Project: Ghana Emergency Medicine Collaborative

Document Title: Shock

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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 X HR

Thus,

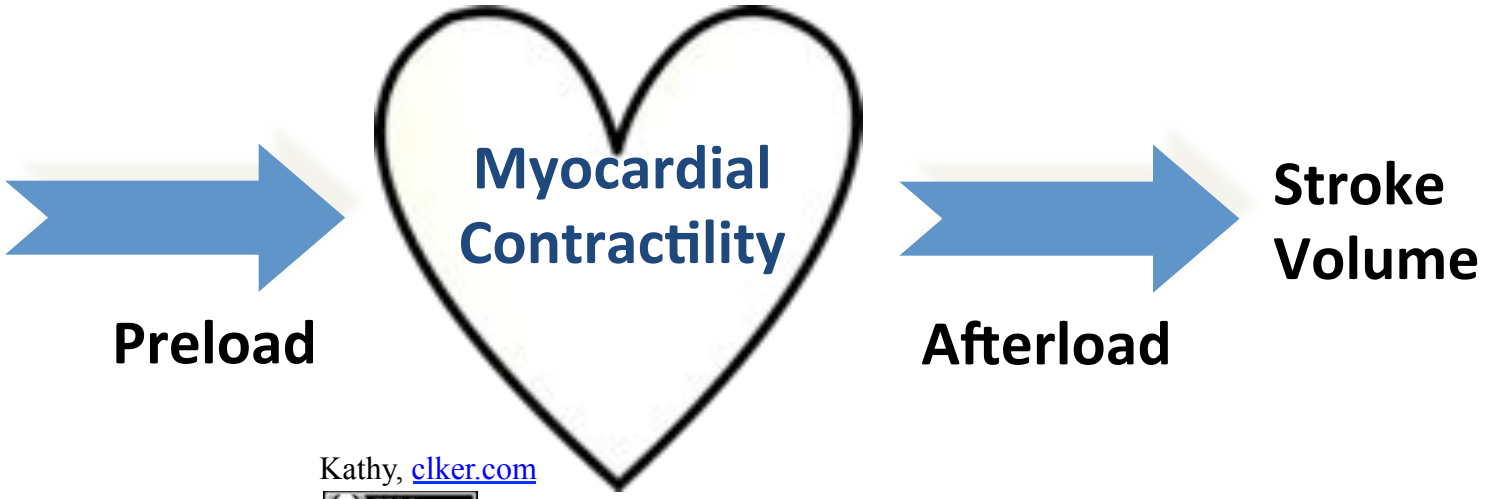
Blood Pressure = SV x HR x PVR

Blood Pressure = Stroke Volume X Heart Rate X 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?



