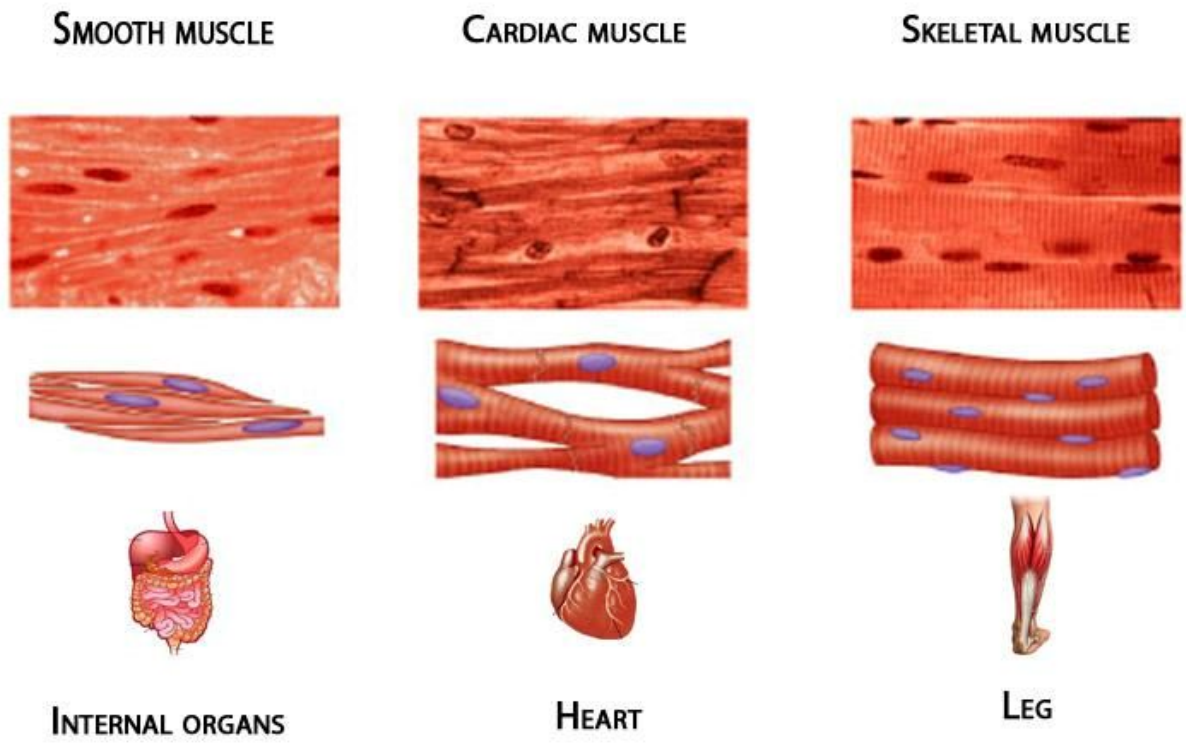
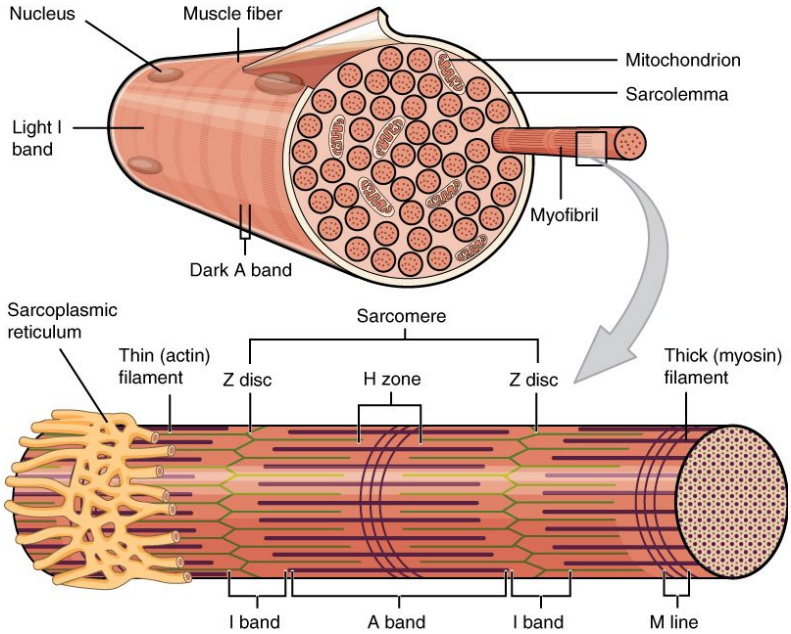


Anesthesia

Structure/Function of Skeletal Muscle

Muscle:



Muscle fiber: large multinucleate cells that are fused myblasts → contractile proteins = **myofibril**, striation due to arrangement of myofibrils

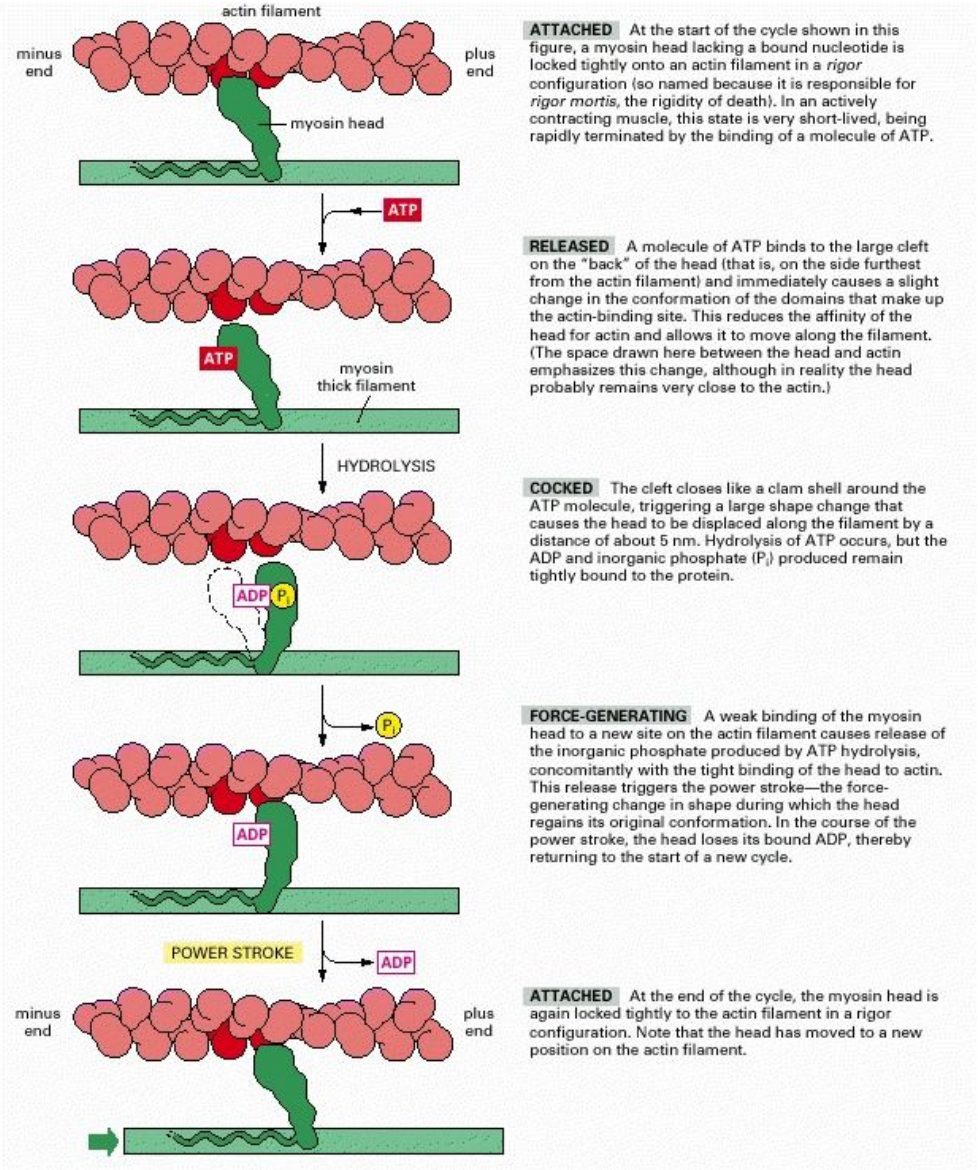
Sarcomere: Thin **actin** filaments with regulatory **troponin** and **tropomyosin** anchored to z-disk. Thin **myosin** filaments which consist of two globular heads and a long tail.

