CHAPTER 21

Kidney Disease

KEY CONCEPTS

- Kidney disease interferes with the normal capacity of nephrons to filter the waste products of metabolism.
- Short-term kidney disease requires basic nutrition support for healing rather than dietary restriction.
- The progressive degeneration of chronic kidney disease requires dialysis treatment and nutrient modification in accordance with each individual's disease status.
- Current therapy for kidney stones depends more on basic nutrition and health support for medical treatment than on major food and nutrient restrictions.

More than 100,000 Americans are diagnosed with end-stage renal disease (ESRD) annually, and this results in 84,000 deaths per year.[1] Decreased kidney function often goes undiagnosed. A review of the National Health and Nutrition Examination Survey found that less than 6% of individuals with compromised kidney function were aware of their condition.[2] These kidney problems are costly as a result of lost work, time, pay, and quality of life.

This chapter looks at the nutrition care of people with kidney disease and primarily focuses on the extensive medical nutrition therapy (MNT) of chronic kidney disease (CKD). Dialysis extends the lives of patients with this irreversible disease; however, it does so at an emotional, physical, and financial cost.

BASIC STRUCTURE AND FUNCTION OF THE KIDNEY

Tremendous quantities of fluid (approximately 1.2 L) are filtered through the kidneys every minute. Most of this fluid is reabsorbed back into the vascular system to maintain circulating blood volume. As the blood circulates through the kidneys, these twin organs repeatedly "launder" it to monitor and maintain its quantity and quality. Indeed, the composition of various body fluids is determined not as much by what the mouth takes in as by what the kidneys keep; they are the master chemists of the internal environment.

Structures

The basic functional unit of the kidney is the nephron. Each human kidney is made up of approximately 1 million nephrons, all of which are independently capable of forming urine. Key parts of the nephron include the glomerulus and the tubules (Figure 21-1).

Glomerulus

At the head of each nephron, a cup-shaped membrane referred to as Bowman's capsule holds the entering blood capillary and its clump of smaller vessels. Within Bowman's capsule, the afferent arteriole branches into a

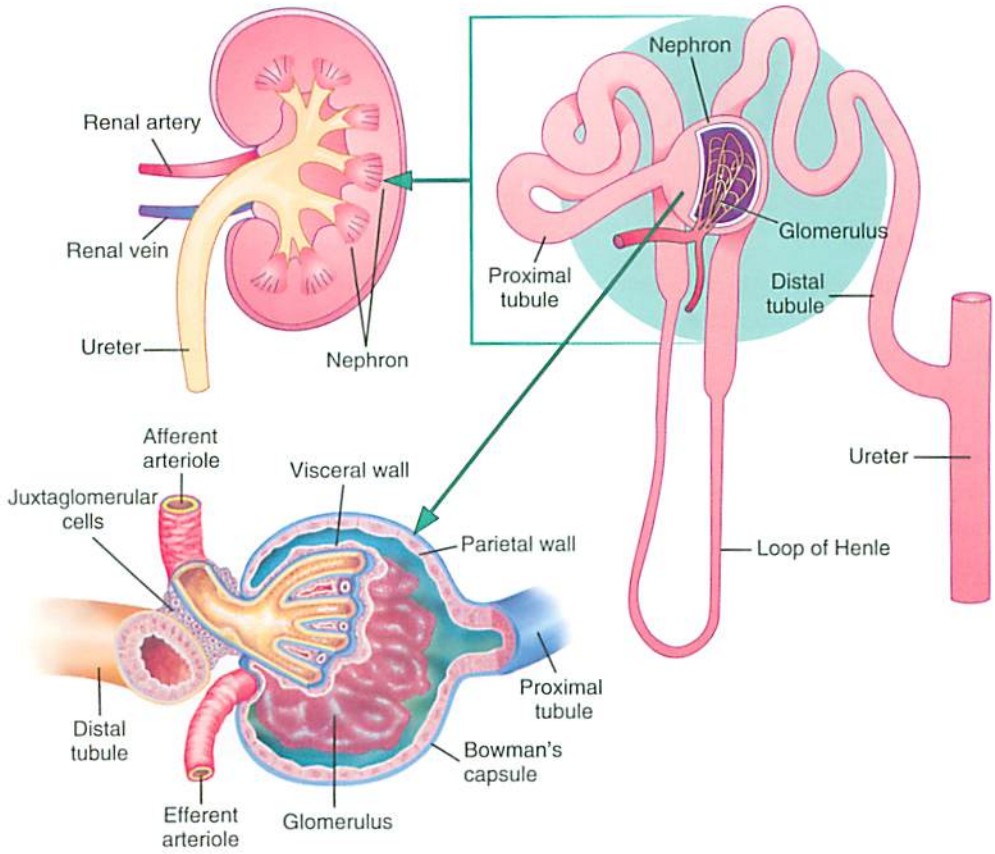

Figure 21-1 Anatomy of the kidney. (*Top*, Reprinted from Peckenpaugh NJ: *Nutrition essentials and diet therapy*, ed 10, Philadelphia, 2002; *Bottom*, reprinted from Thibodeau GA, Patton KT. *Anatomy & physiology*. 6th ed. St. Louis: Mosby; 2007.)

cluster of capillaries to form the **glomerulus** (see Figure 21-1). Only the larger blood proteins and cells remain behind in the circulating blood as it leaves the glomerulus via the efferent arteriole. The rate at which blood is filtered through the glomerulus, which is called the ***glomerular filtration rate*** **(GFR)**, is the preferred method for monitoring kidney function and for defining stages of kidney disease. CKD is defined as a GFR of less than 60 mL/min (adjusted to a standard body surface area of 1.73 m^2) for 3 or more months or a urinary albumin-to-creatinine ratio of more than 30 mg/g.[3]

Tubules

From the cupped head of each nephron, a small tubule carries the filtered fluid through its winding pathway and empties into the central area of the kidney medulla. Specific substances are reabsorbed and secreted along the way in each of the four parts of these tubules (Table 21-1).

Proximal Tubule. Most of the needed nutrients are reabsorbed in this first part of the tubule and returned to the blood. The surface area of the tubule is greatly

dialysis the process of separating crystalloids (i.e., crystal-forming substances) and colloids (i.e., glue-like substances) in solution by the difference in their rates of diffusion through a semipermeable membrane; crystalloids (e.g., blood glucose, other simple metabolites) pass through readily, and colloids (e.g., plasma proteins) pass through slowly or not at all.

nephron the functional unit of the kidney that filters and reabsorbs essential blood constituents, secretes hydrogen ions as needed to maintain the acid-base balance, reabsorbs water, and forms and excretes a concentrated urine for the elimination of wastes.

Bowman's capsule the membrane at the head of each nephron; this capsule was named for the English physician Sir William Bowman, who in 1843 first established the basis of plasma filtration and consequent urine secretion in the relationship of the blood-filled glomeruli and the filtration across the enveloping membrane.

TABLE 21-1 REABSORPTION AND SECRETION IN PARTS OF THE NEPHRON

Part	Function	Substance Moved
Proximal tubule	Reabsorption (active)	Sodium, glucose, amino acids
	Reabsorption (passive)	Chloride, phosphate, urea, water, other solutes
Loop of Henle		
Descending limb	Reabsorption (passive)	Water
	Secretion (passive)	Urea
Ascending limb	Reabsorption (active)	Sodium
	Reabsorption (passive)	Chloride
Distal tubule	Reabsorption (active)	Sodium
	Reabsorption (passive)	Chloride, other anions, water (in the presence of antidiuretic hormone)
	Secretion (passive)	Ammonia
	Secretion (active)	Potassium, hydrogen, some drugs
Collecting duct	Reabsorption (active)	Sodium
	Reabsorption (passive)	Urea, water (in the presence of antidiuretic hormone)
	Secretion (passive)	Ammonia
	Secretion (active)	Potassium, hydrogen, some drugs

From Thibodeau GA, Patton KT. *Anatomy & physiology*. 6th ed. St Louis: Mosby; 2007.

increased by a brush border membrane that contains thousands of microvilli. Glucose and amino acids as well as approximately 80% of the water and other substances are usually reabsorbed here. Approximately 20% of the filtered fluid remains to enter the next section of the tube.

Loop of Henle. The tubule's midsection narrows and dips down into the central part of the kidney. Here, the important exchange of sodium, chloride, and water occurs. This fluid environment maintains the necessary osmotic pressure to concentrate the urine as it passes through the distal tubule and ureter on its way to the bladder for elimination.

Distal Tubule. The latter part of the tubule winds back up into the outer area of the kidney cortex. Here, the secretion of hydrogen ions occurs as needed to control the acid-base balance. Sodium is also reabsorbed as needed under the influence of the adrenal hormone **aldosterone**.

Collecting Tubule. In this final section of the tubule, concentrated urine is produced by the following water-reabsorbing actions: (1) the influence of the **antidiuretic hormone**; and (2) the osmotic pressure from the more dense surrounding fluid in the central area of the kidney. The urine, which is now concentrated and ready for excretion, only amounts to 0.5% to 1% of the original fluid and materials that have been filtered through the glomerulus.

Function

Nephron structure is adapted in fine detail to balance the internal fluids that are necessary for life. At birth, each person has far more nephrons than are actually needed, but they are gradually lost with advancing age. Diabetes and long-term use of high-protein diets tend to exacerbate damage to the glomerulus and to increase the rate of lost functioning nephrons.[4]

Excretory and Regulatory Functions

The following tasks are performed while blood flows through the nephron:

- *Filtration:* Most particles in blood are filtered out, except for the larger components of red blood cells and proteins.
- *Reabsorption:* As the filtrate continues through the winding tubules, substances that the body needs are

glomerulus the first section of the nephron; a cluster of capillary loops that are cupped in the nephron head that serves as an initial filter.

glomerular filtration rate (GFR) the volume of fluid that is filtered from the renal glomerular capillaries into Bowman's capsule per unit of time; this term is used clinically as a measure of kidney function.

aldosterone a hormone of the adrenal glands that acts on the distal nephron tubule to stimulate the reabsorption of sodium in an ion exchange with potassium; the aldosterone mechanism is essentially a sodium-conserving mechanism, but it also indirectly conserves water, because water absorption follows sodium resorption.

antidiuretic hormone a hormone of the pituitary gland that acts on the distal nephron tubule to conserve water by reabsorption; also called *vasopressin*.

selectively reabsorbed and returned to the blood to maintain the electrolyte, acid-base, and fluid balances.

- *Secretion:* Along the tubules, additional hydrogen ions are secreted as needed to maintain the acid-base balance.
- *Excretion:* Waste materials are excreted in the now-concentrated urine.

