Little Rock Critical Care Program

CCRN/PCCN Review

October 30th

Day 1 Oct 30th		
0800-0810	Course Overview	*Lynnette Flynn*
0810-1200	Respiratory Pulmonary edema Pulmonary embolus ARDS Respiratory infection COPD (bronchitis, emphysema, status asthmaticus Ventilation complications Pleural space abnormalities Hemorrhage PHTN Trauma Thoracic surgery	Kelly
1200-1245	*Lunch*	
1245-1345	Multisystem OB complications (eclampsia, HELLP, maternal/fetal transfusion, placental abruption, placenta previa TTM/ Malignant hyperthermia Comorbidity with transplant history EOL, palliative care, failure to thrive Bariatrics complications Pain Sleep disruption Submersion injuries	Kelly Urban
1345-1500	Musculoskeletal / Trauma / Burns / Rhabdo Compartment syndrome Fractures Muscular deconditioning Musculoskeletal trauma, Rhabdomyolysis	Kelly Urban
1500-1630	Shock, Sepsis, MODS Anoxic injury Burns Multi-organ dysfunction syndrome Sepsis Shock states (distributive, obstructive, septic) SIRS	Kelly

Respiratory Monitoring & Beyond:

ABGs, SPO_2, & $ETCO_2$

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Arterial Blood Gas

- Arterial blood gases are used to measure the amount of oxygen, carbon dioxide, and bicarbonate in the blood, as well as the pH.
- ABGs provide information regarding physiologic phenomena

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Acids/Bases

Acids

- Substances capable of releasing a hydrogen ion (H^+) into solution.
- Volatile acids
  - excreted through the lungs (CO_2)
- Fixed or nonvolatile acids
  - excreted by the kidneys (ketoacids and lactic acid)

Bases

- Substances capable of combining with H^+ in solution.
- Bicarbonate (HCO_3)
  - Most important base in the blood
  - regulated by the kidneys
- Hemoglobin and plasma proteins.
- Bases are reflected in the ABGs as the HCO_3 and the base excess or base deficit.

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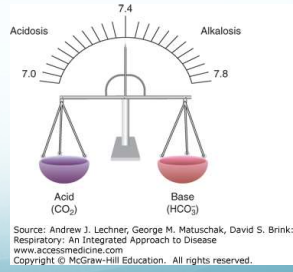
Elements of ABGs: Normal Values

- pH--7.35 to 7.45
  - represents a combined effect of metabolic and respiratory factors.
  - low pH indicates acidosis
  - high pH indicates alkalosis



Source: Andrew J. Lechner, George M. Matuschak, David S. Brink: Respiratory: An Integrated Approach to Disease
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Elements of ABGs: Normal Values

- PCO_2--35 to 45 mmHG
  - A measure of the partial pressure of carbon dioxide dissolved in the plasma.
  - byproduct of metabolism
  - CO_2 is excreted by the lungs and is a measure of the adequacy of ventilation.
  - CO_2 functions as an acid because it combines with water to produce carbonic acid, H_2CO_3.

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Elements of ABGs: Normal Values

- HCO_3--22 to 26 mEq/L
  - Bicarbonate ion is a base regulated by the kidneys
  - It may be adjusted to compensate for respiratory acid-base imbalance, or may be altered by other factors such as kidney disease or metabolic alterations

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Elements of ABGs: Normal Values

- Base Excess (BE) -2 to +2
  - It represents the combined effects of HCO_3 and other bases--plasma proteins, hemoglobin and others
  - A negative base excess is sometimes referred to as a base deficit.

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Elements of ABGs: Normal Values

- PaO_2--80-100 mmHg
  - Is the partial pressure of oxygen dissolved in arterial plasma
  - Only about 1% of total oxygen content is carried in this state, PaO_2 indicates how well oxygen is being taken up in the lungs.

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Elements of ABGs: Normal Values

- SaO_2--95 to 98%
  - SaO_2 represents the percentage of total hemoglobin which is saturated with oxygen.
  - The vast majority of oxygen is carried in this state.
  - While saturation is usually well-correlated with PaO_2, some conditions (pH, temperature) can influence the relationship between these two parameters

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ABG: Summary of Normal Values

pH	**7.35 – 7.45**
PaO_2	**80 – 100 mmHg**
PCO_2	**35 – 45 mmHg**
HCO_3	**22 – 26 mEq/L**
Base Excess (BE)	**-2 - +2**
SaO_2	**95% - 98%**

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Steps in ABG Interpretation

1. Check pH
   - acidotic, alkalotic, or normal
2. Check $PaCO_2$ (respiratory parameter)
   - Elevated (acidotic), decreased (alkalotic), or normal
3. Check HCO_3 (metabolic parameter)
   - Elevated (alkalotic), decreased (acidotic), or normal
4. If abnormalities exist, determine which of the major acid/base imbalances is present

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Steps in ABG Interpretation Cont'd

5. Determine whether any compensation mechanisms are involved
6. Check PO_2 and O_2 saturation
   - normal, elevated, or decreased
7. Observe patient
   - evaluate vital signs and physical parameters

- Evaluate why patient presents any abnormal values which are present and implement appropriate actions to correct the acid/base imbalance

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Respiratory Acidosis (Elevated $PaCO_2$)

Caused by **hypoventilation** of any etiology

- Obstructive Lung Disease (COPD, sleep apnea)
- Oversedation, head trauma, anesthesia, or reduced function of respiratory center
- Neuromuscular disorders
- Chest Trauma (pneumothorax, flail chest)
- Inappropriate mechanical ventilation

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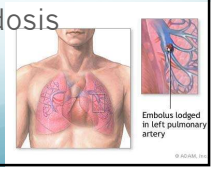
Respiratory Alkalosis (Low $PaCO_2$)

Caused by **hyperventilation** of any etiology

- Hypoxemia
- Nervousness and anxiety
- Pulmonary Embolus
- Pulmonary Edema
- Response to respiratory stimulants (salicylates, theophylline, catecholamines)
- Inappropriate mechanical ventilation
- Compensation for metabolic acidosis
- Pregnancy



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Metabolic Alkalosis (Elevated HCO_3)

- Caused by a loss of nonvolatile acid or increase in HCO3
- Gastric loss of acid (vomiting, prolonged gastric suctioning)
- HCO3 during cardiac arrest
- Baking soda, antacids
- Massive blood transfusion – citrate – lactate · bicarbonate
- Increased excretion of H+, K+, and Cl – due to
  1. Diuretics
  2. Cushings Syndrome
  3. Corticosteroids
  4. Aldosteronism

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Metabolic Acidosis (Low HCO_3)

- Increase in immeasurable anions (high anion gap):
  - Diabetic ketoacidosis
  - Starvation
  - Renal failure
  - Lactic Acidosis
  - Poisoning: salicylates, ethylene glycol, methyl alcohol, paraldehyde
- No increase in immeasurable anions:
  - Diarrhea
  - Drainage of pancreatic fluids
  - Treatment with diamox
  - Treatment with ammonium chloride
  - Renal Tubular Acidosis
  - Hyperalimentation

Caused by a gain in nonvolatile acid which uses up HCO3 or loss of HCO3.

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Clinical Signs of Acidosis (CNS Depression)

- Depressed thought processes
- Delayed reaction times
- Slurred speech
- Somnolence
- Uncoordination
- Confusion
- Semi-coma
- Death

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Clinical Signs of Alkalosis (CNS-Excitation)

- Anxiety
- Paresthesia
- Tremors
- Nausea
- Tetany
- Convulsions
- Death

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Respiratory Acidosis

- pH is low and PaCO2 is high

pH	**7.30** **ACID**
PCO_2	**65** **ACID**
PO_2	**90**
HCO_3-	**26**
BE	**0**
SaO_2	**95%**

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Respiratory Alkalosis

- High pH along with a low $PaCO_2$

pH	**7.5** **BASE**
PCO_2	**30** **BASE**
PO_2	**90**
HCO_3-	**26**
BE	**0**
SaO_2	**95%**

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Metabolic Acidosis

- pH is low with a low HCO_3^- and/or BE

pH	**7.30 ACID**
PCO_2	**35**
PO_2	**92**
HCO_3-	**18 ACID**
BE	**-3 ACID**
SaO_2	**97%**

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Metabolic Alkalosis

- High pH along with a high HCO_3^- and/or BE

pH	**7.5 BASE**
PCO_2	**40**
PO_2	**95**
HCO_3-	**35 BASE**
BE	**+3 BASE**
SaO_2	**96%**

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Compensation

- Respiratory acidosis due to increased $PaCO_2$
  - Compensation: Kidneys excrete more acid and less HCO_3^- resulting in increased HCO_3^-
- Respiratory alkalosis due to decreased $PaCO_2$
  - Compensation: Kidneys excrete HCO_3^-
- Metabolic acidosis due to decreased HCO_3^-
  - Compensation: Hyperventilation to decrease $PaCO_2$
- Metabolic alkalosis due to increased HCO_3^-
  - Compensation: Hypoventilation to increase $PaCO_2$

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Compensation Cont.

- There are two types of compensation
  - Partial Compensation
    - pH, pCO_2, and Bicarb are all abnormal
  - Full Compenstion
    - pH is normal, pCO_2 and Bicarb are abnormal

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ABG Summary

Primary Disorder	Cause	Compensation	Effect on ABGs
Metabolic Acidosis	•Excess nonvolatile acids •Bicarbonate deficiency	Rate & depth of respirations increase → eliminates additional CO2	↓ pH ↓ HCO3 ↓ PaCO2
Metabolic Alkalosis	•Bicarbonate excess	Rate & depth of respirations decrease → retaining CO2	↑ pH ↑ HCO3 ↑ PaCO2
Respiratory Acidosis	•Retained CO2 & excess carbonic acid	Kidneys conserve bicarbonate to restore carbonic acid : bicarbonate ratio 1:20	↓ pH ↑ PaCO2 ↑ HCO3
Respiratory Alkalosis	•Loss of CO2 & deficient carbonic acid	Kidneys excrete bicarbonate and conserve H+ to restore carbonic acid : bicarbonate ratio	↑ pH ↓ PaCO2 ↓ HCO3

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Let's Practice

- pH 7.18
- $pC0_2$ 34
- $HC0_3$ 12
- $Pa0_2$ 84
- $Fi0_2$ .21
- P/F ratio 400

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Let's Practice

- pH 7.22
- $pC0_2$ 59
- $HC0_3$ 35
- $Pa0_2$ 35
- $Fi0_2$ .21
- P/F Ratio 167

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Let's Practice

pH	**7.42**
PCO_2	**50**
PO_2	**80**
HCO_3-	**32**
BE	**2.5**
SaO_2	**95%**

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Let's Practice

pH	**7.37**
PCO_2	**32**
PO_2	**90**
HCO_3	**18**
BE	**-2.5**
SaO_2	**98%**

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Let's Practice

pH	**7.39**
PCO_2	**64**
PO_2	**65**
HCO_3	**37**
$Fi0_2$	**.30**
P/F Ratio	**217**

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Let's Practice

pH	**7.45**
PCO_2	**27**
PO_2	**65.5**
$HC0_3$	**19.1**
$Fi0_2$	**.40**
$SP0_2$	**.88**

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Physiologic Phenomena

- Oxygenation
  - Ability of the lungs to deliver fresh O_2 to the blood in the pulmonary capillary beds
  - Reflected in the partial pressure of oxygen (PaO_2) and the percent saturation of oxygen (SaO_2) in the arterial blood

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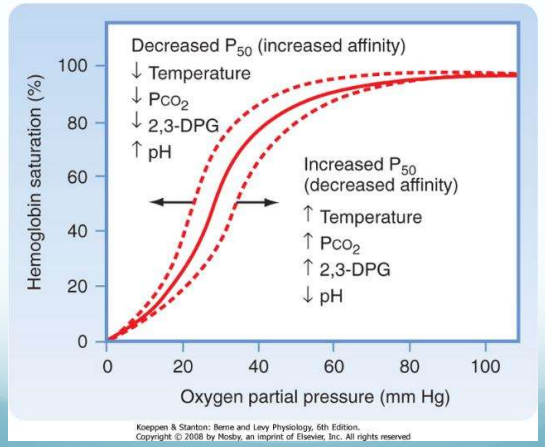
Oxyhemoglobin Dissociation Curve



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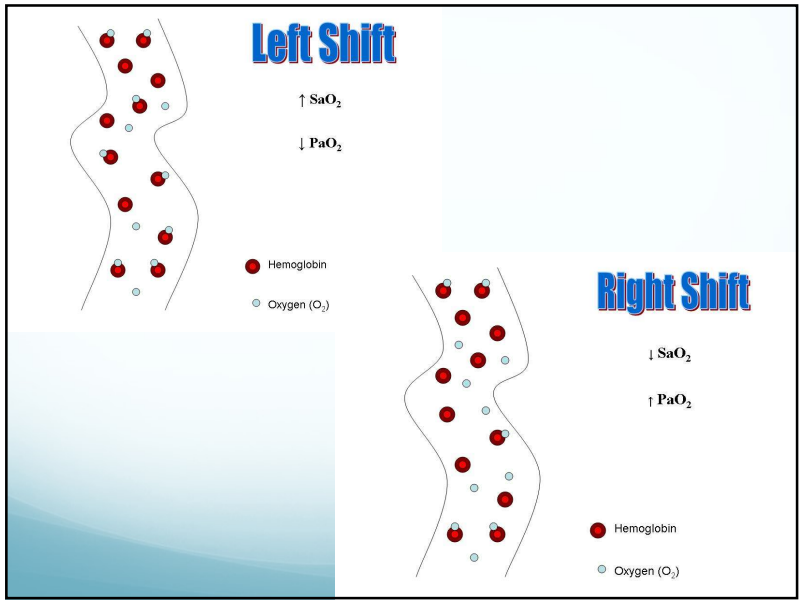

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ETCO2

- The ventilation vital sign

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How $etCO_2$ Works

- $ETCO_2$ monitoring determines the CO_2 concentration of exhaled gases
- Photo detector measures the amount of infrared light absorbed by airway gases during inspiration and expiration
  - CO_2 molecules absorb specific wavelengths of infrared light energy
  - Light absorption increases directly with CO_2 concentration
- A monitor converts this data to a CO_2 value and a corresponding waveform (capnograph)

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Respiratory Cycle

- **Cellular Metabolism** of food into energy → O_2 consumption and CO_2 production
- **Transport** of O_2 and CO_2 between cells and pulmonary capillaries, and diffusion from/into alveoli
- **Ventilation** between alveoli and atmosphere

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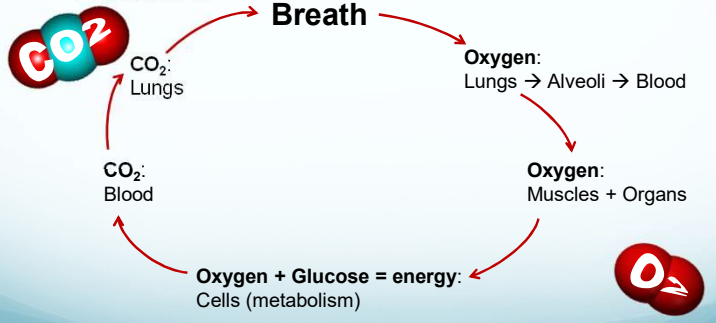
Respiratory Cycle

- **Must have all 3: Metabolism, Perfusion, & Ventilation!**



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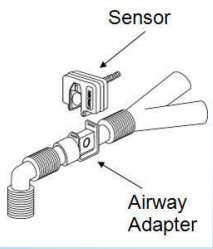
2 Techniques for Monitoring $ETCO_2$

- Mainstream (Flow-through or In-line)
- Sidestream (aspiration)



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$ETCO_2$ Values

- Normal: 35-45 mmHg
- > 45 = Hypoventilation
- < 35 = Hyperventilation

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Why use $etCO_2$?

Pulse Oximetry

- Measures saturation of Hemoglobin with Oxygen
- Reflects Oxygenation
- SPO_2 changes lag when patient is hypoventilating or apneic
- Should be used with Capnography

Capnography

- Carbon Dioxide
- Reflects Ventilation
- Hypoventilation/apnea detected immediately
- Should be used with Pulse Oximetry

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Factors that affect CO_2 levels:

System:	INCREASE IN $ETCO_2$	DECREASE IN $ETCO_2$
Metabolism	• Increased muscular activity (shivering) • Malignant hyperthermia	• Decreased muscular activity (muscle relaxants) • Hypothermia
Perfusion	• Increased cardiac output (during resuscitation) • Bicarbonate infusion • Tourniquet release	• Decreased cardiac output • Pulmonary embolism
Ventilation	• Effective drug therapy for bronchospasm • Decreased minute ventilation (hypoventilation) • Malfunctioning exhalation valve	• Bronchospasm • Increased minute ventilation (hyperventilation) • Circuit leak or partial obstruction • Poor sampling technique

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Questions?

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16. Which of the following ABG values would be most indicative of a diagnosis of acute respiratory failure?

	pH	pCO2	pO2	HCO3
A.	7.18	70	54	26
B.	7.18	80	63	42
C.	7.26	55	80	24
D.	7.34	45	65	23

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21. Which of the following is a correct statement about a shift of the oxyhemoglobin dissociation curve to the right?

A. It can result from an increase in blood pH

B. It can result from an increase in body temperature

C. It results in less oxygen being unloaded from hemoglobin molecules

D. It results in 100% saturation of hemoglobin

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103. Which of the following best defines hypoventilation?

A. An RR less than 10

B. A pCO2 greater than 45

C. A pO2 less than 75

D. An arterial pH greater than 7.35

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106. An ABG sample obtained while a patient is breathing room air reveals the following:

- pH 7.18
- pCO2 80
- pO2 35
- HCO3 29

The ABG indicates:

A. Respiratory acidosis with mild hypoxemia

B. Respiratory acidosis with severe hypoxemia

C. Combined respiratory and metabolic acidosis with mild hypoxemia

D. Combined respiratory and metabolic acidosis

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A patient is admitted to the MICU with the following arterial blood gas results

- pH 7.55
- CO2: 28 mmHg
- PaO2: 88 mmHg
- HCO3: 26 mEq/L

- What is the interpretation?

A. Respiratory Acidosis

B. Compensated Metabolic Alkalosis

C. Non-compensated Respiratory Alkalosis

D. Metabolic Alkalosis

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Which of the following is a complication of mechanical ventilation and peak end expiratory pressure (PEEP) therapy?

A. Atelectasis
B. Oxygen toxicity
C. Reduced cardiac output
D. Acute Respiratory Distress Syndrome

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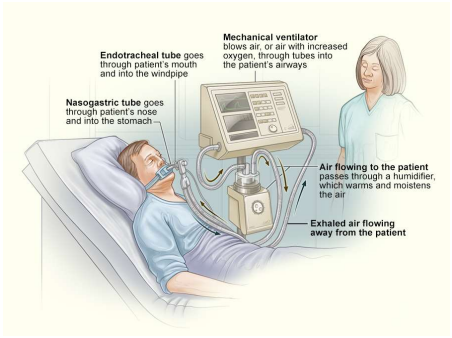

Ventilation Strategies

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UAMS MEDICAL CENTER

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Content

- Modes of Mechanical Ventilation
- Prone positioning
- Noninvasive Ventilation
- Mechanical Ventilation
  - Prevention of Complications
  - Therapeutic Interventions
- Tracheostomy
- Therapeutic Gases
  - Nitric Oxide
  - Heliox

2

Lung Volumes/ Capacities Definitions

Total Lung Capacity (TLC): Volume of gas contained in the lung at the end of maximal inspiration

Vital Capacity (VC): Maximal volume of gas that can be expelled from the lungs following a maximal inspiration

Inspiratory Capacity (IC): Maximal volume of gas that can be inspired from the resting expiratory level

Functional Residual Capacity (FRC): Volume of gas remaining in the lungs at resting end expiration

3

Common Basic Ventilator Settings

- FiO_2 - fraction of inspired Oxygen (21% - 100%).
- Tidal Volume - amt of air that the ventilator has been set to deliver to the patient with each breath.
  - Healthy Lungs: 6-8 ml/kg
  - ALI/ARDS: 5-6 ml/kg
- Respiratory rate- Number of positive pressure breaths the ventilator delivers per minute.

