

SEPSIS/SEPTICEMIA

Sepsis is a syndrome characterized by clinical signs and symptoms of severe infection that may progress to septicemia and septic shock. *Septicemia* implies the presence of an infection of the blood caused by rapidly multiplying microorganisms or their toxins, which can result in profound physiological changes and systemic sepsis. The pathogens can be bacteria, fungi, viruses, or rickettsiae. The most common causes of septicemia are Gram-negative bacteria (and endotoxins), staphylococci, and *Candida*. If the defense system of the body is not effective in controlling the invading microorganisms, septic shock may result, characterized by altered hemodynamics, impaired cellular function, and multiple system failure.

Patients at highest risk for bacteremia and septic shock include the elderly, infants, and immunosuppressed patients with chronic diseases (e.g., diabetes); postoperative patients; and those with ventilators, invasive lines, and catheters. Early signs and symptoms may be vague, and sepsis can develop subtly until sudden, overwhelming septic shock is present, affecting multiple organ systems.

CARE SETTING

Although severely ill patients may require admission to an intensive care unit (ICU), this plan addresses care on an inpatient acute medical-surgical unit.

RELATED CONCERNS

AIDS

Chronic obstructive pulmonary disease (COPD) and asthma

Disaster considerations

Fluid and electrolyte imbalances

Metabolic acidosis (primary base bicarbonate deficiency)

Peritonitis

Pneumonia, microbial

Psychosocial aspects of care

Pulmonary tuberculosis (TB)

Renal Failure: Acute

Surgical Intervention

Total nutritional support: parenteral/enteral feeding

Ventilatory assistance (mechanical)

Patient Assessment Database

Data depend on the type, location, duration of the infective process and organ involvement.

ACTIVITY/REST

May report:	Fatigue, malaise
May exhibit:	Mental status changes, e.g., withdrawn, lethargic Respiration/heart rate increased with activity

CIRCULATION

May exhibit:	Blood pressure (BP) normal/slightly low-normal range (as long as cardiac output remains elevated); profound hypotension (late stage) Peripheral pulses bounding, rapid (hyperdynamic phase); weak/thready/easily obliterated, extreme tachycardia (shock) Heart sounds: Dysrhythmias and development of S ₃ suggest myocardial dysfunction, effects of acidosis/electrolyte imbalance Skin warm, dry, flushed (vasodilation); or pale, cold, clammy, mottled (vasoconstriction)
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ELIMINATION

May exhibit:	Urinary output decreased, concentrated; progressing to oliguria, anuria Urine cloudy, malodorous
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FOOD/FLUID

- May report:** Loss of appetite; nausea/vomiting
May exhibit: Weight loss, decreased subcutaneous fat/muscle mass (malnutrition)
Diminished/absent bowel sounds
Extremity and generalized edema

NEUROSENSORY

- May report:** Headache; dizziness, fainting
May exhibit: Restlessness, apprehension, confusion, disorientation, delirium/coma

PAIN/DISCOMFORT

- May report:** Abdominal tenderness, localized pain/discomfort
Generalized urticaria/pruritus

RESPIRATION

- May report:** Shortness of breath
May exhibit: Tachypnea with decreased respiratory depth, dyspnea; rapid labored respirations
Basilar crackles, rhonchi, wheezes (presence of pneumonia; developing pulmonary complications/onset of cardiac decompensation)

SAFETY

- May report:** History of recent/current infection, viral illness; cancer therapies, use of corticosteroids/other immunosuppressant medications
May exhibit: Temperature: Usually elevated (101°F or higher) but may be normal in elderly or compromised patient; occasionally subnormal (lower than 98.6°F)
Shaking chills
Poor/delayed wound healing, purulent drainage, localized erythema
Mascular erythematous rash, petechiae; oozing/bleeding from invasive line sites, wounds, mucous membranes

SEXUALITY

- May report:** Perineal pruritus
Recent childbirth/abortion
May exhibit: Maceration of vulva, purulent vaginal drainage

TEACHING/LEARNING

- May report:** Chronic/debilitating health problems, e.g., liver, renal, cardiac disease; cancer, diabetes mellitus (DM), alcoholism
History of splenectomy
Recent surgery/invasive procedures, traumatic wounds
Antibiotic use (recent or long-term)
Discharge plan considerations: **DRG projected mean length of inpatient stay: 5.5 days**
May require assistance with wound care/supplies, treatments, self-care and homemaker tasks
Refer to section at end of plan for postdischarge considerations.