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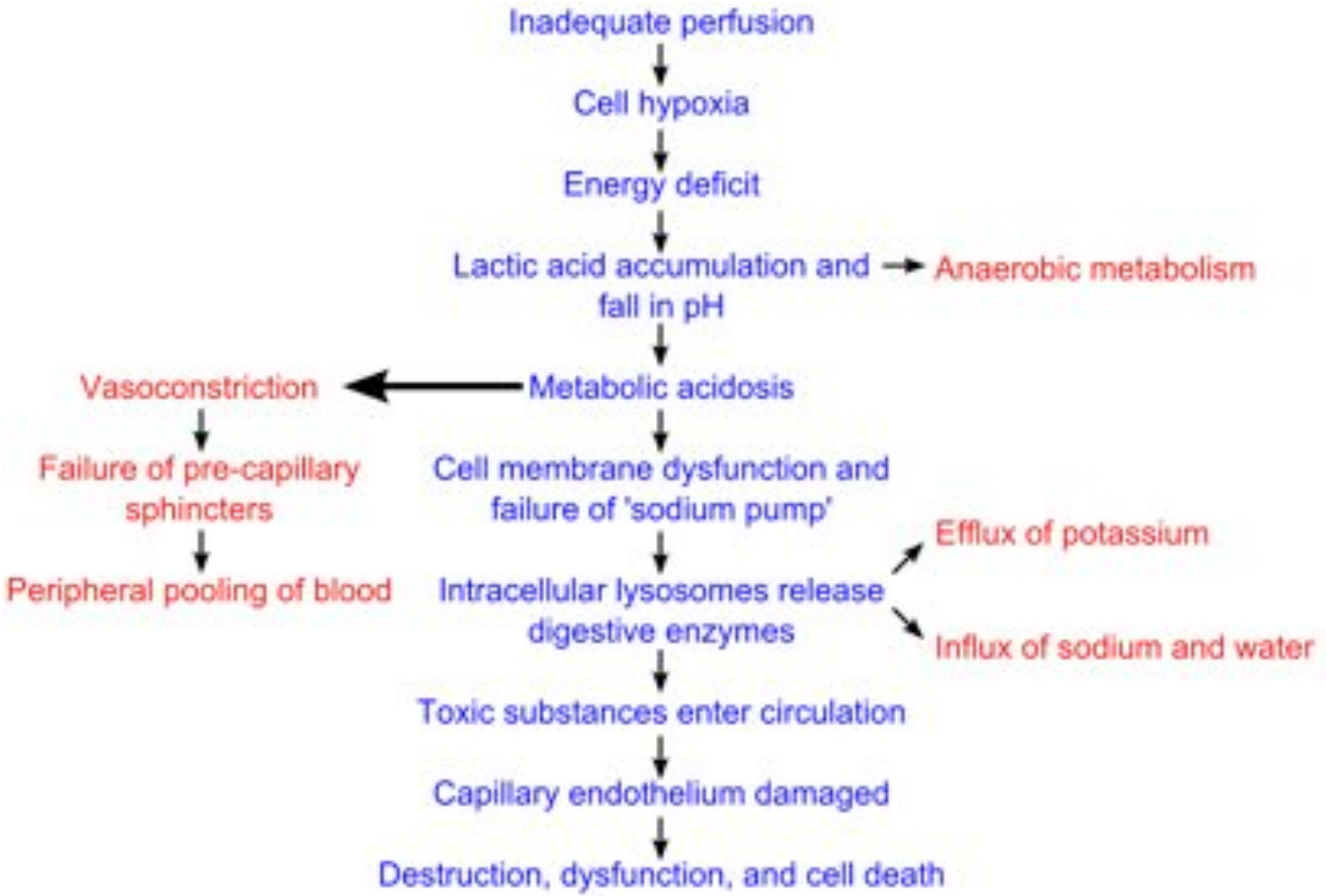
Shock Pathophysiology



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

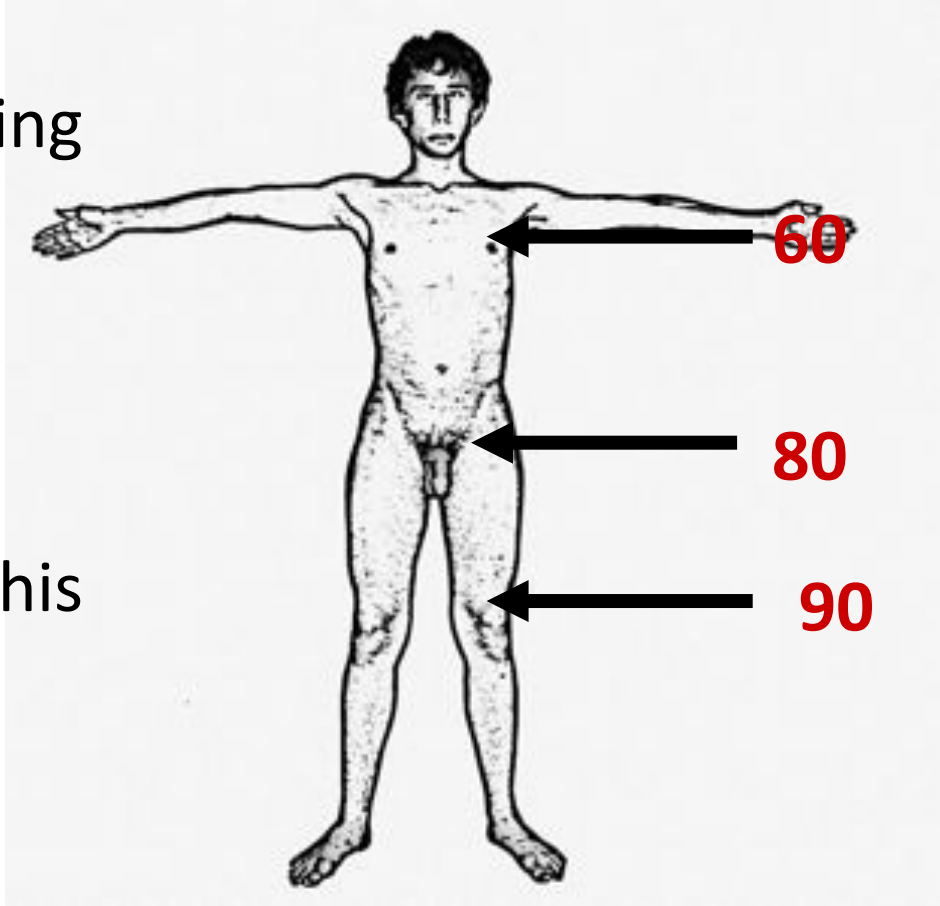
- CT of head/sinuses for occult infections/ abscesses
- Lumbar puncture for meningitis/encephalitis
- Wound cultures
- Acute abdominal series
- Abdominal/pelvic CT or US
- Fibrinogen, FDPs, D-dimer if suspicion for DIC is high

