29 Renal Disease

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Objectives

1. Describe the anatomical features and physiological function of the kidney.
2. List the major causes of acute kidney injury (AKI) and chronic kidney disease (CKD).
3. Review the nutritional requirements of patients with CKD and discuss how these needs change once dialysis is initiated.
4. Construct a parenteral nutrition (PN) formula that considers the metabolic changes that occur in AKI.
5. Compare the calorie needs in AKI to those seen in CKD.

Test Your Knowledge Questions

1. Which of the following metabolic alterations is most commonly observed in AKI?
   A. Decreased energy expenditure
   B. Metabolic acidosis
   C. Decreased serum magnesium concentration
   D. Metabolic alkalosis
2. Which of the following parenteral amino acid preparations is most appropriate for a dialysis-dependent patient with renal failure (RF)?
   A. Essential amino acids only
   B. Nonessential amino acids only
   C. Mixtures of essential and nonessential amino acids
   D. High branched-chain amino acids (BCAAs)
3. Which of the following is a measurement of body iron stores?
   A. Total iron-binding capacity (TIBC)
   B. Ferritin
   C. Transferrin
   D. Ceruloplasmin
4. What percentage of instilled dextrose is typically absorbed from peritoneal dialysate with a 6-hour dwell time?
   A. 25%
   B. 50%
   C. 75%
   D. 100%

Test Your Knowledge Answers

1. The correct answer is B. In AKI, a metabolic acidosis develops from organic and inorganic acid accumulation. Serum potassium concentrations increase as obligate renal clearance is reduced.
2. The correct answer is C. Essential and nonessential amino acids are lost via dialysis solutions used for hemodialysis (HD) and peritoneal dialysis (PD). The ability to synthesize nonessential amino acids is reduced in patients with acute renal insufficiency. Therefore, a solution containing both essential and nonessential amino acids is preferred in this clinical setting. Enriched BCAA solutions have been studied in AKI and have not been shown to improve clinical outcome.
3. The correct answer is B. Ferritin is a serum protein that binds iron and serves as a reliable indicator of total iron stores. Low levels of this protein are typically seen with iron deficiency anemia, while high values occur with iron overload from an excessive intake of iron or the presence of hemochromatosis. Ferritin is an acute-phase protein and is also increased with acute inflammatory diseases of the liver. In CKD, ferritin should be > 100 ng/dL and the transferrin saturation should be > 20%. The TIBC is a measure of serum iron and various proteins that transport iron within the circulation. A saturation index for these proteins is a measure of the available iron within the bloodstream. Transferrin is a circulating transport protein that carries iron that can be used in hematopoiesis. Ceruloplasmin is a serum transport protein that binds zinc and copper. It is not involved in the transport of iron.
4. The correct answer is C. In 6 hours, approximately 75% to 80% of the instilled dextrose of the dialysate solution is absorbed. This can provide a significant source of calories to the patient.

Background

The kidney is responsible for clearing nitrogenous waste and other metabolic byproducts and also regulates fluid, electrolyte, and acid–base balance; eliminates certain drugs; and synthesizes and metabolizes certain hormones. Because of these functions, the development of AKI or CKD can have a profound effect on the nutritional and metabolic status of patients. In AKI, an increase in protein and energy requirements parallels those seen in patients who experience trauma, serious infection, and other acute inflammatory illnesses. Patients with CKD may experience a series of metabolic changes that result in the development of protein–calorie malnutrition (PCM). The advent of renal replacement therapy (RRT) has vastly improved the management of AKI and CKD by improving survival in patients with AKI, enhancing the quality of life in patients with CKD, and by making it possible to provide adequate protein and energy to these patients. When provided in sufficient amounts, nutrition support can reverse PCM (in the absence of injury, inflammation, or the presence of cancer), reduce nutrition-related complications, and perhaps improve overall survival in patients with kidney disease. This chapter reviews the physiological changes that occur in patients with a decreased glomerular filtration rate (GFR), examines the impact of renal replacement therapies on these changes, and discusses the nutritional requirements of patients with RF and how to apply these principles to the provision of nutrition support.

Normal Renal Function

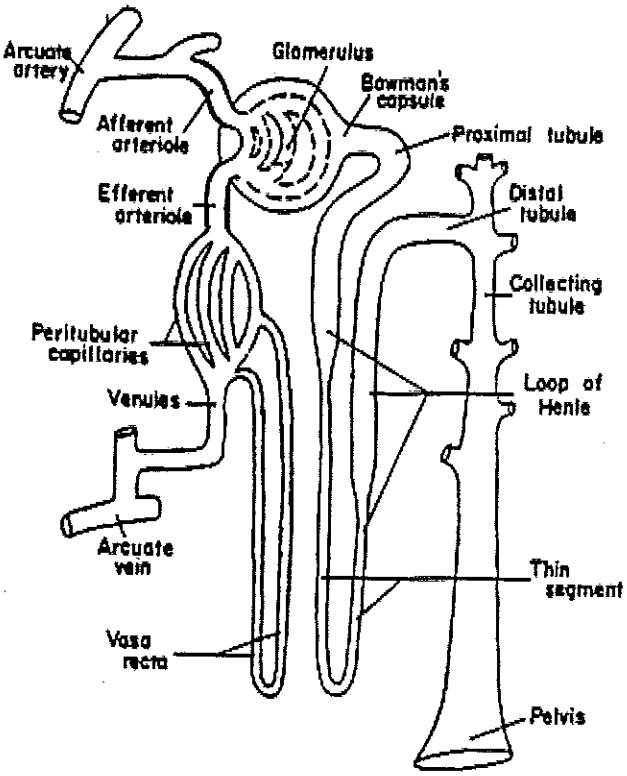
The major function of the kidney is to excrete most end products of metabolism, to regulate the body concentrations of electrolytes and minerals, and to regulate fluid and acid–base balance.[1,2] Other functions include gluconeogenesis, regulation of calcium–phosphorus balance, vitamin activation and metabolism, hormone synthesis and elimination, and drug metabolism and clearance.

The kidneys are two fist-sized organs located in the retroperitoneal space and are attached to the lower portion of the aorta

and vena cava by the renal arteries and veins, respectively.[1] Each kidney is composed of approximately 1 million nephrons, the term used for the filtering, secreting, and reabsorbing part of the kidneys. The nephron can be divided into several functional segments that include the glomerulus through which blood is filtered; the proximal tubule through which various substances are actively or passively reabsorbed, secreted, or metabolized; the distal tubule and loop of Henle that are involved in the secretion of potassium and hydrogen ion, and the regulation of volume and water; and the medulla, where urine is concentrated (Figure 29-1).[3] The majority of fluid and solute reabsorption occurs by active transport in the proximal and distal tubule and by passive transport, which relies on the development of an osmotic gradient that varies along the length of the loop of Henle and collecting tubule. This entire process results in the formation of urine, an ultrafiltrate of plasma.

The nephron functions in conjunction with the liver to clear the plasma of metabolic end products including urea, creatinine, uric acid, and organic and inorganic acids. Electrolytes (sodium, potassium, chloride), minerals (calcium, phosphorus, magnesium), and micronutrients (zinc, selenium) are filtered through the glomeruli and may be reabsorbed or excreted, depending on the metabolic needs of the body.[1,2] Small nutrients that are filtered through the glomerulus, such as glucose, small proteins, amino acids, and vitamins, are reabsorbed by active transport in the proximal tubule.[1]

FIGURE 29-1 Functional Nephron



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Acute Kidney Injury

Definition, Incidence, and Impact of Acute Kidney Injury

AKI is defined as an abrupt decline in kidney function over at least 24 to 48 hours that leads to a reduction of both glomerular filtration and tubular function.[4,5] Clinically, the diagnosis is made by observing a rise in the serum creatinine (Cr) often in association with a decrease in urine output.[6] When severe enough to reduce renal function below 30% of normal, a number of complications may occur including hyperkalemia, hyperphosphatemia, glucose intolerance, fluid overload, acidosis, and azotemia.[4,6] The causes of AKI may be divided into three broad categories depending on the injury. Intrinsic RF occurs with a direct injury to the kidney such as with glomerulonephritis. Prerenal RF results when there is reduced renal blood flow that can be caused by any renal hemoperfusion such as volume depletion, congestive heart failure, shock, acute myocardial injury, nephrotic syndrome, or cirrhosis. Post-RF is caused by a blockage of the urinary drainage such as ureteral obstruction or prostatic hypertrophy.[7,8] A list of the most common etiologies of AKI is presented in Table 29-1.

TABLE 29-1 Causes of Acute Kidney Injury

Category and Disorder	Example
Prerenal	
Hypovolemia	Skin, gastrointestinal, or renal volume loss
	Hemorrhage
	Sequestration
Cardiovascular failure	Congestive heart failure
	Cardiac tamponade
	Capillary leak during sepsis or anaphylaxis
Renal/intrinsic	
Glomerulonephritis	Poststreptococcal, bacterial endocarditis
Acute tubular necrosis	Hemolysis
	Rhabdomyolysis
	Medications
Interstitial nephritis	Drug allergy
Arterial disorder	Hemolytic uremic syndrome
	Malignant hypertension
	Hypotension/shock
	Thrombotic thrombocytopenic purpura
Postrenal	
Ureteral obstruction	Nephrolithiasis
	Neoplasm
Venous occlusion	Renal vein thrombosis
	Neoplasm
Urethral obstruction	Prostatitis
	Foreign object
	Neoplasm

AKI occurs in approximately 0.37% to 5% of all hospitalized patients and is most often caused by a combination of events including sepsis, hypotension, and exposure to nephrotoxic drugs and therapeutic agents.[4,9,10] AKI is associated with a 30% to 80% mortality rate, and the cause of death is usually the result of multisystem organ failure or uncontrolled sepsis, and is generally not directly related to the RF per se.[1,2,4] AKI that occurs in patients in the intensive care unit (ICU) carries a higher mortality than AKI that occurs in patients who are not receiving care in an ICU.[11]

Metabolic Abnormalities Associated with Acute Kidney Injury

Patients with AKI occasionally are both hypermetabolic and hypercatabolic as a result of the neurohumoral responses associated with acute injury.[9] The metabolic complications that occur in AKI are listed in Table 29-2 and include the loss of glucose homeostasis, muscle wasting, protein catabolism, electrolyte imbalance, and the development of metabolic acidosis.[10,12]

TABLE 29-2 Metabolic Complications of Renal Failure

Glucose intolerance
Insulin resistance
Hyperglycemia
Lipid abnormalities
Hypercholesterolemia
Hypertriglyceridemia
Protein and amino acid abnormalities
Decreased protein and amino acid synthesis
Increased protein catabolism
Azotemia
Fluid and electrolyte
Volume expansion and contraction
Hypo- or hypernatremia
Hyper- or hypokalemia
Hyper- or hypophosphatemia
Hypocalcemia
Metabolic acidosis
Gastrointestinal
Anorexia
Nausea and vomiting
Altered taste acuity
Gastroenteritis
Peptic ulceration
Ascites
Endocrine
Renal osteodystrophy
Secondary hyperparathyroidism
Amenorrhea
Infertility
Other
Anemia
Vitamin deficiencies
Hypertension
Atherosclerosis

Muscle wasting may be more obvious in elderly patients due to their inherent scarcopenia.[13] Protein catabolism can result in an accumulation of several byproducts of protein metabolism including an elevated blood urea nitrogen (BUN), and is referred to as azotemia. Besides urea, Cr, aromatic and aliphatic amines, guanidines, and indoles are elevated in the blood of these patients compared to those with normal kidney function.[14] Tissue catabolism also releases intracellular electrolytes such as potassium, phosphorus, magnesium, and protein-bound acids such as sulfuric acid (from sulfur-containing methionine and cysteine) and hydrochloric acid (from the chloride salt amino acids).[14] This accumulation can lead to metabolic acidosis and electrolyte imbalance. Metabolic acidosis increases skeletal muscle release of BCAA that are preferentially used for gluconeogenesis, worsening protein catabolism.

