

RENAL FAILURE: ACUTE

Acute renal failure (ARF) has four well-defined stages: onset, oliguric or anuric, diuretic, and convalescent. Treatment depends on stage and severity of renal compromise. ARF can be divided into three major classifications, depending on site:

Prerenal: Prerenal failure is caused by interference with renal perfusion (e.g., blood volume depletion, volume shifts [“third-space” sequestration of fluid], or excessive/too-rapid volume expansion), manifested by decreased glomerular filtration rate (GFR). Disorders that lead to prerenal failure include cardiogenic shock, heart failure (HF), myocardial infarction (MI), burns, trauma, hemorrhage, septic or anaphylactic shock, and renal artery obstruction.

Renal (or intrarenal): Intrarenal causes for renal failure are associated with parenchymal changes caused by ischemia or nephrotoxic substances. Acute tubular necrosis (ATN) accounts for 90% of cases of acute oliguria. Destruction of tubular epithelial cells results from (1) ischemia/hypoperfusion (similar to prerenal hypoperfusion except that correction of the causative factor may be followed by continued oliguria for up to 30 days) and/or (2) direct damage from nephrotoxins.

Postrenal: Postrenal failure occurs as the result of an obstruction in the urinary tract anywhere from the tubules to the urethral meatus. Obstruction most commonly occurs with stones in the ureters, bladder, or urethra; however, trauma, edema associated with infection, prostate enlargement, and strictures also cause postrenal failure.

Note: Iatrogenically induced ARF should be considered when failure develops during or shortly after hospitalization. The most common causative factor is administration of potentially nephrotoxic agents.

CARE SETTING

Inpatient acute medical or surgical unit

RELATED CONCERNS

Metabolic acidosis (primary base bicarbonate deficiency)

Fluid and electrolyte imbalances

Psychosocial aspects of care

Renal dialysis

Renal failure: chronic

Sepsis/septicemia

Total nutritional support: parenteral/enteral feeding

Upper gastrointestinal/esophageal bleeding

Patient Assessment Database

ACTIVITY/REST

May report: Fatigue, weakness, malaise

May exhibit: Muscle weakness, loss of tone

CIRCULATION

May exhibit: Hypotension or hypertension (including malignant hypertension, eclampsia/pregnancy-induced hypertension)

Cardiac dysrhythmias

Weak/thready pulses, orthostatic hypotension (hypovolemia)

Jugular venous distension (JVD), full/bounding pulses (hypervolemia); flat neck veins (diuretic phase)

Generalized tissue edema (including periorbital area, ankles, sacrum)

Pallor (anemia); bleeding tendencies

ELIMINATION

May report: Change in usual urination pattern: Increased frequency, polyuria (early failure and early recovery), or decreased frequency/oliguria (later phase)

Dysuria, hesitancy, urgency, and retention (inflammation/obstruction/infection)

Abdominal bloating, diarrhea, or constipation

May exhibit: History of benign prostatic hyperplasia (BPH), or kidney/bladder stones/calculi
Change in urinary color, e.g., absence of color, deep yellow, red, brown, cloudy
Oliguria (may last 12–21 days and occurs in 70% of patients); polyuria (2–6 L/day of urine, lacking concentration and regulation of waste products)

FOOD/FLUID

May report: Weight gain (edema), weight loss (dehydration)
Nausea, anorexia, heartburn, vomiting
Metallic taste
Use of diuretics

May exhibit: Changes in skin turgor/moisture
Edema (generalized, dependent)

NEUROSENSORY

May report: Headache, blurred vision
Muscle cramps/twitching; “restless leg” syndrome; numbness, tingling

May exhibit: Altered mental state, e.g., decreased attention span, inability to concentrate, loss of memory, confusion, decreasing level of consciousness (LOC) (azotemia, electrolyte and acid-base imbalance)
Twitching, muscle fasciculations, seizure activity

PAIN/DISCOMFORT

May report: Flank pain, headache

May exhibit: Guarding/distraction behaviors, restlessness

RESPIRATION

May report: Shortness of breath

May exhibit: Tachypnea, dyspnea, increased rate/depth (Kussmaul’s respiration); ammonia breath
Cough productive of pink-tinged sputum (pulmonary edema)

SAFETY

May report: Recent transfusion reaction

May exhibit: Fever (sepsis, dehydration)
Petechiae, ecchymotic areas on skin
Pruritus, dry skin