Endocrine Functions

In addition to major functions in the regulation of the blood constituents and the making and excreting of concentrated urine, the kidneys perform the following other functions:

- *Renin secretion:* When the **arteriole** pressure falls, the kidneys activate and secrete renin, which is an enzyme that initiates the renin-angiotensin-aldosterone mechanism to reabsorb sodium and to maintain hormonal control of the body water balance (see Chapter 9).
- *Erythropoietin secretion:* The kidneys are responsible for producing the body's major supply (80% to 90%) of **erythropoietin**, which is a circulating hormone that is the principal factor in stimulating red blood cell production within the bone marrow in response to decreased tissue oxygen.
- *Vitamin D activation:* The kidneys convert an intermediate inactive form of vitamin D into the final active vitamin D hormone in the proximal tubules of the nephrons (see Chapter 7). This action is stimulated by the parathyroid hormone.

DISEASE PROCESS AND DIETARY CONSIDERATIONS

General Causes of Kidney Disease

Several disease conditions may interfere with the normal functioning of nephrons and result in kidney disease.

Infection and Obstruction

Symptoms of bacterial urinary tract infection may range from the mild discomfort of bladder infections to more involved chronic disease and obstruction from kidney stones. Obstruction anywhere in the urinary tract blocks drainage and may cause further infection and general tissue damage.

Damage From Other Diseases

Diabetes mellitus is the leading cause of ESRD in the United States[1] (Figure 21-2). Hyperglycemia and hypertension associated with diabetes can damage small renal

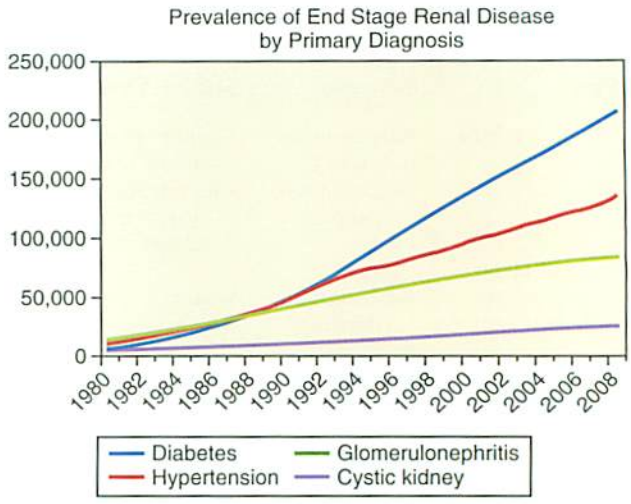

Figure 21-2 Prevalence of chronic kidney disease by primary diagnosis. (Data from the US Renal Data System. *USRDS 2010 annual data report: atlas of chronic kidney disease and end-stage renal disease in the United States*. Bethesda, Md: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases; 2010.)

arteries, thereby leading to glomerulosclerosis (i.e., the loss of functioning nephrons) and eventual CKD.[5] Circulatory disorders such as prolonged and poorly controlled hypertension can cause the degeneration of the small arteries within the kidney and interfere with normal nephron function. More than 80% of patients with advanced CKD have hypertension.[1] Increased demands on other nephrons may in turn cause further hypertension and additional damage to nephrons. Other major causes are glomerulonephritis and cystic kidney disease. Autoimmune diseases such as systemic lupus erythematosus may also lead to compromised function or kidney disease.

Toxins

Various environmental agents (e.g., chemical pesticides, solvents), animal venom, certain plants, heavy metals, and some drugs (e.g., nonsteroidal anti-inflammatory drugs, aminoglycoside antibiotics, radiographic contrast dye) are **nephrotoxic** and can cause kidney damage.

arteriole the smallest branch of an artery that connects with the capillaries.

erythropoietin hormone that stimulates the production of red blood cells in the bone marrow.

nephrotoxic poisonous to the kidney.

Genetic or Congenital Defects

Cystic diseases (e.g., polycystic kidney disease, medullary cystic disease) are genetically linked kidney diseases that often lead to ESRD later in life. Congenital abnormalities of both kidneys can contribute to kidney disease with extensive distortion of kidney structure. Individuals who are born with only one kidney do not necessarily have kidney disease or even impaired function. People with a single kidney are often unaware of the fact and usually lead full lives without compromised kidney function.

Risk Factors

Risks for CKD are higher among individuals who have diabetes, hypertension, or cardiovascular disease (CVD); who are older than 60 years old; who smoke or are obese; and who have a family history of kidney disease. Malnutrition can exacerbate the rate of renal tissue destruction and increase susceptibility to infection. The prevalence of CKD is also higher among individuals with low income or education and among racial and ethnic minority populations.[6] Box 21-1 lists risk factors and common causes of kidney disease.

BOX 21-1 RISK FACTORS AND COMMON CAUSES OF KIDNEY DISEASE

Sociodemographic Factors
- Older age
- Family history of chronic kidney disease
- Member of racial or ethnic minority group
- Exposure to certain chemical and environmental conditions
- Low income or education level

Clinical Factors
- Poor glycemic control in diabetes
- Hypertension
- Autoimmune disease
- Systemic infection
- Urinary tract infection
- Urinary stones
- Lower urinary tract obstruction
- History of acute kidney injury
- Reduction in kidney mass
- Exposure to certain nephrotoxic drugs
- Low birth weight or hereditary diseases

From Levey AS, Eckart KU, Tsukamoto Y, et al. Definition and classification of chronic kidney disease: a position statement from Kidney Disease: Improving Global Outcomes (KDIGO). *Kidney Int.* 2005;67(6):2089-2100.

Medical Nutrition Therapy in Kidney Disease

During the treatment of kidney disease, appropriate MNT is based on the severity of the disease, the GFR, the presence of metabolic abnormalities, and the medical treatment (e.g., renal replacement therapy, medications).

Length of Disease

During short-term acute disease that results from infection, drug therapy with antibiotics usually controls the disease. Nutrition therapy is aimed at optimal nutrition support for healing and normal growth. More specific nutrient modifications may be necessary if the patient is a child or if the disease progresses to become chronic.

Degree of Impaired Kidney Function and Clinical Symptoms

For milder acute disease with few nephrons involved, less interference occurs with general kidney function, because the large number of backup nephrons can meet basic needs. However, with progressive chronic disease, more and more nephrons become involved, which results in CKD. In such cases, extensive MNT is required to help maintain kidney function as long as possible. With continuing disease, nutrient modifications are designed to meet individual needs to address clinical symptoms. Working closely with a registered dietitian for personalized nutrition therapy is especially important when advanced kidney disease is treated with dialysis.

This chapter's discussion focuses primarily on the serious degenerative process of CKD and dialysis that requires MNT. Clinical practice guidelines are discussed for each type of kidney disease in the following sections.

NEPHRON DISEASES

Acute Glomerulonephritis or Nephritic Syndrome

Disease Process

This inflammatory process affects the glomeruli, which are the small blood vessels in the cupped membrane at the head of the nephron. Glomerulonephritis is one of the three most common causes of stage 5 CKD, which is also known as *ESRD.*[1]

Clinical Symptoms

Classic symptoms include **hematuria** and **proteinuria**, although **edema** and mild hypertension also may occur. These patients may experience anorexia in advanced stages, which contributes to feeding problems and

TABLE 21-2 GLOMERULAR SYNDROMES

Syndrome	Clinical Manifestations
Acute nephritic syndrome	Hematuria, azotemia, variable proteinuria, oliguria, edema, and hypertension
Rapidly progressive glomerulonephritis	Acute nephritis, proteinuria, and acute kidney failure
Nephrotic syndrome	> 3.5 g proteinuria, hypoalbuminemia, hyperlipidemia, and lipiduria
Chronic kidney failure	Azotemia and uremia that progress for years
Asymptomatic hematuria or proteinuria	Glomerular hematuria and subnephrotic proteinuria

From Kumar V, Fausto N, Abbas A. *Robbins and Cotran pathologic basis of disease*. 7th ed. Philadelphia: Saunders; 2005.

malnutrition. If the disease progresses to more kidney involvement, signs of **oliguria** or **anuria** may develop. Table 21-2 outlines the five glomerular syndromes and their respective clinical manifestations.

Medical Nutrition Therapy

Nephrologists and dietitians favor overall optimal nutrition support for growth with adequate protein. Diet modifications are not crucial in most patients with acute short-term disease. Fluid intake is adjusted to output and insensible losses.

Nephrotic Syndrome

Disease Process

Nephrotic syndrome or **nephrosis** results from nephron tissue damage to the major filtering membrane of the glomerulus, thereby allowing protein to pass into the tubule. This high protein concentration may cause further damage to the tubule. Both filtration and reabsorption functions of the nephron are disrupted. Nephrosis may be caused by infection, medications, neoplasms, preeclampsia, progressive glomerulonephritis, or diseases such as diabetes and systemic lupus erythematosus.

Clinical Symptoms

Nephrotic syndrome is characterized by a group of symptoms that result from nephron tissue damage and impaired function. The large protein losses (i.e., 3.5 g/day or more in adults) lead to hypoalbuminemia, edema, and **ascites**. The abdomen becomes distended as fluid accumulates, and the plasma protein level is greatly reduced because of losses in the urine. As protein loss continues, tissue proteins are broken down, and general malnutrition follows. Severe edema and ascites often mask the extent of body tissue wasting. Other clinical manifestations include hyperlipidemia and **lipiduria**.

Medical Nutrition Therapy

Diets with moderate amounts of protein reduce albuminuria and albumin catabolism, with no change in the GFR. Nutrition therapy is directed toward controlling major symptoms and replacing the nutrients that are lost in the urine. Current standards of care are as follows[7]:

- *Protein:* The diet is usually moderate in protein (0.8 to 1.0 g/kg of body weight/day), with at least 50% of the protein from high biologic value sources, including soy protein. Total protein intake may be modified on the basis of **blood urea nitrogen** and GFR results. If blood urea nitrogen is elevated and urine output is decreased, dietary protein may be restricted.
- *Energy:* Total energy intake should be adequate to support nutrition status. Needs may be as high as 35 kcal/kg/day. To provide sufficient energy in kilocalories, complex carbohydrates should be given liberally, which also helps to combat the catabolism of tissue protein and to prevent starvation **ketosis**.

hematuria the abnormal presence of blood in the urine.

proteinuria an abnormal excess of serum proteins (e.g., albumin) in the urine.

edema the excess accumulation of fluid in the body tissues.

oliguria the secretion of small amounts of urine in relation to fluid intake (i.e., 0.5 mL/kg per hour or less).

anuria the absence of urine production; anuria indicates kidney shutdown or failure.

nephrosis degenerative lesions of the renal tubules of the nephrons and especially of the thin basement membrane of the glomerulus that helps to support the capillary loops; marked by edema, albuminuria, and a decreased serum albumin level.

ascites the accumulation of serous fluid (i.e., blood and lymph serum) in the abdominal cavity.

lipiduria lipid droplets found in the urine that are composed mostly of cholesterol esters.

blood urea nitrogen a test of nephron function that measures the ability to filter urea nitrogen, which is a product of protein metabolism, from the blood.

ketosis the accumulation of ketones, which are intermediate products of fat metabolism, in the blood.

