        - **Solute** = substance dissolved in a solution (ex: salt)
        - **Solvent** = substance that solute is dissolved in (Ex: H2O) (Fig. 3.6)
    - Types of transport:
        - **Passive transport**
            - No NRG required (no ATP)
            - Movement from high [] to low [] (i.e. down its [] gradient) = **DIFFUSION**
            - Diffusion ➔ passive process; no input of NRG required
            - The greater the diff. in [] ➔ the more molecules wants to move
            - LESS [] GRADIENT VS. HIGH [] GRADIENT—depends on the [solute] on one side of the membrane. Check extra notes
            - Types:
                - Simple diffusion (PASSIVE TRANSPORT) (solute movement) (Fig. 3.7a)
                    - Solute diffuses through cell membrane therefore **small, <u>lipid-soluble</u>** (O2, CO2, etc.) OR
                    - Ions diffuse through membrane via protein channels
                - Facilitated diffusion (PASSIVE TRANSPORT) (solute movement) (Fig. 3.7)
                    - **Still from high [] to low []**
                    - For **large, charged molecules or <u>H2O-soluble</u> molecules** (NOTE: lipid soluble molecules diffuse by simple diffusion)
                    - Diffuse across membrane using a specific carrier protein
                        - Ex: glucose (an H2O soluble molecule) into sk. muscle
                    - NOTE: PROTEIN CHANNELS – just pores/hole within membrane vs. CARRIER PROTEINS – they bind to molecule and change shape – carries molecule from one side to the other

- **NOTE:** lower OP = higher [H2O] and vice versa
- Less solute outside (higher [H2O]) of cell
- Cell swells (takes in H2O) + may burst
- NOTE: Think of "Hungry, Hungry, Hippos
- Swelling can rupture cell = LYSIS
- **NOTE:** If the cell is RBC ➔ then it's HEMOLYSIS

  - Hypertonic solution
    - ECF has greater OP (therefore, lower [H2O]) than ICF (= CYTOSOL)
    - Cell shrinks (loses H20)
  - Isotonic solution
    - NOTE: "iso-" = same
    - ECF and ICF have equal OP
    - Cell neither shrinks nor swells
    - RBC ➔ All [solutes] w./in equals a 0.9% saline solution (= normal saline)
    - Amount of solute does matter and the types doesn't matter when we're

- Chemicals (ex: binding of hormone or nt) = chemical gates (=ligand-gated channels)
- (NOTE: gated channels are named after their stimuli)
- Other examples:
    - Temp' = thermal gates
    - Mechanical (physical) deformation = mechanical gates

- **Resting Membrane Potential (RMP)**
    - (Fig. 11.7 + 11.8)
    - **NOTE:** Diff. in [ionic] on diff. side
        - At rest (= not stimulated), a charge diff. (potential difference) exist just across the cell mem' (= **MEMBRANE POTENTIAL**)
        - **NOTE**: Voltage is always measured between 2 points (= potential diff.) greater diff. in charge between 2 points = higher the voltage
        - *** -70 mV (inside of cell is more neg') = **RMP**
    - Factors Establishing RMP (Fig. 11.8):
        - 1.) Na+/K+-ATPase (Na+/K+ pumps)
            - NOT A CHANNEL!!! It's a carrier protein functioning as a pump
            - Breaks down 1 ATP and uses NRG to pump 3 Na+ out and 2 K+ in ➔ both ions are pumped against their [] gradient, therefore, **ACTIVE TRANSPORT**
            - Effects:
                - **IMPORTANT**: maintains [] grad' of Na+ and K+
                - Contributes a little (a few mV) to RMP (pumping more + ions out than in)
        - **2.) Org- inside cell**
            - Can't cross membrane (org- are non-diffusible) **NOTE:** Large org- ions stay inside b/c they're too large to diffuse and transport

