SHOCK STATES

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Define

SHOCK

: a state where tissue perfusion to vital organs is inadequate.

Shock state

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

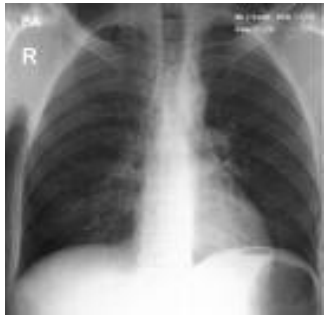
Progressive decompensation

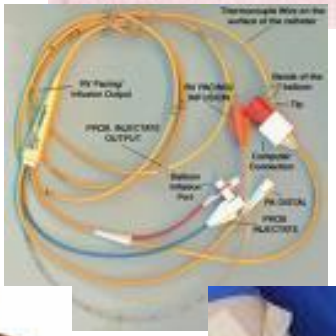
- Failure of compensatory mechanisms in
  - Bowel
  - CNS & autonomic
  - Heart
  - Kidneys
  - Lungs
  - Liver

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

Base Deficit

- 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

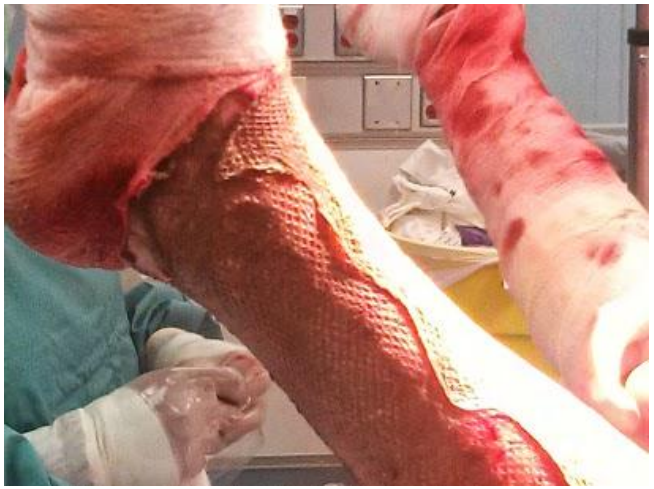
HEMORRHAGIC / HYPOVOLEMIC SHOCK

- Loss of intravascular volume –

HEMORRHAGIC/HYPOVOLEMIC SHOCK

- 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…..

HEMORRHAGIC/HYPOVOLEMIC SHOCK

- **<u>Vasoconstrictor Response</u>**: sympathetic nervous system triggers **adrenal medulla** to secrete –

  ****epinephrine** and
  ****norepinephrine**

  increasing peripheral vascular resistance and reducing size of vascular department.

HEMORRHAGIC/HYPOVOLEMIC SHOCK

- **Kidneys**: decreased blood flow through kidney causes decreased glomerular filtration = decreased urine.
- When blood pressure decreases, kidney **produces renin**.

HEMORRHAGIC/HYPOVOLEMIC SHOCK

Low BP?

Kidney secretes Renin

Renin cleaves angiotensinogen → angiotensin I →

= angiotensin II

Angiotensin II –**vasoconstrictor**

HEMORRHAGIC/HYPOVOLEMIC SHOCK

- 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.

HEMORRHAGIC/HYPOVOLEMIC SHOCK

- 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.”*

HEMORRHAGIC/HYPOVOLEMIC 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%)

HEMORRHAGIC/HYPOVOLEMIC SHOCK

- 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.

HEMORRHAGIC/HYPOVOLEMIC SHOCK

- 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

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

Barash

Massive transfusion guideline

PRBCS	Plasma	Platelets	Cryoprecipitate
6 units	6 units		
6 units	6 units	1 apheresis	
6 units	6 units		10 units
6 units	6 units	1 apheresis	
6 units	6 units		10 units
6 units	6 units	1 apheresis	
6 units	6 units		10 units

Grady Memorial Massive Transfusion protocols

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

CARDIOGENIC SHOCK

- **<u>Early</u> 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).