Decreasing the dietary intake of fat and cholesterol may help to alleviate dyslipidemia and the resulting risk for CVD. Total fat intake should not exceed 30% total kcals/day, 7% of kcals or less should come from saturated fat, cholesterol intake should not exceed 200 mg/day, trans fats should be limited, and up to 10% of kcals should come from polyunsaturated fats, including fish.

- *Sodium and potassium:* Edema is common among patients with glomerular diseases. To reduce symptoms, a sodium restriction of 1 to 2 g/day is advised to maintain the sodium and fluid balance. Sodium overload is difficult to treat because of the characteristic hypoalbuminuria and **hypotension**; therefore, careful monitoring is necessary. The renal clearance of potassium is impaired with oliguria. Thus, potassium intake must be monitored and carefully adjusted in accordance with individual needs.
- *Calcium and phosphorus:* Some calcium is bound to albumin in the blood. As albumin is lost through the tubule, bound calcium is also lost. In addition, low serum vitamin D decreases calcium absorption. Thus, the recommendations are to consume 1 to 1.5 g of calcium per day and to limit phosphorus to 12 mg/kg/day.
- *Fluid:* Fluid intake may be restricted in response to urine output and insensible losses. If restriction is not indicated, fluids can be consumed as desired.

KIDNEY FAILURE

The two types of kidney failure—acute and chronic—have a number of symptoms that reflect interference with normal nephron functions in nutrient metabolism. Both forms are addressed with similar nutrition therapy, depending on the extent of renal tissue damage and the treatment method used.

Acute Kidney Injury

Disease Process

Healthy kidneys may suddenly shut down after metabolic insult or traumatic injury, thereby causing a life-threatening situation. Baseline risk factors for the development of in-hospital acute kidney injury (also known as *acute renal failure*) include older age, diabetes, underlying renal insufficiency, and heart or liver failure.[8] This is a medical emergency in which the dietitian and the nurse play important supportive roles. Depending on the underlying cause, acute kidney injury (AKI) is divided into three categories[7]:

1. *Prerenal:* Prerenal AKI involves inadequate blood flow to the kidneys and subsequent reduced GFR. Common causes include severe dehydration, hypotension, shock, congestive heart failure, and renal vasoconstriction or occlusion.
2. *Intrinsic:* Intrinsic AKI results from damage to a part of the kidney. Common causes include hypertension, infection, acute tubular necrosis or interstitial nephritis, obstruction, or nephrotoxicity from antibiotics, antimicrobial agents, or other drugs.
3. *Postrenal obstruction:* Postrenal obstruction involves the obstruction of urine flow. Common causes include benign prostatic hypertrophy with urinary retention, cancer, ureteral stones, and other obstructions.

AKI can last from days to weeks, with normal function returning when the condition that is causing the failure is resolved. Depending on the extent of renal tissue damage, regaining full function may take months. However, some individuals do not regain normal kidney function, and the disease then progresses to CKD. Patients with a particularly high risk for advancing to CKD are those with diabetes, those of advanced age, those with low serum albumin levels, and those with severe AKI (i.e., chiefly those who require dialysis).[9]

Clinical Symptoms

AKI is classified according to the RIFLE classification system, which assesses the severity of **R**isk, **I**njury, **F**ailure, and the outcomes of either **L**oss or **E**SRD.[10] The major sign of AKI is an increase in serum **creatinine** levels and oliguria, which is caused when cellular debris from the tissue damage blocks the tubules. Diminished urine output may be accompanied by proteinuria or hematuria. Researchers are working to define more sensitive markers for diagnosing AKI, because there are weaknesses involved in the use of the traditional markers of creatinine and urine output.[11] Other symptoms include nausea, vomiting, fatigue, muscle weakness, swelling in the lower extremities, itchy skin, and confusion. Water balance also becomes a crucial factor. Continuous renal replacement therapy, which is a type of dialysis, may be needed to support kidney function.

Medical Nutrition Therapy

Basic Objectives. The major challenge during AKI is to improve or maintain nutrition status while the patient

hypotension low blood pressure.

creatinine a nitrogen-carrying product of normal tissue protein breakdown; it is excreted in the urine; serum creatinine levels are an indicator of renal function.

is faced with marked catabolism. Current standards indicate the need for highly individualized therapy that is focused on the following: (1) treating the underlying cause; (2) preventing further kidney damage and complications from nutrient deficiencies; and (3) correcting any fluid, electrolyte, or uremic abnormalities.[7] Loss of appetite is common, and enteral nutrition may be required. If enteral nutrition is contraindicated, parenteral nutrition may then be necessary (see Chapter 22).

Principles. Preventing protein catabolism, electrolyte and hydration disturbances, acidosis, and uremic toxicity through individualized MNT is thought to play a role in maintaining kidney function while reducing the complications of CVD and progressive kidney deterioration.[12] General recommendations for AKI are presented below. Keep in mind that kidney function and treatment modality may vary greatly among patients; thus, MNT should be adjusted accordingly.[7]

- *Protein:* Adequate protein is important for supporting kidney function and for preserving lean tissue. For patients who are not receiving dialysis and who are not experiencing catabolism, a protein intake of 0.8 to 1.2 g/kg is recommended. For patients who are experiencing catabolism or who are on dialysis, 1.2 to 1.5 g/kg of daily protein is recommended to allow for nutrient replenishment and to account for losses.
- *Energy:* Energy intake in the range of 25 to 35 kcal/kg is suggested. This amount needs to be adjusted on an individual basis, depending on metabolic stress and the nutritional status of the patient. If the patient is on dialysis, energy intake from the **dialysate** should be included in the total energy intake.
- *Sodium and potassium:* During a diuretic phase, patients may lose excessive electrolytes. Losses of both sodium and potassium (2 to 3 g/day each) should be replaced during this phase. These levels are further adjusted depending on blood pressure and the presence of edema.
- *Phosphate and calcium:* Dietary phosphorus intake is determined on the basis of body weight, with a range of 8 to 15 mg/kg of phosphorus. The MNT goal for calcium is to maintain serum value levels within normal limits and to adjust dietary intake accordingly.
- *Vitamins and minerals:* A patient's diet should be balanced to prevent nutrient deficiencies by meeting the Dietary Reference Intakes for all other vitamins and minerals. If the patient is experiencing catabolism or other complications, nutrient intakes may be modified to meet specific needs.
- *Fluid:* Individual fluid needs are highly variable with AKI, and treatment modality, hydration, and fluid loss should be considered. Insensible fluid loss may increase as a result of fever, and sensible fluid loss (e.g., urine output, vomit, diarrhea) will vary considerably among patients. A starting point recommendation is 500 mL of fluid plus urine output daily.

Chronic Kidney Disease

Disease Process

CKD is caused by the progressive breakdown of kidney tissue, which impairs all kidney functions. Few functioning nephrons remain and they gradually deteriorate. CKD develops slowly, and no cure exists.

CKD is most commonly a result of the following:

- Primary glomerular disease
- Metabolic diseases with kidney involvement (e.g., diabetes, hypertension, CVD)
- Inherited diseases (e.g., polycystic kidney disease, congenital abnormality)
- Other causes: immune disease such as lupus, obstructions such as kidney stones, chronic urinary tract infections, and hypertension

Modifiable risk factors include controlling blood pressure, proteinuria, or albuminuria; addressing the HbA1C level and dyslipidemia; and quitting smoking.[6,13] In its clinical practice guidelines, the National Kidney Foundation categorizes CKD into five stages on the basis of the GFR (Table 21-3).

TABLE 21-3 STAGES OF CHRONIC KIDNEY DISEASE

Stage	Description	Glomerular Filtration Rate (mL/min/1.73 m^2)
1	Kidney damage with normal or elevated GFR	≥ 90
2	Kidney damage with mild decrease in GFR	60 to 89
3	Moderate decrease in GFR	30 to 59
4	Severely decreased GFR	15 to 29
5	Kidney failure or end-stage renal disease	< 15 (or dialysis)

GFR, Glomerular filtration rate.

Chronic kidney disease is defined as either kidney damage or a glomerular filtration rate of less than 60 mL/min per 1.73 m^2 for 3 or more months. Kidney damage is defined as pathologic abnormalities or markers of damage, including abnormalities in blood or urine tests or imaging studies.

From Levey AS, Eckart KU, Tsukamoto Y, et al. Definition and classification of chronic kidney disease: a position statement from Kidney Disease: Improving Global Outcomes (KDIGO). *Kidney Int.* 2005;67(6):2089-2100.

Clinical Symptoms

Depending on the nature of the underlying kidney disease, chronic kidney changes may involve the extensive scarring of renal tissue, which distorts the kidney structure and brings vascular changes as a result of prolonged hypertension. As nephrons are lost one by one, the remaining nephrons gradually lose their ability to sustain metabolic balance.

Water Balance. During the early stages of chronic kidney failure, the kidneys are unable to reabsorb water or to properly concentrate urine. Therefore, large amounts of dilute urine are produced (i.e., polyuria). Dehydration is a risk factor at this point, and it may become critical. As the disease progresses, urine production declines to a point of oliguria and finally anuria. Without the urinary excretion of waste products, dangerous levels of urea accumulate in the blood.

Electrolyte Balance. Several imbalances among electrolytes result from decreasing nephron function. The failing kidney cannot appropriately maintain the vital sodium and potassium balance that guards body water (see Chapter 9). A concentration of materials (e.g., phosphate, sulfate, organic acids) is produced by the metabolism of nutrients. Without appropriate filtering, these materials accumulate in the blood, thereby causing metabolic acidosis. The disturbed metabolism of calcium and phosphorus, the abnormal levels of parathyroid hormone, and the lack of activated vitamin D (a process that occurs in the kidneys) leads to bone pain, abnormal bone metabolism, and **chronic kidney disease-mineral and bone disorder** or **osteodystrophy**.

Nitrogen Retention. An increasing loss of nephron function results in elevated nitrogenous metabolites such as **urea** and creatinine. Protein-energy malnutrition is a common complication of protein catabolism.

Anemia. The damaged kidney cannot accomplish its normal initiation of red blood cell production through erythropoietin. Therefore, fewer red blood cells are produced, and those that are produced have a decreased survival time.

