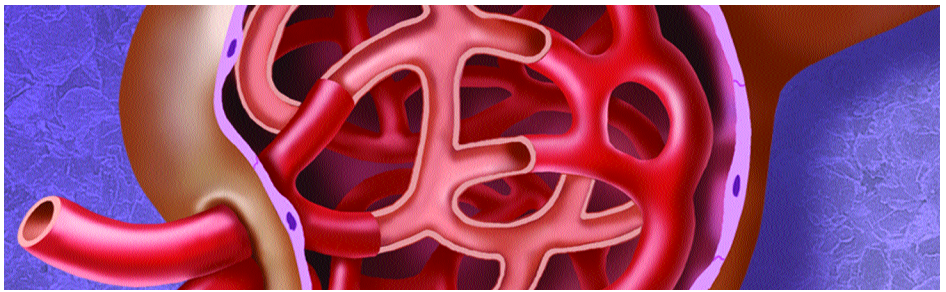


How acute renal failure puts the brakes on kidney function

When you understand the various causes and life-threatening effects, you can provide nursing care that supports recovery.

BY DIANA CAMPBELL, RN, BSN, MPA



ACUTE RENAL FAILURE (ARF) is a sudden loss of kidney function with a buildup of toxic waste products, such as urea and creatinine, in the blood. Unlike chronic renal failure, in which 90% to 95% of nephrons cease to function permanently over time, ARF occurs when the kidneys are taxed suddenly, causing 50% or more of nephrons to lose function so fast that the body can't compensate. (See *Exploring the Nephron* to learn about its key roles.) If the underlying cause is corrected, these nephrons may recover. In some cases, though, damage is permanent and renal failure becomes chronic.

Although ARF most often affects elderly people, the clinical course and survival rate are similar in patients of all ages. Occurring in 5% of hospitalized patients, ARF can lead to complications and death, typically from overwhelming infection or cardiopulmonary problems.

Read on to learn more about how ARF develops and what you can do to support recovery.

Many causes, many effects

Although ARF has many causes, ischemia and toxicity are the most common. Depending on where the problem originates, ARF can be classified as *prerenal*, *intrarenal*, or *postrenal*.

Prerenal ARF, also known as prerenal azotemia, is the most common type. It occurs when decreased blood flow to the kidneys causes ischemia of the nephrons. Blood loss, severe dehydration, septicemia, and cardiogenic shock are common underlying causes. Fortunately, restoring renal blood flow and glomerular ultrafiltration can rapidly reverse prerenal ARF.

Another important cause of prerenal ARF involves use of non-steroidal anti-inflammatory drugs, cyclooxygenase inhibitors, or angiotensin-converting enzyme inhibitors. These drugs can impair renal autoregulatory responses by blocking the production of prostaglandins, which are necessary to maintain renal perfusion. The risk of problems related to therapy with these drugs is high in the elderly and those with renal insufficiency, heart failure, and advanced liver disease.

Intra-renal ARF, also known as intrinsic ARF, is associated with damage to the renal parenchyma. Prerenal ARF can trigger the problem, but a major cause of intra-renal ARF and ARF in general is acute tubular necrosis (ATN)—damage

to the renal tubules caused by ischemia or toxins. Although the terms ARF and ATN are often used interchangeably, they don't mean the same thing. As many as 20% to 30% of patients with ARF caused by ischemia or nephrotoxicity don't have evidence of tubular necrosis.

Diseases involving the large renal vessels (such as renal artery stenosis) and the small vessels and glomeruli (as in glomerulonephritis) are among the ischemic causes of intra-renal ARF. Nephrotoxic substances that can trigger intra-renal ARF include radiocontrast media, cyclosporine, antibiotics (especially aminoglycosides), and chemotherapy. Other nephrotoxic agents include heavy metals, such as mercury and arsenic.

Postrenal ARF occurs as a result of conditions that block urine flow, causing it to back up into the kidneys. Prostatic hypertrophy, ureteral obstruction (usually bilateral), and bladder outlet obstruction are common causes.

Multiple systemic effects
Caring for a patient with ARF is challenging. Renal impairment often affects various body systems, so you'll be dealing with signs and

symptoms of renal disease and problems with other organs and systems. Here's a summary of assessment criteria by body system.

Renal. The key renal effect of ARF is decreased urine output that leads to fluid retention and edema. The classic sign, oliguria (less than 400 ml of urine output in 24 hours) means the kidneys aren't producing enough urine to excrete waste products.

