WMRMC West Michigan Regional Medical Consortium

1675 Leahy St Suite 308B Muskegon, MI 49442

Lesson Plan: EPIC - Online

Topic: EPIC – Online

Presenter: West Michigan Regional Medical Consortium CE Sponsor Program

Location: West Michigan Regional Medical Consortium CE Sponsor Locations

Credit Category: Preparatory – 3-hours
Airway – 1.5 hours
Patient Assessment – 2-hours
Operations – 1-hour

License Level: MEDIC

Credits: 7.5

Format: 8-hour online cognitive learning module, evaluation and assessment

Objectives: At the conclusion of this CE session, the participants will be able to:

Cognitive

1. review cardiovascular medications used in the treatment in Interfacility Transports as presented in the online portion of the course and evaluated by the completion in the online assessments.
2. review Sedation and paralytic medications used in the treatment in Interfacility Transports as presented in the online portion of the course and evaluated by the completion in the online assessments.
3. review Central Nervous System medications used in the treatment in Interfacility Transports as presented in the online portion of the course and evaluated by the completion in the online assessments.
4. review TPN/Insulin/Electrolytes used in the treatment in Interfacility Transports as presented in the online portion of the course and evaluated by the completion in the online assessments.
5. review Obstetric medications used in the treatment in Interfacility Transports as presented in the online portion of the course and evaluated by the completion in the online assessments.
6. review Antimicrobial medications used in the treatment in Interfacility Transports as presented in the online portion of the course and evaluated by the completion in the online assessments.
7. review Pain Control medications used in the treatment in Interfacility Transports as presented in the online portion of the course and evaluated by the completion in the online assessments.
8. review Antidote medications used in the treatment in Interfacility Transports as presented in the online portion of the course and evaluated by the completion in the online assessments.
9. review Gastrointestinal medications used in the treatment in Interfacility Transports as presented in the online portion of the course and evaluated by the completion in the online assessments.
10. identify the different types of infusion pumps and indications for their use as presented in the online portion of the course and evaluated by completion in the online assessments.
11. identify the different risks and safety concerns with each infusion pumps, as presented in the online portion of the course, and evaluated by completion in the online assessments.

12. identify the need to initiate primary and secondary infusion pumps, as presented in the online portion of the course, and evaluated by completion in the online assessments.
13. understand basic hematology as it relates to blood products and blood product transfusion and understand the different types of blood product forms, as presented in the online portion of the course, and evaluated by completion of the online assessments.
14. to identify and understand the indications, equipment, and protocols for blood product administration as presented in the online portion of the course and evaluated by completion of the online assessments.
15. to identify and treat the various possible complications and reactions secondary to blood product transfusions as presented in the online portion of the course and evaluated by completion of the online assessments.
16. understand the basic anatomy of the thoracic cavity, and physiology as it pertains to chest tubes as presented in the online portion of the course and evaluated by completion of the online assessments.
17. understand the indications for the use of chest tubes as presented in the online portion of the course and evaluated by completion of the online assessments.
18. describe the monitoring and maintenance of chest tubes as presented in the online portion of the course and evaluated by completion of the online assessments.
19. identify potential complications with the chest tube and how to address these complications, as presented in the online portion of the course, and evaluated by completion of the online assessments.
20. understand basic ventilator modes and ventilators used in interfacility transfers as presented in the online portion of the course and evaluated by completion of the online assessments.
21. understand the components of BMP, normal and abnormal values and pertinent pathophysiology as presented in the online portion of the course and evaluated by completion of the online assessments.
22. understand coagulation as it pertains to PT/INR and pertinent pathophysiology as presented in the online portion of the course and evaluated by completion of the online assessments.
23. understand the importance of a cardiac injury profile as presented in the online portion of the course and evaluated by completion of the online assessments.
24. understand the components of acid-base in the body and blood gases interpretation as presented in the online portion of the course and evaluated by completion of the online assessments.
25. understand the importance of patient hand-off and patient care reports, as presented in the online portion of the course, and evaluated by completion of the online assessments.
26. understand the importance of communication between facilities of patient care as presented in the online portion of the course and evaluated by completion of the online assessments.

Psychomotor

1. apply principles of pharmacokinetics, in the overall care of a patient as presented in the online portion of the course and evaluated by the completion on on-line assessments.
2. apply principles of pharmacodynamics, in the overall care of a patient as presented in the online portion of the course and evaluated by the completion on on-line assessments.
3. apply principles on medication routes, in the overall care of a patient as presented in the online portion of the course and evaluated by the completion on on-line assessments.
4. apply principles on addiction, in the overall care of a patient as presented in the online portion of the course and evaluated by the completion in the on-line assessments.
5. set and change initial ventilator settings based on patient needs as presented in the online portion of the course and evaluated by completion of the online assessments.

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Affective

1. facilitate communication with intensive care practitioners and Respiratory Therapists as presented in the online portion of the course and evaluated by completion of the online assessments.
2. successfully complete an end-of-course exam for Enhanced Paramedic Interfacility Care, demonstrating competency in cognitive material.

Outline for Online Learning Module:

I. Pharmacology
II. Medications
III. IV Pumps
IV. Blood Products
V. Chest Tube
VI. LTV 1200 Ventilator
VII. Lab Values
VIII. Call Overview / Patient Care Reports

I. PHARMACOLOGY

A. Introduction

a. **Pharmacology**: *The science of drugs as it deals with the interactions of exogenously administered chemical molecule (drugs) with living system.*
b. **Father of Pharmacology:** Oswald Schmiedeberg
c. Two main divisions
   i. Pharmacokinetics: what the body does to the drug
   ii. Pharmacodynamics: what the drug does to the body

B. Pharmacokinetics

a. The quantitative study of drug movement in, through and out of the body.
b. Process:
   i. Transportation > Absorption > Distribution-Storage > Free drug > Metabolism > Receptor Binding (Effect) > Excretion
c. Biological membrane photo*
d. Transportation
   i. *Passive diffusion*
      1. Requires no energy and no carrier
      2. Rapid for lipophilic, nonionic and small molecules
      3. Slow for hydrophilic, ionic or large molecules
   ii. *Specialized transport (Aqueous channels)*
      1. No energy, No carrier
      2. Small hydrophilic drugs (< 200 mw) diffuse along concentration gradient by passing through aqueous channels (pores)
   iii. *Facilitated diffusion*
      1. No energy but needs a carrier
      2. Drugs bind to carrier by noncovalent mechanisms

3. Chemically similar drugs compete for carrier

iv. *Active transport*

1. Needs energy and a carrier
2. Identical to facilitated diffusion, except that ATP powers drug transport against concentration gradient.

e. Absorption

i. Movement of the drug from its site of administration into their circulation

ii. Factors that affect absorption

1. ***Drug-associated factors:*** ionization state, molecular weight, solubility, formulation (solution vs tablet)
    a. Small, nonionized, lipid-soluble drugs permeate plasma membranes most readily
2. ***Patient-associated factors:*** depend on route of administration.
    a. Presence of food in the GI tract, stomach acidity and blood flow to GI tract influence absorption.

f. Bioavailability

i. The fraction of the drug that reaches to systemic circulation from a given dose in an unchanged form

ii. A term typically used for medications given via oral route

iii. IV route gives 100% bioavailability as it directly enters the circulation

iv. *Bioequivalent*:

1. Two formulation of the same drug having equal bioavailability

v. *Bioinequivalent*:

1. If formation differ in their bioavailability

g. Distribution

i. Once a drug has gained access to the bloodstream, it gets distributed to other tissue that initially had no drug. Concentration gradient being in the direction of plasma to tissue.

ii. Factors affecting drug distribution:

1. *Membrane permeability*
    a. Ex. Benzodiazepines are very lipophilic, and readily cross the gut, capillary wall and blood-brain barrier
    b. Ex. Some antibiotics cannot cross the blood-brain barrier
    c. Blood-brain barrier, Blood-testes barrier, Blood-placenta barrier (prevents fetal exposure to some drugs).
2. *Depot storage of lipophilic drugs*
    a. Ex. sedative thiopental accumulates in fat, released slowly from fat stores. So, an obese person may stay sedated for a longer period of time than a skinny person
    b. Ex. Tetracycline, an antibiotic, accumulates in bones and teeth.
3. *Lipid solubility (various barriers)*
4. *Ionization at physiologic pH*
5. *Extent of binding to plasma and tissue protein*
    a. Ex. Albumin – reduces the amount of "free" drug in the blood
6. *Presence of tissue specific transporter*
7. *Difference in the regional blood flow*

8. *First pass metabolism*
    a. blood from GI tract passes through the liver before entering any other organs. A fraction of the drug (in some cases, nearly all) can be metabolized to an inactive or less active derivative.

iii. Movement of the drug proceeds until an equilibrium is established between unbound drug in plasma and tissue fluids.

h. Metabolism (Biotransformation)

i. It means chemical alteration of the drug in the body.
ii. Primary sites of drug metabolism is the liver
iii. Others are the kidney, intestine, lungs and plasma.
iv. *Biotransformation of drug may lead to the following:*
    1. Inactivation
    2. Active metabolite from an active drug
    3. Activation of inactive drug (prodrug)

i. Excretion

i. The removal of the drug and its metabolite from the body
ii. *Drugs and their metabolites are excreted in:*
    1. Urine
    2. Feces
    3. Exhaled air
    4. Saliva and sweat
    5. Milk

j. Half-Life ($t_{1/2}$)
    i. The length of time required for a given blood level to decline by 50%

k. Influence of dosing regimen on plasma drug levels

i. Single Dose (See figure)
    1. Plasma concentration of drug rises as the drug distributes to the bloodstream, then falls as the drug is distributed to tissues, metabolized and excreted
    2. *Distribution half-life ($t_{1/2}a$)*: reflects the rapid decline in plasma drug concentration as a dose of a drug is distributed throughout the body.
    3. Drugs administered orally reach a peak plasma concentration at a later time than drugs administered IV.

ii. Continuous Infusion (See figure)
    1. Steady state (equilibrium) plasma drug concentration is reach after continuous infusion for 4-5 half lives
    2. Increasing the rate of infusion will NOT decrease the time needed to reach steady state.
    3. Increasing the rate of infusion WILL, however, increase the plasma drug concentration at steady state.

iii. Intermittent Dose (See figure)
    1. A drug must be administered for 4-5 half-lives before steady state (equilibrium) is reached.
    2. Peaks: high points of fluctuation. Toxic effects are most likely to occur here
    3. Troughs: low points of fluctuation. Lack of drug effect most likely to occur here.

C. Pharmacodynamics

a. The study of drug effects
b. It attempts to elucidate the complete action-effect sequence and dose-effect relationship.
c. <u>Principles of drug action:</u>
    i. Drugs do not impart new function to any system, organ or cell they only alter the pace of ongoing activity.
    ii. <u>Types of drug action:</u>
        1. Stimulation
        2. Depression
        3. Irritation
        4. Replacement
        5. Cytotoxic action
d. <u>Mechanism of drug action</u>
    i. Majority of the drugs produce their effect by interacting with a discrete target biomolecule
    ii. <u>There are four major target of drug:</u>
        1. Enzymes
        2. Ion-channel
        3. Transporters
        4. Receptors
            a. Generally, proteins or glycoproteins that are on the cell surface, on an organelle within the cell or in the cytoplasm.

D. Drug

a. According to the WHO: Drug is any substance or product that is used or is intended to be used to modify or explore physiological system or pathological states for the benefits of the recipient.

E. Various physiochemical properties of drugs

a. Lipid and water solubility
b. Molecular size
c. Particle size
d. Degree of ionization
e. Physical forms
f. Chemical nature
g. Dosage form
h. Formulation
i. Concentration
j. Shape

F. Drug receptor interaction

a. Receptors are specific protein and have specificity and selectivity
b. No drug is truly specific, but many have a relatively selective action on one type of receptor
c. Drug + receptor --> Drug – receptor complex --> response
d. ***<u>Definitions</u>***:
    i. ***Affinity***: The strength of binding between a drug and its receptor
    ii. Intrinsic activity: The ability of the drug to produce a pharmacological action after combining with the receptor.
    iii. ***Agonist***: A drug that is capable of producing pharmacological action after binding to the receptor.

1. ***Strong Agonist***: An agonist which causes maximal effects, even though it may only occupy a small fraction of receptors on a cell.
2. ***Weak Agonist:*** An agonist which must be bound to many more receptors than a strong agonist to produce the same effect.
3. ***Partial Agonist:*** A drug which fails to produce maximal effects, even when all the receptors are occupied.

iv. ***Antagonist***: Drugs that bind to receptor, but are not capable of producing pharmacological action. These produce receptor blockade.

1. ***Competitive Antagonist:*** Compete with agonists for receptors.
    a. Surmountable as large amounts of agonists can overcome this
2. ***Noncompetitive Antagonist:*** Bind to a site other than the agonist-binding domain. Induces a conformational change in the receptor such that the agonist no longer "recognizes" the agonist-binding domain.
    a. Insurmountable in that larger amounts of agonists cannot overcome this antagonism
3. ***Irreversible Antagonist***: Compete with agonist for the agonist-binding domain, but the binding is permanent. Cannot be "reclaimed" by an agonist.
4. ***Physiological Antagonism***: Two agonists, in unrelated reactions, cause opposite effects and cancel each other out.
5. ***Antagonism by neutralization:*** Two drugs bind to one another, and become inactive.

G. Drug Interactions

a. Addition

i. The response elicited by combined drugs is EQUAL TO the combined responses of the individual drugs (Mathematical Model: 1+1=2)

b. Synergism

i. The response elicited by combined drugs is GREATER THAN the combined responses of the individual drugs (Mathematical Model: 1+1=3)

c. Potentiation

i. A drug which has no effect enhances the effect of a second drug (Mathematical Model: 0+1=2)

d. Antagonism

i. Drug inhibits the effect of another drug. Usually, the antagonist has no inherent activity (1+1=0)

H. Routes of drug administration

a. **Parenteral** = "around the GI tract"

b. Intravenous

i. Rapid onset

ii. Insoluble drugs cannot be administered IV

c. Intramuscular

i. Rate of absorption depends on formulation

1. Oil based = absorb slowly
2. Aqueous preparations = absorb rapidly

d. Subcutaneous

i. Absorption can be controlled by drug formulation

e. Topical

i. local effect (good for meds that have systemic toxic effects)

f. Inhalation
   i. rapid and targeted
g. Per Os (PO)
   i. Most compatible with drugs that are self-administered
   ii. Must be able to withstand the acidic environment for the stomach and must permeate the gut lining before entering the blood-stream.
   iii. Absorption affected by gastric emptying and intestinal motility
   iv. portal circulation --> liver --> first pass metabolism
h. Rectal
   i. 50% first pass
   ii. Absorption is unreliable
   iii. Useful for unconscious, vomiting or small children
i. Sublingual
   i. Rapid absorption
   ii. no first pass
j. Intrathecal
   i. Into the CSF
k. Transdermal
   i. sustained effect

I. Tolerance, Dependence and Withdrawal

a. Tolerance:
   i. Represents a decreased response to a drug.
   ii. Clinically seen when the dose of a drug must be increased to achieve the same effect.
   iii. Metabolic – drug metabolized more rapidly after chronic use
   iv. Cellular – Decreased in number of drug receptors (downregulation)
   v. Behavioral – an alcoholic learns to hide the signs of drinking to avoid being caught by his colleagues
b. Dependence
   i. Occurs when a patient needs a drug to "function normally"
   ii. Clinically, it is detected when cessation of a drug produces withdrawal symptoms.
c. Withdrawal
   i. Occurs when a drug is no longer administered to a dependent patient.
   ii. Symptoms are often opposite the effect achieved by the drug.
   iii. Ex: severe hypertension in a patient after cessation of antihypertensive meds
d. Cross tolerance/Cross Dependence
   i. Occurs when tolerance or dependence develop to different drugs which are chemically or mechanistically related.
   ii. Ex: Methadone relieves the symptoms of heroin withdrawal because patients develop cross dependence to these two drugs.

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II. MEDICATIONS

a. Cardiovascular Drugs

i. **Anti-arrhythmics**

1. **Use:** Treatment and cardioversion of Atrial Fibrillation or Atrial Flutter Treatment and prophylaxis of refractory Ventricular Tachycardia
2. **Adverse Effects:** Hypotension, QT prolongation, Torsades, Ventricular Tachycardia AV Block
3. **Dosing:**
    a. **Magnesium Sulfate**
        i. Bolus: 1-2 grams over 5 minutes Infusion: 6-12 grams over 24 hours
4. **Special Considerations:**
    a. Use infusion pump for drips.
    b. May not be compatible with heparin, lidocaine, amiodarone or bicarb
    c. Frequent BP checks for hypotension
    d. Increased risk for ventricular dysrhythmias if on certain antihistamines or anti-nausea medications

ii. Beta Blocker

1. **Uses:** Slow ventricular response in SVT, Atrial Fibrillation and Atrial Flutter Slow sinus node rate
2. **Adverse Effects:** Hypotension, bradycardia, Hypoglycemia (diabetics on medications); usual signs and symptoms are masked, Bronchospasm, Sinus node arrest
3. **Dosing:**
    a. **Atenolol (Tenormin)**
        i. Infuse: 5 mg over 5 minutes
        May be repeated in 10 minutes
    b. **Esmolol (Brevibloc)**
        i. Bolus: 500 mcg/kg (0.5 mg/kg) over one minute Infusion: 50 mcg/kg/minute for 4 minutes
        ii. If inadequate response, repeat bolus and increase drip rate by 50 mcg/kg/minute up to 3 times **(total dose of 2000 mcg bolus and infusion @ 200 mcg/kg/minute)**
        iii. Drug comes in a 100 mg (10 mg/ml) vial or 2500 mg ampule into 250 ml (10 mg/ml) or 500 ml (5 mg/ml) NS or D5W
    c. **Labetalol (Normodyne)**
        i. Infusion: 2 mg/minute (concentration 1mg/ml; 2ml/min) duration from 25 minutes to 2.5 hours
    d. **Metoprolol (Lopressor)**
        i. Inject: 2.5 mg IV slow push over 2 minutes May repeat dose up to 5 every 5 minutes for a **total dose of 15 mg**
    e. **Sotolol (Betapace)**
        i. Bolus: 1-1.5 mg/kg; followed by
        ii. Infusion: 0.008 mg/kg/minute = 8 mcg/kg/min
4. **Special Considerations:**
    a. Use infusion pump

b. Check BP frequently; monitor heart rate
c. Carefully monitor for hypotension, excessive bradycardia or new AV blocks
d. Patient with DIABETES may have symptoms of hypoglycemia masked; watch carefully for mental status changes
e. Contact MC if develop adverse reaction

iii. Anti-Hypertensives

1. **Uses:** Short term parenteral treatment when oral treatment is not feasible Nitroprusside may be used in CHF to reduce both preload and afterload (reduces work of the heart)
2. **Adverse Effects:** Hypotension, bradycardia, dysrhythmias, Palpitations, flushing, angina, Headache, restlessness, drowsiness, confusion or slurred speech
3. **Dosing:**
    a. **Hydralazine**
        i. Inject: 5- 40 mg IV push over 1-2 minutes
        Usually given as repeat bolus doses every 20-30 min
        ii. Rarely given as drip: 1-10 mg/hour
    b. **Nicardipene (Cardene)**
        i. Dilute to: 0.1 mg/ml
        Infusion: Start @ 50 ml/hr (5 mg/hr)
        ii. May increase rate by 2.5 mg/hr every 15 minutes until desired BP is reached for a **maximum dose of 15 mg/hr**
    c. **Nitroprusside**
        i. Infusion: Continuous to maintain BP
        ii. **See dosage chart below; amount listed is in *ml/hr***
        iii. **ADD NITROPRUSSIDE DOSING CHART**
    d. **Nesiritide (Natrecor)**
        i. Inject: 2 mcg/kg IV push over 60 seconds Infusion: 0.01 mcg/kg/min maintenance infusion
4. **Special Considerations:**
    a. Use infusion pump
    b. Dedicated IV line-should not administer in same IV line as other meds
5. **Nicardipene**:
    a. If hypotensive (BP<60) or tachycardic (HR>140), discontinue drip.
    b. May resume when stable @ 3-5 mg/hr
    c. Infusion site must be changed after 12 hours
    d. Use with caution in patients with liver failure, since it is metabolized in the liver.
    e. May be contraindicated in severe Aortic Stenosis as may decrease preload.
6. **Nitroprusside**:
    a. Small boluses or slight increases in infusion rate may produce profound hypotension
    b. Solution must be wrapped in foil to protect it from light
    c. Do not mix other medications in the same line
    d. Check BP and heart rate every 5 minutes
    e. Hypotension can be alleviated by decreasing the infusion rate
7. **Nesiritide**:
    a. Caution in pregnant or lactating patients
    b. Contact MC for worsening signs/symptoms, significant BP change or if BP<90

iv. Calcium Channel Blockers
    1. **Uses:** Ventricular Rate Control in A Fib, Atrial Flutter, MAT or SVT
    2. **Adverse Effects:** May cause Atrial Flutter, AV Block, Bradycardia, Chest Pain, CHF, Ventricular Arrhythmias, nausea/vomiting, dyspnea or hypotension
    3. **Dosing:**
        a. **Diltiazem (Cardizem)**
            i. Bolus: 0.25 mg/kg over 2 minutes (20 mg for average patient) If needed may repeat bolus in 15 minutes @ 0.35 mg/kg (25 mg in the average patient) over 2 – 5 minutes
            ii. Infusion: Dilute 125 mg (25ml) in 100 ml NS/D5W Drip @ 5 – 15 mg/hour titrated to heart rate
        b. **Nicardipene (Cardene)**
            i. Dilute to: 0.1 mg/ml
            ii. Infusion: Start @ 50 ml/hr (5 mg/hr) May increase rate by 2.5 mg/hr every 15 minutes until desired BP is reached for a **maximum dose of 15 mg/hr**
    4. **Special Considerations:**
        a. Carefully monitor for hypotension/excessive bradycardia/ new A/V block
        b. PVC's can occur with conversion to NSR
        c. Don't use in the presence of a WIDE COMPLEX TACHYCARDIA

v. Glycoprotein IIb/IIIa Inhibitors
    1. **Use:** Unstable Angina Non Q-wave MI Percutaneous Coronary Intervention
    2. **Adverse Effects:** Bleeding (usually at cath sites) possible allergic reactions to ReoPro
    3. **Dosing:**
        a. **Abciximab (ReoPro)**
            i. Loading bolus: 0.25 mg/kg over 10-60 minutes
            ii. Maintenance infusion: 0.125 mcg/kg/min for 12 hours following PCI or 18-24 hours for unstable angina
            iii. Should be administered through a 0.2 or 0.22 micron filter
            iv. Drip rates will vary depending on concentration that was mixed.
            v. Verify drip rates/dosage calculations with the transferring facility staff prior to transport.
        b. **Tirofiban (Aggrastat)**
            i. Loading infusion: 0.4 mcg/kg/min for 30 minutes
            ii. Maintenance infusion: 0.1 mcg/kg/min
            iii. Rate will be halved for patients with renal insufficiency.
        c. Eftifibatide (Integrilin)
            i. Loading bolus: 180 mcg/kg over 1-2 minutes
            ii. Maintenance infusion: 2 mcg/kg/min up to 72 hours
    4. Special Considerations:
        a. Use infusion pump
        b. Should always be given WITH heparin
        c. If bleeding occurs, need to turn off heparin as well as the GPIIb/IIIa drug.
        d. All settings on this medication are to be determined by the ordering physician.