Hypertension. When blood flow to the kidney tissues is increasingly impaired, renal hypertension develops. In turn, hypertension causes cardiovascular damage and the further deterioration of the nephrons.

Azotemia. Elevated blood urea nitrogen, serum creatinine, and serum uric acid levels are reflected in the characteristic laboratory finding of **azotemia**.

General Signs and Symptoms

Increasing loss of kidney function causes progressive weakness, shortness of breath, general lethargy, and fatigue. Thirst, anorexia, weight loss, diarrhea, and vomiting may occur. Increasing capillary fragility may cause skin, nose, oral, and gastrointestinal bleeding. Nervous system involvement brings muscular twitching, burning sensations in the extremities, and convulsions. Irregular cyclic breathing (i.e., Cheyne-Stokes respiration) indicates acidosis. Acidosis may cause mouth ulcers, a foul taste, and bad breath in the patient. Malnutrition lowers resistance to infection, and some patients may experience bone and joint pain.

Medical Nutrition Therapy

Basic Objectives. Treatment must always be individual and adjusted according to the progression of the illness, the type of treatment, and the patient's response. The Kidney Disease Outcomes Quality Initiative dietary guidelines recommend monitoring the nutrition status of patients with CKD at regular intervals: every 1 to 3 months for patients with a GFR of less than 30 mL/min per 1.73 m^2 and every 6 to 12 months for patients with a GFR of 30 to 59 mL/min per 1.73 m^2 to identify anorexia and to help prevent malnutrition.[14]

Principles. Nutrition for CKD involves several nutrient adjustments that should be made in accordance with individual need. The MNT recommendations for patients with CKD are as follows[7]:

- *Protein:* The goal is to provide adequate protein to maintain tissue integrity while avoiding excess. Protein is generally limited to 0.6 to 0.8 g/kg/day for individuals who are not on dialysis with a GFR of less than 50 mL/min per 1.73 m^2. Patients with GFRs of

dialysate the cleansing solution used in dialysis; contains dextrose and other chemicals similar to those in the body.

chronic kidney disease-mineral and bone disorder a clinical syndrome that develops as a systemic disorder of mineral and bone metabolism in patients with chronic kidney disease; results from abnormalities of calcium, phosphorus, parathyroid hormone, or vitamin D metabolism; causes abnormalities in bone turnover, mineralization, volume, linear growth, strength, and soft-tissue calcification.

osteodystrophy an alteration of bone morphology found in patients with chronic kidney disease.

urea the chief nitrogen-carrying product of dietary protein metabolism; urea appears in the blood, lymph, and urine.

azotemia an excess of urea and other nitrogenous substances in the blood.

less than 20 mL/min per 1.73 m^2 may be limited to dietary protein of 0.3 to 0.5 g/kg/day with additional keto acid analogs (i.e., nitrogen-free copies of essential amino acids) to meet protein requirements.[15] At least 50% of this amount should come from high biologic value protein (see Chapter 4) to ensure an adequate intake of essential amino acids.

- *Energy:* Carbohydrate and fat must provide sufficient nonprotein kilocalories to supply energy and spare protein for tissue synthesis. For individuals who are younger than 60 years old with CKD and GFRs of less than 25 mL/min per 1.73 m^2, the recommended energy intake is 35 kcal/kg/day. Energy needs are less for individuals who are 60 years old or older (30 to 35 kcal/kg/day). Because cardiovascular disease is accelerated in patients with CKD, the remaining calories should support cardiovascular health principles (e.g., substitute monounsaturated and polyunsaturated fats for saturated and trans fats, reduce total cholesterol intake; see Chapter 19).
- *Sodium and potassium:* If hypertension and edema are present, sodium intake must be restricted. Sodium intake is usually limited to 1 to 3 g/day. As CKD advances, potassium is not cleared adequately from the blood. Dietary intake is determined by assessing laboratory values. If blood levels of potassium are elevated and other nondietary causes are eliminated, then a potassium-restricted diet may be indicated.
- *Phosphorus and calcium:* Inappropriate blood phosphorus and calcium levels negatively affect bone composition. As the kidney loses function, the activation of vitamin D and the control of blood calcium levels decline. This problem is worsened by excess blood phosphorus, which results in calcium resorption from the bone. Thus, moderate dietary phosphorus restriction depends on laboratory values in the patient who is not undergoing dialysis, and it is generally limited to 800 to 1000 mg/day when serum phosphorus is 4.6 mg/dL or more or when the parathyroid hormone level is elevated. Calcium recommendations are 1.0 to 1.5 g/day and not to exceed 2 g/day, including both dietary and supplemental calcium sources.
- *Vitamins and minerals:* A protein-restricted diet does not contribute the full daily requirement of all essential nutrients (review the Clinical Applications box, "Case Study: A Patient with CKD"). Supplemental fat-soluble vitamins A and E are not recommended, because they may accumulate to toxic levels in patients with kidney failure. Excesses of vitamins D and K are contraindicated, because the kidney cannot convert vitamin D to its active form, and vitamin K can adversely affect clotting time. The specific MNT recommendations are to help patients meet their Dietary Reference Intakes for the B-complex vitamins and vitamin C and to determine the patient-specific needs for vitamin D, iron, and zinc.
- *Fluid:* Fluid intake should be sufficient to maintain adequate urine volume in patients who are not undergoing dialysis. Intake usually is balanced with output, and it is not otherwise restricted.

End-Stage Renal Disease

Disease Process

When CKD advances to its end stages, life-support decisions face the patient, the family, and the physician. ESRD occurs when the patient's GFR decreases to less than 15 mL/min per 1.73 m^2. This decrease is caused by irreversible damage to a majority of the kidneys' nephrons. At this point, the patient has two options: long-term kidney dialysis or kidney transplant. The lives of an estimated 500,000 people in the United States are prolonged by dialysis and kidney transplants annually.[1] Dialysis is the chief treatment for ESRD.

Two forms of dialysis are used: hemodialysis and peritoneal dialysis. For a thorough understanding of the treatment options that are available for ESRD, please refer to the article listed in the "Further Reading and Resources" section at the end of this chapter.

Treatment Options and Respective Medical Nutrition Therapy

Hemodialysis. Hemodialysis is the use of an "artificial kidney machine" to remove toxic substances from the blood and to restore nutrients and metabolites to normal blood levels (Figure 21-3). To prepare a patient for hemodialysis therapy, vascular access must be established. This procedure ideally takes place 4 to 16 weeks before treatments begin to allow for adequate healing. The three basic kinds of vascular access for hemodialysis are arteriovenous fistula, arteriovenous graft, and a venous catheter (Figure 21-4). An arteriovenous fistula is the preferred access for long-term dialysis, and it is made by joining an artery and a vein on the forearm just beneath the skin.[16] After the fistula has healed, a cannula (i.e., a large-bore needle) is inserted through the tissue and connected by tubes to the dialysis machine.

A patient with ESRD who is on hemodialysis usually receives three treatments per week, each of which lasts 3

CLINICAL APPLICATIONS

CASE STUDY: A PATIENT WITH CHRONIC KIDNEY DISEASE

Gary, who is 49 years old, is an active man who works at a large company and has begun to tire more easily. He has little appetite, and he generally feels ill most of the time. He recently noticed some ankle swelling and blood in his urine. At his family's insistence, he finally decided to see his physician.

After a complete workup, the physician's findings included the following:

- No prior illness except a case of the flu with a throat infection during his overseas service in the Army
- Laboratory tests: presence of albumin, red blood cells, and white blood cells in the urine; high blood potassium, phosphorus, creatinine, and urea levels; and low glomerular filtration rate of 20 mL/min per 1.73 m^2
- Other symptoms: hypertension, edema in the lower legs, headache, occasional vision blurring, and low-grade fever

The physician discussed the findings and the serious prognosis of stage 4 chronic kidney disease with Gary and his wife. Together with the dietitian, they explored the immediate medical and nutrition needs for treatment. They also discussed the ultimate need for medical management with dialysis or transplantation. The physician prescribed medications to control Gary's growing symptoms and discomfort.

Over the next 10 months, Gary's symptoms worsened. He lost more weight, became anemic, and had increased bone and joint pain and gastrointestinal bleeding. Nausea increased, and he had occasional muscle twitching and spasms. Small mouth ulcers made eating a painful effort. Gary and his wife made an appointment with their dietitian to learn how to manage his present predialysis diet at home.

Questions for Analysis

1. What metabolic imbalances in chronic kidney disease do you think account for Gary's symptoms?
2. What are the objectives of the treatment of chronic kidney disease?
3. What are the basic principles of Gary's predialysis diet? Describe this type of diet. What foods would be included? Plan a 1-day menu for Gary with the use of the dietary analysis program that is included with this text.

to 4 hours. However, a recent study found that patients who were receiving hemodialysis up to six times per week with shorter sessions (i.e., about 2.5 hrs/each) experienced significant health benefits with no reported loss in quality of life as a result of the frequency of treatments. However, the patients who received frequent hemodialysis in this study did require more interventions related to vascular access.[17]

During each treatment, the patient's blood makes several complete cycles through the dialyzer, which removes excess waste to maintain normal blood levels of life-sustaining substances, a function the patient's own kidneys can no longer accomplish. Two compartments in the machine are separated by a filter. One compartment contains blood from the patient with all of the excess fluids and waste; the other contains the dialysate, which is a type of "cleaning fluid." As during normal capillary filtration, the blood cells are too large to pass through the pores in the filter. However, the remaining smaller molecules in the blood pass through the filter and are carried away by the dialysate. If the patient's blood is deficient in certain nutrients, these may be added to the dialysate.

