Anatomy and Physiology

- Kidneys
  - Location
- Retroperitoneal area
  - Structure
- Cortex
- Medulla
- Nephron
  - Receives 20% to 25% of cardiac output
  - Performs numerous functions

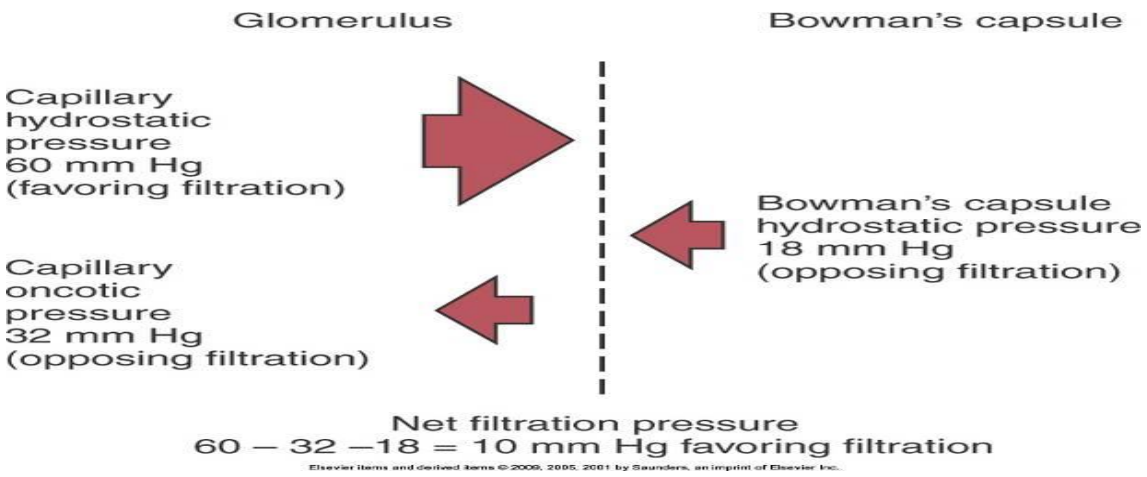
Functions of the Kidney

- Regulation of fluid volume
- Regulation of electrolyte balance
- Regulation of acid-base balance
- Regulation of blood pressure
- Excretion of nitrogenous waste products
- Regulation of erythropoiesis
- Metabolism of vitamin D
- Synthesis of prostaglandin
- Secretion of hormones:

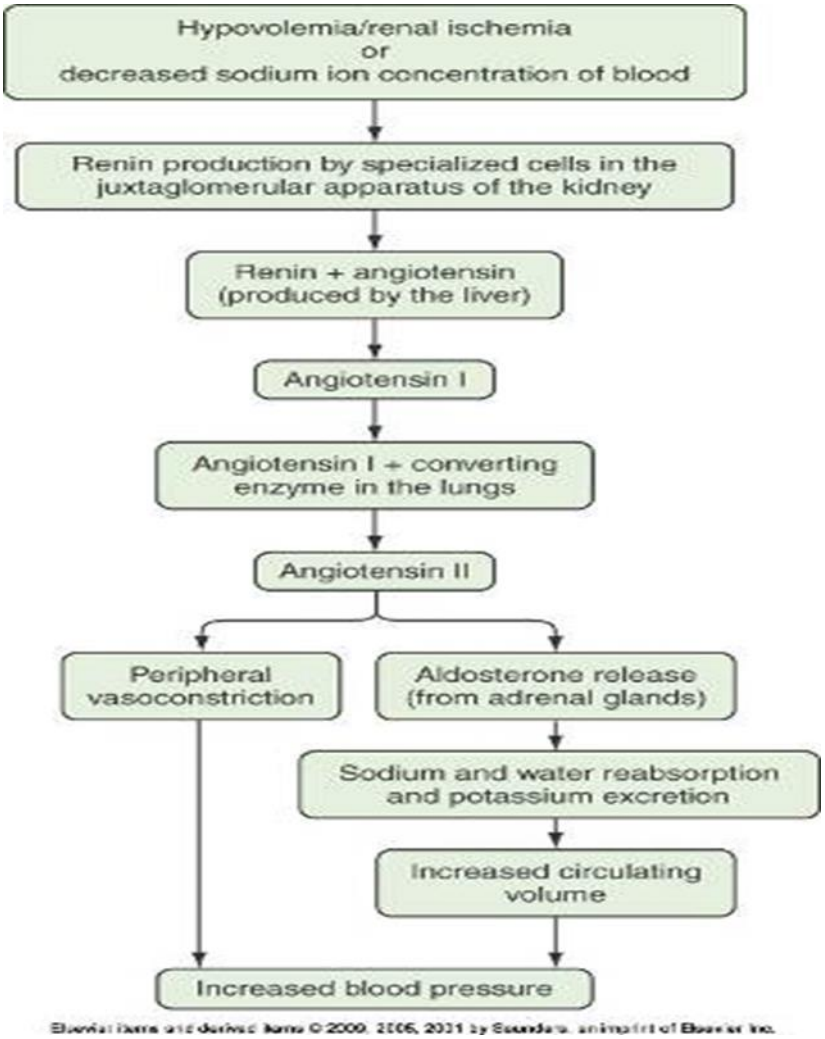
A.Erythropoietin (which is also synthesized in the kidney), which stimulates the bone marrow to produce red blood cells.