TEACHING/LEARNING

May report: Family history of polycystic disease, hereditary nephritis, urinary calculus, malignancy
History of exposure to toxins, e.g., drugs, environmental poisons; substance abuse
Current/recent use of nephrotoxic drugs, e.g., aminoglycoside antibiotics, amphotericin B; anesthetics; vasodilators; nonsteroidal anti-inflammatory drugs (NSAIDs)
Recent diagnostic testing with radiographic contrast media
Concurrent conditions: Tumors in the urinary tract, Gram-negative sepsis; trauma/crush injuries, hemorrhage, disseminated intravascular coagulation (DIC), burns, electrocution injury; autoimmune disorders (e.g., scleroderma, vasculitis), vascular occlusion/surgery, diabetes mellitus (DM), cardiac/liver failure

Discharge plan considerations: **DRG projected mean length of inpatient stay: 5.9 days**
May require alteration/assistance with medications, treatments, supplies; transportation, homemaker/maintenance tasks
Refer to section at end of plan for postdischarge considerations.

DIAGNOSTIC STUDIES

Urine:

Volume: Usually less than 100 mL/24 hr (anuric phase) or 400 mL/24 hr (oliguric phase), which occurs within 24–48 hr after renal insult. Nonoliguric (more than 400 mL/24 hr) renal failure also occurs when renal damage is associated with nephrotoxic agents (e.g., contrast media or antibiotics).

Color: Dirty, brown sediment indicates presence of RBCs, hemoglobin, myoglobin, porphyrins.

Specific gravity: Less than 1.020 reflects kidney disease, e.g., glomerulonephritis, pyelonephritis with loss of ability to concentrate; fixed at 1.010 reflects severe renal damage.

pH: Greater than 7 found in urinary tract infections (UTIs), renal tubular necrosis, and chronic renal failure (CRF).

Osmolality: Less than 350 mOsm/kg is indicative of tubular damage, and urine/serum ratio is often 1:1.

Creatinine (Cr) clearance: Renal function may be significantly decreased before blood urea nitrogen (BUN) and serum Cr show significant elevation.

Sodium: Usually increased if ATN is cause for ARF, more than 40 mEq/L if kidney is not able to resorb sodium, although it may be decreased in other causes of prerenal failure.

Fractional sodium (F_{eNa}): Ratio of sodium excreted to total sodium filtered by the kidneys reveals inability of tubules to reabsorb sodium. Readings of less than 1% indicate prerenal problems, higher than 1% reflects intrarenal disorders.

Bicarbonate: Elevated if metabolic acidosis is present.

Red blood cells (RBCs): May be present because of infection, stones, trauma, tumor, or altered glomerular filtration (GF).

Protein: High-grade proteinuria (3–4+) strongly indicates glomerular damage when RBCs and casts are also present. Low-grade proteinuria (1–2+) and white blood cells (WBCs) may be indicative of infection or interstitial nephritis. In ATN, proteinuria is usually minimal.

Casts: Usually signal renal disease or infection. Cellular casts with brownish pigments and numerous renal tubular epithelial cells are diagnostic of ATN. Red casts suggest acute glomerular nephritis.

Blood:

BUN/Cr: Elevated and usually rise in proportion with ratio of 10:1 or higher.

Complete blood count (CBC): Hemoglobin (Hb) decreased in presence of anemia. RBCs often decreased because of increased fragility/decreased survival.

Arterial blood gases (ABGs): Metabolic acidosis (pH less than 7.2) may develop because of decreased renal ability to excrete hydrogen and end products of metabolism. Bicarbonate decreased.

Sodium: Usually increased, but may vary.

Potassium: Elevated related to retention and cellular shifts (acidosis) or tissue release (red cell hemolysis).

Chloride, phosphorus, and magnesium: Usually elevated.

Calcium: Decreased.

Serum osmolality: More than 285 mOsm/kg; often equal to urine.

Protein: Decreased serum level may reflect protein loss via urine, fluid shifts, decreased intake, or decreased synthesis because of lack of essential amino acids.

Radionuclide imaging: May reveal caliectasis, hydronephrosis, narrowing, and delayed filling or emptying as a cause of ARF.

Kidney, ureter, bladder (KUB) x-ray: Demonstrates size of kidneys/ureters/bladder, presence of cysts, tumors, and kidney displacement or obstruction (stones).

Retrograde pyelogram: Outlines abnormalities of renal pelvis and ureters.

Renal arteriogram: Assesses renal circulation and identifies extravascularities, masses.

Voiding cystoureterogram: Shows bladder size, reflux into ureters, retention.

