Renal Nutrition Forum
A PUBLICATION OF THE RENAL DIETITIANS DIETETIC PRACTICE GROUP
Renal Dietitians
A DIETETIC PRACTICE GROUP OF AMERICAN DIETETIC ASSOCIATION
"Your link to nutrition and health."SM
VOLUME 24
NUMBER 3
SUMMER 2005

Nutrition Management of the Patient with Acute Renal Failure

By Marcia Kalista-Richards, MPH, RD, CNSD, Robert Pursell, MD, Robert Gayner, MD. *Marcia is a renal/nutrition support dietitian at Easton Hospital, Easton, Pa., and an instructor at Pennsylvania State University-Lehigh Campus, Fogelsville, Pa. Dr. Pursell and Dr. Gayner are members of Lehigh Valley Nephrology Associates, Bethlehem, Pa.*

Abstract

Acute renal failure (ARF) is associated with a mortality rate up to 50% despite advances in clinical practice and technology. ARF is an abrupt cessation or decline in normal renal function that is manifested by elevations of blood urea nitrogen (BUN) and serum creatinine that may be associated with decreased urine output. ARF is designated as prerenal, renal (intrarenal or intrinsic renal disease,) and postrenal, each of which has different causes. Systemic effects of ARF can be extensive, with associated metabolic alterations affecting carbohydrate, lipid, and protein metabolism. The initiation of renal replacement therapy (RRT) is an important consideration in patients who have ARF and are receiving nutrition support. This article addresses nutritional requirements and considerations of patients with ARF.

Table 1. Systemic or Physiologic Effects of ARF (4, 5)

Kidney	Decreased urine production leading to fluid retention, edema. Accumulation of nitrogenous waste.
Cardiac	Pulmonary or peripheral edema, arrhythmias. Loss of muscle tone, weakness, neuromuscular irritability, tingling lips/fingertips, electrocardiographic changes with hyperkalemia. Potassium levels >6 mg/dL could trigger bradycardia, heart block, asystole or other arrhythmia, or heart failure.
Respiratory	Dyspnea at rest or exertion. Crackles in lungs.
Neurologic	Accumulation of metabolic waste can affect mental status. Changes in cognitive function and level of consciousness, sensory change, and weakness of extremities can be signs of uremic neuropathy. Paresthesias related to hypocalcemia. Headaches, syncope, and seizures.
Hematology	Impaired red blood cell production, hemolysis, bleeding, hemodilution, and reduced red blood cell survival (60 versus 120 d) contributing to anemia, fatigue, and malaise. Less production of erythropoietin. Low hemoglobin/hematocrit causing a decrease in oxygenation, leading to dyspnea and platelet dysfunction.
Gastrointestinal	Anorexia, nausea, vomiting, constipation, diarrhea, dysgeusia, and stomatitis affecting overall nutrition. Weight loss masked by fluid retention. Uremia can trigger colitis, gastric ulcer, and bleeding. Increased urea causes foul breath.
Visual	Blurring related to electrolyte and fluid imbalance, hypertension.
Skin	Dryness, pitting edema, anasarca. Pruritus, ecchymosis, petechiae, delayed healing, pallor, yellowness related to uremia.

The Kidneys

The primary functions of the kidneys are to excrete end products of metabolism, regulate electrolyte and mineral concentrations, and maintain fluid and electrolyte balance (1). Other functions include urine production, dilution, and concentration; maintenance of blood pressure; concentration of extracellular and intracellular fluids; gluconeogenesis; maintenance of Ca/PO4 balance; and activation of vitamin and hormone synthesis (1). The kidney has approximately 1 million nephrons, each of which is composed of several functional segments, including the glomeruli, proximal tubules, distal tubules, loop of Henle, and collecting duct, which drains into the renal pelvis. The nephron clears plasma of the end products of metabolism (urea, creatinine, uric acid, inorganic and organic acids). Electrolytes (sodium, potassium, chloride, bicarbonate), minerals (calcium, phosphorus, magnesium), and micronutrients (zinc, selenium) are filtered through the glomeruli and are reabsorbed or excreted based on needs. Small nutrients such as glucose, small proteins, amino acids, and vitamins are filtered through the glomerulus and reabsorbed via active transport in the proximal tubule of the kidney (1).

Acute Renal Failure (ARF)

ARF is an abrupt cessation or decline in normal renal function manifested by

Continued on page 4

continued from page 1

elevations of blood urea nitrogen and serum creatinine that may be associated with decreased urine output (2,3). Urine flow or output rates used in the diagnosis of ARF include anuria (<100mL/d), oliguria (100 to 500mL/d), and polyuria nonoliguria (>500mL/d); normal urine output is 1 to 1.5L/d (3). ARF occurs when taxation of kidneys causes a sudden loss of function in at least 50% of nephrons (4). The problem may be specific to the kidney or part of multiple organ dysfunction syndrome (MODS). If the underlying cause is corrected, the nephrons may recover. In some cases, damage is permanent, and renal failure becomes chronic. ARF also may be superimposed on pre-existing renal insufficiency.

