Nutritional Support in the Perioperative Period

Module 17.5

The Traumatized Patient

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Learning Objectives

- Understand the mechanisms behind the metabolic effects of trauma;
- How does the stress response after traumatic injury lead to hypermetabolism;
- What does this mean for protein metabolism;
- Can certain aspects of the stress response after acute non-surgical trauma be avoided and how can they be treated;
- Insights into the relationship between hypermetabolism, alterations in protein metabolism and complications in surgery for trauma.

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Key Messages

- Trauma leads to overall catabolism in the body;
- Anabolism occurs in splanchnic organs, immune system and in wounds;
- Nutrition should support these processes;
- Specific nutrients like glutamine may be considered.
1. Introduction

This chapter discusses the effects of acute un-anticipated traumatic injury on human metabolism. This is to make a distinction with surgical trauma as well as with the critically ill. In elective surgery, the trauma inflicted by the surgical procedure is foreseeable and therefore, appropriate measures can be taken to counteract any unwanted effects of the host response to trauma. This is not the case in acute traumatic injury, such as a long bone fracture due to a traffic accident (Fig. 1, Fig. 2). It is not intended to give an extensive review of various types of trauma, such as single versus multiple injuries blunt versus penetrating, hypothermia versus burns or fractures versus soft tissue trauma. The scope is to focus on skeletal muscle injury and its effects on metabolism.

2. What happens after trauma?

From observations in traumatized wild animals in their natural surroundings, three distinct features become clear. The animal retreats into a hiding place and exhibits little mobility. A second striking phenomenon is anorexia, not exclusively due to the inability to catch food, but also present when easy access to food is offered. Finally the animal exhibits a catabolic state, reflected in loss of weight, largely consisting of loss of muscle (Fig. 3, Fig. 4).

When the trauma is not too extensive, the traumatized area heals, where after the animal resumes normal activity, starts eating and regains muscle mass and function. In the following, we will explain why these adaptations after trauma are a logic and obligatory event.
The response of the human being to acute trauma is essentially the same as in animals. It differs from surgical trauma in that it is unanticipated and therefore, many of its effects can not be prevented, although we can treat them.

Accidental trauma may range from a simple long bone fracture to any combination of injuries in multiple trauma victims (Fig. 5). As such, symptoms may vary widely and many organs may be affected. The general principle in treating these patients is to protect life and preserve function of vital organs. Once this has been achieved, nutritional support may be started. Obviously, the route of administration may be affected by the specific trauma (1, 2).

3. Clinical symptoms

Symptoms of accidental injury are mainly related to the local effects of trauma, such as a fracture, and the systemic effects of a particular injury (3). Pain and shock are two dominant features that initiate and sustain a chain of adverse events that may be described as the stress response.

Two different phases are distinguished in shock after trauma: an initial hypodynamic phase and a later hyperdynamic phase (4).

The initial hypodynamic phase may be quite pronounced after trauma and leads to hypoperfusion of all organs. Subsequent transmembrane fluid shifts will lead to intracellular dehydration and subsequent acidification, which precludes adequate cell function including regulation of protein turnover (5) (Fig. 6).
Also, hypovolemic shock affects intestinal barrier and this may facilitate translocation of bacteria or their components and a subsequent generalized inflammatory response (6-8) (Fig. 7).

The clinical picture of the stress response, including increased capillary leakage, the development of tissue edema, increased cardiac output and vasodilatation is generally considered a harmful side effect of trauma. It leads to compromised alveolar diffusion of oxygen, to intravascular hypovolemia, renal insufficiency and many other disturbances of organ function (3).

4. Protein kinetics

During acute disease and following trauma, growth is inhibited and the organism becomes catabolic, which is specifically exemplified by muscle breakdown and atrophy (9).

It is now clear that the neuroendocrine response including the cytokine response to disease leads to an obligatory loss of muscle that cannot be blocked by nutrition alone (10).

In fact, the normal response to moderate trauma or disease includes immobility, anorexia, and catabolism (Fig. 8).

Muscle tissue disappears, but at the site of injury or in tissues such as liver and the immune system, there is protein accumulation that is modulated by pro-inflammatory cytokines. Although the individual is anorectic, the anabolic accumulation of tissue in the liver, immune system, and site of injury can only occur with substrate that is derived from other tissues such as muscle. This is an easily detectable situation, and it clearly indicates that muscle catabolism during acute trauma or disease is a useful adaptive phenomenon because the substrate derived from this catabolism is utilized for the healing response. As a consequence, modulation of the "catabolic" hormonal pattern with the intention of blocking muscle catabolism should not block the anabolic actions in the wound and immune system (11).

