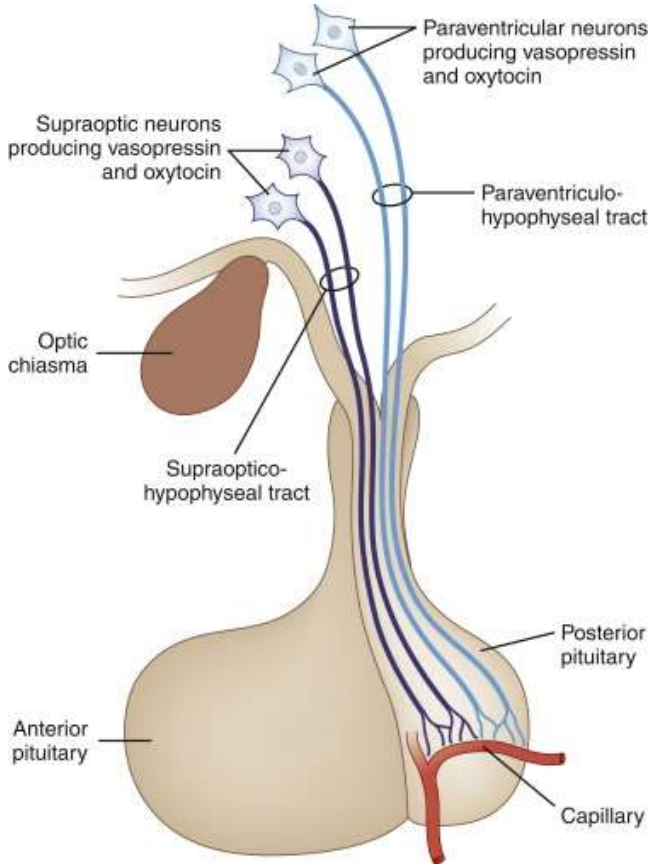
POSTERIOR PITUITARY

BY

DR. NOUR A. MOHAMMED
MUT'AH SCHOOL OF MEDICINE



- Posterior pituitary hormones are synthesized in hypothalamus
- Posterior pituitary just store and release the hormones.
- 2 polypeptide hormones; ***ADH (Vasopressin)*** **&** ***Oxytocin*** *(formed of 9 aa).*
- They are formed in the cells of the ***supraoptic &paraventricular*** *nuclei of hypothalamus respectively*

