ENDOCRINE MODULE
PHYSIOLOGY (LECTURE 3)
POSTERIOR PITUITARY GLAND

BY

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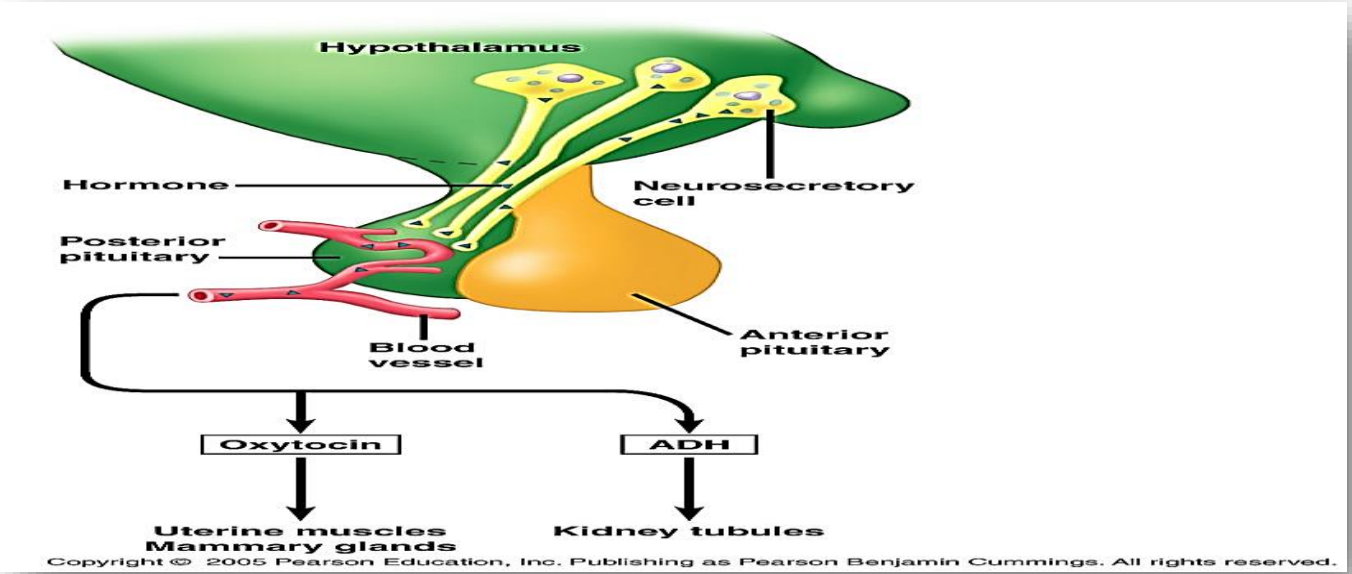
2023-2024



THE POSTERIOR PITUITARY GLAND (NEUROHYPOPHYSIS)

The posterior pituitary is connected to the **hypothalamus** by the pituitary stalk which contains the **hypothalamo-hypophyseal tract**. The tract is formed of the axons (nerve fibers) of the **supraoptic** and **paraventricular** nuclei of the hypothalamus. These fibers terminate in the posterior pituitary.

The posterior pituitary releases 2 hormones :

1. **Anti- diuretic hormone (ADH or vasopressin)**.
2. **Oxytocin**.

