Perioperative nutritional support: does it reduce hospital complications or shorten convalescence?

During the 1960–1970s we learnt how artificial feeding could rescue a few severely undernourished patients. Next, research characterised the integrative biochemistry of illness and sought ways by which the catabolic responses could be influenced nutritionally and hormonally. More recently the large proportion of hospital patients suffering from undernutrition, as judged by both weight for height and by micronutrient status, has become widely appreciated and there has been an interest in more routine supplementation of hospital patients. As the practice of supplementation increases there has been a growing need for randomised controlled trials to assess its clinical effectiveness.

The physiological effects of undernutrition in the otherwise normal human have been well known for about 50 years. As far as sip supplements are concerned there is now excellent evidence that they do not simply replace the intake of normal food but effectively increase nutrient intake and improve nutritional status.

Surgeons and gastroenterologists need to know the best nutritional strategies for patients undergoing major abdominal surgery. Preoperative undernutrition has long been known to predict poor surgical outcome. Preoperative feeding can reduce the increased risk of postoperative complications in nutritionally depleted patients. Enteral feeding is at least as good as parenteral feeding in this context but it is not clear if the time cost of preoperative feeding is justified by its being more effective than early postoperative support. Postoperatively, parenteral feeding should not be used routinely because it increases complication rates in well nourished patients and this technique should be reserved for the severely undernourished and those unable to take nutrients enterally during a prolonged and complicated postoperative course. However, routine very early postoperative enteral feeding of complete polymeric feeds (unlike “immune enhanced” feeds) has been effective in reducing postoperative complications. Furthermore, encouraging the intake of sipfeed supplements containing a full and balanced complement of nutrients as the patient begins to eat seems to reduce even early postoperative complications whether or not the patient is undernourished. Although undernutrition correlates with length of stay, it may be asking much of a routine nutritional supplement to reduce this given that modern average surgical lengths of stay are little over a week.

In the surgical context sip supplements could benefit the patient not only by reducing postoperative complications and hospital stay but also by speeding recovery after discharge from hospital. One study tested the idea that convalescence might be hastened with sip supplements but was unable to demonstrate such an effect as judged by a “well being” visual analogue score. In this study postoperative complications and convalescent weight loss were reduced significantly by supplementation.

The randomised controlled trial described by Beattie et al in this issue of Gut also concentrates on postoperative convalescence. From a total of nearly 2500 patients admitted for elective gastrointestinal or vascular surgery to Ninewells Hospital, Dundee, 450 were screened; 109 were included of whom 101 completed the study and contributed to the results. Patients were either underweight on admission or had lost 5% or more of their body weight from admission to the eight postoperative day and represented a smaller proportion of undernourished patients than had previously been seen at Ninewells. On resumption of oral feeding postoperatively, patients were randomised to receive or not to receive a nutritionally complete liquid supplement of 400 ml (600 kcal). Patients continued on the supplements during the remainder of their admission and during convalescence at home for 10 weeks. Antibiotic use was marginally reduced, length of stay (which seems long in this study) was not altered but, most strikingly, patients regained weight much quicker and had improved physical and mental quality of life scores by the end of the study.

The trial deserves close scrutiny. As the authors emphasised, it was not analysed on an intention to treat basis and this will worry some. This group used a body mass index (BMI) of <20 kg/m² (which is close to the fifth centile for an adult British population) or the 15th centile for arm anthropometric measurements. The arm anthropometric inclusion data and the perioperative weight loss criteria contrived to include about 20% of patients with a BMI above 20. It is not clear whether such patients fared differently from the more clearly undernourished; personal communication from the authors suggests not. The age difference between the groups was unfortunate and may produce some bias.

None the less the study is an important reminder to surgeons of the prolonged impact of surgery on weight and quality of life after discharge from hospital and demonstrates a potential need for continuing care as patients move from the surgical ward back to the community. Weight loss in the control group continued for eight weeks (longer by several weeks than was found in the control group of Keele et al’s study), but was reversing at two to four weeks with nutritional intervention.

Much is made of the economical advantages to the NHS of rapid hospital turnover but less often is the economic impact of illness and surgery on the patient. A faster convalescence time should be an important goal for every surgical team, and hospital dietitians linking with community services may be in a position to help deliver this. We can be confident that such an approach will speed up regain in weight, but whether this speeds general convalescence remains a matter of dispute between this study and that of Keele et al.

