BP = 120/80 mm Hg = SYSTOLIC / DIASTOLIC

SYSTOLIC → cardiac output
DIASTOLIC → Total peripheral resistance

Systolic pressure ∝ cardiac output

- At systole phase Aortic valve is fully opened & the pressure in Lt ventricle is freely transferred to the major vessels.
- If cardiac output is ↑ → more blood is pushed into the arteries → more pressure in major arterial tree ∴ ↑ Systolic pressure.
- During diastole when Aortic valve closes, relationship B/W major arterial tree & the ventricle [communication] is cut off, reducing pressure in the ventricles not influencing pressure in major arteries.
- During diastole the aorta undergoes elastic recoil.
- During diastole if the arterioles are dilated & offering less resistance to incoming blood flow

During diastole the blood pressure in Aorta not influenced by ventricular pressure

- If the TPR is less ie the arterioles are dilated blood in arteries is easily shifted to capillaries & veins & the diastolic pressure falls

∴ If TPR is ↑ ⇒ diastolic pressure ↑
TPR is ↓ ⇒ diastolic pressure falls.

- Mean Pulse pressure :-

MAP = diastolic pressure + $\frac{120-80}{3}$

$= 80 + \frac{40}{3} = 93$ mm Hg

MAP = diastolic pressure + $\frac{\text{Pulse pressure}}{3}$

- Mean AP => Pressure only in Systemic Arteries

Mean Systemic pressure => Pressure in arterial + capillaries + veins ie complete systemic circulation

MSP << MAP

- ⭐ MSP :- Pressure maintained in systemic circulation when there is no cardiac output & but is not functioning as a pump.

V low pressure

MAP
i) High
ii) Maintains blood flow towards the tissue

93 - 100 mm

MSP
i) Low
ii) Maintains the venous return from tissue

6.5 mm

- Factors affecting BP :-

- BP is the product of two values
  1. Cardiac output
  2. TPR

BP = Cardiac output (SV) × TPR

Diastolic Pressure

Cardiac output (SV)
- Preload
  - EDV
  - EDP
  - ⇓ Venous return
    - RAP
      - → venomotor tone
      - → Blood volume
    - Filling time ie diastolic interval
- Contractility
  - CO ∝ contractility
- Afterload
  - TPR

- Pressure in circulation depends on "stressed volume" in arterial system.

Both venoconstriction & arteriolconstriction ↑ stressed volume

- venoconstriction → Blood flows towards ht ie unstressed vol shifts towards stressed volume
- arteriolconstriction → Blood is trapped in arteries ie stressed not allowed to become unstressed volume.

$$MAP = CO \times TPR$$

CO & TPR are interdependent variables

∴ If CO is doubled MAP is ↑ but not doubled as TPR will be ↓

& when TPR is 2x MAP ↑ but is not doubled.

Regulation of BP

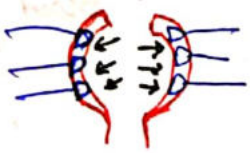
Short term / Rapid ⇓ Neurological

- If BP ↑
- If BP ↓

Long term ⇓ Renin angiotensin aldosterone Axis

- Neural Regulation :-

- There are BP sensors in the arterial system.
- Carotid sinus → Baroreceptors → carotid sinus branch of 9th CN Nerve [Glossph]
- Aorta → Baroreceptors → 10th CN [Vagus]
- If BP ↑ ⇒ carotid sinus stretched more → Nerve ending also stretched ⇒ Na^+ channels open ⇒ Na^+ influx ⇒ depolarisation ⇒ Action pot.



- Central regulator is a spl type of nucleus in the medulla → Nucleus of Tractus solitarius
  ↳ Receives the info from 9th & 10th

When BP ↑ ⇒ Heart rate ↓ → Cardioinhibitory center

Signals thru vagus
Rt vagus to SAN
Lft vagus to AVN

When BP ↑ ⇒ (1) cardioinhibitory centre is activated by NTS
Hrt rt ↓

⇒ (2) cardioaccelerator nucleus is inhibited by NTS
↓
generally when active Hrt rate ↑ by sympathetic outflow.

⇒ (3) Vasomotor centre is also inhibited by NTS
Symp outflow is inhibited
① Venodilatation
② Arteriolodilation
③ Sympathetic outflow to Adrenal medulla inhibited.
④ Symp outflow to Juxtaglomerular apparatus is also reduced

When BP ↓ ⇒ Reverse activity with vagal inhibition.

