AMINO ACID SOLUTIONS FOR THE TREATMENT OF ACUTE RENAL FAILURE ("NEPHRO" SOLUTIONS)

	Rose-Requirements	RenAmin (Clintec)	Aminess (Clintec)	Aminosyn RF (Abbott)	NephrAmine (McGaw)	Nephrotect (Fresenius)
Amino acids (g/L)		65	52	52	54	100
(= g/%)		6.5	5.2	5.2	5.4	10
Volume (mL)		500	400	1000	1000	500
(mOsm/L)		600	416	475	435	908
Nitrogen (g/L)		10	8.3	8.3	6.5	16.3
Essential amino acids (g/L)						
Isoleucine	1.40	5.00	5.25	4.62	5.60	5.80
Leucine	2.20	6.00	8.25	7.26	8.80	12.80
Lysine acetate/HCl	1.60	4.50	6.00	5.35	6.40	12.00
Methionine	2.20	5.00	8.25	7.26	8.80	2.00
Phenylalanine	2.20	4.90	8.25	7.26	8.80	3.50
Threonine	1.00	3.80	3.75	3.30	4.00	8.20
Tryptophan	0.50	1.60	1.88	1.60	2.00	3.00
Valine	1.60	8.20		5.20	6.40	8.70
Nonessential amino acids (g/L)						
Alanine		5.60				6.20
Arginine		6.30	6.00	6.00		8.20
Glycine		3.00				6.30*
Histidine		4.20	4.12	4.29	2.50	9.80
Proline		3.50				3.00
Serine		3.00				7.60
Tyrosine		0.40				3.00†
Cysteine					0.20	0.40

* Glycine is a componenet of the dipeptide.

† Tyrosine is included as dipeptide (glycyl-L-tyrosine).

FIGURE 18-33

Amino acid (AA) solutions for parenteral nutrition in acute renal failure (ARF). The most controversial choice regards the type of amino acid solution to be used: either essential amino acids (EAAs) exclusively, solutions of EAA plus nonessential amino acids (NEAAs), or specially designed "nephro" solutions of different proportions of EAA and specific NEAA that might become "conditionally essential" for ARF (*see* Fig. 18-11).

Use of solutions of EAA alone is based on principles established for treating chronic renal failure (CRF) with a low-protein diet and an EAA supplement. This may be inappropriate as the metabolic adaptations to low-protein diets in response to CRF may not have occurred in patients with ARF. Plus, there are fundamental differences in the goals of nutritional therapy in the two groups of patients, and consequently, infusion solutions of EAA may be sub-optimal.

Thus, a solution should be chosen that includes both essential and nonessential amino acids (EAA, NEAA) in standard proportions or in special proportions designed to counteract the metabolic changes of renal failure ("nephro" solutions), including the amino acids that might become conditionally essential in ARF.

Because of the relative insolubility of tyrosine in water, dipeptides containing tyrosine (such as glycyl-tyrosine) are contained in modern nephro solutions as the tyrosine source [22, 23]. One should be aware of the fact that the amino acid analogue *N*-acetyl tyrosine, which previously was used frequently as a tyrosine source, cannot be converted into tyrosine in humans and might even stimulate protein catabolism [21].

Despite considerable investigation, there is no persuasive evidence that amino acid solutions enriched in branched-chain amino acids exert a clinically significant anticatabolic effect. Systematic studies using glutamine supplementation for patients with ARF are lacking (*see* Fig. 18-11).

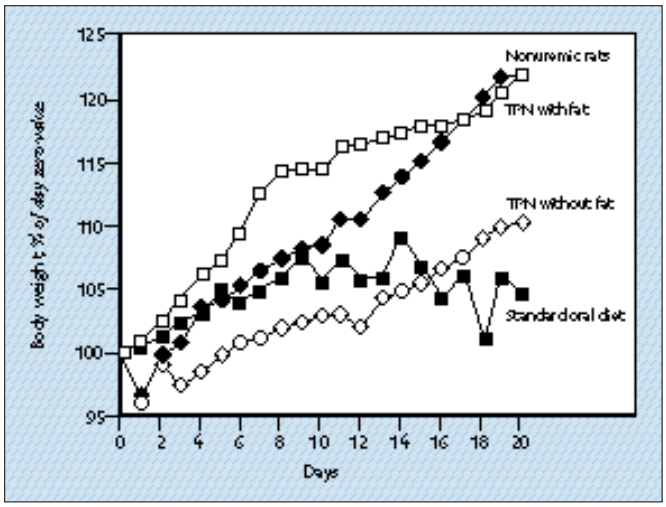

FIGURE 18-34

Energy substrates in total parenteral nutrition (TPN) in acute renal failure (ARF): glucose and lipids. Because of the well-documented effects of overfeeding, energy intake of patients with ARF must not exceed their actual energy expenditure (*ie*, in most cases 100% to 130% of resting energy expenditure [REE]; *see* Figs. 18-3 and 18-4) [2].

