

The Arterial Blood Pressure

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Arterial Blood Pressure (BP)

- Definition: It is the lateral pressure of the blood on the walls of aorta & arterial blood vessels
- Measured in (mmHg).
- Pressure inside big arteries (aorta & big vessels).
- It has 2 components:
 - **Systolic** ... (= max press reached) = 110-130 mmHg.
 - **Diastolic** ... (= min press reached) = 70-90 mmHg.
- **In normal adult = 120/80 mmHg.**

Definitions

- **SYSTOLIC BLOOD PRESSURE** (systolic pressure):
 - It is the **maximum pressure** exerted in the arteries **during systole** of heart.
 - *Normal systolic pressure:* (90 mm Hg to 140 mm Hg). Mainly=120 mm Hg.
- **DIASTOLIC BLOOD PRESSURE** (diastolic pressure):
 - It is the **minimum pressure** exerted in the arteries **during diastole** of heart.
 - *Normal diastolic pressure:* (60 mm Hg to 80 mm Hg). Mainly = 80 mm Hg

Definitions

- **PULSE PRESSURE**

- it is the difference between the systolic pressure and diastolic pressure.
- *Normal pulse pressure: 40 mm Hg (120 – 80 = 40).*

- **MEAN ARTERIAL BLOOD PRESSURE**

- MABP = Diastolic pressure + $\frac{1}{3}$ of pulse pressure

PHYSIOLOGICAL VARIATIONS in ABP

1. Age

⇒ Arterial blood pressure increases as age advances.

2. *Sex*

- ⇒ In females, up to the period of menopause, arterial pressure is 5 mm Hg, less than in males of same age.
- ⇒ After menopause, the pressure in females becomes equal to that in males of same age.

PHYSIOLOGICAL VARIATIONS in ABP

3. Body Built

⇒ Pressure is more in obese persons than in lean persons.

4. Diurnal Variation

⇒ In early morning, the pressure is slightly low. It gradually increases and reaches the maximum at noon.

5. After Meals

⇒ Arterial blood pressure is increased for few hours after meals due to increase in cardiac output.

6. During Sleep

⇒ Usually, the pressure is reduced up to 15 to 20 mm Hg during deep sleep. However, it increases slightly during sleep associated with dreams.

PHYSIOLOGICAL VARIATIONS in ABP

7. Emotional Conditions

⇒ During excitement or anxiety, the blood pressure is increased due to release of adrenaline.

8. After Exercise

⇒ After moderate exercise, systolic pressure increases by 20 to 30 mm Hg above the basal level due to increase in rate and force of contraction and stroke volume.

⇒ Normally, diastolic pressure is not affected by moderate exercise. It is because, the diastolic pressure depends upon peripheral resistance, which isn't altered by moderate exercise.

⇒ After severe muscular exercise, systolic pressure rises by 40 to 50 mm Hg above the basal level. But, the diastolic pressure reduces because the peripheral resistance decreases in severe muscular exercise.

PATHOLOGICAL VARIATIONS in ABP

Pathological variations of arterial blood pressure are:

- Hypertension.
- hypotension.

Factors determining ABP

$$ABP = COP \times PR$$

$$R = \frac{8\eta l}{\pi r^4}$$

FACTORS MAINTAINING ARTERIAL BLOOD PRESSURE

A. Central factors: related to the heart

1. Cardiac output.
2. Heart rate.

B. Peripheral factors: related to blood vessels and blood itself

3. **Peripheral resistance:** Diameter of blood vessels and Viscosity of blood
4. Blood volume.
5. Venous return.

1. *Cardiac Output*

- Systolic pressure is **directly proportional** to cardiac output.
- Cardiac output increases in muscular exercise, emotional conditions, etc. So in these conditions, the systolic pressure is increased.
- In conditions like myocardial infarction, the cardiac output decreases, resulting in fall in systolic pressure.

2. Heart Rate

- Moderate changes in heart rate do not affect arterial blood pressure much.
- However, marked alteration in the heart rate affects the blood pressure by altering cardiac output.