- 3.) More non-gated K+ channels than non-gated Na+ channels (this is not the Na+/K+-ATPase pumps)
    - (Membrane is more permeable to K+ than Na+ at rest. Therefore, K+ is the MAJOR determinant of RMP)
    - K+ diffuses out of cell down its [] grad'. Tf, cell loses +ve charge (inside becomes more –ve inside)
    - Unlike charges attract and K+ diffusion slows as inside becomes increasingly negative – negativity inside the cell pulls the positive K+ back in
    - Na+ diffusion ↑ due to increasing attraction to –ve interior of cell – which is due to K+ efflux
    - Until -70 mV is reached, +ve charge going out (K+ diffusion out of cell) is greater than +ve charge going in (Na+ diffusion into cell) ➔ greater K+ permeability
    - Once at -70mV, amount of +ve (K+) moving out equals the amount of the +ve (Na+) moving in (at exactly -70 mV ➔ we have equal leaving and entering)
    - Therefore, net movement of charge (ions – Na+ and K+) is zero (equal in both directions) ➔ **RMP -- -70 mV**

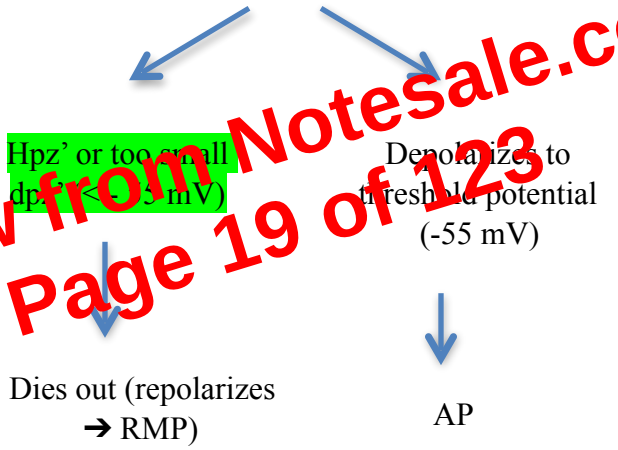
- **Electrically Excitable Cells**
    - **ONLY** muscle and nerve cells
    - Capable of producing departures from RMP (-70 mV = set point) in response to stimuli (= changes in ext. or int. envr')
    - **When a neuron is stimulated:**
        - A.) Gated ion channels (voltage-gated channels) (ex: Na+/ K+ channels) open (IMP. in establishing membrane potential (MP = a charge/voltage diff. across membrane))
        - B.) MP changes = producing a graded potential (NOTE: graded b/c of various levels/grades due to strength of stimulus – ex: small changes in MP – either +ve or -ve) (i.e open lots of gates ➔ strong stimulation)
        - C.) GP ➔ Triggers an action potential (AP) (Fig. 11.11)
            - **NOTE**: -55 mV = THRESHOLD POTENTIAL

- If GP causes dpz' and if it's large enough (i.e. caused by a critical stimulus or multiple GPs summing to become a large GP) ➔ leads to an AP
- Steps:

1.) Critical stimulus
⬇
2.) GP reaching threshold (= -55 mV)
⬇
3.) AP

- The story so far…

Stimulate neuron
⬇
Gated ion channels open at the site of stimulation
⬇
GP (change in membrane potential)



- **Action Potential (AP)**
  - A nerve impulse (signal)
    - How neuron transmits a signal
    - Large change in MP that propagates along an axon w. no change in intensity
  - Initiates at trigger zone (Table 11.1)
    - Ex: axon hillock of multipolar + bipolar neurons; just past the dendrites of unipolar neurons
  - Steps (Check back to notes for an imp' drawing) (Fig.11.11):
    - 1.) GP – MP at the axon hillock reaches -55 mV
    - AP…

