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Kidney Care in the Hospitalized Patient Primer

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Introduction/ Purpose Statement

Patients with renal failure are often challenging and complex to manage in a hospital setting. The purpose of this home study is to review the basic anatomy and physiology of the renal system, and discuss renal failure, laboratory tests, cultural concerns and the psychosocial implications associated with this complicated illness.

Target Audience

This home study was designed for nurses with no familiarity with kidney care in the hospitalized patient; however, all health care professionals are invited to complete this packet.

Content Objectives

1. Describe normal kidney function.
2. Describe the pathophysiologic process of renal insufficiency and failure.
3. Identify acid-base disturbances based on blood gas analysis.
4. Identify common laboratory tests and imaging studies utilized in renal failure.
5. Identify the common nutritional management guidelines for renal failure.
6. Discuss the psychosocial and cultural issues that are frequently associated with renal failure.

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Introduction

Patients with renal failure are often challenging and complex to manage in a hospital setting. The purpose of this home study is to review the basic anatomy and physiology of the renal system, and discuss renal failure, laboratory tests, cultural concerns and the psychosocial implications associated with this complicated illness.

Normal Kidney Function

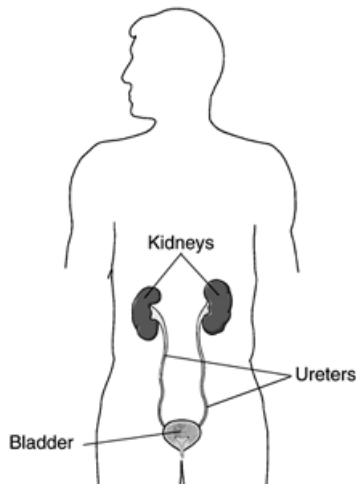
Normal kidneys act as a filtering system. As blood passes through the kidneys, the body's waste products and excess fluid are removed. Waste products and excess fluid are excreted through the ureters and the bladder as urine.

Healthy kidneys provide the body with many important functions. First, they remove the end products of metabolism from the blood, such as urea, creatinine and uric acid.

The kidney also regulates vascular and extravascular volume by controlling the amount of water that is excreted. This in turn regulates the electrolyte balance of the body fluids, as well as the body's acid-base balance.

Healthy kidneys also regulate the secretion of certain hormones. For example, blood pressure is regulated by the production of the hormone, renin, in the kidneys. Other examples include erythropoietin which regulates bone marrow production of red blood cells and eicosanoid which helps regulate renal blood flow is secreted by the kidneys. Lastly, vitamin D is synthesized into its active form in the kidneys.

Image from: www.niddk.nih.gov



What anatomical structures are involved in renal failure?

Renal Blood Flow

The renal artery divides into segmental arteries which further divide into interlobar arteries which travel alongside the pyramids into the cortex. At the junction of the cortex and medulla, the interlobar arteries bend at right angles; arcuate arteries begin at this point. The arcuate arteries branch into interlobular arteries which travel further into the cortex. In the cortex, the interlobular arteries divide into afferent arterioles. Each afferent arteriole divides into a tuft of about 12 capillaries called the glomerulus. The glomerular capillaries rejoin to form the efferent arteriole, which then becomes the peritubular capillary network. The microscopic circulation begins with the afferent arteriole.

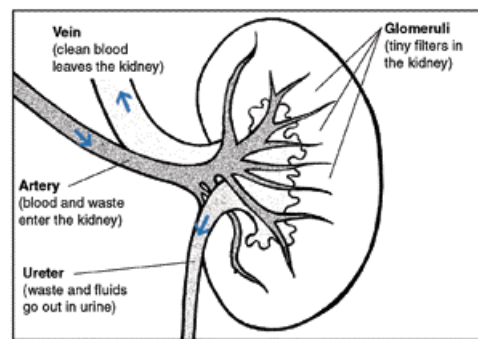


Image from: www.niddk.nih.gov

Lymphatic Drainage

Lymphatic drainage from the kidneys and upper ureters flows into the aortic and paraaortic lymph nodes and then into the thoracic lymph duct, which communicates with the systemic blood circulation.

Nephron

The nephron is the structural and functional unit of the kidney. The main function of the nephron is to filter the blood, reabsorbing what is needed and excreting the rest as urine. Each nephron consists of two components. The first component is the vascular component, which includes the afferent arteriole, glomerulus, efferent arteriole, peritubular capillary network, and vasa recta.

The second component is the tubular component, which includes Bowman's capsule, proximal convoluted tubule (PCT), descending and ascending limbs of the loop of Henle, distal convoluted tubule (DCT), cortical collecting tubule, and medullary collecting duct.

