Despite the improved diagnosis and treatment of kidney disease, late diagnosis with resultant permanent damage to the kidneys still occurs. A work group established by the National Kidney Foundation, the Kidney Disease Outcome Quality Initiative (KDOQI), was asked to develop clinical practice guidelines and a uniform classification system for chronic kidney disease (CKD). CKD is a public health issue because of its increasing incidence and the high cost of interventions. An additional concern is the increasing incidence of kidney disease in African Americans and Native Americans.

According to the 2004 report from the US Renal Data System, the number of patients with CKD receiving therapy in 2002 was 431,284. This number is a 4.6% increase over the number for the year 2001. The adjusted rate for CKD was 1435 cases per million population—72% of patients were undergoing dialysis; the other 122,374 had a functioning transplant. The adjusted rate of CKD for the white population was 1060 cases per million; for the African American population, 4467 cases per million; and for the Native American population, 2569 cases per million.

In 2002, Medicare expenses for CKD treatment increased 11% over the level in 2001; Medicare expenses were $17 billion and non-Medicare expenses were $8.2 billion. From the individual perspective, Medicare costs per year are approximately $53,000, with deductibles and copayments bringing the total to $63,000 per year. Total cost for the entire CKD program was approximately $25.2 billion at the end of 2002.

The KDOQI has identified 5 stages of kidney failure (Table 1) on the basis of glomerular filtration rate (GFR). Normal GFR in men is 125 to 150 mL/min per 1.73 m² (1.73 m² is considered the standard normal body surface area). Chronic kidney failure...
is defined by the KDOQI as having kidney damage lasting for 3 months or more or having a GFR less than 60 mL/min per 1.73 m² for 3 months or more, with or without kidney damage. End-stage renal disease (ESRD) is described as the stage of CKD when damage to the kidneys is permanent and kidney function cannot maintain life; consequently, patients at this time require dialysis or transplantation. ESRD is at the far end of the spectrum of progressive renal dysfunction.

Healthy People 2010 identifies the 5 risk factors for ESRD as diabetes mellitus, hypertension, proteinuria, family history, and increasing age. Unfortunately, patients are often asymptomatic in the early stages of kidney failure when renoprotective strategies that could slow or reverse the process of kidney damage could be implemented. Because of the lack of early signs and symptoms and interventions, patients may come to an acute care facility with signs and symptoms of newly diagnosed ESRD.

What do critical care nurses need to know about CKD and its treatment to improve patients’ outcomes? A thorough assessment of this complex situation by critical care nurses is essential, because multiple body systems are altered when kidney function is impaired. In this article, we use a case study to review the pathophysiological changes that occur when the kidneys fail, resulting in admission to the critical care unit. The immediate interventions and the expected outcomes are also presented.

### Case Study

J.M., a 34-year-old African American man, came to the emergency department with a 6-day history of increasing swelling in both lower extremities. A similar swelling had occurred once before recently but had cleared up spontaneously. J.M. said he had no history of headaches, hypertension, nausea, vomiting or diarrhea, fever and chills, shortness of breath, chest pain, urinary problems, weight loss, confusion or other neurological changes, or exposure to toxic substances. He also stated that he was not taking any medication.

On physical examination, his blood pressure was 222/142 mm Hg, his heart rate was 110/min with S3 and S4 gallops, and his respiratory rate was 24/min with bibasilar crackles. Electrocardiography showed left ventricular hypertrophy and ST-T waves consistent with a strain pattern. A funduscopic examination showed bilateral chronic and new hemorrhages (cotton wool hemorrhages and exudates), arteriolar narrowing, and arteriovenous nicking. Laboratory results are reported in Table 2.

### Table 1: Chronic kidney disease defined by the National Kidney Foundation

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
<th>Glomerular filtration rate (GFR), mL/min per 1.73 m²</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Kidney damage with normal or increased GFR</td>
<td>≥90</td>
</tr>
<tr>
<td>2</td>
<td>Kidney damage with mild decrease in GFR</td>
<td>60-89</td>
</tr>
<tr>
<td>3</td>
<td>Moderate decrease in GFR</td>
<td>30-59</td>
</tr>
<tr>
<td>4</td>
<td>Severe decrease in GFR</td>
<td>15-29</td>
</tr>
<tr>
<td>5</td>
<td>Kidney failure</td>
<td>&lt;15 (or dialysis)</td>
</tr>
</tbody>
</table>

ESRD is defined by the KDOQI as having kidney damage lasting for 3 months or more or having a GFR less than 60 mL/min per 1.73 m² for 3 months or longer. Kidney damage is defined as pathologic abnormalities or markers of damage, including abnormalities in blood or urine tests or imaging studies. Reprinted from the National Kidney Foundation, with permission.

