

Nutritional management of the burn patient

Nutritional balance is disrupted in burns patients as a result of profound alterations in basal metabolic rate and nitrogen excretion.

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Burn injury, the most severe type of injury from a metabolic point of view, is characterised by the most profound alterations in basal metabolic rate and urinary nitrogen excretion.¹ When the burn injury exceeds 15 - 20% total body surface area (TBSA) it results in systemic disturbances including a major stress response, impaired immunity and massive fluid shifts.¹ The severe muscle wasting due to accelerated proteolysis results in muscle weakness that predisposes the patient to pneumonia by limiting his or her ability to cough and clear secretions.

The major improvement in burn survival can be attributed to many factors, one being the development and implementation of improved methods of nutrition support that optimise host defences, enhance wound healing and support the metabolic response to stress.¹

Effect of metabolic response on energy and nutrients

With burns the metabolic rate increases proportionally to the increase in burn size. This relationship is relevant in patients with up to 50 - 60% TBSA, after which there is minimal further increase, although smoke inhalation and other insults such as sepsis increase metabolic rate further.² The catabolism starts within 5 days of injury and may continue for as long as 9 months after injury.³ Free radicals are formed through various processes after burn injury and overpower the inherent free-radical scavenging systems. These oxygen-derived free radicals are associated with local wound response, development of burn shock and distant organ injury.⁴ Patients with major burn injuries develop numerous defects in their immune system that predispose them to infection and multi-organ failure.¹

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After burn injury there is altered glucose oxidation. Burn patients have insulin resistance and an increased glucagon:insulin ratio.⁵ The hyperglycaemia associated with burns is associated with

impaired immune function, poor wound healing and protein catabolism.⁵ The stress hormone and cytokine release result in massive mobilisation of lean body mass and muscle weakness that seems to prolong ventilatory requirement and inhibit sufficient cough reflexes with an increased risk for pulmonary sepsis.⁵

Micronutrients

Marked differences in key micronutrients occur after burn injury as a result of losses through the wound, consumption during metabolism, utilisation for wound healing, inadequate replacement and altered intestinal absorption. Micronutrients play an essential role in antioxidant defences and a deficiency will amplify the already burn-induced metabolic derangements and ongoing catabolism.² The acute phase response is characterised by a decrease in serum iron (Fe), zinc (Zn) and selenium (Se), as well as an increase in serum copper (Cu). This is accompanied by an increase in ferritin and ceruloplasmin.⁶ After burn injury there is the added effect of large trace element losses in the exudates during the first 7 days after injury.⁷ Serum copper decreases contrary to the increase seen with the acute phase response.⁷

All patients with burns exceeding 20% TBSA should receive nutritional support.

Low serum vitamin C, tocopherol, retinol, β -carotene and vitamin A levels have been documented after burn injury.⁸ Vitamin E levels further decline with inhalation injury.⁹ Vitamin E supplementation (100 mg/day) resulted in an increase in serum vitamin E and a significant decrease in lipid peroxides.⁸ Decreased vitamin A levels have been linked to the incidence of diarrhoea in burn patients but it must be remembered that retinol-binding protein is decreased during the acute phase response and supplementation may result in toxicity.^{9,10}

Nutrient requirements

All patients with burns exceeding 20% TBSA should receive nutritional support. Additional factors which may influence the burn patient's requirements:

- age – vulnerable groups are children, the elderly and teenagers
- pregnancy and lactation
- nutritional status prior to the burn
- underlying diseases such as TB, HIV/AIDS, diabetes

- electrolyte abnormalities
- renal failure
- stress diabetes
- fever/infection/sepsis.

Environmental temperature, humidity, pain, and anxiety also need to be effectively controlled to avoid a further increase in metabolic rate.¹¹

The goals of nutrition support include:

- Maintain body mass, particularly lean body mass.
- Prevent starvation and specific nutrient deficiencies.
- Improve wound healing.
- Improve immunity.
- Restore visceral and somatic protein losses.
- Avoid or minimise complications associated with enteral and parenteral nutrition.
- Provide the correct amount and mix of nutrients to limit or modulate the stress response and complications.

It is equally important that a progressive exercise programme is combined with adequate nutrition to enhance restoration of muscle mass and strength.

