25 Nutrition and f_l uid therapy

ARTIFICIAL NUTRITIONAL SUPPORT

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Given the importance of adequate nutrition in recovery from illness and surgery , consideration for artificial nutritional support should be given in any patient who has had inadequate nutritional intake for 5 days or more. In patients with pre-existing chronic malnutrition, this should be instituted earlier, and ideally in the preoperative period if feasible. Patients due to undergo major surgery for head and neck or abdominal cancers (such as laryngeal or pharyngeal resections, oesophagectomies, gastrectomies and pancreaticoduodenectomies) are more likely to have di ffi culty consuming any or su ffi cient oral nutrition postoperatively because of oedema, obstruction, delayed gastric emptying and paralytic ileus. These patients are also more e ff ects of the underlying disease. Forethought should be given preoperatively in these patients regarding the placement of intra venous access, nasojejunal tubes or feeding jejunostomies intraoperatively to facilitate postoperative nutrient delivery (Figure 25.3). ARTIFICIAL NUTRITIONAL SUPPORT

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Anthropometry

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Anthropometry uses several di ff erent parameters to obtain an estimate of body composition as a surrogate for nutritional status. These parameters can include weight and percentage 2 2 weight change, body mass index (BMI) (weight [kg]/height [m]), mid-upper arm circumference (MUAC), skinfold thickness (TSF) and mid-arm muscle circumference (MAMC), where ments are indirect assessments of energy and protein stores and are not su ffi ciently accurate to facilitate planning of nutritional support regimens. BMI, in particular, has often been used as a quick screening measure to identify those who are malnourished. A BMI of less 2 than 18.5 /uni00A0 kg/m and unintentional weight loss greater than 10% within the last 3–6 months or a BMI of less than 20 /uni00A0 kg/m and unintentional weight loss greater than 5% within the last 3–6 months are indicators of a need for nutrition support. It is important to note, however, that both BMI and body weight can be altered by major changes in fluid balance, and thus may not be reliable indicators of nutritional status in critically ill patients. Anthropometry

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Biochemistry

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Biochemical tests can be used in conjunction with clinical history , examination, comorbidities and drug history . Albumin, C-reactive protein and white cell counts can be markers of infection or inflammation, which can compromise nutritional status. Hypoalbuminaemia can be associated with malnutrition; however, it is easily a ff ected by fluid balance and is not a reliable parameter of nutritional status in the acute setting. Haemoglobin levels can indicate the presence of anaemia related to a lack of appropriate vitamins. Glycated haemoglo bin can reflect diabetes and blood glucose control. Electrolytes such as sodium and urea can reflect under lying renal function, while calcium and phosphate are useful baseline measurements in anticipation of potential refeeding syndrome (discussed in more detail later in this chapter). Specific micronutrient lev such as vitamin D levels can also be measured in the appropri ate clinical contexts. Biochemistry

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Clinical evaluation

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Clinical assessment of nutritional status should begin by consideration of any important symptoms that may suggest malnutrition. Upper abdominal symptoms such as nausea and vomiting, early satiety , dysphagia, reflux or bloating as well as lower gastrointestinal symptoms of diarrhoea or constipation can all indicate inadequate nutritional intake or absorption. A thorough assessment of the past medical history and comorbidities is also essential in assessing nutritional status, as conditions such as cancer, gastrointestinal pathologies (e.g. inflammatory bowel disease and liver disease) and neurological conditions (e.g. stroke , Parkinson’s disease and dementia) can all contribute to a ff ect nutritional status. Nutrient absorption can be impaired by conditions directly a ff ecting the bowel such as short bowel syndrome, high-output stoma and enterocutaneous fistulae, and also by disorders more proximally in the gastrointestinal tract such as pancreatic insu ffi ciency , in which absorption is impaired because of a lack of pancreatic enzyme secretion into the bowel. These conditions and the appropriate management are dealt with in the relevant chapters in this book. James Parkinson , 1755–1824, general practitioner of Shoreditch, London, UK, published The total daily calorie intake of an individual can be estimated via a diary of their food and fluid intake, taking into account the quality of the food or fluid consumed. For patients who are unwell, this needs to take into account any di ff erences in current food and fluid consumption compared with their 2 typical intake when well. Their caloric intake can be assessed against their calculated energy requirements, estimated with the calculation of 25–35 /uni00A0 kcal/kg lean body weight and taking into account any metabolic stresses and activity level. Patients whose caloric intake falls short of their caloric requirements or who are anticipated to eat little or nothing for over 5 continuous days in the near future (e.g. owing to upcoming abdominal surgery) are likely to requir e nutritional support. The Malnutrition Universal Screening Tool (MUST), developed by the British Association for Parenteral and Enteral Nutrition (BAPEN), is a rapid screening tool that can be used in both hospitals and the community , and takes into account a combination of the above factors to identify those individuals a t risk of malnutrition (Figure 25.2). Patients who are found to be likely to be malnourished require referral to a dietician or nutritional support team, with regular reviews to ensure sus - tained improvement in nutritional status. - Clinical evaluation

