complications, duration of stay, mortality, and costs,⁹ and the Kings Fund reported that attention to malnutrition might save the NHS £266 million annually at 1992 prices¹⁰ (equating to at least £400 million savings at 2003 costs).

5.0 INDICATIONS FOR ENTERAL SUPPORT

ETF is only likely to benefit nutritionally depleted patients or those at risk of becoming depleted. These individuals need to be identified. At its simplest, nutritional screening involves consideration of a patient’s weight for height and recent history of weight loss. However, nutritional support should also be considered in all patients with excessive nutrient losses (for example, vomiting, diarrhoea, or fistulae) along with those who have high potential demands for nutrients (for example, surgical stress, trauma, infection, metabolic disease, bedsores). It should be hospital policy to record the results of nutritional screening in all patients suffering from serious illness or due for major surgery on or shortly after admission. Specific tools can be used for this purpose, the simplest of which is the malnutrition universal screening tool developed by BAPEN (appendix 1).¹¹ Although biochemical measurements can contribute to nutritional screening, none is specific (for example, a low albumin usually reflects an acute phase response rather than malnutrition).

Once risk is identified, nutritional help should be provided. Verbal encouragement and physical assistance with eating may be needed and special diets and/or food supplements are useful. However, problems such as loss of appetite or swallowing difficulties may limit these approaches, and artificial nutrition support using either ETF or intravenous nutrition is then needed. ETF is preferred whenever patients have adequate accessible gastrointestinal absorptive capacity as it is both more physiological and cheaper. It may also help to maintain gut barrier function^{12–13} although there is little evidence that it reduces bacterial translocation in humans.¹⁴

Post surgical ETF is being used with increasing frequency when oral intake is limited. Early support is usually given via a post pyloric NJ tube or surgical jejunostomy placed pre, inter, or postoperatively. Feeding is usually possible for

although gastric and colonic function may be impaired for several days after surgery, small bowel function is often normal.^{15–16} Absent bowel sounds are not necessarily a contraindication as they relate to gastric and colonic activity.

Concerns about anastomotic integrity with early ETF are probably unwarranted, although anastomoses involving the stomach, small bowel, biliary tree, or pancreas may be more vulnerable than lower gastrointestinal anastomoses. Jejunostomy feeding is particularly useful after oesophago-gastric surgery.¹⁷ A systematic review and meta-analysis of randomised controlled trials which compared any type of enteral feeding started <24 hours after elective gastrointestinal surgery versus nil by mouth management concluded that early feeding reduced infective risks by approximately 30% and mean length of hospital stay by nearly one day.¹⁸ The risk of vomiting however was increased among patients fed early by nearly 30%. ETF also appears to be beneficial in patients with pancreatitis, although it may need to be avoided in cases complicated by fistulation or pseudocyst formation.¹⁹

Perioperative ETF may have advantages over parenteral nutrition (PN) feeding. A meta-analysis comparing these methods of support concluded that enteral nutrition (EN) reduced infective risks by about one third.²⁰ The apparent superiority of EN is usually ascribed to maintained gut integrity but, as mentioned above, the evidence that PN feeding causes either villous atrophy or increased bacterial translocation is mixed. The apparent superiority of EN over PN in the perioperative period may therefore relate to problems of early overfeeding in the PN arms of studies.²¹

Although there is little hard evidence, it seems reasonable to start postoperative ETF within 1–2 days in patients who are severely malnourished (BMI <16 and/or weight loss >15%) and not yet tolerating oral intakes. Moderately malnourished patients (BMI <18.5 and/or weight loss >10%) should probably be fed within 3–5 days of surgery when oral intake remains restricted, with normally or over nourished patients receiving support if they have not met 50% of estimated requirements within 5–7 days. If ETF is not tolerated, PN may be needed, although continued minimal ETF (10 ml/h) may help to stimulate or maintain gut function and decrease the chances of cholestasis.

Common indications for ETF are shown in table 1.

6.0 ETHICAL AND LEGAL CONSIDERATIONS

Artificial nutrition support is fraught with ethical and legal difficulties, and hospital clinicians should be familiar with these. The following points are taken from a report commissioned by the BAPEN.²² The British Medical Association have also provided guidance.²³

Providing adequate and appropriate fluid and nutrients is a basic duty to sick patients. While a patient can swallow and expresses a desire or willingness to drink or eat, fluid and nutrients should be given unless there is a medical contraindication. Treatment plans for patients with existing or probable future fluid or nutrient deficits should include decisions on fluid and/or nutrient provision.

If the plan is to maintain adequate intakes, the ethical duty is to take appropriate measures to achieve this aim. Administration of nutrients and/or fluid via a tube must be considered if the patient cannot consume or absorb adequate amounts orally. However, legally this is considered a medical treatment (even though some professionals would argue that ETF is a part of basic medical care).

If an illness is regarded as being in a terminal phase and the plan is to provide only compassionate and palliative care, ethical considerations indicate that a tube supply of nutrients or fluid need only be given to relieve symptoms. This does not mean that it should necessarily be used to prolong survival. In cases where benefits are in doubt, a planned “time

Table 1 Indications for enteral tube feeding

Indication for feeding	Example
Unconscious patient	Head injury, ventilated patient
Swallowing disorder	Post-CVA, multiple sclerosis, motor neurone disease
Physiological anorexia	Liver disease (particularly with ascites)
Upper GI obstruction	Oesophageal stricture
Partial intestinal failure	Postoperative ileus (see section 5.0), inflammatory bowel disease, short bowel syndrome
Increased nutritional requirements	Cystic fibrosis, renal disease
Psychological problems	Severe depression or anorexia nervosa

GI, gastrointestinal; CVA, cerebrovascular accident.
Hospital nutrition support teams and/or dietitians should be involved as early as possible in the assessment and instigation of enteral tube feeding.

limited" trial of feeding may be useful. Consent of a competent adult patient must be sought for such treatment and a patient's competent refusal is binding.

Competence depends on adequate thought processes to make the decision needed. It is ethically and legally wrong for a carer to underestimate the capacity of a patient in order to achieve what the carer believes to be in the patient's best interest. For an incompetent adult, the doctor undertaking care is responsible in law for any decision to withhold, give, or withdraw a medical treatment, including fluid and/or nutrient provision via a tube. The doctor's duty is to act in the patient's best interest. Before making a decision about starting, stopping, or continuing enteral tube feeding and or fluid provision, the doctor should seek to ascertain whether the patient has expressed any previous views about the type of treatment he or she would wish to receive should the present state of incompetence occur.

All decisions on tube provision of food and/or fluids should involve full consultation with the family and all members of the health care team from the outset. At present, however, under English Law, relatives or a nominated proxy cannot make a decision on behalf of an adult patient and hence cannot override the doctor's decision. Special considerations apply in relation to children and application to the court should be made regarding the legality of withdrawing artificial hydration and nutrition from a patient in a persistent vegetative state.

Under specified circumstances, it can be legal to enforce nutritional treatment for an unwilling patient with a mental disorder. This includes anorexia nervosa in which it is considered that severe malnourishment per se can render a patient incompetent of making rational decisions regarding their care.

7.0 ACCESS TECHNIQUES

Gastrointestinal access for up to 4–6 weeks is usually achieved using NG or NJ tubes, although placement of percutaneous gastrostomy or jejunostomy access should be considered sooner if feeding is very likely to be prolonged (see section 7.3). Oroenteral tubes are also used occasionally and, since the advent of endoscopic placement, percutaneous gut access has become popular for longer term use.²⁴

7.1 Nasogastric (NG) tubes

Most enteral feed is given into the stomach to allow the use of hypertonic feeds, higher feeding rates, and bolus feeding. Fine bore 5–8 French gauge NG tubes are now used unless there is a need for stomach aspiration, or administration of high fibre feeds or drugs via the tube.²⁵ Large bore PVC tubes should be avoided as they irritate the nose and oesophagus and increase the risks of gastric reflux and aspiration. They

Box 1 Placing a nasogastric tube.

- Explain the procedure to the patient.
- Mark the tube at a distance equal to that from the xiphisternum to the nose via the earlobe (50–60 cm).
- Lubricate the tube externally with gel/water and internally with water if a guidewire is present. Check the guidewire moves freely.
- Check nasal patency by "sniff" with each nostril occluded in turn. The clearer nostril can be sprayed with lignocaine to minimise discomfort.
- Sit the patient upright with the head level. Slide the tube gently backwards along the floor of the clearer nostril until visible at the back of the pharynx (10–15 cm).
- If the patient is cooperative, ask them to take a mouthful of water and then advance the tube 5–10 cm as they swallow.
- Repeat the water swallow/advance until the preset mark on the tube reaches the nostril.
- Withdraw the tube at any stage if the patient is distressed, coughing, or cyanosed.
- If there is difficulty passing the tube, ask the patient to tilt their head forwards or turn it to one side.
- Once in place, remove any guidewire and secure carefully.
- Check position of the tube before use (this does not usually require an x ray (see text)).
- Document tube insertion in the patient's notes.

also need frequent replacement as they degrade on contact with gastric contents. Polyurethane and silicone tubes last for at least one month.

