Use + Share + Adapt

{ Content the copyright holder, author, or law permits you to use, share and adapt. }

- **Public Domain – Government**: Works that are produced by the U.S. Government. (17 USC § 105)
- **Public Domain – Expired**: Works that are no longer protected due to an expired copyright term.
- **Public Domain – Self Dedicated**: Works that a copyright holder has dedicated to the public domain.
- **Creative Commons – Zero Waiver**
- **Creative Commons – Attribution License**
- **Creative Commons – Attribution Share Alike License**
- **Creative Commons – Attribution Noncommercial License**
- **Creative Commons – Attribution Noncommercial Share Alike License**
- **GNU – Free Documentation License**

Make Your Own Assessment

{ Content Open.Michigan believes can be used, shared, and adapted because it is ineligible for copyright. }

- **Public Domain – Ineligible**: Works that are ineligible for copyright protection in the U.S. (17 USC § 102(b)) *laws in your jurisdiction may differ*

{ Content Open.Michigan has used under a Fair Use determination. }

- **Fair Use**: Use of works that is determined to be Fair consistent with the U.S. Copyright Act. (17 USC § 107) *laws in your jurisdiction may differ*

  Our determination **DOES NOT** mean that all uses of this 3rd-party content are Fair Uses and we **DO NOT** guarantee that your use of the content is Fair.

  To use this content you should **do your own independent analysis** to determine whether or not your use will be Fair.
Objectives

- Shock Definition
- Normal Physiology
- Pathophysiology
- Clinical Presentation
- Treatment of Shock Patients
- Types of Shock
Shock Definition

- Inadequate oxygen delivery to meet metabolic demands
- Results in global tissue hypoperfusion and metabolic acidosis
- Shock can occur with a normal blood pressure and hypotension can occur without shock
Normal Physiology

Tissue perfusion is driven by blood pressure

Blood Pressure = CO X PVR

CO – Cardiac Output
PVR – Peripheral Vascular resistance
Normal Physiology

Cardiac Output = SV \times HR

Thus,

\textbf{Blood Pressure} = SV \times \text{HR} \times \text{PVR}

\textit{Blood Pressure} = \text{Stroke Volume} \times \text{Heart Rate} \times \text{Peripheral Vascular Resistance}
Normal Physiology

• Stroke Volume = Volume of blood pumped by the heart during 1 cardiac cycle

Stroke Volume is a function of what factors?
Shock Pathophysiology

Inadequate perfusion → Cell hypoxia → Energy deficit → Lactic acid accumulation and fall in pH → Anaerobic metabolism

Vasoconstriction

Failure of pre-capillary sphincters → Peripheral pooling of blood

Cell membrane dysfunction and failure of 'sodium pump'

Intracellular lysosomes release digestive enzymes

Efflux of potassium

Influx of sodium and water

Toxic substances enter circulation

Capillary endothelium damaged

Destruction, dysfunction, and cell death
Physiologic Compensation

• Heart rate increases as a compensatory response to Shock
• Inadequate systemic oxygen delivery activates autonomic responses to maintain systemic oxygen delivery
  – Sympathetic nervous system
    • NE, epinephrine, dopamine, and cortisol release
      – Causes vasoconstriction, increase in HR, and increase of cardiac contractility (cardiac output)
  – Renin-angiotensin axis
    • Water and sodium conservation and vasoconstriction
    • Increase in blood volume and blood pressure
• Goal is to maintain cerebral and cardiac perfusion
  – Vasoconstriction of splanchnic, musculoskeletal, and renal blood flow
Multi-organ Dysfunction Syndrome (MODS)

• Inability of O₂ to meet metabolic demands despite attempts at physiological compensation leads to triad of lactic acidosis, cardiovascular insufficiency and increased metabolic demands

• Downward spiral continues with progression of physiologic effects:
  – Cardiac depression
  – Respiratory distress
  – Renal failure
  – Disseminated Intravascular Coagulation (DIC)

