Define

SHOCK

: a state where tissue perfusion to vital organs is inadequate.
In all shock states, the ultimate result is inadequate tissue perfusion, leading to a decreased delivery of oxygen and nutrients to cells.... and, therefore, cell energy.
Clinical recognition of shock

- **Symptoms**
  - dizziness, nausea, visual changes, thirst, dyspnea

- **Signs**
  - cold clammy skin, pallor, confusion, agitation, diaphoresis, weak thready pulse, obvious injury
Compensatory stages of shock

- Sympathetic nervous system
- Renin-angiotensin system
- Pituitary-antidiuretic hormone release
- Shunting from less critical areas to brain and heart
Progressive decompensation

- Failure of compensatory mechanisms in
  - Bowel
  - CNS & autonomic
  - Heart
  - Kidneys
  - Lungs
  - Liver

What will we see?
Shock diagnosis

- Clinical examination

- Diagnostics:
  - CXR
  - CBC
  - blood chemistry
  - EKG
  - ABG
  - vital signs
Monitoring organ perfusion in shock states

- Base deficit
- Blood lactate levels

Normalization of these markers are the end point goals of resuscitation!
Base Deficit

- Reflects severity of shock, the oxygen debt, changes in oxygen delivery, and the adequacy of fluid resuscitation.

- 2-5 mmol/L suggests mild shock
- 6-14 mmol/L indicates moderate shock
- > 14 mmol/L is a sign of severe shock
The base deficit reflects the likelihood of multiple organ failure and survival.

An admission base deficit in excess of 5-8 mmol/L correlates with increased mortality.
Lactate Levels

- Blood lactate levels correlate with other signs of hypoperfusion.
- Normal lactate levels are 0.5-1.5 mmol/L.
- >5 mmol/L indicate significant lactic acidosis.
Lactate Levels

- Failure to clear lactate within 24 hours after circulatory shock is a predictor of increased mortality.
Types of shock

- Hemorrhagic/hypovolemic
- Cardiogenic
- Neurogenic
- Septic
- Anaphylactic
HEMORRHAGIC / HYPOVOLEMIC SHOCK

- Loss of intravascular volume –
Causes:

- Hemorrhage
  - Low filling pressures lead to decreased cardiac output
  - Low hemoglobin levels lead to a reduction in tissue oxygen delivery
HEMORRHAGIC/HYPOVOLEMIC SHOCK

- **Causes:**
  - Hypovolemia
    - Severe dehydration
    - Secondary to fluid redistribution

i.e. burns, surgery (3rd spacing)
Symptoms of hypovolemic shock

- Anxiety, irritability, decreased level of consciousness, tachycardia, hypotension, tachypnea.

Hemodynamics:
- Decreased CVP, PAP, PCWP, CO
- Increased SVR
HEMORRHAGIC/HYPOVOLEMIC SHOCK

- Any major volume loss causes compensatory mechanisms to kick-in to maintain BP and tissue perfusion.

These include.....
Vasoconstrictor Response:
sympathetic nervous system triggers
adrenal medulla to secrete –
**epinephrine** and
**norepinephrine**

increasing peripheral vascular resistance and reducing size of vascular department.
Kidneys: decreased blood flow through kidney causes decreased glomerular filtration = decreased urine.

When blood pressure decreases, kidney produces renin.
Low BP?

Kidney secretes Renin
Renin cleaves angiotensinogen →
angiotensin I →

= angiotensin II

Angiotensin II – \textit{vasoconstrictor}
Angiotensin II stimulates adrenal cortex to produce aldosterone: conserves water and sodium and decreases secretion of water.

Decreased blood volume also stimulates hypothalamus, which regulates ADH (antidiuretic hormone/Vasopressin) decreasing amount of urinary output.
Early and appropriate resuscitation may avert damage to individual organs as adaptive mechanisms act to preserve the organism.
HEMORRHAGIC/HYPOVOLEMIC SHOCK

- 1st and foremost...

- Identify underlying cause of bleeding or hypovolemia and stop it.

  Hold pressure over source of external bleeding.

- Head down position to move blood out of legs and into thorax and head
HEMORRHAGIC/HYPOVOLEMIC SHOCK

FILL ‘ER UP!

“Heart rate, systemic blood pressure, pulse pressure, respiratory rate, urine output, and mental status remain the best available early clinical indicators of the severity of hemorrhagic shock.”
The response of the pulse and blood pressure to initial fluid therapy also aids in the assessment of hypovolemia.

