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# Identifying Acute Organ Dysfunction as a Marker of Severe Sepsis

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# Identifying Acute Organ Dysfunction as a Marker of Severe Sepsis

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## Introduction:

The current definition of sepsis is suspected or documented infection plus at least one systemic manifestation of infection. Severe sepsis has been defined as sepsis plus evidence of organ dysfunction. Identifying acute organ dysfunction is a marker of severe sepsis. This topic was chosen after witnessing a number of patients fall into septic shock before severe sepsis was diagnosed and treatment initiated. Diagnosis of severe sepsis is important for specific protocol to be implemented in a timely fashion. The current differentiation between sepsis and severe sepsis is evidence of organ dysfunction. There are many examples of organ dysfunction that are not commonly considered when doing a patient workup. Increasing knowledge of acute organ dysfunction markers could potentially lead to earlier diagnosis of severe sepsis. The earlier it is diagnosed, the sooner treatment protocol can be initiated. Recent research showed that if severe sepsis is diagnosed timely, administration of timely drugs improved cardiovascular performance, lessened the inflammatory response, tissue hypoperfusion and multi-organ injury, and most importantly reduced mortality (Lin, Lee, & Wu, 2013).

## Signs and Symptoms:

The individual patient response to severe sepsis is dependent on characteristics of both the host (comorbidities and immunosuppression) and the pathogen (virulence and organism load). Coagulation abnormalities form from severe sepsis, such as intravascular coagulation and fibrinolysis that result in endothelial dysfunction, microvascular thrombi, and impaired tissue oxygenation. This impairment, combined with the systemic vasodilation and hypotension, causes tissue hypoperfusion and decreased tissue oxygenation, further complicated by impaired mitochondrial oxygen utilization secondary to oxidative stress. These mechanisms result in further tissue damage and ultimately contribute to multi-organ failure. Signs and symptoms of organ dysfunction commonly includes hypotension, acute respiratory distress syndrome, altered mental status, acute kidney injury, ileus, hepatic dysfunction, disseminated intravascular coagulation, adrenal dysfunction, and erythroid sick syndrome. The patient will have multiple indicators of organ dysfunction.

Examples include:

- altered consciousness, confusion
- tachypnea, SaO<sub>2</sub> decreased, decreased oxygen extraction ratio
- tachycardia, hypotension
- jaundice, decreased hepatic function evidenced by increased bilirubin
- oliguria, decreased platelets and reduced kidney function evidenced by increased creatinine
- plasma IL-6 and IL-10 levels will show increase (Tutak, Ozer, Demirel & Bayar, 2014)
- Increased lactate/albumin ratio is another value that should be considered because it correlates with the mortality in patients with severe sepsis and septic shock (Wang, Chen, Cao, Xue, Li, & Wu, 2015)

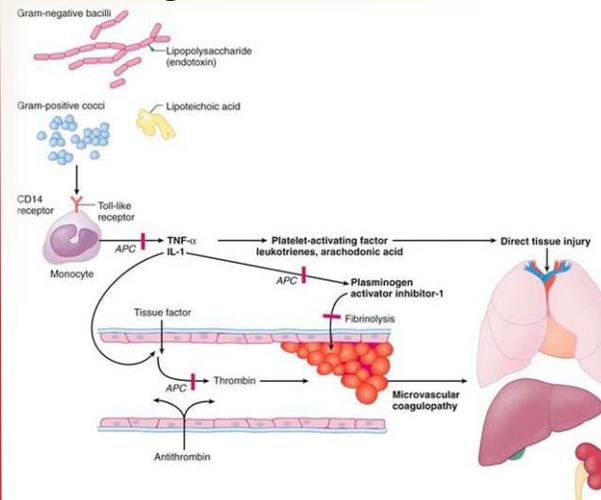
## Underlying Pathophysiology:

Sepsis, severe sepsis, and septic shock may be secondary to either community-acquired, health care associated or hospital-associated infections. The most common underlying causes are pneumonia, intra-abdominal infections, and urinary tract infections. Etiologic organisms cover the spectrum of pathogens, with bacteria and fungi being predominant. However, Staphylococcus aureus, Staphylococcus epidermidis, Enterococcus species, Streptococcus pneumoniae, Escherichia coli, Pseudomonas aeruginosa, species within the Klebsiella family, and Candida species account for most of the pathogens described (Cawcutt & Peters, 2014). Infection triggers both pro-inflammatory and anti-inflammatory processes that ultimately contribute to the clearance of infection and the tissue damage that lead to organ failure. In general, the pro-inflammatory processes are triggered by the infectious agent and are focused on the elimination of the pathogen, whereas the anti-inflammatory processes are triggered by the host to promote tissue repair and healing. An imbalance of these mechanisms lead to excess tissue damage.