DIAGNOSTIC STUDIES

Cultures (wound, sputum, urine, blood): May identify organism(s) causing the sepsis. Sensitivity determines most effective drug choices. Catheter/intravascular line tips may need to be removed and cultured if the portal of entry is unknown. *Note:* Diagnosis does not require positive blood cultures because up to 40% of patients who develop septic shock do not display an identified bacterium by culture.

Complete blood count (CBC): Hematocrit (Hct) level may be elevated in hypovolemic states because of hemoconcentration. Leukopenia (decreased white blood cells [WBCs]) occurs early, followed by a rebound leukocytosis (15,000–30,000) with increased bands (shift to the left), indicating rapid production of immature WBCs. Neutrophils (also called granulocytes, polys, or polymorphonuclear neutrophils [PMNs]) may be elevated or depressed. Counts below 500/mL indicate immune system exhaustion.

Serum electrolytes: Various imbalances may occur because of acidosis, fluid shifts, and altered renal function.

Clotting studies:

Platelets: Decreased levels (thrombocytopenia) can occur because of platelet aggregation.

Prothrombin time (PT)/activated partial thromboplastin time (aPTT): May be prolonged, indicating coagulopathy associated with liver ischemia, circulating toxins, shock state.

Serum lactate: Elevated in metabolic acidosis, liver dysfunction, shock.

Serum glucose: Hyperglycemia occurs, reflecting gluconeogenesis and glycogenolysis in the liver in response to cellular starvation/ alteration in metabolism.

Blood urea nitrogen (BUN)/Creatinine (Cr): Increased levels are associated with dehydration, renal impairment/failure, and liver dysfunction/failure.

Arterial blood gases (ABGs): Respiratory alkalosis and hypoxemia may occur early. In later states, hypoxemia, respiratory acidosis, lactic and metabolic acidosis occur because of failure of compensatory mechanisms.

Urinalysis: Presence of WBCs/bacteria suggests infection. Protein and red blood cells (RBCs) are often present.

X-rays: Abdominal and lower chest films indicating free air in the abdomen may suggest infection due to perforated abdominal/ pelvic organ.

Electrocardiogram (ECG): May show ST-segment and T-wave changes and dysrhythmia resembling myocardial infarction.

NURSING PRIORITIES

1. Eliminate infection.
2. Support tissue perfusion/circulatory volume.
3. Prevent complications.
4. Provide information about disease process, prognosis, and treatment needs.

DISCHARGE GOALS

1. Infection eliminated/controlled.
2. Homeostasis maintained.
3. Complications prevented/minimized.
4. Disease process, prognosis, and therapeutic regimen understood.
5. Plan in place to meet needs after discharge.

NURSING DIAGNOSIS: Infection, risk for [progression of sepsis to septic shock, development of opportunistic infections]

Risk factors may include

Compromised immune system

Failure to recognize/treat infection and/or exercise proper preventive measures

Invasive procedures, environmental exposure (nosocomial)

Possibly evidenced by

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

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Infection Status (NOC)