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EFFECTS OF INTESTINAL RESECTION ON FLUID AND NUTRI

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The main role of the intestine is the absorption of fluid, nutrients and electrolytes, and as such it has a large capacity for adaptation to the loss of intestinal length by increasing the absorptive surface area as well as molecular changes increasing nutrient transporter levels. This may be due to either surgical resection or a reduction in functional capacity associated with severe cases of chronic inflammatory intestinal conditions such as Crohn's disease or ulcerative colitis. Patients with reduced functional intestinal length may therefore require supplemental parenteral nutrition, intravenous fluid or both, depending on the site and extent of a ff ected bowel. Resection of the proximal jejunum can be compensated by the ileum and colon adapting to absorb the additional fluid and electrolyte load, hence these patients do not require supplemental nutrition. Resection of the ileum, however, may have more significant consequences. The ileum is responsible for bile salt reabsorption and loss of even 100 /uni00A0 cm of ileum may cause steatorrhoea, which can be treated by the administration of cholestyramine for bile salt binding. If greater lengths of ileum are a ff ected, dietary fat restriction may also be necessary . Ileal resection also increases - volume of fluid and electrolytes reaching the colon, causing symptoms of diarrhoea. The greatest consequences of loss of functioning intestinal length occur in patients with remnant small intestine of less than 200 /uni00A0 cm. This results in significantly reduced absorptive capacity , with the associated metabolic and nutritional consequences of short bowel syndrome. Short bowel syndrome (discussed in more detail in Chapter 74) is characterised by symptoms of diarrhoea, malnutrition and dehydration, with variable severity depending on the extent and function of the remaining small bo wel. The acute stage of short bowel syndrome occurs in the first few weeks following the insult. It is characterised by high intestinal losses, gastric hypersecretion and hypergastrinaemia and can result in acute renal failure and acid–base imbalances. The subsequent adaptation stage occurs over 1–2 years and is a consequence of the structural and functional changes within the remnant bowel, allowing increased absorptive capacity and ameliorating some of the earlier symptoms. Su ffi cient recovery may occur in some patients to render parenteral nutrition no longer necessary; however, some features of intestinal insu ffi ciency may still remain, requiring special diets, supplementation of nutrients and some pharmacological treatments. Intestinal rehabilitation programmes have been developed over the last decade to optimise intestinal function in short bowel syndrome as much as possible; however, recovery of function to allow weaning from home parenteral nutrition becomes unlikely beyond 3 years of onset. Intestinal transplantation is an option in those dependent on lifelong parenteral nutrition; this is covered in greater detail in Chapter 91 . Patients who have less than 100 /uni00A0 cm of total residual bowel have a particularly severe form of short bowel syndrome as they will lose more water and electrolytes from their bo wel than consumed by mouth. Daily bowel losses can exceed 4 /uni00A0 litres in a 24-hour period. Consumption of oral fluids with sodium concentrations of less than 90 /uni00A0 mmol/L will result in a net e ffl ux of sodium from plasma into the bowel lumen, hence hypotonic fluids should be restricted to less than 1 litre per day and patients should be encouraged to drink glucose and saline replacement solutions such as oral rehydration salts. Fluid balance needs to be carefully monitored; while some of the fluid intake will be covered by parenteral nutrition, further intravenous fluid supplementation may also be necessary in cases of particularly high bowel output. EFFECTS OF INTESTINAL RESECTION ON FLUID AND NUTRIENT ABSORPTION

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Enteral nutrition

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Enteral nutrition (the delivery of nutrients into the gastrointes - tinal tract) should always be the preferred route of administra - tion of nutrition where possible. Benefits of enteral nutrition include preservation of the gut mucosal barrier and immunity and prevention of gut atrophy . The use of enteral n utrition is also associated with reduced infection rates, better wound healing and a reduced length of stay compared with parenteral nutrition. Supplementary enteral nutrition can be in the form of oral supplements as well as via tube-feeding techniques such as feeding gastrostomies or jejunostomies and nasogastric or nasojejunal tubes. Enteral feeds contain variable nutrient formulations with respect to the content of energy , fat and nitrogen, as well as the osmolarity and nutrient complexity . In general, most feed for - mulations contain 1–2 /uni00A0 kcal/mL and up to 0.6 /uni00A0 g/mL protein. Oral supplements Many liquid oral supplements are commercially available, supplying around 200 /uni00A0 kcal and 2 /uni00A0 g of nitrogen per 200-mL carton. These can be used to increase daily caloric intakes in addition to that provided by diet alone, and are useful when weaning patients o ff tube-feeding regimens.

Nasogastric/ duodenal/jejunal tube Whole food PPN TPN by mouth Gastrostomy tube Jejunostomy tube Figure 25.3 Routes available for delivery of arti /f_i cial nutritional sup

port. PPN, partial parenteral nutrition; TPN, total parenteral nutrition. (Redrawn with permission from Rick Tharp, rxkinetics.com.)

