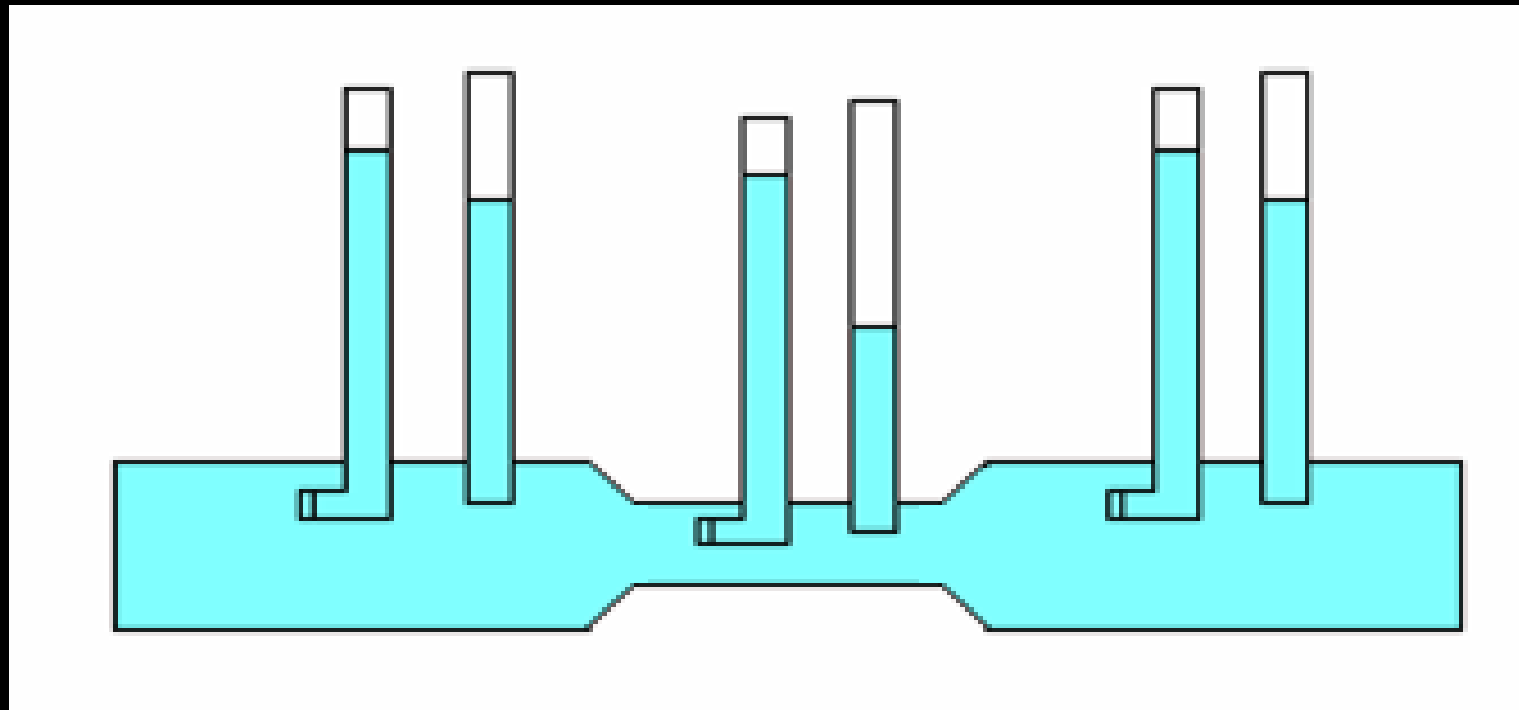


CARDIO-VASCULAR SYSTEM

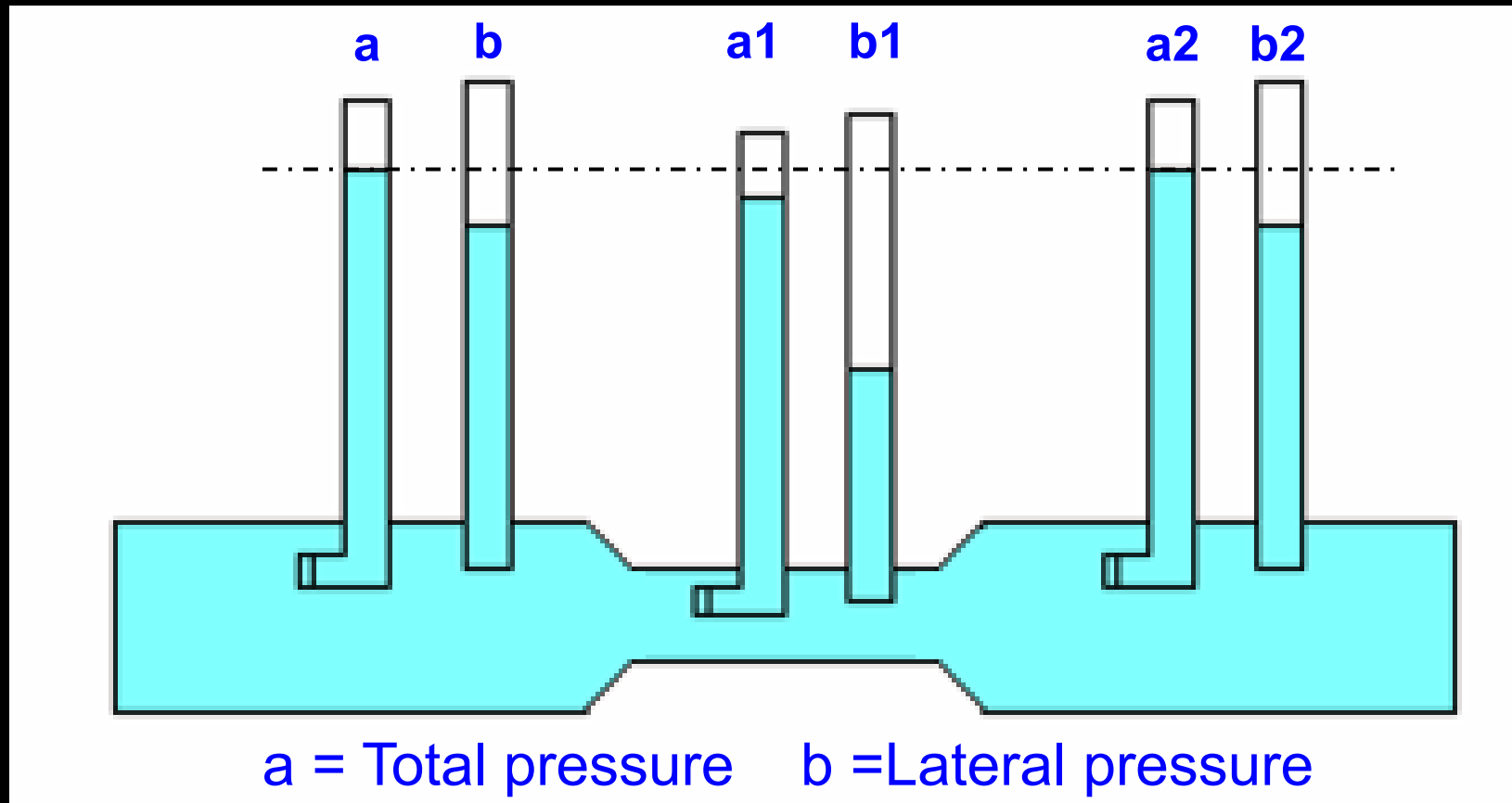
- Dr. Chetna Ramanuj



BLOOD PRESSURE:

