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### Sepsis to Septic Shock

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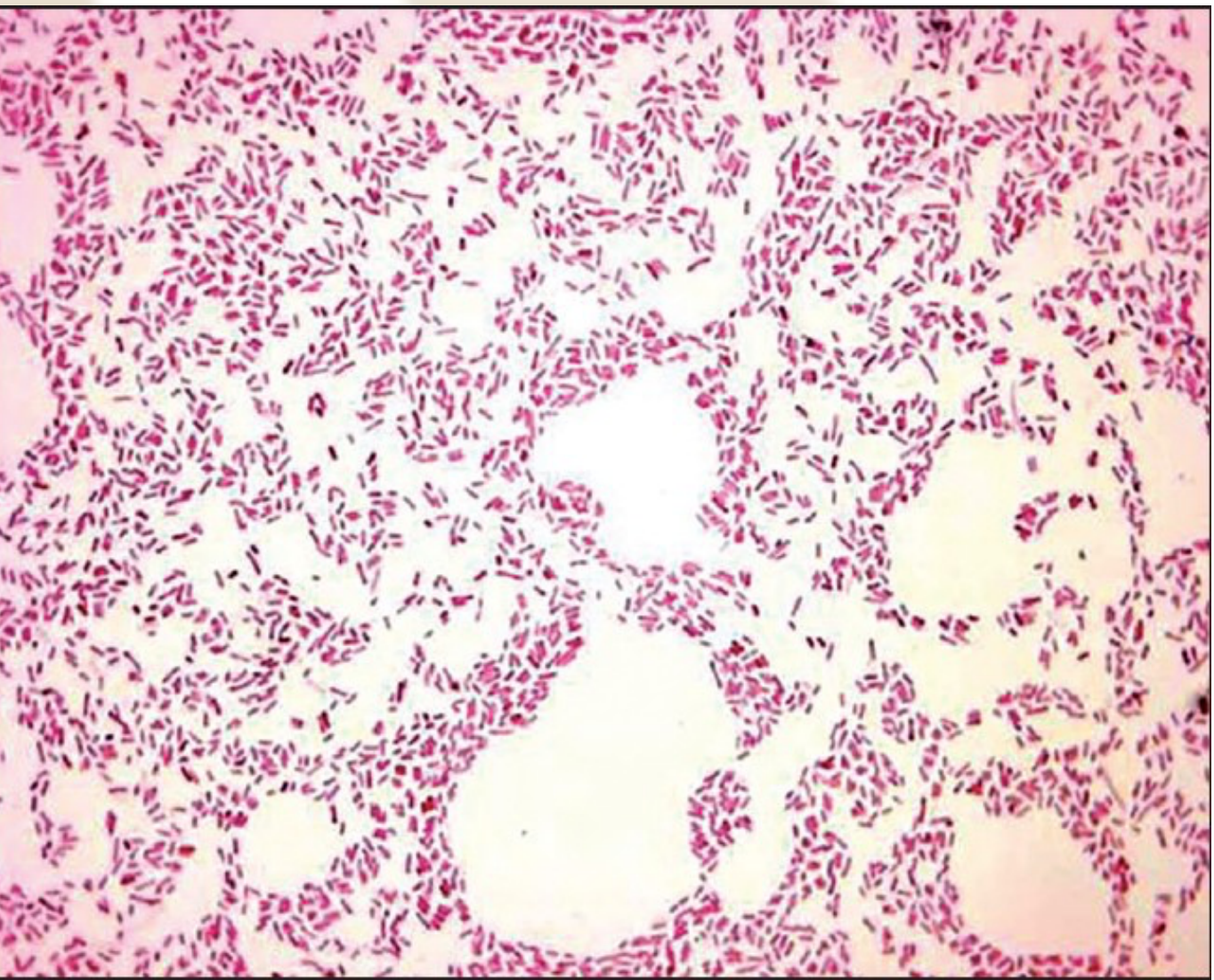
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## Background