Patients who are unable to maintain adequate nutritional intake with oral supplements will need administration of enteral feed via tube feeding. This can be prepyloric either via a conventional nasogastric (Ryle’s) tube or a fine-bore feeding tube inserted into the stomach or via a surgical or endoscop ically placed gastrostomy . Feed can also be delivered beyond the pylorus via a nasojejunal tube or surgical or endoscopic feeding jejunostomy . The enteral feeding regime is best planned and managed by a trained dietician as administration of enteral feed r equires calculation of the patient's nutritional requirements to allow caloric requirements to be met but at a su ffi ciently g radual rate of increase to prevent the onset of refeeding syndrome in the chronically malnourished patient. The rate of feeding typ ically starts at 10–20 /uni00A0 mL/h and can increase to approximately 75 /uni00A0 mL/h if tolerated. Enteral feeding protocols should include aspiration of the tube, if of su ffi ciently wide calibre, to reduce the risk of nosocomial aspiration pneumonia by reducing or stopping the administration of enteral feed if aspirate v olumes are high. Tube blockage is common and can be pr evented by regular flushing with water daily . Specific agents such as chymotrypsin may be used to unblock a partially obstructed tube; however, guidewires should not be used because of the risk of perforation of the tube and thus damage to the lumen of the stomach or bowel. A radio-opaque nasogastric or Ryle's tube can be used for short-term feeding in the majority of patients and provides the advantage of also having a wide enough calibre to allow aspiration; however, the high-grade polyvinylchloride (PVC) material used can become brittle over time and thus should be changed every 2 weeks. For longer term feeding a fine-bore feeding tube (8–12Fr) may be preferable to minimise the risk of rhinitis, pharyngitis and gastric and oesophageal erosions. These tubes are also less likely to interfere with eating and drinking and are often better tolerated by patients. Techniques for establishment of tube feeding Insertion of nasogastric and nasojejunal feeding tubes Nasogastric tubes can usually be inserted in the ward setting; however, in patients in whom there may be any concerns regarding the oropharyngeal or oesophagogastric anatomy , endoscopic insertion under direct visualisation may be needed. Patients are positioned in a semirecumbent position and the distance between the xiphisternum and the tip of the nose measured. The tube is inserted into the chosen nostril and advanced gently to the 10-cm point. Patients are then encour aged to swallow and the tube simultaneously advanced down the oesophagus with successive swallows until the distance measured to the xiphisternum is reached. The position of the nasogastric tube will need to be checked before feed is adminis tered, either by pH testing (pH <5 is considered safe) or with a c hest radiograph to confirm that the tip of the nasogastric tube is below the diaphragm and well past the bronchial bifurcation. Fine-bore feeding tubes can be inserted in a manner similar to John Alfred Ryle , 1889–1950, Regius Professor of Medicine, University of Cambridge, Cambridge, and later Professor of Social Medicine, University of Oxford, Oxford, UK, introduced the Ryle's tube in 1921. insertion; this must be confirmed to have been removed after . insertion of the tube (Figure 25.4) Feed can also be delivered directly to the jejunum via either tube feeding or surgically created jejunostomies. The advantage of this is that it bypasses the stomach and can - thus overcome problems of delayed gastric emptying without necessitating the use of total parenteral nutrition (TPN). Nasojejunal feeding can also be used in patients who are unable to have a gastrostomy as this is the least invasive form of nutrient delivery into the jejunum. The siting of nasojejunal tubes requires either endoscopic or radiological (fluoroscopic) guidance; therefore, unlike nasogastric tubes, these cannot be inserted in the typical ward setting. Abdominal radiographs can confirm the position of the nasojejunal tube if there is - any concern regarding proximal migration or displacement (Figure 25.5) . Gastrostomy Gastrostomy tubes are generally reserved for patients who require longer term feeding. The decision for insertion of these tubes is increasingly discussed in the multidisciplinary context because of the long-term physical, psychological and lifestyle implications. Gastrostomy insertion can be endoscopic (percutaneous endoscopic gastrostomy [PEG]), radiological (radiologically inserted gastrostomy [RIG]) or surgical (

Figure 25.6) . A PEG involves the insertion of the gastrostomy tube through the abdomen and stomach under vision via an endo - scope, avoiding a surgical incision and a general anaesthetic. The endoscopist is able to visualise a cannula entering the - -

Figure 25.4 A /f_i ne-bore feeding tube with its guidewire.

insu ffl ated stomach via the anterior abdominal wall, through which a guidewire is passed. Then either the gastrostomy tube can be inserted through the anterior abdominal wall over the guidewire or the guidewire can be pulled out via the mouth and the tube secured to the guidewire, pulled down into the stomach and then pulled out through the abdominal wall. The stomach wall is pulled up to the anterior abdominal wall and held in place by a cu ff , balloon or plastic bumper to minimise the risk of intraperitoneal leakage (Figure 25.7). A RIG is an option in patients who are unable to have a PEG because of di ffi culty with oesophageal intubation, compromised respiratory function or oropharyngeal anatomy distortion such as fr om head and neck cancers. A nasogastric tube is inserted to insu ffl ate the stomach and a cannula is inserted under radiographic guidance to facilitate insertion of the gastrostomy device, which is retained internally via a balloon or a pigtail. Contrast can be administered via the RIG to confirm the correct site of placement. A surgical gastrostomy may be necessary in patients who are unable to have either a PEG or a RIG, most commonly because of distorted intra-abdominal anatomy , usually from pr evious surgical intervention. This will require either a laparotomy or a laparoscopy with a small gastrostomy to allow insertion of the feeding tube, which can be held in place either by insu ffl ation of a balloon or by a plastic ‘bumper’. The stomach wall is fixed to the anterior abdominal wall with sutures to minimise intraperitoneal leakage. Some gastrostomy devices also allow the fitting of jejunal extensions, thus allowing venting of stomach contents and simultaneous delivery of nutrients into the jejunum. Complications of a gastrostomy , regardless of the technique of placement, include perforation, bleeding and peritonitis. Localised sepsis around the insertion site is very common and may require systemic antibiotics. Gastrostomies that have been in place f or a long period are likely to develop a persistent gastric fistula on removal owing to epithelialisation of the tract, which may require surgical intervention for closure. Tube blockage may occur, as well as tube displacement. Nasojejunal tubes and jejunostomies Surgical jejunostomies are often created at the time of resection in patients undergoing major oesophagogastric surgery who are likely to have insu ffi cient oral intake in the immediate postoperative period. Jejunostomies require a general anaesthetic and either a laparotomy or a laparoscopy , facilitating the insertion of a feeding tube through the anterior abdominal wall into the pro ximal jejunum. The site of insertion in the jejunum is usually fixed to the anterior abdominal wall to further reduce the risk of leakage. A more recent development is the siting

