SHOCK

Hemodynamic manifestation of cellular metabolism insufficiency

Poor Perfusion, cellular Anaerobic Metabolism
Release of mediators which damage tissue
Perfusion

Dependent on:

1. Pump
2. Fluid volume
3. Container
The Pump

- Receives blood from venous system
- Pump blood to lungs to get O2
- Pump blood to peripheral tissues
Fluid

- Blood
- Must have adequate volume to fill system
- Transports O2, CO2, nutrients, hormones, etc
Blood vessels
Continuous, closed, pressurized pipeline moves blood
Under control of ANS, regulate blood flow to different areas by adjusting size and rerouting blood flow through microcirculation
Microcirculation

- Responsive to local tissue needs
- Cap beds adjust size to supply undernourished tissue and bypass tissue with no immediate need
- Precap sphincters and postcap sphincters open and close to feed or bypass tissue
Compensated Shock

Vital organ dysfunction maintained by activation of homeostatic mechanisms. These pathways include:

- Neural
- Hormonal
- Chemical
NEURAL COMPENSATION

Baroreceptors
Monitor for changes in BP
Trigger sympathetic nervous system to vasoconstrict

Blood vessels in skin, kidney, GI tract contract
Sweat glands stimulated

↓ Cardiac Output
↓ Blood Pressure
Baroreceptors

SYMPATHETIC NERVOUS SYSTEM ACTIVATION

Blood vessels in skeletal muscles dilate
Coronary arteries dilate

Heart
HR
FOC

Lungs
Rate & depth

Pupils dilate
HORMONAL COMPENSATION

Sympathetic Nervous System Activation

↓ Renal Blood Flow  Anterior Pituitary Gland  Anterior Pituitary Gland

↑ Renin

↑ Aldosterone

↑ ADH

↑ Sodium & Water Retention

ACTH

↑ Cortisol

Liver

↑ Blood Glucose

Adrenal Medulla

↑ Epinephrine & Norepinephrine

Liver

↑ Blood Glucose

Sodium & Water Retention

↓ Renal Blood Flow
CLASSIFICATION & DIFFERENTIATION

- Cardiogenic shock
- Obstructive shock
  - Narrow pulse pressure
- Hypovolemic shock
- Distributive shock
  - Neurogenic
    - Bradycardia
  - Anaphylactic
  - Septic
    - Wide pulse pressure
COMMON PATHOPHYSIOLOGY

CARDIOGENIC SHOCK
- L Ventricular function
  - MI
  - Cardiomyopathy
  - Etc.

HYPOVOLEMIC SHOCK
- Intravascular volume
  - Bleeding
  - Fluid shifts into interstitial
  - Dehydration

VASOGENIC SHOCK
- Vasodilatation
  - Sepsis
  - Neurologic damage
  - Anaphylaxis

COMMON PATHOPHYSIOLOGY
- VENOUS RETURN
- STROKE VOLUME
- CARDIAC OUTPUT
- BLOOD PRESSURE
- TISSUE PERFUSION
HYPOVOLEMIC SHOCK

Decreased circulating volume through external or internal fluid loss.

Sources of external loss of body fluid:
- Hemorrhage
- GI tract output
- Renal output

Sources of internal loss of body fluid:
- Internal hemorrhage
- Movement of body fluids interstitial spaces
HYPOVOLEMIC SHOCK

INTERVENTION:
- Correct the problem
- Replace the volume

- With What?
  - RBC
    • Stay in space
    • Inflammatory
  - Colloid
    • Stay in space 24h, osmotic pull
    • Less edema
    • Volume limit, expensive
  - Crystalloid
    • Stay in space 1h, trauma
    • Edema, dilution, compartment syndrome
  - Hgb substitutes
    • Polyheme, Oxygent
# Classes of Hypovolemic Shock