i. Heparin gtt

a. **Uses:** Prevents blood clotting, especially in the following situations: Acute MI, Pulmonary Embolus, Deep Vein Thrombosis
b. **Adverse Effects:** Hemorrhage from various sites including needle sticks, GI tract, CNS bleeds
c. **Dosing:**
    i. Bolus: 15-18mg/kg
    ii. Infusion: 800-1600 mg/hour
    iii. Infusion rates may be outside this range and should not require adjustment during transport
d. **Special Considerations:**
    i. Use infusion pump
    ii. D/C immediately for onset of major bleeding or acute mental status change
    iii. Contact MC for any bleeding such as IV sites or gums

ii. Inotropes

a. **Uses:** Short term intravenous treatment of patients with acute decompensated heart failure Severe CHF/Cardiogenic Shock To increase cardiac output by increasing myocardial contractility and stroke volume Hemodynamically significant hypotension not resulting from hypovolemia
b. **Adverse Effects:** May develop hypokalemia resulting from increased cardiac output and/or diuresis. May have tachycardia, ventricular dysrhythmias or ectopy, hypertension, angina or ischemic chest pain. Dobutamine may also cause hypotension. Dopamine may cause nervousness, headache, palpitations, dyspnea, nausea or vomiting
c. **Dosing:**
    i. **Inamrinone (Inocor):**
        1. Loading dose over 2-3 minutes: 0.75 mcg/kg
        2. Maintenance infusion: 5-10 mcg/kg/min
    ii. Milrinone (Primacor)
        1. Loading dose over 10 minutes: 50 mcg/kg
        2. Maintenance infusion: See dosing chart
    iii. Dobutamine
        1. Infusion: 2.5-20 mcg/kg/min continuous
        2. Onset may be 10 minutes
    iv. Dopamine
        1. Infusion: 1-20 mcg/kg/min continuous
        2. Onset may be 10 minutes
    v. Epinephrine
        1. Infusion: 1-10 mcg/min titrated to effect
    vi. Norepinephrine (Levophed)
        1. Infusion: 0.5-1.0 mcg/min; titrated up to 30 mcg/min
d. Special Considerations
    i. Use infusion pump
    ii. For Inamrinone/Milrinone: do not mix with Lasix or dextrose-containing solutions
    iii. Monitor for cardiac dysrhythmias; these may be caused by hypokalemia, pre-existing arrhythmias, abnormal drug levels, catheter placement, etc.
    iv. Check blood pressure and heart rate frequently.

v. Discontinue briefly if develop hypotension secondary to vasodilatation.
vi. Contact MC for any adverse affects

iii. Thrombolytic Therapy
a. **Uses:** Dissolves clots in blood vessels Generally used in the setting of Acute MI or CVA; occasionally used Pulmonary Embolus
b. **Adverse Effects:** Minor hemorrhages from IV sites and gums Major hemorrhage from GI and intracranial or spinal sites Reperfusion dysrhythmias often occur about 30-60 minutes after staring infusion.

5. **Dosing**:
   a. tPA
      i. Dose to be determined by transferring physician
      ii. Rate should not require adjusting en route
6. **Special Considerations:**
   a. Use infusion pump
   b. Monitor heart rhythm
   c. Check BP and HR frequently
   d. Do not mix with other medications in the same line
   e. D/C infusion immediately if there is cardiac arrest, major hemorrhage, anaphylaxis or change in mental status **AND call MC**.

b. Sedation and Paralytic Agents

i. Benzodiazepine gtt

1. **Uses:** Sedation for patients who are intubated (and often concurrently on a paralytic drip) May be used to treat Status Epilepticus
2. **Adverse Effects:** May be more prone to hypotension if used with an opioid drug Can cause paradoxical agitation, hypertension or tachycardia
3. **Dosing:**
   a. **Lorazepam (Ativan):**
      i. Loading dose: 0.5-4.0 mg IV bolus; may be repeated in 10 min
      ii. Infusion: 0.02-0.1 mg/kg/hour
   b. **Midazolam (Versed):**
      i. Loading dose: 0.01-0.1 mg/kg IV bolus
      ii. Infusion 0.02-0.1 mg/kg/hour
4. **Special Considerations:**
   a. Only to be used in intubated patients

ii. Moderate Sedation Agents

1. **Uses:** Sedation for patients who are intubated (and often concurrently on a paralytic drip) May also be used for refractory seizures or therapeutic coma
2. **Adverse Effects:** May be more prone to hypotension if used with an opioid drug. Can cause paradoxical agitation, hypertension or tachycardia
3. **Dosing:**
   a. **Propofol:**
      i. Loading dose: 0.5-5 mg/kg
      ii. Maintenance infusion: 2-10 mg/kg/hour
   b. **Barbiturates:** *Pentobarbital* is most commonly used
      i. Loading dose: 10 mg/kg; infuse up to 25 mg/min

ii. Maintenance: 1-2 mg/kg/hour

c. **Ketamine:**

i. Loading dose: 1-5 mg/kg

ii. Maintenance infusion: 0.01-0.05 mg/kg/hour

4. **Special Considerations:**

a. Only to be used in intubated patients

iii. Opioid gtt

1. **Uses:** *Typically **part of** a sedation combination* for patients who are intubated. Occasionally for pain control
2. **Adverse Effects:** May cause hypotension, especially in volume depleted patients or those with right-sided heart failure
3. **Dosing:**

a. Morphine

i. Bolus: 0.5-10 mg

ii. Infusion: 2-30 mg/hr

b. Fentanyl

i. Bolus: 1-3 mcg/kg

ii. Infusion: 25-250 mcg/hr

4. **Special Considerations:**

a. Not advisable to give patients on narcotic drips Naloxone, as this may precipitate acute withdrawal

b. Antihistamines (both H1 and H2) may counteract hypotension.

iv. Paralytic Agents

1. **Uses:** Total muscular paralysis when patient movement may:

a. Compromise airway control (e.g. causing unwanted extubation)

b. Exacerbate a real or potential illness or injury (e.g. spinal cord injury from a spine fracture)

c. Endanger the patient, EMS care provider or others

2. **Adverse Effects:** Bronchospasm, flushing, hypotension and tachycardia have been rarely reported
3. **Dosing:**

a. **Pancuronium:**

i. Loading dose: 10 mg/kg

ii. May repeat dose every 1-2 hours as needed

b. **Vecuronium:**

i. Initial dose: 10 mg IV push

ii. Repeat dose of 10 mg IV push every 20-40 minutes as needed

iii. Maintenance infusion may be an alternative: 0.01mg/kg/min

c. **Rocuronium:**

i. Loading dose 0.6 mg/kg

ii. May rebolus 0.2 mg/kg every 30-45 minutes

iii. Maintenance infusion may be an alternative: 0.01–0.15 g/kg/min

4. **Special Considerations:**

a. Produces COMPLETE APNEA; therefore an intact airway (e.g. endotracheal intubation), and adequate ventilation/oxygenation **MUST BE ESTABLISHED PRIOR TO**

ADMINISTRATION. Likewise, personnel and equipment with the ability to restore an airway, ventilation and oxygenation must be available during transport.

b. Causes paralysis only; **therefore concomitant use of a sedative/hypnotic is indicated**
c. Note: Paralysis may alter the clinical exam. For example, motor seizure activity will not be seen, but the brain will continue to undergo seizure activity, and this must be treated! Also, conditions such as shock, hypoxia, pain, intracranial injury, hypoglycemia, etc. maybe the cause of this unwanted, spontaneous patient movement in the first place. **These conditions must be addressed but may be masked by the paralytic agent!**

c. CNS Drugs
   i. Anticonvulsants
      1. **Uses:** Prevention and treatment of seizures
      2. **Adverse Effects:**
         a. If intravenous phenytoin is given too rapidly, may result in:
            i. Cardiac dysrhythmias including ventricular fibrillation or asystole
            ii. Hypotension
         b. Subcutaneous extravasations of intravenous phenytoin may cause tissue necrosis or pain at the IV site
      3. **Dosing:**
         a. **Phenytoin:**
            i. 100-1200mg IV piggyback in normal saline;
            ii. Rate not to exceed 50 mg/min
         b. **Fosphenytoin:** *dose expressed in* ***phenytoin equivalents (PE)***
            i. 15-20 PE/kg; rate up to 100-150 PE/min
         c. **Valproic Acid:**
            i. 40-60 mg/kg Rate up to 3 mg/kg/min
         d. Phenobarbital:
            i. 5-15 mg/kg IV, then 0.5-10 mg/kg/hour
      4. **Special Considerations:**
         a. Use infusion pump
         b. Monitor heart rhythm
         c. Check BP frequently; vital sign monitor recommended
         d. D/C infusion and contact MC for any adverse effects
   ii. Mannitol
      1. **Uses:** Treatment of increased intracranial pressure or selected fluid overload states
      2. **Adverse Effects:** Hypernatremia, Volume Depletion
      3. **Dosing:**
         a. 25-50 grams IV push or bolus infusion (in 50cc D5W over 20 minutes)
      4. **Special Considerations:**
         a. Patients receiving mannitol should have a Foley to monitor fluid status
   iii. Steroids
      1. **Uses:** Spinal cord injury to decrease edema. Cerebral edema due to injury or CNS mass or lesion
      2. **Adverse Effects:** GI Bleed, Electrolyte disturbance and hyperglycemia, Hypertension, Acute CHF, Agitation, Corticosteroid hormonal suppression (hypoglycemia, hypotension,

hypothermia), Higher risk for infection or masking symptoms of infection

3. **Dosing:**
    a. **Methylprednisolone (Solumedrol):**
        i. Initial bolus: 30 mg/kg over 15 minutes
        ii. Start infusion 45 minutes later: 5.4 mg/kg/hour for 23 hours
    b. **Dexamethasone (Decadron):**
        i. Cerebral Edema: start with 10 mg IV x 1, then 4 mg IM/IV q 6 hrs
4. **Special Considerations:**
    a. Contact MC for question of adverse effects

d. HAL/TPN, Insulin and Electolytes

i. Hyperalimentation/TPN

1. **Uses:** Intravenous nutrition
2. **Adverse Effects:** Catheter related sepsis, Air embolism if central venous IV tubing becomes disconnected, Subcutaneous extravasations of solution can cause tissue necrosis Discontinuation of infusion may cause hypoglycemia
3. **Dosage:** Continuous infusion usually through central venous catheter but occasionally through a peripheral IV line. Rate should not require adjustment enroute.
4. **Special Considerations:**
    a. Use infusion pump.
    b. Do not administer any other medication through the same IV line.
    c. Contact MC for any adverse effects listed above
    d. Consider use of a cardiac monitor

ii. Insulin gtt

1. **Uses:** Lowers blood glucose. Used in diabetics especially with ketoacidosis or hyperosmolar nonketonic coma.
2. **Adverse Effects:** Hypoglycemia related (tachycardia, diaphoresis, mental status changes, and seizures)
3. **Dosage:** 5-15 units per hour but dosages outside this range may be used.
4. **Special Considerations:**
    a. Use infusion pump
    b. Do not administer medications in the same IV line except D50.
    c. If symptoms of hypoglycemia develop:
        i. turn off infusion, perform a D-Stick, administer 25 grams, (one AMP) D50) if glucose <80 and contact MC.
        ii. Monitor blood sugar every 30 minutes during transport
        iii. Cardiac monitoring required

iii. Potassium Chloride

1. **Uses:** Replacement therapy for hypokalemia
2. **Adverse Effects:** Cardiac dysrhythmias (prolonged PR interval; wide QRS complex; depressed ST segment; tall, peaked T-waves; heart block; cardiac arrest). Subcutaneous extravasations of solution can cause tissue necrosis
3. **Dosage:**
    a. Usual range is up to 20 mEq / hr., continuous infusion.
    b. May be mixed with various IV solutions in various sized bags including “piggy back” solutions.

c. Rate should not require adjustment en route.

4. **Special Considerations:**
   a. Monitor heart rhythm
   b. Often causes burning during infusion; contact MC if this is problematic
   c. Contact MC for changes in EKG configuration and/or dysrhythmias.

e. Obstetric Drugs
   i. Magnesium Sulfate
      1. **Uses:** Treatment of pre-eclampsia and eclamptic seizures. Premature rupture of membranes
      2. **Adverse Effects:** Lethargy, nausea, vomiting, hypotonia, respiratory depression, dysrhythmia
      3. **Dosing:**
         a. Loading dose: 2 – 6 grams IV over 15 minutes (may give 2 gms over 5 minutes)
         b. Followed by maintenance infusion: 1 – 2 gms/hr
      4. **Special Considerations:**
         a. Monitor reflexes
         b. For symptomatic toxicity: 10 mLs of 10% Calcium Chloride and contact MC
         c. MC may also request furosemide and/or NS bolus as MCO
         d. In renal failure, patient may require emergency dialysis
   ii. Oxytocin (Pitocin)
      a. **Uses:** Stimulates post-partum contraction of the uterus to control bleeding
      b. **Adverse Effects:** Hypertension, tachycardia, dysrhythmias
      c. **Dosing:** 10-40 units added to 1000 mL IV Fluid to control hemorrhage. Usual rate is 10-20 milliunits/min
      d. **Special Considerations:**
         i. Use infusion pump
         ii. Monitor heart rhythm
         iii. Check BP frequently; vital sign monitor recommended
         iv. Contact MC for any adverse effects

f. Antimicrobial Therapy
   i. Antibiotics and Antiviral
      1. **Uses:** Bacterial or Viral infections (treatment and prophylaxis)
      2. **Adverse Effects:** Allergic signs and symptoms, including anaphylaxis
      3. **Dosage:** Vary depending on the antibiotic. Generally given as a “piggyback” solution. Rate should not require adjustment en route
      4. **Special Consideration:** D/C infusions if there are any allergic signs or symptoms, then contact MC.
      5. ***Most Commonly used:***
         a. ***Acylcovir***
         b. ***Azithromycin (Zithromax),***
         c. ***Cefazolin (Ancef)***
         d. ***Ceftriaxone (Rocephin)***
         e. ***Gentamicin***
         f. ***Levofloxacin (Levaquin)***
         g. ***Metronidazole (Flagyl)***
         h. ***Piperacillin/Tazobactam (Zosyn)***
         i. ***Vancomycin***

ii. Antifungal
    1. **Uses:** Fungal infections. Often in immune-compromised patients, those on chemotherapy or chronic antibiotics
    2. **Adverse Effects:** Nausea or diarrhea. Amphotericin-fever, rigors, chills.
    3. **Dosing:**
        a. **Amphotericin B, Azoles or "Fungins":**
            i. Usually given as bolus dosing once daily to TID.
            ii. May be given as continuous bladder irrigation: 50 mg/liter Over 24 hours @ 42 ml/hour
    4. **Special Considerations:**
        a. Drug interactions may occur with statins, coumadin, antivirals, benzodiazepines, oral hypoglycemic drugs and transplant anti-rejections drugs
        b. Side effects can be pre-treated with Acetaminophen or Diphenhydramine

g. Pain Control
    i. **Uses:** Control of pain
    ii. **Adverse Effects:** May cause hypotension, especially in volume depleted patients or those with right-sided heart failure Respiratory Depression
    iii. **Dosing:**
        1. **Morphine:**
            a. Loading dose: 2 mg increments given every 5-10 minutes until adequate pain control; typically max dose is 10 mg-may be higher in patients on chronic pain therapy
            b. Infusion: 1-10 mg/hour
        2. **Fentanyl:**
            a. Loading dose: 1-5 mcg/kg given IV push
            b. Infusion: 1-5 mcg/kg/hour
        3. **Hydromorphone (Dilaudid):**
            a. Loading dose: 0.5-4 mg IV slow push
            b. Continuous infusion: 1-10 mg/hour
    iv. **Special Considerations:**
        1. Avoid Naloxone as this could precipitate acute withdrawal
        2. Pump malfunction could precipitate withdrawal
        3. Antihistamines (both H1 and H2) may counteract hypotension; this is an MC option

h. PCA pumps and subcutaneous pumps
    i. **Uses:** Treatment for patients with palliative care or chronic pain conditions. Often PO analgesia is not feasible
    ii. **Adverse Effects:** Hypotension, Respiratory depression, Catheter site infection or irritation
    iii. **Dosing:**
        1. **PCA (Patient Controlled Analgesia) Pumps**
            a. **Morphine, Fentanyl and Hydromorphone are most commonly used.**
            b. Pre-programmed settings for patient
            c. Patient may require assistance to "self-administer" medication
        2. **Subcutaneous Catheter Pumps**
            a. **Morphine most commonly used.**
            b. Up to 2 mLs volume at a time regardless of concentration

c. May also give IV fluids at a usual rate of 1-10 mLs/hour; MAX of 25 mLs/hr

iv. **Special Considerations:**

1. Encourage patient to use medication as needed
2. Avoid Naloxone as this could precipitate acute withdrawal
3. Pump malfunction could precipitate withdrawal
4. Subcutaneous catheter sites need to be changed every 7 days

i. Antidotes

i. N-Acetyl Cysteine/NAC (Mucomist)

1. **Uses:** Acetaminophen overdose-toxic quantities
2. **Adverse Effects:** Anaphylactoid type reactions (urticaria, flushing, hypotension and bronchospasm)
3. **Dosing:**
   a. Loading dose: 150 mg/kg over 15-20 minutes
   b. Maintenance infusion: 50 mg/kg over 4 hours
   c. Then: 100 mg/kg over 16 hours
4. **Special Considerations:**
   a. Ideal time of onset of treatment is within 8-10 hours of ingestion
   b. Anaphylactoid reactions may be treated with IV diphenhydramine
   c. Maintenance infusion must be doubled at the 4 hour period

ii. Cyanide Antidote Kit

1. **Uses:** Cyanokit is indicated for the treatment of known or suspected cyanide poisoning.
2. **Adverse Effects:** Allergic reaction, HTN, Hematochezia
3. **Dosing:**
   a. The starting dose: for adults: 5 g (i.e., both 2.5g vials)
   b. Administered as an intravenous infusion over 15 minutes (approximately 15 mL/min), i.e., 7.5 minutes/vial.
   c. Depending upon the severity of the poisoning and the clinical response, a second dose of 5 g may be administered by intravenous infusion for a total dose of 10 g.
   d. The rate of infusion for the second dose may range from 15 minutes (for patients in extremis) to two hours, as clinically indicated.
4. **Special Considerations:**
   a. Caution should be exercised when administering other cyanide antidotes simultaneously with Cyanokit,
   b. If a decision is made to administer another cyanide antidote with Cyanokit, these drugs should not be administered concurrently in the same intravenous line.
   c. Comprehensive treatment of acute cyanide intoxication requires support of vital functions.
   d. Cyanokit should be administered in conjunction with appropriate airway, ventilatory and circulatory support.
   e. Once reconstituted, hydroxocobalamin is stable for up to 6 hours at temperatures not exceeding 40°C (104°F). Do not freeze.
   f. Any reconstituted product not used by 6 hours should be discarded.

iii. Thiamine

1. **Uses:** Wernicke's encephalopathy
2. **Adverse Effects:** Possible anaphylactic reactions

3. **Dosing:** 100 mg IV over 15-30 minutes
4. **Special Considerations:**
    a. Glucose administration in nutritionally depleted patients should be accompanied by thiamine

i. Bicarbonate gtt
    a. **Uses:** Tricyclic, aspirin or other acidotic overdoses. Renal protection after IV contrast or with sever e muscle breakdown (rhabdomyolysis)
    b. **Adverse Effects:** Sodium load
    c. **Dosing:** Titrated to urine pH >7 by hospital staff
    d. **Special Considerations:**
        i. Usually will have a Foley to check urine pH and output
        ii. May be associated with hypokalemia

ii. Pyridoxine
    a. **Uses:** Isoniazide (INH) Overdose
    b. **Adverse Effects:** GI upset Headache or sleepiness Tingling or burning of hands/feet
    c. **Dosing:** 5 grams IV over 3 – 5 minutes; repeat every 5-20 minutes until seizures resolve
    d. **Special Considerations:**
        i. Often patient is in status epilepticus; seizures may respond to benzodiazepines

i. Glucagon
    a. Uses: Beta-blocker reversal, Calcium Channel Blocker reversal, hypoglycemia
    b. Adverse Effects: Nausea and Vomiting, anaphylaxis, hypertension or hypotension.
    c. Dosing:
        i. Bolus: 3-10 mg IV
        ii. Infusion: 1-10 mg/hr
    d. Special Considerations:
        i. Given with an antiemetic

ii. Intralipid
    a. Uses: Beta-blocker, Calcium Channel blocker or other highly lipid soluble overdose.
    b. Adverse Effects: Hyperglycemia
    c. Dosing:
        i. Bolus: 1.5 mL/kg IV,
        ii. Infusion: 0.25 mL/kg/min
    d. Special Considerations:
        i. Fat overload syndrome may result if given too rapidly. Usually reversible upon discontinuation.
        ii. Contraindicated if known allergy to egg or soybean proteins

j. GI Drugs
    i. Acid Reduction
        1. **Uses:** Decrease secretion of gastric acid or chronic reflux. Patients with UGI Bleed
        2. **Adverse Effects:** (all rare). Occasional CNS symptoms-more so in the elderly. Jaundice. GI upset
        3. **Dosing:**
            a. **Pantoprazole (Protonix**)
                i. Bolus: 80 mg over 5 minutes
                ii. Infusion: 8 mg/hr

b. **Lansoprazole (Prevacid)**
    i. Bolus: 30 – 60 mg over 30 minutes
    ii. Infusion: 6 mg/hr
c. **Ranitidine (Zantac)**
    i. Bolus: 50 mg over 20 -30 minutes
    ii. Infusion: 150 mg over 24 hours

4. **Special Considerations:**
    a. May be used for antihistamine effects

ii. GI Bleed related medications
1. **Uses:** Variceal Upper GI Bleed
2. **Adverse Effects:** Gall Bladder sludging or stones. Diarrhea and GI Upset. Hypoglycemia
3. **Dosing:**
    a. **Octreotide:** 50 mcg IV bolus, then 50 mcg/hour
4. **Special Considerations:**
    a. Alters the balance between insulin/glucagon; could result in either hypoglycemia or hyperglycemia
    b. **Vasopressin** is presently rarely used due to its potent vasoconstrictive and catecholamine inducing properties

III. IV PUMPS

1. <u>External infusion pump</u>

a. a medical device used to deliver fluids into a patient's body in a controlled manner.
b. Infusion pumps may be capable of delivering fluids in large or small amounts, and may be used to deliver nutrients or medications – such as insulin or other hormones, antibiotics, chemotherapy drugs, and pain relievers.
c. Operated by a trained user who programs the rate and duration of fluid delivery through a built-in software interface.