Nephron: Vascular Component

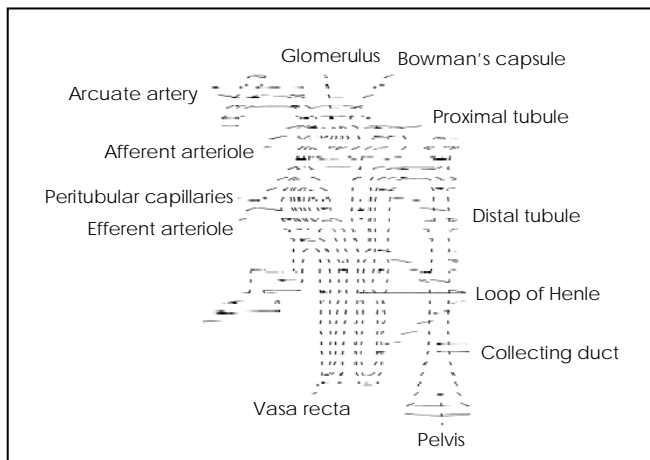
Each afferent arteriole divides into a tuft of capillaries called the glomerulus which protrudes into Bowman's capsule. Blood in the glomerular capsule is separated from fluid in Bowman's capsule only by its capillary

membranes. Thus water and solutes filter from glomerular capillaries into Bowman's capsule. The glomerular capillary permeability and selectivity are related to the structure of the glomerular capillary membrane. Glomerular capillaries reunite to form the efferent arteriole, which has two branches, peritubular capillaries and vasa recta capillaries.

Nephron: Tubular Component

Bowman's capsule is a concave sac that surrounds the glomerular capillaries. One side Bowman's capsule shares cells with the glomerulus and on the other side it opens to the proximal convoluted tubule (PCT). The PCT straightens and narrows to become the descending limb of the loop of Henle. The loop of Henle makes a hairpin turn and ascends through the cortex parallel with the descending limb.

The loop of Henle empties into the distal convoluted tubule (DCT). Several DCTs coalesce to form the cortical collecting tubule, which descends downward through the cortex into the medulla where it is then called first the outer and then the inner medullary collecting duct. Several collecting ducts join and open through the papilla of the pyramid into a minor calyx of the ureter.



How do all these anatomical structures work together?

The kidneys have an extraordinarily unique system of perfusion. The renal system receives 20-25% of the cardiac output (about 1200 ml/min) at any given time. The descending aorta is the first blood vessel involved in delivering arterial blood to the kidneys. The aorta gives off a branch called the renal artery, which enters the kidney beside the ureter.

The afferent arteriole branches to form the capillary network. This network sits inside "Bowman's capsule." The afferent (incoming) and efferent (outgoing) capillaries, endothelial membrane and Bowman's capsule make up the glomerulus.

Blood flows through the kidneys at a regulated rate; the renal vasculature has an autoregulating mechanism. As the blood flows through the glomeruli, osmotic, hydrostatic, and electrical gradients will determine the elements to go into the urine. Blood will exit the glomerulus through the efferent arteriole after water products and/or water have been excreted. Filtered blood exits through the interlobular vein into the arcuate vein, the interlobar vein and finally into the renal vein.

Disorders of the Kidney

Definitions

- *Glomerular filtration rate (GFR)*: Rate at which solutes are filtered from the glomerulus into the nephron. Measured by the creatinine clearance.
- *Acute renal failure (ARF)*: A broad term used to denote a rapid decrease in glomerular filtration rate (GFR) as a result of insult to renal parenchyma.
- *Acute tubular necrosis (ATN)*: A specific form of ARF in which insult to renal parenchyma (e.g., renal ischemia, hemorrhage, drug effects) results in necrosis of renal tubules. There is an abrupt decrease in GFR. Regeneration of renal function can take weeks to months following removal of the insult.
- *Azotemia*: Presence of nitrogenous waste products (urea, creatinine) in the blood at elevated levels.
- *Uremia*: Toxic condition in which patient develops clinical symptoms resulting from nitrogenous waste build-up.

Classification of Acute Renal Failure (ARF)

There are three types of conditions which may cause acute renal failure:

1. Pre-renal
2. Intra-renal
3. Post-renal

Pre-Renal ARF

In acute renal failure caused by pre-renal etiologies, there is a decrease in renal blood flow that results in decreased GFR. Decreased renal perfusion leads to:

- Afferent (incoming) arteriolar dilatation that is prostaglandin dependent.
- Efferent (outgoing) arteriolar constriction that is angiotensin dependent. This is an autoregulatory response the body makes to help maintain local blood flow in the kidney.

There are three major causes of pre-renal ARF:

1. *Decreased intravascular volume* from hemorrhage, sepsis, and extra-cellular volume depletion/dehydration
2. *Cardiac dysfunction with decreased cardiac output*, such as MI, arrhythmias, tamponade, cardiogenic shock, CHF, and afterload reduction therapy
3. *Obstruction of flow to the kidney*, such as by a renal artery embolus

Interventions for patients with Pre-Renal ARF include:

1. Avoid non-steroidal anti-inflammatory (NSAIDs), angiotensin converting enzymes inhibitors (ACE-inhibitors), and angiotensin receptor blockers (ARB's).
2. Volume resuscitation- crystalloids (normal saline) are first line agents.