Although ARF is a disorder of the kidneys, it can have systemic effects (Table 1, page 1) and result in significant metabolic alterations (Table 2).

The prevalence of ARF is 1% in all hospitalized patients, 3% to 5% in general medical/surgical patients, 5% to 25% in intensive care unit (ICU) patients, 5% to 20% with open heart surgery, 20% to 60% in severe burns, 10% to 30% with aminoglycoside therapy, 20% to 30% in rhabdomyolysis, and 15% to 25% among those treated with the chemotherapeutic drugs cisplatinum, bleomycin, and vinblastine (3, 8).

Despite advances in clinical practice and technology, the 50% mortality rate associated with ARF remains constant (3, 4, 8). Death typically is due to severe infection or cardiopulmonary problems. Among predictive factors for a worse prognosis are age, previous health status, pre-ICU hospitalization, delayed occurrence of ARF, sepsis, oliguria, and severity of illness (including need for ventilation and hypotension)(9). The predictive value of nearly each factor influencing outcome in acute renal failure is still debated and continues to require further studies (9).

Causes of ARF

ARF can be defined as prerenal, postrenal, and renal (intrarenal or intrinsic renal disease), and the causes vary by type.

Prerenal ARF results from a decrease in renal perfusion that may be due to volume depletion (gastrointestinal fluid loss, excess diuresis, or salt-wasting nephropathy), volume redistribution (vasodilation from sepsis or antihypertensives), burns, peritonitis, pancreatitis, hypoalbuminemia (nephrotic syndrome, hepatic disease), reduced cardiac output (pericardial tamponade, myocardial infarction complications, acute/chronic valvular disease, cardiomyopathies, arrhythmias), or an embolus (thromboembolic or cholesterol) (3). Restoring renal blood flow and glomerular ultrafiltration can reverse prerenal ARF rapidly (3, 4).

Use of certain medications may contribute to the incidence of prerenal ARF. For example, nonsteroidal anti-inflammatory drugs (NSAIDs), cyclooxygenase inhibitors, or angiotensin-2 receptor blockers can impair renal autoregulatory responses by blocking the prostaglandin production that is needed to maintain renal perfusion. The risk for ARF associated with these medications is high in the elderly and those who have renal insufficiency, heart failure, and advanced liver disease (4).

Postrenal ARF results from obstruction of urine flow. Prostatic hypertrophy, ureteral obstruction (usually bilateral), and bladder outlet obstruction are common causes. Malignancy, inflammatory processes, vascular diseases, papillary necrosis, intratubular crystals (from methotrexate, acyclovir, calcium phosphate, sulfonamide antibiotics) also can be causative (3). Postrenal ARF is found in 1% to 14% of hospitalized azotemic patients, most often among the elderly, and generally can be reversed with proper medical management (3).

Intrarenal ARF (intrinsic ARF) is associated with renal parenchyma damage. Prerenal ARF can trigger the problem, but a major cause of intrarenal ARF and ARF in general is acute tubular necrosis (ATN), which usually is associated with damage to renal tubules caused by ischemia or nephrotoxins such as radiocontrast or aminoglycoside (3). The terms ARF and ATN frequently are used interchangeably, although they are not the same condition.

Predisposing factors to ATN include renal ischemia from prolonged prerenal azotemia, nephrotoxins (radiocontrast agents, aminoglycoside antibiotics, amphotericin, vancomycin, NSAIDs, cyclosporine), chemotherapeutic agents (cisplatin, carboplatin, methotrexate), pigmenturia (rhabdomyolysis, hemolysis), interstitial nephritis (ciprofloxacin, thiazides, furosemide, phenytoin, tetracycline, penicillin), and infections (streptococcal, staphylococcal)(3). Some clinical conditions associated with ATN are abdominal aortic aneurysm repair, major trauma, postoperative states, and gram-negative bacterial sepsis (3).

Diagnosing ARF

A variety of tests are used to diagnose ARF, including a history and physical examination, determination of urine flow, laboratory investigations such as blood urea nitrogen or serum creatinine, urinalysis,

Continued on page 8

Table 2. Metabolic Alterations Associated with ARF (6, 7)

Carbohydrate	Hyperglycemia, insulin resistance, accelerated hepatic gluconeogenesis despite exogenous glucose infusions.
Lipid	Impaired lipolysis causing increased triglycerides and low-density lipoprotein and very low-density lipoprotein cholesterol; decreased total and high-density lipoprotein cholesterol.
Protein and Amino Acids	Acidosis and insulin resistance stimulating protein catabolism, skeletal muscle release of amino acids, and negative nitrogen balance; increased gluconeogenesis, ureagenesis, liver secretion of acute-phase proteins.