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PEEP

- Allows the patient to exhale while maintaining a preset positive pressure at the end of expiration.
- Allows for greater gas exchange before the next breath. It is the application of positive pressure to the airway at end expiration.
- First used early in the 1970s as a treatment for Respiratory distress syndrome in newborns

Auto-PEEP: occurs when expiration is not long enough to empty the lungs

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Inspiratory Time: Expiratory Time Relationship (I:E Ratio)

- During spontaneous breathing, the normal I:E ratio is 1:2, indicating that for normal patients the exhalation time is about twice as long as inhalation time.
- If exhalation time is too short "breath stacking" occurs resulting in an increase in end-expiratory pressure also called auto-PEEP.
- Depending on the disease process, such as in ARDS, the I:E ratio can be changed to improve ventilation

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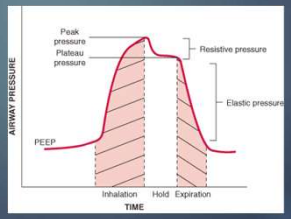
Pressures

- PEAK Airway Pressures
  - Measured at airway opening
  - Norm: < 40
- PLATEAU Pressures
  - Measured at end of inspiration
  - Norm: < 30



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Conventional Modes of Mechanical Ventilation

- Ventilation Modes considerations:
  - Trigger: What controls the tidal breath?
    - Pressure or Volume
  - Limit: What determines the size of the breath?
  - Cycle: What actually ends the breath?
    - Usually time

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Conventional Modes of Mechanical Ventilation – Volume Modes

- Tidal volume and minute ventilation are ensured
  - Volume is preset
  - Pressure varies with patient compliance and resistance
- Assist-Control Ventilation (ACV)
  - CMV
  - **All breaths** (patient initiated and pre-set) are same volume
- Synchronized Intermittent Mandatory Ventilation (SIMV)
  - **Pre-set breaths are set volume**, patient-initiated volume determined by patient
  - Vent synchronizes the mandatory breaths with patient's own breaths

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Conventional Modes of Mechanical Ventilation – Pressure Modes

- Tidal volume is determined by the selected pressure level, airway resistance, and lung compliance
  - Pressure is preset
  - Volume varies
- Pressure Controlled Ventilation (PCV)
  - Does not allow for patient-initiated breaths
  - Applies constant pressure for a preset time
  - Variable tidal volumes (flow depends on lung resistance, lung compliance, and patient effort)
  - Used in ARDS to reduce barotrauma
- Pressure Support Ventilation (PSV)
  - Patient determines inflation volume and respiratory rate
  - Used to augment spontaneous breathing

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Conventional Modes of Mechanical Ventilation – Pressure Modes Continued

- Airway Pressure Release Ventilation (APRV)
  - Bi-level mode providing 2 levels of CPAP with an inverse ratio (very short expiration time – inverse ratio)
  - Requires increased amounts of sedation
  - Considered Rescue Method for patients with lung compliance and oxygenation issues
    - Helps prevent alveolar collapse and maintain recruitment
  - Risks:
    - Pneumothorax
    - Ventilator trauma

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Conventional Modes of Mechanical Ventilation – Dual modes

- Pressure Regulated Volume Control (PRVC)
  - A control mode, which delivers a set tidal volume with each breath at the lowest possible peak pressure.
  - Delivers the breath with a decelerating flow pattern that is thought to be less injurious to the lung…… "the guided hand".

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Ventilator Modes

MODE	FUNCTION	CLINICAL USE
Assist-Control Ventilation (A/C)	Delivers breath in response to patient effort and if patient fails to do so within preset amount of time	Usually used for spontaneously breathing patients with weakened respiratory muscles
Synchronous Intermittent Mandatory Ventilation (SIMV)	Ventilator breaths are synchronized with patient's respiratory effort	Usually used to wean patients from mechanical ventilation
Pressure Controlled Ventilation (PCV)	Pressure limited ventilation; can be combined with inverse ratio (watch for auto-PEEP)	
Pressure Support Ventilation (PSV)	Preset pressure that augments the patient's inspiratory effort and decreases breathing work	Often used with SIMV during weaning
Airway Pressure Release Ventilation (APRV)	Biphasic ventilation with short expiratory time	
Pressure Regulated Volume Control (PRVC)		

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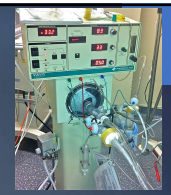
Non-conventional Modes of Mechanical ventilation



- High Frequency Oscillatory Ventilation (HFO)
  - Used for refractory hypoxemia
  - Oscillates the lung around a constant mean airway pressure higher than conventional ventilation
  - Delivers breaths at high frequencies and low tidal volumes
    - Hertz (Hz): 1 Hz = 60 breaths
  - Disadvantages:
    - Require heavy sedation/neuromuscular blockade
    - Cannot transport patient
    - Cannot auscultate breath/heart/bowel sounds

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Prone Positioning

- Redistributes pulmonary blood flow
- Indications
  - ARDS – need to improve oxygenation
- Key Points
  - Patient to be proned at LEAST 16 hours/day

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Prone Positioning

- Complications
  - Worsen chest wall compliance
  - Airway obstruction
  - Endotracheal tube dislodgement
- Contraindications
  - Unstable vertebral fractures (absolute)
  - Elevated intracranial pressure (relative)
  - Hemodynamic instability (relative)
  - Unstable pelvic/long bone fractures (relative)
  - Open abdominal wounds (relative)

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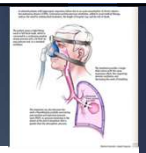
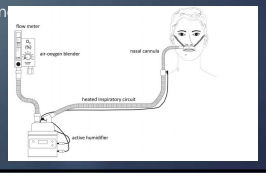
Non-invasive positive pressure ventilation (NPPV)



- BPAP (Bilevel Positive Airway Pressure)
  - Inspiratory Positive Airway Pressure (IPAP)
  - Expiratory Positive Airway Pressure (EPAP)
  - Indications: Respiratory Failure
- CPAP (Continuous Positive Airway Pressure)
  - Delivers 1 specified positive pressure
  - Indications: Resp. Failure d/t cardiogenic pulmonary edem
- High Flow Nasal Cannula
  - Up to 60 L/min
  - Indications: mild-mod hypoxemic respiratory failure



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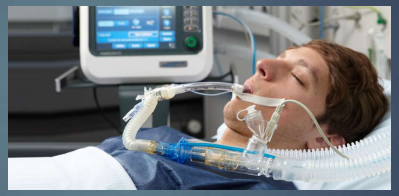
Pulmonary Therapeutic Interventions Associated with Mechanical Ventilation

- Intubation
- Weaning
- Extubation



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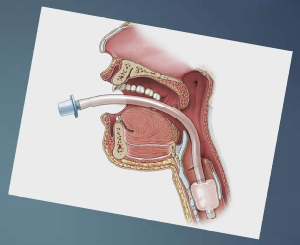
Intubation



- Indications
  - Airway
    - Obstruction
    - Protection
  - Ventilatory Failure
    - PaCO2 > 50 mmHg
  - Hypoxia
    - PaO2 < 50 mmHg
  - Respiratory Distress (high RR, use of accessory muscles)

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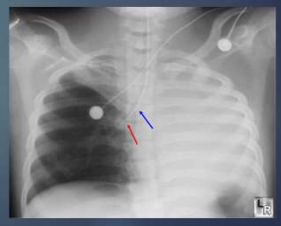
Verify tube placement

- Auscultation
- Confirmation Device
- eTCO2
- Cxr - tube should be between the 3rd and 4th ICS (2 - 3 cm above the carina) – gold standard



20

Inflation of ET tube cuff

- Inflate cuff pressure to 25 mmHg using Wright manometer
- Pressure on the wall of the trachea in excess of 30 mm Hg occludes arterial blood flow causing ischemia/necrosis.
- Minimal Leak Test
  - Inflate cuff until no leak is heard in trachea.
  - Withdraw 0.2 - 0.5 cc air or until a slight leak is heard.

21

Nursing Care for Intubated Patient

- Frequent oral care
- Suction as needed
- Tube placement checks
- Adequate humidity
- Assess oral mucosa

22

Mechanical Ventilation – prevention of complications

- Ventilator Induced Lung Injury
- Cardiovascular Complications
- VAP
- Sinusitis

23

Ventilator Induced Lung Injury

- Oxygen toxicity
  - Use of O2 greater than 60% longer than 48 hours
- Barotrauma / Volutrauma
  - Peak Pressure
  - Plateau Pressure
  - Shear Injury (atelectrauma)
  - PEEP

24

Cardiovascular Complications

Positive End Expiratory Pressure

↑ Intrathoracic Pressure

↓ Venous Return to Heart

↓ Preload

↓ Cardiac Output

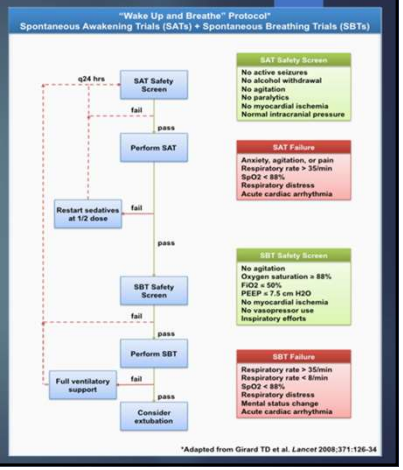
Reduced Cardiac Output:

- ↓ Blood Pressure
- ↓ Urine Output

25

VAP Bundle

- Head of Bed Elevation (~ 30 degrees)
- Oral Care
- Stress Ulcer Prophylaxis
- Sedation Wake-Up
- Spontaneous Breathing Trial



26

Trouble Shooting the Vent

- High peak pressure differential:

High Peak Pressures Low Plateau Pressures	High Peak Pressures High Plateau Pressures
Mucus Plug	ARDS
Bronchospasm	Pulmonary Edema
ET tube blockage	Pneumothorax
Biting	ET tube migration to a single bronchus
	Effusion

27

Weaning

- Improvement of respiratory failure
- Absence of major organ system failure
- Appropriate level of oxygenation
- Adequate ventilatory status
- Intact airway protective mechanism (needed for extubation)

28

Weaning

- Definitions
  - Rapid Shallow Breathing index (RSBI): respiratory rate divided by tidal volume.
    - Most studied of the weaning parameters
    - The faster you breathe with small volumes the higher the number
  - Negative Inspiratory Force (NIF): (or MIP – maximal inspiratory pressure)
    - Global assessment of strength of respiratory muscles
  - Minute Ventilation: respiratory rate x tidal volume
    - Estimates demand on the respiratory system. Normal is 5-6L/min in healthy adults.
    - Increased CO2 production from fever, hypermetabolic state, hypoxemia, etc will increase minute ventilation

29

Predictors of Weaning Outcome

Predictor	Value
Ventilatory muscle capability:	
▪ Vital capacity	▪ > 10 mL/kg
▪ Maximum inspiratory pressure	▪ < -30 cm H_2O
Ventilatory performance	
▪ Minute ventilation	▪ < 10 L/min
▪ Maximum voluntary ventilation	▪ > 3 times V_E
▪ Rapid shallow breathing index	▪ < 100
▪ Respiratory rate	▪ < 30 /min
▪Vt	▪ 5-7 ml/kg

30

Extubation

- Prior to extubation:
  - Confirm minimal FIO2 and PEEP
  - Evaluate upper airway complications
    - Check cuff leak
    - Check that cough and gag are present
- Have equipment ready
  - NC/facemask/bipap
- Suction secretions
- Extubate!

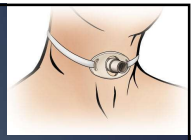
31

Late Post-Extubation Complications

- Fibrotic Stenosis of the Trachea
  - Caused by prolonged use of any tube with a rigid inflatable cuff
  - Follows earlier ulceration and necrosis of site
  - Tracheoesophagel fistula may form
  - Prevention – low-pressure cuffs and proper monitoring of cuff pressures
- Stenosis of Larynx
  - Caused by discrepancy between the anatomy of the larynx and size/shape of the tube
  - Treatment – dilation of surgical intervention or permanent tracheostomy

32

Tracheostomy Indications



- Ventilation
  - Long-term mechanical ventilation
  - Interval between oral intubation to trach varies
    - Consider trach if patient requires endotracheal tube > 21 days (American College of Chest Physicians)
- Airway obstruction
  - Tumors
  - Paralyzed vocal cords
  - Swelling
  - Stricture
  - Unusual anatomy
  - Trauma
- Airway protection
  - Insufficient cough and/or gag
    - High spinal cord injury
    - Cerebrovascular accident
    - Traumatic brain injury
- Secretions

33

Tracheostomy Care (Consensus Statements)

- All supplies to replace trach should be at bedside or within reach
- The first change of a tracheostomy tube should normally be performed by an experienced physician with assistance from another clinician
- Use of a defined trach care protocol will help decrease complications
- In an emergency, patients with a dislodged tracheostomy tube that cannot be reinserted should be intubated
- Acute occlusion of a tracheostomy tube is most likely caused by a mucous plug, obstructing granuloma, or insertion of the tube into a false track
- A patient can be turned in bed once the security of the tube has been assessed to avoid accidental decannulation

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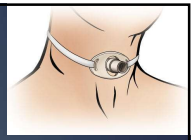
Tracheostomy Care: Mobilization of Secretions



- Mobilization consists of 3 primary factors:
  - Adequate hydration
    - To keep secretions thin and mobile
    - Humidified trach collar provides some moisture
  - Physical mobility
    - Progressive mobility (out of bed, sitting in chair, walking)
    - Range of motion exercises
  - Removal of secretions
    - Suctioning
    - Encouraging patient to cough

35

Therapeutic Gases – Nitric Oxide

- Inhaled
- Improves oxygenation but does not improve outcome
- Vasodilator - ↓ PAP which reduces shunt fraction and ↑ PaO_2
  - Short half-life
  - No systemic effects
- Indications
  - ARDS
  - Persistent Pulmonary Hypertension

36

Therapeutic Gases – Heliox

- Helium + Oxygen
- Indications:
  - COPD
  - ALI
  - ARDS

37

References

- Modes of Mechanical Ventilation https://www.openanesthesia.org/modes_of_mechanical_ventilation/
- Henderson, Griesdale, Dominelli et al. (2014). Does prone positioning improve oxygenation and reduce mortality in patients with acute respiratory distress syndrome? Canadian Respiratory Journal 21(4).
- Hartjes, TM. (2006). AACN Core Curriculum for High Acuity, Progressive, and Critical Care Nursing 7th Edition.

38

Which of the following is a complication of mechanical ventilation and peak end expiratory pressure (PEEP) therapy?

A. Atelectasis
B. Oxygen toxicity
C. Reduced cardiac output
D. Acute Respiratory Distress Syndrome

39

Right main stem intubations are more likely than left main stem due to the fact that the:

- Right main stem bronchus has more ciliary clearance
- Left main stem bronchus is located several inches below the right
- Left main stem bronchus is posterior to the right
- Right main stem bronchus is wider and has less angulation than the left

40

Which of the following is an indication for positive end expiratory pressure therapy?

- To improve CO2 elimination
- To treat a metabolic acidosis
- To reduce post-operative bleeding
- To allow reduction in FiO2 support

41

Which of the following is a complication of mechanical ventilation and PEEP therapy?

- Atelectasis
- Oxygen toxicity
- Reduced cardiac output
- Acute Respiratory Distress Syndrome

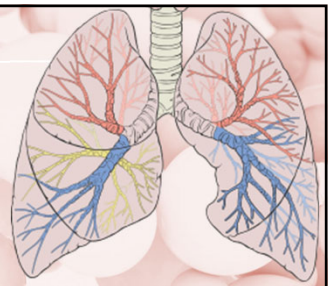
42

A 70kg patient is on mechanical ventilation with Fio2 40%, tidal volume 500 ml/kg, and IMV rate 8. Patient's respirations are 10 breaths per minute. The ABG pH 7.38, PaO2 88mmHg, PaCO2 55mmHg. What is the expected change in vent settings?

- FiO2 increase
- Increase the respiratory rate
- Increase the tidal volume
- No change necessary

43

ACUTE RESPIRATORY FAILURE & OTHER RESPIRATORY EMERGENCIES



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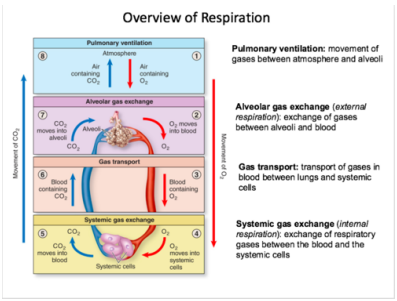
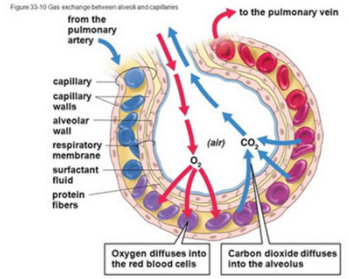
1

Overview of Respiration/ Gas Exchange





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Acute Respiratory Failure (ARF)

- Failure
  - Oxygenation
  - Ventilation
  - Both of the above
- Altered gas exchange (room air)
  - PaO_2 < 60 mm Hg
  - $PaCO_2$ > 50 mm Hg
  - pH ≤ 7.30

3

Failure of Oxygenation

- Hypoventilation
- Intrapulmonary shunting
- Ventilation-perfusion mismatch
- Diffusion defects
- Decreased barometric pressure
- Low cardiac output (nonpulmonary hypoxemia)
- Low hemoglobin level (nonpulmonary hypoxemia)

4

Hypoventilation

- Drug overdose
- Neurological disorders
- Abdominal or thoracic surgery

5

Intrapulmonary Shunting

- Blood shunted from right to left side of heart without oxygenation
- Qs/Qt disturbance
- Causes: atrial or ventricular septal defect, atelectasis, pneumonia, pulmonary edema
- Why does administration of higher levels of oxygen *not* help in shunt disorders?

6

Critical Thinking Challenge

- Why does administration of higher levels of oxygen *not* help in shunt disorders?

7

V/Q Mismatch

- Most common cause of low O_2
  - Normal ventilation (V) is 4 L/min
  - Normal perfusion (Q) is 5 L/min
  - Normal V/Q ratio is 4/5 or 0.8
- A mismatch occurs if either
  - V is decreased or
  - Q is decreased
- What are causes of this condition?

8

Diffusion Defects

- Diffusion of O_2 and CO_2 does not occur
  - Fluid in alveoli
  - Pulmonary fibrosis

9

Low Cardiac Output

- Cardiac output must be adequate to maintain tissue perfusion
- Normal delivery is 600 to 1000 mL/min of oxygen

10

Low Hemoglobin

- Hemoglobin necessary to transport oxygen
- 95% of oxygen is bound to hemoglobin

11

Tissue Hypoxia

- Some conditions prevent tissues from using oxygen despite availability
  - Cyanide poisoning
- Tissue hypoxia results in anaerobic metabolism and lactic acidosis

12

Quick Quiz

The nurse suspects respiratory failure secondary to hypoventilation in a patient with:

A. Anxiety
B. Neuromuscular disease
C. Pulmonary embolism
D. Volume A/C ventilation at rate of 20 breaths/min

13

Critical Thinking Challenge

- How does hemoglobin affect oxygenation?
- Why would a low cardiac output result in hypoxia?

14

Failure of Ventilation

- Hypercapnia
- Related to:
  - Alveolar hypoventilation—decrease in ventilation and hypoxemia
  - V/Q mismatch

15

Assessment of Respiratory Failure

- Neurological—shows earliest signs of hypoxemia and hypercapnia
- Respiratory
- Cardiovascular
- Nutrition
- Psychosocial
- Chest x-ray
- Pulmonary function tests
- Laboratory studies
- Arterial blood gases (ABGs)
- Pulse oximetry and end-tidal CO_2

16

Interventions

- Maintain a patent airway
- Optimize O_2 delivery
- Minimize O_2 demand
- Identify and treat the cause of ARF
- Prevent complications

17

Critical Thinking Challenge

- What nursing interventions assist in reducing oxygen demands?

18

Nursing Diagnoses

- Impaired ventilation
- Ineffective airway clearance
- Infection
- Anxiety
- Impaired skin integrity
- Ineffective coping
- Ineffective breathing pattern
- Impaired gas exchange
- Impaired breathing pattern
- Fluid volume excess
- Altered nutrition

19

Medical Management

- Oxygen
- Bronchodilators
- Corticosteroids
- Sedation
- Transfusions
- Therapeutic paralysis
- Nutritional support
- Hemodynamic monitoring

20

Respiratory Failure Concerns

CLINICAL ALERT

Acute Respiratory Failure

Concern	Symptoms	Nursing Actions
Respiratory muscle fatigue	Diaphoresis Nasal flaring Tachycardia Abdominal paradox Muscle retractions • Intercostal • Suprasternal • Supraclavicular Central cyanosis	Improve O_2 delivery: • Administer O_2 • Ensure adequate cardiac output and blood pressure • Correct low hemoglobin • Administer bronchodilators Decrease O_2 demand: • Provide rest • Reduce fever • Relieve pain and anxiety • Decrease work of breathing • Position patient for optimum gas exchange and perfusion • Prepare for possible intubation and mechanical ventilation
Cerebral hypoxia and carbon dioxide narcosis from increased CO_2 retention	Lethargy Somnolence Coma Respiratory acidosis	Maintain airway patency Prepare for possible intubation and mechanical ventilation

21

Quick Quiz

A nursing intervention to maximize airway clearance is which of the following?

A. Administer supplemental oxygen.
B. Elevate the head of bed.
C. Provide oral care every 4 hours.
D. Reposition patient every 2 hours.

22

ARDS

- Noncardiogenic pulmonary edema
- Diagnostic criteria
  - PaO_2/FiO_2 ratio of less than 200
  - Bilateral infiltrates
  - Pulmonary capillary wedge pressure < 18 mm Hg
- Acute lung injury scoring

23

Case Study

Mrs. J. is a 31-year-old female admitted to the critical care unit with respiratory distress after getting the "flu." Her condition worsens; SpO_2 is 85% on Venturi mask at 0.50. ABGs show a PaO_2 of 50 mm Hg. Her chest x-ray is showing infiltrates.

- Calculate Mrs. J.'s PaO_2/FiO_2 ratio, and interpret the findings.

24

Case Study (Cont.)

Mrs. J. is placed on noninvasive positive-pressure ventilation (NPPV).

Why is this decision made?

25

Case Study (Cont.)

Within 2 hours of NPPV, Mrs. J. is getting worse. Her SpO_2 remains at 85%, and the oxygen via NPPV was 80%. Her chest x-ray shows bilateral "white out."

What treatment is indicated?

26

Quick Quiz

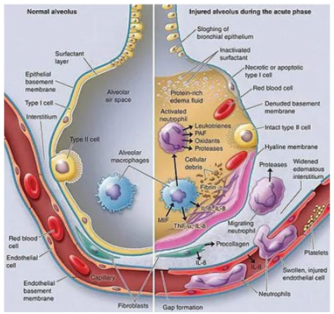
If the PaO_2 is 60 mm Hg and the FiO_2 is 0.6, the PaO_2/FiO_2 ratio is:

A. 100
B. 1000
C. 360
D. 3600

27

ARDS Pathophysiology

- Insult—systemic inflammatory response syndrome (SIRS)
- Release of inflammatory mediators
- Damage to alveolar-capillary membrane
- Increased capillary permeability
- Pulmonary edema (noncardiogenic)



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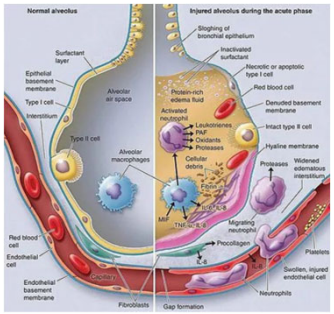
ARDS Pathophysiology (Cont.)