<table>
<thead>
<tr>
<th>Blood Loss</th>
<th>15%  (750ml)</th>
<th>15-30% (750-1500 ml)</th>
<th>30-40% (1500-2000 ml)</th>
<th>&gt;40% (&gt;2000 ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP</td>
<td>Normal</td>
<td>Normal</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Heart Rate</td>
<td>&lt;100</td>
<td>&gt;100</td>
<td>&gt;120</td>
<td>&gt;140</td>
</tr>
<tr>
<td>LOC</td>
<td>Slightly anxious</td>
<td>Mildly anxious</td>
<td>Anxious &amp; confused</td>
<td>Confused &amp; Lethargic</td>
</tr>
<tr>
<td>Resp. Rate</td>
<td>14-20</td>
<td>20-30</td>
<td>30-40</td>
<td>&gt;40</td>
</tr>
<tr>
<td>Urine Output</td>
<td>&gt;30 ml/hr</td>
<td>20-30 ml/hr</td>
<td>5-15 ml/hr</td>
<td>&lt;5 ml/hr</td>
</tr>
</tbody>
</table>
Mal-distribution of intravascular volume

3 Types:

**Neurogenic shock**
- Loss of vasomotor tone regulated by the sympathetic nervous system causing massive peripheral vasodilatation and bradycardia.

**Anaphylactic shock**
- antigen / antibody reaction causing histamine- / serotonin-induced massive vasodilatation and increased capillary permeability.

**Septic shock**
SHOCK

Principles of management

- Correction of the underlying cause of shock
- Improve oxygenation
- Restore adequate tissue perfusion
Review?

- Which patient is illustrating a compensated response to shock?
  a. Patient is flushed, warm and clammy
  b. Patient with decreased HR and BP (parasympathetic response)
  c. Increased reabsorption of sodium and water
  d. Patient with third spacing interstitial space
Review?

- Pt admitted with fractures femur. Carotid pulse not palp, no spont resp. ECG shows NSR with HR 130. What is initial therapy?
  a. PRBC, vent, bicarb
  b. Vent, atropine, calcium
  c. CPR, epi, IV fluid
  d. CPR, dopamine, verapamil
Sepsis???

- Bacterial
- Line sepsis
- Nosocomial infections
  - UTI
  - pneumonia
- Immunosuppression

- 60% patient septic negative blood culture
Relationship of Infection, SIRS, Sepsis, Severe Sepsis

SIRS

- Systemic Inflammatory Response Syndrome

- To a variety of clinical insults

2 or more of the following criteria:
  - Temperature, >38 or <36
  - Tachycardia, HR > 90
  - Tachypnea, RR > 20
  - WBC 12,000 or < 4000 or 10% bands
Sepsis

- Confirmed or suspected infection plus ≥ 2 SIRS criteria
- Hypotension is not part of the definition
Septic Shock

- Sepsis induced hypotension
- despite adequate fluid resuscitation
- perfusion abnormalities
MODS

- Multiple Organ Dysfunction Syndrome

- Progressive organ failure such that the person cannot live without intervention
Organ Failure

- One organ dysfunction = 20% mortality
- For every organ dysfunction thereafter is 15 – 20% increase mortality
- Increase 10% for over 65
- Mortality rate highest in those with CV compromise

- By the time 1 system shows dysfunction, other organs are effected
- Common dysfunction seen first CV and respiratory systems
Incidence of sepsis

- >750,000 new cases annually
- Leading cause of non-cardiac ICU death
- 10\textsuperscript{th} leading cause of death in US
- 139\% increase from 1979 - 1987
Risk Factors for Sepsis:

- Patient related
  - Young, old
  - Malnourished
  - Chronic health problems
  - ETOH
  - Trauma
  - Burns

- Treatment related:
  - Invasive equipment
  - Immunosuppression
  - Surgery
  - Medications
Sepsis Pathophysiology

Infection (Bacterial, viral, fungal, or parasitic infection/endotoxin) → Inflammation → Coagulation → Fibrinolysis → Endothelial Dysfunction and Microvascular Thrombosis → Hypoperfusion Ischemia → Acute Organ Dysfunction
Endothelium & sepsis