B. Renin, which controls the production of angiotensin and aldosterone. These cause, respectively, systemic vasoconstriction and renal salt and water retention to maintain effective circulating volume. This contributes to the regulation of blood pressure and fluid balance.

- Excretion of drugs and byproducts of metabolism, nitrogen, urea, creatinine.



Blood Pressure Regulation



Acute Renal Failure/Acute Kidney Injury

Acute renal failure (ARF) [also known as acute kidney injury (AKI)] is defined as a relatively sudden (over hours to days) decrease in renal function leading to serious derangements of body fluid homeostasis.

Acute renal failure (ARF):Is a reversible clinical condition where there is a sudden and almost complete loss of kidney function (decreased GFR) over a period of hours to days with failure to excrete nitrogenous waste products and to maintain fluid and electrolyte homeostasis.

AKI worsens.

Causes of ARF

- Prerenal
- Renal: intrinsic; parenchymal
- Postrenal

BOX 31-1 Precipitating Causes of Acute Kidney Injury

Prerenal
- Decreased intravascular volume
  - Dehydration
  - Hemorrhage
  - Hypovolemic shock
  - Hypovolemia (gastrointestinal losses, diuretics, diabetes insipidus)
  - Third spacing (burns, peritonitis)
- Cardiovascular failure
  - Heart failure
  - Myocardial infarction
  - Cardiogenic shock
  - Valvular heart disease
  - Renal artery stenosis or thrombosis
- Drugs
  - ACE inhibitors
  - NSAIDs—inhibit prostaglandin-mediated afferent arteriolar vasodilation
  - Calcineurin inhibitors (eg, tacrolimus, cyclosporine)—cause preglomerular vasoconstriction
- Decreased "effective renal perfusion"
  - Sepsis
  - Cirrhosis
  - Neurogenic shock

Intrarenal
- Acute glomerulonephritis
  - Immune complex–mediated (postinfectious, lupus nephritis, cryoglobulinemia, immunoglobulin A [IgA] nephropathy)
  - With vasculitis (Wegener's granulomatosis, antiglomerular basement membrane disease, polyarteritis nodosa)
- Vascular disease
  - Malignant hypertension
  - Microangiopathic hemolytic-uremic syndrome (HUS)
  - Thrombotic thrombocytopenic purpura (TTP)
  - Scleroderma
  - Eclampsia
  - Atheroembolic disease
  - Acute cortical necrosis
- Acute interstitial disease
  - Allergic interstitial nephritis
  - Acute pyelonephritis
- Tubular obstruction
  - Multiple myeloma
  - Acute urate nephropathy
  - Ethylene glycol or methanol toxicity
- Acute tubular necrosis (ATN)
  - Ischemia
  - Nephrotoxins (contrast dye, drugs, heme pigments)
  - Kidney transplant rejection

Postrenal
- Ureteral obstruction
  - Intrinsic (stones, transitional cell carcinoma of the ureter, blood clots, stricture)
  - Extrinsic (ovarian cancer; lymphoma; metastatic cancer of the prostate, cervix, or colon; retroperitoneal fibrosis)
- Bladder problems
  - Tumors
  - Blood clots
  - Neurogenic bladder (spinal cord injury, diabetes mellitus, ischemia, drugs)
  - Stones
- Urethral obstruction
  - Prostate cancer or benign prostatic hypertrophy
  - Stones
  - Stricture
  - Blood clots
  - Obstructed indwelling catheter

