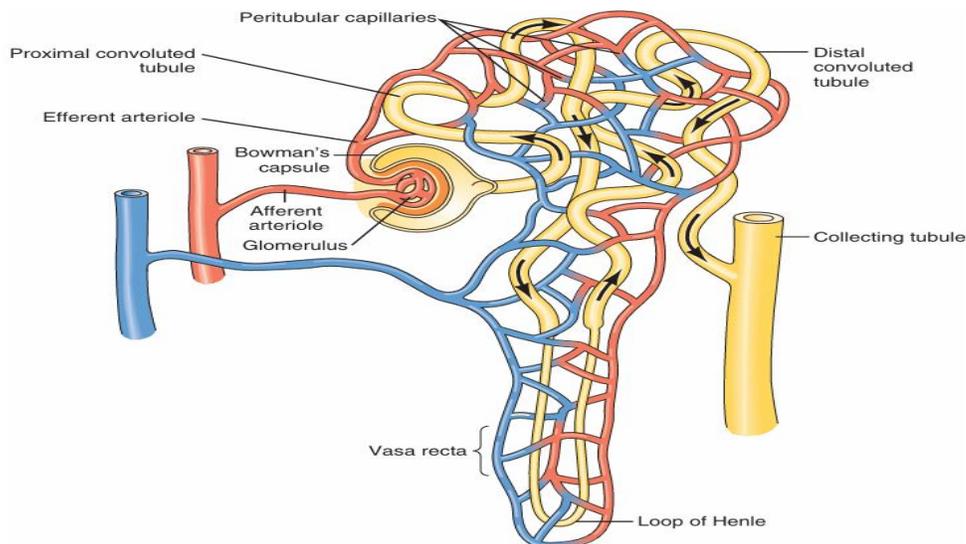


## Acute Kidney Injury

### Review Of Anatomy and Physiology

- The kidneys are a pair of highly vascularized, bean-shaped organs that are located retroperitoneally on each side of the vertebral column, adjacent to the first and second lumbar vertebrae. The right kidney sits slightly lower than the left kidney because the liver lies above it.
- Each kidney is divided into two regions: an outer region, called the cortex, and an inner region, called the medulla.
- An adrenal gland sits on top of each kidney and is responsible for the production of aldosterone, a hormone that influences sodium and water balance.
- The nephron is the basic functional unit of the kidney. A nephron is composed of a renal corpuscle (glomerulus and Bowman's capsule) and a tubular structure
- The kidneys receive approximately 20% to 25% of the cardiac output, which computes to 1200 mL of blood per minute. Blood enters the kidneys through the renal artery, travels through a series of arterial branches, and reaches the glomerulus by way of the afferent arteriole (afferent meaning to carry toward). Blood leaves the glomerulus through the efferent arteriole (efferent meaning to carry away from), which then divides into two extensive capillary networks called the peritubular capillaries and the vasa recta. The capillaries then rejoin to form venous branches by which blood eventually exits the kidney via the renal vein.
- The glomerulus is a cluster of minute blood vessels that filter blood.





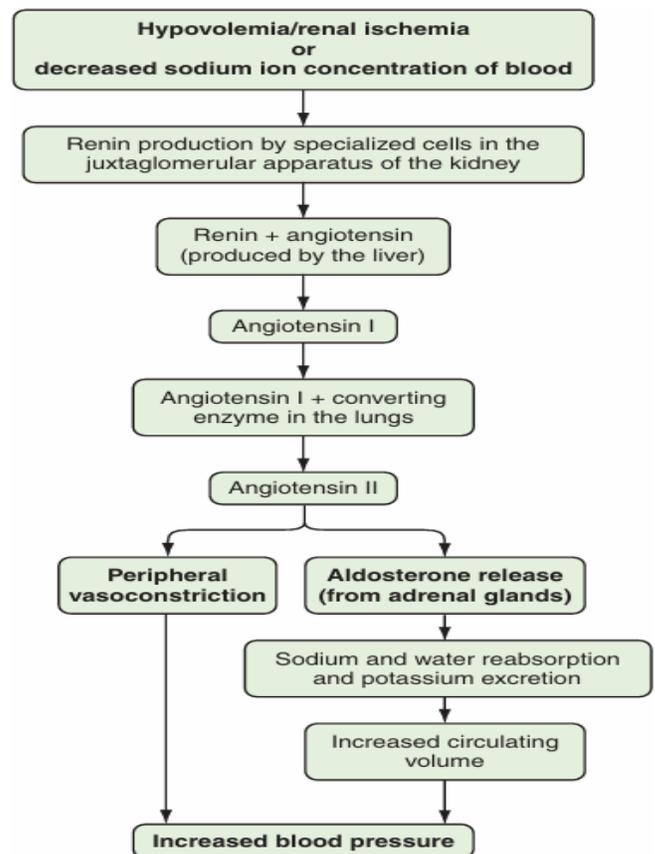
**kidney functions:** The kidneys perform numerous functions that are essential for the maintenance of a stable internal environment.

