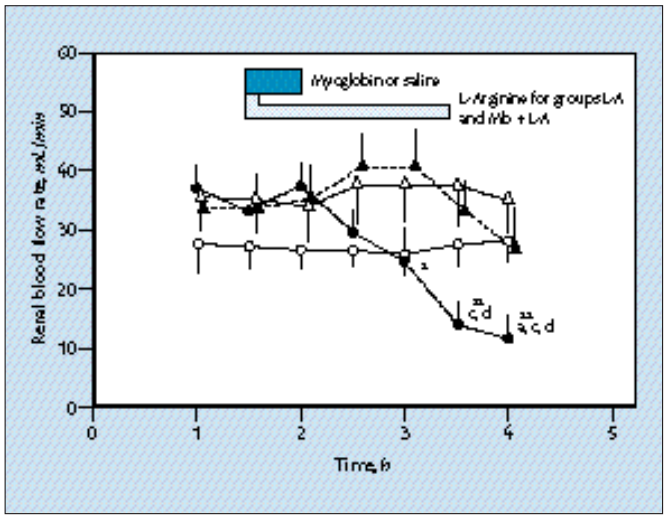
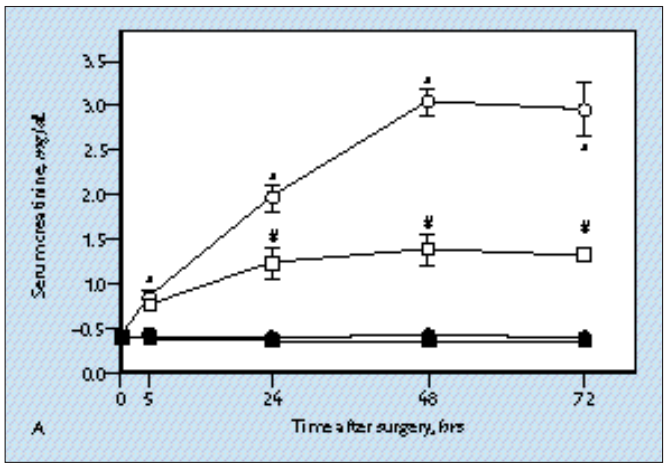


FIGURE 18-26

Impact of nutritional interventions on renal function in acute renal failure (ARF). Amino acid infused before or during ischemia or nephrotoxicity may enhance tubule damage and accelerate loss of renal function in rat models of ARF. In part, this therapeutic paradox [53] from amino acid alimentation in ARF is related to the increase in metabolic work for transport processes when oxygen supply is limited, which may aggravate ischemic injury [54]. Similar observations have been made with excess glucose infusion during renal ischemia. Amino acids may as well exert a protective effect on renal function. Glycine, and to a lesser degree alanine, limit tubular injury in ischemic and nephrotoxic models of ARF [55]. Arginine (possibly by producing nitric oxide) reportedly acts to preserve renal perfusion and tubular function in both nephrotoxic and ischemic models of ARF, whereas inhibitors of nitric oxide synthase exert an opposite effect [56,57]. In myoglobin-induced ARF the drop in renal blood flow (*black circles*, ARF controls) is prevented by L-arginine infusion (*black triangles*) [57]. (*From* Wakabayashi *et al*. [57]; with permission.)



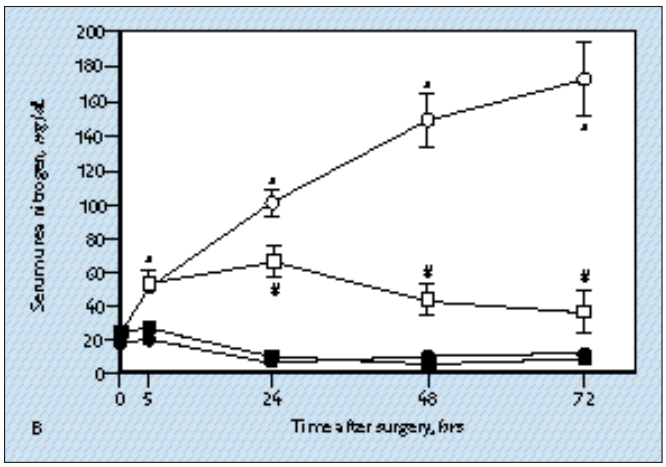


FIGURE 18-27

Impact of endocrine-metabolic interventions on renal function and course of acute renal failure (ARF). Various other endocrine-metabolic interventions (*eg*, thyroxine, human growth hormone [HGH], epidermal growth factor, insulin-like growth factor 1 [IGF-1]) have been shown to accelerate regeneration after experimental ARF [51]. In a rat model of postischemic ARF, treatment with IGF-1 starting 5 hours after induction of ARF accelerates recovery from ischemic ARF, **A**, but also reduces the increase in BUN and improves nitrogen balance, **B**, [58]. (*open circles*) ARF plus vehicle; (*black circles*, sham-operated rats plus vehicle; *open squares*, ARF plus rhIGF-I; *black squares*, sham operated rats plus rhIGF-I.) Unfortunately, efficacy of these interventions was not uniformly confirmed in clinical studies [59, 60]. (*From* Ding *et al*. [58]; with permission.)

