Recognizing the signposts for sepsis

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Sepsis is the number one cause of death in the ICU, the 10th leading cause of death worldwide, and the 11th leading cause of death in the United States. Older patients with chronic illnesses are at high risk for developing this condition. We discuss what you need to know about sepsis and your role in caring for a patient who has it.

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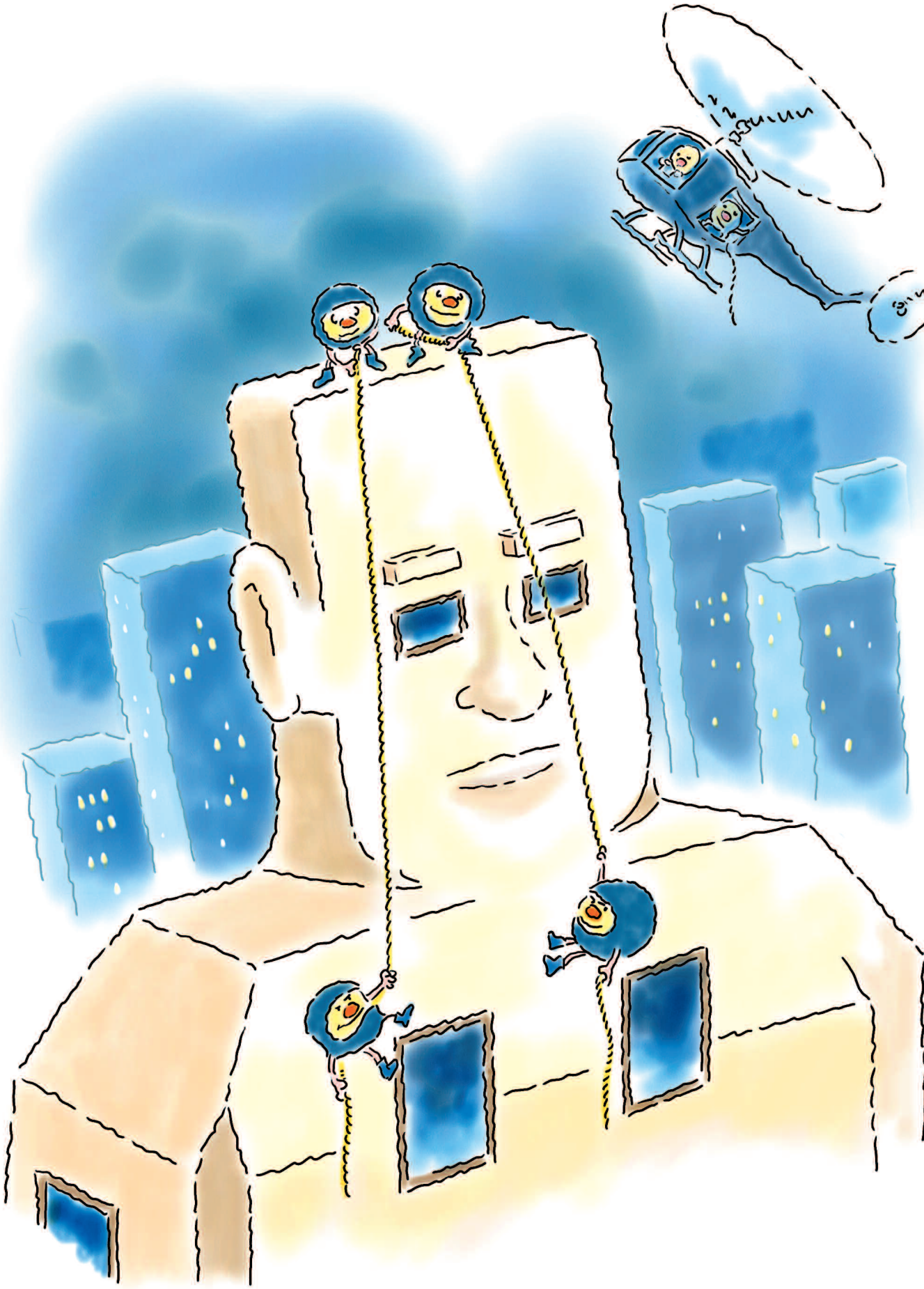
The author has disclosed that she has no significant relationships with or financial interest in any commercial companies that pertain to this educational activity.

Today is the fourth admission in a month for Mr. Hammill, 70, who has a recent medical history of coronary artery bypass graft surgery and atrial fibrillation and also a history of coronary artery disease, chronic obstructive pulmonary disease, diabetes, gastroesophageal reflux disease, anemia, and anxiety. Well known to you as an interactive and communicative man, you find that during this admission he's irritable and less talkative. You also note that he has generalized edema, coarse rhonchi in the anterior lobes, and crackles in the bases of both lungs, and he's coughing up tan-colored sputum. This morning, he's confused upon awakening, and throughout the course of the day you need to reorient him to his surroundings.

His ability to answer questions correctly earlier in your shift suddenly declines. He's tachypneic with a respiratory rate of 32 breaths/minute, an irregular pulse, and a heart rate of 110 beats/minute. His serum lactate level is 12 mmol/L. What's going on with Mr. Hammill? The healthcare team sus-



pects sepsis on the basis of his age and clinical findings.

To start, let's review what sepsis is and how it evolves.

Sepsis = serious

A systemic inflammatory response to the presence of infection (such as Gram-positive or Gram-negative bacteria, fungi, viruses, mycobacteria, or parasites) that can progress to circulatory systemic dysfunction, multiple organ failure, and death, sepsis is a complex disease process that carries a high degree of morbidity and mortality.

Older patients are at increased risk for sepsis and it may be more difficult to diagnose clinically in this age group. Babies and immunocompromised patients are also at increased risk.

