Shock and Hemodynamic Monitoring

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Shock

- Multiple different strategies for classifying shock, but all forms of shock result in impaired oxygen delivery secondary to either one or both:
  - reduced cardiac output (cardiogenic, septic)
    - OR
  - loss of effective intravascular volume (hypovolemic, neurogenic, anaphylactic, septic).
Septic Shock – Gram Negative

• Gram negative septic shock most studied form a shock
  – Lipopolysaccharide (LPS) in bacterial cell wall binds to LPS binding protein.
  – LPS-LBP complex then binds to cell surface CD14 receptors on monocytes and macrophages.
  – The LPS-LBP-CD14 complex then activates cells via Toll-like receptor-4 (TLR4).
  – TLR4 then “activates” cells which produce a cytokine “cascade” of proinflammatory mediators.
Septic Shock – Gram Negative

• Tumor Necrosis Factor (TNF)
  – First cytokine produced in response to gram negative sepsis
  – Principal mediator for acute response to gram negative bacteria
  – Major source of TNF is from activated macrophages
  – High levels of TNF predict mortality and can cause apoptosis.
Septic Shock – Gram Negative

- Interleukin-1 (IL-1)
  - Levels of IL-1 increase soon after TNF production in gram negative sepsis (second cytokine to be elevated)
  - IL-1 produced by macrophages, neutrophils and endothelial cells
  - IL-1 increases levels of next proinflammatory cytokines in cascade, IL-2 and IL-12.
  - IL-1 does NOT cause apoptosis
Septic Shock – Gram Negative

- Interleukin-10
  - Anti-inflammatory cytokine
  - Inhibits production of IL-12
  - Inhibits T-cell activation
Septic Shock – Gram Positive

• Gram positive sepsis
  – Gram positive cell wall components are also known to be involved in septic response
    – Peptidoglycans
    – Teichoic Acid
    – Likely act in a similar manner as LPS, but less potent on a weight bases.
  – Gram positive bacteria also produce exotoxins
    – Act directly with Class II major histocompatibility complex (MHC) on antigen presenting cells and T cell receptors (TCR).
Septic Shock – Treatment
(Surviving Sepsis Guidelines)

• Resuscitation (over the first 6 hours)
  – Begin resuscitation immediately in patients with hypotension or elevated lactate.
  – Resuscitation goals:
    • CVP 8-12 (for nonintubated patients)
    • CVP 12-15 (for intubated patients)
    • Mean arterial pressure > 65
    • Urine output > 0.5 cc/kg/hr
    • Central venous oxygen saturation > 70% or mixed venous > 65%
Septic Shock – Treatment
(Surviving Sepsis Guidelines)

• Vasopressors
  – Norepinephrine and dopamine are the initial vasopressors of choice
  – Vasopressin (0.03 units/min) can be added to norepinephrine
• Do not use low-dose dopamine for renal protection
• Use Crystalloids or colloids
• Recombinant human activated protein C (Drotrecogin alfa (activated)) is NOT recommended for treatment of severe sepsis and has been withdrawn from the market.
Septic Shock – Treatment (Surviving Sepsis Guidelines)

• Do not use low-dose dopamine for renal protection
• Use Crystalloids or colloids
• Do not increase cardiac index to predetermined supranormal levels
• In patients requiring vasopressors, insert an arterial catheter as soon as practical
Anaphylactic Shock

- Immediate hypersensitivity reaction
- Circulating IgE binds to the antigen causing anaphylaxis
- Antigen-bound IgE then activates FcεRI receptor on mast cells and basophils
- IgE- FcεRI complex then mediates degranulation of mast cells and basophils, leading to the release of inflammatory mediators (such as histamine)
Anaphylactic Shock - Treatment

• Immediate supportive care (protect airway, establish IV access, volume resuscitation, etc.)

• Epinephrine is first-line treatment for severe anaphylaxis
  – Can be given IM in the mid-anterolateral thigh
  – Can also be given in nebulized form for laryngeal edema.

