# Shock

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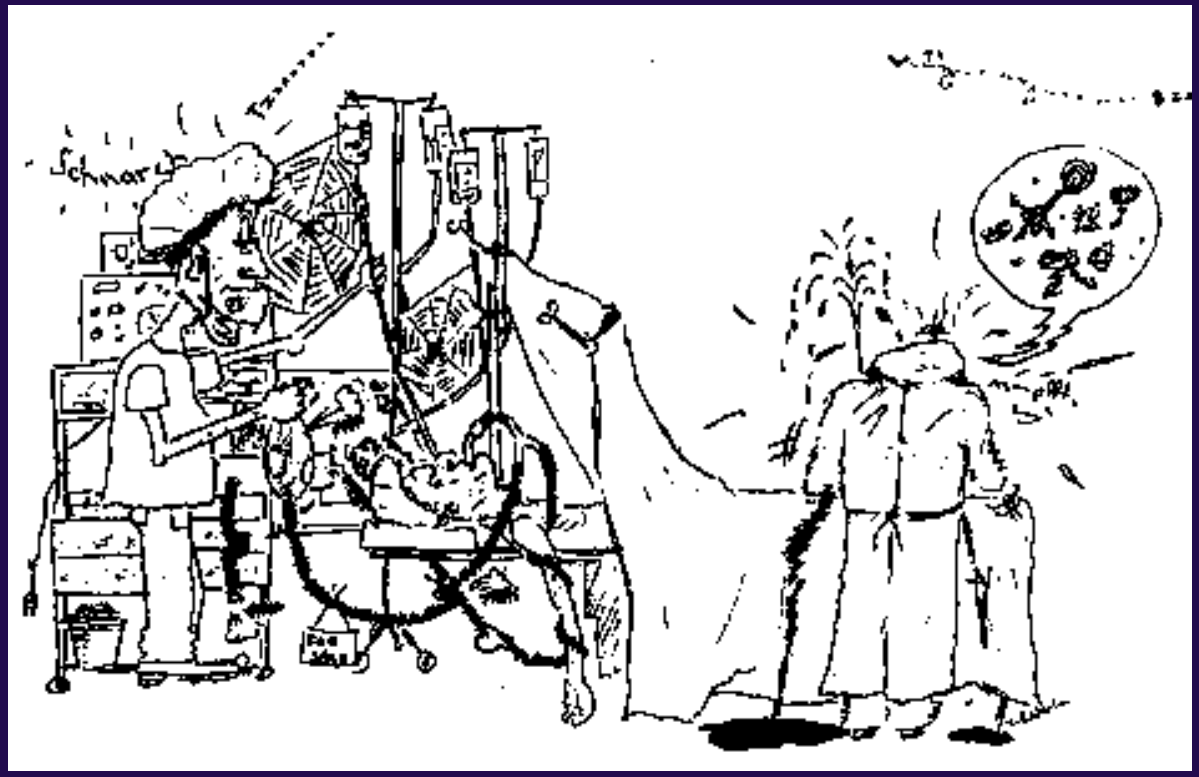
# Shock

**“A rude unhinging of the machinery of life”**



**“A brief pause in the act of dying”**

# Shock

**Defined: Inadequate Tissue Perfusion**

Inadequate peripheral perfusion leading to failure of tissue oxygenation

$\Rightarrow$may lead to anaerobic metabolism

# Shock

- Homeostasis
  - cellular state of balance
  - perfusion of cells with oxygen is one of its cornerstones

# Shock

- Adequate Cellular Oxygenation
  - Red Cell Oxygenation
  - Red Cell Delivery To Tissues

**Fick Principle**

# Fick Principle

**Air's gotta go in and out.**
**Blood's gotta go round and round.**

**Any variation of the above is not a good thing!**

# Shock

- Red Cell Oxygenation
  - Oxygen delivery to alveoli
    - Adequate $F_iO_2$
    - Patent airways
    - Adequate ventilation