Insertion

NG tubes can be placed on the ward by experienced medical or nursing staff (see box 1).²⁶

The position of an NG tube should be confirmed every time it is used for feeding or drug administration. This does not need an x ray as long as the external length of tube remains unchanged and the tube aspirate has a pH <5.²⁷ If aspiration is difficult, change the patient's position or, if safe, give a drink to increase the volume of gastric contents. Advancing the tube slightly may also help. The pH test is valueless if patients are on acid suppression, and if there is any doubt, or any other reason, an x ray is needed. Checking the position of a tube by injecting air through it and listening for bubbles with a stethoscope is unreliable.

7.2 Nasojejunal (NJ) tubes

Jejeunal feeding may be indicated if there are problems with gastric reflux or delayed gastric emptying. It should also be used in unconscious patients who have to be nursed flat. All NJ tubes are fine bore (6–10 French gauge). Some have a shorter second lumen for gastric aspiration.

Insertion

Post pyloric placement can be difficult and various techniques are used.^{28–29} The tube is passed in the same way as an NG tube but once it is well into the stomach (60 cm) the patient is turned onto their right side before the tube is advanced a further 10 cm. This may result in successful passage through the pylorus.²⁴ If this fails, try repeating the manoeuvre after inflating the stomach with 500–1000 ml of

air. Creating a 30° bend, 3 cm from the end of the tube, and rotating it clockwise during insertion may also help. Some NJ tubes (Bengmark) develop a spiral coil once the guidewire has been removed.³⁰ These will usually pass spontaneously into the small bowel if patients have adequate gastric motility and some units place them before gastrointestinal surgery for use postoperatively. Tubes with weighted tips do not help in achieving post pyloric access but intravenous prokinetics such as metoclopramide or erythromycin may be helpful.^{31 32}

Direct endoscopic placement of NJ tubes is difficult as the tube is usually displaced during withdrawal of the endoscope, even when a guidewire is left in situ. An alternative approach is to use a long guidewire, which is passed through the endoscope into the jejunum and then left in place while the endoscope is removed. The wire is then re-routed from the mouth to the nose (using a short tube passed through the nose and out of the mouth), before a well lubricated nasoenteric tube is passed over it.²⁴ However, this is sometimes difficult without fluoroscopic screening and, if fluoroscopy is to be used, endoscopic assistance is usually unnecessary. The position of an NJ tube should generally be confirmed by x ray 8–12 hours after placement as auscultation and pH aspiration techniques can be inconclusive.²⁷

7.3 Percutaneous gastrostomy tubes

If enteral feeding is likely to be needed for periods of more than 4–6 weeks, a gastrostomy tube can be inserted directly into the stomach through the abdominal wall, using relatively simple endoscopic or radiological procedures.³³ Gastrostomy tubes allow feeding without the inconvenience, discomfort, and embarrassment of NG access, and patients receive more of their prescribed feed. This is largely because NG tubes “fall out” easily (see section 10.1). Although gastrostomy placement has a low immediate morbidity, the overall mortality within a few weeks of percutaneous endoscopic gastrostomy (PEG) placement is very high (see section 10.1) and many PEGs are placed inappropriately.³⁴ Deaths are usually due to the nature of the underlying condition and poor patient selection (for example, a severe stroke).

Patients selected for gastrostomy should be at high risk of malnutrition and unlikely to recover their ability to feed orally in the short term. Most authorities consider placement if problems are likely to persist for more than 4–6 weeks but one trial has suggested placement at 14 days post acute dysphagic stroke³⁵ (this suggestion is currently being assessed in multicentre trials). The patient’s gastrointestinal function must be adequate to absorb and tolerate the proposed feeding. The ethical issues involved in PEG placement are no different to those involved in the instigation of artificial nutrition support by any other means (see section 6.0) but the invasive and potentially dangerous nature of the procedure make it obligatory to think these through very carefully.

The concept of gastrostomy feeding must be acceptable to the patient and their family or carers. Suitability for gastrostomy placement should therefore be confirmed by an experienced gastroenterologist or a suitably trained member of a nutrition support team.^{36 37} The prognosis of any swallowing difficulty should be assessed by a specialist.

Common indications for gastrostomy placement are shown in table 2. In patients where cosmetic considerations are important, low profile “button” PEGs can be used which contain a built-in antireflux valve to prevent leaks when feeding extension tubes are disconnected.³⁸

Insertion

Most gastrostomies are placed endoscopically using sedation and local anaesthetic.²⁴ Radiological or ultrasound guided

placement can be used if endoscopy is contraindicated and gastrostomies can also be inserted surgically.³⁹ Relative contraindications to gastrostomy include gastro-oesophageal reflux, previous gastric surgery, ascites, extensive gastric ulceration, neoplastic/infiltrative disease of the stomach, gastric outlet obstruction, small bowel motility problems, malabsorption, peritoneal dialysis, hepatomegaly, gastric varices, coagulopathy, and late pregnancy. Crohn’s disease was thought to be a contraindication due to fears of disease occurrence within the gastrostomy tract. However, a number of studies have now suggested that it should be used where necessary.⁴⁰ Obesity can make gastrostomy technically difficult. The BSG currently recommend giving antibiotics (for example, a single dose of 2.2 g co-amoxiclav) 30 minutes before gastrostomy insertion to reduce the incidence of peristomal wound infections.⁴¹

Removal

Percutaneous gastrostomies should not be removed for at least 14 days after insertion to ensure that a fibrous tract is established that will prevent intraperitoneal leakage.²⁴ Gastrostomy tubes held in place by a balloon usually come out with gentle traction after the balloon is deflated whereas those held in place by a deforming device may need vigorous pulling. Tubes with rigid fixation devices are usually removed endoscopically, although recent evidence suggests that if they are cut off close to the skin and pushed through into the stomach they will pass through the gut spontaneously.⁴² This method should not be used if there is any suspicion of distal stricturing and, overall, 2% will not pass.

7.4 Percutaneous gastrojejunostomy and jejunostomy tubes

These tubes can be useful if patients are at risk of oesophageal reflux, although that risk is not eliminated.⁴³ They are also used for early postoperative feeding (see section 5.0). In the non-surgical patient, jejunal access is usually established transgastrically using radiological techniques. Percutaneous endoscopic transgastric jejunostomies (PEGJs) can also be placed by passing a jejunostomy tube through a gastrostomy and carrying it through the pylorus. Similarly, existing gastrostomies can be converted to jejunostomies using a jejunal extension.²⁴

Direct, percutaneous, endoscopically guided jejunal puncture is now being performed more frequently and can be used in patients who have had a gastrectomy. It is technically difficult and specific training in insertion techniques is required. Leakage problems may occur. Surgical jejunostomies are usually placed at the time of other surgery, although laparoscopic placement has also been described.^{44 45}

Table 2 Indications for gastrostomy

Indications for gastrostomy	Example
Neurological disorders of swallowing	CVA, multiple sclerosis, motor neurone disease, Parkinson’s disease, cerebral palsy.
Cognitive impairment and depressed consciousness	Head injury
Mechanical obstruction to swallowing	Oropharyngeal or oesophageal cancer, radiation enteropathy
Long term partial failure of intestinal function requiring supplementary intake	Short bowel, fistulae, cystic fibrosis

CVA, cerebrovascular accident.

8.0 FEED ADMINISTRATION

8.1 Modes of feeding

Enteral tube feeds can be administered by bolus, or by intermittent or continuous infusion.⁴⁶ Bolus feeding entails administration of 200–400 ml of feed down a feeding tube over 15–60 minutes at regular intervals. The technique may cause bloating and diarrhoea and bolus delivery into the jejunum can cause a “dumping” type syndrome and should therefore be avoided (see section 10.4). Bolus feeding can be performed using a 50 ml syringe, either with or without the plunger. If the latter is removed, the syringe can be hung up to allow gravity feeding. Continuous infusion may help with diarrhoea or prevent “dumping” in some patients but it also results in higher intragastric pH levels than bolus feeding which can promote bacterial growth (see section 10.4). It is commonly used for very ill patients but it should be changed for intermittent infusion as soon as possible. Continuous feed should not be given overnight in patients who are at risk of aspiration. Intermittent infusion provides moderate rates of feed provision via either gravity or pump. Breaks in feeding of six hours or more are used, depending on patients’ needs (for example, overnight feeding). Post pyloric feeding necessitates continuous administration due to the loss of the stomach reservoir.

8.2 Choice of feeds

The choice of feed to be given via ETF is influenced by a patient’s nutritional requirements, any abnormality of gastrointestinal absorption, motility, or diarrhoeal loss, and the presence of other system abnormality, such as renal or liver failure.⁴⁶ Most commercial feeds contain 1.0 kcal/ml, with higher energy versions containing 1.5 kcal/ml. They are generally available in fibre free and fibre enriched forms. They are nutritionally complete but expert dietetic advice should be sought. Producing feeds locally by using a liquidiser is not recommended due to the high infective risks and potentially poor nutritional quality in terms of micro-nutrient provision. The following feeds are generally used.

