



Management of Sepsis in the Adult

Two (2.0) Contact Hours

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Purpose and Objectives

The purpose of this course is to inform the healthcare provider of the updated guidelines regarding early identification, diagnosis, and goal directed therapy from evidenced based research from the international Surviving Sepsis Campaign. This Campaign is an international initiative to improve patient outcomes.

After successful completion of this course, you will be able to:

1. Define infection, systemic inflammatory response syndrome (SIRS), sepsis, severe sepsis, septic shock, and multiple organ dysfunction syndrome (MODS)
2. Identify early signs and symptoms of sepsis
3. Recognize how to diagnosis sepsis
4. Identify the importance of early intervention according to latest standards when identifying sepsis
5. List the treatment for severe sepsis and septic shock

Introduction

Severe sepsis and septic shock are major healthcare problems, affecting millions of people around the world each year, killing one in four (and often more), and increasing in incidence (Dellinger, et al., 2013). Sepsis affects over 26 million people worldwide each year and kills more people than breast, cancer, and lung cancer combined, yet most people haven't heard of it (Sepsis Alliance, 2014). Every year, severe sepsis strikes more than a million Americans. It's been estimated that between 28 and 50 percent of these people die. (National Center for Health Statistics Data Brief No. 62, 2011). Septicemia was the most expensive reason for hospitalization in 2009, totaling nearly \$15.4 billion (AHRQ, 2011).

This course reviews the general shock response, sepsis, systemic inflammatory response syndrome (SIRS), and septic shock including multiple organ dysfunction syndrome (MODS). New guidelines for the identification and management of sepsis were recently released that advocate for implementation of care based on recent evidence-based practice (Kleinpell, Aitken & Schorr, 2013).

According to the CDC (2014), anyone can get sepsis, but the risk is higher in:

- People with weakened immune systems
- Infants and children
- Elderly people
- People with chronic illnesses, such as diabetes, AIDS, cancer, and kidney or liver disease
- People suffering from a severe burn or physical trauma

Definitions

Sepsis occurs when microorganisms invade the body and initiate a systemic inflammatory response. This host response often results in perfusion abnormalities with organ dysfunction (severe sepsis) and eventually hypotension (septic shock). To understand terms used in this course, the following definitions will be reviewed:

- Infection and bacteremia
- SIRS
- Sepsis
- Severe sepsis
- Sepsis-induced tissue hypoperfusion
- Pathophysiology of shock
- Global indicators of systemic perfusion and oxygenation
- Septic shock
- Multiple organ dysfunction syndrome (MODS)

The Dangerous Progression from Infection to Septic Shock

Definitions of Infection and Bacteremia

Infection is the microbial phenomenon characterized by an inflammatory response to the presence of microorganisms or the invasion of normally sterile host tissue by these organisms (Neviere, 2014).

Bacteremia is the presence of viable bacteria in the blood (Neviere, 2014).

Definition of SIRS

Systemic inflammatory response (SIRS) is an intense host response characterized by generalized inflammation in organs remote from initial insult. SIRS causes massive inflammatory dysfunction involving activation of leukocytes and endothelial cells and the release of inflammatory mediators and toxic oxygen free radicals of intracellular and extracellular origin (Neviere, 2014).

This mechanism leads to abnormalities in tissue perfusion and tissue hypoxia that result in tissue destruction, multiorgan failure, and death of critically ill patients in the ICU (Visvanathan, 2013).

Definition of Sepsis

Sepsis occurs when microorganisms invade the body and initiate a systemic inflammatory response.

It is the body's systemic response to infection and is a serious healthcare condition (Kleinpell, Aitken & Schorr, 2013).

Sepsis is caused by a wide variety of microorganisms, including gram-negative and gram-positive aerobes, anaerobes, fungi, and viruses. The respiratory system is the most common site of infection. Over 50% of sepsis cases is caused by gram-positive organisms; therefore, gram-positive bacteria are the predominant cause of sepsis. (Neviere, 2014).