Intra-Renal ARF

A severe parenchymal insult from disease or nephrotoxic agent resulting in damage to glomerulus and/or tubules may cause acute renal failure. There are many etiologies of this type of ARF, including:

1. *Glomerulonephritis* (5-10%)
 2. *Acute Malignant Hypertension*
 3. *Multiple Myeloma*
 3. *Vasculitic diseases*, such as polyarteritis nodosa, Wegener's granulomatosis, scleroderma, lupus
 4. *Acute tubular necrosis* from ischemia, nephrotoxic agents (drugs, contrast media, heavy metals), myoglobins and hemoglobin in the urine, rhabdomyolysis
- a) *Heme pigment ATN*, which is an episode of hemolysis resulting in the release of heme pigment, seen in transfusion reaction, venous snake/insect bites, extracorporeal

circulation, and faulty heart valves.

- b) *Nephrotoxic ATN*: drugs implicated in nephrotoxicity:

Drugs That May Cause Nephrotoxicity	
Arsenic	Contrast media
Acetaminophen	Dextran
Cyclosporine	Ethylene Glycol
Mercury	Lead
Aminoglycosides	Lithium
Amphotericin B	Methotrexate
Analgesics	NSAIDs
Bismuth	Penicillins
Cephalothin	Plicamycin
Cisplatin	Tetracyclines

Interventions for Intra-Renal ARF depend on the cause. For example, treating an acute ingestion of acetaminophen with acetylcystine is used to minimize the likelihood of liver and kidney problems associated with an overdose of this medication.

Post-Renal ARF

Post-renal ARF is caused by an obstruction anywhere along the urinary tract (renal pelvis to urethra). This obstruction results in the blockage of flow of urine and ultimately, damage to renal parenchyma secondary to hydronephrosis.

Causes of post-renal failure include:

1. ureteral and pelvic blood clots, stones, and fungus balls;
2. ureteral and pelvic malignancy and retroperitoneal fibrosis;
3. bladder stones, blood clots, carcinoma;
4. urethral strictures or prostatic hypertrophy
5. neurogenic bladder with inadequate emptying
6. crystal induced from uric acid crystals, during chemotherapy for malignancies with high cell turnover (leukemia, lymphoma, sarcoma)

Interventions for Post-Renal ARF consists of prompt identification of the etiology. Treatment is dependent on the etiology and include placing a foley catheter or percutaneous nephrostomy tube and treating the infection or obstruction.

Acid-Base Balance and the ABG

The kidneys and the lungs are crucial in the maintenance of a normal acid-base balance. This section discusses

how these systems work in cooperation to regulate the body's acid-base balance. Since it is impossible to evaluate acid-base balance as it relates to kidney function in isolation, this section also describes the effect respiratory system has on maintaining a normal acid-base balance.

pH

The pH on the ABG is the inverse logarithmic number of hydrogen ions in the blood. **Normally, the pH should be 7.35-7.45.** If the number of hydrogen ions rises, the blood is more acidotic. If the number of hydrogen ions falls, the blood is more alkalotic.

Maintaining a Normal pH

The body really likes to keep a normal pH. In order to maintain the blood pH between 7.35-7.45, the body has a buffering system. There are two major chemical buffers, regulated by the respiratory and renal systems, in the body:

- carbon dioxide (CO₂): **the normal PaCO₂ on the ABG is 35 - 45 mm/Hg**
- bicarbonate (HCO₃⁻): **the normal HCO₃⁻ level on the ABG is 22 - 26 mEq/L**

The respiratory system responds within 1-3 minutes to changes in acid-base balance. If the chemo-receptors sense too many hydrogen ions (acidosis), it will stimulate the respiratory center to breathe faster and deeper – to “**blow off**” CO₂. If the chemoreceptors sense too few hydrogen ions (alkalosis), it will depress the respiratory center to **keep** CO₂.

The kidneys compensate over 24-48 hours to correct imbalances. If the kidneys see acidosis, they will retain, regenerate or synthesize HCO₃⁻ and excrete H⁺. If the kidneys see alkalosis, they will excrete HCO₃⁻ and retain H⁺.

If the body sees acidosis, it will:

Increase the respiratory rate to blow off CO₂

Retain, regenerate or make bicarbonate

Excrete hydrogen ions

If the body sees alkalosis, it will:

Decrease the respiratory rate to keep CO₂

Excrete bicarbonate

Retain hydrogen ions

When there is an acid-base disturbance and either the lungs or kidneys react, it is called **compensation**. **Compensation** can be complete or partial. The body will compensate so that the pH reaches the edges of normal. For example, if the pH is 7.10 (acidosis), the body will try to compensate so that the pH will reach 7.35, not greater than 7.35. Partial compensation means that the pH has not reached a normal level.

Respiratory Acidosis

In **acute** respiratory acidosis, the lungs don't get rid of enough CO₂.

Causes: oversedation, head trauma, respiratory and cardiac arrest

What to look for: ↑ PaCO₂, ↓ pH, normal HCO₃⁻

Examples:

- pH 7.29, PaCO₂ 57, HCO₃⁻ 28
- pH 7.06, PaCO₂ 98, HCO₃⁻ 28

In **compensated** respiratory acidosis, the lungs still don't get rid of enough CO₂, but the kidneys have had enough time to save bicarbonate.