However, some patients experience nonoliguric ARF (usually seen in prerenal azotemia and rarely in intra-renal ARF). In this condition, blood urea nitrogen (BUN) and creatinine build up in the blood and the volume of urine output varies. Restoring intravascular volume and maintaining adequate cardiac output and blood pressure generally stop progression of nonoliguric ARF, so renal replacement therapy—hemodialysis or continuous arteriovenous hemofiltration—isn't necessary. (See *The Ins and Outs of Renal Replacement Therapy* to learn about these options.)

The kidneys' impaired ability to remove waste products from the blood causes levels of toxins such as BUN and creatinine to rise. Because many processes, such as protein metabolism, internal bleeding, and infection, increase BUN levels, this is a less accurate indicator of renal function than creatinine, which is a by-product of muscle metabolism alone. Keep in mind, however, that if muscle mass is decreased, as in a thin or debilitated patient, so is creatinine production, and creatinine levels may stay in the normal range even though his kidneys are impaired. *Trends* in creatinine levels are a better gauge of renal function.

Other abnormal lab results suggesting ARF include metabolic acidosis and electrolyte imbalances, such as hyponatremia, hyperkalemia, hyperphosphatemia, hypocalcemia, and hypermagnesemia.

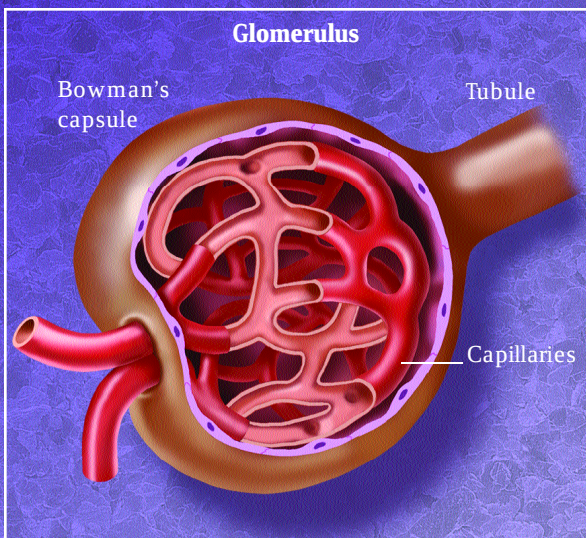
The ins and outs of renal replacement therapy

A patient with acute renal failure may rely on one of these options until normal kidney function returns.

Hemodialysis uses a machine and an artificial kidney to remove excess fluid and waste products from the blood, but it doesn't regulate blood pressure (BP) or other renal functions linked to hormonal control. It's the preferred method when quick removal of water or toxins is indicated and the patient can tolerate the procedure. Because hemodialysis requires removing a substantial amount of fluid from the patient's intravascular system, he could develop hypotension from hypovolemia.

Continuous arteriovenous hemofiltration is based on a simpler concept. Driven by the patient's own BP, it rarely causes hypotension. As the patient's blood is removed, an anticoagulant is added. The blood passes through a porous filter where fluid or solutes are removed, then the blood is returned to the patient. Easier on the body and slower than hemodialysis, the process allows time for fluids to move into the vasculature from the tissues but dramatic results take longer.

Either of these options requires vascular access via a temporary catheter inserted in a large blood vessel.



Exploring the nephron

The kidneys have 2 to 3 million functional units called nephrons made up of components that play key roles in the body.