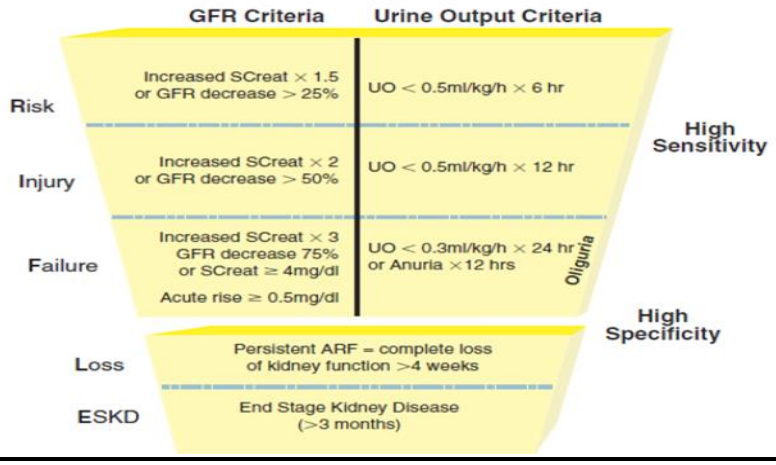
Table 44-2 summarizes common clinical characteristics in all three categories:

Table 44-2 COMPARING CLINICAL CHARACTERISTICS OF ACUTE RENAL FAILURE

Characteristics	Categories		
	Prerenal	Intrarenal	Postrenal
Etiology	Hypoperfusion	Parenchymal damage	Obstruction
Blood urea nitrogen value	Increased (out of normal 20:1 proportion to creatinine)	Increased	Increased
Creatinine	Increased	Increased	Increased
Urine output	Decreased	Varies, often decreased	Varies, may be decreased, or sudden anuria
Urine sodium	Decreased to <20 mEq/L	Increased to >40 mEq/L	Varies, often decreased to 20 mEq/L or less
Urinary sediment	Normal, few hyaline casts	Abnormal casts and debris	Usually normal
Urine osmolality	Increased to 500 mOsm	About 350 mOsm similar to serum	Varies, increased or equal to serum
Urine specific gravity	Increased	Low normal	Varies

RIFLE Criteria: The risk of critically ill patients developing AKI has been classified by a multinational group of nephrologists. The classification

uses the acronym RIFLE—risk, injury, failure, loss, and end-stage kidney disease (ESKD). The RIFLE system classifies AKI in three categories of increasing severity (R, I, F) and two outcome criteria (L, E) based on GFR status reflected by the change in urine output or loss of kidney function2 (Table 27-1).



Pathophysiology Summary

- Prerenal: decreased blood supply
- Renal: failure of nephrons
- Postrenal: obstruction of outflow

Categories of Acute Renal Failure

1. Prerenal- occurs in 60%-70% of cases, is the result of impaired blood flow to that leads to hypoperfusion of the kidney and a decrease in the GFR.

2. Intra-renal is the result of actual parenchymal damage to glomeruli or kidney tubules.

3. Post renal – is usually the result of an **obstruction distal to the** kidneys. Pressure rises in the kidney tubules and eventually, the GFR decreases.

Phases of Acute Renal Failure:

There are four phases of ARF: initiation, oliguria, diuresis, and recovery.

1. Initiation phase:

- Time from event to signs of decreased renal perfusionFew hours to 2 days
- Potentially reversible.

2. Maintenance phase (oliguria/anuria):

➢ BUN and creatinine increase daily (Oliguria is common urine output less than 400 mL/day).