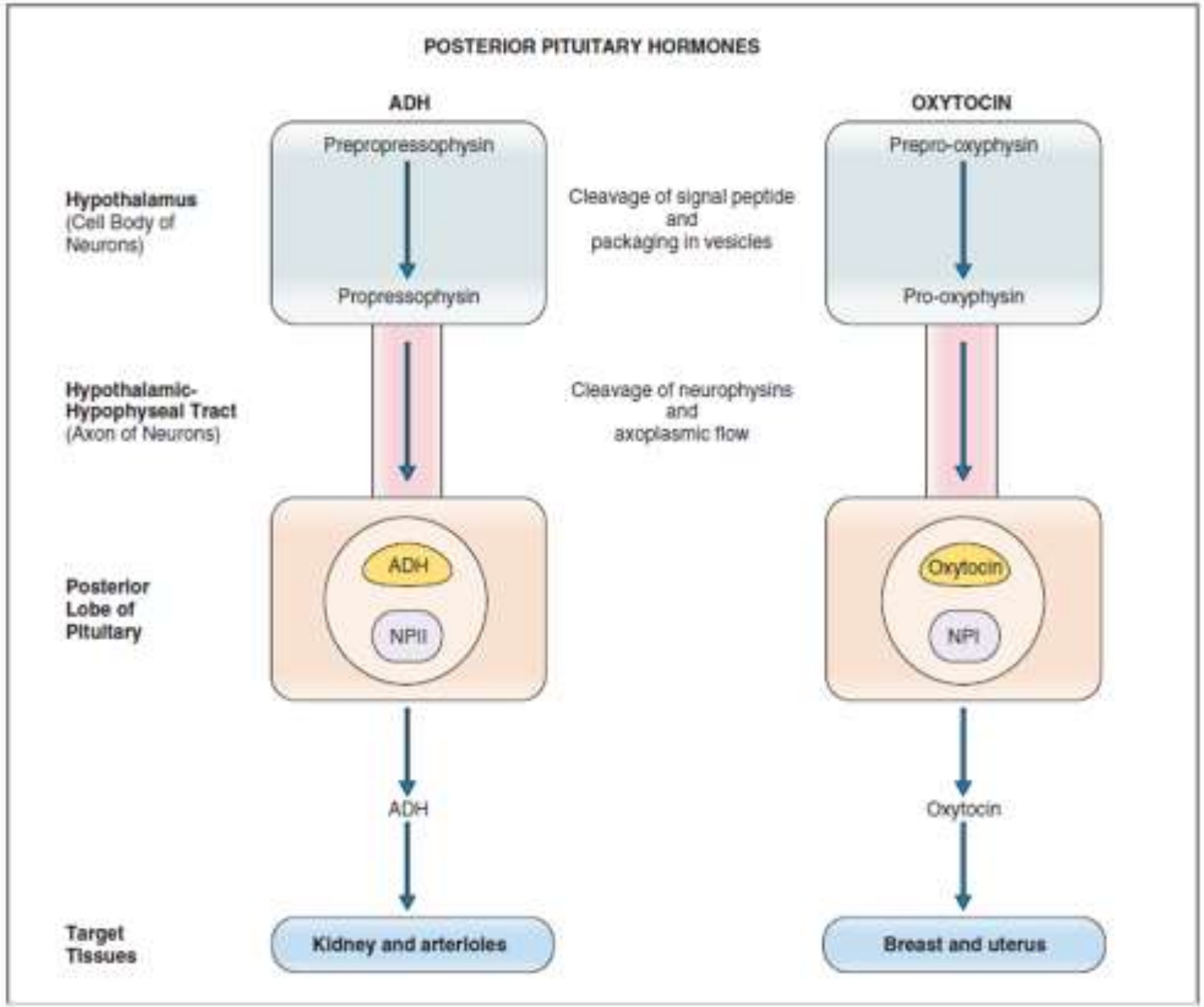
- Their precursor molecules called ***Neurophysin*** that include:

a) **Preprooxyphysin** → **Oxyphysin** or **Neurophysin I** → **oxytocin.**

b) **Prepropressophysin** → **Pressophysin** or **Neurophysin II** → **Vasopressin**.

- Then they are **transported** as granules by ***axoplasmic flow*** to the nerve endings in the posterior pituitary, where they are **stored** as ***Herring bodies***.

- They are **released** by nerve impulses from hypothalamus *(by help of Ca++ ions)*


POSTERIOR PITUITARY HORMONES
ADH
OXYTOCIN
Hypothalamus
(Cell Body of
Neurons)
Prepropressophysin
Propressophysin
Cleavage of signal peptide
and
packaging in vesicles
Prepro-oxyphysin
Pro-oxyphysin
Hypothalamic-
Hypophyseal Tract
(Axon of Neurons)
Cleavage of neurophysins
and
axoplasmic flow
Posterior
Lobe of
Pituitary
ADH
NPII
Oxytocin
NPI
ADH
Oxytocin
Target
Tissues
Kidney and arterioles
Breast and uterus

