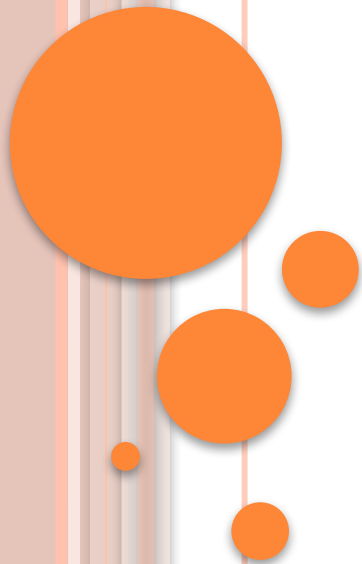


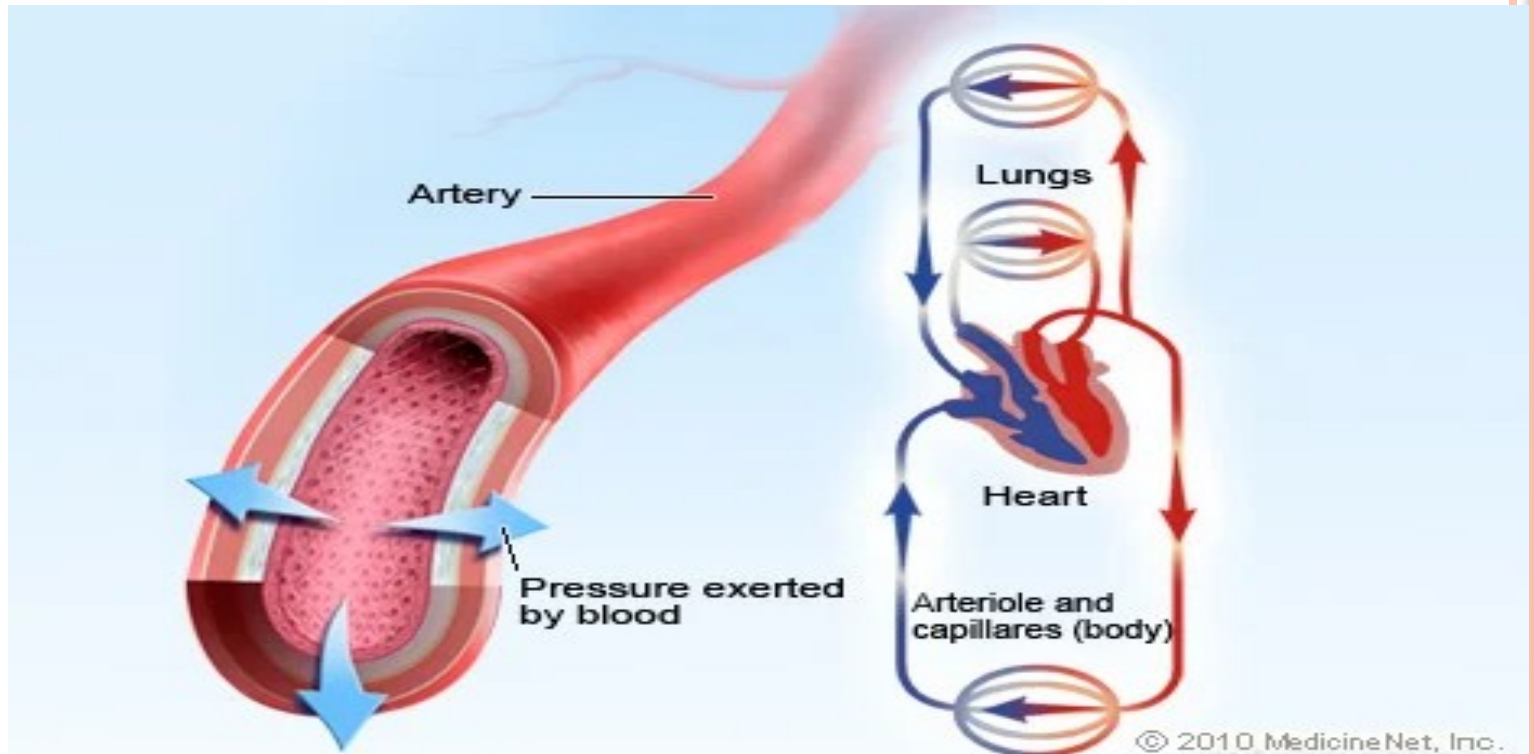


BLOOD PRESSURE



BLOOD PRESSURE -DEFINITION

- *The lateral pressure exerted by the column of blood on the wall of arteries*



SYSTOLIC BLOOD PRESSURE

- **Maximum** pressure recorded during cardiac cycle
- Normal systolic BP :- **100-119 mm Hg**
- Depends mainly on the **cardiac output**



DIASTOLIC BLOOD PRESSURE

- **Minimum** pressure recorded during cardiac cycle
- Normal value :- **60-80 mm Hg**
- Depends primarily on **peripheral resistance**



PULSE PRESSURE

- **Difference** between systolic & diastolic blood pressure
- **PP = SBP – DBP**
- Normal value:- **20-50mm Hg**
- Maintains the **normal pulsatile nature** of blood flow required for **tissue perfusion**



MEAN ARTERIAL PRESSURE

- **Average** pressure recorded during cardiac cycle
- **MAP = DP + 1/3 PP**
- Normal value :- **75 – 105 mm Hg**
- MAP helps in **forward movement of blood** in the lumen of blood vessels → maintains **tissue perfusion**



BLOOD PRESSURE CLASSIFICATION	SBP MMHG	DBP MMHG
NORMAL	<120	and <80
PREHYPERTENSION	120–139	or 80–89
STAGE 1 HYPERTENSION	140–159	or 90–99
STAGE 2 HYPERTENSION	≥160	or ≥100



FACTORS AFFECTING BLOOD PRESSURE

- **Blood Pressure = Cardiac output × Peripheral resistance**
- Alteration in **cardiac output** mainly affects **systolic pressure**
- Alteration in **peripheral resistance** mainly affects **diastolic pressure**



FACTORS AFFECTING CARDIAC OUTPUT

- **Cardiac Output = Stroke Volume × Heart Rate**
- **Stroke volume** is affected by preload, afterload & myocardial contractility
- **Heart rate** is mainly affected by autonomic influences



FACTORS AFFECTING PERIPHERAL RESISTANCE

◦ 1. Diameter of Blood Vessels

- ❖ Depends primarily on the vasomotor tone – **the sympathetic vasoconstrictor tone**
- ❖ Vasoconstriction – increases PR → Increases BP
- ❖ Vasodilation – decreases PR → Decreases BP
- When blood vessels become stiff → PR ↑ → BP ↑



FACTORS AFFECTING PERIPHERAL RESISTANCE

2. Viscosity

- Determines the resistance to flow
- Alteration of blood viscosity affects DBP by its effect on total PR
- Depends on:-
 - ❖ Composition of plasma
 - ❖ Total number of cells in blood
 - ❖ Resistance of cells to deformation
 - ❖ Temperature



○ Factors that increase viscosity:

- ❖ Polycythemia
- ❖ Hyperproteinemia
- ❖ Hereditary spherocytosis
- ❖ Decreased temperature

○ Factors that decrease viscosity:

