ENDOCRINE MODULE
PHYSIOLOGY (LECTURE 4)
POSTERIOR PITUITARY GLAND

BY

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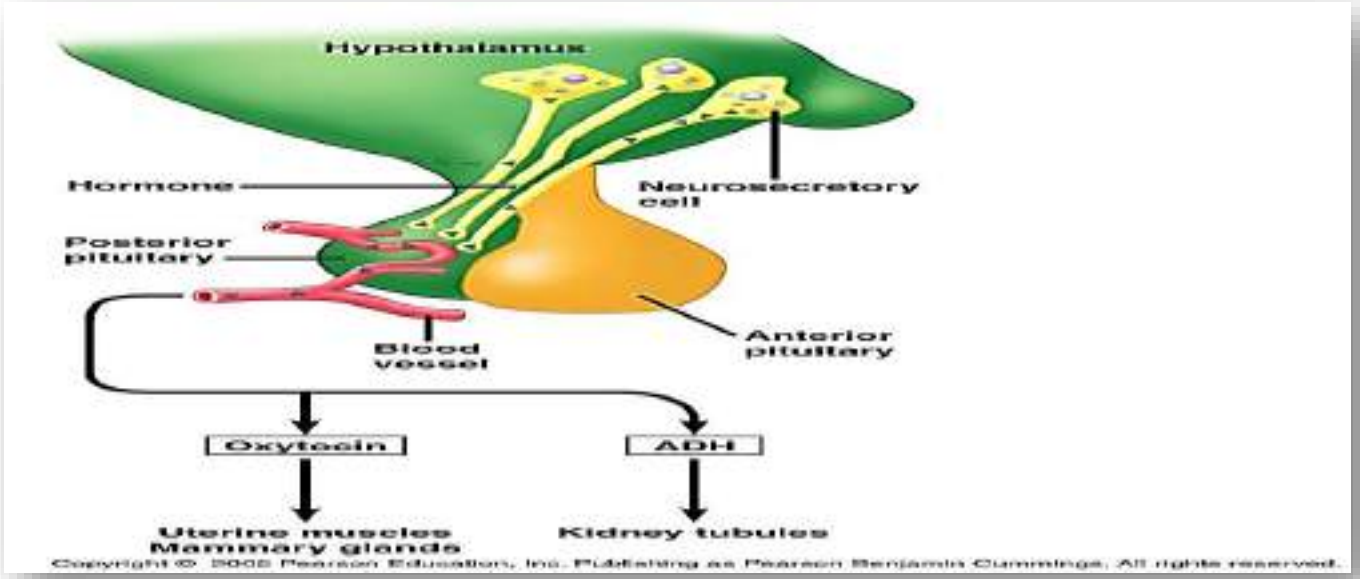


THE POSTERIOR PITUITARY GLAND (NEUROHYPOPHYSIS)