Calcium $^{2+}$ release:

Ca^{2+} stored in the sarcoplasmic reticulum → action potential reaches muscle cells → spreads down T-tubules → conformational change in the voltage sensitive DHP receptor → RYR receptor (Ca^{2+} channel) opens → Ca^{2+} release causes muscle contraction

Cross-Bridge Cycling:

- Hydrolysis of 1 ATP molecule required → rigor mortis without ATP
- Myosin cross-bridge attached to actin → ATP binding leads to dissociation of myosin from actin → shape changed to mysoin head is “cocked” after hydrolysis although ADP and Pi bound → myosin binds to actin + weak initial binding releases P → powerstroke occurs and ADP releases; myosin head returns to low energy conformatino → generates force and pulls actin towards the center of the sarcomere
    - **Tension** (amount of force produced by muscle) depends on proportion of active cross-bridges



Calcium regulation of contraction

Ca^{2+} released from the sarcoplasmic reticulum and binds to **troponin**. Troponin holds **tropomyosin** in lace and blocks myosin binding sites

Tension is a function of Ca^{2+} concentration → relaxation due to CaATPase pumping Ca out of cytoplasm

Excitation-Contraction Coupling:

T-tubules - invaginations of sarcolemma in muscle fiber → AP in muscle fiber conducted into interior of muscle cell along T-tubles → lumen continuous w/ extracellular fluid

DHP is voltage gated Ca2+ channel and directly connected to RYR on SR → pulls open

Action potentials determine rate of Ca^{2+} leaving SR

Gradient is a Ca^{2+} EC gradient, resistance is number of open channels

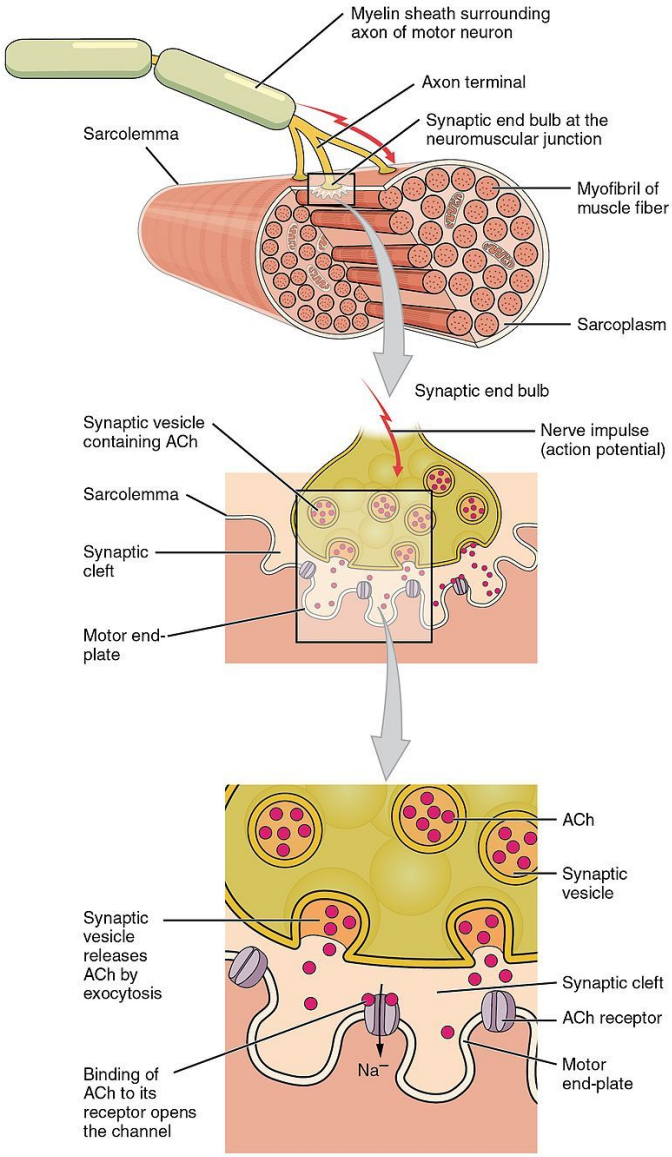
Neuromuscular Junction:

Motor neuron to muscle cell synapse → Ach released → binds to muscle cell → causes "end plate" graded potential = always action potential in healthy muscle cell

Acetylcholine binds to AchR, a cation channel that moves both Na^+ and K^+ → more Na^+ in that potassium out → depolarization