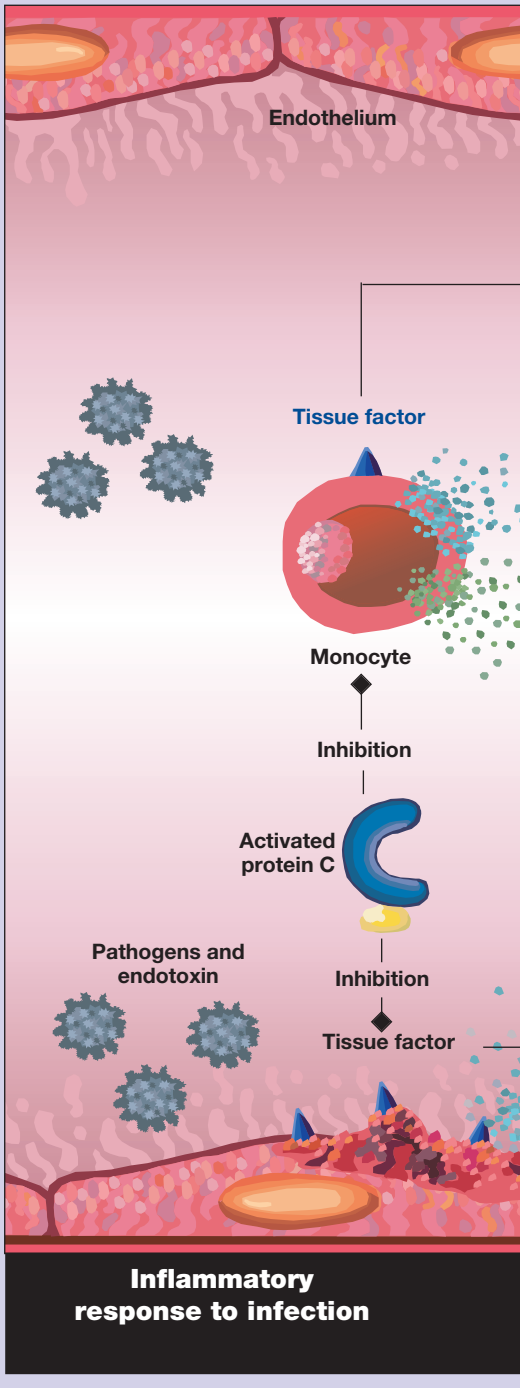
The incidence of sepsis is reported as three cases per 1,000 people; in hospitalized patients, the incidence is 2%. The rise in sepsis cases is believed to be related to the growing number of immunocompromised patients, a greater number of invasive procedures being performed in the ICU, an increased number of resistant organisms, and a rise in the number of older patients with critical illnesses.

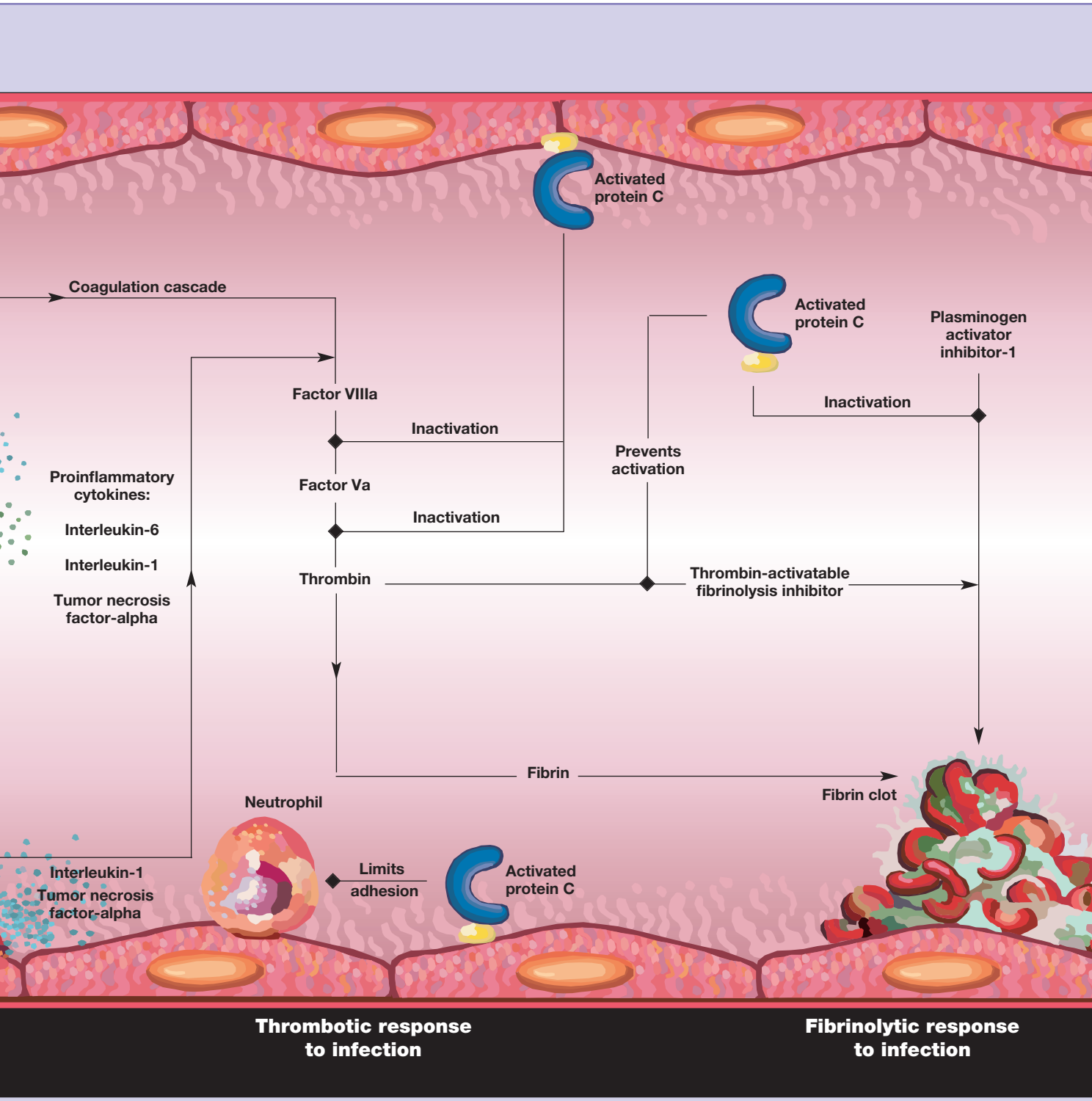
What are the events that can lead to sepsis? Let's find out.