2. <u>Routes</u>

a. Intravenous*
b. Subcutaneous
c. Arterial
d. Epidural
e. Enteral

3. <u>Advantages</u>

a. Ability to deliver fluids in very small volumes
b. Ability to deliver fluids at precisely programmed rates or automated intervals.
c. Can deliver nutrients or medications, such as insulin or other hormones, antibiotics, chemotherapy drugs, and pain relievers

4. <u>Types of infusion pumps by Volume</u>

a. Large Volume Infusion Pumps

i. These infuse large volumes of substances, mostly nourishment, into a patient. They usually use peristaltic computerized pumps. Usually, there is a computer controlled roller or a set of fingers sequentially pressing a rubber tube through which the medication flows. This permits different rates of flow of the medication depending on the patient's need.

b. Small Volume Infusion Pumps/Syringe Pumps

i. Small volume pumps stay true to the name. They deliver smaller medication substances such as hormones by a controlled motor mechanism that uses a plunger-like pumping effect. This pushes the medication through a syringe and into the tube (syringe infusion pumps) to the patient.

5. Specialty Infusion Pumps

a. ***Enteral Pump:*** A pump used to deliver liquid nutrients and medications to a patient's digestive tract.
b. ***Patient-Controled Analgesia (PCA) Pump:*** A pump used to deliver pain medication, which is equipped with a feature that allows patients to self-administer a controlled amount of medication, as needed.
c. ***Insulin Pump:*** A pump typically used to deliver insulin to patients with diabetes. Insulin pumps are frequently used in the home.

6. Types of Infusion pumps based on Mechanism

a. ***Elastometric Pump:*** fluid is held in a stretchable balloon reservoir, and pressure from the elastic walls of the balloon drives fluid delivery.
b. ***Peristaltic pump:*** a set of rollers pinches down on a length of flexible tubing, pushing fluid forward.
c. ***Multi-channel pump:*** fluids can be delivered from multiple reservoirs at multiple rates.

7. Safety Concerns

a. Under-infusion
b. Over-infusion
c. Missed treatments
d. Delayed therapy
e. Between 2005-2009, the FDA has approximately 56,000 reports of adverse events associated with the use of infusion pumps, including 500 deaths.
f. Due to both user error, and device design deficiencies.

8. **Safety Features**

a. Alarms
b. Operator alerts (ex. Downstream occlusion or air in tubing alerts)
c. ***Smart-pump:*** is equipped with safety features, such as user-alerts that activate when there is a risk of an adverse drug interaction, or when the user sets the pump's parameters outside of specified safety limits.
d. Batteries, so the pump can operate if the power fails or is unplugged.
e. Anti-free-flow devices prevent blood from draining from the patient, or infusate from freely entering the patient, when the infusion pump is being set up.
f. A "down pressure" sensor will detect when the patient's vein is blocked.
g. An "air-in-line" detector. A typical detector will use an ultrasonic transmitter and receiver to detect when air is being pumped. Some pumps actually measure the volume and may even have

configurable volumes, from 0.1 to 2 ml of air. None of these amounts can cause harm, but sometimes the air can interfere with the infusion of a low-dose medicine.

h. An "up pressure" sensor can detect when the bag or syringe is empty, or even if the bag or syringe is being squeezed.
i. A drug library with customizable programmable limits for individual drugs that helps to avoid medication errors.
j. Mechanisms to avoid uncontrolled flow of drugs in large volume pumps (often in combination with a giving st based free flow clamp) and increasingly also in syringe pumps (piston-brake)
k. Many pumps include an internal electronic log of the last several thousand therapy events. These are usually tagged with the time and date from the pump's clock. Usually, erasing the log is a feature protected by a security code, specifically to detect staff abuse of the pump or patient.
l. Many makes of infusion pump can be configured to display only a small subset of features while they are operating, in order to prevent tampering by patients, untrained staff and visitors.

9. Risk Reduction

a. Plan ahead

i. Have a back-up plan in case of an infusion pump failure that details:
ii. How to handle infusions when the infusion pump fails in vulnerable patient populations (e.g., individuals sensitive to fluid overload).
iii. This may include clamping and disconnecting the infusion tubing from the patient to prevent over-infusion prior to obtaining a new infusion pump.

b. Label

i. Label the infusion pump channels with the name of the medication or fluid, if your infusion pump does not display the name.
ii. Label the infusion pump tubing at the port of entry with the medication or fluid name.

c. Check

i. Verify that the infusion pump is programmed for the right dosage, at the right rate and volume to be infused.
ii. Monitor for signs of over- or under-infusion of high-risk medications by using other patient monitoring systems such as cardiac, pulse oximetry, end tidal CO_2, and glucose meters, when applicable.

d. Use

i. Use the drug library when applicable. Promptly respond and pay close attention to displayed alerts and cautions.
ii. Use the "5 rights" for safe medication administration: the right patient, the right drug, the right dose, the right route, and the right time.

IV. BLOOD PRODUCTS

1) Introduction and Objectives

a) Blood Products: understand the different types of blood product forms
b) Blood administration procedure and protocol: Understand the indications, equipment and protocols for blood product administration
c) Basic Hematology: Understand the basics of hematology as it relates to blood products and blood product transfusions

d) Transfusion reactions and treatment: identify and treat the various possible complications and reactions that occur secondary to blood product transfusions

2) Protocol Overview

a) Blood Product Transfusion Protocol:

b) **Procedure**:

i) **Indications**:

(1) Significant hypovolemia as the result of acute blood loss
(2) Symptomatic anemia
(3) Decreasing hemoglobin level
(4) Decreasing hematocrit value
(5) To increase oxygen-carrying ability
(6) Decrease clotting factors
(7) Presurgical care in select cases

ii) Equipment

(1) Physician orders
(2) Blood product, typed and crossmatched (in some cases may be cryoprecipitate, platelets, or plasma)
(3) Dedicated venous access line (18-guage or larger needle)
(4) Filtered administration set
(5) Normal saline solution
(6) Thermometer

iii) **Complications**:

(1) Anaphylaxis
(2) Hemolytic reaction
(3) DIC
(4) Transfusion reaction
(5) Infection
(6) Signs of complications include the following:
(a) Body temperature of 2°F (1°C) or more above the baseline temperature
(b) Hives, itching, or skin symptoms
(c) Swelling, soreness, or hematoma at the venous site
(d) Flank pain
(e) Tachycardia
(f) Respiratory distress (wheezing and dyspnea)
(g) Hypotension
(h) Bleeding from widely varied sites or previously clotted wounds
(i) Blood in urine
(j) Anaphylaxis
(k) Nausea and vomiting

iv) Steps: Blood product should be initiated by the transferring facility;

(1) Prior to leaving the transferring facility, physically look at the product with the transferring nurse and confirm you have the right product for the right patient. Review the order with the transferring nurse.
(2) ALL PRODUCTS MUST BE ADMINISTERED VIA AN IV PUMP
(3) Re -confirm the order or protocol prior to administering
(4) Check the patient for the following: right patient, right blood product, and right type. **Have a second provider confirm steps c and d with you**.
(5) Assess baseline vital signs and temperature

(6) Ensure suitable venous access (usually requires 18 – gauge or larger). At this point, patient preparation is complete and the transfusion procedure begins.
(7) Check the blood for the following: right patient, right blood product, right type, and expiration date.
(8) Asses the patient for the possibility of a transfusion reaction, and consider prophylactic administration of ibuprofen or acetaminophen and diphenhydramine.
(9) Maintain the temperature of the blood product
(10) Flush the primary tubing with normal saline
(11) Cover the administration filter with blood
(12) Connect the blood to the tubing
(13) Piggyback into the IV line of normal saline
(14) Start the transfusion slowly
(15) Monitor every 5 minutes for adverse reactions
(16) NOTE:
  (a) DO NOT MIX BLOOD WITH 5% DEXTROSE IN WATER (CAUSES HEMOLYSIS).
  (b) DO NOT MIX WITH LACTATED RINGERS (CAUSES CLOTTING)
  (c) DO NOT MIX WITH MEDICATIONS (MAY REACT)
  (d) HAVE A SECOND VENOUS ACCESS AVAILABLE

3) <u>Blood Products</u>

a) Blood Product Overview
  i) Blood compositions
  ii) Blood product manufacturing overview

b) <u>Types of Blood Products</u>

i) Whole Blood (450 – 500 ml/unit)
  (1) Shelf life of 21-35 days
  (2) Refrigerated
  (3) Uses: Trauma or Surgery

ii) Packed Red Blood Cells (250 ml/unit)
  (1) Composition: 1 unit contains same red cell mass as 1 unit of whole blood in half the volume
  (2) Shelf life: up to 42 days
  (3) Refridgerated
  (4) Effect: 1 unit will raise hemoglobin by 1 g/dL in an adult
  (5) Dose: In stable patients, usually transfused 1-2 units at a time, with each unit transfused over 2 hours
  (6) Key uses: Trauma, surgery, anemia with a Hgb < 7, any blood loss, blood disorders such as sickle cell

iii) Platelets (30-50 mL)
  (1) Composition: 1 unit contains most of the platelets from 1 unit whole blood in 30-50 mL plasma
  (2) Shelf life: 5 days
  (3) Kept at room temperature with constant agitation to prevent clumping
  (4) Effect: 1 unit will raise platelet count by 5,000-10,000/mm3 in an adult
  (5) Dose: 1 unit per 10 kg (6-10 units in an adult)
  (6) 6 units platelets contain the equivalent of 1 unit FFP. More platelents are needed with massive blood transfusions due to dilution from transfused RBC's
  (7) Key uses: Cancer treatments, Organ transplants, trauma, surgery, organ transplant, sepsis

iv) Fresh Frozen Plasma (200-250 mL)
  (1) Composition: 1 bag contains coagulations factors II, V, VII, VIII, IX, XI and XII; fibrinogen,

antithrombin, protein C and vWF
(2) Shelf life of 1 year
(3) Frozen
(4) Effect: 1 bag FFP will approximately increase all coagulation factor levels by 2-3% in an average adult.
(5) Dose: 15-20 ml/kg (4-10 bags in and adult). Typically given in a 1:1:1 ration of PRBC's:Platelets:FFP in massive blood transfusion protocols.
(6) Key uses: Trauma, burn patients, bleeding disorders with an INR > 1.6, Coumadin reversal, TTP, HUS, Hereditary angioedema, sepsis

v) Prothrombin Complex Concentrate
(1) Composition: Vitamin K-dependent factors II VII, IX, and X (3 factor preparations lack factor VII)
(2) Dose: varies
(3) Primarily given to patients on warfarin with life-threatening bleeding
(4) Advantage over FFP is higher concentration of clotting factors (25X compared to FFP)

vi) Cryoprecipitate (10-25 mL)
(1) Composition: 1 bag contains coagulation factors from single-donor plasma: 100-250 mg fibrinogen; 80-100 units factor VIII, 50-60 mg fibronectin and 40-70% vWF (but this degrades during storage)
(2) Shelf life of 1 year
(3) Frozen
(4) Effect: 1 bag will raise factor VIII level by 3%
(5) Dose: 1-2 bags per 10 kg (7-15 bags in an adult)
(6) Key uses: Massive hemorrhage or transfusion, surgical bleeding, Hemophilia A, Von Willebrand disease (most common hereditary coagulation abnormality), Factor XIII deficiency and as a source of fibrinogen in patients with congenital fibrinogen deficiency.

vii) Albumin
(1) Derived from large pools of human plasma
(2) Albumin 20% is indicated in emergency treatment of hypovolemia with or without shock
(3) Effect in reversing hypovolemia depend largely upon its ability to draw interstitial fluid into the circulation and is most effective in patients who are well hydrated
(4) Used in treatment of refractory hypotension in the setting of sepsis after fluid hydration; following large volume paracentesis of cirrhotic ascites; treatment adjuncts in ARDS, Acute Nephrosis, Hyperbilirubinemia secondary to Hemolytic disease of the newborn and ovarian hyperstimulation syndrome
(5) Dose largely differs based on indication. When used for hypovolemia in adults, 25 g IV is given and may be repeated in 15-30 minutes if adequate response is not seen
(6) Adverse Effects:
(a) Most adverse effects are anaphylactoid type reactions. The most serious reactions are anaphylactic shock, circulatory failure, cardiac failure and pulmonary edema

viii) Plasma Protein Fraction
(1) Prepared from large pols of human plasma.
(2) Contains 88% Albumin and 12% globulins
(3) Is a plasma expander, and in normal human volunteers, has resulted in an increased blood volume which has lasted up to 48 hours.
(4) Uses: hypovolemic shock due to burns, crush injuries, abdominal emergencies and any other cause where a predominant loss of plasma fluids and not red blood cells.
(5) Contraindicated on patients on cardiopulmonary bypass, severe anemia, CHF or increased blood

volume

(6) Dose: usual minimal effective dose in adults in 250-500 mL

ix) Synthetic Blood Substitutes

(1) AKA: artificial blood or blood surrogate

(2) Rarely used, most are still in clinical trials or through clinical trials but failed FDA approval

(3) A synthetic substatnce used to mimic and fulfill some functions of biological blood. Its aim is to provide an alternative to blood transfusion. There are no well-accepted oxygen-carrying blood substitutes available.

(4) Assist clinicians avoid the risks of disease transmission and immune suppression, address concerns of Jehovah's Witnessess and others who have religious objections to receiving transfused blood.

(5) Types:

(a) Hemoglobin-based oxygen carriers (HBOC)

(i) Use Hemoglobin removed from RBC's and treated to with cross-linking, polymerization and encapsulation to carry and release oxygen independent of the RBC

(b) Perflurocarbon-based Oxygen Carriers (PFBPC)

(i) Chemical compounds that carry and release oxygen. Small size gives theoretical benefit as they can traverse locations that RBC's cannot go. In theory this can benefit damaged, blood-starved tissue, which conventional red cells cannot reach. PFC solutions can carry oxygen so well that mammals, including humans, can survive breathing liquid PFC solution, called liquid breathing.

x) Tranexamic Acid

(1) Composition: Synthetic derivative of lysine which binds plasminogen thereby inhibiting fibrinolysis

(2) Dose: (from CRASH-2 trial) 1 gram IV over 10 min, then another 1 gram over 8 hours

(3) Recently found to cause small but statistically significant decrease in mortality from hemorrhage in severe trauma if given within 3 hours of injury. With each 15 minute delay in administration, the benefit of this decreases by 10%

4) Basic Hematology

a) Blood Typing – A, B, AB and O

i) Antigen – Definition

ii) Antibody Definition

iii) Universal recipient

iv) Universal donor

v) Co-dominance

vi) Rh Antigen

vii) RhoGam

viii) Agglutination

ix) Blood Type Compatibility Table

b) Blood Composition

i) Plasma (55% of Total Blood Volume):

(1) 91% Water

(2) 7% Blood Proteins (Albumin, Antibodies), Fibrinogen and Clotting Factors

(3) 2% Nutrients (amino acids, sugars and lipids), Hormones (erythropoietin, insulin, etc.) and Electrolytes (sodium, potassium, calcium, etc.)

*(4) This makes up the patients serum, except for Fibrinogen and clotting factors**

ii) ***Buffy Coat (< 1%) of Total Blood Volume):***
(1) WBC's and Platelets

iii) ***Red Blood Cells (45% of total blood volume)***
(1) Hemoglobin – Function to move O2 and CO2
(2) Hematocrit = Volume of RBC's/Total volume

c) How blood clots are formed and the coagulation cascade

i) Endothelial cell injury → exposure of blood to collagen
(1) vWF binds to collagen → Platelet adhesion to both exposed collagen and the vWF by glycoprotein G IIb/IIIa
(2) Fibrinogen → fibrin
(a) Prothrombin → Thrombin
(b) Coagulation cascade
(i) Extrinsic: III (tissue factor) → VII → X → II (Thrombin) → I (Fibrin)
1. Smarter pathway: gets activated at the source of initial insult and starts the coagulation cascade. Gets a little bit of thrombin going. Thrombin then, in turn, activates a whole lot of coagulation factors (5, 7, 8, 11, 13)
(ii) Intrinsic: XII → XI → IX +VIII → X+ V → II (Thrombin) → I (Fibrin)
1. Workhorse pathway
(iii) Factor XIII
1. Factor 13 binds fibrin into its fibrin mesh
(3) Negative Feedback loop
(a) Governed by Thrombin
(b) Thrombin → stimulates production of Plasmin → acts directly on mesh networks of Fibrin and breaks them apart. Helpful, but doesn't prevent the continued production of Fibrin
(c) Thrombin → stimulates production of antithrombin → decreases production of Thrombin from prothrombin and impedes the amount of activated Factor X.
(4) Hemophilia – Targets the intrinsic pathway
(a) Hemophelia A → Factor VIII deficiency
(b) Hemophelia B → Factor IX deficiency
(c) Hemophelia C → Factor XI deficiency

5) Transfusion Reactions

a) Transfusion reactions overview

b) Acute Transfusion Reactions

i) ***Allergic reactions and anaphylaxis***
(1) Allergic reactions range from mild (urticarial) to life threatening (anaphylactic).
(2) (mild): sensitivity to infused plasma proteins
(a) Incidence 1:100
(b) S/S (Mild): chills, facial and laryngeal edema, pruritus, urticaria, and wheezing
(3) (Severe): antibody – antigen reaction
(a) Incidence: 1:20,000-50,000
(b) S/S (Severe): Dyspnea, bronchospasm, chest pain, circulatory collapse, cardiac arrest
(c) Patients with anaphylactic transfusion reactions, like those with urticarial reactions, may present with hives, but they are distinct in that they also develop hypotension, bronchospasm, stridor, and gastrointestinal symptoms. Anaphylaxis occurs in response to a recipient's presensitization

to a variety of proteins in donor plasma. For example, anaphylaxis occurs because of donor IgA being infused into a recipient who is IgA deficient and has preexisting circulating anti-IgA. Prevention of anaphylactic transfusion reactions includes avoiding plasma transfusions with IgA in patients known to be IgA deficient. Cellular products (e.g., RBCs, platelets) may be washed to remove plasma in patients with an IgA deficiency. The best precaution is observation of the patient during the initial 15 minutes of transfusion.

(4) **Treatment**:
   (a) Stop the transfusion. Disconnect the blood administration set from the adapter or hub of venous access device.
   (b) Keep the vein open with normal saline
   (c) Monitor vital signs
   (d) Notify med control or EMS MD if able
   (e) Follow **Allergic Reaction Protocol**

ii) Febrile nonhemolytic transfusion reaction

(1) Defined as a rise in body temperature of at least 1.8°F (1°C) above 98.6°F (37°C) within 24 hours after a transfusion. Mechanism isn't completely known but two mechanisms have been proposed that describe an interaction between recipient and donor non-RBC components.
(2) May be difficult to distinguish from eary acute hemolytic reactions and is a diagnosis of exclusion, after other causes of fever are rules out (sepsis, hemolysis, etc.).
(3) S/S: Fever (as high as 104°F), chills.
(4) Occurs in 1-35% of transfusions
(5) Occurs more often in patients who have been transfused repeatedly and in patients who have been pregnant.
(6) Caused by platelet transfusions more often than RBC transfusions
(7) ***Leukoreduction***, which is the removal or filtration of WBC's from the donor blood, has decreased FNHTR rates
(8) **Treatment**:
   (a) Discontinue the transfusion immediately
   (b) Give antipyretics (Tylenol 650 mg to 1 gm PO or Ibuprofen 400-800 mg PO
   (c) Keep the vein open with Normal Saline
   (d) Notify MD

iii) Acute Hemolytic transfusion reaction

(1) Caused by immune destruction of transfused RBCs, which are attacked by the recipient's antibodies.
(2) Can be acute or delayed.
(3) In acute hemolytic transfusion reactions, there is a destruction of the donor's RBCs within 24 hours of transfusion. The antibodies to the antigens of the ABO blood group or alloantibodies to other RBC antigens are produced after immunization through a previous transfusion or pregnancy.
(4) Incidence of minor reaction: 1/6,000 transfusions. Incidence of fatal reaction: 1/100,000
(5) S/S: Chills, fever, headache, backache, dyspnea, cyanosis, chest pain, tachycardia, hypotension
(6) Treatment:
   (a) Discontinue the transfusion immediately. NOTE: When the transfusion is discontinued, the blood tubing must be removed as well. Use new tubing for the normal saline infusion.
   (b) Keep the vein open with normal saline.