Definition of Severe Sepsis

Severe sepsis is sepsis that has progressed to cellular dysfunction and organ damage or evidence of hypoperfusion (Kleinpell, Aitken & Schorr, 2013). According to Dellinger, et al. (2013), severe sepsis definition has any of the following which are thought to be due to the infection:

- Sepsis-induced hypotension
- Lactate above upper limits laboratory normal
- Urine output < 0.5 mL/kg/hr for more than 2 hours despite adequate fluid resuscitation
- Acute lung injury with Pao₂/Fio₂ < 250 in the absence of pneumonia as infection source
- Acute lung injury with Pao₂/Fio₂ < 200 in the presence of pneumonia as infection source
- Creatinine > 2.0 mg/dL (176.8 μmol/L)
- Bilirubin > 2 mg/dL (34.2 μmol/L)
- Platelet count < 100,000 μL
- Coagulopathy (international normalized ratio > 1.5)

Evaluation tool for severe sepsis can be found at

<http://www.survivingsepsis.org/SiteCollectionDocuments/ScreeningTool.pdf>

Brief Pathophysiology Review of the Stages of Shock

Shock involves ineffective tissue perfusion and acute circulatory failure. The shock syndrome is a pathway involving a variety of pathologic processes that may be categorized as four stages: initial, compensatory, progressive, and refractory (Urden, Stacy, & Lough, 2014).

- **Initial stage** - cardiac output (CO) is decreased, and tissue perfusion is threatened.
- **Compensatory** - Almost immediately, the compensatory stage begins as the body's homeostatic mechanisms attempt to maintain CO, blood pressure, and tissue perfusion.
- **Progressive** - The compensatory mechanisms begin failing to meet tissue metabolic needs, and the shock cycle is perpetuated.
- **Refractory** - Shock becomes unresponsive to therapy and is considered irreversible.

According to Urden, Stacy, & Lough (2014), as the individual organ systems die, MODS occurs. Death occurs from ineffective tissue perfusion because of the failure of the circulation to meet the oxygen needs of the cell.

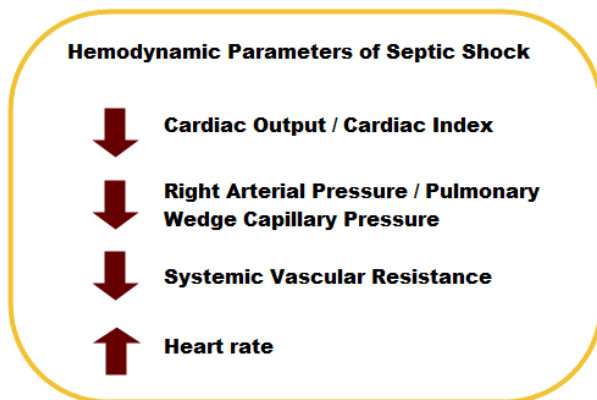
Global Indicators of Systemic Perfusion & Oxygenation

According to the Urden, Stacy, & Lough (2014), global indicators of systemic perfusion and oxygenation include:

- **Serum lactate**- Inadequate cellular oxygenation with anaerobic metabolism and increased metabolic lactate production increase the serum lactate level. The level and duration of this hyperlactatemia are predictive of morbidity and mortality, and management guided by lactate levels has been effective in improving outcomes.
- **Arterial base deficit**- The base deficit derived from arterial blood gas (ABG) values also reflects global tissue acidosis and is useful to assess the severity of shock.
- **Serum bicarbonate**- Studies have demonstrated serum bicarbonate to be an equivalent alternative to arterial base deficit in predicting mortality in surgical and trauma patients.
- **Central or mixed venous oxygen saturation levels**- Equivalent alternative to arterial base deficit in predicting mortality in surgical and trauma patients. The use of mixed venous oxygen saturation (Svo₂) measured by means of a pulmonary artery catheter or central venous oxygen saturation (Scvo₂) measured with a central venous catheter allows assessment of the balance of oxygen delivery and oxygen consumption and the ratio of oxygen extraction.

Definition of Septic Shock

Septic Shock is sepsis with persistent hypotension despite adequate fluid resuscitation (Kleinpell, Aitken & Schorr, 2013). The patient with a mean arterial blood pressure (MAP) less than 60 mm Hg or with evidence of global tissue hypoperfusion is considered to be in a shock state.



Sepsis-Induced Tissue Hypoperfusion

Sepsis- Induced Tissue Hypoperfusion: is defined as hypotension persisting after initial fluid challenge or blood lactate concentration ≥ 4 mmol/L.

Test Yourself

Septic shock results from initiation of systemic inflammatory response due to microorganisms entering body.

- A. True
- B. False

The correct answer is: True. Sepsis occurs when microorganisms invade the body and initiate a

systemic inflammatory response. This host response often results in perfusion abnormalities with organ dysfunction (severe sepsis) and eventually hypotension (septic shock).