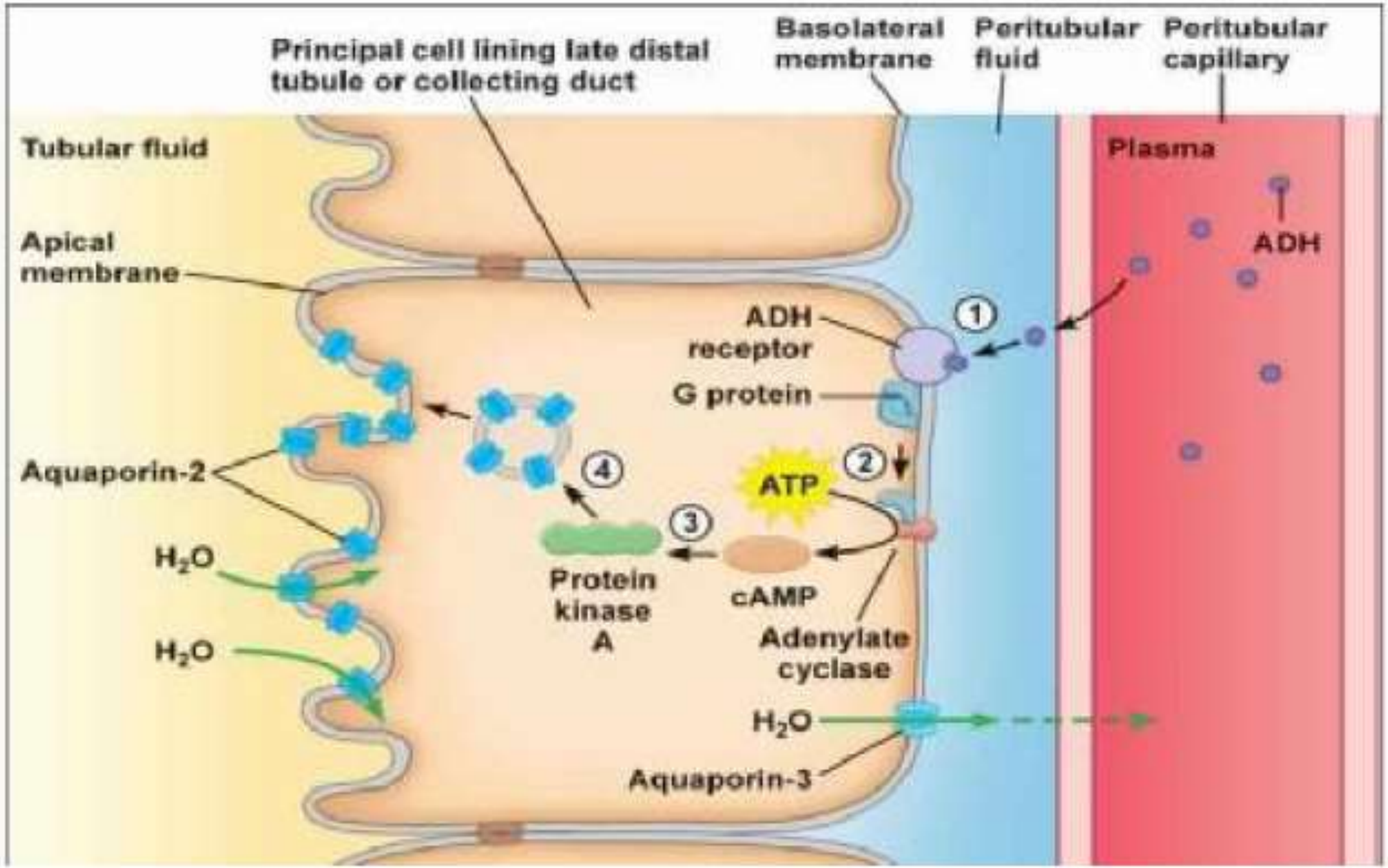
Functions of ADH (Vasopressin)