(c) Send the remaining blood, a sample of the client's blood and a urine sample to the laboratory
(d) Notify transferring MD, receiving MD or EMS MD as soon as possible
(e) Monitor vital signs
(f) Monitor fluid intake and output

iv) Transfusion Related Acute Lung Injury (TRALI)

(1) TRALI is noncardigenic pulmonary edema causing acute hypoxemia
(2) Occurs within 6 hours of transfusion
(3) Incidence: 1:500,000
(4) Patients do not have any other risk factors for acute lung injury
(5) Due to patient antibodies that activate the immune system, resulting in massive pulmonary edema.
(6) Donor products that contain large amounts of plasma from multiparous women are associated with TRALI and some groups advocate using male predominant plasma for transfusions.
(7) Leading cause of transfusion-related mortality.
(8) Treatment:
(a) Respiratory support during transfer
(b) Notify transferring MD, receiving MD or EMS MD as soon as possible
(c) Monitor vital signs

c) Delayed Transfusion Reactions

i) Delayed hemolytic reaction

(1) Caused by immune destruction of transfused RBCs, which are attacked by the recipient's antibodies.
(2) S/S: Chills, fever, headache, backache, dyspnea, cyanosis, chest pain, tachycardia, hypotension
(3) Typically occurs 7-10 days after transfusion, but may occur up to 3-4 weeks
(4) Treatment:
(a) Supportive
(b) Keep the vein open with normal saline.
(c) Notify transferring MD, receiving MD or EMS MD as soon as possible
(d) Monitor vital signs
(e) Monitor fluid intake and output

ii) Transfusion-associated graft-versus-host disease

(1) A consequence of a donor's lymphocytes proliferating and causing an immune attack against the recipient's tissues and organs.
(2) It is fatal in more than 90 percent of cases.
(3) Patients vulnerable to this condition are those who are immunocompromised or immunocompetent and who are receiving transfusion with shared HLA haplotypes (i.e., donor is a relative)
(4) Symptoms include rash, fever, diarrhea, liver dysfunction, and pancytopenia
(5) occurring one to six weeks after transfusion.
(6) Gamma irradiation of blood products keeps the donor lymphocytes from proliferating and can prevent transfusion-associated graft-versus-host disease.

iii) Bacterial contamination

a. Due to administration of blood products contaminated with pathogens. Rarely identified in the acute setting.

b. Risk of transmission:
   i. Hep C: < 1:1.8 million
   ii. Hep B: 1:350,000
   iii. HTLV 1 or 2: 1 in 2 million
   iv. HIV: 1: 2.3 million
   v. CMV/EBV: rare * (exact risk unknown)
c. S/S: chills, fever, chills, vomiting, abdominal cramps, bloody diarrhea, shock.
d. Treatment:
   i. Stop the transfusion.
   ii. Disconnect the blood administration set from the adapter or hub of venous access device.
   iii. Keep vein open with normal saline
   iv. Hold and send the remaining blood to a laboratory (at receiving hospital if in transit or at transferring hospital if haven't left yet)
   v. Administer IV fluids to maintain SBP > 90 mm Hg
   vi. Further orders per med control (transferring MD, receiving MD, or EMS MD)

d) Massive Transfusion Reactions

i) Transfusion Associated Circulatory Overload (TACO)

(1) TACO is the result of a rapid transfusion of a blood volume that is more than what the recipient's circulatory system can handle.
(2) Not an antibody-mediated reaction
(3) Those with underlying cardiopulmonary compromise, renal failure or chronic anemia are at the highest risk.
(4) S/S: Tachycardia, cough, dyspnea, hypertension, widened pulse pressure.
(5) Diagnosis is clinical
(6) Transfusion of lower volumes, or at a slower rate, may help prevent it.
(7) Treatment
   (a) Largely supportive during transfer
   (b) Provide supplemental oxygen 2-4 L/Min
   (c) Notify transferring MD, receiving MD or EMS MD as soon as possible
   (d) Patient will ultimately requite diuresis to decrease volume overload

ii) Metabolic changes

(1) Hypocalcemia

(a) Due to calcium binding to citrate preservative
(b) S/S: arrhythmias, hypotension, muscle cramping, nausea, vomiting, seizure activity, and/or tingling sensation in the fingers. Prolonged QT
(c) Treatment:
   (i) Contact medical control
   (ii) Calcium gluconate 1 gm IV insused slowly, if ordered by medical control
   (iii) Slow or stop the infusion

(2) Hyperkalemia

(a) From lysed RBC's
(b) S/S: Diarrhea, intestinal colic, flaccidity, muscle twitching, oliguria, signs of renal failure,

bradycardia, EKG changes (tall peaked T waves, prolonged pr interval, prolonged QRS and/or cardiac arrest).

(c) See Hyperkalemia Protocol

(d) Treatment:

(i) Perform 12 lead EKG

(ii) Contact medical control

(iii) Treatment per hyperkalemia protocol

(3) Hypokalemia

(a) From citrate metabolism to bicarbonate and resultant alkalosis

(b) S/S: Paresthesias, weakness, hyporeflexia, Dysrhythmias, EKG changes (flat T-waves, U-waves, ST depression).

(c) Treatment:

(i) Obtain EKG

(ii) Contact medical control

iii) Hypothermia

(1) Due to large transfusion of blood products below ideal body temperature

(2) S/S: chills, shivering, hypotension, arrhythmias, bradycardia, and/or cardiac arrest if temp falls below 30° C (86°F)

(3) Treatment:

(a) Stop the transfusion

(b) Warm the patient

(c) Obtain 12 lead EKG

(d) Warm the blood if the transfusion is resumed

iv) Coagulopathy

(1) From low platelets or inadequate transfusion of all blood products during massive transfusion

(2) S/S: Bleeding and oozing from breaks in the skin or gums, abnormal bruising and petechiae.

(3) Treatment:

(a) May be ordered to give platelets, FFP (fresh frozen plasma) or cryoprecipitate, if initiated by transferring facility.

V. CHEST TUBES

1. Objectives:
   a. Identify indications for the use of chest tubes and accompanying signs and symptoms
   b. Describes the risks/complications associated with chest tubes and chest drainage units
   c. Describe the monitoring of chest tubes and chest drainage systems
   d. Describe considerations in caring for the patient who has a chest tube, including chest tube maintenance.
   e. Identify factors that indicate when it is appropriate to discontinue the use of a chest tube.
   f. Describe how to assist with discontinuation of a chest tube.
2. Basic anatomy and physiology

a. Pleural Space: The cavity between the membrane lining of the lung (visceral pleura) and the inner lining of the chest cavity (parietal pleura).
    i. Function:
        1. Prevent friction between the outer lining of the lung and the inner lining of the thoracic cavity during respiration
        2. Hold the two pleural surfaces together, creating negative pressure (a vacuum) that keeps the lungs expanded
b. The lungs are elastic and naturally tend to collapse or recoil, but in normal conditions, the pleural space causes the outer lining of the lung to adhere to the lining of the chest cavity, keeping the lungs expanded to proper position during inspiration and expiration.
c. The pleural space is normally filled with approximately 50 mL of fluid, only enough to essentially provide a thin coating of fluid for the lubrication of the opposing surfaces. Small increases in volumes of air and/or fluid can be absorbed by the body, whereas larger volumes prevent the lung from expanding to the full potential. Breathing is obviously compromised when this excess air and/or fluid enter the pleural space.

3. Definitions
    a. ***Pneumothorax***: A collection of air in the pleural space
    b. ***Tension Pneumothorax***: Occurs when air accumulates in the pleural space to the point of causing mediastinal shift pushing the heart, great vessels, trachea, and lungs toward the unaffected side of the thoracic cavity.
    c. ***Hemothorax***: A collection of blood in the pleural cavity
    d. ***Hemopneumothorax***: An accumulation of both air and blood in the pleural cavity
    e. ***Pleural effusion***: Excess fluid in the pleural cavity
    f. ***Chylothorax***: Accumulation of lymphatic fluid in the pleural space
    g. ***Empyema***: A collection of purulent material from an infection such as pneumonia
4. Chest tube purpose
    a. The chest cavity is normally under negative pressure
    b. When there is an accumulation of positive pressure, a chest tube is needed
    c. Drain body fluids or facilitate the re-expansion of a lung
    d. Certain therapy-related patient management – Intrinsic rewarding in Hypothermia
5. Indications
    a. Pneumothorax, Tension PTX, Hemothorax, Hemopneumothorax, Plaural effusions, Chylothorax, Penetrating chest trauma, Pleural empyema, Post cardiac surgery drainage, hypothermia, need for pleurodesis, chemotherapy administration
6. Contraindications
    a. No absolute contraindications
    b. Multiple adhesions, giant bless, coagulopathies, risks outweigh the benefits
7. Assessment question
    a. What is a collection of air under the skin called
        i. A. Bronchopleural fistula
        ii. B. Empyema
        iii. C. Lung perforation
        iv. D. ***Subcutaneous Emphysema***
8. Chest tube placement:
    a. The position of the chest tube is related to the function of the tube.

b. If used to drain air → the tube may be placed anteriorly near the apex of the lung
c. If used to drain fluid → It may be positioned posteriorly near the base of the lung
d. If used for Hemothorax → may be positioned at apex as well as the lung base.

9. Chest tube sizes:
    a. 8FR – 12 FR → Infants, young children
    b. 16FR – 20 FR → Children, young adults
    c. 24FR – 32 FR → Most popular adult sizes
    d. 36FR – 40 FR → Larger adult sizes
10. Insertion
    a. Video on a chest tube insertion
11. Basics of Chest Drainage Units (CDUs)
    a. A chest drainage unit is a device used to collect chest drainage (air, blood, effusions) and connects to the end of the chest tube. Most commonly, drainage devices use a single unit that has 3 chambers, based on the old 3-bottle system.
    b. Types of systems
        i. Wet system: Use water seal and water column regulator
            1. Regulate suction pressure by the height of the column of water in the suction control chamber. The amount of negative pressure that is transmitted to the patients chest is determined by the height of the water in this chamber, not the level of vacuum set on the regulator.
            2. Disadvantages: can tip over and spill.
        ii. Wet-Dry system: Use water seal and a mechanical regulator
        iii. Dry system: Uses a mechanical regulator, and NO water seal
            1. Regulator mechanically regulates suction pressure
            2. Advantages: Safer during transport, higher suction pressure levels, easier setup, quiet operation without continuous bubbling), no fluid that can evaporate
            3. Disadvantages: Does not provide the same level of patient assessment information as a wet system device.
    c. 3 chamber drainage device (provide separate functions):
        i. Fluid collection
            1. In a traditional water seal operating system, fluids drain from the patient directly into a large collection chamber via a 6-foot patient tube.
        ii. Underwater seal (serves as a simple 1-way valve)
            1. One way valve that allows air to exit the chest and prevents air returning to the patient.
            2. IonAir bubbling through the water seal chamber intermittently is normal when the patient coughs or exhales.
            3. Continuous air bubbling in the chamber can indicate a leak that should be evaluated.
            4. The water seal chamber is connected in series to the collection chamber, and allows air to pass down through a narrow channel and bubble out through the bottom of the water seal.
            5. Since air cannot return to the patient, an UWS is considered one of the safest ways of protecting the patient, in addition to being a very useful diagnostic tools.
            6. The UWS column is calibrated and acts as a water manometer for measuring intrathoracic pressure. As changes in intrathoracic pressure occur, fluctuations in the

water level can be observed in this calibrated column and may provide clinicians with an indication of how the patient is progressing.

iii. Suction control
   1. The use of suction helps overcome an air leak by improving the rate of air and fluid flow out of the patient.
   2. The simplest and most cost effective means of controlling suction is by using a suction control chamber, which is an atmospherically vented section containing water and is connected in series with the water seal chamber and collection chamber.
   3. By adding or removing water in a suction control chamber, the chest drain effectively controls the amount of suction imposed on the patient.
   4. The lower the water content, the lower the imposed suction.
   5. The higher the water level, the higher the imposed suction.

d. Setting up a Traditional (Wet) Water Seal System
   i. Step 1: fill the water seal chamber to the specified level from the manufacturer (usually the 2 cm mark).
   ii. Step 2: Fill Suction control: The suction control chamber is filled with sterile water to the water level that is marked for the desired suction pressure (typically – 20 cm H2O).
   iii. Step 3: Patient connection: Remove patient tube connector cap and insert stepped connector into patient catheter. Remove the "Y" connector insertion.
   iv. Step 4: Applying suction: The tubing on the suction control chamber is attached to wall suction. Start at the lowest suction level, and gradually increase until a gentle bubbling in the suction control chamber is noticed. Adjust the suction control stopcock or suction control source as needed to increase or decrease suction control bubbling. To operate the suction container at -20 cm H2O, wall suction must have at least -80 mm Hg of vacuum.
   v. Step 5: Open suction control stopcock: The suction control stopcock conveniently regulates vacuum to the chest drain. It provides control of suction bubbling and allows efficient use with any unregulated suction source. The stopcock must be on for initial system setup and should not be turned off during patient use.
   vi. Step 6: Placement of unit: For optimum drainage results, always place the chest drain below the patient's chest in an upright position. Avoid accidental knock-over, it is prudent to swing the floor stand open for secure placement on floor or to hang the system bedside, if hangers are provided.

e. Setting up a Dry Suction Water Seal System
   i. Step 1: Fill the water seal chamber to the specified level by the manufacturer (usually the -2 cm mark).
   ii. Step 2: Physician will order the amount of suction (usually -20 cm H2O). Using the dial, place the arrow at the correct amount of suction.
   iii. Step 3: The tubing on the suction control chamber is then attached to wall suction. Start with a lower suction level and gradually increase suction until a gentle bubbling is the suction control chamber is noticed. To operate the suction container at -20 cm H2O, wall suction must have at least -80 mm Hg of vacuum. The bellow must be expanded to mark or beyond for a -20 cmH20 or higher regulator setting. If the bellows is observed to be expanded, but less than the mark on the bellows chamber, the vacuum source pressure must be increased to -80 mmHg or higher.

iv. Note: since there is no bubbling in the dry suction control chamber, the orange bellows are used as a visual indicator of suction operation.

f. Occlusive Dressing:
   i. Dressings are not universal, but the concept is the same
   ii. Steps to applying a chest tube dressing
      1. Use sterile technique
      2. Slide a pre-slit 4x4 around the chest tube on the skin around the tube.
      3. Follow the slit drainage pad, place a nun-slit 4x4 on top.
      4. With ¾ inch tape, secure the dressing with an airtight seal

g. Skin Assessment
   i. Subcutaneous emphysema
      1. Occurs when air or CO2 is trapped in the SubQ tissues
      2. Frequently seen on the face, neck or chest
      3. Appreciated as crepitus (crackling sensation under skin) sometimes described as "Rice Krispies" under skin
      4. Usually painless.
      5. In most cases, surrounding tissue will absorb the SCE after underlying cause is treated

h. Patient assessment
   i. Assess vital signs, respiratory rate, respiratory status, respiratory depth, respiratory pattern, oxygen saturation and ease with respirations.
   ii. Palate skin to assess for SCE
   iii. Signs of respiratory distress: Tachypnea, dyspnea, shortness of breath, tachycardia, decreased or absent breath sounds, accessory muscle use.

i. Monitoring the Chest Drainage Unit
   i. Observe water seal operation
      1. The water seal must be filed and maintained at the 2 cm level to ensure proper operation
      2. Additional sterile water may be added by syringe via the grommet located on the back
   ii. Verify Suction operation
      1. Wet System: Continuous gentle bubbling
         a. Adjust suction control stopcock if necessary.
      2. Dry System: Verify suction operation via the suction monitor bellows
         a. Will not expand if suction is not operating or disconnected.
         b. Bellows must be expanded to the triangular mark or beyond for a -20 cmH20 or higher regulator setting.
   iii. Check the connection source to ensure the unit is suctioning properly
   iv. Tidaling
      1. The water level should fluctuate in the water seal chamber
      2. Corresponds to respiration
      3. Patients not on Mechanical Ventilation
         a. Inhalation = water level should rise
         b. Exhalation = water level should fall
      4. Patients on Mechanical Ventilation

a. The opposite occurs

5. If the lung is re-expanded, tidal in may not be present

j. Monitoring intrathoracic pressure
   i. If the chest tube is not connected to suction, it is utilizing gravity to drain fluid from the chest cavity.
   ii. Assessing Intrathoracic pressure while OFF suction
      1. To accurately read intrathoracic pressure when using gravity only (no suction), the clinician should read directly from the water seal
      2. A rise in water seal = negative pressure is present = patient is healing (good)
      3. Bubbling = positive pressure is present = air leak (not good)
   iii. Calculating intrathoracic pressure (on suction)
      1. Add the readings of suction control chamber + the level of the water seal chamber.
      2. Ex: -20 cm H2O + - 5 cm H2O = -25 cm H2O

k. Monitoring drainage output
   i. Note any sudden increase in output
   ii. Note any sudden changes in output (ex: drainage changes from serous to bloody fluid

l. Air Leaks
   i. Assess for air leaks
   ii. It is important to rectify air leaks because an airtight system is necessary to effective lung expansion (develop negative pressure in the thoracic cavity).
   iii. Assessing for air leak (should only be done under direction of physician)
      1. Clamp off suction for 1 minute
      2. Air leak is present if there is constant bubbling in the water-seal chamber
      3. Starting away from the patient, check all connections. work up towards the patient. Finally, assess the dressing.
   iv. Intermittent bubbling with respiration is expected if the pleural space is leaking (such as in a pneumothorax). However, this should decrease as the pneumothorax improves.

m. Chest tube maintenance
   i. Keep all tubing patent and free of kinks
   ii. Avoid dependent loops as they obstruct drainage and increase positive pressure in the chest cavity
   iii. It is acceptable to gently milk the tubing if a visible clot is obstructing drainage (squeezing hand-over-hand).
   iv. There is no benefit to stripping the tubing as it can result in transient high negative pressure in the pleural space.
   v. When ambulating the patient, the drainage unit must be carried at a level below the patient's chest

n. Dislodgement or Disconnection
   i. Chest tube accidentally falls out
      1. Instruct the patient to perform the Valsalva maneuver
      2. At end expiration, immediately cover the insertion site with Vaseline gauze, a dry sterile dressing and occlusive tape.
   ii. In the event of chest-tube disconnection with contamination
      1. Submerge the tube 1" to 2" (2-4 cm) below the surface of a 250 mL bottle of sterile water or saline solution until a new CDU is set up.

2. This establishes a water seal, allows air to escape, and prevents air re-entry.
   iii. Evaluate the patient for life threatening situations, such as a tension PTX
o. Changing the Chest Drainage Unit
   i. Instruct the patient to exhale and hold his or her breath (perform the valsalva)
   ii. Clamp the chest tube with a padded Kelly clamp 1-2" from the patient.
   iii. Place a second clamp dismally. Aseptically, disconnect tubing from old chest drainage unit and connect to the new chest drainage unit.
   iv. When completed, remove clamps within one minute and have patient breath normally.
   v. Secure all connections with tape.

12. Heinrich chest drainage valve (aka: flutter valve)
   a. Alternative to chest drainage units
   b. Connects to the chest tubing and allows air to pass in one direction only
   c. Functions in any position
   d. Does not need to be clamped
   e. Regulated suction can be attached, if necessary
   f. The valve drains into a plastic bag which can also be held in any position
   g. Most commonly for smaller pneumothorax
   h. Potential Problems
      i. More prone to clogging of the tube
      ii. Fluid commonly leaks from valve (can attach a sputum collection cup to catch)

VI. LTV 1200 VENTILATOR

Purpose: From time to time EMS will be called upon to transport patients whose ventilations are being assisted by a mechanical ventilator. Ventilators are used to provide respiratory support for patients who are unable to effectively breathe on their own. This protocol will guide the caregiver in maintaining proper settings involved in providing adequate ventilatory assistance to the patient.

Indication:

1. Continuation of ventilator controlled respirations on chronic ventilator dependent patients
2. Assist/Control ventilations on any intubated patient in respiratory failure/arrest that is being transported to a care facility.

Adverse Effects/Complications:

1. Increased intra-thoracic pressure
2. Decrease venous return to the heart and decrease cardiac output (hypotension, tachycardia)
3. Increased V/Q ratio (ventilation/perfusion ratio)
4. Decrease blood flow to the kidney with resultant fluid retention (edema)
5. Air trapping and intrinsic PEEP (auto PEEP)
6. Barotrauma
7. Nosocomial infections of the lungs and sinuses
8. Respiratory alkalosis
9. Agitation and increased respiratory distress
10. Increased work of breathing

Lesson Plan: EPIC - Online

General Comment: *There are many commercial ventilators on the market. Most of the ventilators used in the pre-hospital settings are fairly simple to use. Most if not all have built-in safety features, which prevent over inflating the lungs and causing barotrauma. Everyone must be familiar and in-serviced on the particular ventilator being used.*

General Ventilator settings for transport ventilators:

For the most part, there are a few settings that are common/standard to all ventilators:

1. **FIO_2** (Percent of inspired oxygen (room air is 21%): **21% - 100%.** Titrate to maintain pulse ox between 92% - 94%
2. **Tidal Volume**: **7 – 10 ml/kg** (ideal body weight)
3. **Select Mode**: CPAP, Intermittent mandatory ventilation (IMV, Synchronized Intermittent mandatory ventilation (SIMV)
    a. To manage work of breathing, use assist/control mode. If patient is paralyzed and sedated, there is no difference between assist control (AC) and SIMV
4. **Respiratory Rate: Set between 10-12 breaths/minute**
    a. Selection varies on ventilators to accommodate a range of patient ages and conditions
    b. NOTE: On some ventilators, inspiratory flow rate (usually 40-60 L/second) is determined by tidal volume, respiratory rate, and in the Inspiratory:Expiratory (I:E) ratio. (The I:E ratio is generally 1:2 to allow for complete exhalation and prevent air trapping). On other ventilators, flow rate is independently set, which allows adjustment of air-flow to the flow wave pattern that Is most comfortable for the patient. If the patient is having difficulty with spontaneous breathing, increasing the flow rate may be indicated. However, a higher flow rate means a shorter inspiratory time and usually a higher respiratory pressure secondary to increased resistance, with a lower flow rate requiring a longer inspiratory time with a decreased inspiratory pressure. The paramedic should always consult with medical control before changing the flow rate on any ventilator device.
5. **Adjust the peak flow rate or inspiratory time** to accommodate the patients inspiratory flow demand and to allow for sufficient expiratory time and avoidance of auto-PEEP.
6. **Adjust the sensitivity to -1cm H2O**
7. **Pressure suppor**t: Usually set at **10 cm H_2O**
8. **PEEP (Positive End Expiratory Pressure): Usual setting is 5 cm H_2O**

Procedure

A. **Patients already on Ventilator**
    a. As part of your initial patient assessment inquire if patient has any spontaneous respiratory effort or is 100% dependent on the ventilator
    b. Make note of patient's vital signs before any change over occurs. This includes the pulse ox.
    c. Assess the ET tube or Trachea tube placement to assure they are properly secured
    d. Acquire the patient's current ventilator settings from the nurse or RT caring for the patient. Try to match these settings on the transport ventilator to be used (do this before patient is switched to transport ventilator).
        i. IF unable to match the settings and there is a significant discrepancy, contact medical control for assistance.

e. Patient should already be on cardiac monitor and pulse ox prior to switching ventilators.
f. Depending on reason for transport and patients condition, IV access should be considered.
g. Have an Ambu-Bag and suction available for unexpected emergencies
h. Switch patient over to the transport ventilator and observe for any distress. It may take a minute or so for the patient to become accustomed to the new ventilator. If necessary, ventilate with an ambu bag for several minutes.
i. Closely monitor pulse ox, signs of labored respirations, chest rise for any signs of hypoxia/distress. Remove patient from ventilator and assist respirations with an Ambu-bag if there are ANY concerns or problems with ventilation after patient was switched to transport ventilator.
j. Once patient has been switched to the transport ventilator and is tolerating this well, then move patient over to the EMS stretcher for transport.
k. If alarm on ventilator sounds, immediately check patient. Reasons for alarm:
    i. Low Battery/power source: sounds when electrical supply to the ventilator is inadequate or the gas inlet pressure is low. It is corrected by restoring the proper power supply.
    ii. Low-pressure alarm:
        1. Leak or disconnection (reconnect or tighten connections)
        2. Cuffed tube may be leaking
        3. Check O_2 supply (self explanatory)
    iii. High-pressure alarm:
        1. Ventilator uses too much pressure to deliver the tidal volume
            a. Bronchospams
            b. Secretions in airway that increased the resistance/pressure in airway (suction airway)
            c. Kinks in ET tube (unkink tube)
            d. Biting on ET tube
            e. Coughing
            f. Gagging
            g. Breathing asynchronously or bucking the vent
            h. Alveolar over distention
            i. Improper ventilator settings (High or low tidal volumes, excessive rate causing stacking and auto PEEP) (Consult medical control for change)
            j. Water in the ventilator tubing (disconnect the tubing, empty water, reconnect tubing)
            k. Pneumothorax (notify hospital to set up for this if you are en route. If tension pneumothorax, go to that protocol)
            l. Patient anxiety (contact medical control for sedation order)
    iv. IF unable to identify the cause of the ventilator alarm and/or patient's condition deteriorates, disconnect from ventilator and assist respiratons via the Anbu-bag.
l. Upon arrival at the care facility, follow above steps when transferring from EMS stretcher to care facility stretcher. Report any problems to accepting staff.
m. Document vent settings used, vital signs, pulse ox, any changes in the patient's condition during transport.
n. Contact medical control during any of the above steps for assistance as needed.

