Pharmacology 2 is.

Sure shot
↓
PYQs
↓
main (extra)
↓
Buffer.

PHYSIOLOGY BINGE REVISION

Medsynapse by Dr. Nikita

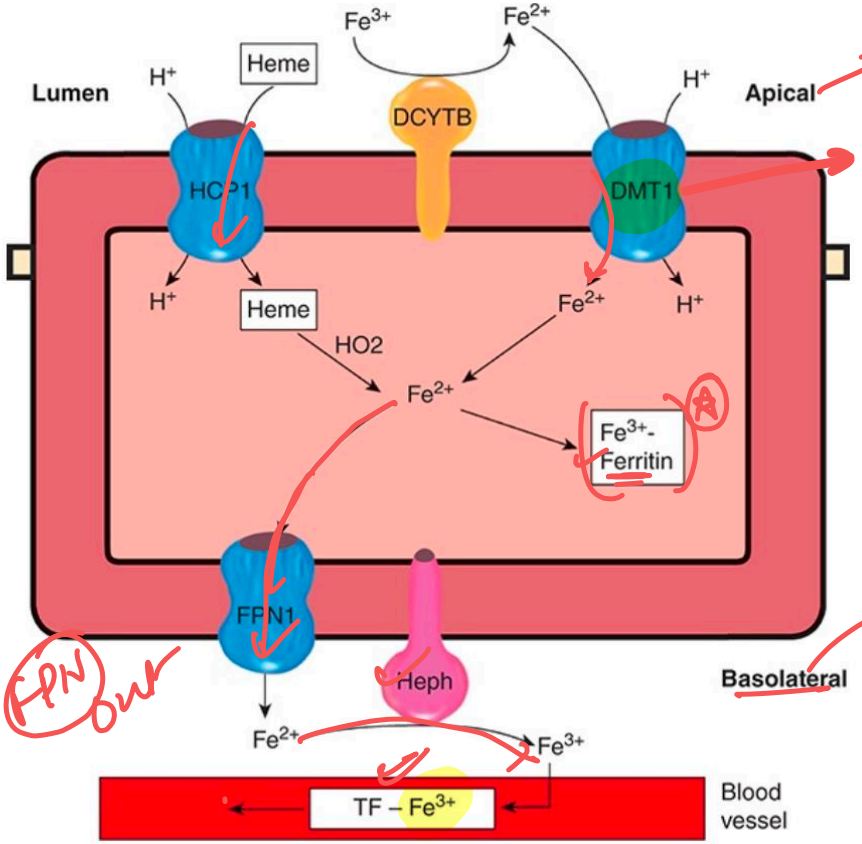


FIGURE 26–8 Intestinal absorption of iron. Fe^{3+} is converted to Fe^{2+} by the ferric reductase DCYTB, and Fe^{2+} is transported into the enterocyte by the apical membrane iron transporter DMT1. Heme is transported into the enterocyte by a separate heme transporter (most likely heme carrier protein 1, HCP1), and heme oxygenase-2 (HO2) releases Fe^{2+} from the heme. Some of the intracellular Fe^{2+} is converted to Fe^{3+} and bound to ferritin. The rest binds to the basolateral Fe^{2+} transporter ferroportin-1 (FPN1) and is transported to the interstitial fluid. The transport is aided by hephaestin (Heph) which converts Fe^{2+} to Fe^{3+}. In plasma, Fe^{3+} is transported bound to the iron transport protein transferrin (TF).

$$Q = \frac{\pi \Delta P r^4}{8 \eta l}$$

Flow

vessel → length 2x ↑ → flow − 1/2

diameter/radius 2x → 2r⁴/d⁴ → 16 times

• Resistance → flow 16x resist 1/16.

- Cardiac output can be computed as $CO = \frac{V_{O_2}}{C_a - C_v}$ (O₂ uptake / Oa − Ov.)
- Where V_{O_2} is oxygen uptake, and C_a and C_v are oxygen content of the arterial and venous blood.
- V_{O_2} is determined by breathing or mechanical method using a spirometer or indirectly by a calorimeter monitor.

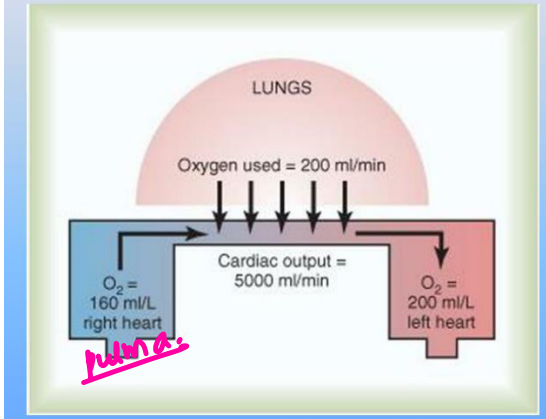
★ (INI) AV O2 gradient

man → heart

mini → kidney

(C.O) numerical

FICK PRINCIPLE



Output of Left Ventricle

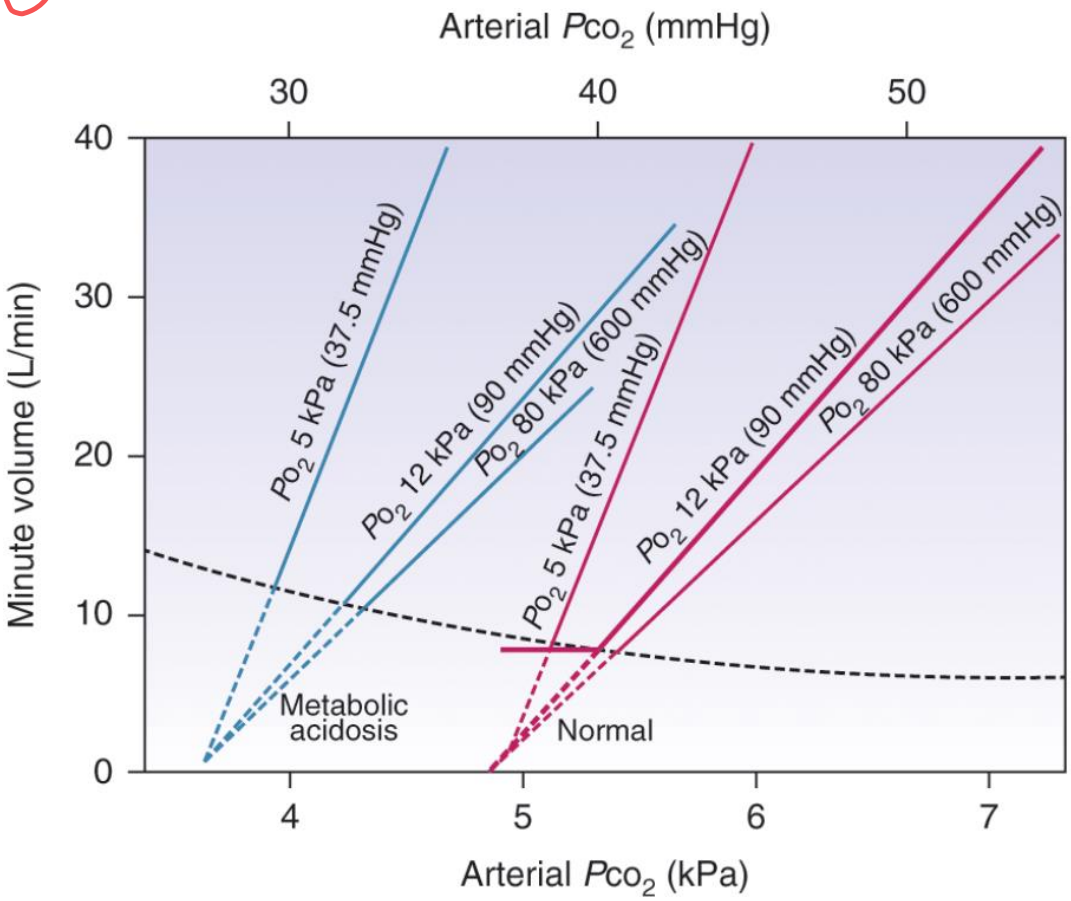
= Oxygen Uptake by lungs ml/min / $A_{O_2} - V_{O_2}$

= 200 ml / min / (200 ml / L − 160 ml / L)

Art blood − {Venous blood [Pul artery}} ★.

= 200 ml/min / 40ml / liter

= 5 L/min ★.



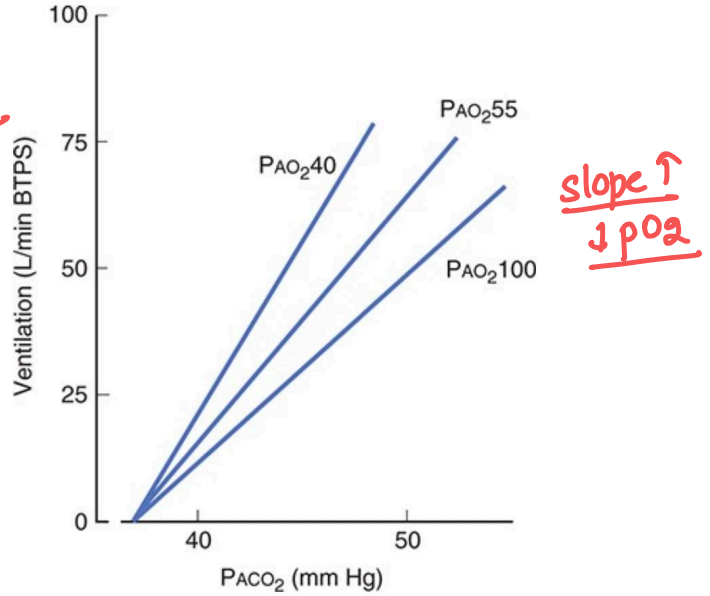
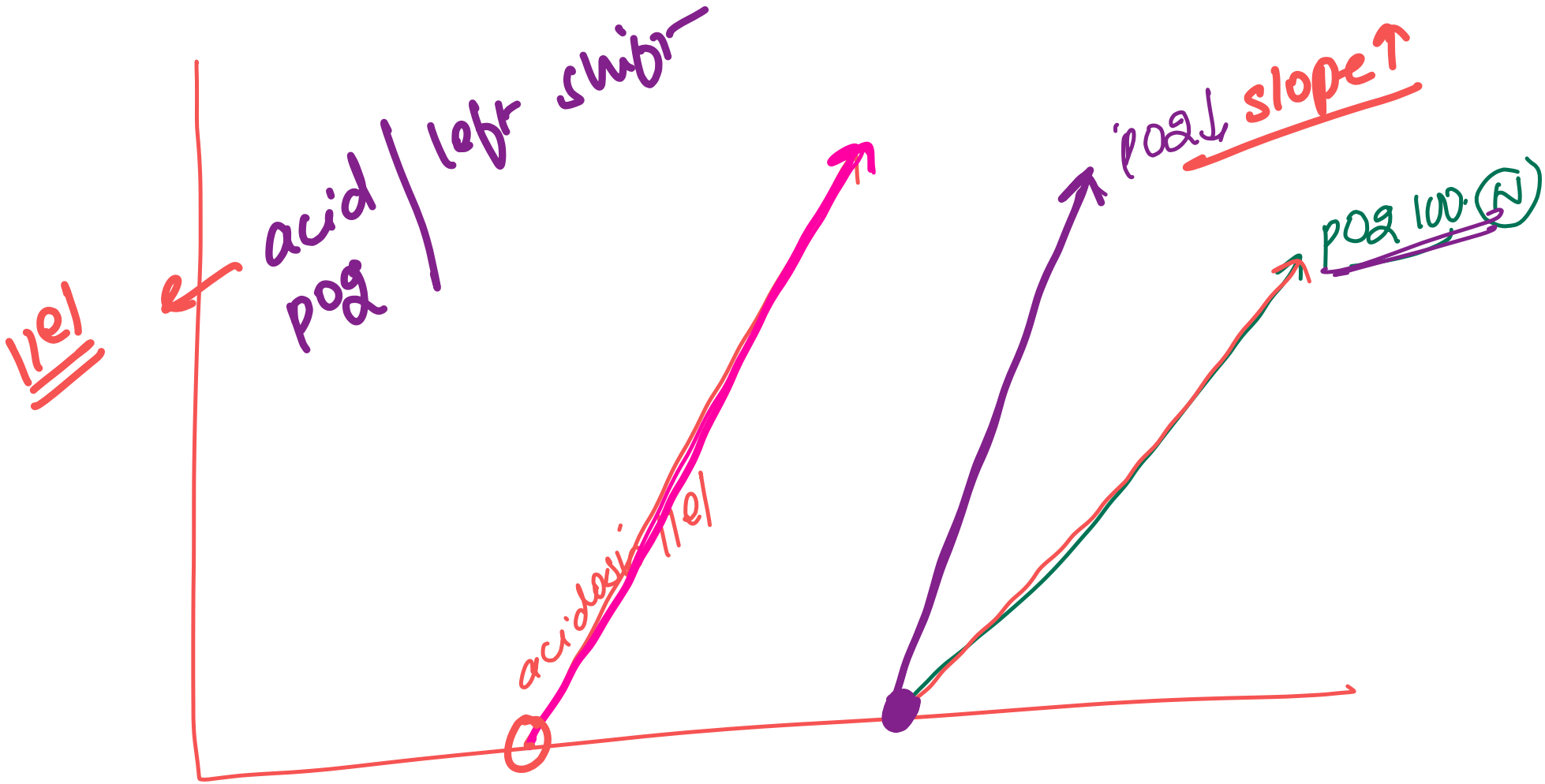


