Chapter 15
Renal Disease

The Principles

The kidney is also a key metabolic organ. Consequently, renal failure affects not only the quantity but also the composition of nutritional requirements. The kidney's most critical functions include the control of fluid and electrolyte balance and the excretion of potentially toxic metabolites. For example, the kidney is responsible for the excretion of water, electrolytes, and nitrogenous breakdown products, including urea, creatinine, and ammonia. As diet and body stores influence the production rate of all these substances, it was not surprising that early management principles advised the restriction of their intake, particularly with regard to protein, which is the precursor for urea and ammonia. However, as with liver failure, the restriction of protein intake was eventually shown to worsen survival as no or low protein diets accelerate the catabolism of body proteins, and patients became progressively malnourished. Infusions of glucose or high-carbohydrate diets were shown to reduce the rate of catabolism as outlined in Chapter 4: "Protein Catabolism" but couldn't prevent the progressive depletion of body protein. Hypothesizing that metabolic balance would best be achieved if only essential amino acids (EAA) were given for the protein component of nutritional support allowing the liver to reincorporate nitrogen oxidative products into the synthesis of nonessential amino acids (NEAA), while simultaneously infusing glucose to prevent protein catabolism, Abel et al performed a prospective double-blind study in patients with acute renal failure (ARF) [269]. Results were encouraging, demonstrating the apparent ability of infusions of EAA and dextrose to improve survival. However, a later study showed that a balanced amino acid mixture was equally effective in maintaining nitrogen balance and suppressing blood urea nitrogen (BUN) concentrations, with no difference in survival [270, 271]. Further concern about the use of EAA was raised by the observation of mental status changes associated with metabolic side effects in children and adults with ARF treated with EAA. Investigation revealed hyperammonemia and hyperchloremic metabolic

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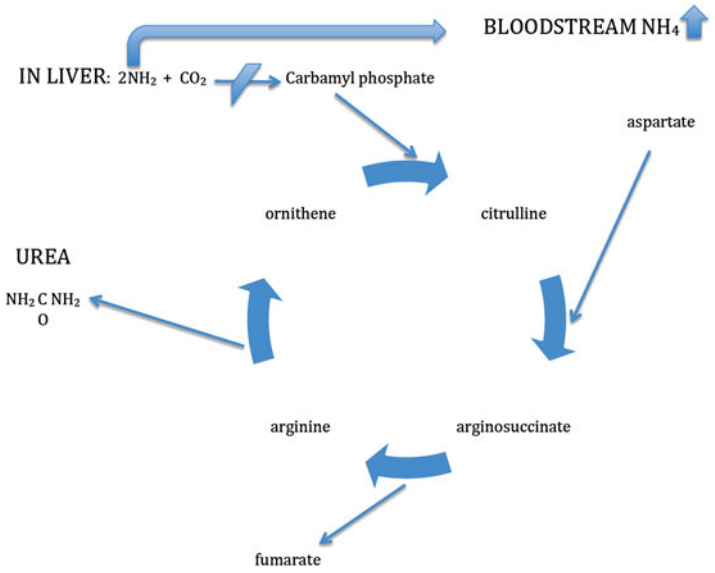

Fig. 15.1 Explanation for the metabolic complications caused by giving essential amino acids alone as a protein source in patients with acute renal failure. Insufficient dietary inessential amino acids result in depletion of citrulline, ornithine, and arginine which are essential for function of the urea cycle. Failure of the urea cycle to incorporate ammonia derived from protein catabolism results in hyperammonemia and mental status changes

acidosis, elevated plasma methionine and ammonia, and depressed plasma citrulline, ornithine, arginine, and histidine levels [272, 273]. The explanation for this appeared to be impairment of the capacity of key urea cycle intermediates (ornithine and citrulline) to accept ammonia from carbamoyl phosphate (Fig. 15.1). In general, controlled clinical trials of the use of specialized nutritional formula for ARF have yielded variable and disappointing results. For example, a recent Cochrane Database meta-analysis examined 8 eligible studies (257 participants) but were unable to perform a pooled analysis because different interventions and different outcomes were employed [274].

The Practice

Appreciation of the results of the above studies has led to the recommendations that, despite the ability of protein restriction to suppress BUN increases, protein restrictions below 0.5 g/kg/day should be avoided. The use of EAA alone should be avoided, but proteins of high biological value (i.e., high EAA:NEAA) should be provided with adequate quantities of energy, chiefly in the form of carbohydrate (4 g/kg/day). As with every other clinical condition, nutritional support should be given by enteral

route. Anorexia and delayed gastric emptying are common in chronic renal failure, and supplemental tube feeding may be necessary. In acute renal failure, protein restriction must be avoided as patients are catabolic: increases in BUN, phosphate, and potassium should be treated with timely dialysis. Once established on dialysis, nutritional requirements return to those used in any form of critical illness, namely, protein (or balanced amino acid formulae in PN-fed patients) infusion rates of 1.5 g/kg ideal body weight/day, energy 25 kcal/kg/day. Additional protein supplements of up to 20 g of amino acids may be necessary to cover amino acid loses during dialysis. "Specialized" formula diets ("Nepro," Abbott Nutrition, Columbus, OH) are designed to counteract the metabolic (high biological value protein, complex carbohydrates), electrolyte (low sodium, potassium, and phosphate), and fluid (concentrated) defects and so have theoretical advantages, but whether these translate into better survival compared to standard formula plus dialysis remains to be shown.