Nutrition support plays an important role in the setting of AKI and may influence the patient's course and ultimately clinical outcome.[4,15–17] Some authors have suggested that nutrition support may promote renal functional recovery.[14,15,18] Sufficient nutrition may decrease the degree of protein catabolism and decrease the severity of negative nitrogen balance.[10,15,16] Although it might seem necessary to achieve a positive nitrogen balance to enhance survival in patients with AKI, achieving positive energy balance and neutral nitrogen balance seems to be more important. As with all forms of nutrition support, the success of this therapy depends on the ability to reverse or control the patient's underlying illness, and positive protein balance cannot be attained in patients with AKI or CKD until all acute inflammatory processes are controlled.

Chronic Kidney Disease

Definition, Incidence, and Impact of Chronic Kidney Disease

CKD is defined as persistent kidney damage for more than 3 months that is associated with a decreased GFR or confirmed by renal biopsy or markers of kidney damage.[19,20] CKD is classified into five stages. Stage 1 is defined as kidney damage with GFR > 90 mL/min/1.73 m^2 and that ranges through Stages 2, 3, and 4 to Stage 5 as kidney failure with a GFR of < 15 mL/min/1.73 m^2. CKD currently affects one in nine Americans or more than 20 million people with another 20 million at increased risk of developing CKD.[19,20] Diabetes mellitus (see Chapter 34) and hypertension account for 65% to 80% of all new cases of CKD.[2,10,20] The remaining 20% are comprised of patients with primary renal diseases such as a glomerulonephritis, adult polycystic kidney disease, progressive CKD from a preceding episode of AKI, and some patients with genitourinary obstruction. A limited list of causes of CKD is presented in Table 29-3.

Diminished glomerular function leads to altered renal fluid, electrolyte, and mineral metabolism. Metabolism or clearance of certain drugs and the production of certain hormones and vitamins may be reduced as well. If a patient has CKD, the remaining nephrons increase their workload in an attempt to maintain the highest level of renal function possible. This increase in single-nephron GFR results in intraglomerular hypertension, stretching of the mesangial supporting cells, and an increase in oxygen-free radicals and other inflammatory

TABLE 29-3 Causes of Chronic Kidney Disease

Diabetes mellitus
Hypertension
Intrinsic renal disease
Glomerulonephritis
Vasculitis
Urinary tract obstruction
Malformation
Tumors
Vascular disease
Hemolytic uremic syndrome

metabolic products. This causes progressive scarring of the remaining glomeruli.[21] Protein restriction is a mainstay in the therapeutic armamentarium in the management of CKD.[15,20,21] This is largely based on the concept that a low protein intake may preserve renal function by reducing intraglomerular pressure, solute load, mesangial stretch, toxic products of metabolism, and overall nephron activity. However, the only firm result of protein restriction in patients with CKD is to prevent or postpone the development of uremic symptoms.

Metabolic Abnormalities Associated with Chronic Kidney Disease

There are multiple contributors to the development of protein energy wasting (PEW) in CKD.[22–26] Cytokines such as tumor necrosis factor, interleukin-1, and other neuroendocrine factors are effectors (see Chapter 23). Abnormalities reported to occur with CKD include reduced protein synthesis and increased protein breakdown, glucose intolerance with a diabetic-like state, and abnormal lipid metabolism with hypertriglyceridemia.[23]

Many of the metabolic complications of CKD are similar to those seen with AKI (Table 29-2). These complications include impaired electrolyte clearance, abnormal acid–base status, and altered fluid status.[1] Calcium and phosphorus metabolism is altered as a result of decreased conversion of 25-hydroxy- to 1,25-dihydroxyvitamin D, a fall in proximal tubular phosphate clearance, and changes in parathyroid responses to serum calcium concentrations. Anemia is common and is in part due to decreased erythropoietin synthesis and a decrease in the erythrocyte life span. Patients treated with HD and PD are prone to the development of water-soluble vitamin deficiency, and those treated with HD are subject to iron deficiency as a result of chronic blood loss. Patients treated with PD also can develop iron deficiency secondary to the required blood loss associated with repetitive blood sampling (30 to 60 mL/month depending on the patient's underlying disease processes). Uremia can secondarily worsen PEW through the development of anorexia, food intolerance, nausea, and vomiting.[25,26] Inflammatory factors associated with chronic RF (CRF) and other comorbid conditions can worsen PEW.[25,26] These factors should be considered when developing a nutrition program for individuals with CKD.

Renal Replacement Therapy

There are three main types of RRT: HD, PD, and continuous RRT (CRRT).[1,2] The decision to perform RRT and the type of RRT depends on a number of factors including the patient's condition, the availability of RRT, vascular and peritoneal access; the serum levels of various electrolyte and metabolic waste products, and the characteristics of the RRT methodology. Nutritionally, 8% of patients with RF have cachexia, adding to the practitioner's challenge.[13] Dietary protein restriction and RRT have been used together to reduce azotemia and signs and symptoms of uremia in CKD.[21,24] Dietary protein restriction decreases the severity of uremic symptoms, can delay the onset of dialysis need, and preserves GFR.[20,23] The type of RRT selected can also have an impact on the nutritional status of the patient. The characteristics of the various forms of RRT are outlined in Table 29-4.

TABLE 29-4 Features of Various Renal Replacement Therapy Methods

	Typical Regimen		Typical Use		Clearance of Fluid and Solutes						
	Time (h)	Frequency	ARF	CRF	Fluid	Urea (ml/min)	E'lytes[a]	Pro	AA	Quantitative Pro + AA	Effect on Calorie Needs
HD	3–6	2–4/wk	Y	Y	> 1 L/h	100–250	++	+	++	10–13 g/d	Increase
Peritoneal Dialysis											
CAPD	24	daily	Y	Y	≤ 1 L/d	20	++	+	+++	5–24 g/d	Decrease
Continuous RRT											
CAVHF/CVVHF	24	daily	Y	N	≥ 20 L/d	5–15	+	++	+		V[b]
CAVHD/CVVHD	24	daily	Y	N	≥ 20 L/d	20	+	++	+		V[c]
SCUF	24	daily	Y	N	≥ 20 L/d	20	+	++	+		V[d]

AA, amino acids; ARF, acute renal failure; CAPD, continuous ambulatory peritoneal dialysis; CAVHD, continous arteriovenous hemodialysis; CAVHF, continuous arteriovenous hemofiltration; CRF, chronic renal failure; CCCHD; CVVHF; E'lytes, electrolytes; HD,hemodialysis; N, no; Pro, protein; RRT, renal replacement therapy; SCUF, slow continuous ultrafiltration; V, variable; Y, yes.

[a]Specifically potassium, magnesium, and phosphorus.

[b]Depends on dextrose use in replacement intravenous fluids.

Hemodialysis

HD is highly effective in treating uremia and other metabolic complications associated with AKI and CKD.[1,2] While this therapy can be provided in-center or at home (home hemodialysis), in-center HD accounts for 80% of all RRT used to manage patients with end-stage renal disease (ESRD). Dialysis is used in RF to maintain fluid and electrolyte balance, and to prevent the accumulation of nitrogen waste products. This form of dialysis can also remove a variety of medications that would otherwise be excreted by the kidney and thus prevents the accumulation and potentially toxic effects of these drugs. In HD, blood is removed from a venous catheter or arteriovenous fistula, and is pumped through a filter that contains a semipermeable membrane.[27] A dialysate solution is pumped through the filter on the other side of the membrane in the opposite direction. The dialysate is an isotonic sodium chloride solution with calcium, magnesium, potassium, bicarbonate, and dextrose. Modern dialysis machines allow hypertonic concentrations of sodium to be attained that can stabilize the blood pressure in some patients on HD who are prone to the development of hypotension across the HD procedure. The counter-current flow of dialysate to blood increases dialysis efficiency. Solutes such as urea, creatinine, and potassium are cleared by diffusion or convective transport and in general, molecular substances with a size < 5000 daltons can be efficiently removed with HD. Excess volume (the equivalent of isotonic saline) is removed by the application of a pressure gradient across the dialysis membrane. HD clears amino acids and small peptides from the blood with losses as high as 10 to 13 g per session.[28] HD also removes some water-soluble vitamins (such as vitamin C and pyridoxine), minerals, and electrolytes.

Magnesium is dialyzable and phosphorus can be removed as well. Calcium, potassium, sodium, and bicarbonate concentrations in the dialysate can be adjusted as needed based on the patient's condition. In ESRD, HD is typically administered 3 to 4 hours per day, 3 days per week to provide effective RRT.

Peritoneal Dialysis

PD is useful for the management of CKD.[29,30] Although this form of RRT is quite effective, only 15% of dialysis patients use PD because of access problems, difficulty complying with the prescription and thus achieving adequate clearance of solute, the potential for peritonitis, and hyperglycemia.[1] PD has the advantage of being relatively "low-tech," portable, and useful for home therapy.[1] The peritoneum acts as a semipermeable membrane where solute and water cross between the blood and peritoneal fluid. Elevated concentrations of urea and other solutes pass from the blood into the dialysate, which is initially free of urea. Potassium, urea, glucose, and other electrolytes pass between the peritoneal capillaries and the dialysate by diffusion based on the concentration gradient.[2] Hypokalemia (see Chapter 7) can occur on occasion because most commercially available solutions do not contain potassium. Potassium can be added to the dialysate, usually in amounts ranging from 3 to 5 mEq/L of solution to prevent or treat hypokalemia.