FIGURE 36–12 Fan of lines showing CO_2 response curves at various fixed values of alveolar Po_2. Decreased P_{AO_2} results in a more sensitive response to P_{ACO_2}.


acid
pog
left shift
vel
acidosis
pog ↓ slope ↑
pog 100 (N)

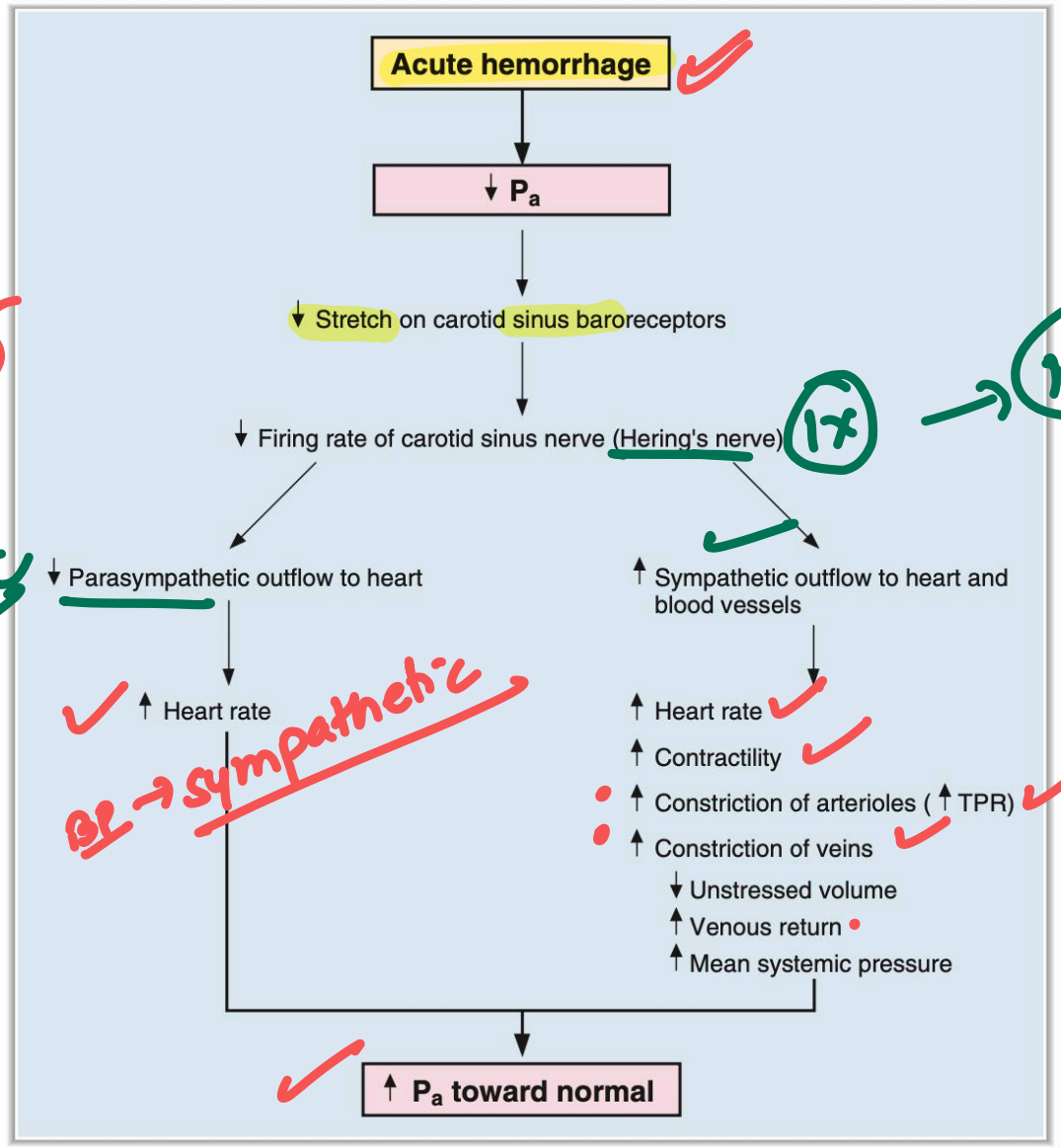
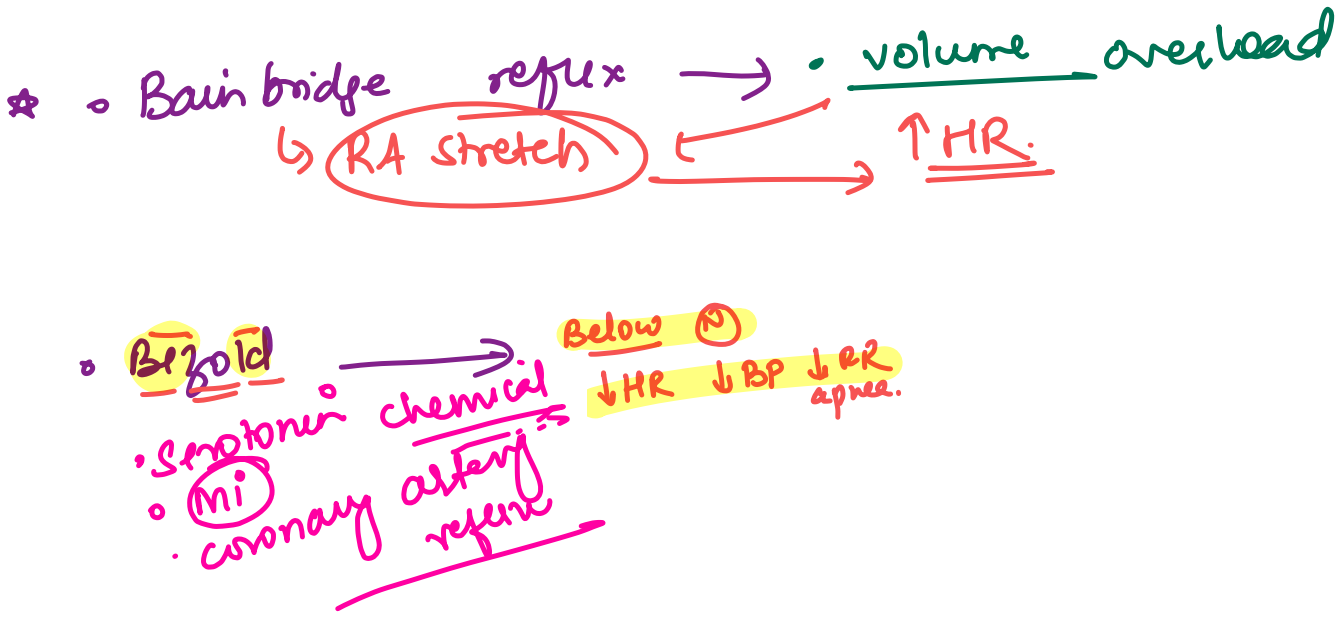
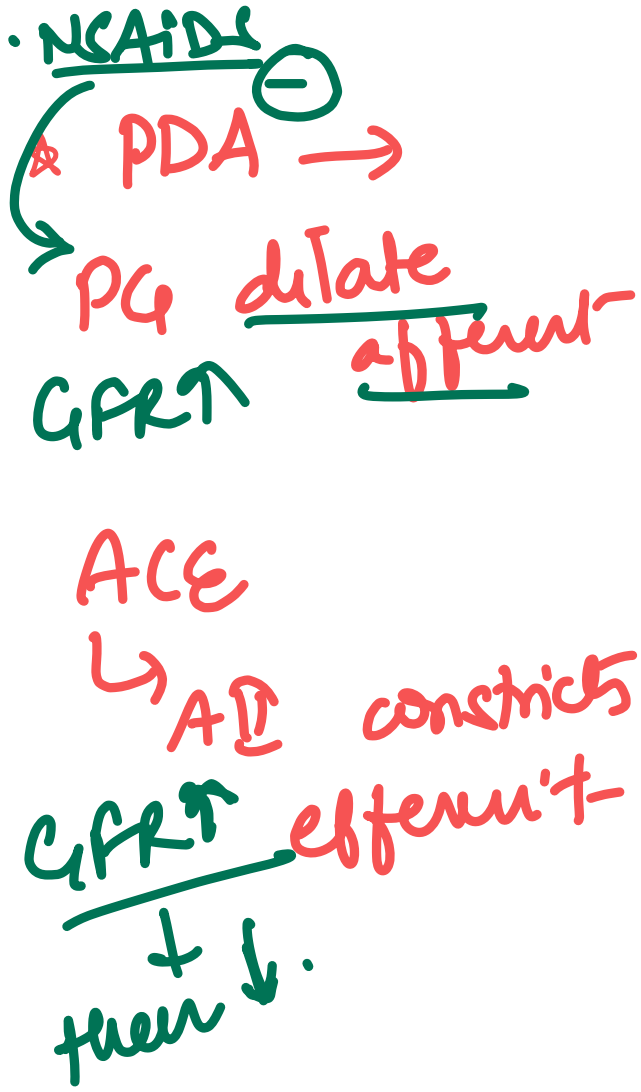


FIGURE 3.16 Role of the baroreceptor reflex in the cardiovascular response to hemorrhage. P_a = mean arterial pressure; TPR = total peripheral resistance.

Hormone	Stimulus for Secretion	Time Course	Mechanism of Action	Actions on the Kidneys
PTH	↓ plasma [Ca^{2+}]	Fast	Basolateral receptor Adenylate cyclase cAMP→urine	↓ Phosphate reabsorption (proximal tubule) ↑ Ca^{2+} reabsorption (distal tubule) Stimulates 1α-hydroxylase (proximal tubule)
ADH	↑ plasma osmolarity ↓ blood volume	Fast	Basolateral V_2 receptor Adenylate cyclase cAMP (Note: V_1 receptors are on blood vessels; mechanism is Ca^{2+}–IP_3)	↑ H_2O permeability (late distal tubule and collecting duct principal cells)
Aldosterone	↓ blood volume (via renin–angiotensin II) ↑ plasma [K^+]	Slow	New protein synthesis	↑ Na^+ reabsorption (ENaC, distal tubule principal cells) ↑ K^+ secretion (distal tubule principal cells) ↑ H^+ secretion (distal tubule α-intercalated cells)
ANP	↑ atrial pressure	Fast	Guanylate cyclase cGMP	↑ GFR ↓ Na^+ reabsorption
Angiotensin II	↓ blood volume (via renin)	Fast		↑ Na^+–H^+ exchange and HCO_3^- reabsorption (proximal tubule)

Kidney hormones -


Bain bridge
reflex
volume overload
RA stretch
↑HR.
Bezold
serotonin
chemical
Below
↓HR ↓BP ↓RR
apnea.
MI
coronary artery
reperf


NSAIDs
PDA
PG dilate
efferent
GFR↑
ACE
AII constricts
efferent
GFR↑
then ↓.

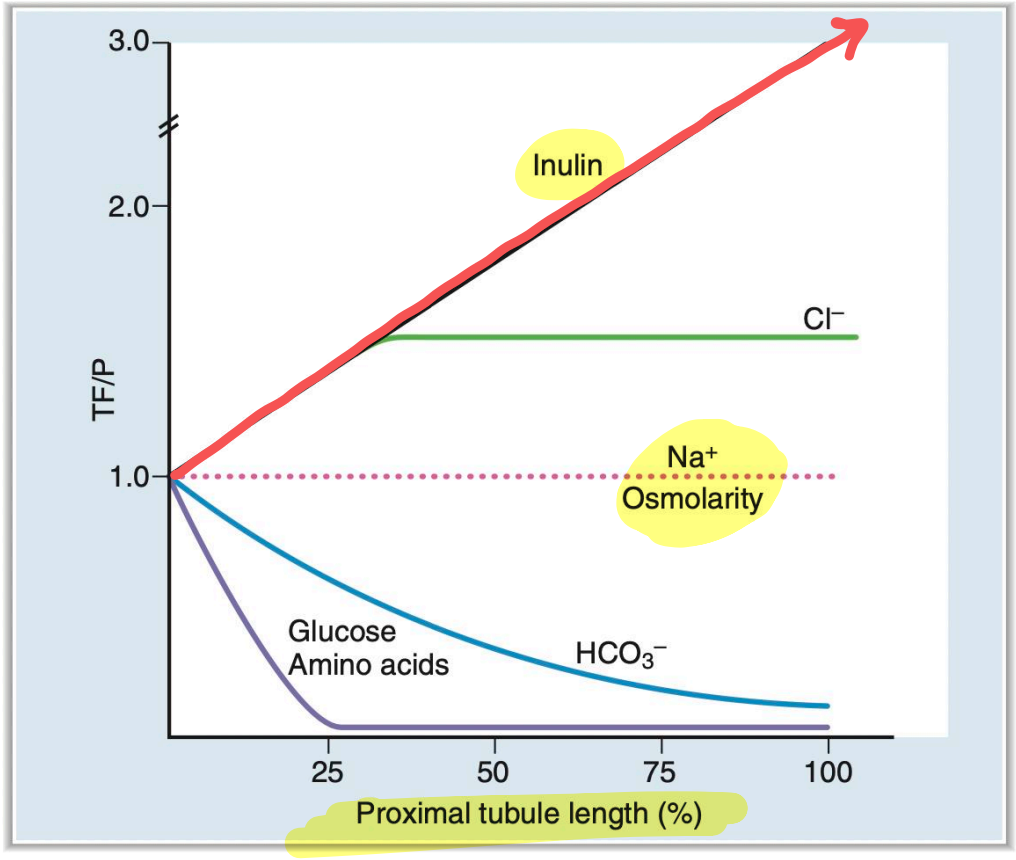


FIGURE 5.10 Changes in TF/P concentration ratios for various solutes along the proximal tubule.