- *Polymeric feeds*—These contain nitrogen as whole protein. The carbohydrate source is partially hydrolysed starch and the fat contains long chain triglycerides (LCTs). Their content of fibre is very variable and although most authorities recommend that fibre should be included⁴⁶ the evidence that higher levels are of real benefit is not strong (see section 9.4).
- *Predigested feeds*—These feeds contain nitrogen as either short peptides or, in the case of elemental diets, as free amino acids. Carbohydrate provides much of the energy content with the content variable in both quantity and the proportion provided as LCTs and medium chain triglycerides (MCTs). The aim of “predigested diets” is to improve nutrient absorption in the presence of significant malabsorption. Their importance is probably greater in maldigestive (for example, pancreatic disease) rather than malabsorptive states, and in patients with a short gut and no colon their high osmolality can cause excess movement of water into the gut and hence higher stomal losses.⁴⁷
- *Disease specific and pharmaco nutrient feeds*—Detailed guidelines on the use of specific formulations for patients with organ failure is beyond the remit of these guidelines, as are descriptions of feeds containing large quantities of nutrients with potential pharmacological activity. Patients with respiratory failure are often given feeds with a low carbohydrate to fat ratio in order to minimise carbon dioxide production, but it should be recognised that this type of feed requires higher oxygen availability, and avoidance of overfeeding is probably the more important in limiting respiratory demands. Renal patients

will often require modified protein, electrolyte, and volume feeds while liver patients may need low sodium low volume feeds. There is no good evidence that patients with hepatic encephalopathy should have low protein intakes and the evidence for the benefit of feeds rich in branch chain amino acids is weak. Sodium supplemented enteral or sip feeds are not available commercially but can be very useful in the management of patients with high output stomas who tend to become salt depleted. Addition of sodium chloride to achieve concentrations >100 mmol/l are needed with due care to avoid potential bacterial contamination. Consequent instability of feed components may be an issue and checks should be made with the manufacturer.

8.3 Energy and nitrogen requirements

An individual patient’s nutrient needs vary with current and past nutritional state, and the nature and complexity of their condition. As both inadequate or excessive feeding can be harmful (see section 10.5), dietitians or others with expertise should be consulted regarding feed prescription. If no expert advice is available, 30 kcal/kg/day (30 ml/kg/day of standard feeds) is likely to be adequate⁴⁶ but very undernourished patients should start at rates of <10 kcal/kg/day to prevent refeeding syndrome. Some experts would always commence feed cautiously in severely ill patients (see section 10.5).

In healthy individuals, a protein intake of well under 0.15 g N/kg/day (1 g N = 6.25 g protein) is adequate to maintain nitrogen balance but this changes dramatically in acute illness and catabolic patients have very high nitrogen losses. In the past, this led to the use of very high protein feeds in patients who were very ill or undernourished but recent thinking suggests that this is unwise.^{48–49} Most authorities therefore recommend early feeding at maximum levels of 0.2–0.3 g N/kg/day⁴⁶ and some recommend even lower levels during early feeding. When calculating energy provision for artificial nutrition support by either ETF or PN, there is no logical justification for considering energy provided as protein as separate from energy given as non-protein calories.

8.4 Micronutrients

Micronutrients are required for the prevention or correction of recognised deficiency states and maintenance of normal metabolism and antioxidant status. Standard enteral feeds are supplemented with vitamins and trace elements at levels which ensure that all micronutrients are likely to be met if the patient is on ETF at a level meeting their entire energy needs. Many patients however do not receive full ETF and may have pre-existing micronutrient deficits, poor absorption, and increased demands. It therefore seems reasonable to give additional balanced micronutrient supplements during the early days of ETF when full feeding may not be tolerated and additional micronutrients may be needed to replenish any deficits or to meet the increased demands of illness.

8.5 Fluid and electrolytes

Fluid needs can usually be met by giving 30–35 ml/kg body weight although allowance must be made for excessive losses from drains, fistulae, etc. Most feeds contain adequate electrolytes to meet the daily requirements of sodium, potassium, calcium, magnesium, and phosphate, although specific requirements can vary enormously. Malnourished or metabolically stressed individuals are often salt and water overloaded and excess sodium intake is a frequent problem in patients with renal problems, liver derangement, and cardiac

failure. High salt intakes may be needed when intestinal losses are excessive.

Potassium requirements are often high in malnourished or sick patients and normal plasma levels do not rule out total body depletion. Approximately 6 mmol of potassium is needed per g N for protein synthesis and needs are higher in patients who are postoperative, or on glucose/insulin infusions or diuretics. Feeding after a period of starvation also leads to high potassium requirements (see section 10.5). If hypokalaemia is persistent, concurrent hypomagnesaemia should be sought as renal and gastrointestinal potassium losses are high in patients with magnesium depletion. Calcium levels, adjusted for albumin, may need specific correction and magnesium losses can be enormous in patients with fistulae or high stomas. The daily requirement for phosphate is about 0.3 mmol/kg/day but requirements may be much greater when refeeding after starvation.

8.6 Monitoring enteral feeding

Patients receiving ETF should be closely monitored, particularly early after instigation. Monitoring allows quantification of losses to enable daily estimation of replacement requirements, maintenance of metabolic balance, detection of toxicity/deficiency states, and early detection of complications. As well as recording the volume and type of feed administered, early monitoring requires blood glucose to be checked at 4–6 hour intervals and plasma sodium, potassium, magnesium, and phosphate to be checked daily. This is especially true in patients who have had a prolonged period with little or no nutrient intake (see section 10.5). Liver function tests and full blood counts must be repeated weekly until the patient is stable. Blood pressure, pulse, and temperature records are also needed regularly and careful fluid balance records are essential. Body weight should be measured weekly, unless more frequent weighing is indicated, in order to monitor fluid status. If possible, trace element and vitamin levels should be measured on commencing ETF and patients on long term feeding should have periodic checks of vitamin and trace element status.

8.7 Stopping enteral tube feeding

ETF should be stopped once the patient has recovered swallowing, gastrointestinal, or general function to a level that permits an adequate oral intake. Dietetic review during the transition to oral feeding is recommended and dysphagic patients will need to be observed closely, ideally by a speech and language therapist with a specialist interest in swallowing difficulties. Video fluoroscopic assessment may be needed.

9.0 HOSPITAL DISCHARGE ON ENTERAL TUBE FEEDING

Increasing numbers of patients are now discharged to their home or community care on continued enteral nutrition. Outlining the management of such patients is beyond the remit of these guidelines but BAPEN have produced guidance.⁵⁰ Prior to discharge, it is the duty of the hospital care team to ensure that there is adequate liaison with the community carers in order to ensure that prescription feeds and feeding equipment is available. The patient, carers, district nurses, community dietitians, and GPs should all be fully informed and adequate training in pump use, infection control, feeding stoma care, etc., must have been provided before discharge. The hospitals should follow written protocols to ensure that discharge goes smoothly. The patient or carer should have a list of expert contacts.

10.0 COMPLICATIONS OF ENTERAL TUBE FEEDING

Although ETF is effective and safe in the majority of patients, feeding carries a number of significant risks summarised in table 3.

10.1 Tube insertion related complications

Although nasal intubation may cause discomfort, traumatic complications are uncommon if using fine bore NG or NJ tubes.^{25–51} Nevertheless, perforation of a pharyngeal or oesophageal pouch can occur and intracranial insertion of feeding tubes has been reported.^{52–53} NG tube insertion should probably be avoided for three days after acute variceal bleeding⁵⁴ and although oesophageal, gastric, or small bowel perforation is unusual, it may occur if a guidewire is reinserted and accidentally exits via a side port. Perforation has also been reported when using polyvinyl or polypropylene tubes without guidewires. Accidental bronchial insertion is relatively common in patients with reduced levels of consciousness or with impaired gag/swallowing reflexes. Endotracheal tubes in ventilated patients do not necessarily prevent bronchial insertion, and ETF into the lungs or pleural space can be fatal.⁵⁵

Approximately 25% of nasogastric tubes “fall out” or are pulled out by patients soon after insertion and tubes, especially those that are fine bore, can be displaced by coughing or vomiting. There is however no evidence to support the use of weighted NG tubes in terms of either placement or maintenance of position.³¹

Problems related to insertion of percutaneous gastrostomy and jejunostomy tubes include abdominal wall or intraperitoneal bleeding and bowel perforation. Free air is visible on x ray in 38% of patients but significant surgical intervention is needed in fewer than 5%.^{56–57} Early procedure related mortality of up to 2% has been reported but this is mainly ascribable to the risks of endoscopy in a vulnerable population group. Later mortality rates are very high (see section 10.2).