Water crosses from the blood to the dialysate as of the result of a glucose-mediated osmotic pressure gradient that is provided by using a dextrose-based dialysis solution. Commercially prepared dialysis solutions contain dextrose in concentrations of 1.5 to 4.25 g/dL or 1500 to 4250 mg/dL, which is considerably higher than the normal serum glucose of 60 to 110 mg/dL. As water moves from the blood into the peritoneal dialysate, solutes transfer to the peritoneal fluid based on their size and electrical charge. This transfer is termed convection or solvent drag, and is responsible for a large part of the clearance of small molecules from the uremic patient on PD. Although PD is not as efficient as HD per unit of time in removing solutes and water from the body, its application on a continuous basis allows it to be as effective as HD as a chronic therapy. Continuous ambulatory PD (CAPD) is the most commonly used form of PD.[29] Adult patients will typically instill (and drain) 2 to 3 L of dialysate four to five times each day with dwell times of 4 to 6 hours. Another form of PD is continuous cycling PD (CCPD) that uses a cycling machine that automatically fills and drains the patient's abdomen of dialysate usually over the nighttime while the patient sleeps. The machine is set up with the appropriate amount of peritoneal dialysate, and the patient connects to the dialysate circuit. The cycler warms the peritoneal fluid, measures and delivers the prescribed amount of dialysis fluid, controls the time that the dialysate dwells in the abdomen, and drains the abdomen of dialysate over a preselected time period. Although CCPD was originally prescribed as a procedure that drained the patient's abdomen at the end of the sleep cycle leaving no fluid during the daytime, many if not most CCPD patients will leave the last dwell in place during the day; some patients require a manual exchange of dialysate as if the patient were on CAPD.

PD can provide a significant glucose load to the patient via the dialysate. The amount absorbed depends on the volume infused, the dwell time, the dialysate dextrose concentration, and the condition of the patient's peritoneal membrane.[30] The energy derived from the dialysate dextrose must be considered when formulating a nutrition care plan. Protein and amino acid losses via PD can be significant and typically range from 5 to 24 g/d.[31] Protein losses can be higher if therapy is complicated by peritonitis. PD is effective in removing electrolytes, trace elements, and vitamins, and is inefficient at removing medications.

Patients receiving PD can remain ambulatory, provide their own care, and have reduced dietary restriction because their waste products are constantly filtered. Patients with heart disease experience fewer complications with PD compared to HD because fluid shifts are not as rapid as those that occur with HD. Common complaints include bloating, abdominal fullness, and loss of appetite from the indwelling dialysate.

Continuous Renal Replacement Therapies

CRRTs are ideal for patients who are unable to tolerate standard HD.[32,33] Although HD remains the most common form of dialysis during AKI, CRRT has become the preferred method of dialysis for patients with AKI in some centers. Slow low efficiency dialysis (SLED) or continuous veno-venous HD (CVVH) may be appropriate. SLED is a modified HD treatment that uses low blood flow rates, low dialysate flow rates, and an extended dialysis time of usually 8 to 24 hours. SLED is an emerging technique because it can achieve adequate solute and volume removal while causing less hemodynamic instability than conventional HD but does not require special equipment other than standard HD equipment.[1]

Continuous arteriovenous hemofiltration, CVVH, and continuous veno-venous hemodiafiltration are forms of CRRT that are currently utilized for the therapy of AKI in the United States. Of these, CVVH is the most commonly used procedure. The "VV" types of CRRT rely upon a blood pump to drive blood flow for the therapy and the "AV" CRRT uses the energy from the patient's own arterial pressure to drive the blood flow. All of these therapies require a specialized filtration or diafiltration membrane that is highly permeable to fluid, electrolytes, minerals, and certain drugs. Because of this, solutes can be removed by convection. This can require large volumes of intravenous fluid to maintain adequate circulating blood volume. The filters are expensive, and patients require continuous anticoagulation; clotting of the extracorporeal circuit is a frequent occurrence. However, trained ICU staff can maintain the procedure, and the provision of enteral and parenteral feeding solutions is made easier because volume overload and electrolyte imbalance can be corrected over the entire procedure without having to make changes common to those needed for the provision of HD such as highly concentrated feeding solutions, electrolyte imbalances, and worsening metabolic acidosis between HD treatments. The decision to use a particular continuous form of HD or HF, SLED, or HD relies upon the experience and comfort of the attending physician and nursing staff with a particular form of RRT.

The effectiveness of SLED and CRRT makes it possible to provide nutrition support without the need to restrict protein or fluid to a great degree.[34] Patients with AKI can have increased protein needs because of the associated inflammatory response to the RF or other comorbid conditions.[25,26] CRRT allows increased protein to be given to patients.[35] The amount of dialysis delivered to a patient should be designed to remove the metabolic wastes created from catabolism and nutrition support. The nutrition regimen should never be designed to decrease the need for dialysis.

Nitrogen amounts lost during CRRT are dependent on the method used. However, the differences in amounts lost between methods are not significant.[36] Protein requirements for adults on CRRT are at least 1 g/kg/d and may be as high as 2.5 g/kg/d because small peptide and amino acid losses can be high.[34,37,38] However, the losses with SLED are minimally increased over those losses from standard HD. In one study, patients with AKI treated with CRRT had serum levels of amino acids that were below their normal range until the protein intake reached 2.5 g/kg/d.[39] Fluid losses can be as high as 20 L per day; therefore, fluid replacement is important for fluid balance.[34,38] Serum electrolytes need to be measured frequently throughout the therapy period.

Energy needs for patients receiving CRRT can be calculated as 1.3 times above their resting energy requirement or as 30 to 35 kcal/kg.[25,34,38] Dextrose-containing dialysate solutions have been used for CRRT and may also represent a significant calorie source.[34] Because CRRT may require up to 20 L of replacement fluid, the choice of replacement fluid can be significant. The dextrose load should be calculated and converted to carbohydrate calories, and this calorie load in the replacement fluid should be subtracted from the dextrose provided in the PN solution, or the enteral nutrition (EN) rate (or formula) should be adjusted to maintain a stable dextrose intake. The amount of glucose transferred across the dialysis or filtration membrane can range from 35% to 45% or higher depending on the dextrose concentration, dialysate flow rate, and the blood glucose concentration.[34] As an example, a 1 L/h dialysate flow at a concentration of 1.5% and a 40% uptake provides about 500 cal/d, as illustrated in the following equation.[34]

$$\text{L/h} \times 24 \text{ hrs} = 24 \text{ hrs} \times 15 \text{ g/L dextrose (1.5\%)} = 360 \text{ g} \times 0.4 \text{ (40\%)} = 144 \text{ g} \times 3.4 \text{ kcal/g} = 490 \text{ kcal/day from the dialysate}$$

Use of dextrose-containing dialysate with a physiological concentration of dextrose (0.1% to 0.15%) or no dextrose may provide a better strategy to reduce calorie intake and minimize problems with glucose control.[34] CAPD fluids may also be used as CRRT dialysate solutions.[34] Wooley and colleagues provide a good review of available solutions.[34] The calories provided from both the dialysate and replacement fluid should be reduced from the total carbohydrate calories provided in PN or from EN feedings. The EN rate reduction can drop the protein intake, and additional protein powder replacement may be needed.

Glucose losses may occur in the ultrafiltrate fluids removed by CRRT. Glucose losses into the dialysate are equivalent to the serum blood glucose concentration they are dialyzed against.[39] These losses should be calculated as the PN solution dextrose concentrations or EN formula rates may need to be increased to compensate for the loss of the dextrose in the dialysate.

Standard adult doses of potassium, phosphorus, magnesium, and minerals may be required in patients on CRRT because of the large losses in the dialysate.[38,40] Premixed dialysate and replacement fluids are available to meet the electrolyte and fluid needs but not the mineral needs. Standard vitamin and mineral (trace element) packages should be provided to patients receiving CRRT to meet the recommended daily intake and the losses in the dialysate.[17,38]

Nutrition Assessment

Nutrition assessment is necessary to determine the appropriateness of the nutrition support regimen and response to therapy.[14,16] This topic is discussed in Chapter 9 and is reviewed in Table 29-5. The cause(s) of AKI/CKD and any associated complications should be noted. Medications must be evaluated to make certain that nephrotoxic agents are reduced or discontinued, and that renally cleared medications are appropriately adjusted. Current weight, change in weight, body mass index, and serum visceral protein concentrations like albumin and transferrin, C-reactive protein, creatinine, subjective global assessment (SGA), normalized protein equivalent to nitrogen appearance, and diet history are all useful in combination to establish the severity of malnutrition.[25,26] The combination of these measurements provides an assessment of visceral and somatic protein pools, body weight and hence fat mass, and nutrient intake.[25,26]

Serum albumin is recommended for routine measurement because there is a large body of literature that defines the normal serum albumin values, characterizes the nutritional and clinical factors affecting serum albumin concentrations, and demonstrates the relationship between serum albumin concentrations and outcome.[25,26] However, its usefulness is only certain when properly interpreted. In acute illnesses, the serum albumin will fall precipitously and will not reflect the patient's nutritional status. In patients with CKD, a slow fall or rise in the serum albu-

TABLE 29-5 Nutrition Assessment and Monitoring in Renal Failure

Medical history	Physical examination
	General appearance
Dietary history	Blood pressure
Interview	Fluid status
Diet recall	Skin turgor
	Urine output
Anthropometrics	Intake and output
Current weight	
Usual weight	Signs of nutrient deficiency
Dry weight	Muscle wasting
Height	
	Diminished fat stores
Medications	Chilosis
	Glossitis
Nitrogen Status	Poor wound healing
Albumin	Rash
Urinary nitrogen	
Appearance (UNA)[a]	Laboratory measures
	Electrolytes
Renal function	Sodium
BUN	Potassium
Creatinine	Chloride
	Bicarbonate
Creatinin clearance[a]	
	Minerals
	Calcium
	Phosphorus
	Magnesium
	Glucose
	Triglycerides/cholesterol
	Complete blood count

BUN, blood urea nitrogen; UNA, urea nitrogen appearance.
[a]See text for equation.

min may usually be interpreted as the development of protein malnutrition or improving protein nutrition, respectively.[25,26] The serum albumin can only be considered as a true marker of visceral protein metabolism when there is no evidence of any disorder that causes inflammation or a change in albumin metabolism (cirrhosis or nephritic syndrome). The transferrin concentration is best used to evaluate the state of circulating iron available for hematopoiesis and cannot be reliably interpreted as a marker of nutritional status except when there is no evidence of inflammation or iron deficiency. The C-reactive protein is useful in defining the presence of an inflammatory process that would have an adverse effect on a person's nutritional status. A normal C-reactive protein has no relationship to a normal nutritional status. The serum creatinine (in the absence of rhabdomyolysis or muscle injury) is reflective of the patient's skeletal muscle mass and can suggest long-standing malnutrition. The patient's age and sex must be factored into the interpretation of the serum creatinine. Normalized protein equivalent to nitrogen appearance is of little help outside the research arena and has little clinical use.