A complex cascade

Inflammation is the body's response to insults that arise from chemical, traumatic, or infectious stimuli. The inflammatory cascade is a complex process that involves humoral and cellular responses and complement and cytokine cascades (see *A complex cascade*). What does this mean? Following an insult to the body, local cytokines are produced and released into the circulation to promote a local response. Growth factor stimulation and recruitment of macrophages and platelets occur. The goal is homeostasis; however, if homeostasis doesn't occur from the local response to inflammation, the result is a sys-

A complex cascade




Activated protein C
Coagulation cascade
Activated protein C
Plasminogen activator inhibitor-1
Factor VIIIa
Inactivation
Inactivation
Prevents activation
Proinflammatory cytokines:
Interleukin-6
Interleukin-1
Tumor necrosis factor-alpha
Factor Va
Inactivation
Thrombin
Thrombin-activatable fibrinolysis inhibitor
Fibrin
Fibrin clot
Neutrophil
Limits adhesion
Activated protein C
Interleukin-1
Tumor necrosis factor-alpha
Thrombotic response to infection
Fibrinolytic response to infection

temic inflammatory reaction.

The most prominent systemic manifestation of inflammation is known as the acute phase response. This constellation of systemic effects usually begins within hours or days of the onset of inflammation. The release of cytokines, such as interleukin (IL)-1, IL-6, and tumor necrosis factor (TNF), causes the thermoregulatory center in the hypothalamus to produce an elevation in the patient's body temperature, resulting in a fever. During the acute phase response, the bone marrow produces more immature neutrophils and the liver increases fibrinogen and C-reactive protein production. Skeletal muscle catabolism provides amino acids that can be used in the repair of tissue. IL-8 may perpetuate tissue inflammation. IL-6 and IL-10 augment the acute phase response by generating additional proinflammatory mediators.

The unregulated release of proinflammatory mediators or cytokines can elicit toxic reactions and promote endothelial cell-leukocyte adhesion, releasing cell-damaging proteases and prostaglandins, which participate in the generation of fever, tachycardia, ventilation/perfusion abnormalities, and lactic acidosis, and activating the clotting cascade. Endotoxins cause tissue damage by releasing prostaglandins, and the resulting vasodilation and increased capillary permeability allow fluid to enter the interstitial space, causing edema and hypotension. The lack of oxygen at the cellular level causes the respiratory system to fail, followed by renal, gastrointestinal (GI), and liver failure. Platelet aggregation and thrombi form because of the increased viscosity of the blood. This can result in decreased tissue perfusion and the development of disseminated intravascular coagulation (DIC), or idiosyncratic bleeding and clotting. This can ultimately lead to multiple organ damage and dysfunction.

Important definitions

cheat sheet

Bacteremia: the presence of bacteria in the blood.

Infection: the presence of microorganisms that trigger an inflammatory response.

Hypotension: a systolic BP of less than 90 mm Hg or a drop in systolic BP of greater than 40 mm Hg from the patient's baseline BP.

Sepsis: a systemic response to infection; may occur after a burn, surgery, or serious illness and is manifested by two or more clinical signs and symptoms:

- temperature greater than 100.4° F (38° C) or less than 96.8° F (36° C)
- heart rate of greater than 90 beats/minute
- respiratory rate of greater than 20 breaths/minute
- partial pressure of carbon dioxide level of less than 32 mm Hg
- WBC count greater than 12,000 cells/mm^3, less than 4,000 cells/mm^3, or greater than 10% immature WBC (bands)
- hyperglycemia and abnormal clotting and bleeding.

Severe sepsis: the presence of signs and symptoms of sepsis-related organ dysfunction, hypotension, or hypoperfusion; clinical signs and symptoms include those of sepsis as well as:

- lactic acidosis
- oliguria
- thrombocytopenia
- altered level of consciousness.

Septic shock: shock associated with sepsis; characterized by symptoms of sepsis plus hypotension and hypoperfusion despite adequate fluid volume replacement.

SIRS: a syndrome resulting from a severe clinical insult that initiates an overwhelming inflammatory response by the body.

MODS: the presence of altered function of one or more organs in an acutely ill patient requiring intervention and support of the organs to achieve physiologic functioning required for homeostasis.

Pinning down the culprit

Sepsis can occur in people of all ages, but it's more prevalent in older patients. In one nationwide study, it was found that patients older than age 65 accounted for 64.9% of all sepsis cases. A possible reason for this high incidence is the fact that older patients are more likely to have Gram-negative infections, particularly associated with pneumonia, and comorbid conditions. Possible causes of sepsis include:

- pneumonia
- urinary tract infection (UTI)
- diarrhea or distension
- meningitis
- cellulitis

- septic arthritis
- wound infection
- endocarditis
- catheter-related infection.

Sepsis may start with systemic inflammatory response syndrome (SIRS). The diagnosis of SIRS requires two or more of the following clinical findings:

- body temperature of greater than 100.4° F (38° C) or less than 96.8° F (36° C)
- heart rate of greater than 90 beats/minute
- respiratory rate of greater than 20 breaths/minute or a partial pressure of carbon dioxide measurement of less than 32 mm Hg
- white blood cell (WBC) count of greater than 12,000/mm^3 or less than 4,000/mm^3 or greater than 10% immature forms of neutrophils or bands.