10.2 Post insertion tube complications

Nasopharyngeal discomfort occurs frequently in patients with nasoenteral tubes and many suffer sore mouths, thirst, swallowing difficulties, and hoarseness.⁵⁵ Mouthwashes, sucking ice cubes, or using artificial saliva can help. Local pressure effects from tubes may cause nasal erosions, abscess formation, sinusitis, and otitis media. Avoidance of larger tubes helps and swapping of the tube to the other nostril when fine bore tubes need replacement (every 4–6 weeks) prevent these problems. Short term oesophageal damage can include oesophagitis and ulceration from local abrasion and gastro-oesophageal reflux although, once again, such problems are rare with fine bore tubes. Longer term damage includes significant stricturing. Large stiff tubes can cause fistulation to the trachea, especially when an endotracheal tube is present. Larger tubes are also unsafe in the presence of varices even if they have not bled recently.⁵⁴

Post insertion tube related complications from gastrostomies and jejunostomies differ from those seen with NG and NJ tubes.^{55–61} They include infection at the insertion site, peristomal leaks, accidental tube removal, tube fracture, gastro-colic fistula, peritonitis, septicaemia, and necrotising fasciitis. PEGJ tubes can also fall back into the stomach or become disconnected with the whole tube passing through the PEG and into the gut. Of even more concern however is the very high mortality rates of approximately 20%³⁷ or even 40%³⁶ seen in PEG patients within one month of insertion. These suggest that PEGs are often placed inappropriately^{34–36} and it has been shown that review of patients referred for gastrostomy by an experienced gastroenterologist results in a much lower 30 day mortality.⁶²

Table 3 Complications of enteral tube feeding

Type	Complication
Insertion	Nasal damage, intracranial insertion, pharyngeal/oesophageal pouch perforation, bronchial placement, variceal bleeding
PEG/PEJ insertions	Bleeding, intestinal/colonic perforation
Post insertion trauma	Discomfort, erosions, fistulae, and strictures
Displacement	Tube falls out, bronchial administration of feed
Reflux	Oesophagitis, aspiration
GI intolerance	Nausea, bloating, pain, diarrhoea
Metabolic	Refeeding syndrome, hyperglycaemia, fluid overload, electrolyte disturbance

PEG, percutaneous endoscopic gastrostomy; PEJ, percutaneous endoscopic jejunostomy; GI, gastrointestinal.

Complications of surgically placed enteral feeding tubes are quite common and include dislodgement, intraperitoneal leakage, and small bowel obstruction. Surgical jejunostomies should be left in for 3–5 weeks, even if feeding has stopped, so that a tract can become established and the purse string suture holding the tube has dissolved.⁵⁵

Feeding tubes block easily, especially if they are not flushed with fresh tap, cooled boiled, or sterile water before and after every feed or medication. Any drugs administered through a tube should ideally be elixirs or suspensions rather than syrups and should only be given after establishing compatibility. Hyperosmolar drugs, crushed tablets, potassium, iron supplements, and sucralfate are particularly likely to cause problems. A tube can often be unblocked by flushing with warm water or, if this fails, by using an alkaline solution of pancreatic enzymes.⁶³ Carbonated drinks, pineapple juice, and sodium bicarbonate solution may cause tube degradation.

Unlike NG and NJ tubes, gastrostomy tubes are sometimes occluded by gastric mucosal overgrowth. Tube blockage or intraperitoneal leakage can be assessed using water soluble contrast, and passing a soft guidewire may be helpful. Blockage may necessitate replacement or surgical removal, although loosening and rotating a gastrostomy tube every week helps to prevent any problem. When splits or breakages occur in gastrostomy or jejunostomy tubes, it is often possible to cut the tube and then replace the luer/funnel lower down.

10.3 Reflux and inhalation problems

Gastro-oesophageal reflux occurs frequently with ETF. It is more common when patients are NG fed in the supine position⁶⁴ and reflects a combination of gravitational back flow and impairment of gastro-oesophageal sphincter function induced by pharyngeal stimulation and the presence of the tube across the cardia. It is very common in patients with impaired consciousness or poor gag reflexes, occurring in up to 30% of those with tracheostomies⁶⁵ and 12.5% of neurological patients.⁶⁶ Aspiration may occur with no obvious vomiting or coughing, and pneumonia can develop silently.

To minimise risks of aspiration, patients should be fed propped up by 30° or more, and should be kept propped up for 30 minutes after feeding.⁵⁵ Acid suppression or sucralfate may help with symptoms of oesophagitis, but they do not prevent aspiration pneumonia. There is an increased risk of aspiration if gastric residues accumulate, and therefore if a four hour aspirate is >200 ml, the feeding regimen should be reviewed. Although continuous pump feeding reduces gastric pooling, it is often used overnight and may therefore be more risky than bolus or intermittent feeding.⁶⁷ Iso-osmotic feeds cause less delayed gastric emptying than high osmotic feeds⁶⁸ and promotility drugs such as metoclopramide or erythromycin may be helpful.

Post pyloric feeding makes aspiration less likely, but does not eliminate the problem. PEG feeding may reduce but will not eliminate the risk of aspiration, although PEGJ feeding does reduce the risk further.⁵⁵

10.4 Gastrointestinal problems

ETF commonly causes gastrointestinal symptoms. Nausea occurs in 10–20% of patients^{69–70} and abdominal bloating and cramps from delayed gastric emptying are also common.⁵⁵ ETF related diarrhoea occurs in up to 30% of enterally fed patients on medical and surgical wards and more than 60% of patients on intensive care units.^{70–74} It can create serious problems from nutrient, fluid, and electrolyte losses, and from infected pressure sores and general patient distress.⁵⁵ Parenteral nutrition may be required if elimination of all other causes of gastrointestinal upset and/or administration of simple symptomatic treatments fails to resolve the problem. Constipation, with or without overflow, also occurs with ETF.

The causes of gastrointestinal discomfort and ETF diarrhoea are multiple and are summarised in table 4.

Feed delivery site and rate

Gastrointestinal discomfort often relates to excessive feed administration rates, delayed gastric emptying, or decreased small bowel motility. Continuous infusion rather than bolus administration of feeds may therefore help. Feeding rates should be reduced if gastric residual volumes are >200 ml, although aspiration through fine bore tubes is unreliable. Prokinetic agents may be helpful but if persistently high aspirates prevent effective feeding, jejunal access should be considered.

Bolus feeding is often thought to cause more diarrhoea⁷⁵ than continuous intragastric feeding but this may be untrue.^{55–76} Enteral feeds taken orally cause less diarrhoea in healthy volunteers than the same quantities given by NG tube⁷⁷ suggesting that cephalic and gastrocolic reflexes are important in the aetiology of ETF diarrhoea. If this is true, bolus intragastric feeding should cause fewer problems than infused feeds as a bolus will stimulate more normal distal colonic motor suppression and promote water absorption in the ascending colon.⁵⁵ There is no evidence that starter regimens with diluted or hypotonic diets are helpful and

Table 4 Causes of gastrointestinal intolerance

Cause	Examples
Feed delivery site and rate	High rate, post pyloric feeding
Feed type	Low fibre feeds
Drug related	Laxatives, antibiotics, NSAIDs, PPIs, antiarrhythmics, antihypertensives, drugs containing magnesium and sorbitol fillers, etc.
Infective	Contaminated feeds, small bowel overgrowth, <i>Clostridium difficile</i>
Lactase deficiency	Primary and secondary
Fat malabsorption	Pancreatic dysfunction, liver disease, coeliac disease.

NSAIDs, non-steroidal anti-inflammatory drugs; PPIs, proton pump inhibitors.

these approaches can delay the provision of adequate nutrition unnecessarily.⁷⁸

Feed type

Most enteral tube feeds are available in standard and fibre enriched forms. The standard feeds contain little or no fibre and hence lead to reduced short chain fatty acid (SCFA) production in the colon, due to both limited substrate availability and decreased induction of bacterial polysaccharidase. SCFAs promote salt and water reabsorption in the colon and also limit growth of pathogenic bacteria due to lower colonic pH.^{55–79} The fibre enriched feeds aim to increase the overall colonic bacterial population and hence stool mass and water absorptive capacity.⁸⁰ However, although they do seem to normalise transit times, there is little evidence that this often helps with ETF diarrhoea, perhaps due to the fact that the diarrhoea has nothing to do with the feed per se.^{55–81–82} Lack of definite benefit may also relate to some problems when manufacturing fibre enriched feeds, which need to contain small particles of non-starch polysaccharide or other insoluble carbohydrate components in order to limit viscosity. The small particles ferment easily and hence little fibre reaches the distal colon where it can help to absorb faecal water.

Feed temperature

Some studies suggested that feed temperature influences ETF diarrhoea but there is little evidence that either refrigeration or warming alters gastrointestinal complications significantly.⁸³

Drug related ETF diarrhoea

Whenever diarrhoea occurs with ETF, all laxatives must be stopped, including drugs containing magnesium such as antacid preparations and drugs containing active fillers, such as sorbitol.⁵⁵ Diarrhoea is also a recognised side effect of many drug classes, including H₂ blockers, proton pump inhibitors, antibiotics, antiarrhythmics, antihypertensives, and non-steroidal anti-inflammatory drugs.

Antibiotics can cause diarrhoea in patients eating normally but the incidence is far higher in patients on ETF.^{55–84} The exact cause is unclear, but it seems likely that they alter intestinal flora to allow overgrowth of pathogenic species. *Clostridium difficile* toxin is found in 20–50% of patients with antibiotic related diarrhoea.⁸⁵ Antibiotics also reduce colonic bacterial production of SCFAs from insoluble carbohydrates and fibre.