3. Peripheral Resistance

- Peripheral resistance is the important factor, which maintains diastolic pressure.
- Diastolic pressure is **directly proportional** to peripheral resistance.
- When peripheral resistance increases, diastolic pressure is increased and when peripheral resistance decreases, the diastolic pressure is decreased.

3. Peripheral Resistance

- ***Diameter of Blood Vessels***

Arterial blood pressure is **inversely proportional** to the diameter of blood vessel.

- ***Viscosity of Blood***

Arterial blood pressure is **directly proportional** to the viscosity of blood.

4- Elasticity of the blood vessels

- Blood pressure is **inversely proportional** to the elasticity of blood vessels.
- Due to elastic property, the blood vessels are distensible and are able to maintain the pressure.
- When the elastic property is lost, the blood vessels become rigid (**arteriosclerosis**) and pressure increases as in old age. Deposition of cholesterol, fatty acids and calcium ions produce rigidity of blood vessels.

5. Blood Volume

- Blood pressure is **directly proportional** to blood volume.
- Blood volume maintains the blood pressure through the venous return and cardiac output.
- If the blood volume increases, there is an increase in venous return and cardiac output, resulting in elevation of blood pressure

5. Venous Return

- Blood pressure is **directly proportional** to venous return.
- When venous return increases, there is an increase in ventricular filling and cardiac output, resulting in elevation of arterial blood pressure.

REGULATION OF ARTERIAL BLOOD PRESSURE

- A. Nervous mechanism or short term regulatory mechanism
- B. Intermediate control of ABP
- C. Renal mechanism or long term regulatory mechanism
- D. Hormonal mechanism
- E. Local mechanism.