        - 3.) Rise Ca++ triggers exocytosis of nt-containing vesicles
        - 4.) Nt crosses synaptic cleft, binds to specific receptors on postsynaptic membrane
            - Receptors = chemically gated ion channels = open in response to nt (=ligands)
        - 5.) Gated ion channels open ➔ allowing movement of ions into (or out of) postsynaptic membrane (Fig. 11.16)
            - Creates graded potential (GP) called a **POSTSYNAPTIC POTENTIAL** (PSP)
            - **NOTE:** Characteristics of PSPs is SAME as GP – any type of GP has same characteristics as ordinary GP
    - **Postsynaptic potentials (PSPs)**
        - PSPs may be:
            - 1.) Excitatory PSPs (EPSPs)
                - **NOTE:** EPSPs = GP
                - EPSPs ➔ depolarizing GP (Fig. 11.19 + 11.18) due to opening of Na+ (or Ca++) channels or closing of K+ channels
                    - **NOTE:** Some stimuli can close channels
                - Nt is often **ACETYLCHOLINE** (ACh) or **GLUTAMATE** (Note: there are other types of nt)
            - 2.) Inhibitory PSPs (IPSPs)
                - = GP ➔ causes hpz' (moving away from -55 mV to more –ve charge)
                - Due to opening of K+ or Cl- channels
                - Inhibits neuron from reaching AP
                - Nt is often **GLYCINE** (DON'T MIX THIS WITH EXCITATORY GLUTAMATE) or **GABA**
        - PSPs occur on CELL BODY or DENDRITES (Like any GP)
            - Many neurons can synapse on to one (postsynaptic neuron) (Fig. 11.16) ➔ If many EPSPs ➔ summation

- **Reflex pathway/arc** = pathway of impulses
- Fig. 13.15

Stimulus ➔ R (specific to a type of stimulus; responds to a specific stimulus) ➔ CNS ➔ E (effectors)

- **Reflexes**
  - Reflexes are categorized according to:
    - A.) Effector
      - i.) SOMATIC = effector is SKELETAL muscle (Fig. 13.17)
      - ii.) VISCERAL (AUTONOMIC) = effector is SMOOTH muscle, CARDIAC muscle, or GLANDS
    - B.) Which side of the body the sensory + motor neurons are located (Fig. 13. 20)
      - i.) IPSILATERAL reflex = sensory + motor neurons on same side
        - Ex: stimulus on right side = response/output on right side
      - ii.) CONTRALATERAL reflex = sensory + motor neurons are on opp. side
        - Ex: stimulus on right side = response/output on left side
    - C.) Number of synapses (+ neurons) in are:
      - MONOSYNAPTIC reflex = 1 synapse between 1 sensory neuron and 1 motor neuron
      - POLYSYNAPTIC reflex = 2 or more synapses between 3 or more neurons (**NOTE**: # of synapses doesn't equal the # of neurons)
- Examples:
  - A.) SOMATIC spinal reflexes (somatic – E = sk. muscle)
    - **1.) Stretch reflex**
      - Ex: Knee jerk reflex
        - Extensor muscle contracts (Fig. 13.18)
        - Stimulus = tapping the patellar ligament, which stretches the quadriceps femoris muscle (quad')
        - Receptor = muscle spindle (in quad') (i.e. stretch receptor)

- Ex: epinephrine on liver cells ➔ activates cAMP ➔ causes breakdown of glycogen to glucose ➔ released to blood
- **Why use 2nd messenger systems?**
  - A.) Hormone can't enter cell (water soluble hormones can't enter phospholipid bilayer)
  - B.) Rapid acting (enzymes are already present – just activate – you don't have to make the protein – you just activate them)
  - C.) 1 hormone molecule ➔ many enzyme molecules are activated (ex: G protein = enzyme molecule) ➔ multiple signals produced
  - D.) Limited – messengers (i.e. hormones) can be broken down or removed
    - Breakdown of hormones