Body weight is useful because of the clear association between body weight and body fat mass and because body weight is correlated with clinical outcome.[25,26] Whenever possible, the patient's dry weight (without edema or ascites) should be used because it best represents the metabolically active lean tissue of an individual. Overweight and obese patients may need to use an adjusted dry weight when calculating Cr clearance to determine medication adjustments.[25,26] Finally, fluid status should be monitored with daily weight and intake and output records because most patients will require some degree of fluid restriction. SGA is recommended because it gives a comprehensive overview of nutritional intake and body composition, including a rough assessment of both muscle mass and fat mass, and because it is correlated with mortality rates.[25,26] The SGA is also easily calculated and requires no special tools or measurements.

Assessment of nutrient intake is essential for assessing the probability that a patient will develop PEW, for evaluating the contribution of inadequate nutrient intake to existing PEW, and for developing strategies to improve protein-energy nutritional status. Also, nutrient intake is correlated with clinical outcome.[25,26] A dietary history should be obtained because anorexia is common, and avoidance of certain foods may identify patients at risk for specific micronutrient deficiencies.

It is recommended that patients with AKI and patients with Stage V CKD be assessed for an inflammatory response because it can lead to weight loss and a depressed appetite, and is linked to increased mortalilty.[16,17] Acute-phase reactant protein (cytokines, C-reactive protein) as markers of inflammation may be useful measures for assessment, but more information is needed.[16]

Renal function should be assessed by the Cr and Cr index (release of muscle Cr as a function of body weight). An adult woman (in the absence of muscle injury) releases 15 to 20 mg Cr/kg body weight per 24 hours, while a similar adult man will release 20 to 25 mg/kg body weight of Cr per 24 hours. Old and frail individuals may release less, and body builders or those with a markedly increased muscle mass may release more. BUN and serum Cr are readily measured and are easy to monitor during the course of therapy. A low serum Cr suggests a low dietary protein intake and/or diminished skeletal muscle mass, and is associated with increased mortality rates.[25]

The Modification Diet in Renal Disease (MDRD) study developed a GFR formula that has less variability and is more accurate than other equations.[1,20] The MDRD formula factors in serum Cr, age, gender, ethnicity, BUN, and albumin. Some data suggest that the MDRD formula be modified for increased precision. The Cockcroft–Gault is also a useful equation that factors in age, weight, height, and gender.[20,41] The National Kidney Foundation (NKF) Kidney Disease Outcome Quality Initiative (www.kidney.org) provides both formula calculators for use. The serum Cr must be stable and unchanging for the calculation of either equation to be clinically relevant as an unstable Cr makes the calculation unreliable. The value of the MDRD and Cockcroft-Gault formulas lies in the fact that one can predict what alterations in renal function can be predicted by the result of the formula. For instance, it is not necessary to know that the GFR is 28 mL/minute or 24 mL/minute. The importance is that the GFR < 30 mL/minute and that

hyperkalemia, hyperphosphatemia, volume overload, medication clearance and metabolism, impaired 1-alpha hydroxylation of 25-OH vitamin D, secondary hyperparathyroidism, and a decreased clearance of insulin can be expected. It would also signal that planning for HD or PD as well as renal transplantation should begin. In a patient with AKI, the daily increase of the serum Cr means that the GFR is impaired such that the daily production of Cr cannot be cleared and the patient should be treated as if the GFR < 10 mL/minute.

Urea nitrogen appearance (UNA) is a measure of net protein degradation in patients with RF. This determination is based on the fact that urea is the major nitrogenous product of protein metabolism. The UNA is used to determine nitrogen balance because urinary nitrogen is usually very low (in patients with AKI or those with advanced CKD), and therefore cannot give a reliable measure of nitrogen metabolism. The UNA can be calculated as follows:

$$\text{UNA (g/d)} = \text{UUN} + \text{BUN} = \text{SUN}_f - \text{SUN}_i \times 0.60 \times \text{BW}_i + (\text{BW}_f - \text{BW}_i) \times \text{SUN}_f$$

where UUN is urinary urea nitrogen, BUN is blood urea nitrogen, SUN is serum urea nitrogen, BW is body weight (kg), 0.60 is the estimated fraction of body weight that represents body water, i is initial, and f is final values. All nitrogen measures are in g/d. This measure can be adjusted to represent total nitrogen appearance (TNA) as follows:

$$\text{TNA (g/d)} = 1.19\ \text{UNA} + 1.27$$

TNA attempts to account for all nitrogen output including urinary, fecal, change in BUN, and hemodialytic losses. There are no data on the efficacy of TNA with CRRT. This value can be converted to protein equivalents by dividing the result by 6.25. This difference between protein intake and TNA is a good measure of nitrogen economy in patients with RF. To promote a positive nitrogen balance in patients with AKI, protein intake should be adjusted according to catabolic rate, renal function, and dialysis losses.[16] However, positive nitrogen balance in AKI patients cannot be attained until the active, inflammatory disease processes have been controlled.

In CKD with GFR < 30 mL/min, several mechanisms controlling acid–base balance are ineffective. Patients develop an inability to normally secrete H1 in the distal tubule because of distal tubular damage and a decreased renal production of ammonia. Most patients also have a proximal tubule disorder resulting in mild bicarbonate wasting. Untreated metabolic acidosis worsens secondary hyperparathyroidism and causes skeletal muscle release and wasting of BCAAs.

Anemia is common in RF because of the associated decrease in erythropoietin production. Iron deficiency may occur because of blood loss associated with HD. Folate deficiency is rare, but vitamin B_{12} deficiency may be more common than previously thought.

Nutrition Requirements

Providing adequate calories, protein, and electrolytes to meet the needs of patients with CKD can both minimize the metabolic complications associated with their RF but also retard the progression of renal disease.[10,20,23] Table 29-6 lists the nutrition requirements for adults. Nutrition support should be considered for all normally nourished patients with CKD in whom oral intake is not anticipated for 5 to 7 days.

Energy

Energy needs for patients with AKI and CRF are similar to those of nonuremic patients (see Chapter 3). Typically, there is no appreciable increase in energy needs with RF alone.[15] Rather, it is the physiological stress of associated medical conditions and surgical interventions that increase the energy needs of patients with AKI or CKD. For patients with AKI, it is recommended that they receive 25 to 35 kcal/kg or 1.3 times their resting energy expenditure (REE).[34,37] For significantly underweight or obese patients, the European Society for Clinical Nutrition and Metabolism recommends a energy intake of 20 to 30 kcal/kg for enteral feeding adjusted to individual needs while for patients with AKI and stable CKD, 30 to 35 kcal/kg for PN is recommended.[17] For patients receiving CRRT, Kopple[9] suggests 35 to 38 total kcal/kg/d, while Seidner and colleagues[10] recommend a range of 30 to 40 total kcal/kg/d for most patients with CKD. Conditions such as severe sepsis, respiratory failure, and other hypercatabolic conditions may increase calorie requirements to 1.5 to 2 times the REE or 30 to 45 total kcal/kg/d.[40] Energy needs, in summary, for patients with AKI and CKD are similar to those of nonuremic patients and must be adjusted based on the severity of their illness, comorbid conditions, and type of dialysis.[13,40]

Indirect calorimetry is recommended for determining energy needs in patients with RF.[16] It is a noninvasive means for measuring the oxygen consumed compared to the carbon dioxide expired and then calculating the patient's resting metabolic rate.[42] Because indirect calorimetry has limited availability and may be expensive, equations are commonly used to calculate energy needs.[42] For either AKI or CKD, energy requirements can also be calculated using the Harris-Benedict equation for REE and an appropriate stress factor.[37] An adjusted weight should be used for patients over 120% of their ideal body weight. Patients with CKD receiving HD or CAPD may have PCM and may need additional calories for weight gain if they are underweight. Calorie distribution and fluid needs must be considered when determining an appropriate PN regimen or choosing an enteral tube feeding formula. Energy requirements are usually met by providing a balanced formula that provides protein (10% to 15% total calories), carbohydrate (55% to 70% total calories), and fat (20% to 30% total calories). Excessive lipid calories should be avoided to minimize risk of hypertriglyceridemia caused by diminished clearance rates in CKD. Fluid restriction is often necessary when RF is associated with oliguria or anuria. When nutrition support is provided via tube feeding, concentrated enteral formulas, which provide 1.5 to 2.0 kcal/mL, are generally preferred.

Energy delivery from RRT must be considered when deciding amounts to provide via enteral and parenteral solutions. PD can provide a significant number of calories with amounts being as great as 500 to 1000 kcal/d.[43] Figure 29-2 illustrates several methods that can be used for estimating calorie uptake from PD.[34] CRRT can be an indirect source of calories for patients with

TABLE 29-6 Adult Renal Failure Nutrition Requirements[10,13,16,17,21,34,37,38,40,45,51,53]