• Second-line medications
  – Antihistamines
  – Glucocorticoids
  – Nebulized bronchial dilators
Neurogenic Shock

- Loss of peripheral vasomotor tone secondary to injury to autonomic pathways within the spinal cord.
- Vasodilation below the level of spinal cord injury
- Unopposed vagal stimulation of heart leads to bradycardia
Neurogenic Shock - Treatment

• Treatment
  – Can be poorly fluid responsive
  – Phenylephrine first line vasopressor
  – Atropine for critical bradycardia
Hypovolemic Shock

- Acute loss in intravascular volume leads to poor venous return to heart and decreased diastolic filling pressures. This leads to inadequate stroke volume and cardiac output.
  - Hemorrhage
  - GI losses
Hypovolemic Shock - Treatment

• Replace fluid losses with isotonic fluids and blood products
• Stop acute intravascular volume loss
  – Control sources of hemorrhage
  – Control GI losses
    • Treatment of infectious diarrhea
    • Relief of bowel obstruction
Cardiogenic Shock

- Cardiac output not adequate for metabolic demands of body
  - Myocardial ischemia and infarction (most common).
  - Cardiac tamponade
  - Spontaneous cardiac dysrhythmias.
Cardiogenic Shock - Treatment

- Treatment
  - Relieve tamponade
  - Treat dysrhythmias as per ACLS protocol
  - Inotropic agents as tolerated to improve stroke volume
  - Decrease afterload if BP allows
  - Intraaortic Balloon Pump
    - Decreases afterload during systole
    - Increases coronary artery perfusions during diastole.
  - Ventricular assist devices
    - Placed in parallel with ventricle
    - Increases cardiac output
    - Used as a “bridge” to heart transplantation
Hemodynamic Monitoring

• All invasive hemodynamic monitoring devices require tubing with a continuous, unobstructed fluid column from tip of catheter to pressure transducer.
  – Pressure transducer needs to be leveled (“zeroed”) at appropriate phlebostatic axis.
    • Transducers positioned to low will artificially raise hemodynamic measurements.
    • Transducers positioned to high will artificially lower hemodynamic measurements.
Hemodynamic Monitoring

• Overdamped or underdamped systems
  – Overdamped system-
    • attenuated systolic peak, narrow pulse pressure, and widened systolic waveform
    • Artificially lower systolic readings
    • Often from bubbles trapped in tubing
  – Underdamped system-
    • sharp systolic peak, increased pulse pressure, and narrow systolic waveform
    • Artificially raises systolic readings
    • Often from stiff tubing that amplifies waveforms as the incoming waveform approaches the resonant frequency of the tubing system
  – Use MEAN arterial pressure readings
Hemodynamic Monitoring
Invasive Arterial

• Invasive Blood Pressure Monitoring
  – Advantages
    • Continuous, instantaneous measurements of SBP, DBP, MAP
    • Ability to easily and repeatedly sample arterial blood
  – Disadvantages
    • SBP increases and DBP decreases the further away the site of measurement is from the aortic root (brachial > radial > femoral)
    • Complications of intraarterial catheters
      – Infection
      – Thrombosis / embolization
      – Arterial injury
Hemodynamic Monitoring

• Invasive Intravascular Volume / Cardiac Function Monitoring
  • Central Venous Catheter
    – Advantages
      » Can measure central venous pressure (CVP) and central venous oxygenation (ScvO2).
      » CVP used by Surviving Sepsis Guidelines for guiding resuscitation
      » Shown to improve outcome when used in Early Goal Directed Therapy in the Treatment of Severe Sepsis and Septic Shock[i] based on values of CVP and continuous ScvO2.

Hemodynamic Monitoring

• Pulmonary artery catheter
  – Can directly measure pulmonary artery pressures, pulmonary artery occlusion pressure, right ventricular cardiac output, and mixed venous oxygenation.
  – Can indirectly calculate SVR, PVR, CI, oxygen delivery, oxygen consumption

• PA Catheter Controversy
  – Prospective randomized trials have failed to show improvement in mortality with the use of PA catheters in patients with:
    • Shock and ARDS[i]
    • High Risk Surgical Patients[ii]
    • Acute Lung Injury[iii]
    • PA Catheter use decreased by 65% from 1993 to 2004.[iv]

Hemodynamic Monitoring

- Arterial Waveform Analysis
  - Stroke volume variation (SVV)
    - \( SVV = \frac{SV_{\text{max}} - SV_{\text{min}}}{SV_{\text{mean}}} \)
    - Uses variations in stroke volume cause by changes in intrathoracic pressure during the respiratory cycle.
    - As intravascular volume decreases, stroke volume variation increases.
    - Has been validated in mechanically ventilated patients only.
    - Arrhythmias can dramatically affect SVV