➢ Fluid overload, electrolyte imbalances, and acidosis

–Renal replacement therapy required

4. Recovery phase

➢ Return of tubular function

➢ 4 to 6 months for BUN and creatinine to return to normal

➢ Residual impairment of GFR

➢ Early dialysis may prevent the traditional "diuretic" phase of ARF

Systemic Manifestations of Acute Kidney Injury

SYSTEM	MANIFESTATION	PATHOPHYSIOLOGICAL MECHANISM
Cardiovascular	Heart failure	Fluid overload and hypertension
	Pulmonary edema	↑ Pulmonary capillary permeability Fluid overload Left ventricular dysfunction
	Dysrhythmias	Electrolyte imbalances (especially hyperkalemia and hypocalcemia)
	Peripheral edema	Fluid overload Right ventricular dysfunction
	Hypertension	Fluid overload ↑ Sodium retention
Hematological	Anemia	↓ Erythropoietin secretion Loss of RBCs through GI tract, mucous membranes, or dialysis ↓ RBC survival time Uremic toxins' interference with folic acid secretion
	Alterations in coagulation	Platelet dysfunction
	↑ Susceptibility to infection	↓ Neutrophil phagocytosis
Electrolyte imbalances	Metabolic acidosis	↓ Hydrogen ion excretion ↓ Bicarbonate ion reabsorption and generation ↓ Excretion of phosphate salts or titratable acids ↓ Ammonia synthesis and ammonium excretion
Respiratory	Pneumonia	Thick tenacious sputum from ↓ oral intake Depressed cough reflex ↓ Pulmonary macrophage activity
	Pulmonary edema	Fluid overload Left ventricular dysfunction ↑ Pulmonary capillary permeability
Gastrointestinal	Anorexia, nausea, vomiting	Uremic toxins Decomposition of urea releasing ammonia that irritates mucosa
	Stomatitis and uremic halitosis	Uremic toxins Decomposition of urea releasing ammonia that irritates oral mucosa
	Gastritis and bleeding	Uremic toxins Decomposition of urea releasing ammonia that irritates mucosa, causing ulcerations and increased capillary fragility
Neuromuscular	Drowsiness, confusion, irritability, and coma	Uremic toxins produce encephalopathy Metabolic acidosis Electrolyte imbalances
	Tremors, twitching, and convulsions	Uremic toxins produce encephalopathy ↓ Nerve conduction from uremic toxins
Psychosocial	Decreased mentation, decreased concentration, and altered perceptions	Uremic toxins produce encephalopathy Electrolyte imbalances Metabolic acidosis Tendency to develop cerebral edema
Integumentary	Pallor	Anemia
	Yellowness	Retained urochrome pigment
	Dryness	↓ Secretions from oil and sweat glands
	Pruritus	Dry skin Calcium and/or phosphate deposits in skin Uremic toxins' effect on nerve endings
	Purpura	↑ Capillary fragility Platelet dysfunction
	Uremic frost (rarely seen)	Urea or urate crystal excretion
Endocrine	Glucose intolerance (usually not clinically significant)	Peripheral insensitivity to insulin Prolonged insulin half-life from ↓ renal metabolism
Skeletal	Hypocalcemia	Hyperphosphatemia from ↓ excretion of phosphates ↓ ↓ GI absorption of vitamin D Deposition of calcium phosphate crystals in soft tissues

Diagnosis of ARF

1. Assessment of Patient History

➢ **Predisposing factors**

➢ Disease states

- Hypertension
- Diabetes
- Immunologic disease
- Hereditary disorders

➢ Hypotensive episodes

➢ Exposure to nephrotoxic agents

➢ Vital signs may be altered

- Blood pressure changes depending on etiology
- Hyperventilation to compensate for metabolic acidosis
- Body temperature may be altered

➢ Assess for volume depletion and volume overload

2. Laboratory Tests for ARF

• Serum creatinine

• Serum BUN

– Affected by catabolism, bleeding, and dehydration

• Bun: creatinine ratio

– Normal 10:1 to 20:1

– More than 20:1, suspect nonrenal causes of laboratory abnormalities

• Urine creatinine clearance

- Estimate of GFR
- 24-hour urine; specific collection protocol
- Normal 84 to 138 mL/min
- Can calculate an estimated value with serum lab values (Cockroft and Gault formula)
- Urine Tests

➢ Urine electrolytes

- Urine specific gravity
- Urine osmolality

3. Daignostic Tests for ARF

➢ **Non-invasive tests**

- X-ray of kidneys, ureter, and bladder (KUB)(Size, shape, and position of kidneys, Calculi, cysts, and tumors
- Renal ultrasound(Size of kidneys, Obstruction)

➢ **Invasive tests**

- IV pyelogram
- Computed tomography (Structures, accumulation of fluid)
- Renal angiography(Abnormalities in blood flow; infarction, masses)
- Renal scan (Renal uptake of isotopes)
- Renal biopsy (Histologic changes)

