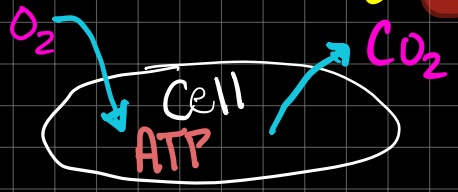
Bio-336 Test2
SDSU '22

# Cardiovascular System

## Pump

Evolved due to multicellular animals needing $O_2$ to each cell



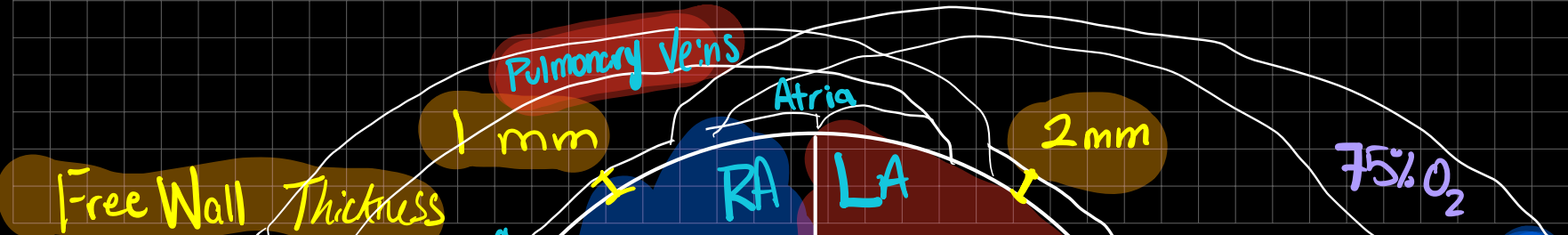

## Blood vessel

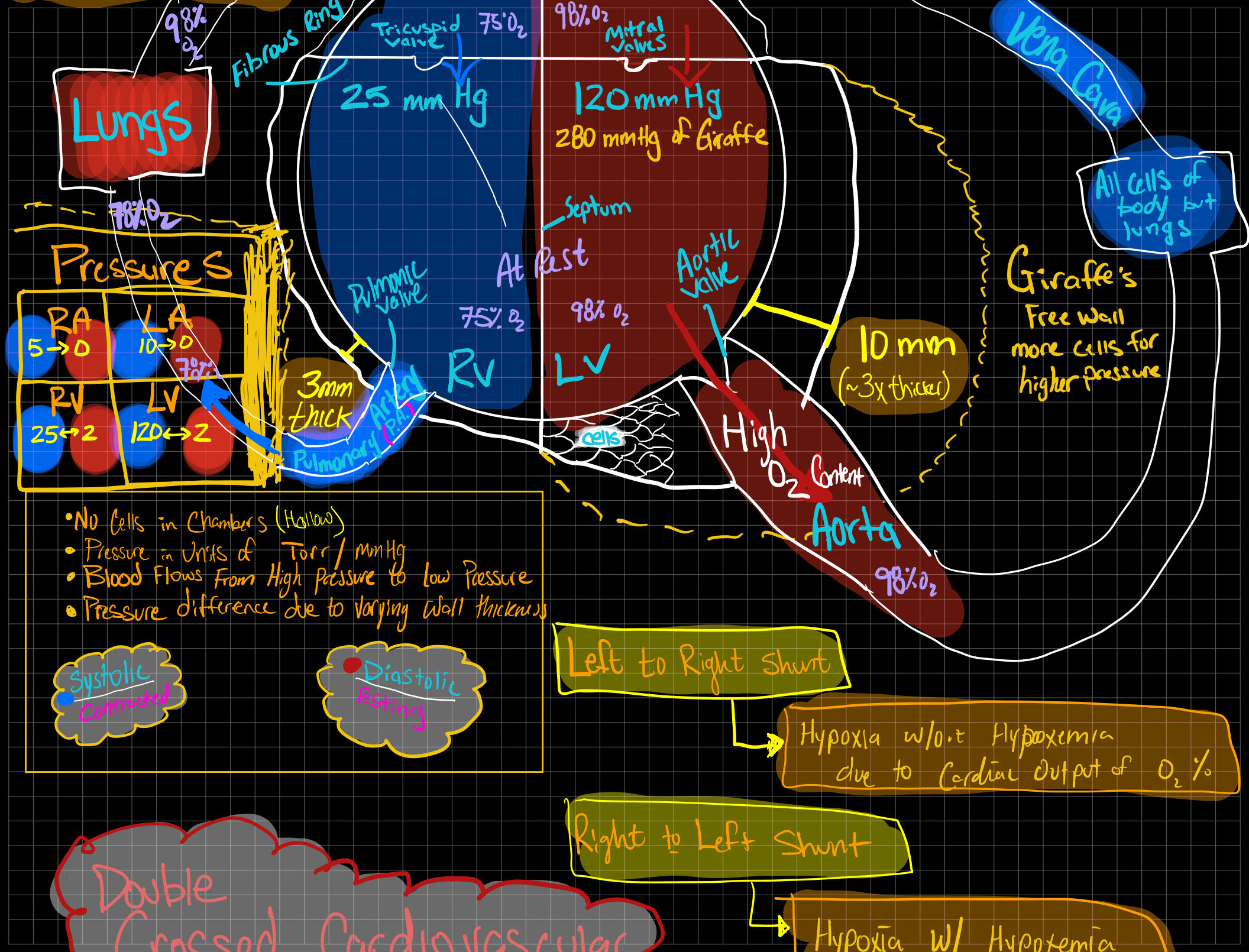
93% or lower

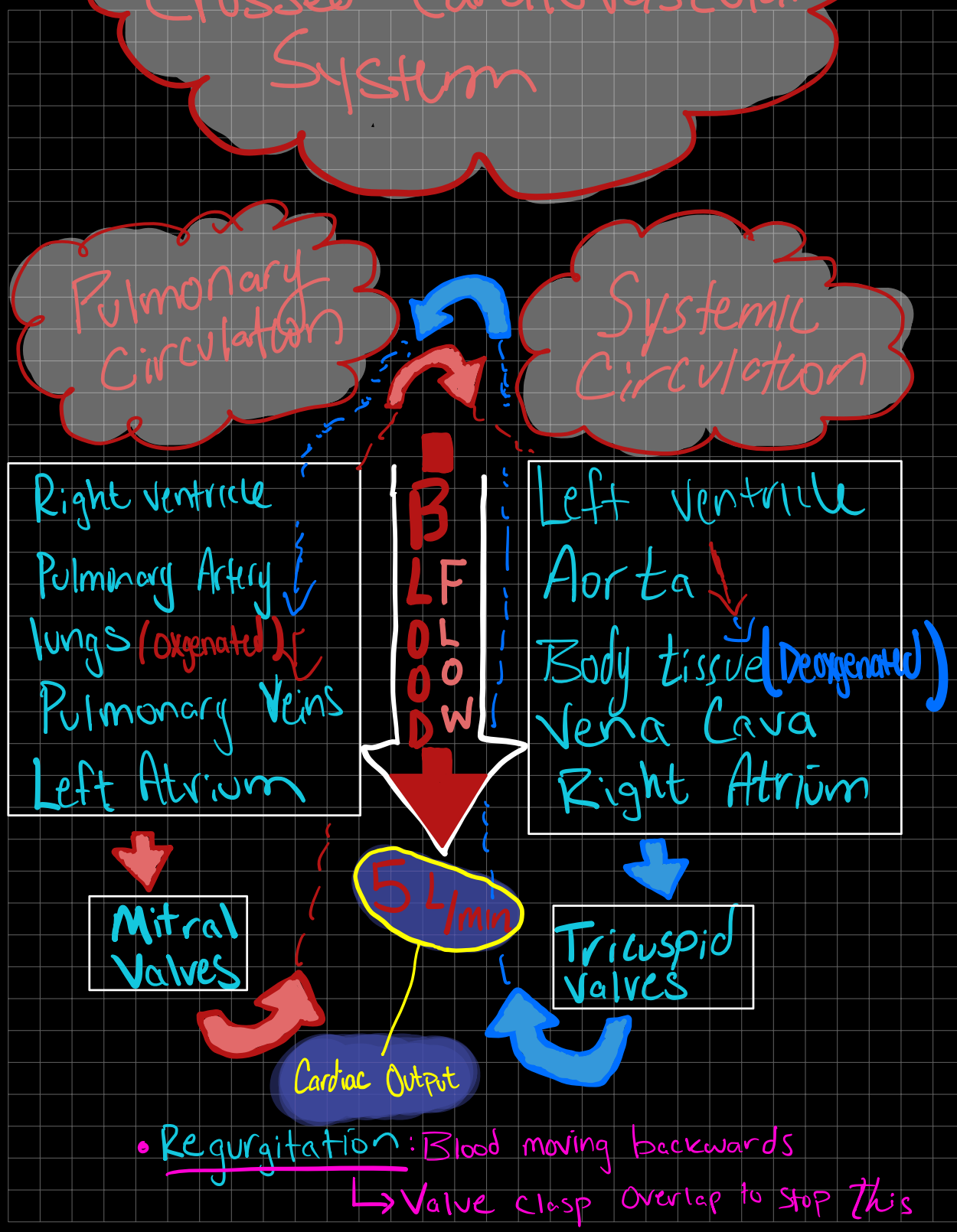
**Hypoxemia**: Lack of $O_2$ in Blood stream. Leads to Hypoxia. Measured by $O_2$ Saturation

**Hypoxia**: Lack of $O_2$ at Cells (mitochondria) and decrease in ATP production

> no more $Na^+/K^+$ ATPase Activity; Decrease in Action Potentials




Lungs
98% $O_2$
Fibrous Ring
Tricuspid Valve
75% $O_2$
25 mm Hg
98% $O_2$
Mitral Valves
120 mm Hg
280 mm Hg of Giraffe
Vena Cava
All cells of body but lungs
78% $O_2$
Pressures
RA 5→0
LA 10→0
RV 25↔2
LV 120↔2
Septum
At Rest
Pulmonic Valve
75% $O_2$
RV
98% $O_2$
LV
Aortic Valve
3mm thick
Pulmonary Artery (PA)
cells
10 mm (~3x thicker)
Giraffe's Free wall more cells for higher pressure
High $O_2$ Content
Aorta
98% $O_2$
•No Cells in Chambers (Hollow)
• Pressure in Units of Torr / mmHg
• Blood Flows From High Pressure to Low Pressure
