Nutrition and Metabolism in Acute Renal Failure

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Adequate nutritional support is necessary to maintain protein stores and to correct pre-existing or disease-related deficits in lean body mass. The objectives for nutritional support for patients with acute renal failure (ARF) are not much different from those with other catabolic conditions. The principles of nutritional support for ARF, however, differ from those for patients with chronic renal failure (CRF), because diets or infusions that satisfy minimal requirements in CRF are not necessarily sufficient for patients with ARF.

In patients with ARF modern nutritional therapy must include a tailored regimen designed to provide substrate requirements with various degrees of stress and hypercatabolism. If nutrition is provided to a patient with ARF the composition of the dietary program must be specifically designed because there are complex metabolic abnormalities that affect not only water, electrolyte, and acid-base-balance but also carbohydrate, lipid, and protein and amino acid utilization.

In patients with ARF the main determinants of nutrient requirements (and outcome) are not renal dysfunction per se but the degree of hypercatabolism caused by the disease associated with ARF, the nutritional state, and the type and frequency of dialysis therapy. Pre-existing or hospital-acquired malnutrition has been identified as an important contributor to the persisting high mortality in critically ill persons.

Thus, with modern nutritional support requirements must be met for all nutrients necessary for preservation of lean body mass, immunocompetence, and wound healing for a patient who has acquired ARF—in may instances among other complications. At the same time the specific metabolic alterations and demands in ARF and the impaired excretory renal function must be respected to limit uremic toxicity.

In this chapter the multiple metabolic alterations associated with ARF are reviewed, methods for estimating nutrient requirements are discussed and, current concepts for the type and composition of nutritional programs are summarized. This information is relevant for designing nutritional support in an individual patient with ARF.

CHAPTER

18

NUTRITION IN ACUTE RENAL FAILURE

Goals
- Preservation of lean body mass
- Stimulation of wound healing and reparatory functions
- Stimulation of immunocompetence
- Acceleration of renal recovery (?)

But not (in contrast to stable CRF)
- Minimization of uremic toxicity (perform hemodialysis and CRRT as required)
- Retardation of progression of renal failure

Thus, provision of optimal but not minimal amounts of substrates

FIGURE 18-1

Nutritional goals in patients with acute renal failure (ARF). The goals of nutritional intervention in ARF differ from those in patients with chronic renal failure (CRF): One should not provide a minimal intake of nutrients (to minimize uremic toxicity or to retard progression of renal failure, as recommended for CRF) but rather an optimal amount of nutrients should be provided for correction and prevention of nutrient deficiencies and for stimulation of immunocompetence and wound healing in the mostly hypercatabolic patients with ARF [1].

METABOLIC PERTURBATIONS IN ACUTE RENAL FAILURE

Determined by	Plus
Renal dysfunction (acute uremic state)	Specific effects of renal replacement therapy
Underlying illness	Nonspecific effects of extracorporeal circulation (bioincompatibility)
The acute disease state, such as systemic inflammatory response syndrome (SIRS)	
Associated complications (such as infections)	

FIGURE 18-2

Metabolic perturbations in acute renal failure (ARF). In most instances ARF is a complication of sepsis, trauma, or multiple organ failure, so it is difficult to ascribe specific metabolic alterations to ARF. Metabolic derangements will be determined by the acute uremic state plus the underlying disease process or by complications such as severe infections and organ dysfunctions and, last but not least by the type and frequency of renal replacement therapy [1, 2].

Nevertheless, ARF does not affect only water, electrolyte, and acid base metabolism: it induces a global change of the metabolic environment with specific alterations in protein and amino acid, carbohydrate, and lipid metabolism [2].