- ❖ Anemia
- ❖ Hypoproteinemia
- ❖ Increased temperature



3. Velocity of blood

- Total PR is **inversely** related to velocity of blood flow



PHYSIOLOGICAL VARIATIONS

- **Age**

BP increases with age

- **Gender**

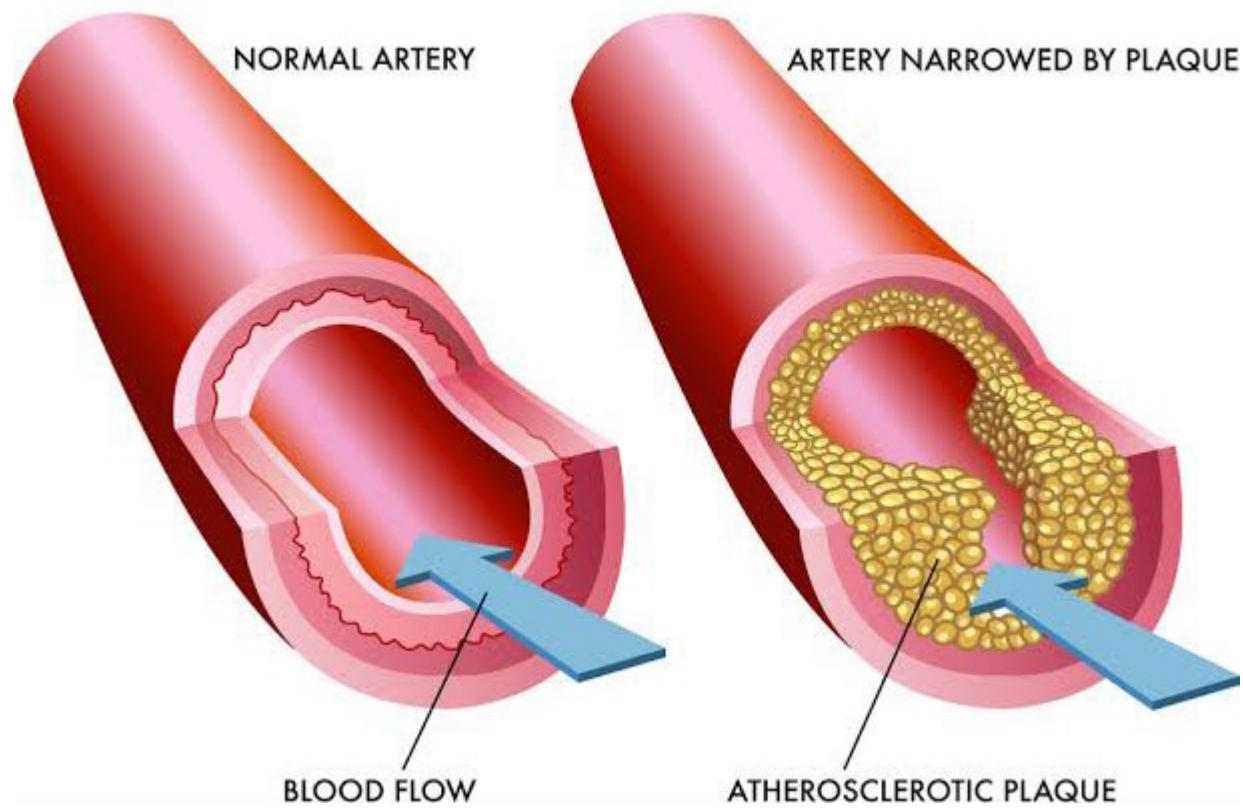
BP is comparatively **less in females** during their reproductive life – Why?

- ❖ **Due to the effect of:-**

- a) **Progesterone** :- relaxes BV smooth muscles

- b) **Estrogen** :- prevents atherosclerosis





- **Eating**

BP increases after a meal

- **Sleep**

BP less during sleep

- **Emotion & Excitement**

↑ed sympathetic discharge → ↑es BP

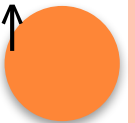


○ Exercise

- ❖ **Systolic BP** – **always rises** – due to increased CO
- ❖ **Diastolic BP** – depends on degree of exercise

Mild exercise – vasoconstriction → **DBP** ↑

Moderate-severe exercise – metabolic vasodilation & increased body temperature → **DBP** ↓ → **PP** ↑



◦ POSTURE

- ❖ Sudden change from supine to standing → **peripheral pooling** of blood in lower limbs
→ Venous Return ↓ es → CO ↓ es → **BP ↓ es**
- ❖ But, *returns to normal* or *may mildly increase* due to immediate corrections initiated by **Baroreceptor reflex**



○ Temperature

- ❖ **Hot environment $\rightarrow$ cutaneous VD $\rightarrow$ BP $\downarrow$ es**
- ❖ **Cold environment $\rightarrow$ cutaneous VC $\rightarrow$ BP $\uparrow$ es**



○ **Pregnancy**

- ❖ Blood volume ↑ es → CO ↑ es → **SBP** ↑ es
- ❖ Progesterone → relaxes smooth muscles → VD → PR ↓ es → **DBP** ↓ es
- ❖ PP increases in pregnancy



REGULATION OF BLOOD PRESSURE

- To maintain the normal MAP within a narrow range of 95-100 mm Hg
- **1. Rapid BP control mechanism/ Short-term regulation / Neural regulation**
 - a) Baroreceptor reflex**
 - b) Chemoreceptor reflex**
 - c) CNS ischaemic response**



- **2. Intermediate BP control mechanisms**

- a) Stress Relaxation mechanism**

- b) Reverse Stress Relaxation mechanism**

- c) Capillary fluid shift mechanism**



○ 3. Long-Term BP control mechanisms

- a) **Direct mechanism:-** Renal-body fluid feedback mechanism
- b) **Indirect mechanism:-** Renin-Angiotensin-Aldosterone system



SHORT-TERM REGULATION OF BP



BARORECEPTOR REFLEX / SINOARTIC REFLEX

- Most important among all the cardiovascular reflexes
- Regulates BP within seconds
- Life-saving reflex



BARORECEPTOR REFLEX

- **Receptors** – high pressure baroreceptors → **Carotid sinus & Aortic arch**
- Baroreceptors detect change in pressure in blood vessels in their walls
- **Afferents** – IXth cranial nerve from carotid sinus
Xth cranial nerve from aortic arch



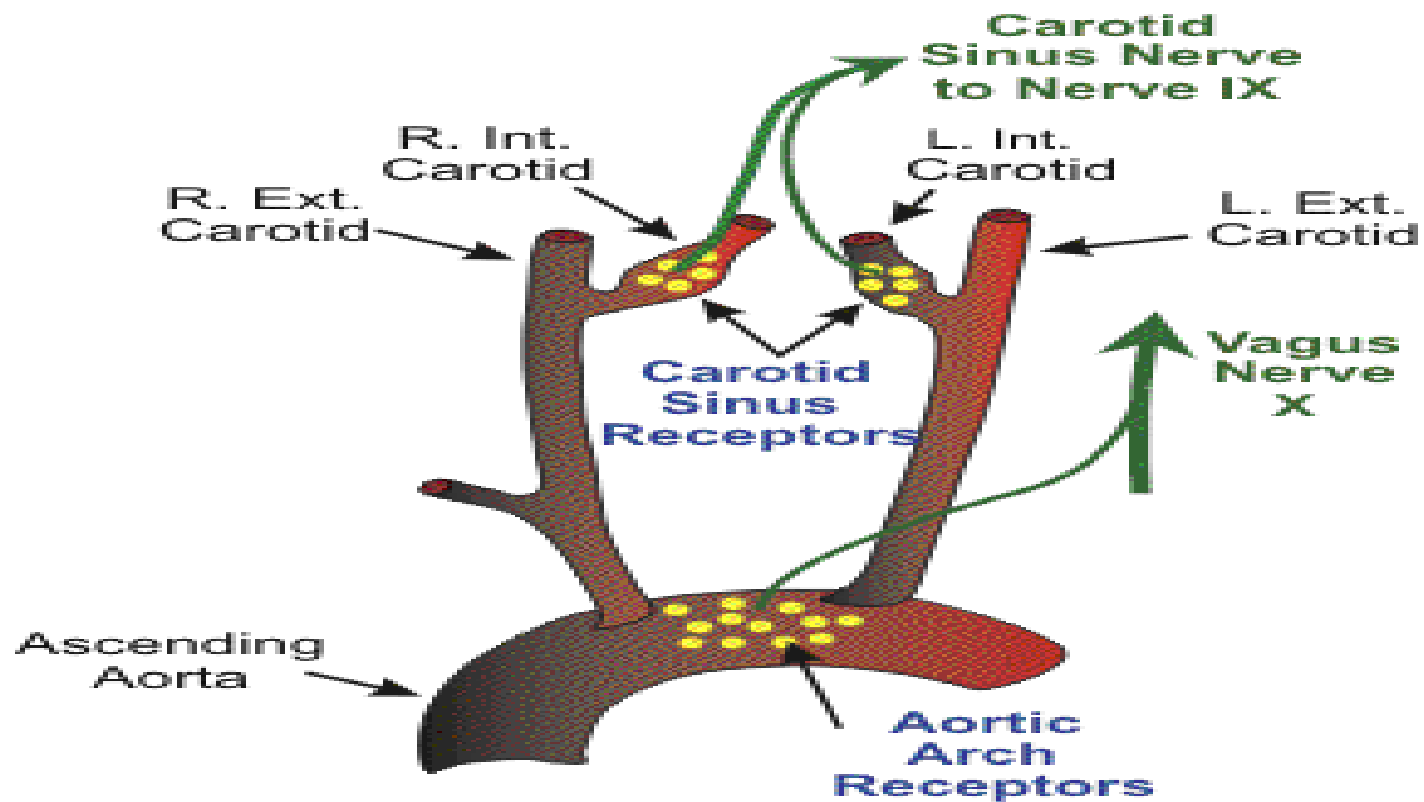


Figure 1. Location and innervation of arterial baroreceptors.