• Pressure difference due to varying Wall thickness
Systolic Contracted
Diastolic Resting
Left to Right Shunt
Hypoxia w/o.t Hypoxemia due to Cardiac Output of $O_2$ %
Right to Left Shunt
Hypoxia W/ Hypoxemia
Double Crossed Cardiovascular




- Regurgitation: Blood moving backwards
  - ↳ Valve clasp overlap to stop this

due to $O_2$ & Cardiac Output

- Biological Valves: Pigs or humans
  - → NO blood clots
  - → 25yr lifespan
  - ↳ dealing w/ left ventricle
- Blood clot is a Thrombus
  - → A piece broken off, now in bloodstream
  - → Creates an Embulism
- Endothelium - Cells along blood vessels
  - ↳ NO into bloodstream to stop clotting
- Damage to Blood vessels causes Thrombuses
- Mechanical Valves: Manmade (Metal)

- **dyspnea**: Difficulty breathing b/c Mitral regurgitation
- **Phepato meglia**: Liver swelling.
- **Leaflets**: another name for Valve Clasps.
- **Pittededema**: Swelling of body parts due to tricospid regurgitation
- **Semi-lunar Valve**: Aortic & pulmonic valves
- **Aortic / pulmonic / Tricuspid**: 3 leaflets (more leaks)
- **Mitral valve**: 2 leaflets (Harder to Open)
- **Stenosis**: Narrowed open valve