Causes: COPD, spinal cord injury, respiratory muscle paralysis

What to look for: ↑ PaCO₂, ↓ pH, ↑ HCO₃⁻

Examples:

- pH 7.31, PaCO₂ 76, HCO₃⁻ 39
- pH 7.34, PaCO₂ 60, HCO₃⁻ 33

Respiratory Alkalosis

In **acute** respiratory alkalosis, the lungs are “blowing off” too much CO₂, leading to an increased pH.

Causes: stress, pain, fever, and hypoxemia

What to look for: ↓ PaCO₂, ↑ pH, normal HCO₃⁻

Examples:

- pH 7.52, PaCO₂ 27, HCO₃⁻ 22
- pH 7.65, PaCO₂ 23, HCO₃⁻ 24

Compensated respiratory alkalosis occurs when the lungs "blow off" too much CO₂, but the kidneys have time to excrete bicarbonate and save hydrogen ions.

Causes: uncommon, but can occur in the patient with neurological damage

What to look for: ↓ PaCO₂, ↑ pH, ↓ HCO₃⁻

Examples:

- pH 7.49, PaCO₂ 16, HCO₃⁻ 11
- pH 7.45, PaCO₂ 23, HCO₃⁻ 16

Metabolic Acidosis

Metabolic acidosis occurs where there is either too much acid (such as in shock, hypoxemia, diabetes, overdose, renal failure) in the system, or when there is a loss of bicarbonate (diarrhea, NG suction, renal tubular acidosis).

Acute metabolic acidosis without compensation may be seen in the mechanically ventilated, sedated, or comatose patient. Because of the altered mental status, there is no compensatory response by the respiratory system.

What to look for: normal PaCO₂, ↓ pH, ↓ HCO₃⁻

Examples:

- pH 7.05, PaCO₂ 37, HCO₃⁻ 7
- pH 7.23, PaCO₂ 40, HCO₃⁻ 12

Compensated metabolic acidosis is much more common. The respiratory rate and depth increases to blow off CO₂. There is a limit to how much the respiratory system can compensate. The PaCO₂ may be quite low, but it is still not able to bring the pH back to normal.

What to look for: ↓ PaCO₂, ↓ pH, ↓ HCO₃⁻

Examples:

- pH 7.19, PaCO₂ 22, HCO₃⁻ 8
- pH 6.96, PaCO₂ 9, HCO₃⁻ 2

Metabolic Alkalosis

In metabolic alkalosis, there is a gain of base or increased loss of acid, resulting in an increased pH. If there is a gain of base, such as in sodium bicarbonate (baking soda) ingestion or administration of NaHCO₃ during CPR, the HCO₃⁻ will be elevated. If there is loss of an acid, such as in vomiting or NG suction, the HCO₃⁻ will be normal in the acute phase.

Acute metabolic alkalosis is uncommon, but can be seen if the patient is not neurologically intact and is unable to increase the respiratory rate.

What to look for: normal PaCO₂, ↑ pH, ↑ HCO₃⁻

Examples:

- Gain of a base: pH 7.55, PaCO₂ 40, HCO₃⁻ 42
- Loss of acid: pH 7.52, PaCO₂ 37, HCO₃⁻ 28

Compensated metabolic alkalosis can look like:

What to look for: ↑ PaCO₂, ↑ pH, ↑ HCO₃⁻

Examples:

- Gain of a base: pH 7.47, PaCO₂ 46, HCO₃⁻ 42
- Loss of acid: pH 7.46, PaCO₂ 44, HCO₃⁻ 26

There is also a limit to the compensation of the respiratory system in metabolic alkalosis. The body will not tolerate CO₂ levels over 50-55 mm Hg, and will increase the rate and depth of breathing after that point.

Now, you might notice that metabolic alkalosis from loss of an acid and respiratory acidosis look a lot alike. Here's how to tell the difference. There is an increase in CO₂ in both metabolic alkalosis and respiratory acidosis, but the pH will be relatively normal. In compensated respiratory acidosis, though, the pH will be on the **low** side of normal, not the high, and the HCO₃⁻ level will be high, not normal.

Analyzing the ABG

1. Look at the PaO₂.

2. Look at the pH.

- a) Is it normal?
- b) Is it low normal or high normal? Look for changes in the PaCO₂ and HCO₃⁻ to see if there is compensation for a problem.
- c) If it is low (less than 7.35), the patient is in acidosis.
- d) If it is high (more than 7.45), the patient is in alkalosis.

3. Look at the PaCO₂.

- a) The pH and PaCO₂ have a "teeter-totter" relationship. If the problem is **respiratory**, one will be up, and the other will be down.
- b) If the pH and PaCO₂ are both up or both down, the problem is **metabolic**. The teeter-totter isn't there, so it can't be a primary respiratory problem, instead, it is a metabolic problem with respiratory compensation.

4. Look at the HCO₃⁻.

- a) The pH and HCO₃⁻ go up and down together in a **metabolic** problem.
- b) If the pH and the HCO₃⁻ are opposite (one is up and the other is down), the problem is primarily respiratory, and the HCO₃⁻ is trying to compensate.