- Microatelectasis
- Decreased compliance (stiff lungs)
- Decreased surfactant (damage to type II pneumocytes)
- Impaired gas exchange
- V/Q mismatch



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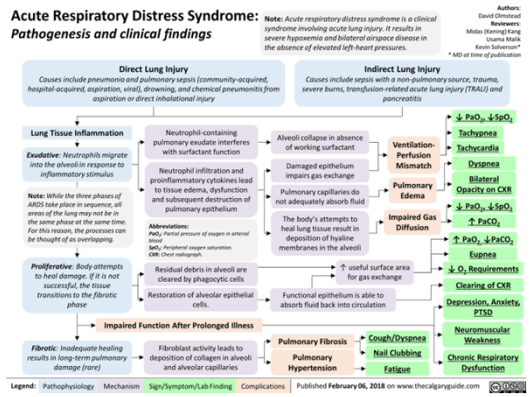
ARDS Pathophysiology



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Berlin Definition – ARDS

Timing	Within 1 week of a known clinical insult of new/worsening respiratory symptoms		
Chest Imaging (x-ray or CT)	Bilateral opacities – not fully explained by effusions, lobar/lung collapse, or nodules		
Origin of Edema	Respiratory failure not fully explained by cardiac failure or fluid overload		
Oxygenation	Mild	Moderate	Severe
	P/F Ratio 201-300 (PEEP ≥ 5 cmH2O)	P/F Ratio 101-200 (PEEP ≥ 5 cm H2O)	P/F Ratio ≤ 100 (PEEP ≥ 5 cm H2O)

31

Critical Thinking Challenge

- What patients would you identify as having a high risk of developing ARDS?
- Why is hyperventilation an early sign seen in patients developing ARDS?
- What related ABG abnormality will be seen?

32

Case Study (Cont.)

- What is a possible etiology of ARDS in Mrs. J.?

33

Symptoms of ARDS

- Dyspnea and tachypnea
- Hyperventilation with normal breath sounds
- Respiratory alkalosis
- Increased temperature and pulse
- Worsening chest x-rays that progress to "white out"
- Increased PIP on ventilation
- Eventual severe hypoxemia

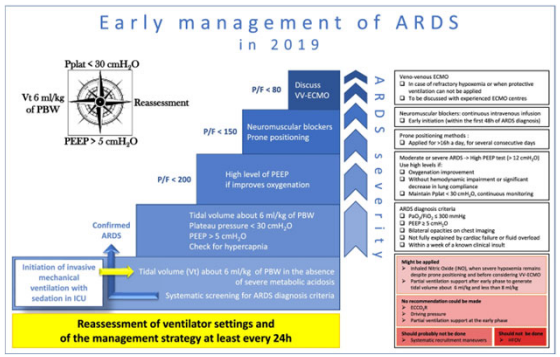
34

Treatment of ARDS

- Treat the cause
- Oxygenation and ventilation
  - Positive end-expiratory pressure (PEEP)
  - Possible nontraditional modes of ventilation: high-frequency, pressure-control, and inverse-ratio

35

ARDS Treatment/Management



36

Case Study (Cont.)

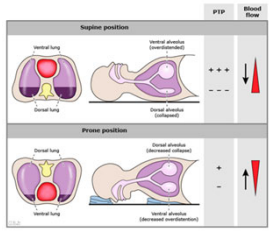
Mrs. J. is placed on volume assist/control (V-A/C) ventilation: rate 16 breaths/min, V_T 8 mL/kg, FiO_2 0.80, and PEEP 10 cm.

What is the rationale for these settings, including PEEP?

37

Treatment of ARDS

- Comfort
  - Sedation
  - Pain relief
  - Neuromuscular blockade
- Decrease O_2 consumption
- Positioning
  - Prone positioning
  - Continuous lateral rotation therapy



38

Case Study (Cont.)

Mrs. J.'s condition does not improve. SpO_2 is 85%, and the FiO_2 is increased to 0.90 to maintain this. A decision is made to increase the PEEP to 20 cm H_2O. About 10 minutes after the PEEP is increased, Mrs. J.'s blood pressure drops to 80/50 mm Hg. Breath sounds are equal bilaterally.

39

Question

What is the likely rationale for the drop in Mrs. J.'s blood pressure?

What is the significance of the bilateral breath sounds?

40

Treatment of ARDS

- Fluid and electrolyte balance
- Adequate nutrition
- Pharmacologic intervention
- Psychosocial support

41

ARDS

- Be alert for complications:
  - Multiple organ dysfunction syndrome
  - Renal failure
  - Disseminated intravascular coagulation
  - Long-term pulmonary effects associated with high oxygen and other therapies

42

ACUTE RESPIRATORY FAILURE IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

43

Pathophysiologic process for development of ARF in COPD

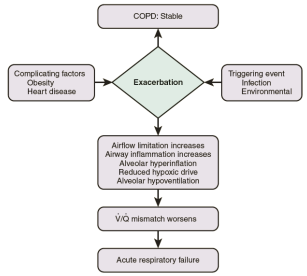
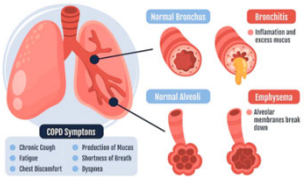


FIGURE 15-3 Pathophysiologic process for the development of ARF in COPD. *COPD*, Chronic obstructive pulmonary disease; *V/Q*, Ventilation-perfusion ratio. (Modified from Brill SE, Wedzicha JA. Oxygen therapy for acute exacerbations of COPD. *Int J Chron Obstruct Pulmon Dis* 9:1241–1252, 2014.)



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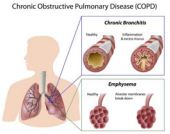
ARF in Chronic Obstructive Pulmonary Disease (COPD)

- Worsening V/Q mismatch (e.g., secretions and bronchoconstriction can lead to ARF)
- Causes: acute exacerbations, CHF/ pulmonary edema, dysrhythmias, pneumonia, dehydration, and electrolyte imbalances

45

COPD – Comparison of Chronic Obstructive Bronchitis and Emphysema



Chronic Obstructive Bronchitis

- "Blue Bloater"
- Productive Cough
- Stocky Build
- Onset 40-50 yr
- Normal RR
- Hypoxemia
- Increased PaCO2
- Cyanosis
- Polycythemia
- Corpulmonale (x-ray – cardiomegaly)
- Peripheral Edema
- Risk for PE

Emphysema

- "Pink Puffer"
- Cough uncommon
- Thin
- Onset 50-70 yr
- Tachypnea (PaCO2 usually low or normal until end stage)
- PaO2 normal or slightly low
- Barrel chest
- Accessory muscle use
- Leans forward while sitting
- Pursed-lip breathing
- Hyperresonance on percussio
- Lung overinflation, diaphragm low

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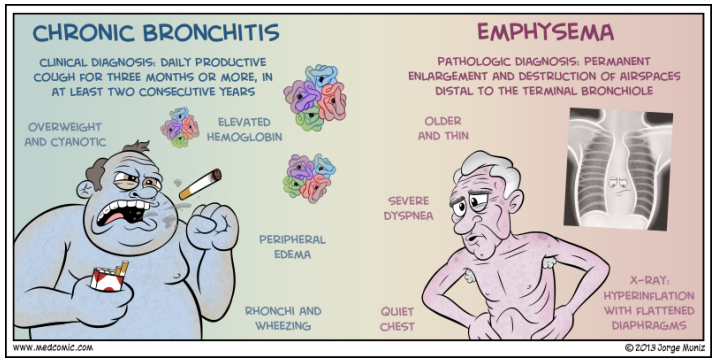
COPD



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Assessment

- Dyspnea
- Chronic cough
- Sputum production
- Postbronchodilator spirometry limitations
- Pulmonary function studies
- Chest wall changes (barrel chest)
- Accessory muscles used for breathing
- Clubbing of the fingers
- Wheezing and crackles
- ABG (hypoxemia and hypercapnia)

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Medical Management of ARF in COPD

- Correct hypoxemia
  - Cautious administration of O_2
  - Noninvasive positive-pressure ventilation
  - Ventilatory assistance
- Medications
  - $Beta_2$ agonists (bronchodilators)
  - Corticosteroids
  - Antibiotics (depends on cause)
  - Cautious administration of sedatives

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Oxygen Guidelines in COPD Exacerbation

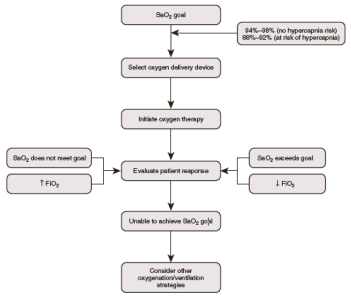

Figure 15-4 General guidelines for titration of oxygen therapy in COPD exacerbation. (Modified from Brill, SE, & Wedzicha, JA. Oxygen therapy for acute exacerbations of COPD. *International Journal of Chronic Obstructive Pulmonary Disease*,9:1241-1252, 2014.)

50

Pharmacologic Therapy

- Short-acting inhaled $beta_2$-agonists
- Long-acting $beta_2$-agonists
- Corticosteroids (prednisone)
- Antibiotics

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Ventilatory Assistance

- NPPV
- Intubation
- End-of-life issues
- Advance directives

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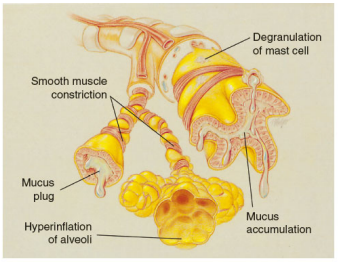
Airway Obstruction caused by Bronchial Asthma



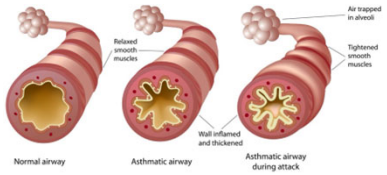


FIGURE 15-5 Airway obstruction caused by asthma. Thick mucus, mucosal edema, and smooth muscle spasm cause obstruction of small airways in bronchial asthma. (Modified from Des Jardins T, Burton GC. *Clinical manifestations and assessment of respiratory disease.* 3rd ed. St Louis: Mosby; 1995.)

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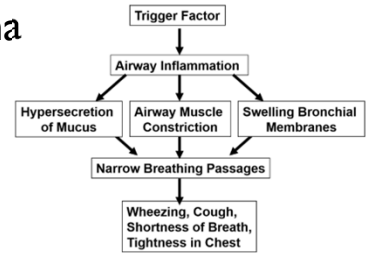
Exacerbation of Asthma



- Wheezing
- Dyspnea
- Chest tightness
- Use of accessory muscles
- Nonproductive cough
- Hyperventilation initially
- Peak expiratory flow reading is less than 50% of normal values

What are some triggers?

54

Exacerbation of Asthma (Cont.)

- Causes
  - Bronchodilators no longer working
  - Noncompliance with medications
- Effects
  - Hyperventilation with air trapping results in respiratory acidosis
  - Severe hypoxemia

55

Medical Management

- Oxygen; ventilation in severe cases
- IV corticosteroids
- Inhaled bronchodilators; rapid-acting beta$_2$-agonists
- Teaching

56

Critical Thinking Challenge

- How can you alleviate anxiety in the patient with status asthmaticus?
- What positioning will facilitate gas exchange?
- What discharge teaching is essential to prevent future episodes?

57

Pneumonia

- Types
  - Community-acquired
  - Health care–acquired
  - Hospital-acquired
  - Ventilator-associated
- Increased risk: elderly, alcoholic, smokers, chronic diseases, head injury, immunosuppression

58

Pathophysiology Pneumonia

- Organisms in lower respiratory tract to overwhelm defense mechanisms
- Causes
  - Aspiration
  - Inhalation
  - Spread from another infected area
- Impaired mucociliary clearance

59

Prevention of Pneumonia

- Influenza vaccine
  - All persons over 6 months
  - People at high risk for complications of influenza
  - People in contact with those at high risk
  - Health care providers
- At age 65, pneumococcal vaccination to prevent *Streptococcus* or *pneumococcus*
  - Conjugate dose
  - Polysaccharide dose

60

Presentation of Pneumonia

- Fever
- Cough
- Purulent sputum
- Hemoptysis
- Dyspnea/tachypnea
- Chest pain (pleuritic)
- Adventitious breath sounds

61

Quick Quiz

Arterial blood gas alterations in pneumonia include which of the following?

A. Hypoxemia and respiratory alkalosis
B. Normal oxygen and respiratory acidosis
C. Hypoxemia and metabolic acidosis
D. Normal values

62

Ventilator-Associated Pneumonia (VAP)

- Aspiration of bacteria from oropharynx or gastrointestinal tract
- Many potential causes
- Controversies about best way to diagnose—no "gold standard"

63

Ventilator-Associated Pneumonia (Cont.)

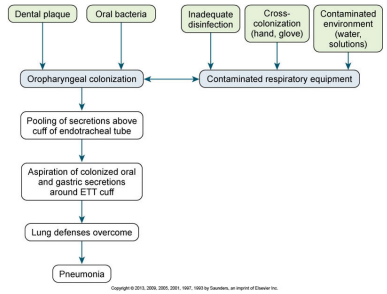


Figure 15-6. Role of airway management in the pathogenesis of ventilator-associated pneumonia.

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VAP Bundle

- Elevate head of bed 30 to 45 degrees
- Awaken daily and assess readiness to wean and extubate
- Stress ulcer disease prophylaxis
- Venous thromboembolism (VTE) prophylaxis
- Oral care

65

NHSN Surveillance Algorithm for Ventilator-Associated Events.

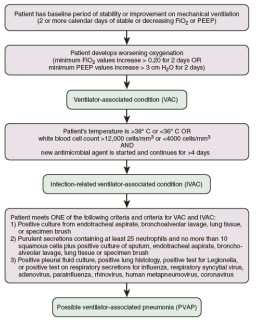


FIGURE 15-7 National Healthcare Safety Network: NHSN surveillance algorithm for ventilator-associated events. FIO_2, Fraction of inspired oxygen; *PEEP*, positive end-expiratory pressure. (Modified from Centers for Disease Control and Prevention. Device-associated module VAE. http://www.cdc.gov/nhsn/PDFs/pscManual/10-VAE_FINAL.pdf, January 2015 Accessed December 17, 2015.

66

Prevention of VAP

- Hand washing and standard precautions
- Surveillance
- Ventilator bundle
- Prevent transmission
  - Sterile water in circuit
  - Drain condensate AWAY from patient
  - Avoid normal saline during suctioning

67

Prevention of VAP (Cont.)

- Prevent infection and aspiration
  - Avoid reintubation
  - Oral intubation
  - ETT with continuous aspiration of subglottic secretions
  - Sedation and weaning protocols
  - Aseptic suctioning of endotracheal tube (ETT)
- Nutrition
- Mobilization

68

Treatment of VAP

- Bacteria-specific antibiotic therapy

69

Quick Quiz

To prevent VAP, it is recommended that the head of bed be elevated to at least:

A. 15 degrees
B. 20 degrees
C. 30 degrees
D. 45 degrees

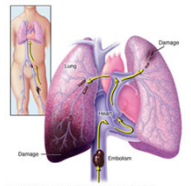
70

Pulmonary Embolus

- Free-floating Thrombus: material that travels to the vascular system in the lungs where it lodges and occludes a vessel
- Can be blood clots, tumor cells, cardiac vegetation, fat, amniotic fluid, air, or nitrogen
- Causes partial or total occlusion of pulmonary artery vessel – infarction
- Affected area of the lung is ventilated but inadequately perfused

71

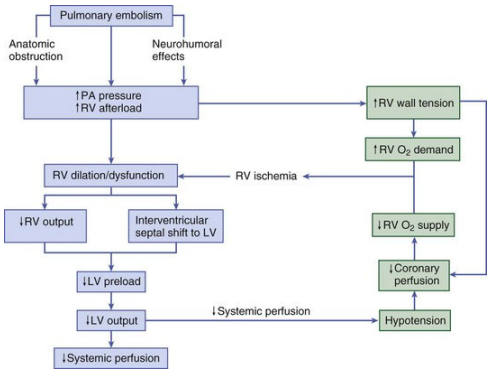
ARF: Pulmonary Embolus (PE)



- Virchow's triad
  - Venous stasis
  - Altered coagulability
  - Damage to vessel wall
- Embolus results in a lack of perfusion to ventilated alveoli (V/Q mismatch)

72

Effects of PE



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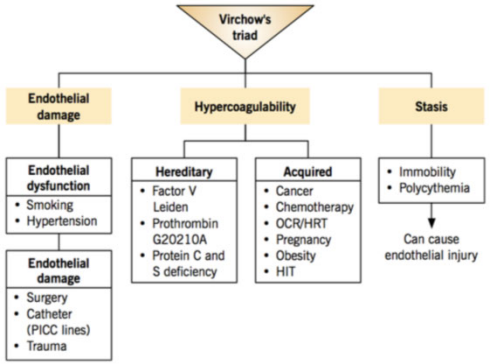
PE Risk Factors



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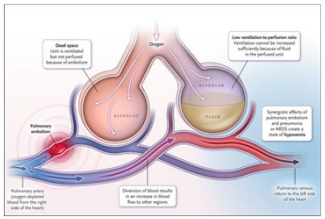
PE Assessment

- Symptoms of deep venous thrombosis
- Chest pain (worse on inspiration)
- Dyspnea
- Tachycardia
- Tachypnea
- Cough; hemoptysis
- Crackles, wheezes
- Hypoxemia

75

Diagnosis of PE



- Clinical signs and symptoms
- D-dimer assay (positive)
- V/Q scan with high probability of PE
- Duplex ultrasound (DVT)
- High-resolution multidetector computed tomography angiography (MDCTA; spiral CT)
- Pulmonary angiogram

76

Prevention of PE

- Medications
  - Heparin, low–molecular weight heparin
- Mechanical
  - Sequential compression devices
  - Foot pumps
  - Compression stockings
- Position changes
- Treatment of atrial dysrhythmias
- Prophylactic anticoagulant therapy
  - Warfarin; long-term prevention

77

Complications of PE

- Heart failure
- Obstructive shock
- Death

78

Treatment for PE

- ABCs; oxygen
- Thrombolytics (dissolve the clots)
- Heparin
- Monitor laboratory results for
  - Bleeding
  - Thrombocytopenia
- Surgical procedures
  - Embolectomy
  - Vena cava umbrella (prevention)

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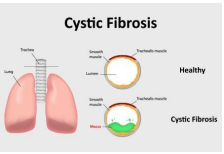
Cystic Fibrosis

- Genetic disorder
- Mutation in chloride transport results in "sticky" mucus that obstructs glands:
  - Lungs (greatest effect)
  - Pancreas
  - Liver
  - Salivary glands
  - Testes



- Thick mucus in lungs is medium for infection, chronic bronchitis, and ARF

80

Cystic Fibrosis (Cont.)

- Considered to be a disease of childhood
- Improvements in care have prolonged life expectancy

81

Cystic Fibrosis (Cont.)

- Cornerstones of care for a patient with CF
  - Antibiotic therapy
  - Airway clearance
  - Nutritional support
  - Ventilatory support
  - *Pseudomonas aeruginosa* is the most common pathogen found in adult patients with CF

82

Transplant for CF

- One treatment for CF is lung transplantation

83

Pulmonary Hypertension

84

CHEST TRAUMA

Kelly Urban, PhD, MEd, RN, CCRN-K, TCRN, CEN
University of Arkansas for Medical Sc

1

CHEST TRAUMA

- Can involve cardiovascular system, respiratory, chest wall, rib cage, CNS and gastrointestinal system

2

CHEST TRAUMA MECHANISMS OF INJURY

Blunt

- Acceleration
- Deceleration
- Shearing
- Crushing/Compression

Penetrating

- GSW
- Stab wounds
- Impalement
- Avulsion/Degloving

3

PULMONARY TRAUMA

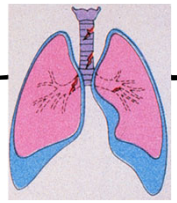
- Pneumothorax
- Hemothorax
- Pulmonary Contusion
- Diaphragmatic Injury
- Rib Fractures/Flail Chest
- Rupture of the Trachea/Bronchus
- Chest Drainage

CV TRAUMA

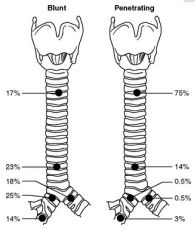
- Myocardial Contusion (Blunt Cardiac Injury)
- Aortic Rupture
- Pericardial Tears
- Electrical Injuries

4

TRACHEOBRONCHIAL INJURY



- Mechanism: Penetrating or clothesline
- Assessment findings
  - Hoarseness
  - Subcutaneous emphysema
  - Hemoptysis



5

TRACHEOBRONCHIAL INJURY

Diagnosis

- Diagnosis
  - Bronchoscopy
  - CT

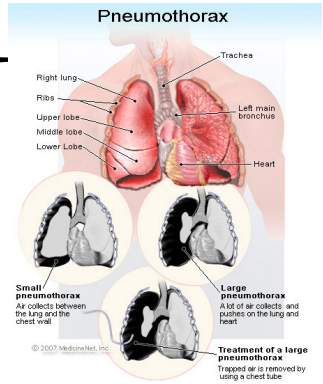
Interventions

- Flex bronchoscopy for intubation

6

PNEUMOTHORAX (SIMPLE & OPEN)

- Simple or open
- Mechanism: Simple-blunt; open-penetrating
- Assessment findings
  - Decreased or absent breath sounds
  - Dyspnea, tachypnea
  - Tachycardia
  - Subcutaneous emphysema
  - Sucking chest wound
- Interventions
  - Depends on severity



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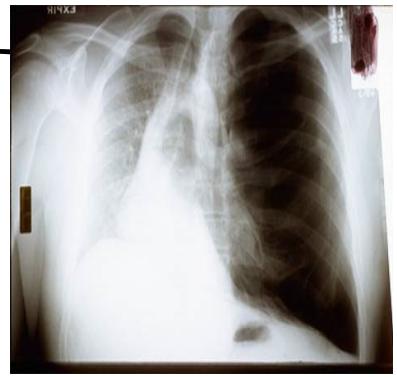
TENSION PNEUMOTHORAX

- Life threatening
- Assessment findings
  - Severe respiratory distress
  - Diminished or absent breath sounds on injured side
  - Hypotension
  - Distended neck, head, and upper extremity veins
  - Tracheal deviation (late sign)
  - Cyanosis (late sign)



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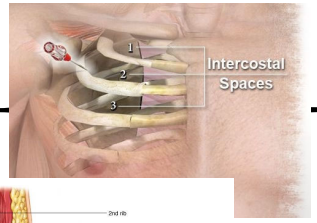
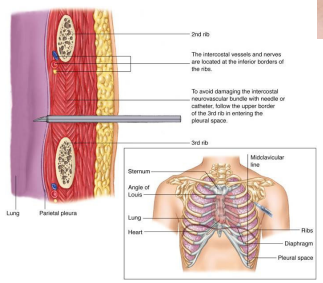
TENSION PNEUMOTHORAX

- Interventions
  - Immediate needle decompression
    - 14-gauge needle
    - Second intercostal space, midclavicular line
    - Placement controversies
  - Prepare for chest tube





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MANAGEMENT OF PNEUMOTHORAX

- Small closed
  - monitor closely
  - maybe supplemental O2
  - Restrict activity
- Small closed symptomatic
  - O2
  - needle puncture
  - Heimlech valve
- Moderate or large/open or closed
  - O2, chest-tube to underwater seal or suction
- Tension
  - Immediate chest tube or needle decompression
  - O2 or vent

10

MANAGEMENT OF PNEUMOTHORAX

- Recurrent Spontaneous
  - Treat immediate problem
  - For long term intervention, will need pleurodesis.