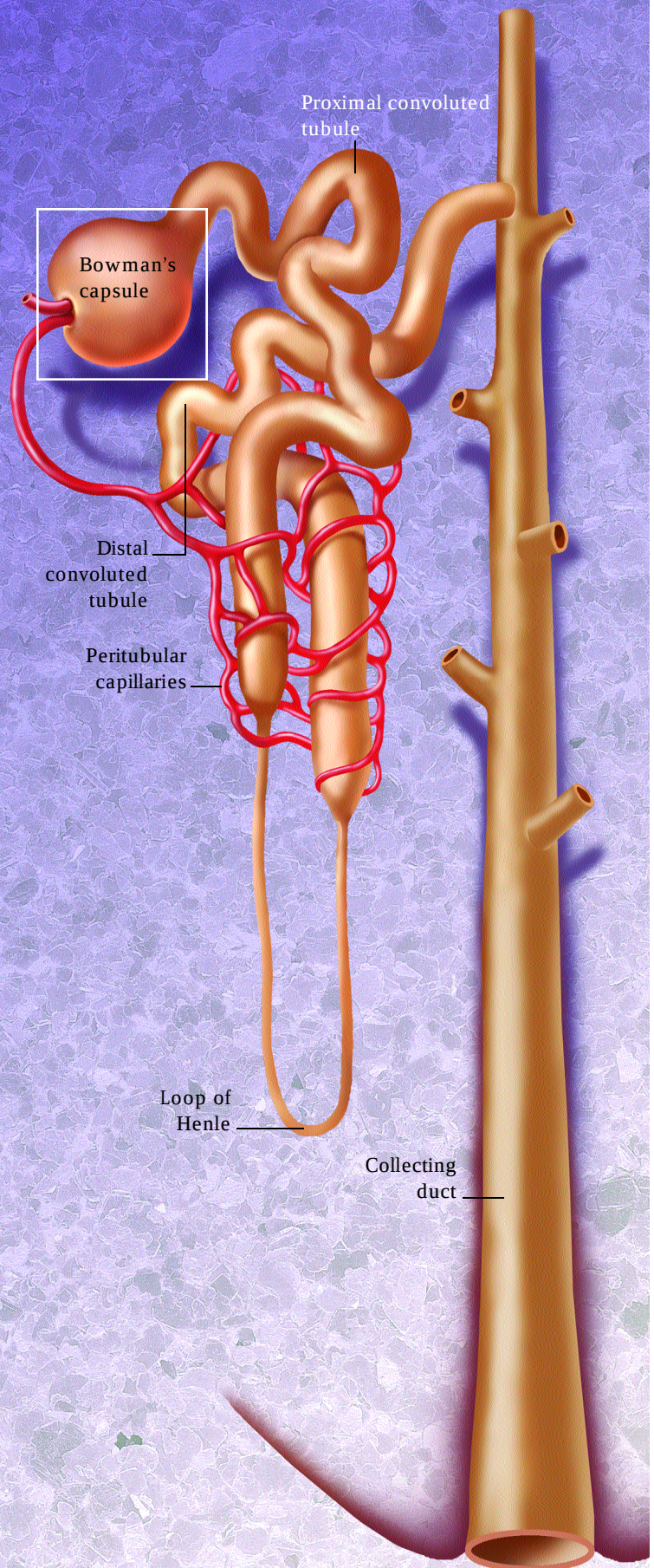
The **glomerulus** is a group of tiny capillaries with very thin walls. As blood passes through, particles and waste products are filtered out into a liquid filtrate. Blood cells and proteins, too large to penetrate the glomerular walls, return to the general circulation.

Bowman's capsule, which surrounds the glomerulus, is the starting point of the tubule, where reabsorption and secretion take place. Filtrate passing into Bowman's capsule is channeled into the tubule.

The **tubule** consists of a proximal convoluted tubule, loop of Henle, and a distal convoluted tubule. Here, 99% of the filtrate—composed of water, electrolytes, and other substances—is reabsorbed into the blood while the rest becomes urine. The loop of Henle concentrates and dilutes the urine.

Peritubular capillaries allow the exchanges between the tubules.

The **collecting duct** receives urine from the tubules of several nephrons and passes it to the renal pelvis, then the ureters.



Cardiovascular Hypertension, pulmonary edema, peripheral edema, and arrhythmias are among the cardiovascular effects of ARF. As decreased urine output causes fluid retention, the patient develops hypertension. If his heart can't pump the additional volume, heart failure ensues. Renin overproduction also causes hypertension.

Edema results when extra fluid moves from his blood vessels into the interstitial space or into his tissues. Because blood albumin is essential to regulate the passage of water and solutes through the capillaries and to prevent fluid from shifting into the interstitial spaces, a low serum albumin level can lead to edema.

Hyperkalemia occurs when the kidneys fail to excrete excess potassium. Be on guard for signs and symptoms such as muscle weakness, loss of muscle tone, and neuromuscular irritability, includ-

for changes in his level of consciousness, which could progress to coma. Sensory changes and weakness in the extremities signal uremic neuropathy.

Hematologic Anemia is the main hematologic effect of ARF. Contributing factors include impaired red blood cell (RBC) production, hemolysis, bleeding, hemodilution, and reduced RBC survival. The normal life span of RBCs, about 120 days, is shortened to about 60 days in ARF. And because the damaged kidneys produce less and less erythropoietin to stimulate RBC production, the lost RBCs aren't replaced. Monitor your patient for decreased hemoglobin and hematocrit levels and dyspnea due to insufficient oxygenation.

