# ENDOCRINOLOGY

## Posterior pituitary

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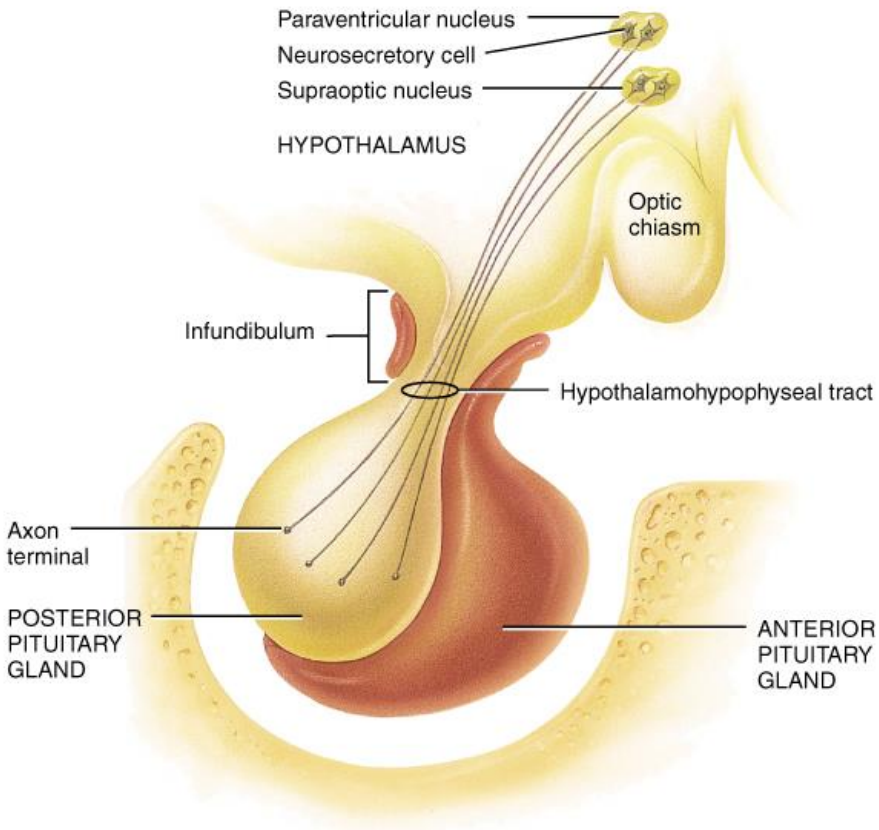
# PHYSIOLOGY OF POSTERIOR PITUITARY GLAND

- Hypothalamic control
- Posterior pituitary hormones
  - ADH
    - Physiological functions
    - Control of secretion
      - Osmotic stimuli
      - Non-osmotic stimuli
  - Oxytocin
    - Physiological functions
    - Control of secretion