Infective causes

Enteral feed is an ideal culture medium and once contaminated, bacteria will rapidly multiply. Stool samples must therefore be checked whenever ETF patients develop diarrhoea. Bacterial feed contamination can also cause sepsis, pneumonia, and even urinary tract infections as well as gastrointestinal related problems.^{86–89} Open bottles or cans of feed get infected during handling and delivery^{90–91} and so it is vital that no part of the delivery system or feed is in contact with the hands, clothes, skin, or other non-disinfected surface. Feeds should not be decanted before use and if continuous feeding is used, bacteria can also spread up the giving set from gastric or enteral sources, especially as the continuous infusion raises gastric pH and promotes bacterial overgrowth. Administration sets and nutrient containers should therefore be discarded every 24 hours.^{92–93} Avoiding simultaneous acid suppression and allowing breaks in feeding to let the pH of the stomach fall may be helpful. With 8 hours fasting/24 hours, the incidence of pneumonia on an intensive therapy unit fell from 54% to 12%.⁹⁴ Post pyloric feeding is particularly prone to infective complications

as the food bypasses the protective gastric acid barrier. Full enteral feed and associated equipment handling guidelines are beyond the remit of this document.

Lactase deficiency

Primary lactase deficiency is common in many parts of the world and a secondary deficiency can occur when there is gut damage from inflammation or infection, a reduced small bowel absorptive area, or rapid small bowel transit.⁹⁵ Carbohydrate malabsorption may then cause gastrointestinal problems although most commercial enteral feeds are lactose free. If a patient with diarrhoea is also taking oral food as well as enteral feeds, it is important to limit milk and milk products.

Fat malabsorption

Fat malabsorption may cause diarrhoea in ETF patients, especially those with pancreatic deficiency, biliary obstruction, or extensive ileal resection. Terminal ileal problems may also cause diarrhoea through bile salt malabsorption. Patients with a jejunostomy or ileostomy do not need to reduce their fat intake but if the colon remains in continuity with a short small bowel, steatorrhoea can develop. Using a feed with a low fat content can then be helpful but may limit the energy provided to the patient. Feeds containing MCTs may be better absorbed although patients often tolerate them poorly.

Hypoalbuminaemia

There is considerable debate over whether hypoalbuminaemia can cause ETF diarrhoea through intestinal oedema.⁵⁷ Rather than a direct causation however it seems more likely that both the low albumin and gut dysfunction reflect a generalised membrane leakiness, often due to a systemic inflammatory response. Certainly, patients with very low plasma albumin due to nephrotic syndrome or cirrhosis do not necessarily have loose stools, and albumin supplements fail to correct ETF diarrhoea.

Treatment of ETF diarrhoea

If diarrhoea remains a problem after attention to the above causes, loperamide in high doses may be used. If this fails, codeine phosphate may control symptoms and there are anecdotal reports of live yoghurt or other probiotics being helpful. If vomiting/bloating or diarrhoea (not related to antibiotic therapy) are problematic, feed rates can be reduced for a trial period.

10.5 Metabolic complications of ETF

Artificial feeding of patients may cause a variety of metabolic problems, including deficiencies or excess of fluid, electrolytes, vitamins, and trace elements.^{55–96–97} Over hydration occurs frequently, particularly if ETF patients are also receiving supplementary intravenous nutrition or fluids.

Hyponatraemia is a common problem when enteral nutrition is given to sick patients.⁹⁸ It is often accompanied by the development of oedema and is usually due to a combination of excessive use of intravenous fluids, such as 5% dextrose, in combination with the adverse effects from malnourishment and severe illness on normal membrane pumping. Patients end up with excess body water in combination with very high total body sodium. As a consequence, rather than administering further sodium in feeds or intravenous fluids, treatment should usually entail fluid restriction. Generous amounts of potassium to encourage cell membrane sodium exchange may be helpful. Hypernatraemia can also occur and is usually due to excess water loss or transient diabetes insipidus in neurosurgical patients.⁹⁷

Between 10% and 30% of tube fed patients are hyperglycaemic⁹⁶ and may need oral antidiabetic agents or insulin,

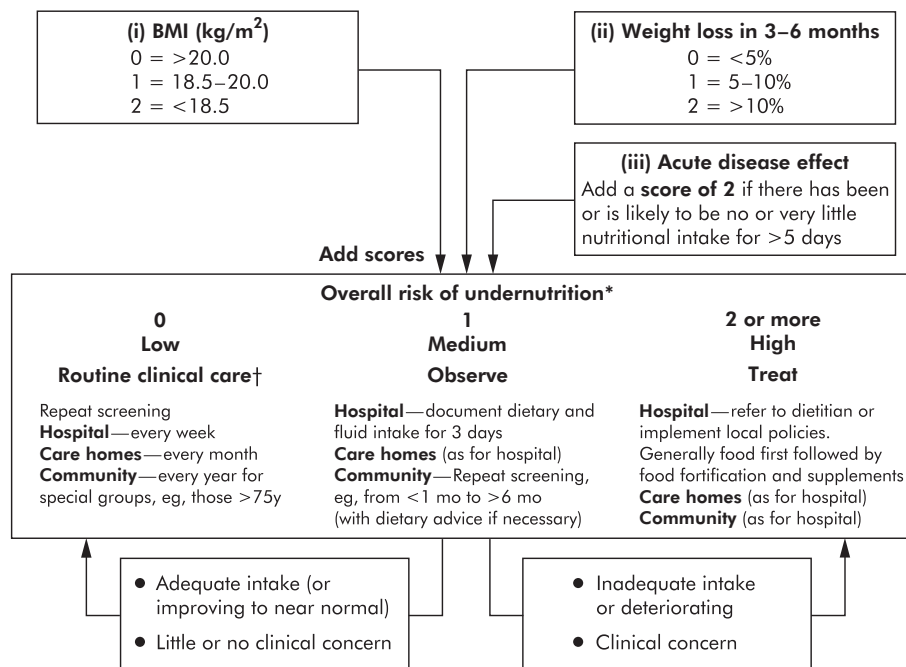


Figure A1 Malnutrition universal screening tool (MUST). *If height, weight, or weight loss cannot be established, use documented or recalled values (if considered reliable). When measured or recalled height cannot be obtained, use knee height as a surrogate measure. If neither can be calculated, obtain an overall impression of malnutrition risk (low, medium, high) using the following: (i) clinical impression (very thin, thin, average, overweight); (ii) clothes and/or jewellery have become loose fitting; (iib) history of decreased food intake, loss of appetite, or dysphagia up to 3–6 months; and (iiic) disease (underlying cause) and psychosocial/physical disabilities likely to cause weight loss. Involves treatment of underlying condition, and help with food choice and eating when necessary (also applies to other categories).

before and during feeding. Rebound hypoglycaemia may also occur in tube fed patients if feeding is stopped abruptly, especially if they are on antidiabetic therapy.

When commencing feeds in patients who have recently starved, there is the danger of inducing refeeding syndrome.^{99–100} This condition is poorly understood but occurs, in part, because the body adapts to undernutrition by down-regulating membrane pumping in order to conserve energy. This in turn causes leakage of intracellular potassium, magnesium, calcium, and phosphate, with subsequent whole body depletion. Simultaneously, sodium and water leak into the cells.

The sudden onset of artificial nutritional support appears to reverse the above processes and along with insulin driven movements of electrolytes into cells, can lead to precipitous falls in circulating levels of potassium, magnesium, calcium, and phosphate. There may also be an accompanying acute increase in circulating and extracellular fluid due to exogenous administration, the endogenous movement of sodium and water out of cells, and the diminished ability of undernourished kidneys to excrete a salt and water load. Furthermore, specific micronutrient deficiencies can compound the problems (for example, thiamine deficiency and cardiac function). As a result of all of these processes, there is a considerable danger of cardiac and respiratory failure, lethargy, confusion, coma, and even death.

Refeeding problems can usually be avoided by feeding for the first few days at very low levels while generously supplementing and closely monitoring potassium, magnesium, calcium, and phosphate. Instigation of feeds at levels of approximately 20 kcal/kg/day is often suggested but some authorities believe that even this may be too high. The situation is particularly dangerous in patients who have abnormal plasma electrolytes before feeding has even started. In such cases, many authorities suggest that correction of the electrolyte abnormalities using intravenous or oral electrolyte

supplements should be undertaken before feeding starts. This approach however may provide a false sense of security as improvement in plasma levels could occur with no significant change in overall electrolyte status. A severely malnourished individual may have intracellular electrolyte deficits which total hundreds of mmol, yet be unable to correct intracellular status unless simultaneous feeding is given to encourage transmembrane transfer. It therefore seems more logical to provide initial generous potassium, magnesium, calcium, and phosphate supplements with feeding at around 10 kcal/kg/day in very high risk groups. Thiamine and other B vitamins must also be given intravenously starting before any feed is started, continuing for at least the first three days of feeding.

It has also been suggested that commencing high levels of feeding shortly after major surgery or during sepsis and/or multiorgan failure can also cause metabolic problems similar to those of refeeding, as well as the problems of insulin resistance seen in such patients. Liver dysfunction can also be triggered or worsened by feeding as the high influx of nutrients to the liver can lead to excessive storage of fat and glycogen. This is particularly problematic if continuous ETF is used.¹⁰⁰

11.0 CONCLUSIONS

Malnourishment is common in adult patients in UK hospitals and ETF is an effective and generally safe means of offering many of them nutritional support. Access options need careful consideration in each patient as well as levels of feeding, rates of administration, and the type of feed to be used. Complications can usually be avoided if care is taken. Feeding should not be undertaken unless it is in the patient's best interests and all relevant ethical issues have been taken into account. A time defined trial of feeding to see if benefit is obtained may be appropriate in difficult ethical situations.