Nursing Diagnosis

1. Excess Fluid Volume related to sodium and water retention and excess intake

• Body weight within 2 lb of dry weight

• Intake and output balanced; bilateral breath sounds clear; vital signs normal

Nursing Interventions

1. Obtain daily weights. Weight gain is best indicator of fluid gain.

2. Maintain accurate intake and output records .Identify imbalances.

3. Monitor respiratory status, including respiratory rate and crackles. Assess volume overload.

4. Assess heart rate, blood pressure, and respiratory rate. Indicate volume overload.

5. Administer all fluids and medications in the least amount of fluid possible.

6. Monitor blood and urine laboratory tests . Levels are altered in acute kidney injury.

2. Risk for Infection related to depressed immune response secondary to uremia and Impaired Skin Integrity.

Infection is absent • Patient is afebrile • WBC count and differential are normal • All cultures are negative.

Nursing Interventions

1. Monitor WBC count and culture results.

2. Monitor temperature.

3. Avoid invasive equipment whenever possible, such as indwelling urinary catheters and central lines.

4. Use aseptic technique for all procedures.

5. Perform pulmonary preventive techniques (turn, cough, deep breathing).

6. Assess potential sites of infection (urinary, pulmonary, wound, intravenous catheters).

3. Imbalanced Nutrition: Less Than Body Requirements related to uremia, altered oral mucous membranes, and dietary restrictions.

• Body weight at patient's baseline • Energy level appropriate

Nursing Interventions

1. Monitor body weight and caloric intake daily.

2. Collaborate with dietitian about nutritional needs.

3. Provide diet with essential nutrients but within restrictions.

4. Remove noxious stimuli from room.

4.Deficient Knowledge related to disease process and therapeutic regimen.

Patient and family have sufficient, accurate information related to condition to be informed participants in the care.

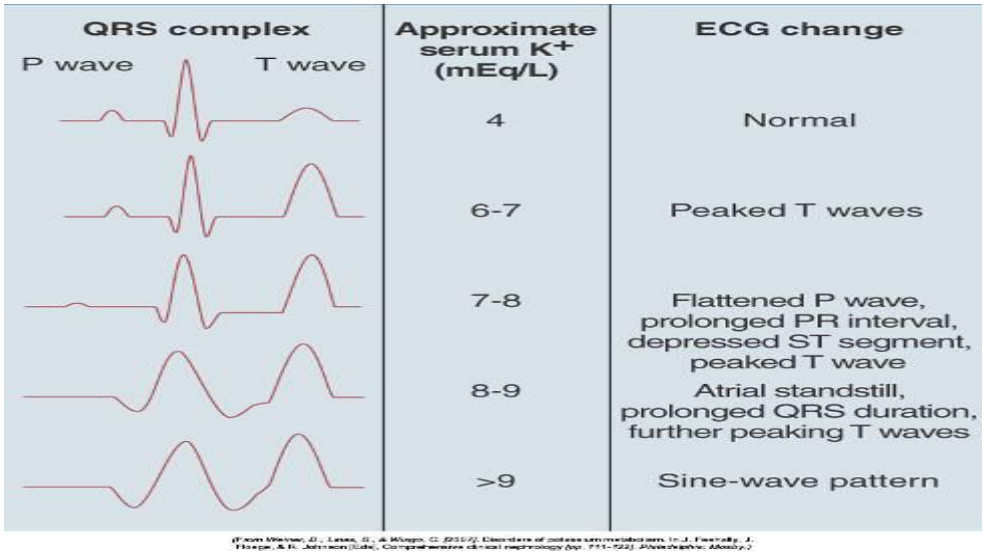
NURSING INTERVENTIONS

1. Provide specific, factual information on acute kidney injury, impact on the patient, and treatment plan.

2. Encourage patient and family to ask questions.

3. Encourage patient and family members to participate in care.

5. Fluid and Electrolyte Imbalances



- Hyperkalemia (Low excretion)
- Hyponatremia (Fluid retention)
- Hypocalcemia (Low excretion of phosphorus)
- Decreased level of vitamin D
- Hyperphosphatemia (Low excretion
- Hypermagnesium(Low excretion)

Medical Management of Acute Kidney Injury

Prerenal Causes

1. Early recognition and prompt treatment are essential.

2. Prompt replacement of extracellular fluids and aggressive treatment of shock may help prevent AKI.

3. Hypovolemia is treated in various ways, depending on the cause. Blood loss may necessitate blood transfusions, whereas patients with pancreatitis and peritonitis are usually treated with isotonic solutions such as normal saline.