# Shock

- Red Cell Oxygenation
  - Oxygen exchange with blood
    - Adequate oxygen diffusion into blood
    - Adequate RBC flow past alveoli
    - Adequate RBC mass/Hgb levels
    - Adequate RBC capacity to bind $O_2$
      - pH
      - Temperature

# Shock

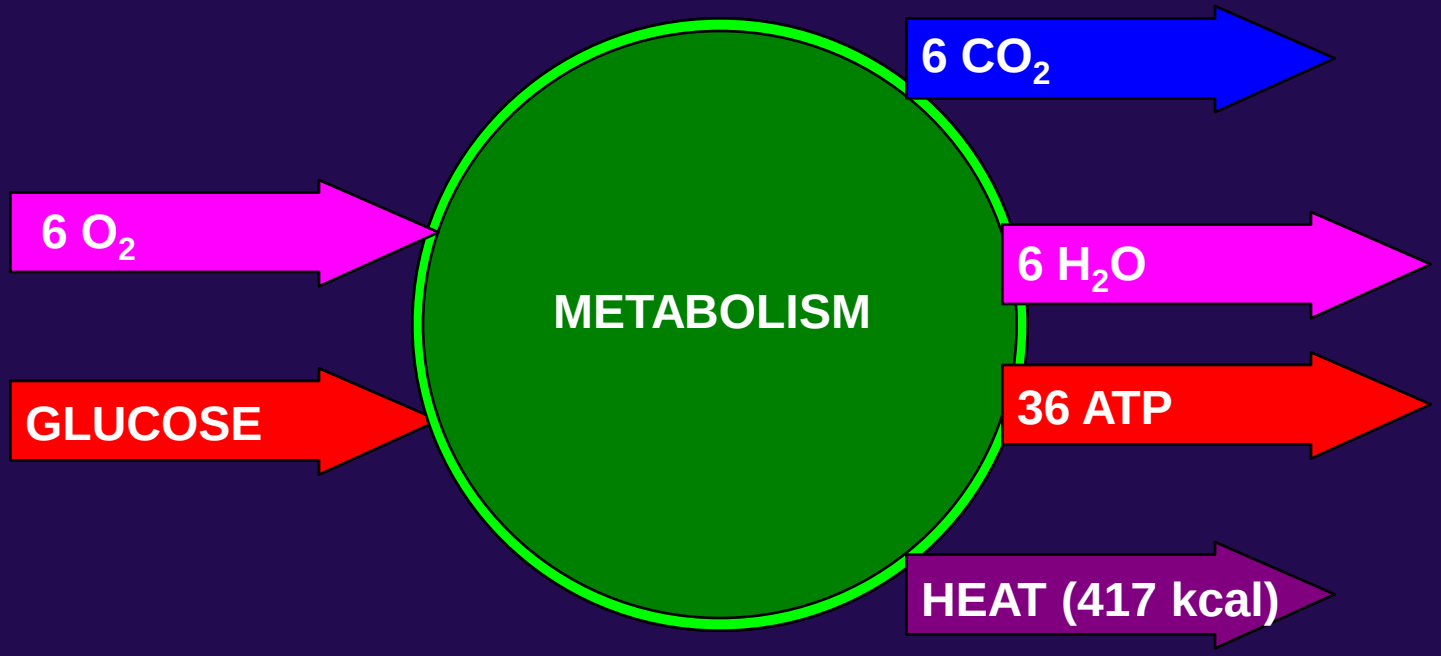
- Red Cell Delivery To Tissues
  - Adequate perfusion
    - Blood volume
    - Cardiac output
      - Heart rate
      - Stroke volume (pre-load, contractility, after-load)