It is the lateral pressure exerted by the flowing blood on the walls of the vessels

Bernoulli's Principle



- ❖ If the velocity of flow is low than the magnitude of lateral pressure and total pressure are almost same
- ❖ If the velocity of flow is high than the lateral pressure exerted by flowing fluid is much less than total pressure

Systolic blood pressure :

The maximum arterial pressure during systole

Occurs during ventricular ejection

Significance:

It is a function of cardiac output, so it represents the extent of work done by the heart

Normal in young adult is : 120 mm Hg

(Range: 105 - 135 mm Hg)

Diastolic blood pressure :

The minimum arterial pressure during diastole

Occurs just before the onset of ventricular ejection

Significance:

It is a function of total peripheral resistance, so it indicates the constant load against which heart has to work.

Normal in young adult is : 80 mm Hg

(Range: 60 - 90 mm Hg)

Pulse pressure :

It is the arithmetic difference between the systolic and diastolic blood pressures.

Systolic BP (- Minus) Diastolic BP

Normally it is 40 mm Hg

Significance:

High pulse pressure indicates systolic hypertension

Mean Arterial Pressure (MAP):

It is the average of all pressure measured millisecond by millisecond throughout the cardiac cycle

Practically, it is roughly equal to Diastolic pressure (DP) plus 1/3rd of pulse pressure (PP).

$$\text{MAP} = \text{DP} + 1/3 \text{ PP}$$

Normal value is 93 mm Hg (Range : 90 – 100 mm Hg)

DETERMINANTS OF THE ARTERIAL BLOOD PRESSURE

The BP is a function of the product of CO and Total Peripheral Resistance (PR)

$$\text{Arterial BP} = \text{CO} \times \text{PR}$$

So, some of the important determinants of BP are :

1. Heart rate:

2. Stroke volume:

3. Arterial blood volume:

MECHANISM

Increase in heart rate



Increase in CO



Increased BP

DETERMINANTS OF THE ARTERIAL BLOOD PRESSURE

4. Arterial elastic constant :

it refers to the stiffness of the arterial system which progressively increases from birth until death.

↑↑ in arterial elastic constant : ↑↑ pressure during systole

5. Peripheral resistance :

↑↑ PR = ↑↑ BP and ↓↓ PR = ↓↓ BP

PHYSIOLOGICAL FACTORS AFFECTING BLOOD PRESSURE

1. AGE : Both SBP and DBP rises with age

2. GENDER :

Before menopause, females have lower BP than males of the same age group

After menopause, females have higher BP than males of the same age group

3. EFFECTS OF MEALS : SBP increases about 4 – 6 mmHg after meals

PHYSIOLOGICAL FACTORS AFFECTING BLOOD PRESSURE

4. **EMOTIONS** : Increased sympathetic activity leads to increase in SBP
5. **TEMPERATURE** : Exposure to cold temp. produces rise in the BP
6. **DIURNAL VARIATION** : SBP shows variation of about 6 – 10 mmHg. It is lower in the morning
7. **EXERCISE** : SBP rises and DBP falls in muscular exercise

PHYSIOLOGICAL FACTORS AFFECTING BLOOD PRESSURE

8. EFFECT OF CHANGE IN POSTURE :

Immediately on standing there occurs pooling of blood in dependent parts leading to ↓ CO and thereby fall in SBP