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 Hemoptysis
  - 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

CLASS	I	II	III	IV
BVL	< 15%	15 - 30%	30 - 40%	> 40%
AMOUNT	750 cc	750 - 1500 cc	1500 - 2000 cc	> 2000 cc
PULSE	<100	> 100	>120	>140
BP	No change	Narrowed pulse pressure	Consistent decrease in SBP	Decreased SBP and narrowed pulse pressure or no DBP
RESP	No change	20-30	30-40	>35
CNS	No change	Anxiety	Anxious, confused	Confused. lethargic
Urine	>30cc per hr	20-30cc per hr	5-15cc per hr	negligible
TX	Replace fluid loss	2L NS IV	2 L NS IV, usually requires blood transfusion	Rapid transfusion of blood and NS, requires immediate intervention to stop hemorrhage

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

	Rapid Response	Transient Response	No Response
Vitals	Return to normal	Transient improvement with return to previous	Remain Abnormal
Estimated Blood loss	10-20%	20-40% with ongoing likely	Severe >40%
Need for more Fluid	Low	High	High
Need for Blood	Type and cross	Type specific	O neg
Need for surgery	Possible	Likely	Highly likely

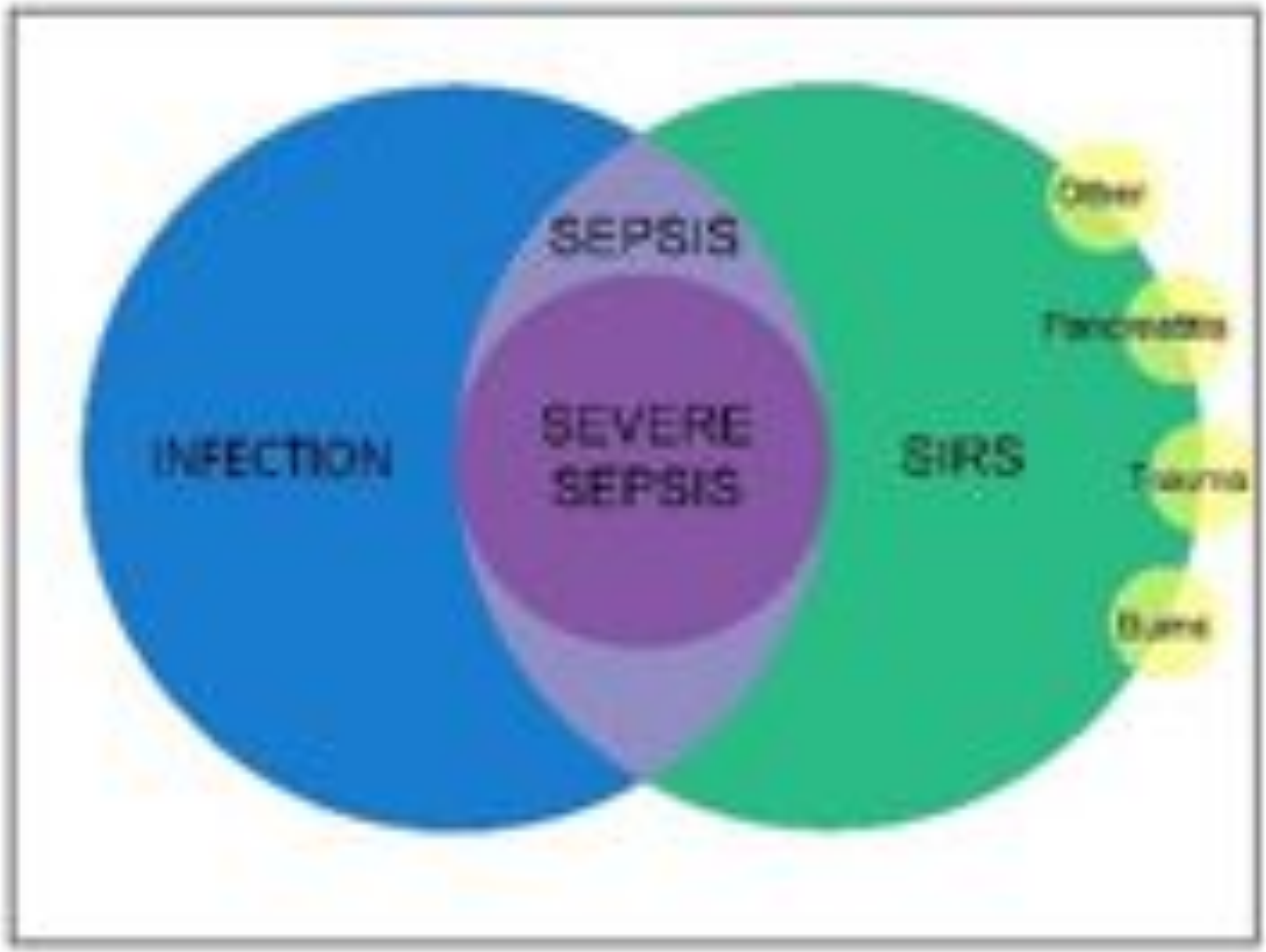
SIRS

- 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/mm^3 or > 12,000 cells/mm^3 or greater than 10% bands