Examples:

- 1) *pH 7.01; PaCO₂ 69; HCO₃⁻ 24*
 - a) The pH is very low, so it is acidosis.
 - b) The PaCO₂ is high, making a teeter-totter with the pH, so it is a respiratory problem.
 - c) The HCO₃⁻ is normal, so there is no compensation.
 - d) Respiratory acidosis without compensation.
- 2) *pH 7.33; PaCO₂ 72; HCO₃⁻ 36*
 - a) The pH is low, so it is acidosis.
 - b) The PaCO₂ is high, making a teeter-totter with the pH, so it is a respiratory problem.
 - c) The HCO₃⁻ is high, so there is compensation, but not enough to bring the pH to normal.
 - d) Respiratory acidosis with partial compensation.
- 3) *pH 6.99; PaCO₂ 20; HCO₃⁻ 2*
 - a) The pH is very low, so it is acidosis.
 - b) The PaCO₂ is low; it is not a teeter-totter with the pH, so it is a metabolic problem with respiratory compensation.
 - c) The HCO₃⁻ is low, confirming a metabolic problem.
 - d) Metabolic acidosis with partial compensation.
- 4) *pH 7.35; PaCO₂ 65; HCO₃⁻ 32*

- a) The pH is low normal.
- b) The PaCO₂ is high, making a teeter-totter with the pH, so it is a respiratory problem.
- c) The HCO₃⁻ is high, so there is compensation.
- d) Respiratory acidosis with compensation.

5) *pH 7.51; PaCO₂ 15; HCO₃⁻ 8*

- a) The pH is high, so it is alkalosis.
- b) The PaCO₂ is low, making a teeter-totter with the pH, so it is a respiratory problem.
- c) The HCO₃⁻ is low, so there is compensation.
- d) Respiratory alkalosis with partial compensation.

6) *pH 7.78; PaCO₂ 59; HCO₃⁻ 40*

- a) The pH is very high, so it is alkalosis.
- b) The PaCO₂ is high; it is not a teeter-totter with the pH, so it is a metabolic problem with respiratory compensation.
- c) The HCO₃⁻ is high, confirming a metabolic problem.
- d) Metabolic alkalosis with partial compensation.

7) *pH 7.45; PaCO₂ 37; HCO₃⁻ 24*

- a) The pH is normal.
- b) The PaCO₂ is normal
- c) The HCO₃⁻ is normal.
- d) Normal acid-base balance.

Common Laboratory Tests

You have probably noticed how many blood and urine tests are performed on patients with renal failure. Why are they important? Is there anything special to know when collecting that 24 hour urine? What do they all mean? This section summarizes the most important laboratory tests performed on patients with renal disease.

Electrolytes

For each electrolyte, we'll review its role in the body and discuss what happens when an imbalance occurs.

Sodium

Normal	Role
135 - 145mEq/L	Sodium is one of the most important elements in the body. It is involved in acid-base balance, water balance, transmission of nerve impulses and contraction of muscles.

Hypernatremia

- Symptoms: flushed skin, thirst, weakness dry mucous membranes, fever, hypotension, lethargy, and disorientation
- Causes: dehydration, administration of hypertonic solutions, polyuria
- Treatment: administer free water (D5W) slowly, monitor neurologic status

Hyponatremia

- Symptoms: muscle cramps or twitching, headache, lethargy, hyporeflexia, seizures, anorexia, and nausea
- Causes: SIADH, volume overload, renal failure, and diuretic misuse
- Treatment: fluid restriction and diuretics if patient volume overloaded; hypertonic saline (not with SIADH)

Potassium

Normal	Role
3.5 - 5.0mEq/L	Potassium is a mineral that helps the kidneys function normally. It also plays a key role in cardiac, skeletal, and smooth muscle contraction.

Hyperkalemia

- Symptoms: muscle cramps, nausea, weakness, peaked T waves, prolonged PR, wide QRS, bradycardia, and asystole
- Causes: acidosis, renal failure, hemolysis, and cell death
- Treatment: 1) emergency: CaCl, insulin and D50, NaHCO₃, albuterol, dialysis 2) non-emergency; Kayexalate

Hypokalemia

- Symptoms: fatigue, nausea, GI hypomotility, ectopy, hypotension, broad T waves, ST depression, paresthesia, cardiac arrest
- Causes: diuresis, correction of acidosis, nasogastric suction
- Treatment: intravenous or oral replacement; hypokalemia will be refractory to treatment unless low Ca and Mg corrected

Calcium

Normal	Role
8.5 – 10.5 mg/dL	Calcium is the 5 th most abundant element in the body. It plays a role in transmission of nerve impulses, muscle contraction, blood coagulation, and cardiac functions.

Hypercalcemia

- Symptoms: weakness, hypotonia, constipation, nausea, seizure, short QT, heart blocks, renal failure, confusion and coma
- Causes: malignancy, hyperparathyroidism
- Treatment: administer normal saline, diuretics, mithramycin, phosphates; watch for signs of digoxin toxicity

Hypocalcemia

- Symptoms: paresthesia, long QT interval on the ECG, Chvostek's and Trousseau's signs, tetany, bradycardia, hypotension, confusion, seizures, bronchospasm
- Causes: sepsis, chelation (citrate of phosphorus), PTH insufficiency
- Treatment: intravenous or oral replacement, treatment of underlying cause

Phosphorus

Normal	Role
3.0 – 4.5 mg/dl	Phosphorus assists in the metabolism of protein, calcium and glucose in the body. It is essential to the body for the production of adenosine triphosphate and for the process of glycolysis.