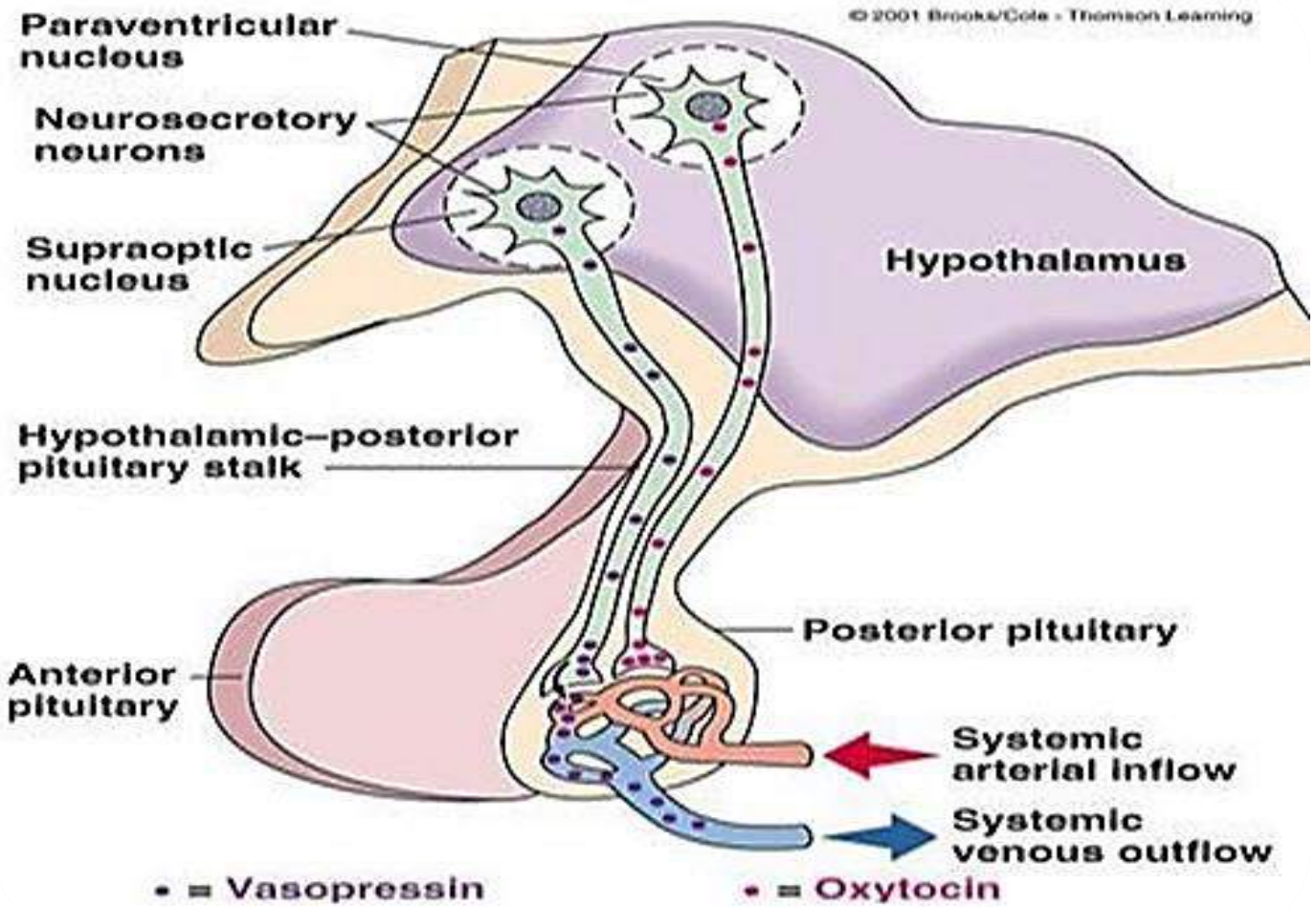
- **The posterior pituitary gland is really a neural extension of the hypothalamus.**
- **The posterior pituitary gland is connected to the hypothalamus by the pituitary stalk (infundibulum) which contains the hypothalamo-hypophyseal tract.**
- **The tract is formed of the axons (nerve fibers) of the supraoptic and paraventricular nuclei of the hypothalamus. These fibers terminate in the posterior pituitary.**
- **The hormones are synthesized not in the posterior pituitary itself but in the hypothalamus—specifically, in the cell bodies of the supraoptic and paraventricular nuclei, whose axons pass down the infundibulum and terminate in the posterior pituitary.**
- **Enclosed in small vesicles, the hormone** moves down the axons to accumulate **at the axon terminals in the posterior pituitary gland**.

POSTERIOR PITUITARY HORMONES

- ✓ **The posterior pituitary gland releases 2 hormones :**
  - o **Antidiuretic hormone (ADH or vasopressin)**.
  - o **Oxytocin**.
- ✓ **The supraoptic nucleus forms mainly ADH (more than 80%), while the paraventricular nucleus forms mainly oxytocin (more than 80%).**
- ✓ Both hormones are peptides, each contains 9 amino acids.
- ✓ They are structurally similar except for 2 amino acids.