CARDIOGENIC SHOCK

- **<u>Late</u> changes in cardiogenic shock** due to:
  - MDF (myocardial depressive factor)
  - blood pooling
  - platelet aggregation
  - released toxins
  - anaerobic metabolism and lactic acidosis

CARDIOGENIC SHOCK

- **<u>Late</u> 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

CARDIOGENIC SHOCK

- **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

CARDIOGENIC SHOCK

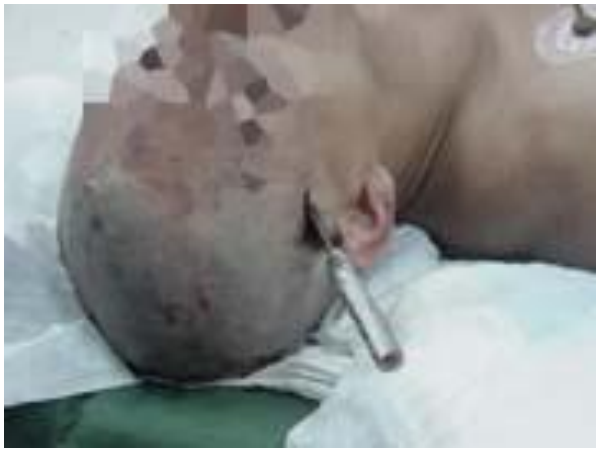
- Obstruction of cardiac filling:
  - cardiac tamponade
  - tension pneumothorax
  - massive pulmonary embolism

CARDIOGENIC SHOCK

- Hemodynamics
  - Increased CVP
  - Decreased CO
- Treatment of obstructive cardiogenic shock
  - Get rid of the obstruction

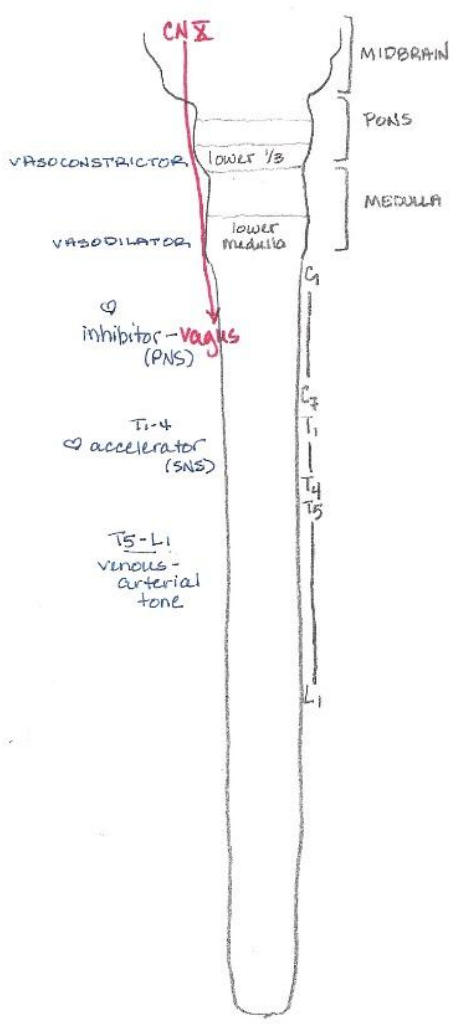
NEUROGENIC SHOCK

- 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

NEUROGENIC SHOCK

- 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

SEPSIS - SEPTIC SHOCK

- 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 ****

SEPSIS - SEPTIC SHOCK

- 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 1ST 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.