1. **Regulation of Fluid and Electrolytes and Excretion of Waste Products:**

As blood flows through each glomerulus, water, electrolytes, and waste products are filtered out of the blood across the glomerular membrane and into Bowman's capsule, to form what is known as filtrate. Normal glomerular filtrate is basically **protein free** and **contains electrolytes**, including **sodium, chloride, and phosphate**, and nitrogenous waste products, such as **creatinine, urea, and uric acid**. The **Red blood cells, albumin, and globulin** are too **large to pass** through the healthy glomerular membrane.

2. **Regulation of Acid-Base Balance:** The kidneys help to maintain acid-base equilibrium in three ways: by **reabsorbing filtered bicarbonate, producing new bicarbonate**, and **excreting small amounts of hydrogen ions** (acid) buffered by phosphates and ammonia.

3. **Regulation of Blood Pressure:** Specialized cells in the afferent and efferent arterioles and the distal tubule are collectively known as the juxtaglomerular. These cells are responsible for the production of a hormone called **renin**. Renin is released whenever **blood flow** and through the afferent and efferent arterioles **decreases or decrease in the sodium ion** concentration of the blood flowing stimulates the release of renin. Renin activates the renin-angiotensin aldosterone cascade, which ultimately results in angiotensin II production. Angiotensin II causes vasoconstriction and release of aldosterone from the adrenal glands, thereby raising blood pressure and flow and increasing sodium and water reabsorption in the distal tubule and collecting ducts.





4. Effects of Aging The most important renal physiological change that occurs with aging is a decrease in the GFR. After age 40 years, renal blood flow gradually diminishes at a rate of 10% per decade.

**Acute Kidney Injury:** Acute kidney injury is defined as the **sudden decline** in kidney function causing **disturbances in fluid, electrolyte, and acid base balance** because of a loss in small solute clearance and decreased glomerular filtration rate. The cardinal features of AKI are azotemia and oliguria. Azotemia refers to increases in blood urea nitrogen and serum creatinine. Oliguria is defined as urine output less than 0.5 mL/kg/hr.

### Classifications of Acute Kidney Injury

Classification criteria for AKI include assessment of three grades of severity and two outcome-level classifications. This 5-point system is known as the RIFLE classification system. RIFLE stands for risk, injury, failure, loss, and ESKD (. Risk, injury, and failure are considered grades of AKI severity, whereas loss and ESKD are considered outcomes of loss that require some form of RRT, at least temporarily

**TABLE 48-2** The RIFLE Classification for Acute Kidney Injury

Class	GFR Criteria	Urinary Output Criteria
R (Risk)	Increased serum creatinine $1.5 \times$ baseline OR GFR decreased $\geq 25\%$	0.5 mL/kg/h for 6 hrs
I (Injury)	Increased serum creatinine $2 \times$ baseline OR GFR decreased $\geq 50\%$	0.5 mL/kg/h for 12 hrs
F (Failure)	Increased serum creatinine $3 \times$ baseline OR GFR decreased $\geq 75\%$ OR Serum creatinine $\geq 354$ mmol/L with an acute rise of at least 44 mmol/L	$<0.3$ mL/kg/h for 24 hrs OR Anuria for 12 hrs
L (Loss)	Persistent acute kidney injury = complete loss of kidney function $>4$ wks	
E (ESKD)	ESKD $>3$ mo	



## Etiology

The etiology of AKI in critically ill patients is often multifactorial and develops from a combination of **hypovolemia, sepsis, medications, and hemodynamic instability**. The etiology of AKI is classified as either prerenal, postrenal, or intrarenal depends on where the precipitating factor exerts its pathophysiological effect on the kidney

1. **Prerenal Causes of Acute Kidney Injury:** Conditions that result in AKI by interfering with renal perfusion are classified as prerenal. Most prerenal causes of AKI are related to:
  - a. Intravascular volume depletion
  - b. Decreased cardiac output
  - c. Renal vasoconstriction
  - d. Pharmacological agents that impair autoregulation and GFR.