Achieve timely healing, be free of purulent secretions/drainage or erythema, and be afebrile.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Infection Control (NIC)</p> <p>Independent</p> <p>Provide isolation/monitor visitors as indicated.</p> <p>Wash hands with antibacterial soap before/after each care activity, even if gloves are used.</p> <p>Encourage/provide frequent position changes, deep-breathing/coughing exercises.</p> <p>Encourage patient to cover mouth and nose with tissue during coughs/sneezes. Place in private room, if indicated. Wear mask when providing direct care as appropriate.</p> <p>Limit use of invasive devices/procedures when possible. Remove lines/devices when infection is present and replace if necessary.</p> <p>Inspect wounds/site of invasive devices daily, paying particular attention to parenteral nutrition lines. Document signs of local inflammation/infection, changes in character of wound drainage, sputum, or urine.</p> <p>Investigate reports of pain out of proportion to visible signs.</p> <p>Maintain sterile technique when changing dressings, suctioning, providing site care, e.g., invasive line, urinary catheter.</p> <p>Wear gloves/gowns when caring for open wounds/anticipating direct contact with secretions or excretions.</p> <p>Dispose of soiled dressings/materials in double bag.</p>	<p>Body substance isolation (BSI) should be used for all infectious patients. Wound/linen isolation and handwashing may be all that is required for draining wounds. Patients with diseases transmitted through air may also need airborne and droplet precautions. Reverse isolation/restriction of visitors may be needed to protect the immunosuppressed patient.</p> <p>Reduces risk of cross-contamination because gloves may have unnoticeable defects, get torn or damaged during use. Some pathogens may survive on hands for 3+ hr after exposure. <i>Note:</i> Methicillin-resistant <i>Staphylococcus aureus</i> (MRSA) is most commonly transmitted via direct contact with healthcare workers who fail to wash hands between patient contacts.</p> <p>Good pulmonary toilet may reduce respiratory compromise.</p> <p>Prevents spread of infection via airborne droplets.</p> <p>Reduces number of sites for entry of opportunistic organisms.</p> <p>May provide clue to portal of entry, type of primary infecting organism(s), as well as early identification of secondary infections. <i>Note:</i> High nutrient content of total parenteral nutrition (TPN) provides excellent medium for bacterial growth.</p> <p>Pressure-like pain over area of cellulitis may indicate development of necrotizing fasciitis due to group A beta-hemolytic streptococci (GABS), necessitating prompt intervention.</p> <p>Prevents introduction of bacteria, reducing risk of nosocomial infection.</p> <p>Prevents spread of infection/cross-contamination.</p> <p>Reduces contamination/soilage of area; limits spread of airborne organisms.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Infection Control (NIC)</p> <p>Independent</p> <p>Monitor temperature trends.</p> <p>Observe for shaking chills and profuse diaphoresis.</p> <p>Monitor for signs of deterioration of condition/failure to improve with therapy.</p> <p>Inspect oral cavity for white plaques (thrush). Investigate reports of vaginal/perineal itching or burning.</p> <p>Collaborative</p> <p>Obtain specimens of urine, blood, sputum, wound, invasive lines/tubes as indicated for Gram stain, culture, and sensitivity.</p> <p>Monitor laboratory studies, e.g., WBC count with neutrophil and band counts.</p> <p>Administer medications as indicated: Anti-infective agents: broad-spectrum antibiotics, e.g., methicillin; Gram-negative agents, e.g., ticarcillin disodium (Ticar); Gram-positive, e.g., nafcillin (Nafcil), vancomycin (Vancocin); aminoglycosides, e.g., tobramycin (Nebcin), gentamicin (Garamycin); cephalosporins, e.g., cefotaxime (Claforan);</p> <p>Immune globulins as appropriate.</p> <p>Assist with/prepare for incision and drainage of wound, irrigation, application of warm/moist soaks, as indicated.</p> <p>Prepare for hyperbaric therapy as appropriate.</p>	<p>RATIONALE</p> <p>Fever (101°F–105°F/38.5°C–40°C) is the result of endotoxin effect on the hypothalamus and pyrogen-released endorphins. Hypothermia (lower than 96°F/36°C) is a grave sign reflecting advancing shock state, decreased tissue perfusion, and/or failure of the body’s ability to mount a febrile response.</p> <p>Chills often precede temperature spikes in presence of generalized infection.</p> <p>May reflect inappropriate/inadequate antibiotic therapy or overgrowth of resistant or opportunistic organisms.</p> <p>Depression of immune system and use of antibiotics increase risk of secondary infections, particularly yeast.</p> <p>Identification of portal of entry and organism causing the septicemia is crucial to effective treatment.</p> <p>The normal ratio of neutrophils to total WBCs is at least 50%; however, when WBC count is markedly decreased, calculating the absolute neutrophil count is more pertinent to evaluating immune status. Likewise, an initial elevation of band cells reflects the body’s attempt to mount a response to the infection, whereas a decline indicates decompensation.</p> <p>Specific antibiotics are determined by culture and sensitivity results, but therapy is usually initiated before obtaining results, using broad-spectrum antibiotics and/or based on most likely infecting organisms. Concomitant use of antimicrobials is often beneficial, but dosage must be balanced against renal function/clearance. <i>Note:</i> Vancomycin is drug of choice for MRSA because ciprofloxacin (Cipro) is losing effectiveness and generating widespread resistance.</p> <p>May boost/provide temporary immunity to general infection or specific illness, e.g., varicella zoster, rabies.</p> <p>Facilitates removal of purulent material/necrotic tissue and promotes healing.</p> <p>Exposure to increased ambient oxygen tension enhances oxygen delivery to cells to combat anerobic infections.</p>

