

The Endocrine Physiology

Posterior Pituitary

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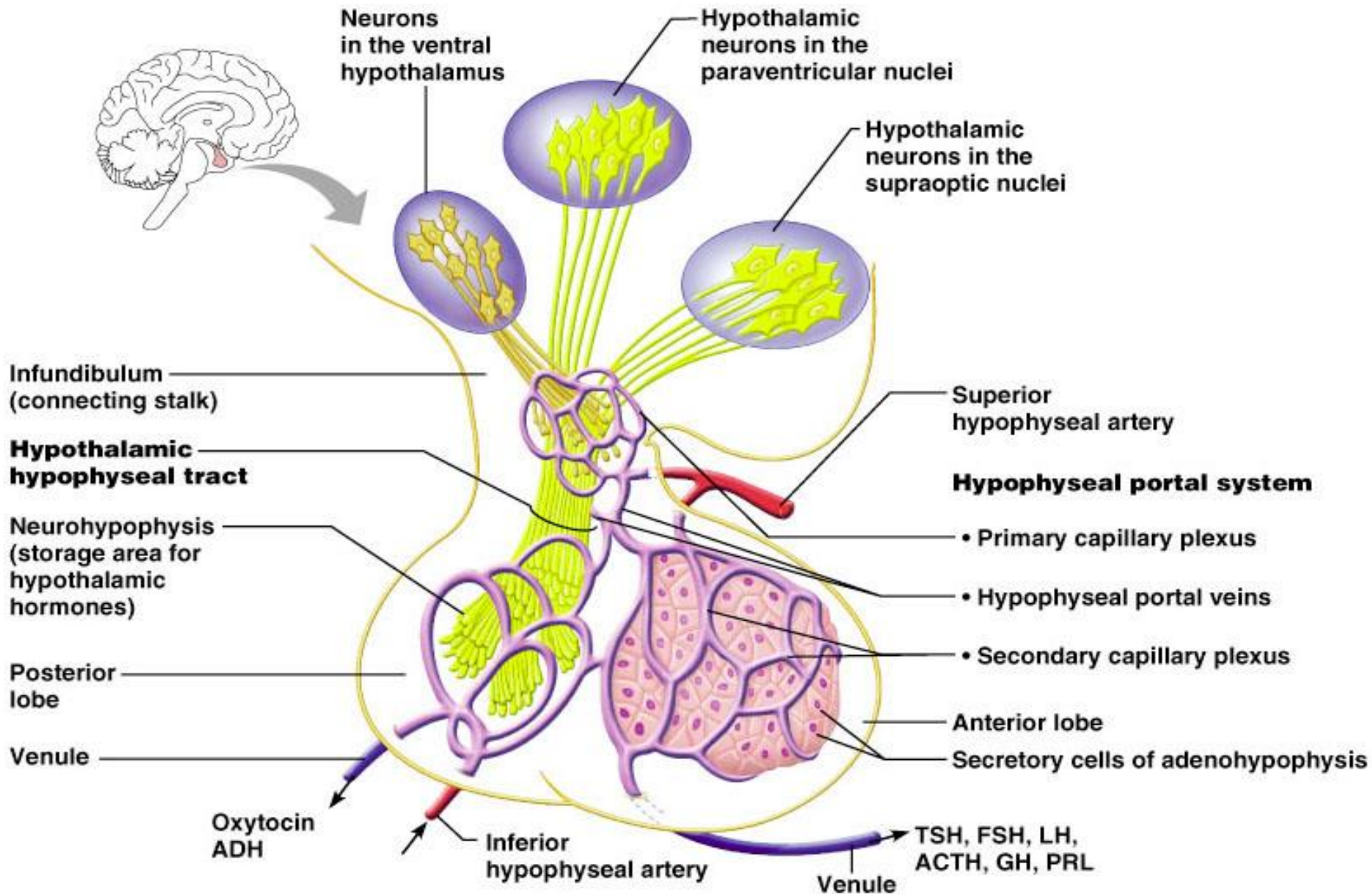
Learning Objectives

- Describe the posterior pituitary relationship with the hypothalamus
- List the target organs and functional effects of oxytocin.
- Name the stimuli for oxytocin release in relation to its reproductive and lactation functions.
- List the target cells for vasopressin and explain why vasopressin is also known as antidiuretic hormone.
- Describe the stimuli and mechanisms that control vasopressin secretion.
- Identify disease states caused by a) over-secretion, and b) under-secretion of vasopressin and list the principle symptoms of each.

