Sepsis is a common diagnosis, a frequent cause of hospitalization, and a leading cause of death in the United States (Kochanek, Xu, Murphy, Mininio, & Kung, 2011). Becoming familiar with sepsis is essential for nurses in all settings. The scope of sepsis, including incidence, prevalence, and associated costs, will be reviewed. Sepsis terminology and the increasing severity of sepsis on a continuum will be explained through clinical examples. Causes of sepsis, as well as a brief review of pathophysiology and early treatment recommendations, will be discussed.

Background

Approximately 24% of patients who develop severe sepsis or septic shock will do so on a medical-surgical unit (Sebat et al., 2005). Sepsis was the principal reason for hospitalization in 836,000 hospital stays and the secondary diagnosis in an additional 829,500 hospital stays in 2009 (Elixhauser, Friedman, & Stranges, 2011). Levit, Stranges, Ryan, and Elixhauser (2008) tracked sepsis data from 1997 to 2006, noting that sepsis as a principal diagnosis increased 48%. The rate of hospitalization for sepsis as a primary or secondary diagnosis rose 70% from 2000 to 2008 (Hall, Williams, DeFrances, & Golosinskiy, 2011). Between 1997 and 2008, hospital stays for septicemia increased by 91% (Healthcare Cost and Utilization Project [HCUP], 2010). Sepsis is present, or develops, in approximately 1 of every 23 hospital admissions (Elixhauser et al., 2011). The prevalence of sepsis outlines the enormity of this problem.

Terminology Related to Sepsis

Definitions relevant to the discussion are included in Table 1 (Bone et al., 1992). They were developed as part of a consensus conference of critical care practitioners, and are still endorsed by the American College of Chest Physicians after 20 years. Understanding the definitions related to sepsis is paramount because if a nurse is unable to clinically correlate a defined condition with a patient’s symptoms, signs of sepsis may go unnoticed. Figure 1 illustrates the sepsis continuum. Patients may begin with sepsis and quickly decompensate into septic shock.

<table>
<thead>
<tr>
<th>TABLE 1. Definitions</th>
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<tr>
<td><strong>Systemic Inflammatory Response Syndrome (SIRS)</strong></td>
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<td>Clinical inflammatory response from a non-specific insult, including two or more of the following:</td>
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<td>• Temperature &gt; 38° C or &lt; 36° C</td>
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<td>• Heart rate &gt; 90 beats/minute</td>
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<td>• Respiratory rate &gt; 20/minute or PaO₂ &lt; 32 mm Hg</td>
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<td>• White blood cell count &gt;12,000/mm³ or &lt; 4,000/mm³, or &gt; 10% immature neutrophils</td>
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<td><strong>Sepsis</strong></td>
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<td>• SIRS response with presumed/confirmed infection</td>
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<td><strong>Severe Sepsis</strong></td>
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<td>Sepsis associated with organ dysfunction:</td>
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<td>• Perfusion abnormalities (altered mental status, lactic acidosis, oliguria, etc.) or</td>
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<td>• Hypoperfusion (systolic blood pressure &lt; 90 mm Hg or systolic blood pressure drop of 40 mm Hg)</td>
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<td><strong>Septic Shock</strong></td>
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<tr>
<td>Sepsis with perfusion abnormalities and</td>
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<td>• Hypotension despite adequate fluid resuscitation</td>
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Source: Bone et al., 1992.

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Diagnosis of systemic inflammatory response syndrome (SIRS) is based on the presence of at least two of eight criteria (Bone et al., 1992). SIRS is an inflammatory response that is common in bacterial infections, but non-infectious causes may include burns, trauma, and hemorrhage. If a patient with any of these diagnoses is not improving, the nurse should remain vigilant for signs and symptoms of sepsis.

