SEPSIS: A Focus on Fluid Resuscitation, Vasopressors/Inotropes and Antibiotics

Trauma Patient Considerations 2014
Objectives

At the end of this presentation, the participant will be able to:

• Explain the diagnosis of shock and the pathophysiological mechanisms of shock
• Recognize the differences between the fluid resuscitation agents that are available
• Discuss the pharmacology, dosing and adverse effects of the vasoactive agents
• Verbalize the importance of antibiotic initiation, selection and de-escalation
Shock

- Inadequate perfusion of tissues and lack of adequate oxygen delivery to vital organs
- Shock must be treated immediately to prevent multisystem organ failure and death
- Blood pressure = Cardiac output (CO) x Systemic vascular resistance (SVR)
- Patients in shock will have either an inadequate cardiac index (CI) or a low SVR due to arterial vasodilation (rarely both)
- Classification of shock depends on the etiology of the physiologic state

Shock

- Diagnosis is based on clinical, hemodynamic and biochemical signals
  - Systemic arterial hypotension
    - Systolic blood pressure < 90mmHg or mean arterial pressure is less than 70mmHg
    - Along with tachycardia
  - Clinical signs of tissue hypoperfusion
    - Cutaneous
      - Skin that is cold and clammy, with vasoconstriction and cyanosis
    - Renal
      - Urine output <0.5 ml/kg body weight/hour
    - Neurologic
      - Altered mental state
  - Hyperlactatemia
    - Indication of abnormal cellular oxygen metabolism
    - Level is increased to ≥ 1.5 mmol/L

Shock

- Results from four pathophysiological mechanisms
  - Hypovolemia
    - Internal or external fluid losses
  - Cardiogenic factors
    - Acute myocardial infarction, end-stage cardiomyopathy, advanced valvular heart disease, myocarditis, cardiac arrhythmias
  - Obstruction
    - Pulmonary embolism, cardiac tamponade, tension pneumothorax
  - Distributive Factors
    - Severe sepsis or anaphylaxis from the release of inflammatory mediators

## Overview of Shock States

<table>
<thead>
<tr>
<th>Shock States</th>
<th>Examples</th>
<th>Primary Problem</th>
<th>Compensatory Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiogenic</td>
<td>CHF</td>
<td>↓ CO</td>
<td>↑ SVR</td>
</tr>
<tr>
<td>Hypovolemic</td>
<td>Hemorrhage, dehydration</td>
<td>↓ CO</td>
<td>↑ SVR</td>
</tr>
<tr>
<td>Distributive</td>
<td>Sepsis, neurogenic, anaphylaxis</td>
<td>↓ SVR</td>
<td>↑ CO</td>
</tr>
<tr>
<td>Obstructive</td>
<td>Cardiac tamponade</td>
<td>↓ CO</td>
<td>↑ SVR</td>
</tr>
</tbody>
</table>
Initial Management

- VIP rule
  - Ventilate- oxygen administration
  - Infuse- fluid resuscitation
  - Pump- administration of vasoactive drugs

Vincent JL and De Backer D. Circulatory Shock. NEJM. 2013. 369;1726-1734.
FLUIDS
Fluid therapy in critically ill patients

- Total body water (TBW) makes up ~60% of body weight
  - 42 Liters (for a 70 kg person)
  - 3 compartments
    - Intracellular fluid (ICF) (2/3 of TBW)
    - Extracellular fluid (ECF) (1/3 of TBW)
      - Intravascular (IVF) (25%)
      - Interstitial (ISF) (75%)

Fluid therapy in critically ill patients

2 main pressures that govern the movement of fluid within the body

• Osmotic pressure
  – Movement of fluid across a semipermeable membrane occurring when the concentrations of solutes on opposing sides of the membrane are unequal
  – Principal force that maintains the relative size of ECF and ICF compartments
  – Primary solute involved in generating the osmotic gradient is sodium
    • With the ECF, electrolyte concentration and oncotic pressure affect the maintenance of the IVF in relation to the ISF
    • Oncotic pressure gradient develops when there is a difference between the protein concentration of the 2 compartments
    • Oncotic pressure generated by the higher concentrations of albumin and other plasma proteins in the IVF draws fluid from the ISF into the IVF

• Hydrostatic pressure
  – The force exerted by columns of blood as they are driven through the capillaries
  – Force is higher on the arteriolar side and lower on the venous side
  – Causes fluid to move out of the IVF and into the ISF