Glucose should be the principal energy substrate because it can be utilized by all organs, even under hypoxic conditions, and has the potential for nitrogen sparing. Since ARF impairs glucose tolerance, insulin is frequently necessary to maintain normoglycemia. Any hyperglycemia must be avoided because of the untoward associated side effects—such as aggravation of tissue injury, glycation of proteins, activation of protein catabolism, among others [2]. When intake is increased above 5 g/kg of body weight per day infused glucose will not be oxidized but will promote lipogenesis with fatty infiltration of the liver and excessive carbon dioxide production and hypercarbia. Often, energy requirements cannot be met by glucose infusion without adding large amounts of insulin, so a portion of the energy should be supplied by lipid emulsions [2].

The most suitable means of providing the energy substrates for parenteral nutrition for patients with ARF is not glucose or lipids, but glucose *and* lipids [2]. In experimental uremia in rats, TPN with 30% of nonprotein energy as fat promoted weight gain and ameliorated the uremic state and survival [63]. (*From* Wennberg *et al*. [63]; with permission.)

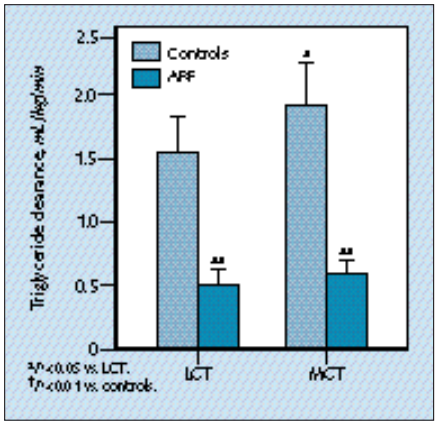

FIGURE 18-35

Energy substrates in parenteral nutrition: lipid emulsions. Advantages of intravenous lipids include high specific energy content, low osmolality, provision of essential fatty acids and phospholipids to prevent deficiency syndromes, fewer hepatic side effects (such as steatosis, hyperbilirubinemia), and reduced carbon dioxide production, especially relevant for patients with respiratory failure.

Changes in lipid metabolism associated with acute renal failure (ARF) should not prevent the use of lipid emulsions. Instead, the amount infused should be adjusted to meet the patient's capacity to utilize lipids. Usually, 1 g/kg of body weight per day of fat will not increase plasma triglycerides substantially, so about 20% to 25% of energy requirements can be met [1]. Lipids should not be administered to patients with hyperlipidemia (*ie*, plasma triglycerides above 350 mg/dL) activated intravascular coagulation, acidosis (pH below 7.25), impaired circulation or hypoxemia.

Parenteral lipid emulsions usually contain long-chain triglycerides (LCT), most derived from soybean oil. Recently, fat emulsions containing a mixture of LCT and medium-chain triglycerides (MCT) have been introduced for intravenous use. Proposed advantages include faster elimination from the plasma owing to higher affinity to the lipoprotein lipase enzyme, complete, rapid, and carnitine-independent metabolism, and a triglyceride-lowering effect; however, use of MCT does not promote lipolysis, and elimination of triglycerides of both types of fat emulsions is equally retarded in ARF [34]. (*Adapted from* [34]; with permission.)

SUGGESTED SCHEDULE FOR MINIMAL MONITORING OF PARENTERAL NUTRITION

	Metabolic Status	
Variables	**Unstable**	**Stable**
Blood glucose	1–6 × daily	Daily
Osmolality	Daily	2× weekly
Electrolytes (Na^+, K^+, Cl^+)	Daily	Daily
Calcium, phosphate, magnesium	Daily	3× weekly
Daily BUN increment	Daily	Daily
Urea nitrogen appearance rate	Daily	2 × weekly
Triglycerides	Daily	2 × weekly
Blood gas analysis, pH	Daily	1× weekly
Ammonia	2 × weekly	1 × weekly
Transaminases + bilirubin	2 × weekly	1 × weekly

FIGURE 18-36
Complications and monitoring of nutritional support in acute renal failure (ARF).

Complications: Technical problems and infectious complications originating from the central venous catheter, chemical incompatibilities, and metabolic complications of parenteral nutrition are similar in ARF patients and in nonuremic subjects. However, tolerance to volume load is limited, electrolyte derangements can develop rapidly, exaggerated protein or amino acid intake stimulates excessive blood urea nitrogen (BUN) and waste product accumulation and glucose intolerance, and decreased fat clearance can cause hyperglycemia and hypertriglyceridemia. Thus, nutritional therapy for ARF patients requires more frequent monitoring than it does for other patient groups, to avoid metabolic complications.

Monitoring: This table summarizes laboratory tests that monitor parenteral nutrition and avoid metabolic complications. The frequency of testing depends on the metabolic stability of the patient. In particular, plasma glucose, potassium, and phosphate should be monitored repeatedly after the start of parenteral nutrition.

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