NURSING DIAGNOSIS: Hyperthermia

May be related to

Increased metabolic rate, illness

Dehydration

Direct effect of circulating endotoxins on the hypothalamus, altering temperature regulation

Possibly evidenced by

Increase in body temperature higher than normal range

Flushed skin, warm to touch

Increased respiratory rate, tachycardia

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Thermoregulation (NOC)

Demonstrate temperature within normal range, be free of chills.

Experience no associated complications

ACTIONS/INTERVENTIONS	RATIONALE
<p>Fever Treatment (NIC)</p> <p>Independent</p>	
<p>Monitor patient temperature (degree and pattern); note shaking chills/profuse diaphoresis.</p>	<p>Temperature of 102°F–106°F (38.9°C–41.1°C) suggests acute infectious disease process. Fever pattern may aid in diagnosis; e.g., sustained or continuous fever curves lasting more than 24 hr suggest pneumococcal pneumonia, scarlet or typhoid fever; remittent fever (varying only a few degrees in either direction) reflects pulmonary infections; intermittent curves or fever that returns to normal once in 24-hr period suggests septic episode, septic endocarditis, or tuberculosis (TB). Chills often precede temperature spikes. <i>Note:</i> Use of antipyretics alters fever patterns and may be restricted until diagnosis is made or if fever remains higher than 102°F (38.9°C).</p>
<p>Monitor environmental temperature; limit/add bed linens as indicated.</p>	<p>Room temperature/number of blankets should be altered to maintain near-normal body temperature.</p>
<p>Provide tepid sponge baths; avoid use of alcohol.</p>	<p>May help reduce fever. <i>Note.</i> Use of ice water/alcohol may cause chills, actually elevating temperature. In addition, alcohol is very drying to skin.</p>
<p>Collaborative</p>	
<p>Administer antipyretics, e.g., acetylsalicylic acid (ASA) (aspirin), acetaminophen (Tylenol).</p>	<p>Used to reduce fever by its central action on the hypothalamus; fever should be controlled in patients who are neutropenic or asplenic. However, fever may be beneficial in limiting growth of organisms and enhancing autodestruction of infected cells.</p>
<p>Provide cooling blanket.</p>	<p>Used to reduce fever, usually higher than 104°F–105°F (39.5°C–40°C), when brain damage/seizures can occur.</p>

NURSING DIAGNOSIS: Tissue Perfusion, risk for ineffective

Risk factors may include

Relative/actual hypovolemia

Reduction of arterial/venous blood flow: selective vasoconstriction, vascular occlusion (intimal damage/microemboli)

Possibly evidenced by

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

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Circulation Status (NOC)