## 4 Congenital Heart Conditions

### ① Transposition of the Great Arteries

→ Flipped positions of Aorta & pulmonary Vein

- Blood develops Hypoxemia / Hypoxia

→ **Ross Procedure**: Arotic valve regurgitation: Body Tissue → Hypoxia

→ move pulmonic valve to aortic & put a biological (AKA Pig) Valve

→ Unlimited lifespan

→ Need prescription for drug to Stop Thromboemboli

- Anticoagulant → **Warfarine**
- → Vitamin K Antagonist

- Blood going back to heart → Venous Return (2 veins)

@ cells

- Systemic & Pulmonic circuits don't cross so no reoxygenation by lungs

in Pulmonic Spot

★ (for longer lasting)

★ Because pressure is lower in Right Ventricle

Pig Valve can sustain pressure longer

## ② Ventricular Septal Defect

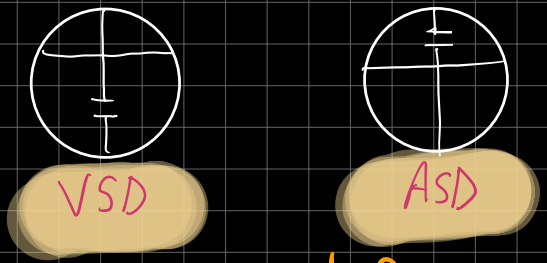
→ Hole in the Septum (lower half)

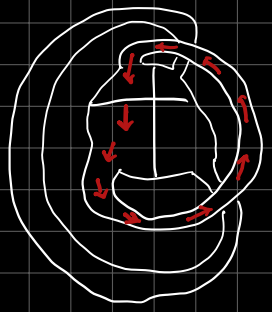
→ L.V. blood to R.V. (due to pressure difference)

→ less blood down to Cells/tissues

## ③ Atrial Septal Defect

- Hole in Septum (upper half)



★ A Septal Hole can be used for ① to allow Oxygenated blood to cross

↳ Septostomy - Bridge to Surgery

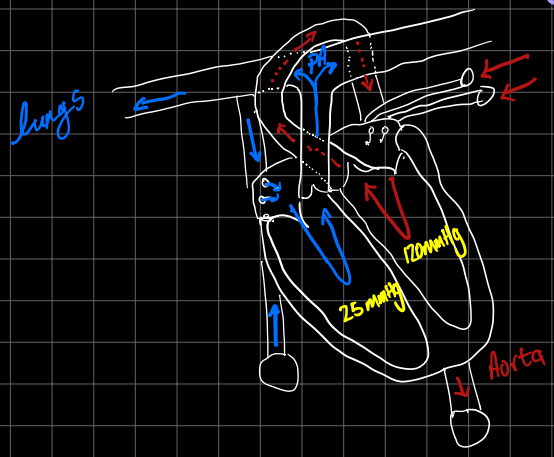
L→R ⟶ 98% Oxgenation restored = Hypoxia

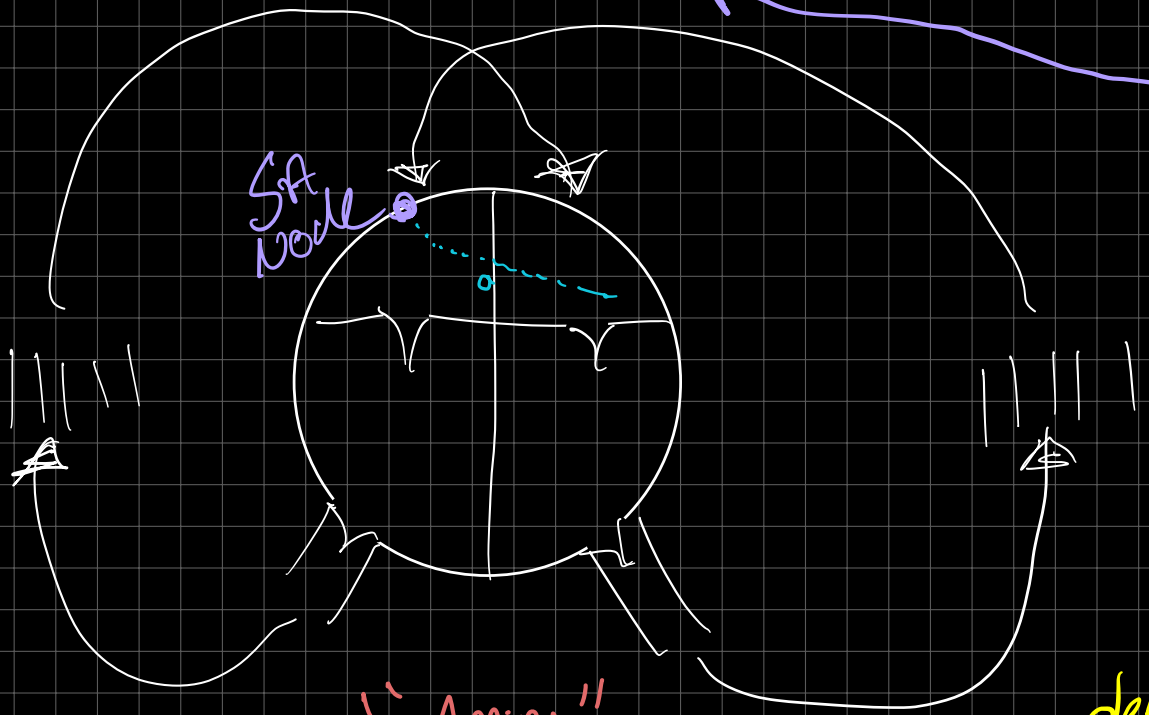
R→L ⟶ decrease in Oxgenation % = Hypoxemia + Hypoxia

# 4 A Tricuspid Mitral Valve

→ Results in leaks or "Backflow" into Right Atrium (Regurgatate) then ↓

→ Oxgenated Blood Regurgatated into lung developing Dyspnea - Difficulty Breathing B/C




Heart failure

"Amine"

Dopamine

depolarizes faster

Positive Chronotrope

Negative Chronotrope

Make funny Channels open faster

Make funny Channels open slower → "olol"

- S-A node (in wall of R.A.)
  → Special group of cells that have...
  "Funny Sodium Channels"
  → Open 1x per Sec.