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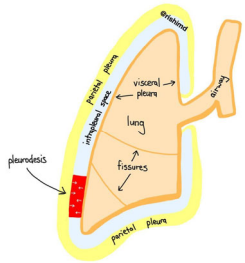
PLEURODESIS



Agents	Advantages	Disadvantages
Talc	Cheap, easily available, highest efficacy	Reports of ARDS, renal failure
Bleomycin	Efficacy similar to talc	Very costly, chest pain, fever nausea
Povidone iodine	Cheap, easily available	Anaphylaxis, randomized study required involving larger number of patients
Tetracycline/ doxycycline	Easily available	Very painful, ARF

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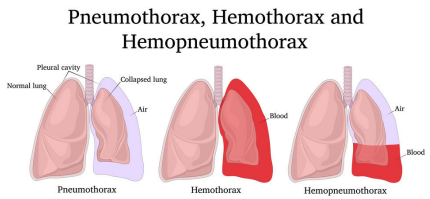
HEMOTHORAX

- Collapse of a lung from an accumulation of blood in pleural space
- Can be from blunt or penetrating trauma
- May occur alone, or with a pneumothorax
- 50% of hemothoraces are from rib fractures, injury the pleural parenchyma, or thoracic aorta.



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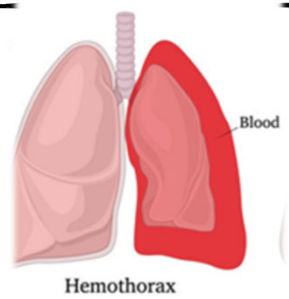
HEMOTHORAX CAUSES

- Subclavian line insertion
- Mediastinal tumors
- Anticoagulant therapy
- Blood dyscrasias
- Sudden changes in intra-thoracic pressure



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HEMOTHORAX CLINICAL PRESENTATION

- May have no s/s early on
- ABGs may be normal early on
- Respiratory distress
- SOB
- Decreased breath sounds on affected side – like pneumothorax
- Dull to percussion
- Increase PIP on vent
- Later on PO_2 will decrease and PCO_2 will increase

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MANAGEMENT OF HEMOTHORAX

- Small
  - O2
  - monitor H&H
  - may need needle thoracentesis
- Moderate to large
  - large bore chest tube usually 4th,5th,or 6th ICS at MAL to 20cm water suction
- Massive
  - above, plus hemodynamic support
  - Autotransfusion if available
  - Surgical repair

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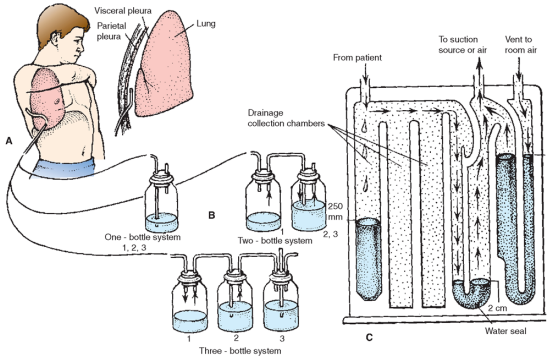
CHEST DRAINAGE (CHEST TUBES)



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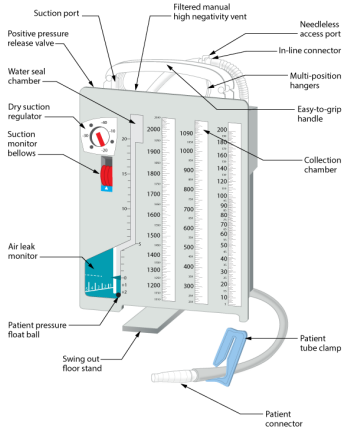
CHEST DRAINAGE (CHEST TUBES)

- Sterile Tube inserted into pleural space and connected to drainage system
- 3 Chambers:
  - Collection chamber
  - Water-seal chamber
  - Wet/Dry suction control chamber



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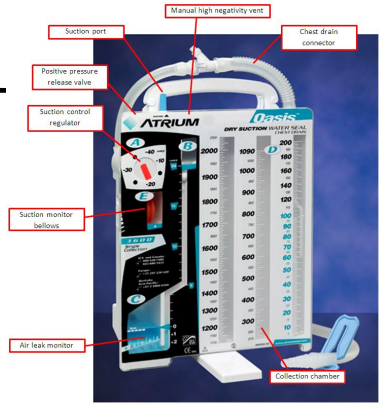
FOCA

- F (fluctuation)
- O (obstruction)
- C (color)
- A (air leak)

https://www.youtube.com/watch?v=RzOMLpWE0M



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CHEST DRAINAGE SYSTEMS

- Notify physician
  - Initial output is > 500 mL
  - More than 200 mL/hour for 2-4 hours
- Clamping chest tube is contraindicated

20

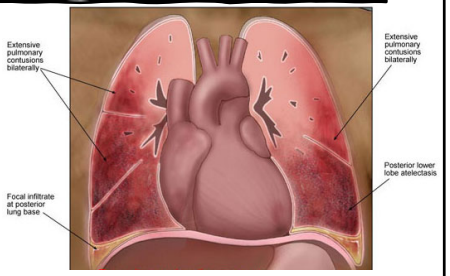
NURSING CARE

- Maintain Closed drainage system
- Avoid kinks in tubing
- Maintain correct fluid level with minimal bubbling required
- Watch for bubbling in underwater seal with expiration only
- Never clamp chest tube without specific MD order
- Assess chest tube insertion site - there should be no fluid leaking from around the site or sounds of air leaking

21

PULMONARY CONTUSION

- Damage to the lung parenchyma, resulting in localized edema and hemorrhage.
- Etiology: Blunt trauma (deceleration) or high-velocity missile



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PULMONARY CONTUSION MECHANISM AND PATHOPHYSIOLOGY

- Usually occurs after rapid deceleration
- Alveolar capillary damage occurs with interstitial and intra-alveolar extravasation of blood

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PULMONARY CONTUSION MECHANISM AND PATHOPHYSIOLOGY

- Bruising of lung causes:
  - Capillary hemorrhage
  - Leukocyte and platelet aggregation in pulmonary vasculature, leads to release of vasoactive substances
  - Loss of pulmonary capillary integrity
  - Extravasation of water and plasma proteins into the alveolar and interstitial spaces
  - Congestive atelectasis
  - Surfactant dilution resulting in decreased lung compliance
  - Decreased FRC due to pulmonary physiologic shunt, which causes venous admixture and hypoxemia

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PULMONARY CONTUSION ASSESSMENT FINDINGS

- Dyspnea
- Ineffective cough, often hemoptysis
- Possible chest wall contusion or abrasions
- Decreased pulmonary compliance = Increased Airway Pressure
- PaO_2/FIO_2 ratio continues to decrease
- Hypoxia/Respiratory Acidosis

25

INTERVENTIONS

- Decrease pulmonary shunting
- Administer oxygen (use lowest FiO_2)
- Increase FRC with PEEP
- Increase effective compliance with diuretics
- Improve oxygenation
- Limit fluids

26

COMPLICATIONS

- Irreversible hypoxia and acidosis (ARDS)
- Pleural effusion
- Infection
- Co-existing cardiovascular injury

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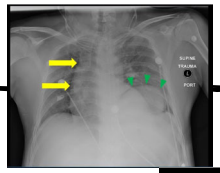
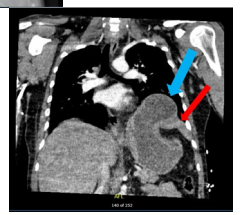
DIAPHRAGMATIC INJURY





- Symptoms:
  - Heart sounds shifted to right side of chest
  - Signs of obstructive shock
  - Dysphagia
  - Dyspnea
  - Decreased breath sounds on affected side
  - Bowel sounds in middle to lower chest
  - Lower chest, abdominal, or epigastric pain radiating to left shoulder (Kerr's sign)

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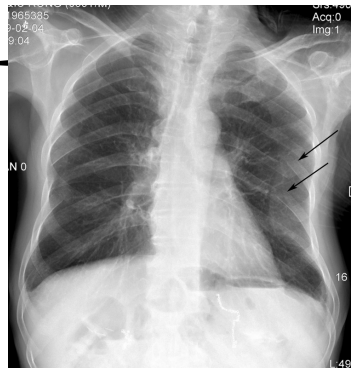
RIB FRACTURES

- Mechanism: Blunt
- Associated injuries
  - Sternal fracture
  - Brachial plexus or thoracic outlet vascular injuries
  - Liver
  - Spleen
  - Lung contusion or laceration



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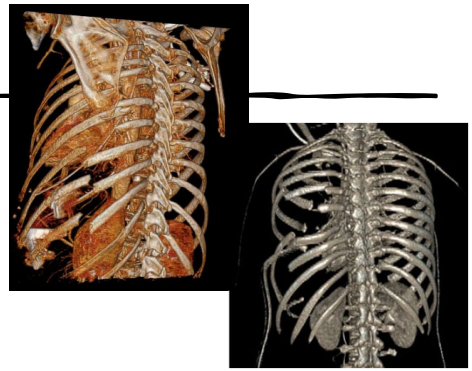
RIB FRACTURES

- Assessment findings
  - Dyspnea
  - Pain
  - Bony crepitus or deformity
  - Chest wall contusions or ecchymoses
- Interventions
  - Supplemental oxygen
  - Analgesic medications
  - Assess for underlying injuries

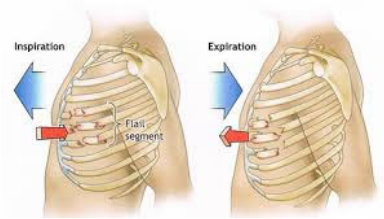


Ped vs Train

30

FLAIL CHEST DEFINITION

- 2 or more fractures of 3 or more adjacent ribs and/or sternal fracture
- Subatmospheric, intrathoracic, pressure during inspiration causes segment to go inward (paradoxical movement)



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FLAIL CHEST ETIOLOGY

- Most common is blunt trauma from steering wheel injury
- Assault
- Motor vehicle crashes
- Lateral impact-crush injuries
- Chest wall is stable at about 2 to 3 weeks after injury

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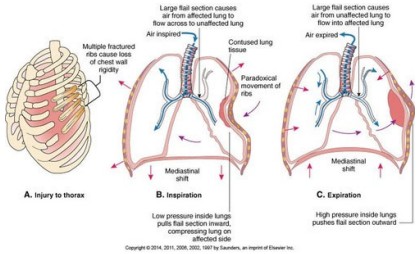
FLAIL CHEST DIAGNOSTIC INDICATORS

- Rapid and labored breathing
- Paradoxical chest wall movement
- Patient splints chest wall and muscle spasms occur
- Palpation of crepitus, fracture
- Pain on inspiration or palpation
- Hypoxia
- Absent or decreased breath sounds on affected side
- Dyspnea, tachypnea, respiratory failure



https://youtu.be/uJHfX1R5kfQ

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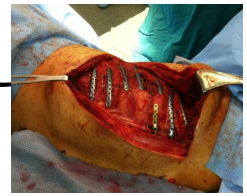
FLAIL CHEST INTERVENTIONS



- Stabilize flail segment
- Intubate and Ventilate (PEEP is often required)
- Control pain to allow full lung expansion (decreases incidence of atelectasis)
- Prevent hypoxemia; correct respiratory acidosis
- Often requires intubation
- Rib plating

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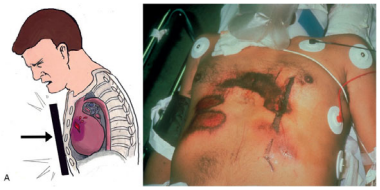
MYOCARDIAL CONTUSION (BLUNT CARDIAC INJURY)

- Etiology
  - Usually acceleration/deceleration injury from Motor Vehicle Crash
    - Sternum may hit steering wheel or dashboard
  - Auto/pedestrian collisions
  - Assault with blunt instrument
  - Explosion
  - Vigorous CPR



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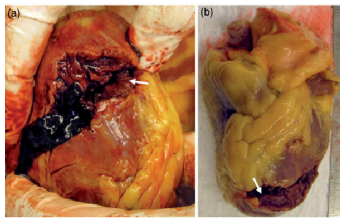
BLUNT CARDIAC INJURY PATHOPHYSIOLOGY

- Bruising, bleeding into myocardium
- Right ventricle primary site of injury due to location
- Bleeding into pericardial sac: Cardiac Tamponade
- Often accompanied by other thoracic trauma: Fx ribs, etc



36

BLUNT CARDIAC INJURY SIGNS & SYMPTOMS

- Cardiac dysfunction
- Stiff Ventricle: S_3 or S^4
- Right sided symptoms:
  - Distended neck veins
  - ↑ Central Venous Pressure
  - Engorged liver, peripheral edema

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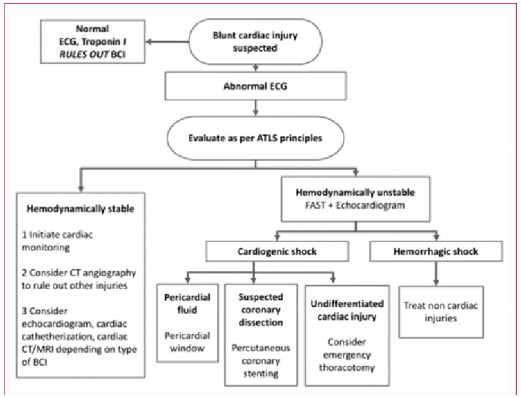
BLUNT CARDIAC INJURY DIAGNOSIS



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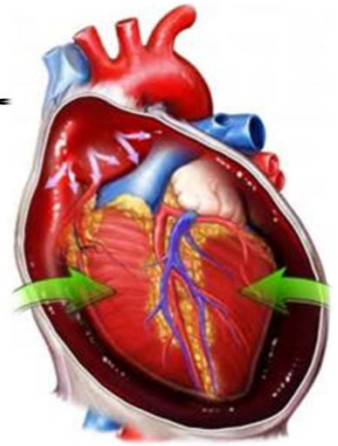
BLUNT CARDIAC INJURY TREATMENT

- Increase oxygen supply: supplemental O2
- Decrease myocardial oxygen demand
  - Bed Rest
  - Anxiolytics
  - Treat pain

39

CARDIAC TAMPONADE: ETIOLOGY

- Cardiac Trauma
- Post cardiac surgery or PTCA
- Post removal of epicardial pacing wires
- Pericarditis
- Infections
- Anticoagulant therapy
- Infection, metabolic disease, connective tissue disease



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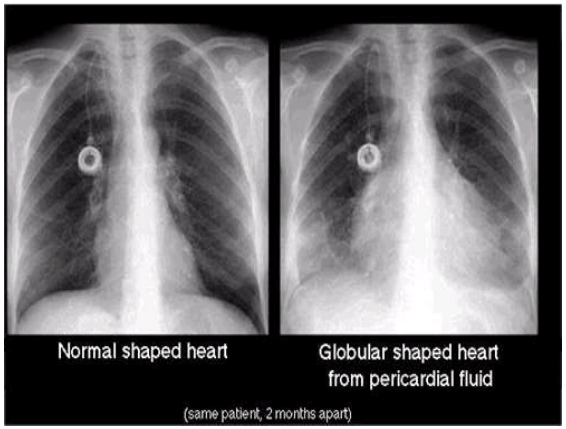
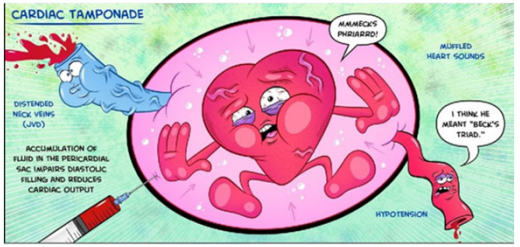


41

CARDIAC TAMPONADE: SIGNS & SYMPTOMS

- Muffled heart tones
- Tachycardia
- Distended jugular veins
- Hypotension
- Narrowing pulse pressure



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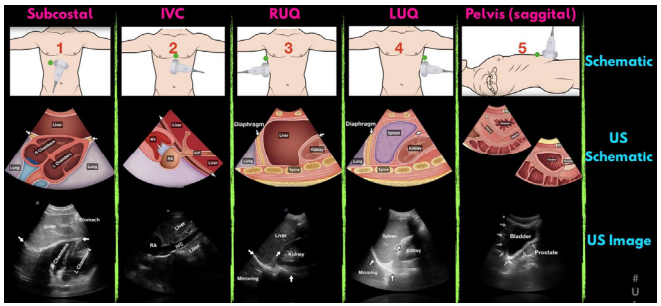
E-FAST

(EXTENDED FOCUSED ASSESSMENT WITH SONOGRAPHY FOR TRAUMA)



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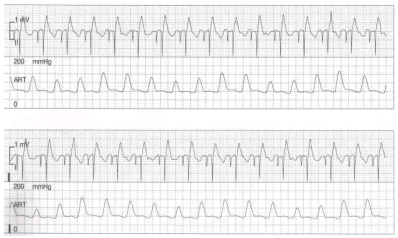
CARDIAC TAMPONADE: SIGNS & SYMPTOMS

- Pericardial friction rub
- Pulsus Paradoxus (auscultator gap > 20 mmHg when measuring blood pressure or a visual with Art Line)



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CARDIAC TAMPONADE TREATMENT

- Pericardiocentesis
- Surgery: window

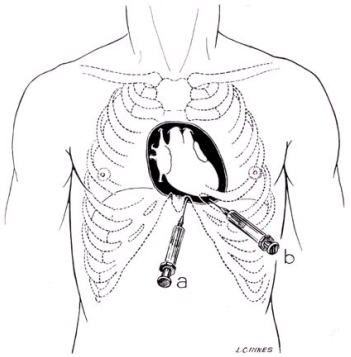

FIGURE 20.—Management of cardiac tamponade by aspiration: Substernal transdiaphragmatic aspiration (a), and left lateral aspiration (b).

45

ELECTRICAL INJURIES

- Tissue damage due to conversion of electrical energy into thermal energy
  - Large release of catecholamines
  - Myocytes may be stunned, injured, or damaged
  - Results in decrease of contractility and CO
- Treatment
  - Manage dysrhythmias
  - Manage volume status/heart failure

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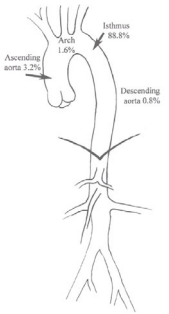
GREAT VESSEL INJURY: AORTIC TRANSECTION

- Exceleration/deceleration injury
- Car passenger hits steering wheel or dash
- Ligamentum arteriosum
  - Anchor



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TRANSECTION OF AORTA

- Rapid triage and
- Rapid treatment
- Signs and symptoms often covered up by pain of the cause: hitting the steering wheel or dash
- Suspect this trauma when the situation suggests the possibility

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QUESTIONS?