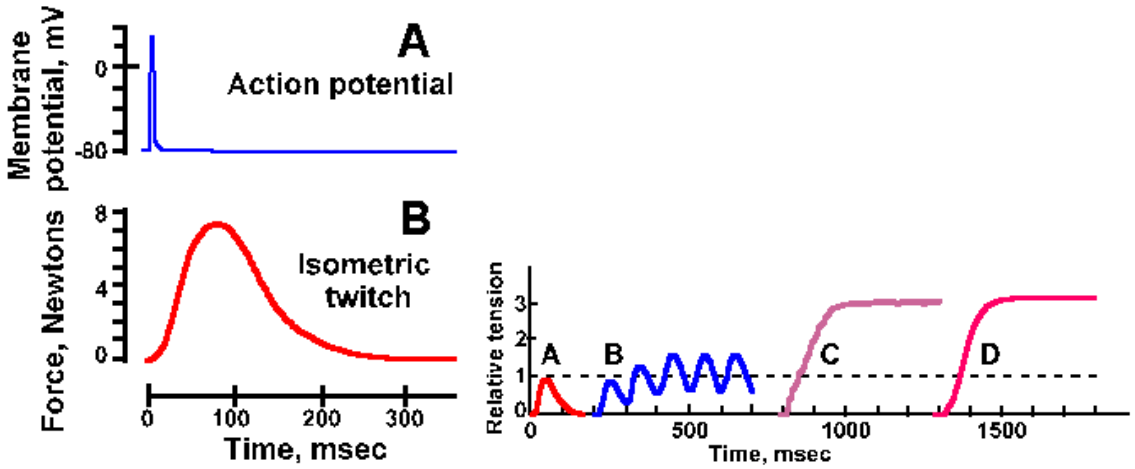
Acetylcholinesterase breaks down acetylcholine

End plate potential: depolarizations of skeletal muscle fibers caused by neurotransmitters binding to the postsynaptic membrane in the neuromuscular junction ---> no AP



- Each muscle cell has one neuromuscular junction = signals from one somatic efferent neuron
- Action potential in neuron releases ACh which binds to DHP and Na^+ enters the cell, causing depolarizing EPSPs above threshold and triggering AP
    - In muscle cell EPSP *always* above threshold so *always* triggers AP → degree that EPSP exceeds threshold = **safety factor**
- **Motor Unit**: A somatic efferent neuron and all the fbers it innervates

Skeletal muscle twitch: single contraction and relaxation cycle produced by an action potential within the muscle fiber itself; doesn't occur in healthy individual



Tetany: Summation of action potentials = tentany → repeated simulation → steady state of tension

Control Systems general model of physiology
Sense + Respond to internal/external environment

1. Stimulus
2. Receptor
3. Input
4. Integrator
5. Output
6. Target
7. Response

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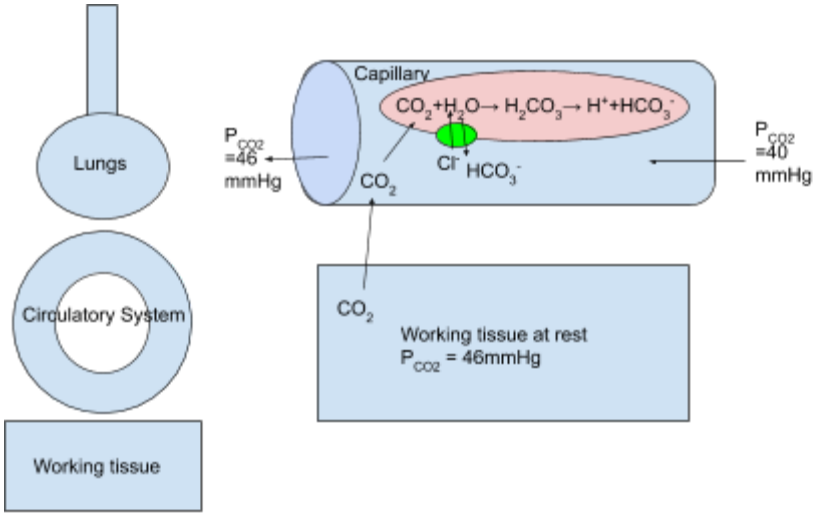
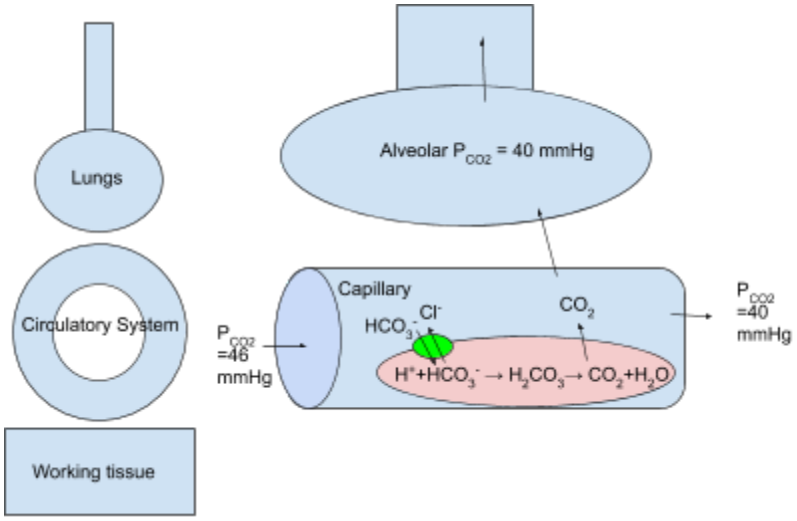
CO_2 and O_2 in the Blood

CO_2 enters capillary then blood cell, converted to bicarb in blood cell through set of reactions
Carbonic anhydrase catalyzes carbon dioxide and water to carbonic acid
Cl- moved in for charge balance

CO2 enters blood and stored as bicarb



CO2 out of bicarb then out of blood at lungs



Bicarb → RBC → H2CO3 → CO2 → plasma → aveoli

Chemosensors: surface of medulla, sensitive to PCO2 and pH of cerebrospinal fluid
Chemosensors on large blood vessels leaving heart sensitive to decreased O2 availability

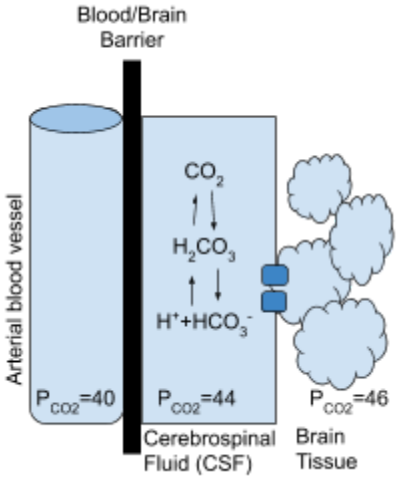
Bodies sense high arterial pCO2 + increase ventilation

- Stimulus: low pH, low CO2

- Receptor: H+ gated sodium channel on central chemoreceptor
- Input: AP from central chemoreceptor (sensory neuron)
- Integrator: respiratory center
- Output: phrenic nerve
- Target: diaphragm
- Response: Increase ventilation

Involuntary Ventillation Regulation: Central chemoreceptors

Amount of CO2 in CSF determined by amount in from tissue and amount out into artery by diffusion (mass balance)