- Fibers from carotid sinus that joins glossopharyngeal nerve → **carotid sinus nerve/ Hering's nerve**
- Fibers in the vagus nerve carrying sensation from aortic arch form the **aortic nerve**
- Nerve fibers arising from carotid sinus & aortic arch receptors are also called **Buffer nerves/ Sinoaortic nerves**



- **Centers :- Medullary cardiovascular centers**

a) Vasomotor center

b) Cardioinhibitory center



○ Vasomotor center

- present in the *rostral ventrolateral medulla (RVLM)*

- central **sympathetic** center

- projects to intermediolateral gray column of spinal cord via **bulbospinal pathway** from where **sympathetic fibers** originate



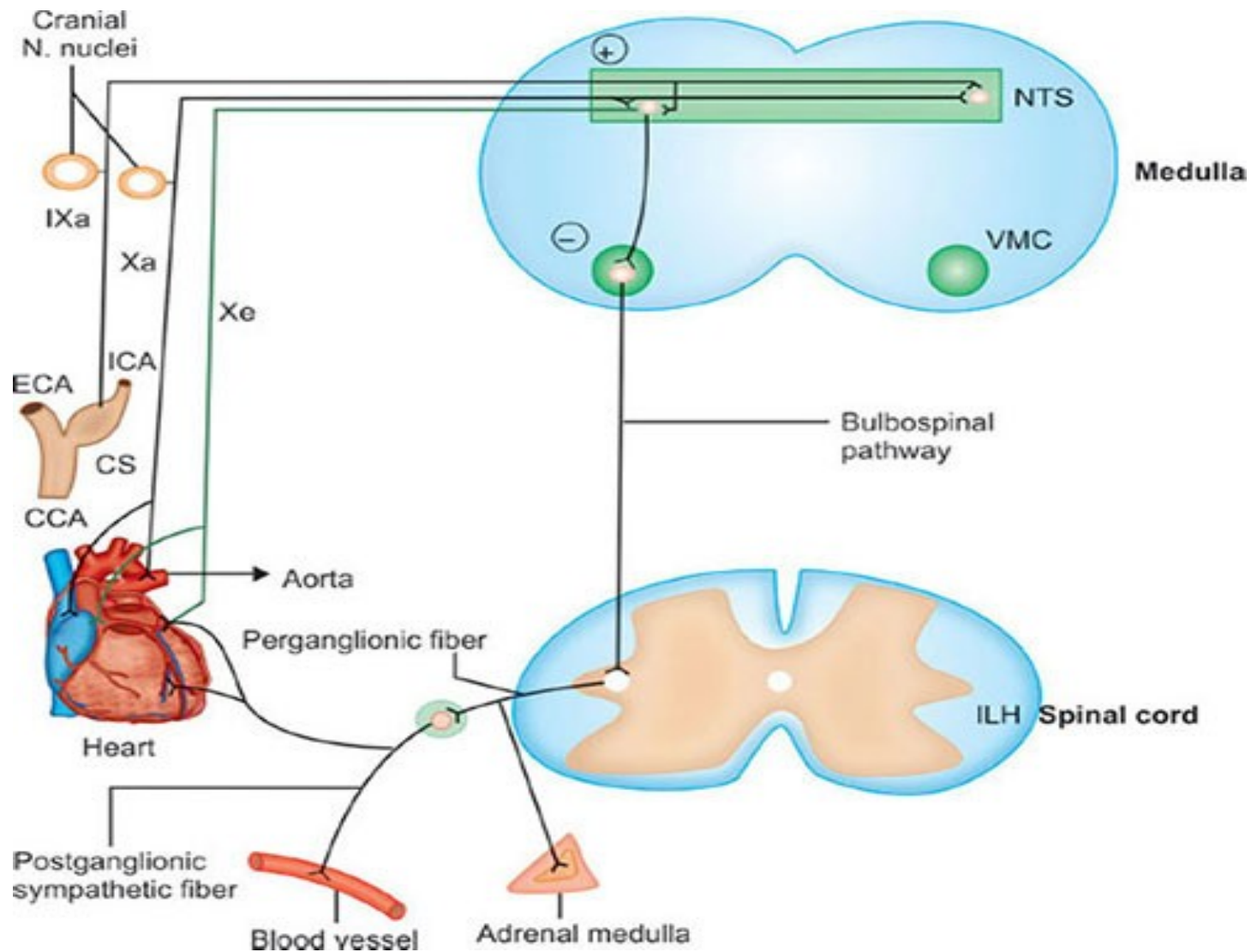
○ Cardioinhibitory center (CIC)

- ❖ formed by *NTS (mainly), Nucleus ambiguus & dorsal motor nucleus of vagus*
- ❖ *Main parasympathetic center* as vagus nerve originates from NTS
- ❖ NTS inhibits VMC via interneurons (CVLM)
- ❖ Thus, excitation of CIC stimulates vagus N & via inhibition of VMC inhibits sympathetic fibers



- **Efferents – sympathetic fibers & vagus N**
- Sympathetic fibers originate from the intermediolateral gray column of sp.cord
- Vagus N originates from NTS
- **Vagus N** innervates **heart**
- **Sympathetic fibers** innervate **heart & blood vessels**