9. SLEEP : in relaxed state, there is a fall in BP

10. BODY BUILT : SBP is slightly higher in Obese people

PATHOLOGICAL FACTORS AFFECTING BLOOD PRESSURE

1. HYPERTENSION

2. HYPOTENSION

MEASUREMENT OF BLOOD PRESSURE

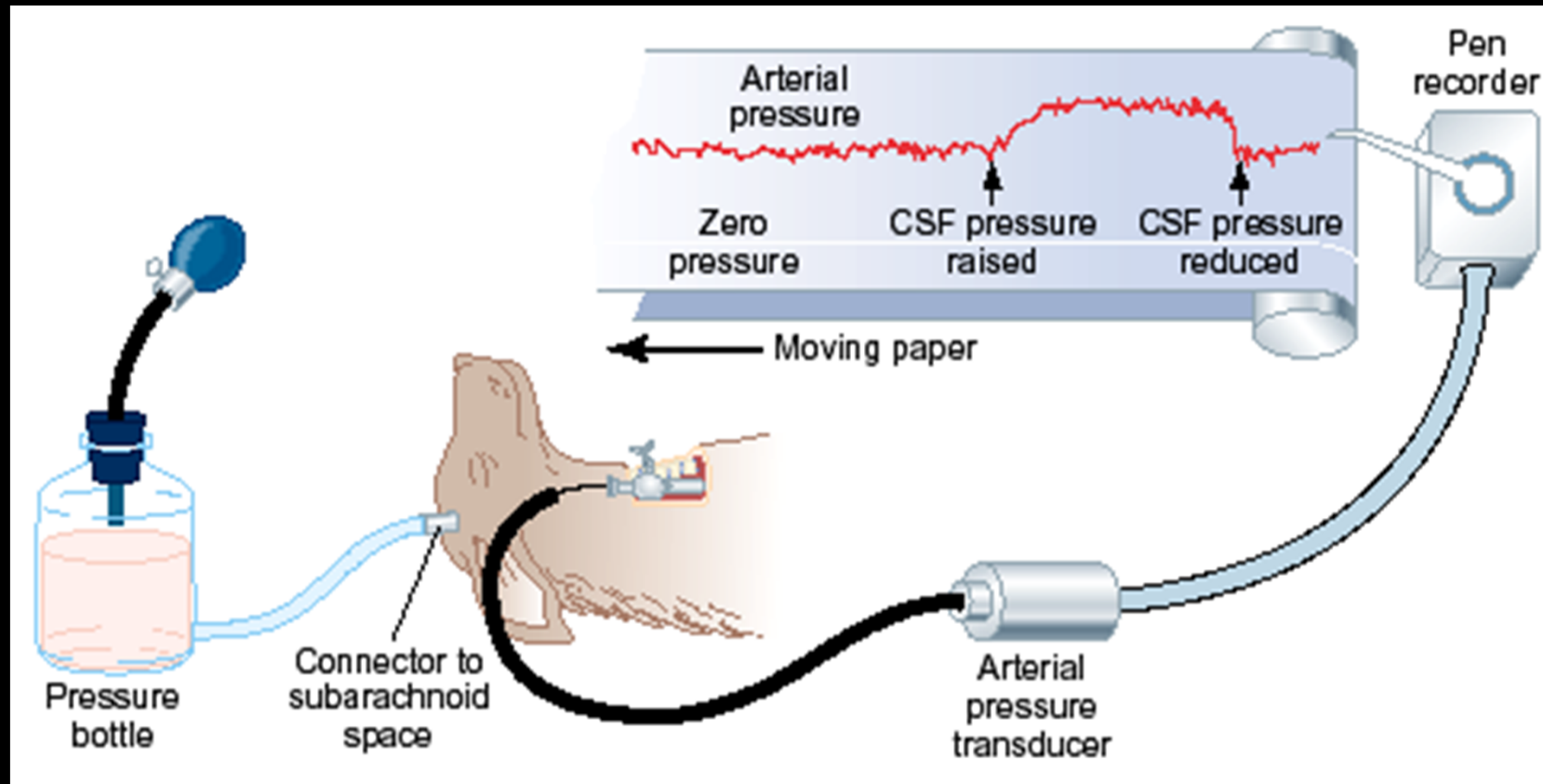
DIRECT : By a cannula or T-tube

INDIRECT : Sphygmomanometer