	Predialysis	PD	HD/CRRT	PN[a]
Energy	ARF:Harris-Benedict: or 35–50 kcal/kg CRF:Harris-Benedict: or 35–38 kcal/kg	Stress factor 1.5–2.0 Stress factor 1.1–1.2	Same as predialysis or PD	Same as predialysis or PD
Protein	0.6–0.8 g/kg[a]	1.2–1.3 g/kg[a] Up to 1.5–1.8 g/kg	1.2–1.3 g/kg (HD)[a] Up to 1.5–1.8 g/kg (HD) > 1 g/kg (CRRT) Up to 2.5 g/kg (CRRT)	0.6–0.8 g/kg[a] Up to 2.5 g/kg
Fluid	As tolerated	As tolerated	As tolerated (CRRT) Urine output[b] 500 mL (HD)	As tolerated
Electrolytes				
Sodium	As tolerated	As tolerated	As tolerated (CRRT) 2 g/d (HD)	35–75 mEq/L
Potassium	As tolerated	3–4 g/d	2–3 g/d (HD)	10–40 mEq/L
Minerals				
Calcium	1.0–1.5 g/d	< 2000 mg/d	< 2000 mg/d (HD)	4.8–9 mEq/L
Phosphate	≤ 10 mg/kg/d	≤ 17 mg/kg/d	≤ 17 mg/kg/d (HD)	3–15 mM/L
Phosphorus restricted to 800–1000 mg/day for elevations of serum phosphorus or plasma PTH: Stage 3 and 4: Serum phosphorus > 4.6 mg/dL Stage 5: Serum phosphorus > 5.5 mg/dL OR Stage 3: Plasma intact PTH > 70 pmol/L Stage 4: Plasma intact PTH > 110 pmol/L Stage 5: Plasma intact PTH > 300 pmol/L				
Magnesium	DRI[c]	DRI[c]	DRI[c]	2–12 mEq/L
Vitamins	DRI[c]	DRI[c]	DRI[c]	Standard additive
Vitamin supplements		DRI with dialysis	DRI with dialysis	DRI with dialysis
Vitamin C	——	75–100 mg/d	75–100 mg/d	——
Pyridoxine	——	5–10 mg/d	——	——
Folic Acid	——	1 mg/d	1 mg/d	——
DHCC	As needed	As needed	As needed	As needed
Trace elements	DRI	DRI	DRI	Standard additive
Supplements				
Iron	As needed[d]	As needed[d]	As needed[d]	As needed[d]
Other				
Carnitine	——	As needed[e]	As needed[e]	——

ARF, acute renal failure; CRRT, continuous renal replacement therapy; DHCC, dihydroxycholecalciferol; DRI, dietary reference intake; HD, hemodialysis; PD, peritoneal dialysis; PN, parenteral nutrition; PTH, parathyroid hormone.
[a]Initial needs, increased needs with dialysis/stress/hypercatabolic states.
[b]Standard needs, adjust based on serum levels.
[c]Initially, adjust based on serum levels.
[d]For anemia, follow iron/ferritin status.
[e]Losses occur, but a standard dose has not been established.

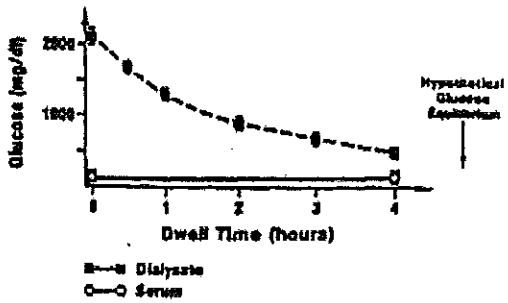
FIGURE 29-2 Glucose Absorption Estimates

1. Dwell time data – Wideroe

Dwell time (hr)	% Glucose absorbed from dialysate
1	29 ± 4
4	74 ± 4
8	86 ± 3

Need glucose concentration and volume to determine dialysate glucose administered.

2. Diagram estimate – Mactier



Intraperitoneal glucose concentrations (mean ± SEM) during four hour exchanges in 18 CAPD patients using 2L of 2.5% dextrose dialysis solution.

Percentage of glucose absorbed is based on identifying the dwell time and comparing glucose (mg/dL) level remaining to the starting glucose. The glucose lost represents glucose absorbed.

3. Must reduce parenteral/enteral calorie intake by added dialysis calories to prevent overfeeding.

Reprinted with permission from Macmillan Publishers Ltd: Wideroe TE, Sneby LC, Berg KJ, et al. Intraperitoneal insulin absorption during intermittent and continuous peritoneal dialysis. *Kidney International* 1983;23:22–28.

RF. Some CRRT solutions do contain a small amount of glucose, and a substantial glucose load can be delivered with replacement intravenous (IV) fluids (up to 20 L/d) given when fluid replacement is necessary. Hyperglycemia and inappropriate weight gain can occur if the glucose amount received during PD and CRRT is not considered. During HD, approximately 30 g of glucose is delivered during each HD dialysis session and usually is not considered when determining a nutrition support regimen.

Protein

Low-protein diets (0.6 to 0.8 g/kg) have traditionally been used in RF to reduce uremic symptoms, slow the progression of renal dysfunction, and obviate or delay the need for dialysis.[10,13,16,21] Although protein restriction can slow the progression of renal dysfunction in some subgroups of patients with CKD, there is no evidence that these diets provide any benefit in AKI. In fact, patients with AKI should be fed the amount of protein and calories needed by their overall condition. Very-low-protein diets, which only contain essential amino acids (EAAs) or a combination of EAA and keto-acids, have not been shown to provide any advantage over conventional protein restriction except in very small, limited studies of highly motivated patients and a single group of nephrologists. Patients with AKI and CRF and receiving dialysis need additional protein because of losses that occur through the dialysate.[10,21] Recommended protein requirements for patients receiving HD and PD are 1.2 to 1.3 g/kg.[10,16,21,37] The recommendations are outlined in Table 29-6. These are recommendations for well-nourished, stable, unstressed patients. Patients on CRRT should receive at least 1 g/kg/d of protein and may need as much as 2.5 g/kg/d.[16,34,37]

The use of amino acids designed for AKI has been controversial.[40] A preliminary study by Abel and colleagues in patients with AKI suggested that a parenteral solution of EAA and hypertonic dextrose could reduce the need for dialysis.[44] Lower serum concentrations of potassium and phosphorus in these patients suggested that protein synthesis was increased. A subsequent randomized controlled trial in hospitalized patients with AKI compared a similar formulation to hypertonic dextrose alone and found the EAA solution improved recovery of renal function, but not overall survival.[18] Following this study, Feinstein and colleagues compared a parenteral solution containing EAA to a mixture of EAA and nonessential amino acids and showed no difference in the rate or frequency of recovery

from AKI or survival.[45] None of the patients was able to attain a positive nitrogen balance in this study, which provided up to 42 g of amino acids/d. A subsequent study with a mixture of EAA and amino acids that provided 82 g of amino acids/d was unable to demonstrate a positive nitrogen balance.[46] Others have shown no advantage of EAA over standard amino acids (SAAs) in AKI.[47] Formulations providing only EAA are not recommended.[40] Finally, BCAAs have demonstrated no advantage over SAA preparations in patients with AKI. Provision of these formulas for longer than 3 weeks and in larger amounts is not advised because serum essential and conditionally EAA imbalances may occur.[48,49] The nonprotein caloric intake may need to be as high as 150 to 250 kcal/g of nitrogen to promote protein anabolism. It is now recommended to provide standard mixed amino acid formulations for AKI patients.[16]

Fluid and Sodium Requirements

Fluid status must be closely monitored in all patients with RF. Urine, gastrointestinal (GI), and insensible losses should be considered when determining fluid requirements. Typical adults require 1200 to 2500 mL/d to maintain fluid balance. The need for fluid restriction often depends on the presence of oliguria or anuria. Patients who are anuric, defined as urine output of < 75 mL/d, are typically fluid restricted to 1000 to 1200 mL/d, which reflects usual insensible water losses/day.[2] Somewhat larger volumes of fluid may be tolerated in patients with oliguria, which is defined as urine output < 400 mL/d.[2] Concentrated EN or PN solutions are often required to meet these restrictions. Concentrated formulas may not be necessary in nonoliguric patients or those receiving RRT.[12,14] RF oliguria often leads to a decrease in urine sodium and potassium losses. In general, sodium or water should not be restricted in patients with nonoliguric AKI unless there is evidence of volume overload or hyponatremia. Patients with oliguric AKI will require sodium restriction once normovolemia has been attained. Water should be restricted only for patients with hyponatremia. Occasionally, fluid in excess of maintenance needs must be given to patients undergoing CRRT or during the diuretic phase of AKI. Fluid therapy adjustments should be determined by daily measurements of intake and output, weight, and serum sodium.

Electrolytes and Minerals

Patients with RF lose the ability to maintain normal serum concentrations of sodium, potassium, phosphate, and magnesium because of impaired excretion. Before nutrition support is instituted, tissue catabolism typically leads to elevated serum potassium, magnesium, and phosphate concentrations because the intracellular concentrations of these electrolytes are high. These electrolytes must be closely monitored during nutrition support and may need to be limited when providing enteral and parenteral solutions. If nutrition support occurs in the setting of protein anabolism, concentrations of these electrolytes may decline within the first few days of feeding (see Chapter 7). Electrolyte doses are predicated on the degree of anabolism and catabolism that must be considered when determining electrolyte requirements. In patients who are not severely malnourished or once feeding is stabilized in others, electrolyte requirements are generally low and dependent on renal and GI losses. Electrolytes (potassium, magnesium, calcium, phosphorus) should be adjusted based on the regular measurement of serum levels.[16]

RRT can be effective in clearing potassium, sodium, and magnesium. Certain dialyzer membranes will provide excellent phosphate clearance, but the effect is membrane dependent. Potassium can be added to both HD and PD solutions to maintain normal serum levels. With CRRT, electrolyte clearance may be high, and electrolyte supplementation may be necessary. Knowing the type of RRT used and the amount of fluid removed is useful in planning the solution volume, dextrose, and amino acid concentrations and additive amounts needed when formulating nutrient solutions. Table 29-6 lists the suggested ranges for electrolyte and mineral requirements at various stages of RF and types of RRT.

Calcium excretion is decreased in both AKI and CKD; however, blood levels are also affected by parathyroid hormone concentration and serum phosphate levels. Serum calcium does not increase because it becomes bound to phosphate, which is frequently elevated in RF. Serum calcium may decline slightly, but the decrease is great enough to result in increased parathyroid hormone levels. Under normal circumstances, an increase in serum parathyroid hormone levels leads to calcium release from bone, increased renal phosphate clearance, and increased conversion of 25-hydroxyvitamin D_3 to 1,25-dihydroxyvitamin D_3.[50] The active metabolite of vitamin D promotes calcium absorption from the gut. In CKD, renal phosphate clearance is suboptimal, and the activity of 1-a-hydroxylase is diminished. This leads to elevated serum phosphate concentrations and lower than expected levels of 1,25-dihydroxyvitamin D_3.