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SA Node cells

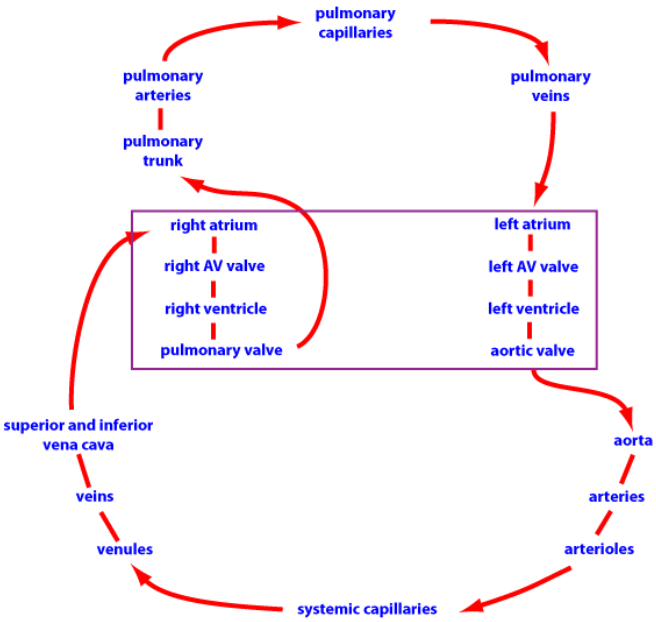
F-Na+ depolarizes cell automatically

Norepinephrine (NE): increases permeability to Na+ in F-Na+ channels → increases heart rate

Acetylcholine: slows V-K closing (stronger hyperpolarization) = lower heart rate (opens K+ channels)

Blood pressure: measurement of force applied to artery walls

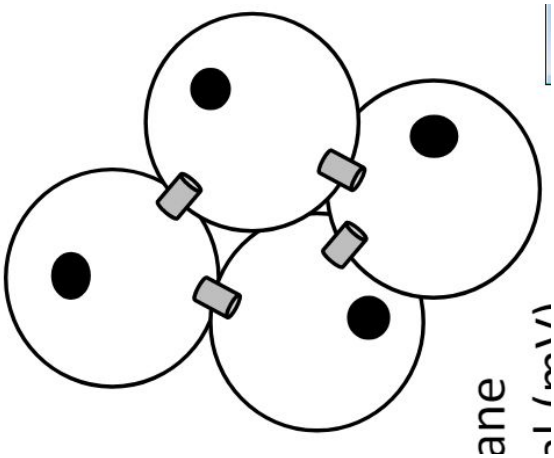
- peripheral resistance, blood volume, and cardiac output
- PR= Blood vessel diameter, blood viscosity, and total vessel length

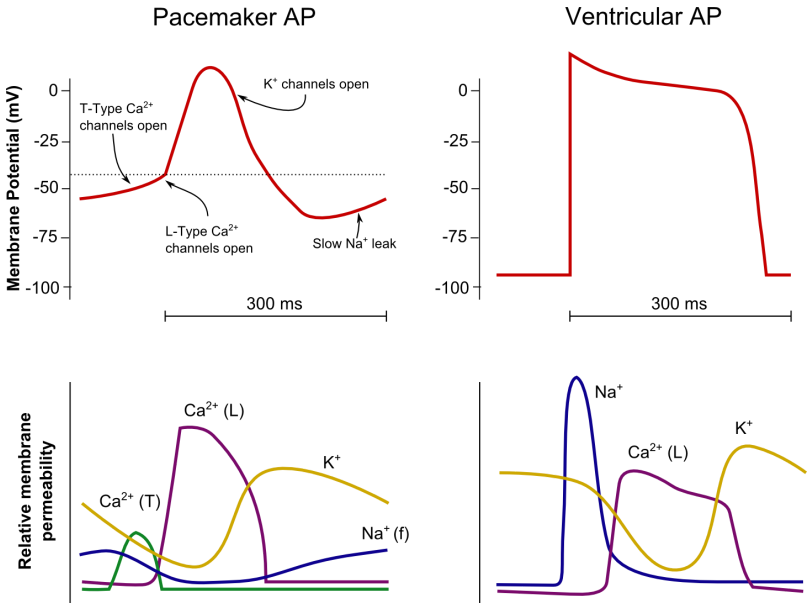


Mean Arterial Pressure:
CO x TPR = MAP
CO = HR x SV

CO = cardiac output
HR = heart rate
SV = stroke volume
TPR = total peripheral resistance

SV changes due to **end-diastolic volume (EDV)** = how much total volume heart has before contraction/end of filling





Frank-Starling: stroke volume of the heart increases in response to an increase in the volume of blood in the ventricles

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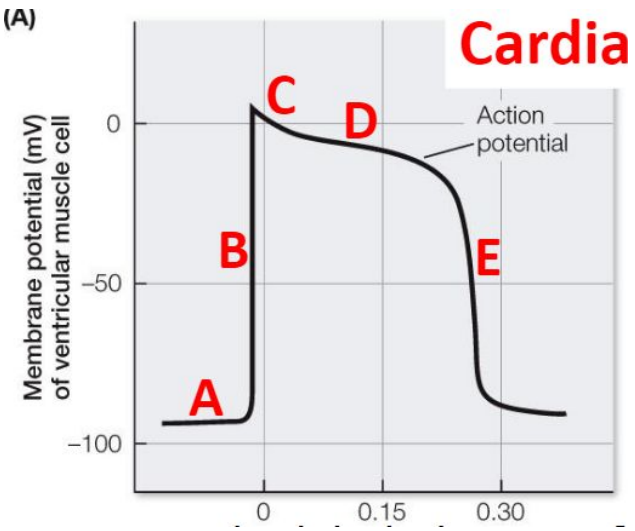
Cardiac muscle cells = different to skeletal muscle but still striated due to sarcomeres
Function: pump blood → intercalated disks, 1 or 3 nuclei, branched

Intercalated disks connect cardiac muscle cells and are critical for the flow of electrical signals from cell to cell and, thus, coordination of heartbeat

Gap junctions = electrical coupling
Desmosomes = provide strength



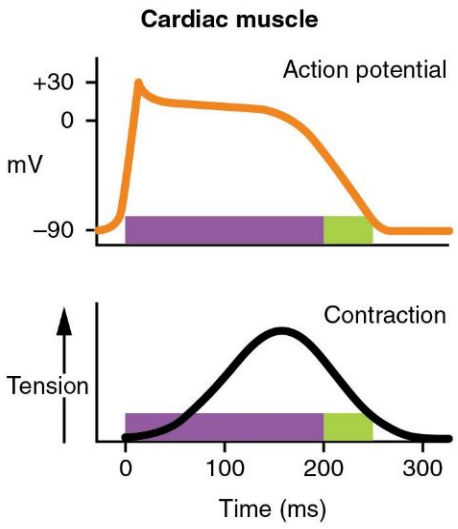
Heartbeat: SA node initiates signal → AV node contracts → bundle of His → perkinje fibers → ventricular cells contract