Renal ultrasound: Determines kidney size and presence of masses, cysts, obstruction in upper urinary tract.

Nonnuclear computed tomography (CT) scan: Cross-sectional view of kidney and urinary tract detects presence/extent of disease.

Magnetic resonance imaging (MRI): Provides information about soft tissue damage.

Excretory urography (intravenous urogram or pyelogram): Radiopaque contrast concentrates in urine and facilitates visualization of KUB.

Endourology: Direct visualization may be done of urethra, bladder, ureters, and kidney to diagnose problems, biopsy, and remove small lesions and/or calculi.

Electrocardiogram (ECG): May be abnormal, reflecting electrolyte and acid-base imbalances.

NURSING PRIORITIES

1. Reestablish/maintain fluid and electrolyte balance.
2. Prevent complications.
3. Provide emotional support for patient/significant other (SO).
4. Provide information about disease process/prognosis and treatment needs.

DISCHARGE GOALS

1. Homeostasis achieved.

2. Complications prevented/minimized.
3. Dealing realistically with current situation.
4. Disease process/prognosis and therapeutic regimen understood.
5. Plan in place to meet needs after discharge.

NURSING DIAGNOSIS: Fluid Volume excess

May be relate to

Compromised regulatory mechanism (renal failure)

Possibly evidenced by

Intake greater than output, oliguria; changes in urine specific gravity

Venous distension; blood pressure (BP)/central venous pressure (CVP) changes

Generalized tissue edema, weight gain

Changes in mental status, restlessness

Decreased Hb/hematocrit (Hct), altered electrolytes; pulmonary congestion on x-ray

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Fluid Balance (NOC)