Progressive endothelial dysfunction

- Microvasc permeability
- Plt sludge
- Coagulation system activated
- Dysregulation of vasodilation and vasoconstriction mechanisms
- Profound shock
- Organ dysfunction
Sepsis

- Vicious cycle
- Protective mechanisms cause:
  - Maldistribution of volm
  - Imbalance O2 supply & demand
  - Alterations in metabolism
Sepsis = Upset balance

- Favors coagulation
  - fibrin clots lead to:
    - microvascular hypoperfusion
    - diminished oxygen delivery
    - tissue necrosis
    - organ failure
    - further endothelial damage
    - release of more neutrophils and inflammatory cytokines.
Other VS of Shock

**Lactate**
- Determine degree of anaerobic metabolism
  - Norm $\leq 2$
  - $\frac{1}{2}$ life 16h
- Progressive clearing
- Global value

**Base Deficit**
- Nonrespiratory component of ABG
- How much stores are used to maintain norm pH to buffer against lactate
- More lactate, more O2 debt, more base deficit
- Norm $0 \pm 2$
- Global value
Lack of oxygen

• Cellular level
  – tissue perfusion is impeded (clotting) and oxygenation is impaired

• Tissue oxygenation is a critical indicator of sepsis
  – can be detected before organ failure occurs by:
    • SVo2, ScV o2
Patient Identification: What to Look for?

- Patient’s with known or suspected infection
  - Change in UOP?
  - High FiO2?
  - Tachypnea?
  - Tachycardia?
  - Vasopressors? After fluid
  - Change in LOC?
  - Low or ↓ plt count
- If yes, to any, something is WRONG!!

Recognizing patients at risk for sepsis is difficult due to limited signs and symptoms!

- 113/48, 122, 32, 102.1
- O2 sat 90% on 60%
- UOP 50cc/4h

Does your patient have any of these signs?
Identifying Acute Organ Dysfunction as a Marker of Severe Sepsis

- Tachycardia
- Systolic BP ≤ 90, or MAP ≤ 70 despite fluids & Vasopressors
- Urine Output < 0.5 mL/kg/hr despite fluids
  - Creatinine > 50% from baseline
  - Acute dialysis
- Altered Consciousness
  - Reduced GCS
- Tachypnea
  - PaO₂/FiO₂ ≤ 250
  - Mechanical Ventilation
  - PEEP > 7.5
- Liver Enzymes
  - > 2x ULN
- Low pH with high lactate (e.g., pH, 7.3 & lactate > ULN)
- Platelets ↓ < 100,000/mm³
- PT/aPTT ↑
- D-dimer ↑

Metabolic Alterations

Hypermetabolic state needs adequate nutrition

• Cytokine mediated autocannibalism
  – Hyperglycemia with insulin resistance
    • Altered glucose metabolism
  – Profound negative nitrogen balance
  – Protein diversion
  – Altered blood flow to key metabolic sites
  – Lactic acidosis

• Treatment – tight glycemic control, 80-110 glucose via insulin drip
Sepsis Resuscitation Bundle:

All elements occur within 6h presentation of sepsis.

• Serum lactate level
• Blood cultures prior to antibiotics
• Broad spectrum antibiotics within 3 hours for ED admissions and 1 hour for non-ED admissions
• End points of resuscitation: In the event of hypotension and/or lactate > 4 mmol/L:
  • Fluid resuscitation of 20ml/kg of crystalloid
  • Vasopressors if not responding fluid resuscitation to maintain MAP > 65 mm Hg
• If persistent hypotension despite fluid resuscitation (septic shock) and/or lactate > 4 mmol/L:
  • CVP >8
Sepsis Management Bundle:

All elements completed within 24h of presentation of sepsis.