Decision Making, Patient Classification, and Nutritional Requirements

DECISIONS FOR NUTRITION IN PATIENTS WITH ACUTE RENAL FAILURE

Decisions dependent on
- Patients ability to resume oral diet (within 5 days?)
- Nutritional status
- Underlying illness/degree of associated hypercatabolism

1. What patient with acute renal failure needs nutritional support?
2. When should nutritional support be initiated?
3. At what degree of impairment in renal function should the nutritional regimen be adapted for renal failure?
4. In a patient with multiple organ dysfunction, which organ determines the type of nutritional support?
5. Is enteral or parenteral nutrition the most appropriate method for providing nutritional support?

FIGURE 18-28
Nutrition in patients with acute renal failure (ARF): decision making. Not every patient with ARF requires nutritional support. It is important to identify those who will benefit and to define the optimal time to initiate therapy [1].

The decision to initiate nutritional support is influenced by the patient's ability to cover nutritional requirements by eating, in addition to the nutritional status of the patient as well as the type of underlying illness involved. In any patient with evidence of malnourishment, nutritional therapy should be instituted regardless of whether the patient will be likely to eat. If a well-nourished patient can resume a normal diet within 5 days, no specific nutritional support is necessary. The degree of accompanying catabolism is also a factor. For patients with underlying diseases associated with excess protein catabolism, nutritional support should be initiated early.

If there is evidence of malnourishment or hypercatabolism, nutritional therapy should be initiated early, even if the patient is likely to eat before 5 days. Modern nutritional strategies should be aimed at avoiding the development of deficiency states and of "hospital-acquired malnutrition." During the acute phase of ARF (the first 24 hours after trauma or surgery) nutritional support should be withheld because nutrients infused during this "ebb phase" are not utilized, could increase oxygen requirements, and aggravate tissue injury and renal dysfunction.

The nutritional regimen should be adapted for renal failure when renal function is impaired. The multiple metabolic alterations characteristic of ARF occur when kidney function is below 30% of normal. Thus, when creatinine clearance falls below 50 to 30 mL per minute/1.73 m^2 (or serum creatinine rises above 2.5 to 3.0 mg/dL) the nutritional regimen should be adapted to ARF. With the exception of severe hepatic failure and massively deranged amino acid metabolism (hyperammonemia) or protein synthesis (depletion of coagulation factors) renal failure is the major determinant of the nutritional regimen in patients with multiple organ dysfunction.

Enteral feeding is preferred for all patients, including those with ARF. Nevertheless, for a large portion of patients, parenteral nutrition—total or partial—will be necessary to meet nutritional requirements.

PATIENT CLASSIFICATION AND SUBSTRATE REQUIREMENTS IN PATIENTS WITH ACUTE RENAL FAILURE

	Extent of Catabolism		
	Mild	**Moderate**	**Severe**
Excess urea appearance (above nitrogen intake)	>6 g	6–12 g	>12 g
Clinical setting (examples)	Drug toxicity	Elective surgery ± infection	Severe injury or sepsis
Mortality	20 %	60%	>80%
Dialysis or hemofiltration frequency	Rare	As needed	Frequent
Route of nutrient administration	Oral	Enteral or parenteral	Enteral or parenteral
Energy recommendations (kcal/kg BW/d)	25	25–30	25–35
Energy substrates	Glucose	Glucose + fat	Glucose + fat
Glucose (g/kg BW/d)	3.0–5.0	3.0–5.0	3.0–5.0 (max. 7.0)
Fat (g/kg BW/d)		0.5–1.0	0.8–1.5
Amino acids/protein (g/kg/d)	0.6–1.0	0.8–1.2	1.0–1.5
	EAA (+NEAA)	EAA + NEAA	EAA + NEAA
Nutrients used	Foods	Enteral formulas	Enteral formulas
		Glucose 50%–70% + fat emulsions 10% or 20%	Glucose 50%–70% + fat emulsions 10% or 20%
	EAA + specific NEAA solutions (general or "nephro")		
	Multivitamin and multitrace element preparations		

BW—body weight; EAA—essential amino acids; NEAA—nonessential amino acids.

FIGURE 18-29

Patient classification: substrate requirements. Ideally, a nutritional program should be designed for each individual acute renal failure (ARF) patient. In clinical practice, it has proved useful to distinguish three groups of patients based on the extent of protein catabolism associated with the underlying disease and resulting levels of dietary requirements.

Group I includes patients without excess catabolism and a UNA of less than 6 g of nitrogen above nitrogen intake per day. ARF is usually caused by nephrotoxins (aminoglycosides, contrast media, mismatched blood transfusion). In most cases, these patients are fed orally and the prognosis for recovery of renal function and survival is excellent.

Group II consists of patients with moderate hypercatabolism and a UNA exceeding nitrogen intake 6 to 12 g of nitrogen per day. Affected patients frequently suffer from complicating infections, peritonitis, or moderate injury in association with ARF. Tube feeding or intravenous nutritional support is generally required, and dialysis or hemofiltration often becomes necessary to limit waste product accumulation.

Group III are patients who develop ARF in association with severe trauma, burns, or overwhelming infection. UNA is markedly elevated (more than 12 g of nitrogen above nitrogen intake). Treatment strategies are usually complex and include parenteral nutrition, hemodialysis or continuous hemofiltration plus blood pressure and ventilatory support. To reduce catabolism and avoid protein depletion nutrient requirements are high and dialysis is used to maintain fluid balance and blood urea nitrogen below 100 mg/dL. Mortality in this group of patients exceeds 60% to 80%, but it is not the loss of renal function that accounts for the poor prognosis. It is superimposed hypercatabolism and the severity of the underlying illness. (*Adapted from* Druml [1]; with permission.)