

Blood pressure

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

⇒ Systolic Blood pressure

max pressure recorded during cardiac cycle

Normal Systolic BP 100-120 mm Hg

depends mainly on cardiac output.

⇒ Diastolic Blood pressure

min pressure recorded during cardiac cycle

Normal value 80-80 mm Hg

depends primarily on peripheral resistance

Pulse pressure

difference b/w systolic & diastolic BP.

$$PP = SBP - DBP$$

Normal value 20-50 mm Hg

• maintains the normal pulsatile nature of blood flow required for tissue perfusion

⇒ Mean Arterial pressure

Avg pressure recorded during cardiac cycle.

$$MAP = DP + \frac{1}{3} PP$$

Normal value = 75-105 mm Hg

MAP helps in forward movement of blood in lumen of blood vessels → maintain tissue perfusion

Blood Pressure classification

	SBP	DBP
• 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 BP

$$BP = \text{cardiac output} \times \text{peripheral resistance}$$

- Alteration in cardiac output mainly affects systolic pressure
- Alteration in peripheral resistance mainly affects diastolic pressure

Factors affecting cardiac output

$$\text{Cardiac output} = \text{stroke Volume} \times \text{heart rate}$$

- Stroke volume is affected by preload, afterload and myocardial contractility.
- Heart rate is mainly affected by autonomic influences

Factors affecting peripheral resistance

1) diameter of blood vessels

• depends primarily on vasomotor tone - the sympathetic vasoconstrictor tone

vasoconstriction - increase PR → increase BP
 vasodilation - decrease PR → decrease BP

- when blood vessels become stiff $\rightarrow$ PR $\uparrow$ BP $\uparrow$

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 no of cells in blood
- temperature
- Resistance of cells to deformation

3) Velocity of blood

Total PR is inversely related to velocity of blood flow

$\Rightarrow$ Physiological Variations

1) Age

BP increases with age

2) Gender

BP is comparatively less in females during their reproductive life - why?

due to effect of

a) Progesterone : Relax BV smooth muscles

b) Estrogen : prevents atherosclerosis

3) Exercise

- Systolic BP always rises due to increased CO
- Diastolic BP depend on degree of exercise

Mild exercise : Vasoconstriction $\rightarrow$ DBP $\uparrow$

Moderate

- Metabolic vasodilation & increased body temp $\rightarrow$ DBP $\downarrow \rightarrow$ PP $\uparrow$

•) eating

BP increases after a meal.

•) sleep

BP decreases during sleep.

•) Emotion and Excitement

↑ sed sympathetic discharge

→ ↑ sed BP

•) Posture

sudden change from supine to standing →
Peripheral pooling of blood in lower limbs

→ venous return ↓ sed → CO ↓ sed → BP ↓ sed

But, returns to normal or may mildly
increase due to immediate corrections
initiated by Baroreceptor reflex

•) Temperature

Hot environment → cutaneous VD → BP ↓ sed

Cold environment → cutaneous VC → BP ↑ sed

•) Pregnancy

• Blood volume ↑ sed → CO ↑ sed → SBP ↑ sed.

• Progesterone → relaxes smooth muscles →

VD → PR ↓ sed → DBP ↓ sed.

• PP increases in pregnancy

Regulation of BP

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 Relaxn mechanism
- c) capillary fluid shift mechanism

• 3) long term BP control mechanisms

- a) Direct mechanism - Renal body fluid feedback mechanism
- b) Indirect mechanism - RAA system

Short term Regulation of BP

- 1) Baroreceptor reflex / ~~receptor~~ sinoaortic reflex
 - Most imp among all the cardiovascular reflexes
 - Regulates BP within seconds
 - life saving reflex

Receptors - high pressure baroreceptors → carotid sinus & Aortic Arch.

- Baroreceptors detect change in pressure in blood vessels in their wall
- Afferents → IXth cranial nerve from carotid sinus
Xth cranial nerve from aortic arch
- Fibres from carotid sinus that joins glossopharyngeal nerve → carotid sinus nerve / Hering's nerve

- Fibres in the vagus nerve carrying sensation from aortic arch form the aortic nerve.
- Nerve fibres arising from carotid sinus and aortic arch receptors are also called baroreceptors / Sinu-aortic nerves

⇒ centers : medullary cardiovascular centres.