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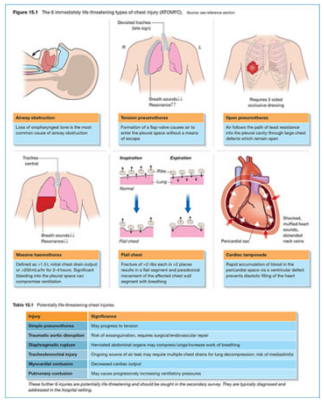


Multisystem Issues

KELLY URBAN, PHD, MED, RN, CCRN-K, TCRN, CCRN

1

Content

- OB Complications
- TTM/Malignant hyperthermia
- Comorbidity with transplant history
- EOL, palliative care, failure to thrive
- Bariatrics complications
- Pain
- Sleep disruption
- Submersion injuries

2

OB Complications

- Eclampsia
- HELLP
- Maternal/fetal transfusion
- Placental Abruption
- Uterine Rupture
- Placenta Previa

3

Preeclampsia

Definition:

- Multisystem disorder associated with decreased oxygenation and perfusion
  - Associated with coagulopathies, liver function abnormalities
  - Characterized by gestational hypertension, gestational proteinuria
  - Mild to severe
  - Not common, but can present postpartum

Assessment:

- Urgent obstetric consult
- Continuous fetal monitoring

4

Preeclampsia - Interventions

- Support maternal ABCs
- Minimize stimulation
- Antihypertensives, magnesium infusion
- Admission to OB unit

- Magnesium infusion
  - Loading dose
  - VS every 5 minutes
  - Infusion
  - Assess neuro status hourly
  - Monitor urinary output
- Magnesium toxicity
  - Loss of patellar reflexes
  - Shortness of breath, hypoxia
  - CNS depression
  - Nausea
  - Treat with calcium gluconate

5

Mild-Moderate Preeclampsia

Clinical manifestations

- SBP greater than 140 mm Hg or DBP greater than 90 mm Hg
- Proteinuria, oliguria
- Edema
- Weight gain of 2 pounds per week or more
- Headaches
- Nausea
- Epigastric or RUQ pain

6

Severe Preeclampsia

Clinical manifestations

- SBP above 160 mm Hg or DBP above 110 mm Hg
- Proteinuria above 2 g/24 hours
- Creatinine above 1.2 mg/dL
- Platelet count below 100,000 per microliter of circulating blood
- Elevated LDH, ALT, AST
- Persistent headache, visual disturbances, epigastric pain
- Oliguria
- Pulmonary edema

Assessment
Urgent obstetric consult
Continuous fetal monitoring

7

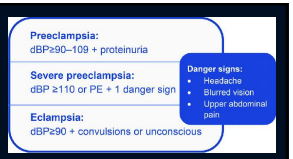
Eclampsia

Definition

- Preeclampsia that has progressed to convulsive state
- Delivery is only cure
- At risk for up to 3 weeks postpartum

Clinical manifestations

- Symptoms of preeclampsia
- Generalized seizures
- Significantly elevated BP
- Decreased fetal heart rate

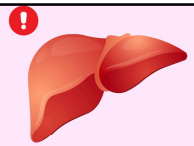


Interventions

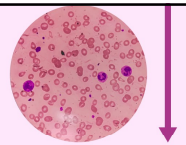
- Support ABCs
  - Patent airway
  - High-flow supplemental oxygen
- Continuous fetal monitoring
- Administer magnesium sulfate
  - Monitor for toxicity
- Antihypertensives
- Admission to OB unit

8

Haemolysis



Increased liver enzymes



Low platelet levels

HELLP Syndrome

- Life-threatening pregnancy complication
- Usually considered to be a variant of severe preeclampsia that involves the liver
- A group of symptoms that occur in pregnant women including:
  - H-Hemolysis
  - EL-Elevated liver enzymes
  - LP-Low platelets

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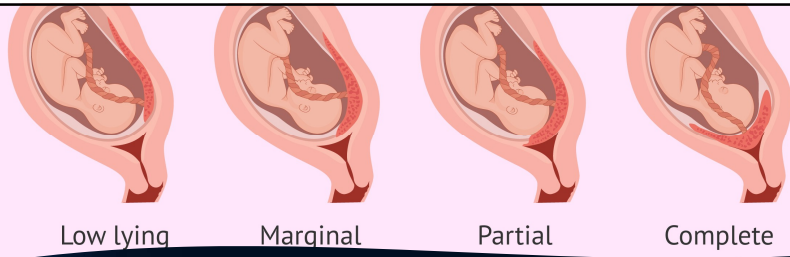


Low lying Marginal Partial Complete

Placenta Previa

- Definition: Implantation of placenta in lower uterine segment or over internal os
  - Placenta may bleed as fetus grows and uterus expands
  - Associated with life-threatening hemorrhage, fetal loss
- Clinical manifestations
  - Sudden, painless bleeding
  - Bright red blood
  - Maternal hemorrhagic shock

10

Placenta Previa – Interventions

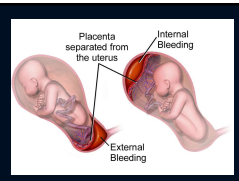
- Urgent obstetric consult
- Left lateral recumbent position
- Large-bore IVs, fluid bolus
- CBC, type and crossmatch with Rh
- No vaginal examination until ultrasound completed
- Prepare for possible C-section delivery

11

Abruptio Placentae

Placental separation from uterine wall

- Rupture of arterial vessels
- Major cause of obstetric hemorrhage, hypovolemic shock
- Inhibits supply of oxygen, nutrients to fetus
- Most common cause of fetal demise from maternal trauma



Clinical Manifestations

- Backache, painful contractions, abdominal pain
- Uterine rigidity
- Frank, dark-red vaginal bleeding or concealed bleeding
- Maternal hemorrhagic shock
- Coagulation panel, elevated D-Dimer

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Abruptio Placentae

ASSESSMENT

- Urgent obstetric consult
- CBC
- Type and crossmatch with Rh
- Continuous electronic fetal heart monitoring

INTERVENTIONS

- Transport to tertiary center if appropriate; or to obstetrics
- High-flow supplemental oxygen
- Fluid resuscitation and transfusion
- Emergent delivery

13

Targeted Temperature Management (TTM)

- Also known as Therapeutic Hypothermia
- Indications: all adult patients with ROSC after cardiac arrest who are comatose

Exclusion Criteria:
- Terminal conditions
- DNR status
- Resuscitation lasted > 1 hour

14

TTM – Process

- Induction – target temp 32° C and 36° C (target reached within 4 hours)
- Maintenance – 24 hours (avoid large deviations in fluctuation)
- Rewarming – slowly rewarm (0.2-0.5° C per hour) to 3.65° to 37° C

15

TTM – Nursing Care

- Eye care
- VTE ppx
- Prevent infection (bundles)
- Maintain skin integrity
- Nutrition and hydration management
- Prevention of fever

Management of Shivering:
- Nonpharmacologic: cover head, hands, feet
- Pharmacologic: NSAIDs, analgesics, sedatives, opiates, neuromuscular blockers, & magnesium sulfate

Effects of Hypothermia on medications:
- Lower metabolism/excretion
- Increased protein binding

16

Malignant Hyperthermia

Rare, inherited disorder that can cause a life-threatening reaction to certain anesthetics and muscle relaxants used during surgery.

- Signs/Symptoms:
  - Rapidly rising body temperature
  - Muscle rigidity
  - Increased heart rate and blood pressure
  - Rapid breathing
  - Confusion or altered mental status
  - Dark-colored urine

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Malignant Hyperthermia - Treatment

- Stop the triggering anesthetics and muscle relaxants
- Administer dantrolene
  - Dantrolene blocks the release of calcium in the muscles, preventing further muscle contractions and heat production.
- Other supportive measures may include:
  - Cooling the body down
  - Giving fluids and electrolytes
  - Treating complications such as AKI

18

Comorbidity with Transplant History

- Chronically immunosuppressed
- Risk of Infection

19

End of Life, Palliative Care

- Palliative Care
  - Can be initiated anytime within course of disease or life-threatening illness
  - Most beneficial when initiated early
- Hospice
  - Focus is on needs of those with < 6 months to live
  - Grief and bereavement services are included
- End-of-Life Care
  - Supports needs of those who are imminently facing death

20

Transitioning GOC to EOLC

- 5 aspects of quality EOLC include
  1. Pain and symptom management
  2. Avoid prolongation of the dying process
  3. Sense of self-control
  4. Relief of burden on the family
  5. Strengthened relationships with loved ones

21

Failure to Thrive

Syndrome of generalized decline, including weight loss, poor nutrition, and functional disability, often linked to an underlying acute or chronic medical condition

22

Failure to Thrive - Characteristics

- Unexplained weight loss
- Nutritional impairment
- Functional decline
- Symptoms: general weakness, fatigue, confusion, inactivity
- Underlying conditions: infections, malignancies, organ diseases
- Multifactorial

23

Bariatrics Complications

- Severe Obesity (BMI 40 or greater)
- Obesity etiology is complex and multifaceted
- Prevalence: > 1/3 of US adults are obese
- Increased risk for all complications related to immobility (i.e. skin breakdown, cardiac deconditioning, atelectasis, DVT, muscle atrophy, urinary stasis, constipation, bone demineralization)

24

Pathophysiology & Health Problems associated with Obesity

PULMONARY

- ↓ Vital Capacity, FRC
- ↓ Alveolar ventilation
- ↓ Expiratory reserve volume
- ↓ Thoracic and pulmonary compliance
- ↑ Work of Breathing
- ↑ Respiratory Drive, chronic CO2 retention

CARDIOVASCULAR

- ↑ L ventricular mass, hypertrophy dilatation
- ↑ Total blood volume (accumulated adipose tissue increases SV and CO)
- ↑ Cardiac deconditioning

Possible chronic hypoxemia, polycythemia, and pulmonary hypertension due to sleep apnea

25

Pathophysiology & Health Problems associated with Obesity

ENDOCRINE

- ↑ Metabolic requirements of excess adipose tissue
- ↑ Insulin resistance
- Stress of critical illness may deplete protein rather than glucose stores

GASTROINTESTINAL

- ↑ Intra-abdominal pressure
- ↑ Gastric volumes
- ↑ Nutritional requirements affected by mobilization of protein rather than lipid stores for ↑ energy needs
- Hypermetabolism associated with critical illness may lead to malnutrition and depleted protein reserves

26

Pathophysiology & Health Problems associated with Obesity

IMMUNE

- Protein-energy malnutrition that may coexist with obesity can impair cell-mediated immunity, phagocyte function, complement system, and antibody concentrations
- Impaired immune response

MUSCULOSKELETAL

- ↑ Joint trauma
- ↑ Pain with movement
- ↑ Disuse atrophy of musculature

Impaired, low, or no mobility

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Pathophysiology & Health Problems associated with Obesity

GENITOURINARY

- ↑ Intra-abdominal pressure
- ↑ Estrogen levels

PSYCHOSOCIAL

- ↑ Perceived or actual social rejection or lack of compassionate care from health care providers
- ↑ Anxiety, self-induced social isolation

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Pain

- Treating pain 1st can prevent the development of agitation and delirium
- Pathophysiological effects and manifestations of acute pain
  - Amplifies body's stress response
  - Causes endocrine and metabolic abnormalities
  - Impedes a patient's recovery
  - Can become refractory

29

Types of Pain

- Acute Pain
  - Directly related to tissue damage (sudden onset, short duration)
  - Can transition to chronic pain
- Chronic Pain
  - Gradual onset
  - Can be chronic musculoskeletal pain, neuropathic pain, malignant pain)
- Nociceptive Pain
  - Visceral pain (poorly localized and can be referred pain)
  - Somatic pain (localized pain)
- Neuropathic Pain
  - Stimuli abnormally processed by the nervous system
  - Examples include: phantom limb pain, post-mastectomy syndrome, post-thoracotomy syndrome, diabetic neuropathy, carpal tunnel syndrome

30

Pain – Summary of WHO Pain Relief Ladder

- Nonpharmacological methods
- Nonopioids +/- adjuvant analgesic
- Weak opioids
- Strong opioids

31

Sleep Disruption (Sleep Deprivation)

- Caused from light, noise, and clinical care
- The more critically ill, more likely to have sleep deprivation
- Treating sleep disruption may decrease delirium development

32

Sleep Disruption (Sleep Deprivation)

- Signs & Symptoms
  - Altered mental status (confusion, delusions)
  - Decreased alertness
  - Irritability
  - Aggressive behavior
  - Restlessness
  - Anxiety
  - Exhaustion

33

Sleep Interventions

- Increase total sleep time
- Cluster activities
- Prioritize activities
- Decrease noise level
- Decrease overhead lighting
- Provide adequate pain relief
- Decrease use of antihistamines, benzodiazepines, narcotics
- Promote nonpharmacologic sleep hygiene
  - Music therapy
  - Back rub
  - Bathing
  - Aromatherapy

34

Submersion Injuries (Near-Drowning)

- Primary respiratory impairment from submersion in a liquid
- 3rd leading cause of unintentional injury
- Electrolyte change:
  - Salt water: hemoconcentration
  - Fresh water: hemodilution

35

Submersion Injuries (Near-Drowning) – Pulmonary Effects

- Aspiration effects:
  - Fresh water: water rapidly enters circulation
  - Salt water: hypertonic sea water draws fluid from circulation into the lungs
- Organic & inorganic contents aspiration
- Destruction of surfactant → atelectasis
- Regional hypoxia → hypoxic vasoconstriction → ↑ pulm vasc pressures → further interstitial fluid → pulm edema

36

Submersion Injuries (Near-Drowning) – Major Insults

- Hypoxemia, tissue hypoxia
- Hypoxic brain injury with cerebral edema
- Hypercapnia
- Acidemia
- Hypothermia
- PNA
- DIC (rare)
- Acute Renal Failure (Acute Kidney Injury)
- Hemolysis

37

Submersion Injuries (Near-Drowning) – Goals of Care

- Effective ventilation and perfusion
- Rewarmed
- Gastric contents evacuated
- Complications are minimized

Potential Complications:
- Hypothermia
- Fluid & Electrolyte imbalances
- Seizures
- ARDS
- Aspiration PNA
- Pulmonary Edema
- Sepsis

38

Questions?

39

Musculoskeletal

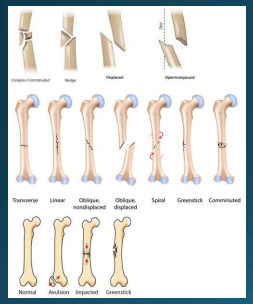
1

Musculoskeletal Content

- Compartment Syndrome
- Fractures
- Muscular Deconditioning
- Musculoskeletal Trauma

2

Types of Fractures



Fracture	Classification
Open	Fracture site is accompanied by compromised skin integrity near or over the fracture
Closed	Skin is intact over or near fracture site
Complete	Bony cortex is completely interrupted
Incomplete	Bony cortex is not completely interrupted
Comminuted	Bone is splintered into fragments
Greenstick	Bone bends or is buckled
Impacted	Bone is wedged into distal and proximal fracture sites
Displaced	Bone fracture sites are not aligned

3

Open Fractures

- Assessment findings:
  - Open wound over or near a fracture
  - Open wound with protrusion of bone
  - Pain
  - Neurovascular compromise
  - Controlled or severe bleeding
- Considered contaminated, at risk for infection

4

Fractures & Dislocations Clavicle/Scapular

- Injury: Clavicular and scapular fractures
- Symptoms:
  - Clavicle: obvious deformity to shoulder
  - Scapula: pain to area exacerbated by movement
- Treatment: sling and rest

5

Fractures & Dislocations Clavicle/Scapular

- Associated Injuries:
  - Large vessel injury
  - Pulmonary injury
  - Rib fractures
  - Cervical spine fractures
- Scapular fractures are rate and associated with great force
  - May cause brachial plexus injuries, splenic injuries, and humerus fractures

6

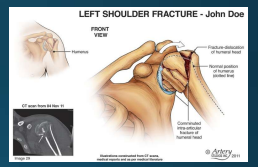
Fractures & Dislocations Shoulder

- Shoulder Fracture
- Anterior Shoulder Dislocation
- Posterior Shoulder Dislocation

7

Fractures & Dislocations Shoulder Fracture

- Symptoms
  - Pain and deformity
- Treatment
  - Shoulder immobilizer
  - Sling with swathe to body
  - 20% will require surgical intervention



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Fractures & Dislocations
Anterior Shoulder Dislocation

- Symptoms
  - Visible deformity with abduction and external rotation of the arm
  - Unable to bring affected arm high enough to touch ear on opposite side of dislocation
- Treatment
  - Relocated under moderate sedation
  - Sling with swathe to body
- Notes
  - Often caused by falling on an outstretched arm

9

Fractures & Dislocations
Posterior Shoulder Dislocation

- Symptoms
  - Arm held in adduction with internal rotation and visible deformity
- Treatment
  - Dislocations are relocated with moderate sedation
  - Sling with swathe to body
- Notes
  - Rare injury caused by arm being forced while extended (i.e. during seizure)

10

Fractures & Dislocations
Mid-Shaft Humerus Fracture

- Symptoms
  - Obvious deformity and pain
- Treatment
  - Allow arm to hang so weight of the elbow helps reduce the fracture
  - May require casts/slings
  - May require surgery (depending on severity and nerve involvement)
- Common Complications:
  - Brachial artery injury
  - Radial nerve injury
  - Fat emboli

11

Fractures & Dislocations
Complete Elbow Dislocation

- Radius and ulna dislocation
- Symptoms
  - Obvious loss of arm length
  - Rapid swelling
- Treatment
  - Reduction and application of supportive splint
- Notes
  - Fall on outstretched hand
  - Frequently involves neurovascular involvement (brachial artery laceration, median nerve impingement or damage)

12

Fractures & Dislocations
Fractured Elbow

- Symptoms
  - May or may not have deformity
  - Pain at joint
- Treatment
  - Non-displaced: sling and encouragement of early use
  - Displaced or Fracture with Nerve Involvement: surgery
- Notes
  - Assess for brachial artery involvement
  - Assess for nerve involvement (typically median nerve)

13

Fractures & Dislocations
Forearm Fracture

- Fracture with obvious deformity
- Monteggia's Fracture
  - Fracture of the proximal 1/3 of the ulna with radial head dislocation
- Nightshift Fracture
  - Fracture of the mid-shaft of the ulna from a direct blow

14

Fractures & Dislocations
Forearm Fracture

- Treatment
  - Closed reduction with cast application
  - Cast should be applied with elbow at 90 degrees
- Patient Teaching:
  - Prevent dependency of arm or drooping of wrist inside sling after cast application

15

Fractures & Dislocations
Wrist

- Deformity
  - Smith's Fracture:
    - looks like a hoe when visualized laterally
    - Upward displacement of the distal radius and ulna
  - Colle's Fracture:
    - looks like a fork when visualized laterally
    - Downward displacement of distal radius and ulna
- Treatment
  - Manipulation, closed reduction, application of cast
  - Assess for involvement of median nerve

16

Fractures & Dislocations
Scaphoid Fracture

- Symptoms
  - Wrist pain/pain with pressure on snuff box area of hand
- Treatment
  - Cast with thumb in opposition

17

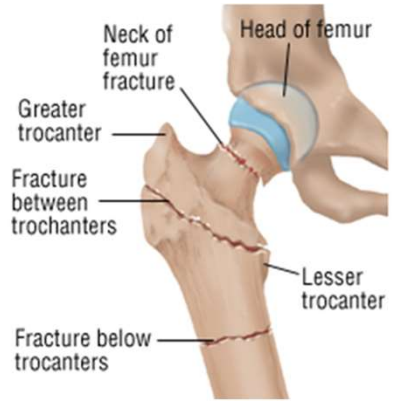
Fractures & Dislocations
Hip

- Hip Fracture
- Anterior Hip Dislocation
- Posterior Hip Dislocation

18

Fractures & Dislocations
Hip Fracture

- Symptoms
  - External rotation
  - Leg shortening
  - Pain
- Treatment
  - Surgery



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Fractures & Dislocations
Anterior Hip Dislocation

- Symptoms
  - Extreme external rotation
  - Less pronounced abduction and flexion
- Treatment
  - Reduce within 6 hours to prevent femoral head necrosis

20

Fractures & Dislocations
Posterior Hip Dislocation

- Symptoms
  - Hip flexed
  - Internally rotated and adducted
- Treatment
  - Reduce within 6 hours to prevent femoral head necrosis

21

Fractures & Dislocations
Femur Fracture

- Symptoms
  - Obvious deformity and pain
- Treatment
  - Traction splint
    - For mid-shaft and upper 1/3 fractures of tibia
    - Do NOT apply if have hip injury or if involving lower tibia/fibula or ankle
  - Surgery

22

Fractures & Dislocations
Femur Fracture

- Complications
  - Hypovolemia (up to 3L blood loss)
  - Damage to peroneal nerve, sciatic nerve, and popliteal nerve

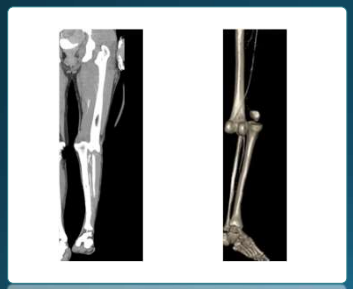
23

Fractures & Dislocations
Knee

- Knee fracture/dislocation
- Patella dislocation
- Patella fracture

24

Fractures & Dislocations
Knee Fracture/Dislocation



- Symptoms
  - Obvious deformity
  - Pain
  - Inability to bear weight
- Treatment
  - Knee immobilizer
  - Surgery
- Common Complications
  - Damage to peroneal/tibial nerves
  - Popliteal artery involvement

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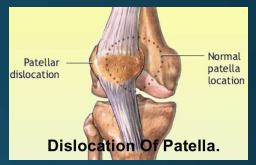
Fractures & Dislocations
Patella Dislocation

- Symptoms
  - Inability to straighten the joint (lateral dislocation)
- Treatment
  - Straighten the leg to relocate
  - Apply knee immobilizer
- Common mechanism: frontal MVC



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Fractures & Dislocations
Patella Fracture

- Symptoms
  - Pain
  - Obvious deformity of patella
  - Swelling
- Treatment
  - Knee immobilizer
  - Surgical intervention
- Common mechanism: Frontal MVC

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Fractures & Dislocations
Tibia/Fibula Fracture

- Symptoms
  - Obvious deformity
  - Pain
  - Inability to bear weight
- Treatment
  - Cast
- Common Mechanism: Auto vs Pedestrian
- Common Complications
  - Blood loss (up to 2 L)
  - Infection (open fracture)
  - Soft tissue damage
  - Neurovascular compromise

