METABOLIC CHANGES AFTER SURGERY AND TRAUMA

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The catabolic phase begins at the time of injury and lasts for approximately 24–48 hours. It may be attenuated by proper resuscitation and is characterised by hypovolaemia, decreased basal metabolic rate, reduced cardiac output, hypothermia and lactic acidosis. The predominant hormones regulating the catabolic phase are catecholamines, cortisol and aldosterone (following activation of the renin–angiotensin system). The magnitude of this neuroendocrine response depends on the degree of tissue damage, blood loss and the stimulation of somatic a ff erent nerves at the site of injury . The main physio logical role of the catabolic phase is to conserve both circulating volume and energy stores for later recovery and repair. Following resuscita tion, the catabolic phase evolves into a hypermetabolic flow phase, which corresponds to SIRS. This phase involves the mobilisation of body energy stores for recovery and repair, and the subsequent replacement of lost or damaged tissue. It is characterised by tissue oedema (from vasodilatation and increased capillary leakage), increased basal metabolic rate (hypermetabolism), increased cardiac output, raised body temperature, leukocytosis, increased oxygen con sumption and increased gluconeogenesis. During the catabolic phase, the increased production of counter-regulatory hormones (including catecholamines, IL-6 and TNF α) results in significant fa t and protein mobil - isation, leading to significant weight loss and increased uri - nary nitrogen excretion. During shock, insulin lev els do not rise as expected to combat the hyperglycaemia that occurs in response to stress hormone release and plasma insulin can even fall after sever e injury . Within a few days, insulin production is increased but is associated with significant insulin resistance and, therefore, injured patients often exhibit poor glycaemic control. Importantly , the combination of pronounced or pro - longed catabolism in association with insulin resistance places patients within this phase at increased risk of septic and other complications. Ob viously , the development of complications will further aggravate the neuroendocrine and inflammatory stress responses, thus creating a vicious catabolic cycle and management of blood sugar levels remains an important step. Summary box 1.4 Purpose of neuroendocrine changes following surgery or trauma /uni25CF /uni25CF /uni25CF -

Peripheral tissues Muscle Amino acids Adipose tissue especially Gln

and Ala Skin The constellation of neuroendocrine changes following sur

gery or trauma acts to: Provide essential substrates for survival from tissue breakdown Postpone anabolism Optimise host defence These changes may be helpful in the short term, but may be harmful in the long term, especially to the severely injured or critically ill patient.

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