Display appropriate urinary output with specific gravity/laboratory studies near normal; stable weight, vital signs within patient's normal range; and absence of edema.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Fluid/Electrolyte Management (NIC)</p>	
<p>Independent</p>	
<p>Record accurate intake and output (I&O). Include “hidden” fluids such as IV antibiotic additives, liquid medications, ice chips, frozen treats. Measure gastrointestinal (GI) losses and estimate insensible losses, e.g., diaphoresis.</p>	<p>Low output (less than 400 mL/24 hr) may be first indicator of acute failure, especially in a high-risk patient. Accurate I&O is necessary for determining renal function and fluid replacement needs and reducing risk of fluid overload. <i>Note:</i> Hypervolemia occurs in the anuric phase of ARF.</p>
<p>Monitor urine specific gravity.</p>	<p>Measures the kidney’s ability to concentrate urine. In intrarenal failure, specific gravity is usually equal to/less than 1.010, indicating loss of ability to concentrate the urine.</p>
<p>Weigh daily at same time of day, on same scale, with same equipment and clothing.</p>	<p>Daily body weight is best monitor of fluid status. A weight gain of more than 0.5 kg/day suggests fluid retention.</p>
<p>Assess skin, face, dependent areas for edema. Evaluate degree of edema (on scale of +1–+4).</p>	<p>Edema occurs primarily in dependent tissues of the body, e.g., hands, feet, lumbosacral area. Patient can gain up to 10 lb (4.5 kg) of fluid before pitting edema is detected. Periorbital edema may be a presenting sign of this fluid shift because these fragile tissues are easily distended by even minimal fluid accumulation.</p>
<p>Monitor heart rate (HR), BP, and JVD/CVP.</p>	<p>Tachycardia and hypertension can occur because of (1) failure of the kidneys to excrete urine, (2) excessive fluid resuscitation during efforts to treat hypovolemia/hypotension or convert oliguric phase of renal failure, and/or (3) changes in the renin-angiotensin system. <i>Note:</i> Invasive monitoring may be needed for assessing intravascular volume, especially in patients with poor cardiac function.</p>
<p>Auscultate lung and heart sounds.</p>	<p>Fluid overload may lead to pulmonary edema and HF evidenced by development of adventitious breath sounds, extra heart sounds. (Refer to ND: Cardiac Output, risk for decreased, following.)</p>
<p>Assess level of consciousness; investigate changes in mentation, presence of restlessness.</p>	<p>May reflect fluid shifts, accumulation of toxins, acidosis, electrolyte imbalances, or developing hypoxia.</p>
<p>Plan oral fluid replacement with patient, within multiple restrictions. Intersperse desired beverages throughout 24 hr. Vary offerings, e.g., hot, cold, frozen.</p>	<p>Helps avoid periods without fluids, minimizes boredom of limited choices, and reduces sense of deprivation and thirst.</p>
<p>Collaborative</p>	
<p>Correct any reversible cause of ARF, e.g., replace blood loss, maximize cardiac output, discontinue nephrotoxic drug, relieve obstruction via surgery.</p>	<p>Kidneys may be able to return to normal functioning, preventing or limiting residual effects.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Fluid/Electrolyte Management (NIC)</p> <p>Collaborative</p> <p>Monitor laboratory/diagnostic studies, e.g.: BUN, Cr;</p> <p>Urine sodium and Cr;</p> <p>Serum sodium;</p> <p>Serum potassium;</p> <p>Hb/Hct;</p> <p>Serial chest x-rays.</p> <p>Administer/restrict fluids as indicated.</p> <p>Administer medication as indicated: Diuretics, e.g., furosemide (Lasix), bumetanide (Bumex), torsemide (Demadex), mannitol (Osmitol);</p>	<p>Assess progression and management of renal dysfunction/failure. Although both values may be increased, Cr is a better indicator of renal function because it is not affected by hydration, diet, and tissue catabolism. <i>Note:</i> Dialysis is indicated if ratio is higher than 10:1 or if therapy fails to correct fluid overload or metabolic acidosis.</p> <p>In ATN, tubular functional integrity is lost and sodium resorption is impaired, resulting in increased sodium excretion. Urine creatinine is usually decreased as serum creatinine elevates.</p> <p>Hyponatremia may result from fluid overload (dilutional) or kidney's inability to conserve sodium. Hypernatremia indicates total body water deficit.</p> <p>Lack of renal excretion and/or selective retention of potassium to excrete excess hydrogen ions leads to hyperkalemia, requiring prompt intervention.</p> <p>Decreased values may indicate hemodilution (hypervolemia); however, during prolonged failure, anemia frequently develops as a result of RBC loss/decreased production. Other possible causes (active or occult hemorrhage) should also be evaluated.</p> <p>Increased cardiac size, prominent pulmonary vascular markings, pleural effusion, infiltrates/congestion indicate acute responses to fluid overload or chronic changes associated with renal and heart failure.</p> <p>Fluid management is usually calculated to replace output from all sources plus estimated insensible losses (metabolism, diaphoresis). Prerenal failure (azotemia) is treated with volume replacement and/or vasopressors. The oliguric patient with adequate circulating volume or fluid overload who is unresponsive to fluid restriction and diuretics requires dialysis. <i>Note:</i> During oliguric phase, "push/pull" therapy (push IV fluids and diurese with diuretics) may be tried to stimulate kidney function.</p> <p>Given early in oliguric phase of ARF in an effort to convert to nonoliguric phase, flush the tubular lumen of debris, reduce hyperkalemia, and promote adequate urine volume.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Fluid/Electrolyte Management (NIC)</p> <p>Collaborative</p> <p>Antihypertensives, e.g., clonidine (Catapres), methyldopa (Aldomet), prazosin (Minipress);</p> <p>Calcium channel blockers;</p> <p>Prostaglandins.</p> <p>Insert/maintain indwelling catheter, as indicated.</p> <p>Prepare for dialysis as indicated, e.g., hemodialysis, peritoneal dialysis, or continuous renal replacement therapy (CRRT).</p>	<p>May be given to treat hypertension by counteracting effects of decreased renal blood flow and/or circulating volume overload.</p> <p>Given early in nephrotoxic ATN to reduce influx of calcium into kidney cells, thereby helping to maintain cell integrity and improve GFR.</p> <p>Vasodilatory effect may improve circulating volume and reestablish renal blood flow to aid in clearing nephrotoxic agents from nephrons.</p> <p>Catheterization excludes lower tract obstruction and provides means of accurate monitoring of urine output during acute phase; however, indwelling catheterization may be contraindicated because of increased risk of infection.</p> <p>Done to correct volume overload, electrolyte and acid-base imbalances, and to remove toxins. The type of dialysis chosen for ARF depends on the degree of hemodynamic compromise and patient's ability to withstand the procedure. (Refer to CP: Renal Dialysis)</p>