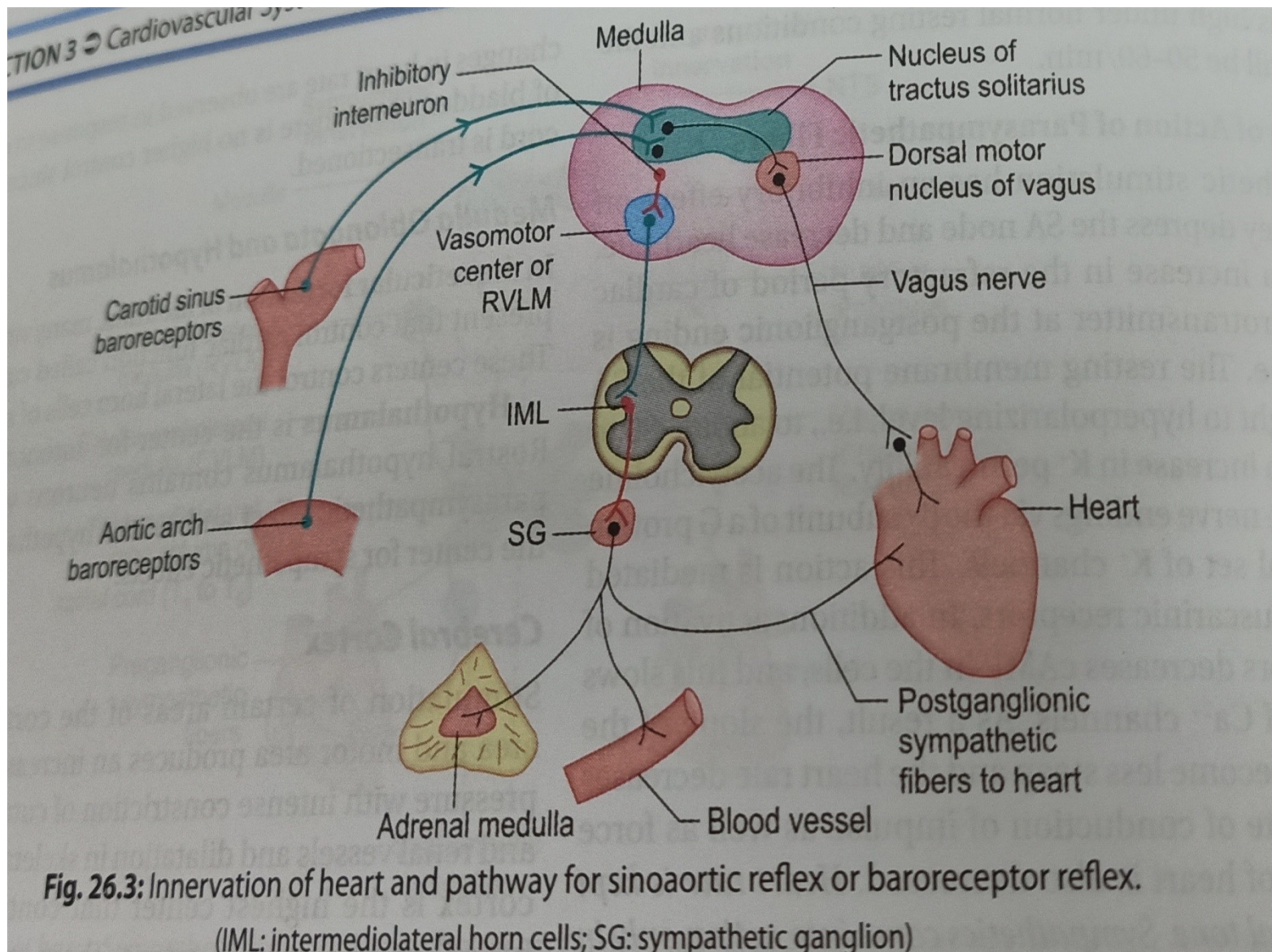
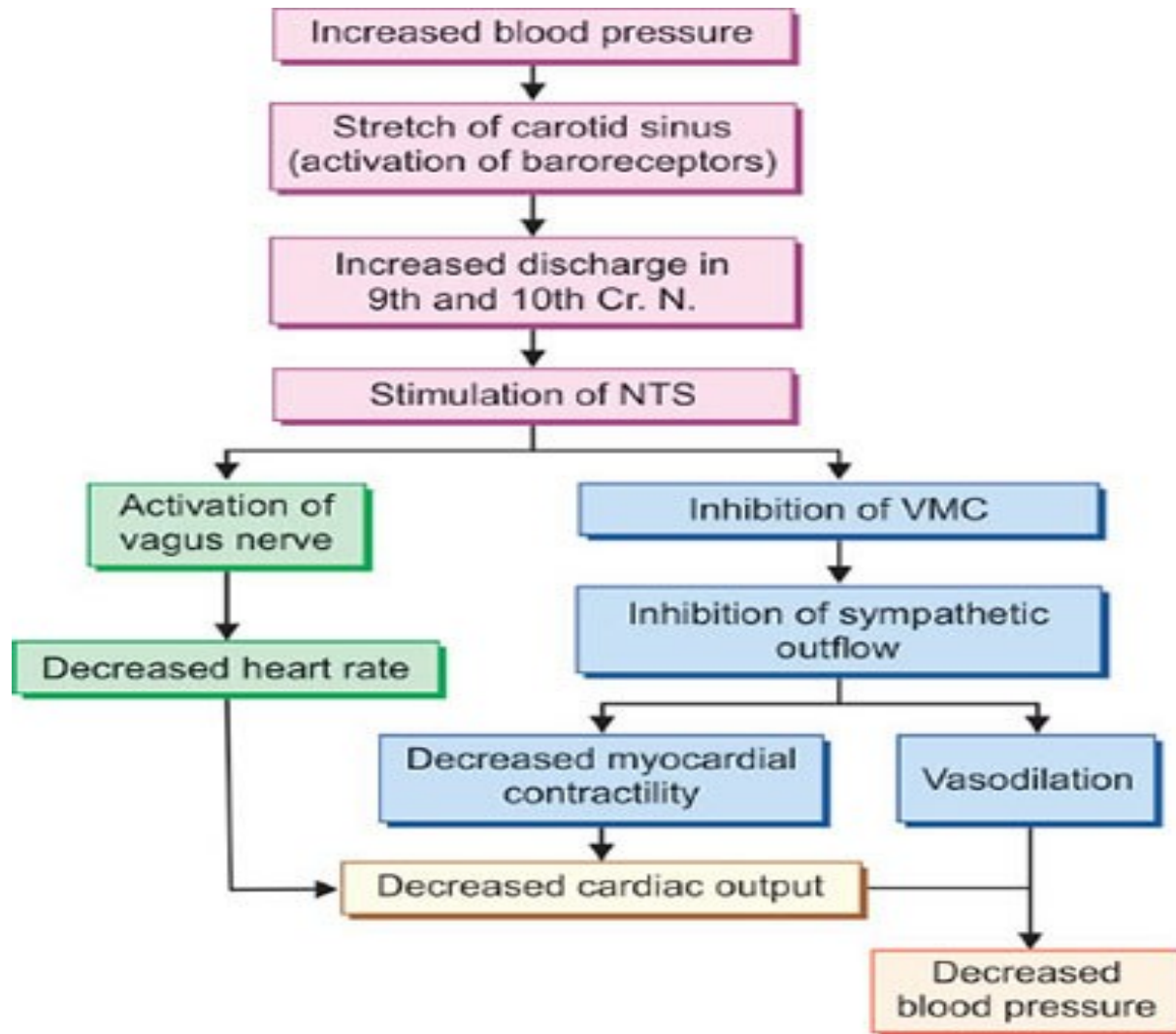


Fig. 26.3: Innervation of heart and pathway for sinoaortic reflex or baroreceptor reflex.

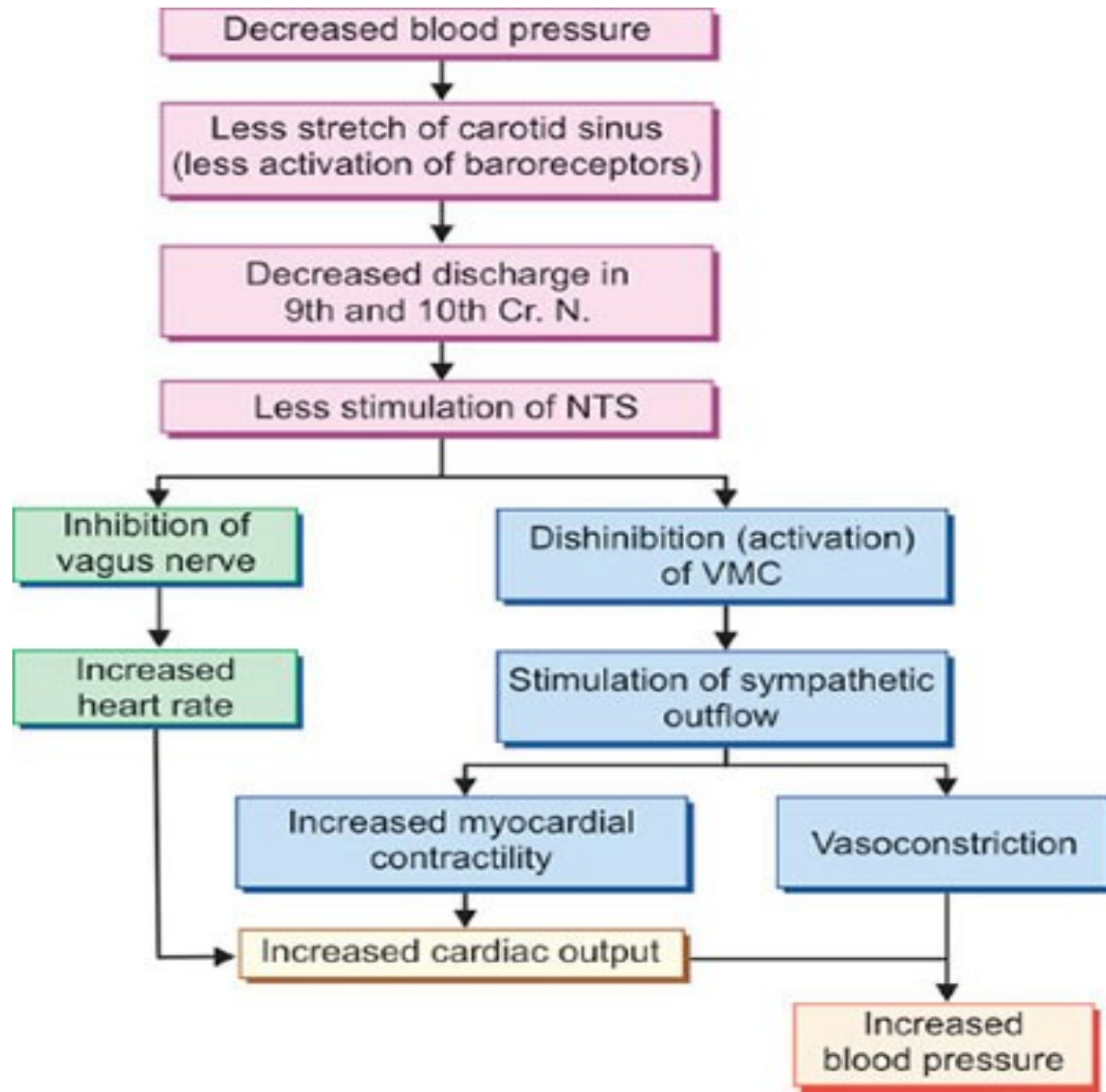
(IML: intermediolateral horn cells; SG: sympathetic ganglion)