A patient with systemic manifestations of infection plus a documented infection has ***sepsis.*** A patient with sepsis complicated by organ dysfunction, tissue hypoperfusion (an elevated serum lactate level or oliguria), or sepsis-induced hypotension (a systolic BP of less than 90 mm Hg or a mean arterial pressure [MAP] of less than 70 mm Hg, or a decrease in systolic BP of greater than 40 mm Hg below normal for the patient's age in the absence of other causes) has ***severe sepsis***. A patient with sepsis-induced hypotension that persists despite adequate fluid resuscitation and isn't explained by other causes has ***septic shock***.

Signs of acute organ system failure

Cardiovascular

- Tachycardia
- Arrhythmias
- Hypotension
- Elevated central venous and pulmonary artery pressures

Respiratory

- Tachypnea
- Hypoxemia

Renal

- Oliguria
- Anuria
- Elevated creatinine

Hematologic

- Jaundice
- Elevated liver enzymes
- Decreased albumin
- Coagulopathy

GI

- Ileus (absent bowel sounds)

Hepatic

- Thrombocytopenia
- Coagulopathy
- Decreased protein C levels
- Increased D-dimer levels

Neurologic

- Altered consciousness
- Confusion
- Psychosis

Complication station

Complications associated with sepsis include acute respiratory distress syndrome (ARDS), acute renal failure, GI complications, DIC, and multiple organ dysfunction syndrome (MODS). Let's take a closer look.

ARDS is defined as the abrupt onset of respiratory distress accompanied by three components: severe hypoxemia, bilateral pulmonary infiltrates seen on X-ray, and the absence of heart failure or fluid overload. Hypoxemia may be present before the onset of other clinical signs. Tachypnea and tachycardia are seen during the first 12 to 24 hours, followed by a dramatic increase in the work of breathing. The three phases of ARDS are the acute exudative phase, which is characterized by profound hypoxemia and associated with inflammation and diffuse alveolar damage; the fibroproliferative phase, which is associated with decreased compliance and increased dead space; and the resolution phase, which may take 6 to 12 months or longer to resolve.

Acute renal failure may develop as a result of endotoxins, which are powerful vasoconstrictors that can cause intravascular

Common medications used to treat sepsis

Classification of drug	Indications	Nursing care
Isotonic crytalloids		
0.9% sodium chloride solution	Used for fluid resuscitation	Monitor cardiac, renal, and pulmonary function; watch for edema in the extremities and signs of changes in mental status
Lactated Ringer's solution	Used for fluid resuscitation	Monitor hemodynamic response
Colloids		
Albumin 5%	Used for volume expansion	May exacerbate renal insufficiency
Antibiotics		
Cefotamine	Used for Gram-negative coverage against *Escherichia coli and Proteus, Klebsiella*, and *Pseudomonas* species	Adjust dose in severe renal failure
Ceftriaxone	Used because of increased prevalence of penicillinase-producing microorganisms	Adjust in renal impairment; use caution if the patient is allergic to penicillin
Cefuroxime	Used for Gram-positive coverage against *E. coli*, *Klebsiella peneumoniae, Proteus mirabilis*, and *Haemophilus influenzae*	Monitor renal function; administer a half dose if creatinine clearance is 10 to 30 mL/minute
Ticarcillin	Used as an antipseudomonal penicillin and beta-lactamase inhibitor	Monitor renal function and adjust dosage in patients with severe colitis
Clindamycin	Used against anaerobes (may have some activity against streptococcus and methicillin-sensitive *S. aureus*)	Monitor PT, digoxin, and theophylline levels
Metronidazole or ciprofloxacin	Used against Gram-positive and aerobic Gram-negative organisms in GI infections and infectious diarrhea	Metronidazole can potentiate warfarin; ciprofloxacin may increase digoxin and theophylline levels
Activated protein C analogues		
Drotrecogin alfa	Used for severe sepsis with acute organ dysfunction	Monitor for hypersensitivity; contraindicated if the patient is at increased risk for bleeding, has had a recent stroke, or has head trauma, has an epidural catheter, or is on heparin therapy
Vasopressors		
Dopamine	Used to treat hypotension in fluid resuscitated patients; stimulates adrenergic and dopaminergic receptors	Hemodynamic effects are dose related; monitor urine output, cardiac output, pulmonary wedge pressure, and BP.
Norepinephrine	Used to treat hypotension following fluid volume replacement; stimulates $beta_1$-adrenergic and alpha-adrenergic receptors and increases cardiac contractility	Use in caution in patients with occlusive vascular disease; correct blood volume depletion before administration; extravasation may result in severe tissue necrosis
Vasopressin	Increases vasomotor tone in patients with septic shock; also increases water reabsorption at the distal renal epithelium and promotes smooth muscle contraction throughout the vascular bed of the renal tubular epithelium	Dosage is one-tenth of what's used to treat an upper GI bleed from esophageal varices; monitor BP and urine output

clotting. The degree of renal damage is relative to the severity and duration of the shock. Acute tubular necrosis may occur because of severe ischemia to the kidneys. With careful monitoring of urine output and serum creatinine and blood urea nitrogen levels, acute renal failure is reversible.

GI complications can develop when there's a redistribution of blood flow to the mucosal layer of the GI tract. Superficial lesions can cause stress ulcers in the stomach. Bleeding is a common symptom, and hemorrhage can occur 2 to 10 days after the insult.

DIC is caused by activation of the coagulation cascade, resulting in the formation of fibrin clots and thrombotic occlusion of small and midsized vessels. The delayed removal of fibrin occurs because of impaired fibrinolysis. Depletion of platelets and coagulation factors increase the risk of bleeding. Fibrin deposits in organs can cause ischemic damage and organ failure.

MODS occurs when multiple organs, such as the kidneys, liver, lungs, brain, and heart, are damaged as a consequence of septic shock. The mortality rate increases with the number of failing organs. See *Signs of acute organ system failure* for more information.