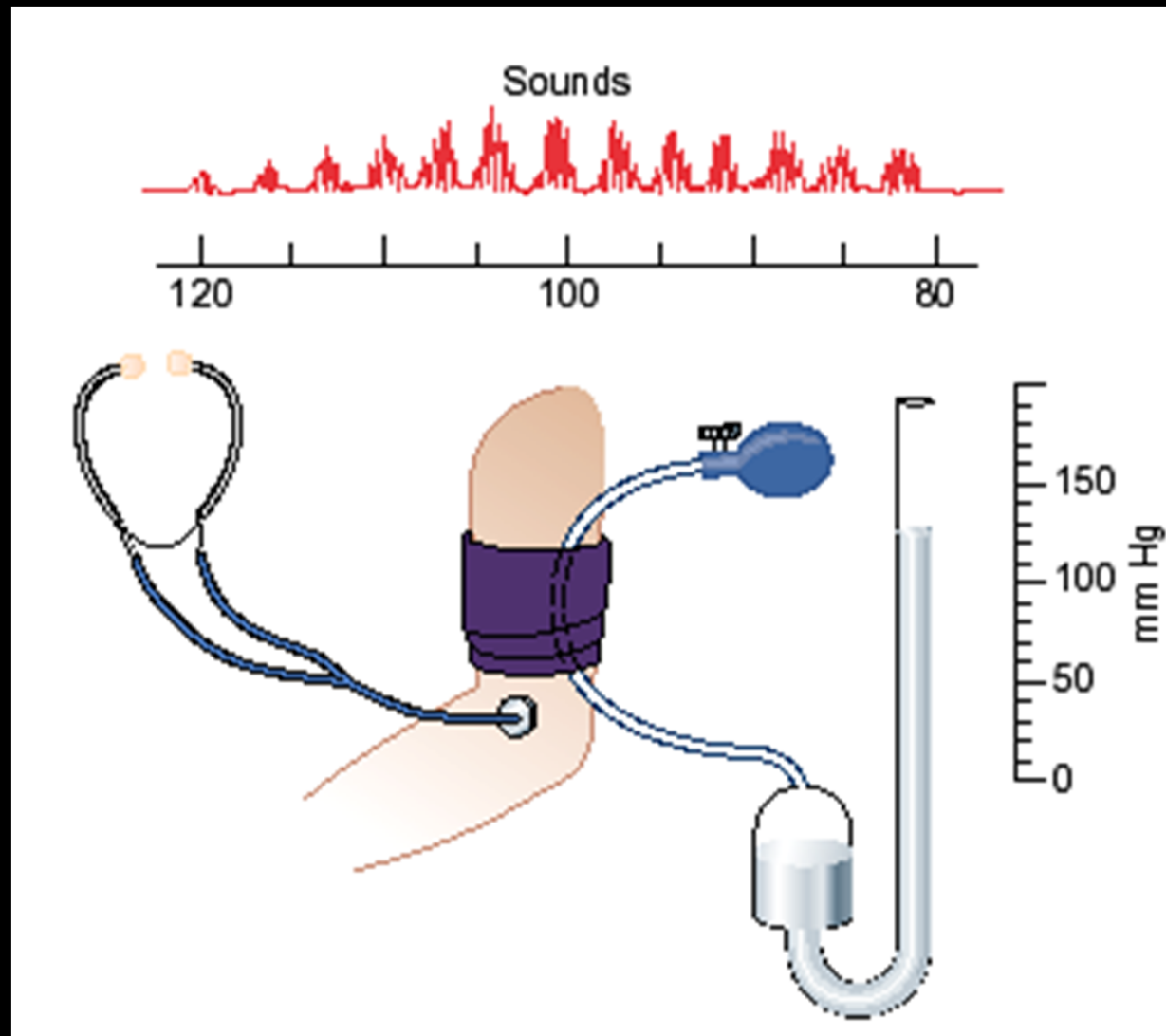
DIRECT MEASUREMENT

Mercury manometer: pressure is recorded on the kymograph

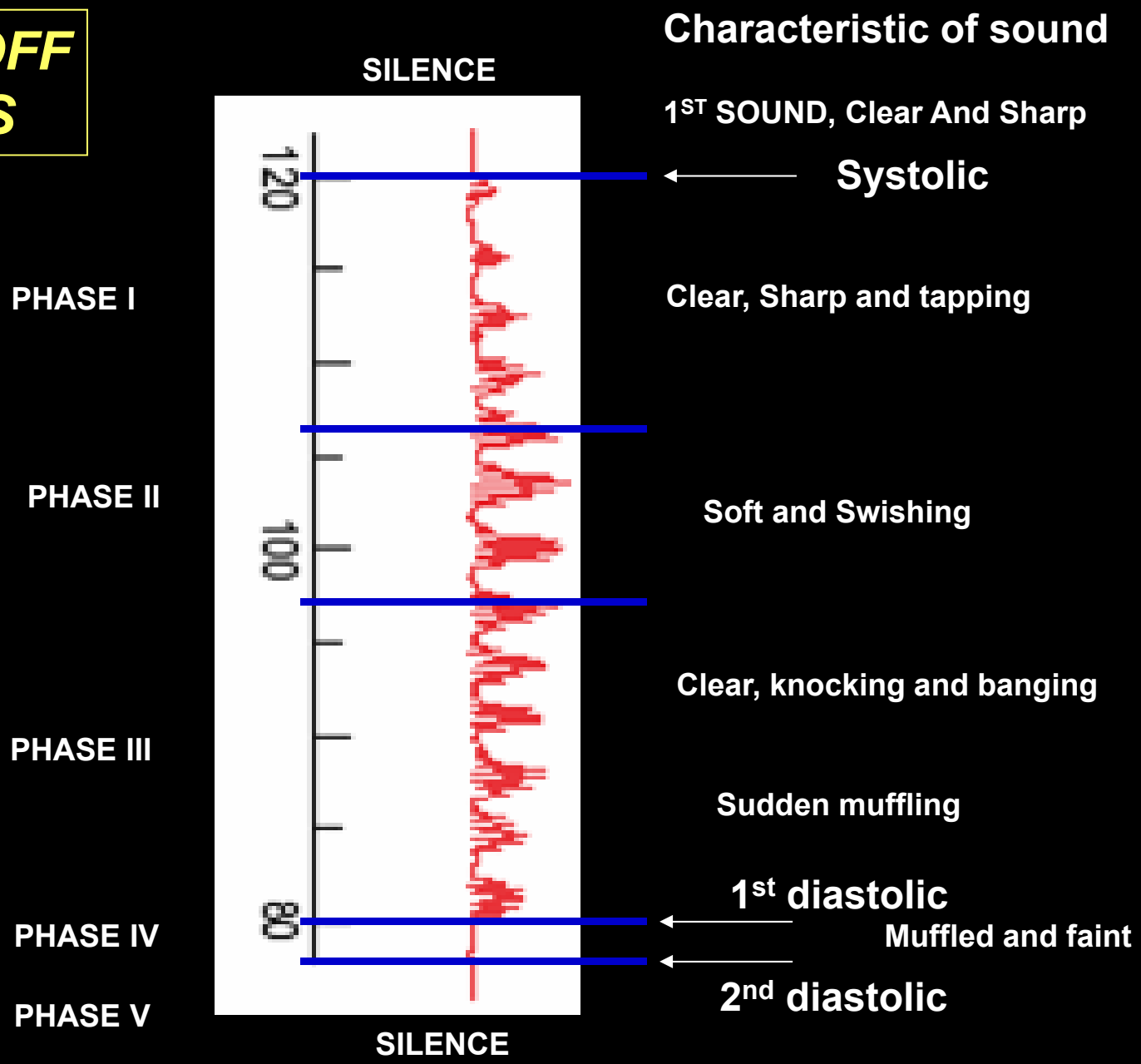
Pressure transducer: pressure is recorded on the polyrite

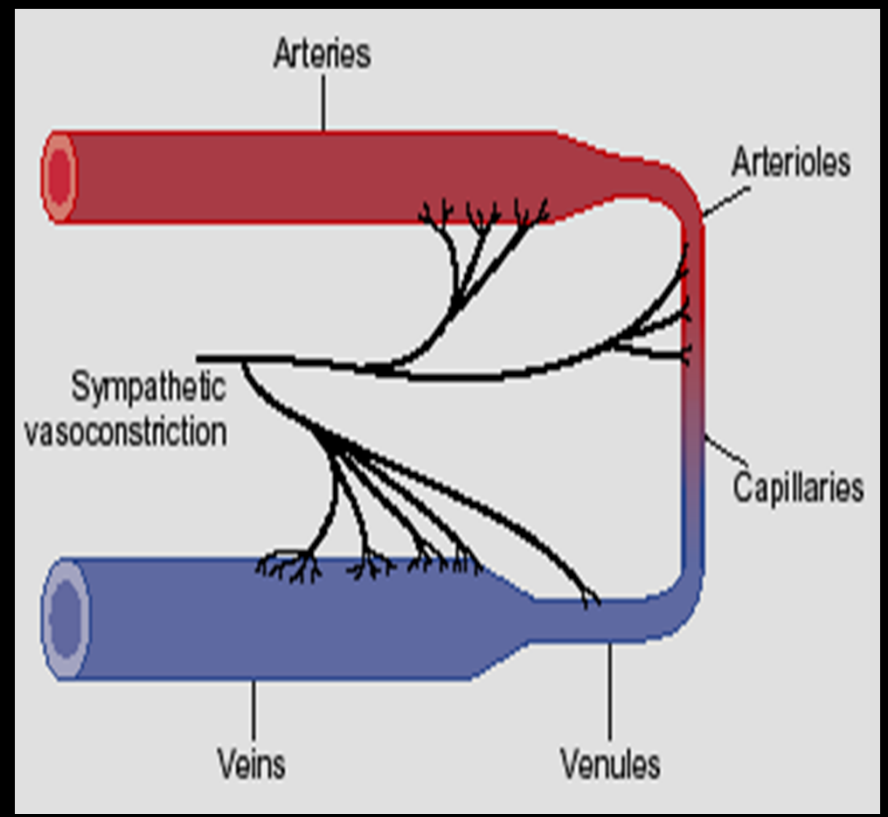
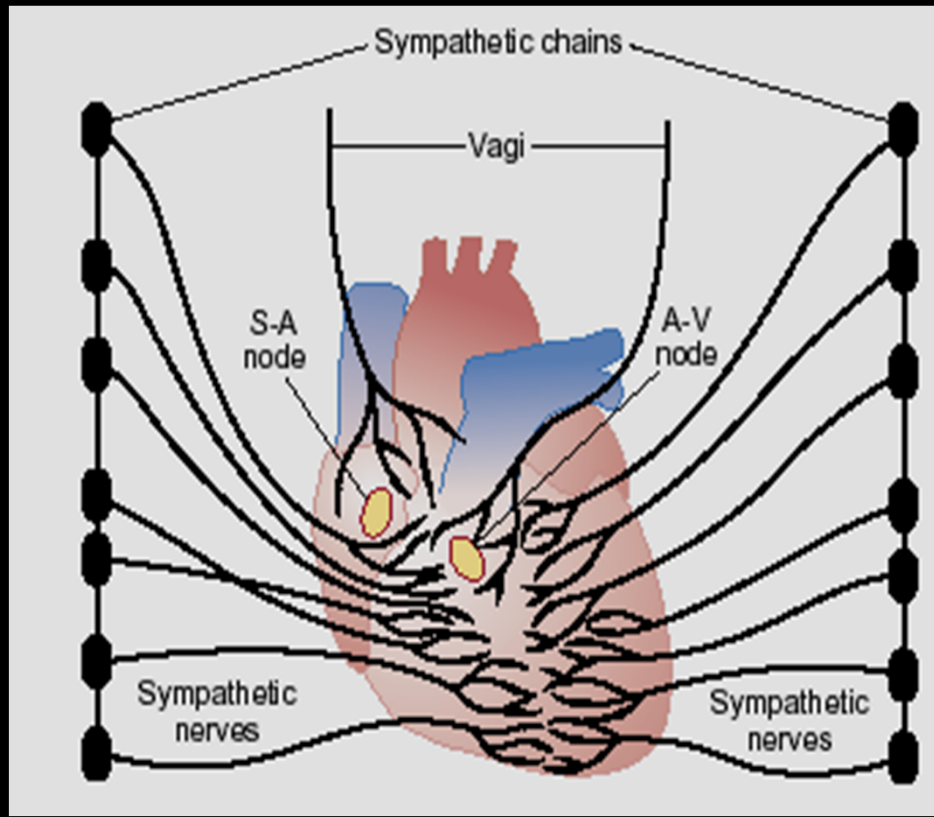