- Low dose steroids for septic shock in accordance with a standardized ICU policy
- Glucose control < 150 mg/dl
- Low tidal volume ventilation
Why Steroids for Sepsis? Adrenal Insufficiency (AI)

• Relative or Functional AI
  – Rare in general population
  – Reported in 28% critically ill pts
  – Failure of appropriate neurohormonal response leads to:
    • ↓d cortisol production from adrenal
      – Vasopressor dependent refractory hypotension
        » ↑d CO and ↓d SVR
Diagnosis of Adrenal Insufficiency

• High-dose corticotropin stimulation test
  – Can be done at any time of day
  – Collect baseline cortisol
    • <15 micrograms/dl = adrenal failure needs replacement therapy
  – Administer 0.25 mg cosyntropin IVP over 1-2 minutes
  – Collect another cortisol level 60 minutes after administration of cosyntropin
  – Cortisol levels post cosyntropin:
    • <9 microgram/dl rise = adrenal failure needs replacement
Treatment of AI: Hydrocortisone

- Hydrocortisone is preferred to other glucocorticoids.
  - synthetic equivalent to the physiologic final active cortisol
- Hydrocortisone taper over 7-11 days.
  - Hydrocortisone 100mg IV every 8h, then 50mg every 8h, to 50mg every 12h

GOAL: Restore Vascular Tone by replacing cortisol
Treatment goals for septic shock

• Fill vascular space
• Prevent secondary organ damage
  – Fluids
  – Pressors
    • Dopamine, levophed (best to vasoconstrict), neosynephrine, vasopressin
  – Xigris
• Steroids stress dose
Review?

- Pt h/o alcohol and heroin abuse admit for treatment of cellulitis. Pt is flushed, moist skin and slow to respond to verbal stimuli. Affected arm is edematous, hard to touch, with yellow exudate from puncture wounds. BP 88/45, HR 124, RR 26, T 102

- What initial orders should you anticipate?
  a. Antipyretic, dopamine
  b. CT head, drug screen
  c. Bld culture, AB, fluid bolus
  d. Monitor signs withdrawal, antipyretic, IV fluid with vitamins
CCRN Review

- Multisystem Trauma
- Trauma triad
- Compartment syndromes
- Burns
- Poisons
Case Study

- 25 year old male with previous medical history of asthma, 1ppd smoker
- Restrained driver of small SUV
- Struck on side of vehicle by pick up traveling 60 mph
- 20 inch intrusion into driver side of vehicle
  - Patient pinned between steering wheel and seat, 25 minute extrication
  - Steering wheel deformity
Initial Treatment & Assessment at the Scene

• Patient complaining of shortness of breath
  – States: “Need breathing treatment”
  – Received albuterol nebulizer

• Continues with difficulty breathing
  – States: “Need to sit up to breathe”

• Assessment:
  – Respiratory: No chest wall deformity, BBS diminished and wheezing
  – Skin: Cool & pale, deformed L thigh, immobilized
At the Scene / During Transport

<table>
<thead>
<tr>
<th>Time</th>
<th>BP</th>
<th>HR</th>
<th>RR</th>
<th>GCS</th>
<th>Events</th>
</tr>
</thead>
<tbody>
<tr>
<td>1625</td>
<td>110/80</td>
<td>100</td>
<td>28</td>
<td>15</td>
<td>100% Non-Rebreather</td>
</tr>
<tr>
<td>1630</td>
<td>93/56</td>
<td>123</td>
<td>28</td>
<td>14</td>
<td>O2 sat not picking up</td>
</tr>
<tr>
<td>1635</td>
<td>123/35</td>
<td>110</td>
<td>24</td>
<td>12</td>
<td>No sat. Asks “Am I going to die?”</td>
</tr>
<tr>
<td>1640</td>
<td>57/25</td>
<td>110</td>
<td>20</td>
<td>13</td>
<td>Loaded in helicopter</td>
</tr>
<tr>
<td>1645</td>
<td>52</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>Needle decompression.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>100% Bag / Valve / Mask (BVM)</td>
</tr>
<tr>
<td>1650</td>
<td>65/20</td>
<td>55</td>
<td>10</td>
<td>4</td>
<td>Unable to intubate. BVM</td>
</tr>
<tr>
<td>1700</td>
<td>57/35</td>
<td>101</td>
<td>8</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>1706</td>
<td>92/55</td>
<td>97</td>
<td>18</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>1713</td>
<td>90/57</td>
<td>90</td>
<td>14</td>
<td>4</td>
<td></td>
</tr>
</tbody>
</table>
Manage Multisystem Trauma