Lab tests at the ready

Early detection of sepsis is critical so that appropriate intervention can be implemented. Aggressive treatment protocols have been shown to decrease mortality rates by 30% for severely septic patients and by 50% for patients who haven't yet developed the disease.

Lab tests to diagnose sepsis include:

• ***metabolic studies, including evaluation of serum electrolyte levels.*** Often patients with sepsis have hypocalcemia, hyper- or hypoglycemia, an elevated blood urea nitrogen level, and mild hyperbilirubinemia.

• ***complete blood cell (CBC) count with differential and platelet count.*** An adequate hemoglobin level (7 to 9 g/dL for adults) is necessary to ensure oxygen delivery in patients with septic shock. Platelets are acute phase reactants and usually rise at the onset of any serious stress. The platelet count will fall with persistent sepsis. An elevated WBC count may predict bacterial infection. The patient may have leukocytosis, leukopenia, azotemia (an accumulation of nitrogenous waste products in the blood), thrombocytopenia, anemia, or hypoxemia.

• ***coagulation studies.*** Assess prothrombin time (PT) and partial thromboplastin time. Patients with sepsis often have a prolonged PT time. Patients with clinical evidence of coagulopathy require additional tests to detect the presence of DIC.

• ***arterial blood gas (ABG) analysis.*** Measure serum lactate levels to assess tissue perfusion. An elevated serum lactate level (above 4 mmol/L) indicates significant tissue hypoperfusion and a shift from aerobic to anaerobic metabolism. In severe cases, the patient may have lactic acidosis.

• ***cultures of sputum, urine, cerebrospinal fluid, wound drainage, or respiratory secretions.*** Tissue Gram staining from the site of the possible infection can provide guidance in the choice of antibiotic therapy. A sputum culture can determine the presence of pneumonia; a urine culture can determine the presence of UTI.

How's sepsis treated? That's up next.

Bundle up

According to the Surviving Sepsis Campaign's guidelines for the management of severe sepsis and septic shock, using a sepsis bundle can reduce mortality in severe cases. A bundle is defined as a group of interventions related to a disease process that, when implemented together, result in better outcomes than when implemented individually. Treatment for sepsis includes the following six interventions:

• ***give 100% oxygen via non-rebreather mask.*** Because the metabolic demands for oxygen are massively increased in sepsis, the need for intubation and mechanical ventilation may be required if ABG levels dete-

riorate and blood pH decreases.

• ***obtain two separate blood cultures before antibiotic therapy is initiated.*** At least two blood cultures should be drawn before antibiotic therapy initiation, with at least one drawn percutaneously and one drawn through each vascular access device, unless the device was recently inserted (less than 48 hours). Source control is also an important strategy for certain types of sepsis patients. Recent studies done on patients with sepsis show that removing any infected device and debriding necrotic or infectious tissue, especially in skin and soft tissue infections, are recommended. Drainage and debridement may also be appropriate for patients with empyema (the collection of pus within an anatomic cavity), sinusitis, and infections of the chest and mediastinum.

• ***initiate antibiotic therapy.*** Because sepsis is caused by an infectious disease, one of the cornerstones of patient management is antibiotic therapy. The appropriateness and timing of antibiotic coverage is important. Initially, a broad-spectrum antibiotic should be used, with emphasis on the most likely pathogens, but should be discontinued within 3 to 5 days. Antibiotic therapy is modified after cultures are available and antibiotic susceptibility patterns are known. Single antibiotic therapy may last for 7 to 10 days and in certain cases may be longer, such as in patients with a slow response, those who are immunologically deficient, or those with an area of infection that's undrainable. Dosage adjustments may be based on renal function.

• ***initiate fluid resuscitation.*** Another cornerstone of treatment is the infusion of I.V. fluid to restore the circulating volume lost as fluid leaks from capillaries. Crystalloid solutions, such as 0.9% sodium chloride or lactated Ringer's solution, or colloids, such as albumin, will help to keep the MAP above 65 mm Hg. Wedge pressure should be maintained at 6 to 12 mm Hg; central venous pressure, 8 to 12 mm Hg; and systemic vascular resistance at 800 to 1,200 dynes/sec/cm-^{5} The typical amount of crystalloid solution for both severe sepsis and septic shock ranges from 4 to 8 liters. Fluid challenges with 300 to 500 mL of crystalloids or colloids may be given based on BP and urine output. This extra fluid will help blood flow to the organs. Careful monitoring is necessary to prevent overload in a patient with known heart failure.

• ***measure the patient's lactate and hemoglobin-A lactate levels.*** Septic shock is diagnosed when the lactate level is greater than 4 mmol/L in the presence of severe sepsis. A low hemoglobin value means that the amount of oxygen carried to the organs and tissue is reduced. If the hemoglobin level is less than 7 g/dL, a blood transfusion should be considered.

• ***insert a urinary catheter to monitor hourly urine output.*** Urine output is a good indicator of how well the patient's kidneys are being perfused. If the amount of urine drained by the catheter is low, it could mean that circulation to the body is impaired and fluid challenges are needed to restore perfusion.

Now let's delve deeper into antibiotic therapy and other medications used to treat sepsis (see *Common medications used to treat sepsis*).