- Nerve endings in carotid sinus & Aorta are very sensitive to rate of change of BP
- Glossopharyngeal & Vagus — Buffer Nerves — regulate BP

	Aff activity	Parasym NS	BP	Sym NS
↑ BP	↑	↑		↓
↓ BP	↓	↓		↑
↓BP ← carotid occlusion	↓	↓		↑
↑BP ← carotid sinus massage	↑	↑		↓
Afferent cut	↓	↓	↑	↑

- From lying position, a person suddenly stands up blood tends to pool down — venous return less — CO ↓ afferent fires less → Perceived as low BP → Sympathetic outflow ↑ → BP restored → Hrt rate ↑

This restores venous return by venoconstriction

— ☆ If a person with low blood volume suddenly stands up from lying position he'll feel blackout = vitigo.

Why?

Inspite of sympathetic mediated venoconstriction venous return is less as the blood volume is less. Cardiac output is less — BP not restored

- Cerebral blood flow reduced.

A person who is vol depleted / has ↓ sympathetic activity

Blood will gravitate → significant fall in systolic blood pressure by more than 10 mm Hg — Orthostatic hypotension

— If a person is volume overloaded BP is ↑ → afferent activity ↑ → Parasym act. ↑ → Sym activity ↓ → BP restored.

— Long Term Regulation :-

- Dependent on RAAS

BP ↓ → reduced renal perfusion

At the beginning of DCT there is Macula densa

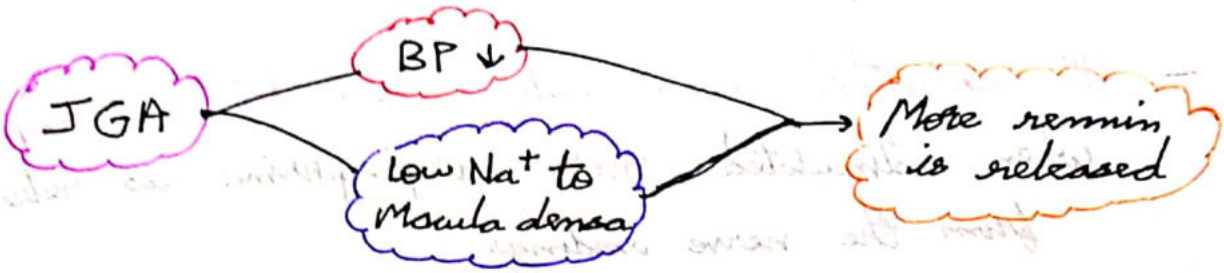
Modified smooth muscles near Macula densa — Polkisin cells of Aff arteriodes

Polkisin cells + Macula densa → JGA. ↳ Act as baroreceptors

BP ↓ → JGA activates → A spl type of enzyme is released called Renmin.

BP ↓ → GFR ↓ → luminal fluid is less and is moving v. slowly thru the nephron → excessive retention of Na^+ → v little Na^+ reaches the macula densa

Macula densa is sensitive to Na^+ → stimulates the JGA → more renin



- JGA — β_1 adrenergic receptors — More renin stimulated

- Liver → Angiotensinogen —Renin→ Angiotensin I [10 aa]

- A unique type of enzyme is syn by endothelial cells of pulmonary circulation & pulmonary system this converts

AT I —Angiotensin converting enzyme→ AT II

- Vascular smooth muscle - AT II acts on 7 pass AT-II receptor coupled with Gq → IP_3 & diacylglycerol (DAG)

↓

Ca^{2+} ↑ in smooth muscle

↓

Construction

Hence Angiotensin II - Powerful vasoconstrictor

Veins

↓

Venous return ↑

↓

EDV ↑

↓

↑ Stroke vol

↓

↑ C.O.

↑ Systolic BP

Arterioles

↓

↑ TPR

↓

↑ diastolic BP

- AT II receptors ↑ hypothalamus [Sub fornical region]
  ↳ Sense of thirst is produced.

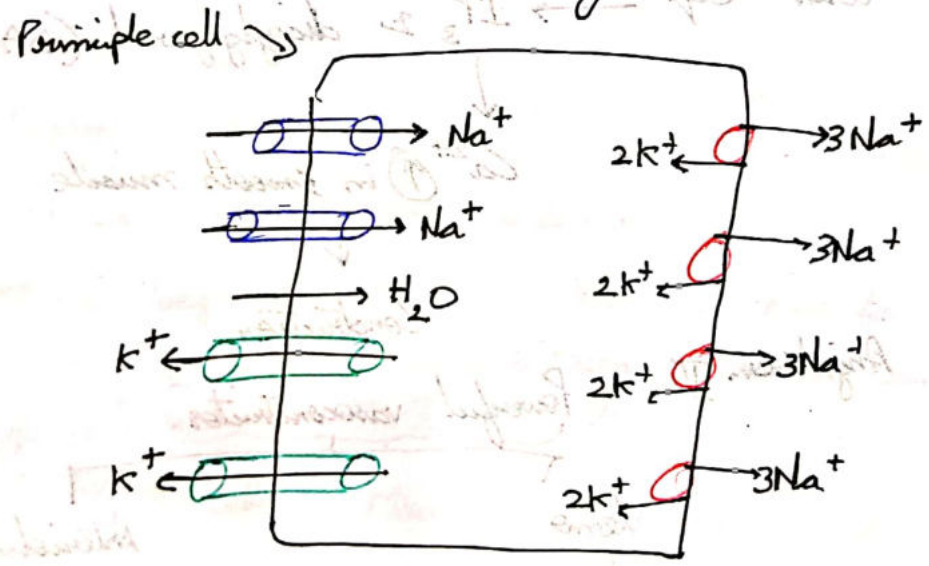
← All sympathetic nerve endings have AT-II receptor
when stimulated more norepinephrine is released from the nerve endings