SPYGMOMANOMETER



KOROTKOFF SOUNDS





Organ	Parasympathetic	Sympathetic
<u>Heart</u> -SA -Atria -A-V -Ventricles	<u>Decreases</u> heart rate contraction & conduction	<u>Increases</u> b1 - heart rate b1 - conduction & conductivity
Arterioles	dilatation only in face & external genitals	α- constriction of all vessels b2 - dilatation
Veins	<p style="text-align: center;">—</p>	alpha - constriction b2 - dilatation

VASOCONSTRICTOR FIBERS:

- sympathetic nerves : secrete noradrenalin

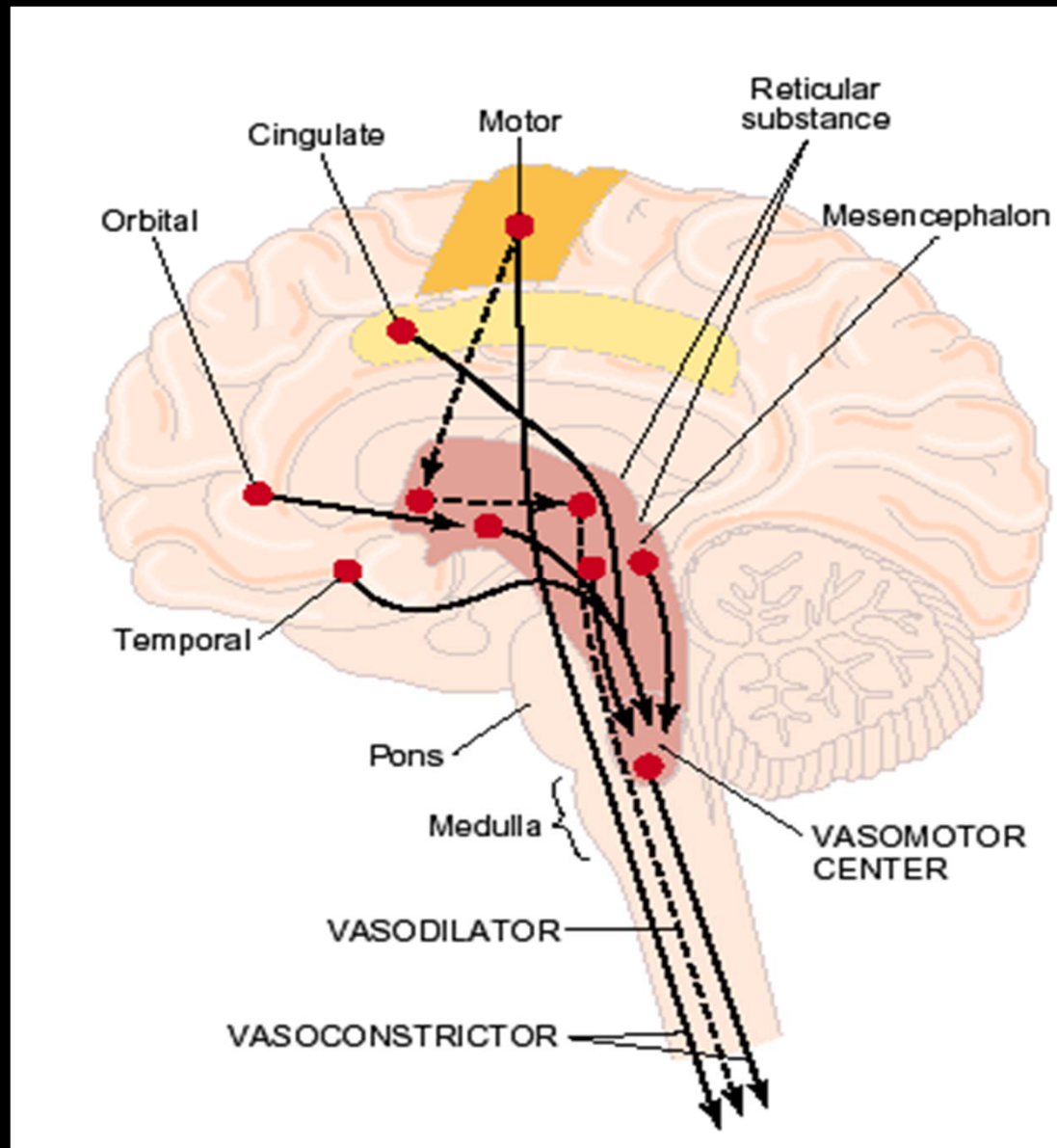
VASODILATOR FIBERS:

- parasympathetic : secrete Ach
- sympathetic vasodilator : secrete Ach.