- PCT →
- isoosmolar
- ADH independ.

TF/P ↓ → reabs

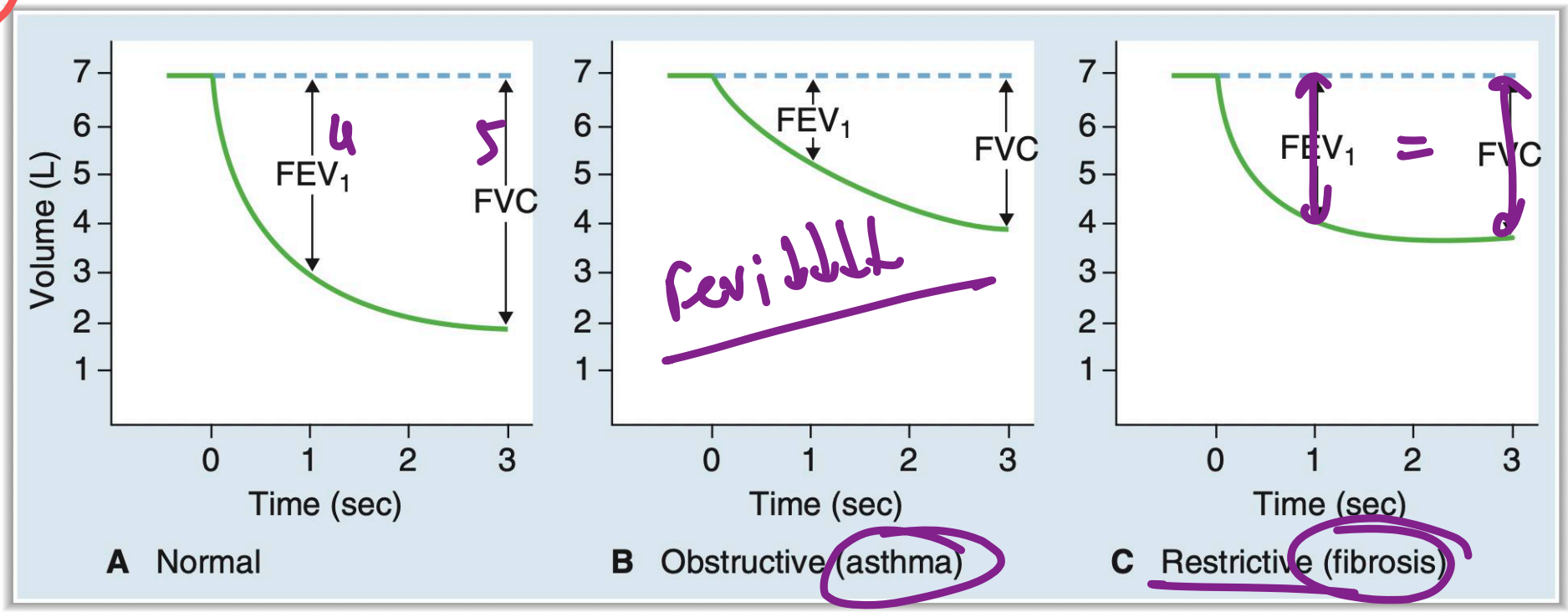


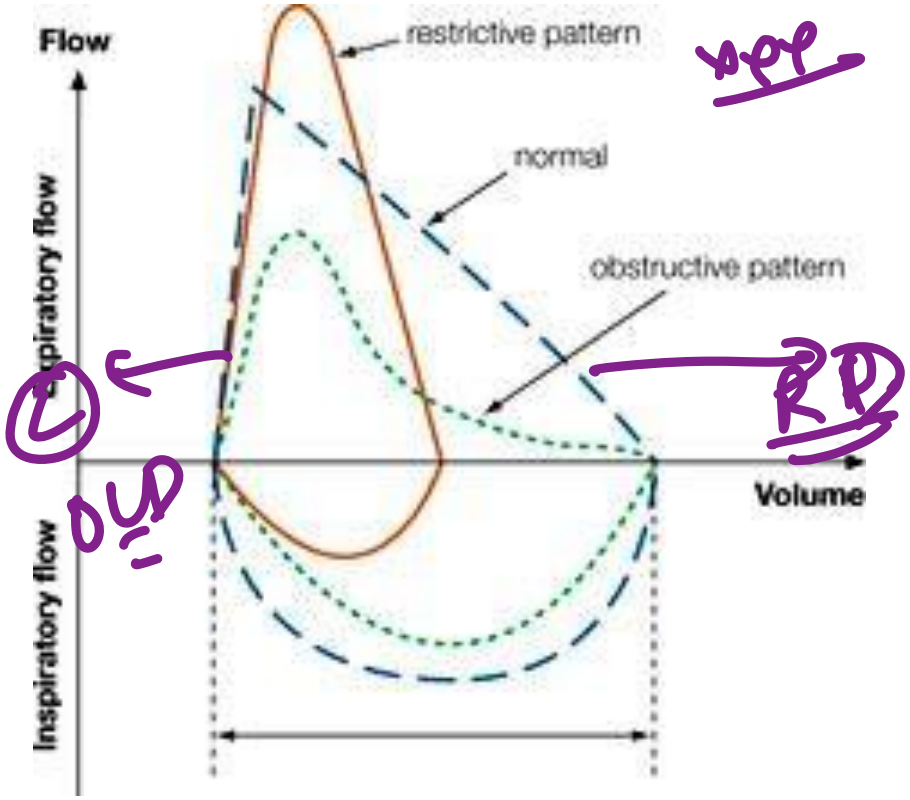
FIGURE 4.2 Forced vital capacity (FVC) and FEV_1 in normal subjects and in patients with lung disease. FEV_1 = volume expired in first second of forced maximal expiration.

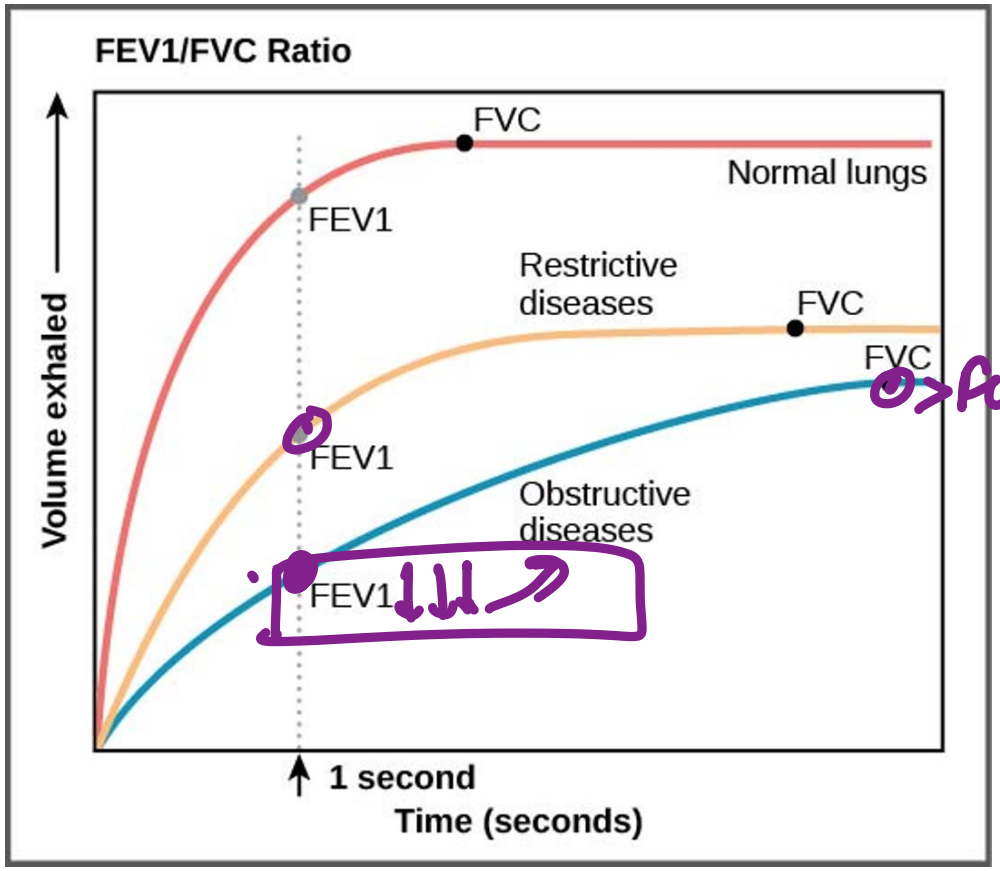
OLD

- FEV1 ↓↓↓↓↓.
- RV ↑
  - TLC ↑

RLD

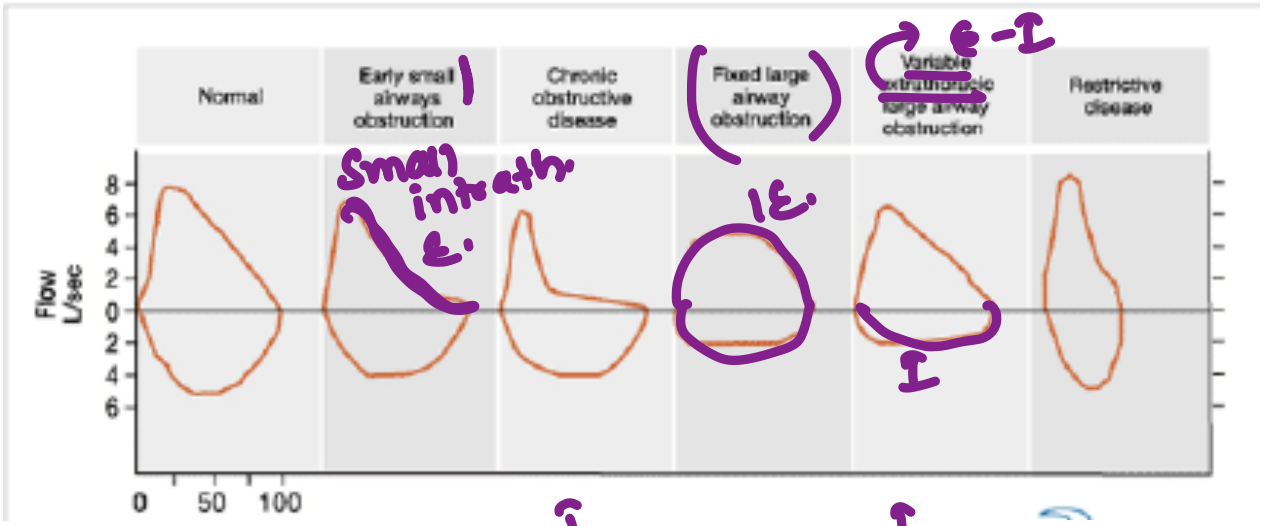
- all volumes ↓.
  - FEV1 ↓
  - FVC ↓.