Fluid therapy in critically ill patients

- Disposition of Administered Fluids
  - D5W contains “free water” and glucose without additional solutes and distribute into all the fluid compartments
    - ~2/3 of administered D5W enters the ICF compartment and 1/3 stays in the ECF
    - Of the volume in the ECF, only 25% stays within the intravascular space
  - Normal saline and lactated Ringers distribute primary into the IVF
    - Primary solute of these fluids is sodium chloride
    - Most of the volume distributes into the ISF compartment
  - Albumin and hetastarch add to the oncotic pressure within the vascular space
    - Contain colloidal proteins or other large macromolecules
    - Maintain the administered volume primarily in the IVF
    - Fluids with supra-physiologic colloid content (Albumin 25%) can expand plasma volume by more than the administered volume because the oncotic pressure generated by the addition of that much protein into the IVF pulls fluids from other compartments

Ideal Fluid

Chemical composition close to extracellular fluid

Predictable and sustained increase in intravascular volume

Metabolized and excreted without accumulation in tissues

Minimal adverse effect profile

# Types of fluids

<table>
<thead>
<tr>
<th>Crystalloids</th>
<th>Colloids</th>
</tr>
</thead>
<tbody>
<tr>
<td>• First line resuscitation fluids</td>
<td>• Volume sparing effect compared to crystalloids</td>
</tr>
<tr>
<td>• Inexpensive</td>
<td>• More effective in expanding intravascular volume</td>
</tr>
<tr>
<td>• Widely available</td>
<td>• Decreased risk of peripheral and pulmonary edema</td>
</tr>
<tr>
<td>• Easily administered</td>
<td>• Greater expense and higher potential for allergic reactions compared to crystalloids</td>
</tr>
<tr>
<td>• Lack of anaphylaxis</td>
<td></td>
</tr>
<tr>
<td>• May cause peripheral and pulmonary edema due to large volumes needed</td>
<td></td>
</tr>
<tr>
<td>• NaCl solution may cause hyperchloremic metabolic acidosis</td>
<td></td>
</tr>
</tbody>
</table>
# Types of Fluids


<table>
<thead>
<tr>
<th>Variable</th>
<th>Human Plasma</th>
<th>Colloids</th>
<th>Crystalloids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osmolarity (mOsm/liter)</td>
<td>291</td>
<td>250</td>
<td>0.9% Saline</td>
</tr>
<tr>
<td>Sodium (mmol/liter)</td>
<td>135–145</td>
<td>148</td>
<td>Compounded Sodium Lactate</td>
</tr>
<tr>
<td>Potassium (mmol/liter)</td>
<td>4.5–5.0</td>
<td>3.0</td>
<td>Balanced Salt Solution</td>
</tr>
<tr>
<td>Calcium (mmol/liter)</td>
<td>2.2–2.6</td>
<td>5.0</td>
<td></td>
</tr>
<tr>
<td>Magnesium (mmol/liter)</td>
<td>0.8–1.0</td>
<td>0.9</td>
<td></td>
</tr>
<tr>
<td>Chloride (mmol/liter)</td>
<td>94–111</td>
<td>128</td>
<td></td>
</tr>
<tr>
<td>Acetate (mmol/liter)</td>
<td>34</td>
<td>24</td>
<td></td>
</tr>
<tr>
<td>Lactate (mmol/liter)</td>
<td>1–2</td>
<td>28</td>
<td></td>
</tr>
<tr>
<td>Malate (mmol/liter)</td>
<td></td>
<td></td>
<td>5</td>
</tr>
<tr>
<td>Gluconate (mmol/liter)</td>
<td></td>
<td></td>
<td>23</td>
</tr>
<tr>
<td>Bicarbonate (mmol/liter)</td>
<td>23–27</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Octanoate (mmol/liter)</td>
<td></td>
<td></td>
<td>6.4</td>
</tr>
</tbody>
</table>
Fluid resuscitation

- One component of the hemodynamic resuscitation strategy
- Targeted at restoring intravascular volume
- Improves microvascular blood flow and increases cardiac output
- Goals during the first 6 hours of resuscitation
  - CVP 8-12 mmHg
  - MAP ≥ 65 mmHg
  - Urine Output ≥ 0.5 mL/kg/hour
  - Superior vena cava oxygenation (Scvo2) 70% or mixed venous oxygen saturation (Svo2) 65%