• Result is end organ failure and death
Clinical Presentation

- **History**
  - Recent illness
  - Fever
  - Chest pain, SOB
  - Abdominal pain
  - Co-morbidities
  - Medications
  - Toxins/Ingestions
  - Recent hospitalization or surgery
  - Baseline mental status

- **Physical examination**
  - Vital Signs
  - CNS – mental status
  - Skin – color, temp, rashes, sores
  - CV – JVD, heart sounds
  - Resp – lung sounds, RR, oxygen sat, ABG
  - GI – abd pain, rigidity, guarding, rebound
  - Renal – urine output
Diagnostic Tools

• Physical exam
  • VS, mental status, skin color, temperature, pulses, etc
  • Clinical signs and symptoms vary depending on the severity of disease and early recognition is key to diagnosis and intervention

• Infectious source

• Basic Labs:
  • CBC
  • Chemistries
  • Lactate
  • Coagulation studies
  • Cultures
  • ABG
Additional Diagnostic Tools

- CT of head/sinuses for occult infections/abscesses
- Lumbar puncture for meningitis/encephalitis
- Wound cultures
- Acute abdominal series
- Abdominal/pelvic CT or US
- Fibrinogen, FDPs, D-dimer if suspicion for DIC is high
Initial Approach to the Patient in Shock

• ABCs
  – Cardiorespiratory monitoring
  – Pulse Oximetry
  – Supplemental oxygen
  – Large bore IV access x 2
  – ABG, labs
  – Foley catheter
  – Vital signs including rectal temperature
Estimating Blood Pressure in shock patients

- Quick method of estimating the blood pressure in patients
- If you palpate a pulse in these regions, then you know the SBP is at least this number:
Shock Treatment Goals

• ABCDE
  – Airway
  – Control work of Breathing
  – Optimize Circulation
  – Adequate oxygen Delivery
  – Monitor End points of resuscitation
Airway

• Assess airway patency and intervene in critically ill patients to decrease work of breathing and support airway control
• Intubation and mechanical ventilation can initially worsen hypotension
  – Sedatives for RSI can lower blood pressure
  – Positive pressure ventilation decreases preload
• May need aggressive volume resuscitation prior to intubation to prevent hemodynamic collapse
Breathing

• Respiratory rate increases with shock to compensate for metabolic acidosis
• Respiratory muscles consume a significant amount of oxygen
• Tachypnea can further exacerbate lactic acidosis
• Mechanical ventilation and sedation will decrease work of breathing and improve overall survival
Circulation

• Isotonic crystalloids (Normal Saline or Lactated Ringers) is optimal first-line fluid

• Titrate to:
  – CVP 8-12 mm Hg if you have central venous access
  – Maintain urine output 0.5 – 1.0 ml/kg/hr (30 ml/hr)
  – Improve heart rate (Goal HR < 100)

• Often requires large amounts of fluids or blood products (>4-6 Liters)

• No survival or outcome benefit from colloids
Oxygen Delivery

• Decrease oxygen demand for patients
  – Provide analgesia and anxiolytics to relax muscles
  – Avoid shivering

• Maintain and increase arterial oxygen saturation
  – Give supplemental oxygen
  – Maintain hemoglobin > 10 g/dL

• Tissue oxygen extraction can be measured with serial lactate levels on an ABG or central venous oxygen saturations if equipment is available
Resuscitation End-Points

• Resuscitation goal is to maximize patient survival and minimize morbidity
• Use objective hemodynamic and physiologic parameters to guide specific therapy
  – i.e. Check vital signs and physiologic markers frequently
• Directed parameters to follow
  – Urine output > 0.5 mL/kg/hr (simplest measure)
  – CVP 8-12 mmHg (if central venous access available)
  – MAP 65 to 90 mmHg
  – Central venous oxygen concentration > 70% (if central venous access available)
What if they don’t get better?