Energy

The estimation of energy requirements is challenging for the clinician because of hypermetabolism. There are numerous

formulae, but they either over- or underestimate requirements. The most unbiased methods are those of Xie (Table I) and Milner.¹² Indirect calorimetry is more accurate, but expensive, unavailable in most hospitals and needs to be interpreted by a trained technician taking all factors into consideration.¹ Variations of the Harris-Benedict formula (Table II), although not accurate, are often used for practical reasons. Ideal rather than actual body weight must be used in the calculation of formula. Excessive energy supply results in fat mass increase without an increase in muscle mass.

Table I. Formula Xie

Energy expenditure (kcal/d) = (1 000 kcal x BSA [m²]) + (25 x %BSAB)
 BSA: Body surface area
 BSAB: Percentage of total body surface area burn

Contrary to previous beliefs, wound closure does not immediately decrease requirements, and the increased requirements may persist for 9 - 12 months after injury. It is equally important that a progressive exercise programme is combined with adequate nutrition to enhance restoration of muscle mass and strength.¹

Carbohydrates

About 60 - 70% of energy should be administered as carbohydrates. Care must be taken not to exceed the patient's ability to metabolise carbohydrate.¹ It is particularly important not to exceed 5 - 7 mg/kg/min in parenteral nutrition. The metabolic consequences of excessive CHO administration include glucose intolerance, increased carbon dioxide production, increased fat synthesis and the development of a fatty liver. In addition, fat synthesis is an energy-requiring process and overfeeding of glucose is energy inefficient.¹

Protein

Optimal protein administration is essential since improved survival has been found with high-protein diets. The NPE:N ratio is also important. A ratio of 100:1 improved survival over the traditional 150:1-200:1.¹³ There are many formulae for the calculation of protein requirements, but many overestimate requirements: approximately 2 - 2.5 g/kg/day should meet requirements of major burns.

Lipids

Although lipids are essential in the diet of burn patients, metabolism of fats is ineffective. In addition, over-supplementation results in immune suppression and fatty liver. Fat supplies at 15% of total energy reduce infectious morbidity and shorten hospitalisation time when compared with 35% supplied as fat. The correct mix of lipids is essential since ω-3 fatty acids are less immunosuppressive. Care must be taken with parenteral lipids. These must be infused slowly and lipid clearance must be monitored to ensure that triglyceride levels do not rise more than 10 - 20% over baseline values.¹

Immunonutrition

The potential benefits of arginine and glutamine for the burn patient are their specific effects on wound healing and immunity. Glutamine is an important energy source for immune cells and enterocytes. It is also a vital cell-signalling molecule in states of illness and injury. It decreases length of ICU stay and may prevent adult respiratory distress syndrome (ARDS). Supplementation of 0.5 g/kg/day glutamine granules via the enteral route, for 14 days, significantly increases serum glutamine plasma pre-albumin and transferrin levels.

Table II. Estimated nutritional requirements

Extent of burn	Adjustments for burn severity		
	BEE*	Protein	NPE:N ratio
Healthy individual		1.0 g/kg/d	150:1
Moderate burn (15 - 30% TBSA)	× 1.5	1.5 g/kg/d	100 - 120:1
Major burn (30 - 50% TBSA)	× 1.5 - 1.8	1.5 - 2 g/kg/d	100:1
Massive burn (≥50%)	× 1.8 - 2.1	2 - 2.3 g/kg/d	100:1

Activity factors: In bed = 1.2; ambulatory = 1.3; ventilated = 1.05
 Harris Benedict formula:
 * Male: BEE = 66 + (13.7 x W) + (5 x H) - (6.8 x age)
 * Female: BEE = 655 + (9.6 x W) + (1.9 x H) - (4.7 x age)