• Primary survey
  – A, B, C, D
• Trimodal distribution of death in trauma & causes:
  – “Immediate”
  – “Golden Hour”
  – Beyond the hour
Emergency Management

- Primary Survey
  - C-spine immobilization
  - A
    - Unresponsive
  - B
    - Not breathing
    - Intubated
  - C = STOP THE BLEEDING
    - Bleeding, major sources
    - Weak carotid pulse with extreme bradycardia
    - IV Atropine
    - Chest opened via Left Thoracotomy
    - Direct cardiac massage, cross clamp aorta
    - Lines, fluid, 3uPC, BP 100/80
  - D
Review?

- Pt just returned from surgery after repair great vessel from penetrating knife wound. VS 60/40, HR 136. Most recent CXR shows widened mediastinum. What is the most appropriate intervention?
  a. Administer .5mg digoxin
  b. Place pt in trendelenburg
  c. Prepare for chest tube insertion
  d. Prepare for sternal re-exploration
Further Assessment

• Secondary Survey
  – Laceration to posterior head
  – Diminished Breath Sounds
  – Blood in ETT
  – Abdominal ecchymosis and distention
  – Left leg: Thigh edema, grossly deformed / shortened, rotated externally, no distal pulses, poke hole to posterior left thigh

• Vital signs:
  – 1730: 123/50, 130, post chest tube & thoracotomy
  – 1738: 133/109, 97, post clamp aorta and 3uPC

• TO OR at 1738
• Surgery started at 1745.
  Procedures performed:
  – Splenectomy for shattered spleen
  – Evacuation of 2 liters of blood from abdomen
  – Repair of ruptured pulmonary vessel
  – Closure of thoracotomy and placement of 2 chest tubes
  – Incision and drainage of open left femur fracture / placement of traction pin
  – Incision and drainage of left elbow
  – 12 units of PRBC’s
  – 2 units of cryoprecipitate
  – 10 units of platelets
  – 2 units of fresh frozen plasma
Now What??

• He is coming to the ICU.

• What are your priorities?

• What are you going to be diligently assessing?
Initial ICU Management

- Aggressive correction and prevention of trauma triad of death

- Laboratory monitoring every 4 hours

- Monitor abdominal pressures every 4 hours
Predisposing Factors for Trauma Triad:

- Degree of transfusion, more than 10 units
- Injury severity score higher than 35
- pH less than 7.2
- Hypotension for longer than 70 minutes.
- Temperatures less than $35^\circ C$
- Base deficit 15 mmol/L
Consequences hypothermia

- cardiac dysrhythmias
- cardiac output
- myocardial ischemia
- systemic vascular resistance
- left shift in the oxygen-hemoglobin saturation curve
- lactic acid production
- coagulopathic bleeding***
Metabolic Acidosis

- inadequate perfusion
- tissue hypoxia
  - normal cell physiology altered
  - shift from aerobic to anaerobic metabolism resulting in lactic acidosis
  - amount of lactate produced correlates with the total oxygen debt
- hypoperfusion and severity of shock.

Temperature accounted for six times more variation in acidosis than injury severity.
Coagulopathy:

- Caused by hypothermia & massive transfusion
- Progressive core hypothermia with persistent metabolic acidosis is precursor to severe coagulopathy.
- Insidious process
- Accounts for 50% hemorrhagic deaths
Physiology of Compartment Syndromes

• Elevated pressure within any enclosed space

• Due to:
  – Fluid
  – Blood
  – Air
  – Mass
Physiologic Insult: Cellular Injury

- Any interruption in blood supply
- Ischemia
- Inflammatory response (histamine)
- Edema, further cellular injury
- Ischemia – decrease oxygen to cells
  - Anaerobic metabolism
The vicious cycle of compartment syndrome