### Pathophysiology

The kidneys act as regulators for many of the body’s functions and control complex processes that maintain homeostasis (Figure 1). The kidneys receive approximately 20% to 25% of the cardiac output per minute; blood is filtered through the nephrons, the functioning units of the kidneys. Each kidney has approximately 1 million nephrons, providing a large reserve of nephrons that enables homeostasis to be maintained, even when nephrons have been damaged. However, when 90% of nephrons are lost, renal function is significantly impaired, resulting in ESRD. The best laboratory indicator of kidney function is the estimated GFR. Several equations are available to estimate GFR. The National Kidney Disease Education Program of the National Institute of Diabetes and Digestive and Kidney Diseases, the National Kidney Foundation, and the American Society of Nephrology have recently recommended the Modification of Diet in Renal Disease Study equation as the formula to calculate GFR:

\[
GFR = 186 \times \frac{\text{serum creatinine (mg/dL)}}{1.154} \times \text{age (years)}^{-0.203} \times 0.742 \times 1.210 \text{ (if African-American)}
\]
On the basis of this formula, J.M. had a GFR of 3 mL/min per 1.73 m², meeting the definition of kidney failure. Whatever the underlying cause of CKD, the effects of kidney failure on the body’s homeostatic mechanisms are the same. The next section provides a comparison of the normal homeostatic regulations and the alterations assessed in J.M.

**Alterations in Regulatory Functions**

**Body Water Regulation**

Fluid volume is altered when the kidney loses its ability to excrete water because of damaged nephrons and the resultant decreased GFR. Other factors that contribute to the development of fluid volume overload are proteinuria and increased renin. Proteinuria occurs in response to damage of the glomeruli. High blood pressure can cause sclerotic changes in the glomeruli with a resultant loss of protein, especially albumin in the urine.10 This damage to the kidneys from hypertension is also known as hypertensive

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<table>
<thead>
<tr>
<th>Laboratory test</th>
<th>Admission results</th>
<th>Results after dialysis</th>
<th>Reference intervals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum urea nitrogen, mmol/L</td>
<td>67.47 (189)</td>
<td>66.04 (185)</td>
<td>2.9-6.4 (8-18)</td>
</tr>
<tr>
<td>Creatinine, µmol/L</td>
<td>1909.4 (21.6)</td>
<td>1865.2 (21.1)</td>
<td>53-106 (0.6-1.2)</td>
</tr>
<tr>
<td>Sodium, mmol/L</td>
<td>127</td>
<td>129</td>
<td>135-147</td>
</tr>
<tr>
<td>Potassium, mmol/L</td>
<td>5.8</td>
<td>3.9</td>
<td>3.5-5.0</td>
</tr>
<tr>
<td>Chloride, mmol/L</td>
<td>90</td>
<td>89</td>
<td>95-105</td>
</tr>
<tr>
<td>Carbon dioxide, mmol/L</td>
<td>9.9</td>
<td>12</td>
<td>23-30</td>
</tr>
<tr>
<td>Uric acid, µmol/L (mg/dL)</td>
<td>1142.0 (19.2)</td>
<td>Not determined</td>
<td>237.9-505.6 (4-8.5)</td>
</tr>
<tr>
<td>Calcium, mmol/L (mg/dL)</td>
<td>2.05 (8.2)</td>
<td>2.35 (9.4)</td>
<td>2.20-2.58 (8.8-10.3)</td>
</tr>
<tr>
<td>Phosphorus, mmol/L (mg/dL)</td>
<td>3.91 (12.1)</td>
<td>3.87 (12)</td>
<td>0.81-1.61 (2.5-5.0)</td>
</tr>
<tr>
<td>Hemoglobin, g/L</td>
<td>83</td>
<td>Not determined</td>
<td>140-180</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>0.252</td>
<td>Not determined</td>
<td>0.42-0.52</td>
</tr>
<tr>
<td>Arterial blood gases on room air</td>
<td>pH 7.32 Pco2 22.9 mm Hg Bicarbonate 11.9 mmol/L Base excess -12 Po2 45.9 mm Hg</td>
<td>pH 7.44 Pco2 18.1 mm Hg Bicarbonate 12.5 mmol/L Base excess -9 Po2 81.7 mm Hg</td>
<td>pH not determined Pco2 33-44 mm Hg Bicarbonate 21-28 mmol/L Base excess not determined Po2 75-100 mm Hg</td>
</tr>
<tr>
<td>Urinalysis</td>
<td>4+ protein 2+ hematuria</td>
<td>Not determined</td>
<td>Not determined</td>
</tr>
<tr>
<td>Alkaline phosphatase, U/L</td>
<td>536</td>
<td>378</td>
<td>30-120</td>
</tr>
</tbody>
</table>