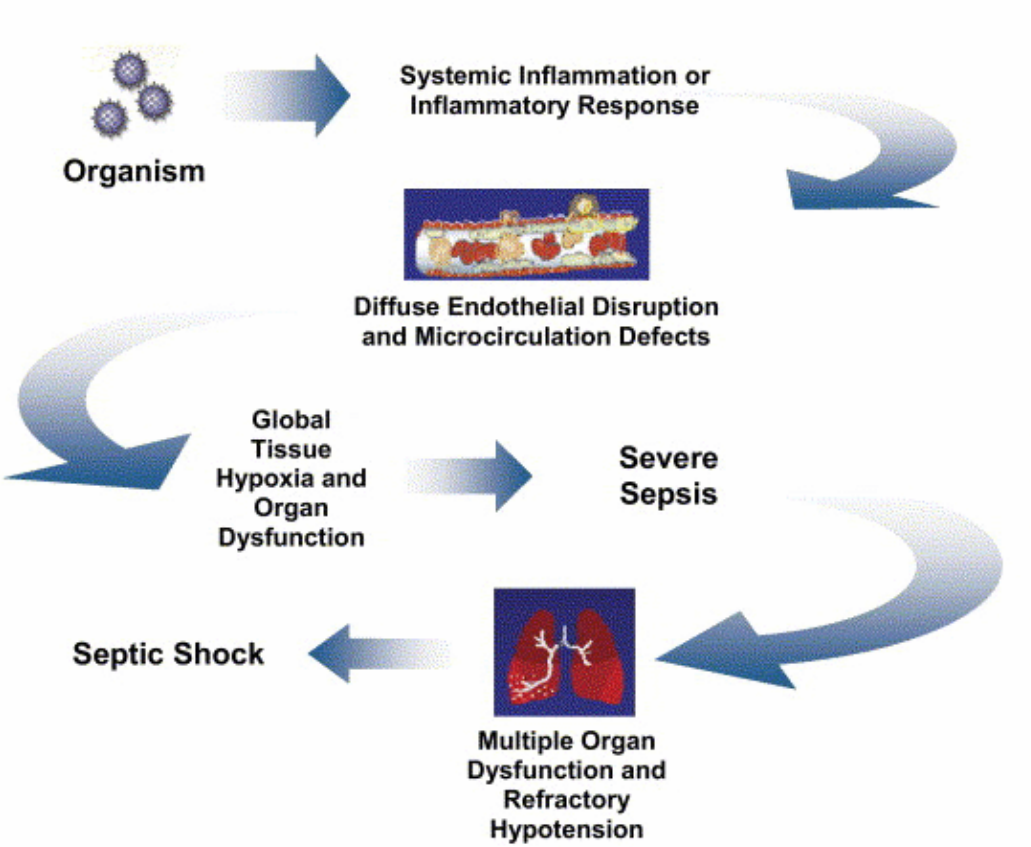
Sepsis

- 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



Nguyen H et al. Severe Sepsis and Septic-Shock: Review of the Literature and Emergency Department Management Guidelines. Ann Emerg Med. 2006;42:28-54.