Metabolic Alterations in Acute Renal Failure

Energy metabolism

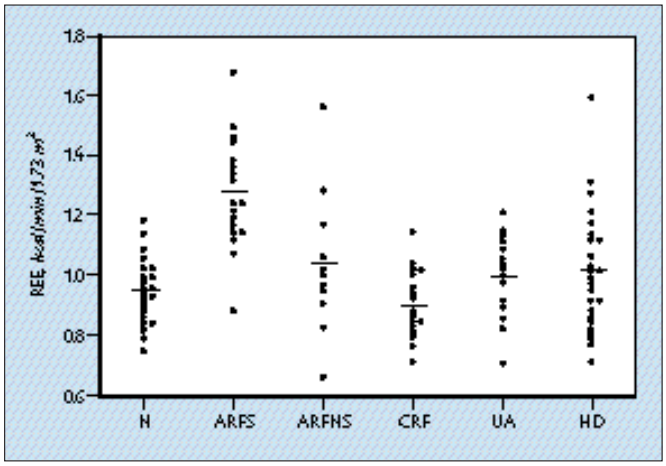

FIGURE 18-3

Energy metabolism in acute renal failure (ARF). In experimental animals ARF decreases oxygen consumption even when hypothermia and acidosis are corrected (uremic hypometabolism) [3]. In contrast, in the clinical setting oxygen consumption of patients with various form of renal failure is remarkably little changed [4]. In subjects with chronic renal failure (CRF), advanced uremia (UA), patients on regular hemodialysis therapy (HD) but also in patients with uncomplicated ARF (ARFNS) resting energy expenditure (REE) was comparable to that seen in controls (N). However, in patients with ARF and sepsis (ARFS) REE is increased by approximately 20%.

Thus, energy expenditure of patients with ARF is more determined by the underlying disease than acute uremic state and taken together these data indicate that when uremia is well-controlled by hemodialysis or hemofiltration there is little if any change in energy metabolism in ARF. In contrast to many other acute disease processes ARF might rather decrease than increase REE because in multiple organ dysfunction syndrome oxygen consumption was significantly higher in patients without impairment of renal function than in those with ARF [5]. (*From* Schneeweiss [4]; with permission.)

ESTIMATION OF ENERGY REQUIREMENTS

Calculation of resting energy expenditure (REE) (Harris Benedict equation):
- Males: 66.47 ÷ (13.75 × BW) ÷ (5 × height) − (6.76 × age)
- Females: 655.1 ÷ (9.56 × BW) ÷ (1.85 × height) − (4.67 × age)
- The average REE is approximately 25 kcal/kg BW/day

Stress factors to correct calculated energy requirement for hypermetabolism:
- Postoperative (no complications) 1.0
- Long bone fracture 1.15–1.30
- Cancer 1.10–1.30
- Peritonitis/sepsis 1.20–1.30
- Severe infection/polytrauma 1.20–1.40
- Burns (= approxim. REE + % burned body surface area) 1.20–2.00
- Corrected energy requirements (kcal/d) = REE × stress factor

FIGURE 18-4

Estimation of energy requirements. Energy requirements of patients with acute renal failure (ARF) have been grossly overestimated in the past and energy intakes of more than 50 kcal/kg of body weight (BW) per day (*ie*, about 100% above resting energy expenditure (REE) haven been advocated [6]. Adverse effects of overfeeding have been extensively documented during the last decades, and it should be noted that energy intake must not exceed the actual energy consumption. Energy requirements can be calculated with sufficient accuracy by standard formulas such as the Harris Benedict equation. Calculated REE should be multiplied with a stress factor to correct for hypermetabolic disease; however, even in hypercatabolic conditions such as sepsis or multiple organ dysfunction syndrome, energy requirements rarely exceed 1.3 times calculated REE [1].

Protein metabolism

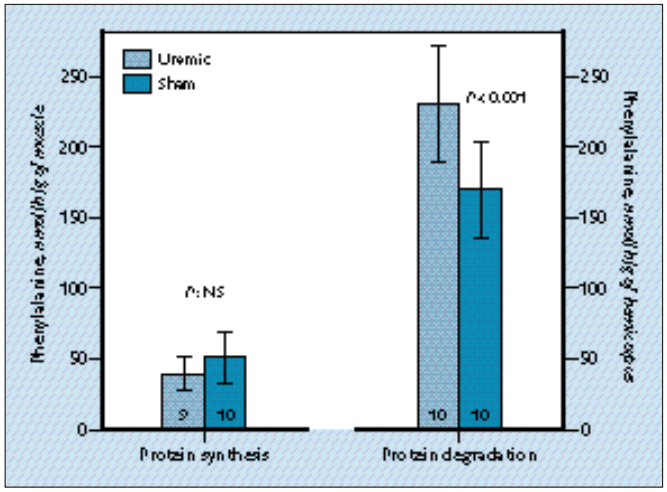

FIGURE 18-5

Protein metabolism in acute renal failure (ARF): activation of protein catabolism. Protein synthesis and degradation rates in acutely uremic and sham-operated rats. The hallmark of metabolic alterations in ARF is activation of protein catabolism with excessive release of amino acids from skeletal muscle and sustained negative nitrogen balance [7, 8]. Not only is protein breakdown accelerated, but there also is defective muscle utilization of amino acids for protein synthesis. In muscle, the maximal rate of insulin-stimulated protein synthesis is depressed by ARF and protein degradation is increased, even in the presence of insulin [9]. (*From* [8]; with permission.)