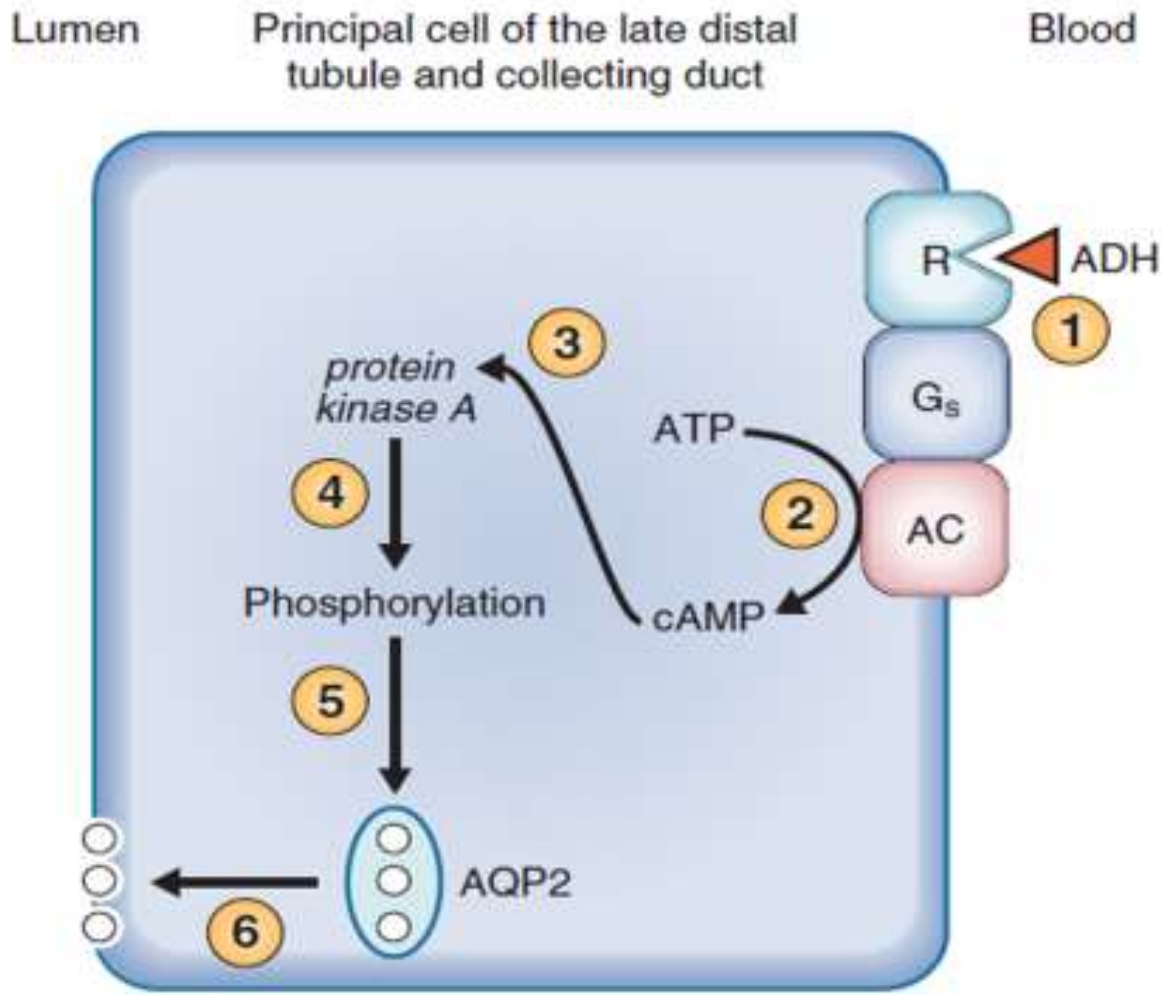
1. Anti-diuresis:

- **ñ H2O reabsorption** ⇨ ⇩ H2O excretion by kidney ⇨ ⇩ urine volume •
- ⇧ H2O reabsorption ⇨ **ñ Plasma volume ð ò Plasma osmolarity** •
- ADH increases H2O reabsorption **only** (no effect on salts) •
- ***Site of action:*** ADH ⇨ ⇧ permeability of the distal convoluted tubules & principal cells of collecting ducts (P-cells) to H2O ⇨ ⇧ H2O reabsorption

- ***Mechanism of action:***

• Acting on V**2** receptors on the blood side membrane of the tubular cells ⇧ c**A**MP in the cells → increase protein kinase and increase formation of **microtubules** in the cell membrane called (**A**quaporin channels type **2** → Increase in the permeability of the luminal side of the cell • membrane to water


Principal cell lining late distal tubule or collecting duct
Basolateral membrane
Peritubular fluid
Peritubular capillary
Tubular fluid
Plasma
ADH
Apical membrane
ADH receptor
1
G protein
2
ATP
Aquaporin-2
4
3
H_2O
Protein kinase A
cAMP
Adenylate cyclase
H_2O
H_2O
Aquaporin-3


Lumen
Principal cell of the late distal tubule and collecting duct
Blood
R
ADH
1
Gs
AC
ATP
2
cAMP
3
protein kinase A
4
Phosphorylation
5
AQP2
6

2. Vasoconstrictor effect:

- Normally ADH has no effect on blood vessels.

- But, in **large dose** it causes vasoconstriction all over the body **Except cerebral & renal blood vessels.**

- This is because V1 receptor is less sensitive than V2.

- 10% decrease in blood volume is sufficient to cause the release ADH to participate in blood volume & blood pressure control.

3. ADH stimulates corticotrophin (ACTH) release:

ADH increase ACTH from the anterior pituitary.

4. ADH inhibits renin release:

- ADH decrease renin from the juxta-glomerular apparatus.

- It is a -ve feed back mechanism.