- **Lipid soluble hormones**
  - Still binds to receptors (= intracellular receptors) – even if they diffuse through the plasma membrane, they still bind to receptors in the cytoplasm of cell (Fig. 16.3)
  - Steroid (ex: cortisol ➔ released for long-term stress event) and thyroid hormones (NOTE: water soluble hormones – peptides, proteins, catecholamines)
  - Trigger protein synthesis
    - Takes time. Tf, slow, but longer-lasting response
  - Steps for action:
    - A.) Enter target cell and bind to INTRACELLULAR (= nuclear – in cytoplasm or nucleus, NOT intercellular) receptors
    - ** Hormone receptor complex binds to a specific region on DNA (activate genes) ➔ starts **gene transcription** (transcribing from DNA strand to mRNA) ➔ produces messenger RNA (mRNA)
    - mRNA attaches to ribosomes to produce proteins (translation ➔ translating a series of nucleotides from mRNA to a. acids [aa] – DON'T CONFUSE IT W. TRANSCRIPTION)
- **Regulation of Hormone Secretion into Blood**:
  - Fig. 16.4

NE converts glycogen to glucose IN LIVER)

- Low insulin ➔ glucose is then not taken up well, esp. by sk. muscle at rest and adipose tissue (fat). Therefore:
    - Glucose is spared for NS
    - Non-NS (other cells of the body) use fats for NRG (control: GH, cortisol – they signal non-NS to use fats for NRG)
        - If stress continues, cortisol inhibits GH release and then protein is used
    - Overall: ↑ in blood FA and a. acids (b/c you're breaking down proteins and fats and then use them as NRG source) ➔ NRG (except brain – brain uses glucose)
- B.) Inhibition of immune system, inhibition of formation of connective tissue (ex: bone) = delayed healing
- C.) Release of aldosterone + antidiuretic hormone (ADH)
    - Reduce kidney Na+ and water loss to maintain blood volume

- **Long Term Effects**
    - ↓ weight (b/c fats and proteins are being used), ↑ bp, ↑ h.r., ↓ bone density, ↑ risk of Type 2 diabetes (b/c of ↑ in blood glucose due to cortisol inhibiting the release of glucose)

- **3.) Phase 3: Exhaustion**
    - Results from:
        - Depletion of body resources (i.e. lipid reserves)
        - Aldosterone causes loss of Na+ (**NOTE:** Na+ is used in causing AP)

        - Bone growth, closure of epiphyses (epiphyseal plates close up)
        - (NOTE: in males: testosterone goes into bones then converted to estrogen to stop growth = closure of plates – becomes epiphyseal line)
    - **Progesterone**
        - From CORPEUS LUTEUM
        - Prepares uterus for pregnancy
- **Ovarian and Uterine Cycle**
    - Both occurs simultaneously
        - **1a.) Follicular (pre-ovulatory) phase: Days 1-14**
            - Early on: low progesterone. Tf, LH and FSH are secreted (from the ant. pit.) (Check the chart in extra notes)
                - Tf, high levels of LH and FSH
                - Some 1° follicles turn to 2° follicles (due to high levels of FSH)
            - Follicles secrete estrogen
                - Tf, blood estrogen rise
            - Later on: one (usually 2°) follicle becomes a Graafian follicle (**NOTE:** Graafian (= tertiary) follicle (w. secondary oocyte floating around the fluid of antrum)
        - **1b.) Uterus**
            - occurs at same time as 1a
            - Menstrual phase (days 1-5)
                - Stratum functionalis shed (layer of endometrium) and bleeds
                - Tf, menstrual flow = blood cells and secretions
            - **Proliferative phase (days 6-14)**
                - NOTE: ≈ day 14 = ovulation
                - Estrogen causes ➔ repair and proliferation of st. functionalis (due to mitosis of st. basalis)
                    - Peak in progesterone vascularized the endometrium and causes build up of glycogen
        - **2.) Ovulation (Day 14)**
            - Due to LH surge
            - LH triggers:
                - Completion of meiosis 1

(High NRG)

- NOTE: Break P bonds – NRG released
- NRG produce used for reactions (ex: protein synthesis, etc.)
- Little ATP is stored
  - Tf, it is constantly made