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Fractures & Dislocations
Ankle Fracture/Dislocation

- Symptoms
  - Edema
  - Tenderness
  - Loss of alignment
  - Loss of function
  - Possible deformity
- Treatment
  - Depends on severity
    - Closed reduction with walking cast
    - Open reduction and fixation
- Common Complications:
  - Neurovascular compromise – peroneal nerve

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Fractures & Dislocations
Calcaneus Fracture

- Symptoms
  - Pain with hyperflexion of foot
- Treatment
  - Compression dressing
  - Crutches
- Notes
  - Associated with falls resulting in feet-first landing → axial loading with potential vertebral fractures and tibial plateau fractures

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Crush Injury

- Direct injury resulting from crush
- Crush Syndrome is the systemic manifestation of muscle cell damage resulting from pressure or crushing

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Crush Injury

- Complications
  - Compartment syndrome
  - Hyperkalemia
  - Rhabdomyolysis
  - Hypovolemia

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Amputations

- Partial or complete
- Priority is to focus on overall assessment and resuscitation of patient (control hemorrhage)

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Amputations

- Assessment Findings
  - Obvious tissue loss
  - Pain
  - Controlled or severe bleeding
  - Evidence of hypovolemic shock may or may not be present

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Amputations

- Treatment – Control Hemorrhage
  - Priority: control hemorrhage
  - Direct pressure over stump
  - Compress artery above the site
  - Elevate extremity
  - Apply tourniquet as needed

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Amputations

- Treatment – Care of Amputated Part
  - Remove dirt and debris from the exposed end
  - Wrap in slightly saline-moistened sterile gauze
  - Place in sealed bag
  - Place on ice (½ water and ½ ice)

Do NOT allow water on the amputated part → edema

Do NOT allow the part to freeze

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Potential Complications

- Blood Loss
- Nerve Impingement
- Fat Emboli
- Compartment Syndrome
- Hyperkalemia
- Rhabdomyolysis

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Blood Loss

- Pathophysiology
  - Disruption of arteries, veins, and capillaries

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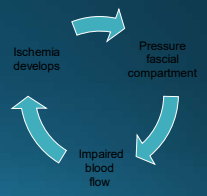
Compartment Syndrome

- Increased pressure in fascial compartment
- Increased pressure inhibits blood flow → muscle and nerve damage or destruction
- Muscle necrosis can occur in 4-6 hours
- Most frequent sites:
  - Muscles of lower leg
  - Muscles of forearm



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Compartment Syndrome

- Common Causes:
  - Hematoma formation (secondary to fractures)
  - Internal Sources
    - Hemorrhage
    - Edema
  - External Sources
    - Casts
    - Dressings
    - Traction/air splints

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Compartment Syndrome Symptoms

Symptom	Phase	Description
Pain	Early	• Pain out of proportion to the injury • Not relieved by opioids • Pain on passive motion
Pressure	Early	• Limb feels tight or tense on palpation • Skin may appear taut or shiny
Pallor	Late	• Indication of poor perfusion
Pulses	Late	• Weak or absent pulses are a poor indicator or positive outcomes
Paresthesia	Late	• Numbness, tingling, loss of sensation
Paralysis	Late	• Indicates injury to nervous system

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Compartment Syndrome

- Diagnosis
  - Measurement of intra-compartmental pressures

Pressure	Description
0-10 mmHg	Normal
> 20 mmHg	Elevated
> 30 mmHg	Ischemia to muscles/nerves
> 40 mmHg	Need to open compartment!

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Compartment Syndrome

- Treatment
  - Remove external pressure (casts, splints, dressings)
  - Elevate limb TO the level of the heart
  - Anticipate fasciotomy, surgical debridement, or amputation

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Hyperkalemia

- Peaks 12-36 hours after the injury then steadily decreases
- Treatment:
  - Cardioprotection (if dysrhythmias):
    - Calcium gluconate
  - Temporary potassium reduction:
    - Insulin/glucose
    - Nebulized beta-adrenergic agonists
  - Permanent potassium reduction:
    - Diuresis
    - Intestinal potassium binders (kayexalate)
    - Dialysis

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Rhabdomyolysis

- Release of myoglobin from severe muscle or cellular destruction

- Symptoms
  - Muscle pain
  - Numbness
  - Changes in sensation
  - Weakness/paralysis
  - **Dark red/brown urine**
  - **Elevated CK levels (5-10 x > normal)**

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Rhabdomyolysis

- Treatment
  - Isotonic Crystalloids
    - Goal: urine output 100 ml/hour
  - Alkalinization of Urine
    - Sodium bicarbonate
    - Osmotic diuretics (mannitol)
    - Goal: urine pH > 8.0 (prevents crystallization of myoglobin)
  - Hemodialysis/Peritoneal Dialysis/ Renal Replacement Therapy
    - Goal: preservation of the kidneys

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Fat Emboli

May occur up to 12 hours – 2 weeks after a long bone fracture (most commonly 24-72 hours)

Most cases are asymptomatic

5-15% are lethal

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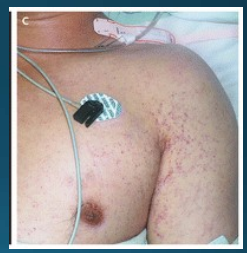
Fat Emboli

- Symptoms:
  - Altered mental status
  - Respiratory distress
  - Tachycardia/hypotension
  - Petechial rash on head, neck, anterior thorax, conjunctivae, buccal mucous membranes, and axillae



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Fat Emboli

Diagnosis
- Helical CT
- Chest Xray (patchy infiltrates)
- ABGs

Treatment
- Oxygenation
- Ventilation
- Correction of hemodynamic instability

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Muscular Deconditioning

- Decline in muscle strength, size, and endurance due to prolonged inactivity or reduced physical activity
- Causes:
  - Prolonged bed rest or inactivity
  - Hospitalization
  - Aging
  - Lack of exercise
  - Malnutrition

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Muscular Deconditioning

- Effects:
  - Reduced muscle strength and endurance
  - Decreased muscle mass (atrophy)
  - Increased risk of falls and injuries
  - Impaired mobility and function
  - Reduced quality of life

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Muscular Deconditioning

- Prevention
  - Get out of bed
  - Walk
  - In-bed or in-chair exercises
  - Position changes

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Care of the Burn Patient

KELLY URBAN, PHD, MED, RN, CCRN-K, TCRN, CEN

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Burn Statistics

Worldwide, an estimated 265,000 deaths every year are caused by burns

Non-fatal burn injuries are a leading cause of morbidity

Deaths from burn injury increase with advancing age and burn size, and presence of inhalation injury

In the US, 486,000 people receive medical treatment for burns
- Survival Rate: 96.7%
- Gender: 69% male, 31% female
- 69% occur in the home

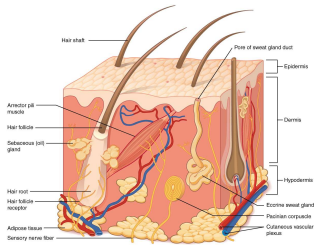
2

Skin

FUNCTIONS

- Protect from infection
- Prevention of loss of body fluids
- Thermoregulation
- Production of vitamin D
- Excretion
- Determination of identity
- Sensation reception

LAYERS



Epidermis

Dermis

Hypodermis (subcutaneous)

3

Sources of Burn Trauma

Thermal
- Flame
- Scald
- Contact

Chemical

Electrical

Radiation

4

Chemical Burns (Acids/Alkalis)

Type of Chemical	Notes
Dry Chemicals	• Remove clothing 1st using caution not to scatter any of the agent • Brush remaining chemical off skin before cleansing with water
Hydrofluoric Acid	• Irrigate with water for 30 minutes • Apply calcium chloride gel to exposed area (changing every 15 min until burning stops) • Monitor for hypocalcemia and administer IV calcium as needed
Tar and Asphalt	• Immediately cool the tar/asphalt with cool compresses or cool water • Remove cooled tar/asphalt with a fat emollient (mayo, cooling oil, mineral oil, etc…)
Phenols	• Irrigate copiously with water followed by application of 50% polyethylene glycol (PEG) • Phenol burns cause a thick eschar to the affected area if not removed quickly
Hydrocarbons	• Cause cell membrane dissolution and skin necrosis, leaving burns that are typically superficial and heal spontaneously. Skill will appear reddened and blistered. • Complications include respiratory failure and hepatic injury (delayed)

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Electrical Burns

- Electricity will heat up at the point of entry & exit creating surface burn wounds
  - Patients may have visible entrance and exit wounds, but tissue damage between these wounds may be internal and not visible
- Electricity seeks the path of least resistance
  - Skin and bone resist electricity
  - Muscle, blood, and nerve do not resist electricity
- Electricity will seek the ground
- AC energy causes muscle tetany which produces severe burns while DC decreases contact with the electrical source and may lead to less severe burns

6

Electrical Burns Care

- Monitor for onset of Compartment Syndrome
- Monitor for fractures and cervical spine injuries due to violent muscular contractions
- Damaged muscle may cause hyperkalemia, rhabdomyolysis, and elevated CK levels
- Damage to the heart:
  - 25% have dysrhythmias (most are benign)
  - Usually occurs immediately after shock but may occur up to 12 hours later
  - T wave and ST abnormalities may occur and resolve spontaneously
  - Fatal dysrhythmias are more common in horizontal (arm to arm) injuries

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Electrical Burns Care Cont.

- Fluid replacement will be more difficult due to inability to see all body areas burned.
  - Use Modified Parkland Formula but start with **4** ml x kg x TBSA
  - May consider starting with 20 ml/kg bolus and adjust as needed based on urine output
    - Desired Urine Output – 100 ml / hr
- Continuous Cardiac Monitoring
- Osmotic diuretics
- Sodium bicarbonate to alkalinize urine
- Monitor for compartment syndrome

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Radiation Exposure

Causes include: sun, radiation beams

Surface burns: skin will be red and moist like a partial thickness burn but without blistering

Indications of Radiation Sickness:
- Nausea/Vomiting
- Diarrhea
- Malaise
- Anorexia
- GI bleeding

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Thermal Burns

Burns caused by an external heat source

- Flame
- Flash
- Scald
- Contact

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Initial assessment of a major burn

- STOP the Burning Process!
- Perform an ABCDEF primary survey
- Assess burn size and depth
- Establish good intravenous access and give fluids
- Give analgesia
- Catheterize patient or establish fluid balance monitoring
- Take baseline blood samples for investigation
- Dress wound
- Perform secondary survey, reassess, and exclude or treat associated injuries
- Obtain thorough history
- Arrange safe transfer to specialist burns facility

A—Airway with cervical spine control
B—Breathing
C—Circulation
D—Neurological disability
E—Exposure with environmental control
F—Fluid resuscitation

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Airway management

SIGNS OF INHALATIONAL INJURY

- Erythema or swelling of oropharynx on direct visualization
- Full thickness or deep dermal burns to face, neck, or upper torso
- Singed nasal hair
- Hoarse voice
- Stridor
- Carbonaceous sputum or carbon particles in oropharynx

INDICATIONS FOR INTUBATION

- History of flame burns or burns in an enclosed space
- Change in voice, with hoarseness or harsh cough
- Stridor, tachypnea, or dyspnea

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Breathing

All burn patients should receive 100% oxygen through a humidified non-rebreathing mask on presentation.

- *Mechanical restriction of breathing*: Deep dermal or full thickness circumferential burns of the chest can limit chest excursion and prevent adequate ventilation. This may require escharotomies
- *Blast injury*—If there has been an explosion, blast lung can complicate ventilation. Penetrating injuries can cause tension pneumothoraces, and the blast itself can cause lung contusions and alveolar trauma
- *Smoke inhalation*—The products of combustion, though cooled by the time they reach the lungs, act as direct irritants to the lungs, leading to bronchospasm, inflammation, and bronchorrhoea. The ciliary action of pneumocytes is impaired, exacerbating the situation. The inflammatory exudate created is not cleared
- *Carboxyhaemoglobin*—Carbon monoxide binds to deoxyhaemoglobin with 40 times the affinity of oxygen. It also binds to intracellular proteins, particularly the cytochrome oxidase pathway. These two effects lead to intracellular and extracellular hypoxia.

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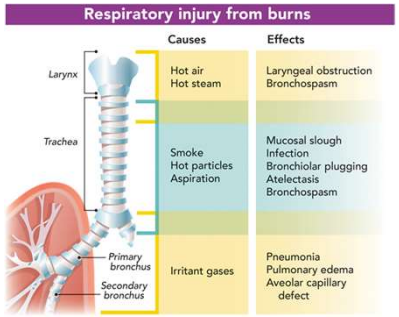
Inhalation Injury



2 requirements for inhalation injury diagnosis:

1. Exposure to combustible agent
2. Signs of exposure to smoke in lower airway (below vocal cords) confirmed by bronch

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Inhalation Injury Grade

Grade	Injury Severity	Signs and Symptoms
0	No injury	• Absence of carbonaceous deposits, erythema, edema, bronchorrhea, or obstruction
1	Mild injury	• Minor or patchy areas of erythema • Carbonaceous deposits in proximal or distal bronchi
2	Moderate injury	• Moderate degree of erythema • Carbonaceous deposits • Bronchorrhea • With or without compromise of bronchi
3	Severe injury	• Severe inflammation with friability • Copious carbonaceous deposits • Bronchorrhea • Bronchial obstruction
4	Massive injury	• Evidence of mucosal sloughing, necrosis, endoluminal obliteration

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Inhalation Injury Treatment

Table 6. Pharmacologic Treatment for Inhalation Injuries

Pharmacologic Modality	Effect
Nitric oxide	Decreases microvascular permeability by decreasing pulmonary vascular pressures and by decreasing the endovascular damage by reactive oxygen species. Also improves oxygenation by decreasing ventilation-perfusion mismatch
Albuterol	Decreases peak inspiratory pressures, improves pulmonary compliance, decreases shunt fraction, and increases the arterial to alveolar oxygen gradient
Inhaled epinephrine	Bronchodilatory effect along with a vasoconstrictive effect to reduce mucosal edema
Aerosolized antithrombin	Decreases the degree of airway obstruction in an ovine model of cutaneous and inhalation burn (Prospective human data not available.)

Adapted from: Fidkowski CW, et al. Inhalation burn injury in children. *Pediatric Anesthesia* 2009;19(Suppl. 1):147-154.

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Signs of Carboxyhaemoglobinaemia

COHb levels	Symptoms
0-10%	Minimal (normal level in heavy smokers)
10-20%	Nausea, headache
20-30%	Drowsiness, lethargy
30-40%	Confusion, agitation
40-50%	Coma, respiratory depression
>50%	Death

**Oxygen saturation will be normal

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Carboxyhaemoglobinaemia Treatment

- 100% oxygen (until asymptomatic or COHb < 10%)
  - Decreases the CO ½ life to 1 hour (normal ½ life is 4 hours when breathing room air)
- Monitor cardiac rhythm and COHb level
  - Hypoxic dysrhythmias
- Hyperbaric chamber may be required for carbon monoxide poisoning resistant to oxygen or for pregnant patients

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Pulmonary Injury

Caused by inhalation of noxious substances such as carbon or noxious fumes

- Damages mucosal cells or bronchioles
- Increases pulmonary capillary permeability (ARDS)
- Sloughing of damaged cells causing obstructed airways
- Decreased production of surfactant causing atelectasis
- Symptoms may be delayed as long as 24 hours after injury
- Pneumonia could result as a secondary complication

Treatment
- O2 with a goal of an SpO2 between 94-98%

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Circulation

- Any patient with burns > 20% TBSA require fluid resuscitation
- Two Large bore IVs through unburned tissue
- Blood for checking full blood count, urea and electrolytes, blood group, and clotting screen
- Peripheral circulation must be checked
- ECG to monitor for cardiac dysrhythmias (may be first sign of hypoxia or electrolyte/Acid-Base imbalance)

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Circulation

- Burns covering more than 20% of body surface may cause a systemic inflammatory response with systemic capillary leakage
- Fluid resuscitation may initially be guided by the Modified Parkland Burn formula- ***only used for partial or full thickness burns***

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Circulation – Modified Parkland Formula

Adult: TBSA x 2 ml x body weight in kg

Pediatric: TBSA x 3 ml x body weight in kg

Electrical: TBSA x 4 ml x body weight in kg

½ of volume to be given in 1st 8 hours (from time of burn)
Remaining ½ volume to be given over remaining 16 hours

Fluid of choice is Lactated Ringers

This is a guide to begin resuscitation, but fluid may be ↑ or ↓ based on urine output.
The adult target is 0.5 ml/kg/hr

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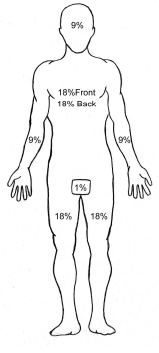
Adult TBSA using "Rule of Nines"

- Head (front and back) – 9%
- Chest & Abdomen – 18%
- Back – 18%
- Arms – 9% each
- Legs – 18% each
- Perineum – 1%



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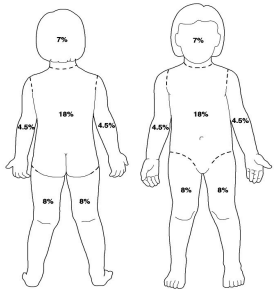
Pediatric TBSA



- Back of head – 7%
- Face – 7%
- Trunk – 18%
- Back – 18%
- Arms – 9% each (4.5 – front, 4.5 – back)
- Legs – 16% each (8 – front, 8 – back)

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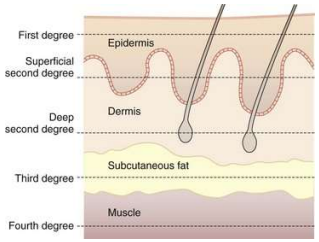
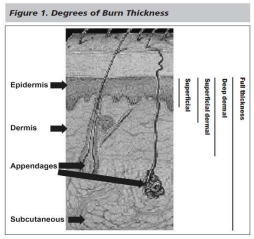
Burn Wound Assessment: Depth





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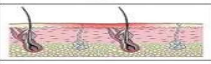
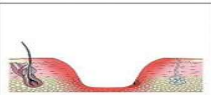
Depth Review

Degree	Anatomic correlate	Schematic aspect	Clinical aspect
I	Reddening, swelling, pain (epidermis)		
IIa	Reddening, blistering, pain (superficial dermis)		
IIb	Pallor, blister, pain (partial dermis)		
III	Greyish white or black necrosis, analgesia (complete dermis)		
IV	Carbonization (may extend to the bones and joints)		

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Burn Wounds

Depth	Level of Injury	Clinical Features	Result/Treatment
Superficial (first degree)	Epidermis	Dry, red; blanches; painful	Healing time 3–6 days, no scarring
Superficial partial thickness (superficial second degree)	Papillary dermis	Blisters; moist, red, weeping; blanches; severe pain to touch	Cleaning; topical agent; sterile dressing; healing time 7–21 days; hypertrophic scar rare; return of full function
Deep partial thickness (deep second degree)	Reticular dermis; most skin appendages destroyed	Blisters; wet or waxy dry; reduced blanching; decreased pain sensation to touch, pain present to deep pressure	Cleaning; topical agent; sterile dressing; possible surgical excision and grafting; scarring common if not surgically excised and grafted; earlier return of function with surgery
Full thickness (third degree)	Epidermis and dermis; all skin appendages destroyed	Waxy white to leathery dry and inelastic; does not blanch; absent pain sensation; pain present to deep pressure; pain present in surrounding areas of second-degree burn	Treatment as for deep partial-thickness burns plus surgical excision and grafting at earliest possible time; scarring and functional limitation more common if not grafted
Fourth degree	Involves fascia and muscle and/or bone	Pain to deep pressure, in the area of burn; increased pain in surrounding areas of second-degree burn	Healing requires surgical intervention

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First Degree Burns

Level of Injury
- Epidermis

Clinical Features
- Dry & Red but BLANCHES
- Painful

Result/Treatment
- Healing 3-6 days
- No scarring

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Second Degree (Superficial Partial Thickness) Burns

Level of Injury
- Reticular Dermis
- Most skin appendages destroyed

Clinical Features
- Blisters
- Moist, red, weeping
- BLANCHES
- Very painful

Treatment
- Keep Clean
- Healing time – 7-21 days

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Second Degree (Deep Thickness) Burns

Level of Injury
- Involves Epidermis and deep Dermis

Clinical Features
- Mottled white or white immediately after injury
- Blister or appear dry with cherry red color
- Does NOT blanch
- White and dry by day 2

Treatment
- Healing time: > 3 weeks
- May require skin grafting

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Third Degree (Full Thickness) Burns

Level of Injury
- Involves all layers of skin and subcutaneous fat

Clinical Features
- "leathery"
- "firm"
- Depressed compared to adjoining unaffected skin
- Insensitive to light touch or pin prick

Treatment
- Requires surgical grafting

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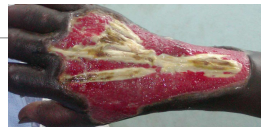
Fourth Degree Burns



Level of Injury
- Extends into underlying fat, tissue, muscle, and bone

Clinical Features
- Similar to full thickness burn but with obvious tissue loss

Treatment
- Surgical grafting

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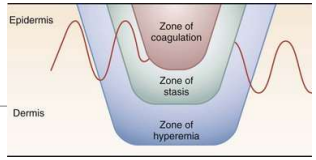
Histopathology (Zones of Burn Injury)



- Zone of Coagulation
  - Nonviable area of tissue at the epicenter of the burn
- Zone of Stasis
  - Surrounding tissues (both deep and peripheral) to the coagulated areas, which are not devitalized initially, but, due to microvascular insult, can progress irreversibly to necrosis over several days if not resuscitated properly
- Zone of Hyperemia
  - Peripheral tissues that undergo vasodilatory changes due to neighboring inflammatory mediator release (more information next slide) but are not injured thermally and remain viable

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Disability

Glasgow coma scale; they may be confused because of hypoxia or hypovolemia.