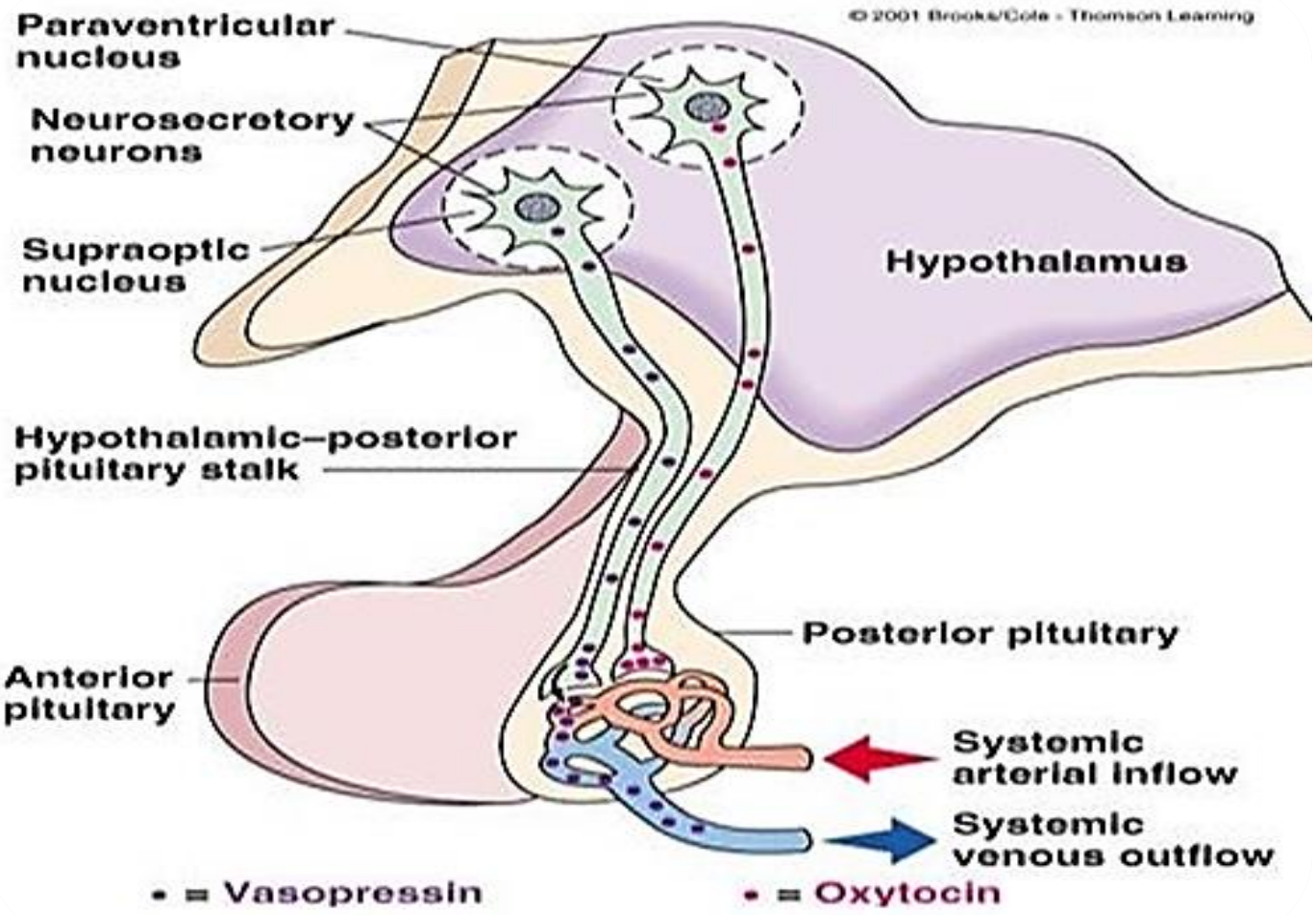
Both ADH and oxytocin are formed by the neurons of the supraoptic and paraventricular nuclei of the hypothalamus.

The supraoptic forms mainly ADH (more than 80%), while the paraventricular forms mainly oxytocin (more than 80%).

- Both hormones are peptides, each contains 9 amino acids.
- They are structurally similar except for 2 amino acids.
- The two hormones are then transported along the hypothalamo-hypophyseal tract to reach their nerve terminals in the posterior pituitary gland where they are stored.
- The two hormones are released from their storage granules in the nerve terminals of the posterior pituitary gland according to nerve impulses descending from the hypothalamus along the hypothalamo-hypophyseal tract.
- For this reason, these hormones are typical neural hormones (hormones secreted into circulation by nerve cells).

- **Mechanism of release of hormones:**

Stimulation of hypothalamic nuclei initiates action potentials in their neurons which on reaching the nerve endings, cause release of hormones (by Ca^{2+} dependent exocytosis), and all are absorbed into the local blood vessels, from which they reach the general circulation.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Paraventricular nucleus
Neurosecretory neurons
Supraoptic nucleus
Hypothalamus
Hypothalamic–posterior pituitary stalk
Posterior pituitary
Anterior pituitary
Systemic arterial inflow
Systemic venous outflow
• = Vasopressin
• = Oxytocin

(1) ANTI- DIURETIC HORMONE (ADH OR VASOPRESSIN)