• Search for other causes of persistent hypotension including:
  • Inadequate volume resuscitation
  • Occult bleeding
  • Pneumothorax
  • Cardiac tamponade
  • Adrenal insufficiency
  • Allergic reaction
Types of Shock

- Hypovolemic/Hemorrhagic
- Septic
- Cardiogenic
- Anaphylactic
- Neurogenic
- Obstructive
Hypovolemic Shock

• Hemorrhagic
  • Trauma
  • GI Bleed
  • Massive Hemothysis
  • AAA rupture
  • Ectopic pregnancy or Post-partum bleeding
• Non-hemorrhagic
  • Vomiting/Diarrhea (Gastroenteritis)
  • Small and Large Bowel obstruction
  • Pancreatitis
  • Burns
  • Environmental (Dehydration)
# Classes of Hypovolemic Shock

<table>
<thead>
<tr>
<th>Class</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>BVL</td>
<td>&lt; 15%</td>
<td>15 - 30%</td>
<td>30 - 40%</td>
<td>&gt; 40%</td>
</tr>
<tr>
<td>Amount</td>
<td>750 cc</td>
<td>750 - 1500 cc</td>
<td>1500 - 2000 cc</td>
<td>&gt; 2000 cc</td>
</tr>
<tr>
<td>Pulse</td>
<td>&lt;100</td>
<td>&gt; 100</td>
<td>&gt;120</td>
<td>&gt;140</td>
</tr>
<tr>
<td>BP</td>
<td>No change</td>
<td>Narrowed pulse pressure</td>
<td>Consistent decrease in SBP</td>
<td>Decreased SBP and narrowed pulse pressure or no DBP</td>
</tr>
<tr>
<td>Resp</td>
<td>No change</td>
<td>20-30</td>
<td>30-40</td>
<td>&gt;35</td>
</tr>
<tr>
<td>CNS</td>
<td>No change</td>
<td>Anxiety</td>
<td>Anxious, confused</td>
<td>Confused. Lethargic</td>
</tr>
<tr>
<td>Urine</td>
<td>&gt;30cc per hr</td>
<td>20-30cc per hr</td>
<td>5-15cc per hr</td>
<td>Negligible</td>
</tr>
<tr>
<td>TX</td>
<td>Replace fluid loss</td>
<td>2L NS IV</td>
<td>2 L NS IV, usually requires blood transfusion</td>
<td>Rapid transfusion of blood and NS, requires immediate intervention to stop hemorrhage</td>
</tr>
</tbody>
</table>
Hypovolemic Shock

- ABCs, IV/O2/Monitor
- Establish 2 large bore IVs or a central line
- Crystalloids
  - Normal Saline or Lactate Ringers
  - Up to 3 liters in adults
  - Pediatrics = 20 cc/kg boluses (may need multiple boluses)
- Blood Products (Whole Blood, PRBC’s/FFP/Platelets)
  - O negative or cross matched if type specific is not available
- Control any sources of active bleeding
- Arrange definitive intervention for hemorrhagic shock (Operating Theatre)
# Hypovolemic Shock

- Evaluate response to treatment

<table>
<thead>
<tr>
<th></th>
<th>Rapid Response</th>
<th>Transient Response</th>
<th>No Response</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vitals</strong></td>
<td>Return to normal</td>
<td>Transient improvement with return to previous</td>
<td>Remain Abnormal</td>
</tr>
<tr>
<td><strong>Estimated Blood loss</strong></td>
<td>10-20%</td>
<td>20-40% with ongoing likely</td>
<td>Severe &gt;40%</td>
</tr>
<tr>
<td><strong>Need for more Fluid</strong></td>
<td>Low</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td><strong>Need for Blood</strong></td>
<td>Type and cross</td>
<td>Type specific</td>
<td>O neg</td>
</tr>
<tr>
<td><strong>Need for surgery</strong></td>
<td>Possible</td>
<td>Likely</td>
<td>Highly likely</td>
</tr>
</tbody>
</table>
SIRS

- Systemic Inflammatory Response Syndrome (SIRS)
  - Defined by the presence of two or more of the following:
    - Body temp < 36 °C (97 °F) or > 38 °C (100 °F)
    - Heart Rate > 90 bpm
    - RR > 20 bpm
    - WBC < 4,000 cells/mm³ or > 12,000 cells/mm³ or greater than 10% bands
Sepsis

• Sepsis = Defined as SIRS in response to a confirmed infectious process.