<p>NURSING DIAGNOSIS: Cardiac Output, risk for decreased</p> <p>Risk factors may include</p> <p>Fluid overload (kidney dysfunction/failure, overzealous fluid replacement)</p> <p>Fluid shifts, fluid deficit (excessive losses)</p> <p>Electrolyte imbalance (potassium, calcium); severe acidosis</p> <p>Uremic effects on cardiac muscle/oxygenation</p> <p>Possibly evidenced by</p> <p>[Not applicable; presence of signs and symptoms establishes an <i>actual</i> diagnosis.]</p> <p>DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:</p> <p>Circulation Status (NOC)</p> <p>Maintain cardiac output as evidenced by BP and HR/rhythm within patient's normal limits; peripheral pulses strong and equal with adequate capillary refill time.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Hemodynamic Regulation (NIC)</p> <p>Independent</p> <p>Monitor BP and HR.</p> <p>Observe ECG or telemetry for changes in rhythm.</p> <p>Auscultate heart sounds.</p> <p>Assess color of skin, mucous membranes, and nailbeds. Note capillary refill time.</p> <p>Note occurrence of slow pulse, hypotension, flushing, nausea/vomiting, and depressed level of consciousness (central nervous system [CNS] depression).</p> <p>Investigate reports of muscle cramps, numbness/tingling of fingers, with muscle twitching, hyperreflexia.</p> <p>Maintain bedrest or encourage adequate rest and provide assistance with care and desired activities.</p>	<p>Fluid volume excess, combined with hypertension (often occurs in renal failure) and effects of uremia, increases cardiac workload and can lead to cardiac failure. In ARF, cardiac failure is usually reversible.</p> <p>Changes in electromechanical function may become evident in response to progressing renal failure/accumulation of toxins and electrolyte imbalance. For example, hyperkalemia is associated with peaked T wave, wide QRS, prolonged PR interval, flattened/absent P wave. Hypokalemia is associated with flat T wave, peaked P wave, and appearance of U waves. Prolonged QT interval may reflect calcium deficit.</p> <p>Development of S₃/S₄ is indicative of failure. Pericardial friction rub may be only manifestation of uremic pericarditis, requiring prompt intervention/possibly acute dialysis.</p> <p>Pallor may reflect vasoconstriction or anemia. Cyanosis is a late sign and is related to pulmonary congestion and/or cardiac failure.</p> <p>Using drugs (e.g., antacids) containing magnesium can result in hypermagnesemia, potentiating neuromuscular dysfunction and risk of respiratory/cardiac arrest.</p> <p>Neuromuscular indicators of hypocalcemia, which can also affect cardiac contractility and function.</p> <p>Reduces oxygen consumption/cardiac workload.</p>
<p>Collaborative</p> <p>Monitor laboratory studies, e.g.:</p> <p style="padding-left: 20px;">Potassium;</p> <p style="padding-left: 20px;">Calcium;</p> <p style="padding-left: 20px;">Magnesium.</p>	<p>During oliguric phase, hyperkalemia is present but often shifts to hypokalemia in diuretic or recovery phase. Any potassium value associated with ECG changes requires intervention. <i>Note:</i> A serum level of 6.5 mEq or higher constitutes a medical emergency.</p> <p>In addition to its own cardiac effects, calcium deficit enhances the toxic effects of potassium.</p> <p>Dialysis or calcium administration may be necessary to combat the CNS-depressive effects of an elevated serum magnesium level.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Hemodynamic Regulation (NIC)</p>	
<p>Collaborative</p>	
<p>Administer/restrict fluids as indicated. (Refer to NDs: Fluid Volume excess p. 000, and Fluid Volume, risk for deficient)</p>	<p>Cardiac output depends on circulating volume (affected by both fluid excess and deficit) and myocardial muscle function.</p>
<p>Provide supplemental oxygen if indicated.</p>	<p>Maximizes available oxygen for myocardial uptake to reduce cardiac workload and cellular hypoxia.</p>
<p>Administer medications as indicated: Inotropic agents, e.g., digoxin (Lanoxin);</p>	<p>May be used to improve cardiac output by increasing myocardial contractility and stroke volume. Dosage depends on renal function and potassium balance to obtain therapeutic effect without toxicity.</p>
<p>Calcium gluconate;</p>	<p>Serum calcium is often low but usually does not require specific treatment in ARF. Calcium gluconate may be given to treat hypocalcemia and to offset the effects of hyperkalemia by modifying cardiac irritability.</p>
<p>Aluminum hydroxide gels (Amphojel, Basaljel);</p>	<p>Increased phosphate levels may occur as a result of failure of glomerular filtration and require use of phosphate-binding antacids to limit phosphate absorption from the GI tract.</p>
<p>Glucose/insulin solution;</p>	<p>Temporary measure to lower serum potassium by driving potassium into cells when cardiac rhythm is endangered.</p>
<p>Sodium bicarbonate or sodium citrate;</p>	<p>May be used to correct acidosis or hyperkalemia (by increasing serum pH) if patient is severely acidotic and not suffering from fluid overload.</p>
<p>Sodium polystyrene sulfonate (Kayexalate) with/without sorbitol.</p>	<p>Exchange resin trades sodium for potassium in the GI tract to lower serum potassium level. Sorbitol may be included to cause osmotic diarrhea to help excrete potassium.</p>
<p>Prepare for/assist with dialysis as necessary.</p>	<p>May be indicated for persistent dysrhythmias, progressive HF unresponsive to other therapies.</p>