Hyperphosphatemia

- Symptoms: paresthesia, long QT, Chvostek's and Trousseau's signs, tetany, bradycardia, hypotension, confusion, seizures, bronchospasm
- Causes: renal failure, hypoparathyroidism, excess intake
- Treatment: limit intake, administer phosphate binders, monitor (Ca)(PO₄) product

Hypophosphatemia

- Symptoms: decreased cardiac contractility, respiratory failure, rhabdomyolysis, weakness, paresthesia, seizures, low O₂ delivery to tissues (2,3-diphosphoglycerate)
- Causes: diuresis, high PTH, cellular shifting, malnutrition
- Treatment: intravenous or oral replacement, treat underlying cause

Magnesium

Normal	Role
1.5 – 2.5 mEq/L	Magnesium is the 2 nd most abundant element in the body. It is excreted mainly by the kidneys. It affects the central nervous and neuromuscular and cardiac systems.

Hypermagnesemia

- Symptoms: hypotension, lethargy, respiratory depression, bradycardia, AV block, short QT, coma
- Causes: renal failure, excess intake
- Treatment: hemodialysis

Hypomagnesemia

- Symptoms: paresthesia, long QT, Chvostek's and Trousseau's signs, tetany, bradycardia, hypotension, confusion, seizures, bronchospasm, torsades de pointes
- Causes: diuresis, malnutrition, intracellular shift, chelation, acute pancreatitis, chronic alcoholism, DKA, diuretic therapy
- Treatment: IV or PO replacement; treat the underlying cause

BUN (Blood Urea Nitrogen)

The normal range for the BUN is approximately 10-20mg/dl.

BUN is the end product of endogenous or exogenous protein metabolism. It is influenced by fluid volume changes, dietary protein intake, and catabolism. BUN rises with renal failure, increased protein breakdown, and fluid volume depletion. BUN is primarily excreted by the kidneys. When your kidneys are not able to remove urea from the blood normally, the BUN rises. BUN must be

correlated with changes in creatinine to assess renal failure. BUN and creatinine will rise simultaneously.

Creatinine

The normal range for the creatinine is approximately 1mg +/- 0.3mg/dl.

Creatinine is the end product of phosphocreatine, an important part of the muscle. It is regulated and excreted by the kidneys. When kidney function is not normal, creatinine levels in the blood rise because it is not adequately excreted by the kidney.

Plasma creatinine is an excellent indicator of renal failure, once it is established that no muscle breakdown exists.

Hemoglobin/Hematocrit

The hemoglobin and hematocrit in patients with chronic renal failure usually falls slowly over several months. The hematocrit usually stabilizes at about 20-25% and the hemoglobin is usually about 6-8gm%.

Urinalysis

The urinalysis may be used to screen for or diagnose renal failure. If abnormal findings are detected, additional testing is usually required. Several significant findings that may be seen on a urinalysis include:

- Cloudiness results from phosphates, urates, pH changes, bacteria, cells, crystals, or lipids that are present in the urine. Cloudiness indicates that there is a problem that may need to be addressed. It is possible that the kidneys are not functioning properly.
- Protein in the urine could be caused by renal failure and deserves further investigation.
- Specific gravity and osmolality are often used in assessing a patient for renal failure. Urine specific gravity normally ranges from 1.005-1.030 and osmolality from 300-1200 mOsm/kg H₂O.

Osmolality is the most accurate measurement of the kidneys ability to concentrate or dilute urine and it measures the number of solute particles per kilogram of water. If the specific gravity is higher or lower than the normal range, it may indicative of renal failure.

- FeNa (Fractional excretion of sodium). This is the amount of sodium excreted relative to the creatinine. It tells us how the body is handling sodium compared to creatinine.

When the FeNa is less than 1%, this is an indicator of pre-renal acute renal failure (ARF), (the body is holding onto sodium). When the FeNa is greater than 1%, this is linked to acute tubular necrosis (ATN).

Commonly Ordered Imaging Studies

- CXR (PA/Lat) or Portable Chest X-Rays are used to assess volume status.
- Bilateral Renal Ultrasounds are used to evaluate for acute renal failure and chronic kidney disease.
- CT Scans (Spinal CT) are especially useful to evaluate for kidney stones.
- Magnetic Resonance Angiography (MRA) is performed to evaluate for renal artery stenosis (RAS). The exam may show smaller kidneys and a decrease in blood flow through the renal artery.
- Vein Mapping of Bilateral Upper Extremities is performed to evaluate veins for access for dialysis.
- Renograms are useful in determining if there is decreased blood flow to the kidneys. The value of the test is increased if it is done twice, once after a dose of captopril and once without captopril.
- Kidney Biopsy is used to obtain tissue and glomeruli for analysis and diagnosis.