**Table 2 J.M.’s laboratory results**

---

Figure 1 Homeostatic functions of the kidney.
nephrosclerosis and may cause damage not only to the glomeruli but also to the arteriolar walls. The loss of albumin in the urine contributes to fluid shifting from the intravascular space to the interstitial space because of decreased oncotic pressure. As a response to decreased GFR, aldosterone is released from the adrenal cortex, causing the kidneys to reabsorb sodium and water. Fluid retention in turn results in the development of respiratory and cardiovascular clinical manifestations. In J.M., cardiovascular assessment findings included 3+ edema of the lower extremities, an albumin level of 32 g/L, blood pressure of 222/142 mm Hg, the presence of S3 and S4 heart sounds, sinus tachycardia (heart rate 108-112/min), crackles in the lungs, and a decreased sodium level (dilutional hyponatremia).

**Acid–Base Balance**

Metabolic acidosis is associated with CKD because the tubules cannot excrete hydrogen ions (H\(^+\)) resulting in the use of bicarbonate (HCO\(_3^–\)) anions to maintain acid-base balance. Two other buffering systems are in place that assist in compensating for the acidosis. Hydrogen ions combine with ammonia produced in the renal tubule cells to form ammonium, which combines with chloride and is excreted in the urine. This mechanism helps to remove H\(^+\) while generating HCO\(_3^–\). However, because of impaired nephron function, excretion of ammonium is decreased. The third mechanism involved with acid-base balance results in H\(^+\) combining with phosphate (one of the body’s buffering systems). Metabolic acidosis also contributes to a shift of calcium from the bone, allowing H\(^+\) to enter and be buffered in the bone. Results of J.M.’s laboratory tests revealed a pH of 7.32, HCO\(_3^–\) 11.9 mmol/L, base excess -12, and serum carbon dioxide 9.9 mmol/L, all indicating metabolic acidosis. A decreased arterial PCO\(_2\) of 22.9 mm Hg results from an increased respiratory rate as the body attempts to compensate for the acidosis by exhaling the respiratory acid carbon dioxide. The anion gap can be used to determine the cause of the metabolic acidosis. Normally the kidney conserves HCO\(_3^–\) and excretes H\(^+\). In CKD, when the glomeruli are damaged, metabolic acids such as sulfuric and phosphoric acid are retained, causing a widening of the anion gap. The anion gap is calculated mathematically:

\[
\text{Anion gap} = \text{Na}^+ – (\text{Cl}^- + \text{HCO}_3^-)
\]

The normal anion gap is approximately 12 mmol/L. J.M.’s anion gap was 127 – (90 + 11.9) = 25.1 mmol/L, supporting the diagnosis of metabolic acidosis due to kidney failure.

**Electrolyte Balance**

Multiple electrolyte levels are altered in patients with CKD. Potassium levels may be normal until late in ESRD, and elevated potassium levels are often associated with CKD because of the inability of the kidney to excrete potassium as a result of decreased GFR. In addition, when metabolic acidosis is present, potassium ions shift from the intracellular compartment to the extracellular space in exchange for H\(^+\), in an effort to maintain extracellular acid-base balance. The kidneys normally excrete 40 to 60 mmol of potassium daily. J.M.’s potassium level was 5.8 mmol/L, increasing his risk for fatal dysrhythmias. Serum phosphorus and calcium levels are also altered in CKD. When GFR is less than 30 to 50 mL/min per 1.73 m\(^2\), phosphorus excretion is impaired. Because of the reciprocal relationship between phosphorus and calcium, this increased retention of phosphorus results in a decrease in the serum level of calcium. Three additional mechanisms can affect calcium level. Calcium is found in 3 forms in the blood: attached to protein, attached to other complexes, and free or ionized. Because some calcium is bound to protein, total serum calcium level can decrease when albumin level decreases. J.M. had proteinuria. This loss of albumin can contribute to a decreased serum level of calcium. CKD also has an effect on vitamin D synthesis. The kidneys normally convert inactive vitamin D to its active form: 1,25-dihydroxycholecalciferol. Impaired vitamin D synthesis results in decreased absorption of calcium in the gastrointestinal tract. The third mechanism that affects serum levels of calcium is the endocrine system. When the serum level of calcium decreases, the parathyroid gland increases its secretion of parathyroid hormone, causing calcium to be released from the bone and compensating for the decreased serum level of calcium. Results of J.M.’s laboratory tests showed a calcium level of 2.05 mmol/L (8.2 mg/dL) and a phosphorus level of 3.91 mmol/L (12.1 ng/dL), indicating impaired phosphorus excretion and a reciprocal decrease in calcium level.