Gastrointestinal (GI) Uremia causes anorexia, nausea, and vomiting, which lead to poor nutrition and loss of body mass and muscle. Because uremia also can trigger

blood flow to the kidneys for a pre-renal condition, treating intrinsic renal disease such as acute glomerulonephritis, or removing a postrenal obstruction. Regardless of the type of ARF affecting your patient, provide the following supportive measures:

Maintain fluid and electrolyte balance. Accurately assessing fluid balance is critical, so strictly monitor your patient's weight along with his fluid intake and output. Be sure to include vomitus and liquid stools in output measurements.

Assess him for edema, which is often dependent (in the legs and feet if he's sitting or in the sacral region if he's supine) but also may appear around his eyes. Document the color and clarity of his urine.

A patient with ARF has an increased risk of hyperkalemia, hyponatremia, and volume overload, so closely monitor his fluid and electrolyte levels. A trial of diuretic therapy may remove excess fluid and electrolytes.

Remove nitrogenous wastes Monitor his BUN and creatinine levels. If ARF progresses to the point where waste products are building up in his body, he may need renal replacement therapy such as hemodialysis or continuous arteriovenous hemofiltration to remove them.

Keeping in mind that your patient may need an arteriovenous fistula for hemodialysis, try to limit peripheral intravenous (I.V.) access to the dorsal aspect of his hands to preserve the cephalic veins. If he already has vascular access for dialysis, reserve it strictly for this purpose; using it for other I.V. therapies could threaten its integrity and patency. (See *Clinical Do's and Don'ts*: "Protecting a Hemodialysis Fistula," in the November issue of *Nursing2002*.)

Sustain nutrition Nutritional support is critical to combat malnutrition and water and electrolyte imbalances. Protein-calorie malnu-

Glomerular filtration rate—the key to kidney function

Glomerular filtration rate (GFR), the amount of blood filtered through the glomeruli of the kidneys, is the driving force of urine production. Normally 120 to 125 ml/minute, GFR decreases when renal insult occurs. Increased blood urea nitrogen and creatinine levels are indirect indicators of reduced GFR.

ing tingling in the lips or fingertips. Changes in the electrocardiogram, such as a flattened p wave, prolonged QRS complex, and tall, tented T waves, also signal hyperkalemia. Often, though, hyperkalemia doesn't cause symptoms, so monitoring the patient's serum potassium level is critical. A level above 6 mEq/liter could trigger bradycardia, heart block, asystole, or another arrhythmia.

Respiratory. If fluid overload continues, your patient may develop dyspnea, indicating pulmonary edema. Assess him for dyspnea at rest or on exertion and auscultate his lungs for crackles.

Neurologic. Metabolic wastes building up in his blood can affect your patient's mental status. Look

colitis and gastric ulcers, your patient is at risk for GI bleeding. His breath may take on a foul urine odor caused by an increase in urea.

To confirm ARF, the primary care provider will perform a thorough history and physical examination, blood and urine tests, and possibly a renal ultrasound. If testing confirms ARF, prepare to intervene rapidly to prevent permanent renal damage.

Eliminating the cause In most cases of ARF, normal kidney function returns naturally within weeks. The treatment goals are to eliminate the cause of ARF and support the patient's kidney function and other affected body systems. This means reestablishing

trition is highly prevalent in patients with ARF. Catabolism due to the stress of critical illness is extensive, with the rate proportional to the severity of renal failure. When a patient is in the hypercatabolic state, his body breaks down muscle for protein, causing his BUN and creatinine levels to increase even further.

To prevent a greater protein burden on your patient's kidneys, his nutritional support should provide adequate calories without increasing the protein load. The recommended diet is low in protein and sodium, higher in fats and carbohydrates. Administer feedings orally if his GI tract is functional; otherwise, use the parenteral route.

Dietary restrictions typically include 2 to 4 grams/day of sodium to prevent further water retention, reduced potassium intake to decrease the risk of cardiac arrhythmias, and limited phosphorus intake and possibly use of phosphate binders with

meals to prevent further reductions in blood calcium levels. Restrict fluid intake to the amount of the patient's urine output plus 500 to 700 ml.

Provide emotional support and teaching. For both your patient and his family, ARF is sudden, unexpected, and traumatic. Provide emotional support and teach them about his medications, nutritional needs, fluid restriction, and the role of dialysis.

If all goes well, his recovery from ARF may take 3 to 12 months. But if the underlying cause of ARF can't be corrected and nephron damage continues, he'll develop chronic renal failure, which calls for maintenance dialysis or a kidney transplant.