(renin increase Angiotensin II which in turn increase ADH So, ADH decrease renin).

Regulation of ADH secretion

1. Osmotic regulation:

- Increase Solutes concentration increases osmotic pressure of blood (by 1-5%) causes stimulation of osmoreceptors in hypothalamus which send impulses to stimulate **supraoptic nuclei** increasing ADH so, increasing water reabsorption **while** electrolytes continue to be lost so, dilutes ECF and restores normal osmotic pressure.
- Dilution of ECF inhibits **ADH** secretion.

2.Alcohol:

- Inhibits ADH secretion causes marked diuresis (alcohol diuresis) •

3. Hypothalamic factors:

- **Temperature: Hot ⇨ ⇧ ADH while Cold ⇨ ⇩ ADH (cold diuresis).** •
- **Pain & trauma & anxiety & morphine & nicotine ⇨ ⇧ ADH secretion.** •

4. Effective plasma volume (effect of hemorrhage): •

➢ *Receptors:* •

- The volume receptors (low pressure receptors) •

- **Site:** Present in the right and left atria & great veins & pulmonary vessels •

- Normally send **tonic inhibitory impulses** to supraoptic nuclei to inhibit ADH •

➢ *Effect of stimulation:* •

- ⇩ Blood volume **(by 10%)**⇨ ⇩ the frequency of inhibitory impulses from the volume • receptors ⇨ stimulates the release of ADH

- ADH ⇨ ⇧ **H2O** reabsorption ⇨ ⇧ the extracellular fluids ⇨ restore the normal blood • volume.

➢ *Inhibition:* •

- Volume expansion e.g. (transfusion) ⇨ inhibition of release of ADH. •

➢ *Primary stimulus:* •

- The primary stimulus is ⇩ blood flow to hypothalamus after **hemorrhage.** •

5. Angiotensin II:

- **Stimulus:**

- Renal ischemia ⇨ release of renin ⇨ formation of angiotensin II ⇨ ⇧ ADH secretion.

- **Mechanism:**

- Angiotensin II ⇨ ⇧ size & number of Na+ channels in the osmoreceptor cells in the hypothalamus ⇨ ⇧ Na+ influx to the receptors.

- Na+ entering the cell of osmoreceptor ⇨ depolarization ⇨ ⇧ ADH secretion.

- **So, Ang II ⇨ causes stimulation of osmoreceptor even with normal osmolarity**.

Functions of oxytocin hormone

1. Effect on the uterus: •

- Stimulates the pregnant uterus at end of pregnancy (during **L**abor) •

⇨ powerful **tonic** contraction and helps delivery of fetus •

2. Effect in primary fertilization of the ovum: •

- Sexual stimulation during intercourse ⇨ reflex stimulation of the paraventricular nuclei ⇨ • ⇧ oxytocin ⇨ **rhythmic** uterine contractions (during orgasm) ⇨ uterine suction of semen toward the fallopian tubes.

3. Effect on Milk Ejection: •

- Oxytocin ⇨ contraction of the myoepithelial cells around the alveoli of mammary glands • (during **L**actation) ⇨ milk **E**jection.

- **No role in milk formation (no role in synthesis of milk).**

4. In the Male (Ejaculation):

- Oxytocin ⇨ increases the contractility of vas deferens and seminal vesicle •

⇨ semen transport during **E**jaculation. •

- **No role in semen formation (no role in spermatogenesis).** •

Regulation of oxytocin secretion

- Oxytocin is regulated by +ve feedback reflexes.

1. Dilatation (stretch) of uterus & cervix & vagina: •

- It occur during **L**abor.