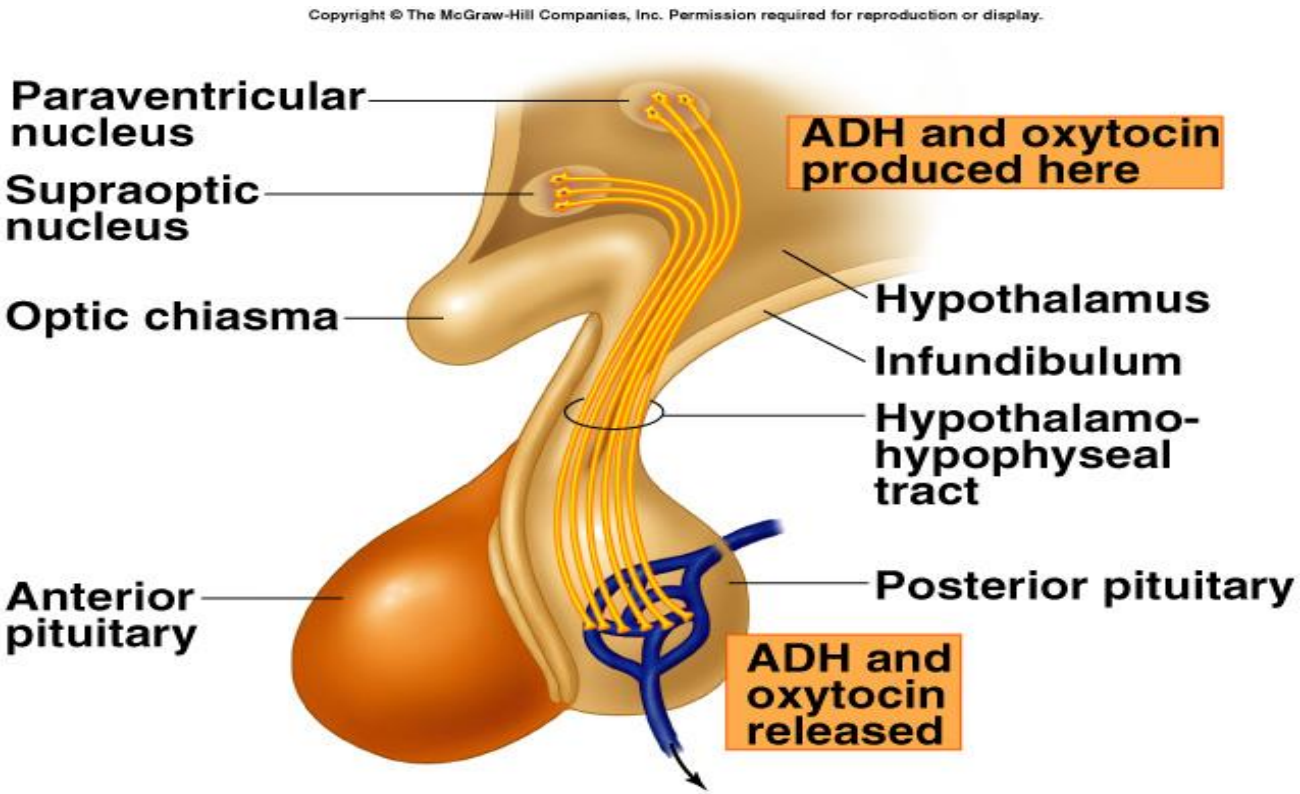
# POSTERIOR PITUITARY GLAND



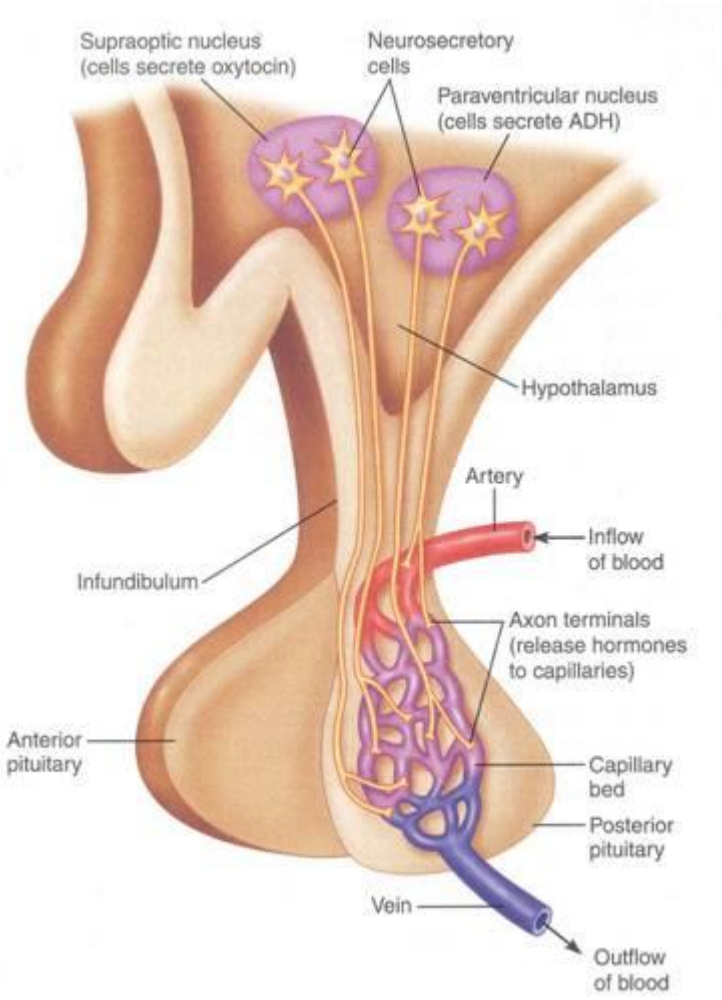

- Does not synthesize hormones
- Consists of axon terminals of hypothalamic neurons

# HYPOTHALAMIC CONTROL OF PITUITARY SECRETIONS

- Secretions of the posterior pituitary are controlled by
  - Nervous signals from hypothalamus



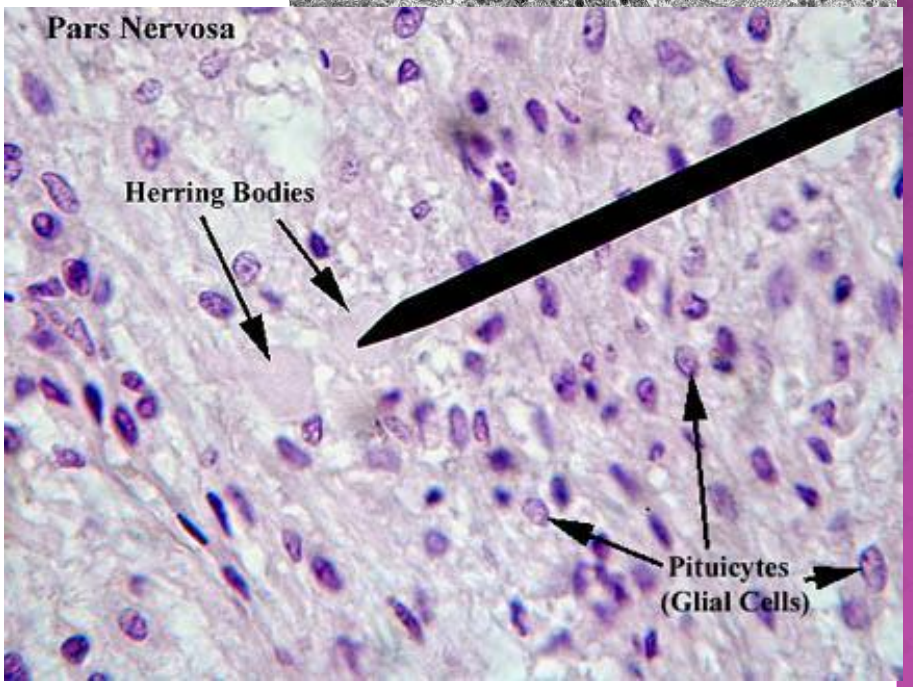
# OXYTOCIN AND VASOPRESSIN ARE MANUFACTURED IN THE HYPOTHALAMUS, BUT RELEASED IN THE POSTERIOR PITUITARY