Slows heartrate lowers Blood pressure

Lecture ②

Resting Heartbeat 60-120 Beats/Min

Bradycardia - Heartbeat < 60 b/m too slow

Tachycardia - Heartbeat > 100 b/m too fast

Each bpm = 1 Cardiac Cycle

Shock - low blood pressure leading to Hypoxic Cells

# Phases of the Cardiac Cycle

① Passive Filling (140 mL: End diastolic volume)

→ Venous return of blood to atrias

→ Volume ↑ Pressure ↑ higher than Pressure in V

→ Mitral/tricuspid valves open to fill V's to 80%

Systole Con. Demarcater: S.A. node Action Potential → gap Junction → Atrial Contraction "Atrial systole

② Active Filling (70 mL: end systolic volume) dif between ① & ②

→ funny Channels open → S-A node depolarizes

→ Sequential Contraction of cells across Atriums from SA Node by Gap Junctions

→ 20% more blood into Ventricles

Demarcater: Sound ① - Closure of AV valves.

Fibrous ring has no gap Junctions

→ 60 B/m / A.P. on its' own

// → Gap Junction

— → A.P. travel through — Cells

→ Sequential Contract

- Norepinephrine
  - → funny Channels open faster
- Acetlycholine
  - → funny channels open slower

- As you age you lose funny channel
  - → Sick Sinus Syndrome

SA Node → Gap Junctions → AV node ← Simultaneously

A-V node Bundle of HIS Tissue piercing the fibrous ring

↳ • Bundle of His

↓

Splits to 2 sides

↓

• Bundle Branches

•Purkinje fibers
•: all large in in diameter

③ Isovolumetric Contraction (Simultaneous)
→ HIS receives Av node Action Potential & Spreads across Fibrous ring
→ BB splitting & Action Potential travels down to Purkinje fibers
→ All heart cells recieve Action Potential at Same time (All (4) Valves closed)

Demarcater: Opening at Semilunar Valves
↳ Aortic + Pulmonary Valves

④ Ejection Phase (70 ml/beat)
→ Pressure in Ventricles exceed Pressure in Semilunar valves
→ Pressure(↓) blood is ejected out into the Pulmonic & Aortic Arteries

Demarcater: Sound (2) - Closure of Semilunar Valves

⑤ Ventricular Relaxation
→ Venous Return
Demarcater: Av Valves open (Mitral & tricuspid)

★ Stroke Volume - Amount of Blood ejected (70ml/Beat)

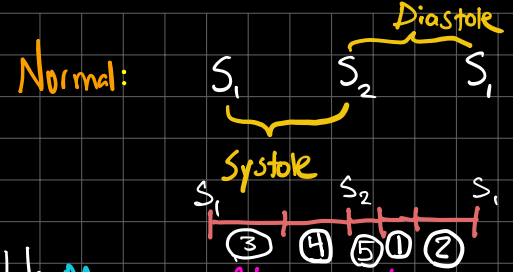
| | |
|---|---|
| End diastolic volume | EDV - ESV Stroke volume |
| End Systolic Volume | 140 - 70 = 70 |

Ejec Fraction - SV/EDV


Aortic Stenosis: Narrow Aortic valve lowers stroke volume

Bundle Branch Block: One branch isn't working → Action Potential not Simultaneous across cells
↳ Weak Contraction

Murmer: A sound by the heart, other than $S_1$ or $S_2$ → From damage to a valve
- Stenotic Valves: Make a sound when they are open / fluid turbulance
- Regurgitant Valves: Make a sound when they are closed / fluid turbulance




4 Murmers: All come in 6 stages ( Grade 1 is lowest & Grade 6 is the worst)

(Higher Stroke Vol) (More dysp)

① Mitral Regurgitation

$S_1$ wwww $S_2$ AV valves close

- Pan Systole
- Systolic Murmer After
- less than 70 $m^2$/min → dyspnea & Hypoxia

② Mitral stenosis

$S_2$ mmm $S_1$ AV valves open

(late)

- Diastolic Murmer

③ Aortic Regurgitation — SV same C.O. decrease

$S_2$ mm $S_1$ Semilunar Valves Closed

(early)

- Diastolic Murmer

④ Aortic Stenosis — Ejection Phase Semilunar valves open

$S_1$ ww $S_2$

(late)

- Systolic Murmer

Ross Procedure

Pig valve → Pulmonary → Aortic

Heart Failure: EF less than 40%

(Sys) Can't Contract ESV↑
(dias) Can't fill EDV↓

60-100 normal heart beat

Thrombus - Clot in heart
Embolus - Piece of Thrombus

| | | | |
|---|---|---|---|
| Positive Chronotropic Drug: | Increase heart beat → Tachycardiac | Time | - Amine |
| Negative Chronotropic Drug: | decrease heart beat → Bradycardiac | | -olol |
| Positive Inotropic Drug: | Increases beat strength → SV & EF↑ | Force | - Amine drugs |
| Negative Inotropic Drug: | Decreases beat strength → SV & EF↓ | | - olol drugs |

- Amine Drugs work on funny channels / SA node (slower)
- Propranolol works on Cardia Output
- Multiple drugs will = better results
- SSS (Sick Sinus Syndrome): SA Node loses funny channels → Slower Depolarization
  - ↳ Slower heartbeat
    Bradycardia

- Ep: pen / Epinephrin - Positive Chrono / Ino (↑ C.O. + ↑ BP)
- Nifedipin - Vasodialators