**Alterations in Excretory Function**

In CKD, nitrogenous waste products from protein metabolism
are retained in the body, resulting in azotemia, as evidenced by the increased serum levels of urea nitrogen and creatinine. The tubules, which are permeable to urea, normally reabsorb little urea. However, as GFR decreases, more urea is reabsorbed. Although elevated serum levels of urea nitrogen alone can indicate other abnormalities, such as dehydration, elevation of serum levels of both urea nitrogen and creatinine indicates kidney failure. The serum urea nitrogen-creatinine ratio (normal 10:1 to 20:1) was once used to assess kidney function, but the ratio is not considered as an important indicator today. J.M.’s serum level of urea nitrogen was 66.05 mmol/L (185 mg/dL), and his serum creatinine level was 1909.4 μmol/L (21.6 mg/dL), resulting in a ratio of 9:1. This slightly decreased ratio could reflect fluid overload from the CKD or an undiagnosed liver disease, because J.M.’s alkaline phosphatase level was also elevated.

Proteinuria and hematuria are associated with glomerulonephritis and result from damage of the glomeruli with the resultant increased permeability. Albumin is a sensitive indicator of CKD related to diabetes, glomerular disease, and hypertension. Although J.M. had no history of kidney disease, he did have hypertension, which can damage the glomeruli. Glomerular damage was evidenced by the 4+ protein and 2+ hematuria.

Uric acid is an end product of purine metabolism that is filtered in the glomeruli and secreted into the distal tubule. Impaired glomerular function results in decreased excretion of uric acid by the kidney and may result in the development of gouty arthritis with deposits of uric acid in joints or soft tissue. J.M.’s uric acid level was 1142 μmol/L (19.2 mg/dL). This elevated level of uric acid increased J.M.’s risk of gout developing with symptoms such as joint pain, redness, and swelling, particularly in the great toe.

Alterations in Metabolic/Endocrine Functions

Anemia results from several factors in patients with CKD. The peritubular capillary endothelium in the kidneys produces erythropoietin, which is needed to stimulate bone marrow to release red blood cells. In addition, uremia inactivates erythropoietin. Failure of this mechanism results in a normochromic, normocytic anemia. Uremia can also contribute to anemia by shortening the life span of the red blood cells. Finally, the low hemoglobin level contributes to acidosis, because less hemoglobin is available in the body to buffer acids. Additionally, uremia causes impaired platelet aggregation, increasing the potential for bleeding. J.M.’s hemoglobin level was 83 g/L, his hematocrit was 0.252, and his platelet counts were adequate. The decreased hemoglobin level and hematocrit could have caused signs and symptoms of anemia, and although his platelet count was adequate, the platelets would not function effectively, increasing the risk for bleeding.

Renin is released in response to changes in intravascular pressure or sympathetic stimulation. The resultant stimulation of the renin-angiotensin-aldosterone system contributes to the retention of water and elevated blood pressure. Renin is an enzyme released from the juxtaglomerular cells in response to decreases in blood flow to the kidney, changes in tubular fluid composition, or stimulation by the sympathetic nervous system. Renin then acts on angiotensinogen, a plasma protein, and converts it to angiotensin I. Angiotensin I in turn is converted to angiotensin II by angiotensin-converting enzyme in the lungs. Angiotensin II produces 2 outcomes. The first is a short-acting vasoconstriction. The second action is an increase in blood pressure through stimulation of the adrenal cortex, which releases aldosterone, causing sodium reabsorption and concomitant water reabsorption by the kidneys. J.M.’s blood pressure was 221/142 mm Hg. It was unknown on admission whether J.M. had a primary hypertension that had led to kidney failure or if the kidney failure had contributed to the hypertension. The retention of water and production of angiotensin certainly contributed to the hypertension and the cardiovascular and respiratory findings from fluid overload.

Assessment Findings

Because the pathophysiological changes associated with CKD affect every body system (Figure 2), a thorough nursing assessment of patients with CKD is essential. A systems approach to assessment is used here.