Flow
restrictive pattern
normal
obstructive pattern
Volume
Inspiratory flow
Inspiratory flow


FEV1/FVC Ratio
FVC
Normal lungs
FEV1
Restrictive diseases
FVC
FVC
FEV1
Obstructive diseases
FEV1
Volume exhaled
1 second
Time (seconds)

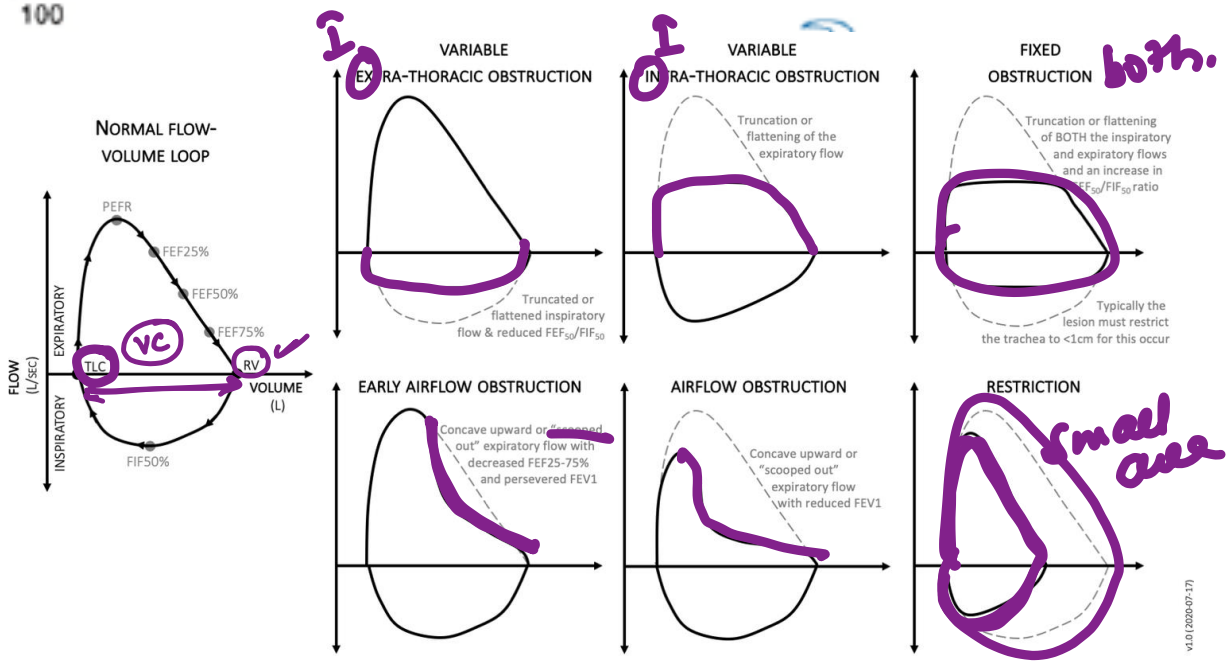
- OLD where DLco ↓ → emphysema (↓ surface area ↓ diffusion)

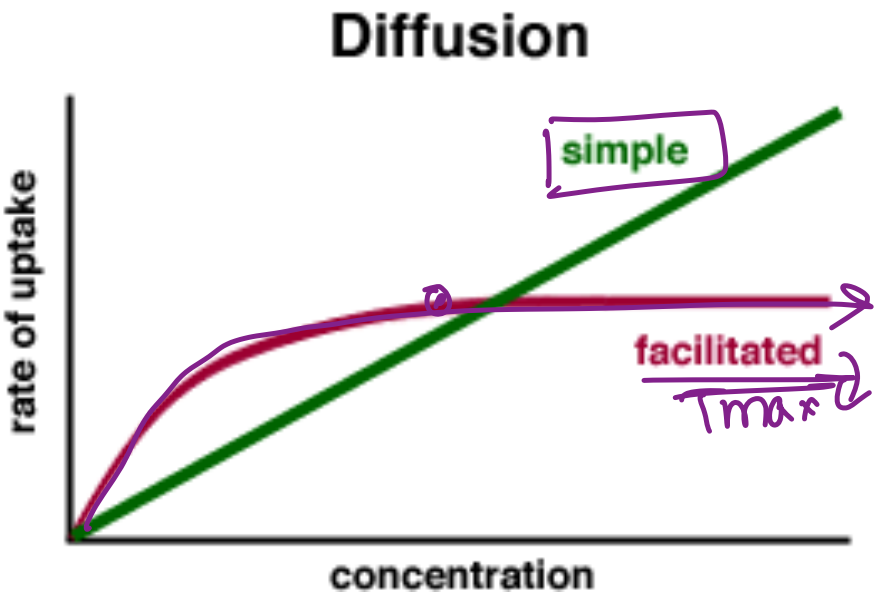
DLco ↓ → fibrosis (Rest)

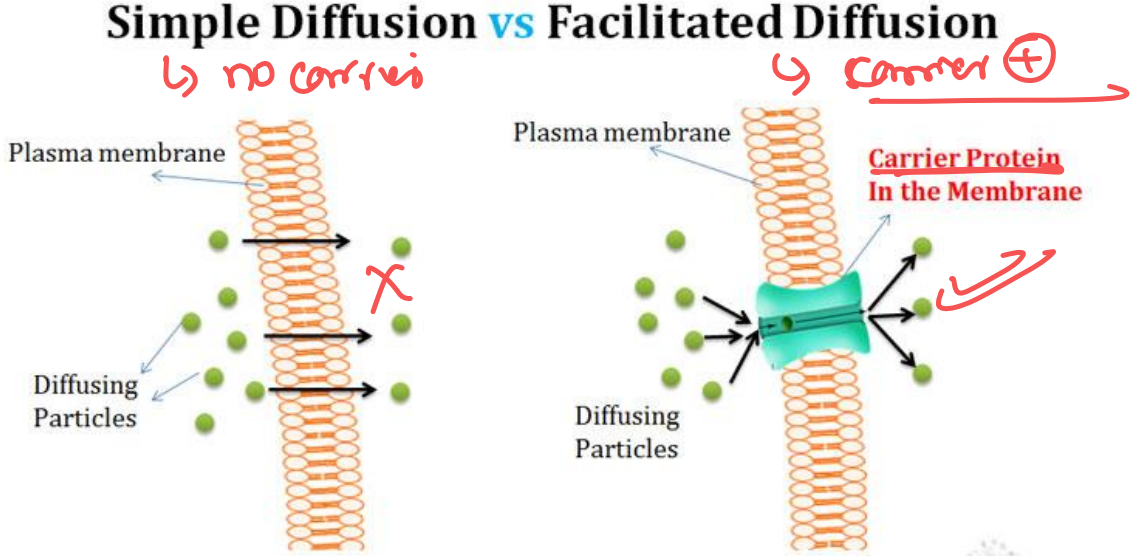


intrath.
i → Exp

E → ⊘ I exp
extrathor.

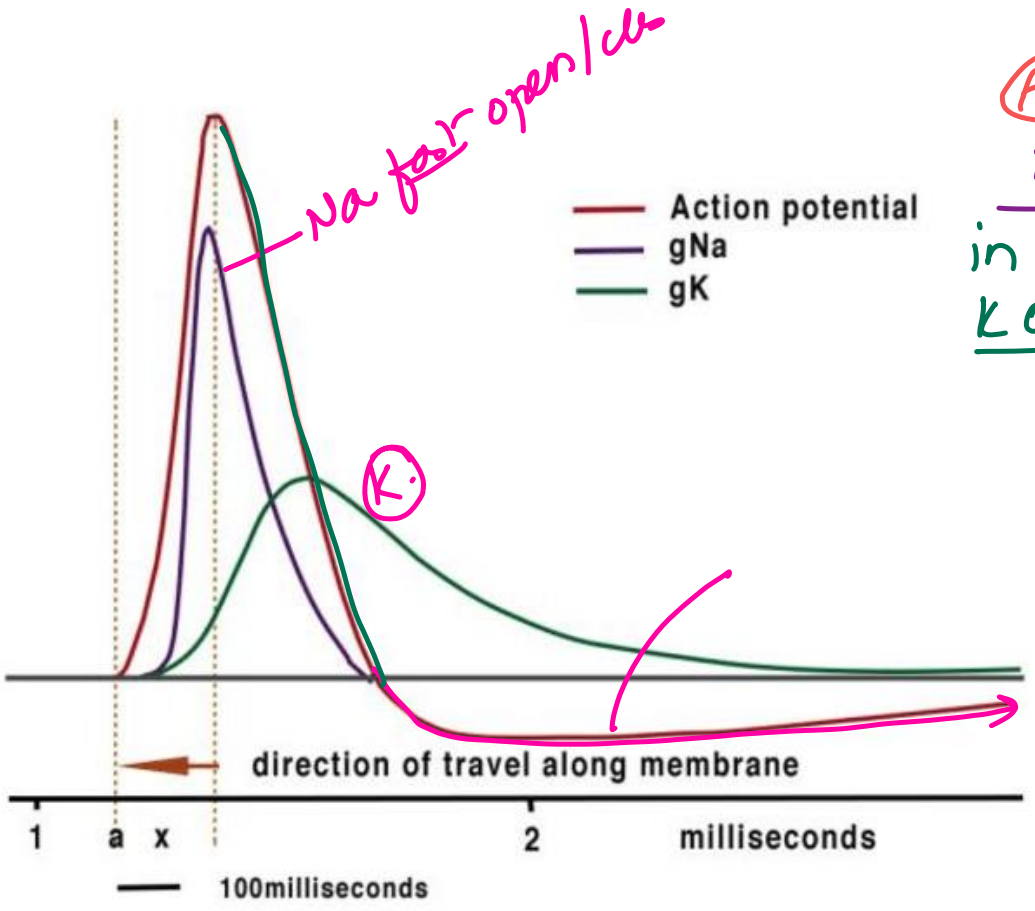






- GLUT → facilitated diff carrier → active ATP
- SGLT → sec. active transport – cotransp

GLUT 1	Basal glucose uptake	Placenta, blood-brain barrier, brain, red cells, kidneys, colon, many other organs
GLUT 2	β-cell glucose sensor, transport out of intestinal and renal epithelial cells	β cells of islets, liver, epithelial cells of small intestine, kidneys
GLUT 3	Basal glucose uptake	Brain, placenta, kidneys, many other organs
GLUT 4	Insulin-stimulated glucose uptake	Skeletal and cardiac muscle, adipose tissue, other tissues
GLUT 5	Fructose transport	Jejunum, sperm
GLUT 6	Function not known	Brain, spleen, leucocytes
GLUT 7	Glucose-6-phosphate transporter in the endoplasmic reticulum	Liver