4. Patients with cardiac instability usually require positive inotropic agents, antidysrhythmic agents, preload or afterload reducers, or an intraaortic balloon pump. Hypovolemia from intense vasodilation may require vasoconstrictor medications, isotonic fluid replacement, and antibiotics (if the patient has sepsis) until the underlying problem has been resolved.

5. . Invasive hemodynamic monitoring with a central venous catheter or pulmonary artery catheter may be considered in the management of fluid balance.

2. Intra-renal Causes: Acute Tubular Necrosis

Common interventions for the patient with ATN include:

1. Drug therapy.

2. Dietary management such as protein and electrolyte restrictions, management of fluid and electrolyte imbalances.

3. Renal replacement therapies such as intermittent hemodialysis or continuous renal replacement therapy (CRRT).

3. Post-renal Causes

1. Post-renal conditions are usually resolved with the insertion of an indwelling bladder catheter, either transurethral or suprapubic.

2. a ureteral stent may have to be placed if the obstruction is caused by calculi or carcinoma.

Pharmacological Management

Diuretics: Diuretic therapy in the treatment of patients with AKI is controversial

1. Diuretics may be used to manage volume overload.

2. A loop diuretic is commonly ordered. Large doses of furosemide are often needed to induce diuresis.

3. Mannitol, an osmotic diuretic often used in AKI caused by rhabdomyolysis, increases plasma volume and is believed to protect the kidney by minimizing postischemic swelling.

- **Dopamine**. The role of dopamine is controversial in the treatment of AKI.

- increase renal blood flow and GFR by stimulating the dopaminergic receptors in the kidney.

- Acetylcysteine, fenoldopam, theophylline(Prevent contrast-induced ARF.
- Epoetin alfa (Treat anemia, Must adjust dosages and timing of medication if patient on dialysis).
- Dietary Management

- Expenditure in catabolic patients with acute kidney injury is much higher than normal.

-Adequate energy, protein, and micronutrients to maintain homeostasis in patients who may be extremely catabolic.

Nutritional recommendations include the following;

• Caloric intake of 25 to 35 kcal/kg of ideal body weight per day

• Protein intake of no less than 0.8 g/kg. Patients who are extremely catabolic should receive 1.5 to 2.0 g/kg of ideal body weight per day—75% to 80% of which contains all the required essential amino acids.

• Sodium intake of 0.5 to 1.0 g/day

• Potassium intake of 20 to 50 mEq/day

• Calcium intake of 800 to 1200 mg/day

• Fluid intake equal to the volume of the patient's urine output plus an additional 600 to 1000 mL/day.

Management of Fluid, Electrolyte, and Acid-Base Imbalances

•Hyperkalemia: is common in AKI, especially if the patient is hypercatabolic.

-Hyperkalemia occurs when **potassium excretion** is reduced as a result of **the decrease in GFR**. Sudden changes in the serum potassium level can **cause dysrhythmias**, which may be fatal.

Three approaches are used to treat hyperkalemia:

(1) Reduce the body potassium content.

(2) Shift the potassium from outside the cell to inside the cell

– Glucose and insulin

– Alkali (sodium bicarbonate)

(3) Antagonize the membrane effect of the hyperkalemia.

– Calcium gluconate

– Regular insulin

– Albuterol 10 to 20 mg given by nebulized inhalation over 15 minutes.

Hyponatremia

-Generally occurs from water overload.

-However, as nephrons are progressively damaged, the ability to conserve sodium is lost, and major salt-wasting states can develop, causing hyponatremia.

- Hyponatremia is treated with fluid restriction, specifically restriction of free water intake.

Acid-base Imbalance

Treatment of metabolic acidosis depends on its severity.

–Intravenous sodium bicarbonate

–Rapid correction of the acidosis should be avoided, because tetany may occur as a result of hypocalcemia.

–Intravenous calcium gluconate may be prescribed.