- Cells in the Zona Glomerulosa have AT II receptors when stimulated the cells release hormone Aldosterone

Aldosterone → steroid hormone
↳ works on principle cells of DCT & CD

Retention of salt & water.
Na^+ absorption
K^+ secretion
Aldosterone
① Na/K ATPases on Basolateral mem
Na^+ channels on Apical cells membrane
K^+ channels on Apical membrane.

RAAS mech takes a long time.



- PCT cells are also rich in AT-II receptors
  ↳ Na^+ absorption H^+ secretion

$$HCO_3^- + H^+ \longrightarrow H_2CO_3^* \longrightarrow H_2O + CO_2$$

CO_2 absorbed in the cell → HCO_3^- formed & absorbed in cell.

- AT - II receptors on efferent arteriole → constriction
  when stimulated → GFR maintained

Drugs :-

① ACE - I inhibitors → act on ACE on endothelial cells of Pulmo microcirculation.

② Aldosterone inhibitors → Spironolacton - structural inhibitor

Note :- ACE - I inhibitors must not be given to patients with Renal stenosis

Renal stenosis → GFR ↓ → ↑ AT - II → efferent arteriole constrict → GFR normal

③ AT - II receptor blocker - Losartan.

- Other Types of Regulation

① Chemoreceptors

Peripheral
- Carotid body
  - Most sensitive to PO_2
  - PO_2 ↓
  - CN IX & X
  - NTS
  - Vagal outflow ↓
  - Sym outflow ↑
  - Cardiac output ↑
- Aortic body

Central
- Sensitive to PCO_2 & pH
- Medulla near 4th ventricle
- ↑ PCO_2 → $CO_2 + H_2O \rightarrow H_2CO_3$
  - ↓
  - ↑ $H^+ + HCO_3^-$
- ↑ H^+
  - Symp ← → Parasymp
  - Rising BP → falling pulse rate

↑ Intracranial Pressure

↓ Cerebral Blood flow

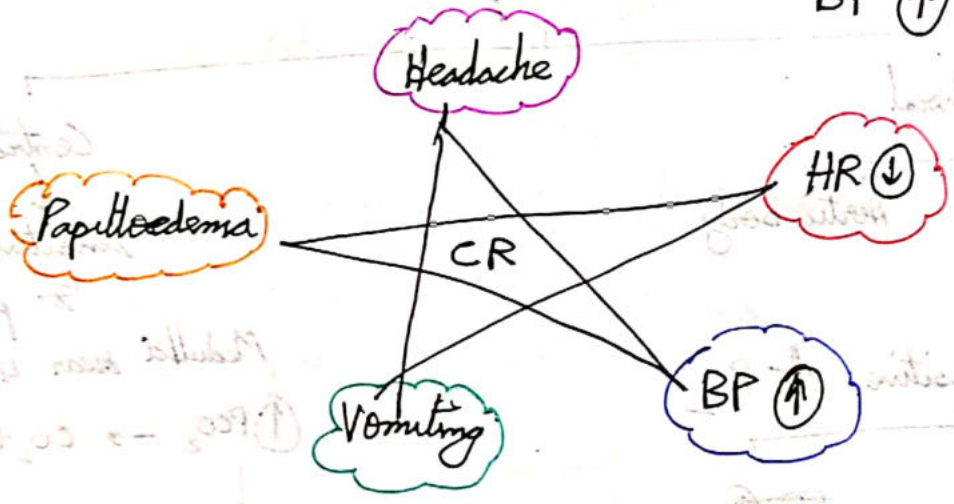
↑ PCO_2

$CO_2 + H_2O \longrightarrow H_2CO_3 \longrightarrow H^+ + HCO_3^-$

↑ H^+ stimulates central chemoreceptors

↑ Vagal output ⇓ Hrt rate ↓

↑ Sympathetic output ⇓ BP ↑



(2) Anti diuretic hormone :-

ADH → V_2 receptors on last part of Nephron → ↑ H_2O absorption

→ high conc → Arteriodconsbriction

(3) Atrial Natriuretic peptide :-

i) Loss of H_2O & salt ii) High conc – Vasodilation.