## Significance of Pathophysiology:

Understanding the pathophysiology of severe sepsis is significant to good patient outcomes. The number of septic cases in the United States exceeds 750,000 per year and was recently reported to be rising. Sepsis is the 10th leading cause of death in the United States and the leading cause of death in non-cardiac ICUs. One patient in the United States presents to an emergency department every minute with severe sepsis and accounts for 20% of all ICU admissions (Palleschi, Sirianni, O'Connor, Dunn, & Hasenau, 2014). Knowledge of organ dysfunction markers could help identify severe sepsis quickly which means that treatment protocol can be initiated earlier. Comprehending the etiologic origins of sepsis and infectious process they take, means appropriate anti-infective measures can be implemented and patient outcome improved. One example of the significance of pathophysiology of severe sepsis is the symptom of low oxygen extraction ratio. Recent research shows initial low OER (Oxygen Extraction Ratio) was associated with severe organ dysfunction that resulted in high mortality with severe sepsis and septic shock. When patients had initial abnormally low OER, their in-hospital mortality was higher than in normal OER patients. Therefore, the OER should be considered when attempting to predict the outcome of septic patients (Kim, Lee, Lee, Han, Moon, & Hong, 2015).

## Sepsis leading to Organ Failure



## Nursing Considerations:

Increased nursing knowledge of organ dysfunction markers could improve severe septic patient outcomes. The earlier the patient is identified as severe sepsis, the sooner that they can receive protocol treatment. Recent studies show that nurses play a key role in the initial triage and care of patients with potentially life-threatening sepsis. The research showed the impact of a nurse-initiated emergency department sepsis protocol to initial antibiotic administration, and had an impact on in-hospital sepsis mortality rates (Bruce, Maiden, Fedullo, & Kim, 2015). A recent study by Gaieski and colleagues supports the nursing clinical relevance by showing that elapsed time from triage to administration of antimicrobials is a primary determinant of mortality of patients with severe sepsis and septic shock (Gaieski et al., 2010). The knowledge of a severe sepsis screening tool significantly decreased the mean time to antibiotics in patients presenting to the ED with suspected severe sepsis or septic shock (Patocka, Turner, Xue, & Segal, 2014). This research demonstrates that increased nursing understanding of sepsis and severe sepsis markers can significantly improve patient care and outcomes.

## Conclusion:

Despite advances in the development of numerous drugs and supportive care therapies, severe sepsis remains an unconquered challenge for clinical investigators and physicians with an unacceptable high mortality rate of 28% to 50%. Sepsis is the most common cause of death in the non-cardiac intensive care unit (Mingming, Zhan, Mian, Jun, Zhi, Jin, & Zhenju, 2015). Treatment protocol differs when a patient is determined to have severe sepsis as opposed to sepsis. For this reason, it is significant that health care professionals be able to identify markers of organ dysfunction as a sign of severe sepsis. Knowledge of these markers and the pathophysiology associated with severe sepsis can improve patient outcomes significantly.

Table 1. BRUSSELS ORGAN DYSFUNCTION DEFINITIONS

Organs	Clinically Significant Organ Dysfunction				
	Normal	Mild	Moderate	Severe	Extreme
Cardiovascular (systolic blood pressure, mmHg)	> 90	≤ 90 fluid responsive	≤ 90 not fluid responsive	≤ 90 pH 7.3	≤ 90 pH 7.2
Pulmonary (PaO <sub>2</sub> /FiO <sub>2</sub> , mmHg)	> 400	400 - 301	300 - 201	200 - 101	100
Neurologic (GCS)	15	14 - 13	12 - 10	9 - 6	≤ 5
Coagulation (platelet count, x10 <sup>3</sup> /mm <sup>3</sup> )	> 120	120 - 81	80 - 51	50 - 21	≤ 20
Renal (creatinine, μmol/L [mg/dL])	<133 [1.5]	133 - 175 [1.5-1.9]	176 - 300 [2.0-3.4]	301 - 442 [3.5-4.9]	≥443 [≥5.0]
Hepatic (bilirubin, μmol/L [mg/dL])	< 20 [1.2]	20 - 32 [1.2-1.9]	33 - 99 [2.0-5.9]	100 - 199 [6.0-11.9]	≥200 [≥12.0]

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