Definition of MODS

Multiple organ dysfunction syndrome (MODS) results from progressive physiologic failure of two or more separate organ systems in an acutely ill patient such that homeostasis cannot be maintained without intervention. MODS is the major cause of death in patients cared for in critical care units. Mortality is closely linked to the number of organ systems involved. MODS survivors may develop generalized polyneuropathy and a chronic form of pulmonary disease from acute respiratory distress syndrome (ARDS), complicating recovery (Urden, Stacy, & Lough, 2014). The systems that MODS can affect include:

1. Gastrointestinal

Normal gut flora and gut environment are altered in patients with severe SIRS.

2. Hepatobiliary

The liver responds to sustained inflammation by selectively altering carbohydrate, fat, and protein metabolism. Consequently, hepatic dysfunction threatens the patient's survival.

3. Pulmonary

The lungs are frequent and early target organs for mediator-induced injury and are usually the first organs affected in the progression of SIRS to MODS. ARDS is the pulmonary manifestation of MODS. ARDS associated with MODS usually occurs 24 to 72 hours after the initial insult.

4. Renal

The kidney is highly vulnerable to hypoperfusion and reperfusion injury. Consequently, kidney ischemic-reperfusion injury may be a major cause of kidney dysfunction in MODS.

5. Cardiovascular:

The initial cardiovascular response in SIRS or sepsis is myocardial depression; decreased RAP and SVR; and increased venous capacitance, CO, and heart rate. Despite an increased CO, myocardial depression occurs and is accompanied by decreased SVR, increased heart rate, and ventricular dilation. These compensatory mechanisms help maintain CO during the early phase of SIRS or sepsis. An inability to increase CO in response to a low SVR may indicate myocardial failure or inadequate fluid resuscitation, and it is associated with increased mortality.

6. Hematologic

The most common manifestations of hematologic dysfunction in sepsis or MODS are thrombocytopenia, coagulation abnormalities, and anemia. The most severe is coagulation system dysfunction manifesting as disseminated intravascular coagulation (DIC). Low platelet counts and elevated d-dimer concentrations and fibrinogen degradation products are clinical indicators of DIC.

Criteria for Definition of SIRS, Sepsis, and MODS

Overview of Diagnosing Sepsis

The diagnosis of severe sepsis is based on the identification of three conditions:

- Known or suspected infection
- Two or more of the clinical indications of the systemic inflammatory response such as:
 - Temp $>38^{\circ}\text{C}$ or $<36^{\circ}\text{C}$
 - Heart rate >90 bpm
 - Respiratory rate >20 bpm or $\text{PaCO}_2 <32$ mmHg
 - WBC $>12,000$ or $<4,000$ mm
 - Evidence of at least one organ dysfunction

If the patient is **SEPTIC** as defined above measure BP and send lactate level STAT.

If BP <90 mmHg and/or lactate ≥ 4 mmol/L, administer a 20-30 mL/kg fluid bolus immediately. Repeat BP and lactate level (wait 2h before next lactate).

The patient is septic with **hypoperfusion** or **shock** if:

- a) **Lactate ≥ 4 mmol/L after 20-30 mL/kg fluid bolus AND/OR**
- b) **SBP <90 mmHg after 20-30 mL/kg fluid bolus**

Detailed Diagnostic Criteria for Sepsis

According to Surviving Sepsis Campaign, Dellinger et al. (2013) states the diagnostic criteria for sepsis. Infection, documented or suspected, and some of the following variables:

General variables:

- Fever (> 38.3 degrees C)
- Hypothermia (core temperature < 36 degrees C)
- Heart rate > 90 /min
- Tachypnea
- Altered mental status
- Significant edema or positive fluid balance (> 20 mL/kg over 24 hr)
- Hyperglycemia (plasma glucose > 140 mg/dL or 7.7 mmol/L) in the absence of diabetes

Inflammatory variables:

- Leukocytosis (WBC count $> 12,000$ μL^{-1})
- Leukopenia (WBC count < 4000 μL^{-1})
- Normal WBC count with greater than 10% immature forms
- Plasma C-reactive protein more than two standard deviation (sd) above the normal value
- Plasma procalcitonin more than two sd above the normal value

Hemodynamic variables:

- Arterial hypotension (SBP < 90 mm Hg, MAP < 70 mm Hg, or an SBP decrease > 40 mm Hg in adults)

Organ dysfunction variables:

- Arterial hypoxemia ($P_{aO_2}/F_{iO_2} < 300$)
- Acute oliguria (urine output < 0.5 mL/kg/hr for at least 2 hours despite adequate fluid resuscitation)
- Creatinine increase > 0.5 mg/dL or 44.2 $\mu\text{mol/L}$
- Coagulation abnormalities (INR > 1.5 or aPTT > 60 s)
- Ileus (absent bowel sounds)
- Thrombocytopenia (platelet count $< 100,000$ μL^{-1})
- Hyperbilirubinemia (plasma total bilirubin > 4 mg/dL or 70 $\mu\text{mol/L}$)