Renal Replacement Therapy

- The decision to initiate renal replacement therapy is a clinical decision based on the fluid, electrolyte, and metabolic status of each patient.
- Renal replacement therapy options include;
- Intermittent hemodialysis.
- Continuous renal replacement therapy CRRT
- r peritoneal dialysis

Indications for dialysis:

The most common reasons for initiating dialysis include

- acidosis.
- Hyperkalemia.
- volume overload.
- Uremia.
- Dialysis is usually started early in the course of the renal dysfunction before uremic complications occur.
- dialysis is may be started for fluid management when total parenteral nutrition is administered in patients with impaired renal function

Principles and mechanisms: Dialysis therapy is based on two physical principles that operate simultaneously: **diffusion and ultrafiltration.**

➢ Diffusion (or clearance) is the movement of solutes such as urea from the patient's blood to the dialysate cleansing fluid, across a semipermeable membrane (the hemofilter). Substances such as bicarbonate may also cross in the opposite direction, from the dialysate through the semipermeable membrane into the patient's blood.

➢ Ultrafiltration is the removal of plasma water and some low– molecular weight particles by using a pressure or osmotic gradient.

➢ Ultrafiltration is primarily aimed at controlling fluid volume, whereas dialysis is aimed at decreasing waste products and treating fluid and electrolyte imbalances.