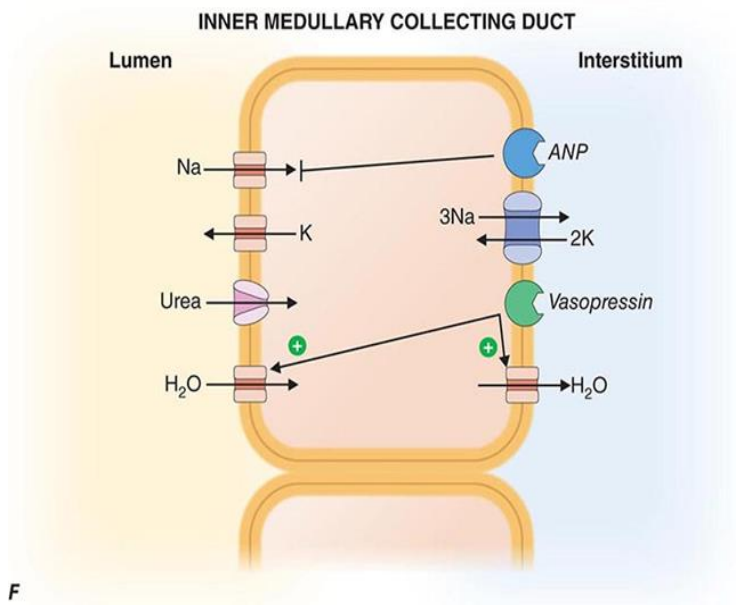
Functions of ADH:

(1) Kidney:

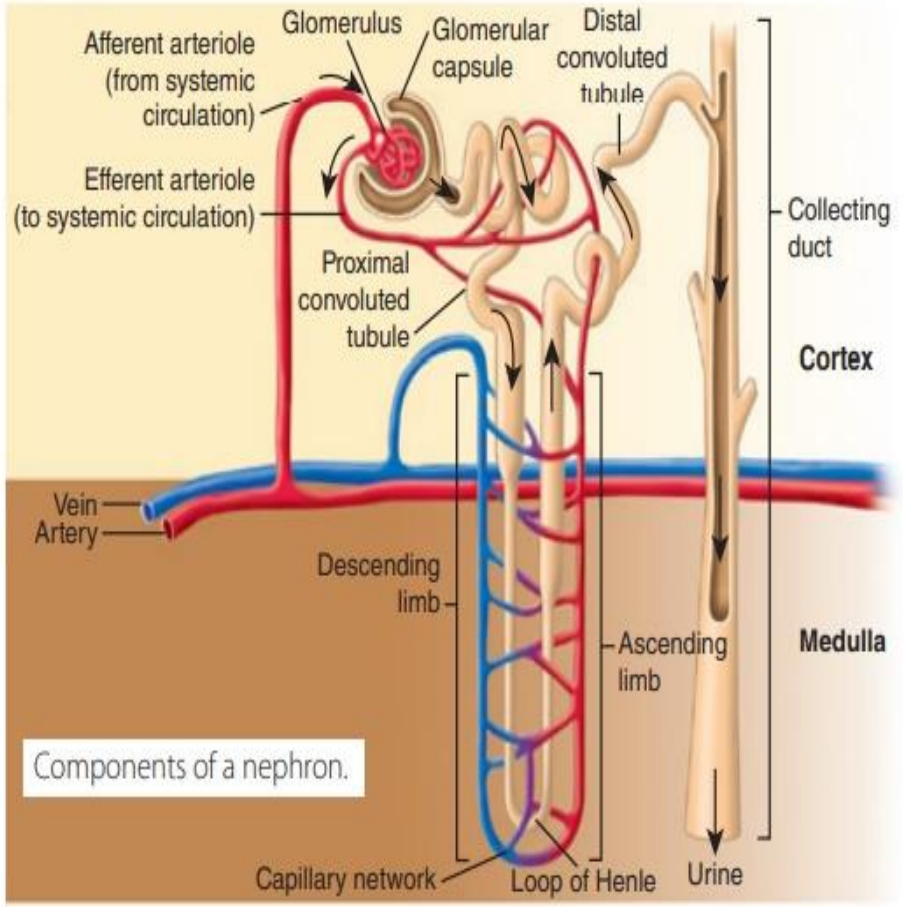
water retention (an antidiuretic effect):

ADH increases permeability of distal segments of renal tubules ……..Increases water reabsorption from the renal tubules (mainly collecting tubules)……decreases the urine volume (concentrated urine).

- This effect is regulated by **V_2 receptors**, through the action of **cAMP** ……causes insertion of protein water channels called **aquaporin 2** into the luminal border of tubular cells ……..increases permeability of cells to water……..water reabsorption by passive diffusion under influence of hyperosmolarity of renal medullary interstitium that is produced by counter current function of loops of Henle of juxtamedullary nephrons.