# Shock

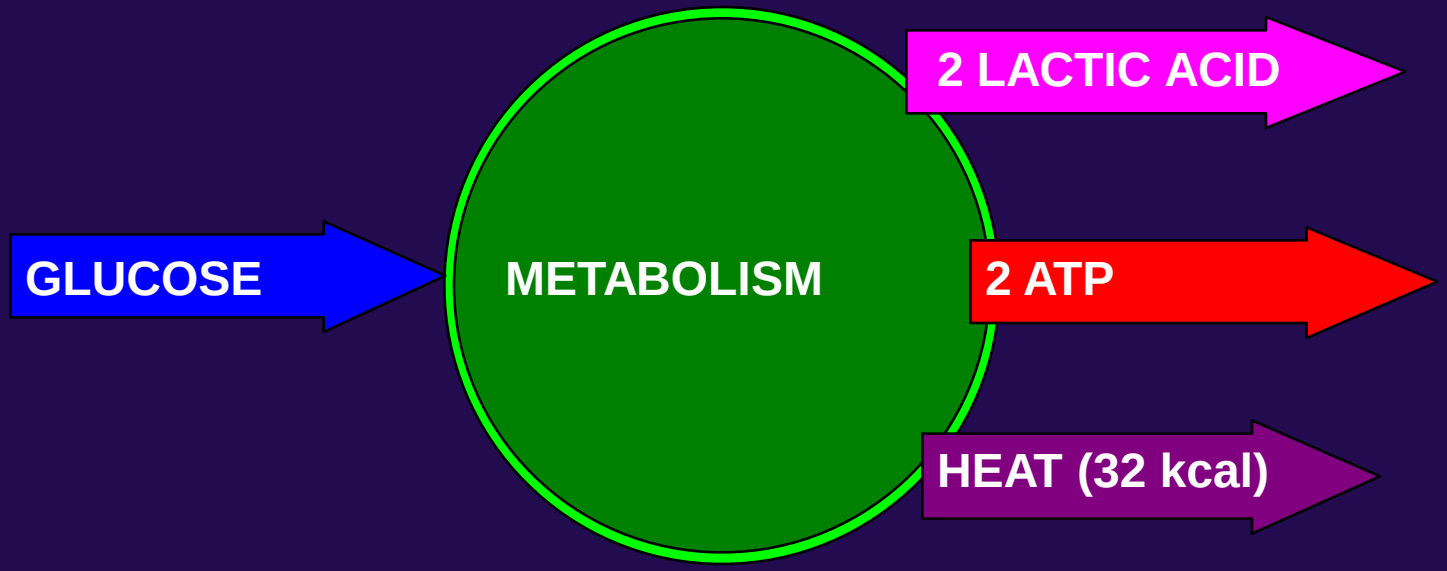
**Inadequate oxygenation or perfusion causes:**

- **Inadequate cellular oxygenation**
- **Shift from aerobic to anaerobic metabolism**