Table 3. Tests Used to Diagnose Acute Renal Failure (ARF) (3, 4, 5, 10, 11)

History and Physical Examination

Chief complaint
History
General review of body systems, including pain, skin integrity, weight, hemodynamic parameters (including blood pressure, pulse)

Determination of Urine Flow (Normal urine output: 1 to 1.5 L/d)

Urine output		*Associated with:*
Anuria	<100 mL/d	Obstruction; bilateral renal, arterial, or venous occlusion; bilateral cortical necrosis; overwhelming acute tubular necrosis (ATN); severe acute glomerulonephritis
Oliguria	< 500 mL/d	Prerenal azotemia, intrinsic ARF, tubular necrosis, interstitial nephritis, glomerulonephritis, partial obstruction An inability to excrete waste occurs with outputs of <500 mL/d
Nonoliguria	>500 mL/d	Tubular necrosis, interstitial nephritis, partial obstruction
Polyuria excessive urine	3 L/d	Prerenal ARF if excessive urine output is the cause of prerenal state (rare), as in adrenal or mineralocorticoid deficiency states and excessive diuresis; more often indicative of polyuric acute tubular necrosis (2)

Laboratory Data

- Blood urea nitrogen (BUN)
- Creatinine
- BUN/Creatinine ratio
- Glomerular filtration rate
- Creatinine clearance
- Electrolytes
- Bicarbonate
- Acid base parameters
- Hemoglobin
- Hematocrit
- Calcium
- Phosphorus
- Magnesium
- Creatine kinase (CK-MM) (CK-MM is the dominant isoform in skeletal muscle, making it most sensitive to confirm rhabdomyolysis [10]).

Urinalysis

Specific gravity (Normal, 1.010 to 1.025)	>1.020=prerenal failure 1.10 (isosthenuric)=intrinsic or postrenal failure
Proteinuria:	>3+ suggests intrinsic glomerular injury
Urine pH (Normal, 4..5 to 8.0)	More acidic in prerenal azotemia than other forms; acidic in rhabdomyolysis
Glycosuria without hyperglycemia:	Suggests proximal tubular injury
Blood:	Positive reaction on dipstick=acute glomerular or tubular injury, urinary tract infection, or nephrolithiasis. If blood is present on dipstick but not microscopically, a pigment nephropathy (hemoglobinuria or myoglobinuria) should be considered.
Urine sediment:	Unremarkable findings in pre- and postrenal azotemia except for occasional hyaline cysts Blood and crystals in postrenal due to stones Active sediment= intrinsic ARF Brown granular casts=rhabdomyolysis Microscopic evaluation can distinguish between glomerulonephritis, ATN, and tubulointerstitial nephritis Eosinophils in large amounts in urine suggest drug-induced tubulointerstitial nephritis
Urine-to-plasma creatinine ratio:	>40=prerenal; the urine concentration is higher than plasma concentration and kidney can concentrate urine 20 to 40=borderline <20=ARF

Urine Electrolytes

Fractional excretion of sodium (less helpful in patients who are not oliguric or receiving diuretics):	<1% in oliguric patient suggests avid tubular sodium reclamation and prerenal azotemia with functional renal tubule >3% in oliguric patient suggests tubular injury and inability to absorb normal amounts of sodium >1% reflects tubular dysfunction and inability to absorb normal amounts of sodium Low value with diuretic use suggests volume depletion and prerenal ARF; elevated value may be result of ATN or diuresis
Urine sodium concentration (Normal range: varies widely)	<20 mEq/L associated with volume depletion, decreased blood flow to kidneys; suggests renal tubular mechanics intact, conserving sodium and able to concentrate urine >40 mEq/L suggests kidneys are losing the ability to concentrate as in some ARF, chronic renal failure, ATN. <10 mEq/L as an isolated measurement, often used as evidence of prerenal state; dependent on the state of water and sodium balance

Radiography

Plain films: kidney size, stones, skeletal abnormalities of secondary hyperparathyroidism
Intravenous pyelography: hazards make test of limited benefit
Renal ultrasonography: sensitive and specific for hydronephrosis detection, obstruction
Computed tomography can clarify cause of obstruction; not advantageous over ultrasonography

Pulmonary Artery Catheterization

Invasive hemodynamic monitoring that is an invaluable diagnostic adjunct in assessing and monitoring fluid balance (volume overload/hypovolemia)

Renal Biopsy

Recommended for ARF of unexplained cause or prolonged course. Used when other tests have not confirmed correct diagnosis and to decide on most appropriate therapy and prognosis.

continued from page 4

measurement of urine electrolytes, and radiography (Table 3). Invasive hemodynamic monitoring via a pulmonary artery catheter may aid in assessing and monitoring fluid balance of critically ill patients. A renal biopsy is recommended for ARF in which a cause cannot be determined or for ARF of prolonged duration.

Assessment of Nutritional Status

History

No single tool or measurement accurately reflects the nutritional status of a patient. Therefore, a comprehensive assessment should evaluate all available parameters, with ongoing monitoring of serial measurements. The use of a nutrition-focused physical assessment or Subjective Global Assessment is appropriate (12, 13).