AP

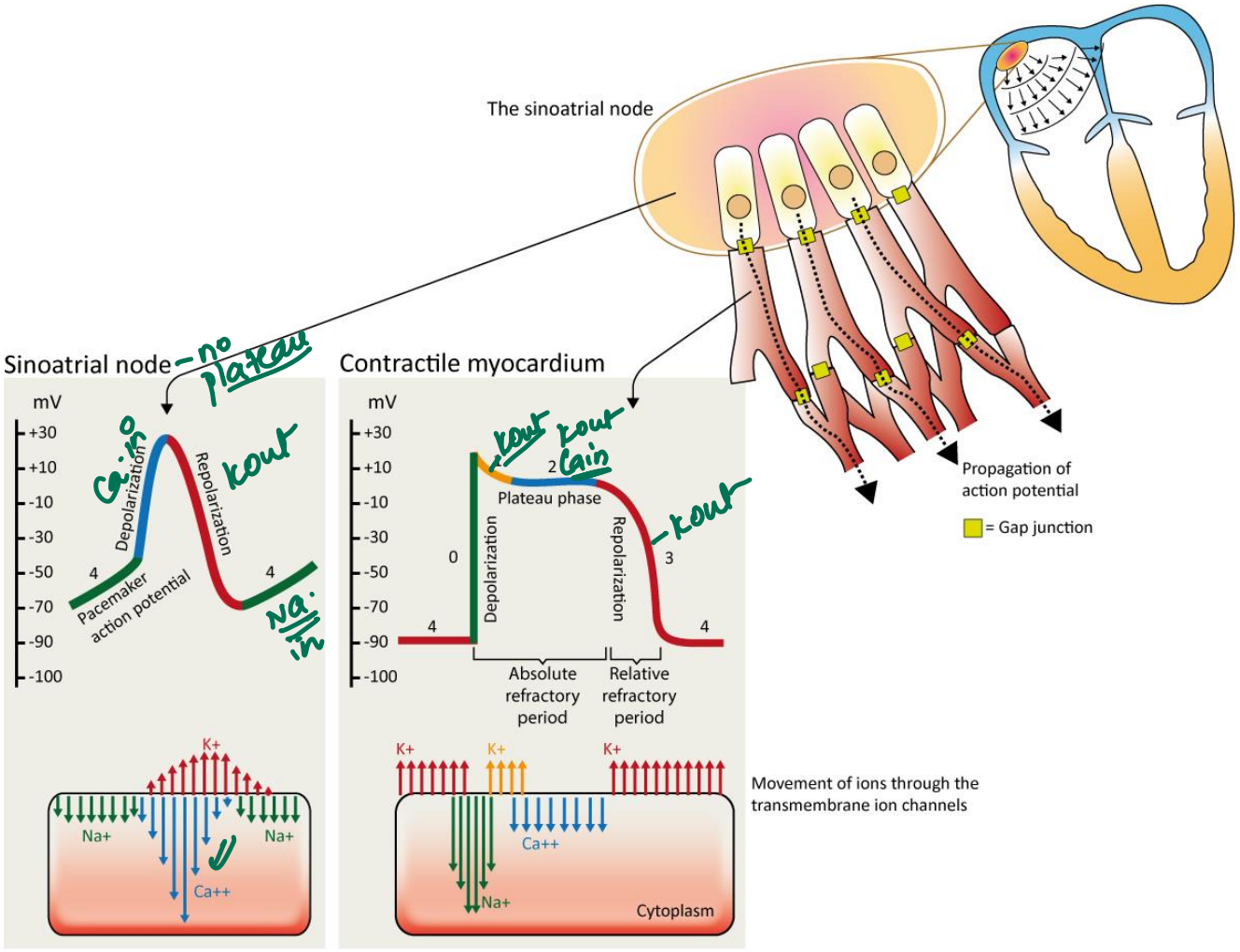
↑ Na K ↓

↑ Na

K out ↓ eff

Na K ATPase

- Nokia
  3 Na^+ out
  2 K^+ in
  ] against conc
  active



AP → Na/Ca in
Poul

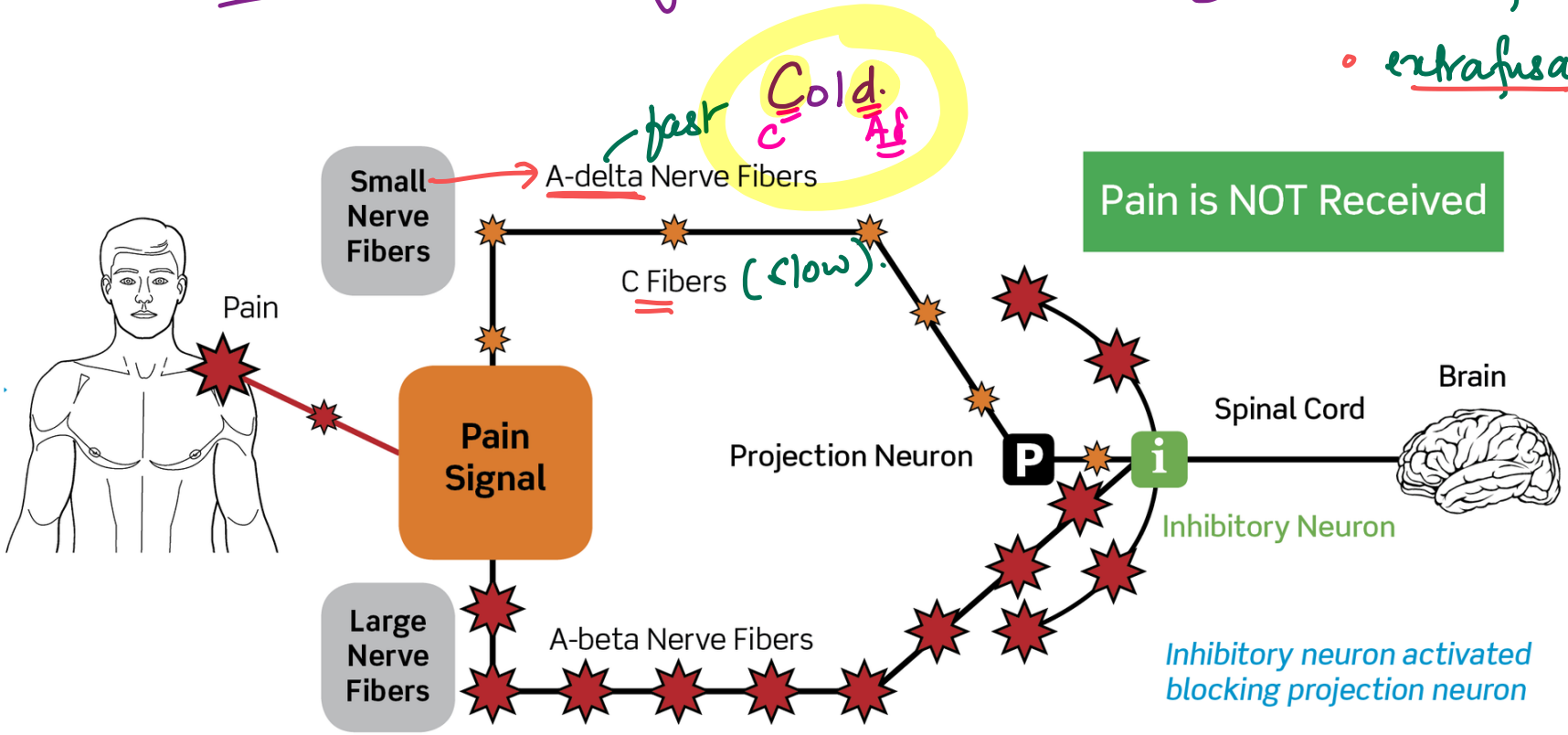
iva → funny current

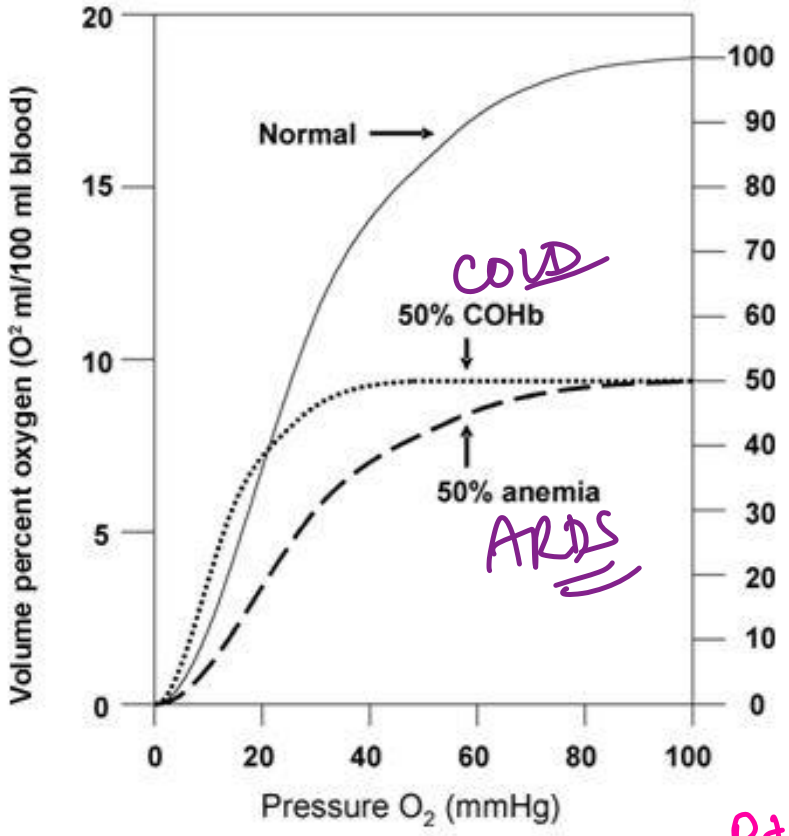
Nc.

✱ TENS.

gate control theory

gamma → muscle spindle
- intrafusal → Aγ.
- extrafusal → Aα



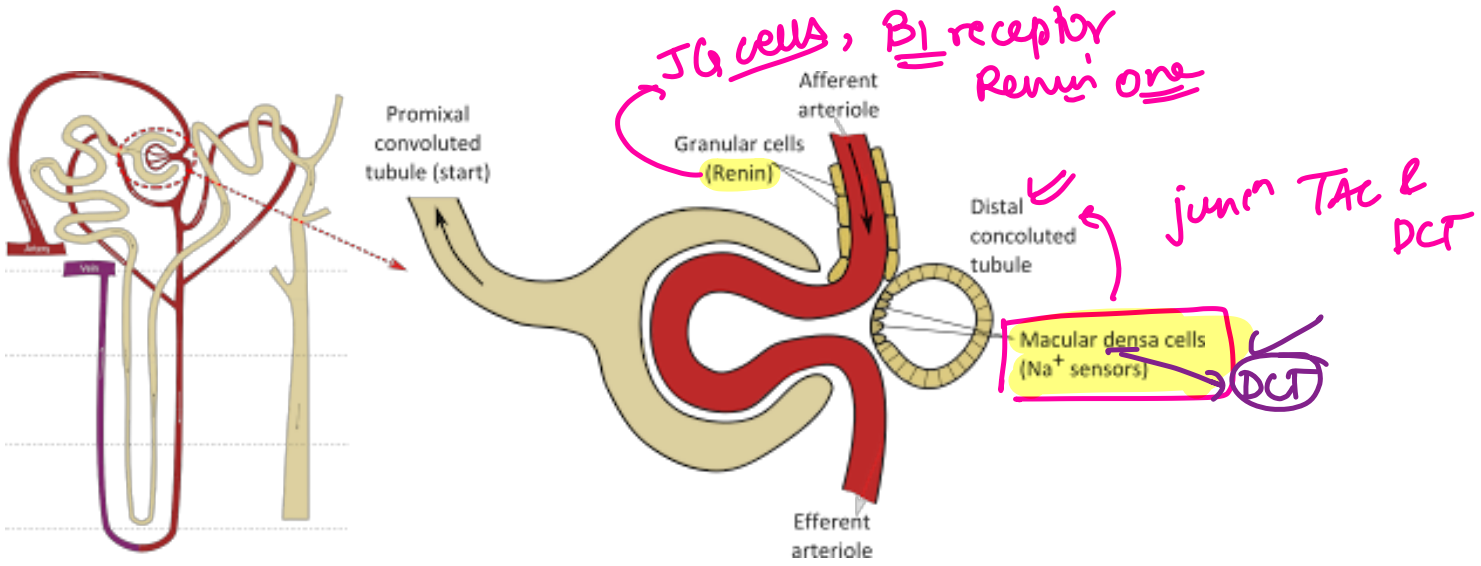


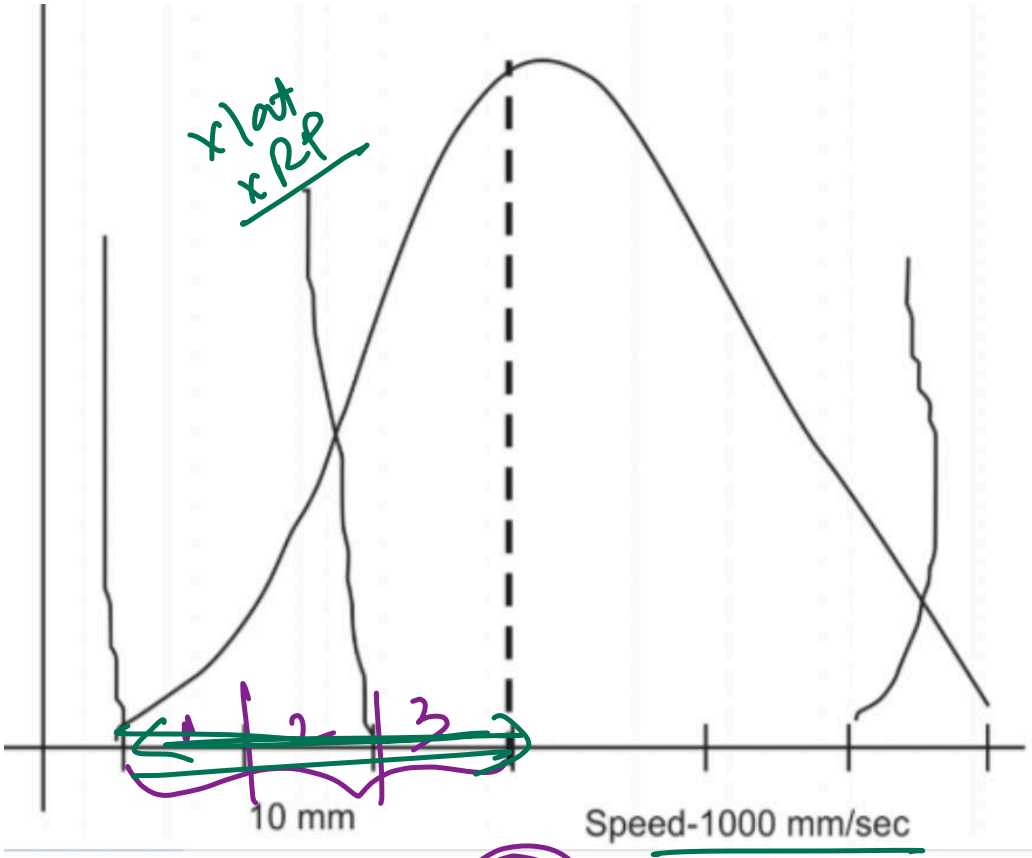
COLD
= left down

ARDS.
= anemia rt down

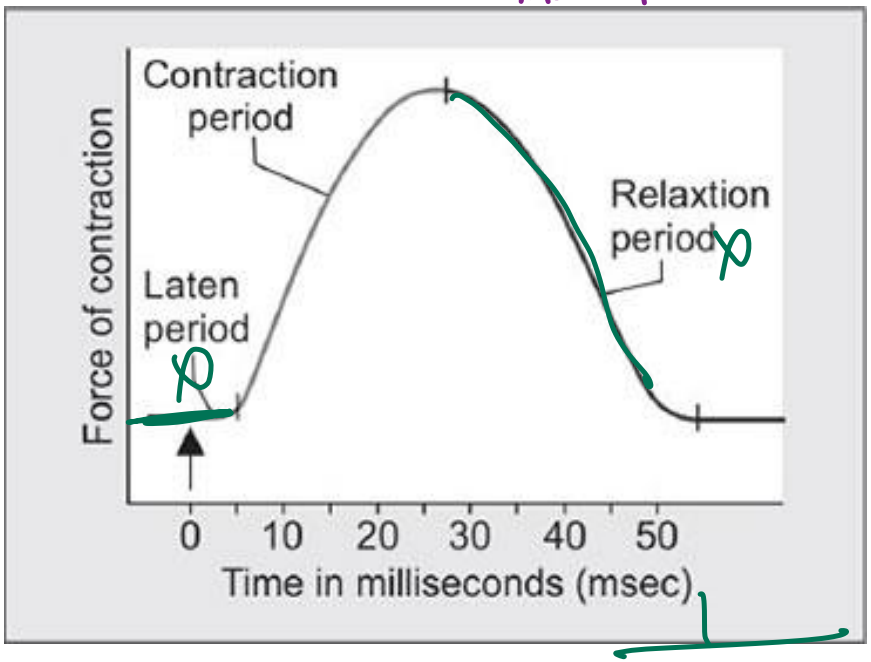
HbF → friend → lower O_2
left shift

Rt shift → release O_2
→ raised values except pH ↓: acidosis dextro
alkalosis left