Display adequate perfusion as evidenced by stable vital signs, palpable peripheral pulses, skin warm and dry, usual level of mentation, individually appropriate urinary output, and active bowel sounds.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Shock Prevention (NIC)</p> <p>Independent</p> <p>Maintain bedrest; assist with care activities.</p> <p>Monitor trends in BP, especially noting progressive hypotension and widening pulse pressure.</p> <p>Monitor heart rate, rhythm. Note dysrhythmias.</p> <p>Note quality/strength of peripheral pulses.</p> <p>Assess respiratory rate, depth, and quality. Note onset of severe dyspnea.</p>	<p>Decreases myocardial workload and O₂ consumption, maximizing effectiveness of tissue perfusion.</p> <p>Hypotension develops as microorganisms invade the bloodstream, stimulating release or activation of chemical and hormonal substances, which initially results in peripheral vasodilation, decreased systemic vascular resistance (SVR), and relative hypovolemia. As shock progresses, cardiac output becomes severely depressed because of major alterations in contractility and preload/afterload, producing profound hypotension.</p> <p>Tachycardia occurs because of sympathetic nervous system stimulation secondary to stress response and to compensate for the relative hypovolemia and hypotension. Cardiac dysrhythmias can occur as a result of hypoxia, acid-base/electrolyte imbalance, and/or low-flow perfusion state.</p> <p>Initially the pulse is strong/bounding because of increased cardiac output (CO). Pulse may become weak/thready because of sustained hypotension, decreased cardiac output, and peripheral vasoconstriction if the shock state progresses.</p> <p>Increased respirations occur in response to direct effects of endotoxins on the respiratory center in the brain, as well as developing hypoxia, stress, and fever. Respiration can become shallow as respiratory insufficiency develops, creating risk of acute respiratory failure. (Refer to ND: Gas Exchange, risk for impaired)</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Shock Prevention (NIC)</p>	
<p>Independent</p>	
<p>Investigate changes in sensorium, e.g., mental cloudiness, agitation, restlessness, personality changes, delirium, stupor, coma.</p>	<p>Changes reflect alterations in cerebral perfusion, hypoxemia, and/or acidosis.</p>
<p>Assess skin for changes in color, temperature, moisture.</p>	<p>Compensatory mechanisms of vasodilation results in warm, dry, pink skin, which is characteristic of hyperperfusion in hyperdynamic phase of early septic shock. If shock state progresses, compensatory vasoconstriction occurs, shunting blood to vital organs, reducing peripheral blood flow, and creating cool, clammy, pale/dusk skin.</p>
<p>Record hourly urinary output and specific gravity.</p>	<p>Decreasing urinary output with increased specific gravity indicates diminished renal perfusion related to fluid shifts and selective vasoconstriction. There may be transient polyuria during hyperdynamic phase (while cardiac output is elevated), but this may progress to oliguria. <i>Note:</i> Acute renal failure may herald development of hemolytic uremic syndrome (HUS) resulting from <i>Escherichia coli</i> infection (other manifestations include hemolytic anemia and thrombocytopenia.)</p>
<p>Auscultate bowel sounds.</p>	<p>Reduced blood flow to the mesentery (splanchnic vasoconstriction) decreases peristalsis and may lead to paralytic ileus or possibly trigger multiple organ dysfunction syndrome (MODS).</p>
<p>Monitor gastric pH as indicated. Hematest gastric secretions/stools for occult blood.</p>	<p>Stress of illness and use of steroids increase risk of gastric mucosal erosion/bleeding.</p>
<p>Evaluate lower extremities for local tissue swelling, erythema, positive Homans' sign.</p>	<p>Verous stasis and infectious process may result in the development of thrombosis.</p>
<p>Monitor for signs of bleeding, e.g., oozing from puncture sites/suture lines, petechiae, ecchymoses, hematuria, epistaxis, hemoptysis, hematemesis.</p>	<p>Coagulopathy/disseminated intravascular coagulation (DIC) may occur related to accelerated clotting in the microcirculation (activation of chemical mediators, vascular insufficiency, and cell destruction), creating a life-threatening hemorrhagic situation/multiple emboli.</p>
<p>Note drug effects, and monitor for signs of toxicity.</p>	<p>Massive doses of antibiotics are often ordered. These have potentially toxic effects when hepatic/renal perfusion is compromised.</p>
<p>Collaborative</p>	
<p>Administer parenteral fluids. (Refer to ND: Fluid Volume, risk for deficient, following.)</p>	<p>To maintain tissue perfusion, large amounts of fluid may be required to support circulating volume.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Shock Prevention (NIC)</p> <p>Collaborative</p> <p>Administer drugs as indicated: Corticosteroids;</p> <p>NaHCO₃;</p> <p>Antacids, e.g., aluminum hydroxide (Amphojel).</p> <p>Monitor laboratory studies, e.g., ABGs, lactate levels.</p> <p>Administer supplemental O₂.</p> <p>Maintain body temperature, using adjunctive aids as necessary. (Refer to ND: Hyperthermia)</p> <p>Assist with measurement of partial pressure of CO₂ in the gastric mucosa (PrCO₂) as indicated.</p> <p>Prepare for/transfer to critical care setting as indicated.</p>	<p>Although steroid therapy remains controversial, steroids may be given for the potential advantages of decreased capillary permeability, increased renal perfusion, and inhibition of microemboli formation.</p> <p>Impaired tissue perfusion and production of lactate result in metabolic acidosis, requiring base of replacement therapy.</p> <p>Decreases potential for gastric bleeding related to stress response/altered perfusion.</p> <p>Development of respiratory/metabolic acidosis reflects loss of compensatory mechanisms, e.g., decreased renal perfusion/hydrogen excretion, and accumulation of lactic acid due to circulatory shunting and stagnation.</p> <p>Maximizes oxygen available for cellular uptake.</p> <p>Temperature elevations increase metabolic/oxygen demands beyond cellular resources, hastening tissue ischemia/cellular destruction.</p> <p>Gastric tonometry may be useful in diagnosing hypoperfusion of the gut before ischemic injury occurs, thus reducing risk of developing MODS.</p> <p>Progressive deterioration requires more aggressive therapy (e.g., hemodynamic monitoring and vasoactive drugs).</p>