- 3.) Cellular Respiration
  - Production of ATP using glucose
    - Glucose enters most cells by facilitated diffusion (Tf, no NRG/ATP needed – NOTE: it uses protein but doesn't use ATP) (may be ↑'ed in some cells by insulin since insulin ↓ blood glucose – glucose from blood into cell; in other situations, active transportation (NRG/ATP needed) is used)
    - Overall:

$C_6H_{12}O_6$ (1 glucose) + $6O_2$ ➔ $6CO_2$ + $6H_2O$ + 32 (max.) ATP + heat

- Steps (Fig. 24.5):
  - A.) Glycolysis in cytosol
    - NOTE: Can be aerobic or anaerobic
    - **NOTE**: Anaerobic ➔ doesn't require O2 even if O2 is present
  - B.) Enters mitochondria ➔ becomes aerobic
  - C.) Krebs's cycle
  - D.) Electron Transport Chain (ETC)
    - Check extra notes for chart!!
    - **NOTE:** when glucose is not present, we can still live off proteins and fats
  - In the body (Fig. 24.3):
    - Glycogen ➔ glucose ➔ ATP
    - Proteins
      - Some amino acids can be converted to pyruvic acid or enters Kreb's cycle – depending on the body's need, they may form new glucose (liver, kidney) or ATP (most cells)
    - Fats

- Cycle repeats many times to shorten the sarcomere (Fig. 9.6)

- **Sliding Filament Mechanism**
  - Sarcomere shorten (Fig. 9.6)
    - H zone (=lighter band at center of A band ➔MYOSIN ONLY) and I band (=light band = actin + titin overlap) shorten
    - A band = same length (which is the length of thick filament)
  - Myofibrils (NOTE: myofibrils makes up myofiber = muscle fiber) shorten
  - Thin (actin) + Thick (myosin) myofilaments remain the same length
- **Muscle Fiber Relaxation**
  - Steps (Fig. 9.8):
    - 1.) ACh broken down by AChE on motor end plate (= sarcolemma; which is facing the synaptic cleft)
      - NOTE: GO BACK to the drawing of "Break Down of Neurotransmitters – ACh)
    - 2.) SR actively takes up Ca+2 (via Ca+2-ATPase)
      - NOTE: It requires active transport because Ca+2 is going against its [] gradient, Tf, ATP used
    - 3.) Tropomyosin covers myosin-binding sites on actin (b/c there are no more Ca+2 that binds to troponin)
    - 4.) ATP binds to myosin and release myosin head from actin (no more actin-myosin binding) (Fig. 9.12)
- **ATP necessary for:**
  - 1.) Cross bridge release (**NOTE**: ATP is not broken down yet when cross bridge breaks)
  - 2.) Activation of myosin (when ATP ➔ ADP + Pi)
  - 3.) Pump Ca+2 back into SR (**NOTE:** WHEN Ca+2 IS TAKEN UP: it's going against [] grad'; DURING RELEASE OF Ca+2: don't need ATP b/c we have high [] of Ca++ in SR already which goes down its grad')
  - 3.) Fiber (= muscle fiber) Na+/K+-ATPase activity
    - To reestablish [] gradients (i.e. Na+ and K+)
- **Muscle Tension** (Fig. 9.13)
  - = Force exerted by a muscle or a muscle fiber
    - Determined by a # of cross bridges formed
  - **In a muscle fiber – affected by:**

- **Excitation-Contraction Coupling in Myocardial Cells**
  - 1.) Open v-g Ca+2 channels of AP
    - Small increase in cytosolic Ca+2 (extracellular – FROM OUTSIDE TO INSIDE) ➔ not enough to trigger contraction BUT
  - 2.) Opens CHEMICALLY-GATED Ca+2 channels on SR ➔ Increases cytosolic Ca+2 ➔ Binds to troponin, etc.,etc. ➔ leads to contraction (same events occur just like the events that occur in sk. muscles)
  - 3.) Contraction
    - Refer back to sliding filament mechanism
    - Begins a few msec after AP begins
    - NOTE: we don't want maintained contraction ➔ heart would stop pumping if contraction is maintained
    - Duration of AP = ≈ 250 msec and duration of twitch/ CONTRACTION = ≈ 300 msec
      - Tf, contraction is almost over when AP ends
      - RESULT = NO SUMMATION – REFRACTORY PERIOD PREVENTS TETANUS – CHECK FIG. 18.13
        - Tf, NO TETANUS – you get alternation of contraction and relaxation – once contraction ends, relaxation occurs