Cardiovascular

Hypertension is a result of increased fluid retention and stimulation of the renin-angiotensin-aldosterone system. In addition, hypertension can lead to the development of CKD. For J.M., it was unknown whether the hypertension contributed to the renal failure or the renal failure contributed to the
hypertension. J.M.’s clinical assessment revealed left ventricular hypertrophy and a strain pattern on the electrocardiogram, indicating longstanding left ventricular failure. Auscultation of heart sounds revealed an S3 (fluid overload) and S4 gallop (decreased compliance and hypertension). Funduscopic examination showed arteriovenous nicking and cotton wool hemorrhages with exudates (hypertensive retinopathy), all indicating significant hypertension. The sinus tachycardia could have been a compensatory mechanism for the decreased PaO$_2$, the anemia, the metabolic acidosis, or the fluid overload. Uremic pericarditis, another manifestation of ESRD, often develops from the accumulation of toxins; J.M. did not have this manifestation. Electrolyte imbalances such as hyperkalemia and hypocalcemia can also lead to dysrhythmias. The focus of the nursing assessment is peripheral edema, circulatory overload evidenced by congestive heart failure and pulmonary edema, cardiac dysrhythmias, hypertension, and electrolyte levels.

**Respiratory**

An increased respiratory rate may result from fluid overload, as a compensatory mechanism for metabolic acidosis, or from decreased PaO$_2$. Although not identified as Kussmaul respirations, deep breaths associated with metabolic acidosis occur as a compensatory mechanism to eliminate carbon dioxide in an attempt to reestablish normal pH. Fluid overload with pulmonary congestion was manifested in J.M. by crackles, decreased PaO$_2$, and increased respiratory rate. J.M.’s acid-base results were described previously. The focus of the nursing assessment is breath sounds, respiratory rate and pattern.

**Gastrointestinal**

Anorexia, weight loss, nausea, and vomiting are frequent findings in patients with CKD, although J.M. said that he did not have these signs and symptoms. Halitosis, a metallic taste in the mouth, and ulcers in the mouth may occur because bacteria in the mouth break down urea into ammonia. Gastrointestinal bleeding from altered platelet function and increased gastric acid secretion from increased release of parathyroid hormone may occur. The focus of the nursing assessment includes inspecting oral mucous membranes, monitoring weight, checking stool for...
occult blood, and noting breath odor.

**Neurological**

Central nervous system findings in patients with CKD can range from confusion and difficulty concentrating to seizures and coma. These findings are described as uremic encephalopathy. Impaired thinking processes are sometimes described as “BUN [blood urea nitrogen] blunting.” The effects of CKD on the peripheral nervous system result in peripheral neuropathy, particularly affecting the lower extremities. The cause of these neurological effects is thought to be atrophy and demyelination of the nerves as a result of uremic toxins and electrolyte imbalances. Early findings include restless leg syndrome progressing to pain, sensations of tightness in the legs, and pain in a stocking-like pattern. Finally, motor function may be impaired with resultant changes in gait and fine motor movement. J.M. had none of these neurological signs or symptoms. The focus of the nursing assessment is mental status and motor and sensory function.

**Integumentary**

Pruritus often occurs in patients with CKD because of the excretion of waste products and phosphate through the skin. The skin is often dry because of decreased activity of sweat glands and oil glands, and the skin may undergo changes in color, from pallor related to the anemia to a yellow-brown or gray aspect from urochrome, a urinary pigment. The nails and hair may become brittle. Bruises may also occur because of impaired platelet function and increased capillary fragility. J.M. had none of these manifestations. The focus of the nursing assessment includes inspection of the skin for color changes or impaired integrity as a result of scratching of the skin by the patients.

**Musculoskeletal**

Renal osteodystrophy results from the loss of calcium in the bones and ineffective conversion of vitamin D to allow absorption of calcium. Three bone changes are associated with this syndrome: (1) osteomalacia due to inadequate absorption of calcium from the gastrointestinal tract, (2) osteitis fibrosa or bone demineralization due to increased parathyroid hormone, and (3) osteosclerosis, which is manifested as bands of increased and decreased bone density in the vertebrae. J.M. had no indications of skeletal deficiencies, although his calcium level was 2.05 mmol/L (8.2 mg/dL). The focus of the nursing assessment is monitoring calcium and phosphorus levels. Signs and symptoms of hypocalcemia include neuromuscular irritability manifested by paresthesia and tetany, which is assessed by testing for the Chvostek sign and the Trousseau phenomenon, muscle cramps, hypotension, and prolonged QT interval.