SHORT-TERM CONTROL OF ABP

1. CNS ischaemic response.
2. Baroreceptor reflex. discussed before
3. Chemoreceptor reflex. discussed before

Baroreceptor reflex

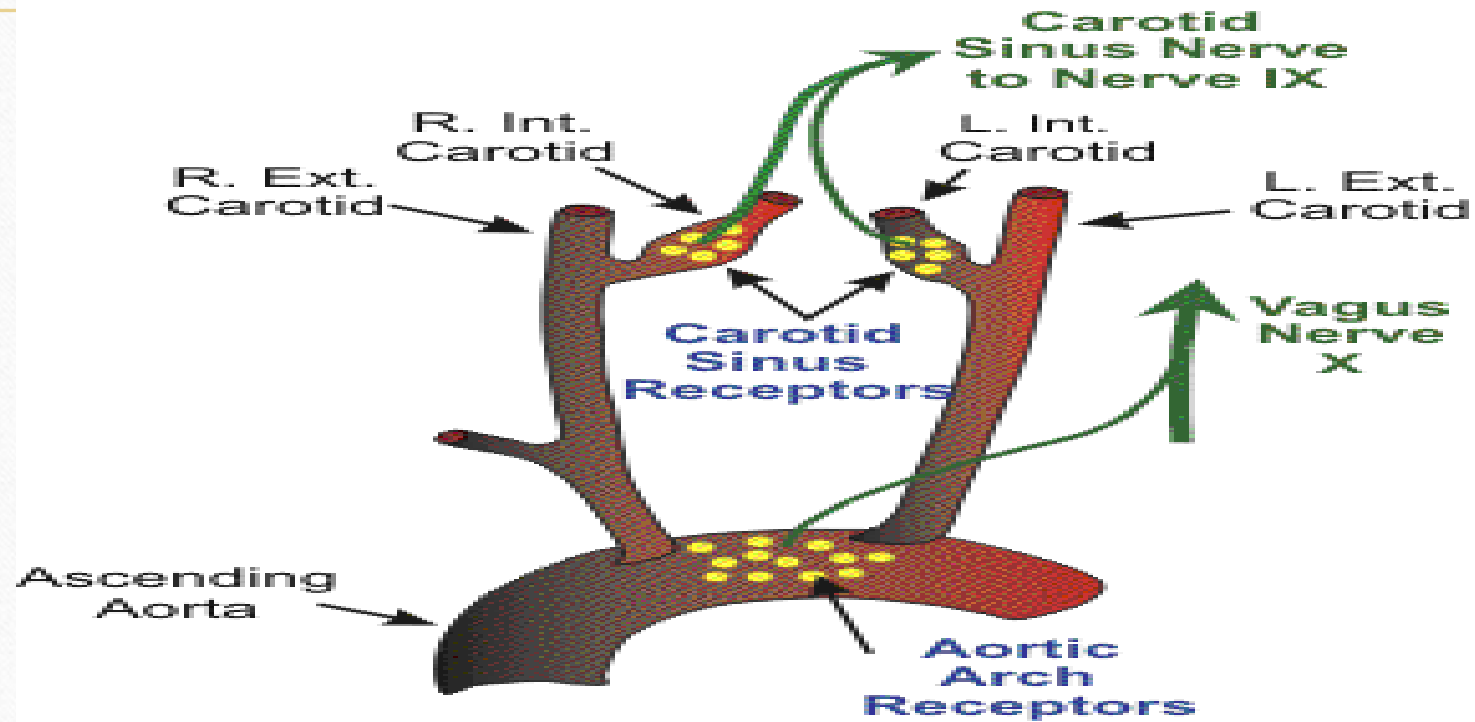
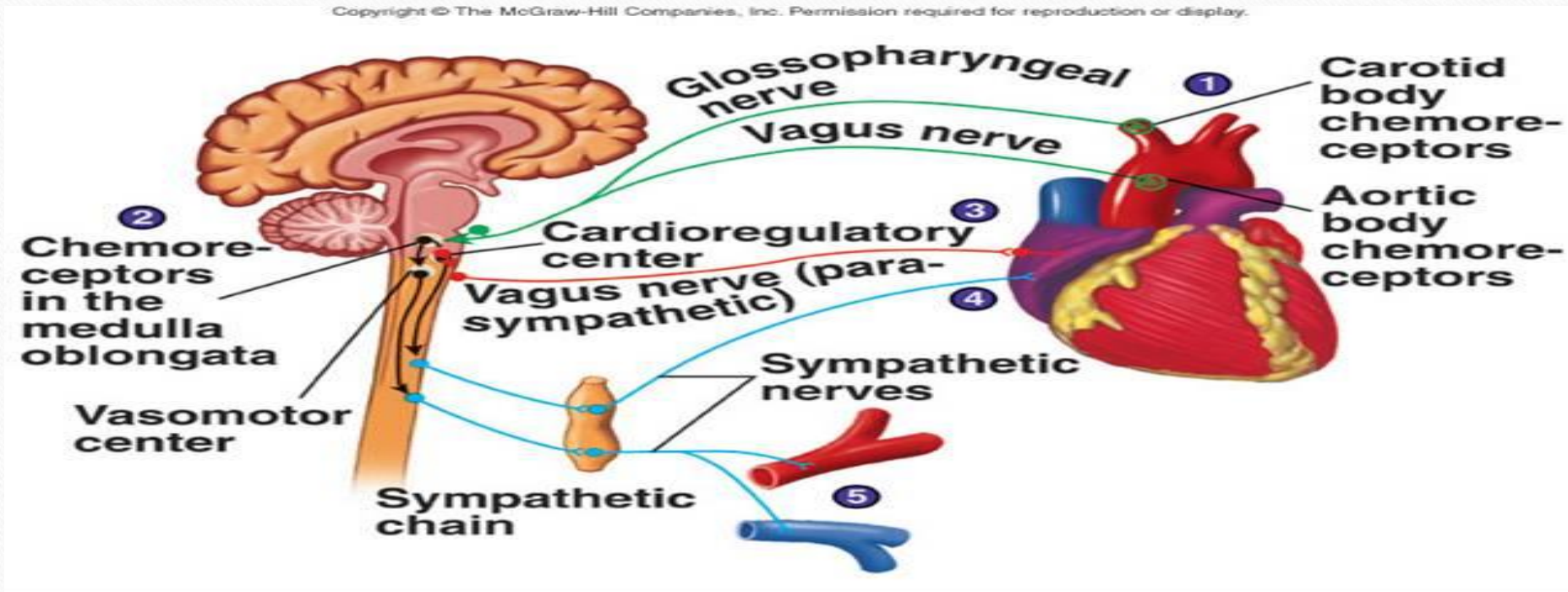