JG cells, B1 receptor
Renin one
Promixal convoluted tubule (start)
Afferent arteriole
Granular cells (Renin)
Distal concoluted tubule
Juxta TAL & DCT
Macular densa cells (Na+ sensors)
DCT
Efferent arteriole



IF = $\frac{1}{\text{time}}$ = $\frac{1}{\text{contractn pd.}}$ × latent



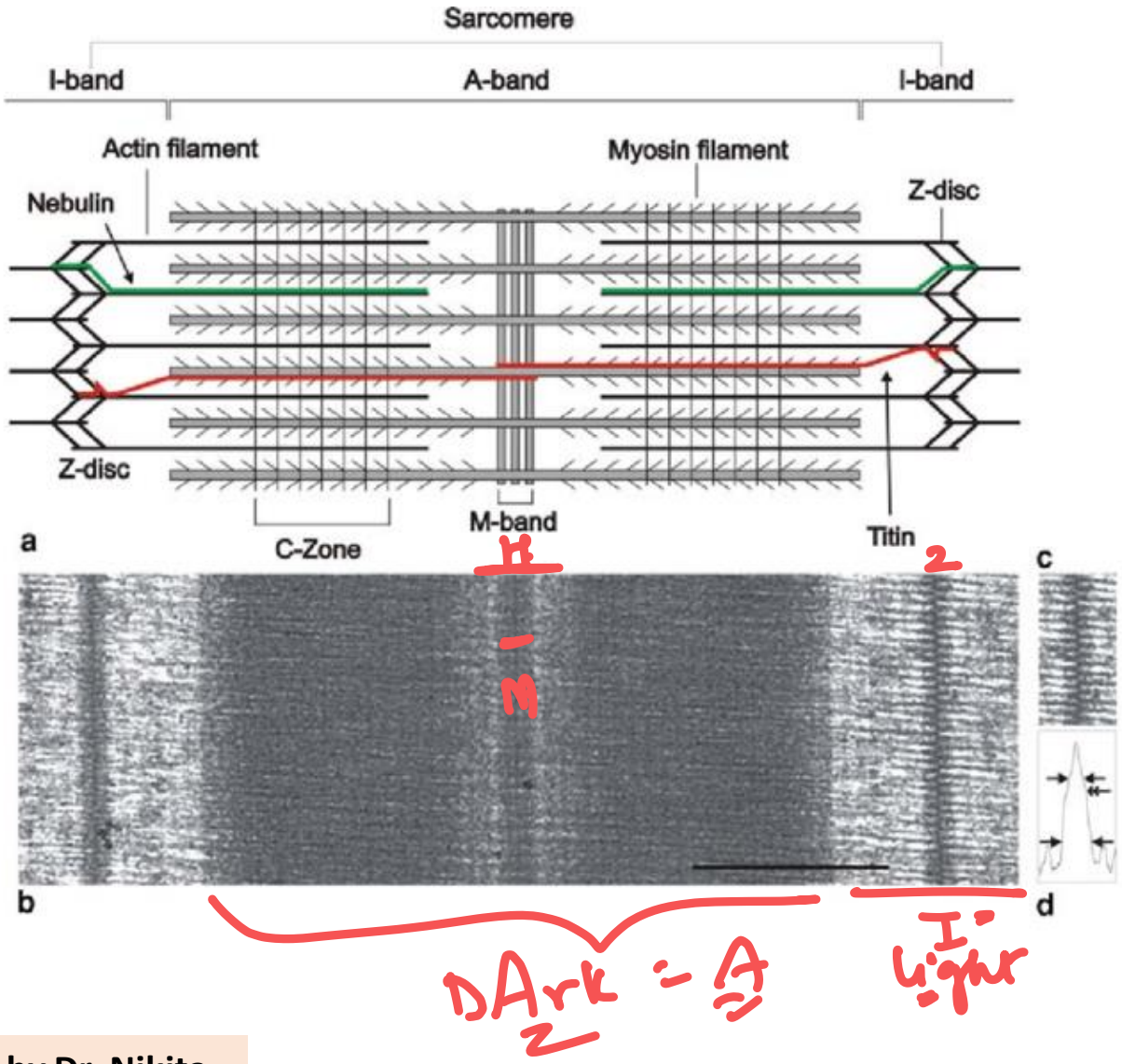
CP 30mm

1000 mm — 1 sec

30 mm — ?

$\frac{30}{1000}$

$\frac{1000}{30}$

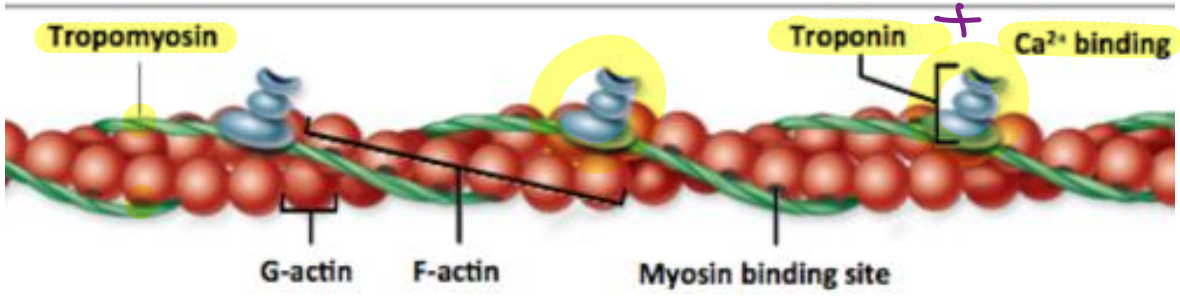


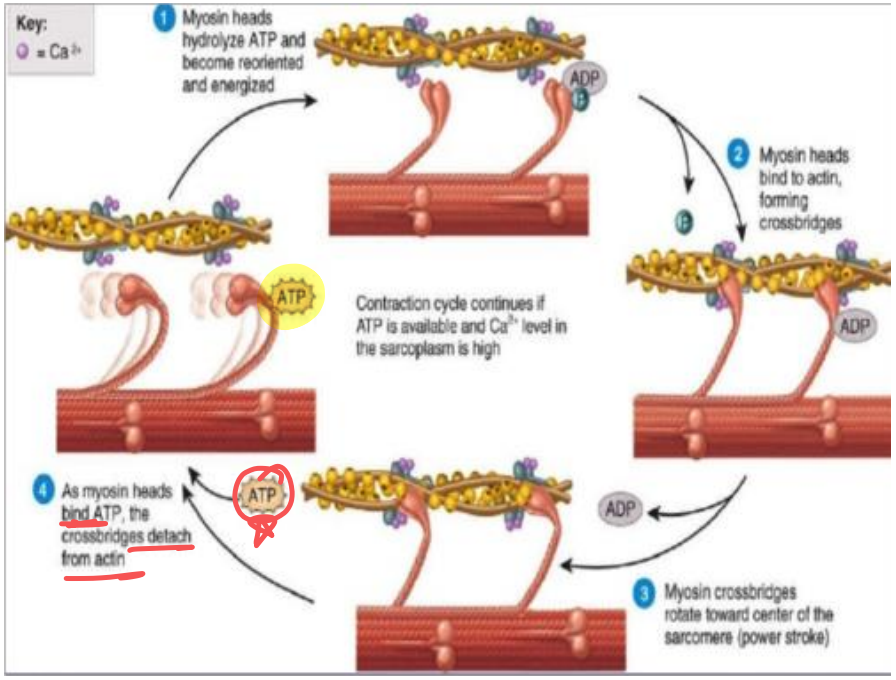
MHAZI

Thick → Mola = myosin

* Binds to myosin binding site → Tropomyosin

H
M
Z
Dark = A
Light = I





Ca → back into SR
↳ SERCA
↓
2° active

• ATP req for relax°

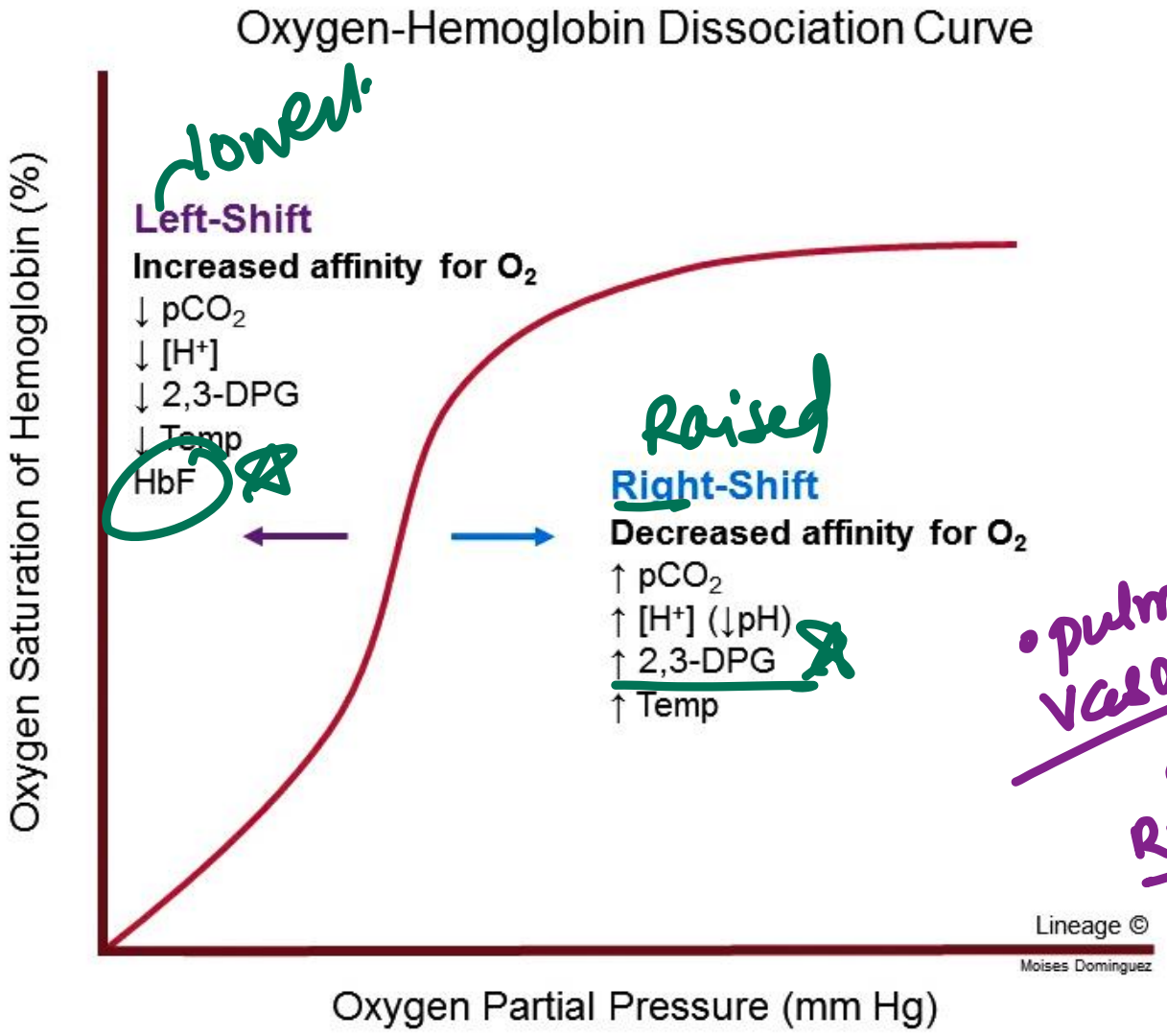
∴ PM → No ATP
↳ rigor mortis

* skeletal muscle → no Ca channel
∴ indep of extracell. Ca.

vs heart → ✓

Increase in all of the following shift O2-Hb dissociation curve to right except

a) pCo2
b) 2,3 BPG
c) pH↓ (acidosis → dextro)
d) Tenperature



lower

Raised

High altitude

– Hypoxia → Rt shift

• pulm vasoconstrict° → RVH

• Resp (alkalosis) → hypocalcemia → perioral tingle

Q9

Which of the following transection results in apneusis

a) Above the pons with vagus cut
b) Below medulla with vagi intact
c) Midpontine with vagus intact
d) Midpontine with vagus cut

apneustic child

⊖ PTC + vagi ✓

Label	Level of transection	Vagi intact	Vagi cut
A	Complete transection above pons	Regular breathing continues	Regular breathing continues but depth of inspiration increases
B	Mid-pontine level section	Regular breathing continues	Apneusis develops
C	Transection midway between pons and medulla	Irregular but rhythmic respiration	Irregular but rhythmic respiration
D	Complete transection below medulla	Spontaneous respiration ceases	Spontaneous respiration ceases