**Hematological**

Decreased erythropoietin levels result in anemia. J.M.’s laboratory results showed decreases in hemoglobin level and hematocrit. The focus of the nursing assessment is detection of signs and symptoms of anemia, including pallor, fatigue, shortness of breath, and tachycardia, and on laboratory evaluation of hematocrit and hemoglobin and iron levels.

**Immunological**

Increased levels of uremic toxins can lead to impaired immune and inflammatory responses with resultant defects in granulocytes, impaired B- and T-cell functioning, and impaired phagocytosis. The focus of the nursing assessment is examination for signs or symptoms of an impaired inflammatory and infectious response. Infection is a common occurrence in patients with CKD that often results in hospitalization and death.

**Renal**

In patients with CKD, urinary signs and symptoms are related to fluid balance; as GFR decreases, urine output decreases. Retention of waste products such as urea nitrogen and creatinine leads to azotemia, whereas uric acid retention may lead to gout. Proteinuria and hematuria were discussed previously. The focus of the nursing assessment is fluid balance (intake and output, daily weight, edema) and monitoring of laboratory results.

**Current Standards of Care**

The clinical manifestations due to the pathophysiological changes of CKD create a need for immediate interventions and constant monitoring. Because J.M.’s serum level of urea nitrogen was 67.47 mmol/L (189 mg/dL) and his creatinine level was 1909.4 μmol/L (21.6 mg/dL), the immediate clinical action required for J.M., according to the KDOQI clinical action plan for CKD, was renal replacement therapy started as soon as possible. After the initial assessment was completed in the emergency department, a nephrologist was consulted and a double-
A vascular access for arterial flow and venous return is needed for the dialysis procedure. Of 3 access types, 1 is temporary and 2 are permanent (Table 3). The central venous catheter is the least desirable because complications are common, particularly infection; however, because of the emergent nature of J.M.’s situation, a temporary access was required. The right internal jugular vein is the preferred site, although the femoral site can cause venous stenosis and thrombosis, interfering with any future access for fistulas or grafts; therefore, it should be used only when the femoral or jugular sites are not accessible.

A temporary access is placed in a large central vein and can be used for as long as 3 months. The procedure is done by using sterile technique in an angiography suite, procedure, or operating suite; however, because of J.M.’s critical condition, the procedure was done at the bedside. The right jugular vein was used, and a 20-cm–long catheter was inserted into the vein and then sutured in place by using “suture wings.” Once the catheter was in place, while maintaining aseptic technique, a nurse instilled heparin into each lumen, per facility protocol, to equal the total volume of each lumen (the lumen volume is printed on each lumen, per facility protocol, to equal the total volume of each lumen). A catheter cap was then placed on each lumen, and a dressing was placed over the site. J.M. was now ready for dialysis after placement of the catheter had been verified with a chest radiograph.

Several brands and types of dialysis catheters are available; those commonly used are double or triple lumen. The triple lumen gives an added lumen for drug therapy, nutritional support, and/or obtaining blood samples. It is important to know which site will be used, as well as the size and body type of the patient, to determine what size and length catheter is needed. If the jugular or the subclavian site is used, catheters must be longer than if the femoral site is chosen, because of the positioning of the catheter. The dialysis catheter has a large lumen, 11.5F or 12F, which allows a smooth flow of blood throughout the hemodialysis procedure.

Additional immediate interventions initiated because of J.M.’s critical condition included oxygen via nasal cannula at 4L/min, cardiac monitoring to assess for dysrhythmias, insertion of a peripheral intravenous catheter and an indwelling Foley catheter. After his initial dialysis, J.M. was placed on a 1000-mL fluid restriction and a renal diet of

<table>
<thead>
<tr>
<th>Access site for hemodialysis</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arteriovenous fistula: A permanent access that is created surgically by connecting an artery to a vein</td>
<td>Preferred route for dialysis; once matured, has good blood flow, few problems It is long lasting and is associated with fewer complications than are other accesses</td>
<td>Long maturation time (3-4 months until it can be used for dialysis) May not enlarge enough to provide a good blood flow for dialysis</td>
</tr>
<tr>
<td>Arteriovenous graft: A permanent access created surgically by using a synthetic material tunneled under the skin One end of graft is connected to an artery; the other, to a vein</td>
<td>Shorter maturation time than fistula; site can be used in 3-6 weeks</td>
<td>Higher incidence of thrombosis and clotting than with arteriovenous fistula Higher incidence of stenosis than with fistula Involves use of synthetic material, creating the potential for an allergic response</td>
</tr>
<tr>
<td>Catheter: A temporary access in which a catheter is placed into a large central vein such as the internal jugular vein</td>
<td>No maturation time Catheter can be used immediately after placement and is often used in emergent situations</td>
<td>Increased risk of infection in catheter, at exit site, and in bloodstream Decreased flow rates with catheter lead to low urea reduction rate Potential for discomfort at catheter exit site and development of poor body image</td>
</tr>
</tbody>
</table>
60 g of protein, 2 g potassium, and 2 g sodium. Fluid volume intake is based on 500 mL/day (insensible fluid loss) plus fluid equal to the urine output of the preceding 24 hours.8