Sepsis occurs when a patient with SIRS has a presumed or confirmed infection (Bone et al., 1992). Infection can be presumed, for example, if a patient has a ruptured appendix, or if a surgical report indicates cultures are pending. The patient with sepsis that does not improve will develop severe sepsis. Severe sepsis is associated with organ dysfunction (Bone et al., 1992). It is present in 6%-15% of patients in the intensive care setting, and consumes almost half the resources (Dombroskiy, Martin, Sunderram, & Paz, 2007). Organ dysfunction manifests as perfusion abnormalities and may include new onset of mental status changes, lactic acidosis, and oliguria. Hypoperfusion can be noted clinically as a systolic blood pressure lower than 90 mm Hg or a systolic blood pressure drop of 40 mm Hg or more. Lastly, the patient with septic shock is recognized clinically as having severe sepsis and, despite receiving adequate fluid resuscitation, continues to have a blood pressure less than 90 mm Hg. Once a patient is in septic shock, he or she has a 39%-60% risk of mortality despite treatment (Sebat et al., 2005).

**Sepsis Causes**

Body systems most frequently affected by sepsis are respiratory (28.4%), cardiovascular (28.4%), and renal (23.1%) (Dombroskiy et al., 2007). Scott (2009) reported 4.5 hospital-acquired infections for every 100 admissions. The U.S. Department of Health and Human Services (2009) indicated three-fourths of all hospital-acquired infections can be associated with four types of infections: surgical site infections, central line-associated bloodstream infections, ventilator-associated pneumonia, and catheter-associated urinary tract infections. Not every patient with a hospital-acquired infection develops sepsis, but the potential exists for this progression. Preventing infections thus can prevent sepsis. Additionally, the average patient is older with more co-morbid diseases, more invasive procedures are performed, and sicker patients are kept alive longer (HCUP, 2010). Approximately two-thirds of patients hospitalized for sepsis in 2008 were age 65 or older. In patients age 85 and older, the sepsis hospitalization was about 30 times that of younger patients. A patient hospitalized for sepsis is eight times more likely to die and half as likely to be discharged home when compared to patients with other diagnoses (Hall et al., 2011). In 2009, sepsis was the 11th leading cause of death in the United States (behind liver disease) with a reported 35,587 deaths (Kochanek et al., 2011). However, occurrence may be underrepresented. For example, if a patient who has cancer also gets sepsis, the cause of death may be reported as cancer instead of sepsis.

**Cost of Sepsis**

Sepsis is not only prevalent, but costly. As hospitals are not reimbursed for many preventable illnesses, including certain infections, nurses have a vested interest in infection prevention. Approximately 1,737,125 hospital-acquired infections occur per year in the United States, with an average cost per patient of $25,903 (Scott, 2009). Sepsis costs were $15.4 billion in 2009 (Elixhauser et al., 2011).

**Pathophysiology of Severe Sepsis and Septic Shock**

Septic shock is a form of distributive shock. There is abnormal distribution due to vasodilation, capillary leakage, maldistribution of blood flow, and the release of myocardial depressant factor (Bridges & Dukes, 2005). The vasodilation results from nitric oxide release and endothelium changes on blood vessel walls. As a result of vasodilation and capillary leakage, the patient exhibits a lower blood pressure that decreases perfusion to vital organs, such as the kidney and brain. Perfusion abnormalities, such as a decrease in urine output or deterioration of mental status, become apparent clinically. On the cellular level, hypoperfusion leads to a change to anaerobic metabolism. This alteration causes changes in the glycolic path and citric acid cycle metabolic pathways, causing high lactate levels and acidosis. As cells continue without oxygen, the sodium potassium pump is impaired, the lysosomal membrane ruptures, and cell death can occur (Porth & Matfin, 2009).
In addition to these changes, coagulation defects occur (see Table 2). The capillary leakage described previously also activates the complement system, which causes additional inflammation and endothelial dysfunction (Bridges & Dukes, 2005). Endothelial damage promotes adhesion on vessel walls, allowing neutrophils to adhere to them. This damage activates tissue factor, the principal activator in coagulation. This causes thrombin to convert from soluble fibrinogen to fibrin. Fibrin clumps with platelets and forms clots that are circulated through the bloodstream. When the body makes clots, fibrinolysis typically helps destroys them. In sepsis, however, endothelial injury and inflammation inhibits fibrinolysis and clots remain in the bloodstream (Porth & Matfin, 2009).