NURSING DIAGNOSIS: Nutrition: imbalanced, risk for less than body requirements

Risk factors may include

Protein catabolism; dietary restrictions to reduce nitrogenous waste products
Increased metabolic needs
Anorexia, nausea/vomiting; ulcerations of oral mucosa

Possibly evidenced by

[Not applicable; presence of signs and symptoms establishes an *actual* diagnosis.]

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Nutritional Status (NOC)

Maintain/regain weight as indicated by individual situation, free of edema.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Nutrition Therapy (NIC)</p> <p>Independent</p> <p>Assess/document dietary intake.</p> <p>Provide frequent, small feedings.</p> <p>Give patient/SO a list of permitted foods/fluids and encourage involvement in menu choices.</p> <p>Offer frequent mouth care/rinse with dilute (0.25%) acetic acid solution; provide gum, hard candy, breath mints between meals.</p> <p>Weigh daily.</p>	<p>Aids in identifying deficiencies and dietary needs. General physical condition, uremic symptoms (e.g., nausea, anorexia, altered taste), and multiple dietary restrictions affect food intake.</p> <p>Minimizes anorexia and nausea associated with uremic state/diminished peristalsis.</p> <p>Provides patient with a measure of control within dietary restrictions. Food from home may enhance appetite.</p> <p>Mucous membranes may become dry and cracked. Mouth care soothes, lubricates, and helps freshen mouth taste, which is often unpleasant because of uremia and restricted oral intake. Rinsing with acetic acid helps neutralize ammonia formed by conversion of urea.</p> <p>The fasting/catabolic patient normally loses 0.2–0.5 kg/day. Changes in excess of 0.5 kg may reflect shifts in fluid balance.</p>
<p>Collaborative</p> <p>Monitor laboratory studies, e.g., BUN, prealbumin/albumin, transferrin, sodium, and potassium.</p> <p>Consult with dietitian/nutritional support team.</p>	<p>Indicators of nutritional needs, restrictions, and necessity for/effectiveness of therapy.</p> <p>Determines individual calorie and nutrient needs within the restrictions, and identifies most effective route and product, e.g., oral supplements, enteral or parenteral nutrition.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Nutrition Therapy (NIC)</p> <p>Collaborative</p> <p>Provide high-calorie, low-/moderate-protein diet. Include complex carbohydrates and fat sources to meet caloric needs (avoiding concentrated sugar sources) and essential amino acids.</p> <p>Restrict potassium, sodium, and phosphorus intake as indicated.</p> <p>Administer medications as indicated:</p> <ul style="list-style-type: none"> Iron preparations; Calcium carbonate; Vitamin D; B complex and C vitamins, folic acid; Antiemetics, e.g. prochlorperazine (Compazine), trimethobenzamide (Tigan). 	<p>The amount of needed exogenous protein is less than normal unless patient is on dialysis. Carbohydrates meet energy needs and limit tissue catabolism, preventing ketoacid formation from protein and fat oxidation. Carbohydrate intolerance mimicking DM may occur in severe renal failure. Essential amino acids improve nitrogen balance and nutritional status, stimulate repair of tubular epithelial cells, and enhance patient's ability to fight systemic complications.</p> <p>Restriction of these electrolytes may be needed to prevent further renal damage, especially if dialysis is not part of treatment, and/or during recovery phase of ARF.</p> <p>Iron deficiency may occur if protein is restricted, patient is anemic, or GI function is impaired.</p> <p>Restores normal serum levels to improve cardiac and neuromuscular function, blood clotting, and bone metabolism. <i>Note:</i> Low serum calcium is often corrected as phosphate absorption is decreased in the GI system. Calcium may be substituted as a phosphate binder.</p> <p>Necessary to facilitate absorption of calcium from the GI tract.</p> <p>Vital as coenzyme in cell growth and actions. Intake is decreased because of protein restrictions.</p> <p>Given to relieve nausea/vomiting and may enhance oral intake.</p>