Once a patient’s condition stabilizes, after the diagnosis of CKD is made and initial treatment has begun, the patient is referred to a surgeon for establishment of a permanent vascular access. The National Kidney Foundation18 recommends the arteriovenous fistula as the preferred type of access; this access is associated with fewer complications and provides longer trouble-free use than does an arteriovenous graft or a catheter. The catheter is the access least recommended because of the frequent complications associated with its use. J.M. had an arteriovenous fistula placed 8 days after admission. Nursing assessment after a fistula is placed focuses on patency of the fistula, determining by palpating a thrill and auscultating a bruit.

**Expected Outcomes**

On the basis of the KDOQI guidelines, the focus of treatment in CKD is specific therapy, dialysis in J.M.’s case, and treatment of comorbid conditions.19 Particularly for J.M., the clinical findings such as hypertension, anemia, calcium and phosphorus balance, and nutritional status required treatment.

The expected outcomes and nursing considerations for J.M. would include the following:

- Adequate hemodialysis20 is determined by measurement of levels of nitrogenous waste products (serum levels of urea nitrogen and creatinine) and the urea reduction ratio, in which the measurements from before and after dialysis are entered into a mathematical formula. Adequate dialysis will also result in correction of acid-base imbalance, fluid volume overload, and electrolyte imbalances (particularly sodium and potassium). Results of J.M.’s laboratory tests after dialysis are found in Table 2. J.M. initially received daily dialysis for 3 days. Correction of the fluid imbalance in turn will correct impaired gas exchange. Nursing implications include fluid restriction, monitoring, and daily weight. The focus of educating patients is teaching about foods that are high in potassium and the importance of potassium restriction in preventing cardiac dysrhythmias. Disregarding potassium restriction can be fatal for a patient with CKD. Immediate referral to a dietician is also appropriate because not only potassium but also sodium and phosphorus are restricted on a renal diet.

- Blood pressure within normal limits21 is related to several interventions, including adequate hemodialysis and fluid restriction. The most important nondrug management for hypertension is fluid removal and restriction of sodium and fluid in the diet. Education of patients focuses on teaching about foods high in sodium that must be avoided and management of fluid intake. Pharmacological intervention includes the use of angiotensin-converting enzyme inhibitors, which decrease not only systemic blood pressure but also intraglomerular pressure by dilating the efferent arteriole.8 Angiotensin-converting enzyme inhibitors must be used with caution because a concomitant increase in serum potassium levels may occur. Calcium channel blockers are also effective in decreasing blood pressure through systemic vasodilatation. J.M. was treated with captopril (Capoten) and nifedipine (Procardia). For patients on dialysis, antihypertensive medications should be given after hemodialysis to prevent hypotension.

- Prevention of other cardiovascular diseases22 is accomplished through control of hypertension and fluid volume, physical exercise, and prevention of end-organ damage. Cardiovascular disease is the leading cause of death in patients with ESRD.22 A physician-approved exercise program for patients with CKD provides the positive benefits of exercise such as decreasing blood pressure, cholesterol and triglyceride levels, and insomnia and the obvious benefit of maintaining weight control. Nursing interventions focus on monitoring weight loss/gain, blood pressure, and peripheral edema and on educating patients about diet and exercise.

- Target hemoglobin and hematocrit levels can be reached23 through the use of iron supplements, multivitamins, and epoetin alfa (Epoegen), which stimulates production of red blood cells. Nursing interventions include monitoring hemoglobin levels and hematocrit and assessing patients for clinical findings of anemia.

- Prevention of bony changes24 is accomplished through the management of calcium and phosphorus levels. In addition to dietary management, phosphate binders (aluminum hydroxide) or calcium salts (PhosLo) may be used to reach this outcome. Because medications are the primary method of removing phosphorus from the body (dialysis removes very little), the necessity of taking the phosphate binder with food to be effective is a significant aspect of patients’ education. Moni-
toring calcium and phosphorus levels would also be part of the nursing role.