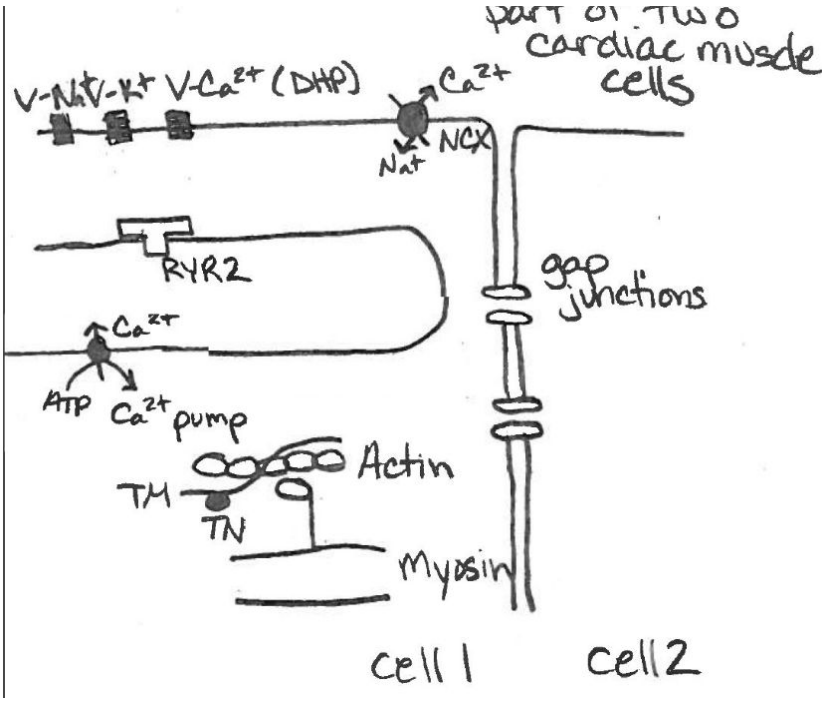
Cardiac muscle cell excitation:

1. V-Na+ opens
2. Na+ enters, increases mV
3. V-Na+ closes, V-K+ opens
4. V-Ca++ opens, V-K+ still open
5. V-Ca++, V-K+ closes

Systole: Contraction while blood pumped into circulation
Diastole: Relaxation as chambers fill with blood

Cardiac muscle cannot reach tetany.





Ca++ enters cytoplasm through V-Ca++ and RYR
Exits through NCX and ATP-Ca++

NE increases stroke volume → binds to NER → RYR stays open longer = more Ca++ in cell = more crosbridge cycling

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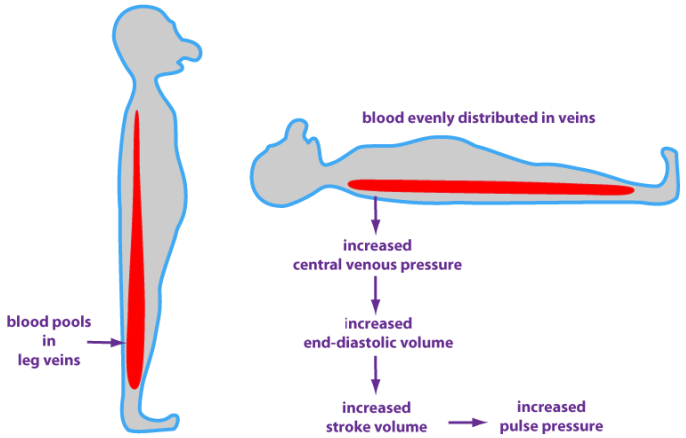
Atrial cells receive signal for AP from gap junctions & SA node
Ventricular muscles receive AP signal from purkinje fibers

Main steps of sketletal muscle contraction:

1. Excitation
2. Ca++ release
3. Contraction/crossbridge
4. Relaxation

Main steps of cardiac muscle contraction:

1. Excitation
2. Ca++ release and crossbridges
3. V-Ca++ Ca in
4. NCX and CaATPase remove Ca++
5. Relaxation



MAP = CO x TPR
CO = HR x SV

HR = SA node, SV = EDV

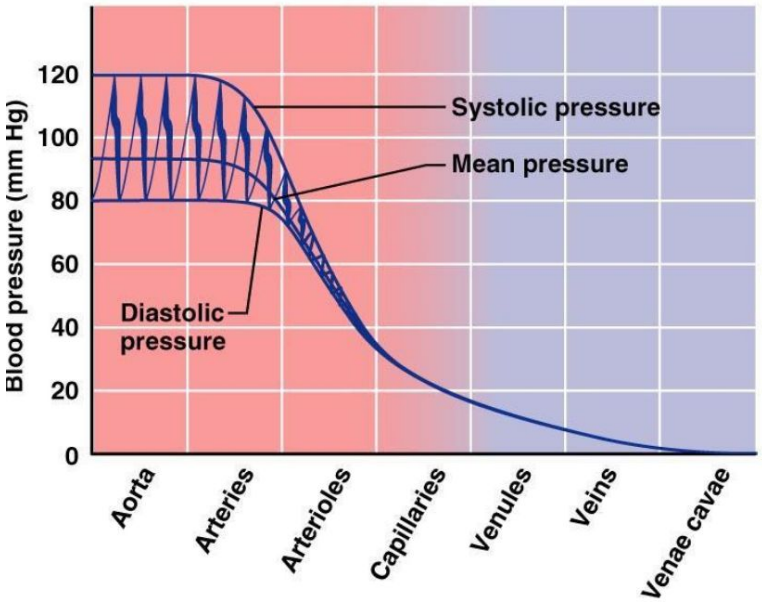
$$TPR \propto \frac{8 \times \text{length of tube} \times \text{blood viscosity}}{Pi \times (\text{vassel radius})^4}$$

Aka driven by **radius of vessel**
→ arteries have highest resistance (largest drop)
→ arterial pressure increases as resistance increases aka vasoconstriction