Understanding the processes Now that you understand how ARF affects many physical processes, you can take appropriate steps to support recovery and teach your patient what he needs to know to endure the crisis. **■**

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Diana Campbell is a renal resource nurse in the Vancouver Island Health Authority Renal Program in British Columbia, Canada.



SELECTED WEB SITES

American Academy of Family Physicians: Acute Renal Failure
<http://www.aafp.org/afp/20000401/2077.html>

MedLine Plus Health Information: Acute Renal Failure
<http://www.nlm.nih.gov/medlineplus/ency/article/000501.htm>

Last accessed on December 3, 2002.

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How acute renal failure puts the brakes on kidney function

PURPOSE To improve nursing practice and the quality of care by providing a learning opportunity that enhances a participant's understanding of ARF.

OBJECTIVES After reading the preceding article and taking this test, you should be able to: 1. Indicate the pathophysiology and causes of ARF. 2. Indicate assessment findings for patients with ARF. 3. Identify treatment goals and nursing interventions for patients with ARF.

CE TEST
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1. Which of the following are characteristics of ARF?

1. permanent kidney damage and progression to chronic renal failure
2. a sudden loss of kidney function with a buildup of toxic waste products
3. permanent loss of function of 95% of nephrons over time
4. abnormally low BUN and creatinine levels

2. Which statement about ARF is correct?

1. It most commonly affects young adults.
2. The clinical course and survival rate vary greatly, depending on the patient's age.
3. It occurs in 25% of hospitalized patients.
4. Death typically results from overwhelming infection or cardiopulmonary problems.

3. Which type of ARF occurs when decreased blood flow to the kidneys causes ischemia of the nephrons?

1. intrarenal
2. postrenal
3. prerenal
4. intrinsic

4. Possible causes of postrenal ARF include

1. absorption of heavy metals.
2. prostatic hypertrophy.
3. injection of radiocontrast material.
4. cyclosporine therapy.

5. Drugs that could cause prerenal ARF include

1. nonsteroidal anti-inflammatory drugs.
2. cyclosporine.
3. aminoglycosides.
4. chemotherapy agents.

6. Which of the following could cause intrarenal ARF?

1. renal artery stenosis
2. blood loss
3. septicemia
4. cardiogenic shock

7. What's the classic sign of ARF?

1. oliguria
2. polyuria
3. anuria
4. dysuria

8. Which of the following correctly describes nonoliguric ARF?

1. It typically occurs with intrarenal ARF.
2. It rarely occurs with prerenal azotemia.
3. The appropriate treatment is renal replacement therapy.
4. Maintaining adequate cardiac output and BP generally halts it.

9. Which of the following can affect serum creatinine levels?

1. protein metabolism
2. muscle mass
3. internal bleeding
4. infection

10. Consider the possibility of ARF when lab tests indicate

1. metabolic alkalosis.
2. hypernatremia.
3. hypokalemia.
4. hypocalcemia.

11. Which ECG changes suggest hyperkalemia?

1. tall, tented p waves
2. shortened QRS complex
3. flattened T waves
4. heart block and other arrhythmias

12. Sensory changes and extremity weakness in a patient with ARF signal

1. pulmonary edema.
2. anemia.
3. uremic neuropathy.
4. colitis.

13. What's an appropriate nursing intervention for a patient with ARF?

1. encouraging oral fluid intake
2. limiting peripheral I.V. access to the

cephalic veins

3. providing a low-protein, low-sodium diet
4. offering frequent protein supplements

14. Which of the following occurs in the glomerulus?

1. Urine passes from the tubules of several nephrons to the renal pelvis.
2. Urine is concentrated and diluted.
3. About 99% of filtrate is reabsorbed into the blood.
4. Particles and waste products pass into a liquid filtrate.

15. What's the starting point of the tubule where reabsorption and secretion occur?

1. peritubular capillaries
2. loop of Henle
3. Bowman's capsule
4. collecting duct

16. Which statement about GFR is correct?

1. It increases after a renal insult.
2. It's normally 90 to 100 ml/minute.
3. Decreased BUN and creatinine levels are direct indicators that it's reduced.
4. It's the driving force behind urine production.

17. Which of the following statements about hemodialysis is correct?

1. It's slower than continuous arteriovenous hemofiltration.
2. It regulates BP.
3. It regulates renal functions linked to hormonal control.
4. It removes a substantial amount of fluid from the intravascular system.

18. Which statement about continuous arteriovenous hemofiltration is correct?

1. The patient's BP is the driving force.
2. It frequently causes hypotension.
3. It's faster than hemodialysis.
4. It's indicated when quick removal of toxins is required.



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