The nutrition health history should examine nutrition-related problems, including intake, food intolerances, diet restrictions, allergies, the use of alternative therapies, and nutrient losses. This should be followed by a thorough physical examination and assessment of weight status that encompasses involuntary loss or gain and amount and percentage of weight change over an identified period of time. Depletion of muscle mass may be indicated by a prominent bony thoracic skeleton; hollow temples; clavicular prominence; or wasting of the scapula, quadriceps area, or interosseous muscle of the hand. Depletion of fat stores is evidenced by hollow eyes or decreased triceps skinfold. The gastrointestinal evaluation should include questions regarding the presence of nausea, vomiting, diarrhea, food intolerances, or malabsorption. Patients should be asked about their functional status, such as activities of daily living, and assessed for evidence of macro- and micronutrient deficiencies, such as skin lesions or hair loss, which can indicate zinc deficiency.

Biochemical Parameters

A thorough nutrition assessment should include evaluation of biochemical parameters and hepatic transport proteins, with careful consideration of factors that can alter results. It is generally accepted that no single parameter can be used as an indication of nutritional status (14).

Albumin, which is easy and inexpensive to obtain and has a 21-day half-life, can serve as a valuable prognostic indicator for the risk of increased morbidity and mortality (14). However, shifting of albumin from the intravascular to extravascular space during stress, hydration, and intravenous albumin administration can limit the value of this measurement in the ICU. Prealbumin, which has a short half life (2 to 3 days), has been observed to have a more rapid response to therapy. Because prealbumin is degraded in the kidney, the values may be higher in ARF, but no specific guidelines have been set for a patient with ARF (2). Prealbumin values of less than 30mg/dL in chronic kidney disease patients on dialysis have been associated with increased mortality (8).

Both albumin and prealbumin are transport proteins. Metabolic insult and inflammation result in a release of inflammatory cytokines and interleukin-1, causing hepatic reprioritization of protein synthesis. This leads to increased production of acute-phase reactants (C-reactive protein, fibrinogen, ceruloplasmin, haptoglobin) and decreased transport protein (albumin, prealbumin) synthesis (14). C-reactive protein is a positive acute-phase protein involved in the immune response (2). Increases can occur within 4 to 6 hours of the onset of acute stress, and declines generally reflect resolution of the inflammatory response (15). The rapid decrease in serum protein levels and increase in C-reactive protein levels reflect severity of illness more so than nutritional status (2, 14).

Transferrin is responsible for iron absorption and transport, although it is strongly influenced by iron status (14). Low transferrin levels might indicate malnutrition, but a patient with iron deficiency anemia can have an elevated transferrin level, which can complicate interpretation of the transferrin concentration.

Nitrogen balance is a useful indicator of the degree of catabolism and guide to nutrition support for critically ill patients with or without ARF. However, the validity of nitrogen balance is affected by severe nitrogen retention disorders (i.e., creatinine clearance <50 mL/min)(8). Nitrogen balance can be determined by calculating the urea nitrogen appearance (UNA) or the protein catabolic rate (PCR) in patients with ARF (8). UNA also can be used for patients receiving continuous renal replacement therapy (CRRT) including continuous ambulatory peritoneal dialysis (CAPD), continuous arteriovenous hemodialysis (CAVHD), and continuous arteriovenous hemofiltration (CAVH)(8).

Blood urea nitrogen (BUN) is the concentration of nitrogen (within urea) in the serum; such serum concentration depends on urea production (which occurs in the liver), tubular reabsorption, and glomerular filtration (11). Clinicians must consider factors other than filtration when interpreting changes in BUN (11). For example, BUN values may be elevated in the presence of internal hemorrhage, protein hypercatabolism, infection, excess protein provision, tetracycline, and steroids (11). On the other hand, BUN values can be low in patients who are malnourished, have profound liver damage due to the inability to synthesize urea, or when fluid overload initially dilutes BUN (11).

Creatinine, a byproduct of muscle catabolism, is a more sensitive indicator of kidney function that assesses urinary excretion. It is important to consider the baseline creatinine value and the patient's muscle mass. For example, a thin, debilitated person who has decreased muscle mass may not appear to have significantly elevated values in the presence of renal failure due to lower baseline values.

Creatinine concentrations may be increased by medications such as trimethoprim, cimetidine, and certain cephalosporins (11). Rhabdomyolysis can cause an elevated creatinine concentration related to muscle breakdown (10).

The normal range of BUN is 8 to 20mg/dL, and the normal value of creatinine is

Continued on page 9

continued from page 8

0.5 to 1.5mg/dL (11). The BUN:creatinine ratio is usually 10:1 (5). In most cases of renal failure, both the BUN and serum creatinine increase; in stable ARF, the ratio is maintained at 10 to 15:1 (5). Certain renal abnormalities characterized by low tubular flow can cause the BUN to increase, and factors such as liver disease may lower BUN, which would alter the BUN:creatinine ratio (5).

Nutrition Support

Treatment goals for ARF are designed to address the systemic effects of the condition (Table 4). Nutrition support is an integral part of such treatment because the nutrition prescription can affect fluid, electrolyte, and mineral status and the patient's nutritional status. Patients who have ARF and are at risk for nutrition disorders should be identified on admission to the hospital and their status and nutrition monitored throughout the hospital course. Patients who cannot or should not take oral foods for an extended time are candidates for nutrition support, with enteral nutrition preferred over parenteral nutrition (6).