## Cardiac Output

~5,000 ml/min Avg.
as high as 40,000 ml/min

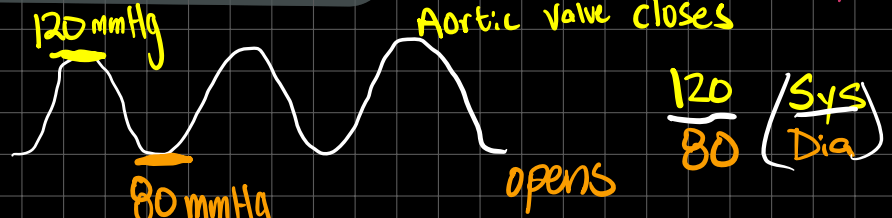
3/4 fold 2 fold

Beat x SV

$\left(70\frac{\text{Beats}}{\text{min}} \times 70\frac{\text{mL}}{\text{beat}}\right)$

## Blood Pressure ↑ in Arteries when Aortic Valve Opens

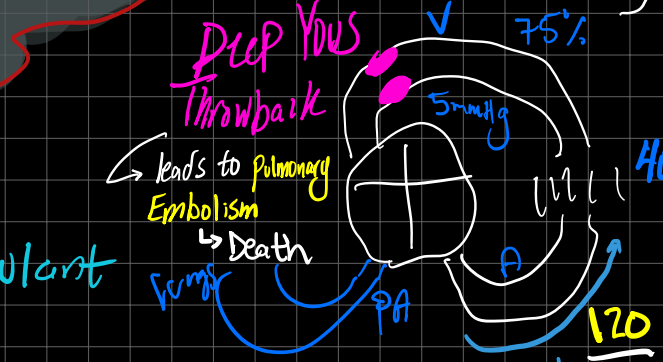
(systolic Blood Pressure)




Warfarsn
↳ stop Thrombus

Heprin
↳ Anticoagulant

Deep Vein Thrombosis
↳ leads to Pulmonary Embolism
↳ Death




Pressure ↓ as Energy loss
Blood Pressure Gradient

40 mmHg

Brain 750 ml/min CBF

Kidney RBF 1000 ml/min

## Blood Pressure Gradient

120 → 40 → 5

## Atrial Fibrillation

- multiple pacemakers
  - Bc Atrials not Contracting fully
    - ↓ SV, static blood / stasis

Hypertension: 130/80

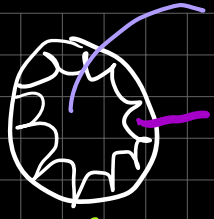
- Atherosclerosis

Very bad b/c damage to Endothelium
→ ↓ NOS
→ Thrombus

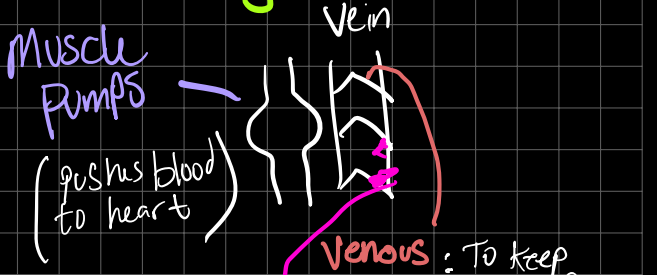
- Atrial thrombosis → Brain (Ischemic (thrombotic Stroke) 2/3

Hemorrage Stroke 1/3 : Blood vessel ruptures
- ↓ Blood to tissue
- Hernia

tPA: Breaks down embol: (thrombolytic)




Cross section of Artery




# How to Lower Blood Pressure?

→ Ohms Law

- $Bp = C.O. \times T.P.R$ (restriction) $R \propto \frac{1}{r^4}$

(Total peripheral restriction)

Size of all blood vessels

- Cells around vein: Vascular Smooth Muscle (VSM)

Vasoconstriction: Constrict
↳ smaller blood vessels
↳ more T.P.R.

Vasodilation: Relax
↳ Wider blood vessels
↳ less T.P.R.

BLOOD PUMP

Artery
thick NO loss of $O_2$

Capillary - Endophilo Cells
Allows for $O_2$ & $H_2O$ Exchange

- Blood Pressure is directly proportional to C.O. & T.P.R.

★ Give olol drugs to lower B.P.
-or- Vasodialators (pine drugs)

↳ Nifedipine: Causes VSM to dialate
↳ ↓ Decrease T.P.R.

(Too much = Syncope)
↳ too low BP
↳ light headed -> Pass out

★ Cannot pump blood against Gravity ★

Vein
Endothilia & VSm

Diuretics
thiazide
↳ Diurisis
↳ Produce more Urine
↓ Blood Volume
↓ SV

Ace inhibitors "Prill"
-> Decrease in Angiotensen II ↓R ↓P lowers → Angiotensen II

Calcium channel blockers
lesartan

VSM


**Hematocrit**: % of Blood made of RBC

Other $Na^+$, $K^+$, $Ca^{2+}$
90% $H_2O$
6-8% Proteins

Male: 40-45%
Female: 35-40%

**Anemia**: less than 35% Crit
- Risk for Hypoxia

**Polycythemia**: 50%
- Greater risk for thromboembol:

# Blood Volume: ~5L (2L RBC & 3L Plasma)

# Plasma: 90% $H_2O$ & 7% Plasma Protien

Mostly **Albumin** (made in Liver)

## Red Blood cells lifespan: 100 Days

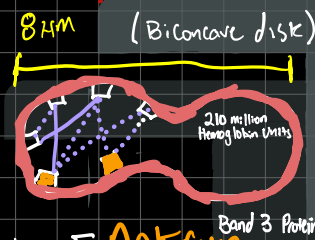
① → **Erythropoiesis**: production of RBC Caused by Enzyme: **erythropoietin**: produced by Kidneys to do 7 day process

② → **Erythrophagocytosis**: Destruction of RBC

① < ②↑ = **Hemolytic anemia** - High bilirubin, low crit, High RC

## Red Blood Cell

(Biconcave disk)




- ↑ SA & Gas Exchange
- Flexibility to fit through 7 μm blood vessels
- Rubs on Endothelium & Allows release of NO (Nitric Oxide)