Derangements in mineral and bone metabolism common to CKD were found to be associated with increased morbidity and mortality. This prompted the development of *Clinical Practice Guidelines for Bone Metabolism and Disease in Chronic Kidney Disease* by the Kidney Disease Quality Outcomes Initiative.[51] The guidelines provide a framework for management of this complex problem. Recommendations are included for the evaluation of serum levels of phosphorus, calcium, and plasma; intact parathyroid hormone (PTH); management and/or treatment with vitamin D; phosphate binders; and dialysate bath. Serum levels of phosphorus, corrected calcium, and plasma PTH should be monitored with guideline recommendations implemented, where applicable, in patients with CKD requiring nutrition support.[17]

Guidelines for Phosphorus Evaluation and Management

The restriction of phosphorus to 800 to 1200 mg/d is based on either elevated serum phosphorus or plasma intact PTH levels. In Stage 3 and 4 CKD, phosphorus restriction is advised for patients with a serum phosphorus > 4.6 mg/dL and > 5.5 mg/dL for patients with Stage 5 CKD.[25,52] The phosphorus restriction is also advised for plasma intact PTH levels > 70 pg/mL for patients with Stage 3 CKD, > 110 pg/mL for patients with Stage 4 CKD, and > 300 pg/mL for patients with Stage 5 CKD. Further

management of elevated serum phosphorus often includes calcium-, noncalcium-, or nonaluminum-based phosphate binders.[51,53] Calcium-based binders include calcium carbonate, calcium acetate, and calcium citrate. All of these agents are effective at reducing hyperphosphatemia while also being a good source of calcium for patients with CKD.[54] Calcium citrate should only be used in patients who are not taking or who are not exposed to aluminum salts including the use of aluminum cookware. Noncalcium, nonaluminum binders, such as magnesium carbonate and sevelamer, are also effective and are used in adults when there is a concern for soft-tissue calcification.[54]

Guidelines for Calcium Evaluation and Management

In CKD, the total amount of elemental calcium should not exceed 2500 mg/d, which includes both nutritional intake and medications (calcium-based binders). Hypercalcemia, as evidenced by a corrected calcium level > 10.2 for patients with Stage 5 CKD, may be treated by several measures, including decrease in total calcium provided (nutrition provision and medication adjustments), decrease or discontinuation of vitamin D preparations, and/or a decrease in calcium dialysate. Low calcium levels (< 8.4 mg/dL) should be corrected for those patients exhibiting clinical symptoms.[51,53]

Acid–Base Balance

Acidosis (see Chapter 7) occurs in RF because of loss of normal acid excretion or loss of bicarbonate.[12] Under normal circumstances, the body generates 1 mEq of acid per kg of body weight each day as a result of the metabolism of a normal diet. Inflammation with its associated catabolism coupled with an excessive dietary protein intake may lead to the development of a metabolic acidosis in patients with advanced CKD. This metabolic acidosis can lead to further protein breakdown, bone reabsorption, and a decrease in the responsiveness of adrenergic beta-receptors. Bicarbonate therapy is recommended for patients with CKD and patients with ESRD with a serum bicarbonate < 22 mEq/L or in acutely ill patients with a bicarbonate < 10 to 15 mEq/L.

Sodium bicarbonate can be given intravenously when the enteral route is not available. Bicarbonate forms an insoluble precipitate with calcium in PN solutions. Acetate is an acceptable salt to be used with PN solutions. A dialysate with a 40 mEq/L bicarbonate concentration is frequently used for HD. Oral citrate use in patients with CKD and ESRD is not recommended because it can markedly increase GI aluminum absorption and cause aluminum toxicity.[2]

Vitamins

Vitamin requirements are not well established for patients with AKI or CKD.[14] Generally, vitamins are supplemented in amounts that provide the dietary reference intake (DRI). Water-soluble vitamins are recommended for all dialysis patients.[37,40] On occasion, additional vitamins may be required depending on the patient's clinical condition and RRT use. Recommendations are outlined in Table 29-6.

Water-Soluble Vitamins

In the past, daily administration of water-soluble vitamins to patients with CKD was considered unnecessary because they are excreted by the kidney and were believed to be retained in CKD. However, it was soon learned that vitamin D and water-soluble vitamins are deficient in CKD. Although the exact requirements for water-soluble vitamins in RF are unknown, the DRI amounts for normal persons are recommended to be given daily to patients with CKD and ESRD. Water-soluble vitamins are not toxic, and there is an increase in clearance of these compounds with RRT. Requirements for these vitamins are met when adequate amounts of enteral tube feeding are given and with standard parenteral multiple vitamin preparations. A water-soluble vitamin supplement should be given to patients on renal-restricted diets. Additional vitamins may be needed in dialysis patients because of loss in the dialysate in combination with poor intake and altered metabolism. Vitamins A, D, and cyanocobalamin (B_{12}) are protein bound and are removed with HD.[55] Other water-soluble vitamins lost during HD include vitamin C, folic acid, thiamin (B_1), and pyridoxine (B_6).[56] CRRT vitamin requirements tend to mirror those needed in HD.

Vitamin C losses may be significant with suppression of white blood cell phagocytosis activity being observed in both HD and PD.[57,58] Amounts of 125 mg/d have been recommended for adults receiving RRT.[56,59] However, doses > 200 mg/d have led to elevated blood oxalate levels, which can result in deposition of oxalate in the heart, kidney, and blood vessels, and precipitate acute pseudogout.[40,59]

Some authors recommend folate supplementation because serum folate levels have been found to be low in patients with CKD receiving HD or CAPD, and the concentration of folate in HD and CAPD dialysis effluent has been found to be high. However, actual tissue levels may not be low because normal concentrations have been found in white and red blood cells.[60] Therefore, the need to supplement folic acid has remained controversial in renal disease. The elderly patient with AKI is at higher risk for folate deficiency.[13] If folate is measured, it should be measured as red blood cell folate, which is more representative of body stores.

Pyridoxine (B_6) has been studied extensively in HD. Normal levels have been reported in granulocytes and serum, but reduced enzyme activity suggests a functional deficiency.[55] Patients on CAPD have also been shown to have reduced enzyme activity. In addition, reduced pyridoxine (B_6) activity can lead to increased oxalate formation.[61] Pyridoxine supplementation of 5 mg/d or 50 mg three times a week has been advised in patients on CAPD, but this remains controversial. Elderly patients with AKI are at higher risk for a pyridoxine deficiency.[13]

Thiamin may be deficient in patients on HD; however, supplementation does not seem to improve enzyme activity in HD. Thiamin losses have been reported in CAPD.[62] Supplementation of riboflavin (B_2), biotin, niacin (B_3), and pantothenic acid does not seem necessary because these vitamins are not cleared by HD.[62] Similar data on these vitamins is not available for CAPD. Normal vitamin levels appear to be maintained in CAPD patients with AKI who received a standard water-soluble vitamin preparation and an appropriate diet.

Fat-Soluble Vitamins

Fat-soluble vitamin excesses or deficiencies should not pose a problem during AKI because of the short-term nature of this illness. Therefore, DRI guidelines should be followed for AKI. Standard enteral formulas and parenteral multiple vitamin preparations are adequate for most patients with AKI and CKD.[17] Vitamin K should be given in sufficient amounts each week as with any other patient on parenteral solutions.[8]

Patients with CKD often need 1,25-dihydroxyvitamin D_3, the most active metabolite of vitamin D metabolism, to maintain normal calcium homeostasis and prevent osteomalacia.[40,50] Active vitamin D therapy is used for the prevention or treatment of vitamin D insufficiency and vitamin D deficiency in CKD. Provision of vitamin D is integrated with serum calcium, phosphorus, and PTH levels. Indications for vitamin D therapy in CKD include serum levels of 25-dihydroxyvitamin D < 30 ng/mL; indications for elevations in plasma intact PTH levels include > 70 pg/mL for Stage 3 CKD, > 110 pg/mL for Stage 4 CKD, and > 300 pg/mL for Stage 5 CKD. Detailed discussion of bone disease management and algorithms for the integration of vitamin D dosing with corrected calcium levels, phosphorus levels, and plasma intact PTH levels are available within the Kidney Disease Quality Outcomes Initiative guideline document.[51]

Vitamin A status should be carefully monitored in patients with CKD, especially patients with ESRD on dialysis.[37] Supplements should not be provided if RRT is not used because excessive amounts can lead to toxicity.[37] If signs of hypervitaminosis A, such as visual disturbances, abnormal liver function tests, irritability, and fatigue occur, fat-soluble vitamins may need to be withheld.[13]

Trace Elements

Trace elements are reviewed in Chapter 8. Trace mineral requirements in RF are not well established.[14] Some trace elements (zinc, selenium, chromium, and iodine) are normally excreted by the kidneys.[63] Excess accumulation of trace elements is unlikely because losses also occur through the GI tract.[64] Commercially prepared enteral formulas and parenteral trace element solutions are generally well tolerated because they provide normal amounts of these substances. Because trace elements are protein-bound, one would imagine that supplementation would be required in RRT; however, this does not seem to be the case.[65] Of interest is recent application of inductively coupled plasma-mass spectrometry techniques in evaluations of Cu, Se, Zn, Mn, and Ni in a large group of patients on HD and age-matched controls. Patients on HD were found to have significantly lower concentrations of Se, Zn, and Mn and significantly higher Ni when compared to controls. Further research work employing these techniques may give insight on trace element imbalances and clinical monitoring needs.

Zinc

Low serum zinc levels have been demonstrated in patients receiving PD.[64] Low zinc levels are most likely the result of compartmental shifts from plasma to the intracellular space. Although patients with symptomatic zinc deficiency have reported improved taste acuity and improved appetite following supplementation, more information is required before routine zinc supplementation can be recommended prophylactically for all patients on PD.[13]

Trace Elements in Renal Replacement Therapy

Aluminum

Aluminum toxicity can occur in patients with Stage 4 and Stage 5 CKD with or without RRT.[13,51] Aluminum excess can lead to osteodystrophy, anemia, and encephalopathy. The best method for management of aluminum excess is to restrict its intake. The frequency of toxicity is lower now that dialysate solutions are commercially prepared with water devoid of aluminum, and PN solutions do not contain protein hydrolysates. Furthermore, aluminum-based phosphate binders have been replaced with calcium and noncalcium-based binders. One potential venue for aluminum accumulation is the use of PN vitamin preparations. Prudence in product selection and proper plasma monitoring of aluminum levels may be warranted. Awareness of diagnostic procedures to identify syndromes of aluminum toxicity is necessary. Detail of the features, causes, and considerations of therapy for aluminum toxicity may be found in the *Clinical Practice Guidelines for Bone Metabolism and Disease in Chronic Kidney Disease.*[51]

Iron

Many patients with CKD and ESRD have iron deficiency anemia because of decreased red blood cell survival, GI blood loss from angiodysplasia, bone marrow toxicity caused by elevated serum PTH levels, diminished oral intake, and losses through repeated laboratory testing of blood (may range from 30 to 90 mL/month). Iron status should be assessed by measuring serum iron, TIBC, iron saturation, and ferritin.[66,67] Ferritin levels must be interpreted with caution because many patients have chronic low-grade inflammation, which can cause falsely elevated values.[67,68] Serum transferrin is less reliable in patients with CKD because of metabolic abnormalities such as malnutrition and altered protein metabolism seen in RF.[69] DRI amounts of iron should be provided to patients with mild CRF. In ESRD, iron dosing should be sufficient to maintain adequate ferritin and iron saturation.[68,69] Iron is also necessary to ensure the effectiveness of erythropoietin (EPO), a hormone given to patients with CRF to improve red cell production and reduce anemia.[67,68] Iron supplementation is recommended if serum ferritin levels are < 100 ng/dL. The goal of therapy is a serum ferritin level > 100 ng/dL and a transferrin saturation > 20%.[70] EPO will be ineffective with a serum ferritin level < 100 ng/dL and a transferrin saturation < 20%. Iron should be held if serum ferritin levels exceed 500 to 800 ng/dL.[67,69,70] Excess iron can exacerbate sepsis by potentiating the growth of certain bacteria and inhibiting neutrophil phagocytic activity. The goal of iron therapy in adults is to maintain the serum hemoglobin at 10 to 11 g/dL or hematocrit at 30% to 33%.[69,70] Iron supplementation frequently is not necessary for patients with AKI or for those receiving CRRT because they often receive blood transfusions during the critical phase of their illness.