INNER MEDULLARY COLLECTING DUCT
Lumen
Interstitium
Na
K
Urea
H_2O
ANP
3Na
2K
Vasopressin
H_2O

F


Afferent arteriole (from systemic circulation)
Glomerulus
Glomerular capsule
Distal convoluted tubule
Efferent arteriole (to systemic circulation)
Proximal convoluted tubule
Collecting duct
Cortex
Vein
Artery
Descending limb
Ascending limb
Medulla
Components of a nephron.
Capillary network
Loop of Henle
Urine

(2) On the blood vessels:

Physiological doses that are normally secreted and produce anti-diuresis but have no effect on smooth muscles of B.V.

While large dose produce vasoconstriction allover the body; so it is called vasopressin (increased blood pressure).

- This effect is regulated by **V_1 receptors** in arterioles, through the action of IP3/Ca^{2+}……smooth muscle contraction and V.C.

N.B. V3 receptors are located in adenohypophysis, where they stimulate ACTH secretion.

(3) On other Smooth muscles:

Intestinal colic and increased the uterine contraction (stimulates all smooth muscles).

Control of ADH release

1. Plasma osmolality (Osmotic pressure):

Changes in plasma osmolality (1-2%), will stimulate **osmoreceptors** in the **anterior hypothalamus**…….increases ADH…….. water retention ……decreases plasma osmotic pressure back to normal level.

Mechanism:

↑osmotic pressure of plasma (as in dehydration)→ stimulate osmoreceptors → sends impulses to stimulate the supraoptic nucleus of the hypothalamus → send impulses along the hypothalamo-hypophyseal tract to nerve terminals in the posterior pituitary → ↑ ADH release → retention of water by the kidney → ↓ osmotic pressure of plasma to normal.

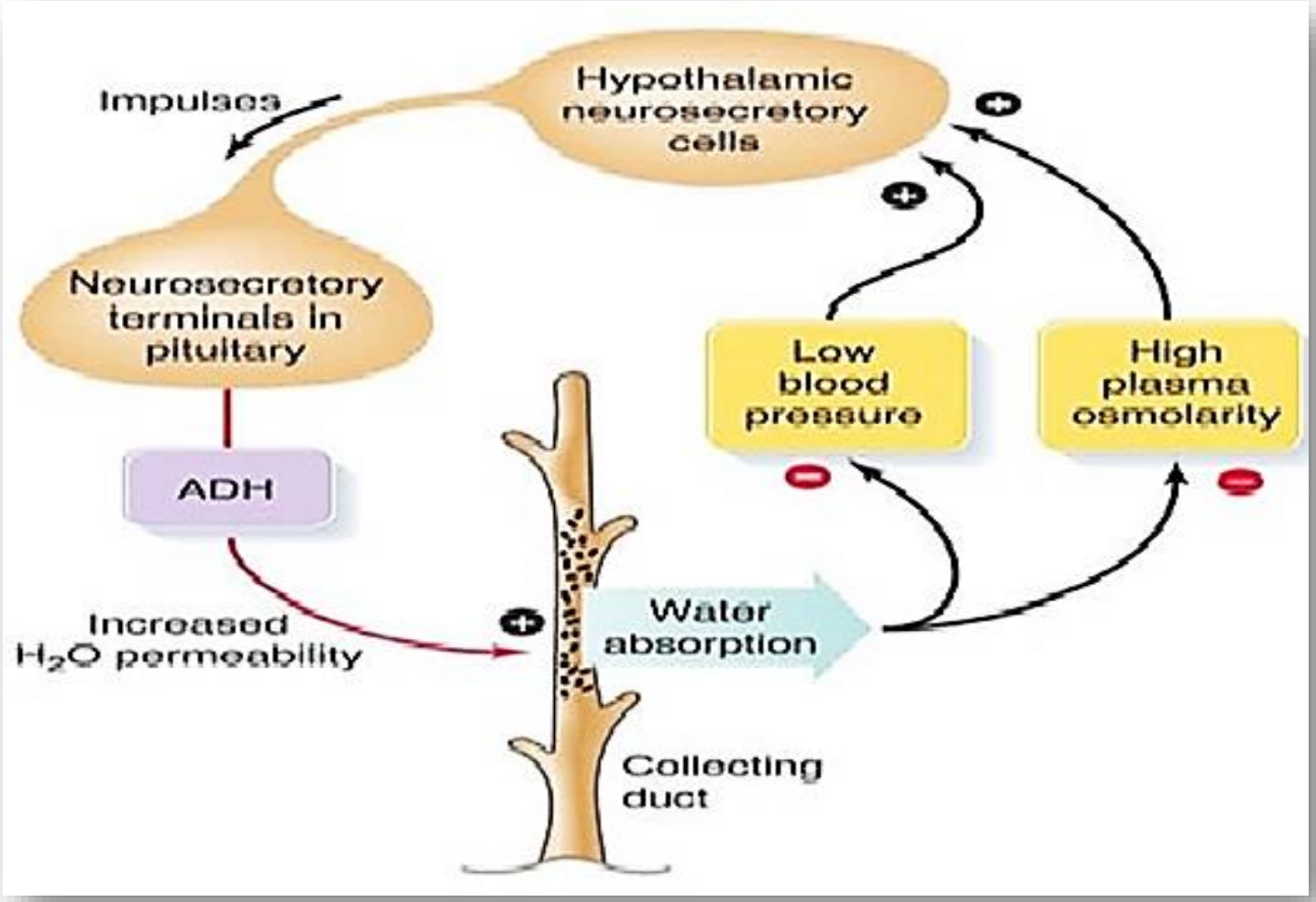
– ↓ osmotic pressure as in overhydration → ↓ADH secretion.