Biconcave disk [ Ankryn / Spectrin

lack of proteins lead to Dismorphic

* → Sickle Cell Anemia - Trapped in Capillaries & restrict Blood Flow

* 1 amino acid in B chain is different
- No Nucleus → NO Duplication
- No Mitochondria
  ↳ NO taking $O_2$, gives to other cells

**HemoGlobin Units** (RBC)
- 210 million $\alpha_2\beta_2$

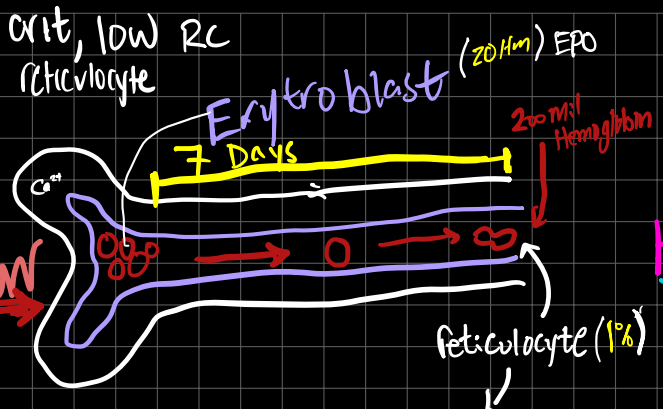
Ex. Heme { Bchain, achain } Tatramer

→ **Globins**: Protien Chain

Each Hemoglobin has 4 protein chains & 4 Hemes

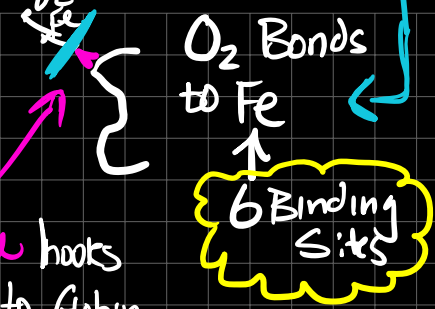

1 $O_2$ - 1 heme - 4 Nitrogen bonds to keep Fe in middle

↓① < ② = Hypo-Prolific anemia - low crit, low RC
- determined by reticulocyte
- (low)




▲ Stem Cells In Red Bone Marrow
↳ Start w/ 0 Hemoglobin

Reticulocyte Count (RC): 1% (Healthy)




Histodine hooks Heme to Globin (α & β Chains)

Sickle Cell Anemia:
Non-flexible cells get stuck in Sleen more often
→ Hemolytic anemia

Iron Deficiency Anemia: Not enough Fe to have efficient Erythropoiesis
↳ Hypoprolific anemia
↳ low Hemotocrit & R.C.

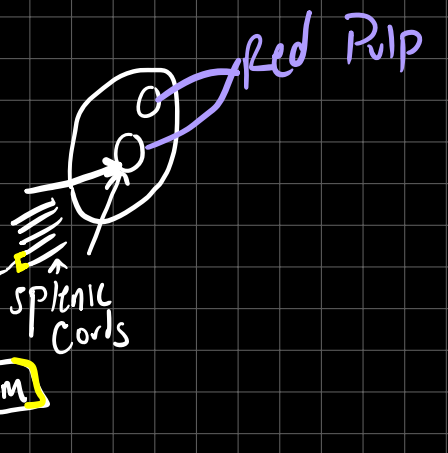
Renal Failure

Metastatic Bone Cancer: Came from a different Area & Destroys stem cells
-Destroys Bone Marrow-
↳ Hypoprolific

■ Procrit: Recombinant EPO, to prevent anemia → too much EPO led to Polyethemia & thrombe embilis (for Hypoprolific)

# Erythrophagocytosis

② Splenic cords in red pulp of the spleen




- 100 day old RBC lose their flexibility due to defective Ankyrn & Spectin and get stocked in these folds

- Amino Acids are released in the Spleen from the α & β Chains
- The Heme left over is turned into bilirubin: Too much turns you yellow → Jaundice
  - → Broken down RBC from Heme

- Hyperbaric chambers used on people who become Hypoxic ($O_2$ Chamber)

Our Blood

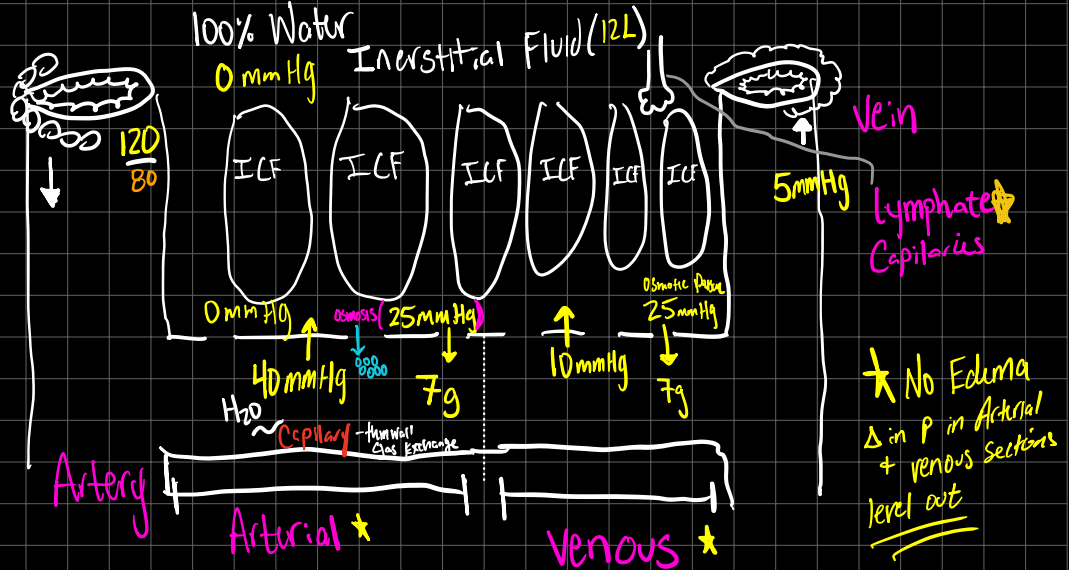
7g = 25mmHg

↳ Plasma Proteins

7% Albumin

93g or 93% $H_2O$

- Capilaries only have endothelium, NO VSM, → Allows $O_2$ & $H_2O$ to leave vessel (Cannot leave arteries or viens)
- As Water is lost, Hemocrit ↑, → DVT