Figure 25.5 Abdominal radiograph con /f_i rming that the position of

the tip of a nasojejunal feeding tube is past the duodenojejunal /f_l exure. Figure 25.6 Percutaneous endoscopic gastrostomy tube, showing the external bumper and tube clamp Adapter Tubing clamp External bumper Skin Fat Muscle Internal bumper Catheter tip Stomach wall Figure 25.7 Cross-sectional appearance of a percutaneous endo

scopic gastrostomy tube in situ , showing the abutment of the stom

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punctured under image guidance and a guidewire inserted, over which the tract is dilated to allow a feeding jejunostomy tube to be passed. The position of the tube is confirmed with fluoroscopy and the tube anchored to the skin with sutures. Complications of jejunostomy insertion in the periopera tive period include bleeding or tube displacement and leakage causing peritonitis. In the longer term, granulation tissue for mation or localised sepsis at the site of insertion is common.

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Enteral nutrition (the delivery of nutrients into the gastrointes - tinal tract) should always be the preferred route of administra - tion of nutrition where possible. Benefits of enteral nutrition include preservation of the gut mucosal barrier and immunity and prevention of gut atrophy . The use of enteral n utrition is also associated with reduced infection rates, better wound healing and a reduced length of stay compared with parenteral nutrition. Supplementary enteral nutrition can be in the form of oral supplements as well as via tube-feeding techniques such as feeding gastrostomies or jejunostomies and nasogastric or nasojejunal tubes. Enteral feeds contain variable nutrient formulations with respect to the content of energy , fat and nitrogen, as well as the osmolarity and nutrient complexity . In general, most feed for - mulations contain 1–2 /uni00A0 kcal/mL and up to 0.6 /uni00A0 g/mL protein. Oral supplements Many liquid oral supplements are commercially available, supplying around 200 /uni00A0 kcal and 2 /uni00A0 g of nitrogen per 200-mL carton. These can be used to increase daily caloric intakes in addition to that provided by diet alone, and are useful when weaning patients o ff tube-feeding regimens.

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FURTHER READING

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British Association for Parenteral and Enteral Nutrition. Parenteral nutrition . Redditch: BAPEN, 2016. Available from https://www . bapen.org.uk/nutrition-support/parenteral-nutrition/. National Institute for Health and Care Excellence. Nutrition support for adults: oral nutrition support, enteral tube feeding and parenteral nutrition . NICE Clinical Guideline 32. London: NICE, 2017. Available from https://www .nice.org.uk/guidance/cg32/chapter/1-Guidance# parenteral-nutrition-in-hospital-and-the-community/. FURTHER READING

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Introduction

INTRODUCTION

Optimal nutritional status, both pre- and postoperatively , is a key factor in reducing perioperative complications and improving surgical outcomes. However, the pathologies requiring surgical intervention often contribute to malnutrition, and a lack of appreciation of preoperative nutritional status can unnecessar ily increase the risk of the operation and compromise recovery from surgery (Figure 25.1). Some operations, particularly Carl Ferdinand Cori , 1896–1984, Professor of Pharmacology and later Biochemistry , Washington University Medical School, St Louis, MI, USA, and his wife Gerty Theresa Cori , 1896–1957, also Professor of Biochemistry at the Washington University Medical School. In 1947, the Coris were awarded a share of the Nobel Prize in Physiology or Medicine for their discovery of how glycogen is catalytically converted. abdominal operations, can compromise absorption from the gastrointestinal tract, at least temporarily if not permanently . It is therefore imperative that the e ff ects of surgical intervention on the patient’s nutritional status are taken into consideration perioperatively , and appropriate intervention taken as early as possible to correct any nutritional deficits. - Appreciation of the importance of nutritional status is becoming more widespread within the healthcare profession and many tools have been developed to help identify poor nutritional status. These assessment tools and the availability of improved options for both enteral and parenteral delivery of nutrients allows any nutritional deficiency to be expeditiously identified and corrected until normal intestinal absorption can resume.