- vasomotor center
- cardioinhibitory center

• Vasomotor center

- Present in the rostral ventrolateral medulla (RVLM)
- central sympathetic center
- Projects to intermediolateral grey column of spinal cord via bulbosacral pathway from where sympathetic fibres originate

• Cardioinhibitory center (CIC)

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

⇒ • Efferents - sympathetic fibres & vagus nerve

- Symp fibres originate from the intermediolateral grey column of spinal cord

- Vagus nerve originates from NTS
- Vagus nerve innervates heart
- Symp fibres innervates heart & Blood vessels

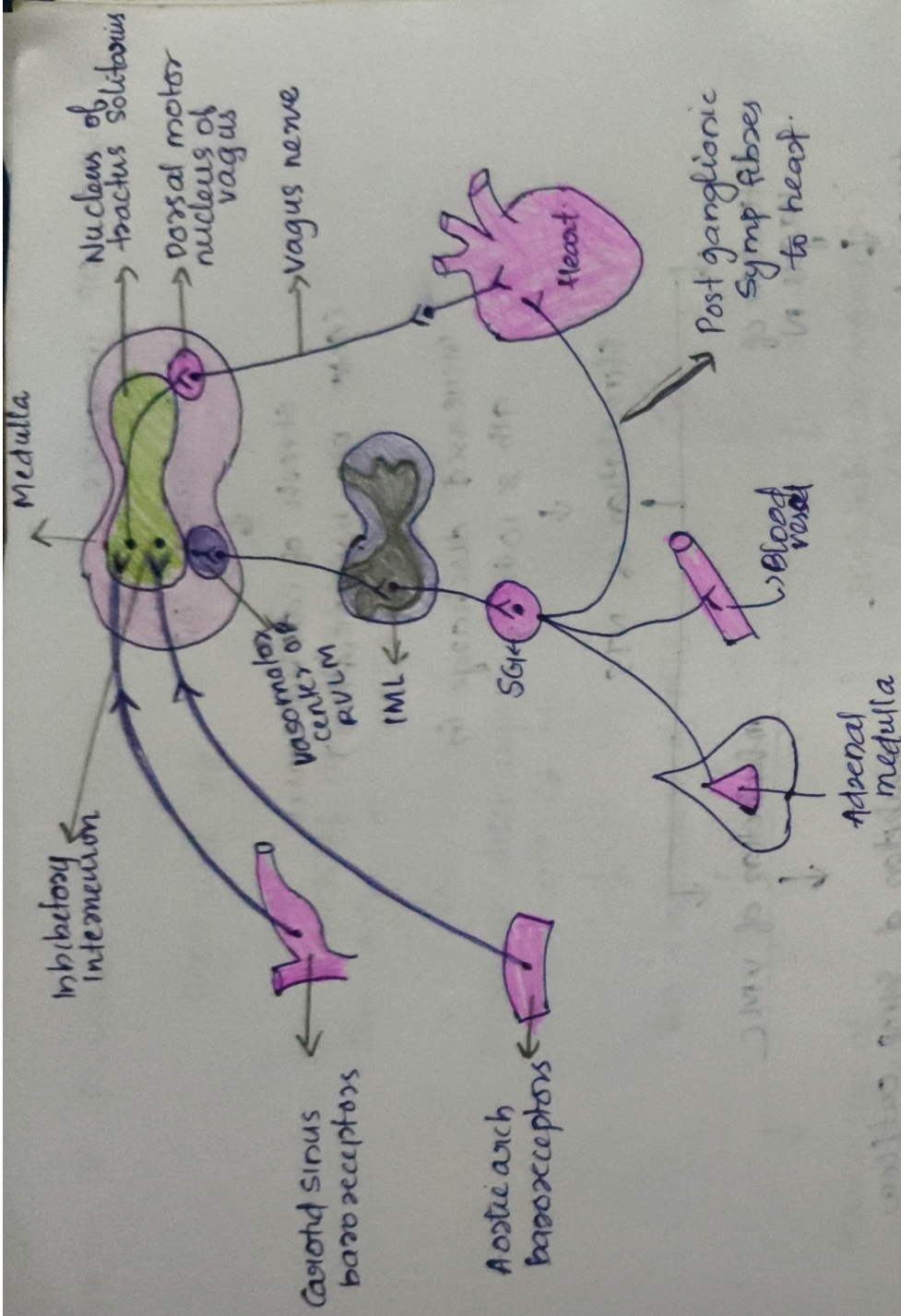


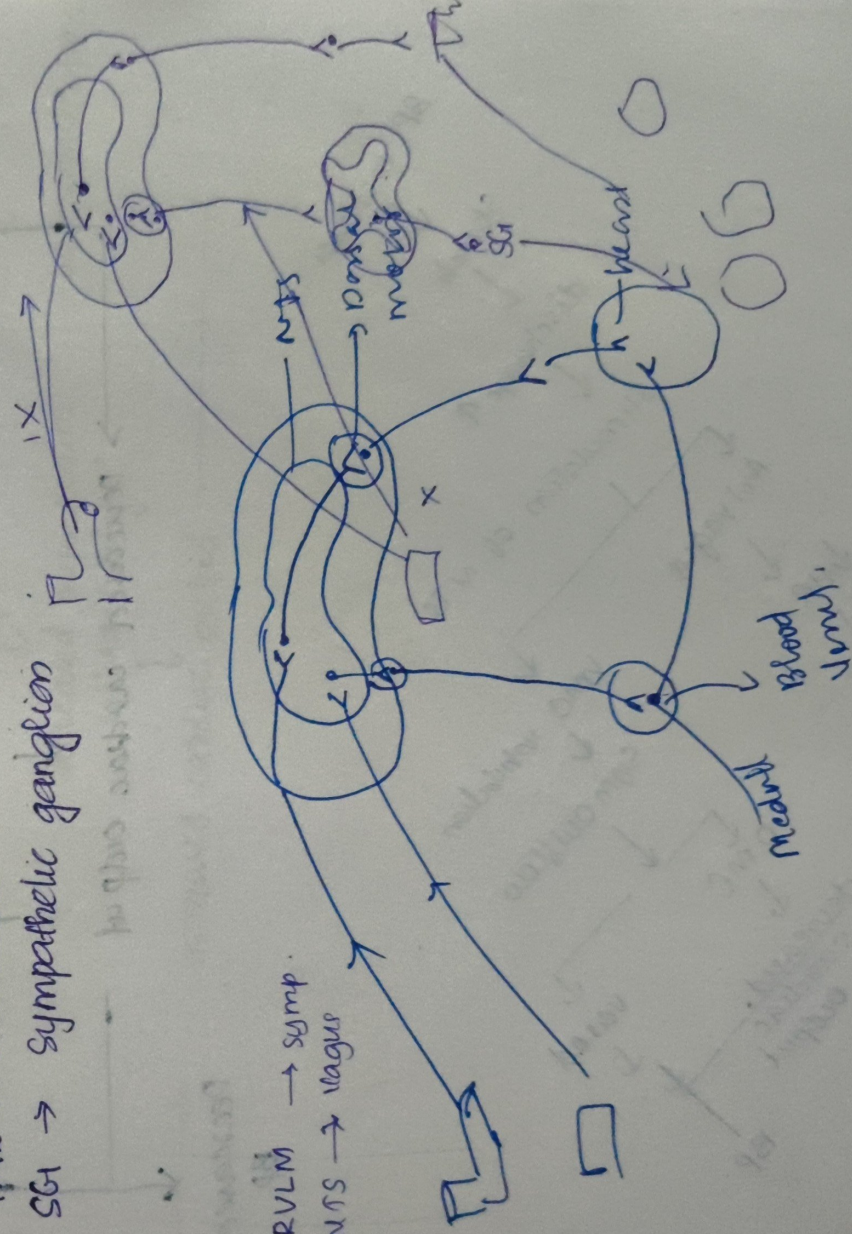
fig: Innervation of heart & pathway for sinoaortic reflex / baroreceptor reflex.

IML $\rightarrow$ Intermedio lateral horn cells

SGH $\rightarrow$ Sympathetic ganglion

Rec -

Aff. cent - Vaso CIC $\rightarrow$ NTS $\rightarrow$ vagus eff. - vagus sy. $\rightarrow$ Symp.



When BP increases

Increased BP



stretch of carotid sinuses
(aortic baroreceptors)