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

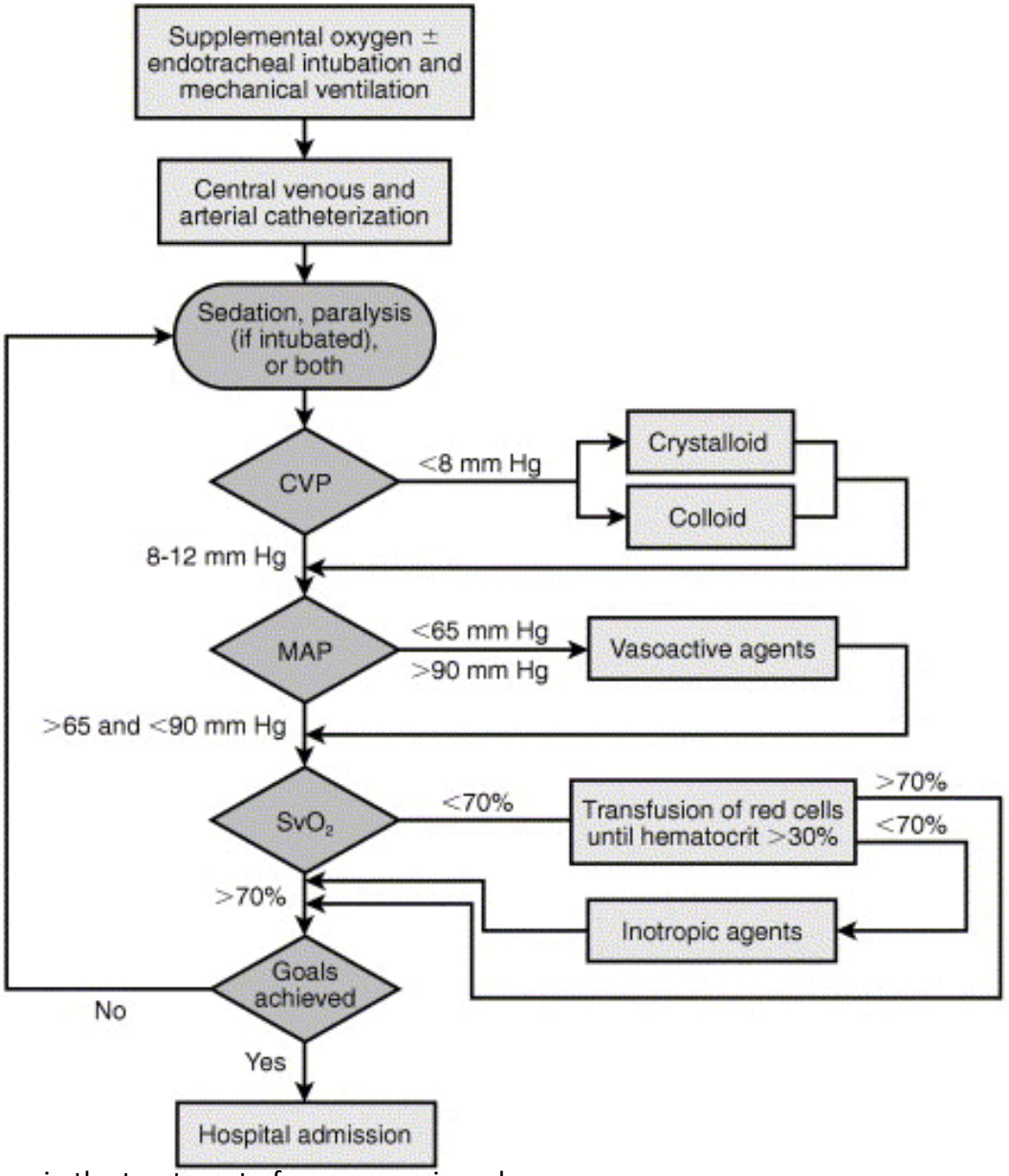
Septic Shock - Treatment

- 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



Rivers E et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock N Engl J Med. 2001:345:1368-1377.

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

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

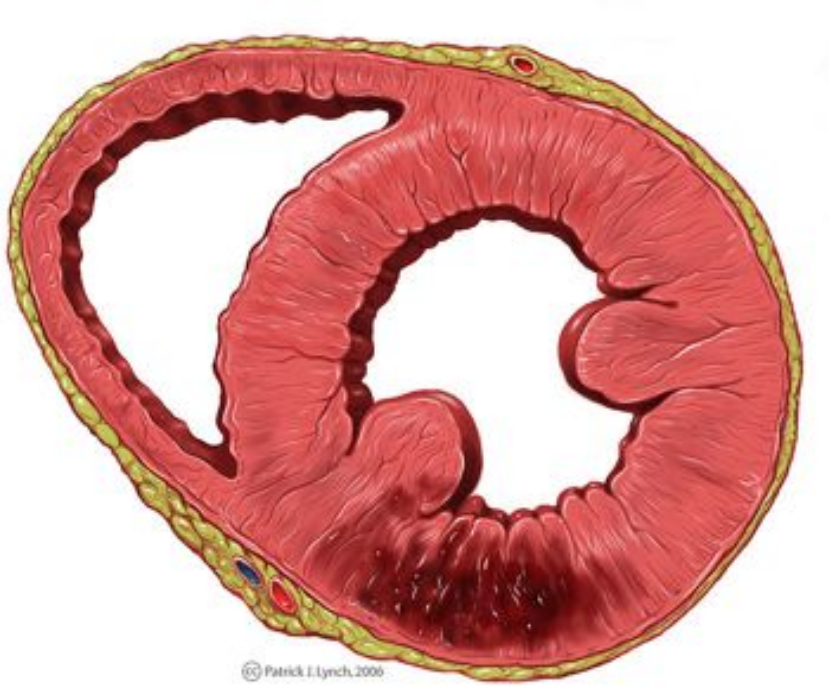
- Epinephrine
  - 0.3 mg IM or SC of 1:1000 dilution for anaphylaxis
  - Repeat every 5-10 min as needed
  - Caution if patient on beta blockers due to unopposed alpha stimulation and resultant severe hypertension
  - For CV collapse (i.e. cardiac arrest), 1 mg IV of 1:10,000 (same as ACLS Dose)
  - 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/m^2
  - 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

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

gunkyboy, Wikimedia Commons

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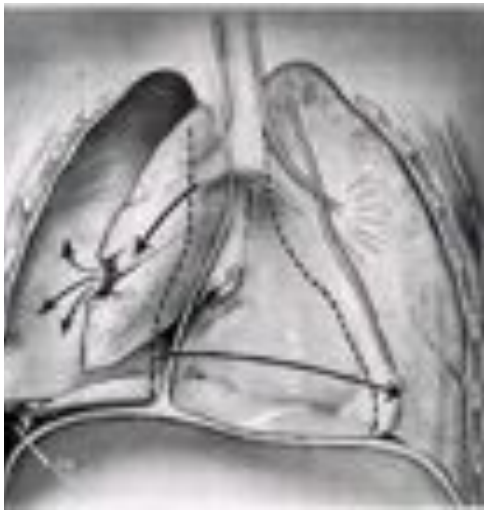
Neurogenic Shock- Treatment