Table III. Micronutrient requirements

Micronutrient	Data	Recommendations
B vitamins and folic acid	Increased utilisation due to hypermetabolism	2 - 3 × RDA
Vitamin D	Risk for deficiency – limited ultraviolet exposure, cimetidine inhibits hepatic hydroxylation	None
Vitamin E	Documented deficiency Increased requirements with inhalation injury	100 mg – only normalise after 2 weeks
Vitamin A	Documented deficiency Associated with diarrhoea during enteral nutrition	10 000 IU 1 - 10 × RDA is safe
Carotenoids (β-carotene)	Deficient – do not normalise without supplementation	30 mg
Magnesium	Deficiency due to exudates losses and aminoglycosides	According to serum levels
Phosphate	Deficiency due to increased ATP formation and exudates, and urine losses	According to levels
Trace elements	Documented deficiency, losses in exudates Significant reduction in infection, nosocomial pneumonia, ventilator-associated pneumonia	Intravenous: copper 2.5 - 3.1 mg/day, selenium 315 - 380 µg/day, zinc 26.2 - 31.4 mg/day IV for 8 - 21 days No recommendation thereafter, but oral supplementation would probably be appropriate
Vitamin C	Wound healing Reduce resuscitation fluid volume	66 mg/kg/h during resuscitation (24 h) 5 - 10 × RDA thereafter

Micronutrients

The micronutrient requirements of burns patients are listed in Table III.

Evidence-based guidelines for micronutrient supplementation are limited. Iron supplementation is not indicated in the critically ill burn patient. Reduced serum iron is the favourable result of the acute phase response to deter bacterial growth. Supplementation results in an increased risk of infections and increased mortality.¹

Oral magnesium sulphate must be avoided since it causes diarrhoea. Oral magnesium chloride suspension restores serum levels more effectively than slow release tablets. Oral supplementation of a sodium hydrogen phosphate solution effectively restores serum phosphate but potassium phosphate should be avoided. The intravenous route is preferable for trace element supplementation. Intravenous supplementation of copper, zinc and selenium is associated with a significant reduction in infection. High-dose vitamin C administration during the first 24 hours of resuscitation (66 ml/kg/h) significantly reduces resuscitation fluid, improves weight gain and decreases wound oedema.¹⁴

Implementation: route and timing

Enteral nutrition is the administration route of choice and initiation should be immediate or at least within 24 - 48 hours. It is associated with less energy expenditure, reduced bacterial translocation, fewer septic complications, better preservation of immune function and marked increase in survival in comparison with total parenteral

nutrition.^{1,2,5} Parenteral feeding should be reserved for prolonged ileus or as an adjunct to enteral nutrition to meet energy requirements. Overzealous enteral feeding in a patient on inotrope support can, however, result in feeding-induced bowel necrosis in trauma patients due to poor gut perfusion.¹⁵

The nutrient requirements of small burns (<20% TBSA) are easily met with a regular ward diet. It is possible to meet the requirement of patients with large burns, who are conscious and co-operative, with a high-protein diet and oral supplementary drinks.¹¹ The hyperalimentation-per-mouth (HAM) drinks are made out of a combination of commercial and household products and administered at set times throughout the day and night.

With the nutrition care plan consider:

- the volume – keep it as small as possible by using concentrated drinks and by adding protein and carbohydrate modules to food
- the area burnt – with facial burns chewing may cause pain, hand burns may cause difficulty in managing cutlery – consider the consistency of the diet and use cutlery with enlarged grips
- the psychological state of the patients – pain and anxiety may influence appetite and need to be adequately controlled
- religious and cultural influences.

Complications of enteral nutrition

The most common complication with enteral nutrition is significant diarrhoea. Diarrhoea

has been shown to correlate with the use of antibiotics, cimetidine, excessive dietary lipid content (mediated by PGE₁ and PGE₂) and diminished dietary vitamin A content. Other factors that contribute to diarrhoea are hypo-albuminaemia with gut oedema, hyperosmolar tube feeds, histamine H₂-receptor antagonists, antacids (particularly magnesium-containing preparations) due to inactivation of pepsin, bacterial overgrowth (test for *Clostridium difficile*), and food deprivation (functional abnormalities). The incidence of diarrhoea is also lower when enteral nutrition is started within 48 hours after injury and when continuous rather than intermittent feeding is used.

Assess/monitor

Assessing and monitoring the nutritional status and the response to feeding is fraught with error and complicated. Traditional markers of nutritional status, such as albumin and transferrin, are influenced by the metabolic response and therefore not valid in the burn patient. Daily weights in the acute phase are inaccurate because of dressings, fluid shifts, and removal of eschar. The gold standard, nitrogen balance studies, is confounded by large amounts of protein being lost through the wounds.