Figure 1. Location and innervation of arterial baroreceptors.

Chemoreceptor reflex



CNS ischemic response & CUSHING REFLEX

1- CNS ischemic response:

- When blood flow to the VMC ↓ markedly → cerebral ischemia → ABP ↑ to its maximum .
- It's an emergency control system that act rapidly & powerfully to prevent further ↓ in CBF & prevent brain damage (**the last ditch**).
- Activated when ABP falls below 50 mmHg & maximumly stimulated at 15-20mmHg.

CNS ischaemic response & CUSHING REFLEX

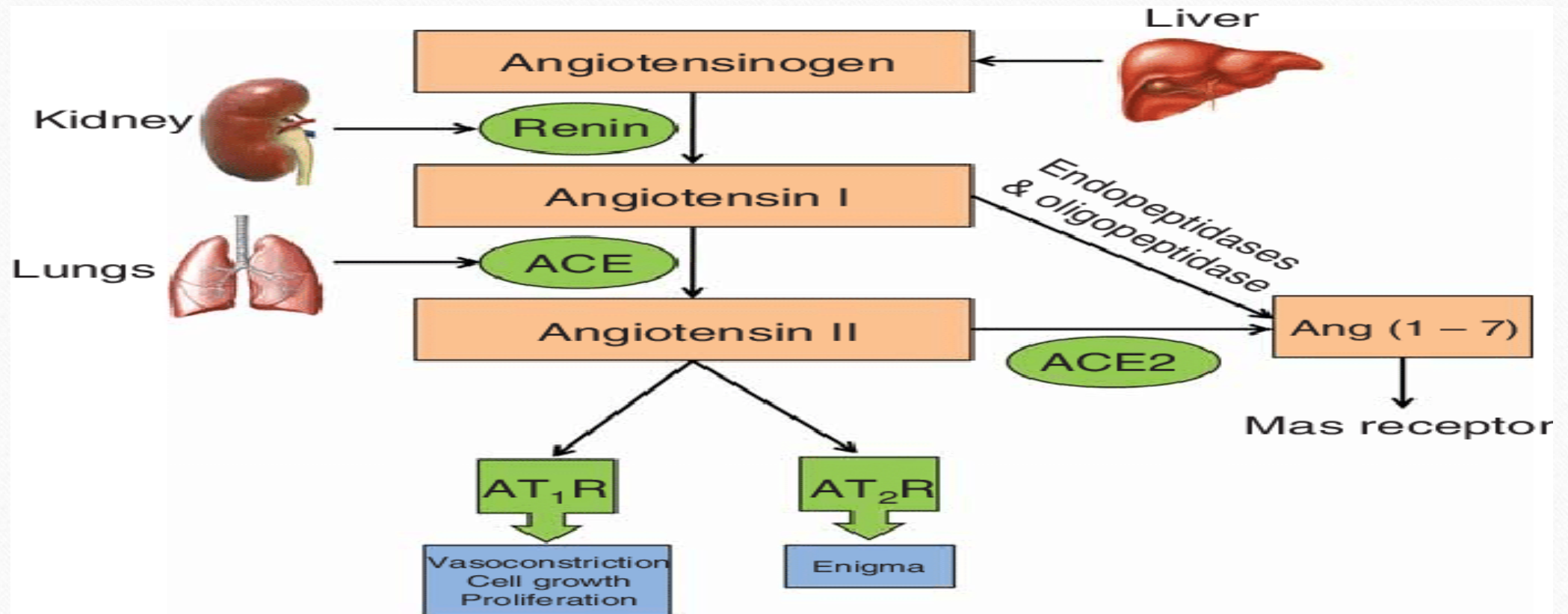
2- The cushing reaction (reflex):