Tissue perfusion variables:

- Hyperlactatemia (> 1 mmol/L)
- Decreased capillary refill or mottling

Making the Diagnosis

The Surviving Sepsis Campaign (Dellinger et al., 2014) lists the recommended diagnostics to promptly make a diagnosis of sepsis:

1. Cultures as clinically appropriate before antimicrobial therapy if no significant delay (> 45 mins) in the start of antimicrobial(s)
2. At least 2 sets of blood cultures (both aerobic and anaerobic bottles) be obtained before antimicrobial therapy with at least 1 drawn percutaneously and 1 drawn through each vascular access device, unless the device was recently (< 48 hours) inserted
3. Use of the 1, 3 beta-D-glucan assay, mannan and anti-mannan antibody assays, if available and invasive candidiasis is in differential diagnosis of cause of infection
4. Imaging studies performed promptly to confirm a potential source of infection

SURVIVING SEPSIS CAMPAIGN BUNDLES

The Surviving Sepsis Campaign (Dellinger et al., 2013) designed the “Surviving Sepsis Campaign Bundle” to create a protocol when there is suspicion of sepsis.

TO BE COMPLETED WITHIN 3 HOURS:

- Measure lactate level
- Obtain blood cultures prior to administration of antibiotics
- Administer broad spectrum antibiotics
- Administer 30 mL/kg crystalloid for hypotension or lactate 4 mmol/L

TO BE COMPLETED WITHIN 6 HOURS:

- Apply vasopressors (for hypotension that does not respond to initial fluid resuscitation) to maintain a mean arterial pressure (MAP) 65 mm Hg
- In the event of persistent arterial hypotension despite volume resuscitation (septic shock) or initial lactate 4 mmol/L (36 mg/dL):
- Measure central venous pressure (CVP)*

- Measure central venous oxygen saturation (ScvO₂)*
- Remeasure lactate if initial lactate was elevated*

**Targets for quantitative resuscitation included in the guidelines are CVP of 8 mm Hg, ScvO₂ of 70%, and normalization of lactate.*

(Dellinger et al. 2013)

This entire chart is part of the national campaign. It is used in all publication that reference the new guidelines.

Septic Shock Algorithm

The septic shock algorithm can be found here:

<http://www.survivingsepsis.org/SiteCollectionDocuments/Protocols-Pocket-Card-StJoseph.pdf>

Initial Resuscitation Goals:

According to the Surviving Sepsis Campaign (Dellinger, 2013), for the patients with sepsis- induced tissue hypoperfusion, the goals during the first 6 hours of resuscitation are:

- CVP 8–12 mm Hg
- MAP ≥ 65 mm Hg
- Urine output ≥ 0.5 mL.kg.hr
- Superior vena cava oxygenation saturation (Scvo₂) or mixed venous oxygen saturation (Svo 70% or 65%)
- In patients with elevated lactate levels, targeting resuscitation to normalize lactate

Treatment Overview of Severe Sepsis/Septic Shock

The goals of treatment are to reverse the pathophysiologic responses, control the infection, improvement and preservation of tissue perfusion, and promote metabolic support. This approach includes supporting the cardiovascular system and enhancing tissue perfusion, identifying and treating the infection, limiting the systemic inflammatory response, restoring metabolic balance, and initiating nutritional therapy. The following treatments to sepsis will be reviewed based on new evidenced based research:

- Source control
- Infection prevention
- Hemodynamic support and adjunctive therapy
 - Fluid therapy
 - Vasopressors
 - Inotropic therapy
 - Administration of blood products
- Supportive therapy for severe sepsis

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- Mechanical ventilation in patients with sepsis-induced respiratory distress syndrome
- Sedation, analgesia, and neuromuscular blockade in patients with sepsis
- Glucose control
- Renal replacement therapy
- Prophylaxis of deep vein thrombosis
- Stress ulcer prophylaxis
- Nutrition
- Goals of care

Source Control

Priority is to identify the source of the infection. Also note:

- During the assessment, pay careful attention to areas of redness and inflammation.
- Look for abscess during skin assessment.
- Drainage at the insertion site of a vascular access for a potential catheter-associated bloodstream infection. Assess for need to discontinue the catheter.
- Drainage of abscess or cholangitis, removal of infected catheters, debridement or amputation of osteomyelitis.