- Edema
- More Inflammation
- Cell death
- Cell hypoxia
- Perfusion
- Compartment pressure
  ↓

↑
# Intra-Abdominal Pressure Grading

<table>
<thead>
<tr>
<th>Grade</th>
<th>Severity</th>
<th>Range of Reading</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>normal</td>
<td>0-15</td>
<td>Rarely require decompression</td>
</tr>
<tr>
<td>II</td>
<td>mild</td>
<td>15-25</td>
<td>Vigilant monitoring, may require decompression</td>
</tr>
<tr>
<td>III</td>
<td>moderate</td>
<td>25-35</td>
<td>Worry, monitor, surgical decompression</td>
</tr>
<tr>
<td>IV</td>
<td>severe</td>
<td>&gt;35</td>
<td>Urgent surgical decompression</td>
</tr>
</tbody>
</table>
Interventions for ACS:

- Abdominal pressure monitoring – belly already open

- Monitor for the effects on the other organ systems
Consequences of ACS:

- Post-op ileus
- Ischemic bowel
- Gastric ulcers
- Hepatic dysfunction
- Pancreatic dysfunction
- Rhabdomyolosis
- Venous thromboembolism
- Cerebral ischemia
- Spinal cord ischemia
- Poor wound healing

Post-op sepsis & MODS
Post Initial Care

- Continues neurologically impaired, not follow commands
- Persistently febrile
  - Panculture
  - Cooling blanket
- Cleared for OOB by ortho, OOB daily
- DAY 5, 10a:
  - 113/48, 122, 32, 102.1
  - O2 sat 90% on 60%
  - UOP 50cc/4h

What is going on?
What red flags are there?
Infection? Sepsis? Severe sepsis?

WHY?
Post Initial Care

• Continues neurologically impaired, not follow commands
• Persistently febrile
  – Panculture
  – Cooling blanket
• Cleared for OOB by ortho, OOB daily
• DAY 5, 10a:
  – 113/48, 122, 32, 102.1
  – O2 sat 90% on 60%
  – UOP 50cc/4h

Infection? Sepsis? Severe sepsis?

What is going on? What red flags are there? Treatment?
Treatment

• Sepsis resuscitation bundle
  – Lactate 5.2
  – 3L fluid, remained hypotensive
  – Dopamine
  – Intubation
  – Cultures, antibiotics

• Transfer to ICU
  – Other labs: BUN 32, creat 1.8, K 5.7

What systems are dysfunctional?
What are your signs?
What continued treatment can you expect?
• Our patient 6 weeks later!
Pt blunt chest injury from MVC.
Confused, cyanotic, distant heart sounds
2h after admit – BP decreased, JVD
What is most likely cause of findings:
  a. Hypovolemia
  b. Cardiac tamponade
  c. Cardiogenic shock
  d. Pulmonary edema
Patient with blunt cardiac trauma, what patient symptom supports diagnosis of myocardial contusion?

a. Cardiac dysrhythmias
b. PMI shift to right
c. Increased myoglobin
d. Bounding pulses
Case Study: Burns

- Betty is 68 y.o. at home alone
- Neighbors note smoke from window, call 911
- 40% BSA burn & severe smoke inhalation
- Admits to EMT “I drank liquid plummer then set the house on fire”.
Burns:

• Causes:
  – Thermal
    • House fires 75% burn deaths
    • Death d/t smoke inhalation, CO poisoning
  – Scald
    • Kids, elderly
  – Chemical
    • Flush
  – Electrical
  – Radiation
Zones of injury

- **Zone of hyperemia**
  - Outer zone
  - Tissue red, blanchable
  - No cell death

- **Zone of stasis**
  - Middle zone
  - Cells can recover or die 24h post injury
  - Tissue red, not blanch
  - Recovery depend on prompt resuscitation

- **Zone of coagulation**
  - Protein coagulation
  - Cell death
  - Tissue gray, black, white, not blanch
Assessment

• A
  – Edema, secretions

• B
  – Respiratory excursion, circumferential burn chest

• C
  – fluid resuscitation, formula
    • 4ml x (% BSA burn) x weight kg
    • ½ 8h, rest in next 16h