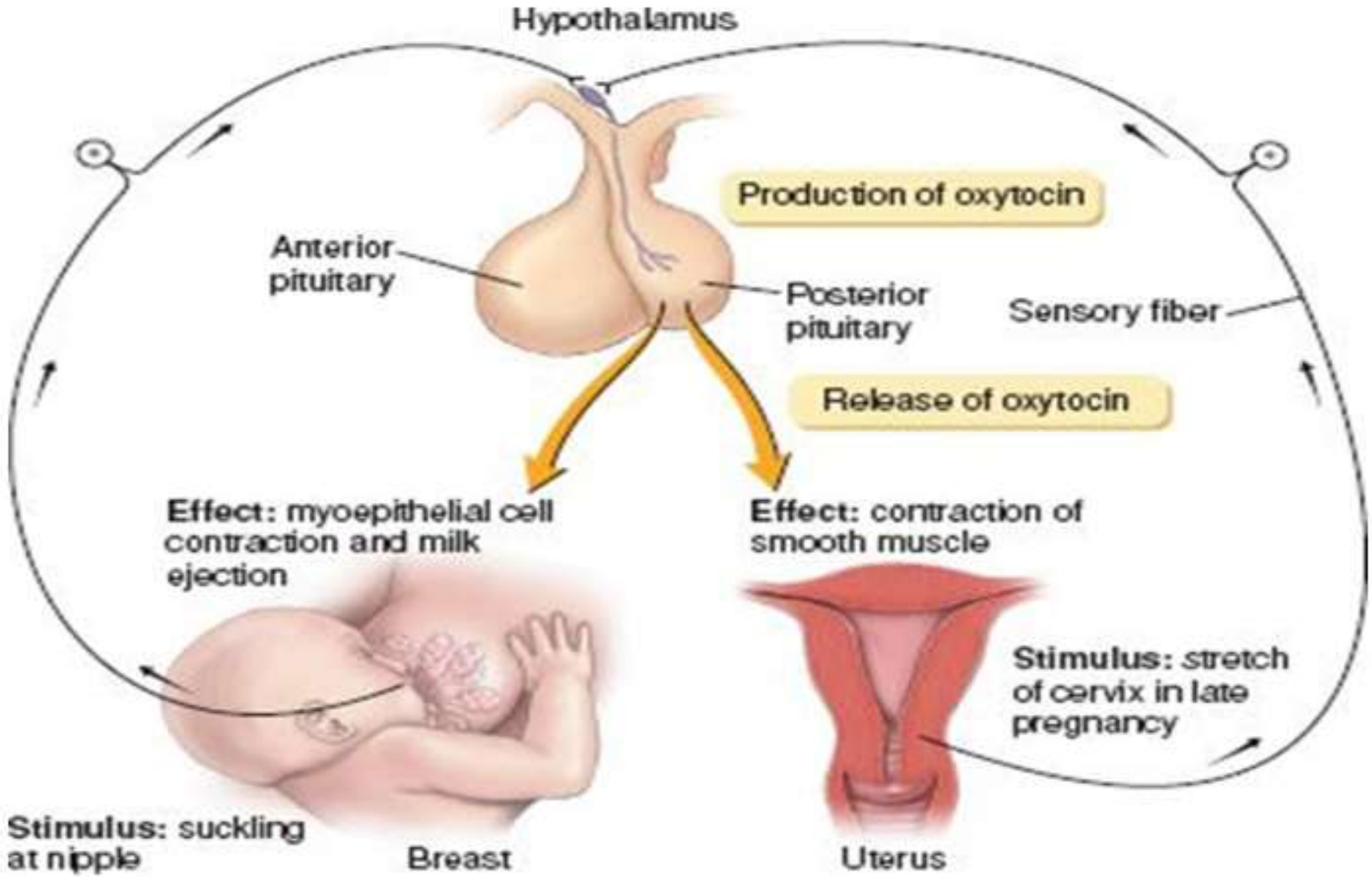
- It is called **positive feed back of labor**. •

a) Stretch of uterus ⇨ stimulate stretch receptors in the wall of the uterus.

b) Dilatation of cervix & vagina after the onset of labor by the head of •

fetus ⇨ stimulate stretch receptors in the wall of the cervix. •

Both (a+b) ⇨ send impulses to hypothalamus ⇨ stimulation of • paraventricular nuclei ⇨ ⇧ oxytocin ⇨ powerful **tonic** contraction ⇨ labor.