12.0 APPENDIX

Figure A1 shows the malnutrition universal screening tool (MUST).

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13.0 REFERENCES

- McWhirter JP, Pennington CR. Incidence and recognition of malnutrition in hospital. *BMJ* 1994;**308**:945–8.
- Robinson G, Goldstein M, Levine GM. Impact of nutritional status on DRG length of stay. *JPEN J Parenter Enteral Nutr* 1987;**11**:49–51.
- Reilly JJ Jr, Hull SF, Albert N, et al. The economic impact of malnutrition: A model system for hospitalised patients. *JPEN J Parenter Enteral Nutr* 1988;**12**:371–6.
- Eccles M, Clapp Z, Grimshaw J, et al. North of England evidence based guidelines project: methods of guideline development. *BMJ* 1996;**312**:760–2.
- Edington J, Kon P, Martyn C. Prevalence of malnutrition in patients in general practice. *Clin Nutr* 1996;**15**:60–3.
- National Diet and Nutrition Survey. People aged 65 years and over. London: The Stationary Office, 1998 (ISBN 0 11 322173 8).
- Veterans Affairs TPN Cooperative Study. Perioperative total parenteral nutrition in surgical patients. *N Engl J Med* 1991;**325**:525–32.
- Potter J, Langhorne P, Roberts M. Routine protein energy supplementation in adults: a systematic review. *BMJ* 1998;**317**:495–501.
- Green CJ. Existence, causes and consequences of disease related malnutrition in the hospital and the community, and the clinical and financial benefits of nutritional intervention. *Clin Nutr* 1999;**18**(suppl 2):3–38.
- Lennard-Jones JE. *A positive approach to nutrition as treatment*. London: Kings Fund Centre, 1992.
- Elia M, Torodovic V. Malnutrition in the community and other health care settings. *Int J Gastro* 2003 (in press).
- Moore FA, Moore EE, Poggetti R, et al. Gut bacterial translocation via the portal vein: a clinical perspective with major torso trauma. *J Trauma* 1991;**31**:629–38.
- Buchman AL, Moukarzel AA, Bhuta S, et al. Parenteral nutrition is associated with intestinal morphologic and functional changes in humans. *JPEN J Parenter Enteral Nutr* 1995;**19**:453–60.
- Macfie J. Enteral versus parenteral nutrition: the significance of bacterial translocation and gut barrier function. *Nutrition* 2000;**16**:606–11.
- Woods JH, Erickson LW, Condon RE, et al. Postoperative ileus: a colonic problem? *Surgery* 1978;**84**:527–32.
- Carr CS, Ling KDE, Boulos P, et al. Randomised trial of safety and efficacy of immediate postoperative enteral feeding in patients undergoing gastrointestinal resection. *BMJ* 1996;**312**:869–71.
- Wakefield SE, Mansell NJ, Baigrie RJ, et al. Use of a feeding jejunostomy after oesophagogastric surgery. *Br J Surg* 1995;**82**:811–13.
- Lewis SJ, Egger M, Sylvester PA, et al. Early enteral feeding versus "nil by mouth" after gastrointestinal surgery: systematic review and meta-analysis of controlled trials. *BMJ* 2001;**323**:773–6.
- Dejong CH, Greve JW, Soeters PB. Nutrition in patients with acute pancreatitis. *Curr Opin Crit Care* 2001;**7**:251–6.
- Braunschweig CL, Levy P, Sheehan PM, et al. Enteral compared to parenteral nutrition: a meta-analysis. *Am J Clin Nutr* 2001;**74**:534–42.
- Stroud MA. Perioperative nutrition. In: Johnson C, Taylor I, eds. *Advances in surgery*. London: Royal Society of Medicine Press, 2002:37–46.
- Jones L. *Ethical and legal aspects of clinical hydration and nutritional support*. Maidenhead: British Association for Parenteral and Enteral Nutrition, 1998.
- Withholding and withdrawing life-prolonging medical treatment: guidance for decision making*. London: BMA, 2000.
- Mensforth A, Nightingale J. Insertion and care of enteral feeding tubes. In: Nightingale J, ed. *Intestinal failure*. London: Greenwich Medical Media Ltd, 2001:281–303.
- Silk DB, Rees RG, Keohane PP, et al. Clinical efficacy and design changes of "fine bore" nasogastric feeding tubes: a seven-year experience involving 809 intubations in 403 patients. *JPEN J Parenter Enteral Nutr* 1987;**11**:378–83.
- Moore DM, Calcaterra TC. Inserting and securing the nasogastric tube. *Laryngoscope* 1987;**97**:1460.
- Metheny NA, Clouse RE, Clark JM, et al. pH testing of feeding-tube aspirates to determine placement. *Nutr Clin Pract* 1994;**9**:185–90.
- Zaloga GP. Bedside method for placing small bowel feeding tubes in critically ill patients. *Chest* 1991;**100**:1643–6.
- Schulz MA, Santanello SA, Monk J, et al. An improved method for transpyloric placement of nasogastric feeding tubes. *Int Surg* 1993;**78**:79–82.
- Benchmark S. Progress in peri-operative enteral tube feeding. *Clin Nutr* 1998;**17**:145–52.
- Keohane PP, Attrill H, Silk DBA. Clinical effectiveness of weighted and unweighted 'fine bore' nasogastric feeding tubes in enteral nutrition: a controlled clinical trial. *J Clin Nutr Gastroenterol* 1986;**1**:189–93.
- Lord LM, Weiser-Mamone A, Pulhamus M, et al. Comparison of weighted vs. unweighted enteral feeding tubes for efficacy of transpyloric intubation. *JPEN J Parenter Enteral Nutr* 1993;**17**:271–3.
- Moran BJ, Taylor MB, Johnson CD. Percutaneous endoscopic gastrostomy. *Br J Surg* 1990;**77**:858–62.
- Hughes AJ, Bowling TE. Is the current service for the insertion of endoscopic feeding tubes appropriate and acceptable? A UK survey. *Gut* 2002;**50**(suppl 1):A20.
- Norton B, Homer-Ward M, Donnelly MT, et al. A randomised prospective comparison of percutaneous endoscopic gastrostomy and nasogastric tube feeding after acute dysphagic stroke. *BMJ* 1996;**312**:13–16.
- Thuraisingham AJ, Cairns SA. Percutaneous endoscopic gastrostomy: prospective clinician review appropriately decreases insertion. *Gut* 2002;**50**(suppl 1):A86.
- Ng DHL, Timmis L, Bowling TE. A two year audit on percutaneous endoscopic gastrostomy: is the nutrition team generating more work? *Gut* 2001;**48**(suppl 1):A40.
- Gauderer MWL, Picha GJ, Izant RJ. The gastrostomy "button" – a simple, skin-level, nonrefluxing device for long-term enteral feedings. *J Pediatr Surg* 1984;**19**:803–5.
- Wollman B, D'Agostino HB, Walusiwigle JR, et al. Radiologic, endoscopic, and surgical gastrostomy: An institutional evaluation and meta-analysis of the literature. *Radiology* 1995;**197**:699–704.
- Nightingale JMD. Crohn's disease and gastrostomies. *Eur J Gastroenterol Hepatol* 2000;**12**:1073–5.
- BSG Guidelines in Gastroenterology. *Antibiotic prophylaxis in gastrointestinal endoscopy*. London: British Society of Gastroenterology, 1996.
- Korula J, Harma C. A simple and inexpensive method of removal or replacement of gastrostomy tubes. *JAMA* 1991;**265**:1426–8.
- DiSario JA, Foutch PG, Sanowski RA. Poor results with percutaneous endoscopic jejunostomy. *Gastrointest Endosc* 1990;**36**:257–60.
- Delany HM, Carnevale NJ, Garvey JW. Jejunostomy by a needle catheter technique. *Surgery* 1973;**73**:786–90.
- Duh Q-Y, Way LW. Laparoscopic jejunostomy using T-fasteners as retractors and anchors. *Arch Surg* 1993;**128**:105–8.
- McAtear CA, Arrowsmith H, McWhirter J, et al. *Current perspectives on enteral nutrition in adults*. A report by the British Association for Parenteral and Enteral Nutrition. Maidenhead: BAPEN, 1999.
- McIntyre PB, Fitchew M, Lennard Jones JE. Patients with a jejunostomy do not need a special diet. *Gastroenterology* 1986;**91**:25–33.
- Jeejeebhoy KN. Bulk or bounce—the object of nutritional support. *JPEN J Parenter Enteral Nutr* 1988;**12**:539–46.
- Elia M. Changing concepts of nutrient requirements in disease: implications for artificial nutrition support. *Lancet* 1995;**345**:1279–84.
- Elia M, Cottee S, Holden C, et al. *Enteral and parenteral nutrition in the community*. Report by BAPEN working party. London: BAPEN, 1994.
- Rees RG, Attrill H, Quinn D, et al. Improved design of nasogastric feeding tubes. *Clin Nutr* 1986;**5**:203–7.
- Iyer V, Reichel J. Perforation of the oesophagus by a fine bore feeding tube. *N Y State J Med* 1984;**84**:63–4.
- Wyler AR, Reynolds AF. An intracranial complication of nasogastric intubation. *J Neurosurg* 1977;**47**:297–8.
- Keohane PP, Attrill H, Grimble GK, et al. Enteral nutrition in malnourished patients with hepatic cirrhosis and acute encephalopathy. *JPEN J Parenter Enteral Nutr* 1983;**7**:346–50.
- Duncan HD, Silk DB. Problems of treatment—enteral nutrition. In: Nightingale J, ed. *Intestinal failure*. London: Greenwich Medical Media Ltd, 2001:477–96.
- Gallagher MW, Tyson KRT, Ashcraft KW. Gastrostomy in pediatric patients: an analysis of complications and techniques. *Surgery* 1973;**74**:536–9.
- Ho CS, Yee CAN, McPherson R. Complications of surgical and percutaneous non-endoscopic gastrostomy: Review of 233 patients. *Gastroenterology* 1988;**95**:1206–10.
- Wolfsen HC, Kozarek RA, Ball TJ, et al. Tube dysfunction following percutaneous endoscopic gastrostomy and jejunostomy. *Gastrointest Endosc* 1990;**36**:261–3.
- Litchfield BL, Nightingale JMD. Outcomes of percutaneous endoscopic gastrostomy tube insertion 1991–7. *Proc Nutr Soc* 1999;**58**:138A.
- Klein S, Heare BR, Soloway RD. The "buried bumper syndrome": a complication of percutaneous endoscopic gastrostomy. *Am J Gastroenterol* 1990;**85**:448–51.
- Hull MA, Rawlings J, Murray FE, et al. Post PEG outcomes—Audit of outcome of long-term enteral nutrition by percutaneous endoscopic gastrostomy. *Lancet* 1993;**341**:869–72.
- Chalmers-Watson TA, McKenzie CA, Burnham SP, et al. An audit of the PEG service provided by a nutrition support team in a DGH over a 3-year period. *Proc Nutr Soc* 2002;**61**:17a.
- Marcuard SP, Stegall KL, Trogdon S. Clearing obstructed feeding tubes. *JPEN J Parenter Enteral Nutr* 1989;**13**:81–3.
- Sriram K, Jayanthi V, Lakshmi RG, et al. Prophylactic locking of enteral feeding tubes with pancreatic enzymes. *JPEN J Parenter Enteral Nutr* 1997;**21**:353–5.
- Nagler R, Spiro SM. Persistent gastro-oesophageal reflux induced during prolonged gastric intubation. *N Engl J Med* 1963;**269**:495–500.
- Winterbauer RH, Duming RB, Barron E, et al. Aspirated nasogastric feeding solution detected by glucose strips. *Ann Intern Med* 1981;**95**:67–8.