HD may significantly affect iron status because of the blood loss in the dialyzer circuit and catheter, anticoagulant-related blood loss, and increased fragility of the red blood cells.[69,71] Patients with ESRD receiving PD may also require supplemental iron, although it does not seem to be lost in the PD dialysate.[67,69] Oral iron is generally preferred if tolerated because of the potential complications associated with intravenous iron. Iron sulfate and other iron salts are available as tablets or as a liquid for oral/enteral therapy. However, these patients frequently receive calcium salts, phosphate binders, proton pump inhibitors, and other medications that might impair iron absorption. If EPO is being used, oral iron usually cannot replete and maintain the body's iron stores of patients with CKD and on hemodialysis.[70,71] Iron dextran is given parenterally and may be used if oral therapy is not successful.[69] Intravenous iron dextran has the potential for inciting an anaphylactic reaction, and thus a test dose must always be given before regular dosing begins.[69] Newer forms of intravenous iron, such as iron sucrose and sodium ferric gluconate complex, are associated with less risk of life-threatening adverse reactions such as hypotension and anaphylaxis. However, multiple investigations report conflicting results. Chertow retrospectively reviewed 1981 adverse drug events reported to the U.S. Food and Drug Administration.[71] This study concluded that parenteral iron-related adverse drug events are rare (< 0.01%) overall. The adverse drug event rate was highest in the ferric gluconate (Ferrlecit™) and high-molecular-weight dextran formulation group as compared to low-molecular-weight iron dextran group such as InFed™. Injectable iron can be given during HD three times a week, intravenously or intramuscularly, at varying intervals.[67] Iron may need to be withheld when sepsis is suspected or documented because of its potential detrimental effect on the control of infection.[64,69]

Carnitine

Serum carnitine levels may be reduced in dialysis because carnitine seems to be lost in HD and PD.[40,72] Carnitine deficiency can lead to intradialytic hypotension, cardiomyopathy, skeletal muscle weakness, and anemia.[73] The mechanism by which carnitine deficiency contributes to anemia or hyporesponsiveness to EPO is unknown. Patients with CKD and on hemodialysis have shown improved exercise tolerance and well-being with carnitine supplementation.[74] Only intravenous carnitine is recommended for use for anemia treatment in patients with CKD and on HD.[73] Oral carnitine is not recommended for EPO-treated patients with CKD and on HD because of its limited bioavailability and lack of demonstrated efficacy.[71,75] The NKF recommends carnitine supplementation only in patients on HD who are unable to maintain a target hemoglobin of 11 to 12 g/dL or hematocrit of 33% to 36%, and who receive > 300 units per week of EPO and have adequate iron stores with no other known cause of anemia.[75] This recommendation has not been changed since hemoglobin targets for EPO therapy have been changed to 10 to 11 g/dL. The NKF recommendations would dose the carnitine at 20 mg/kg IV after each dialysis.

Nutrition Therapy

Oral Diets

A typical diet for a patient with RF generally restricts protein and phosphorus and sometimes sodium and potassium.[20] Restricted intake of meat, dairy, and egg products reduces protein, acid, and phosphorus intake. Phosphorus in adult oral diets should be restricted to 800 to 1200 mg/d. Potassium restriction includes limiting fruits and fruit juices, which are concentrated sources of potassium. Fluid restriction should be based on weight changes, fluid status, and urine output. Patients with a Cr clearance < 25 mL/min may benefit slightly from very-low-protein diets (0.28 g/kg/d), but this diet is difficult to follow and requires close monitoring for nutritional safety. In patients with a Cr clearance < 10 mL/min, a very-low-protein diet, as compared with a low-protein diet, did not significantly slow the progression of RF, but uremic symptoms were ameliorated.[76] Initially, in patients with CKD, protein intake is limited to 0.6 to 0.8 g/kg/d.[20] Intense nutrition intervention early in CKD reduces the risk of diabetes, hypertension, anemia, and dyslipidemias, and may delay the progression of renal dysfunction in some patients.[20]

Enteral Nutrition

Guidelines for nutritional support of patients with CKD receiving dialysis recommend a diet rich in protein (1.0 to 1.2 g/kg body weight/d) and low in phosphate (< 1200 to 1500 mg/d).[23] Diets high in energy and protein and standard enteral formulas tend to deliver a substantial amount of fluid, electrolytes, and phosphorus. Disease-specific formulas for enteral feeding including oral supplementation in patients with CKD were developed with a 2 kcal/mL energy provision and lower potassium, phosphorus, and sodium concentrations. A meta-analysis of 18 studies with a total of 541 patients was conducted to determine the potential benefits of EN in patients with CKD on dialysis.[23] Nutritional measures such as albumin, prealbumin, dietary intake, electrolyte status, and quality of life were evaluated. The use of supplemental oral intake did not affect electrolyte status. The study was unable to demonstrate an improvement in quality of life or clinical outcomes. Although the study group was too small to distinguish a difference between feeding with a disease-specific formula and a standard formula, an increased serum albumin and total dietary intake with the use of supplements was demonstrated.[23]

Disease-specific enteral formulas designed for patients with CKD and not on dialysis are usually not necessary for patients with CRRT. This is because the large volume of fluid and electrolytes removed by CRRT reduces the need for fluid restriction and increases the need for increased electrolyte supplementation. Because of the hypercatabolism seen with AKI, standard formulas may be a better option unless the patient is fluid restricted. Immune-enhancing formulas have been suggested for patients with AKI on CRRT but, because of the lack of controlled data, they are not recommended.[34] Patients who are receiving HD, PD, or CRRTs should not have their protein, energy, or fluid intake restricted, and these should be managed by increasing the amount of dialytic or filtration therapy delivered to the patient (Practice Scenario 29-1).

Practice Scenario 29-1

Question: What is the best approach to nutrition support in a hospitalized malnourished patient on HD?

Scenario: MR is a 66-year-old woman who has been admitted with a diagnosis of a left lower lobe pneumonia and acute respiratory failure with hypoxemia. She has been on HD for 6 years. Over the last 9 months, she has had multiple episodes of vascular access stenosis and clotting. This has resulted in the placement of central HD catheters while her vascular access has been revised. She has had four bacteremic episodes requiring intravenous antibiotics and has had recurring *Clostridium difficile* enterocolitis over this time. She has lost 10.2 kg body weight and now weighs 60 kg. Her last three monthly urea reduction ratios have been 55%, 50%, and 57% (adequate dialysis urea reduction ratio > 65%). The renal dietitian has documented inadequate energy and protein intake for the last 2 months. Her serum albumin has fallen from 3.5 g/dL 3 months ago to 2.2 g/dL today. Her oral intake over the last month has been one can of Glucerna™ daily and part of a tuna sandwich and some cookies daily. Physical examination shows loss of temporal, masseter, and interosseous muscles. She has little subcutaneous fat. She has an infected arteriovenous polytetrafluoroethylene graft in the right upper arm and a left subclavian HD catheter. Her abdomen is soft with good bowel sounds. Her BUN is 118 mg/dL, Cr is 3.4 mg/dL, calcium 7.8 mg/dL, potassium 3.2 mEq/L, and phosphorus 3.2 mg/dL. Her white blood cell count is elevated with a left shift, and blood cultures are positive at 12 hours for gram-positive cocci in pairs. She is placed on broad spectrum antibiotics. She is also committed to daily HD that represents a 25% increase over her outpatient procedures.

Intervention: The patient was started on a standard enteral formula via nasoenteric feeding tube with precautions taken to prevent refeeding syndrome.

Rationale: The patient has been malnourished for the past 3 months. This is a complication of chronic underdialysis and of recurrent inflammatory illness from vascular access thrombosis, infection, and repeated surgeries. The likelihood that diet stimulants will work or that she will take adequate oral supplements is quite unlikely. IDPN and PN are not indicated as she has a functional GI tract. An enteral tube should be placed as soon as her respiratory status is stabilized. Because she will receive daily dialysis, there is little concern for the need to have a concentrated feeding solution. She is at extremely high risk of the refeeding syndrome and the use of a "renal" formula is not needed.

1.2 g/kg × 60 kg = 78 g protein/day, 78/6.25 = 12.5 g N, 150 3 12.5 g N = 1870 kcal/day. As refeeding syndrome is highly likely, the enteral feeding should be started at a slow rate and increased every 12 hours until the goal rate is reached. Stat electrolytes, including phosphorus and calcium, should be obtained 6 hours after starting the enteral feeding and should be measured frequently thereafter. Before the enteral feeding is begun, she should receive enteral thiamine because her intake predisposes her to be thiamine deficient. She should also receive oral multivitamins. After stabilization of her medical, dialysis, and nutritional status, the enteral feeding should be changed to nocturnal or postprandial, and oral intake should be encouraged. Her diet should be liberalized to a totally select diet. She will require nutritional support for several weeks.

Parenteral Nutrition

If nutritional requirements cannot fully be met with EN or if the GI tract is nonfunctional, then PN should be used.[40] Fluid restriction requires concentration of both dextrose and SAA and a restriction of sodium chloride intake. Intravenous fat emulsion is commonly used daily to meet the patient's caloric needs while controlling the amount of fluid administered. Patients on HD are fluid restricted, but CRRT may allow liberalization of the fluid restriction and can simplify the PN formula. Overall intake and output, weight changes, and renal function should be assessed daily and fluids adjusted. Electrolytes should be adjusted based on the extent of the RF, status of RRT, and other medical conditions.[13,40] Water-soluble vitamins should be given, but fat-soluble vitamins with the exception of vitamin K1 should be restricted in short-term PN (< 8 weeks). Trace element supplementation is not needed in patients with AKI who receive blood products or in those in whom oral intake is restricted < 2 weeks. If needed, trace element solution should be given once every 1 to 2 weeks. There are no data to guide this recommendation, and each center may have its own supplementation guidelines. Specifically, selenium deficiency is to be avoided. Chromium, manganese, and copper deficiency are quite unlikely to occur in short-term PN (< 6 to 8 weeks) unless the patient remains hypercatabolic for an extended period of time (eg, burn patient with multiple débridements and grafts) (Practice Scenario 29-2).