Nutritional Management

The main goal of a special diet for kidney failure is to help reduce the build-up of waste products and extra fluid in the blood. Diets for patients with kidney failure are based on what type of treatment the individual is receiving, the patient's height and weight, nutritional status, other medical conditions they have and their laboratory values.

There are three common diets which are based on the patient's disease and method of treatment.

Calories

For all patients the caloric intake is 35cal/kg/d. Each group then follows individual guidelines for protein, sodium, potassium, phosphorus, and fluid intake.

Chronic Kidney Disease

The main objective for patients with chronic renal disease who are not on renal replacement therapy is to slow the

progression of the kidney disease by limiting the amount of protein in the diet. Phosphorus and sodium in the diet are also limited.

The following specific guidelines are recommended for patients with chronic kidney disease:

- Protein 0.6-1g/kg/dl
- Sodium 1-3 g/d,
- Potassium intake is individualized
- Phosphorus intake is 8-12mg/kg/dl.
- Fluid intake is individualized for each patient

Maintaining a healthy weight and preventing malnutrition are also very important.

Hemodialysis

Patients who are on hemodialysis follow a slightly different set of guidelines. Protein intake is liberalized to approximately 1.1-1.4g/kg/day because patients receiving hemodialysis need more protein.

Patients on hemodialysis still need to limit sodium, potassium, phosphorus and fluid.

The following specific guidelines are recommended for patients receiving hemodialysis:

- Protein intake is liberalized to approximately 1.1-1.4g/kg/day
- Sodium intake is limited to 2-3g/d.
- Potassium intake is limited to 40mg/kg or individualized for each patient.
- Phosphorus intake is <17mg/kg/d.
- Fluid intake for a patient on hemodialysis is 500-750cc/d + urine output, as well as for the patient to maintain weight gain between hemodialysis runs of <5%.

Peritoneal Dialysis

Patients receiving peritoneal dialysis need more protein in their diet because a considerable amount of protein is lost when the peritoneal fluid is discarded following each run of peritoneal dialysis.

The following specific guidelines are recommended for patients receiving peritoneal dialysis:

- Protein intake increases to 1.2-1.5g/kg/d.
- Sodium intake is 2-4g/d.
- Potassium intake is individualized
- Phosphorus intake is <17mg/kg/d.

- Fluid intake is individualized for each patient.

In summary, the goal for all patients with renal failure is to prevent malnutrition and to reduce accumulation of uremic toxins (potassium, phosphorus, sodium, fluid and urea).

Psychosocial Implications

Due to the life altering changes that a diagnosis of renal failure brings, patients have many issues to grapple with over time.

Potential threats faced by patients with kidney disease are related to fear of dying, bodily integrity and comfort, one's self-concept and future plans, emotional equilibrium, the fulfillment of customary social roles and activities, and the need to adjust to a new physical and social environment.

Patients experience a wide range of emotions when confronted with a chronic illness such as renal failure, including anxiety, depression and feeling angry.

Anxieties include apprehension about how the illness will affect their family and friends, work and financial concerns and anxiety about quality of life.

Depression may occur as a result of the loss of independence and self-confidence, changes in appearance, sexual dysfunction and concerns about dying.

Anger can be a natural response for a patient undergoing the experiences associated with renal failure. Problems with anger may occur when the patient expresses it in a self-destructive manner or is unable to move through this phase.

How does renal failure affect the patient's significant others?

Illness can affect patients' family in many ways as well. An illness can incapacitate people, damage lifelong values and commitments. It can also destroy social relationships, produce role losses, generate discomfort and pain, result in repeated or continual loss of dignity, force a person to life with debilitating uncertainties, and threaten life itself. One has the sense that there is no refuge.

The goal of effective intervention requires more sophisticated thought than currently exists and a deep respect for the person who needs our help.

Illness behavior is integral in understanding stress and coping. Once illness/breakdown occurs, stress and coping take on new issues. In order to develop a

framework/strategies for illness interventions designed to support positive coping responses, the nurse must see the social and psychological problems faced by the patient and their families in their daily lives.

Cultural Issues Associated with Renal Failure

Ethnicity extends beyond the family belief systems and distinguishes people in terms of race, customs, characteristics, language and culture. The transmission of one's ethnic culture directly affects one's attitudinal and belief systems as it molds, shapes, and influences family roles, relationships, religion, customs, communication patterns, stress, coping mechanisms, appropriate expression of pain and suffering, and means of illness, life and death.

The patient's ethnic affiliation will shape their behavior and will be influenced by circumstances, perception and generational factors. Awareness and understanding of the patient's ethnic affiliations are essential to assessment and interpretation of the patient's behavior and attitudes.

One of the most important things to remember is to treat each patient as an individual and don't make assumptions based on age, race, culture or ethnicity.

Summary

In recent years, our understanding of renal failure has improved. In this home study, you have reviewed normal anatomy and physiology, pathophysiology, acid-base disturbances, laboratory data, nutritional management and common psychosocial and cultural issues associated with renal failure.