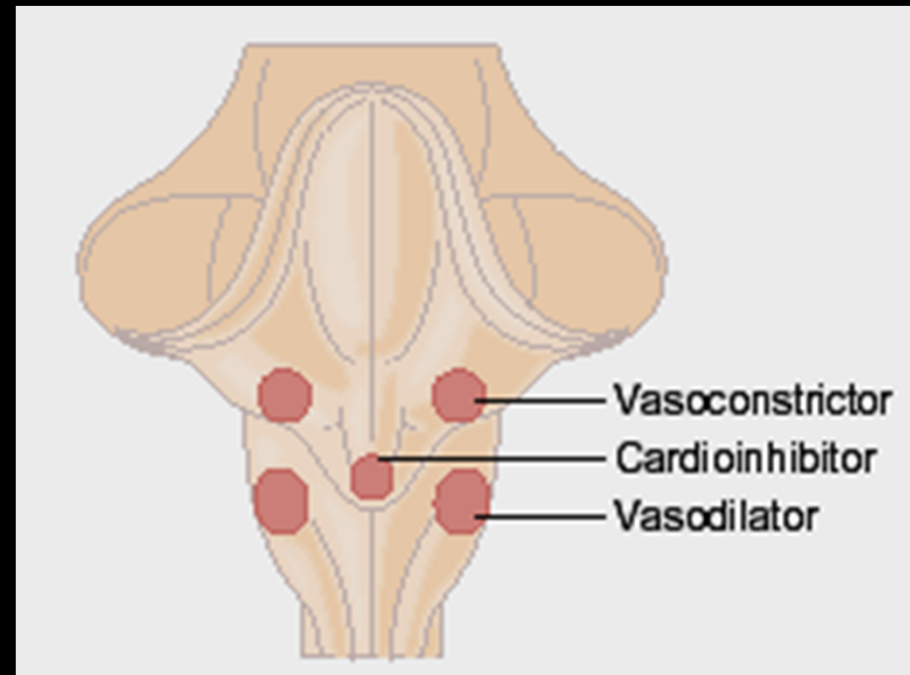
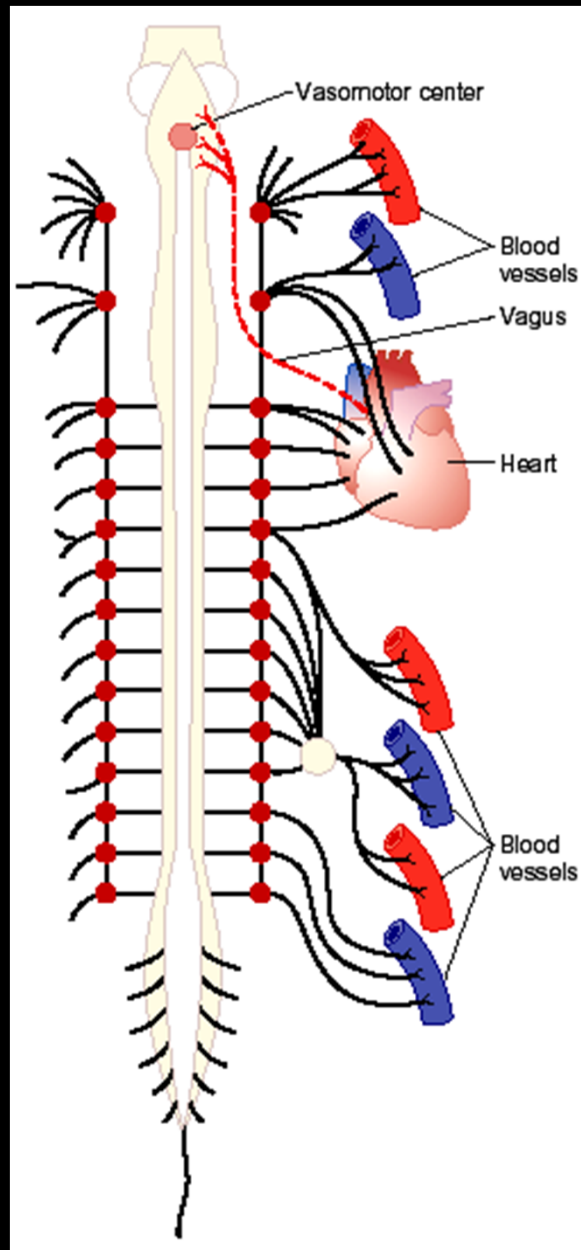
REGULATION OF BLOOD PRESSURE

1. RAPID BLOOD PRESSURE CONTROL MECHANISM
(Nervous regulating mechanism)
2. INTERMEDIATE BLOOD PRESSURE CONTROL
MECHANISM
3. LONG-TERM BLOOD PRESSURE CONTROL
MECHANISM

VARIOUS AREAS IN THE MEDULLA FOR CARDIO VASCULAR CENTRE



VARIOUS AREAS IN THE MEDULLA FOR CARDIO VASCULAR CENTRE



VASOMOTOR CENTRE

Structure of VMC

- 1) **Vasoconstrictor or pressor area or area C-I** : located B/L in the AL part of upper medulla.
- 2) **Vasodilator or depressor area or area A-I** : located B/L in AL part of lower half of medulla.
- 3) **Sensory area or A-2** : located B/L in NTS, in the PL part of medulla and lower pons.

Inputs of VMC

inhibitory inputs-

- from baroreceptors (thro 9 and 10 cranial nerves to NTS),
- lungs & parts of cerebral cortex and limbic system

VASOMOTOR CENTRE

Excitatory inputs-

- from chemoreceptors (thro 9 and 10 cranial nerves to NTS),
- pain receptors from skin and joints,
- parts of cerebral cortex and limbic system
- hypoxia and hypercapnia in brain (due to cerebral ischemia) is strong direct stimulus for VMC

Outputs of VMC

Stimulation of VMC causes stimulation of sympathetic and inhibition of parasympathetic nervous system (vagus) and vice versa.

1. RAPID BLOOD PRESSURE CONTROL MECHANISM (Nervous regulating mechanism)

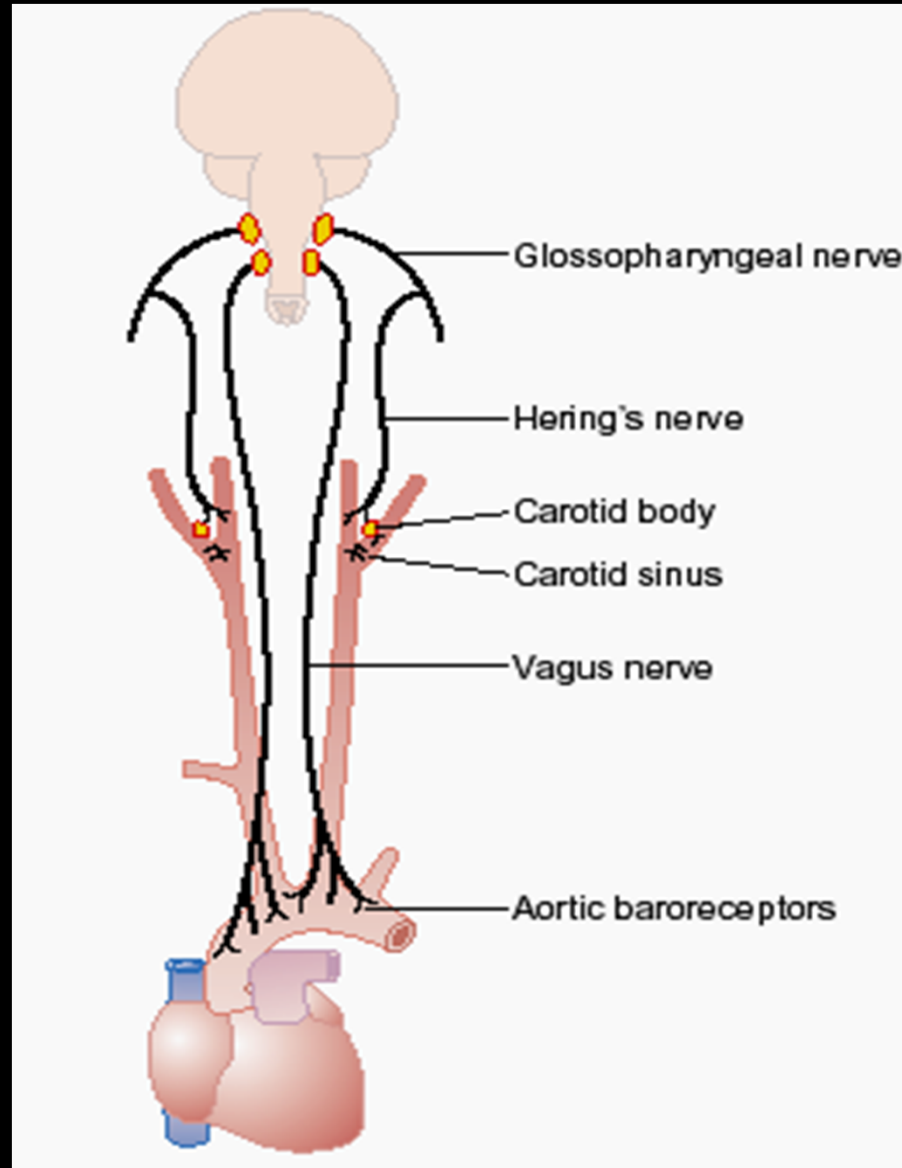
- **BARORECEPTOR REFLEXES**
- **CHEMORECEPTOR REFLEXES**
- **CENTRAL NERVOUS SYSTEM ISCHAMIC
RESPONSE**
- **CARDIO-PULMONARY REFLEX (ATRIAL LOW
PRESSURE REFLEX)**