Figure 25.1 Severely malnourished patient with evidence of fat and muscle wasting. The causes and complications of malnutrition and their • management The options for nutritional intervention and the indications • for enteral versus parenteral nutritional support

Learning objectives

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To understand: The importance of assessment of perioperative nutritional • status and /f_l uid balance The nutritional requirements of surgical patients and the • effects of intestinal resection on nutrition Learning objectives

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Metabolic response to trauma and sepsis

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This is described in greater detail in Chapter 1 , and covered briefly in Summary box 25.2 . It is important to note that the metabolic response to trauma is influenced by the early and rapid rises in sympathetic nervous system activity and circulating catecholamines and elevated levels of glucocorticoids, glucagon and gr owth hormone, as well as insulin. Energy requirements often remain increased to allow tissue repair and inflammatory cell function. Elevated stress hormone levels can lead to net catabolism of tissue protein and thus a negative nitrogen balance. Ketone body production and utilisation may be impaired in the response to trauma, further exacerbating the catabolic response and protein breakdown. Metabolic response to trauma and sepsis

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NUTRITION SUPPORT TEAMS

NUTRITION SUPPORT TEAMS

Multidisciplinary nutrition support teams are essential to ensure that all essential aspects relating to the appropriate - ness of nutritional support, initiation and maintenance are addressed safely . The team should include doctors, dieticians, specialist nutrition support nurses and pharmacists and may also include other allied healthcare professionals suc h as speech and language therapists. Specialist nutrition support nurses, in conjunction with ward nurses and dieticians, should aim to minimise complications related to enteral or parenteral feeding, develop and implement protocols for training of ward nurses in administration of enteral and parenteral nutrition, around the time of discharge from hospital. NUTRITION SUPPORT TEAMS

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TABLE 25.4 Recommended schedule for monitoring feeding regimens. a Daily Observations including: pulse, blood pressure and temperature body weight /f_l uid balance, including volume of urine and/or urine and intestinal losses quantity and type of food consumed, if allowed to eat Plasma levels: sodium, potassium, urea and creatinine blood glucose magnesium and phosphate (if at risk of refeeding syndrome) liver function tests C-reactive protein Weekly to Plasma levels: fortnightly full blood count calcium, zinc, copper plasma proteins including albumin thiamine triglycerides vitamin B12 folic acid 3–6 monthly Ferritin Selenium, manganese 25-hydroxyvitamin D a Could be converted to weekly once the patient is established on a stable feeding regimen.

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PHYSIOLOGICAL RESPONSES TO NUTRITIONAL IMPAIRMENT

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The constant need for glucose by metabolically active tissues in the body , such as the brain, red and white blood cells and the renal medulla, necessitates homeostasis of blood glucose levels even during periods of fasting. During short-term fasting periods, when insulin levels fall and glucagon levels rise, glycogenolysis is the main source of glucose, whereby glycogen stores from the liver and skeletal muscle are converted to glucose via lactate (the Cori cycle). After approximately 24–40 hours of fasting, glycogen reserves are depleted and gluconeogenesis (the de novo synthesis of glucose from non-carbohydrate precursors such as the amino acids glutamine and alanine, as well as fructose, glucose production. The generation of amino acids occurs from catabolism of skeletal muscle, in amounts of up to 75 /uni00A0 g per day for the average-sized individual. Under conditions of even more prolonged fasting (>48 hours), glucose production is met by the breakdown of fat stores (lipolysis); this provides glycerol, which is then converted to fa tty acids and glucose. Fatty acids can be converted to ketones, which can be used as a metabolic substrate by the majority of tissues in circumstances of extended fasting, reducing the need for muscle breakdown. Resting energy expenditure levels significantly decrease in starvation, related in part to reduced conversion of inactive thyroxine (T) to active tri-iodothyronine (T). Nevertheless, 4 3 this reduction is insu ffi cient to obviate the need for metabolic substrates, leaving a glucose requirement of approximately 200 /uni00A0 g per day even during conditions of prolonged fasting. Summary box 25.1 Metabolic response to starvation /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni00A0 /uni00A0

