Management of Shock

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Shock

- Serious, life-threatening medical condition where insufficient blood flow reaches the body tissues
- Condition of severe impairment of tissue perfusion
Shock

- Shock occurs at the cellular level
- Cell dysoxia producing a measurable change in organ function
- $\text{VO}_2 < \text{MRO}_2 = \text{SHOCK}$
Treatment

- Identify cause of shock
- Supportive measures
- Treat underlying mechanism
Etiology

- Shock is caused by problems with
  - Pump (cardiogenic and noncardiogenic obstructive)
  - Tubing (distributive)
  - Fluid (hypovolemic)
Etiology

- Cardiogenic Shock
- Myopathic: acute MI, cardiomyopathy
- Mechanical: valvular, papillary muscle
- Arrhythmia
Etiology

• Extracardiac Obstructive Shock
• Pericardial tamponade
• Massive pulmonary embolism
• Tension pneumothorax
Etiology

- Distributive Shock
- Sepsis
- Toxic
- Anaphylaxis
- Neurogenic
- Endocrinologic
Etiology

- Oligemic Shock
- Hemorrhage
- Volume depletion
- Internal sequesteration
**Etiology**

- Endocrine
- Neurogenic
- Drugs
- Sepsis
- Hypovolemic
- Obstructive
- Cardiac

“END SHOC”
Clinical Manifestations
Clinical Manifestations

- Contingent of etiology
- Typically manifest as:
  - Hypotension
  - Tachycardia
  - Weak pulses
  - Tachypnea
  - Oliguria (< 20 mL / hr)
• Eventual manifestations of anaerobic metabolism
  • Metabolic acidosis
  • Elevated lactate
  • Vasoactive substances
Clinical Manifestations

- Oligemic Shock
  - Flat jugular veins
  - Narrow pulse pressure
  - Cold, clammy extremities
Clinical Manifestations

- Cardiogenic Shock
- Evidence for CHF
  - Left heart failure: pulmonary crackles, SOB
  - Right heart failure: JVD, hepatic congestion, lower extremity edema
- Murmur, irregular rhythm
Clinical Manifestations

• Obstructive:
  • Similar to RHF symptoms/ signs
  • Unilateral chest rise/breath sounds with PTX
  • Muffled heart sounds, pulsus paradoxus with tamponade
Clinical Manifestations

- Distributive Shock:
  - Warm extremities
  - Wheezing / hives with anaphylaxis
  - Fever, chills with sepsis
  - Paralysis, spinal injury with spinal shock
Hypovolemic Shock
Hypovolemic Shock

- Hemorrhage, GI losses, dehydration, burns
- Loss of circulating blood volume
- Decreased preload & cardiac output
• Compensation Mechanisms

• Baroreceptors → centrally mediated peripheral vasoconstriction

• Autotransfusion

• Renin-angiotensin-aldosterone
Starling Curve:
End diastolic volume’s influence on stroke volume
Classification of Hemorrhage

- Class I: up to 15% of blood volume
  - Normal pulse and blood pressure
- Class II: up to 30% of blood volume
  - Tachycardia, decreased UOP, anxiety
Classification of Hemorrhage

- Class III: up to 40% blood volume
  - Tachycardia, hypotension, tachypnea, oliguria, anxiety
- Class IV: > 40% blood volume
  - marked tachycardia & hypotension, tachypnea, anuria, confusion, lethargy
• Blood loss: most helpful sign/symptoms
• Severe postural dizziness or postural pulse increment > 30 bpm
• Vomiting/ diarrhea/ ↓ oral intake
• Postural hypotension, sunken eyes, weakness, unclear speech
Labs & Tests

- Hematocrit / hemoglobin
- Type & cross if hemorrhagic
- BUN / creatinine ratio > 20
- PA catheter:
  - CO ↓, PCWP ↓, SVR ↑, CVP ↓
normal echo clip
Treatment

- High flow oxygen
- Adequate IV access
- Restore blood or fluid loss
- Goal: stabilize hemodynamics & maintain tissue perfusion
TREATMENT

• High flow oxygen
• Adequate IV access
• Restore blood or fluid loss
• Goal: stabilize hemodynamics & maintain oxygen delivery
• Insufficient evidence:
  • Early vs delayed resuscitation
  • Large vs small volume resuscitation
  • Hypertonic vs isotonic fluids
Crystalloid vs Colloid

- Crystalloid Benefits:
  - May replace intravascular & interstitial fluid losses
  - Decreased blood viscosity may improve perfusion
  - Inexpensive
• Crystalloid Problems

• Intravascular 1/2 life is 20-30 minutes

• Potential peripheral / pulmonary edema

• Need for large quantities (3x blood loss)
• Colloid Benefits:
  • Longer intravascular 1/2 life (3-4 hours)
  • Less volume required (1x blood loss)
  • Potential for less peripheral edema
• Colloid Problems

• Expensive

• Leaky capillary membranes may worsen edema

• Adverse effects
• Vs albumin:

• anaphylactoid reactions more common with gelatin, hydroxymethyl starch and dextran (12x, 4x, 2x)

• Pruritis, coagulopathy, bleeding more common with hydroxymethyl starch
• ICU patients: albumin vs saline

• mortality, ICU stay, hospital stay, mechanical ventilation, new organ failure unchanged

• albumin assoc with higher mortality in trauma pts, lower mortality in sepsis pts
• Crystalloids in sufficient amounts are as effective as colloids

• Severe intravascular deficits are more rapidly corrected with colloids
• Consider colloid resuscitation

• Severe intravascular deficit prior to arrival of blood for transfusion

• Resuscitation in presence of protein losing conditions (burns)
Cardiogenic Shock
Etiology