Medical Nutrition Therapy for Hemodialysis. The diet of a patient who is undergoing hemodialysis is an important aspect of maintaining biochemical control. Registered dietitians who specialize in renal care are heavily involved with meal planning and diet education. The goal of the nutrition therapy during hemodialysis is to maintain optimal nutrition while preventing the buildup of waste products in between treatments. In most cases, MNT can be planned with more liberal nutrient allowances, as follows[7]:

- *Protein:* Protein energy malnutrition, as indicated by dietary intake and the biomarkers of protein status, is a major concern for patients on dialysis, and it is considered one of the most significant predictors of overall malnutrition and adverse outcomes.[18-20] For most adult patients on dialysis, a protein allowance of at least 1.2 g/kg is ideal to prevent protein malnutrition. This amount provides nutrition needs, maintains positive nitrogen balance, does not produce excessive nitrogenous waste, and replaces the amino acids that are lost during each dialysis treatment. At least 50% of this daily allowance should consist of protein foods of high biologic value (e.g., eggs, meat, fish, poultry).
- *Energy:* MNT recommendations for energy intake are 35 kcal/kg/day for individuals who are younger than 60 years old and 30 to 35 kcal/kg/day for individuals who are older than 60 years old. Interestingly, the mortality rate decreases as the body mass index increases above normal ranges (i.e., 23 kg/m^2 or more), purportedly as the result of a complex association between malnutrition and clinical

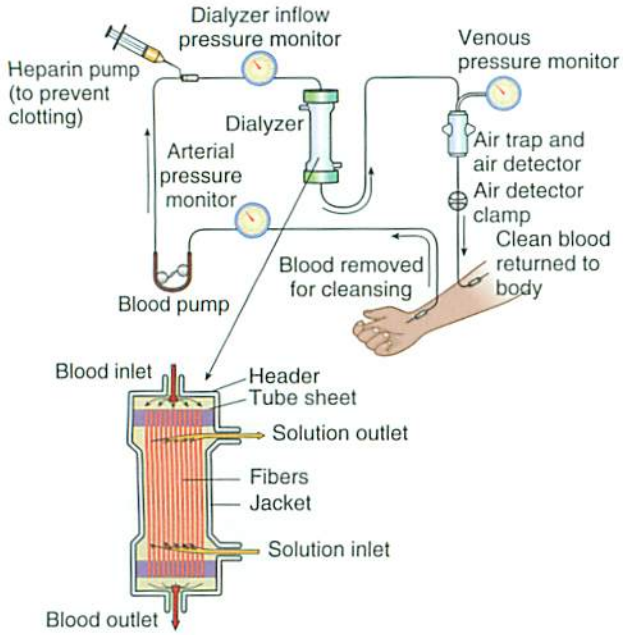

Figure 21-3 Hemodialysis cleans and filters blood with a special filter called a *dialyzer* that functions as an artificial kidney. Blood travels through tubes into the dialyzer, which filters out wastes and extra water, and then the cleaned blood flows through another set of tubes and back into the body. (From the National Institute of Diabetes and Digestive and Kidney Diseases. *Treatment methods for hemodialysis*. National Institutes of Health Publication No. 07-4666. Bethesda, Md: National Institutes of Health; 2006.)

outcomes.[21,22] The unfortunate combination is that ESRD is closely related to a decrease in appetite when the GFR falls below 60 mL/min per 1.73 m^2. A generous amount of carbohydrates with some fat continues to supply needed kilocalories for energy and protein sparing.

- *Sodium and potassium:* To control body fluid retention and hypertension, sodium is limited to 1 to 3 g/day. Sodium intake is not as stringently regulated for patients on dialysis as it is for those with CKD and not yet on dialysis, because the dialysis process rids the body of excess sodium. To prevent potassium accumulation, which can cause cardiac problems, intake is restricted to 2 to 3 g/day, with adjustments that are based on serum levels.
- *Phosphorus and calcium:* With careful monitoring to control for comorbid bone conditions such as chronic kidney disease-mineral and bone disorder, the dietary intake of phosphorus is limited to 800 to 1000 mg/day or 8 to 15 mg/kg when serum phosphorus levels exceed 5.5 mg/dL or when parathyroid hormone is elevated. Calcium intake should not exceed 2 g/day, including the amount received through medications such as binders.
- *Vitamins and minerals:* The general recommendation for all water-soluble vitamins is to achieve the Dietary Reference Intakes. Iron and vitamin D intake are individualized per patient on the basis of

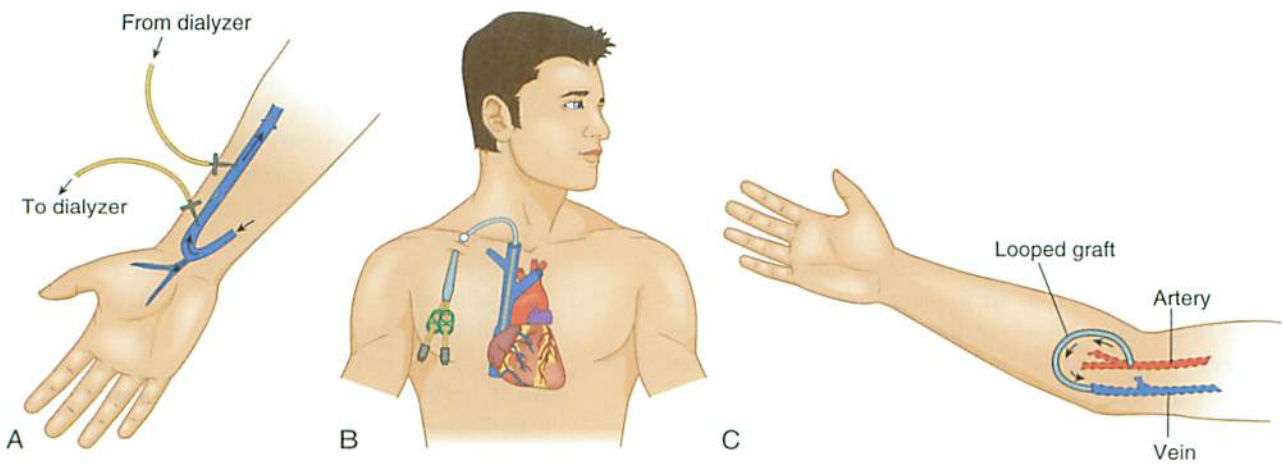

Figure 21-4 Types of access for hemodialysis. **A,** Forearm arteriovenous fistula. **B,** Venous catheter for temporary hemodialysis access. **C,** Artificial loop graft. (From the National Institute of Diabetes and Digestive and Kidney Diseases. *Kidney failure: choosing a treatment that's right for you*. National Institutes of Health Publication No. 00-2412. Bethesda, Md: National Institutes of Health; 2007.)

biochemical markers. Other micronutrients of special interest are as follows:

- Vitamin C: 60 to 100 mg/day
- Vitamin B_6: 2 mg/day
- Folate: 1 mg/day
- Vitamin B_{12}: 3 µg/day
- Vitamin E: 15 IU/day
- Zinc: 15 mg/day

- *Fluid:* Fluid intake is limited to 1000 mL/day plus an amount equal to urine output.

Peritoneal Dialysis. An alternative form of treatment is peritoneal dialysis, which has the convenience of mobility. Approximately 6% of patients with ESRD who are on dialysis use this form of dialysis.[1] During this process, the patient introduces the dialysate solution directly into the **peritoneal cavity**, where it can be exchanged for fluids that contain the metabolic waste products. Because this form of dialysis is continuous within the body, the process is called *continuous ambulatory peritoneal dialysis.*

First, the patient is prepared by surgically inserting a permanent catheter into the peritoneal cavity. Treatments are then carried out by doing the following: (1) attaching a disposable bag that contains the dialysate solution to the abdominal catheter, which leads into the peritoneal cavity; (2) allowing 4 to 6 hours for the solution exchange; (3) lowering the bag to allow gravity to pull the waste-containing fluid into it; and (4) repeating the procedure (Figure 21-5). When the bag is empty, it can be folded around the waist or tucked into a pocket to provide the patient with mobility. The intermittent use of peritoneal dialysis that is self-administered at home gives the patient a sense of control. An automated device is often used to

peritoneal cavity a serous membrane that lines the abdominal and pelvic walls and the undersurface of the diaphragm to form a sac that encloses the body's vital visceral organs.

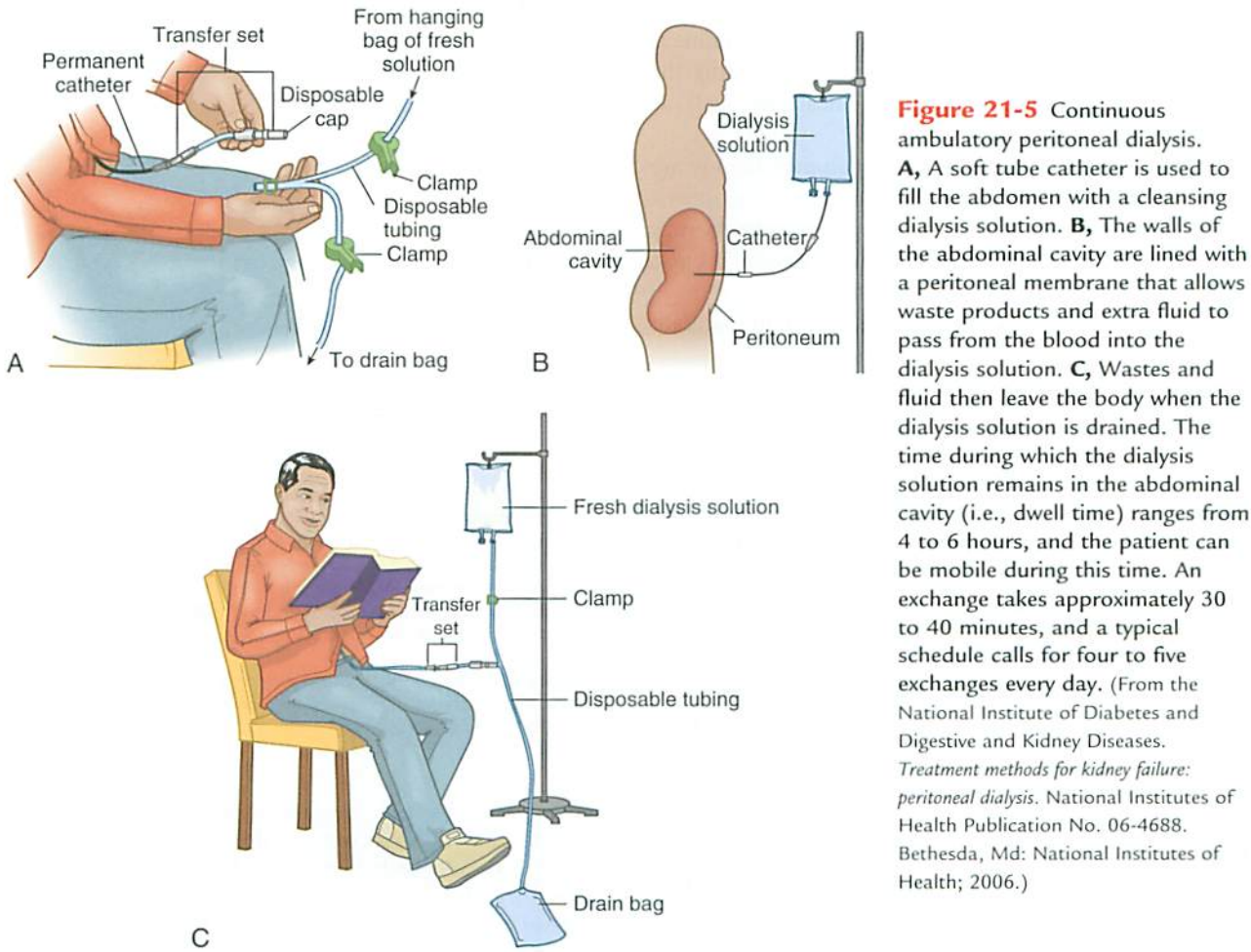

Figure 21-5 Continuous ambulatory peritoneal dialysis. **A,** A soft tube catheter is used to fill the abdomen with a cleansing dialysis solution. **B,** The walls of the abdominal cavity are lined with a peritoneal membrane that allows waste products and extra fluid to pass from the blood into the dialysis solution. **C,** Wastes and fluid then leave the body when the dialysis solution is drained. The time during which the dialysis solution remains in the abdominal cavity (i.e., dwell time) ranges from 4 to 6 hours, and the patient can be mobile during this time. An exchange takes approximately 30 to 40 minutes, and a typical schedule calls for four to five exchanges every day. (From the National Institute of Diabetes and Digestive and Kidney Diseases. *Treatment methods for kidney failure: peritoneal dialysis.* National Institutes of Health Publication No. 06-4688. Bethesda, Md: National Institutes of Health; 2006.)