Meds up to bat

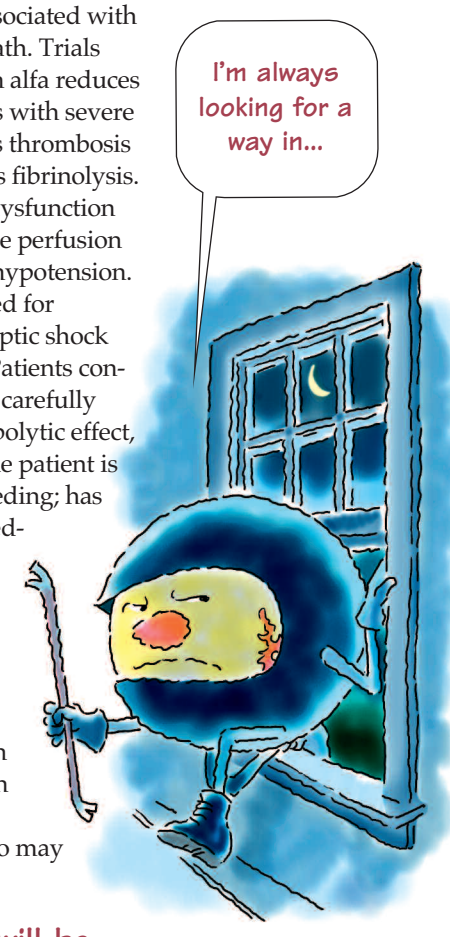
According to the guidelines, ***antibiotic therapy*** should be started within the first hour, if possible. A recent study examined the timing of antibiotic therapy and found that every additional hour without effective antibiotic therapy can increase the risk of death in patients with hypotensive sepsis by 7.6% during the first 6 hours. The choice of antibiotic prescribed by the healthcare provider depends on complex issues related to the patient's history, drug intolerances, underlying diseases, clinical syndromes, and susceptibility patterns of pathogens in the community and hospital. Clinicians should be cognizant of the virulence and

growing prevalence of methicillin-resistant *Staphylococcus aureus* infection and the possibility of candidemia when choosing appropriate antibiotic therapy. It's recommended that patients with severe sepsis receive broad-spectrum antibiotic therapy until the causative organism and antibiotic susceptibilities are found. Although appropriate cultures need to be obtained before starting antibiotics, it shouldn't delay therapy. Premixed antibiotics or bolus antibiotics may facilitate prompt administration.

Vasopressors, such as dopamine and norepinephrine, are used to restore tissue perfusion pressure. Dopamine increases myocardial contractility and is more likely to increase cardiac output; however, it may increase the patient's heart rate and produce tachyarrhythmias. Norepinephrine is a more potent vasoconstrictor and has been found to be more effective than dopamine in restoring hemodynamic stability. It's considered to be a first-line choice of drug therapy for patients with sepsis. The use of vasopressin was recently studied in the Vasopressin and Septic Shock Trial. During this study, it was found that patients requiring vasopressors (norepinephrine or vasopressin) for at least 6 hours who had one dysfunctional organ system had no difference in 28-day survival.

The use of ***corticosteroids*** may be helpful to decrease complement activity, platelet activation, TNF, and proinflammatory cytokines. Guidelines state that the use of corticosteroids is indicated for adult patients with septic shock when hypotension remains poorly responsive to adequate fluid resuscitation and vasopressors.

Drotrecogin alfa was approved for use by the FDA in 2001 for the treatment of severe sepsis. This recombinant form of human activated protein C is the first of a new category of medications that act on the body's response to infection at the level of the blood vessel. The inflammatory cascade that occurs during severe sepsis causes activation of the coagulation system associated with impaired fibrinolysis. This can result in changes in the microvasculature circulation associated with multiorgan dysfunction and death. Trials show that the use of drotrecogin alfa reduces the risk of death among patients with severe sepsis by 20% because it inhibits thrombosis and inflammation and promotes fibrinolysis. It also prevents microvascular dysfunction and coagulation, improves tissue perfusion and oxygenation, and reverses hypotension. Drotrecogin alfa is recommended for patients with severe sepsis or septic shock who are at high risk for death. Patients considered for treatment should be carefully evaluated. Due to its antithrombolytic effect, this drug is contraindicated if the patient is experiencing active internal bleeding; has had recent head trauma, GI bleeding, or surgery; or has an epidural catheter in place. And it shouldn't be given 2 hours before an invasive or surgical procedure. Recently, the FDA has issued a label warning about administering drotrecogin alfa to patients with single organ dysfunction who have recently undergone surgery or those who may not be at high risk for death.



The role of the nurse will be played by...

What should you do when caring for a patient with sepsis?

Nursing interventions include:

- ***infection control measures.*** It's critical that you strictly follow infection control measures, including practicing proper hand hygiene as outlined by the CDC.
- ***assessment and monitoring.*** Assess and document the patient's vital signs at least every hour. Monitor body temperature, respiratory rate, BP, MAP and central venous pressure, heart rate, urine output, and oxygen saturation values. Lab monitoring of a patient's blood glucose level is recommended every 30 to 60 minutes because tight glycemic control (under 150 mg/dL) has been shown to improve out-

comes. Frequently perform neurologic checks to assess any change in mental status. Monitor for coagulation abnormalities. If the patient starts oozing or bleeding from three separate sites (such as the nose, a wound, and the I.V. site) or has petechiae or cyanosis, he may be developing DIC. Also be alert for the presence of cold, mottled, or cyanotic extremities because a microvascular occlusion forms clots to distal tissues. Report these findings to the healthcare provider.

- ***proper documentation.*** Documentation should include the following: the type of respiratory support and results of ABG analysis, the type and location of I.V. access, and the time and amount of I.V. crystalloids, colloids, antibiotics, or vasopressors given. Report and document results of lab tests such as the CBC count; electrolyte, blood glucose, and lactate levels; and coagulation studies.
- ***communication with the patient's family.*** Families of patients with sepsis may feel helpless during this critical time. They need frequent explanations of interventions and procedures, as well as compassionate care. Communication about end-of-life issues and life-sustaining measures ahead of time allows the family to understand what's happening to their loved one and make informed choices and decisions.

On the road to recovery

So what's happening with our patient, Mr. Hammill?