Fluid Resuscitation

- **Surviving Sepsis Campaign (SSC)**
  - Targeting resuscitation to normalize lactate in patients with elevated lactate levels as a marker of tissue hypoperfusion

- **Large fluid deficits in septic shock**
  - 6-10 L of crystalloid solutions or 2-4 L of colloid solutions
  - Administer via boluses

- **Colloids versus crystalloids**
  - Which is best?
  - SAFE Trial

SAFE Trial

- Saline versus albumin fluid evaluation (SAFE)
- Blinded, randomized controlled trial to examine the safety of albumin
- 6997 ICU patients
- Efficacy of resuscitation
  - 4% albumin versus saline
- Primary Outcome
  - Rate of death at 28 days
- Results
  - No significant difference between albumin and saline with respect to rate of death or development of new organ failure

Surviving Sepsis Guidelines 2013 (as related to fluid administration)

- **Crystalloids as the initial fluid of choice**
- **Against the use of hydroxyethyl starches for fluid resuscitation**

**Fluid Therapy in Severe Sepsis**

- **Albumin in fluid resuscitation when patients require substantial amounts of crystalloids**
- **Initial fluid challenge minimum of 30 ml/kg of crystalloids for tissue hypoperfusion and hypovolemia**

Vasopressors and Inotropes
Vasoactive agents

- Indicated when hypotension is severe or persists despite fluid administration
- Two main hemodynamic goals
  - Provide an adequate perfusion pressure that will ensure blood flow to vital organs
  - Provide an adequate level of oxygen delivery
- Pharmacologic agents
  - Vasopressors- increase blood pressure by causing arteriolar vasoconstriction
  - Inotropes- increase cardiac contractility and cardiac index (CI)

Vasoactive agents

• First-line agents are vasopressors in sepsis
  – Adrenergic agonists
    • Rapid onset of action
    • High potency
    • Short half-life allows easy dose adjustment
  – Norepinephrine is the vasopressor of choice
    • Predominately alpha-adrenergic properties
    • Modest Beta-adrenergic effects to help maintain cardiac output

Vasoactive Drugs

- **Adrenergic agents**
  - Phenylephrine
  - Epinephrine
  - Norepinephrine
  - Dopamine
  - Dobutamine
  - Isoproterenol

- **Nonadrenergic agents**
  - Phosphodiesterase inhibitors (PDI)
    - Milrinone
  - Vasopressin
# Receptor Pharmacology

<table>
<thead>
<tr>
<th>Receptor Type</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpha 1</td>
<td>Vasoconstriction</td>
</tr>
<tr>
<td>Beta 1</td>
<td>Increased heart rate and myocardial contractility</td>
</tr>
<tr>
<td>Beta 2</td>
<td>Bronchial smooth muscle dilation and skeletal muscle dilation</td>
</tr>
<tr>
<td>Dopaminergic 1</td>
<td>Renal, coronary and mesenteric vasodilation</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>Vasoconstriction, sodium + water retention</td>
</tr>
</tbody>
</table>

Hemodynamic Support

\[ \alpha = \uparrow SVR \]
- Distributive shock
- Hypovolemic shock
- Code situation

\[ \beta = \uparrow CO \]
- Cardiogenic shock
- Cardiomyopathy

Dopamine
- Epinephrine
- Norepinephrine
- Phenylephrine
- Vasopressin
  \( V1 = \uparrow SVR \)
- Milrinone
  \[ \text{PDE inhibitor} = \uparrow CO + \downarrow SVR \]
Phenylephrine (NeoSynephrine®)

- **Pharmacology**
  - Selective $\alpha$ 1 agonist = vasoconstriction
  - $\uparrow$ SVR = BP, MAP
  - Least arrhythmogenic of all pressors

- **Dose**
  - Starting dose: 50 mcg/min
  - Max dose: 350 mcg/min

- **Clinical Use**
  - Vasodilatory shock
  - Hypotension associated with tachycardia
Norepinephrine (Levophed®)

• Pharmacology
  – Endogenous catecholamine
  – Potent $\alpha$ agonist with some $\beta$ 1 effects = vasoconstriction
  – $\uparrow$ SVR & MAP plus modest increase in HR ($\uparrow$ CO)