- 67 **Olivares L**, Segovia A, Revuelta R. Tube feeding and lethal aspiration in neurological patients: a review of 720 autopsy cases. *Stroke* 1974;**5**:654–7.
- 68 **Jacobs S**, Chang RWS, Lee B, *et al*. Continuous enteral feeding: A major cause of pneumonia among ventilated intensive care unit patients. *JPEN J Parenter Enteral Nutr* 1990;**14**:353–6.
- 69 **Bury KD**, Jambunathan G. Effects of elemental diets on gastric emptying and gastric secretion in man. *Am J Surg* 1974;**127**:59–66.
- 70 **Jones BJM**, Lees R, Andrews J, *et al*. Comparison of an elemental and polymeric enteral diet in patients with normal gastrointestinal function. *Gut* 1983;**24**:78–84.
- 71 **Keohane PP**, Attrill H, Love M, *et al*. Relation between osmolality of diet and gastrointestinal side effects in enteral nutrition. *BMJ* 1984;**288**:678–80.
- 72 **Rees RG**, Keohane PP, Grimble GK, *et al*. Elemental diet administered nasogastrically without starter regimens to patients with inflammatory bowel disease. *JPEN J Parenter Enteral Nutr* 1986;**10**:58–262.
- 73 **Kelly TWJ**, Patrick MR, Hillman KM. Study of diarrhoea in critically ill patients. *Crit Care Med* 1983;**11**:7–9.
- 74 **Benya R**, Layden TJ, Morbarhan S. Diarrhoea associated with tube feeding: the importance of using objective criteria. *J Clin Gastroenterol* 1991;**13**:167–72.
- 75 **Edes TE**, Walk BE, Austin JL. Diarrhoea in tube-fed patients: feeding formulas not necessarily the cause. *Am J Med* 1990;**88**:91–3.
- 76 **Heibert JM**, Brown A, Anderson RG, *et al*. Comparison of continuous versus intermittent tube feeding in adult burn patients. *JPEN J Parenter Enteral Nutr* 1981;**5**:526–7.
- 77 **Kocan MJ**, Hickisch SM. A comparison of continuous and intermittent enteral nutrition in NICU patients. *J Neuroscience Nurs* 1986;**18**:333–7.
- 78 **Duncan HD**, Cole SJ, Bowling TE, *et al*. Does the mode of feeding play a role in the pathogenesis of enteral feeding-related diarrhoea? *Proc Nutr Soc* 1997;**56**:216A.
- 79 **Rees RGP**, Keohane PP, Grimble GK, *et al*. Elemental diet administered nasogastrically without starter regimens to patients with inflammatory bowel disease. *JPEN J Parenter Enteral Nutr* 1986;**10**:258–62.
- 80 **Cummings JH**. Short chain fatty acids in the human colon. *Gut* 1981;**22**:763–79.
- 81 **Stephen AM**, Cummings JH. Mechanisms of action of dietary fibre in the human colon. *Nature* 1980;**284**:283–4.
- 82 **Dobb GJ**, Towler SC. Diarrhoea during enteral feeding in the critically ill: a comparison of feeds with and without fibre. *Intensive Care Med* 1990;**16**:252–5.
- 83 **Patil DH**, Grimble GK, Keohane P, *et al*. Do fibre containing enteral diets have an advantage over existing low residue diets? *Clin Nutr* 1985;**4**:67–71.
- 84 **Gershon-Cohen J**, Shay H, Feks SS. The relation of meal temperature to gastric motility and secretion. *Am J Roentgenol Radium Ther Nucl Med* 1940;**43**:237–42.
- 85 **Silk DBA**. Towards the optimization of enteral nutrition. *Clin Nutr* 1987;**6**:61–74.
- 86 **Guenther PA**, Settle RG, Perlmutter S, *et al*. Tube feeding-related diarrhoea in acutely ill patients. *JPEN J Parenter Enteral Nutr* 1991;**15**:277–80.
- 87 **Levy J**, Van Laethem Y, Verhaegen G. Contaminated enteral nutrition solutions as a cause of nosocomial bloodstream infection: A study using plasmid fingerprinting. *JPEN J Parenter Enteral Nutr* 1989;**13**:228–34.
- 88 **Thurn J**, Crossley K, Gerdtz A, *et al*. Enteral hyperalimentation as a source of nosocomial infection. *J Hosp Infect* 1990;**15**:203–17.
- 89 **Driks MR**, Craven DE, Celli BR, *et al*. Nosocomial pneumonia in intubated patients given sucralfate as compared with antacids or histamine type 2 blockers. The role of gastric colonization. *N Engl J Med* 1987;**317**:1376–82.
- 90 **Bussy V**, Marechal F, Nasca S. Microbial contamination of enteral feeding tubes occurring during nutritional treatment. *JPEN J Parenter Enteral Nutr* 1992;**16**:552–7.
- 91 **Anderson KR**, Norris DJ, Godfrey LB, *et al*. Bacterial contamination of tube-feeding formulas. *JPEN J Parenter Enteral Nutr* 1984;**8**:673–8.
- 92 **Anderton A**, Aidoo KE. The effect of handling procedures on microbial contamination of enteral feeds. *J Hosp Infect* 1988;**11**:364–72.
- 93 **Payne-James JJ**, Rana SK, Bray MJ, *et al*. Retrograde (ascending) bacterial contamination of enteral diet administration systems. *JPEN J Parenter Enteral Nutr* 1992;**16**:369–73.
- 94 **Lee B**, Chang WRS, Jacobs S. Intermittent nasogastric feeding: a simple and effective method to reduce pneumonia among ventilated ICU patients. *Clin Intensive Care* 1990;**1**:100–2.
- 95 **Walike BC**, Walike JW. Relative lactose intolerance: a clinical study of tube fed patients. *JAMA* 1977;**238**:948–51.
- 96 **Woolfson AMJ**, Ricketts CR, Hardy SM, *et al*. Prolonged nasogastric tube feeding in critically ill and surgical patients. *Post Grad Med J* 1976;**52**:678–82.
- 97 **Gault MH**, Dixon ME, Doyle M, *et al*. Hypernatraemia, azotaemia and dehydration due to high protein tube feeding. *Ann Intern Med* 1968;**68**:778–91.
- 98 **Solomon S**, Kirby DF. The refeeding syndrome: a review. *JPEN J Parenter Enteral Nutr* 1990;**14**:90–7.
- 99 **Havala T**, Shronts E. Managing the complications associated with refeeding. *Nutr Clin Pract* 1990;**5**:23–9.
- 100 **Flatt JP**, Blackburn GL. The metabolic fuel regulatory system: implications for protein-sparing therapies during caloric deprivation and disease. *Am J Clin Nutr* 1974;**27**:175–87.