During pure starvation, the reutilization of amino acids derived from protein degradation is efficient. Essential amino acids are degraded to a very limited degree and are reused for protein synthesis. Whole-body protein turnover decreases, and very little protein is lost at the whole-body level.
Following trauma, amino acids derived from muscle catabolism are not released in the circulation as such, but a large part (especially the branched-chain amino acids (BCAAs)) are irreversibly degraded to yield other amino acids such as glutamine and alanine, which are avidly used at the site of injury and in the liver and immune system (11) (Fig. 9). This precludes efficient reutilization of amino acids and obligatorily leads to increased protein catabolism at the whole-body level. It follows that the substrate mix and specifically the amino acid mix that is utilized during disease is essentially different from the substrate mix that is used during pure starvation. This may have consequences for subsequent treatment.

5. Why is there increased protein degradation during trauma?

In traumatized animals, muscle protein synthesis rates are not decreased, but remain stable or are even slightly increased. In view of the fact that muscle protein loss occurs, this must imply that muscle protein degradation increases to an even greater degree. However, muscle protein turnover contributes only 40% to total body protein turnover and this figure may even be less in the stressed condition. It has already been mentioned that in the stressed condition liver protein synthesis is greatly enhanced. The increase in muscle protein degradation may furnish amino acids from peripheral tissues to central tissues to sustain synthesis of crucial proteins. Visceral protein synthesis is increased in response to trauma and these proteins are functional like e.g. clotting factors, fibrinogen, complement factors and many others.

6. Minimizing unwanted effects of trauma and its treatment: hypothermia

More often than not, a laparotomy is part of the treatment of major trauma patients. The open abdomen is a site of major fluid and heat loss, and this is potentially harmful to the patient. Prevention of (intraoperative) hypothermia reduces the severity of the endocrine-metabolic response and sympathetic reflexes, and changes the fibrinolytic-coagulatory balance resulting in reduced bleeding. Several randomised trials have demonstrated that preservation of normothermia by infusion of fluids, heated to body temperature and using an upper body forced-air heating cover reduces wound infections, cardiac complications and bleeding, and transfusion requirements (12).

7. Proactive approach to prevent unnecessary aspects of the surgical stress response

Increased whole body protein degradation partly reflects the increased degradation of muscle protein, and partly increased turnover of visceral proteins, that play crucial functional roles in the response to trauma and other diseases. Protein synthesis rates should be stimulated by nutritional support to synthesize new muscle protein and to meet the demand of crucial visceral protein and proteins in wounds, white cells and macrophages. So essentially, new therapeutic interventions should be more tailored to organ needs and thereby supply organs with their specific needs.
8. What is the actual substrate mix used by the body after trauma?

One of the major reasons why reutilization of amino acids derived from muscle proteolysis leads to net catabolism at the whole-body level is that the increased glutamine and alanine efflux from muscle following trauma (13) is derived in part from the irreversible degradation of the BCAAs. This precludes reutilization of BCAAs for protein synthesis and leads to a catabolic rate that is more pronounced than during starvation unaccompanied by disease or trauma. The observation that more glutamine (and alanine) is released from peripheral (muscle) tissues (14) implies that more glutamine is consumed in central organs such as liver, immune system, and possibly the site of injury (Fig. 9).

9. What metabolic goals should be achieved in traumatized patients?

As stated, the acutely traumatized organism increases its protein turnover and becomes catabolic. Although plasma concentrations of albumin decrease, fractional synthesis rates of albumin and fibrinogen increase after trauma (11). The accumulation of white cells, macrophages, granulation tissue, collagen, and bone matrix at fracture sites is also proof of increased synthetic processes at the site of injury and in the immune system following trauma. These considerations imply that one of the metabolic goals of therapeutic intervention should be to support increased protein turnover.

Another goal is to exogenously furnish the specific nonessential amino acids that the previously healthy organism produces in excess after trauma and acute illness. Glutamine would seem to be a good candidate for this in the traumatized patient, because it has been shown to reduce complications in trauma victims (15) (Fig. 10).

Early enteral nutrition may be beneficial if tolerated (Fig. 11). Finally, we should preserve the differential changes in protein kinetics as observed in the previously healthy organism subject to trauma or acute disease. Endeavors to inhibit net muscle catabolism in patients with burns by the use of growth hormone have been reasonably successful (16) and therefore, preservation of the central anabolic responses and inhibition of muscle catabolism may be a future treatment option, but only when this does not interfere with the central response.
10. Assessment of efficacy of treatment

Essentially, growth stops when organisms are traumatized (11) and all substrate is directed to the healing response. Although the response to trauma is obligatorily catabolic despite nutritional support, nutrition may limit nitrogen losses by increasing the protein content of the food to 1.5 g of protein/kg/24 h limits nitrogen losses.

Glutamine enrichment may limit nitrogen losses and may improve outcome for trauma patients (15). The increased flux and rate of appearance of glutamine from peripheral to central tissues after trauma is such that supplementing the nutritional regimen with as much as 20 or even 40 g/d can still be considered in the physiological range and safe.

Clinically, it is clear that healthy granulation tissue, solid epithelialization of granulating defects, growth of hair, loss of tissue edema, and regaining of muscle tonicity are all convincing signs of benefit.

References