Metoprolol = β-receptor blocker
↓ slope of phase 4 dep / prolongs rep. in nodal tissue
rate control antiarrhythmic
Caution with reactive airway / asthma patients

Diltiazem / Verapmil = slows AP upstroke in phase 0 in nodal tissue
(non-DHP) ↓ excitability of SA node / prolongs AV nodal conduction
↳ dep on freq. of stimulation Rate control antiarrhythmic
↳ ↓ current = ↓ RAP Caution with L. vent. dysfunction
Use-dep block in nodal tissues
Effect at L-type Ca^{2+} channels
Oral + IV

DHPs – indep. of freq. of stim.

Nifedipine = DHP
L-type Ca^{2+} channel blocker

Nicardipine = DHP
L-type Ca^{2+} channel blocker
IV administration

Amlodipine = DHP
L-type Ca^{2+} channel blocker
Largest ½ life
Preferred drug for HTN – use 1st

★ PR int. ↑, rest are unchanged

Drug List

Inhalers: target bronchioles. Faster, less adverse effects, less dose → spare inhaler: children
Inhaled: albuterol, ipratropium, fluticasone less adverse effects
Less deposit in mouth & throat

Bronchodilators	Antiinflammatory Drugs	Others
β_2-adrenoceptor agonists Albuterol ↑ cAMP	Glucocorticoids Fluticasone ↑ anti-inflam. cytokines Prednisone > phosph. A2	Antitussives Dextromethorphan (Codeine) ↓ cough detective ★ dry cough – depresses cough center in medulla
Methylxanthines (both) Theophylline	Leukotriene pathway inhibitors Zileuton – enzyme inhibitor (5-lipoxygenase) Zafirlukast – receptor antagonist	Respiratory stimulants Caffeine (Theophylline) – methylxanthine – antagonist at A_1 + A_{2a} rec. (less preferred)
Antimuscarinics Ipratropium ↳ blocks Ach mediated calcium release	Methylxanthines Theophylline – bronchodilator, anti-inflamm. ★ inhibit phosphodiesterase	caffeine: longer ½ life, ↓ adverse effects

★ Tic cough/sore cough = habit cough, stress ★ C WP → silica

★ Incidence: # of new cases / # at risk to be new → cohort ODDS RATIO = AD/BC

		Disease	
		Yes	No
Risk	Yes	A	B
	No	C	D

★ Prevalence: total # cases / # at risk → Cross-section RR: Incidence exposed / Incidence unexposed = A/(A+B) / C/(C+D)

Levels of Disease Prevention

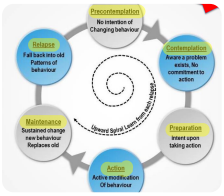
Primordial*	• Prevent risk factors, broad changes in social environment • Example: Policies to improve green space in urban areas to encourage exercise in all youth
Primary	• Prevent disease before it starts, reduce risk factors • Example: Promote healthy food and exercise behaviors that keep blood pressure and cholesterol low
Secondary	• Early detection and treatment for those at risk, stop or slow progression of disease • Example: Screening for early detection and intervention for coronary heart disease
Tertiary	• Manage those already with disease, prevent further complications, improve quality of life • Example: Coronary intervention procedures: angioplasty, stent, bypass surgery

MEDICARE >65 – fed. insurance
MEDICAID lowincome – assistance program

– retrospective = OR
– new cases = RR

< 1 = ↓ risk
> 1 = ↑ risk

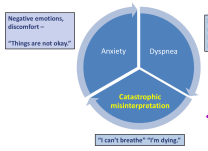
Assessment = monitor, diagnose, investigate
Policy dev = develop policy, inform, educate
Assurance = enforce, evaluate



Endemic: normally found
Outbreak: sudden, new
Epidemic: sudden ↑
Pandemic: multiple countries

± 1 SD = 68
± 2 SD = 96
± 3 SD = 99

Percentile = (100 – SD) ÷ 2
⊕ skew = lower values ∩



2 score = $\frac{X-\bar{X}}{s}$

CI = $\bar{X} \pm 1.96$ SE
★ If 1 in interval = not sig.

Stomach

G-cells ↓ secrete gastrin (hormone that controls release) ↓ HCl (parietal cells) pepsinogen (chief cells)

★ Sjogren Syndrome = dry eyes/mouth

β-defensins promote bacterial lysis via pore formation, are chemotactic for many leukocytes and promote phagocytosis.

Cathelicidins possess broad spectrum antimicrobial activity against many organisms including Gram-positive and Gram-negative bacteria by the disruption of membrane integrity.

Surfactant Proteins A & D are collectins that are produced by type II alveolar cells. They promote phagocytosis and are directly bactericidal by destroying bacterial membranes.

Lysozyme (Muramidase) is found in most secretions and is able to cleave the cell wall of Gram-positive bacteria.

Intestines

Antigen Sampling

★ Enterocyte pathway: enterocytes internalize antigen via bulk uptake + rec. mediated endocytosis.
– processed antigen → presented to intraepithelial lymphoid cells → no CD80/86 → no immune response

★ M cell → overlies peyer's patches → transcytosis → antigen delivery to APC → activ.

★ Antigen presenting dendritic cells → dendritic cell sends dendrites through tight junctions to capture antigen → migrates to lymphatics → l.n. → processed / presented to T lymphocytes (activ.)

↑ TOLLIP = inhibit TLR = tolerance of commensal organisms (comp. inh. of pathogens, vit. synthesis, decarb. of bile salts, production of antimicrobials)

Tolerance Induction:
- T reg: IL-10 / TGF-β
- ↓ co-stim CD80/86
- ↓ TLR by ↑ TOLLIP
- ↓ CD14

Pro-Inflamm: IL-1, 6, 15 TNFα
Anti-Inflamm: IL-10 TGF-β

★ paneth = defensins (intestines)
★ macrophages = long-lived
★ Neutrophils = short-lived

Genitourinary

– TITP → urinary tract epi. cells → flushes bacteria when peeing

★ Clostridium difficile = excessive use of antibiotics = ↓ normal flora

Mucus – goblet cells – mucins – IgA

★ Slowly adapting (Hering Breuer) → terminates insp. + large exp.
★ Rapidly adapting (irritant Receptors) → cough reflex, constrict at high activity
★ C-fiber / J Rec = rapid shallow breaths, constrict (↑ in int. fluid/edema)
★ CCR = stim by H+ (80%) = ↑ PCO_2 = ↑ H+ = ↓ pH = ↑ VT FR
★ PCR = stim by H+ (20%), 100% low PO_2 – narrowing, stenosis – low Hb low O_2 conc. – no ATP

★ Tissue Hypoxia = PaO_2 normal (stagnant, anemic, histotoxic)
★ Arterial Hypoxia / Hypoxemia = PaO_2 < normal (low PIO_2, – high alt Diff. limitation, Hypoventilation, Alv. V / Q mismatch, R→L shunt (↓ no vent.)