WHEN BP INCREASES



WHEN BP DECREASES



- When BP falls → Baroreceptor reflex operates within few seconds to correct it
- Hence, lifesaving in acute hypotension & hemorrhage
- Corrects fall in BP on **change in posture**
- BR reflex is the **first and foremost reflex for BP regulation**



- Baroreceptors regulate BP in the pressure range of **50-200 mm Hg**
- Constitute a feedback mechanism to stabilize BP over a wide range of fluctuation in BP
- BR reflex explains the basis for **Marey's law** (HR is inversely proportional to BP)

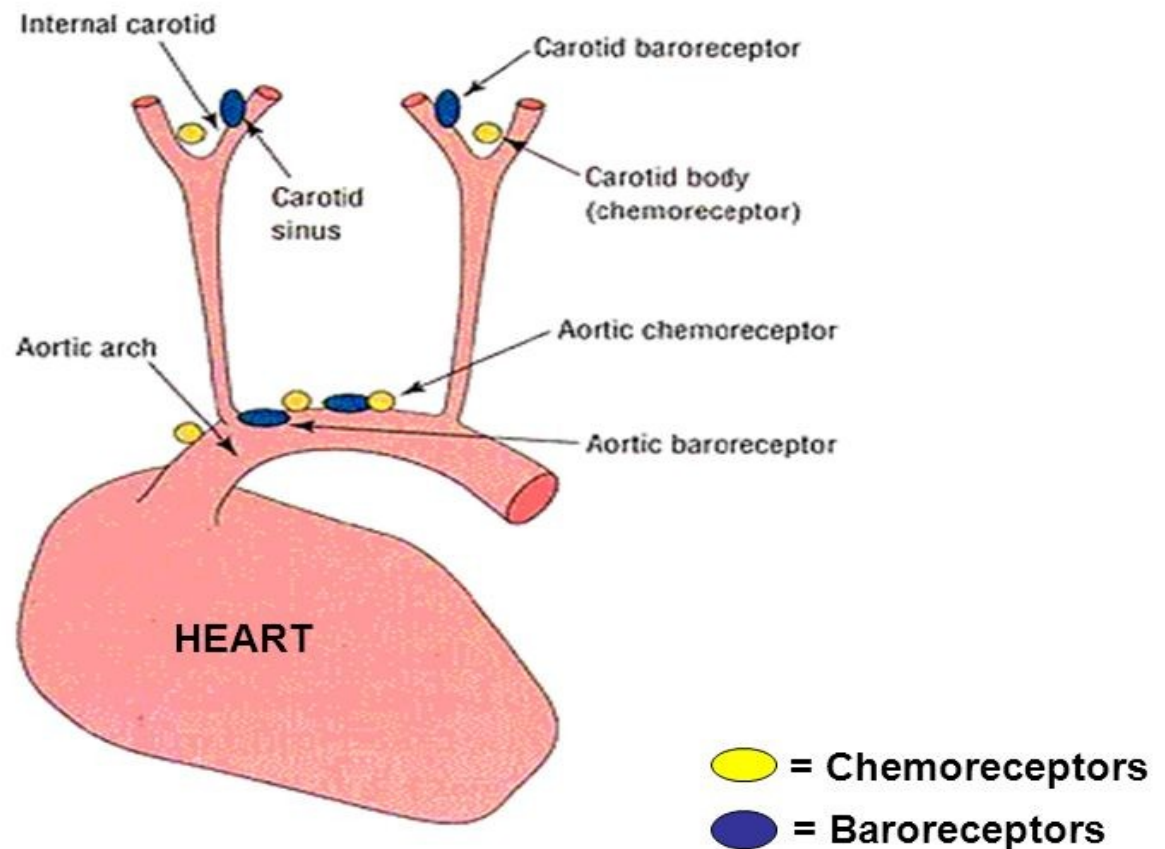


CHEMORECEPTOR REFLEX

- **Receptors :- Carotid bodies & Aortic bodies**
- Respond to change in *chemical composition of blood* occurring in *hypoxia, hypercapnia, acidosis*
- **Afferents :- IXth & Xth cranial nerve respectively**
- **Centers :- Vasomotor center, Cardioinhibitory center & respiratory centers**



Location of Chemoreceptor cells and Baroreceptor cells



- Whenever arterial BP falls below a critical level, chemoreceptors become stimulated-
How?
- Diminished blood flow causes decreased O_2 & excess buildup of CO_2 & H^+ ions that are not removed by the slowly flowing blood



EFFECTS OF CHEMORECEPTOR REFLEX

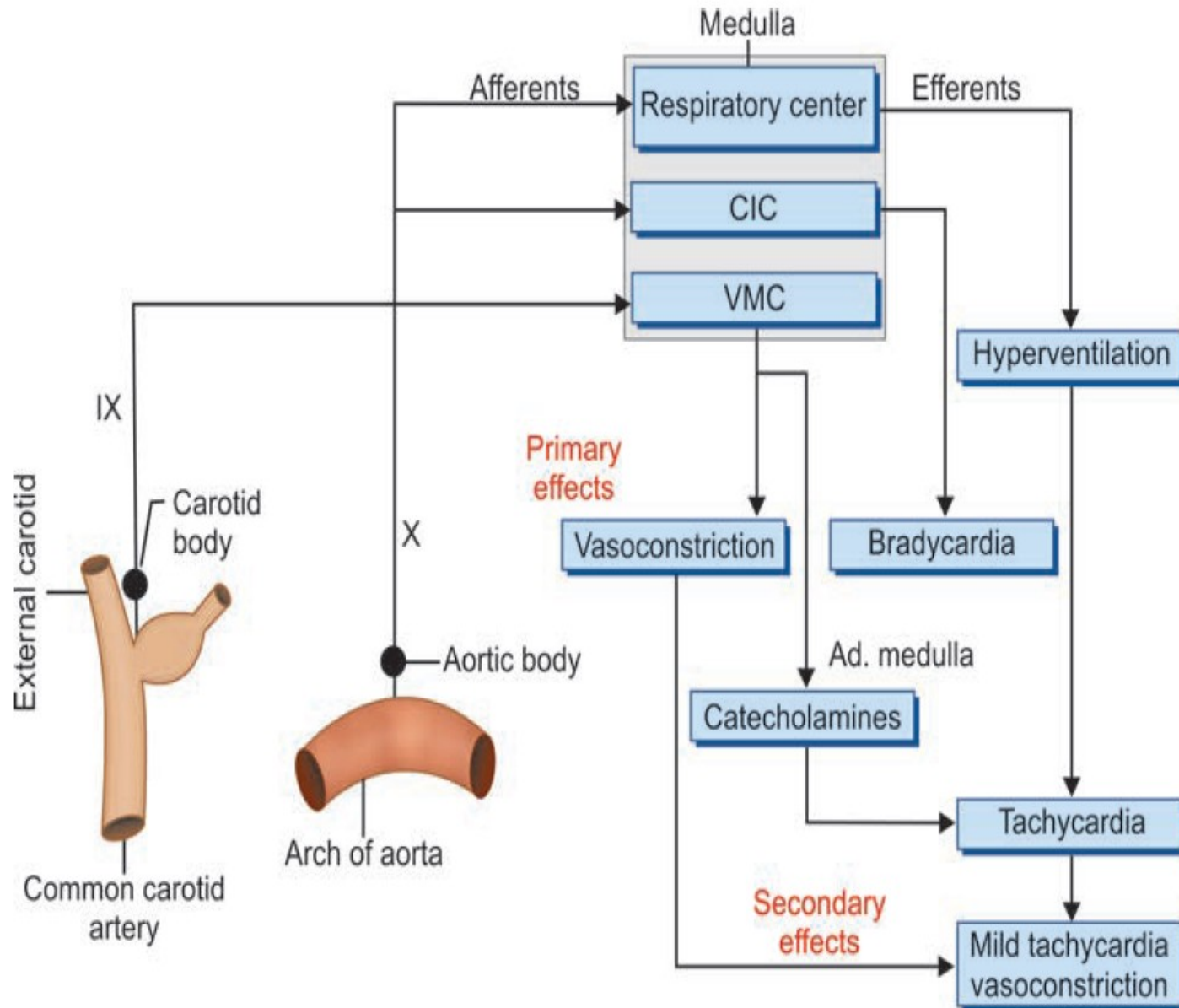
- **Primary effects:-** **Bradycardia & Vasoconstriction**
- ❖ Stimulation of **CIC** :- causes *Bradycardia*
- ❖ Stimulation of **VMC** :- causes *Vasoconstriction*
- **Secondary effects:-** **Tachycardia**
- ❖ Hypoxia → stimulates respiratory center → causes increase in pulmonary ventilation → hypocapnia & increased stretching of lungs → inhibits vagal centers → tachycardia