provide several solution exchanges during sleep hours and one continuous exchange during the day for a technique that is called *continuous cyclic peritoneal dialysis.*

Medical Nutrition Therapy for Peritoneal Dialysis. A slightly more liberal diet may be used with peritoneal dialysis, as follows[7]:

- *Protein:* Increase protein intake slightly to at least 1.2 to 1.3 g/kg with at least 50% of that intake coming from foods with high biologic value protein.
- *Energy:* Maintain a lean body weight by accounting for the energy provided by the dialysate solution in the meal plan.
- *Sodium and potassium*: Sodium intake is slightly more liberal and contingent on fluid balance, with a recommended intake ranging from 2 to 4 g/day. Potassium may be increased to 3 to 4 g/day, depending on serum levels, by eating a wide variety of fruits and vegetables each day.
- *Phosphorus and calcium:* Recommendations for phosphorus and calcium intake remain the same as for hemodialysis: phosphorus, 800 to 1000 mg/day or 8 to 15 mg/kg when serum phosphorus levels exceed 5.5 mg/dL or when parathyroid hormone is elevated; calcium, less than 2 g/day, including the amount received through medications such as binders.
- *Vitamins and minerals:* All recommendations are the same as for hemodialysis, as described previously, with the following exception: patients may need 1.5 to 2 mg/day of thiamin (vitamin B_1) as a result of losses that occur during dialysis.
- *Fluid:* Fluid intake should be adequate to maintain balance.

Table 21-4 presents nutrition laboratory parameter outcome goals for patients with CKD who are either on hemodialysis or peritoneal dialysis.

Transplantation. Kidney transplantation, which is another treatment modality, improves affected individuals' quality of life and survival rates, and it is more cost effective than maintenance dialysis.[1,23] Kidney transplantation has several advantages. Current advances in surgical techniques, immunosuppressive drugs to prevent rejection, and antibiotics to control infection have helped to ensure successful outcomes (see the Drug-Nutrient Interaction box, "Immunosuppressive Therapies After Kidney Transplantation"). Patients who are undergoing kidney transplantation have significantly lower rates of CVD progression, despite the disadvantages of immunosuppressive therapy.

The difficulty with transplantation is that waiting lists can be long and donor matches difficult to find, even when using **expanded-criteria donors** (matches with more liberal criteria).[24] See the Cultural Considerations

TABLE 21-4 NUTRITION LABORATORY PARAMETER OUTCOME GOALS FOR STAGE 5 CHRONIC KIDNEY DISEASE (HEMODIALYSIS AND PERITONEAL DIALYSIS)

Nutrition Laboratory Parameter	Goal	Outcome Prevention Focus
Serum albumin (g/dL)	≥ 4.0	Protein energy malnutrition
Serum prealbumin (mg/dL)	> 30	Protein energy malnutrition
Predialysis serum creatinine (mg/dL)	> 10	Protein energy malnutrition
Serum cholesterol (mg/dL)	> 150 to 180	Protein energy malnutrition
	< 200	Hyperlipidemia
Hemodialysis Prognostic Nutrition Index	≥ 0.8	Increased mortality and morbidity rates
Subjective Global Assessment: 4-item, 7-point scale	≥ 6 to 7	Malnutrition
Serum phosphorus (mg/dL)	3.5 to 5.5	Bone disease
Serum calcium (mg/dL)	8.4 to 10.5	Bone disease
Serum calcium-phosphorus product	≤ 55	Bone disease
Serum bicarbonate (mmol/L)	≥ 22	Metabolic acidosis
Lipid Profile		
Low-density lipoprotein cholesterol (mg/dL)	< 100	Cardiovascular disease
High-density lipoprotein cholesterol (mg/dL)	> 40	Cardiovascular disease
Triglycerides (mg/dL)	< 150	Cardiovascular disease

Based on the Kidney Disease Outcome Quality Initiative recommendations.
Also applicable to stages 1 through 4 of chronic kidney disease, with individualization to the appropriate level of kidney function.
Parameters to be monitored monthly, with the exception of the Hemodialysis Prognostic Nutrition Index, the Subjective Global Assessment (quarterly or when changes are indicated), and the lipid profile (annually).
From Beto JA, Bansal VK. Medical nutrition therapy in chronic kidney failure: integrating clinical practice guidelines. *J Am Diet Assoc.* 2004;104:404-409.

expanded-criteria donors any brain-dead donor who is older than 60 years old or a donor who is older than 50 years old with two of the following conditions: history of hypertension, a terminal serum creatinine level of at least 1.5 mg/dL, or death from a cerebrovascular accident.

DRUG-NUTRIENT INTERACTION

IMMUNOSUPPRESSIVE THERAPIES AFTER KIDNEY TRANSPLANT ATION

The kidney is the most common solid organ that is transplanted worldwide, and the need for kidney transplantation has grown over the past decade. Survival after kidney transplant is largely dependent on a successful immunosuppressive regimen.[1] Several antirejection medications are used after an organ transplant. Most kidney transplant recipients undergo multidrug immunosuppressive therapy that includes corticosteroids to reduce the risk of acute rejection. Over time, the patient may be weaned from steroid use but continue on long-term maintenance regimens that include other antirejection medications. The use of corticosteroids is associated with a number of adverse side effects that specifically affect overall nutritional status, such as the following:

- Peptic ulcer disease
- Hypertension
- Hyperglycemia
- Bone disease
- Increased appetite and weight gain
- Growth retardation (in children)

Corticosteroids also increase the excretion of several nutrients. Additional consumption of vitamins A, B_6, and C; potassium; magnesium; zinc; and protein may be needed in the diet or as supplements. Supplemental calcium and vitamin D are recommended with long-term corticosteroid use. Other antirejection medications are often used concomitantly with corticosteroids, and these may also interact with some of these nutrients. For example, cyclosporine and tacrolimus (Prograf) are calcineurin inhibitors. These immunosuppressants may cause hyperkalemia; thus, high potassium intake from food or supplements should be avoided when these drugs are included in the drug regimen. When the patient is taking these medications, serum drug levels and electrolytes are monitored and the dosage is adjusted for optimal therapeutic benefit. Azathioprine (Imuran) and mycophenolate (CellCept) are other antirejection medications that do not have significant nutrient interactions, but they can cause nausea, vomiting, abdominal pain, and diarrhea in some patients. This can become a concern if the patient is unable to consume adequate nutrition, so the side effects are generally managed by reducing the dose.[2]

Research continues to explore more potent immunosuppressive regimens that avoid or reduce corticosteroid and cyclosporine use. In 2008, only 59% of patients were discharged on corticosteroids and 8% on cyclosporine compared with 96% and 63%, respectively, in 1999.[3] Newer therapies have been associated with fewer episodes of acute rejection, and steroid avoidance is associated with improved growth outcomes in children.[4,5] Further research is needed before new protocols can be accepted.

Kelli Boi

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box entitled "Cultural Disparities in Kidney Transplant Availability and Success in Certain Ethnic and Racial Groups" for more details.

MNT for patients who choose kidney transplantation will be highly individualized. Table 21-5 summarizes nutrition parameters for various levels of kidney disease and treatments.

Complications

Long-term complications of dialysis include bone disorders, malnutrition, anemia, hormonal and blood pressure imbalances, psychologic depression, and diminished quality of life as a result of constant dependence on treatments.

Nutrition Support. There are special considerations for patients on dialysis who are in medical need of nutrition support via enteral or parenteral feedings. A medical necessity of nutrition support usually means that the patient is experiencing severe malnutrition, inflammation, and anorexia. The type of and tolerance of dialysis must be considered when choosing an appropriate nutrition support modality, and the current GFR, metabolic state, stress, and nitrogen balance must also be considered. The American Society for Parenteral and Enteral Nutrition has published clinical guidelines for administering and evaluating nutrition support specifically for patients with CKD.[25]

Osteodystrophy. Bone disease and disorders are prevalent in CKD, and they are an important cause of morbidity. A combination of factors contribute to renal osteodystrophy and chronic kidney disease-mineral and bone disorder. The decreased activation of vitamin D has

CULTURAL CONSIDERATIONS

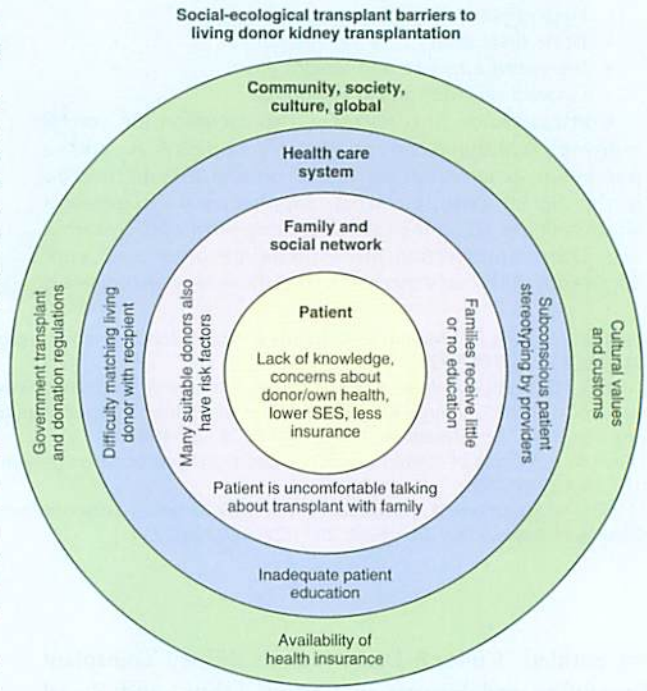
CULTURAL DISPARITIES IN KIDNEY TRANSPLANT AVAILABILITY AND SUCCESS IN CERTAIN ETHNIC AND RACIAL GROUPS

Kidney transplantation is generally considered the optimal treatment for patients with end-stage renal disease. In 2010, nearly 17,000 kidney transplantations were performed in the United States. Almost half of those transplants went to Caucasian recipients; however, Caucasians only make up 38% of those on the waiting list to receive a transplant.[1] Advances in both medical technology and immunosuppressive therapies have led to longer lives for transplant recipients among all ethnic groups over the past 30 years. Despite this increase in survival rates, African-American kidney transplant recipients continue to have a higher incidence of the transplant failing within 10 years as well as a higher rate of death among transplant recipients compared with their white counterparts. Hispanic and Asian transplant recipients are reported to have the best outcomes.[2,3] Several theories exist to explain these disparities in transplantation rates and survival among various ethnic groups.