1. RAPID BLOOD PRESSURE CONTROL MECHANISM (Nervous regulating mechanism)

SALIENT FEATURES:

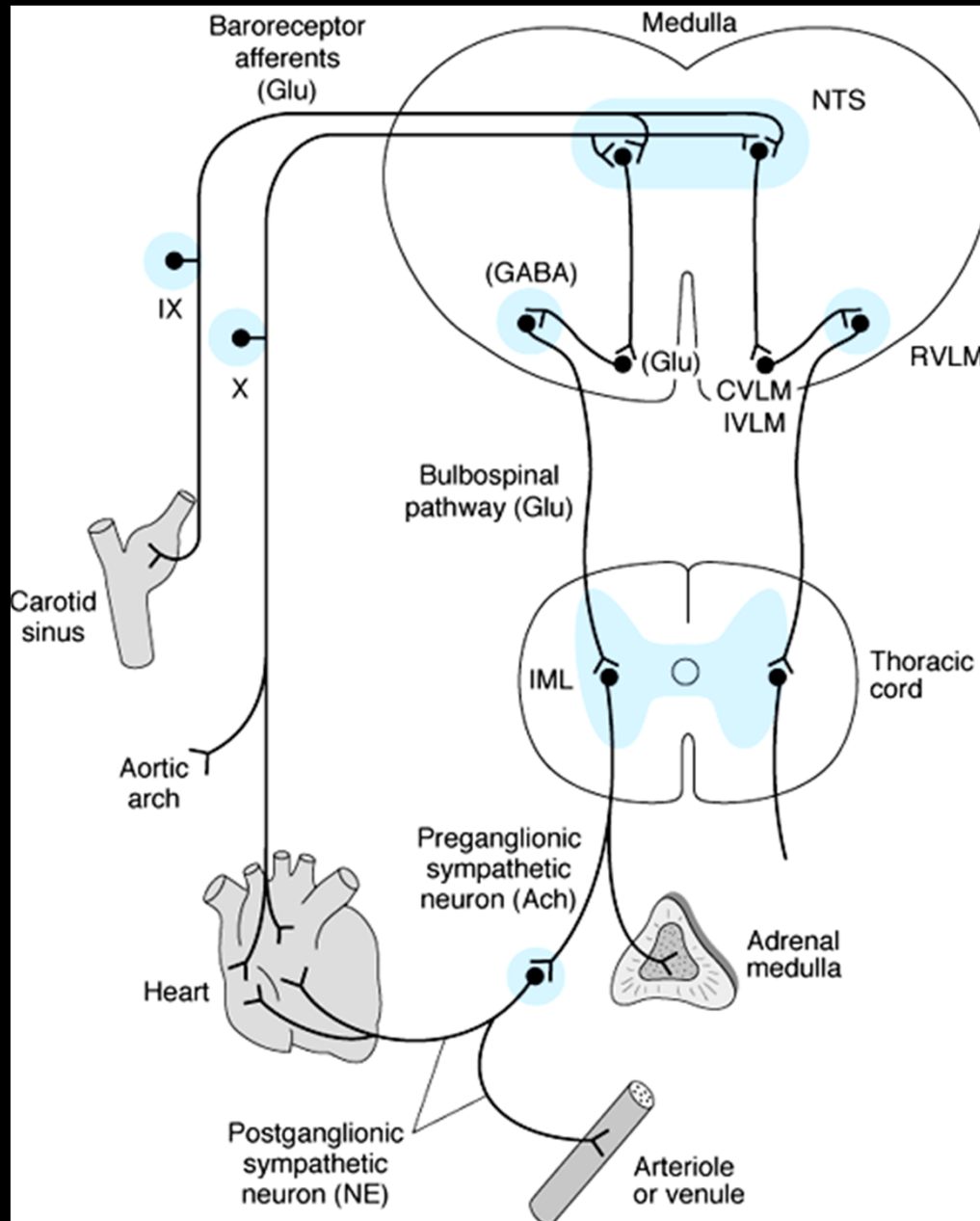
- These act very rapidly (within seconds to few minutes)
- These are short-term mechanisms
- Useful in preventing acute decreases in blood
pressure

BARORECEPTOR REFLEX MECHANISMS



Baroreceptors are located in the walls of the heart and major blood vessels

BARORECEPTOR REFLEX MECHANISMS



BARORECEPTOR REFLEX MECHANISMS

Baroreceptors are stimulated by :

↑↑ in arterial BP

↑↑ in carotid sinus perfusion pressure

Ligation of ICA

Stimulation of Sino-Aortic nerve

Decrease in Baroreceptors activity :