increased discharge in

9th & 10th CN



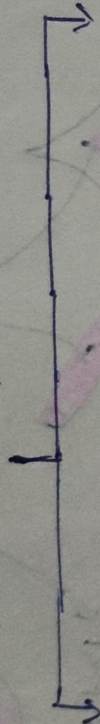
stimulation of NTS



Activation of
vagus N



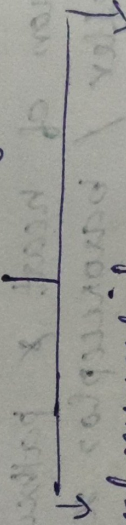
decreased
heart rate



inhibition of VMC



inhibition of symp outflow

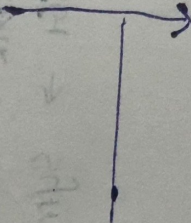


decreased myocardial
contractility

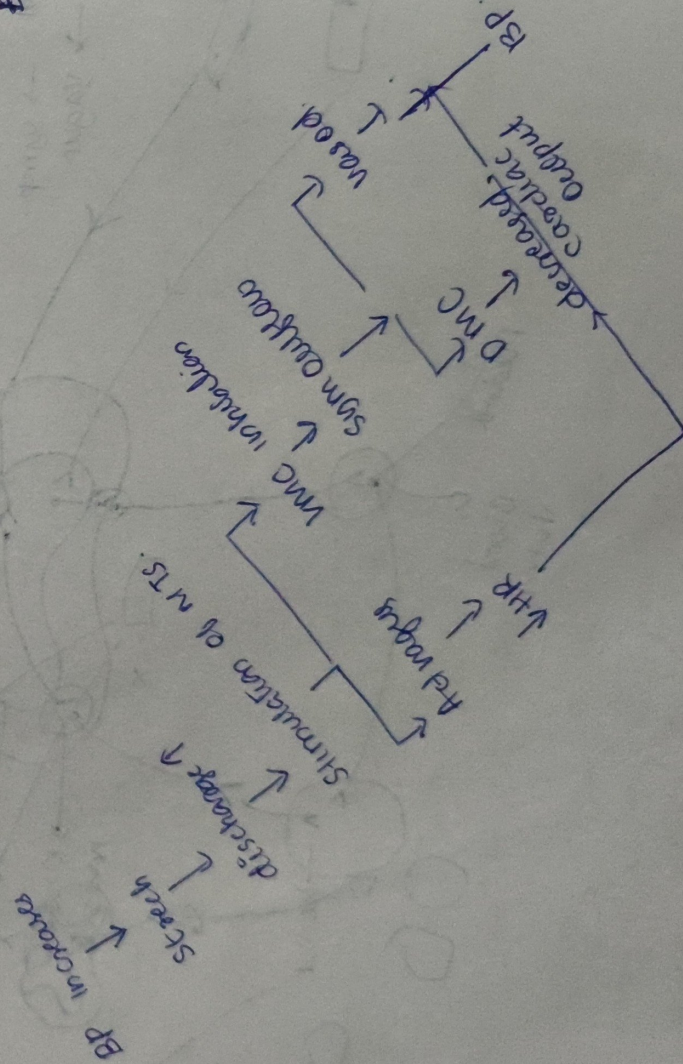


decreased cardiac output

Vasodilation



Decreased BP



When BP decreases

↓
Decreased BP

↓
Less stretch of carotid sinus
(less action of baroreceptors)

↓
Decreased discharge in
Atr & Vagus CN

↓
Less stimulation of NTS

↓
Inhibition of
vagus nerve

↓
Increased
heart rate

↓
Disinhibition (Activation)
of VMC

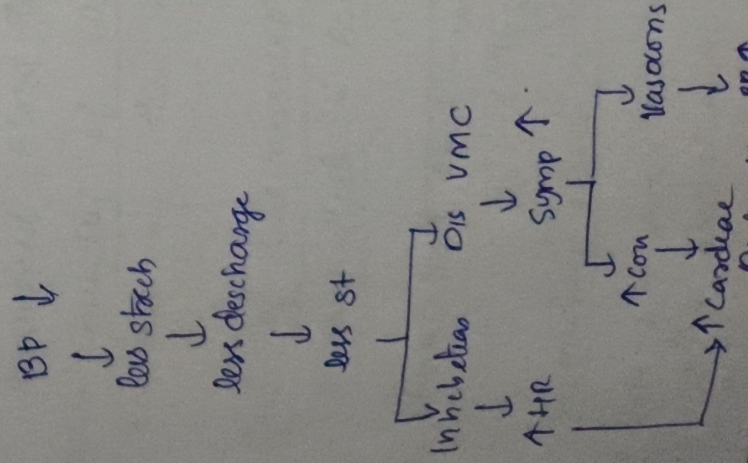
↓
Stimulation of sym outflow

↓
Increased myocardial
contractility

↓
Increased cardiac output

↓
Vasoconstriction

↓
Increased
BP



• When BP falls $\rightarrow$ Baroreceptor reflex operates within few sec $\rightarrow$ correct it

- Hence, life saving in acute hypertension & hemorrhage
- correct 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 of Marey's law (+HR) is inversely proportional to BP (Reverse is not true)