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Parenteral nutrition

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Indications and composition of parenteral nutrition Nutrition may need to be delivered intravenously in patients in whom adequate feeding through the alimentary tract is not possible. This can be either in addition to enteral feeding (supplemental parenteral nutrition) or the sole source of nutri tion (TPN). TPN is indicated in patients who are unable to meet their nutritional requirements via absorption of nutrients from their intestinal tract. The commonest cause for this is in patients with short bowel syndrome related to massive intes tinal r esection or a significant reduction in functional small bowel, often related to intestinal fistulation. In some cases the establishment of TPN is a temporary endeavour for a few days to minimise n utritional depletion until a route of enteral nutrition is established, e.g. awaiting the siting of a nasojejunal tube in patients with delayed gastric emptying. Parenteral nutrition formulations have evolved over the years, but are currently commonly provided by the hospital pharmacy in the form of a 3-litre bag containing a lipid emul sion with a mixture of essential and non-essential amino acids, glucose, electrolytes, trace elements and vitamins. The energy content of parenteral nutrition is in the ratio of 150–250 /uni00A0 kcal Complications of enteral feeding /uni25CF /uni25CF - /uni25CF /uni25CF - /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF . The /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF - per gram of protein nitrogen, with usually 30–50% of the - energy coming from fat. This ensures su ffi cient energy pro - vision for amino acids to be utilised for tissue maintenance. Folic acid is supplemented in the solution once or twice a week at a dose of 15 /uni00A0 mg and other vitamins are given daily . Patients requiring long-term parenteral nutrition (over many months) w ould also benefit from a single-dose injection of vita - - min B12. Phosphate is an essential component of parenteral - nutrition regimens: 20–30 /uni00A0 mmol phosphate is required daily to ensure phosphorylation of glucose and pre vent hypophos - phataemia. The specific composition of parenteral nutrition can be changed daily to reflect the patient’s needs and tailored to address any electrolyte deficiencies and ongoing energy requirements. This is guided by daily assessments (including weight and electrolytes). In addition, the protein content will di ff er in pa tients who are critically ill (requiring more protein) compared with those with chronic renal failure (requiring less protein). Micronutrients such as zinc, copper, selenium, fer - ritin, folate and vitamins B12 and D will need to be checked in patients on parenteral nutrition for more than 28 days and every 3 months in patients on long-term parenteral nutrition. - Administration of parenteral nutrition Parenteral nutrition is usually administered directly into the central venous system (the superior vena cava [SVC] or the - right atrium) to minimise the risk of venous thrombophlebitis, through either a peripherally inserted central catheter (PICC) or central venous catheter (Figure 25.8). PICC lines may be inserted through the basilic (most commonly used), cephalic, brachial or median cubital vein of the arm, and can be used for parenteral nutrition administration over several weeks to months. Femoral lines should be avoided for parenteral nutri - tion because of the high risk of infection at this site. Chest radiographs should be performed after PICC or central venous - catheters are inserted to confirm the correct position of the line tip within the SVC or right atrium prior to commencing the parenteral feed (Figure 25.9).

Tube related Malposition Displacement Blockage Breakage/leakage Local complications (e.g. erosion of skin/mucosa) Gastrointestinal Diarrhoea Bloating, nausea, vomiting Abdominal cramps Aspiration Constipation Metabolic/biochemical Electrolyte disorders, including refeeding syndrome Vitamin, mineral, trace element de /f_i ciencies Drug interactions

In patients who are likely to require long-term parenteral nutrition, an implantable port or a Hickman line may be more appropriate. These are implanted via fluoroscopic or ultra sound guidance with a subcutaneous port or cu ff and a cathe ter attachment sitting within the SVC. Rarely , parenteral nutrition can be administered through a peripheral venous catheter; however, the high osmolarity of parenteral nutrition produces an increased rate of thrombo phlebitis at these sites because of the narrower calibre and low rate of flow in peripheral veins, making it an appropriate option only wher e the duration of parenteral nutrition admin istration is less than 14 days. The risk of thrombophlebitis can be reduced to some extent by the use of soft polyurethane pae diatric cannulae and using feeds of low osmolarity and neutral pH. The cannulae used will need to be changed every few days. T he parenteral nutrition bag should be covered at all times, including during infusion, with an opaque protective bag to pr event the vitamins from degradation. If the parenteral nutrition infusion is disconnected from the line for any reason during administration the bag will need to be discarded. It is important to remember tha t parenteral nutrition administra tion contributes to fluid intake, and thus the volume infused should be carefully recorded on the fluid balance chart to avoid fluid overload. In patients in whom parenteral nutrition is a temporary measure, oral nutritional input or enteral feeding should be monitored, and par enteral nutrition withdrawal planned in a stepwise manner and stopped once the patient is established on adequate oral or enteral support. Complications of parenteral nutrition The complications of parenteral nutrition are best considered to fall within one of three categories: insertion complica tions, line complications and metabolic complications (see Summary box 25.4). Robert O Hickman , 1926–2019, formerly paediatric nephrologist, Seattle Children’s Hospital, Seattle, WA, USA. - - Summary box 25.4 - Complications of parenteral feeding /uni25CF - /uni25CF /uni25CF - /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF - /uni25CF Insertion complications The most common complication of line insertion is an inad - vertent pneumothorax, which occurs in around 0.5–1% of cases, most commonly during insertion of subclavian lines. It is managed by insertion of a chest drain, which can be removed once the pneumothorax has resolv ed. Line misplacement can also occur and is diagnosed on chest radiograph, which is - mandatory following central line insertion. The line is consid - ered to be in the correct place if the tip is in the inferior third of the SVC or at the atriocaval junction (see Figure 25.9).