The Posterior Pituitary and Hypothalamic Hormones

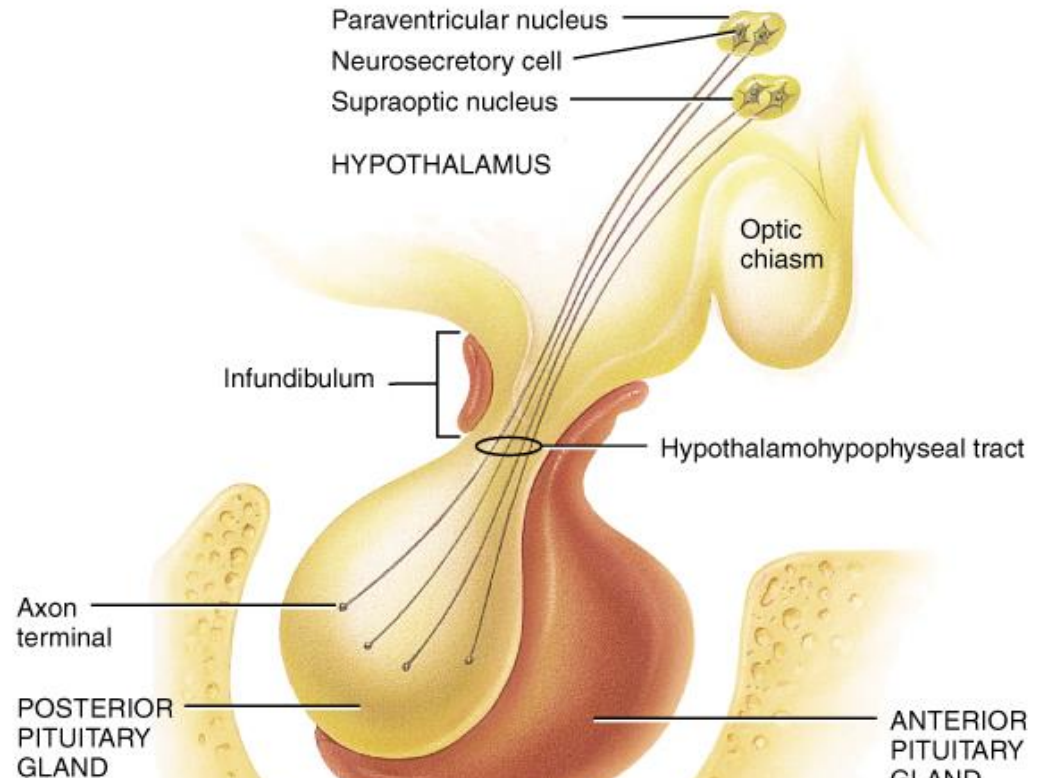
- The posterior lobe is a downgrowth of hypothalamic **neural tissue**
- Has a neural connection with the hypothalamus (*hypothalamic-hypophyseal tract*)
- Nuclei of the hypothalamus synthesize oxytocin and antidiuretic hormone (ADH)
- These hormones are transported to the posterior pituitary

Pituitary (Hypophysis)



Posterior pituitary gland

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



Antidiuretic Hormone (ADH)

(vasopressin)