EFFECTS OF CHEMORECEPTOR REFLEX

- *Thus, the net effect of stimulation of CRs is mild tachycardia & vasoconstriction that increases BP*





- Chemoreceptor reflex is not a powerful arterial pressure controller until BP falls **below 80mm Hg**
- The range of BP regulation is **40-70mm Hg**
- Thus, it is at lower pressures that this reflex becomes important & helps to prevent further fall in pressure



CNS ISCHAEMIC RESPONSE

- Operates between **15-50mm Hg** range of MAP
- Does not operate until BP falls to 50mm Hg



CNS ISCHAEMIC RESPONSE

- Fall in arterial BP < 50mm Hg



CNS ischaemia



CO₂ / lactic acid accumulation in VMC



Directly stimulates VMC



Strong sympathetic stimulation leading to intense VC



HR & BP INCREASE to maintain blood supply to brain



CNS ISCHAEMIC RESPONSE

- Acts as an emergency arterial pressure control system
- Acts as a “**last ditch stand**” to prevent death
- It can elevate the MAP to as high as 250mmHg
- Most powerful among all the activators of sympathetic VC system



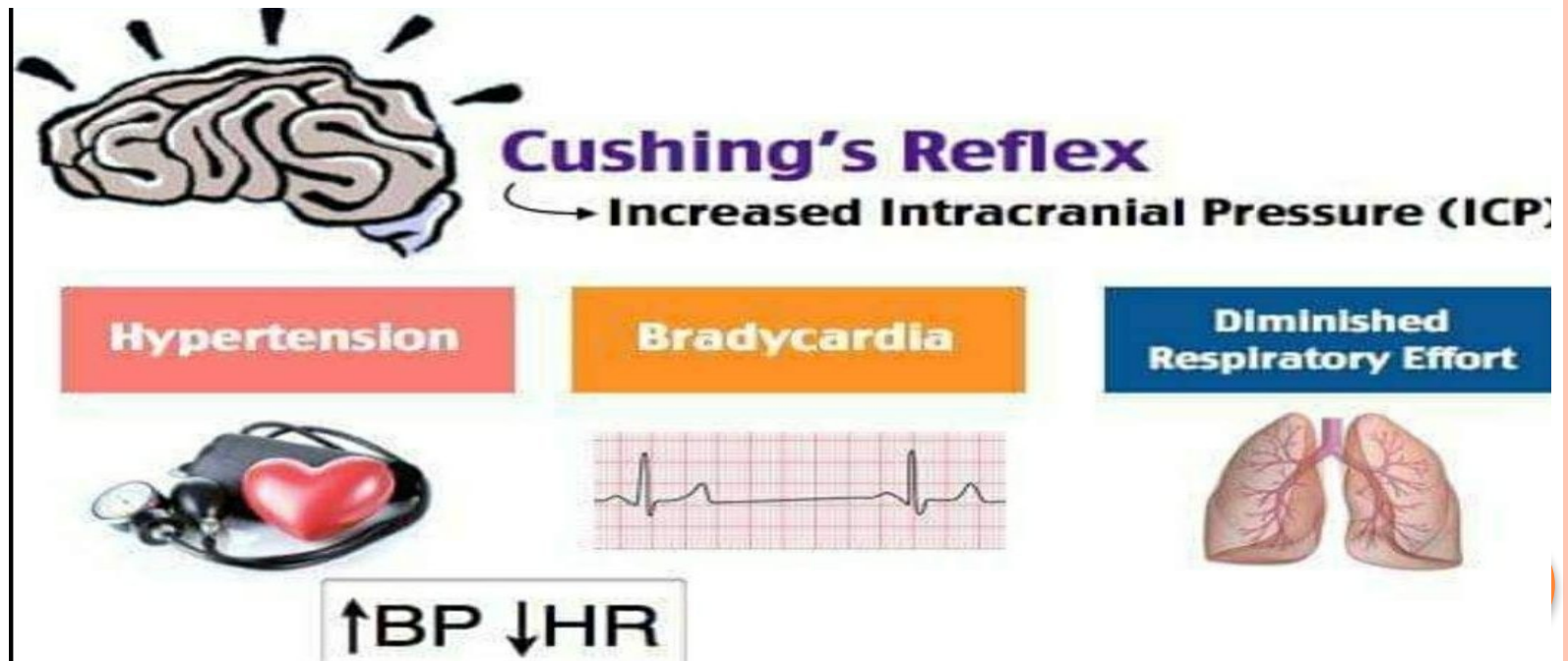
MISCELLANEOUS SHORT-TERM REGULATORY MECHANISMS

- Cushing reflex
- Atrial Stretch Reflex
- Bezold-Jarisch Reflex
- Bainbridge reflex

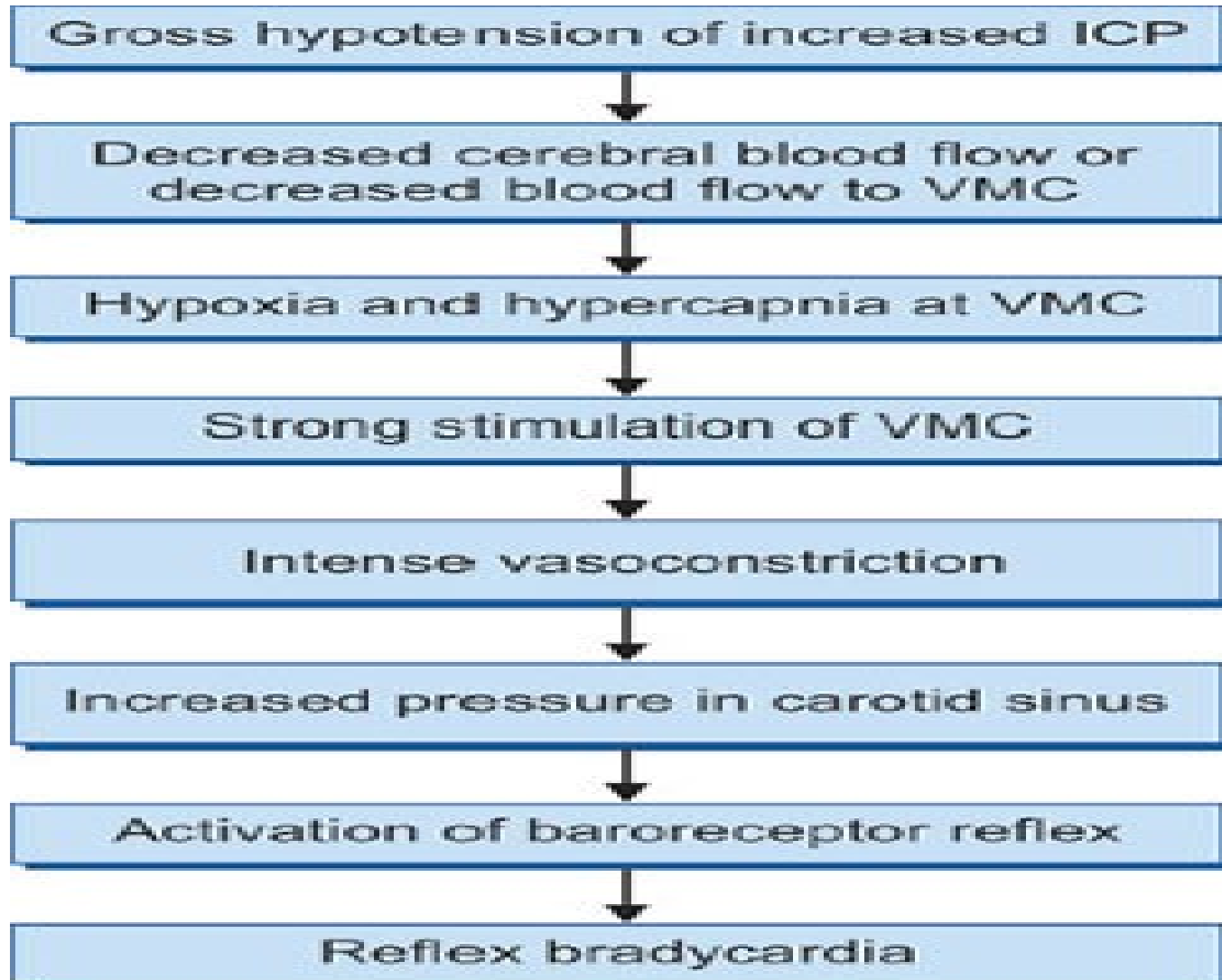


CUSHING REFLEX

- A special type of CNS ischemic response resulting from **increased IntraCranial Pressure (ICP)**



