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 proinflamatory 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 anaphylaxsis
- 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 filing 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.

- [i] Rivers E, et.al. Early Goal-Directed Therapy in the Treatment of Severe Sepsis and Septic Shock. N Engl J Med 2001; 345:1368-77

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]

- [i] Richard C, et.al. Early Use of Pulmonary Artery Catheter and Outcomes in Patients with Shock and Acute Respiratory Distress Syndrome. JAMA. 2003; 290(20):2713-20.
- [ii] Sandham JD, et.al. A Randomized, Controlled Trial of the Use of Pulmonary-Artery Catheters in High-Risk Surgical Patients. N Engl J Med 2003; 348:5-14.
- [iii] Pulmonary-Artery versus Central Venous Catheter to Guide Treatment of Acute Lung Injury. N Engl J Med. 2006;354:2213-24.
- [iv] Wiener RS, et.al. Trends in the Use of the Pulmonary Artery Catheter in the United States, 1993-2004. JAMA 2007; 298(4):423-29.

Hemodynamic Monitoring

- Arterial Waveform Analysis
  - Stroke volume variation (SVV)
    - SVV = (SVmax – SVmin) / SVmean
    - 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