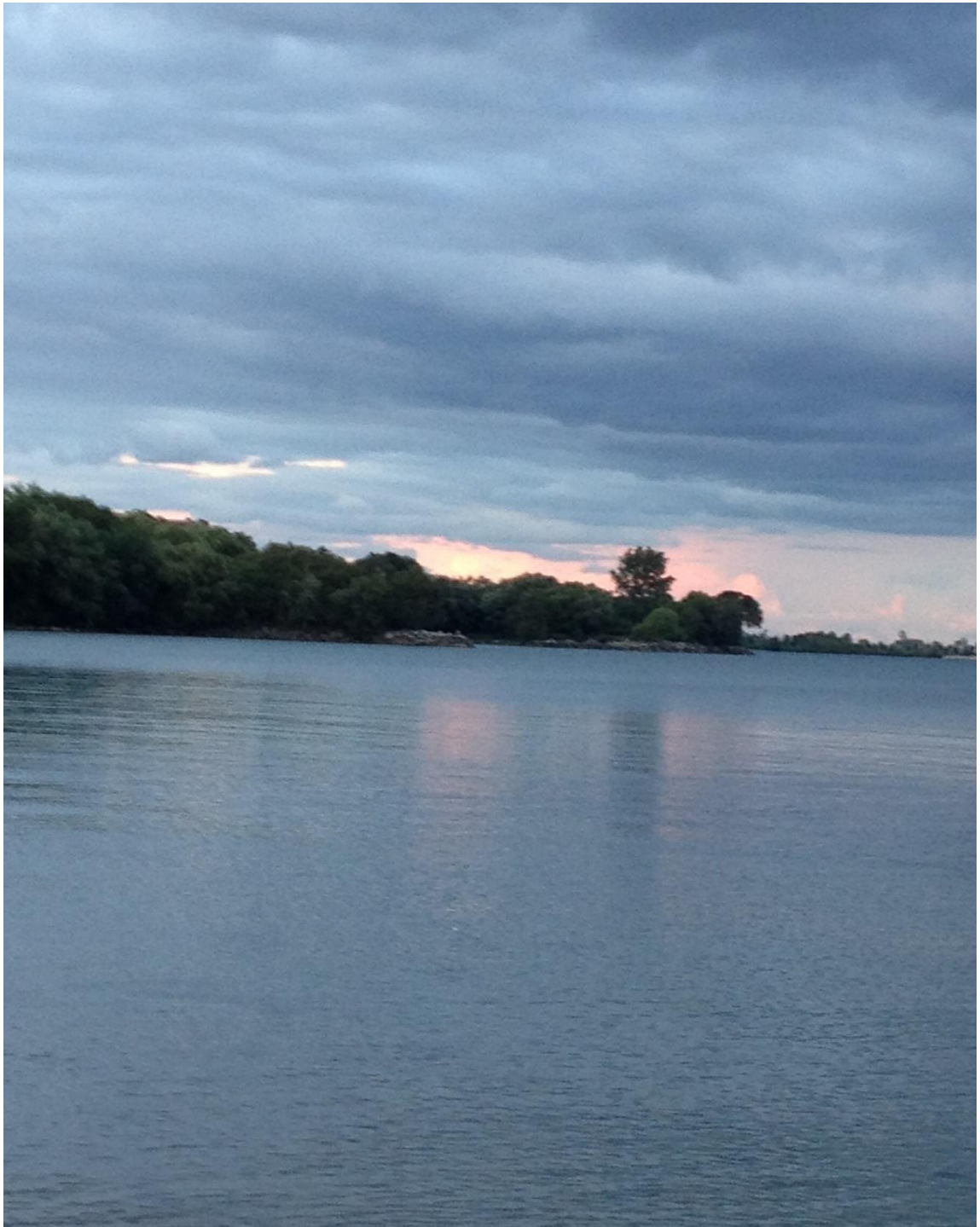


## Herring Body


## Pituicytes function

**It forms physical and chemical barrier between nerve terminal and blood vessels**





# ANTIDIURETIC HORMONE

(vasopressin)

# SYNTHESIS OF ADH

- It is synthesized as pre-prohormone and processed into a nonapeptide (nine amino acids).
- ADH synthesized in the cell bodies of hypothalamic neurons(supraoptic nucleus)
- ADH is stored in the neurohypophysis (posterior pituitary)