Second stage of SEPTIC SHOCK

- **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 2nd stage of SEPTIC SHOCK

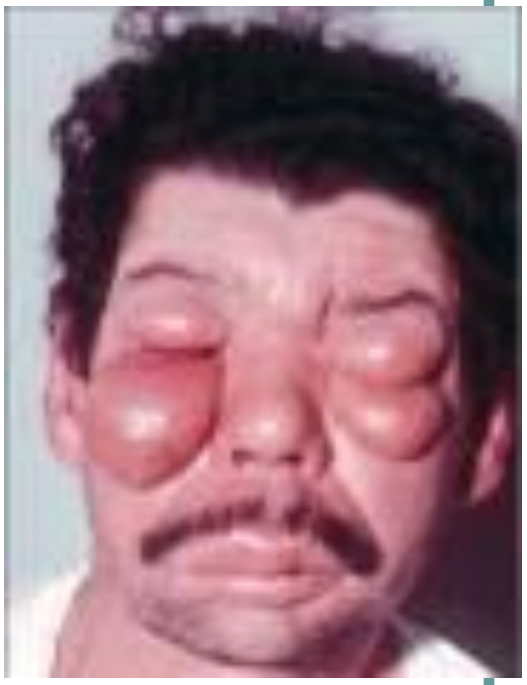
- **S/S:**
  - hypotension
  - decreased pulse
  - cold, mottled skin
  - no urinary output
  - ischemia, arrhythmias, acidosis, decreased stroke volume and cardiac output.

When compensatory mechanisms fail…

Mortality rate in septic shock is 80-90%.

ANAPHYLACTIC SHOCK

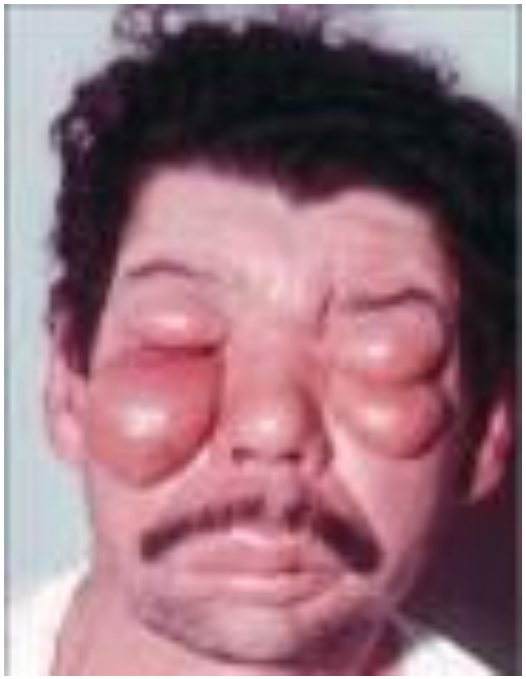
Allergic response triggers mast cells to release immunological mediators (i.e.: histamine, prostaglandins, leukotrienes, etc.) causing:



- systemic vasodilation
- edema of bronchial mucosa, bronchoconstriction, and dyspnea
- angioedema

ANAPHYLACTIC SHOCK

Anaphylactic shock can lead to death in a matter of minutes if left untreated.



ANAPHYLACTIC SHOCK

- 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

FAILURE OF COMPENSATORY MECHANISMS in SHOCK

- 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

FAILURE OF COMPENSATORY MECHANISMS in SHOCK

- Acidosis: worsened by:
  - Increased production of lactic acids with poor tissue perfusion
  - Decreased renal function
  - Decreased respiratory function, hypoxia, and hypercapnia (more acids)

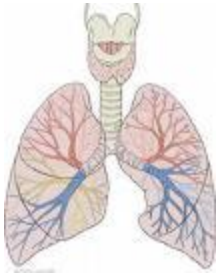
Complications of shock

HYPOTENSION

(cardiac output low)

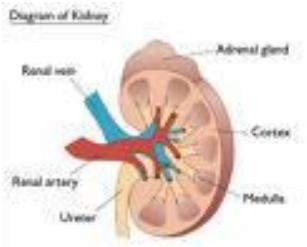
LUNGS

kills Type 2 alveolar cells→ no surfactant → ARDS



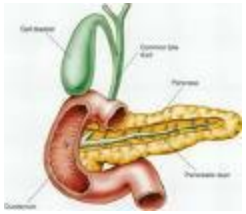
KIDNEYS

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

Go Steelers!!!!

The End