Depth of inspiration controlled by:

a. Pneumotaxic center
b. Posterior medulla
c. Apneustic center
d. Pons

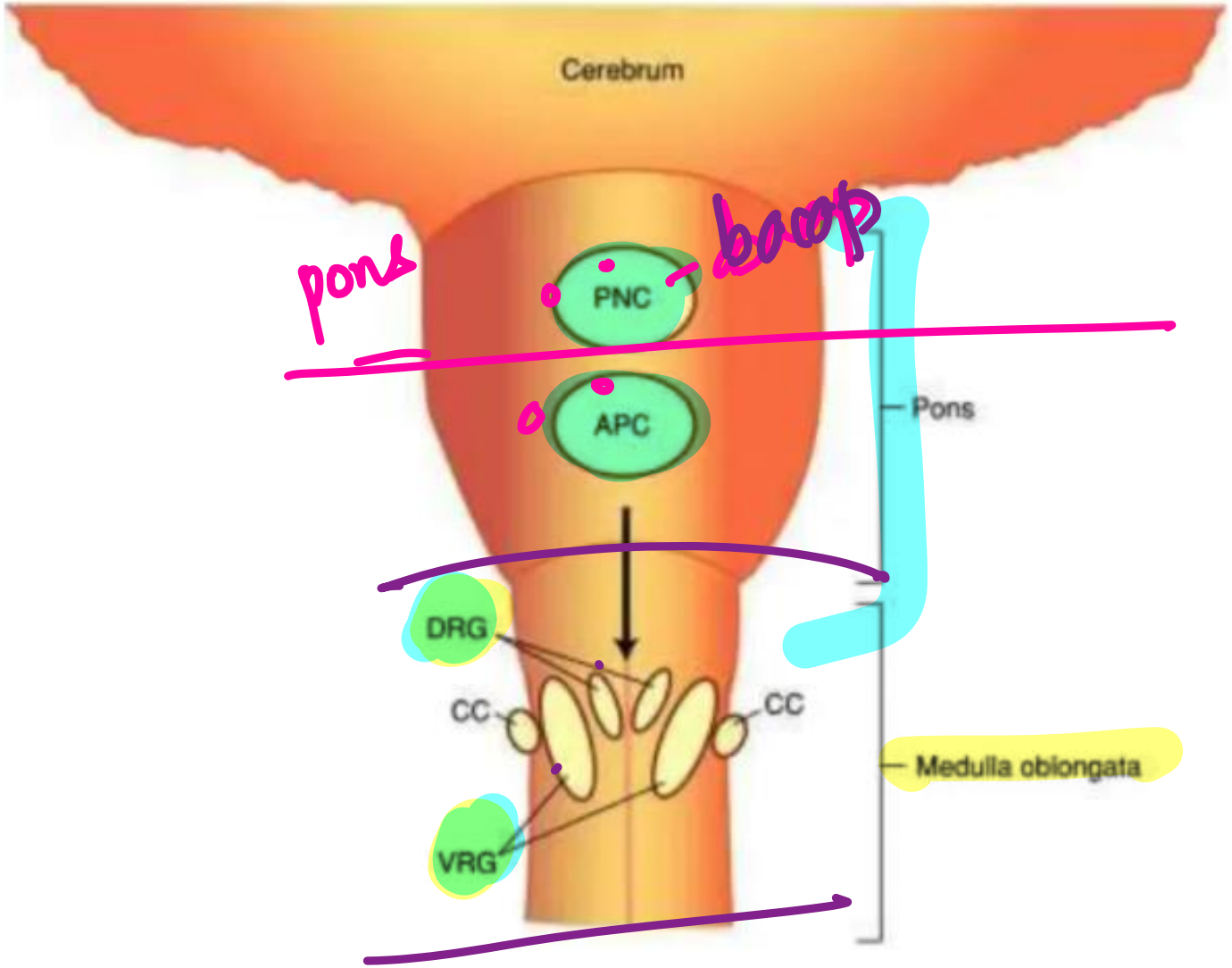
PRAD

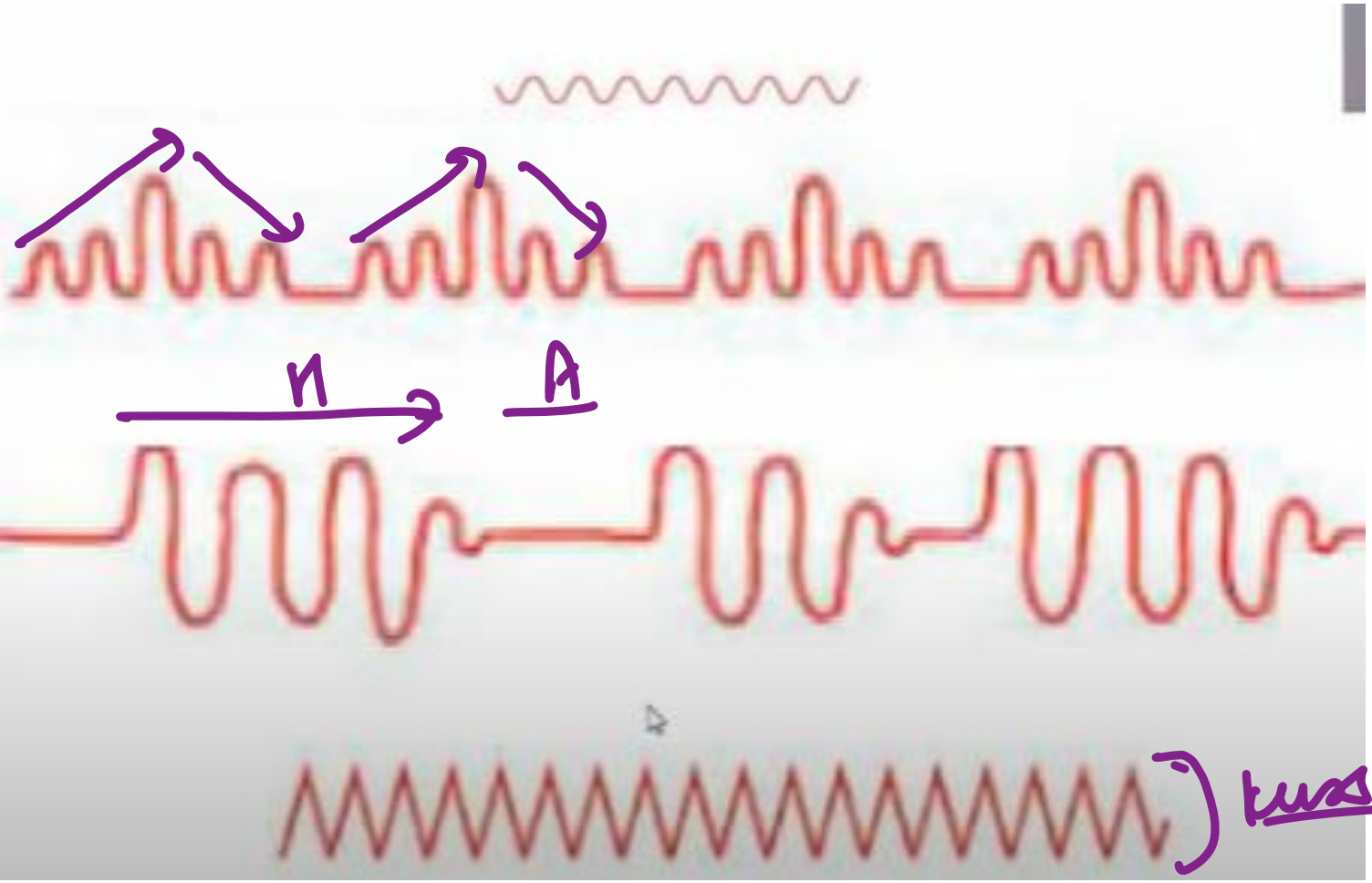
- pneumotaxic → rate
- apneustic → depth

Pacemaker regulating the rate of respiration:

a. Pneumotaxic center
b. Dorsal group of nucleus
c. Apneustic center
d. Pre-Bötzinger

Respiratory centre component	Primary function
Pre-Bötzinger complex	Pacemaker cells of spontaneous or automatic breathing
DRG	Respiratory rhythm generator (inspiratory ramp signal)
VRG	Overdrive mechanism
Pneumotaxic centre	Controls the "switch off" point of inspiratory ramp signal
Apneustic centre	Delays switch off signal-promotes inspiration
Vagi	Inhibits inspiratory discharge