CUSHING REFLEX



ATRIAL STRETCH REFLEX / THE “VOLUME REFLEX”

- Increased Venous Return



Increased atrial filling → stretch of atria



- Causes:**
- a) Reflex dilation of afferent arterioles in kidneys → ↑ fluid filtration
 - b) Decreased secretion of ADH from hypothalamus → ↓ water reabsorption
 - c) Release of Atrial Natriuretic Peptide → act on kidneys to ↑ fluid excretion in urine



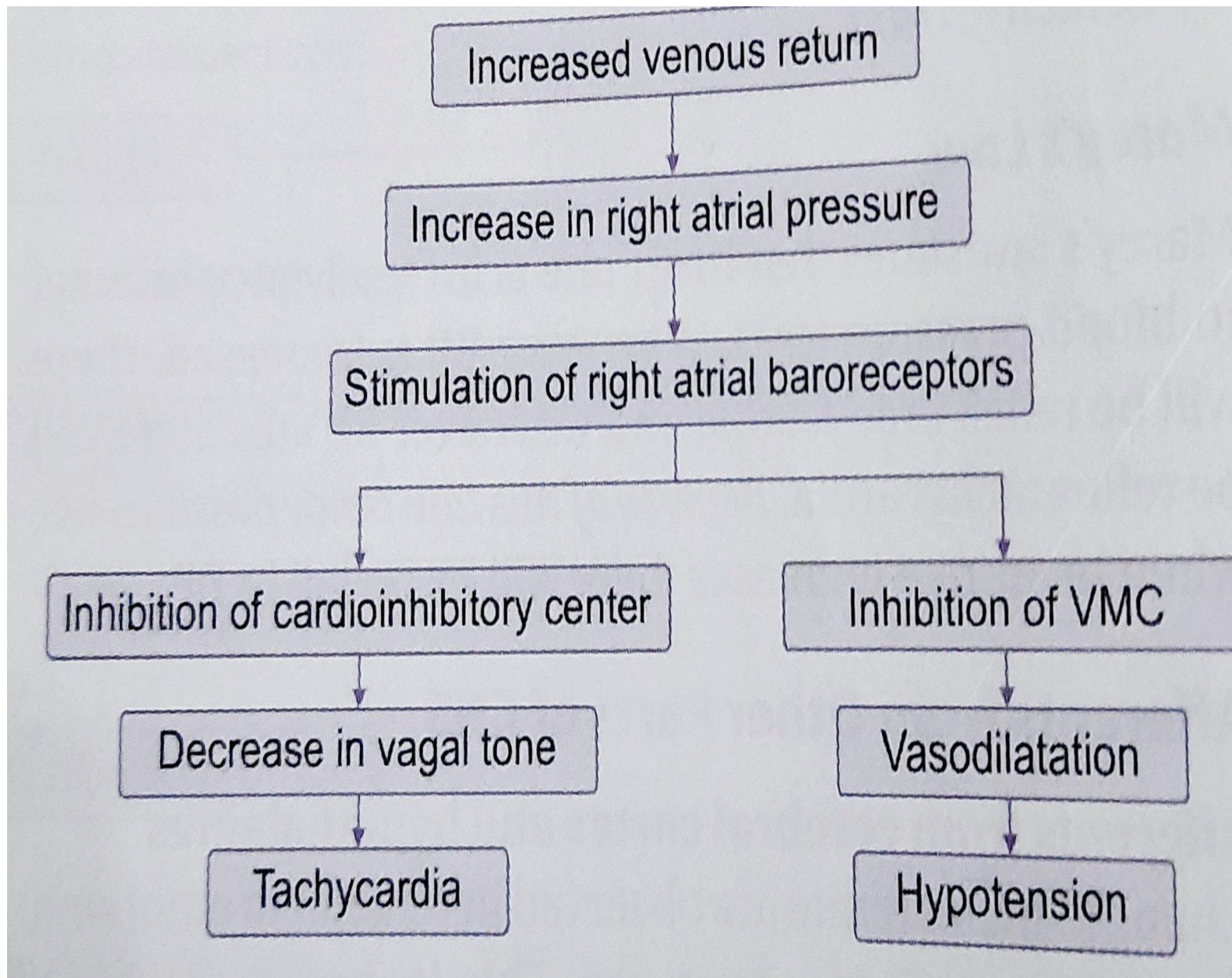
Return of blood volume to normal



BAINBRIDGE REFLEX

- Rapid infusion of blood/saline in anaesthetized animals produces **tachycardia** if the initial heart rate is low





BEZOLD-JARISCH REFLEX / CORONARY CHEMOREFLEX

- Injection of chemicals like **capsaicin**, **veratridine**, **phenyldiguanide** & **serotonin** into **left coronary artery** produces **reflex apnea** followed by **rapid breathing**, **bradycardia**, **hypotension**
- Receptors present in coronary arteries & left ventricles are **C-fiber endings**
- Afferents from these receptors are **vagal fibers**
- In MI → chemicals from infarcted tissue → stimulate ventricular receptors → Bradycardia & Hypotension



INTERMEDIATE REGULATORY MECHANISMS



INTERMEDIATE REGULATORY MECHANISMS

- Act within few minutes to few hours
- Remain functional from few days to a month only
- Corrections are done primarily by altering the blood volume



1. STRESS RELAXATION MECHANISM

- *Rise in arterial BP (eg: following massive slow iv transfusion) → vessels become stretched → causes **relaxation of BVs by vascular tone adjustment** → causes increase in capacity of arterial system → concomitant fall in BP*



2. REVERSE STRESS RELAXATION MECHANISM

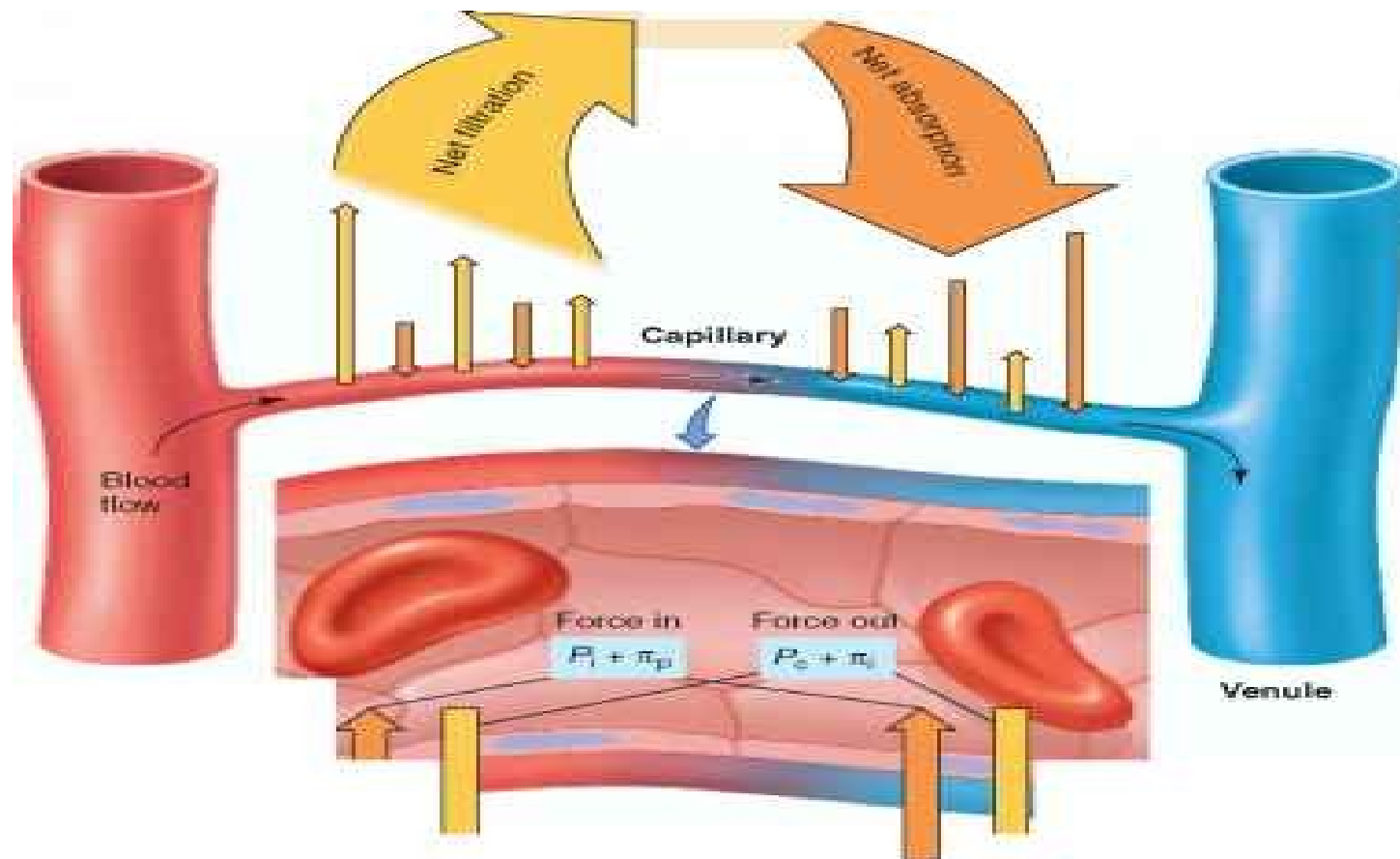
- *Fall in arterial BP (eg: following prolonged slow bleeding) → less stress on vessel wall → causes tightening of BV walls by vascular tone adjustment → restores BP back to normal*