Mechanism of Release of ADH and Oxytocin

- **Stimulation of hypothalamic nuclei initiates action potentials in their neurons which on reaching the nerve endings, cause release of hormones from posterior pituitary by Ca^{2+} dependent exocytosis.**
- **The hormone then enters capillaries to be carried away by the blood returning to the heart. In this way, the brain can receive stimuli and respond as if it were an endocrine organ.**
- **By releasing its hormones into the general circulation, the posterior pituitary can modify the functions of distant organs.**
- **For this reason, these hormones are typical neural hormones (hormones secreted into circulation by neurons).**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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) ANTIDIURETIC HORMONE (ADH OR VASOPRESSIN)

Functions of ADH:

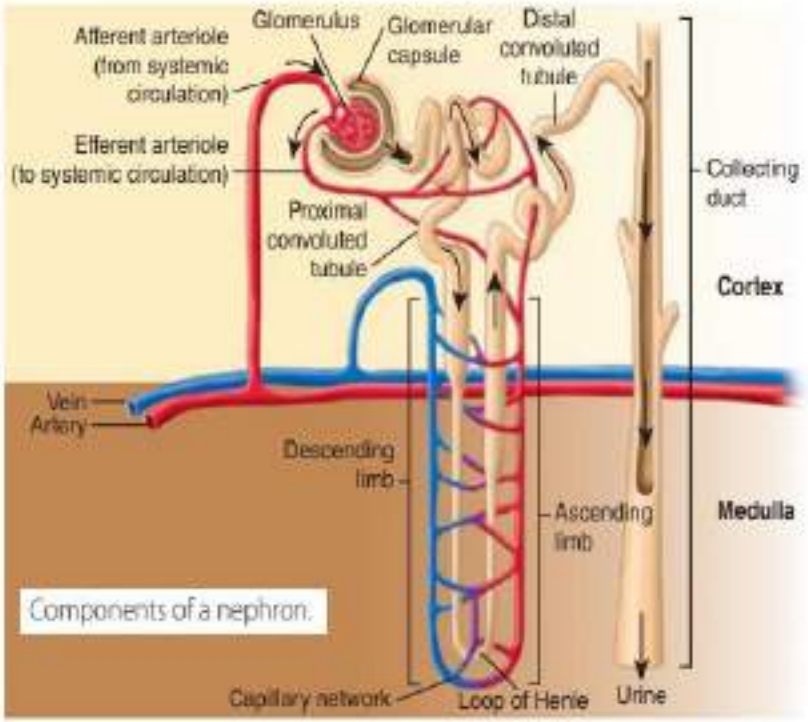
(1) Kidney:

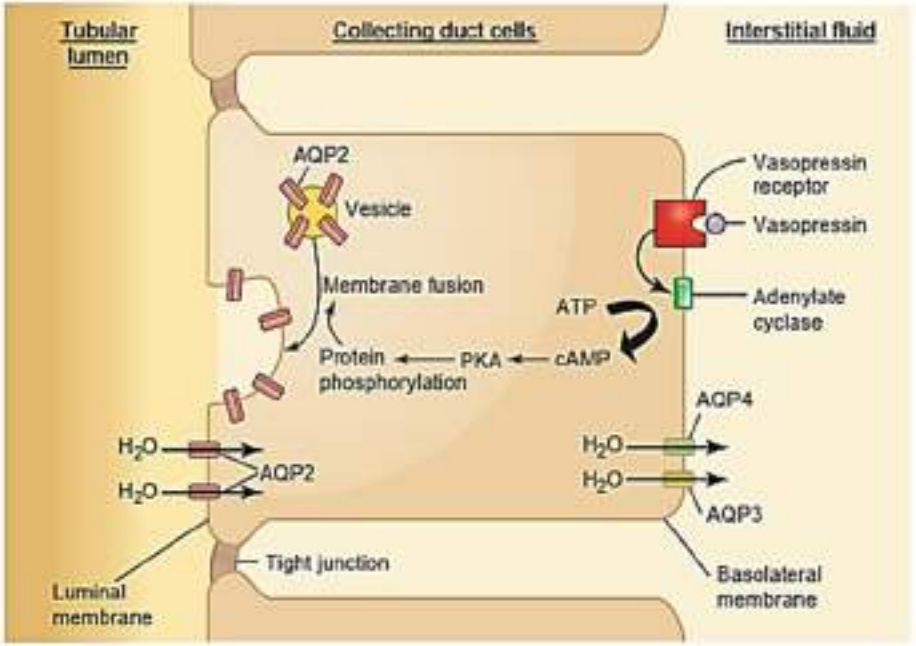
water retention (an antidiuretic effect):