- Adequate nutrition (based on albumin level)\(^5\) is managed through dietary regulation. Proteins of high biological value are essential because total protein intake is restricted. Adequate protein levels result in maintenance of fluid balance, healing and maintenance of skin integrity, and finally maintenance of immune function. Providing dietary education on appropriate protein foods and serving size is an important activity of nurses and/or dieticians, along with monitoring albumin levels.

**Conclusion**

The significant role of critical care nurses in providing care to patients with CKD is clear. A thorough assessment of all body systems is essential in evaluating each patient. This assessment will enable the early detection of systemic alterations related to CKD and the implementation of appropriate interventions. Education of patients about the management of CKD and continued evaluation of patients’ outcomes are also essential so that critical care nurses can determine the effectiveness of interventions.

**Acknowledgments**

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**References**

1. Why is chronic kidney disease (CKD) a public health issue? 
   a. Because of increasing incidence and the high cost of interventions
   b. Because of increasing cost of insurance premiums and loss of insurance
   c. Because of limited access to dialysis centers and limited public education of the disease
   d. Because of limited access to dialysis centers and the high cost of interventions

2. Which of the following are 3 risk factors for end-stage renal disease according to Healthy People 2010? 
   a. Hypertension, proteinuria, and family history
   b. Diabetes mellitus, coronary artery disease, and smoking
   c. Obesity, smoking, and proteinuria
   d. Increasing age, hypertension, and hypercholesterolemia

3. In relationship to cardiac output, what percentage of blood is filtered through the nephrons per minute in normal homeostasis? 
   a. 20% to 25%
   b. 35% to 45%
   c. 45% to 50%
   d. 75% to 80%

4. Which of the following best describes the response to decreased glomerular filtration rate? 
   a. Cortisol is released from the adrenal medulla, resulting in reabsorption of sodium and water.
   b. Aldosterone is released from the adrenal cortex, resulting in excretion of water, sodium, and potassium.
   c. Antidiuretic hormone is released resulting in reabsorption of water, sodium, and potassium.
   d. Aldosterone is released from the adrenal cortex, resulting in reabsorption of water, sodium, and potassium.

5. Which of the following best illustrates the correct anion gap equation? 
   a. Anion gap = Na+ + (Cl- + HCO3-)
   b. Anion gap = Na+ - (Cl- + HCO3-)
   c. Anion gap = 6.1 + log (HCO3- / [0.03 x PCO2])
   d. Anion gap = Cl- + (Na+ / HCO3-)

6. Which of the following best describes the normal excretion of potassium by the kidneys? 
   a. 20 to 40 mmol daily
   b. 40 to 60 mmol daily
   c. 30 to 50 mmol daily
   d. 80 to 100 mmol daily

7. Which of the following best describes the function of the endocrine system in CKD in relationship to hypocalcemia? 
   a. The thyroid gland increases it secretion of thyroid hormone, stimulating the release of cortisol into the blood.
   b. The thyroid gland decreases secretion of thyroid hormone, decreasing the amount of calcium excreted.
   c. The parathyroid glands secrete parathyroid hormone, decreasing the amount of calcium released from the bone into the vascular system.
   d. The parathyroid glands secrete parathyroid hormone, increasing the amount of calcium released from the bone in the vascular system.

8. Which of the following cardiovascular changes do critical care nurses need to monitor during their assessment of the patient with CKD? 
   a. Peripheral edema, cardiac dysrhythmia, and electrolyte levels
   b. Gastroesophageal reflux disease, pulmonary edema, and hypotension
   c. Dehydration, gastrointestinal bleeding, and hypotension
   d. Endocarditis, hyperglycemia, and congestive heart failure

9. Which of the following is associated with renal osteodystrophy? 
   a. Osteomalacia, ostetis fibrosa, and osteosclerosis
   b. Periostitis, osteoporosis, and osteosclerosis
   c. Osteomatosis, osteomyelitis, and dysostosis
   d. Dermatitis, desmosis, and myelolysis

10. As blood completes the circuit during dialysis, approximately how much blood is outside the body at any time? 
    a. 75 to 150 mL
    b. 100 to 300 mL
    c. 200 to 300 mL
    d. 250 to 400 mL

11. The National Kidney Foundation recommends which type of access for permanent vascular access? 
    a. Infusaport
    b. Arteriovenous graft
    c. Arteriovenous fistula
    d. Arteriovenous fistula

Test answers: Mark only one box for your answer to each question. You may photocopy this form.

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