- Giving LR 2,000 ml over 15 minutes in adults, or 20 ml/kg in children, should normalize vital signs if hemorrhage is mild (10-20%)
A transient improvement after fluid infusion suggests a 20-40% decrease in circulating volume or continuing blood loss – more crystalloids and possibly blood transfusion are required in these patients.
If the VS do NOT respond to this initial fluid resuscitation, there has probably been severe (>40%) blood loss – replace by rapid infusion of crystalloids, colloids, and blood.

We also draw labs and transfuse based on the H&H levels.
# Fluid resuscitation

<table>
<thead>
<tr>
<th></th>
<th>Rapid response</th>
<th>Transient response</th>
<th>Minimal or No response</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vital Signs</strong></td>
<td>Return to normal</td>
<td>Transient improvement</td>
<td>Remain abnormal</td>
</tr>
<tr>
<td><strong>Estimated blood loss</strong></td>
<td>Minimal 10-20%</td>
<td>Moderate, ongoing 20-40%</td>
<td>Severe &gt;40%</td>
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<tr>
<td><strong>Need for crystalloid</strong></td>
<td>Low</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td><strong>Need for blood</strong></td>
<td>Low</td>
<td>Moderate to high</td>
<td>Immediate</td>
</tr>
<tr>
<td><strong>Blood preparation</strong></td>
<td>Type &amp; cross</td>
<td>Type specific</td>
<td>Not type-specific</td>
</tr>
<tr>
<td><strong>Need for surgery</strong></td>
<td>Possible</td>
<td>Likely</td>
<td>Highly likely</td>
</tr>
<tr>
<td><strong>Early surgeon presence</strong></td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
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</table>

*Barash*
## Massive transfusion guideline

<table>
<thead>
<tr>
<th>PRBCS</th>
<th>Plasma</th>
<th>Platelets</th>
<th>Cryoprecipitate</th>
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<td>6 units</td>
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CARDIOGENIC SHOCK

- pump failure, or
- obstruction of cardiac filling
  - increased venous return
  - very decreased cardiac output
  - increased afterload (SVR)
CARDIOGENIC SHOCK

- Pump failure:
  - myocardial infarction
  - dysrhythmias
  - ventricular septal defect
  - cardiomyopathies
  - valve disorders
  - pulmonary hypertension
CARDIOGENIC SHOCK

- Hallmark signs in cardiogenic shock characterized by:
  - decreased urine output
  - altered mentation
  - hypotension
CARDIOGENIC SHOCK

- Hemodynamics:
  - Increased: CVP, PCWP, SVR
  - Decreased: CO
Early signs and symptoms are due to strong sympathetic stimulation:
- dilates brain vessels and coronary arteries
- clamps all other arteries
- increased heart rate and blood pressure

BUT
Perfusion pressure low (confused, clammy, decreased urinary output).
Late changes in cardiogenic shock due to:

- MDF (myocardial depressive factor)
- blood pooling
- platelet aggregation
- released toxins
- anaerobic metabolism and lactic acidosis
CARDIOGENIC SHOCK

**Late signs and symptoms:**

- Tachycardia and arrhythmias
- Absent/decreased peripheral pulses
- Cool and clammy skin
- Gallop S3 and S4
- Pulmonary crackles and edema
- Distended jugular veins
Treatment:

- **Inotropes** – increased contractility and decreases SVR (afterload).
  i.e.: dobutamine

- **Milrinone** – phosphodiesterase inhibitor: increases cAMP and calcium

- **Diuretics** – decrease preload and afterload
  i.e.: furosemide
CARDIOGENIC SHOCK

- Oxygen
- Decrease myocardial oxygen demand
- Intra-aortic balloon pump
- Antiarrhythmics
Obstruction of cardiac filling:
- cardiac tamponade
- tension pneumothorax
- massive pulmonary embolism
CARDIOGENIC SHOCK

- Hemodynamics
  - Increased CVP
  - Decreased CO

- Treatment of obstructive cardiogenic shock
  - Get rid of the obstruction
NEUROGENIC SHOCK

- Caused by the sudden loss of the autonomic nervous system signals to the smooth muscle in vessel walls.
Loss of background sympathetic stimulation. Blood vessels suddenly relax resulting in a sudden decrease in peripheral vascular resistance and hypotension.