HOMA of the relationships between IS and typical correlates, such as obesity, insulin secretion and glucose tolerance.² Under these conditions, the mathematical modelling approach based on 24 h circadian rhythm of glucose and insulin suggested by Nobili has a different meaning to “stressing” glucose homeostasis during an oral glucose test. This test is more physiological and reflects the effects of insulin throughout the day. Also, measuring insulin secretion would add importantly to the understanding of the process, but the test remains extremely cumbersome and unsuitable for clinical studies.

The differential impact of basal and post-load insulin resistance on liver fibrosis might reflect the intrinsic difference in the physiological meaning between HOMA-R and OGIS, although the complex interplay between insulin resistance and liver damage is still unknown. In chronic hepatitis C (CHC), insulin resistance may be attributed both to host factors and to a possible interference of hepatitis C virus with intrahepatic insulin signalling. In genotype-1 CHC, we and others³ failed to identify an independent association of HOMA-R with liver fibrosis. On the contrary, this association was found in genotype-3 CHC patients, with rare or no components of the metabolic syndrome, where the low degree of insulin resistance might reflect a virus-related hepatic insulin resistance, quantitatively measured by HOMA-R.

In the analysis, we introduced both HOMA-R and OGIS into the model without evidence of collinearity. This is further evidence suggesting that the two surrogate indices, although statistically correlated with each other and both with the clamp, clearly measure two different processes.

Insulin sensitivity has a gaussian distribution in the general population. As such, for each method a population reference is needed, derived from subjects with similar characteristics (ethnicity, BMI, etc). Although investigators commonly use cut-offs published in large studies, none of them can be taken for granted. The cut-offs of HOMA-R and OGIS we used are derived from our personal experience (HOMA-R) or from the large experience of the group that described OGIS. We apologise for a mistake in the reference of the HOMA-R cut-off of 2.7. The correct reference study for HOMA-R in our setting was reported elsewhere.⁴

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REFERENCES

1. **Pacini G, Mari A.** Methods for clinical assessment of insulin sensitivity and beta-cell function. *Best Pract Res Clin Endocrinol Metab* 2003;**17**:305–22.

2. **Mari A, Pacini G, Brazzale AR, et al.** Comparative evaluation of simple insulin sensitivity methods based on the oral glucose tolerance test. *Diabetologia* 2005;**48**:748–51.
3. **Camma C, Bruno S, Di Marco V, et al.** Insulin resistance is associated with steatosis in nondiabetic patients with genotype 1 chronic hepatitis C. *Hepatology* 2006;**43**:64–71.
4. **Marchesini G, Pagotto U, Bugianesi E, et al.** Low ghrelin concentrations in non-alcoholic fatty liver disease are related to insulin resistance. *J Clin Endocrinol Metab* 2003;**88**:5674–9.

Is ileocaecal Crohn’s disease L1 or L3 according to the Montreal classification?

In a recent issue of the journal, Satsangi *et al* reviewed the key issues that have emerged from discussions of the Montreal Working Party (*Gut* 2006;**55**:749–53). One problem that I have encountered in my clinical practice is to define ileocaecal Crohn’s disease according to the Montreal classification. In both articles on the Montreal classification, terminal ileum involvement is L1, colonic disease is L2, and ileocolonic involvement is L3.¹ Should we consider ileocaecal Crohn’s disease as L1 or L3 according to the Montreal classification?

I decided to interview 27 French and international experts in the field of inflammatory bowel disease via email asking them “What is ileocaecal Crohn’s disease according to the Montreal classification?” Fifteen out of 27 (55.6%) colleagues classified ileocaecal Crohn’s disease as L1, while the 12 remaining experts (44.4%) responded L3.

What can explain such discrepancy between the experts? Most experts who answered L1 argued that the caecum is the end of the small intestine and that caecal involvement is not sufficient to be considered as colonic disease, while those who classified ileocaecal Crohn’s disease as L3 explained that the caecum is an integral part of the colon.

I think we forget that the Montreal classification is based on the same definitions as the original Vienna classification, as it is a revised version of the Vienna classification.^{1,2} Indeed, it is clearly stated in the original paper on the Vienna classification that the term “terminal ileum” covers disease limited to the lower third of the small bowel with or without spill-over into the caecum.² In this regard, the term “terminal ileum” used in both articles on the Montreal classification may be misleading.¹

Recently, Offerlbauer-Ernst *et al* confirmed that discrepancies in the Vienna classification existed mainly for L1 and L3, and concluded that the presence of coexisting colonic lesions may lead to disagreement between observers.³ The authors proposed an alternative, segment-wise description of Crohn’s disease as ileal, right colonic, transverse colonic, left colonic or rectal disease.³

This might result in an improvement of L1 and L3 interobserver agreement to 85%.³

In conclusion, because it is well established that diagnostic misclassification reduces the ability to detect linkage in inflammatory bowel disease genetic studies,⁴ we should keep in mind that, similarly to the Vienna classification, L1 corresponds to pure ileal or ileocaecal Crohn’s disease according to the Montreal classification.

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REFERENCES

1. **Silverberg MS, Satsangi J, Ahmad T, et al.** Toward an integrated clinical, molecular and serological classification of inflammatory bowel disease: Report of a Working Party of the 2005 Montreal World Congress of Gastroenterology. *Can J Gastroenterol* 2005;**19**(Suppl A):5–36.
2. **Gasche C, Scholmerich J, Brynskov J, et al.** A simple classification of Crohn’s disease: report of the Working Party for the World Congresses of Gastroenterology, Vienna 1998. *Inflamm Bowel Dis* 2000;**6**:8–15.
3. **Offerlbauer-Ernst A, Miehsler W, Eckmuller O, et al.** Impact of interobserver disagreement on phenotype-genotype associations in Crohn’s disease. *Inflamm Bowel Dis* 2007;**13**:156–63.
4. **Silverberg MS, Daly MJ, Moskovitz DN, et al.** Diagnostic misclassification reduces the ability to detect linkage in inflammatory bowel disease genetic studies. *Gut* 2001;**49**:773–6.

CORRECTIONS

Osonnaya C, Osonnaya K, Abdi M, *et al.* Effect of *Helicobacter pylori* eradication on dyspepsia, quality of life and utilisation of health care resources in the Eastern England *Helicobacter Pylori* project: randomised control trial (*Gut* 2007;**56**(Suppl II):A16).

It has come to the editor’s notice that the wording of this abstract closely resembles that of an article published in the *BMJ* (Lane J A, Murray L J, Noble S, *et al.* Impact of *Helicobacter pylori* eradication on dyspepsia, health resource use, and quality of life in the Bristol *Helicobacter* project: randomised controlled trial. *BMJ* 2006;**332**:199–204). We therefore wish to withdraw the abstract by Osonnaya *et al.*

We also wish to withdraw the following abstracts, which closely resemble previously published articles by other authors.

Osonnaya C, Osonnaya K, Swain P. Investigating the link between mast cell density and severity of *Helicobacter pylori* gastritis in the corpus and antrum. *Gut* 2005;**54**(Suppl II):A85. This abstract withdrawn at the request of Professor Swain.

Osonnaya C, Swain P C, Sanderson I R. Mast cell density in the antrum and corpus: increase in *Helicobacter pylori* gastritis. *Gut* 2003;**52**(Suppl V1):A153. This abstract withdrawn at the request of Professor Sanderson.

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P Abdulhannan, J W L Puntis. Iron deficiency anaemia and perianastomotic ulceration as a late complication of ileal resection in infancy

(*Gut* 2007;**56**:1478–9). The first author's name for this letter was published incorrectly and should be Peshang Abdulhannan. Furthermore, the letter should have read "We were interested..." not "I was interested ...".

Committee on Publication Ethics (COPE) – Seminar 2008

9.30am–4.30pm Friday 4 April 2008, Woburn House, London, UK

This year's seminar will focus on three key topics: (1) How does patient privacy legislation affect an editor's ability to publish? (2) What is publication? — the changing definitions of publication. (3) COPE's new Best Practice Guidelines. There will also be a short demonstration of an anti-plagiarism system as it is working in a publishing house.

Invited speakers will discuss legislation on privacy and data protection that editors need to be aware of; how editors should respond to more and more data being available online prior to formal peer-reviewed publication; and what happens to a publication after it appears in print.

The newly designed COPE website will be demonstrated, and there will be interactive workshops on common ethical and editorial dilemmas.

Editors, authors and all those interested in improving the standard of publication ethics are welcome.

The seminar will include invited talks:

- ▶ A Pandora's box of tissues—legislation in relation to tissues and cells
- ▶ The promise and perils of patient privacy
- ▶ Pre-publication or duplicate publication? How to decide
- ▶ What really happens to a publication after it appears in print
- ▶ Screening for plagiarism: the CrossCheck initiative

In addition:

- ▶ Discussion of COPE's new Best Practice Guidelines with experiences from journals who have piloted the audit
- ▶ COPE's new website unveiled
- ▶ Interactive workshops on the key topics of the seminar.
- ▶ Opportunities to network with other editors and share your experiences and challenges

The seminar is free for COPE members and £50.00 for non-members. Numbers are limited and early booking is advisable. For registration or more information please contact the COPE Administrator at cope@bmjgroup.com or call 020-7383-6602.

For more information on COPE visit www.publicationethics.org.uk/