<p>NURSING DIAGNOSIS: Fluid Volume, risk for deficient</p> <p>Risk factors may include</p> <p>Marked increase in vascular compartment/massive vasodilation Capillary permeability/fluid leaks into the interstitial space (third spacing)</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>Hydration (NOC)</p> <p>Maintain adequate circulatory volume as evidenced by vital signs within patient’s normal range, palpable peripheral pulses of good quality, and individually appropriate urinary output.</p>
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ACTIONS/INTERVENTIONS	RATIONALE
<p>Shock Prevention (NIC)</p> <p>Independent</p> <p>Measure/record urinary output and specific gravity. Note cumulative intake and output (I&O) imbalances (including insensible losses), and correlate with daily weight. Encourage oral fluids to tolerance.</p> <p>Monitor BP and heart rate (HR). Measure central venous pressure (CVP) is used.</p> <p>Palpate peripheral pulses.</p> <p>Assess for dry mucous membranes, poor skin turgor, and thirst.</p> <p>Observe for dependent/peripheral edema in sacrum, scrotum, back, legs.</p>	<p>Decreasing urinary output with a high specific gravity suggests relative hypovolemia associated with vasodilation. Continued positive fluid balance with corresponding weight gain may indicate third spacing and tissue edema, suggesting need to alter fluid therapy/replacement components. <i>Note:</i> Excessive diarrhea may lead to a negative fluid balance.</p> <p>Reduction in the circulating fluid volume reduces BP/CVP, initiating compensatory mechanisms of tachycardia to improve cardiac output and increase systemic BP.</p> <p>Weak, easily obliterated pulses suggest hypovolemia.</p> <p>Hypovolemia/third spacing of fluid gives rise to signs of dehydration.</p> <p>Fluid losses from the vascular compartment into the interstitial space create tissue edema.</p>
<p>Collaborative</p> <p>Administer IV fluids, e.g., crystalloids (D₅W, normal saline[NS]) and colloids (albumin, fresh frozen plasma) as indicated.</p> <p>Monitor laboratory values, e.g.:</p> <p style="padding-left: 40px;">Hct/RBC count;</p> <p style="padding-left: 40px;">BUN/Cr.</p> <p>Monitor cardiac output as indicated.</p>	<p>Large volumes of fluid may be required to overcome relative hypovolemia (peripheral vasodilation), replace losses from increased capillary permeability (e.g., sequestration of fluid in the peritoneal cavity) and increased insensible sources (e.g., fever/diaphoresis).</p> <p>Evaluates changes in hydration/blood viscosity.</p> <p>Moderate elevations of BUN reflect dehydration, high values of BUN/Cr may indicate renal dysfunction/failure.</p> <p>CO (and other functional parameters, such as cardiac index, preload/afterload, contractility, and cardiac work) can be measured noninvasively using thoracic electrical bioimpedance (TEB) technique. Useful in determining therapeutic needs/effectiveness.</p>

NURSING DIAGNOSIS: Gas Exchange, risk for impaired

Risk factors may include

Altered O₂ supply: effects of endotoxins on the respiratory center in the medulla (resulting in hyperventilation/respiratory alkalosis); hypoventilation

Altered blood flow (changes in vascular resistance), alveolar-capillary membrane changes (increased capillary permeability leading to pulmonary congestion)

Interference with O₂ delivery/utilization in the tissues (endotoxin-induced damage to the cells/capillaries)

Possibly evidenced by

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

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Respiratory Status: Gas Exchange (NOC)

Display ABGs and respiratory rate within patient's normal range, with breath sounds clear and chest x-ray clear/improving.