- Hydrostatic pressure: Forces $H_2O$ out Capilaries @ 40mmHg
  - → Osmosis: Plasma proteins bring $H_2O$ back in 25mmHg
- An Increase in interstitial space: Edema (Swollen)
  - → decrease ability for $O_2$ to diffuse into Cells → Hypoxic Cells

# 5 Causes to Edema (Swelling)

- 100% → 93% high to low → Osmotic pressure

## ① Hypertension Increase Blood Pressure

→ High Arterial Pressure / more $H_2O$ into interstitial fluid

→ High altitude Pulmonary, High altitude Cerebral, Edema → Nifedepine (Vasodialator)

**Starting Forces of Capilary Fluid Exchange**

- ★ Arterial · B.P. > PPOP → Forcing more Water out that can be brought back in
- ★ Venous · B.P. < PPOP → more Water in than can be forced out
  (10mm Hg due to frictional losses from RBC)
- Water in the blood / interstitial space are the Same

## ② Decrease Plasma Proteins

→ Osmotic pressure decrease

→ Kwashiorkor: lack of proteins in diet

→ liver or renal failure (urinate out)

→ Pregnancy, Alcoholism

## ③ Increase Capilary permeability

→ Plasma proteins decrease (into interstitial) (less venous return) [↓C.O. ↓B.P.]

→ Cell damage / injury: produce Histamine

→ Allergies → anaphalcotic Shock: Edema All over Histamine Everywhere

Stored in Mast cells

Histamine: Makes Capilarius Permeable so White Blood Cells can go out & Help (lose plasma proteins)

## ④ Decrease venous return

→ Pregnancy / venous Capilary B.P. increase (More $H_2O$ out)

→ lay down / feet up

★ Extra → Pitted Edema → 4 grades 1+ 2+ 3+ 4+

↳ Pit formed by Heart failure

→ diuretics + ⊕Ino/cronotropes

## ⑤ Lymphedemda ★ (Blocked or deficient)

→ Damage to lymphatic ducts / capilarius (Comeback 1% of fluid)

→ Surgery / radiation / mosquito bite → Elaphantisis → bad wound healing → Systymic infection by phalarisis

Parasite → Death

→ manual lymphatic draining. Force fluid to go back into ducts by Compressing Area

- Easier for foreign bodies to end up into "R" lung (Aspiration of the R lung)

Trachea - Tidal breathing

Apex

Tracheal rings throughout

R (three lobes)

L (two lobes)

"L" Main Branch

"R" main Branch

Intrapleural Space

(Neg) Pressure 755 mmHg

Intra Pulmonary Space

760 mmHg

Pleural Pleura

Thoracic Cavity

Visceral Pleura

Sternum

Cardiac Notch (L ventricle Space)

Oblique Fissure

Horizontal Fisher

Oblique Fisher

upper

middle

lower

Mediastinum

762
760
758

$P \propto \frac{1}{vol.}$ Boyle's law

(Inspiration) Contracting diaphragm → lung Vol ↑, P ↓ (air in)

(Expiration) Relax diaphragm → lung Vol ↓, P ↑ (air out)

Base

Diaphragm

- 23 generations of tubes over 50,000 bronchioles

Generation - A fork in tubes

- 1-17 generations (Bring in Air) → Conducting bronchioles
- 18-23 generations (gas Exchange) → respiratory bronchioles
- Horizontal fissure → Seperates upper and lower lobes

(3) low bar bronchi (s)

- Vascular Smooth Muscle Surround (1-17)
  - → Bronchodilate
  - → Bronchoconstrict

- Asthma (1) Bronchy Constrict

$R \propto \frac{1}{r^4}$ → R ↑, r ↓

- (1-17): Has Goblet walls cells that Secret mucus where particulate Matter sticks (r ↑, R ↓)

- Bronchitis: Causes (2) Goblet cells to Produce to much mucus ↑ R, r ↓ (cough up Spectum)

- (18-23)

elastin: Hold alveoli open

- Cystic fibrosis damage mucus lungs
- The ribcage Wants chest to expand
- Lungs Want to collapse (b/c elastin)

Creates Negative Pressure

Not enough $H_2O$ → Get too thick Prevents Silia movement

Mucus 50ml/day

"Sputum" - too much Product (mucus) To Swallow

Silia

6 Hm F Sol Gel

2 Hm F Sol Gel

8 Hm

air

Muco Siliary escalator

- 300 Mil alveoli creates a SA of 20 $m^2$ (Both lungs)
- Elastase: Destroys elastin
  - (Cig Smoke) → 19-23 collapse → Emphysema ③ → R↑, r↓

①②③ = ↑ airway resistance → ↑ Dyspnea

COPD: Chronic obstructive Pulmonary disease → Emphazema, bronchitis, Asthma
- $FEV_1$ = < 70% in 1sec (Breath out)

dead space: No ventilation

total Vol. 500 mL → 150 (1-18) / 350 (19-23)

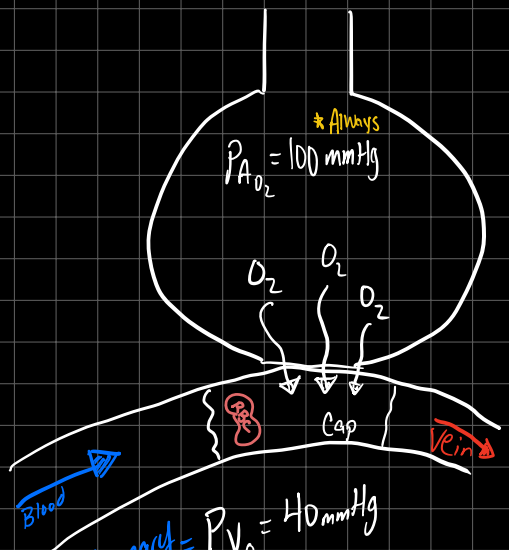
- tissue lining thoracic Cavity → parietal pluera
- "White" line around lungs → Visceral pluera
- 760 mm Hg Barometric/ambient pressure
  - → 760 in lungs (intrapulmonary)
  - → 755/-5 in intraplural Space (stops first 2 bullets)
- Pneumothorax: First 2 bullet hopefully ble air into Plural space
  - (Trauma) - external - Damage to Chest Wall → Chest tube
    - ★ Pressure difference
  - (friction) - Internal - Hole in the lung (bleb)