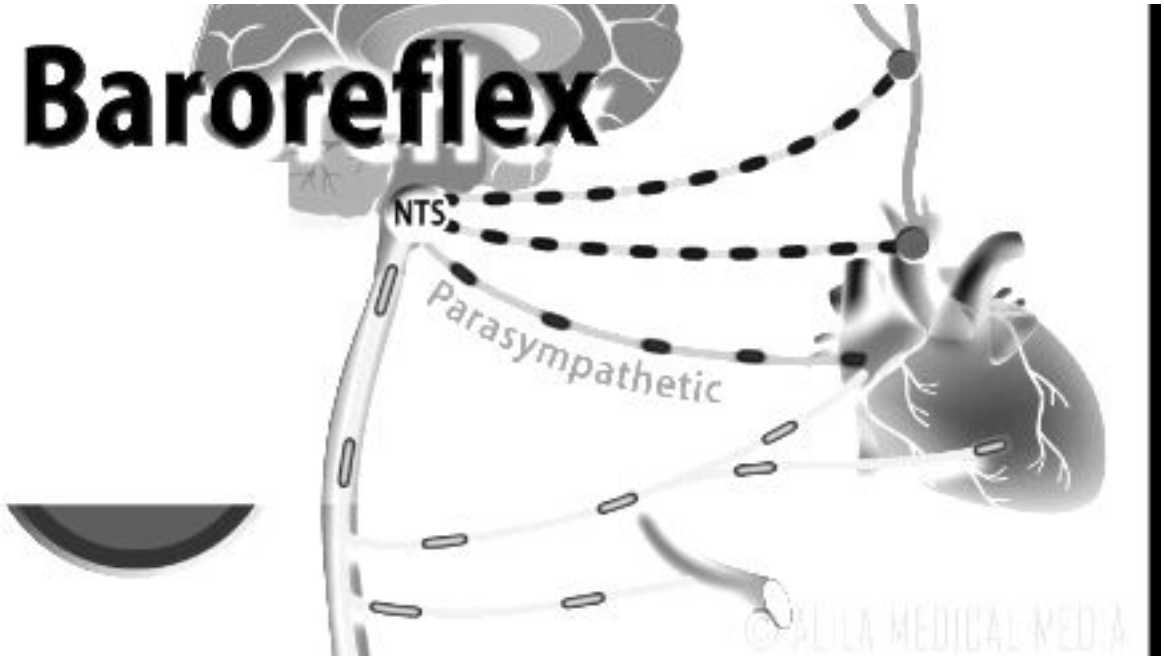
Use mass balance for this

Blood pressure regulated by neurons

1. Stimulus: BP
2. Receptor: Baroreceptor
3. Input: baroreceptor sensory neuron
4. Integrator: Cardiovascular center of branstem
5. Output: Motor neuron
6. Target: cardiac cells
7. Response: Change SV/force of contraction



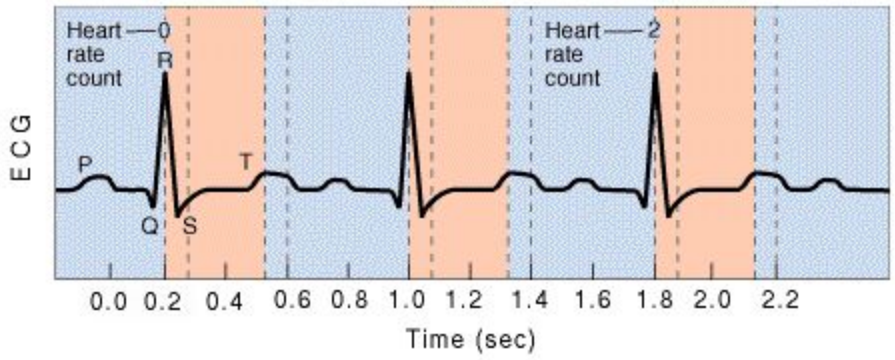
Baroreceptor: functions as a mechanically gated Na+ channel → more stretch = more AP in aorta

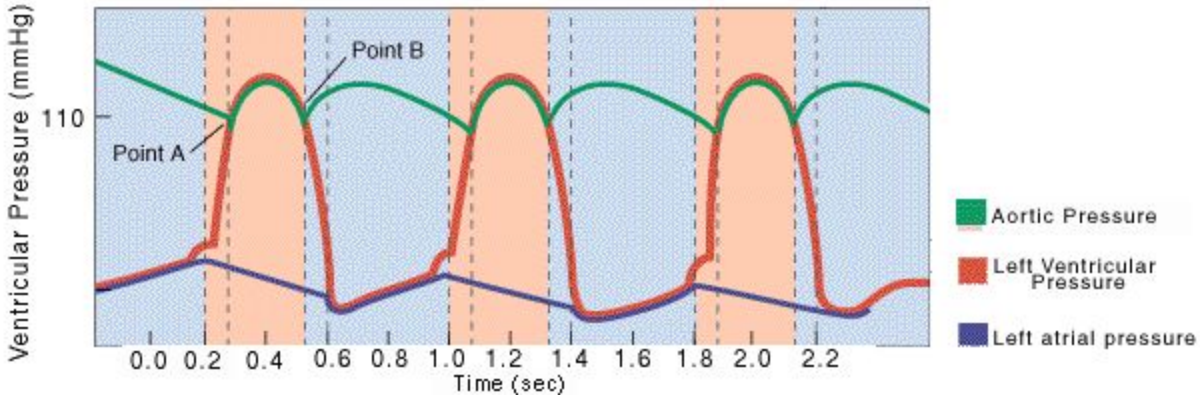


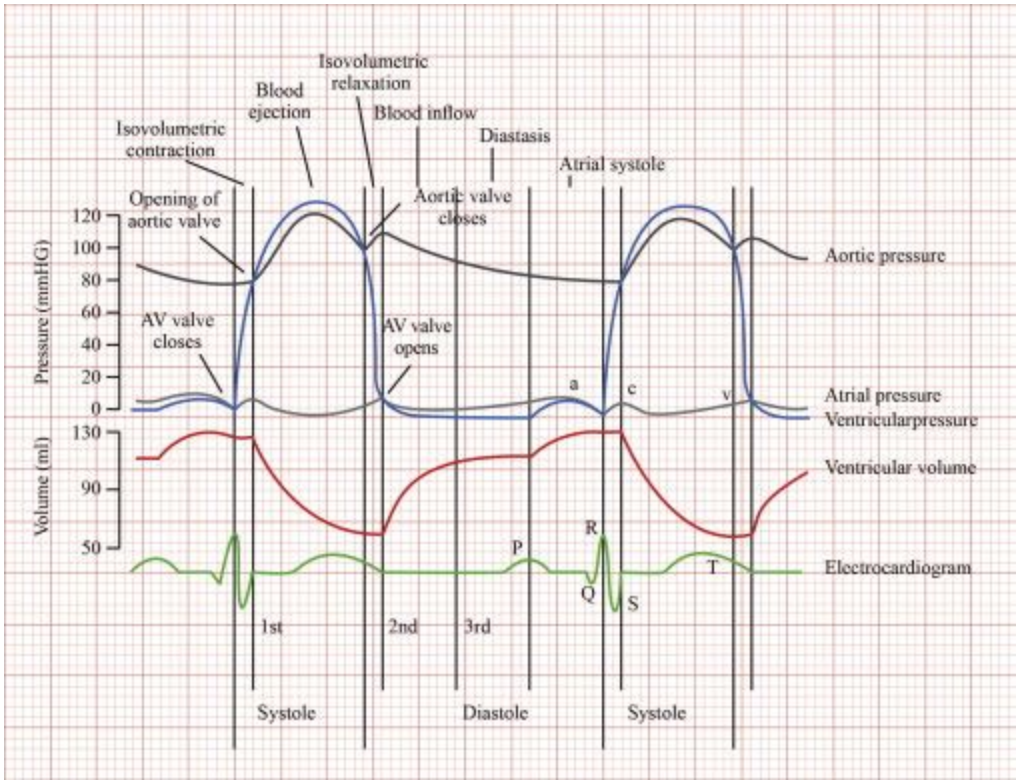
<u>Decrease in blood pressure:</u>
SA node: less ACh → less slowing of HR, more NE → increased HR
Cardiac muscle: NE increases, NER binding increases, more Ca2+, stronger contraction, increase SV
Arterioles: NE increases → more Ca++, vasoconstriction → more TPR