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).

Mechanism:

- 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.


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


Tubular lumen
Collecting duct cells
Interstitial fluid
AQP2
Vesicle
Vasopressin receptor
Vasopressin
Membrane fusion
ATP
Adenylate cyclase
Protein phosphorylation
PKA
cAMP
AQP4
H_2O
H_2O
AQP2
H_2O
H_2O
AQP3
Tight junction
Luminal membrane
Basolateral membrane

(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 which increases arterial blood pressure; so it is called vasopressin (increased blood pressure).

Mechanism:

ADH binds to V1 receptors in arterioles → increases cytoplasmic calcium (through formation of IP3) → smooth muscle contraction and VC.

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).

Factors that Control Release of ADH

1. Plasma osmolality (Osmotic pressure):

Changes in plasma osmolality **(~1%)**, 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 back to its normal level.

↓ osmotic pressure as in hydration → ↓ADH secretion.

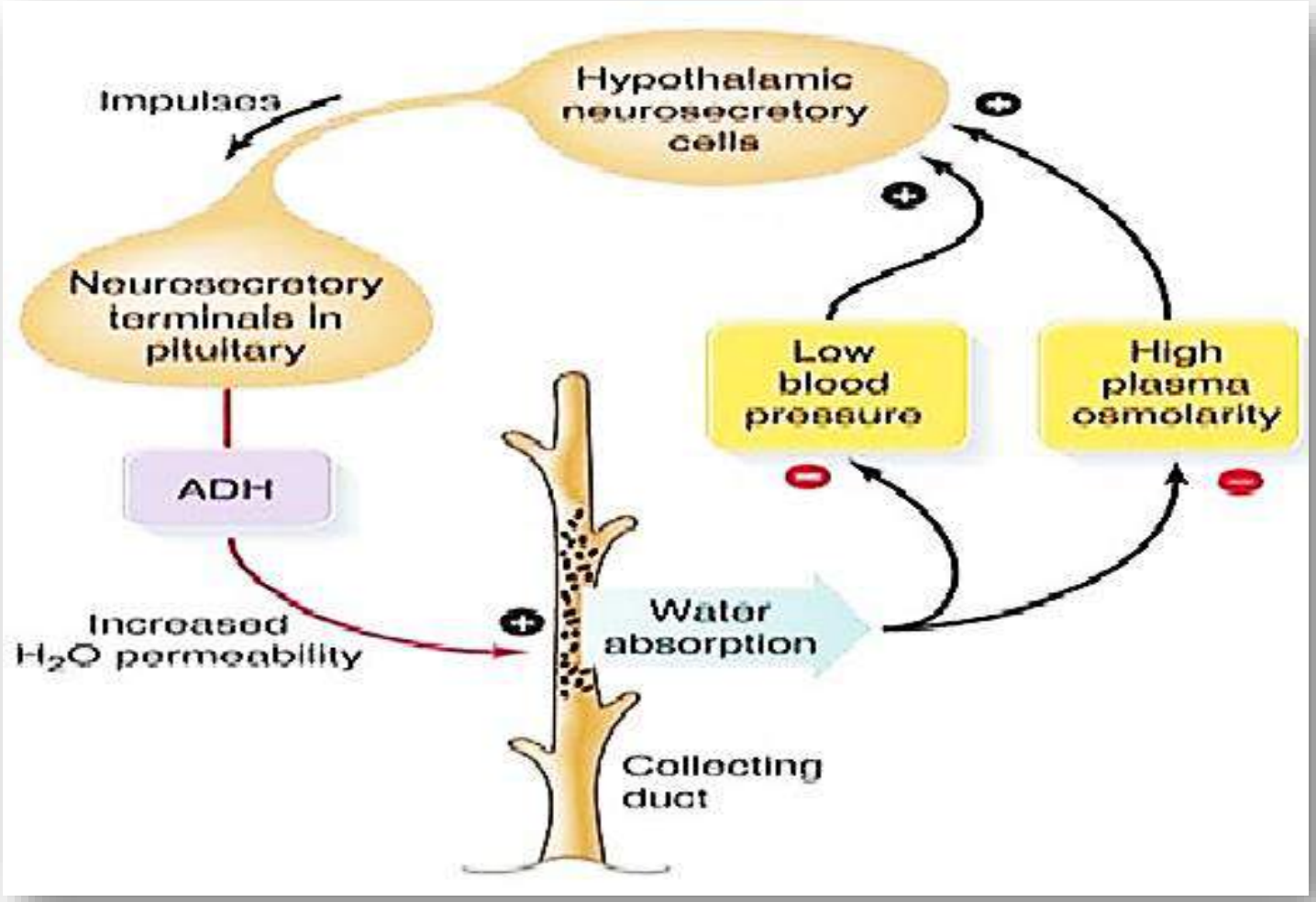
2. ECF (Blood) volume:

- ✓Hypovolemia (greater than 10% loss of blood volume)→ 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.
- ✓Hypovolemia also leads to secretion of renin from kidneys → formation of angiotensin II → ADH release.
- ✓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.
- ✓Arterial baroreceptors "high pressure receptors" in carotid sinus and aortic arch increase ADH Only if changes in ECF volume are great enough to affect ABP.

3. Other factors:

- ✓ Angiotensin II → ↑ ADH release.
- ✓ Stress and sleep → ↑ ADH release.
- ✓ Drugs: morphine& nicotine→ ↑ ADH release.

- ✓ Atrial natriuretic peptide (ANP) → ↓ADH release.
- ✓ Alcohol (ethanol)→ ↓ADH release.
- ✓ Cold → ↓ADH release.


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. Urine is dilute.
- 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 mainly the paraventricular nucleus to release oxytocin.

Source of stimuli :

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

Suckling→ afferent impulses from nipple→ hypothalamus (paraventricular nuclei mainly)→ release of oxytocin→ contraction of myoepithelial cells→ milk letdown.

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

Significance :

Oxytocin stimulates uterine contractions and facilitates labor.

Mechanism :

Cervical dilatation (stretch) by baby head → nerve impulses→ hypothalamus (paraventricular nuclei mainly) → oxytocin release→ uterine contraction→ more cervical dilatation by baby head→ more uterine contraction thus helping delivery of baby.

Positive feedback mechanism.

B) Hormones:

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

C) Other factors:

Secretion of oxytocin is altered by many emotional and stressful stimuli through conditioned reflexes.