- **Cardiac Cycle**
  - 3 components
    - 1.) Electrical Activity (ECG or EKG)
      - NOTE: ECG ➔ we're looking at the whole electrical event of the whole heart
      - Small currents due to dpz' and rpz' of heart movement through salty body fluids
      - Potential measured on body surface using electrodes (= leads)
      - Recording seen as waves
        - = SUM of electrical activity of all myocardial cells (NOT AN AP)
      - ECG Waves (Fig. 18.17 + 18.18):
        - A.) P wave = atrial dpz ➔ followed by contraction
        - B.) QRS wave = ventricular dpz ➔ contraction
          - Also **atrial rpz** (➔ relaxation) – but masked by a larger ventricular electrical event (due

        - Digitalis (drug) -- ↑ Ca+2 inside (Tf, ↑ contract motility)
    - Forced decreased by:
        - Acidosis – excess of H+
        - ↑ external K+ -- not a lot of K+ leaves the cell; Tf, no restoration of RMP
        - Ca+2 channel blockers
            - i.e. drug (Ex: verapamil)

- **Blood Circulation**
    - Blood Flow = volume of blood flowing through any **tissue** per min (mL/min – same unit as CO)
    - BF in a vessel is determined by PRESSURE + RESISTANCE
    - BF is calculated as:
        - Flow = ΔP/R = (b.p. gradient)/R
            - F = Flow
            - ΔP = b.p. gradient from aorta → arterioles (aka resistance vessels) (ΔP going from aorta (low P) to arterioles (high P)) (NOTE: DIAMETER OF LUMEN INCR. ↓ AS YOU REACH CAPS')
            - Large veins (capacitance vessels – HIGH CAPACITANCE -- STRETCHY)
                - NOTE: 60-75% of blood in veins
            - R = resistance
                - Opposes flow – friction of blood rubbing against vessel walls
                - Depends on:
                    - a.) Vessel length (less R when diameter is big and vice versa)
                    - b.) Blood viscosity (↑ viscous = ↑ R)
                        - NOTE: vessel length and blood viscosity doesn't normally change
                    - c.) Radius of arterioles (major RESISTANCE VESSELS) controlled by smooth muscle innervation by **SNS** (NOTE: NO PSNS innervation of b.v.)
                        - Vasodilation = ↑ radius

        - Vasoconstriction ➔ skin, viscera – reinforces SNS
        - Vasodilation ➔ heart, sk. muscle, liver – opposes SNS in just these specific areas b/c we want increased blood flow in these areas
    - Other hormone
        - Angiotensin II, ADH – causes vasocon'
        - Histamine – causes vasodil'
            - Ex: allergy/ inflammatory response

- **Blood Pressure**
    - = Hydrostatic P exerted by blood on wall of blood vessel (clinically on the walls of arteries) – results when F is opposed by R
    - Systolic pressure = pressure due ventricular constriction
    - Diastolic pressure = pressure due to vascular resistance (when ventricles are relaxed – blood are in b.v. already)
    - What we measure in an artery (i.e. 120/80 = systolic/diastolic)
    - Mean Arterial Pressure (MAP) = regulated BY THE BODY – the body regulates MAP by various baroreceptors – aortic arch and carotid artery (i.e what the body measure)
        - = Average b.p. through cardiac cycle
        - But diastole is longer than systole (Check chart w. timeframe in extra notes), so: MAP = diastolic P + 1/3 pulse P
    - MAP regulation:
        - $F = \Delta P/R$ ➔ $\Delta P = (F)(R)$
        -
        -
            - Where TPR = resistance in all arterioles; HR (beats/ min); SV (mL/beat)
        - ΔP = MAP – venous P (P in vein is ≈ 0 ➔ Tf, ΔP = MAP)
        - MAP is regulated by controlling:
            - 1.) Cardiac output
            - 2.) TPR (arteriolar radius)