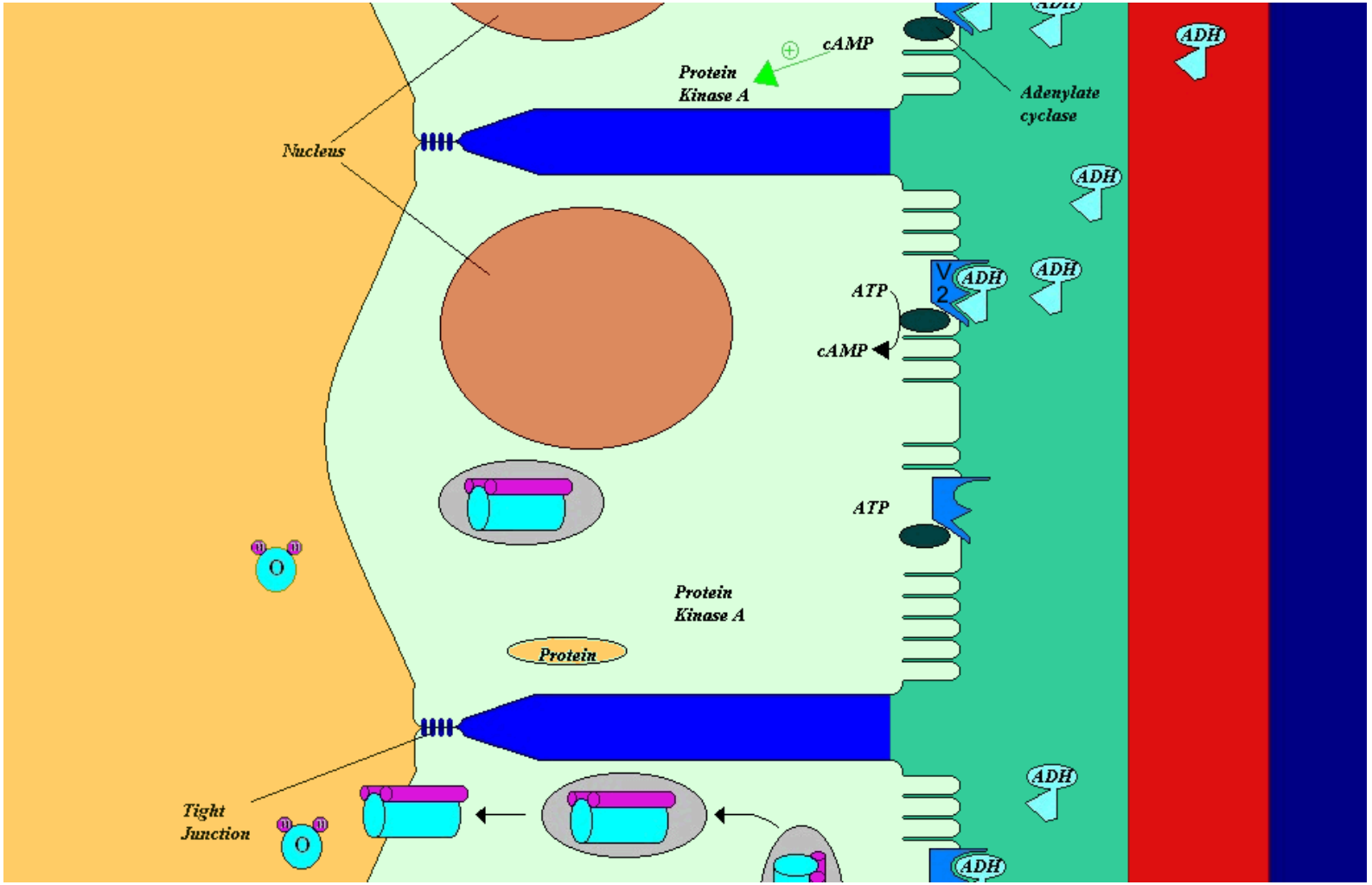
# RECEPTORS OF ADH (VASOPRESSIN)

- There are 3 types of receptors for ADH:
  - $V_{1A}$
  - $V_{1B}$
  - $V_2$
- $V_{1A}$ receptors mediate vasoconstriction
- $V_{1A}$ receptors also found in the liver glycogenolysis
- $V_{1B}$ receptors are unique to anterior pituitary and mediate increased ACTH secretion
- $V_2$ receptors are located in the principle cells in distal convoluted tubule and collecting ducts in the kidneys

# MECHANISM OF ACTION OF ADH: ANTIDIURESIS

- ADH binds to $V_2$ receptors on the peritubular(serosal) surface of cells (principle cells) of the distal convoluted tubules and medullary collecting ducts.
- Via adenylate cyclase/cAMP induces production and insertion of **aquaporin2** into the luminal membrane and enhances permeability of cell to water.
- Increased membrane permeability to water permits back diffusion of solute-free water, resulting in increased urine osmolality (concentrates urine).

