The Pathophysiology of Sepsis

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**Pathophysiology of Sepsis**

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**Sepsis**

Sepsis is still a leading cause of hospital admissions and often leads to death if not identified early and treated properly (Pilcher, Cooper, & Ilitch, 2013). It is important for advanced practice nurses to recognize risk factors for sepsis. By understanding the pathophysiology of sepsis, nurse practitioners will be able to recognize early signs and symptoms of this inflammatory disease process and implement early medical management. Consequently, early medical management of sepsis is crucial to increase patient survival rates.

**The Importance Understanding Sepsis**

There is a high mortality rate linked with the diagnosis of sepsis and septic shock, which ranges from 30% to 40% (Wawrzeniak, Loss, & Netticadan, 2015). By understanding the pathophysiology of sepsis, patients have a greater chance of survival and improved outcomes.

**Pathophysiological Process**

**Disease Process**

The systemic response to severe infections leads to a lowered ability to fight infections, which can result in sepsis or multiorgan failure (Lord, Midwinter, Chen, Belli, Brash, Kovacs, & Sibbald, 2014). Several of the microcirculatory alterations occurring in septic patients include altered glycocalyx, endothelial dysfunction, impaired communication, and alterations in adhesion and production of red blood of the white blood cell, platelets, and leukocytes (De Backer, Gerts, Donadello, & Vincent, 2014). Severe infection is injury as a result of a documented or suspected infection. It causes the body's response to tissue injury, sepsis can be difficult to diagnose and may be due to trauma: an overview of the pathophysiology of sepsis. Infection is not properly identified and controlled, it can progress to sepsis, which leads to septic shock. Sepsis then leads to multiple organ dysfunction syndrome, multiorgan failure and ultimately death (Dunkley & McLeod, 2015). Understanding the pathophysiology of sepsis and recognizing early warning signs of sepsis will allow clinicians to provide appropriate treatment interventions. Many patients either ignore or are unable to recognize early warning signs of an infection therefore, healthcare providers must be advocates for these patients. It is important for advanced practice nurses to obtain a detailed patient history and physical. If an infection is suspected, start appropriate treatment is pertinent. Quick diagnosis and appropriate management can improve patient outcomes and decreased patient mortality in septic patients.

**Signs & Symptoms**

Sepsis can be difficult to diagnose because the signs and symptoms of sepsis can be subtle and often mimic other disorders (Dunkley & McLeod, 2014). To diagnose SIRS a patient must meet 2 of the following 4 criteria:

- Hypothermia or hyperthermia.
- Leukocytosis or leukopenia.
- Tachypnea.
- Increased creatinine.

**Other possible manifestations of sepsis**

- Hyperglycemia in patients who do not have diabetes.
- Increased procalcitonin.
- Altered mental status.

Table 1. Definitions of Sepsis inflammatory response syndrome (SIRS). (Lord, et al., 2014).

<table>
<thead>
<tr>
<th>SIRS</th>
<th>Meets 2 of the following 4 criteria:</th>
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<tr>
<td>Heart rate &gt; 100 bas/min</td>
<td>White blood cell count &gt; 10,000 X 10^9/L or &lt; 4,000 X 10^9/L</td>
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**Complications**

Severe sepsis is associated with complications that can be life-threatening and even fatal. These complications include shock, disseminated intravascular coagulation, and multiorgan dysfunction syndrome (Dunkley & McLeod, 2015).

**Underlying Pathophysiology**

The inflammation response is the body's response to noxious stimuli. Neutrophils mediate cellular changes. Vasodilation and inflammation occurs. The invading pathogenic microorganism stimulates the release of cytokines. Cytokines trigger an exaggerated inflammatory response. Fluid leaks from the circulation into the interstitial space. If a decrease in intravascular volume results in hypotension. Inadequate tissue perfusion leads to cellular hypoxia and lactic acidosis.

**Significance of Pathophysiology**

Understanding the pathophysiology of sepsis is vital to the development of sepsis protocols, which have proven to reduce mortality and improve patient survival. The biggest focus of treatment is early initiation of appropriate therapy and management of these patients. The complement system normally has three primary responsibilities: 1. Mediate the immune response to pathogens. 2. Stimulate the immune response as a result of a documented or suspected infection. 3. Eliminate the pathogen. The pathogen triggers an exaggerated immune response. Cytokines trigger an exaggerated inflammatory response. Fluid leaks from the circulation into the interstitial space. If a decrease in intravascular volume results in hypotension. Inadequate tissue perfusion leads to cellular hypoxia and lactic acidosis. Nitric oxide is produced by the endothelium during hypoxia.

**Altered Compensatory Mechanisms**

- Tachycardia: Tachycardia is a compensatory response to lactic acidosis. The hematocrit is stimulated to expel carbon dioxide in order to try and rid the blood of lactic acid. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulated to stimulate the sympathetic nervous response. Reduced cardiac output and stimulation of baroreceptors in the carotid sinus, upper aorta, and right atrium is stimulate...