3. CAPILLARY FLUID SHIFT MECHANISM

- **When BP is raised** → *mean capillary pressure ↑ es → shift of fluid from circulation to ISF compartments → reduces blood volume → restores BP*
- **When BP is lowered** → *mean capillary pressure ↓ es → absorption of fluid from interstitium to circulation → blood volume increases → restores BP*





3. CAPILLARY FLUID SHIFT MECHANISM

- *This mechanism is two times more effective than baroreceptor reflex but it begins to act much slowly*



LONG-TERM BP CONTROL MECHANISMS

- 1. Renal-body fluid feedback mechanism/
Direct mechanism
 - ❖ Corrects BP by causing appropriate changes in blood volume through **diuresis & natriuresis**
 - ❖ As arterial pressure increases from **60mm Hg to 200mm Hg** → progressive increase in water excretion (*pressure diuresis*) & sodium excretion (*pressure natriuresis*) in urine



- When BP falls too low



Renal ischemia



Decrease in GFR



Water & electrolyte retention



Increases the ECF volume



increase in blood volume



Increase in cardiac output & increase in BP



- When BP rises too high



Pressure natriuresis & diuresis



ECF volume & blood volume ↓ es



BP returns to normal



2. RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM (RAAS)

○ RENIN

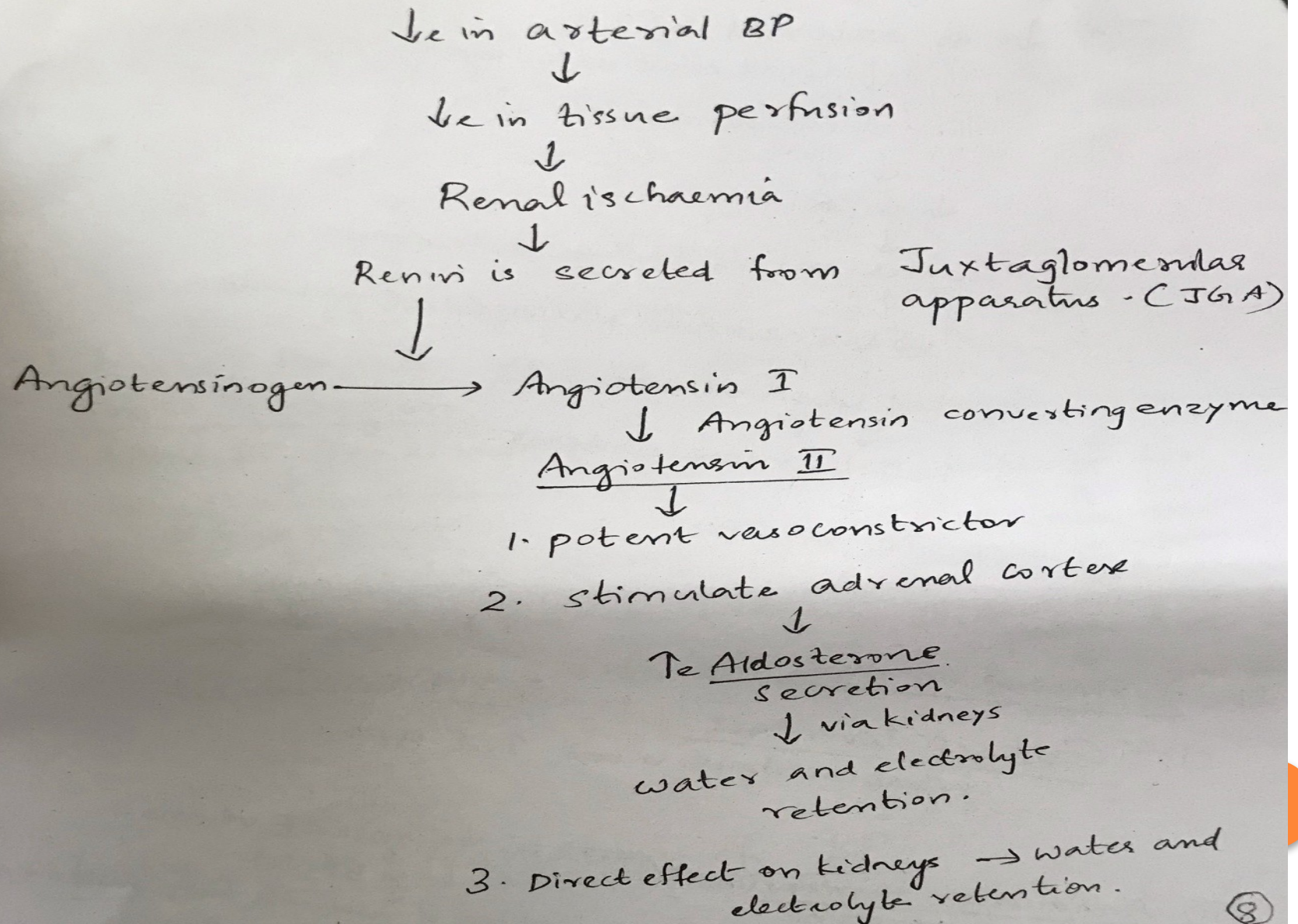
- ❖ A protein enzyme released by kidneys when arterial pressure falls too low
- ❖ Synthesized & stored in an inactive form (prorenin) in juxtaglomerular cells (JG cells)
- ❖ When BP falls → intrinsic reactions in kidneys cause conversion of prorenin to renin



- Renin acts on **renin substrate (or angiotensinogen)** to release **angiotensin-I** (mild vasoconstrictor)
- Angiotensin-I is converted to **angiotensin-II** by ACE (**Angiotensin Converting Enzyme**) present in **endothelium of lung vessels**
- **Angiotensin-II** is a **powerful vasoconstrictor**



RENIN-ANGIOTENSIN ALDOSTERONE



APPLIED ASPECTS

◦ 1. HYPERTENSION

- ❖ Condition in which value of systolic BP is persistently **>140 mmHg** and diastolic BP **> 90mmHg**



❖ 2types:-

a.Primary HTN/Essential HTN

-Raised BP without any underlying disease

-Risk factors:- obesity, heredity, stress, smoking

-Presents in 2 forms

- i) **Benign HTN** :- BP is moderately raised
- ii) **Malignant HTN** :- sudden marked rise in BP
eg- 250/150mmHg



b. Secondary HTN

-BP is raised due to some other underlying disease.

- Common causes are:-

a) **Cardiovascular diseases** - atherosclerosis,
coarctation of aorta

b) **Renal diseases** – Renal artery stenosis, Tumour
of JG cells leading to excess angio-II,
Glomerulonephritis

c) **Endocrine disorders** – Cushing syndrome,
Hyperaldosteronism

d) **Neurological disorders** – Raised ICP

e) **Pregnancy Induced Hypertension (PIH)**