# MECHANISM OF ACTION OF ADH



# THE SINGLE MOST IMPORTANT FUNCTION OF ADH IS TO CONSERVE BODY WATER BY REDUCING URINE OUTPUT

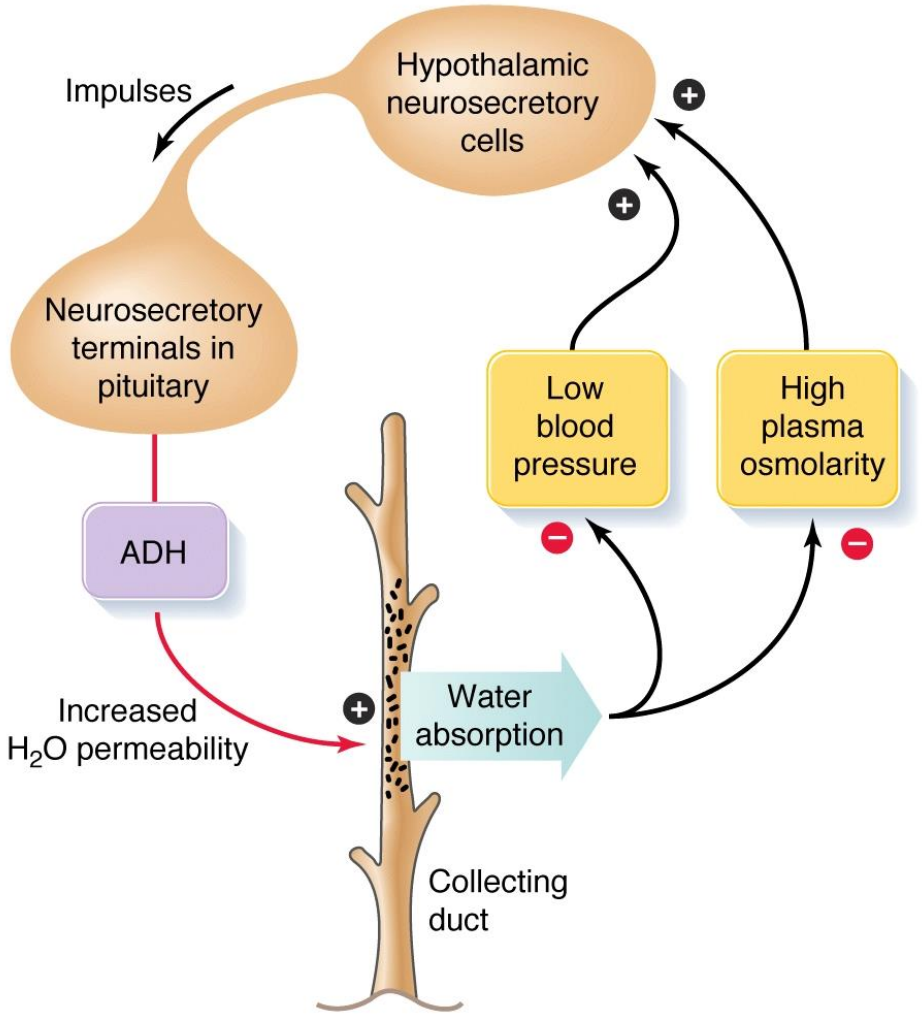
# SECRETION OF ADH
## OSMOTIC STIMULI

- If plasma osmolality is directly increased by administration of solutes, only those solutes that do not freely or rapidly penetrate cell membranes, such as sodium, cause ADH release.
- Conversely, substances that enter cells rapidly, such as urea, do not change osmotic equilibrium and thus do not stimulate ADH release.
- ADH secretion is very sensitive to changes in osmolality.
- Changes of 1-2% result in increased ADH secretion.

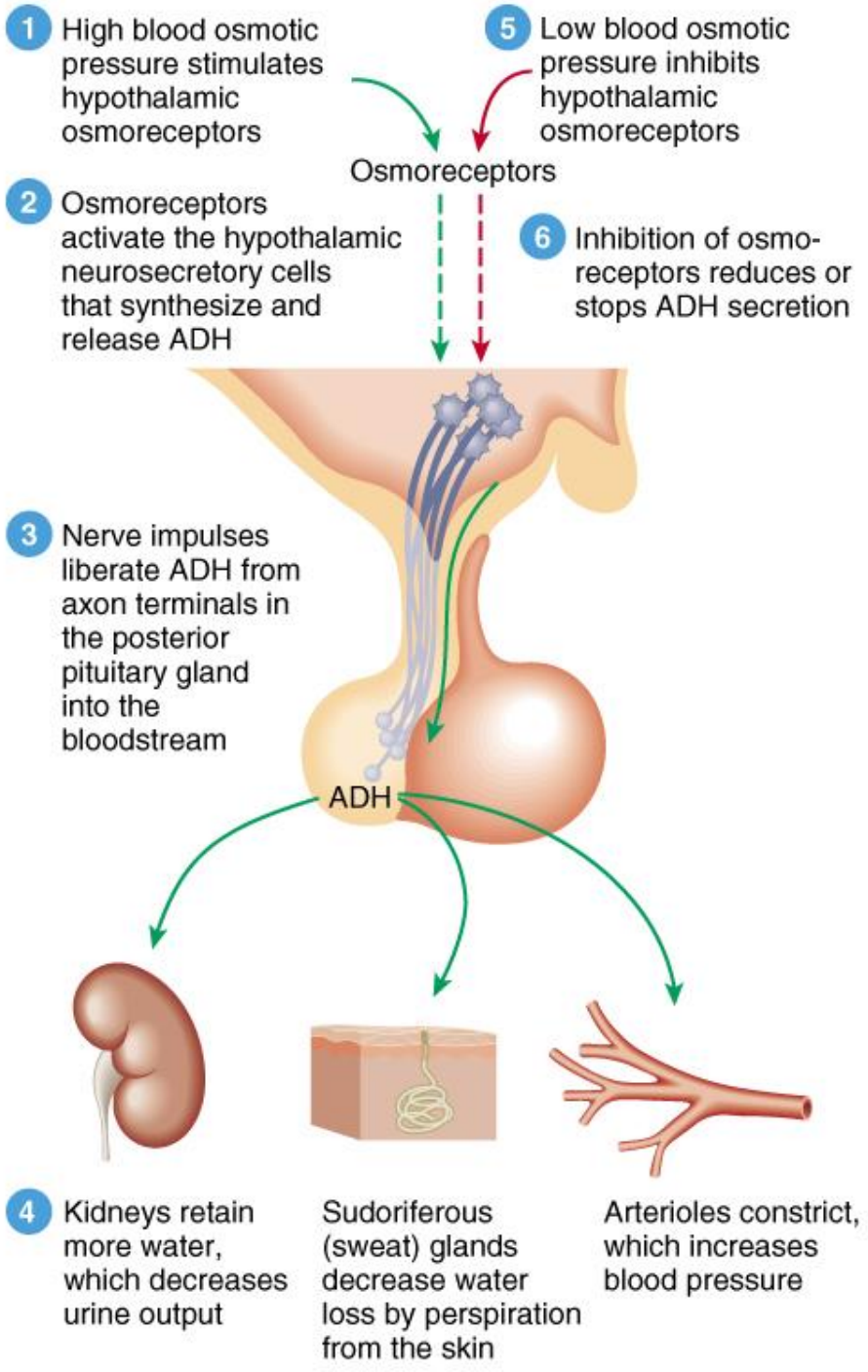
# SECRETION OF ADH
## NON-OSMOTIC STIMULI

- Hypovolemia is perceived by “pressure receptors” -- carotid and aortic baroreceptors, and stretch receptors in left atrium and pulmonary veins.
- Normally, pressure receptors tonically inhibit ADH release.
- Decrease in blood pressure induces ADH secretion by reducing input from pressure receptors.
- The reduced neural input to baroreceptors relieves the source of tonic inhibition on hypothalamic cells that secrete ADH.
- Sensitivity to baroreceptors is less than osmoreceptors- senses 5 to 10% change in volume