Racial variation with regard to transplant success can be attributed in part to differences in immunologic function among different racial and ethnic groups. There are also social factors that affect the disparity of transplantation (see figure). Minorities are less likely to pursue live donor kidney transplants as a result of lack of knowledge, discomfort with talking to family members or individuals from other social networks about the procedure, difficulty with finding suitable donors, insufficient education from health care providers, lower availability of health insurance, and health care provider discrimination.[4] Provider inequity is related to variations in the consensus regarding the appropriate and just allocation of organs. Many providers believe that an organ should go to the recipient who is likely to receive the greatest benefit or live the longest as a result of the transplant. Factors such as African-American ethnicity, lower socioeconomic status, comorbid diseases, and limited access to health care all decrease survival rate and length of life despite transplantation. To compound this issue, the longer that a recipient is on dialysis while waiting for a transplant, the lower the success rate of the transplant.[5]

To decrease these disparities, many interventions have been suggested. Immunosuppression regimens should be modified for different racial groups. Education methods and materials should also be culturally sensitive and consider the patients' cultural beliefs, values, language, socioeconomic status, and social context.[2] In addition, dialysis providers should be educated regarding kidney transplantation so that they can supply quality education to both potential transplant patients and potential live donors. Education about live donor kidney transplant should be offered to families of patients with end-stage renal disease to increase the donor pool and thus ease the matching process for donors for these groups. Research indicates that education that is provided early during the course of treatment and frequently throughout treatment leads to an increase across all ethnic groups with regard to patients' pursuit of transplantation.[4,5]

Jennifer E. Schmidt



From Waterman AD, Rodrigue JR, Purnell TS, et al. Addressing racial/ethnic disparities in live donor kidney transplantation: priorities for research and intervention. *Semin Nephrol.* 2010;30(1):90-98.

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TABLE 21-5 SELECTED NUTRITION PARAMETERS FOR VARIOUS LEVELS OF KIDNEY FAILURE*

Parameter	Normal Kidney Function	Stages 1 Through 4 of Chronic Kidney Disease	Stage 5 Hemodialysis	Stage 5 Peritoneal Dialysis	Transplant
Energy	30 to 37 kcal/kg of body weight	35 kcal/kg if < 60 years old	35 kcal/kg if < 60 years old	35 kcal/kg if < 60 years old	30 to 35 kcal/kg initial
		30 to 35 kcal/kg if ≥ 60 years old	30 to 35 kcal/kg if ≥ 60 years old	30 to 35 kcal/kg if ≥ 60 years old; include calories from dialysate	25 to 30 kcal/kg for maintenance
Protein	0.8 g/kg	0.6 to 0.75 g/kg ≥ 50% HBV	≥ 1.2 g/kg ≥ 50% HBV	≥ 1.2 to 1.3 g/kg ≥ 50% HBV	1.3 to 1.5 g/kg initial 1.0 g/kg for maintenance
Fat (% total kcal)	30% to 35%	Patients considered at highest risk for cardiovascular disease; emphasis on MUFA, PUFA, and 250 to 300 mg of cholesterol per day			< 10% saturated fat
Sodium	Unrestricted	1 to 3 g/day	1 to 3 g/day	2 to 4 g/day; monitor fluid balance	Unrestricted; monitor medication effects
Potassium	Unrestricted	Unrestricted unless serum level is high	2 to 3 g/day; adjust to serum levels	3 to 4 g/day; adjust to serum levels	Unrestricted; monitor medication effects
Calcium	Unrestricted	1 to 1.5 g/day and not to exceed 2 g/day with binder load	≤2 g/day from diet and medications, including binder load	≤ 2 g/day from diet and medications, including binder load	1.2 g/day
Phosphorus	Unrestricted	800 to 1000 mg/day when serum phosphorus level is > 4.6 mg/dL or when parathyroid hormone is elevated	800 to 1000 mg/day when serum phosphorus level is > 5.5 mg/dL or when parathyroid hormone is elevated	800 to 1000 mg/day when serum phosphorus level is > 5.5 mg/dL or when parathyroid hormone is elevated	Unrestricted unless indicated
Fluid	Unrestricted	Unrestricted with normal urine output	1000 mL plus urine output	Maintain balance	Unrestricted unless indicated

*These are guidelines for initial assessment only; individualization to the patient's metabolic status and coexisting metabolic conditions is essential for optimal care.

HBV, High biologic value; *MUFA*, monounsaturated fatty acids; *PUFA*, polyunsaturated fatty acids.

Modified from Beto JA, Bansal VK. Medical nutrition therapy in chronic kidney failure: integrating clinical practice guidelines. *J Am Diet Assoc.* 2004;104:404-409; and the American Dietetic Association. *ADA nutrition care manual*. Chicago: American Dietetic Association; 2010.

a cascading effect that results in elevated parathyroid hormone and reduced serum calcium levels. Patients also have elevated serum phosphorus levels as a result of the inability of the kidney to excrete phosphorus. This combination causes abnormal changes in bone structure and function. Hyperphosphatemia is associated with increased mortality risk; thus, phosphate binders are an important management aspect of CKD. Patients with any level of kidney dysfunction should be evaluated for bone disease and disorders of calcium and phosphorus metabolism. Treatment strategies for bone disorders require a highly individualized management plan and continue to evolve in the light of new research.[26]

Neuropathy. Central and peripheral neurologic disturbances are common among patients at the initiation of dialysis, and they are even more prevalent in patients with diabetes. Symptoms of neuropathy may not be present until the GFR falls to less than 12 to 20 mL/min per 1.73 m^2; however, patients should be periodically assessed for implications of uremia or disease progression.[14]

KIDNEY STONE DISEASE

In the United States, approximately 5% of women and 12% of men form kidney stones at some point during their lives.[27] The basic cause of **nephrolithiasis** is unknown, but many factors that relate to the nature of the urine itself or to conditions of the urinary tract environment contribute to stone formation. Genetics, urine calcium excretion, and urine pH are three factors that are closely linked with the risk for stone formation.[28] The most common types of kidney stones are calcium, struvite, and uric acid. Figure 21-6 illustrates various types of stones. In addition, Box 21-2 lists risk factors that are associated with kidney stone development.

Disease Process

Calcium Stones

Calcium oxalate and calcium phosphate stones are the most common types, and they account for approximately 80% of all kidney stones. High levels of urinary oxalate increase the risk of an individual forming a calcium oxalate stone. Oxalates are derived from endogenous synthesis (relative to lean body mass) and dietary sources (see Box 21-3). Oxalic acid is a metabolite of ascorbic acid. Therefore, the long-term megadosing of vitamin C supplements (more than 3 g/day) may pose a potential health risk for kidney stone formation. A small percentage of the population are "hyperabsorbers" of dietary oxalate and thus are at higher risk of forming stones. The supersaturation of kidney stone materials in the urine may result from the following[29]:

- Excess calcium in the blood (hypercalcemia) or urine (hypercalciuria)
- Excess oxalate (hyperoxaluria) or uric acid in the urine (hyperuricosuria)
- Low levels of citrate in the urine (hypocitraturia)
- Excess animal protein and sodium intake

Significant dietary calcium intake from food or supplements equaling up to 2 g/day is inversely associated with calcium oxalate stones. Essentially, individuals with a low dietary intake of calcium are at a *higher* risk for calcium oxalate stone formation than those who adequately consume calcium. It is a common misunderstanding to restrict the calcium intake of those patients who form calcium oxalate stones.[29,30]

Struvite Stones

Struvite stones, which account for approximately 10% of all stones, are composed of magnesium ammonium phosphate and carbonate apatite. They often are called

nephrolithiasis the formation of a kidney stone.

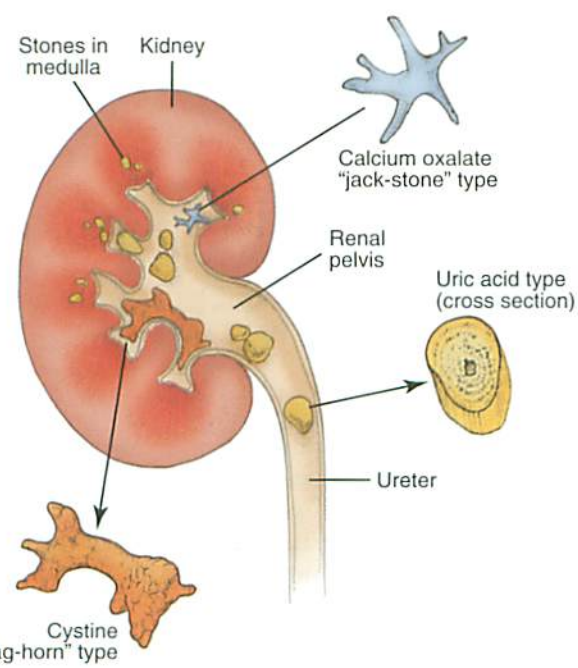

Figure 21-6 Renal calculi: stones in the kidney, renal pelvis, and ureter.

BOX 21-2 RISK FACTORS FOR THE DEVELOPMENT OF KIDNEY STONES

- High dietary intake of the following:
  - Animal protein
  - Purines
  - Oxalate
  - Vitamin C
  - Sodium
- Inadequate intake of the following:
  - Fiber
  - Potassium
  - Fluid
- Family history
- Urinary tract infections
- Kidney disorders such as cystic kidney diseases
- Metabolic disorders such as the following:
  - Hyperparathyroidism
  - Chronic inflammation of the bowel
  - Intestinal bypass or ostomy surgery
  - Higher body mass index

From the American Dietetic Association. *ADA nutrition care manual.* Chicago: American Dietetic Association; 2010.