- Special type of CNS ischemic response.
- When ICP \uparrow to equal ABP \rightarrow reflex \uparrow in ABP up to 250mmHg to maintain CBF & bradycardia.
- Function: protect brain centers from ischemic damage.
- If ischemia become so severe that maximal \uparrow in ABP cannot relieve it; neuronal damage occurs & within 3-10min they become inactive \rightarrow drop of ABP.

INTERMEDIATE CONTROL OF ABP

- A. Renin - angiotensin –vasoconstrictor mechanism.
- B. Stress relaxation of vasculature.
- C. Fluid – shift through the capillary wall.
- D. Right-atrial mechanism.

Renin - angiotensin –vasoconstrictor mechanism



Stress relaxation of vasculature

- When ABP increase → stretch in the wall of the arterioles due to increased tension on their walls
- After some time (minutes to hours) → arteries relax and tension on their walls decrease.

Fluid-shift mechanism

- When ABP decrease due to a decrease in blood volume → decrease in the capillary pressure → fluid is absorbed from tissues to capillaries → increase in blood volume and ABP.
- When ABO increase → increase in capillary pressure → fluid is filtered into tissue space → decrease in blood volume and ABP.

Right-atrial mechanism (volume reflex)

- when blood volume $\uparrow \rightarrow$ stimulation of stretch receptors in Rt atrium & pulmonary arteries leading to
 - 1- Generalized VD & \downarrow ABP.
 - 2- Increase salts and water excretion by kidneys.

Renal mechanism or long term regulatory mechanism

- When arterial blood pressure increase → pressure diuresis → kidneys excrete excess amounts of salts and water.
- When blood pressure falls → kidney reduce urine formation, with thirst stimulation and good fluid intake → restoration of blood volume and ABP.
- Renal mechanism is the strongest and slowest mechanism (several days or weeks).

HORMONAL MECHANISM FOR REGULATION OF BLOOD PRESSURE

increase ABP

- 1- Adrenaline and nor-adrenaline.*
- 2- Thyroxin.*
- 3- Aldosterone.*
- 4- Vasopressin (ADH).*
- 5- Angiotensins.*
- 6- Serotonin.*

Decrease ABP

- 1- Vasoactive Intestinal Polypeptide (VIP).*
- 2- Bradykinin.*
- 3- Prostaglandins (PGE₂).*
- 4- Histamine.*
- 5-Acetylcholine.*
- 6- Atrial Natriuretic Peptide (ANP).*

APPLIED PHYSIOLOGY

1- HYPERTENSION

Hypertension is defined as the persistent high blood pressure.

- Types of Hypertension

Hypertension is divided into two types:

1. **Primary hypertension** or **essential hypertension**
2. **Secondary hypertension.**

Common causes of secondary hypertension

1- Cardiovascular hypertension

- a) Atherosclerosis.
- b) Coarctation of aorta.

2- Endocrine hypertension

- a) Pheochromocytoma.
- b) Hyperaldosteronism.
- c) Cushing syndrome.

Common causes of secondary hypertension

3- Renal hypertension

- a) Stenosis of renal arteries.
- b) Glomerulonephritis.

4- Neurogenic hypertension

Increased intracranial pressure

2-nHYPOTENSION

- **Definition:** Hypotension is the low blood pressure. When the systolic pressure is less than 90 mm Hg, it is considered as hypotension.

- **Types**

1. Primary hypotension
2. Secondary hypotension: is the hypotension that occurs due to some underlying diseases:
 - a) Myocardial infarction
 - b) Hypoactivity of pituitary gland
 - c) Hypoactivity of adrenal glands

Thank you

the end