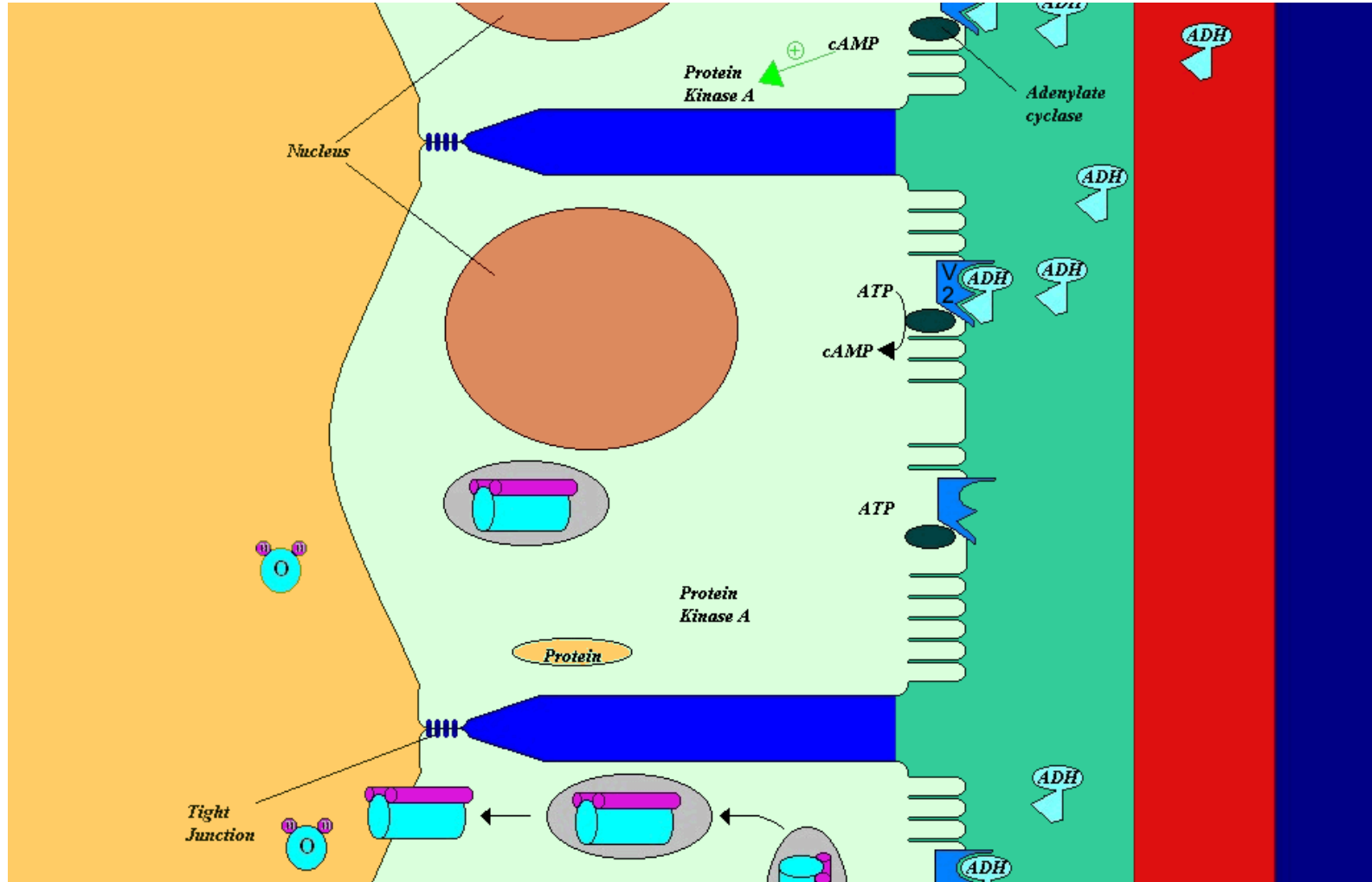
Synthesis of ADH

- It is synthesized as pre-prohormone and processed into a nonapeptide (9 amino acids)
- ADH synthesized in the cell bodies of hypothalamic neurons(**supraoptic nucleus**)
- ADH is stored in the posterior pituitary

Receptors of ADH (vasopressin)

- There are 2 types of receptors for ADH:
 - V_1
 - V_2
- ⊙ **V1** receptors mediate vasoconstriction
- ⊙ **V2** receptors are located in the *principle cells* in distal convoluted tubule and collecting ducts in the kidneys