The best option in the Third-World setting is probably to use pre-albumin, which is less influenced by the acute phase response. If that is unavailable it may be wise to do serial measurements of traditional methods and to plot trends. The combination of albumin, C-reactive protein (CRP), clinical assessment, weight and nitrogen balance may provide some indication of response to feeding over time.

Conclusion

Early enteral nutrition should form the basis of the nutritional support and continue for 12 months after recovery. Exercise is essential to improve muscle mass. Monitoring the nutritional status is not easy. Clinical assessment of the burn patient provides indirect and often valuable information regarding the success of feeding and outcome of the patient.

References

1. Deitch EA. Nutritional support of the burn patient. *Critical Care Clinics* 1995; 11(3): 735-750.
2. Demling RH, Seigne P. Metabolic management of patients with severe burns. *World J Surg* 2000; 24: 673-680.
3. Jeschke MG, Barrow RE, Herndon DN. Extended hypermetabolic response of the liver in severely burned pediatric patients. *Arch Surg* 2004; 139: 641-647.
4. Latha B, Babu M. The involvement of free radicals in burn injury: a review. *Burns* 2001; 27: 309-317.
5. Pereira CT, Murphy K, Jeschke M, Herndon DN. Post burn muscle wasting and the effects of treatment. *Int J Biochem Cell Biol* 2005a; 37: 1948-1961.
6. Kushner I. The phenomenon of the acute phase response. *Ann NY Acad Sci* 1982; 389: 39.
7. Berger, MM, Cavadini C, Bart A, et al. Cutaneous zinc and copper losses in burns. *Burns* 1992a; 18: 373.
8. Mingjian Z, Qifang W, Lanxing G, Hong J, Zongyin W. Comparative observation of the changes in serum lipid peroxides influenced by the supplementation of vitamin E in burn patients and healthy controls. *Burns* 1992; 18(1): 19 - 21.
9. Gottschlich MM, Warden GD. Vitamin supplementation in the patient with burns. *J Burn Care Rehabil* 1990; 11(3): 275-278.
10. Gottschlich MM, Warden GD, Michel M, et al. Diarrhea in tube-fed burn patients: incidence, etiology, nutritional impact and prevention. *JPEN* 1988; 12: 338-345.
11. Prins A. Nutrition support in thermal injury: The ultimate challenge. *Health & Hygiene* 1997; 8(1): 2-3.
12. Xie WG, Wang, SL. Estimation of the caloric requirements of burned Chinese adults. *Burns* 1993; 19(2): 146-149.
13. Alexander WJ, MacMillan BG, Stinett JD, et al. Beneficial effects of aggressive feeding in severely burned children. *Ann Surg* 1980; 192: 505.
14. Tanaka H, Matsuda T, Miyagantani Y, Yukioka T, Matsuda M, Shimazaki S. Reduction of resuscitation fluid volumes in severely burned patients using ascorbic acid administration. *Arch Surg* 2000; 135: 326-331.
15. Marvin R, McKinley B, McGuigan M. Non-occlusive bowel necrosis occurring in critically ill trauma patients receiving enteral nutrition manifests no reliable clinical signs for early detection. *Am J Surg* 2000; 179: 7-12.

In a nutshell

- Enteral nutrition is the feeding method of choice.
- Start within 24 - 48 hours.
- Energy requirements are increased, but overfeeding is detrimental.
- Protein requirements: 1.5 - 2.5 g/kg.
- Nitrogen:non-protein energy – 100:1.
- Arginine is potentially indicated during specific periods.
- Glutamine supplementation is essential.
- Micronutrient requirements are increased – use the parenteral route for trace elements.
- High-dose vitamin C during resuscitation may reduce resuscitation fluid requirements.
- Monitor.
- Progressive exercise programme.

Single suture

No smoking here too

While the West is winning the war against tobacco, the death toll still rises in developing nations as more people take up the habit. An estimated 10 million people will die of smoking-related diseases by the year 2025, according to the WHO.

Now Bill Gates and Michael Bloomberg, the multimillionaire mayor of New York City, have pledged \$375 million towards anti-smoking programmes in developing countries. Bloomberg established a \$125 million initiative, Reduce Tobacco Use, in 2005. This new programme will include funding public information campaigns designed to take on the advertising power of major tobacco companies – who often use forms of advertising banned in the West.

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