        - = Endocytosis from blood into epithelial cell then exocytosis from endothelial cell into ISF
    - C.) Mediated transport
        - Requires a carrier protein
        - Imp. mainly in the brain
- Fluid (H2O) enters (=ABSORPTION) or leaves (= FILTRATION) capillaries by:
- NOTE: movement out of caps' = filtration; into caps' = absorption
    - 1.) Osmosis
    - 2.) Bulk flow – due to P diff.
        - 4 pressures involved (Check extra notes):
            - Blood hydrostatic P (BHP) = b.p.
            - Blood osmotic P (BOP) = due to plasma proteins in blood (mostly albumins)
            - ISF hydrostatic P (IFHP) = 0 mmHg
            - ISF osmotic P (IFOP) = due to ISF proteins
    - Net Filtration Pressure
        - NFP (in mmHg) = (BHP + IFOP) – (BOP +IFHP)
        - NOTE: (BHP + IFOP) = promotes filtration and (BOP +IFHP) = oppose filtration (= absorption)
            - Check extra notes
            - NOTE: Whichever has the largest # in the formula, it'll determine whether you have net filtration or absorption
            - Check extra notes for an example
        - In prev. example:
            - Overall, (across whole cap' – not just one end)
                - 10 mmHg + (-9 mmHg) = 1mm Hg
                    - Tf, net filtration
            - Tf, in the whole body
                - 90% of filtered body fluid (= ISF) reabsorbed to blood
                - 10 % enters the lymph – which goes back to the blood eventually
                - Tf, ISF volume remains constant -- 100% of filtered fluid gets into the blood eventually

- Clinical Application
  - Edema
    - = Accumulation of fluid (ISF) in the tissue
    - ISF causes swelling
    - Due to:
      - High b.p. (= increased in BHP)
      - Leakage of plasma protein into ISF ➔ inflammation (🡅 IFOP)
      - 🡇 plasma proteins (due to ex: malnutrition, burns) (tf, 🡇 BOP)
      - Obstruction of lymph vessels – from ex: elephantitis, surgery
  - **Circulatory Shock**
    - = Inadequate blood flow
    - (🡇 O2, nutrients to cell)
      - 1.) Hypovolemic shock
        - NOTE: "hypovolemic" = "low volume"
        - Decreased blood vol.
        - Due to: blood loss, severe burns, diarrhea, vomiting
      - 2.) Vascular shock
        - Blood vol' normal but blood vessels are expanded
        - Due to: SYSTEMIC vasodilation of blood vessels ➔ 🡇 b.p. (= 🡇 BOP)
        - Examples:
          - a.) Anaphylactic shock = allergic rxns
            - Due to lots of histamine (=vasodilator) released from lots of mast cells
            - NOTE: Epi pen ➔ lots of EpiE ➔ causes vasoconstriction to offset vasodilation
      - 3.) Cardiogenic shock
        - Pump failure = 🡇 cardiac output

        - NOTE: RBC ➔ no nuc' or mitochondria (b/c mitochon' uses O2 for cell resp' ➔ we want O2 in gas transport)
            - Tf, RBC uses anaerobic resp' only (i.e. don't use O2)
- b.) White Blood Cells (WBCs) (= Leukocytes)
    - i.) Granulocytes
        - 1.) Neutrophils
            - First to enter infected area (Fig. 17.10)
        - 2.) Eosinophil
            - Attacks parasites (ex: worms)
            - Break down chemicals released in allergic rxns
        - 3.) Basophils
            - Secrete histamine (a vasodilator) -- ↑ inflammation
            - Secrete heparin – inhibits local clotting
                - NOTE: in order for neutrophils to get into infected areas – blood clot must be inhibited in infected areas
    - ii.) Agranulocytes
        - 1.) Monocytes
            - Enter tissues and enlarge to become phagocytic macrophages
        - 2.) Lymphocytes
            - a.) T-lymphocytes
                - Helper T (TH) + cytotoxic T (CTCs) lymphocytes
            - b.) B lymphocytes
                - When activated, they give rise to plasma cells which