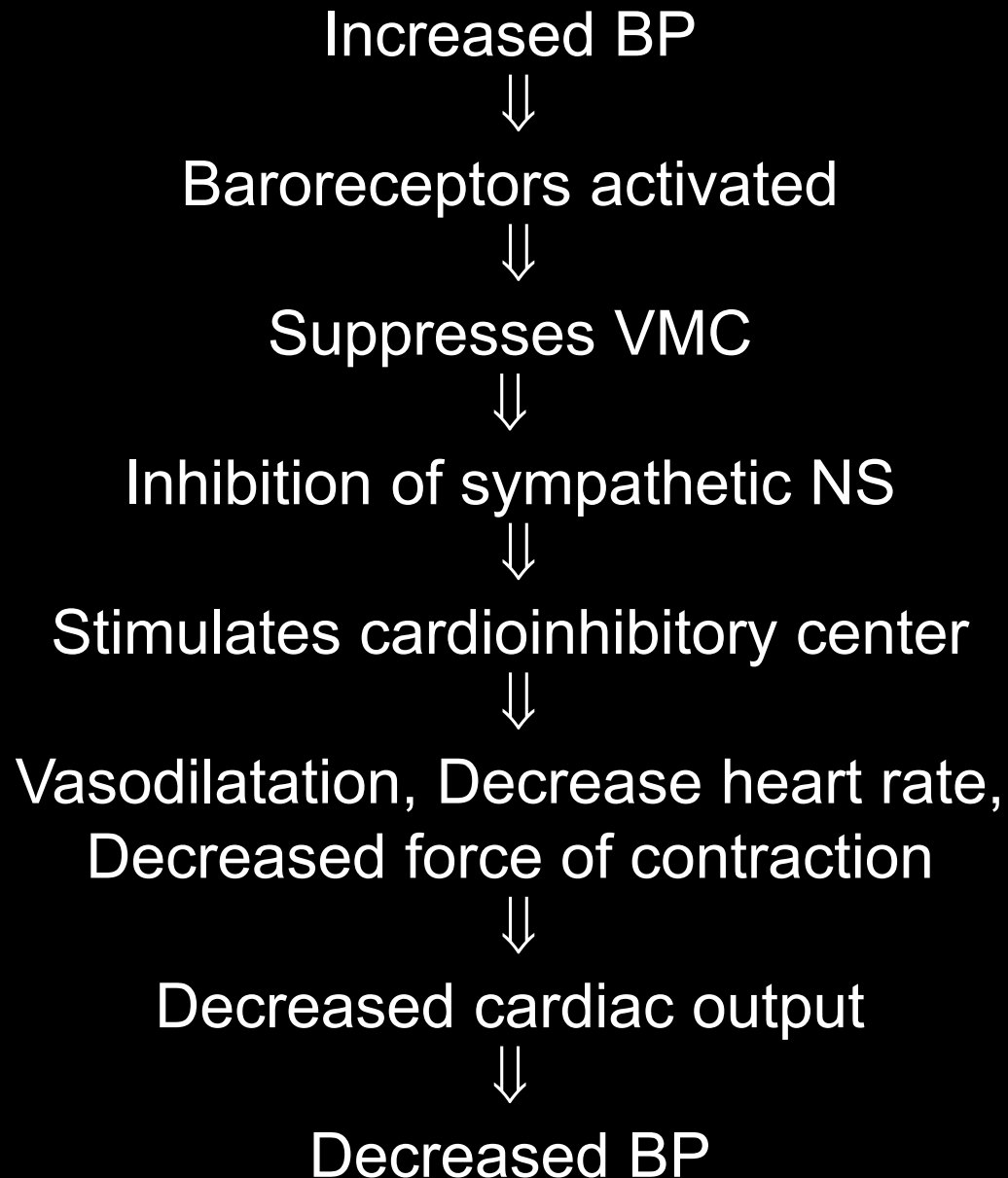
↓↓ in arterial BP

↓↓ in carotid sinus perfusion pressure

Ligation of Common Carotid Artery

Section of Sino-Aortic nerve

BARORECEPTOR REFLEX MECHANISMS



BARORECEPTOR REFLEX MECHANISMS

Characteristics-

- Operates between 60 to 160mmHg (best at 100)
- Respond more to Change in BP
- Act as buffer system (9 & 10 nerves called buffer nerves)
- Correct postural & day to day variation
- Reset in two three days (no role in long term regulation)
- Respond to pulse pressure but most sensitive to MBP

CHEMORECEPTOR REFLEXES

Chemoreceptors are chemosensitive cells that respond to following changes in blood:

- ❖ Oxygen lack (decreased PO_2)
- ❖ Carbon Dioxide excess (increased PCO_2)
- ❖ Hydrogen ion excess (decreased pH)

Location of chemoreceptors:

- Carotid bodies
- Aortic bodies

Functions of chemoreceptors

1. Respiratory control
2. Cardiovascular control

- In hypoxia, there is increased chemoreceptor discharge



Hyperventilation, excitation of VMC (vasomotor centre)



Increase in arterial blood pressure

Functions of chemoreceptors

2. Cardiovascular control (continued....)

- In hypotension due to severe hemorrhage



Increased chemoreceptor discharge



Increase in arterial blood pressure

CENTRAL NERVOUS SYSTEM ISCHAMIC RESPONSE

When blood pressure falls below 60 mmHg, the blood flow to the vasomotor area is decreased enough to cause CNS

ischemia



CO₂, lactic acid accumulated near VMC



Excitation of VMC



Vasoconstriction



Increase in blood pressure

CARDIO-PULMONARY REFLEX (ATRIAL LOW PRESSURE REFLEX)

They play role in minimizing the effect of blood volume on BP (via ADH & renal a arteriolar dilatation) & complement baroreceptor

SUMMARY OF THE REFLEXES

Nervous Reflexes	Stimulus & receptor	Pathway & center	Response
<u>baroreceptors reflex</u> (100 mmHg)	stretch (↑ BP) / spray nerve endings at	9 & 10 CN to NTS to VMC	-VMC (SI & PS)
<u>cardio-pulmonary reflex</u> (atrial low pressure R)	stretch (↑ B volume) / stretch R at	10 CN to NTS to VMC	- VMC (renal A A dilatation & + ADH)
<u>chemo receptor reflex</u> (80 mmHg)	- O ₂ , + CO ₂ , + H / C & A body	9 & 10 CN to NTS to VMC	+ VMC (SS & PI)
<u>CNS-Ischemic reflex</u> (60 mmHg)	CNS-Ischemic (+ CO ₂ & L acid at VMC	directly stimulate VMC	++ VMC (SS & PI)

CLINICAL HYPERTENSION (HT)

What is HT:

3 Consecutive reading more than 140/90 mmHg in adults

Causes:

- Essential (90%)
- Secondary (Renal, Endocrine, CNS disease, Oral Contraceptive pills)

CLINICAL HYPERTENSION (HT)

Complications:

Hypertension if left untreated can cause following lethal effects

- CHD, heart attack, heart failure
- Brain hemorrhages, infarcts
- Hemorrhages in kidneys leading to renal failure, uremia and death

CLINICAL HYPERTENSION (HT)

Treatment:

- life style modification

Drugs (ABCD)-

A = Which block action of renin-angiotensin,
e.g. **A**CE inhibitors

B = Which ↓ activity of sympathetic nervous system,
e.g. **B**eta blockers.

C = Drugs paralyzing the smooth muscle of renal
vasculature, e.g. **C**alcium channel blockers.

D = Which ↓ tubular absorption of salt and water,
e.g. **D**iuretics

DISCLAIMER

- All figures are taken from Guyton and Hall Textbook of Medical Physiology, 12th Edition.