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One of the most important line complications is line sepsis, which can occur in up to 15% of patients and is associated with significant morbidity and mortality . Insertion of the line and administration of parenteral nutrition requires strict aseptic technique as line infections can rapidly progress to septicae mia. Catheter entry sites should be checked daily . Patients with suspected line sepsis will need paired blood cultures taken from the line and a separate peripheral site and use of the line should be stopped until culture results are available. Positive cultures will require line removal and commencement of antibiotics. Fungal line infections in particular can be associated with uveitis and bacterial endocarditis. Line thrombosis is not uncommon and can occasionally occur in major veins in association with line infection, causing serious complications such as SVC occlusion and pulmonary embolism. Treatment is by anticoagulation, rarely requiring fibrinolysis for acute SVC occlusion and endovascular inter vention in the longer ter m. Line blockage is relatively common and can be prevented by regular line flushing after manipula tion and the use of a dedicated parenteral nutrition line or, in the case of a multilumen centrally placed catheter, a dedicated lumen. Blocked lines can be unclogged by locking the a ff ected line with heparin–saline or thrombolytic agents. Metabolic complications Refeeding syndrome One of the most significant meta bolic complications of both parenteral and enteral feeding is refeeding syndrome. This occurs in the first days after feeding is commenced in patients who have been severely malnourished. Patients due to start nutritional support need to be screened f the risk of refeeding syndrome. The degree of risk is related to their BMI, amount and rate of unintentional weight loss, period of starvation and electrolyte levels (see Summary box 25.5 The main underlying pathological process is one of hypophosphataemia, resulting in fluid and electrolyte shifts between the intra- and extracellular compartments. Patients Summary box 25.5 Refeeding syndrome /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF cardiac failure, oedema, lethargy or seizures; at its most severe the syndrome can be fatal. Laboratory tests will reveal low levels of phosphate, potassium, calcium and magnesium and a lactic acidosis. Nutritional support in this group of patients should be started at a maximum of 10 /uni00A0 kcal/kg per day , aiming to - increase levels slowly to meet full needs by 4–7 days. Frequent monitoring and replacement of the electrolytes listed above is essential. Nutritional support should include supplementary thiamine, vitamin B, multivitamins and trace elements. Blood sugar derangement In patients with diabetes and those with impaired blood glucose control owing to critical illness, administration of parenteral nutrition should coincide with a variable insulin infusion regimen to avoid hyperglycaemia. Conversely , insulin dosing should be reduced accordingly when parenteral nutrition is interrupted to avoid hypoglycaemia. - Liver dysfunction Long-term use of parenteral nutrition - is associated with derangement of liver function tests in at least 25% of patients. Fatty liver is a common complication. This is worse in children, and the degree can be reduced by modifying the parenteral nutrition solution, such as alternating the use of lipid-free parenteral nutrition solutions. A smaller percentage of patients may subsequently develop liver fibrosis and cirrho - sis. Once liver disease is established in these patients the term - ‘intestinal failure-associated liver disease’ (IFALD) is used, as these cholestatic changes in liver function profile are di ffi cult to separate from the e ff ects of short bowel syndrome. Factors such as a lack of colonic continuity , extreme short bowel, lack or of enteral intake and high energy and fat content in feed have all been associated with a higher risk of the development of IFALD.). Metabolic bone disease and vitamin deficiencies Osteoporosis or osteomalacia are both known complications of long-term parenteral nutrition, leading to fractures or kidney stones. Supplementation of calcium, phosphate, vitamin D and sometimes bisphosphonates can both prevent and treat this complication. Excess or deficiency of vitamins or trace elements may occur, manifesting with non-specific symptoms such as anaemia, hair loss or neurological symptoms. Regular measurements and replacement, as well as clinical assessment, can prevent this from occurring.

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NJ tube passing below diaphragm Figure 25.9 A chest radiograph showing correct positioning of the internal jugular and peripherally inserted central catheter lines within the superior vena cava, as well as the tip of the nasogastric (NG) tube in the stomach and the nasojejunal (NJ) tube passing into the abdomen. Insertion complications Pneumothorax Misplacement Line complications Sepsis Thrombosis Metabolic complications Electrolyte disorders, including refeeding syndrome Blood sugar derangement Liver dysfunction Metabolic bone disease Vitamin de /f_i ciencies