• D

• Expose
  – Remove burning clothing, jewelry
  – Cover dry sterile dressing, towels, temp control
Secondary Survey

- Neuro
  - pain
- Chest = s/s smoke inhalation
- Abdomen = compartment syndrome
- GU = edema
- Extremities = 5 P’s
- Burn
Assessment of the burn:

- **Location**
  - ↑ severity hands, face, feet, eyes, ears, genitalia

- **Extent**
  - Percentage BSA burned
  - Rule of 9’s
  - Palm
  - Evolution of burn injury
Burn Assessment: Depth

- **Superficial partial-thickness**
  - Pink to red, no blister
- **Moderate partial thickness**
  - Red, mottled, pink with blistering
  - Skin moist, weeping, blanches
- **Deep partial thickness**
  - Pink to pale, blisters
  - Non blanchable
- **Full thickness**
  - White, red, brown, black
  - No blister
Complications of Burns:

- Pulmonary injury consider location at time of injury (inside closed space)
  - Smoke inhalation
  - CO poisoning
  - Direct thermal burn to airway
- Fluid deficit: considerations time & replacement start
  - Replacement formula
- Infection
- Electrolyte imbalance
  - ↑ K, cell death, muscle death
  - ↑ Na, intravascular fluid loss
Carbon Monoxide

- Most seen fall & winter
- Gas emitted from gas, charcoal, oil, wood
  - From incomplete burning of materials
- Displaces oxygen from Hgb
  - 200x affinity
- >10% COHb diagnostic of CO poisoning
- Absence of COHb not rule out inhalation injury
S/S CO poisoning

- Low level exposure:
  - SOB
  - Nausea
  - Mild headache

- Moderate level exposure:
  - H/A
  - Nausea
  - Light headedness, dizzy

- High Level exposure
  - Death within minutes
Treatment CO poisoning

- **Oxygen**
  - Reduces CO HBG ½ life from 4-5h to 1h

- **Pulmonary support**
  - Bronchodilators
  - Postural drainage
  - Intubation

- **Hyperbaric oxygen**
  - Controversial
  - Decreases CO Hgb ½ life to 30 minutes
  - Consider if COHb > 25%
Common substances involved in toxin exposure:

- Analgesics
  - Largest # deaths from poisoning reported

- Cleaning substances
Physiologic Response to Toxins

- Local
- Systemic
  - CNS
  - CV
  - Blood, hematopoietic organs
  - Visceral organs
    - Liver, kidney, lung
  - Skin
- Depends on the toxin
Goals of Care for Poison Ingestion

- A, B, C
- Toxic substance removed, eliminated, effects reversed
- LOC restored
- Organ function preserved
- Normothermia
Ingestion of poisons

- **Emesis**
  - Serious aspiration risk
  - Ipecac takes too long, vomit

- **Gastric lavage helpful if early**
  - Eliminate unabsorbed toxins
  - Not for caustic agents
  - 500-3000cc

- **Activated charcoal**
  - 20% patients will vomit
  - 1 gram/kg, mix with water
  - Give within 1-2h ingestion
  - Absorbs toxins

- **Hemodialysis**
Comatose adult treatment:

- 100ml D50
- Thiamine, 100mg IV
- Narcan, 2mg IV, IM repeat up to 10mg
  - Smaller dose (0.1-0.2mg) if not apneic
  - ½ life 30 minutes
Treatment

- **Antidotes:**
  - Opiates ⇒ Narcan
  - Acetaminophen ⇒ Acetylcysteine
  - Benzodiazepines ⇒ Flumazenil

- **Supportive:**
  - CV = temp, cardiac rhythm, hypovolemia
  - Pulmonary
  - Seizures: phenobarb or valium
  - ↑ ICP: mannitol, dexamethasone
Case Study:

- What are your priorities of care for Betty?
- Assessment
- Treatment
• Multisystem
  – Apply and incorporate all you’ve learned

• Questions????

• Good luck!