B. Intubated patients not on ventilator

a. Ventilate patient with ambu-bag till ventilator can be set up
b. Patient should: be on Monitor, pulse ox, IV access, have suction available
c. Initial vent settings: (Call medical control as soon as possible to verify or assist with settings)
   i. FIO_2: 100% then titrate to pulse ox of 92 – 94%
   ii. Tidal Volume: 7 – 10 ml/kg
   iii. Be aware: If patient is a tight asthmatic, has severe COPD or has had prior lung surgery (partial lung removed), use smaller tidal volume (7 ml/ kg) and faster rate to maintain pulse ox.
   iv. Rate: Adult 10 – 12 bpm, Children 12 – 24, pre-school 20 - 30
   v. Pressure support: 10 cm H_2O (if available on ventilator)
   vi. Peep: 5 cm H_20 (if available on ventilator)
d. Monitor patient/vital signs/pulse ox for signs of adequate ventilations
e. If any distress or concerns, remove from ventilator and assist respirations with ambu-bag.
f. If ventilator alarm sounds, see steps above
g. En-route to hospital; notify the staff so they can have a ventilator set up on your arrival.
h. Document vents settings, vitals, pulse ox, patient response.

A. Introduction and Objectives
B. Protocol Review
C. LTV 1200 Ventilator Operations Overview

a. LTV Ventilator Applications

i. Adult, Pediatric & Infant (> 5kg)
ii. Invasive (ETT, King LT, Combitube or Non Invasive (Mask) Ventilation
iii. Emergency and Non-emergency Transport
iv. Long-Term Care
v. Rehab
vi. Homecare

b. Ventilator Setup

i. Making the connections

1. Left side panel overview
2. Right side panel overview
3. Oxygen Source
   a. The LTV 1200 can be used with either a 50-PSI (wall mount, no flow-regulator) oxygen source or with Low Pressure oxygen (from a Christmas-tree type connector).
   b. 50 PSI
      i. A 50 PSI Oxygen source allows you to use the internal oxygen-air blender to set a specific O_2% from 21-100%.
      ii. The 50 PSI source can be from a regulated oxygen cylinder or off the ambulance or hospital wall source.
      iii. An input O_2 of less than 35 PSI will cause an alarm.
   c. Low Pressure

i. Home ventilator patients may utilize low pressure oxygen (from a flow meter or oxygen concentrator) bled into the unit using a nipple adapter on the oxygen fitting on the unit.
ii. The "Low Pressure O_2 Source" button must be activated and the blender will no longer be active.
iii. An O_2 Input pressure of more than 35 PSI will cause an alarm.

4. Breathing Circuits
   a. LTV 1000
      i. Peep valve is present
   b. LTV 1200
      i. A head and moisture exchanger is usually used.
      ii. Swivel elbow, attaches to the ETT and is always used.
      iii. Exhalation port only, NO PEEP valve
         1. WARNING: if using an LTV 1000 Circuit on the LTV 1200, make sure that the in-line PEEP is set to "0"
   c. Adult vs Pediatric circuit connections
5. Power Sources
   a. External AC Adapter 120VAC/12VDC
   b. External Lithium Ion Battery 12V (3 hours)
   c. Internal Battery (1 hour)

ii. Turning the unit ON
iii. Adjusting the settings
1. Front panel overview
2. LTV 1200 Presets
   a. Presets are loaded in the LTV to facilitate the quick initiation of mechanical ventilation when operators with limited knowledge of the equipment must apply it.
   b. The preset values are simply recommended starting points and should be safe levels for most patients.
   c. Once mechanical ventilation is initiated, adjustments and changes should be made to meet the needs of the patient.
   d. The full range of ventilator settings is available to the operator, regardless of the preset used.
   e. Using Preset Vent Settings
      i. Turn the unit on and the word "SAME" will display in the Display of Monitored Data window.
      ii. If the vent is being used on the same patient for whom it was last used and the settings are the same, press the "Select" button
      iii. If **NOT** being used on the same patient, turn the Set Value Knob (dial) until "NEW" is displayed in the Monitored Data Window and then press the SELECT button.
      iv. If you select "NEW", the options of "INFANT", "PEDIATRIC" and "ADULT" are available. Turn the SELECT VALUE KNOB to choose the appropriate preset for the patient.

1. Infant: >5 kg and <10 kg
2. Pediatric: 10 to 40 kg
3. Adult: >40 kg

3. Changing Settings
   a. When there are variable settings or options:
   b. Press the button by the parameter or setting to be changed
   c. Turn the "Set Value Knob" clockwise or counterclockwise to the desired setting
   d. Press the parameter button again to confirm setting
   e. Before changing a setting, it helps to think of what is a normal setting for that value.
   f. Key LTV Ventilator Settings
      i. Rate: 0 to 80 bpm
      ii. Tidal Volumes: 50 to 2000 ml
      iii. Press. Control: 1 to 99 cmH_2O
      iv. I-time: 0.3 to 9.9 sec
      v. Press. Support: 0 to 60 cmH_2O
      vi. O2%: 21 to 100%
      vii. PEEP: 0 to 20 cmH_2O
4. Basic Operation – Mode selection
   a. ventilator setting box.
   b. Selections are made by pressing the Mode "Select" button. One push and the next mode selection flashes. A second push confirms the mode and makes the change.
   c. The NPPV mode (Non-invasive Positive Pressure Ventilation) is the Bi-Level setting for the LTV.
   d. A non-vented mask is necessary when applying NPPV with the LTV.
5. Basic Ventilator Settings
   a. Breath Rate, Tidal Volume, Pressure Control, Insp. Time, Pressure Support, O2 %, Sensitivity
   b. Set by selecting the parameter button, rotating the set value knob, and pushing parameter button again or waiting 5 seconds
   c. PEEP
      i. 0 to 20 cm H_20
      ii. LTV 1200 will add the pressure setting (if pressure is selected) to the PEEP. Be careful that you are not over-pressurizing the patient!
6. Other settings on the front of LTV
   a. "Insp/Exp Hold"
      i. This button is used to assess the lung compliance of the patient and determine if there is any air-trapping happening during ventilation.
      ii. You will not be utilizing this button.
   b. Manual Breath

i. This can be used to temporarily increase the ventilation for a patient
ii. Sometimes used after a stressful situation to help the patient "catch up" with their ventilation demands
iii. Manual breaths also send a burst of air through sensing lines to clear them of fluid/secretions

c. "Low O_2 Source"
i. This button must be selected when a low pressure O_2 source is used.
ii. You will be utilizing a high pressure source, so this option should not be On, or lit.

d. Control Lock
i. Pressing this button will lock the controls on the unit, so they may not be accidentally (or intentionally) changed.
ii. The indicator is lit when the controls are locked out.
iii. Simply press the button again to turn off the lock, thus allowing changes.
iv. If a quick push will not turn this on and off, press and hold the button for 3 seconds.

iv. Extended Menus
1. Accessed by pressing and holding the 'Select' button
2. The extended menu settings can be preset to standard and acceptable levels for most applications, then accessed only when necessary, by properly trained personnel.
3. O2 Cylinder Duration
a. Particularly useful for transport teams
b. Accessed in: Extended Features → Vent Op → O2 Cylinder Duration
i. Cylinder Type?
ii. Cylinder Pressure?
iii. Calculate >>>

v. Monitoring the patient
1. Measured and calculated values scroll through the monitor display
2. Press the 'Select' button to find to a desired parameter
3. Double click the 'Select' button to resume the automatic scroll
4. Airway pressure is dynamically displayed on the light bar above monitoring display
5. **Treat the patient, not the machine**
6. Troubleshoot, starting with the patient:
a. Look at your patient: distressed, moving, coughing, seizing, disconnected from vent, ???
b. Look at the vent alarm
i. which one is activated?
7. If patient is not being ventilated effectively, solve the problem quickly or ventilate manually (BVM)
a. If intubated, use the D.O.P.E pneumonic to assess ventilation problems:

i. D – Displaced
ii. O – Obstructed
iii. P – Pneumothorax
iv. E – Equipment

8. Providing Oxygen Flush
    a. Pressing and holding the O_2% button for 3 seconds will set the vent to deliver 100% oxygen (oxygen flush) for 2 minutes
    b. This can be used to pre-oxygenate or post-oxygenate the patient during suctioning or a stressful event
9. Suction/Clearing Secretions
    a. Utilize the suction catheter on the patient's existing circuit, or have catheter available
    b. May need to pre-oxygenate and/or post-oxygenate some patients using O_2% button
    c. Use of the 'Manual Breath' button will deliver a breath at set volume or pressure, and will also send burst of air through sensing lines to clear any fluid/secretions blocking ports
    d. Use of a "closed" suction system
        i. ATTACH SALINE BULLET, THEN UNLOCK THUMB VALVE
        ii. WHILE DEPRESSING THUMB VALVE
        iii. SET WALL SUCTION (120-140 mm Hg)
        iv. HOLD CONNECTOR WITH ONE HAND AND INSERT TIP OF CATHETER INTO THE ENDOTRACHEAL TUBE
        v. LAVAGE (DEPENDING ON PROTOCOL)
        vi. PASS CATHETER DOWN THE ENDOTRACHEAL TUBE (measured or until resistance)
        vii. DEPRESS THUMB VALVE ,WAIT FOR 1-2 SECONDS BEFORE *SLOWLY* PULLING THE CATHETER BACK (CONTINUOUS SUCTION)
        viii. WITHDRAW CATHETER UNTIL BLACK STRIPE IS VISIBLE IN SHEATH (*see arrow)
        ix. WHILE CONTINUING TO DEPRESS THE THUMB VALVE, FLUSH THE INSIDE OF THE CATHETER WITH 15 ML OF SALINE ** then release thumb valve**
        x. DISCONNECT SALINE, LOCK THUMB VALVE
        xi. CHANGE CATHETER EVERY 24 HOURS.
10. Alarm Settings
    a. Audible and visual alarm when parameter is violated.
    b. If situation is corrected, audible alarm will silence, but visual will stay lit until "Silence/Reset" button is hit.
    c. The monitor display will show the active alarm violation until reset.
    d. Ventilator Alarms
        i. High pressure limit
            1. Setting High Pressure Limit Alarm:
                a. Usually set within 10 cmH_2O above patient's average Peak Pressure.

b. When activated, ventilator will terminate breath and the patient does not receive full tidal volume

2. Causes of high pressure alarm violation:
   a. Resistance to gas flow:
      i. Kinks in tubing or monitoring lines
      ii. patient coughing
      iii. secretions
      iv. bronchospasm
      v. gagging, "fighting the ventilator"
   b. Decrease in lung compliance:
      i. Atelectasis
      ii. Pneumothorax
      iii. pulmonary edema

ii. Low airway pressure
1. Setting Low Pressure Alarm:
   a. 5-8 cmH_2O less than ventilating pressure
2. Causes of Low Pressure Alarms:
   a. Cuff Leak
   b. Vent Circuit
      i. Check tubing for holes or kinking
      ii. Check monitoring lines for tight fit or kinking (Leur connections can become loose)
      iii. Check connection at "Y" connector
   c. If using a Ballard suction device, check that all connections are secure (cap for saline port)
   d. Vent not meeting patient's inspiratory need (A/C)

iii. Low minute volume
1. **Minute volume** *= total volume of breaths over 1 minute time*
   a. e.g. 10 breaths per minute x 600 ml per breath = minute volume of 6000 ml or 6.0 L/min
2. Setting Low Minute Volume:
   a. Set 3 Liters under patient's minute volume, with a minimal setting of 5L/m.
   b. Ensures that adequate alveolar ventilation is maintained.
3. Causes of Low Minute Volume alarms:
   a. Neurological changes (A/C or PSV)
   b. Sedation issues (A/C or PSV)
   c. Patient fatigue (PSV)
   d. Decrease in compliance (PSV)

e. High pressure alarm active and ventilator dumps delivered tidal volume (A/C or PSV)

iv. Apnea

1. Apnea Parameters
   a. Activated when no exhalation is detected for a selected time period (e.g. 20 seconds)
   b. Tidal volume and pressure control level should be set appropriately for patient, as these will be used for apnea ventilation. *(These are the apnea back up, make certain that they are appropriately set.)*

vi. Turning OFF and processing the unit

c. Sequence of LTV Setup
   i. Connect breathing circuit to the LTV ventilator.
   ii. Make sure ventilator is connected to an adequate power source – battery, UPS, or AC-DC supply *(internal battery should only be used for short transport or during switch to alternate power supply).*
   iii. Connect oxygen source to ventilator (if ventilating at greater than 21% O_2).
   iv. Turn unit ON – UNIT SHOULD NOT BE CONNECTED TO THE PATIENT AT THIS TIME.
   v. Select the patient type (Adult, Pediatric, Infant) using the Presets in the LTV.
   vi. Make any necessary adjustments in the ventilator settings.
   vii. Set proper alarm limits, as appropriate for patient.
   viii. Check the low pressure, high pressure, and disconnect alarms before applying to the patient.
   ix. Connect LTV breathing circuit to the patient and closely monitor the patient.
   x. Utilize HME (heat and moisture exchanger) and closed suction catheter, if on patient circuit at facility.
   xi. Keep flow sensing lines up to avoid water and secretions in these lines.
   xii. Monitor your patient. Make appropriate adjustments, and CALL FOR HELP, if you're uncomfortable with what you see. Use the BVM if necessary.

d. Volume Control vs Pressure Control Modes
   i. ***Volume Control:*** Tidal Volume is controlled; the resulting pressure is based on the physical size of airways and lungs and the patient's lung compliance (stiffness).
   ii. ***Pressure Control:*** Ventilating Pressure is controlled; the resulting tidal volume is based on the physical size of airways and lungs and the patient's lung compliance (stiffness).

e. Ventilator Modes

i. Control Ventilation

1. Assist/Control button is illuminated
2. "Sensitivity" is set to a dashed line
3. Will provide only machine breaths at the set breath rate.
4. Given either to the pressure or tidal volume that you set.
5. No assisted breaths are given.
6. This mode is used in a patient with no respiratory effort of their own.
7. Control Settings
   a. Breath Rate

b. Tidal Volume or Pressure Control level
c. Inspiratory Time (or Peak Inspiratory Flow rate)
d. Oxygen %
e. PEEP
f. Sensitivity set to dashed line

ii. Assist/Control Ventilation

1. The "Asst/Ctrl" mode is illuminated and the sensitivity is set to a numerical value. (Typically around 3)
2. This will provide machine breaths at the set breath rate and will assist any patient initiated breaths. These will be either to the pressure or tidal volume that you set.
3. The patient may breathe faster than the set rate; the breaths will be measured and will be assisted in order to reach the set tidal volume or set pressure.
4. If patient ***DOES NOT*** have any spontaneous respirations, the patient will receive the set number of breaths at either the set pressure or the set tidal volume each minute.
5. If the patient ***DOES*** try to initiate a spontaneous breath, the patient will receive the set tidal volume or pressure.
6. Machine-initiated and/or patient-initiated breaths are *all* delivered at the set parameters (volume or pressure)
7. Assist/Control Settings
    a. Breath Rate
    b. Tidal Volume or Pressure Control level
    c. Inspiratory Time (or Peak Inspiratory Flow rate)
    d. Oxygen %
    e. PEEP
    f. Sensitivity set to a numerical value

iii. NPPV/Non-Invasive Pressure Support Ventilation

1. The patient's spontaneous breathing effort is supported to a set positive pressure from the ventilator
2. There are no mandatory breaths from the ventilator. Patient effort determines respiratory rate, inspiratory time, peak flow, and tidal volume
3. This Mode is used with a non-vented face mask on a non-intubated patient that must be breathing at an adequate rate.
4. Goals
    a. Overcome the work associated with moving gas through the artificial airway and circuit
    b. Improve patient/ventilator synchrony
    c. Augment spontaneous tidal volume
5. Basic NPPV Settings
    a. Select "Pressure" (vs. volume)
    b. Select "NPPV" mode
    c. Set "Pressure Support + PEEP" button
    d. Range 5-25 cmH2O (typical)
    e. Set "PEEP" (0 to 20 cmH_2O)

f. Set O_2% (21% to 100%)
g. Set Sensitivity
h. A non-vented mask must be used
i. This setting is used for a breathing patient that is not intubated

iv. <u>SIMV/Synchronized Intermittent Mandatory Ventilation</u>

1. This ventilation mode provides a mixture of mandatory (controlled) and spontaneous breath types.
2. The LTV will give and/or "sync" with enough breaths to achieve the set breathing rate; the patient may breath in between these mandatory breaths, but the "in between" breaths will not be assisted
3. <u>Basic SIMV Settings</u>
   a. Breath Rate
   b. Tidal Volume or Pressure Control level
   c. Inspiratory Time
   d. Oxygen %
   e. PEEP
   f. Sensitivity

v. <u>CPAP and PEEP</u>

1. ***<u>PEEP</u>*** = Positive End Expiratory Pressure
   a. a technique of assisting breathing by increasing the air pressure in the lungs and air passages near the end of expiration so that an increased amount of air remains in the lungs following expiration
2. ***<u>CPAP</u>*** = Continuous Positive Airway Pressure
   a. a technique of assisting breathing by maintaining the air pressure in the lungs and air passages constant and above atmospheric pressure throughout the breathing cycle
3. Function:
   a. Functionally, CPAP and PEEP do the same thing:
4. CPAP/PEEP is used to "splint" airway structures open (upper airways, alveoli).
5. CPAP also works to "recruit" collapsed or fluid filled alveoli. As a result of this recruitment, the usable surface area of the lung increases and allows for more efficient gas exchange.
6. <u>Basic Settings</u>
   a. Set "Breath Rate" to dashed lines
   b. Select the "SIMV/CPAP" mode
   c. Set the "PEEP" between 1-20 cmH_2O
   d. Typical PEEP for a pediatric would be around 4 cmH_2O
   e. Typical PEEP for an adult with COPD 8–10 cmH_2O
   f. Typical PEEP for an adult with CHF around 10+ cm H_2O

f. <u>Sensitivity</u>

i. Sensitivity determines when the ventilator will recognize a patient's own inspiratory effort. (A patient trigger)
ii. The LTV 1200 offers flow sensitivity.
iii. The ventilator delivers a low level of constant flow (10 lpm) into the patient circuit. This is called the "bias" flow.

iv. As the patient begins to inhale, some of this constant flow is diverted to the patient.
v. Change in flow in the vent circuit will cause the vent to "cycle" and deliver gas to patient.
vi. Sensitivity is usually set at 2-3 liters per minute in the hospital.
vii. May require slightly higher setting during transport in field to avoid "auto-cycling" of the ventilator.
1. Setting too low can cause auto-cycling of the ventilator (usually due to leak in system)
2. Setting too high can "lock out" patient from being able to "trigger" any spontaneous breaths

D. Key Points BEFORE Transporting
a. See how the patient is interacting with their current vent
i. If in PSV
1. Breathing Rate
2. Spontaneous tidal volumes
3. Minute Volume
4. Sensitivity
b. Talk to the patients therapist and nurse:
i. Secretions
1. (If already in place, keep the in-line suction device attached to the patient when you go)
ii. Weaning schedule or ventilator goals for this patient
iii. Any "Quirky" respiratory patterns
1. example: pt will breath 50 times per minute when he/she gets anxious.
2. See what relieves the "quirkiness" (changing modes, settings, favorite medication, reassurance, etc)

E. Key Points WHILE Transporting
a. Set alarms appropriately
i. if set appropriately, alarms can alert you to subtle changes before they become large problems.
b. Monitor patients vent parameters
i. Minute Volume (A/C or PSV)
ii. Peak Pressure (A/C)
iii. Tidal Volumes (PSV)
c. Anticipate what changes you would make if patients vent needs change?

VII. LAB VALUES

1. Basic Metabolic Panel

a. Chemistry panel, also known as the Basic Metabolic Panel which typically contains 8 values.
b. Also known as a Chem-7, Chem-8 or Chem-10 (depending on the number of components of the test).
c. The Comprehensive Metabolic Panel is the same as a Basic Metabolic Panel, but also includes liver function studies.
d. In clinical practice, the Chemistry panel is written as short hand using what is known as a **fish bone.**

i. The numeric readings for each value sits in the designated spot on the fishbone, which is quicker and easier than writing them all out with the associated word.
ii. Universal understanding of the components of the fishbone allows clinicians to efficiently communicate multiple lab values in written documentation.

2. iSTAT

a. An iSTAT is a handheld bedside blood analyzer that allows rapid analysis of many lab values including lactic acid, hematology values such as hemoglobin and hematocrit, Chemistry panel, Cardiac enzymes, Coagulation labs such as the PT/INR, arterial or venous blood gases and a blood pregnancy test.
b. For the purpose of this lecture, most of the lab values we will discuss are pertinent to the management of critical and emergency medical management of patients and we will focus on the most common lab values that will give you the broadest and most complete understanding of these measurements.

