MANAGING THE CATABOLIC STRESS RESPONSE

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There are several key elements that determine the extent of catabolism and thus govern the metabolic and nutritional care of the surgical patient. It must be remembered that, during the response to injury , not all tissues are catabolic. Indeed, the essence of this coordinated response is to allow the body - to reprioritise limited resources away from peripheral tissues (muscle, adipose tissue, skin) and towards key viscera (liver, immune system) and the wound (Figure 1.3) . However the damage to skeletal muscle can be catastrophic. Figure 1.3

Central tissues Liver During the metabolic response to injury, the body reprioritises protein metabolism away from peripheral tissues and towards key central tissues such as the liver, Immune system immune system and wounds. One of the main reasons why the reutilisation of amino

acids derived from muscle proteol

ysis leads to net catabolism is that the increased glutamine and alanine ef /f_l ux from muscle is derived, in part, from the Wound irreversible degradation of branched chain amino acids. Ala, alanine; Gln, glutamine.

The majority of trauma patients (except possibly those with extensive burns, in whom a greater e ff ect can be seen) demonstrate energy e xpenditures approximately 15–25% above pr edicted healthy resting values. The predominant cause appear s to be a complex interaction between the central control of metabolic rate and peripheral energy utilisation. In particular, central thermodysregula tion (caused by the proinflammator y cytokine cascade), increased sympathetic activity , abnormalities from wound circulation (ischaemic areas produce lactate, whic h must be metabolised by the adenosine triphosphate [ATP]-consuming he patic Cori cycle; hyperaemic areas cause an increase in car diac output), increased protein turnover and nutritional support may all increase patient energy expenditure. Theoretically , patient energy expenditure could rise even higher than observed levels f ollowing surgery or trauma, but several features of standard intensive care (including bed rest, paralysis, ventilation and external temperature regulation) limit the hypermetabolic driving forces of the stress r esponse. Furthermore, the skeletal muscle wasting experienced by patients with prolonged catab olism actually limits the volume of metabolically active tissue (see Alterations in skeletal muscle protein metabolism MANAGING THE CATABOLIC STRESS RESPONSE

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