★ High perfusion / ventilation at base of lung → higher compliance
★ Top = higher V_I, more recoil, less compliance = less ventilated
★ VA/Q highest at top

★ $PaCO_2$ ↑ = hypoventilation
★ $Pa\bar{v}O_2$ ↓ = high alt (low PIO_2)
★ PaO_2 < 400 = R→L shunt w/ 100% O_2
★ CO diff. capacity < normal = diffusion problem
★ CO diff capacity = normal = VA/Q mismatch

Neurons controlling Resp:
★ Pneumotaxic = inhibitory
★ VRG = basic rhythm, extra drive
★ DRG = integration, NTS

Lung Volumes

* RV = 1.5 L air
* ERV = 1.5 L (1.5 L = RV, 1.5 L = ERV) = 3L after normal exp.
* V_T = 0.5 L
* IRV = 2.5 L

Inert gas method → $V_L = V_S \frac{F_0 - F_1}{F_1}$ for RV

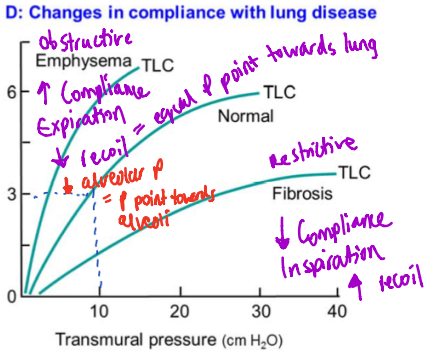
+ IC = V_T + IRV
+ FRC = RV + ERV
* VC = ERV + V_T + IRV – largest breath that can be taken
+ TLC = VC + RV (ERV + VT + IRV + RV) – lung V after max. inspiration

1-16 = conducting/dead space
17-23 = respiratory space

* Lung V at FRC = 3L
  → 0.15 L in dead space
  → rest in resp. space
* Tidal V = 0.5 L
  → 1/3 in dead space (0.15 L)
  → 2/3 in resp. space (0.35 L)
* Physiological D.S. ≥ Anatomical D.S.

Physiol. DS = Anatomic + alv. regions not doing gas exch.

Emphysema:
– lip pursing
= ↑ mouth P
↑ inside P of airways
↑ Trans. P
= avoid ⊖ P
= keep airways open + exhale



* Compliance: • elasticity
  • principle of interdependence
  • surface tension ↓ C
  • surfactant = ↓ ST ↑ C
  ↓ radius = ↑ P ↓ P diff.
  ↑ surfactant density

* Ideal Gas Law: ↑ T = ↑ V
  ↓ P = ↑ V

Measure RV: Helium dilution
$F_0 \cdot V_S = F_1 \cdot (V_S + V_L)$

* Obstructive: ↑ TLC ↑↑ RV ↑ RV/TLC% FEV/FVC ↓
* Restrictive: ↓ TLC ↓ RV Normal FEV/FVC normal or ↑ recoil ↑

* Resting V of lung = below RV, min. lung V (0.5 L)
* Resting V of RS = FRC (3 L)
* Resting V of CW = 60% of TLC

* Chest wall restricts expiration
* Lungs/CW restrict inhalation
* At FRC: lungs inward / chest out
  ↳ P_{PL} = ⊖
* ⊖ flow = inspiration (air from atm → body)

Recoil → elastic tissue
→ ST

* lip pursing (emphysema): shifts equal P point towards mouth (airways open)

$P_X = F_X (P_B - P_{H_2O})$ $F_{O_2} = 0.21$
P_B = 760 (sea level) Alt ↑ = ↓ P_B
P_{H_2O} = 47 mm Hg
P_{IO_2} = 147
P_{IO_2} ↓ with ↑ Alt.

$P_{AO_2} = P_{IO_2} - \frac{P_{ACO_2}}{R}$ $P_{ACO_2} = \frac{\dot{V}CO_2}{\dot{V}_A} \times 863$

* upper airways (1-16) = 80% of resistance
* lower airways (17-23) = little resistance, high total diameter

Airway Resistance: passive = transa. P; active = SM contraction

– ↓ density / viscosity = ↓ Raw
– ↓ CO_2 = ↑ Raw (bronchoconstriction)
– sympathetic: ↓ Raw / parasym: ↑ Raw
– lung volume – V ↑, recoil force ↑, compliance ↓, push out resistance ↓

* FEV_1 = V of air forcibly exhaled from the lungs in 1st second
* FVC = after max inspiration, V of air forcibly exhaled until no more can be exhaled
* $\frac{FEV_1}{FVC} = 0.8$ (80% = normal)
* PEFR = max flow rate achieved during FVC
* $V_T = V_D + (V_T - V_D)$
* Vent = freq x tidal V
  $F_R \cdot V_T = F_D \cdot V_D + F_R \cdot V_A$
  VE (total vent) = $V_D + V_A$
  ↑ V_T = ↑ V_A (efficient for gas exchange) (V_D constant ~150 ml)
  ↑ F_R = ↑ V_A + ↑ V_D

↑ 1° = 10% change in BMR (37°C = 300 / 38°C = 330)

$R = \frac{CO_2 \text{ output}}{O_2 \text{ uptake}}$ RQ = R
Carbs: R = 1
Fats: R = 0.7
Proteins: R = 0.8

Normoventilation: $P_aCO_2 = 40$
Hyperventilation: $P_aCO_2 < 40$
Hypoventilation: $P_aCO_2 > 40$