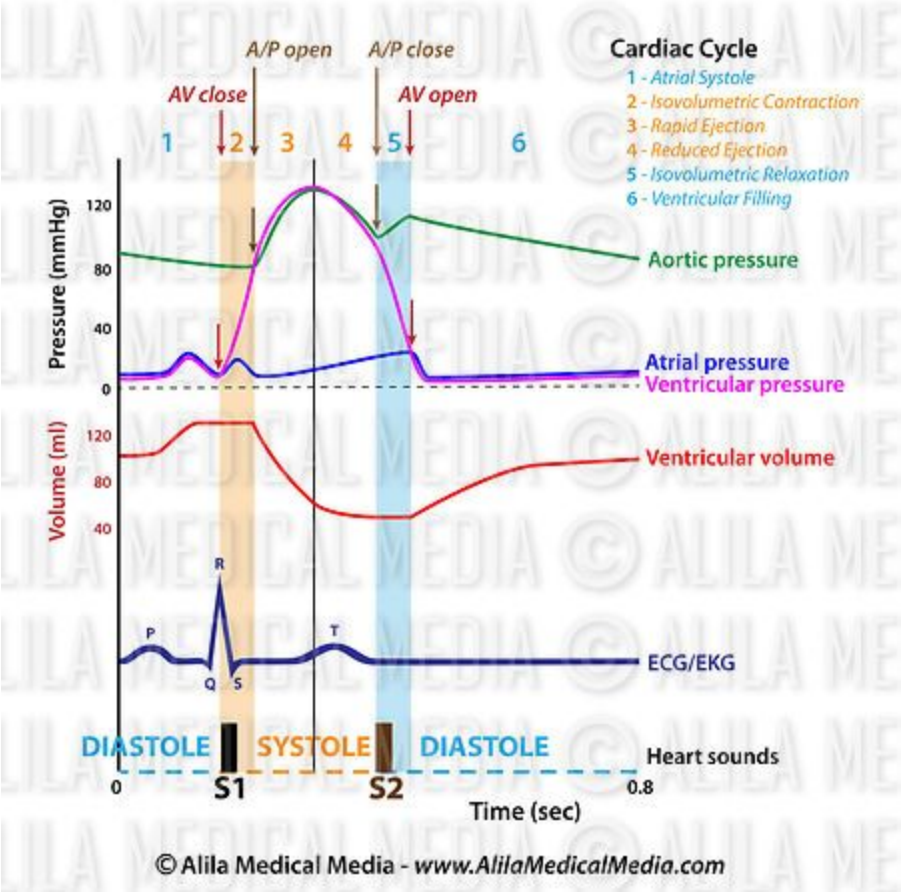
Parasympathetic: ACh → only affects SA node
Sympathetic: NE

Decreased stretch → decrease in graded potentials → less baroreceptor AP → increased sympathetic AP


Heart rate count
0
R
Heart rate count
2
P
T
Q
S
ECG
0.0 0.2 0.4 0.6 0.8 1.0 1.2 1.4 1.6 1.8 2.0 2.2
Time (sec)


Point B
110
Point A
Ventricular Pressure (mmHg)
0.0 0.2 0.4 0.6 0.8 1.0 1.2 1.4 1.6 1.8 2.0 2.2
Time (sec)
Aortic Pressure
Left Ventricular Pressure
Left atrial pressure


Isovolumetric relaxation
Blood ejection
Blood inflow
Isovolumetric contraction
Diastasis
Atrial systole
Opening of aortic valve
Aortic valve closes
120
100
80
60
40
20
0
Pressure (mmHG)
AV valve closes
AV valve opens
a
c
v
Aortic pressure
Atrial pressure
Ventricularpressure
130
90
50
Volume (ml)
Ventricular volume
R
P
T
Q
S
Electrocardiogram
1st
2nd
3rd
Systole
Diastole
Systole


A/P open
A/P close
AV close
AV open
Cardiac Cycle
1 - Atrial Systole
2 - Isovolumetric Contraction
3 - Rapid Ejection
4 - Reduced Ejection
5 - Isovolumetric Relaxation
6 - Ventricular Filling
1 2 3 4 5 6
Pressure (mmHg)
120
80
40
0
Aortic pressure
Atrial pressure
Ventricular pressure
Volume (ml)
120
80
40
Ventricular volume
R
P
T
Q S
ECG/EKG
DIASTOLE
SYSTOLE
DIASTOLE
Heart sounds
0
S1
S2
0.8
Time (sec)
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Lazy Pandas