# AEROBIC METABOLISM



# ANAEROBIC METABOLISM

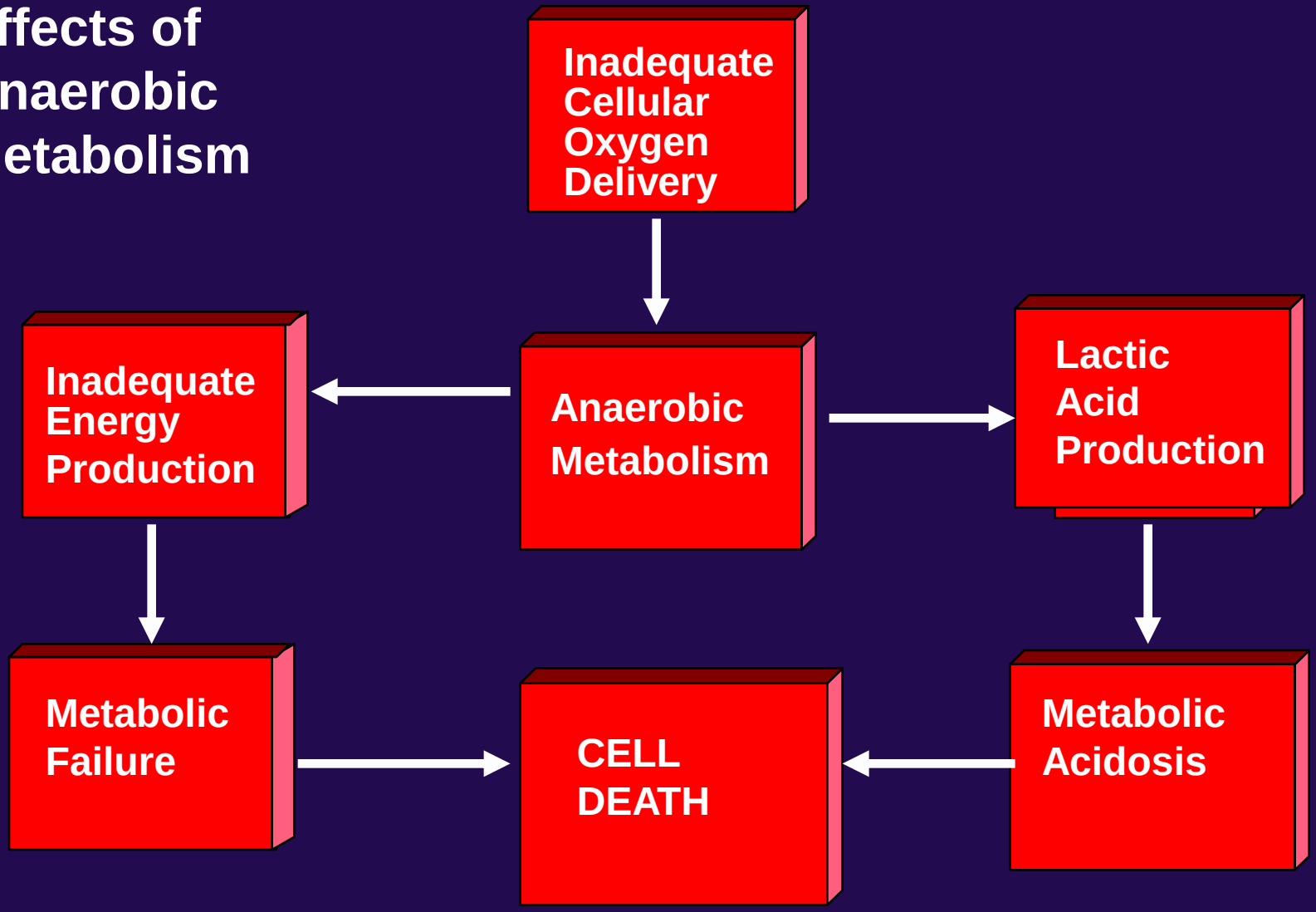



**Glycolysis: Break down of glycogen in the liver when the body is in anaerobic metabolism. Inefficient source of energy production; 2 ATP for every glucose; produces ketones and acid**

# Anaerobic Metabolism

- Occurs without oxygen
  - glycolysis can occur without oxygen
  - cellular death leads to tissue and organ death
  - can occur even after return of perfusion
    - $\Rightarrow$ organ or organism death

# Ultimate Effects of Anaerobic Metabolism



# Maintaining perfusion requires:

- Volume
- Pump
- Vessels
  - Failure of one or more of these causes shock

# Shock

- Hypovolemic Shock = Low Volume
  - –Trauma
  - –Non-traumatic blood loss
    - ♦GI
    - ♦GU
    - ♦Vaginal
  - –Burns
  - –Diarrhea
  - –Vomiting
  - –Diuresis
  - –Sweating
  - –Third space losses
    - ♦Pancreatitis
    - ♦Peritonitis
      - ♦ascites

# Shock

- Cardiogenic Shock = Pump Failure

–Acute MI
–CHF
–Bradyarrhythmias
–Tachyarrhythmias

–Mechanical obstruction
  - ♦Cardiac tamponade
  - ♦Tension pneumothorax
  - ♦Pulmonary embolism