Recommended Web Sites

www.kidney.org
www.niddk.nih.gov
www.nlm.nih.gov

Directions for Submitting Your Post Test for Contact Hours

To obtain a certificate of completion for this home study program, please complete the post-test and evaluation on the next few pages. If you are completing this home study as pre-reading for a TCHP class, please bring your post-test and evaluation to class with you for processing. The date on your certificate of completion will be the date that your home study is received. **Any materials received with a postmark after the expiration will be discarded.**

HealthEast, HCMC, & MVAMC Employees

If you are an employee of HealthEast, HCMC, or MVAMC, you may send the post-test and evaluation to TCHP for processing. Your post-test will be returned to you through your hospital. It cannot be mailed to your home.

Paid Participants

If you are not an employee of one of the TCHP hospitals, please send the post-test and evaluation to TCHP with a check for \$6.00. Please make check payable to **TCHP Education Consortium** and mail to:

**TCHP Education Consortium
Capitol Office Building
525 Park Street, Suite 120
St. Paul, MN 55103**

Your post-test will be returned to you with the certificate of completion.

Kidney Care in the Hospitalized Patient Primer Post-Test

Please print all information clearly and sign the verification statement:

Name _____
(please print legal name above)

Birth date (required)

Format: 01/03/1999

M	M	D	D	Y	Y	Y	Y

For HealthEast, HCMC, or MVAMC, employees only:

Hospital _____ Unit _____

Personal verification of successful completion of this educational activity (required):

I verify that I have read this home study and have completed the post-test and evaluation.

Signature

1. The rate at which solutes are filtered from the glomerulus into the nephron is:
 - a. Creatinine Clearance
 - b. Glomerular Filtration Rate
 - c. Glomerular Capillary Rate
 - d. Glomerular Nephron Rate
2. The presence of elevated nitrogenous waste products, such as urea and creatinine, in the blood is called...
 - a. Uremia
 - b. Creatininemia
 - c. Azotemia
 - d. Proteinuria
3. Which of the following is not a major cause of pre-renal ARF?
 - a. Nephrotoxicity
 - b. Obstruction of flow to the kidney
 - c. Decrease intravascular volume
 - d. Cardiac dysfunction with decreased cardiac output

4. _____ ARF is caused by an obstruction along the urinary tract.
 - a. Intra-Renal
 - b. Obstructive-Renal
 - c. Pre-Renal
 - d. Post-Renal
5. The kidneys under normal functioning compensate over _____ to correct an acid-base imbalance.
 - a. 10-12 hours
 - b. 12-24 hours.
 - c. 20-25 hours
 - d. 24-48 hours
6. _____ occurs when there is either too much acid (such as in renal failure) in the system, or when there is loss of bicarbonate.
 - a. Respiratory Acidosis
 - b. Metabolic Alkalosis
 - c. Respiratory Alkalosis
 - d. Metabolic Acidosis
7. When a patient presents with hyponatremia, what would you expect for the doctor to order?
 - a. Administer free water
 - b. Fluid restriction
 - c. Administer hypotonic solution
 - d. Do nothing
8. If your patient presents with a potassium of 6.3, what is one thing that would you not anticipate being ordered?
 - a. Insulin and D50W
 - b. Kayexalate
 - c. Dialysis
 - d. Intravenous or oral replacement of potassium
9. What do patients with kidney failure have to monitor in their nutritional intake?
 - a. Protein and Calories
 - b. Sodium and Potassium
 - c. Fluid Intake
 - d. Phosphorus
 - e. All of the above

Expiration date: The last day that post tests will be accepted for this edition is **December 31, 2016**—your envelope must be postmarked on or before that day.

Evaluation: Kidney Care in the Hospitalized Patient

Please complete the evaluation form below by placing an "X" in the box that best fits your evaluation of this educational activity. Completion of this form is required to successfully complete the activity and be awarded contact hours.

At the end of this home study program, I am able to:	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
1. Describe normal kidney function.					
2. Describe the pathophysiologic process of renal insufficiency and failure.					
3. Identify acid-base disturbances based on blood gas analysis.					
4. Identify common laboratory tests and imaging studies utilized in renal failure.					
5. Identify common nutritional management guidelines for renal failure patients					
6. Discuss the psychosocial and cultural issues that are frequently associated with renal failure.					
7. The teaching / learning resources were effective. <i>If not, please comment:</i>					

The following were disclosed in writing prior to, or at the start of, this educational activity (please refer to the first 2 pages of the booklet).

	Yes	No
8. Notice of requirements for successful completion, including purpose and objectives		
9. Conflict of interest		
10. Disclosure of relevant financial relationships and mechanism to identify and resolve conflicts of interest		
11. Sponsorship or commercial support		
12. Non-endorsement of products		
13. Off-label use		
14. Expiration Date for Awarding Contact Hours		
15. Did you, as a participant, notice any bias in this educational activity that was not previously disclosed? <i>If yes, please describe the nature of the bias:</i>		

16. How long did it take you to read this home study and complete the post test and evaluation:

_____ hours and _____ minutes.

17. Did you feel that the number of contact hours offered for this educational activity was appropriate for the amount of time you spent on it?

___ Yes

___ No, more contact hours should have been offered

___ No, fewer contact hours should have been offered.

Expiration date: December 31, 2016