Table 4. Treatment Goals (16, 17)

- Eliminate cause of failure
- Prevent further kidney damage
- Achieve renal recovery
- Support function of kidneys and other effected organ systems
  - Establish or maintain blood flow to kidneys for a prerenal condition
  - Treat intrinsic renal disease
  - Remove postrenal obstruction
- Maintain fluid and electrolyte, acid-base, and mineral balance; regulate volume status
  - Observe physical signs of edema: extremities, sacral, eyelids, other; color and amount of urine
  - Monitor for signs of hyperkalemia, hypermagnesemia, hyponatremia, hyperphosphatemia
  - Avoid hypokalemia/magnesemia caused by aggressive diuretic therapy
  - Correct acidosis
  - Improve hemodynamic stability
- Remove nitrogenous wastes
  - Monitor BUN, creatinine levels
  - Assess decision for start of renal replacement therapy based on: 1) presence of uremia, 2) significant renal failure with refractory hyperkalemia, 3) significant renal failure with refractory acidosis, 4) significant renal failure with refractory heart failure, 5) pericarditis
- Manage anemia
- Reassess use of nephrotoxic drugs
- Maintain acceptable nutritional status through provision of adequate nutrition support
  - Prevent compromise to organ systems
  - Prevent wasting syndrome, loss of lean body mass, malnutrition
  - Provide substrates to meet needs of hypercatabolic state and comorbid conditions
  - Prevent deficiencies and/or toxicities
  - Facilitate use of gut as status permits
- Protect and reserve vascular access for dialysis use; may place limits on intravenous access
- Decrease ICU/hospital stay
- Prevent mortality through aggressive treatment of conditions most related to mortality: sepsis, cardiac disease, or hemorrhage
- Address the physical and emotional needs of patient/establish support systems

Nutritional Requirements

Energy Needs

When available, the use of indirect calorimetry (IC) to measure oxygen consumption, carbon dioxide production, resting energy expenditure, and respiratory quotient can help determine energy needs. Certain factors, such as mechanically ventilated patients requiring fractional inspired oxygen concentrations (FiO_2) greater than 60% and leaking chest or endotracheal tubes, may lead to inaccurate results (8). The energy needs of people who have ARF can be influenced by nutrition and metabolic status as well as by the degree of hypercatabolism (e.g., sepsis, infection, cause of renal failure, severity of underlying illness, comorbid conditions). When direct measurement is not available, calorie requirements can be met by providing 35 to 50kcal/kg ideal bodyweight, with the upper level reserved for patients who are severely catabolic and whose nitrogen balance does not improve at lower intakes (1,8). There is no appreciable increase for renal failure alone (1); the increase in energy expenditure is related more to the critical illness than to ARF (2). For patients who are receiving maintenance hemodialysis or chronic peritoneal dialysis, the target energy intakes are 35kcal/kg/d for those who are age 60 years and younger and 30 to 35kcal/kg/d for those older than age 60 years (15).

Protein

Factors to consider when determining protein allowances and restrictions include the loss of amino acids across the membrane with hemodialysis treatments (10 to 12g of amino acids are lost per treatment of high-efficiency hemodialysis (18), amino acid loss in CAVHD (up to 30g/d but usually <15g/day) (19), effectiveness and/or frequency of RRT (number of days per week and hours on treatment), and whether the patient completes fully scheduled dialysis treatments on RRT. Dialysis treatments may not achieve goals of fluid, electrolyte balance, and nitrogenous waste removal when treatments are shortened for reasons of hypotension, cardiac instability, or problems with patency of dialysis access.

Protein-controlled diets (0.6 to 0.8g/kg/d) traditionally have been used to reduce uremic complications, slow renal disease progression, and avoid or delay the need for

Continued on page 10

continued from page 9

dialysis (1). Protein restriction is indicated for patients who have advanced chronic renal insufficiency but are not undergoing dialysis if they are well monitored (18). If ARF is expected to resolve in a few days and dialysis will not be needed, a protein intake of 0.6 to 1g/kg/d is recommended (8). The patient's nutritional and metabolic status and renal diagnosis determine the exact dose (8). Patients with chronic renal failure (CRF) who are undergoing hemodialysis or peritoneal dialysis typically require 1.2 to 1.3g/kg/d (16, 17) or up to 1.5g/kg/d, respectively (15,18).

ARF patients who are severely malnourished and catabolic may require as much as 1.5 to 1.8g/kg/d protein; such levels of protein intake have been associated with increased protein catabolic rates, which is desirable (18).

Patients undergoing continuous hemofiltration therapy require a protein intake of at least 1.0g/kg/d; less than 1g/kg per day of protein has been associated with greater nitrogen deficits (18). Azotemia can be controlled while providing more than 1g/kg/d of protein (18). For patients on CRRT, the recommendation is 1.5 to 2.0g/d protein (8).