The body attempts to normalize renal perfusion by reabsorbing sodium and water. If adequate blood flow is restored to the kidney, normal renal function resumes. If the prerenal situation is prolonged or severe, it can progress to intrarenal damage, acute tubular necrosis (ATN), or acute cortical necrosis.

2. **Postrenal Causes of Acute Kidney Injury:** Acute kidney injury resulting from **obstruction of the flow of urine** is classified as postrenal, or obstructive renal injury. Obstruction can occur at any point along the urinary system. With postrenal conditions, increased intratubular pressure results in a decrease in the GFR and abnormal nephron function.

Most common postrenal Courses:

- a. Benign prostatic hypertrophy (BPH)
  - b. Renal stones or crystals
  - c. Tumors
  - d. Foley catheter obstruction
3. **Intrarenal Causes of Acute Kidney Injury:** Conditions that produce AKI by directly acting on functioning kidney tissue (either the glomerulus or the renal tubules) are classified as intrarenal. The most common intrarenal condition is ATN.

Intrarenal causes of acute kidney injury

- a. Glomerulonephritis



- b. Ischemia
- c. Hypertension
- d. Hypotension from any cause
- e. Hypovolemia from any cause
- f. Severe heart failure
- g. Diabetes mellitus
- h. Advanced age

### Phases of Acute Kidney Injury

The patient with AKI progresses through three phases of the disease process: the initiation phase, the maintenance phase, and the recovery phase.

1. **Initiation Phase:** The initiation phase is the period that elapses from the occurrence of the precipitating event to the beginning of the change in urine output. This phase spans several **hours to 2 days**, during which time the normal renal processes **begin to deteriorate**, but actual intrinsic **renal damage has not yet occurred**.
2. **Maintenance Phase:** During the maintenance phase, renal damage is established, and the GFR stabilizes at approximately 5 to 10 mL/min. Urine volume is usually at its lowest point during the maintenance phase. This phase usually **lasts 8 to 14 days**, but it may last up to 11 months. The longer a patient remains in this stage, the slower the recovery and the greater the chance of permanent renal damage. Complications resulting from uremia, including hyperkalemia and infection, occur during this phase.
3. **Recovery Phase:** This phase is the period during which the renal tissue recovers and repairs itself. A gradual increase in urine output and an improvement in laboratory values occur. Recovery may take as long as 4 to 6 months.

### Laboratory test

1. The serum creatinine level is often used to evaluate kidney function.
2. serum blood urea nitrogen (BUN) level is also used to evaluate kidney function, the BUN level is not a reliable indicator of kidney function because the rate of protein metabolism (urea is a by-product of protein metabolism) is not constant.



- The BUN/creatinine ratio provides useful information. The normal BUN/creatinine ratio is 10:1 to 20:1 (e.g., BUN level, 20 mg/dL, and creatinine level, 1.0 mg/dL). If the ratio is greater than 20:1 (e.g., BUN level, 60 mg/dL, and creatinine level, 1.0 mg/dL), problems other than kidney failure should be suspected.

### Diagnostic Procedures

- Noninvasive diagnostic procedures**
  - Radiography of the kidneys, ureters, and bladder
  - Renal ultrasonography
  - Magnetic resonance imaging (MRI)
- Invasive diagnostic procedure:**
  - intravenous pyelography
  - Computed tomography
  - Renal angiography
  - Renal scanning
  - Renal biopsy

### Medical Management of Acute Kidney Injury

- Prerenal Causes:** Acute kidney injury from prerenal conditions is usually reversible if renal perfusion is quickly restored; therefore, early recognition and prompt treatment are essential.
- Postrenal Causes:** Postrenal obstruction should be suspected whenever a patient has an unexpected decrease in urine volume. Postrenal conditions are usually resolved with the insertion of an indwelling bladder catheter, either transurethral or suprapubic.
- Intrarenal Causes:** Acute Tubular Necrosis Common interventions for the patient with ATN include drug therapy, dietary management such as protein and electrolyte restrictions, management of fluid and electrolyte imbalances, and renal replacement therapies such as intermittent hemodialysis or continuous renal replacement therapy (CRRT).



## General principles in Management for Acute Kidney Injury

The collaborative approach of the healthcare team to the treatment of patients with AKI begins with the recognition of AKI risk. The focus for those at risk is maintenance of adequate renal perfusion and avoidance of renal injury.