The increase in inflammation increases coagulation and inhibits fibrinolysis. The patient has low blood pressure and impaired flow to organs. As microthrombi further impair blood flow to organs, cells die from the lack of oxygen. The bacterium is not the ultimate cause of death in septic shock; instead, death results from organ hypoperfusion as a result of vasodilation, capillary leakage, and thrombus formation (Porth & Matfin, 2009).

**Early Treatment of Severe Sepsis**

Early goal-directed therapy (EGDT) in the treatment of severe sepsis and septic shock has been understood as an effective way to reduce mortality since Rivers and co-authors (2001) found patients with severe sepsis or septic shock who received EGDT had 16.5% less mortality than patients who did not receive EGDT. Multiple studies have reproduced these findings (Levy et al., 2010; Micek et al., 2006; Nguyen et al., 2007; Shapiro et al., 2006; Shorr, Micek, Jackson, & Kollef, 2007). In 2008, Dellinger and co-authors initiated the Surviving Sepsis Campaign as an international effort to provide best practice guidelines. In 2002, before the campaign was introduced formally; they identified the aim to reduce mortality associated with sepsis by 25% by 2009; this goal was not met. The second phase focused on creating treatment guidelines, including plans for initial resuscitation in the first 6 hours after occurrence. An overview of these guidelines follows.

### Overview of Sepsis Guidelines

Initial resuscitation should begin immediately in patients with hypotension or an elevated serum lactate (>4 mmol/l). Treatment should not be delayed pending intensive care admission. Time is crucial, so when the patient experiences severe sepsis or septic shock on the medical-surgical unit, aggressive treatment needs to begin immediately. Calling for a Rapid Response Team or for an intensive care bed would be appropriate, depending on the setting and structure. Basic goals for resuscitation include a central venous pressure (CVP) of 8-12 mm Hg, mean arterial pressure (MAP) of 65 mm Hg, urine output greater than 0.5-1 mL/kg/hr, and mixed venous gas greater than 65%. Fluid resuscitation should begin with crystalloids or colloids to raise MAP and CVP. Initial diagnostics include obtaining two or more blood cultures (one percutaneously, one from each vascular device in place > 48 hours) and cultures from other sites as indicated. Antibiotics should be given intravenously as early as possible and always within the first hour of recognition. The source of the infection should be identified within the first 6 hours; if infection is from intravascular access, the device should be removed. The patient should be recognized as having severe sepsis or septic shock, fluids started, cultures completed, and antibiotics ordered and administered within 1 hour (Dellinger et al., 2008). These treatment guidelines require prompt sepsis identification and a team approach to treatment.

Recently, phase four of the Surviving Sepsis Campaign prompted the review of over 30,000 patients with sepsis. Their treatment according to the guidelines was shown to be successful in reducing the mortality for sepsis. Leaders of the Surviving Sepsis Campaign currently are reviewing guidelines and ways to further disseminate knowledge (Levy et al., 2010).

### Conclusion

Sepsis is deadly and costly, and can occur anywhere in the hospital. Prevention clearly is the best strategy. When severe sepsis and septic shock occur, early goal-directed therapy can decrease mortality; the most recent guidelines should be implemented whenever severe sepsis and septic shock are recognized (Dellinger et al., 2008). Nurses are a critical part of the health care team that provides evidence-based care to prevent, identify, and promptly treat sepsis in the hospital setting.

### REFERENCES


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**TABLE 2. Three-Part Problem**

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<th>Inflammation</th>
<th>Coagulation</th>
<th>Fibrinolysis</th>
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ADDITIONAL READING