ARF patients should be given a balanced mixture of essential amino acids (EAA) and nonessential amino acids (NEAA) (18). Previous studies supported the use of small doses of EAA plus dextrose rather than dextrose alone (18). More recent investigations comparing EAA administration with administration of a balanced mix of EAA and NEAA showed no difference in mortality, nitrogen balance, or BUN (18). Additionally, when EAA formulations are used for longer than 2 to 3 weeks, hyperammonemia and metabolic encephalopathy can occur (7, 18). ARF patients require NEAA such as arginine, ornithine, and citrulline to enable detoxification of ammonia via the urea cycle (18).

Fat

The requirements for lipids are based on the goals of providing adequate nutrition while preventing essential fatty acid deficiency and overfeeding. Omega-6 fatty acid intake must be monitored because high doses are associated with hypoxemia, bacteremia, and suppression of immune function (8). Serum triglyceride levels should be monitored before the initiation of parenteral nutrition and routinely through administration of intravenous lipids, as should serum glutamic oxaloacetic transaminase (SGOT), serum glutamic-pyruvic transaminase (SGPT), and alkaline phosphatase (8). Elevations in these measures suggest impaired hepatic clearance of the lipid load (8). Other lipid sources, such as propofol, which contributes a source of lipid calories, should be calculated in the formula. General recommendations are to provide 60% of total kcal as carbohydrate (not to exceed 5mg/kg/min) and 20% to 35% of total kcal as fat or 1g/kg/d in critical illness (8, 18).

Fluid

Each patient's fluid requirements depend on sodium concentration, fluid status, and renal function. Patients may present with anuria, oliguria, or polyuria. A general guideline for patients making urine is to replace losses (24-hr urine output + 500mL for insensible loss) (8). A weight loss of more than 1kg/d indicates volume loss or catabolism, which may alter the fluid allowance. It is important to assess weight gain for volume expansion because unintentional fluid gains also may place limits on the fluid allowance. When determining fluid needs, the clinician should consider use and success of RRT and comorbid fluid balance issues. In fluid overload states manifested by peripheral edema, hypoxemia due to pulmonary edema, or pleural effusion, intravenous fluid sources and parenteral nutrition substrates can be concentrated. Similarly, enteral products that provide up to 2kcal/mL are useful because they provide nutrient-dense substrates while limiting free water (8).

Micronutrients

Exact vitamin needs have not been established for patients who have ARF. In studies of ARF treated with dialysis and ARF associated with MODS, increased levels of ascorbate, carotene, and selenium were documented, with plasma levels of tocopherol being decreased only in MODS patients (2). The effects of vitamin and mineral supplementation in amounts exceeding the dietary reference intakes during critical illness are not known (2). Additional research is needed before developing guidelines.

Water-soluble vitamin supplementation is indicated with RRT because of increased clearance (1, 8, 18). The need for vitamin B6 and folic acid also is increased due to losses (1, 8, 18). Riboflavin, biotin, niacin, and pantothenic acid do not appear to be cleared by hemodialysis, thereby obviating the need for added supplementation above the dietary reference intake (1). For dialysis patients who have wounds, a standard renal multivitamin should be administered enterally or a multivitamin infusion and multitrace element infusion administered parenterally (20). Oral vitamin preparations have been formulated to meet the needs of patients requiring dialysis and include: vitamin C (40 to 100mg), vitamin B1 (1.5mg), vitamin B2 (1.5 to 1.7mg), niacin (20mg), vitamin B6 (10 to 50mg), vitamin B12 (6mcg/1mg), folic acid (800mcg, 1mg, 5mg), pantothenic acid (5 to 10mg), and biotin (150 to 300mcg). Some preparations also contain iron and or zinc.

The specific needs of vitamin A in ARF are not known, but because vitamin A levels are elevated with CRF, supplementation in ARF is not recommended for at least 2 weeks (8) or when RRT is not used. Excessive amounts can lead to toxicity, which would require withholding of vitamin A (1).

Supplementation of vitamin K may be indicated in ARF, especially when long-term antibiotic therapy that suppresses intestinal growth of bacteria that usually synthesize the vitamin, is prescribed (8). The use of coagulation therapy should be factored into decisions about whether to administer vitamin K and in determining the appropriate amount. The newer multivitamin infusions and lipids include vitamin K.

Vitamin C intake recommendations are 60 to 100mg/d (8) and should not exceed 200mg/d to prevent increased oxalate production, which can result in deposition

Continued on page 11

continued from page 10

of oxalate in the heart, kidney, and blood vessels (1). A dose of 10mg/d of pyridoxine hydrochloride (8.2mg/d pyridoxine) is recommended to avoid deficiency in patients with ARF (8).

Micronutrient recommendations for wound healing for patients with renal failure on dialysis in whom zinc levels are believed to be depleted or who have an increased risk of deficiency include 50mg zinc for 10 days (220mg ZnSO4) (oral/enteral) and 10mg for 10 days (parenteral) (20). Excess zinc supplementation (>150mg/d) interferes with copper absorption (20). Gastrointestinal loss or a catabolic state such as injury or burns can increase requirements.