3. Biomedical Pathways

a. This lecture will include many pathways, which may seem overwhelming depending on your current level of understanding.
b. For the purpose of this lecture, it is important to understand the overall purpose of each pathway, which should give you a better understanding on a particular physiological process, but don't get caught up in the details. At least not until you grasp the overall concepts.
c. The Renin Angiotensin aldosterone system (also known as RAAS for short):
i. A system of different hormones that regulates blood pressure and fluid balance.
ii. Steps:
1. A drop in blood pressure decreases the amount of blood that flows to the kidney. This drop in blood pressure is sensed by specialized cells in the kidney known as the juxtaglomerular cells.
2. The kidney then activates and secretes a hormone known as Renin into circulation.
3. The liver secretes a hormone known as angiotensinogen, which circulates in the blood waiting for the signal from Renin to become activated.
4. Renin then converts Angiotensinogen to Angiotensin I.
5. The lungs contain an Enzyme known as Angiotensin-Converting Enzyme (or ACE for short). This enzyme is the target of the anti-hypertensive medications ACE-inhibitors such as Lisinopril. ACE converts Angiotensin I into Angiotensin II.
6. Angiotensin II is the main player in this pathway and has many functions:
a. First, it directly increases sympathetic activity. The fight or flight mechanism of the body.
b. Secondly, Angiotensin II acts directly on the renal tubules to increase the reabsorption of Sodium, Chloride and Water and at the same time, excretes potassium to maintain electrolyte balance.
c. This mechanism is also managed a second way. Angiotensin II stimulates the adrenal gland, that sits on top of the kidney, to secrete aldosterone, which has the same effect in electrolyte balance.

d. Angiotensin II also acts directly at the smooth muscle cells of blood vessels, causing vasoconstriction, which increased blood pressure.
e. Finally, Angiotensin II acts in the pituitary gland to secrete a hormone known as Anti-Diuretic Hormone (or ADH), which works in the kidney as well, but this time at the collecting ducts and reabsorbs water. So, it is true to its name in that it is the opposite of a diuretic, or an antidiuretic. Another name for ADH, is vasopressin, which I am sure you are all familiar with in its use as a medication given to help increase blood pressure.

7. As blood pressure increases, the increase in renal blood flow is again sensed by the juxtaglomerular cells in the kidney, and renin is no longer produced.

d. Fluid and Solute Shifts

i. Osmosis

1. The spontaneous net movement of solvent molecules, such as water, through a semi-permeable membrane into a region of higher solute concentration.
2. If the body, water shifts in and out of cellular structures based on the concentration of different solutes on each side of that membrane (Solutes include electrolytes such as sodium, or different proteins such as Albumin).
3. Keep in mind, with osmosis, the water molecules are small enough to pass through the cellular membranes, but the different solutes cannot.
4. With osmosis, water will always follow solute, until the concentration of solute is equal on both sides.

ii. Diffusion

1. The movement of molecules from a region of high concentration to an area of low concentration.
2. The gaps in the cellular membrane are large enough for the solute to pass, and therefore, they will move down their concentration gradient until an equal concentration is achieved on each side of the membrane.

4. Basic Metabolic Panel - FISH BONE

a. SODIUM

i. Sodium is the primary extracellular cation in the body.
ii. It regulates osmotic forces, it is involved in membrane transport systems, it helps regulate many chemical reactions as well as aid in acid-base balance.

iii. Na/K pump

1. The picture shown here is of a cell membrane. The Green box in the center is the Na/K pump. This is an enzyme that acts as a pump to pump Sodium out of cells while pumping potassium into cells. Both of these ions are moving AGAINST their concentration gradient, and this movement requires energy which is formed by cleaving a phosphate from the molecule ATP.

2. The movement of these ions gives energy for other transport systems that move amino acids, glucose and other nutrients into cells, it controls signaling within many cells, controls cell volume and controls neuron activity.
3. This small electrolyte shift is important to so many mechanisms in the body, that you can hopefully appreciate the importance that Sodium, Potassium and even phosphorus play in cellular function.

iv. Normal Range of Sodium
1. The normal range of Sodium is typically 138-146 mmol/L.
2. Values outside this range can have detrimental effects on many cellular functions throughout the body.

v. Hypernatremia (sodium that is too high)
1. Causes:
   a. Dehydration (the concentration of sodium is elevated in relation to the amount of water in the body).
      i. In the setting of dehydration, a decrease is blood pressure is sensed by the kidneys and the RAAS system is activated, increasing the absorption of water in the kidneys.
      ii. Further, the chemoreceptors in the brain sense a higher sodium concentration, and the pituitary will secrete ADH, causing more free water to be reabsorbed. It also activates the thirst centers, causing the patient to feel thirsty.
   b. Excess saline administration.
   c. Renal loss of water, such as aggressive diuretic use or Diabetes Insipidus.
2. Treatment:
   a. As clinicians, we can help this process by giving the patient free water. What that means is, we are giving water in the pure form, without sodium.
   b. If the patient can tolerate oral fluids, we can give them water to drink.
   c. It is not uncommon the amount of free water that they are deficient is quite large, in the 7-10-liter range.
   d. This deficit is a calculated value based on a patients gender, age, weight and sodium level.
   e. If they need more free water than they can drink, or they are unable to tolerate PO, we can give them free water through the IV. However, it is not safe to infuse free water into an IV. So IV free water is given in the form of Dextrose and water, or D5W.

vi. Hyponatremia (sodium concentration that is too low)
1. Causes:
   a. Excessive vomiting or diarrhea, where sodium is expelled from the body before it can be absorbed, or reabsorbed by the GI tract).

b. Certain diuretics
c. Inadequate salt intake
d. Drinking too much free water (as seen in diabetes insipidus)

2. Symptoms:
    a. Headaches, confusion, lethargy
    b. If the levels drop below 120 mEq/L, the patient is at risk for seizures, coma and death.
3. Treatment:
    a. Gradually increase in the sodium concentration by slow infusion of 0.9% normal saline.
    b. If sodium is corrected too rapidly, a devastating phemonenon called Central Pontine Myolinolysis can occur, where the myelin sheath on neuronal cells in the pons of the brainstem degenerates, and results in irreversible paralysis, difficulty swallowing, difficulty speaking and other neurological symptoms.
    c. A 1 liter bag of normal saline will increase sodium by about 1 mEq/L.
    d. Our goal in treatment is an increase in serum sodium by 1-2 mEq/L/hr in acute hyponatremia, and no more than 0.5 mEq/L/hr in patients with chronic hyponatremia to avoid central Pontine myolinolysis.
    e. If a patients sodium drops too low, and they start to have significant neurological changes and certainly if they develop seizures, the treatment is to give a higher concentration of sodium. This is done with 3% hypertonic saline given at 25-100 mL/hr with very close monitoring of rate correction.
    f. If seizures occur, treatment of the seizure with benzodiazepines should be attempted, but unlikely to respond until the sodium is brought up to a safer level, typically > 120 mEq/L
4. Correction of Na for Hyperglycemia
    a. Whenever hyponatremia is seen on laboratory evaluation, look at the glucose.
    b. Significant elevations in glucose will cause sodium concentrations to appear low, and this is actually a falsely low reading even though the actual sodium concentration may be normal.
    c. If you notice a significantly elevated glucose level (like in the hundreds) and the sodium is low, you must correct the sodium level with the following equation.

$$\text{Corrected serum sodium} = \underset{(\text{mEq per L})}{\text{sodium}} + \frac{1.65 \times (\text{glucose [mg per dL]} - 100)}{100}$$

i.

    d. *Alternate Method:*
        i. Typically, sodium will falsely read low by 1.6 mEq/L for every 100 mg/dL of glucose AFTER 100.

ii. Ex: if the patients' Sodium level is 129 and blood glucose is 300:
1. Add 1.6 for every 100 mg/dL of glucose AFTER 100.
2. So in this case, we use a glucose level of 200.
3. So we will have to add 1.6 twice (so, 3.2) to the patients measured sodium to get the corrected, and accurate sodium level.
4. Corrected Sodium for a glucose of 300 = 132.2 (round it to 132).

e. Even Easier Method

i. Download the app MDCalc, and you can just plug the numbers in.

b. POTASSIUM

i. The major INTRACELLULAR cation in the body.

ii. Along with sodium, it regulates intracellular volume, plays an important role in not only nerve conduction, but also contraction of skeletal and cardiac muscle cells and plays an important role in many metabolic processes.

iii. The normal range is 3.5-4.9 mEq/L.

iv. Hyperkalemia (concentration of potassium is too high)

1. Causes:
   a. Renal failure (potassium is no longer filtered or excreted through the kidneys)
   b. Excessive potassium replacement.
   c. Trauma - *remember that potassium likes to be inside of cells, so trauma causing massive tissue damage, such as a crush injury that causes large amounts of cellular damage can cause dangerously high levels of potassium to spill into systemic circulation.*
      i. Reperfusion syndrome - patients extremities are crushed and trapped by a large object, such as a fallen building or the dash of a car for a prolonged period of time, and when the object is removed and the blood that was trapped in the pinned extremity is released into systemic circulation, a toxic level of potassium reaches the heart resulting in cardiac arrest.
2. Signs and Symptoms
   a. Cardiac dysrhythmias
      i. Typically not seen until the potassium level reaches a level of about 6.5.
   b. EKG Changes
      i. Potassium level of 6.5-7.5
         1. Prolonging of the PR interval
         2. Shortening of the QT segment
         3. Peaking of the T waves.

ii. Potassium level of 7.5-8.0
    1. P-waves begin to flatten
    2. QRS begins to widen.
iii. Potassium levels greater than 8.0
    1. QRS segment begins to widen so much that it is difficult to appreciate a normal QRS segment, and it starts to become more of a sine wave appearance
    2. Rapidly degrades into v-fib and then asystole.

3. Treatment
    a. Get an EKG!
        i. If there are no significant EKG changes, and the potassium level is significantly elevated, the blood specimen should be redrawn prior to treatment as many factors can lead to a false reading. A prolonged tourniquet time while searching for a vein to start an IV, for example can cause a falsely elevated reading as cells leak potassium into the hypoxic arm, causing a higher local concentration that does not represent the true systemic concentration.
    b. Calcium (either gluconate or chloride)
        i. Always given first
        ii. Stabilizes the cardiac membrane
        iii. Does nothing to actually decrease potassium levels.
    c. Insulin
        i. Will drive potassium into cells, and this hides it.
        ii. This is only a temporizing measure, as it doesn't decrease the actual whole body level of potassium, only temporarily decreases the amount of potassium in systemic circulation.
        iii. Giving 10 units of insulin to a patient who is not hyperglycemic could be a big problem, so we always give glucose with the insulin when it is used for hyperkalemia.
    d. Sodium Bicarbonate
        i. Also helps to hide potassium in cells, providing a temporary fix.
    e. Inhaled albuterol
        i. Also helps to hide potassium in cells, providing a temporary fix.
    f. Lasix
        i. Sometimes used
        ii. Increases the amount of potassium excreted at the kidneys.
    g. Kayexalate
        i. Binds potassium in the GI tract, preventing its re-absorption.
        ii. This process is very slow

iii. Literature is mixed on its actually clinical efficacy.

h. Dialysis

i. Indicated for significant hyperkalemia and will actually rapidly remove potassium from the body.

v. Hypokalemia (serum potassium levels less than 3.5).

1. Causes:
   a. Over-diuresis
   b. Inadequate dietary intake of potassium
   c. GI wasting with diarrhea and vomiting
   d. Other metabolic conditions.
2. Signs/Symptoms
   a. Metabolic alkalosis
      i. potassium is lost as a compensatory mechanism in the kidney in exchange for sodium, which we will talk more about later. Remember that potassium is vital for cell depolarization.
   b. Confusion
   c. Drowsiness
   d. Weakness
   e. Fatigue
   f. More prone to arrhythmias.
   g. Ileus
   h. Nausea and Vomiting
3. Treatment
   a. Replacement of Potassium.
   b. Potassium is significantly more efficiently absorbed through the GI tract, than through IV administration.
   c. So, if the patient can take oral medications, we prefer this route.
   d. However, it may be beneficial to give potassium both PO as well as IV.
   e. IV potassium must be given over a long period of time to help minimize risks associated with the IV infusion and is typically given at a rate of 10 mEq per hour at a maximum of 40-50 mEq per treatment.
   f. There are dextrose containing potassium formulations, however, this should really just be avoided as it can cause an anaphylactic reaction in patients who have an allergy to corn products.
   g. For each 10 mEq dose of potassium given IV, will increase the serum concentration of potassium by 0.1 mEq.
      i. So if a patients potassium level is 3.1, we can estimate that giving them 40 mEq of IV potassium will increase their serum concentration back to 3.5.

ii. Oral potassium replacement is much less predictable, so we can't use this rule for estimating enteral potassium replacement

c. BICARBONATE (HCO3/CO2)

i. The normal range of Bicarbonate is 23-28 mmol/L.

ii. In the majority of the time, you will actually not find Bicarbonate listed on the chemistry panel, but it is there. That is because it is actually listed as CO2.

1. *In the body, most CO2 is actually being carried around in a molecule known as Bicarbonate, or HCO3*
2. *Therefore, a CO2 blood test is really a measurement of your blood bicarbonate level.*
3. *CO2 and HCO3 are used interchangeably when referring to the basic metabolic or chemistry panel.*
4. *It is important NOT TO CONFUSE this with the pCO2, which is the partial pressure of CO2 in the blood and important in blood gas and pH analysis. We will discuss that more later.*

iii. Blood pH is maintained by the bicarbonate buffer system.

1. The lungs and the kidneys are constantly involved in a tug-of-war to help maintain acid-base homeostasis.
2. CO2 reacts with water to form carbonic acid, which readily dissociates into bicarbonate and a hydrogen ion.
3. An increase in the formation of the molecules on any one side of this equation will result in a compensatory increase in the formation of molecules on the opposite side of the equation to keep blood pH at the physiological level of 7.35-7.45.
4. CO2 is actually safely carried by red blood cells in the form of bicarbonate and transported throughout the body and eventually to the lungs to participate in gas exchange.
5. **Response to acidosis**
    a. The body will compensate and do what it can to shift the equation to the right, in favor of bicarbonate formation which will buffer the blood and increase the pH.
    b. This is done by generation of new bicarbonate in the kidney and increased absorption of bicarbonate in the kidney.
    c. The equation can readily move from right to left and left to right, so the kidney will also excrete hydrogen ions to prevent further formation of carbonic acid.
    d. Further, the lungs help out by increasing the respiratory rate and blowing off more CO2.
6. **Response to alkalosis**
    a. The body will compensate and do what it can to shift the equation to the left, in favor of carbonic acid, and decrease the pH.

b. This is done by renal wasting of bicarbonate and decreased bicarbonate production by the kidney.
c. Hydrogen ions are saved at the kidney and the lungs decrease the respiratory rate to increase the amount of CO2 that is now present to allow for more carbonic acid production.

d. CHLORIDE

i. An abundant anion in the extra-cellular fluid.
ii. Mechanisms
    1. Moves in and out of cells with sodium
    2. Involved in many cellular transport systems
    3. Helps transport CO2 to RBC's

e. BLOOD UREA NITROGEN (BUN)

i. Ammonia is a waste product of amino acid catabolism and is quite a toxic substance to the body.
ii. Our body converts the toxic ammonia, to a less toxic substance such as urea nitrogen (Urea Cycle).
iii. Urea nitrogen that is produced is released into the blood where it eventually travels to the kidneys and is excreted in urine.
iv. We can measure the amount of urea nitrogen in the blood as an indirect measurement of how well the kidneys are functioning.
v. If the kidneys are not filtering the way they should, the serum blood urea nitrogen level will be increased.
vi. Azotemia (An elevation of BUN)
    1. However, this really not a specific lab value to completely assess renal function, as other things can make this value elevate such as heart failure, dehydration, and a high protein.
    2. UPPER GI Bleed
        a. Consider if you see levels greater than 30 mg/dL
        b. As blood enters the stomach, the protein components of blood, including hemoglobin and immunoglobulins are digested and just like any other protein, their amino acids are utilized with the same waste product of Ammonia, and eventually Urea Nitrogen.
        c. It is important to note that this is only seen with upper GI bleeds, and not lower GI bleeds as proteins are broken down to amino acids in this portion of the digestive tract.
vii. Clinically, BUN is used in conjunction with creatinine to assess fluid status.
    1. As intravascular volume decreases, so does the oncotic pressures that filter substances through the kidney.
viii. Mechanisms of Kidney Injury

1. Prerenal (an insult occurring before the kidney)
    a. Etiologies: Sudden drop in blood pressure, or dehydration will cause a decrease in the blood flow to the kidney, and will cause an elevation in BUN that typically is elevated significantly higher than the other 2 mechanisms of insult.
    b. Clinically, this is likely the most often utilized benefit of analyzing BUN.
    c. If the kidney injury is due to a prerenal cause the radio on BUN/Creatinine will typically be > 20.
    d. Treatment: IV fluids
2. Renal (direct insult to the kidney)
    a. Etiologies: Direct infectious process, an autoimmune disorder or toxic medication effect.
    b. The BUN will also increase, but not to the level that it is seen with a prerenal etiology.
    c. The ratio of BUN/Creatinine is typically less than 20.
3. Post-Renal (insult offering after the level of the kidney)
    a. Etiologies: Impedance of urine flow from a kidney stone blocking the urinary outflow.
    b. Again, BUN will elevate, but not to the level of that seen in a prerenal etiology.

f. CREATININE

i. Creatinine is a much more specific indicator of how the kidney is doing.
ii. Creatinine is a chemical waste molecule generated from muscle metabolism.
iii. It is transported through the bloodstream to the kidneys and most of this waste product is filtered out through the kidneys and into the urine.
iv. It is more specifically elevated in renal failure, or during a pathologic process that causes injury to the kidney such as profound dehydration and shock, which decreases blood flow to the kidney resulting in injury.
v. The normal value of Creatinine is patient specific, as patients may have chronic renal failure and their “normal” value may be outside of the range listed here.
vi. Clinically, it is the change in creatinine that we use to assess changes in renal function. However, in an otherwise healthy patient with no history of renal disease, their value is typically 0.6-1.3 mg/dL.
vii. Low creatinine levels aren’t typically of clinical concern, as the goal of the body is to get rid of this waste product anyways, but can be seen in pregnancy, starvation states and in patients after dialysis.
viii. AKIN Criteria
    1. Stage 1 AKI - Creatinine increases by more than 1.5 from the patients baseline or an elevation by more than 0.3 mg/dl.

2. So, if you don't remember anything else from this chart just remember that if the patients baseline

ix. Example

1. Cr is 0.7, and today it is 1.1, the patient is in an acute kidney injury.
2. Now lets tie in BUN. If the patients BUN is elevated, we look at the ratio of BUN elevation in relation to Cr elevation.
3. So if the BUN is 24, we divide that by 1.1, and we get a value of 21.8. This is greater than 20.
4. So if the clinical appearance of the patient fits intervascular volume depletion, the treatment for this patient will likely be to initiate IV fluids while other possible causes of renal injury are ruled out.

g. GLUCOSE

i. Glucose levels will change based on fasting vs fed states.

ii. Fasting blood glucose level is 70-105 mg/dL in a nondiabetic patient.

iii. Serves as nearly the only energy source for the brain.

iv. ***As blood glucose drops*** → pancreas promotes glucagon release → stimulates glycogen breakdown into glucose in the liver.

v. ***As blood glucose rises*** → pancreas secretes insulin which stimulates the uptake of glucose into tissue and cells and stimulates he storage of excess glucose as glycogen by the liver.

vi. **Hyperglycemia**

1. Symptoms
   a. Thirst
   b. Urinary output increases – due to increased water secretion
   c. Blurred Vision – due to fluid shifts in the eye
2. Treatment
   a. Normal saline – correct body fluid depletion
   b. Insulin – once hydrated
   c. Potassium – Insulin shifts potassium into cells, so much watch closely.
3. **Diabetic Ketoacidosis**
   a. Increased release of glucose by the liver, but also leads to release of free fatty acids and from adipose tissue which are converted to ketones to try to use as an energy source.
   b. Without insulin, the body cannot uptake glucose into cells.
   c. Ketones cause pH to decrease → metabolic acidosis.
   d. Bicarbonate system rapidly overwhelmed.
   e. Typically occurs in Type 1 diabetics, as they have a complete lack of insulin.
   f. Causes include infection, after exogenous steroid use or following a stroke or MI.
4. **Hyperglycemic Hyperosmolar Syndrome**

a. Seen with a relative insulin deficiency that leads to an increase in serum glucose.
b. Typically seen in Type 2 diabetics that still have some insulin secretion.
c. As insulin is still present in these patients, ketones are not typically produced as glucose can still be used by cells as a source of energy.
d. However, the glucose levels increase to a level that increases the overall serum osmolarity.
e. Typically, glucose levels are significantly higher than those seen in DKA, and not uncommonly greater than 900.
f. Ketones are not produced → no metabolic acidosis
g. Bicarbonate buffer system is not depleted
h. Causes: Infection, or other stress such as exogenous steroid use, stroke and MI.
i. The fluid deficit is usually much more profound than in DKA
j. Mortality is much higher than in DKA

vii. Hypoglycemia

1. Causes
   a. excess in insulin levels
   b. adverse effect of oral hypoglycemic
   c. Infection
   d. Liver failure (as glucose cannot be utilized from glycogen stores)
   e. Alcoholics (inadequate diet)
2. Symptoms
   a. Headache
   b. Dizziness
   c. Extreme fatigue
   d. Seizures
   e. Coma.
   f. Hunger.
   g. Shakiness, Tachycardia, Sweating (autonomic nervous system activated)
      i. Patients on beta blocker may not show these symptoms!
3. Treatment
   a. Glucose replacement with juice or oral glucose gel, paste or tabs if the patient can tolerate PO
   b. IV dextrose
   c. IM glucagon

h. ANION GAP

i. Helps identify causes of metabolic acidosis.
ii. Calculation: **$Na^+ – (Cl^- – HCO_3^-)$**

iii. Normal value = 12

iv. Causes

1. M – methanol
2. U – uremia
3. D – diabetic ketoacidosis/alcoholic ketoacidosis
4. P – propylene glycol
5. I – isoniazide/iron
6. L – lactic acidosis
7. E – ethylene glycol
8. S – salicylates (aspirin overdose)

v. Symptoms

1. Weakness, confusion, loss of consciousness and coma
2. Shortness of breath
3. Tachycardia, arrhythmias
4. Nausea, vomiting and diarrhea

i. CALCIUM

i. 80% of all calcium is bound to albumin in circulation.

ii. Ionized (free) calcium is the biologically active form

iii. Calcium plays many important biological functions, including building strong bones and teeth, clotting of blood, Sending and receiving nerve signals, muscle contraction and relaxation, cardiac contraction and in the release of many hormones from both the anterior and posterior pituitary in the brain.

iv. Normal level is around 10 mg/dL.

v. As blood calcium levels fall → Parathyroid gland PTH

1. Kidney → directly stimulate Calcium uptake in the kidney
2. Kidney → activates Vitamin D → increased calcium uptake by the intestines
3. Bones → stimulates calcium reabsorption from calcium stores in bones

vi. Hypercalcemia

1. Causes
   a. Hyperparathyroidism (excess PTH)
   b. Excessive Vitamin D intake
   c. Thiazide diuretic use (calcium sparing in the kidney)
   d. Adrenal insufficiency
   e. Certain types of cancers (multiple myeloma)
   f. Milk-alkali syndrome (tums ingestion)
2. Symptoms
   a. “stones, bones, groans and psychiatric overtones.”
   b. Kidney stones and kidney disease
   c. Bone loss – from Ca reabsorption and bone remodeling

d. Psychiatric disturbances and abdominal pain

3. Treatment
    a. IV fluids to flush calcium through the kidneys
    b. IV Lasix - a loop diuretic causing calcium wasting at the kidney

vii. Hypocalcemia

1. Ionized calcium levels below 4.6 mg/dL.
2. Causes
    a. Underactive parathyroid gland
    b. Vitamin D deficiency
    c. Renal failure
    d. Any disease process that causes calcium sequestration such as pancreatitis.
3. Symptoms
    a. Muscle spasms
    b. Convulsions
    c. Tetany
    d. Cardiac arrhythmias.
    e. EKG changes: **prolongation of the QT interval**.
4. Treatment
    a. Replace calcium.
    b. Calcium gluconate or calcium chloride IV.
    c. *Caution should be given to patients on Digoxin receiving IV calcium, specifically calcium gluconate, as there is a theoretical risk of causing calcium influx into myocardial cells resulting in a phenomenon known as "stone heart" resulting in malignant dysrhythmias.*

5. PT/INR

a. Prothrombin Time (PT)

i. Evaluates the coagulation factors **1, 2, 5, 7 and 10.**
ii. ***Prolonged in:*** liver disease, vitamin K deficiency or a coagulation factor deficiency.
iii. ***Decreased in:*** patients with high vitamin K intake, oral contraceptive use and in patients on hormone replacement therapy.

b. International Normalized Ratio (INR)

i. A calculated value based on the Prothrombin time
ii. We use this calculated value clinically to measure the therapeutic effects of the anticoagulant medication Warfarin (Coumadin).
iii. < 1.2: Normal in a patient not on Warfarin
iv. 2-3: Therapeutic range for patients with nonvalvular a-fib on Warfarin
v. 2.5-3.5: Therapeutic range for patients with mechanical heart valves on Warfarin
vi. Elevated INR

1. A patient with a supratherapeutic INR is a concern, as they are at a significantly increased risk for major bleeding event.
2. INR < 10 and there is no signs of bleeding, treatment typically includes holding the patients Coumadin and restarting it when the INR falls to therapeutic levels.
3. INR > 10 regardless of bleeding, the Coumadin should be reversed with oral Vitamin K, which serves as a cofactor for the coagulation factors previously listed.
4. Any sign of major bleeding should be treated more aggressively with IV Vitamin K PLUS either fresh frozen plasma or Prothrombin Complex Concentrate.