- decreased preload, CVP
- very decreased afterload
- decreased cardiac output
NEUROGENIC SHOCK

- This can result from severe damage to the
  - Brain (CNS)
  - Spinal cord
    - spinal anesthesia
    - spinal cord injury
S/S of neurogenic shock:
- Profound hypotension
- Bradycardia
- Restlessness, confusion
- Warm, dry extremities – no sweating
- Peripheral vasodilation
- Venous pooling
- Oliguria
NEUROGENIC SHOCK

Treatment:
- Support hemodynamics until neurologic status stabilized
- Large volumes of fluid may be needed to restore normal hemodynamics
- Dopamine
- Vasopressors i.e. epinephrine
- Atropine
Series of events triggered not only by an invading microbe, but also to a larger extent by the substances released from the microbes and the body’s defense against this invasion.
SEPSIS - SEPTIC SHOCK

- SIRS – Systemic Inflammatory Response
  - At least three of the following criteria must be present –
    - Tachycardia (HR > 90 bpm)
    - Tachypnea (or requirement of mechanical vent)
    - Hyper- or hypothermia (< 36 or > 38 degrees C)
    - WBC < 4,000 or > 12,000
SEPSIS - SEPTIC SHOCK

- #1 cause is gram-negative bacteria (Klebsiella, pseudomonas, E.Coli, proteus).
  - Can also be from gram + cocci.
  - 34% from urinary tract infection.

** Overwhelming occurrence from overwhelming infection **
Gram-negative infection releases **endotoxins**. An endotoxin in the blood causes some cells to release **histamine** - a powerful **vasodilator** - this dilates all blood vessels **especially** capillaries.
SEPSIS - SEPTIC SHOCK

- You have the right amount of blood but vessels are so dilated the blood is pooled.

- Microbes or endotoxins trigger the release of:
  - endorphins
  - prostaglandins
  - vasoactive substances:
    - histamine and bradykinin.
First stage of SEPTIC SHOCK

- **Warm Stage / Hyperdynamic Stage**
  - 3 minutes to 12-16 hours long.
    (Almost never picked up)
S/S of 1st stage of SEPTIC SHOCK

- S/S of warm stage of septic shock
  - normal temperature
  - great cardiac output 9-11 liters/minute
    (endotoxins work on myocardium to increase heart contractility 50%)
  - pulse bounding, good blood pressure
  - hyperventilation: endotoxins work on the medulla oblongata to increase respiratory rate
    (not panting but subtle: respiration rate increases to 26-30)
S/S of 1\textsuperscript{ST} stage of SEPTIC SHOCK

- ABG’s are excellent (because respiratory rate is increased)
- Kidneys: vessels vasodilate; Bowman’s capsule filtering increased amounts of blood; patient’s have a great urine output.
- Confused, though, because endotoxins work on brain.
Treatment of 1st stage of SEPTIC SHOCK

- Treatment must be done in WARM STAGE

- FLUID AND ANTIBIOTICS!!!!!
- Give 200cc IV fluid per hour so when patient goes into cold stage, body won’t suffer from low BP
- Give Dopamine: vasoconstricts capillaries so they can’t pool blood
- Find infection and get rid of it.
**COLD STAGE**

All blood vessels vasodilate and pools/stagnates in capillaries.

Pre-capillary sphincter relaxes but not post-capillary – the blood dumps in but not out.
S/S of 2\textsuperscript{nd} stage of SEPTIC SHOCK

- hypotension
- decreased pulse
- cold, mottled skin
- no urinary output
- ischemia, arrhythmias, acidosis, decreased stroke volume and cardiac output.
When compensatory mechanisms fail…

Mortality rate in septic shock is 80-90%.
ANAPHYLACTIC SHOCK

Allergic response triggers mast cells to release immunological mediators (i.e.: histamine, prostaglandins, leukotrienes, etc.) causing:

- systemic vasodilation
- edema of bronchial mucosa, bronchoconstriction, and dyspnea
- angioedema
Anaphylactic shock can lead to death in a matter of minutes if left untreated.
Common causes of anaphylactic shock:

- **Food**: Peanuts, walnuts, cashews, shellfish, fish, milk, and eggs
- **Medications**: NSAIDS, IV contrast, blood products
- **Anesthetics**: NDMR, latex, antibiotics, colloids, induction agents, and local anesthetics
- **Insect stings**: bees, wasps, hornets
ANAPHYLACTIC SHOCK

S/S:
- respiratory distress
- hypotension
- urticaria
- flushed appearance
- angioedema: swelling of lips, face, neck, and throat
- Anxiety
- Tachycardia, hypotension
ANAPHYLACTIC SHOCK

- Hemodynamics
  - Decreased CVP
  - Decreased PCWP
  - Decreased SVR
ANAPHYLACTIC SHOCK

Treatment:

Stop administration or decrease absorption of offending agent if possible.