- under each rib lies a notch → Neurovascular Bundle
- A chest tube has a trocar w/ holes & is placed over a rib into intraplural space
- Hook up to pump & Suck out air → negative space → lung inflates

Hemothorax: Blood collects in intraplural Space → Chest tube (diff)

pleuraldesis: talcom Powder To seal Area
- NO intraspace
- tall thin Guys
- Stops blebs

## Gas Exchange




Composition of the Air we Breathe

| | |
|---|---|
| $O_2$ | 20.93% |
| $CO_2$ | 0.04% |
| $N_2$ | 79% |

Fractional [Conc.] of Inspired Air Types ($FIO_2$)

760 mm Hg

## Daltons Law/Law of Partial P

$P_{IO_2} = 0.2093 \times 760 = 160$ mm Hg

- Top of AA → drops ($P_{A_{O_2}}$ & $P_{a_{O_2}}$) but not the $FIO_2$

- $O_2$ Will diffuse from High (100) to low (40) pressure into the blood & bind to the iron in hemoglobin

- **emphysema**: $P_{I_{O_2}} = 160\ mmHg$
  - bc alveoli are destroyed
    - Elastin destroyed
    - CSA ↓
    - → increase ΔP (Which ↑ $F_{I_{O_2}}$ & $P_{I_{O_2}}$)
  - ABG = ↓ 100 mm Hg
  - $P_{a_{O_2}}$ ↓

- Once its bound ⇒ New PP → $P_{a_{O_2}}$ = 100 mmHg ← A.B.G.
  - $P_{a_{O_2}}$: Arterial
  - A.B.G.: Arterial Blood Gas

## Ficks Law of Diffusion

$$\rightarrow D_{lung} \propto \frac{\Delta P \cdot CSA}{L}$$

- ΔP = 60 Torr = Normal
- CSA = Cross Sectional Area ($70 m^2$)
- L = Length
  - ↑ Distance of alveoli to P.A.
  - normal ↓ 300 mil. vs. 100 mil Alveoli (SA)

- **Pulmonary Fibrosis**
  - ↑ L, Collagen fiber build up
  - → $P_{I_{O_2}}$ & $P_{A_{O_2}}$ are same
  - → $P_{a_{O_2}}$, ABG, Saturation ↓
  - → Increase ΔP

- **Pulmonary edema** (liquid based)
  - ↑ L
  - → give $O_2$ & Diuretics (increase urination)

- **HAPE** (lung & Brain)
  - → ΔP↓ CSA ↑ L
  - → use pure $O_2$ / leave altitude
  - → Nefedipine

- **Pneumothorax** - ↓ CSA
  - → lung shrivel

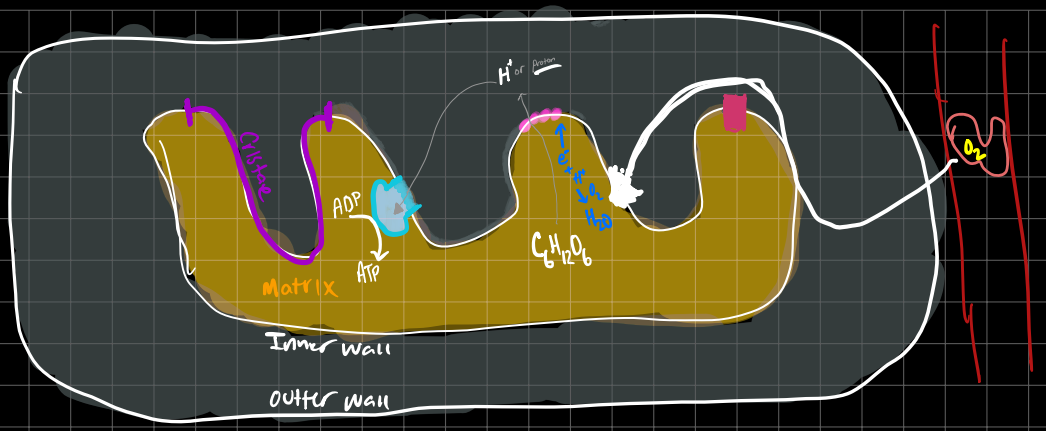
- @ 100 mm Hg of $P_{a_{O_2}}$
  - → Hemoglobin Saturation is 98%

## 4 Types of Hypoxia

1. Lung Problems → Hypoxic Hypoxia → Pulmonary edma → COPD, Altitude
2. Blood Problems → Anemic Hypoxia → Anemia
3. Heart Problems → Circulatory Hypoxia
4. Mitochondria Problems → Histologic Hypoxia

OXPHOS

Brown fat → Natural heat for baby produced in mitochondria by UCP




**Cristae** - fold to increase surface Area
Matrix
Internal membranal space

Inner membrane is composed of 75% Proteins

Cytochrome C blocked by Cyanide uses oxygen to make $H_2O$

- Complex I, II, III, IV = Electron Transport System

  Carbohydrates pushed through → ETS → via Complex Proteins from matrix into Internal membranal space → oxidized

- Complex V = ATP Synthase → Synthesizes ATP from ADP by use of proton

  Hydrogen formed when back inside matrix? forms $H_2O$

75% heat 25% ATP

- Uncoupling Protein ⇒ Allows for free proton transfer into matrix without ATP production → generates heat