Practice Scenario 29-2

Question: How would you formulate a PN solution for a hospitalized patient with nonoliguric AKI who is not going to be starting RRT?

Scenario: A 67-year-old man had recently undergone repair of an abdominal aortic aneurysm. He was admitted to the hospital 2 days before surgery with intense abdominal pain that was unrelated to meals or physical activity. On postoperative day 4, he developed respiratory failure requiring intubation for mechanical ventilation and intravenous vasopressor support. On postoperative day 5, his urine output rapidly declined, and he became oliguric with a continued increase in the serum Cr. The nutrition support team was consulted. He was eating normally until 2 days prior to admission, and he had little intake postoperatively. His height is 175.3 cm, weight 79.3 kg, usual weight 75 kg, and ideal weight 72 kg. On postoperative day 6, his abdomen was distended and tympanic to percussion, and bowel sounds were not audible on auscultation. An abdominal x-ray showed dilated loops of small and large bowel with air–fluid levels in the small bowel. Laboratory values were as follows: potassium = 5.2 mEq/L; BUN = 38 mg/dL; Cr = 4.6 mg/dL; and albumin = 2.3 g/dL. Other laboratory values are normal. The patient remained on nothing by mouth orders.

Intervention: This patient was started on PN. The PN was fluid restricted and supplemented for a patient that is not going to be started on dialysis immediately.

Answer: This patient should be started on nutritional support. In this case, PN because of a presumed small bowel obstruction or Ogilvie syndrome. In formulating a PN solution for a patient in AKI but not receiving dialysis, volume has to be restricted. This is done by limiting the amount of sodium salts used in the formula and by maximally concentrating the PN formula. Concentrated dextrose and 20% lipid emulsion should be used. Considering his insensible fluid losses and his nasogastric output, he should receive about 1800 mL/d. Because other intravenous medications and antibiotics account for 600 mL/d, the maximal PN volume is 1200 mL/d. The Harris-Benedict formula yields an REE of 1200 kcal, and adding a stress factor of 1.3 brings the total needs to approximately 2000 kcal/d. Patients with AKI who will not undergo dialysis require 0.6 to 0.8 g/kg/d of protein intake. His initial protein requirements would be 40 to 72 g/d. Because the PN fluid must be restricted to 1200 mL per day, concentrated sources of dextrose and amino acids should be used to compound this formulation. Standard amino acids should be provided.

In starting PN, half of the estimated energy needs would be provided on the first day in order to assess tolerance to the dextrose load. The initial prescription for this patient was 40 g of amino acids, 1000 kcal as a three-in-one solution with 70% of the nonprotein kcal as dextrose and 30% as lipid. If the patient were hyperglycemic, an argument could be made to increase the lipid calories to 50% of the total. With respiratory insufficiency, the increased lipids might assist because the respiratory quotient is 0.8 compared to the respiratory quotient of 1 for carbohydrate. Electrolytes should be provided based on the serum concentrations. If the serum sodium concentration is normal, then it is appropriate to add sodium chloride sufficient to mimic 0.45% saline solution or roughly 70 to 77 mEq/L. If the patient is hypernatremic, then less sodium chloride should be used; if the patient is hyponatremic, more sodium chloride should be used. The sodium chloride concentration in the PN must be adjusted daily based on daily measurements. Reduced amounts of phosphorus and magnesium should be provided because of the elevated Cr unless the patient is hypophosphatemic or hypomagnesemic. As this patient has had no intake for over 8 days, he is at risk for refeeding syndrome with hypophosphatemia and hypokalemia. In general, provision of phosphorus at 10 mM per 1000 kcal delivered is sufficient to begin PN. Again, phosphorus concentrations should be monitored daily and if the refeeding syndrome is a consideration.

Phosphorus should be checked 6 to 8 hours after the start of PN. Although the patient has mild hyperkalemia, potassium should not be held. Potassium should be administered at 10 to 20 mEq/1000 kcal supplied. In this case, it would be prudent to add 5 mEq/1000 kcal and to measure the potassium concentration 6 to 8 hours after starting the PN, especially if the refeeding syndrome is of concern. The PN regimen should include 10 mL/d standard multivitamin injection/week and TE should not be provided until the patient has been on PN > 2 weeks and has not had any blood products. As standard MVI is not indicated, individual water-soluble vitamins should be given (folic acid 1 to 2 mg, vitamin C 100 to 125 mg, thiamine 100 mg, a B-complex) and vitamin K1 1 to 2 mg/d. Serum glucose, electrolytes, calcium, phosphorus, and magnesium should be ordered 6 hours after the start of the PN and then measured regularly according to the findings. If the patient is hyperglycemic, regular insulin should be added to the PN and increased daily as needed. In general, 6 to 10 units/1000 kcal delivered represents a safe starting point for the addition of insulin.

Rationale: This patient would benefit from nutrition support because he has not had any nutrition for more than 8 days. Patients with severe injury without adequate oral intake for 5 to 7 days should receive either EN or PN. In this particular case, PN would be the best method of nutrition support because the patient has a nonfunctional GI tract. It was decided that RRT was not required at that time because the patient's BUN was well below 100 mg/dL, fluid accumulation was mild with an increase in weight of only 4 kg since admission, and electrolyte abnormalities were only slight. The PN should be planned without dialysis. The need to fluid restrict is based on assessment of his fluid status. In this case, this patient's weight is significantly increased from his preoperative weight because of accumulation of fluids administered during surgery and fluid retention from RF. His preoperative dry weight should be used to calculate estimated protein and energy requirements. Fluid restriction is common in AKI; in this case, intravenous fluids were to be restricted to no more than 1800 mL/d because this was less than GI, urinary, and insensible fluid losses. His calorie calculation would be based on the standard of the Harris-Benedict formula, which provides the basal calorie needs of 1200 kcal.[30] With Harris-Benedict adding a stress factor of 1.3 to 1, the estimate for total energy expenditure is 2000 to 2200 kcal/d. This is consistent with the American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) guidelines of 25 to 35 kcal/kg. Calorie needs in AKI are based not on the AKI per se but on the underlying medical condition when determining the stress factor. The activity factor in a hospitalized patient is 1.1 unless the patient is very active. For patients with AKI who will not need dialysis, 0.6 to 0.8 g of protein initially is recommended. This patient will need his full requirement (1.5 to 1.8 g/kg) but may have to be tapered up to that, based on tolerance. Based on A.S.P.E.N. standards, protein restriction is initially appropriate in patients not on dialysis with a goal of preventing or delaying dialysis while still meeting protein needs. As the patient stabilizes, the protein provided should be increased as tolerated and hopefully as renal function returns. SAAs should be provided.

Starting PN at half of the estimated energy needs facilitates evaluation of tolerance to the dextrose load. The initial prescription for this patient was 40 g of amino acids: 1100 kcal as a three-in-one solution with 70% of the nonprotein kcal as dextrose and 30% as lipid. Electrolytes should be provided based on the electrolyte serum level and current renal function. Additives should include sodium and calcium as needed. Typically in renal dysfunction, reduced amounts of potassium, phosphorus, and magnesium would be provided. Electrolytes should be adjusted based on serum Cr changes, renal function, and the amount of losses. Potassium supplementation was held because of the increased serum level. As the potassium level drops due to utilization, anabolism, and improving renal function additional potassium will be needed. A standard AKI PN regimen could include 10 mL/d standard multivitamin injection and standard trace element package daily to meet basic needs. His needs allowed only specific water-soluble vitamins to be given and trace elements were held. If the patient was being dialyzed then additional supplements may have been required.

Intradialytic Parenteral Nutrition

Intradialytic PN (IDPN) has been utilized for malnourished patients with inadequate oral intake when oral/enteral supplement was not effective and in whom standard PN was not felt to be indicated.[77] The goal of this therapy is to provide additional calories and protein to the malnourished patient on HD during HD as the volume of IDPN administered can be removed immediately by increasing the ultrafiltration rate during the dialysis procedure.[78] IDPN solutions typically range from ~350 to 1000 mL/dialysis.[78–80] IDPN is comprised of dextrose, amino acids, and use of fat emulsion.[78–80] This solution generally provides ~300 to 1200 kcal and 1.2 to 1.4 g of protein/kg per HD treatment.[78–80] The delivery of increased protein will necessitate changes to assure dialysis adequacy. Acidosis as a result of increased protein provision may occur and should be corrected if observed. Glucose monitoring before, during, and after IDPN is necessary, and the addition of short-acting insulin during therapy may be necessary. The protein and lipid calories generally prevent post-IDPN hypoglycemia as long as large amounts of exogenous insulin are avoided.

Lipids are used to provide an increase in caloric intake with minimal effect on volume delivery and hyperglycemia. Although there is concern about adverse effects of lipid delivery in dialysis patients because of preexisting hyperlipidemia, possible carnitine deficiency, and possible free fatty acid toxicity, clinically these have not been reported and do not seem to occur. Optimal solution composition of IDPN has not been defined. Clinical and physical monitoring are necessary for identification and correction of substrate intolerance, electrolyte imbalance, vitamin, and trace element deficiencies.[81]

Most information on the efficacy of IDPN is from small studies or anecdotal reports.[80,82] Some studies have demonstrated improved protein synthesis, reduced proteolysis, and improved energy metabolism, as well as increased serum albumin and weight gain.[78–80] The more malnourished the patient, the greater the improvement of albumin with using IDPN.[77] Nutritional improvements in one study did not extend to patients with behavioral health issues resulting in poor nutritional intake.[72] IDPN is not recommended for routine use at this time but rather should be reserved for those patients on HD who demonstrate malnutrition not amenable to oral or daily EN. It is an expensive therapy with multiple risks that has not clearly been shown to be associated with improved quality of life or survival. It is difficult to justify the use of IDPN except under very unusual circumstances. Proper attention to acute and chronic inflammatory states, impediments to oral intake of nutritious foods, adequate dialysis, medication side effects, and other states that can cause malnutrition, and the failure of EN must be considered. Failing this, daily PN should be considered as well before settling on IDPN.

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