Cerebrum
pons
baap
PNC
APC
Pons
DRG
CC
CC
Medulla oblongata
VRG



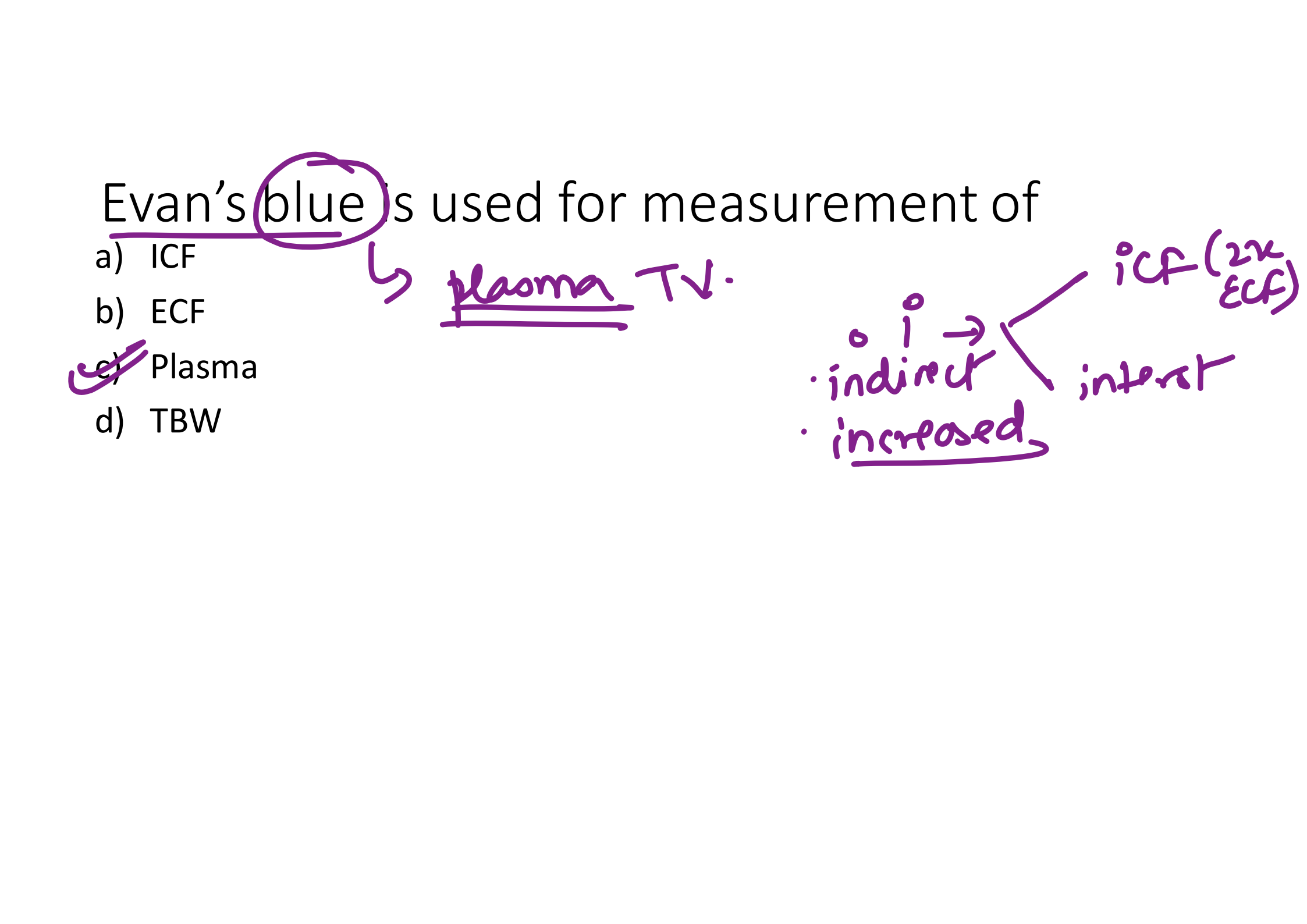
C – D
Cheynestokes

Biphasic
Bjots

kussmaul
DKA

Evan's blue is used for measurement of

a) ICF
b) ECF
c) Plasma
d) TBW



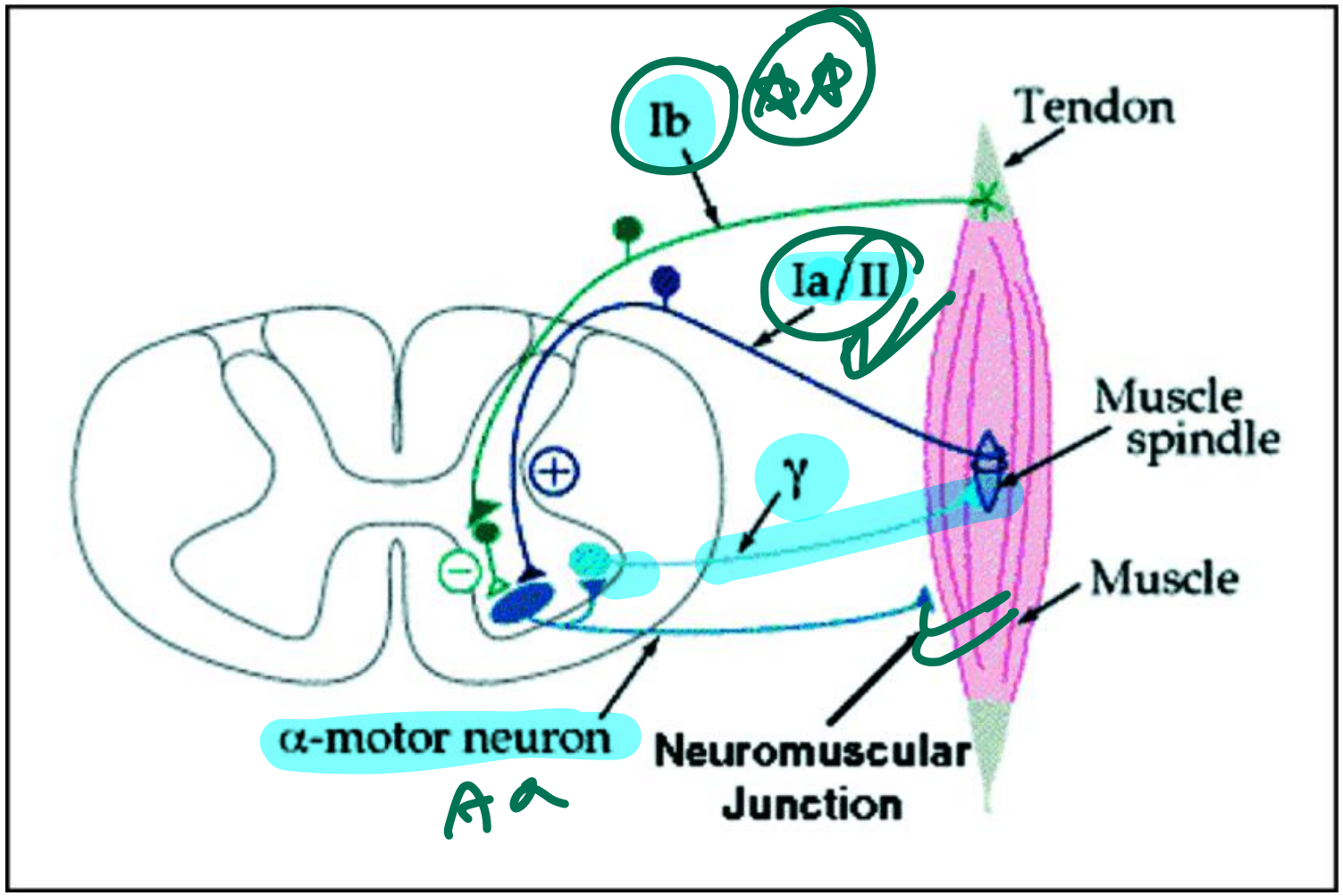
Lung compliance is increased in

a) Pulmonary edema
b) Pulmonary fibrosis
c) Emphysema
d) Interstitial lung disease

$$\frac{\Delta V}{\Delta P}$$

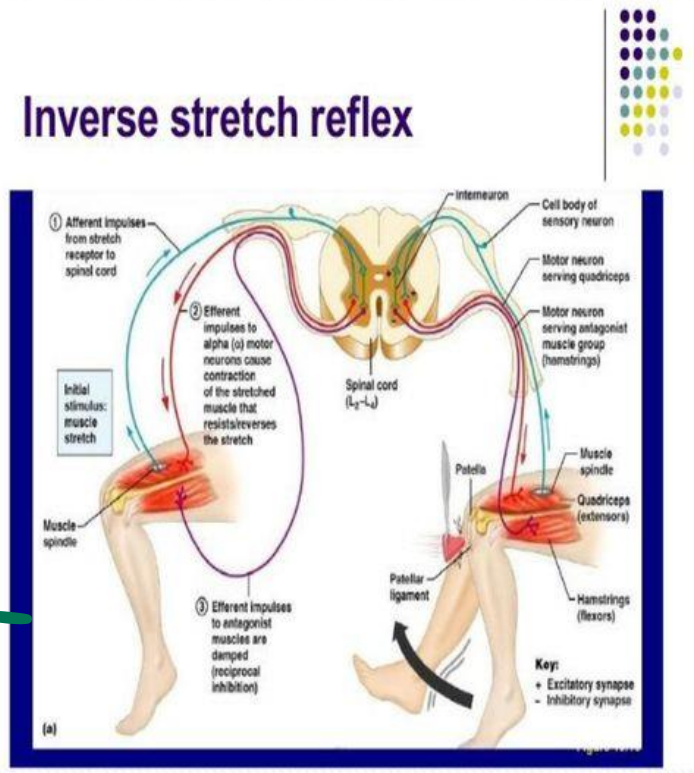
True about Golgi tendon reflex

a) Bisynaptic
b) Afferent is type II fiber
c) Response is muscle contraction
d) Activated by decrease in muscle tension


Ib
AA
Tendon
Ia / II
Muscle
spindle
γ
Muscle
α-motor neuron
Aα
Neuromuscular
Junction

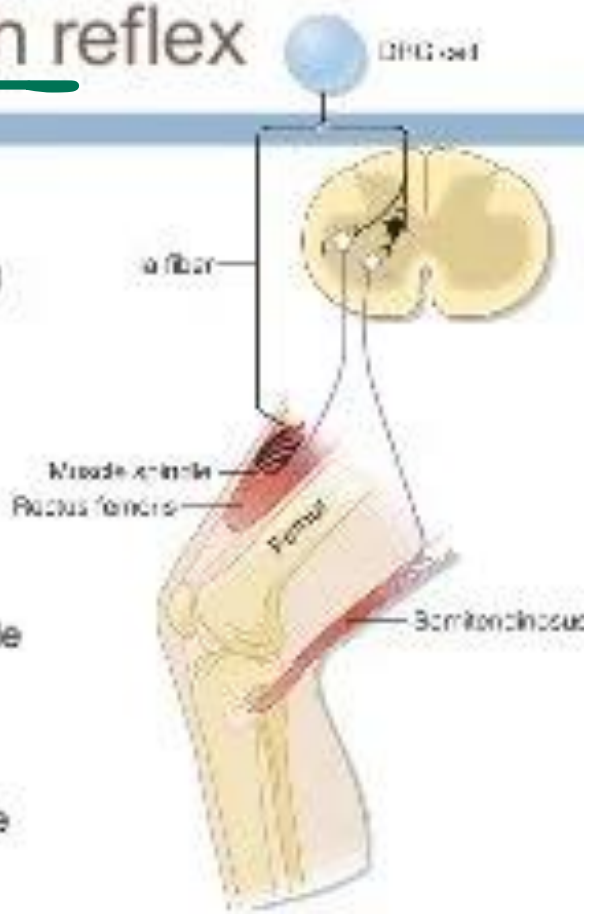
Golgi Tendon Reflex (Inverse Stretch Reflex)-2

- It is called inverse stretch reflex because it is the inverse of stretch reflex
- It is initiated by an increase in muscle tension
- This activates Group Ib nerve fibers in Golgi tendon organ
- The sensory input activates an inhibitory interneuron in the spinal cord
- This interneuron inhibits the activity of motor neuron innervating the same muscle causing muscle relaxation



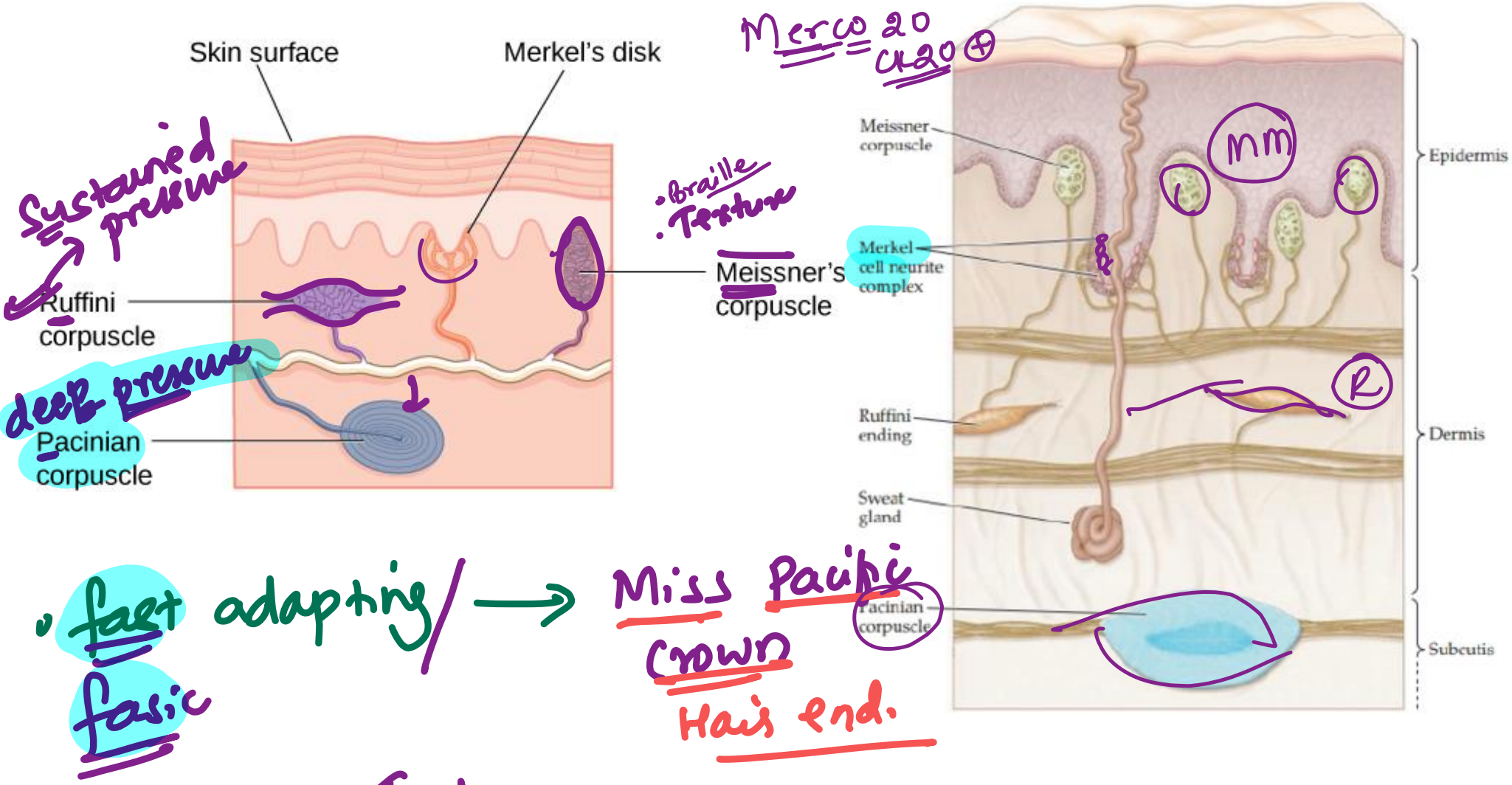
Myotatic or stretch reflex

- Monosynaptic reflex mediated by muscle spindles
  - Contraction in response to lengthening
- Reflex arc:
  1. Muscle stretches
  2. Ia afferent of muscle spindle increase firing
  3. Synapse on α motor neuron and inhibitory interneuron in spinal cord
  4. α motor neuron of homonymous muscle excited, and of antagonist muscle inhibited
  5. Homonymous muscle contracts to oppose lengthening, antagonist muscle relaxes



B&L Figure 9-6

Contract



Sustained pressure

deep pressure

• Braille
• Texture

Merco 20 CH20 ⊕

mm

R

• fast adapting → Miss Pacini

fasic

Crown

Hair end.

- slow - Tonic

	Extracellular fluid	Intracellular fluid
Na^+	**142 mEq/L**	10 mEq/L
K^+	4 mEq/L	**140 mEq/L**
Ca^{++}	**2.4 mEq/L**	0.0001 mEq/L
Cl^-	**103 mEq/L**	4 mEq/L
HCO_3^-	**28 mEq/L**	10 mEq/L
PO_4^{3-}	4 mEq/L	**75 mEq/L**
Glucose	**90 mg/dL**	0-20 mg/dL
Proteins	2 g/dl (5mEq/L)	**16 g/dL (40 mEq/L)**
pH	7.4	7.0
Osmolarity	282 mOsm/L	281 mOsm/L
Major Cation	Na^+	K^+
Major Anion	Cl^-	Phosphate
Buffer	$HCO3^-$	Protein
Most osmotically active ion	Na^+	K^+