1. **Correct of fluid imbalance:** Maintaining fluid balance in the renal failure patient is a challenge. The simultaneous goals of providing enough fluid to ensure adequate renal perfusion and preventing excess fluid and volume overload require close attention. The following interventions are recommended in the management of fluid imbalance:
  - a. **Calculate daily fluid needs:** In prerenal disease, fluid replacement must be matched with fluid loss, both in amount and composition. Insensible fluid losses must be considered in this calculation
  - b. **Obtain accurate intake and output** and include insensible losses in the measurements. Fluid therapy decisions are often based on the patient's output.
  - c. **Obtain daily weights:** Body weight can decrease by 0.2 to 0.3 kg/day as a result of catabolism. If the patient's weight is stable or increasing, volume over load is suspected
  - d. **Administer diuretics** only when the patient is hypervolemic and in consultation with healthcare providers. Increasing dosages may be used in an attempt to determine the optimal dose.
  - e. **Institute renal replacement therapy (RRT) as needed.** There are three types of RRT available: IHD, peritoneal dialysis (PD), and several forms of CRRT.
2. **Preventing the life-threatening electrolyte imbalance:** There are a number of electrolyte imbalances that can occur in AKI, the most common being hyperkalemia, hypocalcemia, hypermagnesemia, hyperphosphatemia, and bicarbonate deficiency.
3. **Treating acidosis:** Patients with AKI often develop metabolic acidosis with a mild respiratory alkalosis compensation.



4. **Preventing additional kidney damage:** In AKI, medications metabolized or excreted by the kidney require adjustment to avoid excessive blood levels and potential nephrotoxicity. Particular attention also must be given to medication scheduling in patients on RRT, as medications may be eliminated or potentiated by dialysis.
5. **Prevention and treating infection:** Patients with AKI are at high risk for infection and often require treatment with antimicrobial agents. Selection and dosing of antimicrobial agents is carefully considered to minimize the risk of additional kidney injury.
6. **Maintaining adequate nutrition:**  
In patients with AKI, the challenge in the management of nutritional status is to provide a balance between sufficient calories and protein to prevent catabolism, while avoiding problems, such as fluid and electrolyte imbalances that increase the requirement for RRT. The following interventions are recommended in Maintaining adequate nutrition
  - a. Restrict the patient's fluid, sodium, potassium, and phosphorus intake. Because patients with AKI cannot eliminate wastes, fluid, or electrolytes, their dietary intake of these substances is typically restricted. The degree of restriction depends on the cause and severity of their disease.
  - b. Administer necessary vitamin supplements. Supplementation of folic acid, pyridoxine, and the water-soluble vitamins is most frequently necessary.
  - c. Consult a dietitian for a diet plan. Dietary requirements change for patients depending on their renal status and the severity of their underlying condition.

### Nursing Management

Nursing care of the patient with acute kidney injury is complex. Multiple nursing diagnoses must be dealt with in these often critically ill patients

1. Nursing diagnosis: **Excess Fluid Volume related to sodium and water retention and excess intake**

Patient outcomes: Stable fluid balance

Interventions:

- a. Obtain daily weights
- b. Maintain accurate intake and output records



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- c. Monitor respiratory status, including respiratory rate and crackles
  - d. Assess heart rate, bloodpressure, and respiratory rate
  - e. Administer all fluids and medications in the least amount of fluid possible
  - f. Monitor blood and urine laboratory tests
2. Nursing diagnosis: **Risk for Infection related to depressed immune response secondary to uremia and Impaired Skin Integrity**
- Patient outcomes: Absence of infection
- Interventions:
- a. Monitor WBC count and culture results
  - b. Monitor temperature
  - c. Avoid invasive equipment whenever possible, such as indwelling urinary catheters and central lines
  - d. Use good hand-washing technique
  - e. Use aseptic technique for all procedures
  - f. Perform pulmonary preventive techniques (turn, cough, deep breathing)
  - g. Assess potential sites of infection (urinary, pulmonary, wound, intravenous catheters).
3. Nursing diagnosis: **Imbalanced Nutrition: Less Than Body Requirements related to uremia, and dietary restrictions.**
- Patient outcomes: Adequate nutritional and caloric intake
- Interventions:
- a. Monitor body weight and caloric intake daily
  - b. Collaborate with dietitian about nutritional needs
  - c. Provide diet with essential nutrients but within restrictions
  - d. Provide oral hygiene every 2 to 4 hours
  - e. Remove noxious stimuli from room