Antimicrobial Therapy

When septic shock and severe sepsis without septic shock is recognized, the effective intravenous antimicrobial should be administered within the first hour. The following are the guidelines from Dellinger, et al. (2013) on the use of antimicrobial therapy with the septic shock or severe sepsis patient:

- Initial empiric anti-infective therapy of one or more drugs that have activity against all likely pathogens (bacterial and/or fungal or viral) and that penetrate in adequate concentrations into tissues presumed to be the source of sepsis.
- Antimicrobial regimen should be reassessed daily for potential deescalation.
- Combination empirical therapy for neutropenic patients with severe sepsis and for patients with difficult-to-treat, multidrug-resistant bacterial pathogens such as *Acinetobacter* and *Pseudomonas* spp. For patients with severe infections associated with respiratory failure and septic shock, combination therapy with an extended spectrum beta-lactam and either an aminoglycoside or a fluoroquinolone is for *P. aeruginosa* bacteremia. A combination of beta-lactam and macrolide for patients with septic shock from bacteremic *Streptococcus pneumoniae* infections.
- Empiric combination therapy should not be administered for more than 3–5 days. De-escalation to the most appropriate single therapy should be performed as soon as the susceptibility profile is known.
- Duration of therapy typically 7–10 days; longer courses may be appropriate in patients who have a slow clinical response, undrainable foci of infection, bacteremia with *S. aureus*; some fungal and viral infections or immunologic deficiencies, including neutropenia.

- Antiviral therapy initiated as early as possible in patients with severe sepsis or septic shock of viral origin.
- Antimicrobial agents should not be used in patients with severe inflammatory states determined to be of noninfectious cause.

Reminder: Starting Antimicrobial Therapy

Before starting antimicrobial therapy, blood cultures PRIOR to antibiotics

- Blood cultures (both aerobic and anaerobic bottles) be obtained before antimicrobial therapy
- 1 blood culture should be drawn percutaneously and 1 drawn through each vascular access device, unless the device was recently (<48 hours) inserted
- IV antibiotics should be given within 3 hours in the Emergency Department
- IV antibiotics should be given within 1 hr in the Intensive Care Unit

Infection Prevention

The following are recommendations for infection control:

- Hand hygiene
- Barrier precautions
- Catheter care
- Head of bed elevation
- Comprehensive oral care with use of subglottic suctioning
- Oral chlorhexidine gluconate and digestive decontamination to reduce ventilator-associated pneumonia (VAP)

Hemodynamic Support and Adjunctive Therapy

The following hemodynamic support and adjunctive therapies for septic shock will be reviewed in detail:

- Fluid therapy
- Vasopressors
- Inotropic therapy
- Administration of blood products

Fluid Therapy

For hemodynamic support, fluid therapy should be initiated in the setting of severe sepsis within 3 hours (Dellinger, et al., 2013).

- Crystalloids are the initial fluid of choice in the resuscitation of severe sepsis and septic shock.

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- Avoid the use of hydroxyethyl starches for fluid resuscitation of severe sepsis and septic shock.
- Albumin in the fluid resuscitation of severe sepsis and septic shock when patients require substantial amounts of crystalloids.
- Initial fluid challenge in patients with sepsis-induced tissue hypoperfusion with suspicion of hypovolemia to achieve a minimum of 30 mL/kg of crystalloids (a portion of this may be albumin equivalent).
- Fluid challenge technique be applied wherein fluid administration is continued as long as there is hemodynamic improvement.

Quick Reminders on Fluid Administration

- Patients will need central line access for fluid hydration and/or vasopressors
- 1st line therapy – fluids, fluids, fluids
- Crystalloid equivalent to colloid
- Initial 1-2 Liters (20mg/kg) crystalloid or 500 mL colloid
- **Careful in CHF patients!**
- Sodium bicarbonate is not recommended in the treatment of shock-related lactic acidosis

Test Yourself

A patient admitted to the ICU with possible septic shock has a blood pressure of 70/40. What should the nurse do first?

- A. Start levophed
- B. Crystalloids
- C. Hydrocortisone
- D. Hespan

The correct answer is: B. Fluids! Crystalloids are the initial fluid of choice in the resuscitation of severe sepsis and septic shock.

Overview of Vasopressors

Norepinephrine - Preferred vasopressor in septic shock. It increases MAP by vasoconstriction. It has a lesser effect on HR and SV compared to other vasopressors.

Vasopressin - Studies have shown improved outcomes when vasopressin is used with norepinephrine.