# Shock

- Vasogenic (distributive) Shock = Low Resistance
  - Spinal cord trauma
    - neurogenic shock
  - Depressant drug toxicity
  - Simple fainting

# Shock

- Mixed Shock
  - Septic Shock
    - Overwhelming infection
    - Inflammatory response occurs
    - Blood vessels
      - Dilate (loss of resistance)
      - Leak (loss of volume)

# Shock

- Mixed Shock
  - Septic Shock
    - Fever
      - Increased $O_2$ demand
      - Increased anaerobic metabolism
    - Bacterial toxins
      - Impaired tissue metabolism

# Shock

- Mixed Shock
  - Anaphylactic Shock
    - Severe allergic reaction
    - Histamine is released
    - Blood vessels
      - Dilate (loss of resistance)
      - Leak (loss of volume)
    - Extravascular smooth muscle spasm
      - Laryngospasm
      - Bronchospasm

# Shock

- Progressive syndrome
- Three phases
  - Compensated
  - Decompensated
  - Irreversible

# Shock

- **Signs and symptoms due to:**
  - **Hypoperfusion**
  - **Compensatory responses**

# Compensated Shock

- Baroreceptors detect fall in BP
  - Usually 60-80 mm Hg (adult)
- Sympathetic nervous system activates
  - What are the primary SNS Neurotransmitters & their effects?
    - ACh
    - Epi Norepi

# Compensated Shock

- Cardiac effects
  - Increased force of contractions
  - Increased rate
  - Increased cardiac output

# Compensated Shock

- Peripheral effects
  - Arteriolar constriction
  - Pre-/post-capillary sphincter contraction
  - Increased peripheral resistance
  - Shunting of blood to core organs

# Compensated Shock

- Decreased renal blood flow
  - Renin released from kidney arteriole
  - Renin & Angiotensinogen combine
  - Converts to Angiotensin I
  - Angiotensin I converts to Angiotensin II
    - Peripheral vasoconstriction
    - Increased aldosterone release (adrenal cortex)
      - promotes reabsorption of sodium & water

# Compensated Shock

- Decreased blood flow to hypothalamus
- Release of antidiuretic hormone (ADH or Arginine Vasopressin) from posterior pituitary
  - Retention of salt, water
  - Peripheral vasoconstriction

# Compensated Shock

- Insulin
  - ⇓ secretion caused by epinephrine
  - contributes to hyperglycemia
- Glucagon
  - ⇑ release caused by epinephrine
  - promotes liver glycogenolysis & gluconeogenesis
    - Body's releasing glycogen stores to produce glucose.

# Compensated Shock

- Peripheral capillaries contain minimal blood
- Stagnation
- Aerobic metabolism changes to anaerobic
- Extracellular potassium shifts begin

# Compensated Shock

- Presentation
  - Restlessness, anxiety
    - Earliest sign of shock
  - Tachycardia
    - Bradycardia in cardiogenic, neurogenic

# Compensated Shock

- Presentation
  - Normal BP, narrow pulse pressure
  - Falling BP = late sign of shock
  - Mild orthostatic hypotension (15 to 25 mm Hg)
  - “Possible” delay in capillary refill

# Compensated Shock

- Presentation
  - Pale, cool skin
    - Cardiogenic
    - Hypovolemic
  - Flushed skin
    - Anaphylactic
    - Septic
    - Neurogenic

# Compensated Shock

- Presentation
  - Slight tachypnea
  - Respiratory compensation for metabolic acidosis

# Compensated Shock

- Presentation
  - Nausea, vomiting
  - Thirst
  - Decreased body temperature
  - Feels cold
  - Weakness

# Decompensated Shock

- Presentation
  - Cardiac Effects
    - Decreased RBC oxygenation
    - Decreased coronary blood flow
    - Myocardial ischemia
    - Decreased force of contraction

# Decompensated Shock

- Presentation
  - Peripheral effects
    - Relaxation of precapillary sphincters
    - Continued contraction of postcapillary sphincters
    - Peripheral pooling of blood
    - Plasma leakage into interstitial spaces

# Decompensated Shock

- Presentation
  - Peripheral effects
    - Continued anaerobic metabolism
    - Continued increase in extracellular potassium
    - Rouleaux formations of RBCs
      - “pile up like coins”
    - Cold, gray, “waxy” skin

# Decompensated Shock

- Presentation
  - Listlessness, confusion, apathy, slow speech
  - Tachycardia; weak, thready pulse
  - Decreased blood pressure
  - Moderate to severe orthostatic hypotension
  - Decreased body temperature
  - Tachypnea

# Irreversible Shock

- Post-capillary sphincter relaxation
- Loss of peripheral vascular resistance

# Irreversible Shock

- Washout of accumulated products
  - Hydrogen ion
  - Potassium
  - Rouleaux formations
  - Carbon dioxide
- Systemic metabolic acidosis occurs
- Cardiac Output decreases further

# Irreversible Shock

- Presentation
  - Confusion, slurred speech, unconscious
  - Slow, irregular, thready pulse
  - Falling BP; diastolic goes to zero
  - Cold, clammy, cyanotic skin
  - Slow, shallow, irregular respirations
  - Dilated, sluggish pupils
  - Severely decreased body temperature

# Irreversible Shock

- Irreversible shock leads to:
  - Renal failure
  - Hepatic failure
  - Disseminated intravascular coagulation (DIC)
  - Multiple organ systems failure
  - Adult respiratory distress syndrome (ARDS)
  - Death

# Disseminated Intravascular Coagulation (DIC)

- Decreased perfusion causes tissue damage/necrosis
- Tissue necrosis triggers diffuse clotting
- Diffuse clotting consumes clotting factors
- Fibrinolysis begins
- Severe, uncontrolled systemic hemorrhage occurs

# Adult Respiratory Distress Syndrome (ARDS)

- AKA: “Shock Lung”
- Decreased perfusion damages alveolar and capillary walls
- Surfactant production decreases
- Fluid leaks into interstitial spaces and alveoli
- Gas exchange impaired
- Work of breathing increases

# Key Issues In Shock

- Tissue ischemic sensitivity
  - Heart, brain, lung: 4 to 6 minutes
  - GI tract, liver, kidney: 45 to 60 minutes
  - Muscle, skin: 2 to 3 hours

**Resuscitate Critical Tissues First!**

# Key Issues In Shock

- Recognize & Treat during compensatory phase

*Restlessness, anxiety, combativeness = Earliest signs of shock*

**Best indicator of resuscitation effectiveness = Level of Consciousness**

# Key Issues In Shock

- Falling BP = LATE sign of shock
- BP is NOT same thing as perfusion
- Pallor, tachycardia, slow capillary refill = Shock, until proven otherwise

# General Shock Management

- Airway
  - Open, Clear, Maintained
  - Consider Intubation

# General Shock Management

- High concentration oxygen
  - Oxygen = Most Important Drug in Shock
- Assist ventilation as needed
  - When in Doubt, Ventilate
    - BVM
- Decompress Tension Pneumothorax

# General Shock Management

- Establish venous access
  - Replace fluid
  - Give drugs, as appropriate
  - Don’t delay definitive therapy
- Maintain body temperature
  - Cover patient with blanket if needed
  - Avoid cold IV fluids

# General Shock Management

- Monitor
  - Mental Status
  - Pulse
  - Respirations
  - Blood Pressure
  - ECG

# Hypovolemic Shock

- Control severe external bleeding
- Elevate lower extremities
- Avoid Trendelenburg

# Hypovolemic Shock

- Fluid Resuscitation aimed at *permissive hypotension*
- 2 Large Bore IV's of NS or LR
- Maintain BP of 90 mmHg
- Some EMS Systems still aim for BP of 100 systolic for fluid resuscitation
  - Not current ACS recommendation