- Cardiac Output = SV * HR
- Stroke volume:
  - failure to receive, failure to eject, inadequate volume
- Heart rate:
  - too fast, too slow, ineffective
Diagnosis

- History and physical
- ECG
- Pulmonary catheter
  - $CO \downarrow$, $PCWP \uparrow$, $SVR \uparrow$, $CVP \uparrow$
- Echocardiography
normal TG SAX
poor ef
Acute Coronary Syndrome

• ST segment myocardial infarction
• Non ST segment myocardial infarction
• Unstable Angina
Risk Factors

- Coronary artery disease
- Hypertension
- Diabetes Mellitus
- Tobacco
- Family history
• Perioperative factors
  • Increased oxygen demand
  • Hypercoaguable state
  • Greatest risk of myocardial ischemia is 3rd postoperative day
Pathogenisis

- Coronary artery plaque rupture
- Thrombus formation
- Decreased or absent coronary blood flow
VO2 < MRO2
Oxygen Supply/ Demand Mismatch

- VO2
  - Cardiac output
  - O2 content: \((Hb \times 1.34 \times SpO2)\)
- MRO2
  - HR
  - Afterload
  - Preload
Detection

- History & Physical
- ECG
  - ST changes
  - T wave inversion
  - New left bundle brach block
  - Arrhythmia
ECG slides
• Hypotension

• Signs of heart failure

• PA catheter: ↑ PCWP
Management

VO2 > MRO2
Management

- Core principles
  - Optimize oxygenation, blood volume and hemoglobin
  - Minimize oxygen demand
**Management**

- **VO2** > **MRO2**
  - **CO**
  - Inotropes
  - **Hb*1.34*SpO2**
    - 100% oxygen
    - Red blood cells
  - **Heart rate**
    - Beta1 blocker
    - Calcium channel blocker
  - **Afterload**
    - Nitroprusside
    - Beta2 blockers
    - Anesthetic
  - **Preload**
    - Nitroglycerine
• Immediate interventions
  • 100% O2
  • Nitroglycerine IV
  • Aspirin
  • Morphine
  • Beta blockade: metoprolol, esmolol
- Antiplatelet therapy
- Heparin
- Weigh bleeding risk
• Reperfusion Therapy
• Thrombolytics
• Coronary Artery Bypass Graft
• Percutaneous Intervention (stents)
• Angioplasty
Endocrine
Adrenal Crisis

• Suspect if
  • septic shock with N. meningitidis
  • recent glucocorticoid therapy
  • disseminated TB
  • AIDS
  • refractory hypotension
Diagnosis

- Random serum cortisol
- < 18-20 mcg/dL: give cosyntropin (ACTH) stimulation test
- Dexamethasone will not interfere with cosyntropin stim test
• Give Cosyntropin (ACTH) 250 ug IVP

• Check cortisol @1hr and @2hrs post-ACTH

• If cortisol inc by 7mcg/dl and absolute value >20mcg/dl

  • Secondary hypoadrenalism

  • Stop steroids
• If cortisol does not increase and is <20 mcg/dL

  • Primary hypoadrenalism

  • Stress dose steroids
**Treatment**

- Dexamethasone 10 mg IV if diagnosis is unknown & planning stimulation test
- Hydrocortisone 100 mg IV every 8 hours
  - d/c dexamethasone
- Fluid resuscitation
• During illness or perioperative period, maintenance therapy must be increased

• Hydrocortisone 50 mg IV every 8 hours

• 1st dose preoperatively

• Reduce to maintenance dose 3-4 days after surgery
Anesthetic Implications
• Anesthetic agents have direct cardiovascular depressant effects

• Also inhibit compensatory hemodynamic mechanisms
  • Baroreflex (neuroregulatory)
  • Central catecholamine output
• IV anesthetics have less baroreceptor depression than inhalational agents

• Thiopental, propofol, ketamine depress mechanism most (~ 10 minutes)

• Opioids have minimal effect

• Etomidate has little effect

• Isoflurane < halothane
• Hemorrhage and hypovolemia

• Higher blood concentrations of given dose

• Preferential distribution of cardiac output to brain & heart

• Increased sensitivity to some anesthetics

• Cerebral hypoxia
Pharmacokinetics/pharmacodynamic response of anesthetic agents is variable in shock models

- Plasma concentrations & central clearance of fentanyl increased
- Increased central / peripheral compartment concentration of etomidate
- Propofol: higher concentration & increased brain sensitivity
• Ketamine

• Stimulatory effects when autonomic nervous system is intact

• Direct myocardial depressant
Anesthesia in Hypovolemic Patients

• Important principles
  • Accurate estimation of degree of hypovolemia
  • Reduction of drug doses accordingly
• Propofol is a poor choice for patients in hemorrhagic shock

• Significant cardiovascular effects, even with adequate fluid resuscitation

• Etomidate has most stable hemodynamic platform in hemorrhagic shock
Induction

- Time constraints / continuing hemorrhage prevent restoration of blood volume
- Airway must be secured with minimal or no anesthesia
Maintenance

• All inhalational agents reduce regional and global blood flow
• Isoflurane, desflurane, sevoflurane cause cardiovascular depression/ impair cardiac output in hypovolemia
• Nitrous oxide

• Hypovolemia may unmask myocardial depressant property

• Reduces FIO2

• Pulmonary vasoconstriction
• Inhalational agents should be used only in small concentrations

• < 1 MAC

• Opioid supplementation usually well tolerated
Bibliography

- Clinical Anesthesia: Fifth Edition; Barash; Lippincott, Williams & Walker 2006
- Colloids versus crystalloids for fluid resuscitation in critically ill patients. The Cochrane Database of Systematic Reviews 2004, Issue 4