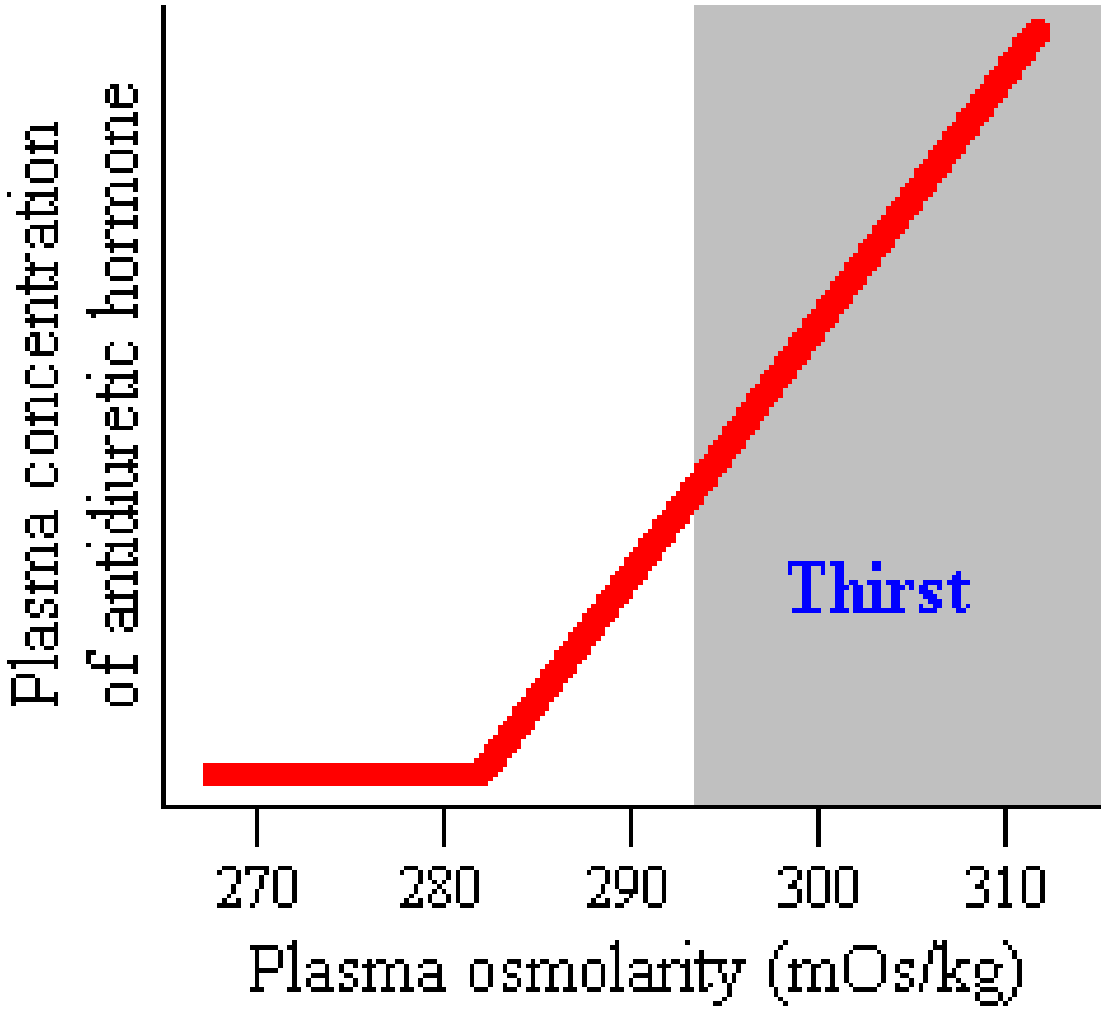
# FUNCTION OF ADH (VASOPRESSIN)



