BAPEN Symposium 9: Choosing enteral feeds: evidence based or gut reaction?

Formulation of enteral diets for use in jejunal enteral feeding

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Nasogastric enteral feeding is not tolerated in patients with gastric atony and in many critically-ill patients in whom gastric emptying may be delayed and in whom gastro-oesophageal regurgitation may lead to pulmonary aspiration of enteral feed and the development of pneumonia. Initial attempts to overcome these problems led to the development of post pyloric enteral feeding techniques with the infusion port of the tubes positioned in the duodenum. In many centres this technique is still the most practised post-pyloric enteral feeding technique. Nasoduodenal feeding tubes often retroperistalse into the stomach. The technique of choice, therefore, in these difficult patients is to position the infusion port of the feeding tube well distal to the ligament of trietz (post ligament of trietz nasojejunal enteral tube feeding). While nasogastric and nasoduodenal enteral feeding techniques have been shown to elicit a stimulatory exocrine pancreatic response, distal jejunal enteral feeding does not. During this mode of feeding the ileal brake is activated and pancreatic exocrine pancreatic secretion inhibited by the action of the released peptide YY and glucagon-like peptide-1 hormones, in turn the inhibition of pancreatic secretion being the result of inhibition of trypsin secretion. In the light of the findings showing the absence of a stimulatory pancreatic exocrine response to nasojejunal enteral feeding these patients should receive a predigested rather than a polymeric enteral diet.

Nasoduodenal feeding: Nasojejunal feeding: Predigested enteral diets

In patients requiring nutritional support who have a normally- or near-normally-functioning gastrointestinal tract enteral nutrition is preferred to parenteral nutrition[1]. Parenteral nutrition may often be associated with increased complications and, in critically-ill patients, an increased mortality[2].

Enteral nutrition is not, however, without problems in some patients. Problems occur, for example, in patients with gastric atony (Table 1) receiving nasogastric feeding. Gastro-oesophageal regurgitation and pulmonary aspiration of the diet is a common complication of nasogastric feeding in patients who are critically ill and ventilated, leading to the development of pneumonia[3,4]. This route of feeding is poorly tolerated in patients with neuromuscular disorders of the gut[5].

The categorisation of intragastric enteral-feeding complications has pointed clinicians to developing techniques of post-pyloric enteral feeding[6]. One of the most common problems with this technique, apart from difficulties of tube placement[7], has been that only approximately one-third of positioned tubes remain in situ for the full course of nasoduodenal feeding; the remainder being either inadvertently removed or recoiling back into the stomach as a consequence of retroperistalsis[6]. Most recently, therefore, techniques of nasojejunal tube feeding have been developed to overcome these problems and the indications for jejunal enteral feeding defined (Table 2). Long, usually single-lumen, feeding tubes are sited using standard upper gastrointestinal endoscopy-assisted techniques; radiologically using newly-developed guide wires (the author's preferred method of tube placement) or with the assistance of a newly-developed nasoendoscopic technique[8]. In some patients who go on to require longer-term jejunal feeding nasojejunal tubes can be replaced by directly-sited jejunal tubes, e.g. a needle-catheter jejunostomy tube sited at mini laparotomy or a percutaneously endoscopically-sited

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Table 1. Patient groups and diseases associated with gastric atony

Diabetes with neuropathy
Hypothyroidism
Head injuries and patients undergoing neurosurgery
Multiple trauma
Patients post abdominal surgery
Intra-abdominal sepsis
Patients in intensive care on ventilators
Some patients with neuromotor deglutition disorders
Some patients following a cerebrovascular accident

Table 2. Indication for jejunal enteral feeding

Patients in the intensive treatment unit
Patients on ventilation
Patients with neuromuscular disorders of gut
Patients with gastric atony
Some patients post-operatively after surgery

tube[7]. One of the issues that arises when considering nasojenunal and other techniques of jejunal enteral nutrition is what type of enteral diet should be employed, i.e. polymeric or predigested? Clearly, the answer to this question will be dependent on what influence intra-jejunally-administered enteral nutrition will have on the pancreatic secretory response. The present article examines this evidence.