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Exposure

The whole of a patient should be examined (including the back) to get an accurate estimate of the burn area

Check for any concomitant injuries.

Burn patients, especially children, easily become hypothermic. This will lead to hypoperfusion and deepening of burn wounds. Patients should be covered and warmed as soon as possible.

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Key points of a burn history: Exact Mechanism

- Type of burn agent (scald, flame, electrical, chemical)
- Is there risk of concomitant injuries (such as fall from height, road traffic crash, explosion)?
- How did it come into contact with patient?
- Is there risk of inhalational injuries (did burn occur in an enclosed space)?
- What first aid was performed?
- What treatment has been started?

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Key points of a burn history: Exact timings

- When did the injury occur?
- How long was cooling applied?
- How long was patient exposed to energy source?
- When was fluid resuscitation started?

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Key points of a burn history: Exact Injury

Scalds

- What was the liquid? Was it boiling or recently boiled?
- What was the voltage (domestic or industrial)?
- If tea or coffee, was milk in it?
- Was a solute in the liquid? (Raises boiling temperature and causes worse injury, such as boiling rice)

Electrocution injuries

- Was there a flash or arcing?
- Contact time

Chemical injuries

- What was the chemical?

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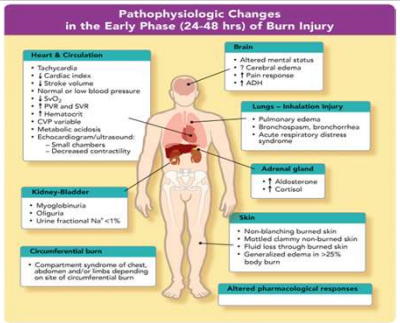
Burn Care after the Initial Assessment



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Burn Care - Brain

- Altered Mental Status?
  - Hypovolemia?
  - Carbon monoxide?
  - Head injury?
  - Cerebral Edema?
- ↑ Pain Response
- ↑ ADH

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Burn Care - Heart & Circulation

- Prevent hypovolemic shock
  - Volume requirements should be guided by urine output after the initial resuscitation
- Persistent acidosis – think about cyanide toxicity
- Tachycardia
- Arrhythmias
  - Electrolyte imbalance
  - Electrical burns
- Monitor distal pulses and nail beds

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Burn Care – Inflammatory Response

Table 2. Burn Mediators

Mediator	Effects
Thromboxane A2	Vasoconstrictor that can cause an increased area of ischemia and worsen the burn injury
Bradykinin	Local inflammatory mediator that increases vascular permeability
Catecholamines	Vasoconstrictors
Serotonin	Smooth muscle constrictor of large blood vessels
Platelet aggranulation factor	Can increase capillary permeability leading to edema
Angiotensin	Potent vasoconstrictor of terminal arterioles
Histamine	Thought to be responsible for the early onset of edema after the burn injury
PGE2	Causes an increase in vascular permeability and is a central mediator of fever
Vasopressin	Causes increased systemic vascular resistance
Corticotropin releasing factor	Natural inhibitor of the acute inflammatory response

Adapted from: Keck M, et al. Pathophysiology of burns. *Wien Med Wochenschr* 2009;159:327-336.

42

Burn Care - Lungs

- Maintain adequate oxygenation & ventilation

43

Burn Care - Adrenal Gland

Hypermetabolic response:

- ↑ Aldosterone
  - May be elevated for several days to weeks
- ↑ Cortisol

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Burn Care - Kidney-Bladder

- Watch for renal ischemia → renal failure
  - Hypovolemia?
- Renal failure may be more prevalent in:
  - Full Thickness burns
  - Electrical Burns
    - Myoglobin released from muscle cells causing Rhabdomyolysis

45

Burn Care - Nutrition

- Insert gastric tube for burns greater than 20% TBSA
  - Gastric distention can cause nausea and vomiting
- Early nutrition is essential
  - To counteract effects of hypermetabolism and increased levels of inflammatory mediators
- Enteral feeding is best practice

46

Circumferential Burn

- Limb is burned all the way around
- Soft tissues under the skin swell, and there is loss of skin expansion
- Pressure inside the limb increases, eventually exceeding the arterial pressure
- Perfusion to the limb is lost

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Circumferential Burn

Escharotomy

- Limb threatening complications can result when a full thickness circumferential injury is present
- Assessment of pulses is paramount
- Escharotomy is performed to release the burn eschar which is encumbering blood flow to distal extremities
- Escharotomies of the chest wall may also be necessary with a circumferential burn.
  - Signs: peak pressures on ventilator and decreasing tidal volumes

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Escharotomy

Figure 4. This figure demonstrates appropriate chest escharotomy. Notice the amount chest wall expansion following escharotomy, i.e. the skin gap seen at the sight of escharotomy.

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Skin

Hypothermia may lead to coagulopathies

- Cover wounds with clean, dry dressing or sheets to reduce air currents
- Do NOT apply ice
- Maintain body temperature at normal (patients are at risk for poikilothermy)

Poikilothermy:
assumption of room temperature

50

Phases of Wound Healing

Phase	Purpose	Characteristics	Key Information
Inflammatory	• Prevent infection • Degrade necrotic tissue	• Vasodilation • Fluid extravasation • Edema	• Neutophils & monocytes go to site of injury initiating the immune response • Immune response also sustained by macrophages
Proliferative	• Begin wound closure	• Wound closure • Revascularization	• Keratinocyte & fibroblast activation
Remodeling	• Wound contracture	• Wound maturation • Scarring	• Collagen and elastin are deposited and reformed as fibroblasts and myofibroblasts

51

Burn Wound Care – Topical Wound Management

- If anticipating early transfer (< 24 hours) to a burn center → do NOT debride the burn or apply topical antimicrobial agents
- Do not leave wet dressings or sheets on patient
- If patient to remain at non-burn facility for > 24 hours:
  - Contact burn center for further wound care
  - General rule: bedside cleaning with soap and water followed by application of silver sulfadiazine cream or alternative

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Burn Wound Care – Wound Coverage and Grafting

- Excision within 24-48 hours after injury is associated with decreased blood loss, infection, length of hospital stay, and mortality
- Standard for rapid and permanent closure of full thickness burns is a split-thickness skin graft from an uninjured donor site on the same patient
- Patients with more extensive burns often require temporary coverage with an allograft, xenograft, skin substitute, or dermal analog

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Burn Wound Care – Dressings

The selection of an appropriate dressing depends on several factors:

- Depth of burn
- Condition of the wound bed
- Wound location
- Desired moisture retention and drainage
- Required frequency of dressing changes
- Cost

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Burn Wound Care – Dressings

Product Name	Classification	Origin	Characteristics
Alloderm	Acellular	Human	Dermal matrix
GraftJacket	Acellular	Human	Tissue scaffold
Integra	Acellular	Bovine/shark	Bilayer matrix
Biobrane	Acellular	Biocomposite	Nylon fibers in silicone with collagen
Dermagraft	Cellular	Neonatal	Bioabsorbable polyglactin mesh scaffold with human fibroblasts
Epicel	Cellular	Keratinocyte-based	Cultured epidermal autograft
Recell	Cellular	Autologous	Cell suspension of keratinocytes, fibroblasts, Langerhan cells and melanocytes

55

Special Burn Wound Situations

- Hand and foot burns have a high risk for post-burn strictures and necessitate burn center care along with intense rehabilitation
- Perineal burns have a high risk for contamination and infection

56

Pain Control

Pain should be differentiated from anxiety

- Pain
  - Opiods given IV (never IM)
- Anxiety
  - Small doses of benzodiazepines

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American Burn Association Burn Center Transfer Criteria

1. Second and third degree burns greater than 10% total body surface area (TBSA) in patients under 10 or over 50 years of age.
2. Second and third degree burns greater than 20% TBSA in other age groups.
3. Second and third degree burns that involve the face, hands, feet, genitalia, perineum, and major joints.
4. Third degree burns greater than 5% TBSA in any age group.
5. Electrical burns including lightning injury.
6. Chemical burns.
7. Inhalation injury.
8. Burn injury in patients with pre-existing medical disorders that could complicate management, prolong recovery, or affect mortality.
9. Any patients with burns and concomitant trauma (such as fractures, etc.) in which the burn injury poses the greatest risk of morbidity or mortality. In such cases, if the trauma poses the greater immediate risk, the patient may be treated initially in a Trauma Center until stable before being transferred to a Burn Center. Physician judgment will be necessary in such situation and should be in concert with the regional medical control plan and triage protocols.
10. Hospitals without qualified personnel or equipment for the care of children should transfer children with burns to a Burn Center with these capabilities.
11. Burn injury in patients who will require special social/emotional and/or long-term rehabilitative support, including cases involving suspected child abuse, substance abuse.

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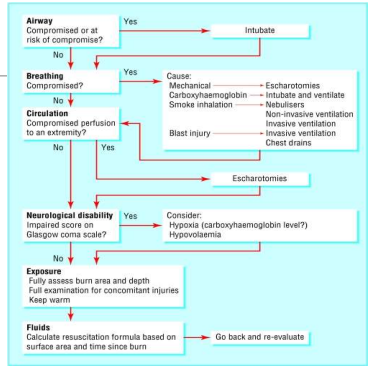
Burn Care Summary



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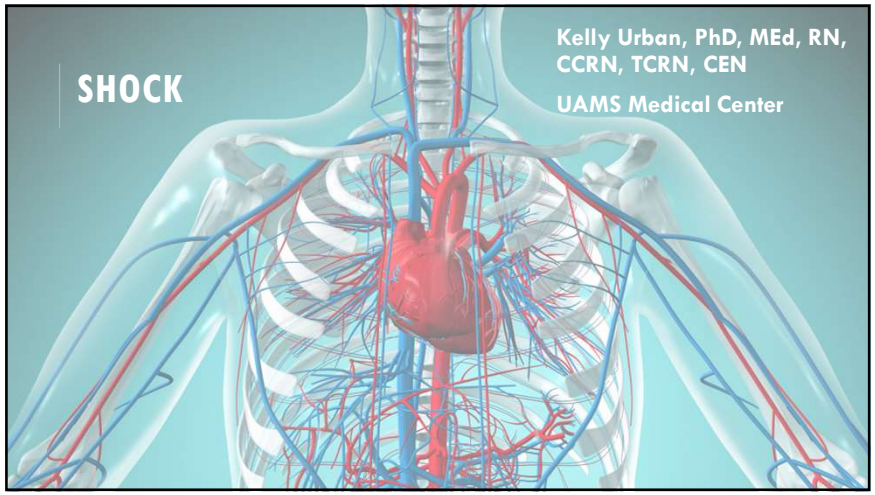


1

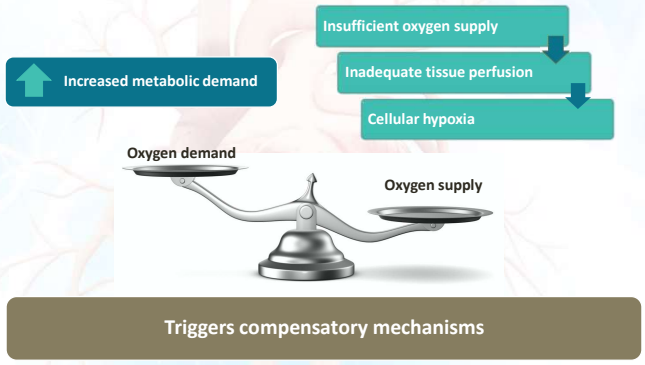
SHOCK - DEFINED

Inadequate tissue perfusion

Caused when oxygen intake, absorption, or delivery fails, or when the cells are unable to take up and use the delivered oxygen to generate sufficient energy to carry out cellular functions.

2

WHAT IS SHOCK?



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SHOCK - DEFINED

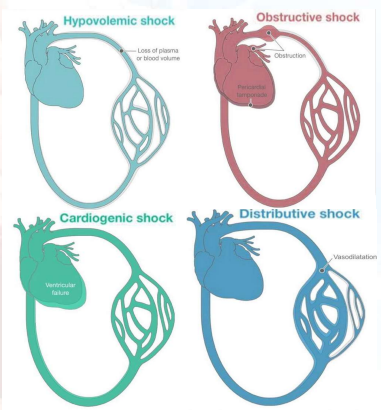
Inadequate tissue perfusion

Volume Problem
- ↓ intravascular volume

Pump Problem
- Failing heart

Pipe Problem
- ↓ vascular tone

Obstruction
- Mechanical barrier to blood flow



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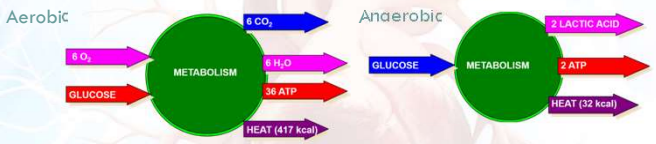
METABOLISM REVIEW



- No oxygen = lactic acid production
- Reduction in number of ATP = reduced energy

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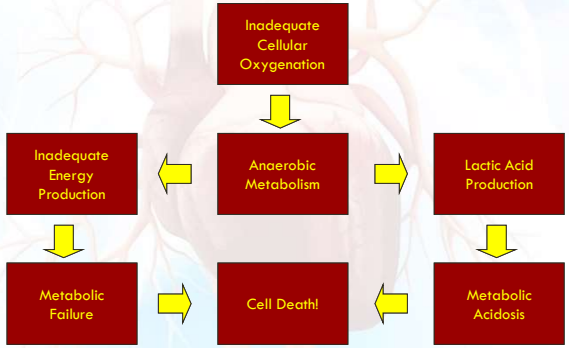
ANAEROBIC METABOLISM – SO WHAT?



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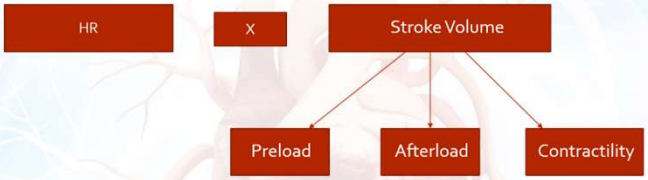
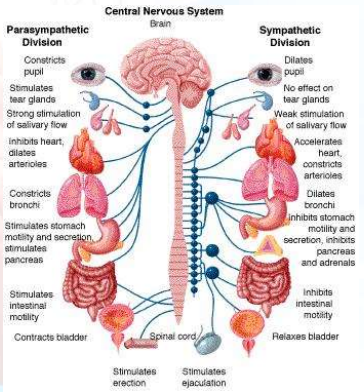
CARDIAC OUTPUT REVIEW



- Preload: amount of stretch at end of diastole (volume ready to be ejected)
- Afterload: resistance in which ventricle has to overcome to contract (vasoconstriction)
- Contractility: ability of the heart to contract

7

AUTONOMIC NERVOUS SYSTEM REVIEW



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EFFECTS OF SYMPATHETIC NERVOUS SYSTEM STIMULATION

Organ	Effect
Heart (muscle)	↑ force of contraction (+ inotropy)
Heart (rate)	↑ heart rate (+ chronotropy)
Peripheral vessels	Vasoconstriction
Pupils	Dilation
Sweat glands (cholinergic)	↑ secretion
Adrenal glands	↑ cortisol and medullary secretion
Bronchi	Dilation
Kidneys	↑ Renin secretion (↓ urine output)
Liver	Glycogenolysis (↑ blood sugar)

9

STAGES OF SHOCK

Compensated

Decompensated

Irreversible

10

COMPENSATED SHOCK

(COMPLEX SERIES OF NEURO-ENDOCRINE RESPONSES TO ↑ CO)

Decreased cardiac output compensatory mechanisms
- Tachycardia

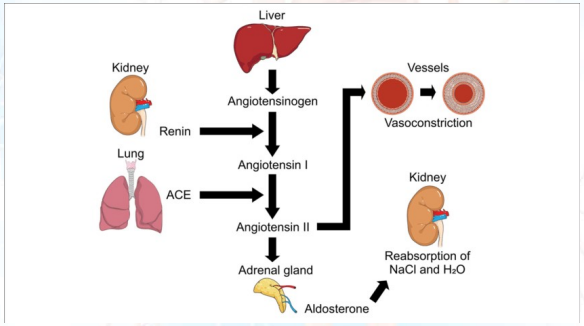
Activation of autonomic nervous system
- Tachycardia
- Vasoconstriction

Activation of renin-angiotensin system (due to reduced blood flow to kidneys)
- Vasoconstriction
- Na/Water retention

Increased rate and depth of respirations

11

ACTIVATION RENIN-ANGIOTENIN-ALDOSTERONE SYSTEM (RAAS)



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COMPENSATED SHOCK – CLINICAL FINDINGS

Normal BP, narrow pulse pressure
Sinus tachycardia
Fast, deep respirations
↓ Urine Output
↑ Urine Specific Gravity
Cool, clammy skin
↓ LOC
Dilated pupils
↑ blood sugar
Respiratory alkalosis with hypoxemia

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DECOMPENSATED (PROGRESSIVE) SHOCK

Decreased oxygen delivery to cells

- Shift to anaerobic metabolism
- Decreased ATP production
- Production of lactic acid = metabolic acidosis
- Failure of Na+/K+ pump
- Arrhythmias
- Alteration of capillary fluid dynamics
- Further decrease in cardiac output
- DIC

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DECOMPENSATED (PROGRESSIVE) SHOCK – CLINICAL FINDINGS

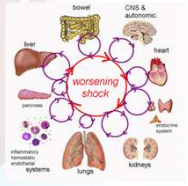
↓ BP with narrow pulse pressure
Continued tachycardia
Acute renal failure
Continued decreasing LOC
Interstitial pulmonary edema
Peripheral edema
Metabolic and respiratory acidosis with hypoxemia

15

IRREVERSIBLE SHOCK – MULTIORGAN DYSFUNCTION SYNDROME

Microvascular and organ damage are now irreversible

There is often a "last ditch" effort from the ischemic midbrain with an enormous discharge of endogenous catecholamines and this can create a last spike of sinus tachycardia



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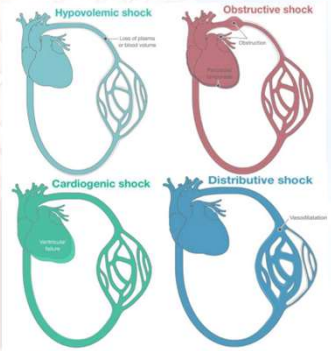
CLASSIFICATION OF SHOCK

- Hypovolemic
- Distributive
- Cardiogenic
- Obstructive



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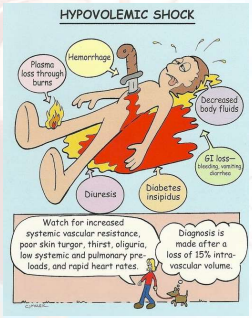
HYPOVOLEMIC SHOCK

Causes

Absolute Fluid Volume Deficit
- Hemorrhage or Blood Loss
  - Surgery
  - Trauma
  - GI Bleed
- Severe Dehydration
  - Sweat
  - Vomiting/Diarrhea
- Skin Loss via Burns

Relative Fluid Volume Deficit
- Severe Ascites
- Severe Burns (interstitial)
- Severe hypoalbuminemia



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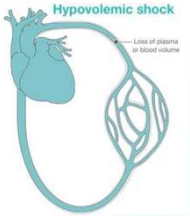
HYPOVOLEMIC SHOCK - PATHOPHYSIOLOGY



- Decreased Intravascular Volume
- Decreased Venous Return
- Decreased Ventricular Filling
- Decreased Stroke Volume/ Cardiac Output
- Inadequate Tissue Perfusion

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HYPOVOLEMIC SHOCK HEMODYNAMICS

Shock Type	CVP	PAWP	SVR	C.O.	HR	Comments
Hypovolemic	↓	↓	↑	↓	↑	Too little volume

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CLASSIFICATIONS OF HYPOVOLEMIC SHOCK (FROM BLOOD LOSS)

Parameter	Class I	Class II	Class III	Class IV
Blood Loss (ml)	< 750	750-1500	1500-2000	>2000
Blood Loss	< 15%	15-30%	30-40%	> 40%
Pulse (bpm)	< 100	100-120	120-140	> 140
Blood Pressure	Normal	Normal	Decreased	Decreased
Urinary Output (ml/hour)	> 30	20-30	5-20	Negligible
CNS	Slightly anxious	Mildly anxious	Anxious and confused	Confused and lethargic
Fluid Replacement	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

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HYPOVOLEMIC SHOCK POINTS TO CONSIDER

Increases in HR may be blunted in patients taking beta-blockers

Pulse pressure and mean arterial pressure (MAP) are better than looking at systolic or diastolic pressure alone

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HYPOVOLEMIC SHOCK TREATMENT

Airway management

Control Hemorrhage

IV access

Fluid Resuscitation

Prevent Hypothermia

23

CALCIUM AND THE DIAMOND OF DEATH

Hypothermia:
Decreased hepatic metabolism of citrate precipitates hypocalcemia. Hypocalcemia is associated with decreased cardiac output and shock.

Acidosis:
Hypocalcemia is associated with acidosis, which in turn worsens coagulopathy.

Diamond of Death

Coagulopathy:
Calcium is critical for proper platelet function and coagulation. Hypocalcemia is directly associated with impaired coagulation in trauma patients.

Hypocalcemia:
Hypocalcemia develops from trauma and hemorrhage, which is worsened by transfusion.

