EFFECTS OF INTESTINAL RESECTION ON FLUID AND NUTRIENT ABSORPTION

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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.

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