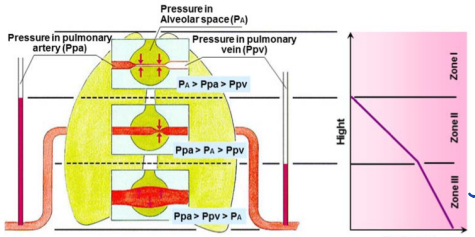
↑ diffusion: ↓ thickness
↑ SA

$D_L CO_2 = 20 \cdot D_L O_2$ (no diffusion problems for CO_2)

$D_L = \frac{\dot{V}gas}{P_A}$

* Hyperoxic / Hypocapnic = Bronchoconstriction → resistance ↑ → gas diverts
* Hypoxic / Hypercapnic = vasoconstriction → resistance ↑ → blood diverts
↳ reduce vent/perfusion mismatch

* BP lower at top of lung



Arterial P higher than venous + alv. P
Alv. P lowest, blood flowing. As go up lung = blood flow ↓

- no blood flow at top
- lots of blood flow at base

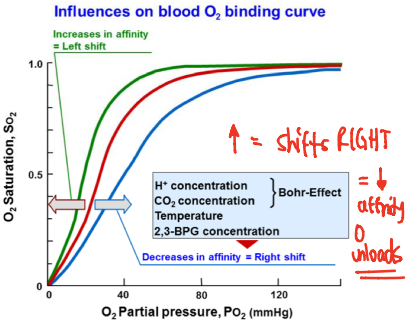
* Diffusing capacity measure with CO

Exercise = ↑ D_L (recruitment / distention)
Body position = supine: ↑ cap. V, more even flow
Body size = ↑ Lung size = ↑ SA = ↑ D_L

L → R Normal shunts: ↓ PO_2
- Thebesian Veins → l. atrium / l. ventricle
- Bronchial Veins → drains into pulm v. → into l. atrium (dew into ox. blood)

* Mixed venous blood = 40 mmHg PO_2
* Arterial blood = 100 mmHg PO_2
* end capillary → $P_A = P_{c'O_2}$

↓ PO_2 = ↓ O_2 sat / conc
↓ Hb = ↓ O_2 conc / not saturation



↑ = shifts RIGHT = ↓ affinity & unloads

- diffusion limitation: alv. PO_2 > arterial PO_2 (A-a) PO_2 difference
- Exercise = ↑ CO = ↓ transit time ↳ with lung disease / high altitude
- Low alveolar PO_2 (hypoxia) - exercise at high altitude → diff. lim.

* If no diff. lim → it is PERFUSION LIMIT. (A-C') PO_2 difference
= ↑ flow

* N_2O = perfusion limited
* O_2 = perfusion limited / diffusion limited w/ intense exercise, disease, thickening of blood-gas barrier, alv. hypoxia
* CO = diffusion limited

* Lung: High affinity → load O_2
* Tissue: Low affinity → unload O_2

→ Carboxyhemoglobin = CO + Hb
→ shifts left = ↓ unloading O_2

→ oxidation of Hb = methemoglobin (Fe^{3+})
↳ methemoglobin reductase for $Fe^{3+} \rightarrow Fe^{2+}$

- Pulmonary circulation = less R, smaller P difference than systemic
- ↓ R = ↑ P (↑ CO)
- ↓ PVR: widening of vessel
  open a closed vessel (recruitment)] passive - occur as CO ↑
  ↑ P in vessels = stretch = ↓ Resistance
  ↑ V = ↓ resistance of airways (LOWEST AT FRC → ↑ V or ↓ V = ↑ R) — vessel resistance

Bohr = CO_2 on O_2
Haldane = O_2 on CO_2

* Regional Hypoxia = vasoconstriction = R ↑ = divert blood
* General Hypoxia = all vessels constrict = no diverted flow = r. vent has to push against ↑ R → pulm. HTN, HF, edema

Systemic = VASODILATION (K^+ channels / NO) / pulm = VASOCONSTRICT ↳ inhibit K^+

Normal HR: 60 - 100 bpm

Bradycardia: < 60

Tachycardia: > 100

- find net 0 or use lead I / AVF

Normal axis: +90 to -30

Left dev: -30 to -90

Right dev: +90 to +180

1st degree = slow AV conduction → long PR int >1 big box

2nd type I = partial AV block → PR int ↑ w each beat ↳ reg. irregular + dropped QRS

2nd type II = partial His block → dropped QRS

3rd degree = complete AV block → atria/vent beat own rhythm

Long QTS = QT_c = >450 men >470 women

- gain sodium
- loss function IKs / IKr

p-P int. Consistent

R-R shorter

broad QRS

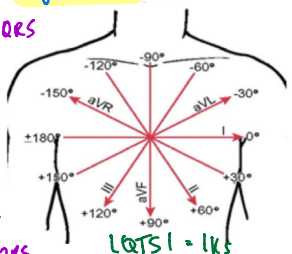
opp ⊕/⊖ of QRS + T

2-4 P waves for every QRS

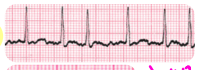
LQTS1 = IKs

LQTS2 = IKr

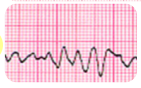
LQTS3 = Na^+



Afib = no p-waves = irregularly irregular



Vfib = - lack of any pattern



V1 → V6 = ⊖ to ⊕ QRS

View of heart	Leads	CAs
Inferior	II, III, aVF	RCA
Lateral	I, aVL, V5, V6	LCX
Anterior	V3, V4	LAD
Septal	V1, V2	LAD

CO = SV x HR

- preload
- afterload
- contractility

- SNS (+)
- PNS (-)

EF = SV / EDV normal = 0.55 - 0.65

- Starling: Stretch ↑ = force/contraction ↑
- ↑ afterload = ↓ shortening velocity

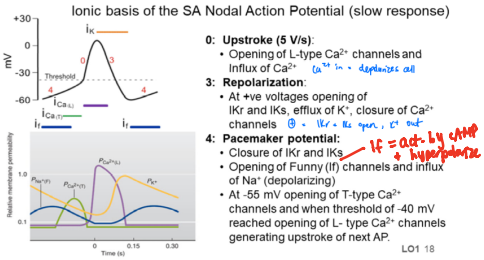
→ V_{max} = lowest afterload

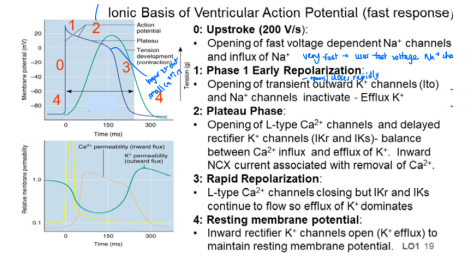
→ F_{max} = highest afterload

T-type = -55 mV

L-type = -40 mV

- Na^{2+} = +60 mV
- Ca^{2+} = +130 mV
- K^+ = -90 mV





- ★ Aortic Stenosis = systolic
- ★ Aortic regurgitation = diastolic
- ★ Mitral stenosis = diastolic
- ★ Mitral regurgitation = systolic

- concentric → pressure → S4
- eccentric → volume → S3
- static compliance = physical, connective/elastic tissue
- dynamic compliance = vascular tone, SM contraction, SNS activity

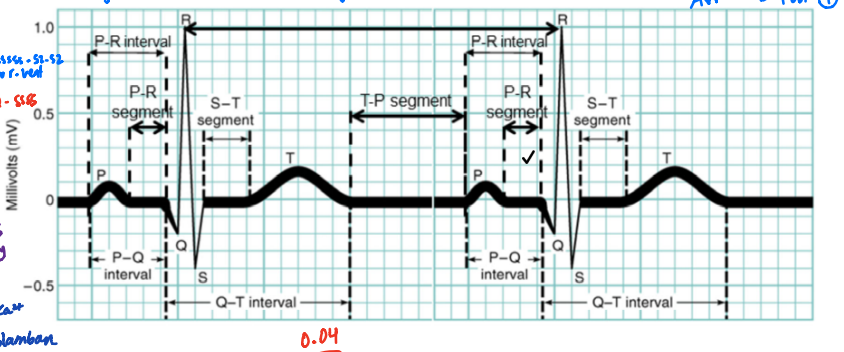
Systolic: between S1/S2

- aortic stenosis
- mitral reg
- ASD/VSD S1 - ssssss - S2-S2 ↳ more blood into r. heart

Diastolic: S1 - S2 - SSS

- Aortic reg
- mitral stenosis

Right arm = both ⊖ / left leg = both ⊕, AVL = Left arm ⊕ / AVR = Right arm ⊕ AVF = L- foot ⊕



Aorta P = 120/80

R. atrium = -4 to 4

Pulm. a = 25/8

L. atrium = 6-12

RV = 25/0

LV = 120/0

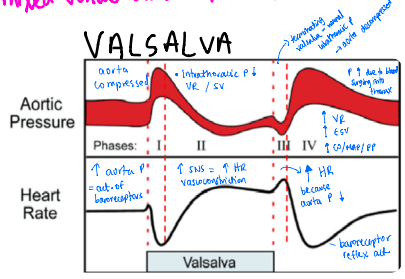
SNS = ↑ contractility

- ↑ cAMP → PKA = ⊕ (L-Type Ca^{2+} channel, RyR, Albopholamban, Troponin I)

= ↑ EDV, ↑ crossbridges → act. cAMP

→ digitalis = ↑ contractility → blocks Na^+-K^+ ATPase → Na/Ca^{2+} exchanger ↓

★ mixed venous blood - pulm. a, r. atrium, r. vent.



R-R interval

- time between successive heart beats
- HR = 60 / R-R * 300 ÷ # of big boxes

T-P segment

- diastole

Hypertrophy

> 7 big boxes (V_1 + V_5 OR V_6) ↳ whichever is taller

0.04

PQ/PR interval = 0.12 - 0.2 s (4 small boxes)

- atrial contraction + time for conduction of AP through AV node
- long = AV node damage

ST segment = 0.12 s

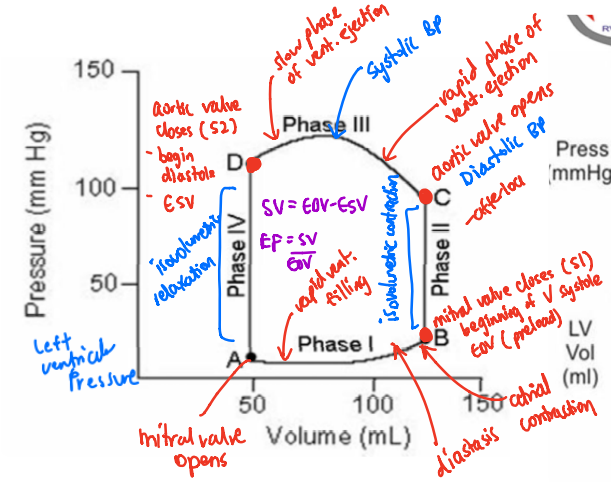
- ventricles fully dep. Plateau phase
- ↑ in MI / ↓ in ischemia (2 or more small)

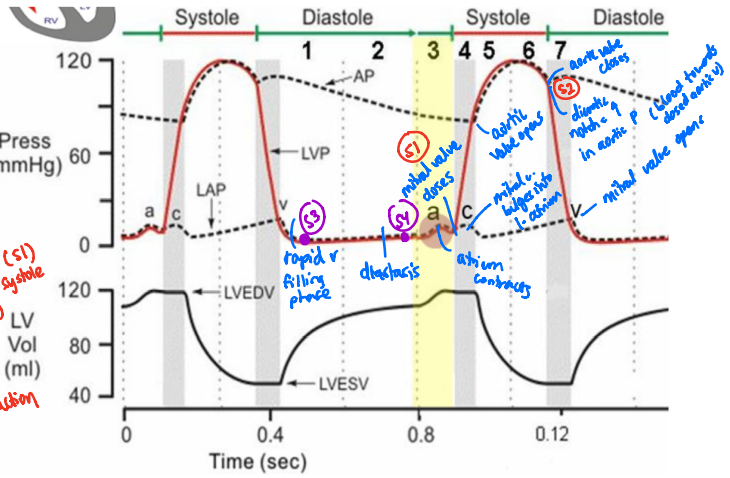
QRS < 0.10 s

QT interval = < 0.4 s (8 sm. boxes)

- vent dep. → rep. (systole)
- long = LQTS > 450 ms

$QT_c = \frac{QT}{\sqrt{RR}}$



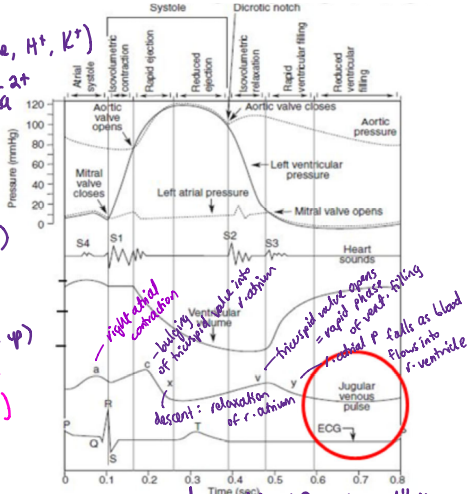


Control of Blood Flow:

- metabolic control - metabolites/paracrine (↓ O_2 = dilation, adenosine, H^+, K^+)
- myogenic control - ↑ flow = ↑ stretch = SM contracts (vasoconstriction) *Ca^{2+}

* autoregulation:
- ability of organ to maintain constant blood flow when arterial P changes
→ heart, brain, kidney (between 60 - 180 mmHg, constant flow)

= ↓ flow OR vasodilation → by NO (↑P
↳ PKG: ① phospholamban Ca^{2+} uptake ↑ = relaxation
= ↑ shear stress → NO stim)



Hyperemia
- accumulation of ions/metabolites in ↓ blood flow = vasodilation (↑ blood flow to get rid of this build up)

- Active Hyperemia → ↑ in blood flow due to ↑ in metabolism (ex: running)
- Reactive Hyperemia → short interruption of blood flow causes vasodilation + ↑ blood flow (ex: weight lifting / BP cuff)

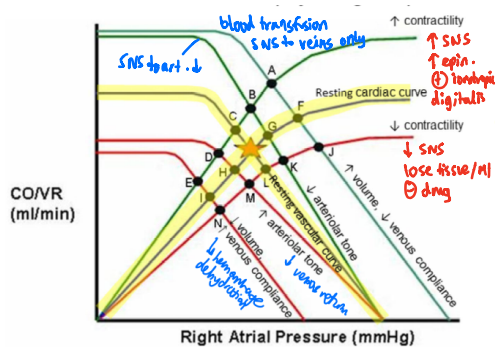
* skeletal: ADENOSINE + K^+
* heart: ADENOSINE/NO
* Brain: HIGH CO_2 (hypercapnia) = vasodilation
→ circle of willis: basilar a + int. carotids → maintain blood flow to brain

Coronary blood flow → l. vent. coronary = highest during diastole (compressed in systole → ↑ muscle → dilation = active hyp. ↳ diastole = reactive hyp.
r. vent coronary = does not ↓ during diastole (less compression)

↑ RAP = ↓ VR

VR = CVP - RAP

↑ VR / ↑ CO by ↑ blood volume
↓ venous compliance
↓ arteriolar resistance



ORTHOSTASIS
↳ gravity + high compliance of veins

Stand → blood pools in LE → ↓ VR, CO, MAP
↓
baroreceptor reflex
↓
↑ HR, contractility, TPR
↓
VR, CO, MAP - normal

* Reduce Venous Pooling:
- Skeletal Muscle Pump → contract → ↑ VR
- Thoracic Muscle Pump → resp. pump inspiration → ↓ RAP → ↑ VR

Exercise ↓ filling time
↳ diastasis sig. reduced, SV ↑ at very high HR
↑ activity → SNS → ↑ CO, ↑ MAP, vasoconstriction, ↑ TPR
local effect → vasodilation → ↓↓↓ TPR → overall ↓ in TPR

= ↑ EDV, ↑ systolic BP, ESPVR steeper, ↓ ESV, ↑ EF, ↑ SV, HR, CO, ↑ stroke work

- ↑ MAP in exercise not counteracted by baroreceptor reflex → reset to higher MAP set point

Pack years = # of cigs ÷ 20 × # of years

HTN: ↑ sym tone / excess Na^+ (↓ BP = ↑ renin)

AS: forms plaque. Risk: high chol, BP, diabetes, smoking
stable = mod/severe narrowing → chronic ischemia / angina
unstable = thin cap rupture → thrombosis → acute ischemia/infarct

IHD: caused by AS plaque

Type	Clinical Presentation
Stable angina pectoris	Chest pain •lasts 2-5 min (<20 min) •alleviated with rest or nitroglycerine
Acute coronary syndrome (ACS) - Unstable angina pectoris - Myocardial infarction (MI)	Chest pain •lasts >20 min and subsides after 2-3 hours •not alleviated with nitroglycerine
Sudden cardiac death (SCD)	(Sudden and unexpected) death •within 1 hour after appearance of symptoms if caused by a cardiac disease

MI:
* absence of nuclei / lots of neutrophils
* ↓ contractility, LV rupture w/ hemopericardium, tamponade
* LV pump function: pulm. edema, cardiogenic shock, cardiac arrest

HF: ↓ cardiac ability to eject blood or be filled with blood.
- symptoms: dyspnea (breathless) / fatigue (tiredness)
- causes: Pressure overload → HTN / stenosis / ↓ contractility - ischemia
volume overload (regurgitation, L → R shunt) / ↓ preload (↓ compliance - hypertrophy)

* LHF = pulmonary circuit (pulm. edema, pleural effusion)
* RHF = systemic circuit (jug. veins, peripheral edema, nutmeg liver)
* Hf cells: RBCs eaten up by macrophages - presence of hemosiderin, excession in macrophages

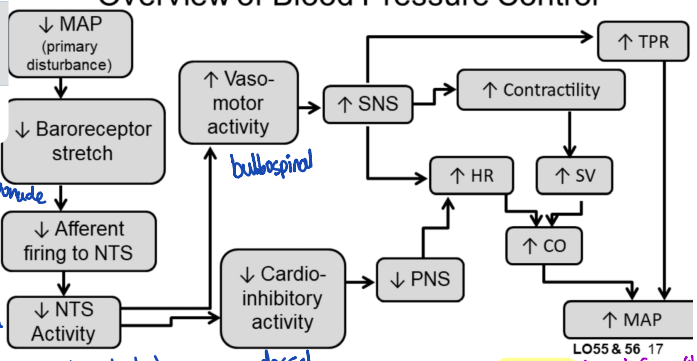
Hypovolemic shock: ↓ blood V
Cardiogenic shock: ↓ CO - HF / MI. LHF = affects pulm. ↓ CO, ↑ pulm. V, CHF
RHF = affects systemic ↓ pulm. V, periph. edema
- compensated = 10 - 20% lost V → reflex can restore MAP, ↓ SV
- decompensated = compensatory mech reach max. ability, can't ↑ further → ischemia
- irreversible = untreated decomp. shock → organ damage / death

$CI = \frac{CO}{\text{body SA}}$

Wedge P = measures pulm. vein and L. atrial P

ΔP Systemic = aorta - R. atrium
ΔP pulmonary = pulm a - L. atrium

Overview of Blood Pressure Control



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Diameter: Lowest for capillaries
Velocity: Lowest for capillaries
CS area: Highest for capillaries
Blood distrib: Highest in veins
TPR: Highest in arterioles
BP: Highest in aorta → Lowest in veins

$P_IO_2 = F_IO_2 (P_B - P_{H_2O})$
$= 0.21 (P_B - 47)$

Transit time = Volume / Flow

* each g Hb binds 1.34 O_2

* Dissolved O_2 conc = $P_{O_2} \times \alpha O_2$

$\alpha O_2 = 0.003$

$P_AO_2 = P_IO_2 - \left(\frac{P_ACO_2}{R}\right)$ Alveolar PO_2

$V_A = \frac{VCO_2}{P_ACO_2} \times 863$ Alveolar Ventilation

$CO = SV \times HR$

$MAP = CO \times TPR$ / $MAP = P_{dias} + 1/3(P_{sys} - P_{dias})$

$R = \frac{VCO_2}{VO_2}$

$SV = EDV - ESV$

$EF = \frac{SV}{EDV}$

Series: add up r

Parallel: $\frac{1}{\frac{1}{r} + \frac{1}{r} \ldots}$

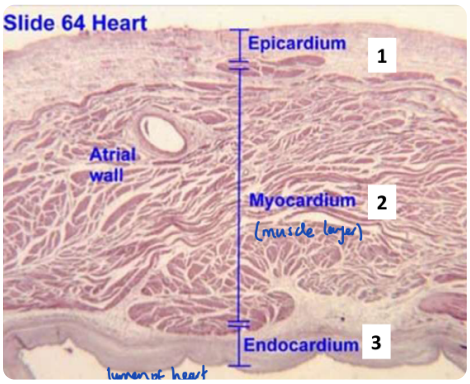
$DL_{CO} = \frac{V_{CO}}{P_{ACO}}$

$CO = \frac{VO_2}{C_A - C_V}$

$PVR = \frac{(\text{Pulm. artery } P - \text{L. Atrial } P)}{CO}$

$(P_C + P_i) - (P_i + \pi_C)$

$F_{He(before)} \times V_{bag} = F_{He(after)} \times (V_{bag} + RV)$



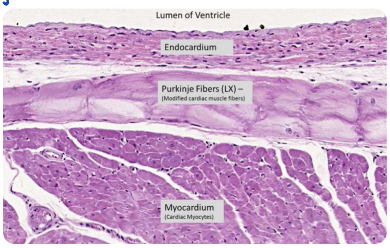
Epicardium - outermost - LCT under
- simple squamous
- secretes pericardial fluid

Myocardium - middle / thickest
- cardiac m. cells

Endocardium - innermost, faces heart chamber
1) inner thin endothelial layer (simple squamous)
2) middle myoelastic layer (SM / CT)
3) deep subendocardial layer (CT / vessels / purkinje)
↳ merges w/ outer myocardium

Heart Valves: fibrous ring, leaflets + chorda tendinae
support valve

Purkinje Fibers: do not contract, propagate waves through myocardium. Linked to cardiac cells by gap junctions / desmosomes



→ between endocardium + myocardium
→ Purkinje are LARGER + PALER than other cardiac cells.

3 Layers of Blood Vessels:

Tunica Intima: innermost
1) endothelium 2) basal lamina 3) subendothelial
- squamous simple
- flat, long, parallel to blood
- tight junctions

LCT - collagen / SMCs
- indep. movement

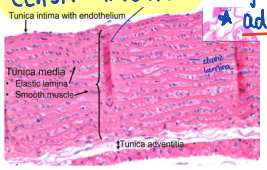
Tunica Media middle
- SMCs joined by gap junctions
- vasoconstriction / vasodilation
- elastic fibers

Tunica Adventitia outermost - CT (type I collagen / elastic fibers)

*vasa vasorum = tunica blood vessels → in adventitia / outer media → arteries / capillaries / venules
*vasi vasorum = tunica nerves → reg. contraction of SMCs

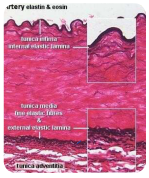
*atherosclerosis = fatty plaques in intima of med/large arteries

ELASTIC ARTERIES - largest, tunica media has elastic fibers - lamina
*adventitia thinner than media

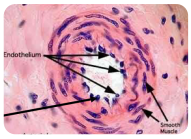


- Elastic fibers
- SMCs (between elastic laminae, joined by gap junctions, produce components of media, proliferate to repair intima)

MUSCULAR ARTERIES - internal elastic lamina between intima + media
- external elastic lamina in large muscular arteries, not small
*adventitia thinner than media - media = 40 layers SM



ARTERIOLES - media = 1-2 layers of SM



METARTERIOLE - connect art → capillaries
- no media
- pre capillary sphincter (single SMC that encircles metarteriole at junction)

CAPILLARIES → only intima layer present (endothelium / basal lamina)

CONTINUOUS CAPILLARIES - most common, least permeable
- tight junctions, continuous layer

*pericytes = contractile, more blood made SMCs (growth / healing)

*Blood Brain Barrier - special type of continuous capillary
- blocks big molecules
- tight junctions : ASTROCYTES - few pinocytic

FENESTRATED CAPILLARIES - fenestrae / windows in endothelial layer
↳ where rapid exchange occurs = kidney / int / pancreas ↳ bridged w/ diaphragms = permeable ↳ pinocytic vesicles

SINUSOIDAL CAPILLARIES - most permeable, least common
- no pinocytotic vesicles - fenestrae, no diaphragms - intercellular gaps
- irregular in shape
- liver, bone marrow, spleen

POST-CAPILLARY VENULES = smallest, only intima, WBC migration - pericytes

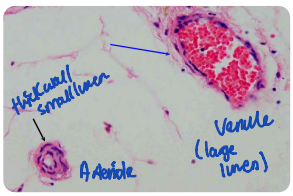
MUSCULAR VENULES = all 3 layers, no pericytes
- media = 1-2 layers SM

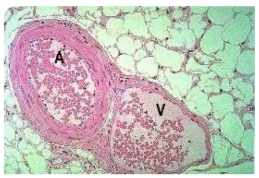
MEDIUM + LARGE VEINS - adventitia thicker than media
↳ contain valves (paired folds of tunica intima)

Arteries	Veins
- thicker media	- thicker adventitia
- more round	- irregular lumen
- elastic lamina	- no elastic lamina

- valves = paired folds of tunica intima

Lymph Vessels = no RBCs, thin walls





General, arteries (A) have thicker walls and smaller lumens than veins (V)
Veins may possess valves.

* Conducting Portion
- humidifies = glands
- filters = vibrissae (hairs)
- warms = blood
- smell / speech

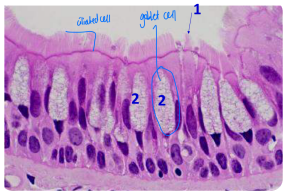
* Respiratory Portion
- gas exchange

Pharynx
- nasopharynx = resp. ep.
- oropharynx / laryngopharynx = non-keratinized stratified squamous

Epiglottis
- elastic cartilage core
- hyaline cartilage everywhere else
- respiration = vertical
- swallowing = horizontal
* 2 surfaces: • Ant. (lingual) - strat. squamous
• Post. (respiratory) - upper = ciliated squamous; lower = resp. epithelium
* glands are marker for resp. side

Nasal Cavity
1) vestibule = external → keratinized stratified squamous epithelium
2) Nasal fossa = internal

* Respiratory epithelium: ciliated pseudostratified columnar epithelium → all touch basal lamina
↳ 5 cell types:
- ciliated columnar → mitochondria – ciliary beating action
- goblet cells → secrete mucus * appear light → mucus color
- brush cells → microvilli, chemoreceptors
- Basal cells → basement membrane, can differentiate
- small granule cells (DNES cells) → resp / GI, releases catecholamine → small cell carcinoma + other hormones

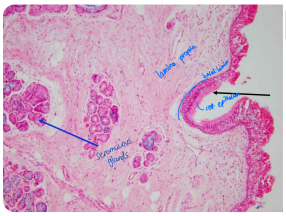


* Olfactory epithelium: senses smell
↳ 3 cell types
- Olfactory receptor cells (neurons) → chemoreceptors for odorants → bipolar, non-motile cilia
- Supporting (sustentacular) cells → ovoid nuclei, wide base microvilli, physical/met. support
- Basal cells → neuronal stem cells, contact basal lamina

* olfactory nerves are NOT myelinated

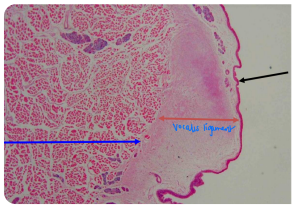
* Bowman's gland = serous exocrine glands
- found in CT deep to olfactory epithelium in lamina propria
- act as solvent for odorous substances → clear cilia

Larynx – hyaline cartilage
1) Vestibular / ventricular folds = false, immovable, respiratory epithelium, seromucous glands
2) true vocal folds = phonation, non-keratinized squamous ep., large elastic fibers, VOCALIS M.



False Vocal fold (cord), mag. 200X

Epithelium is respiratory epithelium (ciliated pseudostratified columnar with goblet cells, black arrow).
Glands are seromucous (blue arrow).



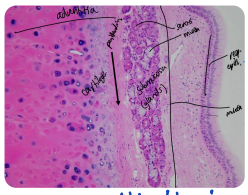
True Vocal fold of Larynx, Mag. 20X

Beneath the epithelium (black arrow) lies the vocal ligament (orange arrow) which is composed of parallel bundles of elastic fibers.

The vocalis muscle (blue arrow) is very prominent beneath the ligament.

* Vocal fold Nodule (Singers Nodule) = ↑ tension of vocal folds → hoarse voice
subepithelial scarring

Trachea – bifurcates at T4
- resp. epithelium
- C-shaped hyaline cartilage (16-20)
- trachealis m.
↳ swallowing = relaxes
↳ coughing = contracts

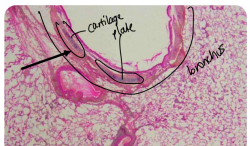


* 3 layers of tracheal wall:
1) Mucosa = resp. epith. / lamina propria * ciliated pseudostrat.
2) submucosa = submucosal glands, LCT * glands
3) Adventitia = hyaline cart. C-shape rings * cartilage (perichondrium)

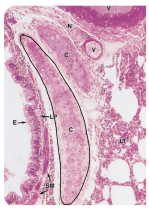
Bronchi – resp. epithelium
- extrapulmonary = not surrounded by lung
- intrapulmonary = surrounded by lung → irregular plates of cartilage

* as bronchi get smaller: Hyaline cartilage ↓
SM + elastic fibers ↑

Feaves of bronchi:
- cartilaginous plates
- SM
- seromucous glands
- respiratory epithelium



↑ Intrapulmonary bronchi ↑



INTRAPULMONARY BRONCHUS
High Mag- H&E
N nerve
LP lamina propria
C cartilage
LT lung tissue
E epithelium
V (blood) vessel

SM smooth muscle
As bronchial lumens narrow, the presence of smooth muscle increase

Bronchioles – Bronchioles simplify + thin = resp. epithelium → ciliated simple columnar/cuboidal

– no cartilage
– no glands
> bronchi have both, bronchioles don't !!

* Goblet cells = large bronchioles (get replaced w/ club cells in terminal bronchioles)

* Club cells = only found in bronchioles
→ dome shaped
→ secrete surfactant components
→ detox airborne toxins – smooth ER
→ secrete antimicrobial peptides
→ stem cells for ciliated/secretory epithelial cells

* Asthma: chronic inflammation of bronchial tree
↳ bronchospasms → sudden constriction
↳ infiltration of bronchiolar wall by eosinophils, lymphocytes, mast cells

Respiratory Bronchioles – 1st part of resp. portion
– ciliated simple column/cuboidal
– club cells
– out pockets for alveoli (simple squamous)

RB have breaks = alveoli
TB are continuous

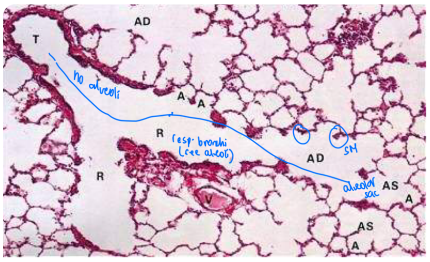
Alveoli — type I pneumocytes – thin, simple squamous, blood-air barrier, tight junctions
— type II pneumocytes – cuboidal cells, round nuclei, found at angles of alveolar wall, secrete surfactant
↳ mitotic activity → replace own cells/replace type I

↳ lamellar bodies secrete by exocytosis

Alveolar Ducts – SM
– end of resp. bronchioles
– lined w/ alveoli
– SM surrounding opening

Alveolar Sacs – no SM
– clusters of alveoli

Blood-Air Barrier
– prevents air bubbles in blood
– prevents blood from entering alveoli
– 4 components: 1) alveolar ep. cells
usually fused [2) basal lamina of alv. epithelium
3) basal lamina of cap. endothelium
4) endothelial cells of capillary network



T= terminal bronchiole
R=respiratory bronchiole
AD=alveolar duct
AS=alveolar sac
↳ no SM pockets mapping sound
A=alveolus

Alveolar Macrophages – phagocytose dust, pollen, RBCs
– darker than type 2 pneumocytes

Alveolar pores (of Kohn) – openings in interalveolar septa
– allow circulation of air from 1 alveolus to another
– in obstructive disease: useful (alveoli by block can be aerated)

* Pulmonary surfactant + Neonatal Respiratory Distress Syndrome
– premature infants
– enough surfactant at 35th week
– surfactant: ↓ surface tension → ↓ alveolar collapse
* leads to RDS

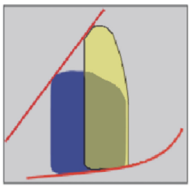
* Emphysema: COPD
– permanent enlargement of airspaces
– destruction of elastic fibers
– ↓ SA (fewer/larger alveoli)
– ↓ ability to absorb O_2 + rid CO_2

* no recoil when stretched

** stimulus = ↑ macrophages = ↑ neutrophil = ↑ elastase = ↓ α1 antitrypsin = destroy elastic fibers = emphysema

Aortic or Pulmonic Valve Stenosis (chronic untreated state)

higher resistance to flow
r. ventricle → pulm.
l. ventricle → aorta



- Increased resistance for ↓ SV ejection of blood (difficult to eject blood)
- Decreased stroke volume
- Increased ESV

- Systolic murmur
- Concentric hypertrophy on ECG
- S4 heart sound change in compliance of ventricle

YouTube Video of Aortic Valve: http://www.youtube.com/watch?v=Ic1vcKDzD7U&NR=1

Note: Aortic valve stenosis is a condition where diastolic aortic pressure is not a good estimate of the actual afterload.

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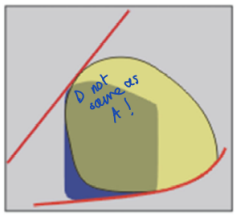
Aortic or Pulmonic Valve Regurgitation (chronic untreated state)

Blood back-flow



- Increased Preload (EDV)
- Increased stroke volume
- Eccentric hypertrophy

Starling Law:
- ↑ SV
- eccentric
- filling issue
- S3

- Diastolic murmur
- S3 heart sound
- Increased Systolic BP due to large volume of blood ejected into aorta
- Blood pressure will reduce as blood regurgitates so pulse pressure will increase.

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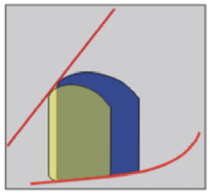
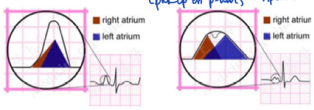
Mitral or Tricuspid Valve Stenosis (chronic untreated state)

trouble w/ getting blood from atrium ↓ ventricle



- Decreased Preload (EDV)
- Decreased stroke volume
- Decreased afterload

- Diastolic murmur associated w/ filling
- No abnormal heart sound
- Atrial dilated hypertrophy - tries to open up on p-vars compensate



↓ cross bridges → below peak of active tension curve → ↓ contraction → ↓ SV

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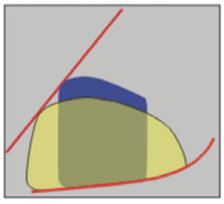
Mitral or Tricuspid Valve Regurgitation (chronic untreated state)

Blood back into atrium



- Increased Preload (EDV)
- Decreased systolic BP
- Increased total SV
- Decreased effective SV (where blood should be going → needs to go to out-flow tract but is going into atrium)
- Decreased ESV

- Systolic murmur
- Eccentric hypertrophy
- S3 heart sound

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Starling Forces:

- **Net Driving Pressure = [(Pc + πi) – (Pi + πc)]**

or

- **Net Driving Pressure = Forces Out – Forces In**

⊕ filtration: capillary → interstitial

⊖ reabsorption: interstitial → capillary

Edema = ↑ filtration = ↑ Pc
↓ reabsorption = ↓ πc

★ exudate → proteins

★ transudate → no proteins

1. ↓ arteriolar resistance → ↑Pc → ↑ filtration → edema
2. ↑ venous resistance → ↑Pc → ↑ filtration → edema (more difficult for fluid to get into veins from capillaries)
3. ↓ plasma proteins → ↓πc → ↓ reabsorption → edema
4. ↓ lymph drainage → ↑ Pi → edema (can't drain interstitial fluid ↑ Pi)

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