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Morphine and biliary pain revisited

Pain in the biliary tract is assumed to originate from either an obstructive event (the gall bladder contracting on a partially obstructed bile duct on ultrasonography) or an inflammatory event (the gall bladder contracting on a partially obstructed bile duct). Pain in the biliary tract is assumed to originate from either an obstructive event (the gall bladder contracting on a partially obstructed bile duct) or inflammation of the gall bladder (closed common duct, blocked by a gall stone or inflammation of the gall bladder). Pain in the biliary tract is assumed to originate from either an obstructive event (the gall bladder contracting on a partially obstructed bile duct) or inflammation of the gall bladder (closed common duct, blocked by a gall stone or inflammation of the gall bladder).

The "post cholecystectomy syndrome" is a poorly defined entity which includes many symptoms that range from the trivial (mild dyspepsia) to severe attacks of pain. Pain in the biliary tract is assumed to originate from either an obstructive event (the gall bladder contracting on a partially obstructed bile duct) or inflammation of the gall bladder (closed common duct, blocked by a gall stone or inflammation of the gall bladder).

Type I
- Biliary pain plus:
  - Delayed drainage of the common bile duct (> 45 minutes)
  - Dilated common bile duct (> 12 mm on ultrasound)
  - Elevated aminotransferase, alkaline phosphatase or bilirubin (> 2×normal on 2 occasions)

Type II
- Biliary pain plus 1 or 2 of:
  - Delayed drainage of the common bile duct (> 45 minutes)
  - Dilated common bile duct (> 12 mm on ultrasound)
  - Elevated aminotransferase, alkaline phosphatase or bilirubin (> 2×normal on 2 occasions)

Type III
- Biliary pain alone

See article on page 838

| Table 1 Milwaukee classification of biliary sphincter of Oddi dysfunction |
|-----------------|-----------------|-----------------|
| Type I          | Type II         | Type III        |
| Biliary pain plus: | Biliary pain plus 1 or 2 of: | Biliary pain alone |
| - Delayed drainage of the common bile duct (> 45 minutes) | - Delayed drainage of the common bile duct (> 45 minutes) | |
| - Dilated common bile duct (> 12 mm on ultrasound) | - Dilated common bile duct (> 12 mm on ultrasound) | |
| - Elevated aminotransferase, alkaline phosphatase or bilirubin (> 2×normal on 2 occasions) | - Elevated aminotransferase, alkaline phosphatase or bilirubin (> 2×normal on 2 occasions) | |
tactical markers of bile to time their clearance from the biliary tree. Difficulties arise in the presence of intrahepatic cholestasis. Such computer assisted choledochoscinography identifies SO dysfunction as a delayed clearance of marker from the biliary system15 or a prolonged transit from the hepatic hilum to the duodenum.15 Although the Rome II consensus recommended this as a valid screening test, its precision is less than ideal.

Thomas et al in this issue13 (see page 838) used morphine provocation to elicite the hypersensitivity of the dysfunctional SO to opiates and so increase the detection accuracy of biliary scanning. In 34 patients with type II (n=21) or type III (n=12) SO dysfunction, routine 99mTc-IDA quantitative biliary scanning could not distinguish between those with normal (n=16) and those with abnormal (n=18) sphincteric tone (abnormal >40 mm Hg). With low dose morphine provocation (0.04 mg/kg intravenously over five minutes), differences became evident with a sensitivity of 83% and a specificity of 81%. Further, 14 of the 18 patients with abnormal SO manometry experienced biliary pain after morphine compared with two of 16 with normal manometry. Whether or not those patients with positive tests would have benefited from sphincterotomy is unknown as the study did not examine this therapeutic end point.

One must be careful in attributing all biliary-type pain to a motility disorder affecting only the sphincter of Oddi. The abnormality could reside elsewhere as a more generalised motor disorder of the gut, from the oesophagus through the duodenum-jejunum,15 or perhaps as part of the spectrum of the irritable bowel syndrome.18 Altered sensation in the form of visceral hypersensitivity is a potential basis for pain in functional gastrointestinal disorders. An abnormal sensitivity to a relatively innocuous stimulus could result from modified receptor sensitivity at the level of the sphincter or adjacent viscus, increased excitability of neurones in the dorsal horn of the spinal cord, and/or altered central modulation of sensory inputs. The bile ducts per se could be the pain trigger zone.15 Conversely, a recent study in patients with type III SO dysfunction implicated duodenal hyperalgesia as the basis for the biliary-type pain.19 The rich neural connections between the gall bladder and SO, and the biliary tract and the stomach and small intestine orchestrate the interplay between motility and sensitivity of the biliary system and the foregut.

Use of biliary scans with morphine provocation should provide a more effective non-invasive screening test before embarking on manometry and/or sphincterotomy. If combined with barostat-type evaluations of duodenal hyper-sensitivity, the mystery of the sphincter of Oddi—a motility disorder or a supersensitive sphincter/viscus as the cause of the pain—might be solved.
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