One of the most important line complications is line sepsis, which can occur in up to 15% of patients and is associated with significant morbidity and mortality . Insertion of the line and administration of parenteral nutrition requires strict aseptic technique as line infections can rapidly progress to septicae mia. Catheter entry sites should be checked daily . Patients with suspected line sepsis will need paired blood cultures taken from the line and a separate peripheral site and use of the line should be stopped until culture results are available. Positive cultures will require line removal and commencement of antibiotics. Fungal line infections in particular can be associated with uveitis and bacterial endocarditis. Line thrombosis is not uncommon and can occasionally occur in major veins in association with line infection, causing serious complications such as SVC occlusion and pulmonary embolism. Treatment is by anticoagulation, rarely requiring fibrinolysis for acute SVC occlusion and endovascular inter vention in the longer ter m. Line blockage is relatively common and can be prevented by regular line flushing after manipula tion and the use of a dedicated parenteral nutrition line or, in the case of a multilumen centrally placed catheter, a dedicated lumen. Blocked lines can be unclogged by locking the a ff ected line with heparin–saline or thrombolytic agents. Metabolic complications Refeeding syndrome One of the most significant meta bolic complications of both parenteral and enteral feeding is refeeding syndrome. This occurs in the first days after feeding is commenced in patients who have been severely malnourished. Patients due to start nutritional support need to be screened f the risk of refeeding syndrome. The degree of risk is related to their BMI, amount and rate of unintentional weight loss, period of starvation and electrolyte levels (see Summary box 25.5 The main underlying pathological process is one of hypophosphataemia, resulting in fluid and electrolyte shifts between the intra- and extracellular compartments. Patients Summary box 25.5 Refeeding syndrome /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF cardiac failure, oedema, lethargy or seizures; at its most severe the syndrome can be fatal. Laboratory tests will reveal low levels of phosphate, potassium, calcium and magnesium and a lactic acidosis. Nutritional support in this group of patients should be started at a maximum of 10 /uni00A0 kcal/kg per day , aiming to - increase levels slowly to meet full needs by 4–7 days. Frequent monitoring and replacement of the electrolytes listed above is essential. Nutritional support should include supplementary thiamine, vitamin B, multivitamins and trace elements. Blood sugar derangement In patients with diabetes and those with impaired blood glucose control owing to critical illness, administration of parenteral nutrition should coincide with a variable insulin infusion regimen to avoid hyperglycaemia. Conversely , insulin dosing should be reduced accordingly when parenteral nutrition is interrupted to avoid hypoglycaemia. - Liver dysfunction Long-term use of parenteral nutrition - is associated with derangement of liver function tests in at least 25% of patients. Fatty liver is a common complication. This is worse in children, and the degree can be reduced by modifying the parenteral nutrition solution, such as alternating the use of lipid-free parenteral nutrition solutions. A smaller percentage of patients may subsequently develop liver fibrosis and cirrho - sis. Once liver disease is established in these patients the term - ‘intestinal failure-associated liver disease’ (IFALD) is used, as these cholestatic changes in liver function profile are di ffi cult to separate from the e ff ects of short bowel syndrome. Factors such as a lack of colonic continuity , extreme short bowel, lack or of enteral intake and high energy and fat content in feed have all been associated with a higher risk of the development of IFALD.). Metabolic bone disease and vitamin deficiencies Osteoporosis or osteomalacia are both known complications of long-term parenteral nutrition, leading to fractures or kidney stones. Supplementation of calcium, phosphate, vitamin D and sometimes bisphosphonates can both prevent and treat this complication. Excess or deficiency of vitamins or trace elements may occur, manifesting with non-specific symptoms such as anaemia, hair loss or neurological symptoms. Regular measurements and replacement, as well as clinical assessment, can prevent this from occurring.

. Patient is considered to be at risk of developing refeeding syndrome with EITHER One or more of the following: 2 BMI <16 /uni00A0 kg/m Unintentional weight loss >15% within the last 3–6 months Little or no nutritional intake for more than 10 days Low potassium, phosphate or magnesium levels prior to feeding OR Two of more of the following: 2 BMI <18.5 /uni00A0 kg/m Unintentional weight loss >10% within the last 3–6 months Little or no nutritional intake for more than 5 days History of alcohol abuse or on medication, including insulin, chemotherapy, antacids or diuretics . . .

SUMMARY

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Appropriate and safe assessment and administration of ï¬‚uid therapy and nutritional support is of key importance in good surgical practice. It is imperative that the preoperative nutritional state of the patient and the impact of any surgical intervention are taken into account when considering nutritional requirements and the mode of nutrient delivery . Appreciation and avoidance of complications of both enteral and parenteral nutrition such as refeeding syndrome are also ensuring the safe administration and weaning of supplemental nutrition in a challenging area of practice. SUMMARY

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The effect of the metabolic response to surgery on nutrition

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The metabolic response to surgery is a ff ected not only by the induced fasting period but also by the phenomenon of insulin resistance, which has been described in surgery and in other similar stresses, including trauma and burn injuries. Metabolic response to trauma and sepsis /uni25CF /uni25CF /uni00A0 /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF These stresses elicit combined hormonal and inflammatory responses to the triggers of pain, immobility , acidosis, tissue damage, hypoxia and impairment of homeostasis. Insulin resistance causes hyperglycaemia as a result of increased gluconeogenesis and reduced peripheral glycolysis. This is further worsened by reduced transport of glucose into muscle cells (the main tissue for uptake of insulin-mediated glucose) owing to reduced activation of the glucose transporter protein GLUT4. Instead, muscle protein is broken down to produce amino acids as substrates for gluconeogenesis, inducing a catabolic state with loss of lean muscle mass. The lack of response to insulin means that the catabolic processes induced by fasting or starvation are not resolved with the provision of glucose, and the inappropriate handling of peripheral glucose and breakdown of lean muscle continues for as long as the triggers for insulin resistance persist. Pre-existing comorbidities such as metabolic syndrome, diabetes, cancer and obesity have been shown to contribute to perioperative insulin resistance, which in turn is associated with an increased risk of major complications, in particular severe postoperative infection. Increased awareness of perioperative insulin resistance has led to the incorporation of specific interventions such as preoperative high-carbohydrate drinks to increase insulin sensitivity , adoption of minimally invasive surgery where appropriate (more invasive surgery appears to trigger a greater degree of insulin resistance) and early mobilisation protocols to minimise the impact and duration of insulin resistance on postoperative outcomes and recovery .

Increased counter-regulatory hormones: adrenaline (epinephrine), noradrenaline (norepinephrine), cortisol, glucagon and growth hormone Increased energy requirements (up to 40 kcal/kg per day) Increased nitrogen requirements Insulin resistance and glucose intolerance Preferential oxidation of lipids Increased gluconeogenesis and protein catabolism Loss of adaptive ketogenesis Fluid retention with associated hypoalbuminaemia

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