Give:

- Epinephrine
- Antihistamines: H1 and H2 blockers
- Racemic Epinephrine for laryngeal edema or laryngospasm
- Airway control: endotracheal intubation or tracheostomy
- Hydrocortisone 100-150 mg IV q 6 hrs. - stabilize cell membrane for persistent symptoms
- Fluid resuscitation
FAILURE OF COMPENSATORY MECHANISMS in SHOCK

- **Myocardial**: ultimately results in:
  - increased myocardial ischemia
  - decreased contractility (acidosis, myocardial depressive factor)
  - increased dysrhythmias
Cerebral ischemia:

- initially results in stimulation of the SNS, if prolonged there will be a loss of sympathetic influences.
FAILURE OF COMPENSATORY MECHANISMS in SHOCK

Kidney

- Decreased blood pressure → kidney tubules necrotic → acute tubular necrosis (ATN)
FAILURE OF COMPENSATORY MECHANISMS in SHOCK

**Microcirculation:**

Thrombosis of small vessels secondary to:

- Blood stagnating within the capillary bed
- Acidosis and catecholamines increases platelet aggregation
- Damage to endothelial lining of cells
Acidosis: worsened by:

- Increased production of lactic acids with poor tissue perfusion
- Decreased renal function
- Decreased respiratory function, hypoxia, and hypercapnia (more acids)
Complications of shock

**HYPOTENSION**
(cardiac output low)

**LUNGS**
kills Type 2 alveolar cells ➔ no surfactant ➔ ARDS

**KIDNEYS**
kidney tubules necrotic ➔ ATN

**PANCREAS**
pancreas releases MDF (myocardial depressant factor) releases harmful enzymes
Complications of shock

- **ARDS**: non-cardiogenic pulmonary edema. Increased capillary permeability and interstitial edema. Due to destruction of Type 2 alveolar cells and decreased surfactant production.

- Findings: dyspnea: often severe and sudden
  - Hallmark: decreased PaO2 (< 50 on FiO2 > 40%)
  - Bilateral diffuse pulmonary infiltrates; decreased lung compliance
  - PCWP > 18 mm Hg

- **Treatment**: mechanical ventilation with PEEP
- Close monitoring of fluid status
- Antibiotics
Complications of shock

- **Acute Tubular Necrosis (ATN):** injury may be from ischemia due to renal hypoperfusion or to toxins as seen with sepsis.
  - Treatment: diuretics; improve renal perfusion by fluids or by increasing cardiac output.

- **Disseminated Intravascular Coagulation (DIC):** Consumptive coagulopathy, microembolization
  - Findings: prolonged bleeding, oozing.
    - Decreased platelets, PT/PTT, used all up in clotting
    - Increased FSP (fibrin split products), released with breakdown of clot.
  Microembolization may lead to multisystem organ failure.
Treatment of Shock

- Initiation of Therapy
  - Airway, Breathing, Circulation
  - Ensure oxygenation and CO$_2$ Removal
  - Reconstitution of Blood Volume

- Evaluate for circulatory disturbances
  - Focused treatment on circulatory abnormalities
Treatment of Shock

- oxygen delivery:
  - intubate/ventilate early
Treatment of Shock

- **fluid replacement**: “replace what you lose”
  - Crystalloids: NS, LR, hypertonic saline
  - Colloids: albumin, synthetic colloids
    - Non-oxygen carrying colloids
  - Blood products
  - Blood substitutes – hemoglobin based oxygen carriers
Treatment of Shock

- **vasopressors:**
  - Neosynephrine: vasoconstrictor with no chronotropic side effects
  - Levophed: alpha stimulator
Treatment of Shock

- **inotropic drugs/IABP**
  - Dopamine 3-20 mcg/kg/min
  - Dobutamine 2.5-40 mcg/kg/min

- **antibiotics / drainage**

- **steroids**: stabilize cell membranes
Treatment of shock states

- Evaluate for different contributing factors.
- Initiate therapy with fluid replacement and evaluate clinical response.
- Initiate invasive monitoring if inappropriate response to volume replacement occurs.
Bottom line…

Expedient and aggressive approach to the patient in shock.
Thank you,

PANA
The End