Phenylephrine - Increases MAP by vasoconstriction. Causes less tachycardia but decreases stroke volume.

Dopamine - Increases MAP and CO by increasing stroke volume and HR. It also increases cardiac oxygen consumption and irritability.

Epinephrine - This medication can be added to the septic shock patient when norepinephrine and dopamine are ineffective in maintaining a MAP>60. It causes tachycardia and hyperlactemia.

Vasopressors

For hemodynamic support in the septic shock patient, vasopressors may be required. All patients requiring vasopressors have an arterial catheter placed as soon as practical if resources are available. Dellinger, et al., (2013), states that:

- Vasopressor therapy is used initially to target a mean arterial pressure (MAP) of 65 mm Hg
- Norepinephrine is the first choice vasopressor
- Epinephrine is used (added to and potentially substituted for norepinephrine) when an additional agent is needed to maintain adequate blood pressure
- Vasopressin can be added to norepinephrine with intent of either raising MAP or decreasing norepinephrine dose; low dose vasopressin not recommended as the single initial vasopressor for treatment of sepsis-induced hypotension and vasopressin doses higher than 0.03-0.04 units/minute should be reserved for salvage therapy (failure to achieve adequate MAP with other vasopressor agents)

Vasoconstrictor agents are used to increase afterload by increasing the systemic vascular resistance (SVR) and improving the patient's blood pressure level.

The Surviving Sepsis Campaign (Dellinger, et al., 2013) made other evidenced based suggestions on other vasopressors: dopamine and phenylephrine.

Dopamine is used as an alternative vasopressor agent to norepinephrine only in highly selected patients such as patients with low risk of tachyarrhythmias and absolute or relative bradycardia.

Phenylephrine is not recommended in the treatment of septic shock except in circumstances where norepinephrine is associated with serious arrhythmias, cardiac output is known to be high and blood pressure persistently low or as salvage therapy when combined inotrope/vasopressor drugs and low dose vasopressin have failed to achieve MAP target.

Low-dose dopamine should not be used for renal protection.

Do NOT use any vasodilators (such as nitroglycerin) with a septic shock patient since the patient is already vasodilated.

Test Yourself

A nursing home patient presents to emergency room with fever, cloudy urine, and chest pain. Vital signs: 104.7degrees F, HR Afib 150's, BP 70/40's. Which of the following is **INAPPROPRIATE**?

- A. Give fluids
- B. Draw cultures and start antibiotics
- C. Place a central line
- D. Give nitroglycerin for chest pain

The correct answer is: D. It would be INAPPROPRIATE to give nitroglycerine for chest pain. Do NOT use any vasodilators (such as nitroglycerin) with a septic shock patient since the patient is already vasodilated.

Inotropic Therapy

Dellinger, et al. (2013) states in their algorithm that patients with a MAP < 65, ScvO₂ < 70%, and Hct > 30%, a trial of dobutamine infusion up to 20 micrograms/kg/min be administered or added to vasopressor (if in use) in the presence of:

- Myocardial dysfunction as suggested by elevated cardiac filling pressures and low cardiac output
- Ongoing signs of hypoperfusion, despite achieving adequate intravascular volume and adequate MAP

(Dellinger, et al., 2013)

Corticosteroids

The recommendation of corticosteroids has been updated based on evidenced based research.

- Do not use intravenous hydrocortisone to treat adult septic shock patients if adequate fluid resuscitation and vasopressor therapy are able to restore hemodynamic stability.
- In the patient when this is not achievable, the Surviving Sepsis Campaign suggest intravenous hydrocortisone alone at a dose of 200 mg per day (Dellinger, et al., 2013).
- In treated patients, hydrocortisone should be tapered when vasopressors are no longer required.
- Corticosteroids should not be administered for the treatment of sepsis in the absence of shock.

It is no longer recommended to use the ACTH stimulation test to identify adults with septic shock who should receive hydrocortisone (Kaufman & Mancebo, 2014; Dellinger, et al., 2013).

Administration of Blood Products

An adequate cardiac output and hemoglobin level are crucial to oxygen transport.

- Blood administration should be considered to augment oxygen transport if the patient's hemoglobin level is critically low
- Hemoglobin levels should have a target of 7–9 g/dL in the absence of tissue hypoperfusion, ischemic coronary artery disease, or acute hemorrhage (Dellinger, et al., 2013)

Supportive Therapy for Severe Sepsis

Besides the hemodynamic support and adjunctive therapy with the use of fluid therapy, vasopressors, inotropic therapy, and the administration of blood products, there are also other supportive therapy for severe sepsis including:

- Mechanical ventilation in patients with sepsis-induced respiratory distress syndrome
- Sedation, analgesia, and neuromuscular blockade in patients with sepsis
- Glucose control

- Renal replacement therapy
- Prophylaxis of deep vein thrombosis
- Stress ulcer prophylaxis
- Nutrition
- Goals of care

Mechanical Ventilation

Supplemental oxygen should be provided to all patients with sepsis and oxygenation should be monitored continuously with pulse oximetry (Schmidy & Mandel, 2014). Adequate pulmonary gas exchange is critical to oxygen transport. The following are guidelines from the Surviving Sepsis Campaign (Dellinger, et al., 2013). Mechanical ventilation in patients with sepsis-induced respiratory distress syndrome should have the following:

- Low tidal volume
- Limitation of inspiratory plateau pressure for acute respiratory distress syndrome (ARDS)
- Application of at least a minimal amount of positive end-expiratory pressure (PEEP) in ARDS
- Higher rather than lower level of PEEP for patients with sepsis-induced moderate or severe ARDS
- Recruitment maneuvers in sepsis patients with severe refractory hypoxemia due to ARDS
- Prone positioning in sepsis-induced ARDS patients with a Pao₂/Fio₂ ratio of ≤ 100 mm Hg
- Head-of-bed elevation in mechanically ventilated patients unless contraindicated
- Conservative fluid strategy for patients with established ARDS who do not have evidence of tissue hypoperfusion

Although prone positioning can help in the optimization of ventilation and perfusion, it can be associated with potentially life-threatening complications, including accidental dislodging of the endotracheal and chest tubes, as well as the development of pressure ulcers (Kleinpell, Aitken & Scorr, 2013).

Sedation, Analgesia, and Neuromuscular Blockade in Patients with Sepsis

When using sedation, analgesia, and neuromuscular blockades in septic patients, it is important for hospitals to have protocols for sedation and weaning of sedation. Some standards from Dellinger et al. (2013) include:

- Minimizing use of either intermittent bolus sedation or continuous infusion sedation targeting specific titration endpoints
- Avoidance of neuromuscular blockers, if possible
- In the septic patient without ARDS, a short course of neuromuscular blocker (no longer than 48 hours) for patients with early ARDS and a Pao₂/Fio₂ < 150 mm Hg

Glucose Control

Glucose control to a target level of 140 to 180 mg/dL is recommended for all critically ill patients. Benefits of glucose control in the critically ill include lower incidences of infection, renal failure, sepsis, Material protected by Copyright

and death. As with other protocols reviewed, the practitioner should follow hospital protocol to approach the blood glucose management in the septic patient.

- Insulin dosing when two consecutive blood glucose levels are > 180 mg/dL. Follow insulin gtt protocols per institution.
- Target an upper blood glucose ≤ 180 mg/dL.

(Dellinger, et al., 2013)

Renal Replacement Therapy

Due to the degree of kidney damage or inability to mobilize fluids in septic patients, it may be necessary for renal replacement therapy. Depending on the hemodynamic stability of the patient, this can be done with continuous veno-venous hemofiltration or intermittent hemodialysis (Dellinger, et al., 2013).

Prophylaxis of Deep Vein Thrombosis

Patients with severe sepsis should receive prophylaxis for deep vein thrombosis. This should be accomplished with daily subcutaneous low-molecular weight heparin (LMWH). Patients with severe sepsis should be treated with a combination of pharmacologic therapy and intermittent pneumatic compression devices whenever possible (Dellinger, et al, 2013).

Septic patients who have a contraindication for heparin use that do not receive pharmacoprophylaxis should receive mechanical prophylactic treatment, such as graduated compression stockings or intermittent compression devices, unless contraindicated. When the risk decreases start pharmacoprophylaxis.

Contraindication for heparin use includes: thrombocytopenia, severe coagulopathy, active bleeding, and recent intracerebral hemorrhage.

Stress Ulcer Prophylaxis

Stress ulcer prophylaxis should be used to prevent upper gastrointestinal bleeding in patients with bleeding risk factors (Dellinger, et al., 2013).

Stress ulcer prophylaxis using H2 blocker or proton pump inhibitor (PPI) should be given to patients with severe sepsis/septic shock who have bleeding risk factors. It is recommended to use the PPI over the H2 receptor antagonists (H2RA).

Patients without bleeding risk factors should not receive prophylaxis.