BOX 21-3 HIGH-OXALATE FOODS AND DRINKS

Drinks
Chocolate drink mixes, soymilk, Ovaltine, instant iced tea, fruit juices of the fruits listed in this table

Fruits
Apricots (dried), red currants, figs, kiwi, rhubarb

Vegetables
Beans (wax, dried), beets and beet greens, chives, collard greens, eggplant, escarole, dark greens of all kinds, kale, leeks, okra, parsley, green peppers, potatoes, rutabagas, spinach, Swiss chard, tomato paste, watercress, zucchini

Breads, Cereals, and Grains
Amaranth, barley, white corn flour, fried potatoes, fruitcake, grits, soybean products, sweet potatoes, wheat germ and bran, buckwheat flour, All-Bran cereal, graham crackers, pretzels, whole wheat bread

Meat, Meat Replacements, Fish, and Poultry
Dried beans, peanut butter, soy burgers, miso

Desserts and Sweets
Carob, chocolate, marmalades

Fats and Oils
Nuts (peanuts, almonds, pecans, cashews, hazelnuts), nut butters, sesame seeds, tahini (a paste made from sesame seeds)

Other Foods
Poppy seeds

From the American Dietetic Association. *ADA nutrition care manual.* Chicago: American Dietetic Association; 2010.

infection stones because they are primarily caused by urinary tract infections and because they are not associated with any specific nutrient. Thus, no particular diet therapy is involved. Struvite stones are usually large "staghorn" stones that are surgically removed.

Uric Acid Stones

The excess excretion of uric acid may be caused by some impairment that involves the metabolism of purine, which is a nitrogen end product of the dietary protein from which uric acid is formed. This impairment occurs with diseases such as gout, and it can also occur with rapid tissue breakdown during wasting disease. Other conditions that are associated with persistently acid urine and uric acid stone formation are diarrheal illness, diabetes, obesity, and metabolic syndrome.[29] Roughly 9% of kidney stones are uric acid stones.

Other Stones

Other rare forms of kidney stones are often reflective of inherited disorders or complications of medications. For example, cystine stones are caused by a genetic defect in the renal reabsorption of the amino acid cystine (as well as other dibasic amino acids), thereby causing an accumulation in the urine (cystinuria). Cystine is not soluble and thus high levels may result in stone formation.

Clinical Symptoms

The main symptom of kidney stones is severe pain. Many other urinary symptoms may result from the presence of the stones, and general weakness and sometimes fever are present. Laboratory examination of the urine and of any passed stones helps to determine treatment.

Medical Nutrition Therapy

General Objectives

MNT may include several aspects, and it will vary depending on the type of stone. General MNT recommendations are as follows[7]:

- *Protein:* Excessive protein intake, especially from animal sources, is a risk factor for stone formation. Thus, patients should normalize their intake to healthy population standard recommendations of 0.8 to 1.0 g/kg/day and should not exceed the Dietary Reference Intake.
- *Calcium:* Low dietary calcium intake is a risk for calcium oxalate stone formation. Thus, patients should be encouraged to normalize calcium intake to 800 mg/day for men and 1200 mg/day for women and balance intake throughout the day.
- *Sodium:* High sodium intake increases the amount of calcium excretion in the urine, thereby precipitating hypercalciuria, and it is associated with an increased risk of stone formation. Sodium intake should not exceed 2300 to 3450 mg/day.
- *Oxalates:* Limiting urinary oxalate significantly reduces the risk of calcium oxalate stone formation.[31] Thus, avoiding foods that are high in oxalates is advised. Intake should be less than 40 to 50 mg/day; see Box 21-3.
- *Vitamins and minerals*: Vitamin C should be limited to the Dietary Reference Intake, and all other vitamin and mineral intake should meet the Dietary Reference Intake standards.
- *Fluid:* A large fluid intake of 2 L/day or more helps to produce more dilute urine and thus to prevent the accumulation of materials that form stones.

TABLE 21-6 SUMMARY OF DIETARY PRINCIPLES IN KIDNEY STONE DISEASE

Stone Chemistry	Nutrient Modification
All types of stones	Increase fluid intake enough to produce ≥ 2.5 L/day of clear urine
Calcium oxalate	Reduce sodium, animal protein, and oxalate-containing food intake; continue normal calcium intake; increase fiber intake; avoid high doses of vitamin C
Calcium phosphate	Reduce phosphorus intake
Uric acid	Reduce animal protein intake and purine intake
Cystine	Reduce sodium intake; avoid excess protein; maintain fluid intake at 4 L/day

Exact fluid intake needs vary by patient, but enough fluids—preferably water—should be ingested to produce at least 2.5 L of clear urine daily. For patients who consume soft drinks, reducing soft-drink intake may lower the risk of recurrent stone formation.[32]

General dietary principles that involve kidney stone disease are summarized in Table 21-6.

Objectives Specific to Type of Stone

The nutrition care plan may be further individualized relative to the nature of the specific stone formed. A variety of medications are useful for the treatment of kidney stones in combination with diet therapy. For medications to be most effective, the specific type of stone must be identified. This is not always possible and therefore limits drug therapy in some individuals.

Calcium Stones. In some cases, dietary control of the stone constituents may help to reduce the recurrence of such stone formation. If a stone is made of calcium oxalate, then foods that are high in oxalate (see Box 21-3) should be limited. If a stone is made of calcium phosphate, additional sources of phosphorus (e.g., meats, legumes, nuts) should be controlled.

In addition to the recommendations listed previously, fiber intake should be considered in the case of calcium stones. Materials that bind potential stone elements in the intestine can prevent their absorption and eliminate them from the body. For example, phytate can bind calcium and thus help to prevent the crystallization of oxalate calcium salts. Phytates are found in high-fiber plant foods such as whole wheat, bran, and soybeans.

Uric Acid Stones. The alkalinization of the urine helps to prevent the formation of uric acid stones. Dietary attempts to alter urinary pH with **acidic or alkaline diets** are unsuccessful.[33] However, potassium salts may be used to raise the urinary pH, which decreases the supersaturation of uric acid.[29] Establishing and maintaining a healthy weight and limiting animal protein (including red meat, fish, and poultry) intake to 40 to 50 g/day are also advisable.[31]

Cystine Stones. Dietary modifications are geared toward reducing urinary cystine concentrations by decreasing intake and diluting the urine. Diluting the urine may require the intake of 4 to 5 L/day of water in adults.[31]

acidic or alkaline diets diets based on the theory that diets high in acidic foods (e.g., animal protein, caffeine, simple sugars) will disrupt the body's normal pH balance, which is slightly alkaline.

SUMMARY

- The nephrons are the functional units of the kidneys. Through these unique structures, the kidney maintains homeostasis in the blood of the materials that are required for life and health. The nephrons accomplish their tremendous task by constantly cleaning the blood, returning necessary elements to the blood, and eliminating the remainder in concentrated urine.
- Various diseases that interfere with the vital function of nephrons can cause kidney disease. Kidney diseases have predisposing factors, such as diabetes, recurrent urinary tract infections that may lead to renal calculi, and progressive glomerulonephritis that may lead to chronic nephrotic syndrome and kidney failure.
- At its end stage, CKD is treated by dialysis or kidney transplantation. Patients who are undergoing dialysis require close monitoring for protein, water, and electrolyte balance.
- Kidney stones may be formed from a variety of substances. For some patients, a change in the dietary intake of the identified substance (e.g., sodium, oxalate, purine) may decrease stone formation.

CRITICAL THINKING QUESTIONS

1. For each of the following conditions, outline the nutrition components of therapy, and explain the effect of each on kidney function: glomerulonephritis, nephrotic syndrome, and CKD.
2. Consider the nutrition factors that must be monitored in patients who are undergoing kidney dialysis. How would you suggest a client self-monitor fluid intake to meet hydration needs while not exceeding restrictions?
3. Outline the medical and nutrition therapy for patients who are on hemodialysis and peritoneal dialysis. What are the critical differences, and why?

CHAPTER CHALLENGE QUESTIONS

True-False

Write the correct statement for each statement that is false.

1. *True or False:* The basic functional unit of the kidney is the nephron.
2. *True or False:* Only a few nephrons are present in each kidney, so metabolic stress can easily cause problems.
3. *True or False:* The functioning of the nephrons relates little to the rest of the body.
4. *True or False:* The main function of the glomerulus is filtration.
5. *True or False:* The tasks of the various parts of the nephron tubules are reabsorption, secretion, and excretion.
6. *True or False:* Dietary modifications in acute glomerulonephritis usually involve crucial restrictions of protein and sodium.
7. *True or False:* The primary symptom in nephrotic syndrome is massive albuminuria.
8. *True or False:* Nephrotic syndrome is best treated by a very-low-protein diet.
9. *True or False:* The multiple symptoms of advanced CKD result from metabolic imbalances in the body's inability to handle protein, electrolytes, and water.
10. *True or False:* Prolonged immobilization (e.g., with full body casts or disability) may lead to the withdrawal of bone calcium and the formation of calcium kidney stones.

Multiple Choice

1. Acute glomerulonephritis is best treated with which of the following methods? *(Circle all that apply.)*
   - **a.** Reducing protein because filtration is impaired
   - **b.** Using a normal amount of protein for optimal tissue nutrition and growth
   - **c.** Restricting sodium to 1500 mg/day
   - **d.** Restricting potassium intake
2. Diet therapy for patients with nephrotic syndrome is designed to perform which of the following functions? *(Circle all that apply.)*
   - **a.** Increase protein to replace the massive losses
   - **b.** Normalize protein intake to reduce albumin losses
   - **c.** Provide adequate kilocalories for energy and spare protein for tissue needs
   - **d.** Moderately restrict sodium to help prevent edema
3. The general diet needs for a patient in stage 5 CKD who is on hemodialysis includes which of the following? *(Circle all that apply.)*
   - **a.** Increased protein intake
   - **b.** Reduced protein intake
   - **c.** Careful control of sodium and potassium according to need
   - **d.** Increased fluids to stimulate kidney function

Evolve Please refer to the Students' Resource section of this text's Evolve Web site for additional study resources.

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FURTHER READING AND RESOURCES

American Urological Association. www.urologyhealth.org

National Institute of Diabetes and Digestive and Kidney Diseases. www.niddk.nih.gov

National Kidney Foundation. www.kidney.org

These Web sites provide additional information about various forms of kidney disease. Several national organizations provide free education and support for health care providers, patients, and family members. Dietary restrictions for patients with kidney disease can sometimes be overwhelming. To fully understand such diets, continuous follow-up and feedback are needed.

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