2. ECF (Blood) volume:

- Blood volume (hypovolemia by about 10%)→ stimulate volume receptors (in atria, big veins and pulmonary vessels)……discharge signals that stimulate hypothalamic ADH secreting neurons …..ADH release from neurohypophysis……water retention……increases ECF volume back to its normal level.
- Hemorrhage is the most potent stimulus of ADH release which helps rise of blood pressure by its V.C. and fluid retention effects.
- Hypervolemia…….opposite effects.
- Baroreceptors "high pressure receptors" carotid sinus and aortic arch baroreceptors increase ADH Only if changes in ECF volume are great enough to affect ABP.

3- Other factors:

Stress and sleep →↑ ADH secretion.
Drugs: morphine& nicotine→ ↑ ADH
Alcohol (ethanol)→ ↓ADH secretion
Cold → ↓ADH secretion


Impulses
Hypothalamic neurosecretory cells
Neurosecretory terminals in pituitary
ADH
Increased H_2O permeability
Water absorption
Collecting duct
Low blood pressure
High plasma osmolarity

ABNORMALITIES OF ADH RELEASE:

DIABETES INSIPIDUS:

- It is a disease caused by ADH deficiency.
- **Types:**

(1) Neurogenic :

A lesion in the hypothalamus destroying the regions of the supraoptic nuclei (mainly) → ↓ ADH secretion.

ADH Levels are low.

(2) Nephrogenic:

A condition in which ADH is secreted at the normal rate but the response of the cell wall of the kidney tubular cells is impaired due to receptor defect (V_2).

Manifestations:

- Urine volume is markedly increased (**polyuria**). It may reach 20 liters / day in severe cases.
- The specific gravity of urine is very low 1002 - 1004.
- Loss of large volume of urine → excessive thirst and intake of water (**polydipsia**).
- Loss of water soluble vitamins in urine.

N.B. neurogenic Diabetes insipidus responds to exogenous ADH administration but nephrogenic diabetes insipidus does not.

Oxytocin

Functions of Oxytocin :

1) Milk ejection (letdown):

It is the most important function oxytocin.

Oxytocin stimulates contraction of the **myoepithelial cells** …….. squeeze the milk outwards through the nipples.

***2) It stimulates contraction of the uterine smooth muscles*.**

Significance:

- It helps the process of normal labor.
- It helps involution of the uterus after delivery.
- It helps the ascent of spermatozoa in the female genital tract.

3) Oxytocin has a slight pressor and antidiuretic effects.

Control of Oxytocin Secretion:

A) Neuroendocrine reflex.

Afferent impulses reach the hypothalamus from different sites and stimulate the paraventricular nucleus to release oxytocin.

Source of stimuli :

1) afferent impulses from the nipple during sucking → initiate the suckling reflex.

Suckling (stimulation of touch receptors around nipple)……afferent (nerve impulse)……..hypothalamus (paraventricular nuclei)…….oxytocin……contraction of myoepithelial cells…….milk letdown.

2) afferent impulses from the female genital tract during delivery.

Significance :

Oxytocin stimulates uterine contractions and facilitates delivery.