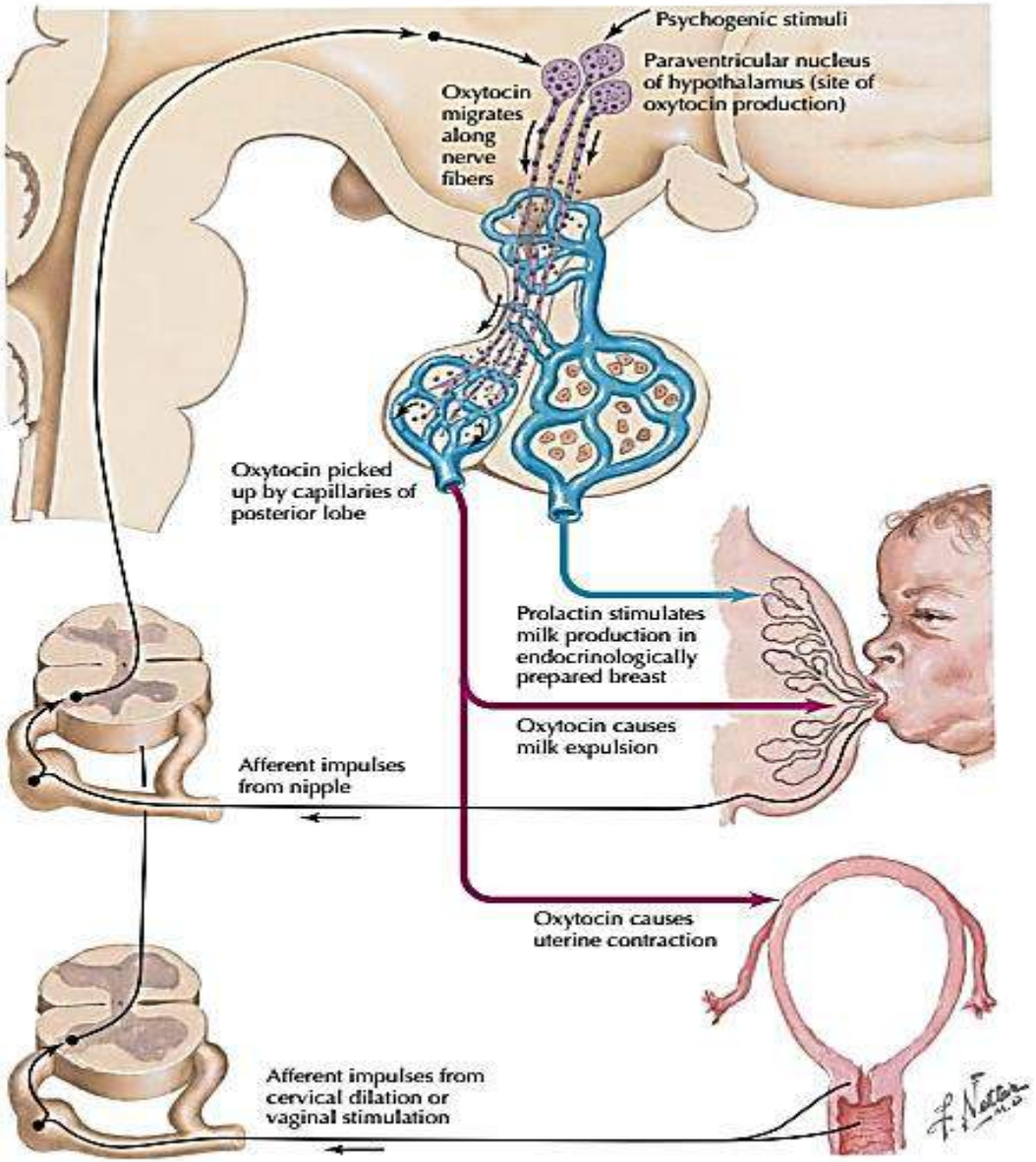


Figure 26.7 **Posterior Pituitary Function (Oxytocin)** Oxytocin is synthesized mainly in the paraventricular nuclei (and also the supraoptic nuclei) of the hypothalamus and is stored and released at the posterior pituitary. Its main functions are to stimulate milk let-down and uterine contraction.


thank
you