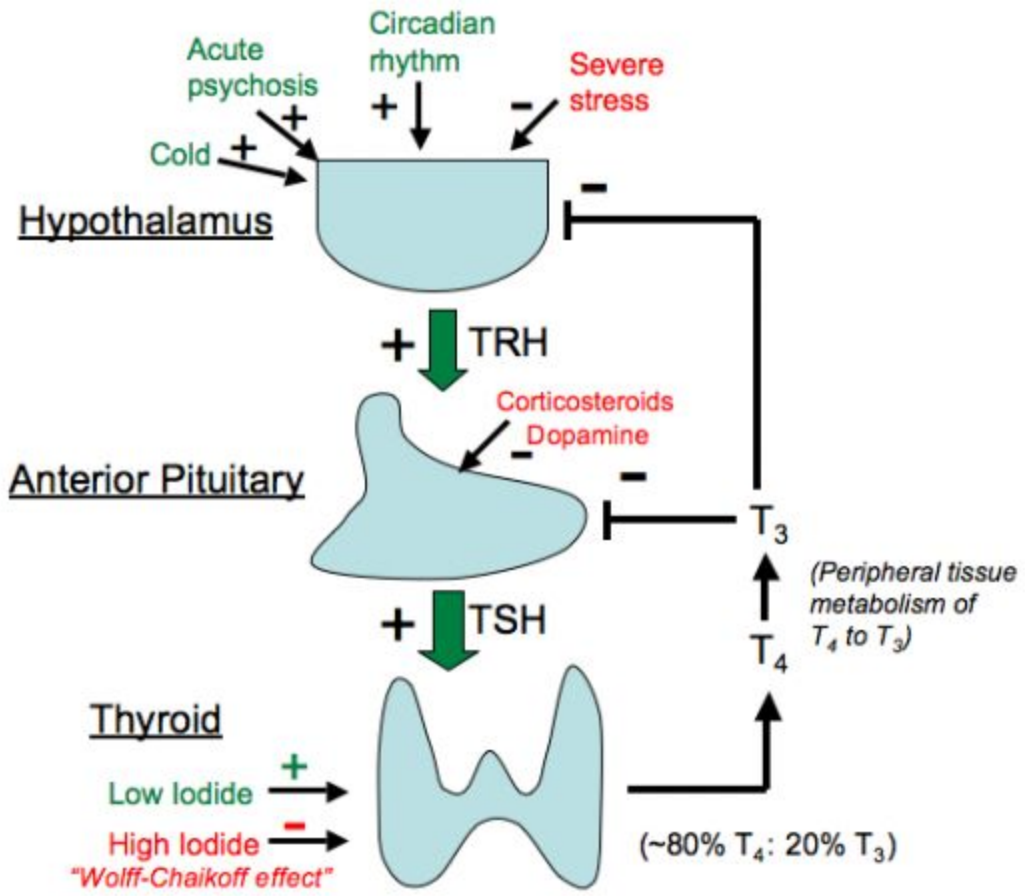
Pandas are in the class carnivora but diet = mostly bamboo → low nutritional quality, specialized digestive system needed

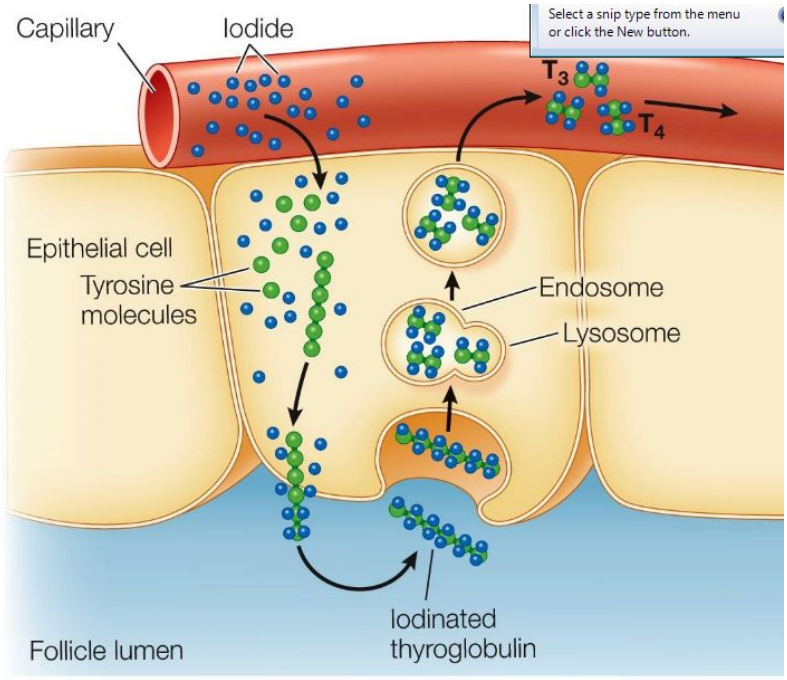
Hypothesis: statement about world views
Prediction: what will be measured/quantified

FALSE morphological changes in digestive tract (long herbivore, short carnivore)
Pandas have shortish → digestive tract not specialized to extract maximum energy from plant material therefore energy in is not greater in rate

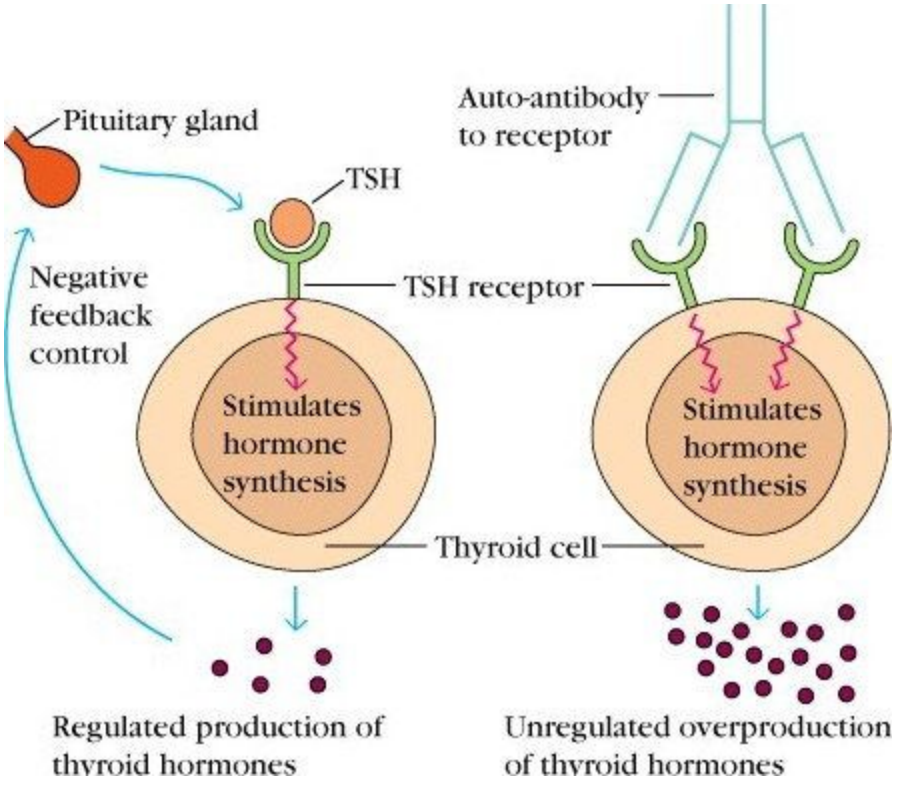
Pandas use less energy relative to other bears/ mammals → lower daily energy expenditure compared to other mammals of same size

How?
Changes in behavior → less activity in wild
Better insultation to reduce lost body heat = less surface temperature
Smaller energy hog organs → brain, liver, kidney…. Heart is bigger than others of similar size
Change in hormone levels (thyroid) = metabolic → low t3/t4





Goiter: low T4, above normal + goiter
→ lots of thyroglobulin in lumen


Pituitary gland
TSH
Auto-antibody to receptor
Negative feedback control
TSH receptor
Stimulates hormone synthesis
Stimulates hormone synthesis
Thyroid cell
Regulated production of thyroid hormones
Unregulated overproduction of thyroid hormones