Food-induced pancreatic secretion

The stimulatory effect of feeding on pancreatic exocrine function has been divided into three phases: the cephalic phase; the gastric phase; the intestinal phase. Following the cephalic phase there is strong vagal stimulation when food enters the mouth, is chewed and swallowed, and further vagal stimulation when food enters the stomach. The movement of food plus gastric secretions through the pylorus into the duodenum results in the maximal stimulation of pancreatic secretion mediated by humoral secretion of cholecystokinin and secretin; the combined stimulation being fortified by cholinergic excitation[9].

Enteral feeding-stimulated pancreatic secretin

The stimulatory effect of polymeric and 'elemental' enteral diets on pancreatic secretion has been examined with diets either being administered orally or infused at the site of the ampulla of Vater with intestinal contents being aspirated 200 mm distally[10]. The secretory response to oral ingestion of the polymeric diet was found to be similar to that of the infused diet. However, pancreatic secretion was found to be reduced by 50 % in response to ingestion of a predigested elemental diet (L-amino acid N source, 6 % energy only derived from fat). It is clear from these basic physiological studies that patients receiving post-pyloric intra-duodenal feeding should receive a polymeric enteral diet, the pancreatic secretory response being entirely adequate to result in assimilation of nutrients from the diet.

Inhibition of pancreatic secretion

In 1984 two research groups (one of which was the author's group) independently described an important humorally-mediated braking mechanism whereby jejunal transit and motility is inhibited by the infusion of nutrients in the terminal ileum[11,12]. This mechanism was termed the 'ileal brake' and the hormones enteroglucagon, neurotensin and peptide YY were thought to mediate the braking effects. Later studies have also implicated glucagon-like peptide-1[13]. Importantly, later studies of the ileal brake have shown that stimulated pancreatic secretion can be inhibited by the infusion of unabsorbed nutrients into the ileum[14,15] and that the inhibitory action is mediated via the release of peptide YY and glucagon-like peptide-1[16]. These very important studies led to an investigation of the effect of the site of jejunal feeding on stimulated pancreatic secretion[17]. Normal subjects received a polymeric enteral diet infused either at the ligament of trietz (proximal jejunum) or 600 mm distally (mid jejunum). Proximal jejunal feeding was found to provide a strong stimulus for lipase, trypsin, amylase and bilirubin secretions. In contrast, feeding at 600 mm down the jejunum was found to have no significant effects on basal secretion rates[16]. Subsequently, it has been confirmed in healthy volunteers that there is no significant pancreatic secretory response to the infusion of a predigested diet at distances of 400–1200 mm from the ligament of trietz[18]. This lack of stimulatory exocrine pancreatic response has been shown to occur in association with elevated peptide YY and glucagon-like peptide-1 responses[18], indicating that the ileal brake is activated during intra-jejunal nutrient infusion. The same research group has developed a technique for measuring trypsin synthetic rates[19] and using this technique have shown that inhibition of pancreatic secretion is the result of inhibition of trypsin synthesis[20].

Predigested and polymeric diets during jejunal enteral feeding

For the reasons discussed, post-pyloric duodenal feeding techniques are not the optimum means of overcoming the problems of complications that occur with intragastric enteral nutrition. Properly-sited access beyond the ligament of trietz is the way forward in those difficult cases; certainly, initially and in the short term nasojejunal enteral-tube feeding is the technique of choice. In patients with normal small intestinal motility nasojejunal tubes, once they are correctly positioned, will be moved in a caudad direction by the phase III migrating motor complex[1]. It must be assumed therefore that in most patients the diet will be infused approximately 300–400 mm distal to the ligament of trietz. Infusion of polymeric diet at this site will activate the ileal brake, causing release of peptide YY and glucagon-like peptide-1. In turn this response will result in an inhibition of exocrine pancreatic enzyme synthesis and secretion, which is likely to have a most deleterious effect on the digestion and absorption of

nutrients from the polymeric diet. Although the evidence points to the fact that the intrajejunal infusion of pre-digested diets also fails to elicit a pancreatic exocrine secretory response[18], near optimum absorption of nutrients can occur from suitably-formulated diets in the absence of pancreatic enzymes[1].

On the basis of the evidence discussed it is concluded that properly-formulated predigested enteral feeds are the diets of choice for patients undergoing post-ligament of trietz intrajejunal enteral feeding.

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