The effect of the metabolic response to surgery on

The effect of the metabolic response to surgery on nutrition

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

The effect of the metabolic response to surgery on nutrition

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

---

Revision #1
Created 2025-12-31 15:11:52 UTC by Omar Ayman
Updated 2025-12-31 15:11:52 UTC by Omar Ayman