Experience no dyspnea/cyanosis.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Respiratory Monitoring (NIC)</p> <p>Independent</p> <p>Maintain patient airway. Place patient in position of comfort with head of bed elevated.</p> <p>Monitor respiratory rate and depth. Note use of accessory muscles/work of breathing.</p> <p>Auscultate breath sounds. Note crackles, wheezes, areas of decreased/absent ventilation.</p> <p>Note presence of circumoral cyanosis.</p> <p>Investigate alterations in sensorium: agitation, confusion, personality changes, delirium, stupor, coma.</p> <p>Note cough and purulent sputum production.</p> <p>Reposition frequently. Encourage coughing and deep-breathing exercises. Suction as indicated.</p>	<p>Enhances lung expansion, respiratory effort.</p> <p>Rapid/shallow respirations occur because of hypoxemia, stress, and circulating endotoxins. Hypoventilation and dyspnea reflect ineffective compensatory mechanisms and are an indication that ventilatory support is needed.</p> <p>Respiratory distress and the presence of adventitious sounds are indicators of pulmonary congestion/interstitial edema, atelectasis. <i>Note:</i> Respiratory complications, including pneumonia and adult respiratory distress syndrome (ARDS), are a prime cause of death.</p> <p>Reflects inadequate systemic oxygenation/hypoxemia.</p> <p>Cerebral function is very sensitive to decreases in oxygenation (e.g., hypoxemia, reduced perfusion).</p> <p>Pneumonia is a common nosocomial infection that can occur by aspiration of oropharyngeal organisms or spread from other sites.</p> <p>Good pulmonary toilet is necessary for reducing ventilation/perfusion imbalance and for mobilizing and facilitating removal of secretions to maximize gas exchange.</p>

ACTIONS/INTERVENTIONS	RATIONALE
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ACTIONS/INTERVENTIONS	RATIONALE
<p>Teaching: Disease Process (NIC)</p> <p>Independent</p>	
Review disease process and future expectations.	Provides knowledge base from which patient can make informed choices.
Review individual risk factors and mode of transmission/portal of entry of infections.	Steroid therapy, kidney/liver dysfunction, neoplastic disease, rheumatic heart disease, valve dysfunction, and diabetes may predispose to septicemia. Awareness of means of infection transmission provides opportunity to plan for/institute protective measures.
Provide information about drug therapy, interactions, side effects, and importance of adherence to regimen.	Promotes understanding of and enhances cooperation in treatment/prophylaxis, and reduces risk of recurrence and complications.
Discuss need for good nutritional intake/balanced diet.	Necessary for optimal healing and general well-being.
Encourage adequate rest periods with scheduled activities.	Prevents fatigue, conserves energy, and promotes healing.
Review necessity of personal hygiene and environmental cleanliness, proper cooking techniques/food storage.	Helps control environmental exposure by diminishing the number of pathogens present. <i>Note:</i> Undercooked meat increases risk of exposure to <i>E. coli</i> .
Discuss proper use or avoidance of tampons with women, as indicated.	Superabsorbent tampons/infrequent changing potentiates risk of <i>Staphylococcus aureus</i> infection (toxic shock syndrome).
Identify signs/symptoms requiring medical evaluation, e.g., persistent temperature elevation(s), tachycardia, syncope, rashes of unknown origin, unexplained fatigue, anorexia, increased thirst, and changes in bladder function.	Early recognition of developing/recurring infection allows for timely intervention and reduces risk for progression to life-threatening situation.
Stress importance of prophylactic immunization/antibiotic therapy as needed.	Used for prevention of infection dependent on individual risk factors, e.g., age, presence of chronic disease, immunosuppression.

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

Infection, risk for recurrence/opportunistic—stasis of body fluids, decreased hemoglobin, leukopenia, suppressed inflammatory response, use of anti-infective agents, increased environmental exposure, malnutrition.

Nutrition: imbalanced, less than body requirements—increased energy needs (hypermetabolic state), anorexia, continuing gastrointestinal (GI) dysfunction, side effects of medication.

Self-Care deficit/Home Maintenance, impaired—decreased strength/endurance, pain/discomfort, inadequate support systems, unfamiliarity with neighborhood resources.