# REGULATION OF ADH



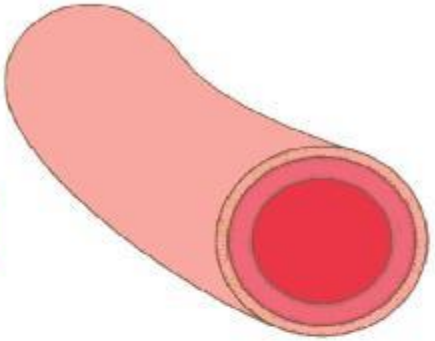

- Dehydration
  - ADH released
- Overhydration
  - ADH inhibited




**Plasma Osmolarity stimulates both ADH release and thirst via OSMORECEOPTORS**

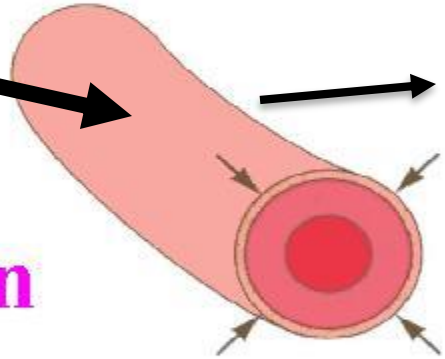
# Effects on Blood Vessels

Normal



**Angiotensin II**

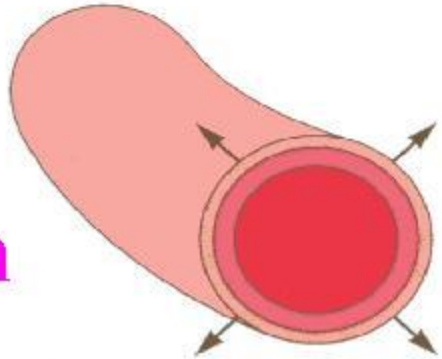
**ADH**



**Increased Blood Pressure**

Vasoconstriction

Vasodilitation



# OTHER STIMULI THAT AFFECT ADH SECRETION

- Stimuli that increase ADH secretion:
  - Pain
  - Nausea
  - Surgical stress
  - Emotional stress
- Stimuli that decrease ADH secretion:
  - Alcohol intake

# TABLE SUMMARIZES THE MAJOR CHARACTERISTICS OF OSMORECEPTORS AND BARORECEPTORS

| Receptors | Osmoreceptors | Baroreceptors |
|---|---|---|
| Location | Anterolateral hypothalamus | Carotid sinus & aortic arch |
| Value Measured | Plasma osmolality | Circulating volume |
| ADH Release Stimulated By | Activation of receptor | Suppression of receptor |
| Change Required for Action | 1% above 280 mosm/kg | 10-15% decrease |
| Resulting Amount of ADH | Small | Large (vasoconstriction) |
| Override Other? | no | yes |

# CONTROL OF ADH RELEASE

- **Osmotic pressure**:
  - Osmoreceptor mediated
  - ↑osmolality → ↑ADH secretion
  - ↓osmolality → ↓ ADH secretion
- **Volume effects**
  - Baroreceptor mediated (vagus nerve)
  - ↑blood pressure → ↓ ADH secretion
  - ↓blood pressure → ↑ ADH secretion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# OXYTOCIN

# FUNCTION OF OXYTOCIN

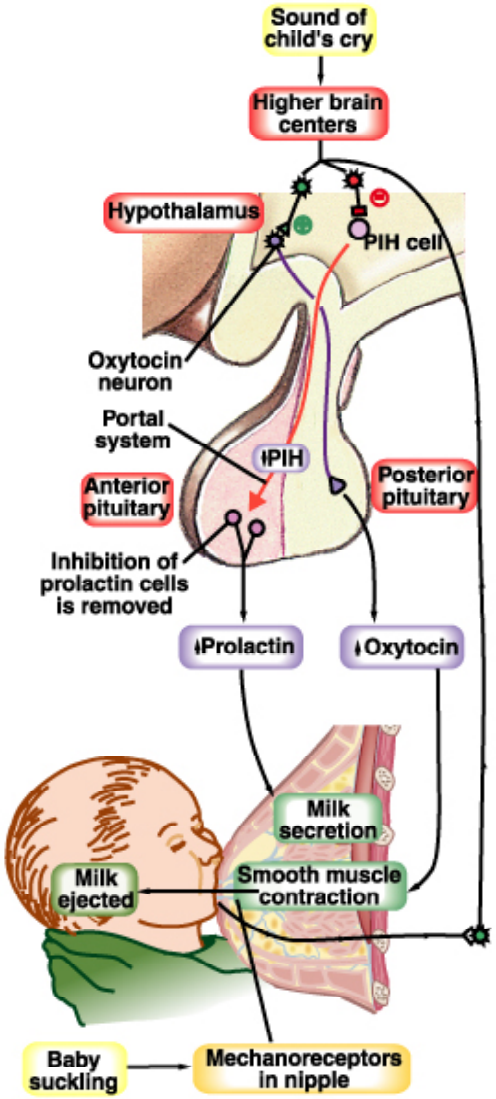
- **Breast-feeding**
  - contracts the myoepithelial cells of the alveoli

  (classic neuroendocrine reflex)

- **Childbirth** (parturition)
  - in late pregnancy, uterine smooth muscle (myometrium) becomes sensitive to oxytocin