<p>NURSING DIAGNOSIS: Infection, risk for</p> <p>Risk factors may include</p> <ul style="list-style-type: none"> Depression of immunologic defenses (secondary to uremia) Invasive procedures/devices (e.g., urinary catheter) Changes in dietary intake/malnutrition <p>Possibly evidenced by</p> <p>[Not applicable; presence of signs and symptoms establishes an <i>actual</i> diagnosis.]</p> <p>DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:</p> <p>Immune Status (NOC)</p> <p>Experience no signs/symptoms of infection.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Infection Protection (NIC)</p> <p>Independent</p> <p>Promote good handwashing by patient and staff.</p> <p>Avoid invasive procedures, instrumentation, and manipulation of indwelling catheters whenever possible. Use aseptic technique when caring for/manipulating IV/invasive lines. Change site/dressings per protocol. Note edema, purulent drainage.</p> <p>Provide routine catheter care and promote meticulous perianal care. Keep urinary drainage system closed and remove indwelling catheter as soon as possible.</p> <p>Encourage deep breathing, coughing, frequent position changes.</p> <p>Assess skin integrity. (Refer to CP: Renal Failure: Chronic; ND: Skin Integrity, risk for impaired.)</p> <p>Monitor vital signs.</p> <p>Collaborative</p> <p>Monitor laboratory studies, e.g., WBC count with differential.</p> <p>Obtain specimen(s) for culture and sensitivity and administer appropriate antibiotics as indicated.</p>	<p>Reduces risk of cross-contamination.</p> <p>Limits introduction of bacteria into body. Early detection/treatment of developing infection may prevent sepsis.</p> <p>Reduces bacterial colonization and risk of ascending UTI.</p> <p>Prevents atelectasis and mobilizes secretions to reduce risk of pulmonary infections.</p> <p>Excoriations from scratching may become secondarily infected.</p> <p>Fever (higher than 100.4°F) with increased pulse and respirations is typical of increased metabolic rate resulting from inflammatory process, although sepsis can occur without a febrile response.</p> <p>Although elevated WBCs may indicate generalized infection, leukocytosis is commonly seen in ARF and may reflect inflammation/injury within the kidney. A shifting of the differential to the left is indicative of infection.</p> <p>Verification of infection and identification of specific organism aids in choice of the most effective treatment. <i>Note:</i> A number of anti-infective agents require adjustments of dose and/or time while renal clearance is impaired.</p>

NURSING DIAGNOSIS: Fluid Volume, risk for deficient

Risk factors may include

Excessive loss of fluid (diuretic phase of ARF, with rising urinary volume and delayed return of tubular reabsorption capabilities)

Possibly evidenced by

[Not applicable; presence of signs and symptoms establishes an *actual* diagnosis.]

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Fluid Balance (NOC)

Display I&O near balance; good skin turgor, moist mucous membranes, palpable peripheral pulses, stable weight and vital signs, electrolytes within normal range.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Fluid Monitoring (NIC)</p> <p>Independent</p> <p>Measure I&O accurately. Weigh daily. Calculate insensible fluid losses.</p> <p>Provide allowed fluids throughout 24-hr period.</p> <p>Monitor BP (noting postural changes) and HR.</p> <p>Note signs/symptoms of dehydration, e.g., dry mucous membranes, thirst, dulled sensorium, peripheral vasoconstriction.</p> <p>Control environmental temperature; limit bed linens as indicated.</p>	<p>Helps estimate fluid replacement needs. Fluid intake should approximate losses through urine, nasogastric/wound drainage, and insensible losses (e.g., diaphoresis and metabolism). <i>Note:</i> Some sources believe that fluid replacement should not exceed two-thirds of the previous day's output to prevent prolonging the diuresis.</p> <p>Diuretic phase of ARF may revert to oliguric phase if fluid intake is not maintained or nocturnal dehydration occurs.</p> <p>Orthostatic hypotension and tachycardia suggest hypovolemia.</p> <p>In diuretic or postobstructive phase of renal failure, urine output can exceed 3 L/day. Extracellular fluid volume depletion activates the thirst center, and sodium depletion causes persistent thirst, unrelieved by drinking water. Continued fluid losses/inadequate replacement may lead to hypovolemic state.</p> <p>May reduce diaphoresis, which contributes to overall fluid losses.</p>
<p>Collaborative</p> <p>Monitor laboratory studies, e.g., sodium.</p>	<p>In nonoliguric ARF or in diuretic phase of ARF, large urine losses may result in sodium wasting while elevated urinary sodium acts osmotically to increase fluid losses. Restriction of sodium may be indicated to break the cycle.</p>