Mr. Hammill's condition is medically complex and he's critically ill. His WBC count is 25,000/mm^3 and sputum cultures identify the source of sepsis as pneumonia. Mr. Hammill is placed on a broad-spectrum antibiotic. Over the course of the day, he becomes more confused and then obtunded. His oxygen saturation value falls to 85% and he becomes acutely hypotensive. He's resuscitated and placed on mechanical ventilation. Despite the administration of I.V. fluids, he remains hypotensive. He's started on vasopressors in an attempt to maintain a MAP of greater than 65 mm Hg. His urine output from the indwelling catheter drops to 10 mL/hr. He's given additional I.V. colloids and a total of 200 mL of albumin. Later that night, his condition begins to improve.

Signposts ahead

Sepsis is a significant cause of morbidity and mortality in older patients. The diagnosis of sepsis can be particularly difficult in this age group, and a high index of suspicion is necessary for early identification and intervention. Deterioration in the clinical condition of an older patient may be subtle and easily missed, so each encounter may provide clinical clues that can alert you to changes in physiologic status, which may be an early manifestation of sepsis. By understanding the signposts for sepsis, you'll be able to recognize and rapidly identify a patient developing this condition so that immediate and appropriate management can be implemented. ■

Learn more about it

Dellinger RP, Levy MM, Carlet JM, et al. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock: 2008. *Crit Care Med*. 2008;36 (1):296-327.

Dombrovskiy V, Martin A, Suderram J, Paz H. Use of drotrecogin alpha (activated) for severe sepsis in New Jersey acute care hospitals. *Am J Health Syst Pharm*. 2006;63(12):1151-1156.

Domino FJ. *The 5 Minute Clinical Consult.* 17th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008.

Kumar A, Roberts D, Wood KE, et al. Duration of hypotension before initiation of effective antimicrobial therapy is the critical determinant of survival in human septic shock. *Crit Care Med.* 2006;34(6):1589-1596.

Martin GS. SAFE, VASST, LIPOS Trial 3, CORTICUS and more: implications for the Surviving Sepsis Campaign guidelines. http://www.medscape.com/viewarticle/555167.

Picard KM, O'Donoghue SC, Young-Kershaw DA, Russell KJ. Development and implementation of a multidisciplinary sepsis protocol. *Crit Care Nurse*. 2006;26(3):43-54.

Porth CM. *Essentials of Pathophysiology: Concepts of Altered Health States*. Philadelphia, PA: Lippincott Williams & Wilkins; 2004:334-335.

Porth CM. *Pathophysiology: Concepts of Altered Health States*. 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006:387-401.

Robson WP, Daniel R. The sepsis six: helping patients to survive sepsis. *Br J Nurs.* 2008;17(1):16-21.

Sakr Y, Reinhart K, Vincent JL, et al. Does dopamine administration in shock influence outcome? Results of the Sepsis Occurrence in Acutely Ill Patients (SOAP) study. *Crit Care Med.* 2006;34(3):589-597.

Sharma S, Eschun G. Multisystem organ failure of sepsis. http://www.emedicine.com/MED/topic3372.htm.

Smeltzer SC, Bare BG, Hinkle JL, Cheever KH. *Brunner and Suddarth's Textbook of Medical-Surgical Nursing.* 11th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2007:373.

Strategies for Managing Multisystem Disorders. Philadelphia, PA: Lippincott Williams & Wilkins; 2005:326-329.

On the Web

These online resources may be helpful to your patients and their families:

- **eMedicine Health: Sepsis (blood infection):** http://www.emedicinehealth.com/sepsis_blood_infection/article_em.htm
- **Mayo Clinic: Sepsis:** http://www.mayoclinic.com/health/sepsis/DS01004
- **Medline Plus: Sepsis:** http://www.nlm.nih.gov/medlineplus/sepsis.html
- **Sepsis.com:** http://sepsis.com/index.jsp
- **Surviving Sepsis Campaign:** http://www.survivingsepsis.org.

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GENERAL PURPOSE: To provide the professional nurse with an overview of how to recognize and care for a sepsis patient. **LEARNING OBJECTIVES:** After reading this article and taking this test, you should be able to: 1. Describe the pathophysiology, signs and symptoms, complications, and lab tests associated with sepsis. 2. Identify the recommended medical and nursing interventions for managing sepsis.

1. Which statement most accurately describes sepsis?
a. It's a severe infection complicated by tissue hypoperfusion.
b. It's a systemic inflammatory response to the presence of infection.
c. It's an infection-induced hypotensive state that doesn't respond to fluid resuscitation.

2. The inflammatory cascade involves all of the following *except*
a. production and release of local cytokines into the circulation.
b. inhibition of macrophages and platelets.
c. release of tumor necrosis factor.

3. Multisystem failure (respiratory, renal, GI, and liver) in shock is due to
a. lack of oxygen at the cellular level.
b. decreased capillary permeability.
c. intravascular fluid overload.

4. Which *isn't* a clinical sign of systemic inflammatory response syndrome?
a. white blood cell count of 3,800 mm^3
b. temperature of 96.2° F (35.7° C)
c. respiratory rate of 20 breaths/minute

5. Which phrase best defines severe sepsis?
a. sepsis-induced hypotension that persists despite adequate fluid resuscitation
b. systemic manifestations of infection plus a documented infection
c. sepsis with organ dysfunction, tissue hypoperfusion, or sepsis-induced hypotension

6. The phase of acute respiratory distress syndrome associated with decreased compliance and increased dead space is
a. the acute exudative phase.
b. the fibroproliferative phase.
c. the resolution phase.

7. Disseminated intravascular coagulation is associated with
a. depleted platelets and coagulation factors.
b. deactivated coagulation cascade.
c. decreased risk of bleeding.

8. A sign of sepsis-induced acute cardiovascular system failure is
a. bradycardia.
b. hypertension.
c. elevated central venous pressure.