Sepsis has long been part of medicine and conceptualized using multiple definitions and criteria. Without consistent and reproducible criteria the extensive pathophysiology associated with sepsis is difficult to diagnose and treat. The CDC reports approximately 1 million patients are diagnosed with sepsis each year (Sepsis, 2016). Treatment guidelines are ambiguous and patients incur a prolonged hospital stay while receiving complex therapy. Patients diagnosed with sepsis reflect an in-hospital mortality risk of 10% and those who develop septic shock increase their mortality risk greater than 40% (Singer, et al., 2016). In 2016, a task force consisting of international healthcare clinicians who lead the world in sepsis pathophysiology, clinical trials and sepsis epidemiology convened a consensus to develop updated definitions and criteria related to sepsis. Singer et al. (2016) proposed sepsis is a “life-threatening organ dysfunction caused by dysregulated host response to infection”. Furthermore, septic shock is the bodies deterioration affecting circulation and cellular metabolic homeostasis thus increasing mortality (Singer, et al., 2016). Outside the critical care arena sepsis is often misdiagnosed thus increasing healthcare costs thru delay in treatment all while increasing patient mortality (Mouncey et al., 2015).



### Infecting Microorganisms

Pathogens associated with sepsis are predominantly derived from the blood stream, lungs, kidneys and GI tract.

- Gram Positive: 47%
- Gram Negative: 62%
- Fungal: 19%

Source: (Gotts & Matthay, 2016)

## Signs / Symptoms

### Sepsis

- Specific to region related to primary offense (ex: cough r/t pneumonia, pain r/t non-healing wound, abdominal pain r/t pancreatitis)
- Hypotension (SBP < 90mmHg, MAP < 70mmHg)
- Temperature > 38.3C or < 36C
- Heart Rate > 90 beats/minute
- Tachypnea – RR > 20breaths/minute

Source: (Rhodes et al., 2017), (Singer et al., 2016)

### Laboratory Markers

- Leukocytosis (WBC>12K) or Leukocytopenia (WBC < 4K)
- Hyperglycemia (serum glucose > 14mg/dl)
- Arterial hypoxemia (PaO2/FiO2 < 300)
- Oliguria (UOP < 0.5ml/kg/hr)
- Thrombocytopenia (platelet < 100K)
- Hyperlactatemia (lactate >2mmol/L)
- Coagulation dysfunction (INR>1.5, aPTT>60 seconds)

Source: (Rhodes et al., 2017), (Laszlo et al., 2015)

### Septic Shock

Clinical presentation includes previous sign/symptoms but the patient requires vasopressor support to maintain mean arterial pressure > 65mmHg and hyperlactitemia > 2mmol/L despite adequate fluid resuscitation. Septic shock represent an advanced diagnosis where circulatory, cellular, and metabolic abnormalities reflect increased patient mortality.

Source: (Laszlo et al., 2015), (Gotts & Matthay, 2016)

## Risk Factors

Sepsis is a diagnosis that can affect anyone susceptible to an infecting organism. Sepsis and septic shock are the host organisms reaction to infection and the inability of an adequate immune response. Risk factors associated with sepsis and septic shock include:

- ICU admission
- Bacteremia
- Age > 65 yo
- African Americans > risk of developing sepsis
- Male > female
- Immunocompromised
- Comordities (ie Diabetes, Cancer)
- Previous hospitalization
- Community acquired pneumonia

Source: (Prescott et al., 2015), (Sepsis, 2016),

### Modifiable Risk Factors

- Alcohol consumption
- Cigarette smoking
- Vitamin B12 deficiency
- Vaccination

Source: (Gotts & Matthay, 2016)

## Pathophysiology

Sepsis is a “life-threatening organ dysfunction caused by dysregulated host response to infection (Singer et al., 2016). The normal host reaction to pathogens involves adequate immune response including innate and adaptive immunity. Sepsis involves the pro-inflammatory mediators transitions beyond the local site to wreak systemic influences which become generalized (Laszlo et al., 2015). Basic sepsis pathophysiology involves increased cardiac output when systemic vascular resistance is decreased given biventricular deterioration. General abnormalities associated with sepsis include excessive alterations in endothelium along with microcirculatory impairment (Gotts & Matthay, 2016).

### Inflammation Pathway Discord

- Macrophage, monocytes, granulocytes, natural killer cells – detect pathogen associated molecular patterns (PAMP) and damage associated molecular patterns (DAMP)
- PAMP + DAMP activate innate immune response after binding to Toll-like receptors C-type lectin receptors, resulting in secretion of pro-inflammatory mediators TNF-a, IL1, and IL6.
- Inflammatory cytokines initiate complement cascades (C5a) which promotes reactive oxygen species (ROS) and nitric oxide to impair cellular proteins and function, DNA, and mitochondria ATP output
- Results: increased endothelium permeability, systemic immunothrombosis with microvascular impaired function (DIC)

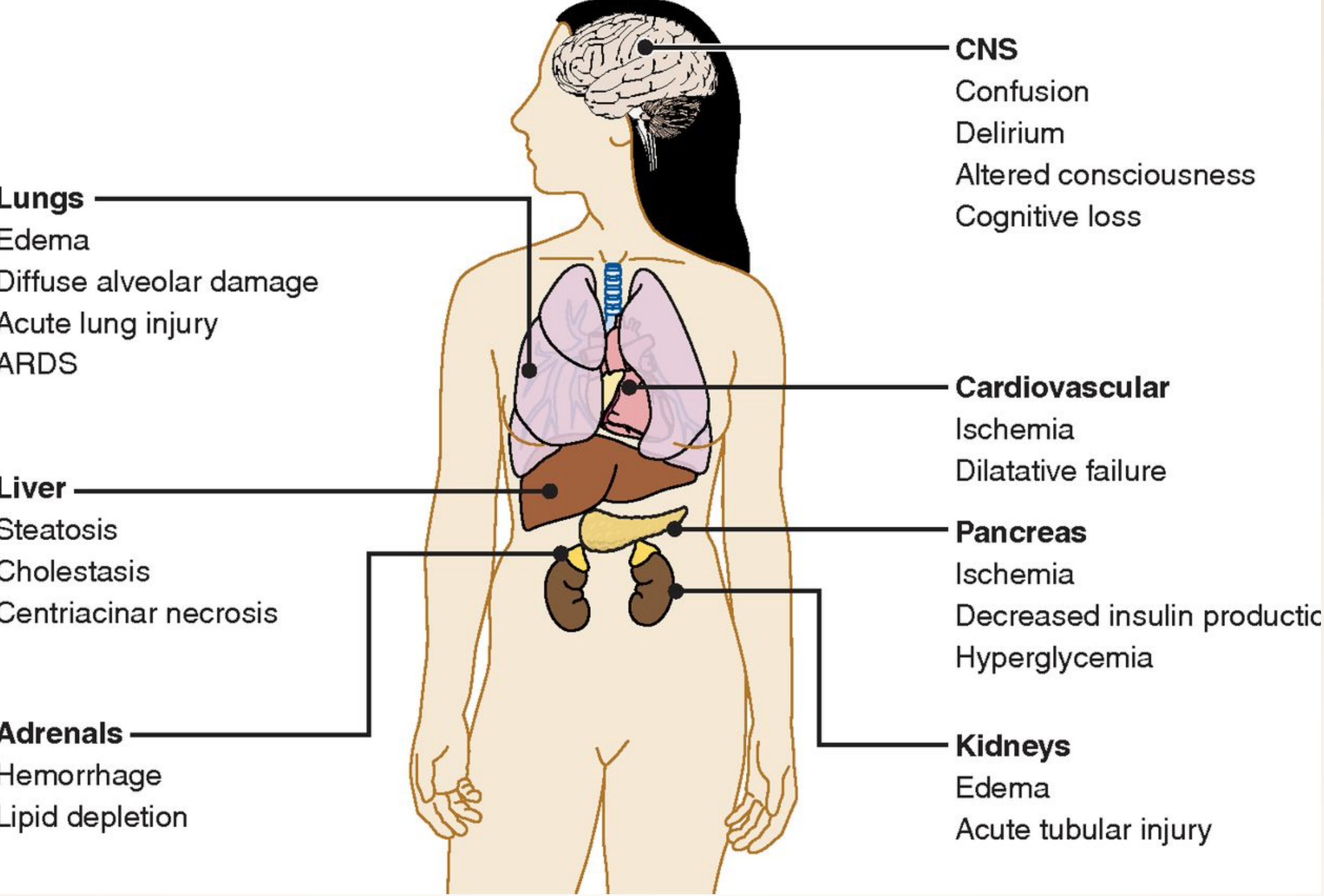
### Metabolic Dysfunction

- Decreased ATP production at cellular level leads to hypoxia
- Results: catabolism with significant reduction in muscle mass, acute kidney injury, myocardial depression, hepatic dysfunction, endothelial and epithelial permeability causing acute lung injury

### Organ / Tissue Impairment

- Alteration to endothelium permeability: Lungs – protein rich edema fills interstitial tissue, leads V/Q mismatch, arterial hypoxemia, ARDS
- Liver: hepatocyte dysfunction; inability to clear bilirubin
- Kidney: cytokine and immune mediated microvascular dysfunction
- Nervous system: encephalopathy related to compromised blood brain barrier endothelium and pathogen infection
- Spleen: depeleted CD4, CD8 Tcells affect long term host immune response

Source (Gotts & Matthay, 2016)



End Stage Organ Dysfunction

Source: (Iskander et al., 2013)

## Clinical Significance

Sepsis has proven to be an elusive diagnosis where one size does not fit all. The inability to stage the disease given the multitude of disease progression continues to cause controversy in the management of sepsis. The Surviving Sepsis Campaign was convened to establish unifying criteria on definition, treatment and prevention protocols proven to combat sepsis (Howell & Davis, 2017). Accepted guidelines call for rapid administration of broad-spectrum antibiotics within 1 hour of suspected sepsis event. Each hour in the delay of antibiotic therapy is associated with a 12% reduction in surviving a septic diagnosis(Gotts & Matthay, 2016). After antibiotic therapy has been initiated, guidelines call for pan-culture to identify the infective microorganism. Crystalloid fluid resuscitation is used to maintain a MAP > 65mmHg (Howell & Davis, 2017). If hypotension persists, first line drug therapy should be norepinephrine. Mechanical ventilation may be indicated for sustained hypoxemia with PaO2 < 75 (Howell & Davis, 2017). In general, the role of the healthcare practitioner is to optimize the host defense and response to an infecting organism (Gotts & Matthay, 2016).

## Conclusions

Patients suffering from invading microorganism develop sepsis that often deteriorates to septic shock. It is s a common disease process with a vast and complex progression through multiple systems in the body. The dynamic nature of sepsis leads to challenges in the management of sepsis. The overwhelming and sometime contrasting nature of proven therapies leads to it being a deadly and expensive diagnosis seen throughout healthcare. Since national consensus and guidelines have been implemented there has been improvements in outcomes, while the rate of sepsis has also increased. This is due to vigilance in disease identification, rapid delivery of antibiotic therapy, and supportive treatments to subsequent organ failure. Critical care nurses play a vital role in early detection and initiation of sepsis protocols. Critical to improving patient mortality requires the bedside nurse to understand sepsis pathophysiology, signs and symptoms, and anticipate disease progression while monitoring the patient clinical response to treatment.

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## Implications to Nursing Care

In 2002, the Surviving Sepsis Campaign started a quality improvement initiative to improve knowledge related to sepsis, improve diagnosis, and develop guidelines of care. Amongst the various strategies is the Surviving Sepsis Campaign Bundles (SSCB). These guidelines focus care to satisfy elements that, when done collectively, improve patient outcomes. Nurses represent a vital role in the healthcare continuum for recognizing early signs and symptoms related to sepsis. Many healthcare institutions have customized the SSCB which allows the ICU nurse to initiate established protocol based on agreed upon criteria (Surviving Sepsis, 2015). One approved diagnostic tool is use of the quick SOFA (sequential organ failure assessment) score. A positive screen reflects 2 of 3 criteria including: respiratory rate > 22 bpm, Glasgow coma scale < 15, and/or SBP < 100 mmHg (Gotts & Matthay, 2016).