• Septic Shock = Sepsis plus refractory hypotension
  • After bolus of 20-40 mL/Kg patient still has one of the following:
    • SBP < 90 mm Hg
    • MAP < 65 mm Hg
    • Decrease of 40 mm Hg from baseline
Sepsis/SIRS Interface
Sepsis Pathophysiology

Septic Shock – Clinical Manifestations

• Clinical features:
  • Hyperthermia or hypothermia
  • Tachycardia (HR > 100)
  • Wide pulse pressure
  • Low systolic blood pressure (SBP<90)
  • Mental status changes

• Some patients may present in compensated shock with appearance of “normal” blood pressure
Septic Shock - Diagnosis

- Cardiac monitor
- Pulse Oximetry
- Laboratory Tests
  - Complete Blood Count
  - Chemistry panel (electrolytes and BUN/Creatinine
  - Coagulation parameters
  - Liver function tests
  - Lipase
  - Urinalysis
- ABG with lactate measurement
- Cultures: Blood culture x 2, urine culture
- Chest x-ray
- Foley catheter
Septic Shock - Treatment

- 2 large bore IVs
  - NS IVF bolus- 1-2 L wide open (if no contraindications)
- Supplemental oxygen
- Empiric antibiotics as soon as possible and based on suspected source
  - Survival correlates with how quickly the correct antibiotic drug is administered
  - Broad spectrum coverage for both gram positive and gram negative bacteria
- Persistent Hypotension Treatment
  - If no response after 2-3 L IVF, consider starting a vasopressor (Norepinephrine, Dopamine, etc) and titrate to MAP
  - Goal: MAP > 65
  - Consider adrenal insufficiency: Hydrocortisone 100 mg IV
Early Goal Directed Therapy

• Septic Shock Treatment Algorithm, Rivers et al. NEJM, 2001
  • Study that examined use of an early goal directed therapy algorithm to treat patients with septic shock (as defined by refractory hypotension or lactate > 4)
  • 263 patients randomly assigned to either treatment arm or to standard resuscitation arms (130 vs. 133)
  • Control arm treated with standard care and admitted to ICU
  • Treatment arm followed protocol in ED for 6 hours and then admitted to ICU

• Findings/Outcome
  • Treatment group received more fluids compared to control group (5 L vs. 3.5 L)
  • 3.8 days less in hospital for treatment group
  • 2 fold less cardiopulmonary complications for treatment group
  • Relative reduction in mortality of 34.4%
Early Goal Directed Therapy

Anaphylactic Shock

- **Anaphylaxis**
  - Severe systemic hypersensitivity reaction
  - Characterized by multisystem involvement
  - IgE mediated

- **Anaphylactoid reaction**
  - Clinically indistinguishable from anaphylaxis
  - Does not require a sensitizing exposure
  - Not IgE mediated

Justin Beck, [Flickr](https://www.flickr.com)
Anaphylactic Shock – Clinical Manifestations

• Clinical Spectrum
  – First Symptoms:
    • Pruritus
    • Flushing
    • Urticaria

  – Progression:
    • Throat fullness
    • Anxiety
    • Chest tightness
    • Shortness of breath
    • Lightheadedness

  – Severe Symptoms:
    • Altered mental status
    • Respiratory distress
    • Circulatory collapse

• Risk factors for fatal anaphylaxis
  – Poorly controlled asthma
  – Previous anaphylaxis

• Reoccurrence rates
  – 40-60% for insect stings
  – 20-40% for radiocontrast agents
  – 10-20% for penicillin

• Most common causes
  – Antibiotics
  – Insects
  – Food
Anaphylactic Shock - Diagnosis