Nutrition

The critically ill patient should be started on enteral nutritional support therapy within 24 to 48 hours after a diagnosis of septic shock or severe sepsis (Dellinger, et al, 2013).

The type of nutritional supplementation initiated varies according to the cause of shock, and it should be tailored to the individual patient's needs, as indicated by the underlying condition, laboratory data, and treatment.

Low dose feeding can be done (up to 500 calories per day), advancing only if tolerated.

Use IV glucose and enteral nutrition rather than total parental nutrition alone or parenteral nutrition in conjunction with enteral feeding in the first 7 days after a diagnosis of severe sepsis/septic shock.

Oral or enteral (if necessary) feedings can be provided, as tolerated, rather than either complete fasting or provision of only intravenous glucose within the first 48 hours after a diagnosis of severe sepsis/septic shock.

Goals of Care

The psychosocial needs of the patient and family dealing with shock are extremely important. These needs are based on situational, familial, and patient-centered variables. Urden, Stacy & Lough (2012), state that nursing interventions for the psychosocial stress of critical illness include providing information on:

- Patient status
- Explaining procedures and routines
- Supporting the family
- Encouraging the expression of feelings
- Facilitating problem solving and shared decision making
- Individualizing visitation schedules
- Involving the family in the patient's care
- Establishing contacts with necessary resources
- Addressing goals of care: including treatment plans and end-of-life planning (as appropriate), as early as feasible, but within 72 hours of intensive care unit admission (Dellinger, et al., 2013).

Test Yourself

Goals of care are not important in the septic shock patient because sepsis is very common in the ICU setting and most families are aware of the risks.

- A. True
- B. False

The correct answer is: false. Addressing goals of care including treatment plans and end-of-life planning (as appropriate), as early as feasible, but within 72 hrs of intensive care unit admission. The psychosocial needs of the patient and family are important to address throughout providing care for the septic shock patient.

Nursing Management of Shock

The nursing management of a patient in shock is a complex and challenging responsibility. It requires an in-depth understanding of the pathophysiology of the disease and the anticipated effects of each intervention, as well as a solid understanding of the nursing process.

Prevention- Prevention of severe sepsis and septic shock is one of the primary responsibilities of the

nurse in the critical care area. These measures include the identification of patients at risk and reduction of their risk factors, including exposure to invading microorganisms.

Management- Nursing interventions include early identification of sepsis syndrome; administering prescribed fluids, medications, and nutrition; providing comfort and emotional support; and preventing and maintaining surveillance for complications.

Case Study

An 85 year old male presents to the Emergency Department from home c/o >2 days with a cough. Temp 103.2. BP 85/40 (55). P 80. RR 28.

Past Medical History (PMHX): Hypertension (HTN), coronary artery disease (CAD)

Home meds: Lopressor 25mg po bid, ASA 81 mg po daily, Lisinopril 10mg po daily

Clinical presentation of patient:

Neuro – restless and confused

CV – warm and flushed

Resp – dyspnea, bilateral crackles

GU – foley placed with <10ml urine in the first hour

Should you be concerned about sepsis even though the HR is normal in the setting of fever?

Yes. There is presence of infection, high respiratory rate, low blood pressure and fever. The heart rate is low because the patient takes Lopressor.

Case Study

Diagnostics and labs are reported on the patient:

Hct 30%; Hgb 10g/dL; WBC 14.0 X10³/μL; PT 13 sec; PTT 38 sec; INR 7.8

Na 140meq/L; K 3.8meq/L; Cl 108meq/L CO₂ 28mmoles/liter; Cr 1.0mg/dl ; BUN 25mg/dL; Lactate 7mg/dL

Chest x-ray: Bilateral lower lobe pneumonia

According to the Surviving Sepsis Care Bundle, what are the next two steps that should be done for this patient within 3 hours?

**Administer broad spectrum antibiotics
Administer 30 mL/kg crystalloid**

The nurse in the Emergency Room should take the following steps:

1. Blood Cultures X2
2. Antibiotics
3. Fluid
4. Oxygen
5. Tylenol

Conclusion

Sepsis is a serious worldwide healthcare condition that is associated with high mortality ratios despite improvements in the ability to manage infection (Kleinpell, Aitken & Schorr, 2013). This continuing education course reviewed the updated guidelines for the management of sepsis that were recently released that advocate for implementation of care based on evidence-based practice.

Resources

Surviving Sepsis Campaign

<http://www.survivingsepsis.org/Pages/default.aspx>

Sepsis Alliance

<http://www.sepsisalliance.org>

Centers for Disease Control: Sepsis

<http://www.cdc.gov/sepsis/>

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