        - Thoracic walls recoils out, lungs recoil in
        - NOTE: lungs are elastic – but fluid holds them together; Tf, P ip is slightly reduced
- **Types** (Fig. 22.13 + 22.14)**:**
    - Quiet Inspiration
        - ACTIVE process ➔ still requires ATP (b/c muscles contract)
        - At start, (before muscle contracts): P atm = P alv (P inside lungs) (760 mmHg) – no air moves, then:
            - i.) Diaphragm, ext. intercostal contract
                - Tf, ↑ vol. of thoracic cavity
            - ii.) Lung resist expansion
                - Tf, P ip ↓ (756 mmHg ➔ 754 mmHg) (Fig. 22.14)
            - iii.) Higher P difference between P alv and P ip pushes lungs out ➔ lungs expand ➔ Tf, P alv ↓ (b/c vol. ↑) (760 ➔ 758 mm Hg)
            - iv.) Air moves in down P gradient (until P alv = P atm)
    - b.) Forced Inspiration
        - Involves diaphragm, ext. intercostal, sternocleidomastoids, pectoralis minors, scalenes (Tf, ACTIVE)
            - All aid in inspiration (Only diaphragm and ext. intercostals for quiet inspr')
        - Big ↑ in vol of thoracic cavity – Tf, P gradient (between P_{alv} and P_{atm}) ↑ and more air moves in
    - c.) Quiet Expiration
        - Relax muscle ➔ lungs to resting size ➔ Tf, ↓ thoracic cavity (PASSIVE PROCESS)
        - Lung vol ↓, P ip ↑ (754 ➔ 756 mm Hg)
        - Tf, P alv ↑ (760 ➔ 762 mm Hg) ➔ air moves out/ down P gradient
    - d.) Forced Expiration
        - Labored or impeded (ex: asthma) breathing
        - Relax diaphragm, ext. intercostals + contract int. intercostals, abdominals (ACTIVE PROCESS)

- b.) Then, inhibitory signals cause:
  - i.) Gastric motility (slows emptying) due to:
    - 1.) CCK – released due to presence of FAs, a. acids
    - 2.) Enterogastric reflex (enteric NS) – triggered by a. acids, peptides, acid, duodenal stretch, hyper-tonicity
      - Directly
      - Signals CNS ➔ SNS
  - ii.) ↓ gastric secretion (acid, enzymes) due to:
    - Secretin released due to acid
    - CCK released due to a. acid, FAs

- Large Intestine (Fig. 23.29)
  - Motility:
    - 1.) Haustral Contractions
      - Slow, weak – move material down the tube – allow mixing, absorption of salts, H2O
    - 2.) Mass movement:
      - Due to food in stomach via **gastrocolic reflex** = powerful waves of contraction from transverse colon to rectum
      - Move fecal mass to rectum:
        - Initiates urge to defecate (NOTE: Autonomic reflex involved)
  - **Rectal (Defecation) Reflex** (Fig. 23.31):
    - Stimulus = feces in rectum (stretch receptor in rectal wall)
    - CNS = sacral segment of s.c. (PSNS)
    - Effector = sm. muscle of rectum contract, internal anal sphincter relaxes
    - External anal sphincter = under voluntary control (i.e. not part of reflex)
    - Digestion (Fig. 23.29):
      - NONE, but get bacterial fermentation of undigested nutrients
      - Bacteria synthesize some vitamins (B_6, B_5, K, Folate, Biotin)
    - Absorption: