

Types of Vessels

1. Wind Kessel $\Rightarrow$ aorta, large arteries
2. Resistance $\Rightarrow$ arterioles, metarterioles, smaller arteries
3. Exchange $\Rightarrow$ capillaries
4. Capacitance $\Rightarrow$ large & small veins

Differences b/w systemic & pulmonary circulation

Intercalated disc

Length-tension relationship

Frank-Starling Law

- Mechanisms $\rightarrow$ $\uparrow$ EDV $\Rightarrow$ $\uparrow$ initial muscle length
- $\rightarrow$ stretch sensitive Ca channels open
 - $\rightarrow$ Ca-induced-Ca channels
 - $\rightarrow$ $\uparrow$ affinity of troponin C for Ca due to stretch

$\left\{ \begin{array}{l} \text{EDV} = \text{end-diastolic volume} \\ \text{N.} = \text{Nerve} \\ \uparrow = \text{increase / rise} \\ \downarrow = \text{decrease / fall} \end{array} \right.$

Parasympathetic (vagus)

- ve chronotropic $\Rightarrow$ $\downarrow$ heart rate
- ve dromotropic $\Rightarrow$ $\downarrow$ rate of conduction
- ve bathmotropic $\Rightarrow$ $\downarrow$ excitability
- ve inotropic $\Rightarrow$ $\downarrow$ myocardial contractility

(70)

• Basal / Resting heart rate $\Rightarrow$ vagal tone $>$ sympathetic tone

• Intrinsic heart rate

(120)

Sympathetic

- Right sympathetic N. $\Rightarrow$ affects heart rate
- Left sympathetic N. $\rightarrow$ " myocardial contractility
- $\rightarrow$ all 4 effects are +ve

Properties of Cardiac Muscle

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- Automaticity
- Rhythmicity
- Conductivity
- Contractility
- Excitability
- Distensibility
- Long Refractory Period (non-tetanzable)
- Extrasystole
- Compensatory pause, PEP
- Staircase phenomenon

(PEP = post-extrasystolic potentiation)

Mechanisms - released Ca^{2+} not completely pumped back

- $\uparrow$ temperature $\Rightarrow \uparrow$ enzymatic activity

- $\downarrow$ viscosity

- Length-Tension Relationship (heterometric autoregulation of CO)
- Frequency-force relationship (Bowditch)
- Load-velocity relationship

Fast Response AP (phase 0 $\rightarrow$ 4)

- phase 0: rapid depol. & overshoot $\rightarrow$ opening of voltage-gated Na^+ channels
- phase 1: initial rapid repol. $\rightarrow$ closure of Na^+ channels; opening of outward rectifying K^+ channels
- phase 2: plateau phase $\rightarrow$ slow Ca^{2+} influx; efflux of K^+
- phase 3: final repol. $\rightarrow$ closure of Ca^{2+} channels; opening of outward delayed rectifying K^+ channels
- phase 4: restoration of RMP $\rightarrow$ opening of inward rectifying K^+ channels

Slow Response AP (phase 0, 3, 4)

phase 0: opening of Ca^{2+} channels (influx) (less steep)

phase 3: closure of " " ; opening of K^+ channels (efflux)

phase 4: (slow diastolic depole.)
early part $\Rightarrow$ closure of K^+ channels
later part $\Rightarrow$ opening of transient Ca^{2+} channels

Pacemaker Potential: automaticity due to - spontaneous diastolic depolarization
↳ pacemaker potential; K^+ decay

Effect of Vagal Stimulation - slope $\downarrow \Rightarrow HR \downarrow$

(HR = heart rate)

ACh acts by 2 mechanisms - G protein activated K^+ channel

- M_2 muscarinic receptors

Effect of Sympathetic stimulation - slope $\uparrow \Rightarrow HR \uparrow$

mechanisms - norepinephrine binding to β_1 receptors

SA node

Internodal pathway

AV node

His bundle

Bundle branches

Purkinje fibres

(LV = Left ventricle
RV = right ventricle)

Initiation of excitation in SA node (60-90 ms)

Slowing in AV node due to - small size of nodal cells & their branching

- slow voltage-gated Ca^{2+} channels $\Rightarrow$ slow upstroke of AP

- few gap junctions (weak electrical coupling)

Importance of AV nodal delay - allows ventricular filling

- maintains low ventricular rate in AF

- drugs & vagal stimulation

Rapid conduction in ventricles (0.08 - 0.1 s)

complete excitation of LV, RV = 75 ms

(AP = action potential) (AF = atrial fibrillation)

Uses of ECG

Technical Aspects

Bipolar limb leads (I, II, III)

$$I \Rightarrow RA(-) \& LA(+)$$

$$II \Rightarrow RA(-) \& LL(+)$$

$$III \Rightarrow LA(-) \& LL(+)$$

Einthoven Δ

Unipolar limb leads - acting/recording & indifferent electrode

$$aVR \Rightarrow RA(+)\& LA+LL(-)$$

$$aVL \Rightarrow LA(+)\& RA+LL(-)$$

$$aVF \Rightarrow LL(+)\& RA+LA(-)$$

$$aVR = \frac{3}{2} VR$$

Chest/Precordial Leads (unipolar)

$$V_1 \rightarrow V_6$$

Indifferent electrode $\Rightarrow RA, LA, LL$

grounding electrode $\Rightarrow RL$

Normal ECG waves & segments

Intervals

$$PR \Rightarrow 0.12 - 0.20 s$$

$$QRS \Rightarrow 0.08 - 0.10 s$$

$$QT \Rightarrow 0.40 - 0.43 s$$

$$ST \Rightarrow 0.32 s$$

PP

RR

Cardiac Dipole As Vector

Heart Rate

$$\text{Atrial rate} = \frac{1500}{\text{PP interval (mm)}}$$

$$\text{Ventricular rate} = \frac{1500}{\text{RR interval (mm)}}$$

% RR interval is irregular $\Rightarrow$ count QRS complexes for 5s & extrapolate for 60s

Cardiac Rhythm

Short PR interval $\Rightarrow$ WPW syndrome

Long PR interval $\Rightarrow$ digitalis effect ; CAD

High amplitude of QRS complex $\Rightarrow$ ventricular hypertrophy

Mean QRS Axis

Right Axis deviation — Right ventricular hypertrophy
 — Left posterior hemiblock
 — WPW syndrome

Left Axis deviation — Left ventricular hypertrophy
 — Left anterior hemiblock
 — WPW syndrome

Sinus Arrhythmia

Mechanisms — alteration in autonomic activity
 — activation of baroreceptor reflex
 — irradiation from inspiratory center
 — activation of atrial stretch reflex

Paroxysmal Supraventricular Tachycardia (PST)

Nodal PST: AV nodal re-entry due to presence of dual AV nodal pathways
 $\rightarrow$ seen in normal individuals

Atrial PST: when an atrial focus discharges regularly (seen in digitalis patients)

Ventricular fibrillation: due to discharge from multiple ventricular ectopic foci

Heart blocks

1st degree: $\uparrow$ av nodal delay

2nd degree — Mobitz type I: PR interval is elongated progressively till a QRS complex is dropped $\Rightarrow$ Wenkebach Phenomenon

incomplete heart block

- acute inferior MI
- digitalis toxicity
- acute carditis

- Mobitz type II: ventricular beat in every 2 or 3 atrial beats (2:1 or 3:1 block)

- acute anterior MI
- degenerative disease of conduction system

(MI = myocardial infarction)

3rd degree: complete heart block

$\rightarrow$ atria & ventricle beat separately (AV dissociation)

- AV nodal block
- infranodal block

Idioventricular rhythm: portion of ventricular muscle becomes pacemaker (15-40 bpm)

$\downarrow$ circulation $\Rightarrow$ cerebral ischaemia $\Rightarrow$ fainting

faints again after sometime

regaining consciousness

circulation to brain improves due to lying posture of fainting

Stokes-Adam syndrome

Causes: - septal MI

- HIS bundle injury during surgery for repair of VSD
- digitalis toxicity
- degenerative disease of conductive system

Treatment: electronic pacemaker

Bundle Branch Block: prolongation of QRS complex

RBB block $\Rightarrow$ depression of ST segment ; inversion of T wave

LBB block $\Rightarrow$ R wave widens with 2 peaks

WPW Syndrome: aberrant pathway (bundle of Kent)

$\downarrow$
transmits impulses faster than usual $\Rightarrow$ ventricle is excited early

- PR interval shortened

- QRS is widened & slurred

Myocardial Ischemia: blood supply $\downarrow$ to ventricular tissue

- cells depolarize to lower RMP due $\downarrow$ K^+ conc. gradient
- ST segment $\Rightarrow$ + or - waves

His Bundle EG: better diagnosis of heart block

(catheter containing electrode introduced into RA close to tricuspid valve through an arm vein)

A deflection: activation of AV node

H " : transmission through His bundle

V " : ventricular depol.

Intervals - PA: time of conduction from SA to AV node

- AH: time " " through AV node

- HV: " " " " His bundle & bundle branches

Phases of Cardiac Cycle

V = ventricle
A = atrium

Atrial systole $\Rightarrow$ contributes to $\approx 20\%$ of ventricular filling

- V vol. $\uparrow$ mildly
- A pressure $\uparrow$
- A pressure $>$ V pressure
- 4th heart sound $\Rightarrow$ influx of blood into V
- mitral valve closes

Atrial Diastole: during ventricular systole

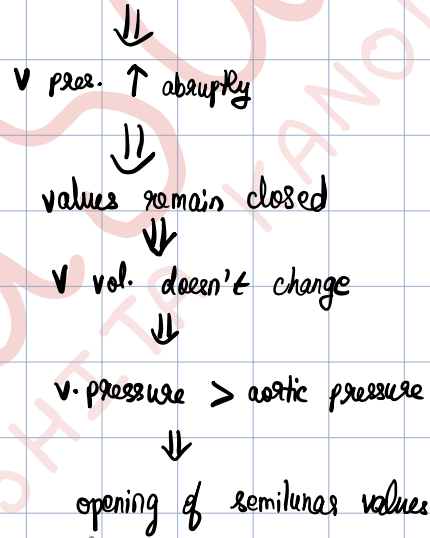
JVP waves

- a: rise of A pressure in atrial systole
- atrial diastole { c: bulging of AV valve into $\Rightarrow$ A pres. $\uparrow$
v: venous return $\uparrow \Rightarrow$ A. pres. $\uparrow$

(V = ventricular)
(A = atrial)

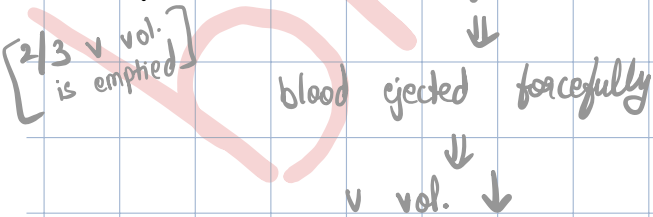
Ventricular Systole:

Isovolumetric Contraction: from peak of QRS complex in ECG / 1st heart sound in PCG



Ventricular Ejection:

Rapid Ejection: aortic blood flow $\uparrow$ abruptly



Reduced Ejection: V & aortic pressure $\downarrow$ due to run-off of blood from aorta to periphery



Ventricular Diastole

Isovolumetric Relaxation: $\rightarrow$ period b/w closure of aortic valve & opening of mitral valve
no change in v. vol.

steep $\downarrow$ in v. pressure



2nd heart sound in PCG due to closure of aortic valve



opening of mitral valve

(PCG = phonocardiogram)

Ventricular filling: $\left\{ \begin{array}{l} \text{rapid} \\ \text{slow (diastasis)} \end{array} \right.$

Rapid filling:

LV pressure $\downarrow$ < LA pressure



opening of mitral valve $\rightarrow$ v. wave in jvp

Diastasis: v. vol. curve $\uparrow$ slowly



v & A pressure remain unchanged

(ESV = end-systolic volume)

Pressure-Volume Relationship

Phase I: filling (50 mL $\xrightarrow{\text{ESV}}$ 120 mL $\xrightarrow{\text{EDV}}$)

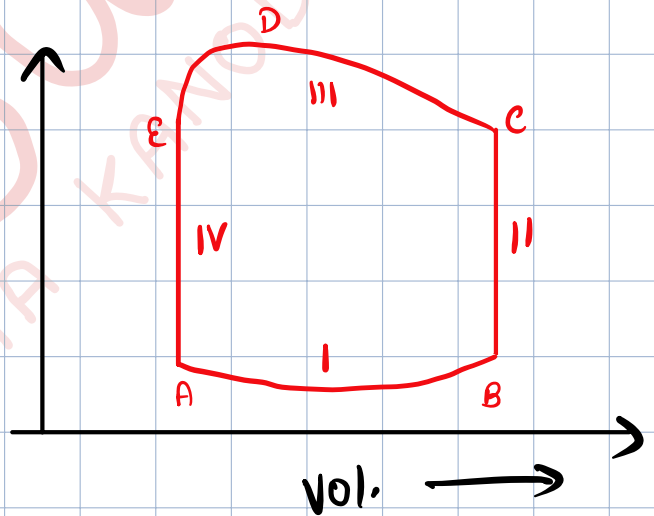
Phase II: isovolumetric contraction (Pressure reaches $\approx$ 80 mm Hg)

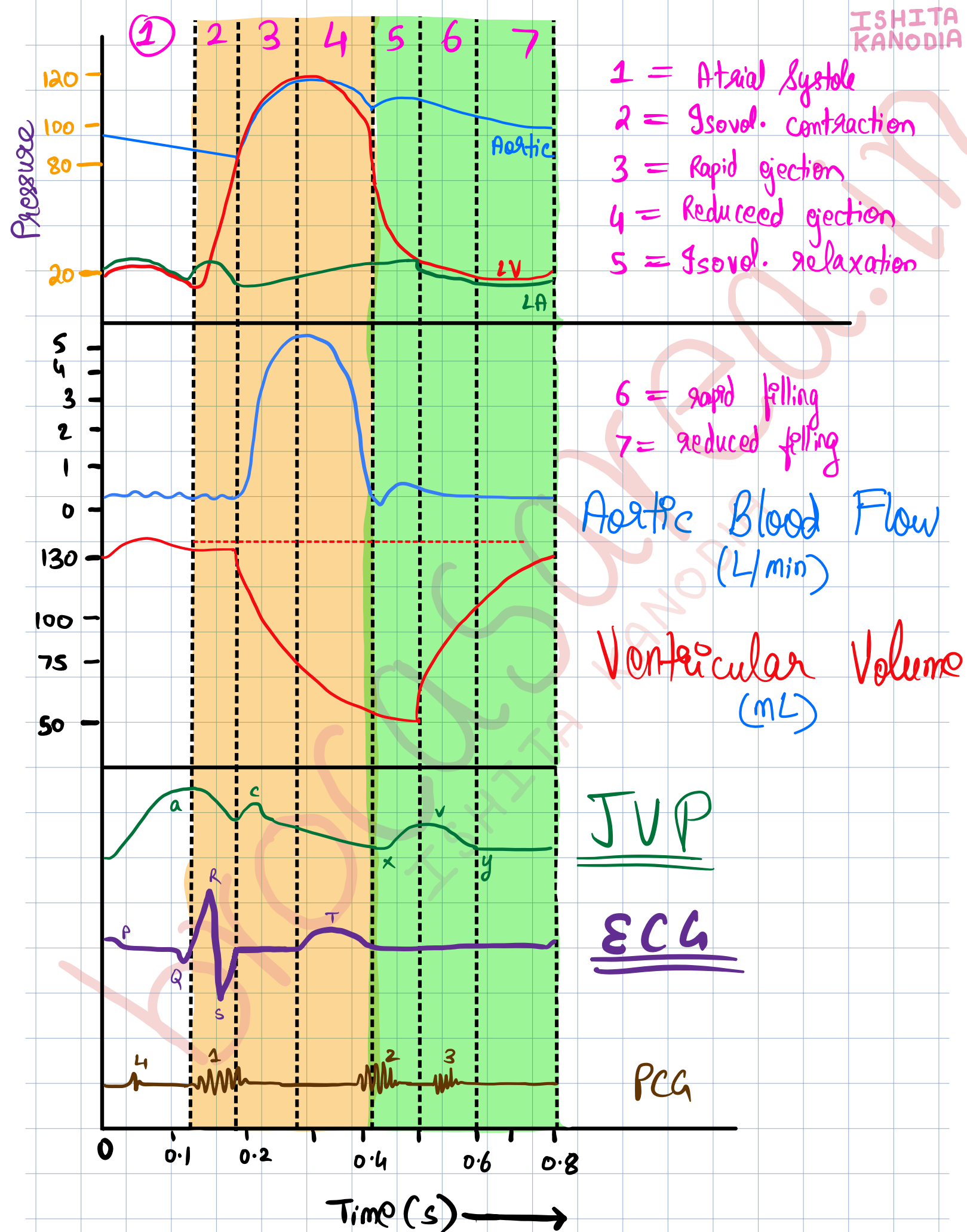
Phase III: ejection

Phase IV: isovolumetric relaxation (return to starting point - 50 ml, 2-3 mm Hg)

Systolic Dysfunction: pressure-volume curve shifts to right
(inability of ventricle to contract)

Diastolic Dysfunction: pressure - volume curve shifts to left
(inability of ventricle to relax)





Duration of Cardiac Cycle: 0.8s (75 bpm)

{ systole $\rightarrow$ 0.3s
diastole $\rightarrow$ 0.5s

- increased HR $\approx$ 200 bpm (shortening of systole < shortening of diastole)
- decreased HR $\approx$ 40 bpm (prolongation of systole < prolongation of diastole)

JVP (pressure change in A is directly reflected in JVs)

3 +ve, 2 -ve waves

(+) **a**: atrial contraction

(+) **c**: coincides with onset of systole (bulging of tricuspid valve into RA as RV pressure $\uparrow$)

(+) **v**: passive rise in pressure in RA as venous return continues while tricuspid valve remains closed

(-) **x**: fall of RA pressure due to RA relaxation

(-) **y**: fall in RA pressure when blood enters RV as tricuspid valve opens

Raised JVP - right sided heart failure

- increase in circulating blood vol. (pregnancy, acute nephritis)

- congestive heart failure

Absence of a wave - atrial fibrillation

Prominent v wave - tricuspid regurgitation

Giant a wave - pulmonary hypertension.

Heart Sounds:

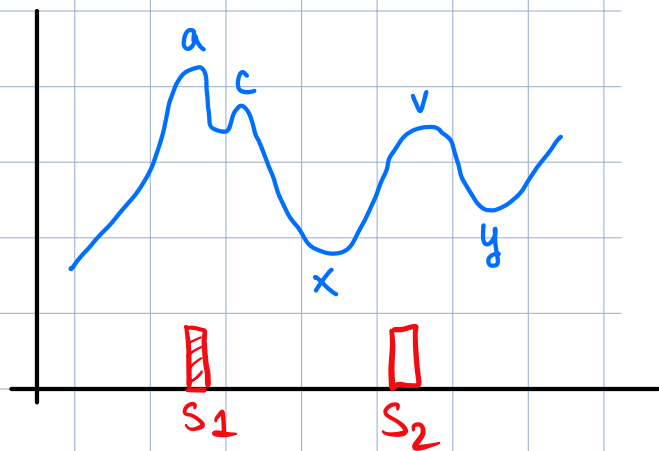
S1: sudden closure of AV valve (at beginning of systole)

$\rightarrow$ 0.15s $\rightarrow$ 1st
 $\rightarrow$ 25-45 Hz

Accentuation: - exercise
- hypertension

Diminution: - shock, obesity, emphysema

splitting: 2 components - mitral closes slightly before tricuspid (pathological).



A₂ (aortic " ")
 ↳ P₂ (pulmonary component)

S₂: closure of semilunar valves

- ↳ 0.12s
- ↳ 50 Hz
- ↳ dup

Loud A₂: systemic hypertension

Diminished A₂: aortic stenosis

Loud P₂: pulmonary hypertension

Diminished P₂: pulmonary stenosis

Splitting: aortic valve closes before pulmonary valve

→ distinctly heard during inspiration

→ becomes lesser in expiration.

S₃: → sign of heart failure
 heard in conditions of hyperkinetic circulation

- ↳ 0.1s
- low-pitched
- best heard in mitral area
- heard in VSD, mitral regurgitation

(VSD = ventricular septal defect)

S₄: atrial sound (produced during A contraction)

- ↳ never heard in normal individuals
- low pitched
- indicates increased stiffness / non-compliance of vessels
- left ventricular hypertrophy
- MI
- pulmonary hypertension

Triple Sound 1st = S₁

2nd = S₂

3rd = S₃ or S₄.

when HR > 100 bpm ⇒ triple sound is called gallop rhythm

Murmurs: occur due to turbulence in blood flow at / near a valve.

Cardiac output (CO) 5-6 L/min

Stroke volume (SV) ≈ 70 mL

Cardiac Index (CI) ≈ 3.2 L

EDV 130 mL (120)

Ejection fraction (EF) 65%

ESV 50 mL

Cardiac Reservoir 15-25 L/min
20-40 L/min $\Rightarrow$ athlete

Physiological factors affecting CO

Age $\downarrow$ in elderly (HR $\downarrow$)

Gender 10% $\downarrow$ in females

Exercise $\uparrow$ (HR $\uparrow$, SV $\uparrow$)

Pregnancy ECF vol. $\uparrow$

Posture standing $\downarrow$ (pooling)

Environmental Temp. $\uparrow$ hot

Direct Measurement of CO

Indirect " " "

Fick method

indicator dilution method (Hamilton's dye dilution method)

Thermo-dilution method

Ballistocardiography

Echocardiography

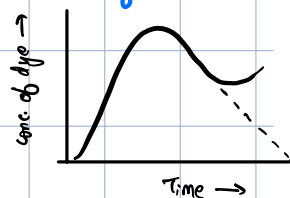
X-ray method

Pulse-pressure method

Evan's blue / Indocyanine green

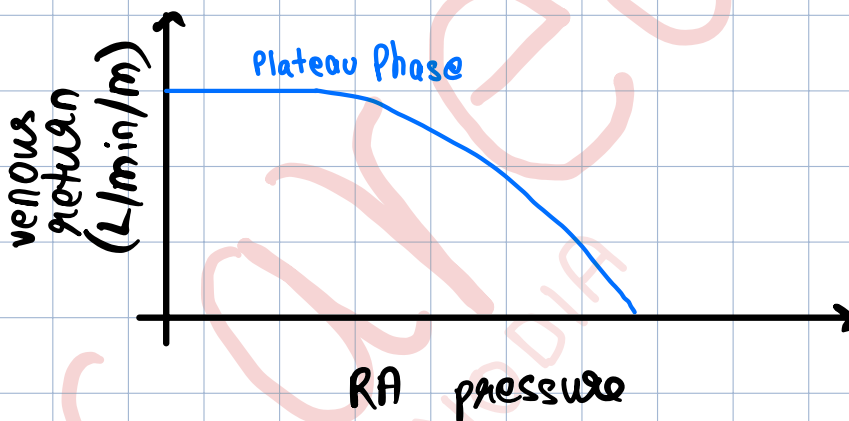
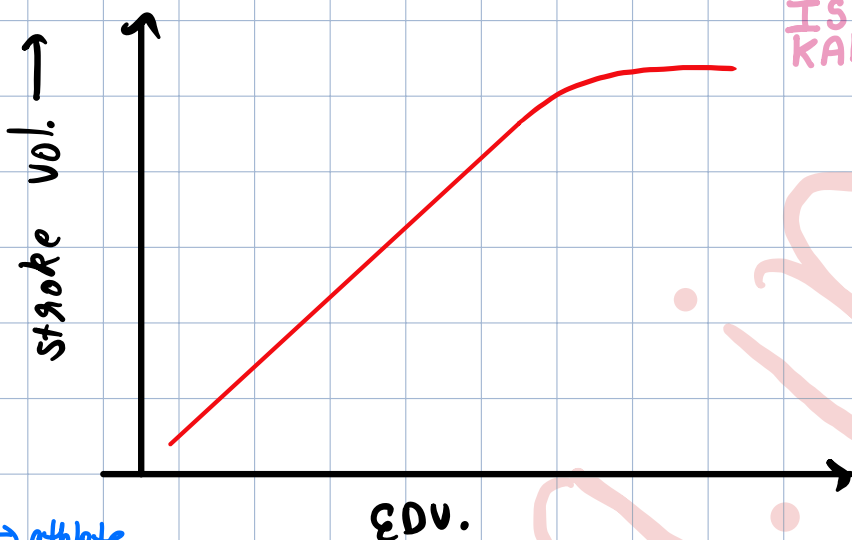
$$F = \frac{I}{C \times t}$$

$$CO = F \times 60$$



Fick's Principle

Amount of substance taken up by an organ/whole body = AV difference of substance $\times$ blood flow



$\uparrow = (+) \Rightarrow \text{increase/rise}$
 $\downarrow = (-) \Rightarrow \text{decrease/fall}$

Factors affecting CO

factors affecting SV

Preload

venous return *

atrial pump activity

20%; 80%

ventricular compliance $\propto$

Inotropic State (myocardial contractility) *

Afterload

(homometric auto-regulation / Anrep effect)

vessel diameter (vascular hindrance)
 viscosity of blood (haematological ")
 (CO increases in anemia) $(-)$

factors affecting HR

CO does not $\uparrow$ proportionately to that of $\uparrow$ of HR

vagal stimulation $(-)$

sympathetic $(+)$

$\therefore$ in severe tachycardia, diastole is shortened

* venous return

skeletal muscle pump $\uparrow$ (walking vs. quite standing)

thoracic pump $\uparrow$ inspiration

abdominal pump $\uparrow$ inspiration

ECF volume $\propto$

sympathetic activity $\propto$

RA pressure $\uparrow / \propto$

Degree of filling of systemic circulation $\propto$

Resistance to blood flow in venous system $1/\propto$

* Factors affecting CO

ventricular muscle mass $\propto$

autonomic activity $(S+)$

hormonal factors

catecholamine $(+)$

Ach $(-)$

Glucagon $(+)$

Insulin $(+)$

Thyroxine $(+)$

chemical factors

$(+)$

$\Rightarrow$ xanthines (caffeine, theophylline)

$(-)$

$\Rightarrow$ hypercapnia, hypoxia, acidosis, general anaesthetics, toxins

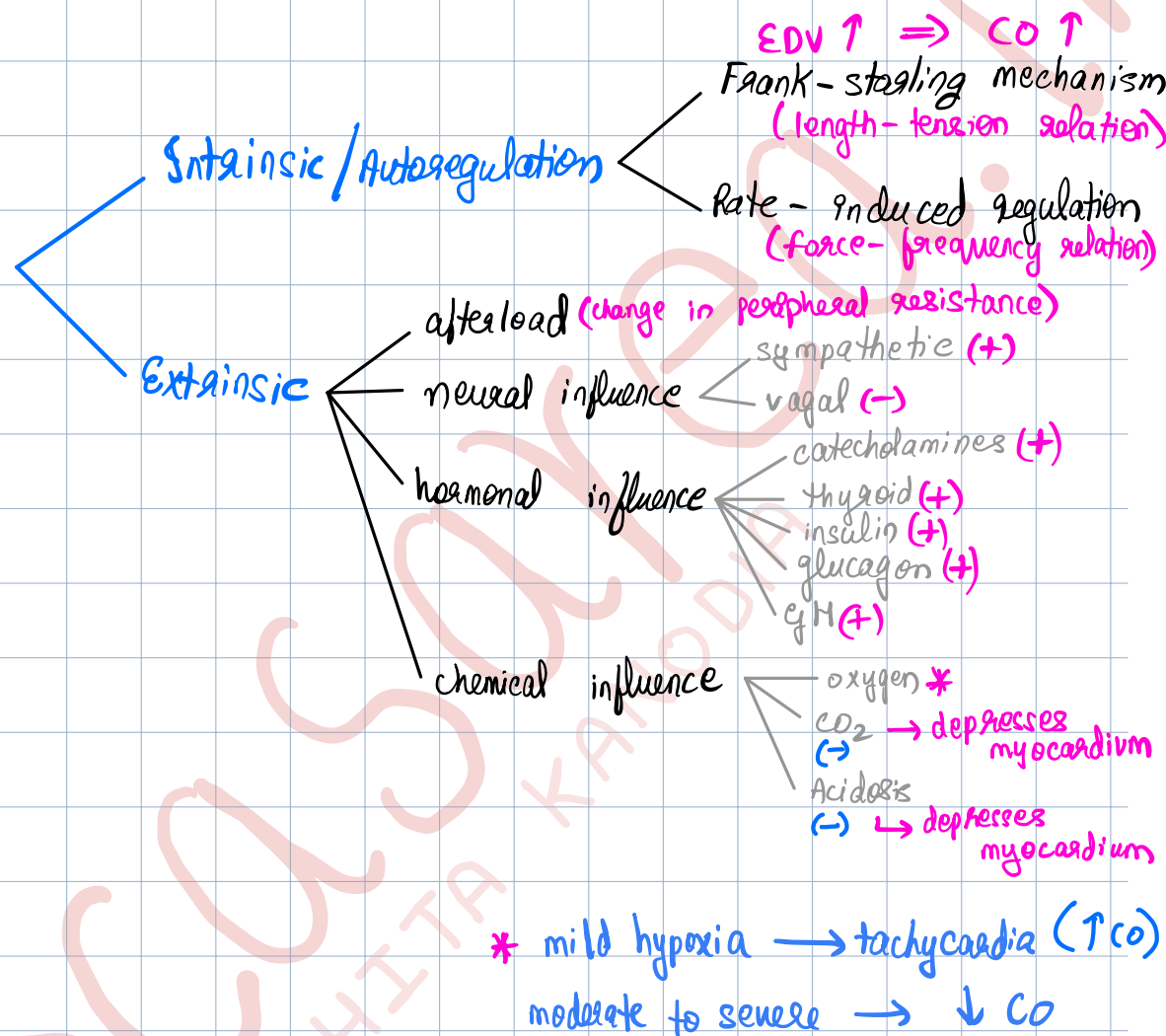
drugs

Digitalis $(+)$

Quinidine, procainamide,

barbiturates $(-)$

Regulation of CO



HR - rate of discharge of SA node

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Bradycardia < 60 bpm
Tachycardia > 100 bpm
Infants & children ↑
geriatric ↓

Physiological Variations

Age - ↑ in children & infants ; ↓ in geriatric

Gender - ↑ in females

Diurnal variation - ↑ in the day (esp. afternoon) ↓ at night

Respiration - sinus arrhythmia ⇒ ↑ during inspiration, ↓ during expiration

Body Temp. - ↑

Env. Temp. - ↑ summer

Food intake - ↑ (∵ rate of metabolism ↑)

Posture - supine to standing posture ↑ (due to ↓ stimulation of baroreceptors)

Exercise - ↑ (due to sympathetic stimulation)

Regulation of HR

Neural

Autonomic (effect of sympathetic stimulation on HR remains for a longer period than vagal stimulation)

Reflex - baroreceptor, chemoreceptor, Cushing's, etc.

Regulation by higher centres - stimulation of motor cortex, frontal lobe, limbic system, thalamus

Humoral

Thyroxine, catecholamines ↑

Hypoxia ↑

Hypercapnia, acidosis ↓

Pulse deficit

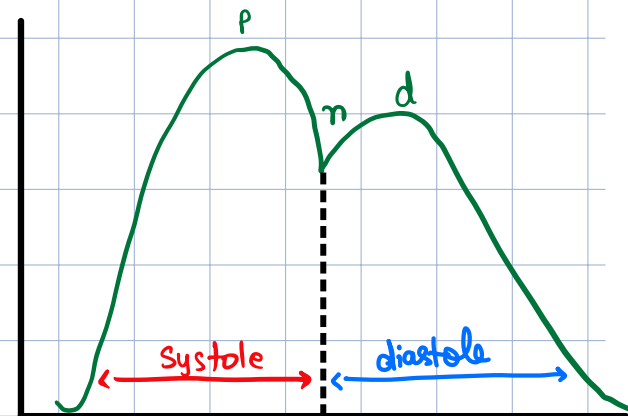
- absent normally
- > 10 ⇒ atrial fibrillation
- < 10 ⇒ other arrhythmias, heart blocks

Normal Pulse Tracing: (two waves, 1 notch)

Percussion (p) wave - ejection of blood during V systole

Dicrotic (d) wave - rebound of blood against closed aortic wall during diastole

Dicrotic (n) notch - closure of aortic valve



Tachycardia (>100)

- Physiological - exercise, anger, emotion, infants & children, pregnancy
- Pathological - fever, anemia, heart failure

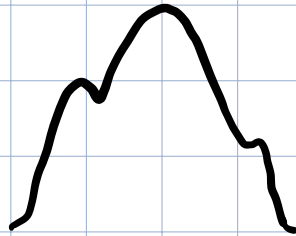
Bradycardia (<60)

- Physiological - athletes, geriatrics, yoga practitioners, grief
- Pathological - myxedema, increased intracranial pressure, obstructive jaundice, digitals

Common Abnormal Pulses:

Anacrotic Pulse: (anadicrotic)

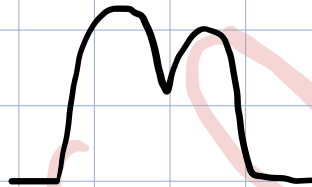
- ↳ two upbeats in p wave
- aortic stenosis



$\left[\uparrow 1^\circ = \uparrow 10 \text{ beats} \right]$
normal.

Dicrotic Pulse: twice-beating pulse

- ↳ d wave is prominent
- typhoid fever



[Pulsus ectardus]

Water-Hammer / Corrigan's / Collapsing pulse:

- ↳ in aortic regurgitation (POA, VSD)
- no dicrotic notch
- rapid upstroke & rapid downstroke



- steep rise $\Rightarrow$ due to $\uparrow$ EDV (filling also occurs from incompetent aortic valve in addition to filling from LA)
- steep fall due to
 - diastolic run-off of blood into V
 - rapid run-off of blood into periphery due to $\downarrow$ vascular resistance

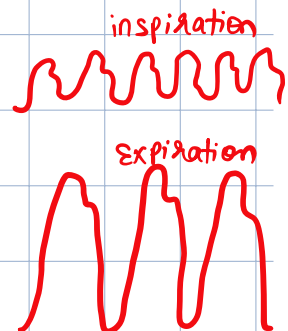
Pulsus Bisferiens: combination of anacrotic & collapsing pulse

- ↳ aortic stenosis

Pulsus Paradoxus: grossly decreased amplitude of pulse during inspiration

- ↳ constrictive pericarditis
 - ↳ pericardial effusion
 - ↳ asthma
 - ↳ massive pleural effusion
- } restriction to expansion of heart chambers $\therefore$ incomplete filling

Mechanisms: during inspiration - intrathoracic pressure becomes more -ve $\Rightarrow$ venous return $\downarrow$
- intrapericardial pressure $\uparrow \Rightarrow$ venous return $\downarrow$

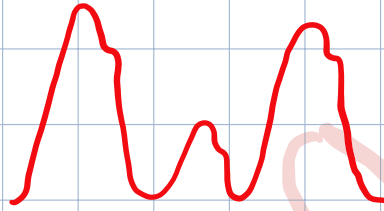
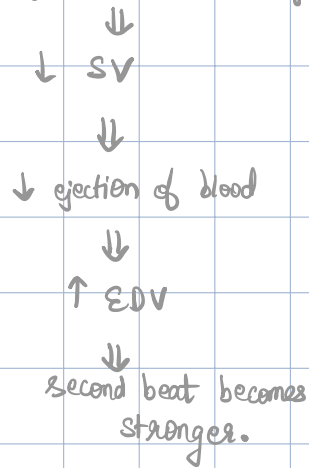


Pulsus Alternans: alternating beats are strong & weak

↳ diff. of 5-20 mm Hg systolic pressure b/w 2 consecutive beats

→ left v failure $\Rightarrow$ $\downarrow$ myocardial contractility

→ toxic carditis



Haemodynamics

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Turbulent flow: occurs when velocity > critical velocity

probability of turbulence is expressed in terms of Re (Reynolds number)

$$Re = \frac{\rho D v}{\eta} \quad \left[\begin{array}{l} Re < 2000 \Rightarrow \text{laminar} \\ Re > 3000 \Rightarrow \text{turbulent} \end{array} \right]$$

Korotkov sounds are produced by turbulent flow

Poiseuille-Hagen Formula: relationship b/w viscosity of fluid with radius & length of tube

$$\text{Resistance (R)} = \frac{8 \eta L}{\pi r^4}$$

$\therefore$ BP $\uparrow$ markedly with vasoconstriction.

Factors affecting Peripheral Resistance $\left\{ \begin{array}{l} \text{Radius of blood vessel (Vascular hindrance)} \\ \text{Viscosity of blood (haematological hindrance)} \end{array} \right.$

Plasma Skimming — cells mostly accumulate at center of flowing column of blood

$\therefore$ periphery of blood vessel has low cell content

— blood entering smaller branches mostly comes from peripheral part of column of blood

$\therefore$ smaller branches receive blood with lower cell content.

Laplace Law: relationship b/w distending pressure (P), tension (T) & radius (r)

• Sphere $\Rightarrow P = \frac{2T}{r}$

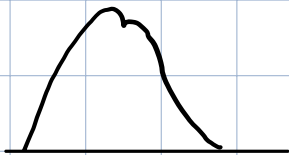
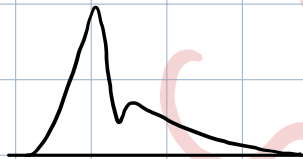
• cylinder $\Rightarrow P = \frac{T}{r}$

$\rightarrow$ if thickness of vessel wall (w) is taken into consideration

$$T = P \times \frac{r}{w}$$

Applications: — wall tension increases when organ gets filled (wall tension $\downarrow$ when thickness $\uparrow$)

- in ventricular hypertrophy, as wall is thick $\therefore$ wall tension is less
- in a dilated heart (as seen in heart failure), more energy is required to pump blood as wall tension is more
 - $\therefore$ dilated heart pumps less efficiently
 - $\therefore$ dilated heart fails faster.

Central Pulse
TracingPeripheral Pulse
Tracing

Arterial Pressure

Pulse Pressure

Capillary circulation: microcirculation (except lens, cornea)

→ single layer of endothelial cells

→ tunica media is completely absent

true capillaries: emerge from arteriole/metarteriole & form anastomosing network that drain into venules

Thoroughfare vessel^(TV): metarteriole is connected directly to venule by TV

Types of Capillaries:

- continuous (non-fenestrated) ⇒ clefts / gap at inter-endothelial junction = 4-10 nm
⇒ in skeletal & smooth muscles
- fenestrated ⇒ pores (20-100 nm)
⇒ in intestinal villi, choroid plexus in brain
- sinusoidal / discontinuous ⇒ gaps > 400 nm
⇒ in spleen, liver, bone marrow

Capillary Exchange:

- diffusion
- vesicular transport
- filtration

Hydrostatic Pressure arterioles end: 37 mm Hg

venules end: 17 mm Hg

interstitial tissues: 1 mm Hg

Osmotic Pressure tissue fluid: nil

blood: 25 mm Hg

Net filtration pressure arterioles end: $[(37-1) - 25] = 11 \text{ mm Hg}$ in outward direction

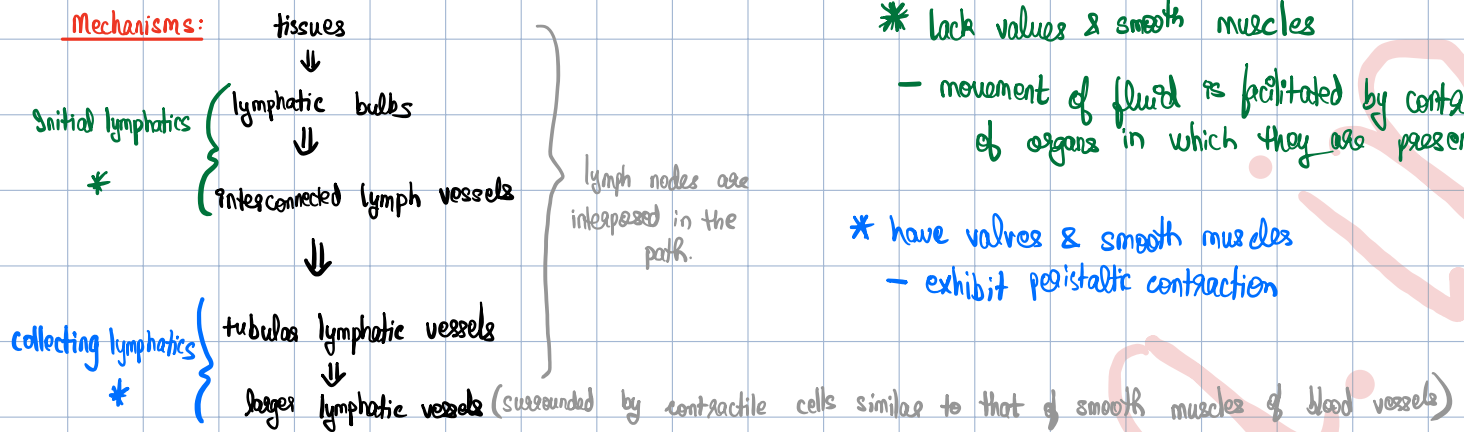
venules end: $[25 - (17-1)] = 9 \text{ mm Hg}$ in inward direction (into capillaries)

Arteriovenous Anastomoses (arteriovenous shunts)

- directly connect arterioles to venules bypassing capillaries
- thick muscular wall
- densely innervated by sympathetic Ns.
- in fingers, palms, ear lobes.

Lymphatic Circulation

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* lack valves & smooth muscles

- movement of fluid is facilitated by contraction of organs in which they are present

* have valves & smooth muscles

- exhibit peristaltic contraction

Lymph: modified tissue fluid

→ chiefly lymphocytes

→ large molecules of fat (chylomicrons)

→ after a fatty meal, fat globules are so numerous that lymph becomes milky (called chyle)

→ formed by transcapillary exchange & exchange b/w tissue fluid & lymphatics

→ lymph flow is much slower (1ml/min in thoracic duct) than blood

→ colloidal osmotic pressure is lesser than that of plasma.

Functions of Lymphatic circulation:

- prevent edema formation
- maintain plasma protein content
- absorption of long-chain fat, cholesterol, fat-soluble vitamins from intestine
- contain mononuclear phagocytes in lymph nodes which remove antigens from lymph.

Edema formation:

increased filtration of fluid into interstitial tissue space

decreased removal of fluid from interstitial tissue space

increased hydrostatic pressure of capillary blood *

decreased oncotic pressure
(hypoproteinemia in liver disease / kidney disease / malnutrition / burns)

increased capillary permeability
(histamine, bradykinin)

infection (filariasis)

surgery (radical mastectomy that removes lymphatics)

diseases of lymphatics (lymphangitis)

increased venous pressure (congestive cardiac failure)

venous constriction

increased ECF vol.

arteriolar dilation (local inflammation)

Edema due to lymphatic obstruction is usually non-pitting type.

Regulation of peripheral blood flow

intrinsic

autoregulation
(60-180 mm Hg)

myogenic theory

metabolic theory
(reactive hyperemia)

tissue pressure theory

endothelium-mediated regulation

extrinsic

neural

sympathetic

parasympathetic

hormonal

 β -receptors (vasodilation) epinephrine α -receptors (vasoconstriction) norepinephrineRegulation of BP:

SBP :- 100 - 119 mm Hg (120 - 139 ; > 140)

DBP :- 60 - 79 mm Hg (80 - 89 ; > 90)

Pulse pressure :- 20 - 50 mm Hg (30 - 60)

MAP :- 75 - 105 mm Hg $\left(DBP + \frac{1}{3} PP \right)$

SBP = Systolic Blood Pressure

DBP = Diastolic " "

MAP = Mean arterial " "

PP = Pulse pressure

Factors Affecting BP: $BP = CO \times PR$ CO $\rightarrow$ SBPPR $\Rightarrow$ DBPFactors that $\uparrow$ viscosity

Polycythemia

Hyperproteinemia

Hereditary spherocytosis

 $\downarrow$ temp.

(temp. = temperature)

Physiological variations of BP

- Age: $\uparrow$ with age
- Gender: less in females during reproductive life
- Eating: $\uparrow$ after a meal
- Sleep: $\downarrow$ during sleep
- Emotion: $\uparrow$ sympathetic discharge $\Rightarrow \uparrow$ BP

• **Exercise:** $\uparrow$ SBP due to $\uparrow$ CO

$\rightarrow$ DBP $\uparrow$ in mild exercise (due to vasoconstriction); DBP $\downarrow$ in moderate/severe exercise due to metabolic vasodilation & $\uparrow$ body temp.

• **Posture:** BP $\downarrow$ on immediate standing

$\rightarrow$ Baroreceptor reflex controls BP in 15-30s.

• **Temperature:** BP $\downarrow$ in hot envi. (due to cutaneous vasodilation)

• **Pregnancy:** CO $\uparrow \Rightarrow$ SBP $\uparrow$

PR $\downarrow \Rightarrow$ DBP $\downarrow$

$\therefore$ pulse pressure $\uparrow$.

Short-term Regulation:
(Seconds - minutes)

Neural

Autonomic

Medullary - VMC, CIC

Hypothalamic
Cortical (+)
Reflex

Sympathetic (S)

Parasympathetic (PS)

Vasodilator

Vasoconstrictor (+ venoconstriction)

systemic blood vessels lack PS fibres

Baroreceptor / Sino-aortic Reflex
Chemoreceptor (40-70 mm Hg)
Cushing
Somatosympathetic
Atrial stretch
Brainbridge

Vascular

Capillary fluid shift

Stress relaxation

Hormonal

Catecholamines +

RAAS +

ADH +

ANP -

Kinin-kallikrein -

Histamine -

Endothelins +

EDRF -

Adrenomedullin -

Ventral medullary centre

* VMC (+)

(caudal ventrolateral medulla)

CVLM

(intermediate)

IULM

(rostral)

RULM (main)

vasoconstriction & cardioacceleration

$\downarrow$

(+) BP

(ILH = intermediolateral horn of spinal cord)

* Hypothalamic (HT)

anterior HT $\rightarrow$ bradycardia (-)

posterolateral HT $\rightarrow$ tachycardia (+)

ADH - long-term regulation (+)

NTS (Nucleus Tractus Solitarius)

NA (Nucleus Ambiguus)

DMN of Vagus

(Dorsal motor nucleus)

CIC (-)

(bradycardia, $\downarrow$ CO)

Cardiac inhibitory centre

RAAS = Renin angiotensin aldosterone system
ADH = Anti-diuretic hormone
ANP = Atrial natriuretic factor
EDRF = endothelin derived release factor

BP ↑

~~Baroreceptor~~
reflex(50-200 mm Hg)
(70-140 ⇒ linear)Stretch of carotid sinus
& aortic arch

↑ discharge of CN 9 & 10

stimulation of NTS

Activation
of vagusInhibition of
VMC↓ Myocardial
contractility

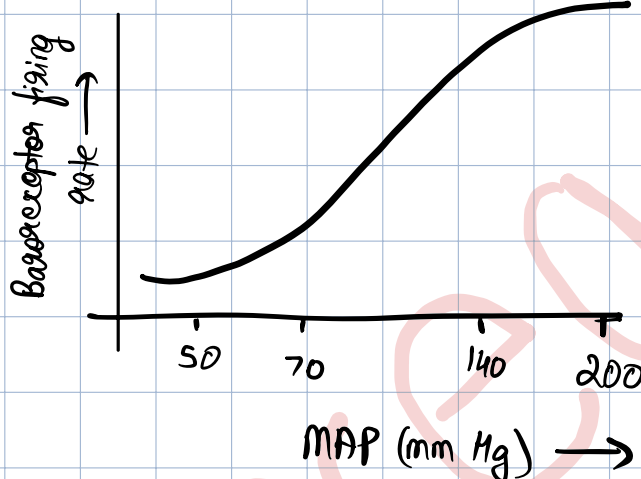
Vasodilation

↓ CO

BP ↓

→ life-saving in daily life in
change in posture

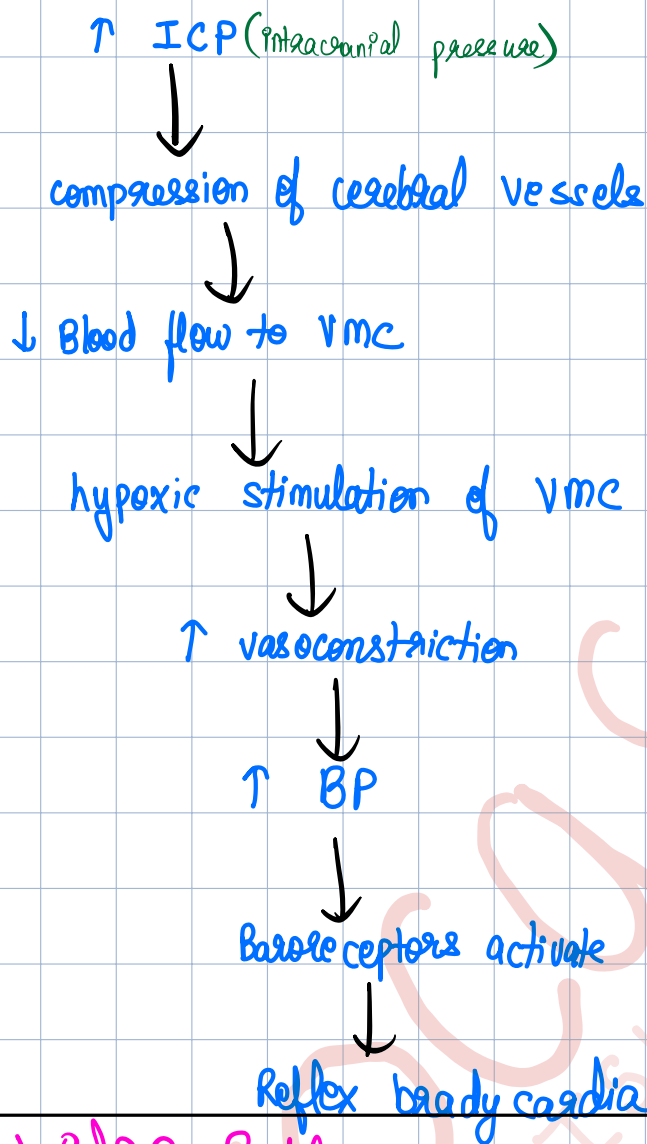
→ life-saving in acute hypotension & haemorrhage



(CN = cranial nerve)

Cushing

(last physiological reflex to correct BP)



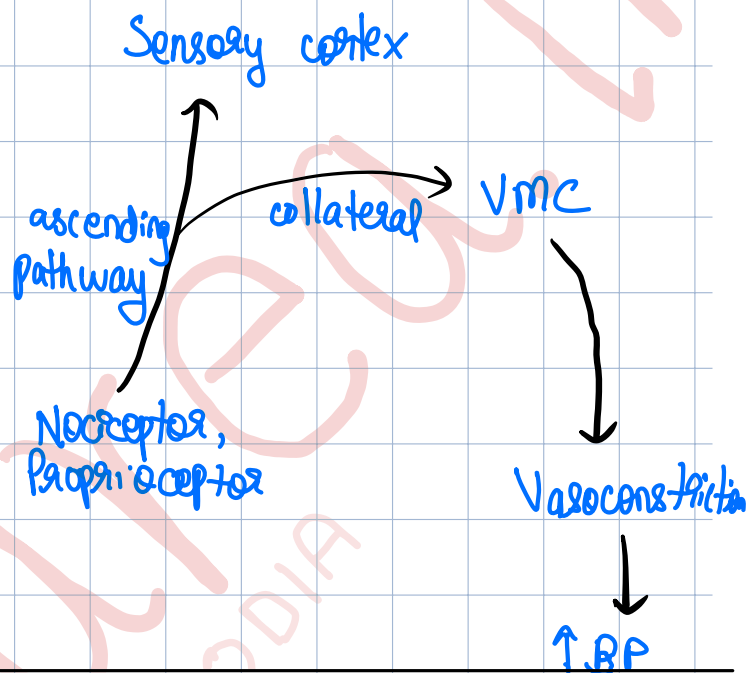
Barbridge Reflex

Infusion of Saline (with initial ↓ HR)