• Dose
  – Starting dose: 5 mcg/min
  – Maximum dose: Contact prescriber for doses greater than 20 mcg/min

• Clinical Use
  – 1st line option for severe hypotension due to septic shock
  – Refractory shock
Epinephrine (Adrenaline®)

• Pharmacology
  – Endogenous catecholamine
  – $\alpha_1$, $\beta_1 > \beta_2$ agonist
  – $\uparrow$ SVR, HR, MAP, CI and stroke volume

• Dose
  – Bolus (cardiac arrest): 1 mg IV every 3-5 min
  – Infusion
    • Starting dose: 1 mcg/min
    • Maximum dose: 10 mcg/min

• Clinical Use
  – 1st line anaphylactic shock
  – 2nd line for septic shock
  – Cardiac arrest, symptomatic bradycardia, heart block
Dopamine (Intropin®)

• **Pharmacology**
  – Norepinephrine and epinephrine precursor
  – Dose dependent effects
    • 2-5 mcg/kg/min (DA-1)
    • 5-10 mcg/kg/min (β-1)
    • > 10 mcg/kg/min (α-1)
  – ↑ cardiac contractility, SVR, HR
  – Dilates mesenteric, renal and cerebral vascular beds

• **Dose**
  – Range: 2 – 20 mcg/kg/min
  – Average initial dose: 5 mcg/kg/min
  – Doses greater than 10 mcg/kg/min associated with tachyarrythmias
Dopamine (Intropin®)

- **Clinical Use**
  - Alternative vasopressor agent to norepinephrine only in highly selected patients
    - Low risk of tachyarrythmias
    - Absolute or relative bradycardia
  - CHF (in combo with other agents)
  - Cardiogenic shock (high dose)

- Low doses are not recommended to maintain renal perfusion
  - ANZICS trial
    - Large-scale randomized, placebo controlled multicenter trial
    - 328 patients with critical illness found that low-dose or “renal-dose” dopamine did not decrease the incidence of renal failure or rule out the need for renal replacement therapy
Vasopressin (Pitressin®)

- **Pharmacology**
  - V2 (renal collecting duct) > V1 (vascular smooth muscle)
  - ↑ sensitivity of blood vessels to constrictor actions of catecholamines = ↓ catecholamine requirement
  - ↑ BP, SVR

- **Dose**
  - Range: 0.01 – 0.04 units/min

- **Clinical Use**
  - Vasodilatory shock
  - Surviving Sepsis Campaign recommends to add vasopressin to norepinephrine to raise the MAP or to decrease norepinephrine requirements
  - Septic shock
    - Vasopressin levels ↓ shock: vasopressin deficiency
Dobutamine

- **Pharmacology**
  - Primary effect as β1 agonist, minimal α1 activity
  - ↑ CO

- **Dose**
  - Average starting dose: 5 mcg/kg/min
  - Maximum dose: 20 mcg/kg/min

- **Clinical Use**
  - Cardiogenic shock
  - 1st line for patients with low cardiac index, decompensated heart failure to increase CO
Isoproterenol

- Pharmacology
  - Pure β-agonist (β1 and β2)
  - ↑ CO by ↑ HR and contractility

- Dose
  - Range: 2-10 mcg/min

- Clinical Use:
  - Increase heart rate in patients refractory to atropine
  - Bradycardia in denervated transplanted heart
  - Bradyarrhythmias, AV block and Torsades
Milrinone

• **Pharmacology**
  – Phosphodiesterase inhibitor (PDI) with inotropic/vasodilator activity
  – ↑ contractility = ↑ CO
  – Vasodilation = ↓ SVR

• **Dose**
  – May require loading dose: 50 mcg/kg over 10 minutes
  – Maintenance dose: 0.25-0.75 mcg/kg/min

• **Clinical Use**
  – Cardiogenic shock, acute exacerbations of CHF
  – Low cardiac output
Adverse Effects

- Cardiovascular
- Metabolic
- Renal and Splanchnic Blood Flow
- Skin Necrosis
- Central Nervous System
- Hematologic
Adverse Effects of Vasopressors

• **Cardiovascular**
  – β-agonists and PDIs can increase heart rate and contractility → increased myocardial oxygen demand
  – Vasoconstrictors (α-agonists and vasopressin) can cause vasoconstriction of coronary vessels and decrease myocardial oxygen supply
  – Sympathomimetic amines that stimulate β-receptors (dobutamine and isoproterenol) can directly cause tachyarrhythmia by increasing myocardial oxygen demand
  – Inotropes and vasodilators may actually decrease blood pressure and induce hypotension