# Permissive Hypotension Physiology

- Body has several protective measures with BP at this level
- Increasing BP causes more rapid blood loss
- Increasing BP may cause clot to dislodge increasing more bleeding
  - Especially if bleeding has stopped
- Still being researched

# Hypovolemic Shock

- **Do NOT delay transport**
- **Start IVs enroute to hospital**

**Where does stabilization of critical trauma occur?**

# Cardiogenic Shock

- Supine, or head and shoulders slightly elevated
- Do NOT elevate lower extremities

# Cardiogenic Shock

- Keep open line, micro-drip set
- Fluid challenge based on cardiovascular mechanism and history
  - Titrate to BP ~ 90 mm Hg

# Cardiogenic Shock

- Treat the underlying cause if possible
- Treat rate, then rhythm, then BP
  - ❶Correct bradycardia or tachycardia
  - ❷Correct irregular rhythms
  - ❸Treat BP
    - Cardiac contractility
      - Dobutamine, Dopamine
    - Peripheral resistance
      - Dopamine, Norepinephrine

# Obstructive Shock

-Treat the underlying cause

- Tension Pneumothorax
- Pericardial Tamponade

– Isotonic fluids titrated to BP w/o pulmonary edema

– Control airway

- Intubation

# Shock Management

**Avoid vasopressors until hypovolemia ruled out, or corrected**

# Shock Management

**Squeezing partially empty tank can cause ischemia, necrosis of kidney and bowel**

# Vasogenic Shock

- Consider need to assist ventilations
- Patient supine; lower extremities elevated
- Avoid Trendelenburg

# Vasogenic Shock

- Infuse isotonic crystalloid
  - "Top off tank"
- Consider possible hypovolemia
- Consider vasopressors
  - Dopamine
  - Levophed preferred for septic shock
    - or Phenylephrine (Neo-synephrine)
    - Rarely Epi drips

# Vasogenic Shock

- Maintain body temperature
- Hypothermia may occur

# Vasogenic Shock

- Anaphylaxis
  - Suppress inflammatory response
    - Antihistamines
    - Corticosteroids
  - Oppose histamine response
    - Epinephrine
      - bronchospasm & vasodilation
  - Replace intravascular fluid
    - Isotonic fluid titrated to BP ~ 90 mm

# Shock in Children

- Small blood volume
  - Increased hypovolemia risk
- Very efficient compensatory mechanisms
  - Failure may cause “sudden” shock
- Pallor, altered LOC, cool skin = shock UPO

# Shock in Children

- Avoid massive fluid infusion
  - Use 20 cc/kg boluses
- High surface to volume ratio
  - Increased hypothermia risk

# Shock in the Elderly

- Poor cardiovascular condition
  - Rapid decompensation
- Sepsis more likely
- Hypoperfusion can cause:
  - CVA
  - AMI
  - Seizures
  - Bowel Infarctions
  - Renal failure

# Shock in the Elderly

- Assessment more difficult
  - Peripheral vascular disease
  - Weak pulses
  - Altered sensorium
  - Hypertension masking hypoperfusion
  - Beta-blockers masking hypoperfusion
- Fluid infusion may produce volume overload/CHF

# Shock in OB Patients

- Pulse increases 10 to 15 bpm
- BP lower than in non-pregnant patient
- Blood volume increased by 45%
  - Slower onset of shock signs/ symptoms
- Fluid resuscitation requires greater volume

# Shock in OB Patients

- Oxygen requirement increased 10 to 20%
- Pregnant uterus may compress vena cava, decreasing venous return to heart
  - Place women in late-term pregnancy on left-side
- Fetus can be in trouble even though mother looks well-perfused

# Transport Considerations

- Indications for Rapid Transport
- Indications for Trauma Center Transport
- Considerations for Air Medical Transport

- Questions?