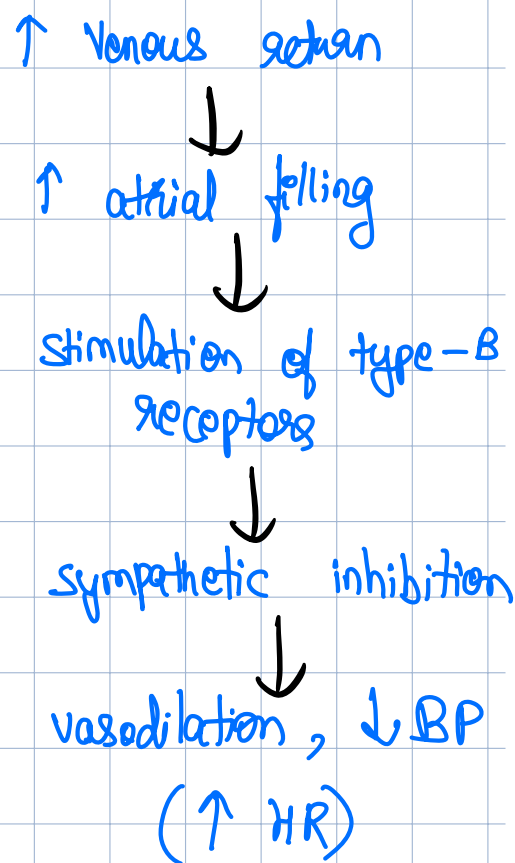


Parasympathetic Reflex

ISHITA KANODIA



Atrial Stretch Reflex



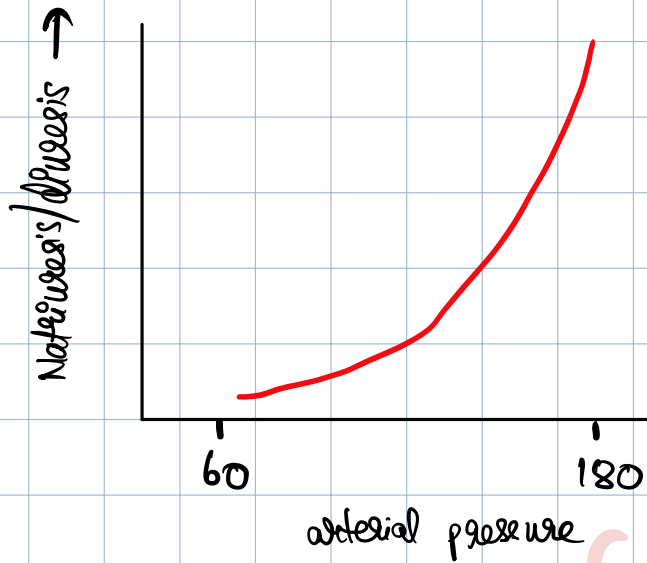
Long-Term Regulation

Renal (60 - 200 mm Hg)

of Arterial pressure $\uparrow \Rightarrow$ pres. natriuresis
(intrinsic property of kidney) $\Rightarrow$ pres. diuresis

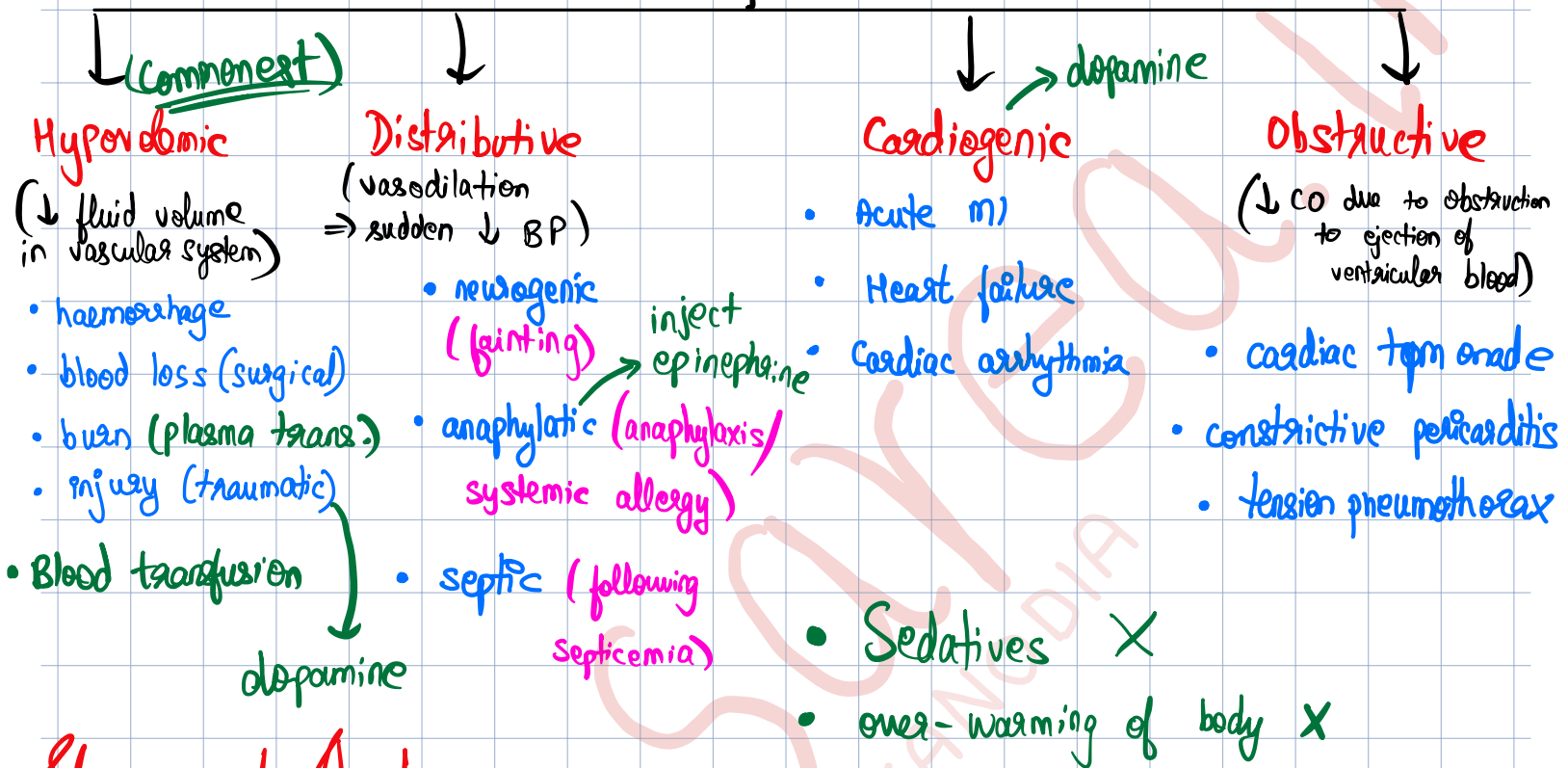
Hormonal

- Aldosterone
- ADH
- ANP



Shock

inadequate tissue perfusion
associated with relative/absolute
decrease in CO



Stages of Shock

Non-progressive / Compensatory ⇒ fluid conservation by kidney, vasoconstriction, tachycardia

Progressive ⇒ failure of compensatory mechanism

⇒ reduced tissue perfusion leading to hypoxia of all body tissues (except brain & heart)

Irreversible / Refractory ⇒ tissue damage cannot be reversed

⇒ MI

⇒ hypoxic & ischemic injury

⇒ acute tubular necrosis in kidney

Heart Failure

ISHITA
KANODIA

Pressure Overload

$\uparrow$ BP $\Rightarrow$ $\uparrow$ afterload $\Rightarrow$ heart pumps against $\uparrow$ resistance

Normal

$$\text{Stress} = \frac{P \times R}{T}$$



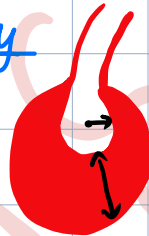
$\begin{cases} P = \text{pressure} \\ R = \text{radius} \\ T = \text{wall thickness} \end{cases}$

Early Stage

$$\uparrow \text{ stress} = \frac{P \uparrow \times R}{T}$$

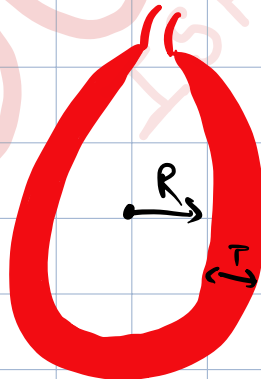
Stage of Concentric Hypertrophy

$$\text{Normal stress} = \frac{\uparrow P \times R \downarrow}{T \uparrow \uparrow}$$



State of hypertrophy & dilation

$$\uparrow \text{ stress} = \frac{\uparrow P \times R \uparrow}{T \uparrow}$$



Volume Overload

$\uparrow$ venous return

Normal

$$\text{Stress} = \frac{P \times R}{T}$$

State of dilation

$$\uparrow \text{ stress} = \frac{P \times R \uparrow}{T}$$

State of dilation & hypertrophy

$$\text{Normal stress} = \frac{P \times R \uparrow}{T \uparrow}$$

State of further dilation

$$\uparrow \uparrow \text{ Stress} = \frac{P \times R \uparrow \uparrow}{T \uparrow}$$