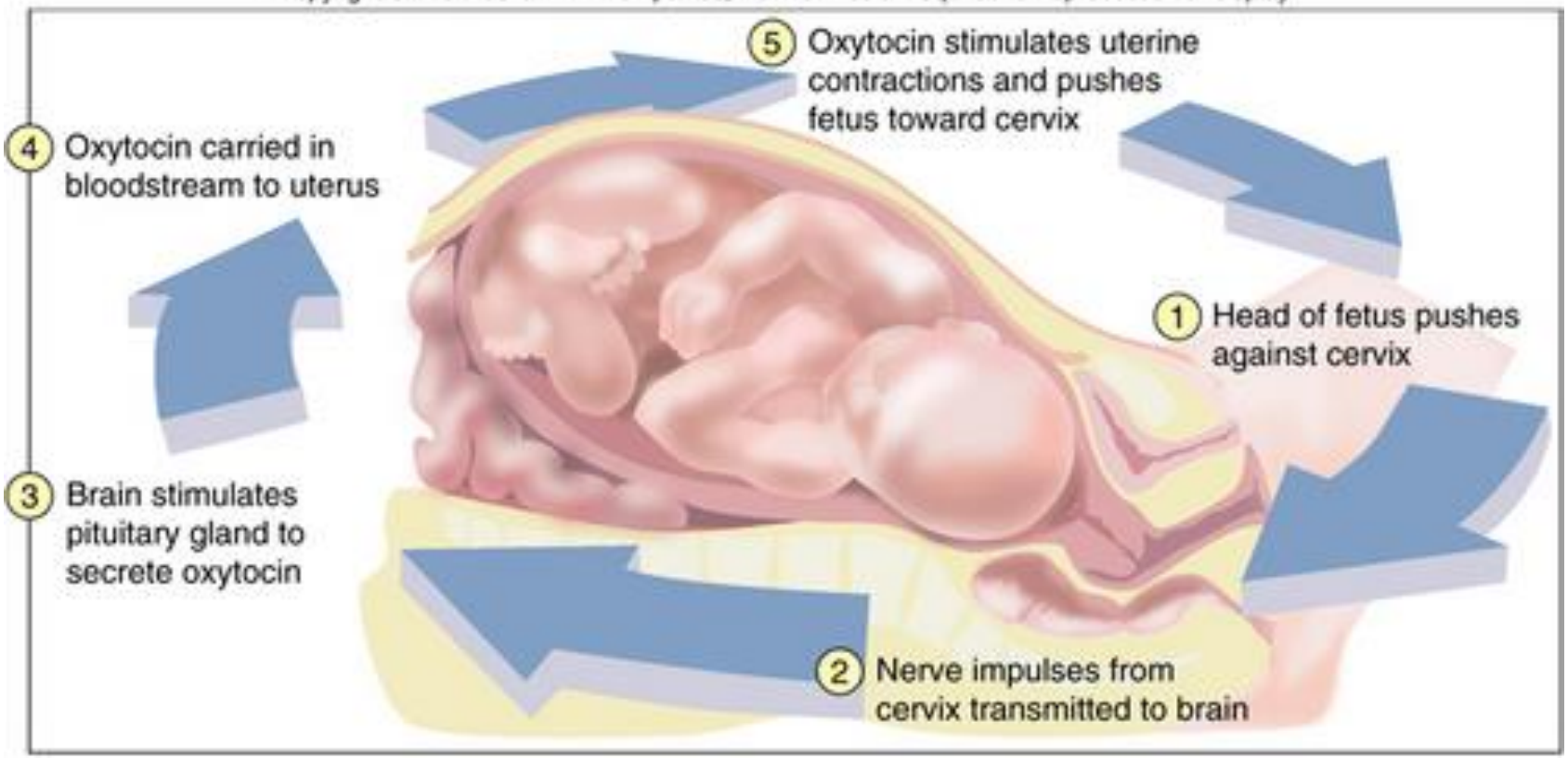
    (positive feedback)

# BREAST FEEDING



# CHILDBIRTH






50 ml
Oxytocin
FERRING - LÉČIVA
Oxytocin

# OTHER STIMULI THAT CONTROL RELEASE OF OXYTOCIN

- In humans, oxytocin is thought to be released during hugging, touching, and orgasm in both sexes.
- Release increased during stress
- Release inhibited by alcohol
- In males secretion increases at time of ejaculation (contraction of smooth muscle of vas deferens)

# OXYTOCIN AND AUTISM

- Autistic group had significantly lower plasma oxytocin levels than in the non-autism group
- Elevated oxytocin was associated with higher scores on social and developmental measures for the non-autistic children
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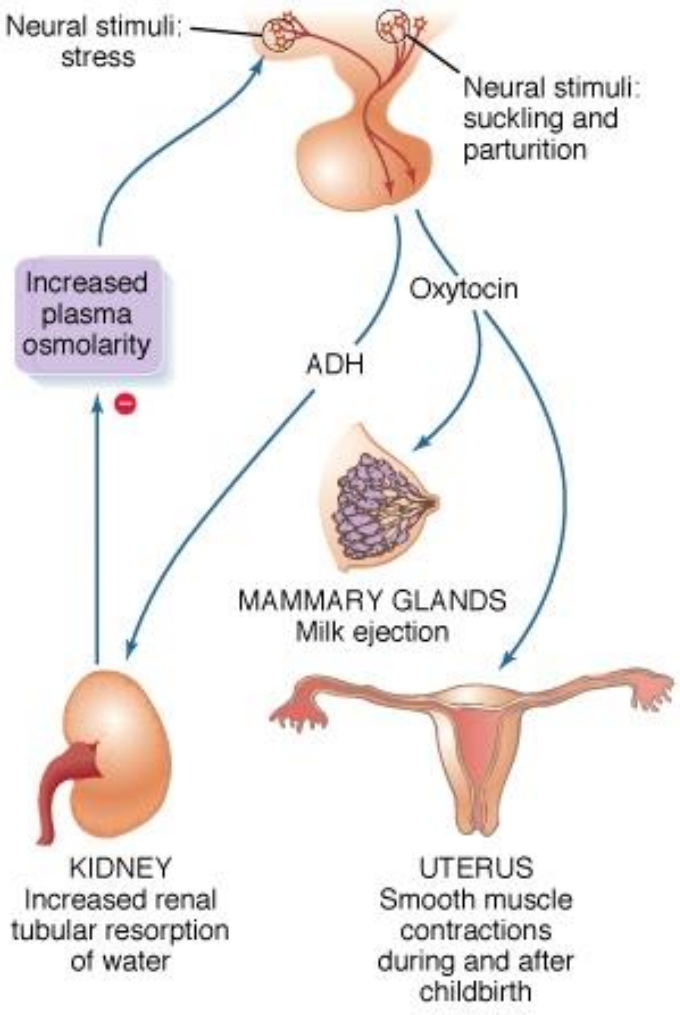
# SUMMARY OF POSTERIOR PITUITARY HORMONES ACTIONS

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