Because of diminished urinary clearance, aluminum provision can be toxic in patients with renal failure if aluminum-containing antacids or phosphate binders are used over long periods of time (21). Excess aluminum can lead to osteodystrophy, anemia, and encephalopathy with dementia (1,21). Patients at highest risk for aluminum toxicity are those who have uremia and those receiving long-term parenteral nutrition (21).

The need for iron supplementation must be based on overall patient status, potential losses, sepsis, and the use of blood transfusions. Intake, dialysis-induced losses, frequent laboratory testing, impaired intestinal iron absorption, and occult gastrointestinal bleeding are likely to cause iron deficiency anemia (8). Added supplementation is needed when erythropoietin is administered (8). Certain laboratory parameters must be evaluated to determine if supplemental iron is needed and the amount required (8). Iron losses can be monitored by assessing serum iron and ferritin concentrations, percent transferrin saturation, and total iron binding capacity (8).

Carnitine

Carnitine is an amine compound required for the transport of long-chain fatty acids into the mitochondria for oxidation and ultimate production of ATP by oxidative phosphorylation (2). Carnitine is lost with dialysis, but the amount lost is unknown, and supplementation of L-carnitine is controversial. Kidney Disease Outcomes Quality Initiative guidelines have concluded that evidence is insufficient for routine supplementation (15). In July 2002, the Centers for Medicare & Medicaid Services issued a national coverage determination for the use of L-carnitine for end-stage renal disease that likely will increase use (22, 23). Patients should be monitored for symptoms related to carnitine deficiency, which include malaise, asthenia, general weakness or fatigue, skeletal muscle cramps, and decreased exercise capacity or low peak oxygen consumption after dialysis (15). Carnitine levels have been noted to be normal with ARF (2).

Electrolytes

Electrolyte supplementation should be individualized. Suggested doses of sodium are 1.1 to 3.3g/d orally, 500mg (22mEq/kg) enterally, and 1 to 2mEq/kg parenterally. Suggested doses of potassium are 780 to 2,000mg/d orally, 2g (51mEq/kg) enterally, and 1 to 2mEq/kg parenterally. The enteral and parenteral doses of both electrolytes are standard and not specific to ARF (8, 24). CRRT causes significant losses of magnesium, calcium, phosphorus, and potassium that require careful monitoring and repletion (25).

The Nutrition Prescription

Enteral

Several tube feeding formula options are available for patients with ARF. Standard 2-kcal/mL formulas can be used for patients requiring fluid restriction. The risk for refeeding syndrome should be assessed, keeping in mind that renal formulas tend to be low in potassium, sodium, and phosphorus compared with standard nonrenal products. Dialysis patients with persistent hyperkalemia or hyperphosphatemia may benefit from renal enteral products.

Parenteral

Concentrated substrates that include 20% to 30% lipid emulsion, 50% to 70% dextrose, and 10% to 15% amino acids generally are administered via a central access to avoid excessive fluid administration with fluid limits. Electrolytes (sodium and potassium) may be added as acetate salts or bicarbonate in the presence of metabolic acidosis. The dialysis bath formula (known as dialysate) has varying ion and mineral compositions that affect serum electrolyte values and, therefore, must be considered when developing the parenteral formula.

Conclusion

Patients with ARF are at nutritional risk and require thorough nutrition assessments to formulate appropriate nutrition prescriptions. The decision to initiate nutrition support should be based on the patient's overall condition, nutritional status, and laboratory results. ARF patients requiring nutrition support must be monitored closely to avoid potential complications.

REFERENCES

1. Wolk R. Nutrition in renal failure. In: Gottschlich MM, Fuhrman MP, Hammond KA, Holcombe BJ, Seidner DL, eds. *The Science and Practice of Nutrition Support: A Case-based Core Curriculum*. Dubuque, Iowa: Kendall/Hunt Publishing Co; 2001:575-600.
2. Charney P, Charney D. Nutrition support in acute renal failure. In: Shikora SA, Martindale RG, Schwaitzber SD, eds. *Nutritional Considerations in the Intensive Care Unit: Science, Rational, and Practice*. Dubuque, Iowa: Kendall/Hunt Publishing Co; 2002:209-218.
3. Hammes M, Gillum D, Brennan S. Acute renal failure. In: Hall JB, Schmidt GA, Wood LDH eds. *Principles of Critical Care*. 2nd ed. New York, NY: McGraw-Hill; 1998:1117-1131.
4. Campbell D. How acute renal failure puts the brakes on kidney function. *Nursing*. 2003;33:59-64.
5. Parker K. Acute and chronic renal failure. In: Parker J, ed. *Contemporary Nephrology Nursing. American Nephrology Nurses' Association*. Pitman, NJ: Jannetti, Inc; 1998:201-265.
6. Druml W. Nutritional management of acute renal failure. *Am J Kidney Dis*. 2001;37(suppl 2):S89-S94.