• **Metabolic**
  – Sympathomimetic amines can increase glucose levels through glycolysis and gluconeogenesis
  – β2 agonists can decrease serum potassium levels

Adverse Effects of Vasopressors

- **Skin Necrosis**
  - All vasoconstrictors can cause severe tissue necrosis if they extravasate
  - Vasopressors should be given through a central line

- **Central Nervous System**
  - Sympathomimetic amines can cause CNS stimulation, tremors, restlessness and even confusion and psychosis

- **Renal and Splanchnic Blood Flow**
  - Excessive vasoconstriction may decrease blood flow to the kidneys and the GI tract

- **Hematologic**
  - Sympathomimetic amines can cause an increase in white blood cell counts (stress response)

Clinical Pearls of Vasoactive Agents

• Hypovolemic and septic shock patients should ALWAYS receive volume resuscitation first
  – Inadequate preload will lead to reductions in cardiac output
  – Inotropes will worsen tachyarrhythmia and induce ischemia

• PDIs and sympathomimetic amines with $\beta$ effects that increase CI should be used in caution in patients with severe aortic insufficiency

• PDIs and sympathomimetic amines can cause arrhythmias and myocardial ischemia
  – Cardiac monitoring
  – Electrolyte replacement

Surviving Sepsis Guidelines 2013
(as related to vasopressor therapy)

Target a mean arterial pressure of 65 mmHg

Norepinephrine as the first choice vasopressor

Vasopressors

Vasopressin can be added to norepinephrine to raise the MAP or decrease norepinephrine dose

Epinephrine can be added to norepinephrine when an additional agent is needed

# JMH Nursing Titration Parameters

<table>
<thead>
<tr>
<th>Drug</th>
<th>Standard Concentration</th>
<th>Fluid Restricted Concentration</th>
<th>Starting Dose</th>
<th>Nursing Titration Parameters</th>
<th>Max Dose for Nursing Titration</th>
<th>Titration Goals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dobutamine (Central line)</td>
<td>500 mg/250 mL (2 mg/mL)</td>
<td>1000 mg/mL (4 mg/mL)</td>
<td>5 mcg/kg/min</td>
<td>Prescriber directed dosing</td>
<td>20 mcg/kg/min</td>
<td>CO/CI</td>
</tr>
<tr>
<td>Dopamine (Central line)</td>
<td>400 mg/250 mL (1.6 mg/mL)</td>
<td>800 mg/250 mL (3.2 mg/mL)</td>
<td>5 mcg/kg/min</td>
<td>2.5 mcg/kg/min every 5 min</td>
<td>20 mcg/kg/min</td>
<td>CO/CI, SBP, MAP, HR</td>
</tr>
<tr>
<td>Epinephrine (Central line)</td>
<td>5 mg/250 mL (20 mcg/mL)</td>
<td>15 mg/250 mL (60 mcg/mL)</td>
<td>2 mcg/min</td>
<td>2 mcg/min every 5 min</td>
<td>10 mcg/min</td>
<td>MAP, CPP, HR, SBP, CO/CI</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>1 mg/250 mL (4 mcg/mL)</td>
<td>4 mg/250 mL (16 mcg/mL)</td>
<td>2 mcg/min</td>
<td>Prescriber directed dosing</td>
<td>10 mcg/min</td>
<td>HR, CO/CI</td>
</tr>
<tr>
<td>Milrinone (Central line)</td>
<td>40 mg/200 mL (200 mcg/mL)</td>
<td>40 mg/100 mL (400 mcg/mL)</td>
<td>0.25 mcg/kg/min</td>
<td>Prescriber directed dosing</td>
<td>0.75 mcg/kg/min</td>
<td>CO/CI</td>
</tr>
<tr>
<td>Norepinephrine (Central line)</td>
<td>4 mg/ 250 mL (16 mcg/mL)</td>
<td>16 mg/250 mL (64 mcg/mL)</td>
<td>5 mcg/min</td>
<td>2.5 mcg/min every 5 min</td>
<td>Contact prescriber for doses ≥ 20 mcg/min</td>
<td>MAP SBP, CPP</td>
</tr>
<tr>
<td>Phenylephrine (Central line)</td>
<td>50 mg/250 mL (200 mcg/mL)</td>
<td>100 mg/250 mL (400 mcg/mL)</td>
<td>50 mcg/min</td>
<td>25 mcg/min every 15 min</td>
<td>350 mcg/min</td>
<td>MAP SBP, CPP</td>
</tr>
</tbody>
</table>
ANTIBIOTIC THERAPY
Initial administration