6. Cardiac Injury Profile (Troponin I, CK-MB, and other CK isoforms)

a. The enzymes are released into circulation as a result of injury to cardiac myocytes.

b. CK and CK-MB

i. Are not specific to cardiac muscles, but also found in skeletal muscle.
ii. Therefore, an elevation of these do not necessarily mean the cardiac myocytes are injured.
iii. Blood levels tend to peak earlier than Troponin in cardiac injury, but also normalize rather rapidly.
iv. Therefore, a patient who has a repeat coronary event with elevated CK isoforms should help with diagnosis

c. Troponin I

i. Is specific to cardiac cells.
ii. Typically takes up to 6 hours to show up in the blood after cellular injury, which is why these labs are often repeated several times in the emergency department.
iii. Troponin goes up after several hours but stays elevated for days.

7. ACID-BASE BALANCE AND BLOOD GAS ANALYSIS

a. Definitions

i. The body pH is strictly regulated within a narrow range of **7.35 and 7.45**
ii. **Acidosis (< 7.35)** = accumulation of acids → decrease in blood pH
iii. **Alkalosis (> 7.45)** = accumulation of a base → increase in blood pH
iv. **Acidemia** = term used when describing a blood pH < 7.35
v. **Alkalemia** = term used when describing a blood pH > 7.45
vi. It is possible to have a mixed process resulting in both an acidosis and an alkalosis

b. Physiology

i. Even minor changes in pH can have devastating effects on protein stability and many biochemical processes.
ii. Normal cellular metabolism constantly produces and excretes CO_2 into the blood.
iii. Buffer System

1. **$CO_2 + H20 \leftrightarrow H_2CO_3 \leftrightarrow HCO_3^- + H^+$**

2. This process is in an equilibrium, meaning all components of the left and right sides co-exist at all times and the concentration of any component is determined by the concentration of the components on the other side of the equation.
3. So, if there is an increase in the concentration of any component on one side, will shift the equation to the other side, leading to an increase in the concentration of all components on that side.
4. This equation is paramount in your understanding of acid base regulation in the body.
5. Continuous Carbon dioxide production by cells within the body drives the equilibrium to the right, resulting in the production of more hydrogen ions.
6. pH is a function of hydrogen ion concentration.
7. More hydrogen = higher acidity and lower pH.
8. Under normal ongoing metabolism, the blood is constantly being made more acidic.
9. The body compensates to keep blood pH at a steady state.
10. There are 2 mechanisms by which this is achieved:
    a. Lungs
        i. A decrease in central and arterial chemoreceptors leads to deeper and faster respirations and CO_2 is eliminated through expired air → less hydrogen is made → blood pH returns to normal.
        ii. This mechanism is fast, occurring in minutes to hours.
    b. Kidney
        i. Bicarbonate is reabsorbed, and Hydrogen ions are excreted in the urine.
        ii. The kidney controls blood pH by regulating the amount of hydrogen ion secretion and the amount of bicarbonate reabsorption.
        iii. This mechanism is slow, taking days to respond and compensate for changes in pH

c. Respiratory Acidosis

i. Due to retention of CO2 secondary to hypoventilation.
ii. Causes
    1. Primary lung disorders decreasing perfusion at the alveoli
    2. Respiratory depression from medications, neuromuscular or neurodegenerative diseases as well as with aggressive breath holding.
iii. Symptoms
    1. Depressed mental status from CO_2 narcosis.
iv. Metabolic Compensation
    1. Occurs at the kidney, which conserves more filtered bicarbonate
    2. By adding new bicarbonate into circulation and by excreting more hydrogen ions.
    3. Other smaller chemical buffer systems also assist taking up hydrogen ions, to take it out of circulation.

v. Treatment
    1. Treat the underlying cause
    2. NIPPV to help drive off CO_2

d. Respiratory Alkalosis

i. Due to a loss of CO_2 due to hyperventilation.
ii. Causes
    1. Fever
    2. Anxiety
    3. Hyperventilation
    4. High altitude with lower partial pressure of oxygen concentrations
    5. As a respiratory compensation for a metabolic acidosis, such as aspirin poisoning
iii. Symptoms
    1. Tachypnea, dizziness, confusion and seizures - *due to vasoconstriction from raised pH.*
iv. Metabolic compensation includes a
    1. Decrease in bicarbonate reabsorption
    2. Less hydrogen wasting at the kidneys
    3. Release of hydrogen ions from other smaller chemical buffer systems.
v. Treatment
    1. Slow the ventilatory rate.
    2. If the patient is hyperventilating, reassuring the patient and letting the patient breath into a paper bag may help.
    3. If the patient is on a ventilator, the breath rate can be turned down resulting in less CO_2 loss at the lungs.

e. Metabolic Acidosis

i. Due to a reduction in the plasma bicarbonate levels.
ii. Causes
    1. Secondary to diarrhea
    2. Due to consumption of other metabolic processes such as DKA with ketoacid formation, lactic acidosis from seizure or exercise.
iii. Symptoms
    1. Kussmaul's respirations - deep, slow and irregular respirations
    2. Tachypnea
iv. Respiratory Compensation
    1. Tachypnea – an attempt to increase the respiratory rate and blow off more CO_2
v. Metabolic Compensation
    1. Kidneys excrete more hydrogen ions and conserving more bicarbonate.
vi. Treatment
    1. Treat the underlying cause
    2. Sodium Bicarbonate may be given if the pH falls below **6.9**

f. Metabolic Alkalosis

i. Caused by an increase in Plasma bicarbonate WITHOUT change in CO_2

ii. Causes

1. Vomiting with loss of HCl
2. Diarrhea with loss of colonic secretions
3. Certain medications.

iii. Symptoms

1. Weakness
2. Neuralgias
3. Altered mental status
4. Seizures.

iv. Respiratory Compensation

1. Decrease in the respiratory rate to retain more CO_2

v. Metabolic Compensation

1. Kidneys conserve more hydrogen ions and excrete more bicarbonate

vi. Treatment

1. Treat the underlying cause
2. Potassium is likely to be low in patients with vomiting and diarrhea and should be replaced
3. Hydrochloric acid can potentially be given for severe alkalosis, and possibly with dialysis where a low bicarbonate dialysate is used

g. Blood Gas Physiology

i. Alveoli

1. As air is inspired into the lung and down the trachea, bronchi and bronchioles, it eventually ends up at the Alveoli where the gas exchange occurs.
2. pAO_2 is about **100 mmHg**
3. $pACO_2$ is about **40 mmHg**

ii. Pulmonary Artery

1. The pulmonary arteries carry deoxygenated blood from the heart to the pulmonary capillary beds where gas exchange occurs.
2. pAO_2 is about **40 mmHg**
3. $pACO_2$ is about **46 mmHg**
4. The higher partial pressure of carbon dioxide in the pulmonary artery in comparison to the lower partial pressure of carbon dioxide in the alveolus, favors movement of CO_2 down its concentration gradient and into the alveoli
5. Oxygen will also move from an area of higher concentration to an area of lower concentration, and this favors the movement of oxygen from the Alveolus and into the pulmonary capillaries.

iii. Pulmonary Vein

1. pAO_2 is about **100 mmHg**
2. $pACO_2$ is about **40 mmHg**
3. The pulmonary vein then pumps blood back to the heart, and it is then subsequently pumped via systemic arteries to target tissue.

iv. Target Tissue

1. pAO_2 is < **40 mmHg**
2. $pACO_2$ is > **46 mmHg**
3. Again, oxygen will move down its concentration gradient from an area of higher concentration to an area of lower concentration, and oxygen will move into the tissue
4. CO_2 will move down its concentration gradient from an area of higher concentration in the tissue, to an area of lower concentration in the capillary bed.

h. ABG Interpretation

i. **ABG Components**

1. **pH** - tells us if there is an acidotic or an alkalotic process going on.
2. **pCO_2** - tells us if the issue is the fault of the respiratory system.
3. **pO_2** - tells us if the patient has adequate oxygenation.
4. **HCO_3^-** - tells us if there pH imbalance is a metabolic issue.
5. **SpO_2** - another assessment of oxygenation, but much more prone to erroneous readings.
6. **Base Excess** - another way of measuring the metabolic component as it contributes to pH homeostasis.
   a. Normal Range: + 2 to – 2
   b. +2 means there is a lot of base present, favoring an alkalotic process, or compensation in the direction of an alkalotic process.
   c. – 2, means there is the lack of base within the blood that is available to participate in the buffer system.

ii. Buffer System Review

1. **$CO_2 + H20 \leftrightarrow H_2CO_3 \leftrightarrow HCO_3^- + H^+$**
2. 2 mechanisms by which this is achieved:
   a. Lungs
      i. A decrease in central and arterial chemoreceptors leads to deeper and faster respirations and CO_2 is eliminated through expired air → less hydrogen is made → blood pH returns to normal.
      ii. This mechanism is fast, occurring in minutes to hours.
   b. Kidney
      i. Bicarbonate is reabsorbed, and Hydrogen ions are excreted in the urine.
      ii. The kidney controls blood pH by regulating the amount of hydrogen ion secretion and the amount of bicarbonate reabsorption.

iii. This mechanism is slow, taking days to respond and compensate for changes in pH

iii. ABG Interpretation Steps:

1. **Step 1**: Give it a last name
    a. The pH will give you this
    b. Is it acidosis or an alkalosis. Is the pH up or down.
2. **Step 2**: Give it a first name
    a. Which part of the buffer system is broken and who can we blame for the abnormal pH.
    b. Important: Use the HCO_3 or CO_2 off of the Basic Metabolic Panel (venous specimen) not the actual ABG for this part.
        i. CO2 combines with water to give carbonic acid, which quickly dehydrates to bicarbonate and hydrogen ions. So, a measurement of bicarbonate is also a measurement of total body CO2. Do not confuse this with the partial pressure of dissolves Carbon dioxide gas that is measured on the ABG. Don't overthink this part. For now, just know that the pCO2, or partial pressure of carbon dioxide indicates respiratory involvement, and an elevation favors acidic Hydrogen ion formation where the CO2 on the venous drawn basic metabolic panel is the bicarbonate measurement, indicates metabolic involvement, and an elevation favors a basic environment with more bicarbonate.
    c. Always start by looking at the pCO2 FIRST.
        i. Is the pCO2 abnormal, and does it fit with the pH?
            1. If YES, the PRIMARY issue is the fault of the respiratory system
                a. Respiratory Acidosis = low pH and a high pCO2
                b. Respiratory Alkalosis = high pH is high and the low pCO2
            2. If NO, THE PRIMARY issue is the fault of the metabolic system
                a. Look at the bicarbonate
                b. Metabolic alkalosis = high pH with a high HCO_3^-
                c. Metabolic Acidosis = low pH and a low HCO_3^-
            3. ROME: Respiratory Opposite and Metabolic Equal
3. **Step 3**: determine if compensation is present.
    a. Once you determine if the primary problem is respiratory or metabolic, look at the other value (the pCO_2 or HCO_3) that is not part of the primary problem.
    b. Is it within normal limits, or is it abnormal?
    c. If it is abnormal, does it move in the opposite direction that you would expect for the pH?
    d. If so, there is compensation by that system.

4. **Step 4**: ensure that the paO_2 and SpO_2 is adequate and not abnormal.
    a. Deoxy-hemoglobin dissociation curve.
        i. *Shift to the left*
            1. You won't see a drop in the pulse oximetry reading until the oxygen concentration in the blood is extremely low.
            2. Causes: High pH, alkalosis, hypothermia
        ii. Shift to the right
            1. Higher concentration of O_2 in the blood at the time the pulse Oximeter reading starts to fall.
            2. Causes: low pH and hyperthermia.
    b. RBC Physiology
        i. Hemoglobin
            1. 4 subunits (2 alpha and 2 beta)
            2. Each subunit contains a Heme group
            3. An oxygen or carbon dioxide molecule can bind to each of these heme compounds
        ii. **Met-hemoglobin**
            1. Contains Fe_3^+ → cannot bind O_2
            2. *Causes: nitrates found in foods, or medications such as lidocaine, sulfonamides, dapsone, benzocaine, pyridium and nitrates can cause a higher concentration of methemoglobin.*
            3. Causes a change in the Hgb protein → causes the normal heme groups to have a much higher affinity for oxygen → cannot release bound O_2
            4. SpO_2 will tend to read in the mid 80's as this is measuring the amount of oxygen being transported around in the blood.
            5. Though, the patient can look profoundly cyanotic. This is because the oxygen that is present in the blood, is not able to be released from the red blood cells at target tissues, so the patient gets cyanotic despite a modest decrease in SpO2.
            6. Treatment:
                a. Methylene blue
        iii. **Carbon Monoxide**
            1. CO molecules can also bind to the heme sites on the hemoglobin protein.
            2. This forms a much more stable complex with the heme group than oxygen does
            3. SpO_2 will still read normal as all heme sites still occupied
            4. Treatment:

a. High flow O_2 in hopes to outnumber the CO molecules and regain their sites on the RBCs

c. Assessment of perfusion

i. Hypoxemia:
   1. determined by PaO_2
   2. Low oxygen concentration measures in a blood sample.

ii. Hypoxia
   1. determined by SpO_2
   2. Low concentration of delivered oxygen to tissue.

iii. We can use these two readings to determine if perfusion is adequate.

iv. PaO2 = FiO_2 X 5.5

1. *I typically just use 5, as it will get you close enough and easier to calculate in my head.*
2. FiO2 is the concentration of oxygen in the air we breathe.
3. Normal room air is made up of 21% oxygen. So if we multiply 21 x 5, we get 105. So, a normal PaO2 in someone breathing room air is about 105 mmHg.
4. Determining FiO_2
   a. Room air 21
   b. Nasal Cannula
      i. 1 L = 24
      ii. 2 L = 28
      iii. 3 L = 32
      iv. 4 L = 36
      v. 5 L = 40
      vi. 6 L = 44
   c. Face Mask
      i. 6 L = 35
      ii. 7 L = 41
      iii. 8 L = 47
      iv. 9 L = 53
      v. 10 L = 59
   d. Non-Rebreather
      i. Add 10% for every Liter of flow
   e. Venti mask
      i. FiO2 is dependent on the adapter piece and the flow rate.

Lesson Plan: EPIC - Online

VIII. CALL OVERVIEW / PATIENT CARE REPORTS

1. Why do we document?
    a. Documentation is a continuation of patient care
    b. You are your patient advocate
    c. 5 Critical Areas
        i. Clinical: For the record
            1. Records assessment and patient care.
            2. Informs the ED or transferring Physicians and Nurses about the scene, condition of patient when found, assessment and treatment.
            3. Becomes part of patients medical record.
            4. Plays role in subsequent treatment in an ED.
            5. Can be held negligent in your documentation.
            6. QA and QI processes.
        ii. Legal:
            1. Your PCR is a legal document
            2. You are responsible for what is or isn't in your report. If you didn't document it, you didn't do it
            3. First thing reviewed in alleged malpractice suits.
            4. Should be written at or as close to time of incident.
            5. Reflects the standard of care provided and can help avoid a case against EMS if done well.
            6. Cases usually months/years after call. Helps remember what happened.
            7. Proper documentation is your best defense against lawsuits of question of your care
            8. The PCR is part of the patient's permanent medical record.
                a. The patient will have their own medical file
                b. Other healthcare providers will be able to access your medical records
                c. Reports should include subjective statements from the patient and cannot include bias
        iii. Operational: Data Drivers
            1. Track performance measurements such as response times, call-to- intervention times, on-scene times, transport times and other such assessments.
            2. Data used for policy making, staffing, deployment, peak-demand utilization.
            3. QA/QI, training and continuing education.
            4. Many states now require EMS agencies to submit data to their state EMS office. This is used to improve the EMS system as a whole
        iv. Financial: The Bottom Line
            1. Role in billing and reimbursement.
            2. Medicare makes payment decision.
        v. Compliance: Following the Law
            1. Verifies The Organization is operating with all applicable contracts and local, state or federal laws.
2. Tips for good documentation

Lesson Plan: EPIC - Online

a. Notebook
    i. Carry a small notebook
b. History
    i. Gather all information on scene before you leave
c. Complete
    i. Ensure a fully complete PCR. Incomplete or unchecked boxes invite questions.
    ii. Don't leave sections blank, fill in all the section blanks. If not applicable, write N/A
d. Accurate
    i. The narrative must be an accurate reflection of what happened. It details the accounts from when EMS arrives on scene to when the patient is turned over to the next higher level of care. Where found: surroundings, and assessments.
    ii. If you don't write it down, you didn't do it or it didn't get done.
e. Spelling
    i. Don't be misled that if you don't write it, they can't get me. False!
    ii. Poor spelling multiple times relays a sense of ignorance or apathy. Illegible raises questions.
f. Standards
    i. When in doubt reference national standards.
g. Vitals
    i. Need multiple set of vitals to determine if patient is stable.
    ii. Blood pressure by palpation provides incomplete information about patient's perfusion.
    iii. Repeated vital signs are rarely the same.
h. Corrections
    i. If you need to correct something, make only one line through it and initial it
i. Never:
    i. Document with pencil
    ii. Scratch something out
    iii. Use white out

3. Writing the narrative
    a. Your goal is to paint a picture with your words
    b. Place the emergency physician in your shoes at the scene
    c. Include the MOI
    d. Include spoken accounts
        i. Indicate who made the statement and use quotation marks around the exact statement
    e. Exclude: Your feelings, nonessential information or wordy statements
    f. Report any treatment and the patient's response to it.
    g. Use: SAMPLE, OPQRST, AVPU
    h. Know your agencies preferred narrative method
        i. CHART Method
        ii. DCHART Method
        iii. SOAP Method
        iv. Chronological Order
    i. DCHART Method
        i. One of the most common ways to write a narrative
        ii. Helps you find a pattern in your PCR's and remember everything
        iii. D – Dispatch Information

1. The dispatch information given to the crew at the time of the call
2. EMD Codes
3. Specific/Specialized instructions

iv. C – Chief Complaint
1. What the patient, family member, caregiver tells you is wrong with him or her. If no info, use the info from dispatch. Your general impression upon arrival.

v. H – History
1. Include HPI, PMH
2. Utilize OPQRST, SAMPLE and AEIOUTIPS
3. Any statement that the patient made about the current event and past pertinent events

vi. A – Assessment
1. Include any information about any assessment that others may have done as well
2. Primary and secondary survey
    a. ABCD
    b. DCAP-BTLS AND OPQRST
    c. Include patient negatives
    d. Include info obtained via: EKG, pulse ok, blood glucose, etc.

vii. R – Rx
1. Interventions given
2. Document procedures, who it was done by, and the time
3. The time, dosage and route of administration
4. Must have time stamp for
    a. Advanced procedures
    b. Medications administered
    c. Vitals

viii. Transport
1. Where you transported to and whether or not you used lights and sirens
2. All treatment done en route to the hospital
3. Any change to the patients condition
4. To whom you transferred the patient and what position they are (MD, RN, etc.)

