I. Homeostasis

a. Concepts

i. pH = potential/power of Hydrogen

ii. Acid: substance that donates a proton, H ion

iii. Base: substance that accepts a proton, H ion

iv. Neutrality → 7.35 – 7.45

1. Concentration of H ions in pure water, with equal amounts of hydroxyl ions
2. At equilibrium: $H_2O \leftrightarrow OH^- + H^+$, where [H+]= 1x 10^{-7} moles/L

v. Acidity: high H concentration, [H+] > 1x 10^{-7} moles/L

vi. pH <7 → 1-7.35

vii. Alkalinity: low H concentration, [H+] < 1x 10^{-7} moles/L

viii. pH >7 → 7.45 – 14

b. pH Regulation

c. Blood pH regulations has a very narrow range: 7.35-7.45, ± 1.15 x 10^{-7} moles/L

d. At extreme pH proteins and organs lose normal function: pH <7.3, pH >7.5

e. Buffers

i. Pka = acid dissociation constant

ii. Henderson-Hasselbalch equation:

$$pH = pKa + \log$$

iii. Buffer is most effective within 2 pH units of its pKa

1. Maximum buffering capacity when pKa = pH
2. You want to equal amount of base and acid

f. Chemical buffers → maintain neutrality

i. Substance that minimizes pH changes of a solution following the addition of small amounts of acid/base

ii. HA= weak acid; A^-= conjugate base of weak acid

iii. Equilibrium of weak acid in solution

1. HA $H^+ + A^-$

iv. K_1: dissociation of HA into H ion and A^-

v. K_2: dissociation of H and A^- into HA acid

g. Chemical buffers

i. After final equilibrium is achieved, a small change in the concentration of one component will cause the equilibrium reaction to shift in an attempt to re-establish the final proportion of components

1. If small amounts of HA are removed from the weak acid equilibrium reaction, the reaction will shift to the left in order to restore the original conditions
2. If small amounts of H or A are removed, the reaction would shift to the right

h. Physiological buffers

i. Hemoglobin
ii. Bicarbonate
iii. Phosphate
iv. Proteins

i. Physiological acids
    i. Body's metabolic pathways produce several acidic waste products
    ii. 2 types of acidic waste
        1. Volatile acid: carbon dioxide
        2. Fixed acids: phosphates, sulfates, organic acids (lactic acid, acetoacetic acid, beta-hydroxybutyric acid) → ketoacids
        3. Hypoventilation → trapping CO2 in the body to increase acidity

II. Acid-Base Regulation
a. Buffer systems: regulate H ion concentration
b. Lungs and kidney: maintain pH homeostasis
c. Bicarbonate Buffer System
    i. Ratio between carbonic acid and bicarbonate is maintained by the lung:

    CO_2 H_2CO_3 $H^+ + HCO_3$
    ii. Carbonic anyhdrase
d. Lungs
    i. Too acidic = hyperventilate
    ii. Too alkaline = hypoventilate
e. Kidneys
    i. Hydrogen and bicarbonate = carbonic acid dissociate H2) + CO2 – urine to Kidney Wall and form carbonic acid and dissociate Hydrogen to urine and bicarbonate to blood

III. Acid-Base Disorders
a. Acidosis → pH = <7.35
b. Alkalosis → >7.45
c. Check out the ABC sheet on webcourses!
d. Metabolic Acidosis
    i. Due to increased production of organic acids
    ii. Decrease in normal 20:1 ratio of HCO_3^-:H_2CO_3
    iii. Pathogenesis → decreased bicarbonate, increase carbonic acid
        1. Increase in acid
        2. Excess base removal/decrease
        3. Combination of acid increase and base decrease
    iv. Compensatory physiological response
        1. Compensatory respiratory alkalosis via hyperventilation: reduce P_{CO2} and partial increase in HCO_3/H_2CO_3 ratio (does not remove metabolic acids from body)
    v. Etiology
        1. Uncontrolled diabetes: excess fatty acid oxidation leads to accumulation of acetoacetic acid and hydroxybutyric acid

2. Fasting: results in increased acetoacetic and hydroxybutyric acid levels
3. Increase in anaerobic metabolism: increase lactic acid production
4. Local tissue hypoxia (low tissue P_{O2}): lactic acid production
5. Renal failure acidosis: failure to exchange H ions for Na ions
6. **Liver disease**: impairs urea and ammonia formation causing H ion retention

vi. Etiology
1. **Medication**: salicylate intoxication (converts to acid before excretion)
2. Ingested poisons: convert to acid metabolites, methanol, ethylene glycol, paraldehyde, ammonium chloride
3. Large quantities of isotonic sodium chloride infusion: high Na competes with H ions for renal excretion
4. **Ingestion of carbonic anhydrase inhibitors**: interferes with bicarbonate
5. **Diarrhea and colitis**: decrease bicarbonate concentration

vii. Clinical manifestations
1. ABG: below normal bicarbonate concentration
2. pH: below normal if uncompensated
3. Headache, abdominal pain
4. CNS depression: confusion, lethargy, stupor, coma
5. Ventricular dysrhythmias: due to myocardial intracellular acidity
6. Decreased cardiac contractility
7. Brainstem dysfunction: when pH <6.9, fatal
    a. Cardiorespiratory arrest
    b. MA caused by hyperventilation

e. Respiratory Acidosis
i. Etiology
1. Disorders that impair ability of lungs to expel carbon dioxide: pulmonary edema, bronchoconstriction, pneumonia, **asthma, apnea**, emphysema, **morphine injection, barbiturate poisoning**
2. **Respiratory distress syndrome (RDS**): common in premature infants who lack sufficient surfactant levels in their lungs, gas exchange inhibited
3. **Adult respiratory distress syndrome (ARDS**)

ii. Clinical manifestations
1. Tachycardia, cardiac dysrhythmias
2. Headache, neurological abnormalities (blurred vision, tremors, vertigo, disorientation, lethargy)

iii. Physiological response