• Administer antibiotics within the first hour of recognition of septic shock and severe sepsis without septic shock
  – Each hour delay in achieving administration of effective antibiotics is associated with an increase in mortality
  – Loading doses should be used depending on the antibiotic
    • Example: Vancomycin

Empiric antibiotics

- Initial empiric anti-infective therapy should include one or more drugs that have activity against likely pathogens
  - Most common hospital acquired infections
    1. Gram positive bacteria
    2. Gram negative and mixed bacterial microorganisms
  - If an infection source is identified then antibiotics should be targeted to the most likely pathogens

- The antimicrobial regimen should be assessed daily
  - Potential de-escalation to prevent the development of resistance
  - Reduce toxicity
  - Reduce costs

Empiric antibiotics

- Empiric combination therapy should not be administered for more than 3–5 days
  - Once the causative agent has been identified de-escalation should be performed
    - Select the most appropriate agent

- Duration of therapy should be between 7-10 days
  - Depends on patient’s response, type of infection and source control

- Source control is also important
  - Effective intervention should be used such as drainage of an abscess

Antibiotic selection

- Multi-drug resistant (MDR) pathogen risk factors
  - Antimicrobial therapy in preceding 90 days
  - Current hospitalization of 5 days or more
  - High frequency of antibiotic resistance in the community or in the specific hospital unit
  - Presence of risk factors for HCAP:
    - Hospitalization for 2 days or more in the preceding 90 days
    - Residence in a nursing home or extended care facility
    - Home infusion therapy (including antibiotics)
    - Chronic dialysis within 30 days
    - Home wound care
    - Family member with multidrug-resistant pathogen
  - Immunosuppressive disease and/or therapy
Antibiotic selection (Continued)

- Empiric therapy for gram positive pathogens
  - Vancomycin
    - Loading dose: 20-25 mg/kg x 1
    - Maintenance dose: 15-20 mg/kg IV BID (normal renal function)

- Empiric therapy for gram negative pathogens
  - Cefepime 2 g IV Q12H (normal renal function)
  - Meropenem 1 g IV Q8H (normal renal function)
Antibiotic selection

• Antifungal risk factors
  – Use of broad-spectrum antibacterial agents
  – Central venous catheters
  – Receipt of parenteral nutrition
  – Receipt of renal replacement therapy by patients in ICUs
  – Neutropenia
  – Use of implantable prosthetic devices
  – Receipt of immunosuppressive agents
    • Glucocorticosteroids, chemotherapeutic agents, and immunomodulators

• Empiric therapy
  – Fluconazole
    • Loading dose: 800 mg IV (12 mg/kg)
    • Maintenance dose: 400 mg IV (6 mg/kg) daily
  – Micafungin 100 mg IV daily
Administer antibiotics within the 1st hour of recognition of sepsis

Initial empiric anti-infective therapy should include drugs with activity against likely pathogens

Antimicrobial regimen should be assessed daily

Duration of therapy 7-10 days depending on infection, patient response and source control

De-escalation to the most appropriate single therapy as soon as the susceptibility profile is known

Empiric therapy should not be administered for more than 3-5 days
Conclusion

• Shock must be treated immediately to prevent multisystem organ failure and death
• Administer fluids to restore intravascular volume and increase cardiac output
  – Crystalloids are the first choice for resuscitation
• Initiate vasoactive agents if fluids fail to maintain perfusion to vital organs
  – Vasoactive selection based on mechanism of shock
• Administer antibiotics within the first hour of recognition of shock
  – Assess antibiotic regimen daily
  – De-escalate antibiotics when culture and sensitivity data is available
• The course evaluation must be completed to obtain contact hours.

• Please be sure to complete the evaluation after completing the Post Test.
References

- Vincent JL and De Backer D. Circulatory Shock. NEJM. 2013. 369;1726-1734.
Gina Riggi, PharmD, BCPS
Clinical Pharmacist Trauma Intensive Care Unit
Jackson Memorial Hospital
June 2014