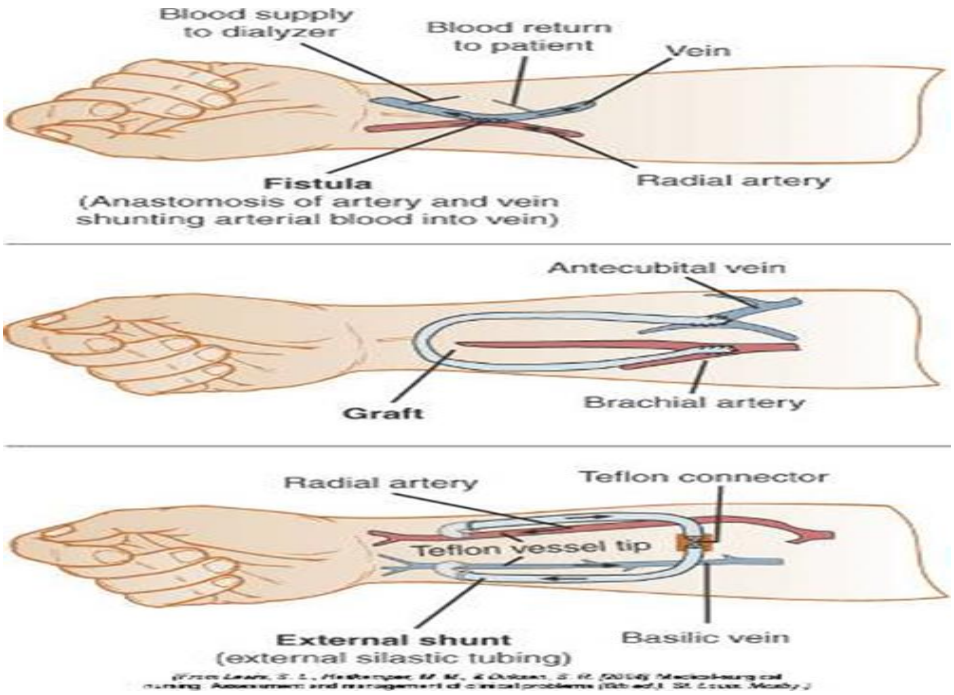
Vascular access

➢ Temporary percutaneous catheters

➢ Arteriovenous (AV) fistulas

➢ Grafts

➢ External shunts



Hemodialysis

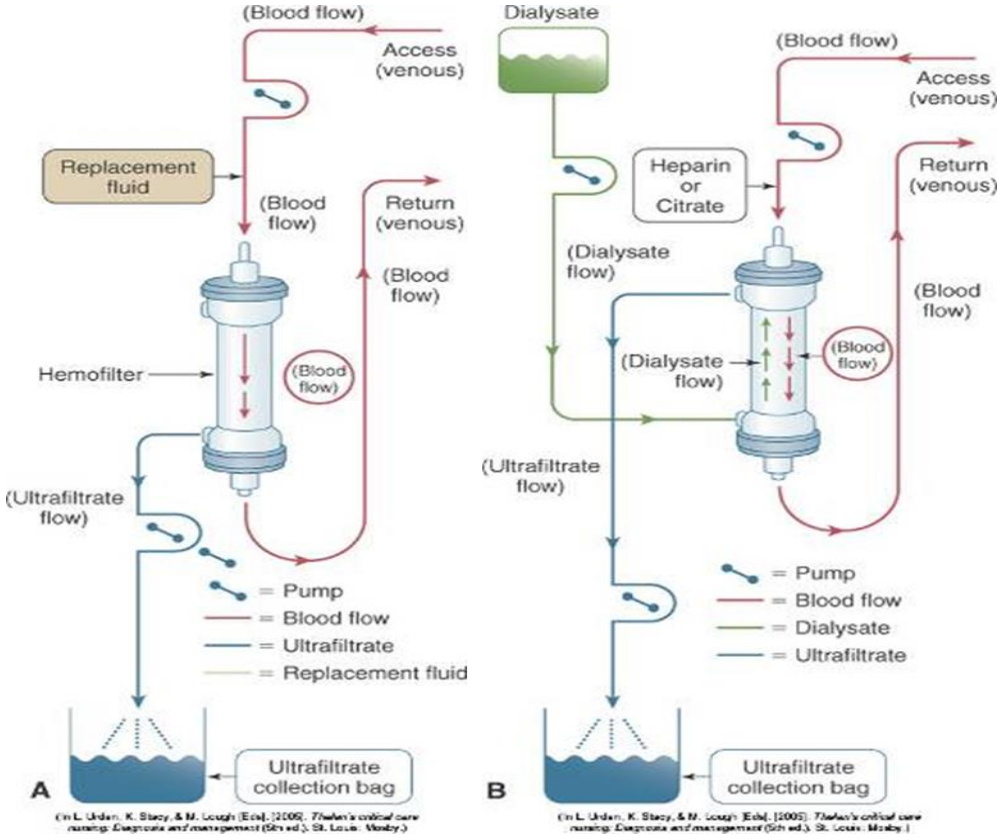
• Usually done at the bedside in the ICU

• Pre- and post-dialysis labs and weight

• Monitor for complications

➢ Cramps

- Bleeding/clotting
- Dialyzer reaction
- Hemolysis
- Dysrhythmias
- Infections
- Hypoxemia
- Pyrogen reactions
- Dialysis disequilibrium syndrome
- Vascular access dysfunction
- Technical errors (incorrect dialysate mixture, contaminated
- dialysate, or air embolism



Continuous renal replacement therapy

• CRRT is a slower type of dialysis that puts less stress on the heart. Instead of doing it over four hours, CRRT is done 24 hours a day to slowly and continuously clean out waste products and fluid from the patient. It requires special anticoagulation to keep the dialysis circuit from clotting.

- Used with patients too unstable for hemodialysis

- Advantages
  - More gradual solute removal
  - Flexible fluid administration
  - Minimal heparin
  - Can be done by staff nurses at the bedside
- Disadvantages
  - Bed rest
  - One-to-one nursing care

Peritoneal dialysis.

- Removal of solutes and fluids using the peritoneal membrane as a filter
- Rarely used in the critical care setting because it is less efficient.
- High risk of peritonitis.

Preventing Acute Renal Failure:

1. Provide adequate hydration to patients at risk for dehydration including: Before, during, and after surgery. Patients undergoing intensive diagnostic studies requiring fluid restriction and contrast agents (eg,barium enema, intravenous pyelograms),especially elderly patients who may have marginal renal reserve Patients with neoplastic disorders or disorders of metabolism (eg, gout) and those receiving chemotherapy.
2. Prevent and treat shock promptly with blood and fluid replacement.
3. Monitor central venous and arterial pressures and hourly urine output of critically ill patients to detect the onset of renal failure as early as possible.
4. Treat hypotension promptly.
5. Continually assess renal function (urine output, laboratory values) when appropriate.
6. Take precautions to ensure that the appropriate blood is administered to the correct patient in order to avoid severe transfusion reactions, which can precipitate renal failure.
7. Prevent and treat infections promptly. Infections can produce progressive renal damage.
8. Pay special attention to wounds, burns, and other precursors of sepsis.
9. To prevent infections from ascending in the urinary tract, give meticulous care to patients with indwelling catheters. Remove catheters as soon as possible.
10. To prevent toxic drug effects, closely monitor dosage, duration of use, and blood levels of all medications metabolized or excreted by the kidneys.