Continued on page 12

continued from page 11

7. Maliakkal R, Bistrian B. Nutritional support of ICU patients with acute renal failure. *J Crit Ill.* 1992;7:1261-1273.
8. Goldstein-Fuchs DJ, McQuiston B. Renal failure. In: Matarese LE, Gottschlich MM, eds. *Contemporary Nutrition Support Practice: A Clinical Guide*. 2nd ed. Philadelphia, Pa: Saunders; 2003:460-483.
9. Brivet FG, Kleinknecht DJ, Loirat P, et al. Acute renal failure in intensive care units: causes, outcome and prognostic factors of hospital mortality. A prospective, multicenter study. *Crit Care Med.* 1996;24:192-198.
10. Russel TA. Acute renal failure related to rhabdomyolysis: pathophysiology, diagnosis and collaborative management. *Nephrol Nurs J.* 2000; 27:567-576.
11. Traub S. The kidneys. In: Traub SL, ed. *Basic Skills in Interpreting Laboratory Data.* 2nd ed. Bethesda, MD: American Society of Health-System Pharmacist, Inc; 1996:131-157.
12. *Assessing the Nutritional Status of Dialysis Patients Using Subjective Global Assessment.* McGaw Park, Ill: Baxter Healthcare Corporation - Renal Division; 1993.
13. The American Dietetic Association Dietitians in Nutrition Support. *Nutrition-focused Physical Assessment Skills for Dietitians Study Guide*. 2nd ed. Chicago, Ill: Dietitians in Nutrition Support; 2000.
14. Cresci GA. Nutrition assessment and monitoring. In: Shikora S, Martindale R, Schwaitzberg S, eds. *Nutritional Considerations in the Intensive Care Unit: Science, Rational, and Practice*. Dubuque, Ia: Kendall/Hunt Publishing Co; 2002:21-30.
15. National Kidney Foundation. Kidney disease outcomes quality initiative clinical practice guidelines for nutrition in chronic renal failure. *Am J Kidney Dis*. 2000;35(suppl 2):S1-S140.
16. Monson P, Mehta R. Nutrition in acute renal failure: a reappraisal for the 1990s. J Renal Nutr. 1994:58-77.
17. Hasse J. Nutrition and organ failure – overcoming obstacles. In: Kalista-Richards M, Marian M, eds. *Sharpening Your Skills as a Nutrition Support Dietitian.* Chicago, Ill: The American Dietetic Association Dietitians in Nutrition Support; 2003:227-236.
18. A.S.P.E.N. Board of Directors and The Clinical Guidelines Task Force. Guidelines for the use of parenteral and enteral nutrition in adult and pediatric patients. *J Parenter Enteral Nutr.* 2002:26(suppl 1):78SA-80SA.
19. Frankenfield DC, Reynolds HN. Nutritional effect of continuous hemodiafiltration. *Nutrition*. 1995;4:388-393.
20. Ross V. Micronutrient recommendations for wound healing. *Support Line*. 2002;24(4):3-9.
21. Klien GL. Nutritional aspects of aluminum toxicity. *Nutr Res Rev*. 1990; 3:117-141.
22. Steinman T, Nissenson A, Glassock R, et al. L-carnitine use in dialysis patients: is national coverage for supplementation justified? What were CMS regulators thinking – or were they? Nephrol News Issues. 2003;17:28-39.
23. Schreiber B. CMS national coverage decision for L-carnitine injection: a reasonable process. *Nephrol News Issues*. 2003;17:40-46.
24. A.S.P.E.N. Board of Directors and the Clinical Guidelines Task Force. Guidelines for the use of parenteral and enteral nutrition in adult and pediatric patients. *J Parenter Enteral Nutr.* 2002:26(suppl 1):22SA- 24SA.
25. Klein CJ, Moser-Veillon PB, Schweitzer A, et al. Magnesium, calcium, zinc and nitrogen loss in trauma patients during continuous renal replacement therapy. *J Parenter Enteral Nutr.* 2002; 26:77-92.

Kalista-Richards M, Pursell R, Gayner R. *Nutrition Management of the Patient with Acute Renal Failure*. Copyright Dietitians in Nutrition Support, a Dietetic Practice Group of the American Dietetic Association. Reprinted by permission from Support Line, Vol 26; Number 6, 2004.

MANY THANKS

Thank you to the following peer reviewers for this issue:

Debbie Benner
Robin Diaz-Grady
Susan DuPraw
Susan Folk
Catherine Goeddeke-Merickel
Sharon Griff
Barbara Hutson
Yolanda Jeffries
Rita Kohse
Linda Lackney
Jayne Whiteside
Martha Wilkins
Laura Yates

Nominations Wanted for American Dietetic Association Renal Practice Group

The Nominating Committee is accepting nominations for enthusiastic and dedicated individuals for the 2006 ADA/RPG Executive Committee. The positions open are for Chair-Elect, Secretary, and Nominating Committee.

Please refer to RPG's website at www.renalnutrition.org after mid-August for descriptions of these positions and the nominations form. Deadline for submitting nominations is October 1. Please contact Susan Knapp, Nominating Committee Chair, with questions regarding nominations at sknapp@davita.com or 918-392-0290 Ext. 19.