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

2. Stimulation of vagina & cervix:

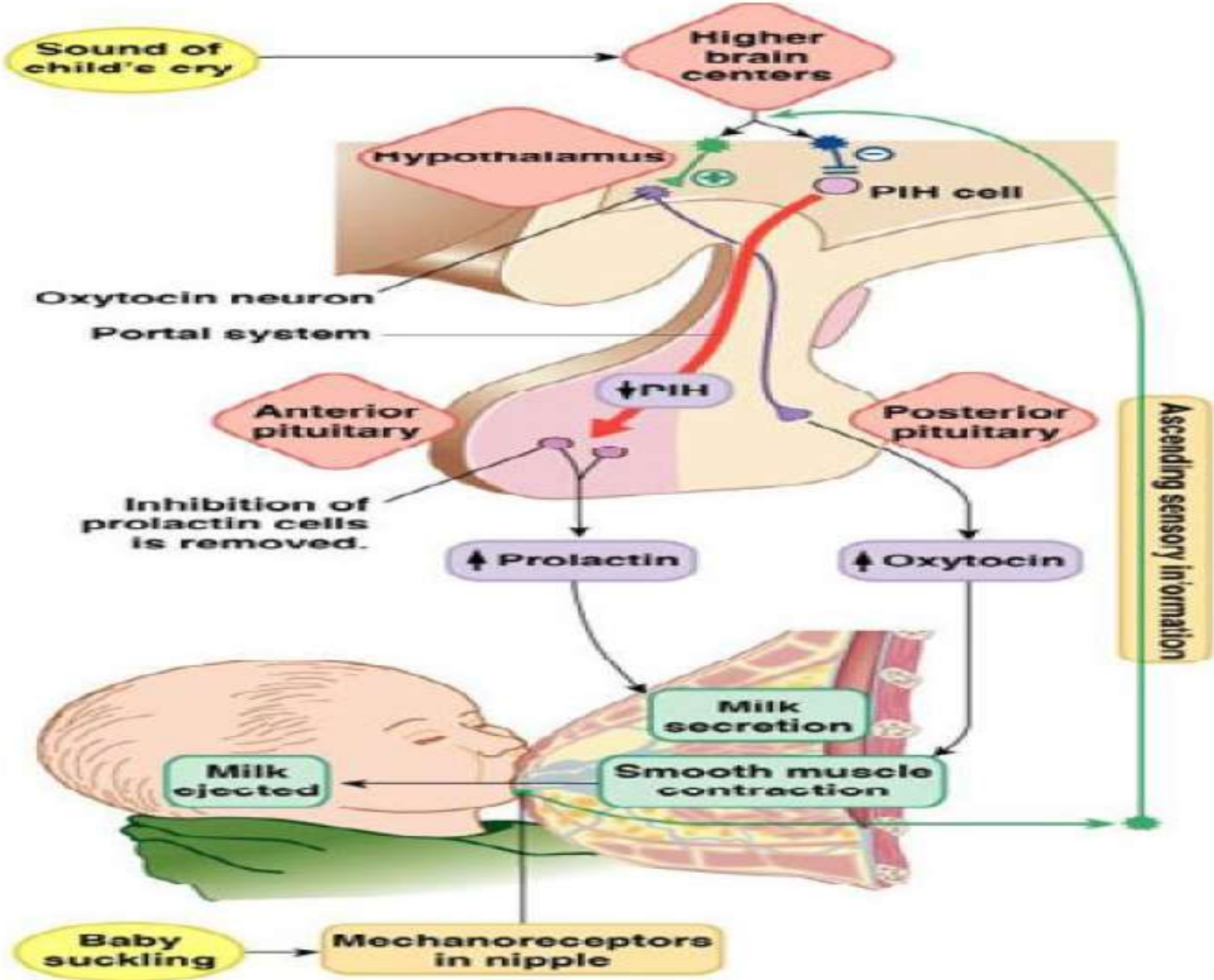
-It occurs during intercourse.

- Vaginal & cervical stimulation ⇨ send impulses to hypothalamus ⇨

- stimulation of paraventricular nuclei ⇨ ⇧ oxytocin ⇨ **rhythmic** uterine
- contractions ⇨ orgasm & suction of semen toward the fallopian tubes.

3. Suckling of the nipple:

- It occur during **L**actation.
- It is called **suckling reflex**.
- Suckling ⇨ send impulses to hypothalamus ⇨ stimulation of paraventricular nuclei ⇨ ⇧ oxytocin ⇨ ejection of milk.


Sound of child's cry
Higher brain centers
Hypothalamus
PIH cell
Oxytocin neuron
Portal system
PIH
Anterior pituitary
Posterior pituitary
Inhibition of prolactin cells is removed.
Prolactin
Oxytocin
Ascending sensory information
Milk secretion
Milk ejected
Smooth muscle contraction
Baby suckling
Mechanoreceptors in nipple


THank you