- Clinical diagnosis
  - Defined by airway compromise, hypotension, or involvement of cutaneous, respiratory, or GI systems
- Mild, localized urticaria can progress to full anaphylaxis
- Symptoms usually begin within 60 minutes of exposure
- Faster onset of symptoms = more severe reaction
- Biphasic presentation occurs in up to 20% of patients
  - Symptoms will return 3-4 hours after initial reaction has cleared

- Look for exposure to drug, food, or insect
- Labs have no role
Anaphylactic Shock - Treatment

• ABC’s
  – Respiratory compromise require immediate intubation
• IV, Cardiac Monitor, Pulse Oximetry
• IVF’s, Oxygen
• Epinephrine for Anaphylaxis/Shock
• Second line Therapies
  – Corticosteroids
  – H1 and H2 blockers
Anaphylactic Shock - Treatment

• Epinephrine
  – 0.3 mg IM or SC of 1:1000 dilution for anaphylaxis
  – Repeat every 5-10 min as needed
  – Caution if patient on beta blockers due to unopposed alpha stimulation and resultant severe hypertension
  – For CV collapse (i.e. cardiac arrest), 1 mg IV of 1:10,000 (same as ACLS Dose)
  – If refractory hypotension and shock, start IV drip
Anaphylactic Shock - Treatment

• Corticosteroids
  – Methylprednisolone 125 mg IV
  – Prednisone 60 mg PO

• Antihistamines
  – H1 blocker- Diphenhydramine 25-50 mg IV
  – H2 blocker- Ranitidine 50 mg IV

• Bronchodilators
  – Albuterol nebulizer
  – Atrovent nebulizer
  – Magnesium sulfate 2 g IV over 20 minutes
Cardiogenic Shock

- Defined as:
  - SBP < 90 mmHg
  - Cardiac Index < 2.2 L/m/m²
  - Pulmonary Capillary Wedge Pressure (PCWP) > 18 mmHg

- Mortality prior to reperfusion therapy = 50-80%

- Signs:
  - Cool, mottled skin
  - Tachypnea
  - Pulmonary vascular congestion
  - Hypotension
  - Altered mental status
  - Narrowed pulse pressure
  - Rales, murmur

Patrick J. Lynch, [Wikimedia Commons](https://commons.wikimedia.org/wiki/File:Cardiogenic_Shock_Heart.jpg)
Cardiogenic Shock

• Cardiogenic Shock Etiology
  – Acute Myocardial Infarction (AMI)
  – Sepsis
  – Myocarditis
  – Myocardial Contusion
  – Acute Aortic Insufficiency
  – Aortic of Mitral Stenosis
  – Hypertrophic Cardiomyopathy
Cardiogenic Shock

• Often results from myocardial ischemia with loss of left ventricular (LV) function
  – Clinical shock ensues after loss of 40% LV
• Resulting cardiac output reduction leads to lactic acidosis and hypoxia
• Stroke volume is reduced
  – Tachycardia develops as compensation for decreased SV to maintain cardiac output
  – Ischemia and infarction worsens creating downward spiral
Cardiogenic Shock - Diagnosis

• EKG
• CXR
• Laboratory:
  – Complete Blood Count
  – Chemistry Panel (Electrolytes, BUN/CR)
  – Cardiac enzymes (Myoglobin, CK, CK-MB, Troponin)
  – Coagulation studies
• Echocardiogram
Cardiogenic Shock - Treatment

• **Treatment Goals**
  – Airway control (if necessary)
  – Improving myocardial pump function
  – Reperfusion (if available)
  – Preventing further myocardial damage

• **Monitoring**
  – Cardiac monitor
  – Pulse Oximetry

• Supplemental oxygen, IV access

• Intubation decreases preload and causes hypotension
  – May need to give fluid bolus to compensate if intubating
Cardiogenic Shock - Treatment