9. Which lab test result is consistent with a diagnosis of sepsis?
a. elevated blood urea nitrogen level
b. hypercalcemia
c. hypobilirubinemia

10. Which blood test is used to assess tissue perfusion in a patient with sepsis?
a. serum lactate levels
b. serum electrolyte levels
c. complete blood cell count

11. Which method of oxygen therapy is best for a patient with sepsis?
a. aerosol mask at 40% oxygen
b. high-flow oxygen mask at 24% oxygen
c. non-rebreather mask at 100% oxygen

12. Antibiotic therapy for suspected sepsis should begin
a. before obtaining blood cultures.
b. immediately after obtaining two separate blood cultures.
c. after blood culture results and sensitivity tests have been received.

13. Which is used for volume expansion during sepsis?
a. vasopressin
b. lactated Ringer's solution
c. albumin 5%

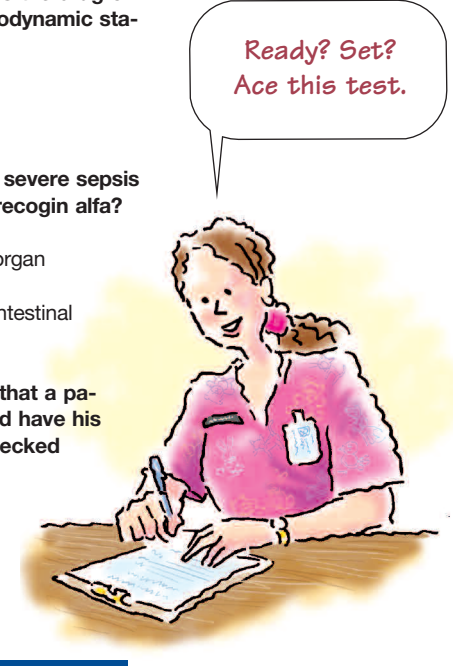
14. In the presence of severe sepsis, septic shock is diagnosed when the patient's lactate level is
a. less than 3 mmol/L.
b. between 3 and 4 mmol/L.
c. greater than 4 mmol/L.

15. Before the causative organism is found, patients with severe sepsis should receive
a. broad-spectrum antibiotic therapy.
b. antibiotic therapy to treat methicillin-resistant *Staphylococcus aureus*.
c. medication to treat candidemia.

16. Which medication is the drug of choice to restore hemodynamic stability?
a. norepinephrine
b. dopamine
c. vasopressin

17. Which patient with severe sepsis *shouldn't* receive drotrecogin alfa?
a. a hypotensive patient
b. a patient with acute organ dysfunction
c. a patient with gastrointestinal bleeding

18. It's recommended that a patient with sepsis should have his blood glucose level checked every
a. 4 to 6 hours.
b. 2 to 3 hours.
c. 30 minutes to 1 hour.



Go to the next page for the CE Enrollment Form.

CE ENROLLMENT FORM *Nursing made Incredibly Easy!* May/June 2009

A. Registration Information:

Last name _____ First name _____ MI ___

Address _____

City _____ State _____ ZIP _____

Telephone _____ Fax _____

E-mail _____

❑ LPN ❑ RN ❑ CNS ❑ NP ❑ CRNA ❑ CNM ❑ other _____

Job title _____ Specialty _____

Type of facility _____ Are you certified? ❑ Yes ❑ No

Certified by _____

State of license (1) _____ License # _____

State of license (2) _____ License # _____

❑ Please fax my certificate to me.

❑ Please check here if you do not wish us to send promotions to your e-mail address.

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Managing heart failure (page 12)

B. Test Answers: Darken one circle for your answer to each question.

	a	b	c		a	b	c		a	b	c		a	b	c		a	b	c
1.	❍	❍	❍	5.	❍	❍	❍	9.	❍	❍	❍	13.	❍	❍	❍	17.	❍	❍	❍
2.	❍	❍	❍	6.	❍	❍	❍	10.	❍	❍	❍	14.	❍	❍	❍	18.	❍	❍	❍
3.	❍	❍	❍	7.	❍	❍	❍	11.	❍	❍	❍	15.	❍	❍	❍	19.	❍	❍	❍
4.	❍	❍	❍	8.	❍	❍	❍	12.	❍	❍	❍	16.	❍	❍	❍				

Registration deadline: June 30, 2011
Contact hours: 2.5
Fee: $24.95
Test code: NMIE0509A

C. Course Evaluation*

1. Did this CE activity's learning objectives relate to its general purpose? ❑ Yes ❑ No
2. Was the journal home study format an effective way to present the material? ❑ Yes ❑ No
3. Was the content relevant to your nursing practice? ❑ Yes ❑ No
4. How long in minutes did it take you to read the article _____, study the material _____, and take the test _____?
5. Suggestion for future topics

_____

Recognizing the signposts for sepsis (page 40)

B. Test Answers: Darken one circle for your answer to each question.

	a	b	c		a	b	c		a	b	c		a	b	c		a	b	c
1.	❍	❍	❍	5.	❍	❍	❍	9.	❍	❍	❍	13.	❍	❍	❍	17.	❍	❍	❍
2.	❍	❍	❍	6.	❍	❍	❍	10.	❍	❍	❍	14.	❍	❍	❍	18.	❍	❍	❍
3.	❍	❍	❍	7.	❍	❍	❍	11.	❍	❍	❍	15.	❍	❍	❍				
4.	❍	❍	❍	8.	❍	❍	❍	12.	❍	❍	❍	16.	❍	❍	❍				

Registration deadline: June 30, 2011
Contact hours: 2.5
Fee: $24.95
Test code: NMIE0509B

C. Course Evaluation*

1. Did this CE activity's learning objectives relate to its general purpose? ❑ Yes ❑ No
2. Was the journal home study format an effective way to present the material? ❑ Yes ❑ No
3. Was the content relevant to your nursing practice? ❑ Yes ❑ No
4. How long in minutes did it take you to read the article _____, study the material _____, and take the test _____?
5. Suggestion for future topics

_____

D. Two Easy Ways to Pay:

❑ Check or money order enclosed (Payable to Lippincott Williams & Wilkins)
❑ Charge my ❑ Mastercard ❑ Visa ❑ American Express

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