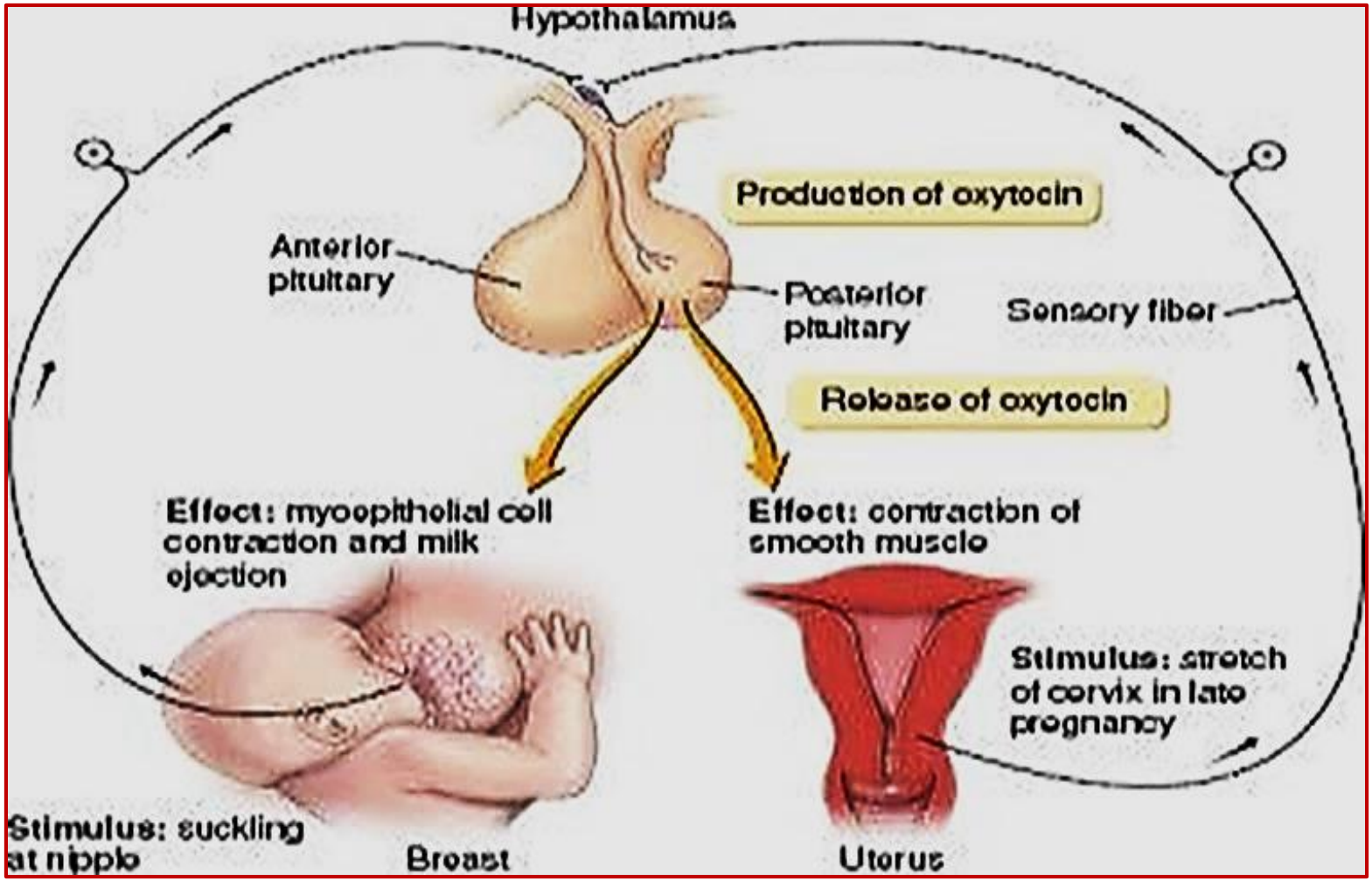
During labor:

Cervical dilatation by baby head……..nerve impulses…….hypothalamus……oxytocin…….uterine contraction……more cervical dilatation by baby head……more uterine contraction.

Positive feedback mechanism.

B) Hormones:

- Progesterone → ↓uterine sensitivity to oxytocin.
- Estrogen → ↑uterine sensitivity to oxytocin.


Hypothalamus
Production of oxytocin
Anterior pituitary
Posterior pituitary
Sensory fiber
Release of oxytocin
Effect: myoepithelial cell contraction and milk ejection
Effect: contraction of smooth muscle
Stimulus: stretch of cervix in late pregnancy
Stimulus: suckling at nipple
Breast
Uterus

THANK YOU