NURSING DIAGNOSIS: Knowledge, deficient [Learning Need] regarding condition, prognosis, treatment, self-care, and discharge needs

May be related to

- Lack of exposure/recall
- Information misinterpretation
- Unfamiliarity with information resources

Possibly evidenced by

- Questions/request for information, statement of misconception
- Inaccurate follow-through of instructions/development of preventable complications

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Knowledge: Disease Process (NOC)

- Verbalize understanding of condition/disease process, prognosis, and potential complications.
- Identify relationship of signs/symptoms to the disease process and correlate symptoms with causative factors.

Knowledge: Treatment Regimen (NOC)

- Verbalize understanding of therapeutic needs.
- Initiate necessary lifestyle changes and participate in treatment regimen.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Teaching: Disease Process (NIC)</p> <p>Independent</p> <p>Review disease process, prognosis, and precipitating factors if known.</p> <p>Explain level of renal function after acute episode is over.</p> <p>Discuss renal dialysis or transplantation if these are likely options for the future.</p> <p>Review dietary plan/restrictions. Include fact sheet listing food restrictions.</p> <p>Encourage patient to observe characteristics of urine and amount/frequency of output.</p> <p>Establish regular schedule for weighing.</p> <p>Review fluid intake/restriction. Remind patient to spread fluids over entire day and to include all fluids (e.g., ice) in daily fluid counts.</p>	<p>Provides knowledge base from which patient can make informed choices.</p> <p>Patient may experience residual defects in kidney function, which may/may not be permanent.</p> <p>Although these options would have been previously presented by the physician, patient may now be at a point when options need to be considered/decisions made and may desire additional input.</p> <p>Adequate nutrition is necessary to promote healing/tissue regeneration; adherence to restrictions may prevent complications.</p> <p>Changes may reflect alterations in renal function/need for dialysis.</p> <p>Useful tool for monitoring fluid and dietary status/needs.</p> <p>Depending on the cause/stage of ARF, patient may need to either restrict or increase intake of fluids.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Teaching: Disease Process (NIC)</p> <p>Independent</p> <p>Discuss activity restriction and gradual resumption of desired activity. Encourage use of energy-saving, relaxation, and diversional techniques.</p> <p>Discuss reality of continued presence of fatigue.</p> <p>Determine/prioritize ADLs and personal responsibilities. Identify available resources/support systems.</p> <p>Recommend scheduling activities with adequate rest periods.</p> <p>Discuss/review medication use. Encourage patient to discuss all medications (including over-the-counter [OTC] drugs) and herbal supplements with physician.</p> <p>Stress necessity of follow-up care, laboratory studies.</p> <p>Identify symptoms requiring medical intervention, e.g., decreased urinary output, sudden weight gain, presence of edema, lethargy, bleeding, signs of infection, altered mentation.</p>	<p>Patient with severe ARF may need to restrict activity and/or may feel weak for an extended period during lengthy recovery phase, requiring measures to conserve energy and reduce boredom/depression.</p> <p>Decreased metabolic energy production, presence of anemia, and states of discomfort commonly result in fatigue.</p> <p>Helps patient manage lifestyle changes and meet personal/family needs.</p> <p>Prevents excessive fatigue and conserves energy for healing, tissue regeneration.</p> <p>Medications that are concentrated in/excreted by the kidneys can cause toxic cumulative reactions and/or permanent damage to kidneys. Some supplements may interact with prescribed medications/contain electrolytes.</p> <p>Renal function may be slow to return following acute failure (up to 12 mo), and deficits may persist, requiring changes in therapy to avoid recurrence/complications.</p> <p>Prompt evaluation and intervention may prevent serious complications/progression to CRF.</p>

POTENTIAL CONSIDERATIONS following acute hospitalization (dependent on patient's age, physical condition/presence of complications, personal resources, and life responsibilities)

Fluid Volume, deficient (specify)—dependent on cause, duration, and stage of recovery.

Fatigue—decreased metabolic energy production/dietary restriction, anemia, increased energy requirements, e.g., fever/

inflammation, tissue regeneration.

Infection, risk for—depression of immunologic defenses (secondary to uremia), changes in dietary intake/malnutrition, increased environmental exposure.

Therapeutic Regimen: ineffective management—complexity of therapeutic regimen, economic difficulties, perceived benefit.