• AMI
  – Aspirin, Beta-blocker, Morphine, Heparin
  – Nitroglycerin for pain control (avoid in inferior MI)
  – If no pulmonary edema, may try IV fluid for BP support
  – If pulmonary edema
    • Dopamine – will ↑ HR and thus cardiac work
    • Dobutamine – May drop blood pressure due to peripheral vasodilatation
      • Combination therapy may be more effective
  – Reperfusion Therapy (if STEMI and available in the clinical setting)
    • Cardiac catheterization with intervention
    • Thrombolytics
Neurogenic Shock

- Results from spinal cord injury with shock lasting from 1-3 weeks
- Loss of sympathetic tone and resultant unopposed vagal tone
- Decreased vasomotor tone
- Results in hypotension and bradycardia
- Patients may remain alert, warm, and dry despite the hypotension
- Spinal shock
  - Temporary loss of spinal reflex activity below a total or near total spinal cord injury
  - Not the same as neurogenic shock, the terms are not interchangeable
Neurogenic Shock- Treatment

• A,B,Cs
  – Remember C-spine precautions
  – High C-spine injuries require mechanical ventilation
    • C3-C4-C5 – Phrenic Nerve innervation

• Fluid resuscitation
  – Keep MAP at 85-90 mm Hg for first 7 days
  – Minimize secondary cord injury
  – If crystalloid is insufficient use vasopressors

• Search for other causes of hypotension
  – Often, SCI results from trauma = r/o Hemorrhagic Shock

• For bradycardia
  – Atropine
  – Pacemaker
Obstructive Shock

- Tension Pneumothorax
- Cardiac Tamponade
- Pulmonary Embolism
- Severe Aortic Stenosis
Obstructive Shock

- Tension pneumothorax
  - Air trapped in pleural space with 1 way valve, air/pressure builds up
  - Mediastinum shifted impeding venous return
  - Chest pain, SOB, decreased breath sounds
  - No tests needed/clinical diagnosis
  - Treatment: Needle decompression followed by chest tube

Source:
www.meddean.luc.edu/lumenMedEd/medicine/pulmonar/cxr/pneumo1.htm
Obstructive Shock

• Cardiac tamponade
  • Blood in pericardial sac prevents venous return to and contraction of heart
  • Etiology: Trauma, Pericarditis, MI
  • Beck’s triad:
    • Hypotension, muffled heart sounds, JVD
  • Diagnosis: Large heart CXR, echo
  • Treatment: Pericardiocentesis
Obstructive Shock

- Pulmonary embolism
  - Virchow triad:
    • Hypercoagulable
    • Venous injury
    • Venostasis
  - Signs:
    • Tachypnea
    • Tachycardia
    • Hypoxia
  - Low risk: D-dimer
  - Higher risk: CT chest or VQ scan
  - Treatment: Heparin
    • Consider thrombolytics if shock from PE

Hellerhoff, Wikimedia Commons
Obstructive Shock

• Aortic stenosis
  • Resistance to systolic ejection causes decreased cardiac function
  • Chest pain with syncope
  • Systolic ejection murmur
  • Definitive diagnosis with echo
  • Vasodilators (NTG) will drop pressure
  • Treatment: Aortic valve surgery
Which pressor should I choose?

- **Hypovolemic shock**
  - Fluids and Blood

- **Cardiogenic shock**
  - Dobutamine - B1 agonist
    - Increases squeeze and heart rate

- **Neurogenic shock**
  - Fluids, phenylephrine, Levophed, look for another type of shock if it is persistent

- **Anaphylactic shock**
  - Fluids and epinephrine

- **Septic shock**
  - Neosyphrine - alpha agonist
    - Increases SVR by arteriolar constriction
  - Norepinephrine/Levophed - alpha and beta agonists

- **Dopamine**
  - Low Dose - increases renal blood supply
  - Medium Dose - beta effects (increases heart rate and squeeze)
  - High Dose - alpha effects (arteriolar constriction)
Questions?

Dkscully, Flickr

(c) BY-NC-SA