- 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



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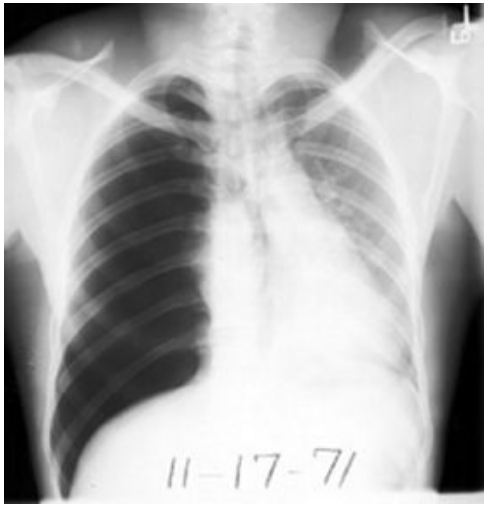

Source: www.meddean.luc.edu/lumenMedEd/medicine/pulmonar/cxr/pneumo1.htm

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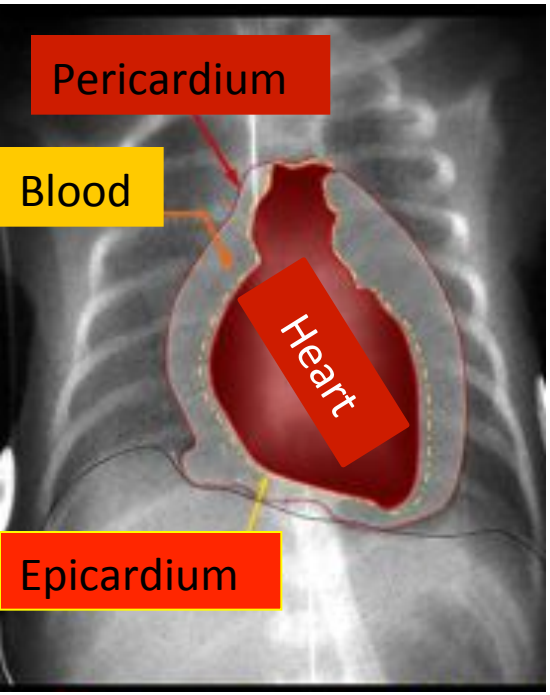
- Tension pneumothorax
  - Air trapped in pleural space with 1 way valve, air/pressure builds up
  - Mediastinum shifted impeding venous return
  - Chest pain, SOB, decreased breath sounds
  - No tests needed/clinical diagnosis
  - Treatment: Needle decompression followed by chest tube

Obstructive Shock

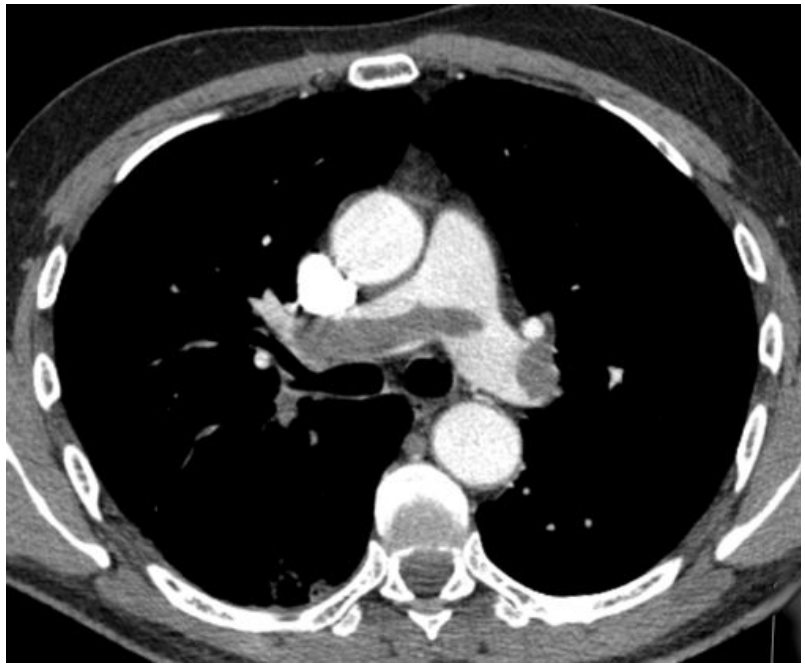


Aceofhearts1968, Wikimedia Commons

- 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



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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
  - Neosynephrine - 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