4. Special Circumstances
    a. Refusal of care reporting
        i. Studies show anywhere from 50-90% of EMS lawsuits result from patients who were evaluated by paramedics and not transported to the hospital
        ii. Proper documentation is your best defense
        iii. If you feel like the patient needs medical attention, make every effort to persuade them to go to the hospital and contact OLMD to speak with patient if they still refuse.
        iv. Competent, adult patients have the right to refuse care
        v. Document every effort in care made, and the patients response
        vi. Know and understand patient rights
        vii. The patient should know
            1. His or her current situation
            2. Consequences of refusal of care
        viii. Information must be given to the patient in a language they understand

ix. Documented on the PCR and witnessed by an observer
x. Documentation should include
    1. Any assessment done and its finding
    2. Any advice you or the MD gave the patient
    3. Persuasive efforts
    4. Patient statement
    5. At least 1 fill set of vital signs and glucose, if able
    6. Clinical information that clearly indicates that the patient is capable of making an informed decision
    7. A release of liability form
    8. Signatures by the patient and a witness (preferably a relative or bystander)
    9. That you have informed the patient that they may call 011 again at any time and that you informed the patient what to look for if his or her condition worsens
xi. Initialed and signed by the patient
xii. The refusal documentation should clearly show:
    1. The process you went through
    2. How the process is documented
    3. Who witnessed the process
xiii. Unresponsive patients may be treated under implied consent.
    1. Be familiar with individual state laws related to consent.
    2. Confirm every effort is made to ensure patient's best interests.

b. Dead on Arrival
    i. Document the following:
        1. Pulseless and apneic
        2. Pupils fixed and midpoint
        3. Dependent lividity
        4. Presence of rigor mortis
        5. The beginning of decompensation
        6. General body temperature (cold or still warm)
        7. Obvious irreversible signs of death (decapitation)

c. MVCs
    i. Approximate rate of speed if known
    ii. Type on impact (frontal, rear, lateral rollover, passenger side or driver
    iii. Type of vehicles involved
    iv. Intrusion to the passenger compartment
    v. Presence of hazardous material
    vi. Use of seat belts
    vii. Deployment of airbags
    viii. Spider webbing or broken windows
    ix. Deformed steering wheel
    x. Broken brake or gas peddles
    xi. Deformities in dash board
    xii. Use of child safety seat
    xiii. Possible loose projectiles
    xiv. Death of another in the same passenger compartment

xv. Entrapment
xvi. Lengthy extrication
xvii. Emergency lifting or carrying techniques

5. Patient Assessment
    a. **Head Exam**
        i. *The Head and Neck Exam is kind of long, but it can be remembered the following way: (1) check for new holes, (2) check the old holes (ears, eyes, nose, mouth/speech), (3) check the face, and (4) check the neck.*
        ii. **Assess for facial and cranial lesions (DCAP-BTLS)**
            1. Check in all neurological and trauma patients.
            2. DCAP-BTLS = Deformities Contusions Abrasions Punctures/Penetrations Burns Tenderness Lacerations Swelling. This is an American acronym and it's useful for students because it proves they know what they are looking for, but once you graduate you can use the all encompassing term 'lesions', which is more concise.
            3. Check for Battles' sign (mastoid ecchymosis) and Raccoon's Eyes (bilateral, periorbital ecchymosis) in head injury.
            4. Be aware that Battles' sign usually occurs DAYS after an injury. If you're pulling someone from a car or other traumatic injury and you discover Battle's sign, you must consider <u>previous</u> trauma. Were they in another accident a few days ago?
            5. Racoon's eyes usually happens HOURS after an injury, so again, you have to wonder how they got that if you're at a 'fresh' trauma.
            6. Technical point: it's not "Battle's" sign, it's "Battles' sign" (note the apostrophe). This is because it's not a 'sign of battle' (which is what most people think), instead it's named after a physician named 'Battle' who first described it.
        iii. **Assess if pupils are equal and reactive to light (PEARL)**
            1. Check in all patients, but use caution in patients who have been having recent seizures (because you might elicit another seizure by flashing a light in their eyes).
            2. Report if unequal, or report as a pertinent negative in the traumatic and neurological patient.
            3. 25% of the population have naturally unequal pupils ("anisocoria"), otherwise the pupils should be equal and reactive. The best way to tell if a patient has anisocoria is to ask them (or someone who knows them, if they can't answer). I don't know of any way to tell just by physical exam.
            4. Be aware that if you shine light in one pupil, both will react.
            5. Please don't use very bright lights to test this. That's pretty uncomfortable for the patient. In this day and age of LED flashlights you can really blind someone. Test your light - if it's too bright for your eyes, it's too bright for theirs.
            6. Generally, one blown and dilated pupil indicates head injury (stroke or trauma).
            7. Bilaterally dilated, non-reactive pupils indicates brain death. However, in the cardiac arrest patient who has received epinephrine and atropine the pupils will be dilated and non-reactive. This does not *necessarily* mean they are brain dead! (although they might be) It could be drug effect. Keep that in mind.
            8. Pinpoint non-reactive pupils generally indicates narcotic overdose, but there are some synthetic narcotics that do NOT cause this, so don't use normal pupils to rule out narcotic overdose.

9. Bilaterally dilated pupils that DO react are often due to fear, dark ambient light or non-endogenous sympathomimetics

iv. **Assess pupils for midline, conjugate gaze**
   1. Check in all patients.
   2. Report as a pertinent negative in the traumatic and neurological patient.
   3. To assess this, hold up one finger and draw a capital 'H' about 60cm (2 feet) or so in front of the patients face. Ask them to follow your fingerS
   4. Be aware that some patients do not have conjugate pupils. If the patient has a dysconjugate gaze, ask them if this is normal.

v. **Assess pupils for nystagmus**
   1. Check in all patients.
   2. To assess this, hold up one finger and draw a capital 'H' about 60cm (2 feet) or so in front of the patients face. Ask them to follow your finger.
   3. Many drugs can cause nystagmus (barbiturates, benzodiazepines, alcohol, lithium, phenytoin) as well as thiamine deficiency (vitamin B1 deficiency - common in alcoholics, best not to give these patients dextrose until you've given thiamine).
   4. MS, strokes and decompression sickness can also cause nystagmus.
   5. As a general rule though, horizontal nystagmus (side to side) and orbital nystagmus (going in circles) is usually due to toxicological causes (think alcohol) and vertical nystagmus (up and down) is due to CNS pathology. I remember this by thinking 'look sideways to the bottle, and look up to the brain'. Maybe I'm crazy, but it works for me. :-)

vi. **Assess for Ottorrhea/Rhinorrhea & Ottorrhagia/Rhinorrhagia**
   1. Check in all neurological and trauma patients.
   2. These mean: ear/nose fluid & ear/nose blood. If you're having trouble remembering the trouble between Otto meaning 'ear' and Rhino meaning 'nose', just remember that Rhinoceroses have big noses (rhino is greek for nose and keras is greek for horn).
   3. Report if positive, or report as a pertinent negative in the traumatic patient.
   4. Whenever there is ottorhagia (ear blood), perform the 'bulls-eye test'. To do this, put some of the blood on a white or pale sheet. If a pinkish centre with a clear fluid area surrounding it forms on the sheet you should assume that there is CSF in the fluid. This is a very ominous sign. I read an article not too long ago stating that this test might not actually be as effective as we are all taught, so take it with a grain of salt. Certainly, don't rule out the presence of cerebrospinal fluid based on a negative bulls-eye test.
   5. In the non-traumatic patient rhinorrhagia can be a 'sentinel bleed' in occult hypertension, so 'nose bleed = check blood pressure'.

vii. **Asses for speech deficits or abnormalities**
   1. Check in all speaking patients, but especially cardiac, neurological and trauma patients.
   2. If in doubt, ask the patient to repeat a simple phrase such as 'the sky is blue today'
   3. This is an important indicator of stroke, if it is present check for ptosis and other facial asymmetry as well as unilateral paralysis or weakness (check pronator drift). These tests are the three elements of the Cincinnati Prehospital Stroke Exam.

viii. **Assess mucous membranes for moistness and color**
   1. Check in all patients, especially the medical and/or dehydrated patient.
   2. In the medical and/or dehydrated patient also check skin turgor.
   3. Mucous membrane colour is especially important to check in patients with dark skin, when pallor may not be noticeable in the skin, check the inner lining of the eye lids or the inside of the lips.

ix. **Assess for oral trauma (bitten lips, cheeks or tongue)**
   1. **C**heck in all neurological, cardiac and trauma patients.
   2. This is a common finding in patients who have seized (they tend to chew up the inside of their mouthes while seizing).

x. **Assess for ptosis and other facial asymmetry**
   1. Check in all neurological and trauma patients.
   2. This is an important indicator of stroke, if it is present check for speech deficits and unilateral paralysis or weakness (check pronator drift) again, this is the Cincinnati Prehospital Stroke exam.

b. <u>Neck Exam</u>

i. **Assess for JVD**
   1. Check in all cardiac, neurological, respiratory and trauma patients.
   2. Be aware: most people who are lying down will have significant JVD (try it and see). This is important to know because if a patient is lying down and they do NOT have significant JVD then this is an abnormal finding.
   3. Ideally, JVD should be assessed when the patient is lying at a 45 degree fowlers position.

ii. **Assess for tracheal deviation**
   1. Check in all cardiac, neurological, respiratory and trauma patients.
   2. Report as a pertinent negative in all respiratory/trauma combination patients.
   3. To assess this properly you must use palpation (not just visualization!). Put a finger on either side of the suprasternal notch and feel to make sure that the trachea is evenly between both fingers. Assessing for tracheal deviation *visually* (only) will result in a failing mark for this assessment if you're doing it in front of me.

iii. **Assess for cervical vertebral body deviation & tenderness**
   1. Check in all neurological and trauma patients.
   2. The cervical bodies should be midline and non-tender.
   3. Practice assessing this on classmates and other healthy people to get the feel of it.

iv. **Assess for cervical muscle spasm & tenderness**
   1. Check in all neurological and trauma patients.
   2. The cervical muscles (the bands on either side of the cervical bodies) should be soft and non-tender.
   3. Practice assessing this on classmates and other healthy people to get the feel of it.

c. **Chest Exam**

i. **Assess for lesions (DCAP-BTLS)**
   1. Check in neurological, respiratory and trauma patients.
   2. DCAP-BTLS = Deformities Contusions Abrasions Punctures/Penetrations Burns Tenderness Lacerations Swelling.

3. Be alert for subcutaneous emphysema, this is a sign that somehow the pleura surrounding the lung have ruptured and air is escaping from the lung parenchyma into the musculature.

ii. **Assess chest wall respiratory movement**
   1. Check in all patients.
   2. Check for symmetry, and paradoxical or diminished movement. This is often an indicator of broken ribs (as in a flail segments

iii. **Assess Anterior/Posterior ratio**
   1. **C**heck in cardiac and respiratory patients.
   2. 'Barrel chested' patients have a higher incidence of cardiac and respiratory problems.

d. **Respiratory Exam**
   i. Note: perform this in all patients. You should be very good at this!
   ii. **Assess for Accessory muscle use**
      1. Report as a pertinent negative in the respiratory patient
   iii. **Assess Breath sounds**
      1. Report as a pertinent negative in the respiratory patient
      2. **Pattern**: (Eupnea, Hyperpnea, Tachypnea, Bradypnea, Cheyne-Stokes, Biots, Kussmauls, Apneustic)
      3. **Adventitia**: (coarse/fine crackles, ronchi, wheezes, stridor, rubs)
      4. **Locations**: (basilar, mid-scapulary, apices)
      5. **Symmetry**: (compare and contrast left and right sides)
      6. **Phase**: (inspiratory, expiratory or both)
   iv. **Assess Color and condition of skin**
      1. If the patient appears pale, ask friends/family if this is normal (if possible)
      2. Assess mucous membranes for colour in the dark-skinned patient
   v. **Assess word Dyspnea**
      1. How many words can they say per breath?
      2. Less than ten words is worrisome, less than five is critical.
      3. This is probably the single best 'field test' of someone's ability to breathe, so learn to pay close attention to it.
   vi. **Assess for Extending position**s
      1. i.e. tripoding, orthopnea - both of which indicate real difficulty breathing.
   vii. **Assess SpO2 using Finger probe**
      1. CO poisoning will produce a falsely high SpO2 reading (100%), so do not use in patients with smoke inhalation (or, at least, be cautious with your findings. If you pull a patient with obvious smoke inhalation injuries who is not responding to pain out of an enclosed, smoky environment and get a sat of 100%, don't blithely assume that everything is fine in terms of PaO2!
      2. Key points…
         a. Large changes in the Sao_2 occur with small changes in the Pao_2.
         b. Pulse oximeters are generally accurate between 80% and 100%.
         c. < 80 %, large changes in Pao_2 occur with small changes in Sao_2.
         d. Sao_2 values at or below 96% have been shown to be 100% sensitive for detection of hypoxia (Pao_2 < 70 torr).

3. Limitations
    a. Will Read falsely high in patients with CO2 and Methemaglobinemia
    b. Tells you nothing about *pH*
    c. Tells you nothing about *$Paco_2$*
    d. Tells you nothing about *ventilation*

viii. **Assess end tidal CO2 Gas values and waveforms**

1. Check if trained, and if the patient is in respiratory distress.
2. This is a GREAT tool and (IMHO) should be used not only on any intubated patients, but in any patient who has SOB.
3. *= measurement of CO2 during Expiration*
4. *$ETco_2$ is usually 2 to 5 mmHg < $Paco_2$ due to the dilution of the end-tidal gases by physiologic dead space gas.*
5. *VQ mismatch ratios (including pulmonary embolism), cardiac arrest, hypovolemia, obstructive lung disease, and the lateral decubitus position can widen the Pa-$ETco_2$ gradient → still valuable in trending values*
6. Key Points
    a. Capnography is the earliest indicator of airway or respiratory compromise.
        i. *It displays an abnormally high or low $ETco_2$ 5 to 240 seconds before pulse ox changes.*
    b. Low $ETco_2$ (hypopneic hypoventilation)
        i. *Commonly seen with sedative-hypnotic agents (especially propofol) and during deep sedation*
        ii. *Represents low–tidal volume breathing and should not be misinterpreted as hyperventilation*
    c. More sensitive than clinical assessment of detection of apnea.
        i. *Soto and colleagues found that during procedural sedation, 26% of patients experienced 20-second periods of apnea that were detected by capnography but not by the clinicians*
7. False elevations
    a. Esophageal intubation after bag or mask ventilation
        i. *Detection of $ETco_2$ usually ceases after six breaths.*
    b. Ingestion of carbonated beverages or antacids.
    c. Falsely elevated for 5 to 10 minutes after injection of sodium bicarbonate.

6. Lack of pulmonary blood flow during cardiopulmonary arrest.
    a. **Heart Rate**
        i. Check in every patient (as a part of vitals), dyspnea usually results in tachycardia, unless they've really crashed - then they can be bradycardic (maybe sinus brad from hypoxia, or an agonal rhythm).
    b. **Assess the inspiratory/expiratory ratio ("i/e ratio")**
        i. Normally it takes twice as long to breath out as it takes to breath in (remember, exhalation is passive), so the normal i/e ratio is about 1:2. This changes depending on the disease processes present. In asthmatics (who have trouble breathing out) the i/e ratio becomes more like 1:3 or 1:4, with long, laboured, wheezy expirations (try it, if you've got field experience this will sound familiar). In pulmonary edema it's the opposite. It takes work to get air into the fluid filled alveoli, so breathing in is difficult and the i/e ratio can change to 2

or 3:1. So paying attention to the i/e ratio (which you can see from across the room) is a good clue to what is happening in your patient.

7. Abdominal Exam
    a. **Assess for lesions (DCAP-BTLS)**
        i. Check in gastrointestinal and trauma patients.
        ii. DCAP-BTLS = Deformities Contusions Abrasions Punctures/Penetrations Burns Tenderness Lacerations Swelling
    b. **Assess for shape**
        i. Check in gastrointestinal and trauma patients
        ii. Normally (non-obese) patients have an s-shape to their stomach, this is called a 'normal scaphoid curve'.
        iii. If the patient seems normally thin, but they have a large belly, it could be because of blood in the belly (especially in trauma patients). If you suspect this, cut a piece of tape and place it on the belly, just above the umbilicus, with the short ends touching (it should look kind of like this: --). If, over time the inner ends of the tape pull apart (so they look more like this: - -) then the belly is expanding and you should suspect an internal bleed.
    c. **Assess for abdominal cough tenderness**
        i. Check in gastrointestinal and trauma patients
        ii. DO NOT use rebound tenderness. This is an antiquated and cruel assessment. There isn't anything additional to be learned using rebound tenderness, and it's really painful.
        iii. If present, cough tenderness suggests blood in the abdominal cavity.
        iv. Often, just bumping the stretcher (or the bouncing of the ambulance) will be enough to elicit abdominal tenderness in the abdominally bleeding patient
    d. **Assess for softness / guarding**
        i. Check in gastrointestinal and trauma patients.
        ii. Do this very gently. Do not use 'rebound' tenderness (see above).
        iii. If present it suggests blood in the abdominal cavity.
    e. **Assess for color and temperature**
        i. Check in gastrointestinal and trauma patients.
        ii. Be wary of the cool, mottled abdomen - this is a sign of shock.
    f. **Assess for ascities/obesity**
        i. Check in cardiac, respiratory, gastrointestinal and trauma patients.
        ii. There's a difference between obesity and ascities and it can be assessed by tapping the abdomen and seeing what type of wave is produced. Have someone experienced demonstrate or explain this to you, or just play around a bit an experiment with this while you're doing your exams. You'll get it.
    g. **Assess for abnormal pulsatile masses**
        i. Check in cardiac and gastrointestinal patients.
        ii. Be very gentle if you find one! DO NOT push on it.
        iii. Finding this is typically considered a diagnostic clue to a dissecting abdominal aortic aneurysm (AAA).
    h. **Assess for appliances**
        i. Check in all patients
        ii. Ask the patient why the appliance was placed (if they are able to respond)
        iii. Be alert for signs of internal bleeding, or of infections

i. **Assess for scars**
    i. **C**heck in all patients.
    ii. Ask the patient what the scar is from (if they are able to respond), often they are from previous surgeries
j. **Assess children for paradoxical respiratory motion**
    i. Children normally have their belly go out during inspiration and in during expiration, if this is reversed it's considered the same as accessory muscle use in the adult
k. **Assess for abdominojugular reflux**
    i. Check in all cardiac patients (especially those with CHF).
    ii. To do this, press on the abdomen (if safe and appropriate to do so) and watch if their JVD goes up (normally it won't). If it does, it suggests that the right ventricle is not working properly and can't accommodate an increased venous return (think: constrictive pericarditis, right ventricular infarction, and/or restrictive cardiomyopathy)
l. **Bowel sounds**
    i. Report as present if you happen to hear them while auscultating the chest, but do not specifically assess for these in the field as a paramedic. They aren't that important to us because in order to *properly* assess for bowel sounds you must listen in each quadrant for a full minute, and that's four minutes that we generally don't have (unless, of course, you do, In which case - knock yourself out).

8. Pelvic Exam
    a. **Assess for lesions (DCAP-BTLS)**
        i. Check in trauma patients.
        ii. DCAP-BTLS = Deformities Contusions Abrasions Punctures/Penetrations Burns Tenderness Lacerations Swelling
    b. **Assess for stability in 3 planes (in up, and down)**
        i. Check in trauma patients.
        ii. Crepitus and/or instability suggests a pelvic fracture.
    c. **Assess for priaprism**
        i. Check in neurological and trauma patients.
        ii. DO NOT check in female patients or people will (rightfully) laugh at you
        iii. If present it suggests spinal cord injury. Anal sphincter tone will also probably be loose, but this is not commonly assessed by paramedics.
        iv. If you discover priaprism, take a look again for a sweat line on your patients torso. Sweating is a sympathetic response, so there should be sweating on the upper part of the torso (it might be pale and mottled too - all indicating SNS innervation). If there is a spinal cord transection, the part of the torso that is not receiving SNS innervation (the lower part) will be well coloured and won't be sweaty. Mark the sweat line with a pen or marker.
    d. **Assess for urinary and fecal incontinence**
        i. Check in neurological and trauma patients.
        ii. Urinary incontinence is not uncommon in patients who lose consciousness, fecal incontinence is less common and more ominous.

9. **Extremity Exam**
    a. **Assess for lesions (DCAP-BTLS)**
        i. Check in all patients.

   ii. DCAP-BTLS = Deformities Contusions Abrasions Punctures/Penetrations Burns Tenderness Lacerations Swelling.

b. **Assess for PMS (pulse, motor, sensory)**
   i. Check in cardiac, neurological and trauma patients.
   ii. You can just check for pulses only in isolated cardiac patients

c. **Assess for clubbing of the fingers**
   i. Check in cardiac and respiratory patients.
   ii. Presence suggests chronic anemia.
   iii. This is a common sign in long-term smokers

d. **Assess for ROM (range of motion)**
   i. Check in neurological and trauma patients.
   ii. Only check if there is no evidence of musculo-skeletal trauma (ie don't move broken bones!

e. **Assess for ambulation if appropriate (ability, posture and gate)**
   i. Check in neurological and trauma patients.
   ii. Assess the ability to ambulate, resting posture, and their gate while walking
   iii. Assess for tremors or ataxia.
   iv. Be careful with this, don't ask your patient to try walking when they are unsteady on their feet, only to let them take a nose-dive because you weren't guarding to make sure they didn't! you and your partner should walk beside them if you're testing this because you have doubts about their ability to walk. Be prepared to catch them!

f. **Assess for strength (grip strength and pronator drift)**
   i. Check in neurological and trauma patients.
   ii. Pronator drift is a more sensitive sign than grip strength and is preferred.
   iii. This is an important indicator of stroke, if it is present check for ptosis and other facial asymmetry as well as for speech deficits.

g. **Assess for bilaterally equal BPs in arms**
   i. Check in ALL cardiac patients.
   ii. Unequal pressures (more than a 10-20 mm/Hg difference) suggests the possibility of subclavian steal from a dissecting aortic aneurysm.

h. **Assess for radio-femoral pulse transmission delay**
   i. Check in cardiac patients, especially in those with suspected abdominal aortic aneurysm (AAA).
   ii. This isn't actually all that difficult to do, check in yourself, other students, or healthy patients
   iii. Also visualize the thighs in the suspected AAA patient (pull down their pants) and check for colour and temperature. Unilateral coolness and pallor suggests the dissecting AAA. Check their big toe too for the classic 'blue toe' sign, indicating lack of perfusion.

i. **Assess for orthostatic hypotension**
   i. Check in cardiac, neurological and trauma patients.

j. **Assess for tremors or ataxia**
   i. Check in neurological patients.
   ii. Presence suggests CNS lesion (especially to cerebellum), possibly due to CVA/TIA, traumatic head injury, CP, MS or tumor.
   iii. May also be due to drug effects, especially sedatives such as alcohol, barbiturates or benzodiazepines. Solvent inhalation (i.e. 'glue sniffing' or other propellants such as those used in spray paint or hair spray) is a commonly encountered cause in EMS.

k. **Assess for edema**
    i. Check in cardiac and respiratory patients.
    ii. Is there any present?
    iii. Is it pitting?
    iv. How high up the legs does it go?
    v. Ensure that sacrum/scrotum is examined for dependent edema too.
    vi. Note: 'Anasarca' is often used in EMS to describe edema from foot to abdomen in the sitting patient (it actually means widespread/globalized edema) .
    vii. Pitting edema suggests liver, kidney or (most commonly) right sided heart failure. Be sure to assess breath sounds for pulmonary edema
l. **Assess for skin turgor**
    i. Check in all patients, especially the medical/dehydrated patients
m. **Assess for Plantar- Babinski reflex**
    i. Check in neurological and trauma patients
    ii. The patients response is *either* plantar (toes go down) *or* babinski (toes go up). Do not use silly terms like 'positive (or negative) plantar response'. These make no sense.

Student Evaluation Method: Students will be evaluated through multiple methods, including formative evaluations within each learning module in the form of section quizzes which require 100 percent before moving to the next section; summative evaluation through an end-course online examination which requires a score of 90 percent or better; once the student has successfully completed the online learning modules and scored at or above 90 percent, they will attend a guided practical and competency evaluation, which will serve as a cumulative summative evaluation.

Evaluation of Presentation: Continuing Education Program Sponsor Evaluation Form will be filled out by all participants.