24

END POINTS TO FLUID RESUSCITATION

Traditional

Invasive Hemodynamic Monitoring

Metabolic Parameters

25

END POINTS TO FLUID RESUSCITATION - TRADITIONAL

Vital Signs
- Blood pressure is **not** a good predictor of tissue perfusion

Urinary Output
- < 0.5 ml/kg/hour is an early sign of inadequate perfusion

Mental Status
- May also be affected by pre-existing conditions, alcohol, or drugs

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END POINTS TO FLUID RESUSCITATION – INVASIVE HEMODYNAMIC MONITORING

CVP
- Measures right ventricular preload (norm 2-6 mmHg)

Wedge Pressure
- Measures left ventricular preload (norm 8-12 mmHg)

Cardiac Index
- Normal 2.5-4 L/min/m^2

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END POINTS TO FLUID RESUSCITATION – METABOLIC PARAMETERS

Lactate
- Byproduct of inadequate tissue perfusion
- Patients who lactate levels do not normalize have a higher mortality rate
- Lactate > 4 mmol/L indicates widespread tissue hypoperfusion

Base Deficit
- Measures buffering capacity of the blood reflecting metabolism and depth of hemorrhagic shock
- Base deficit > 6 mmol/L is a marker of severe injury

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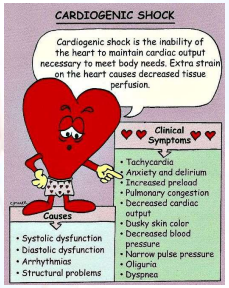
CARDIOGENIC SHOCK

Severe dysfunction of the right or left ventricle that results in inadequate pumping



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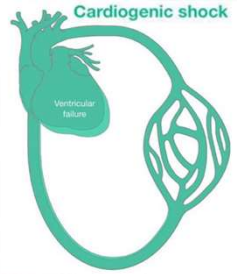
CARDIOGENIC SHOCK

Causes:

- Myocardial Infarction
- Structural Variations
  - Cardiomyopathy
  - Papillary Muscle Dysfunction
- Blunt Cardiac Injury (cardiac contusion)
- Ventricular Ischemia



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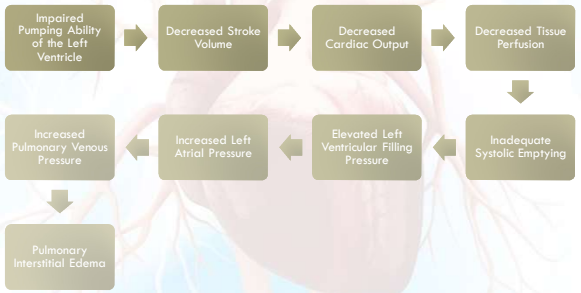
CARDIOGENIC SHOCK - PATHOPHYSIOLOGY



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CARDIOGENIC SHOCK HEMODYNAMICS

Shock Type	CVP	PAWP	SVR	C.O.	HR	Comments
Cardiogenic	↑	↑	↑	↓	↑	Ineffective pump

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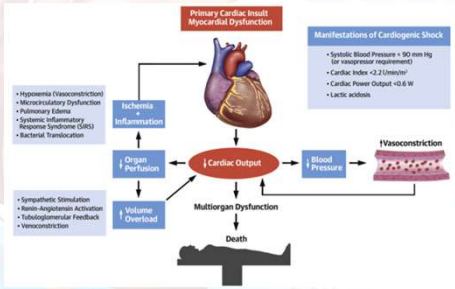
CARDIOGENIC SHOCK

Symptoms:

- ↓ BP (may be normal initially)
- ↑ HR
- ↑ RR/Dyspnea
- Bilateral Crackles
- ↓ Urine Output
- Normal Temp
- Cool, Pale Skin
- ↓ CO/CI
- ↑ PCWP/PA Pressures



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CARDIOGENIC SHOCK - TREATMENT

Hemodynamic Support

- ABCs (Supplemental Oxygen)

Decrease Preload	Increase Contractility	Decrease Afterload	Increase Afterload
• Diuretics • Nitrates • Morphine	• Dobutamine • Milrinone	• Nitroprusside • Nitrates • ACE Inhibitors	• Norepinephrine • Epinephrine

Identify & Treat Cause

- ECG/Cardiac Enzymes (MI/dysrhythmias)
- Chest Xray
- Echo

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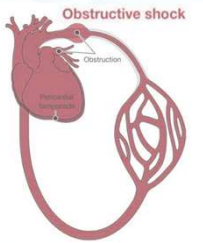
OBSTRUCTIVE SHOCK

Hypoperfusion of tissue due to an obstruction in either the vasculature or heart

Cause	Treatment
Pregnancy	Roll patient to her side
Tension Pneumothorax	Chest Tube/Needle Decompression
Cardiac Tamponade	Pericardiocentesis
Pulmonary Embolism	Thrombolytics
Aortic Aneurysm	Surgical Intervention
Aortic Stenosis	Surgical Intervention
Excessive Positive End Expiratory Pressure	Readjust Ventilator Settings



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OBSTRUCTIVE SHOCK - PE

Source

DVT

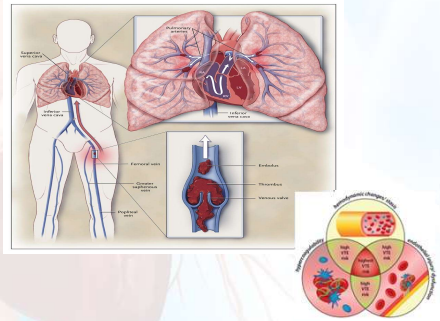
Air embolism

Fat embolism

Amniotic fluid embolism

Septic embolism

Tumor embolism



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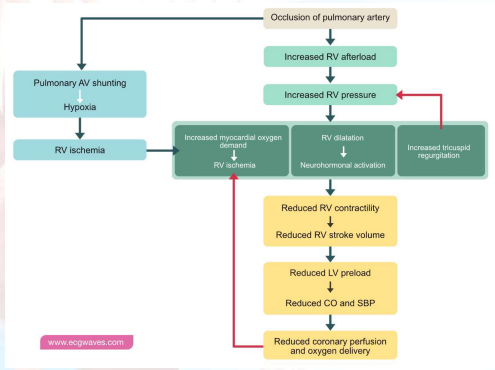
PE PATHOPHYSIOLOGY



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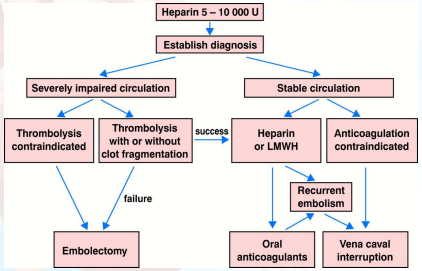
OBSTRUCTIVE SHOCK TREATMENT - PE

ABCs

Thrombolytics

Embolectomy



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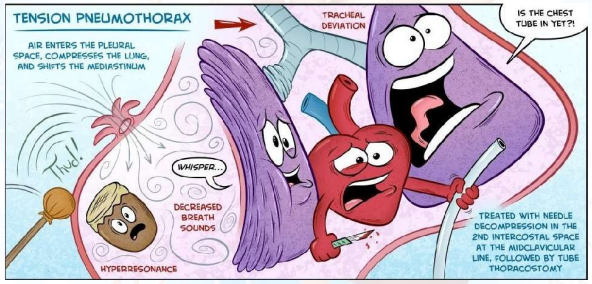
OBSTRUCTIVE SHOCK – TENSION PNEUMOTHORAX



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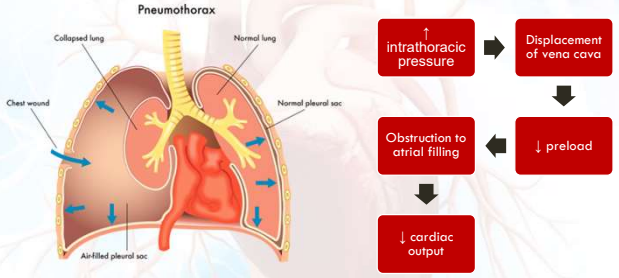
OBSTRUCTIVE SHOCK – TENSION PNEUMOTHORAX



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OBSTRUCTIVE SHOCK TREATMENT – TENSION PNEUMOTHORAX

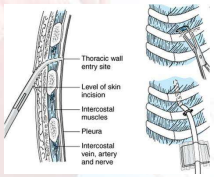
Needle Decompression

Insert 14 g x 3.25 in angiocath into chest wall

- 2nd intercostal space
- Midclavicular line
- Above 3rd rib (to avoid nerves, vein, artery that are located under ribs)

Chest Tube Placement

Thoracostomy tube placed 4th - 5th intercostal space, midaxillary line



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OBSTRUCTIVE SHOCK – CARDIAC TAMPONADE

Accumulation of fluid in the (inflexible) cardiac sac → Impediment of diastolic expansion and filling of ventricle → Low preload, SV, CO, and End-Organ Perfusion

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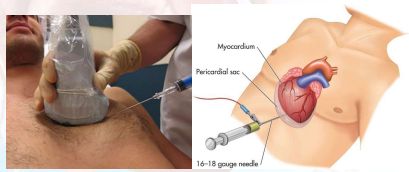
OBSTRUCTIVE SHOCK TREATMENT – CARDIAC TAMPONADE

Pericardiocentesis: procedure in which fluid is removed from the pericardium

Removal of 5-10 ml may ↑ stroke volume by 25-50%



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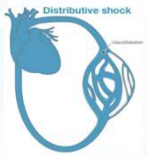
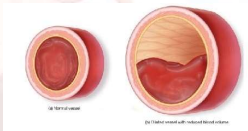
DISTRIBUTIVE SHOCK



Abnormality in the vascular system that produces a maldistribution of blood flow.

3 Types:

- Septic
- Neurogenic
- Anaphylactic



Occurs when blood vessels dilate without subsequent increase in volume

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DISTRIBUTIVE SHOCK HEMODYNAMICS

Shock Type	CVP	PAWP	SVR	C.O.	HR	Comments
Neurogenic	↓	↓	↓	↓	↓	Loss of sympathetic tone
Septic	↓	↓	↓	↑	↑	Endotoxins/exotoxins result in vasodilation
Anaphylactic	↓	↓	↓	↓	↑	Histamine release results in vasodilation

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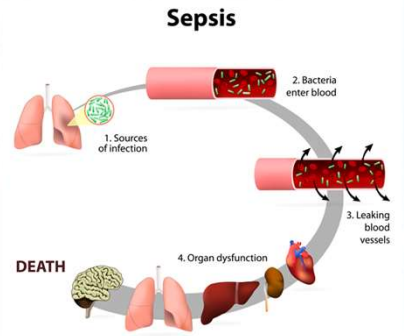
SEPSIS & SEPTIC SHOCK

The inflammatory and coagulation response is rapid and widespread, causing a dysregulated response

- The body's reaction to the pathogen may overwhelm all of the body's systems
- Immune systems that are too strong or too weak are unable to respond effectively to pathogen invasion



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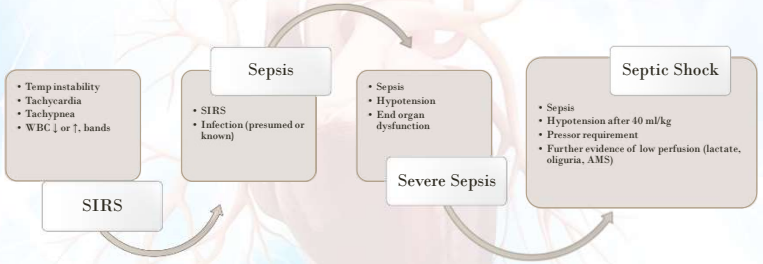
SEPTIC SHOCK (DISTRIBUTIVE)



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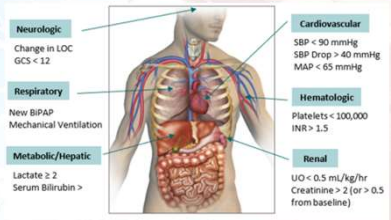
SEVERE SEPSIS

Sepsis + new organ dysfunction = severe sepsis

Organ dysfunction is defined as a condition in which an organ does not function as expected.



Acute Organ Dysfunction Related to Sepsis

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SEPTIC SHOCK

Severe sepsis + refractory hypotension OR lactate ≥ 4 mmol/L = septic shock

Septic shock is a distributive shock

Cytokine release leads to a large-scale inflammatory response

- Massive vasodilation
- Increased capillary permeability
- Decreased systemic vascular resistance
- Blood clots form in the microvasculature
- Hypotension reduces tissue perfusion causing tissue hypoxia

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SURVIVING SEPSIS 2021 ADULT SEPSIS GUIDELINES

SOFA

- 5 point scale to rate presence and severity of indicators of organ failure in 6 categories
  - Respiration
  - Coagulation
  - Liver function
  - Cardiovascular function
  - CNS function
  - Renal function

qSOFA: RR ≥ 22/min, GCS, BP ≤ 100 mmHg

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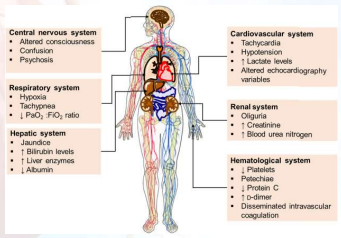
MULTI-ORGAN DYSFUNCTION SYNDROME (MODS)

MODS is altered organ function in an acutely ill patient requiring medical intervention to achieve homeostasis. Can be the end result of septic shock.

Sepsis-related organ dysfunction → No organ system is immune

- Respiratory failure
- Liver failure
- Kidney failure
- Heart failure
- Gut permeability
- DIC (disseminated intravascular coagulation)
- Altered mental status
- Brain death



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TREATMENT – GOAL DIRECTED THERAPY

Fluid Resuscitation

Intensive antimicrobial therapy

Vasopressors (MAP)

Transfusion for bleeding complications

Intensive patient monitoring

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HOUR-1 BUNDLE

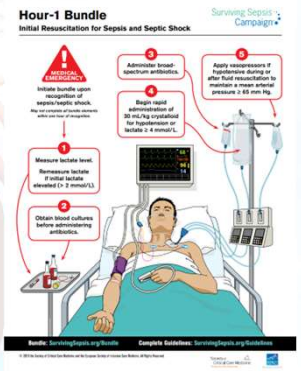
Measure lactate level

Obtain blood cultures before administering antibiotics

Administer broad-spectrum antibiotics

Begin rapid administration of 30 ml/kg crystalloid for hypotension or lactate ≥ 4 mmol/L

Apply vasopressors if hypotensive during or after fluid resuscitation to maintain a mean arterial pressure ≥ 65 mmHg



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LACTATE

With sepsis, lactate is viewed as a marker of global tissue perfusion.

Lactate has some predictive use:

- Sustained > 6 hours, an elevated lactate foreshadows increased mortality
- Mortality increases as lactate levels increase

Lactate Level	Mortality
0-2.5 mmol/L	4.9 percent mortality
2.5-4.0 mmol/L	9.0 percent mortality
> 4.0 mmol/L	28.4 percent mortality

(Nguyen, et al., 2004; Shapiro, et al., 2005)

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FLUID ADMINISTRATION RECOMMENDATIONS

Begin rapid administration of 30 ml/kg crystalloid for hypotension or lactate ≥ 4 mmol/L

Additional fluid should be given as small boluses

To avoid over/under resuscitation, fluid administration (beyond initial resuscitation) should be guided by careful assessment of intravascular volume and organ perfusion

- HR, CVP, SBP are poor indicators of fluid status
- Dynamic measures should be used (passive leg raising combined with CO measurement, fluid challenges against SV, systolic or pulse pressure, and increases of SV in response to changes in intrathoracic pressure

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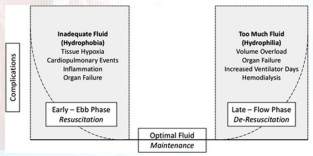
FLUID ADMINISTRATION RECOMMENDATIONS

Crystalloids are 1st-line fluid for resuscitation

- Balanced crystalloids instead of normal saline

Albumin can be used in patients who received large volumes of crystalloids

Do not use starches or gelatin for resuscitation



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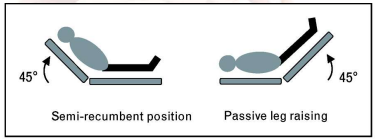
HEMODYNAMIC MONITORING

Dynamic assessments to guide fluid administration may improve outcomes for septic shock



The passive leg-raising test consists of measuring the hemodynamic effects of a leg elevation up to 45°. A simple way to perform the postural maneuver is to transfer the patient from the semi-recumbent posture to the passive leg-raising position by using the automatic motion of the bed.

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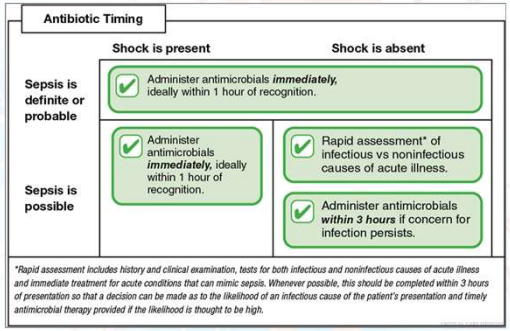
ANTIBIOTIC TIMING



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VASOACTIVE AGENT MANAGEMENT

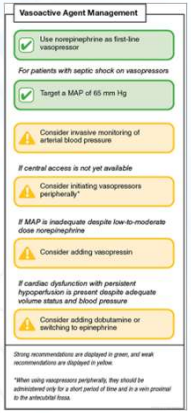
An initial target MAP of 65 mmHg

Norepinephrine is 1st-line agent

Vasopressin is 2nd-line agent

Epinephrine can be used if norepinephrine and vasopressin are inadequate

May also consider using Dobutamine if suspect cardiac dysfunction with persistent hypoperfusion



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ANAPHYLACTIC SHOCK (DISTRIBUTIVE)

Immune system overreaction that results in a host of vasoactive reactions

Causes:

- Repeated exposure to an antigen (antibiotics, other drugs, contrast media, food, insect stings, snake bites)

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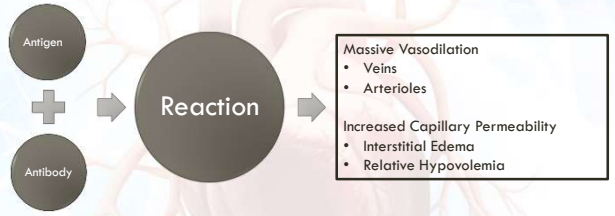
ANAPHYLACTIC SHOCK - PATHOPHYSIOLOGY



Massive Vasodilation
- Veins
- Arterioles

Increased Capillary Permeability
- Interstitial Edema
- Relative Hypovolemia

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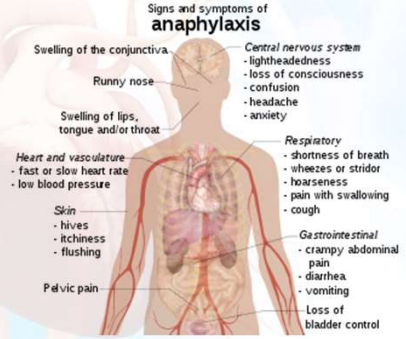
ANAPHYLACTIC SHOCK

Symptoms
- Urticaria/Pruritis
- "Sense of Impending Doom"
- Bronchoconstriction
- Increased Capillary Permeability
- Decreased CO
- ↑ HR/RR
- ↓ PAP/PCWP



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ANAPHYLACTIC SHOCK – TREATMENT GOALS

ID and remove causative agent

Fluids

Epinephrine

Antihistamines

Corticosteroids

Bronchodilators

Patient Education

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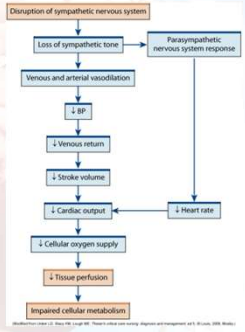
NEUROGENIC SHOCK (DISTRIBUTIVE)

Causes:
- Spinal Cord Injuries above T4-T6
- Brain Injury (if the central sympathetic system has been affected)



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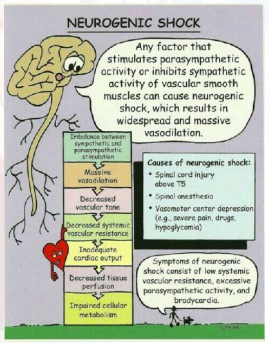
NEUROGENIC SHOCK (DISTRIBUTIVE)

Symptoms:

- Hypotension (due to vasodilation)
- Glyconeogensis
- Dry skin
- Bradycardia
- ↓ Contractility, Automaticity, Respiratory Rate



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NEUROGENIC SHOCK (DISTRIBUTIVE)

Treatment

- 1-2 L fluid bolus
- Vasopressors
- Goal: MAP 85-90 mmHg
- Atropine
- Assist with respirations as needed

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REVIEW OF TYPES OF SHOCK

Shock Type	CVP	PAWP	SVR	C.O.	HR	Comments	Therapy
Hypovolemic	↓	↓	↑	↓	↑	Too little volume	Replace what is lost (volume)
Cardiogenic	↑	↑	↑	↓	↑	Ineffective pump	Increase Contractility, reduce congestion
Neurogenic	↓	↓	↓	↓	↓	Loss of sympathetic tone	Provide Sympathetic tone
Septic	↓	↓	↓	↑	↑	Endotoxins/exotoxins result in vasodilation	Volume, Antibiotics, Vasopressors
Anaphylactic	↓	↓	↓	↓	↑	Histamine release results in vasodilation	Remove cause, Volume, Epi

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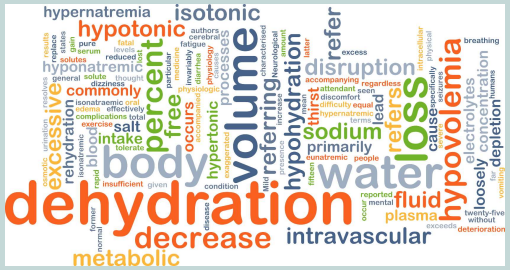

QUESTIONS?

Contact Kelly Urban @ klurban@uams.edu

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