Starvation

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Figure 1.7

Factors that exacerbate the metabolic response to surgical injury include hypothermia, uncontrolled pain, starvation, immobilisation, sepsis and medical complications.

Starvation

During starvation, the body is faced with an obligate need to generate glucose to sustain cerebral energy metabolism (100 /uni00A0 g of glucose per day). This is achieved in the first 24 hours by mobilising glycogen stores and thereafter by hepatic glucone - ogenesis from amino acids, glycerol and lactate. The energy metabolism of other tissues is sustained by mobilising fat from adipose tissue. Such fat mobilisation is mainly dependent on a fall in circulating insulin levels. Eventually , accelerated loss of neogenesis) is reduced as a result of the liver converting free fatty acids into ketone bodies, which can serve as a substitute for glucose for cerebral energy metabolism. Provision of 2 litres of intrav enous 4% dextrose/0.18% sodium chloride as maintenance intravenous fluids for surgical patients who are fasted provides 80 /uni00A0 g of glucose per day and has a significant protein-sparing e ff ect. Avoiding unnecessary fasting in the first instance and early oral/enteral/par enteral nutrition form the platform for avoiding loss of body mass as a result of the varying degrees of starvation observed in surgical patients. Modern guidelines on fasting prior to anaesthesia allow intake of clear fluids up to 2 hours before surgery . Administration of a carbohydrate drink at this time reduces perioperative anxiety and thirst and decreases postoperative insulin resistance. Starvation

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