Mechanism of action of ADH



Control of ADH Release

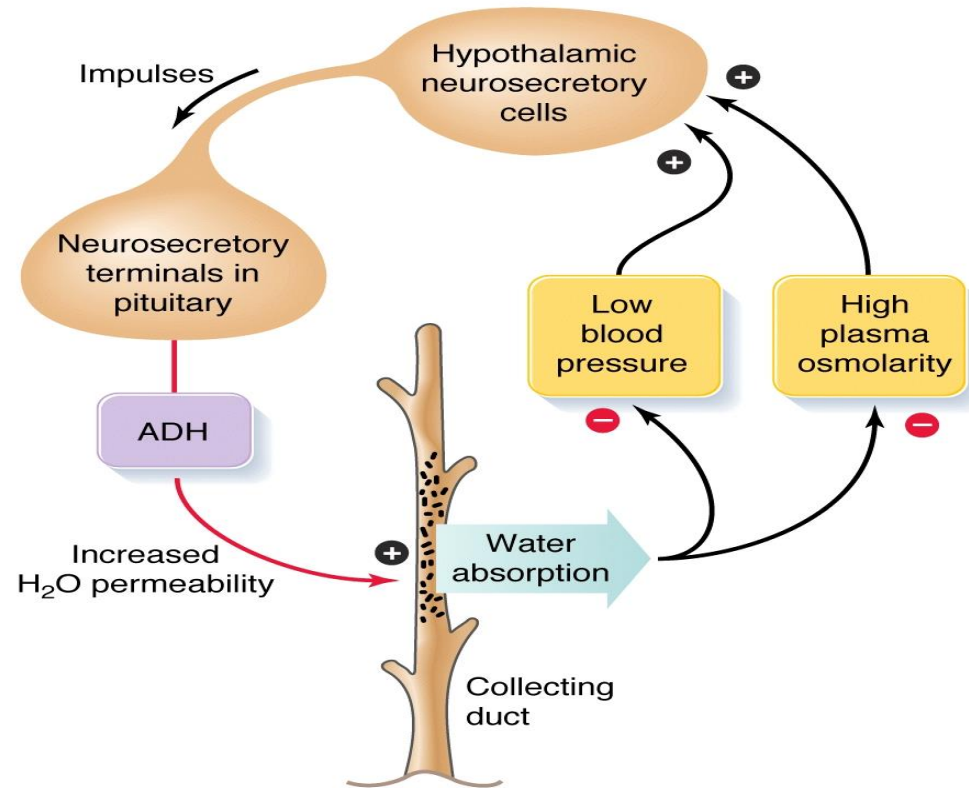
- **Osmotic pressure:**
 - Osmoreceptors in the hypothalamus:
 - \uparrow osmotic pressure \rightarrow \uparrow ADH secretion
 - \downarrow osmotic pressure \rightarrow \downarrow ADH secretion
- **Blood volume :**
 - **Baroreceptor** in carotid artery and aortic arch, and **Stretch receptors** in left atrium:
 - \uparrow blood pressure \rightarrow \downarrow ADH secretion
 - \downarrow blood pressure \rightarrow \uparrow ADH secretion

Regulation of ADH

Hypothalamus receives feedback from:

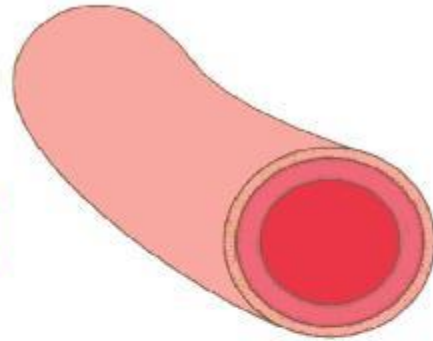
- Osmoreceptors
- Aortic arch baroreceptors
- Carotid baroreceptors
- Atrial stretch receptors

Any increase in osmolality or decrease in blood volume will stimulate ADH secretion from posterior pituitary.

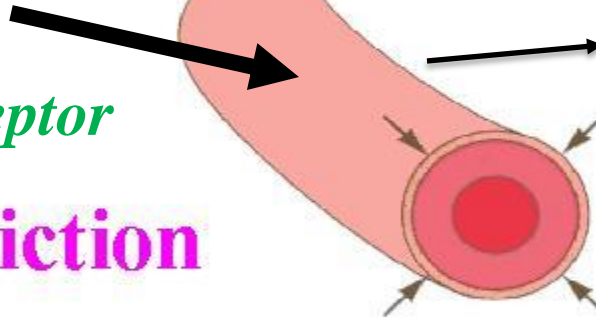


Effects on Blood Vessels

Normal



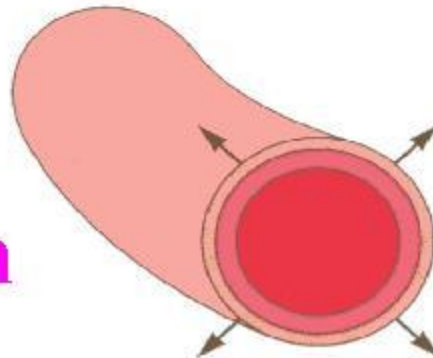
ADH
V1 receptor



*Increased
Blood
Pressure*

Vasoconstriction

Vasodilation



Oxytocin



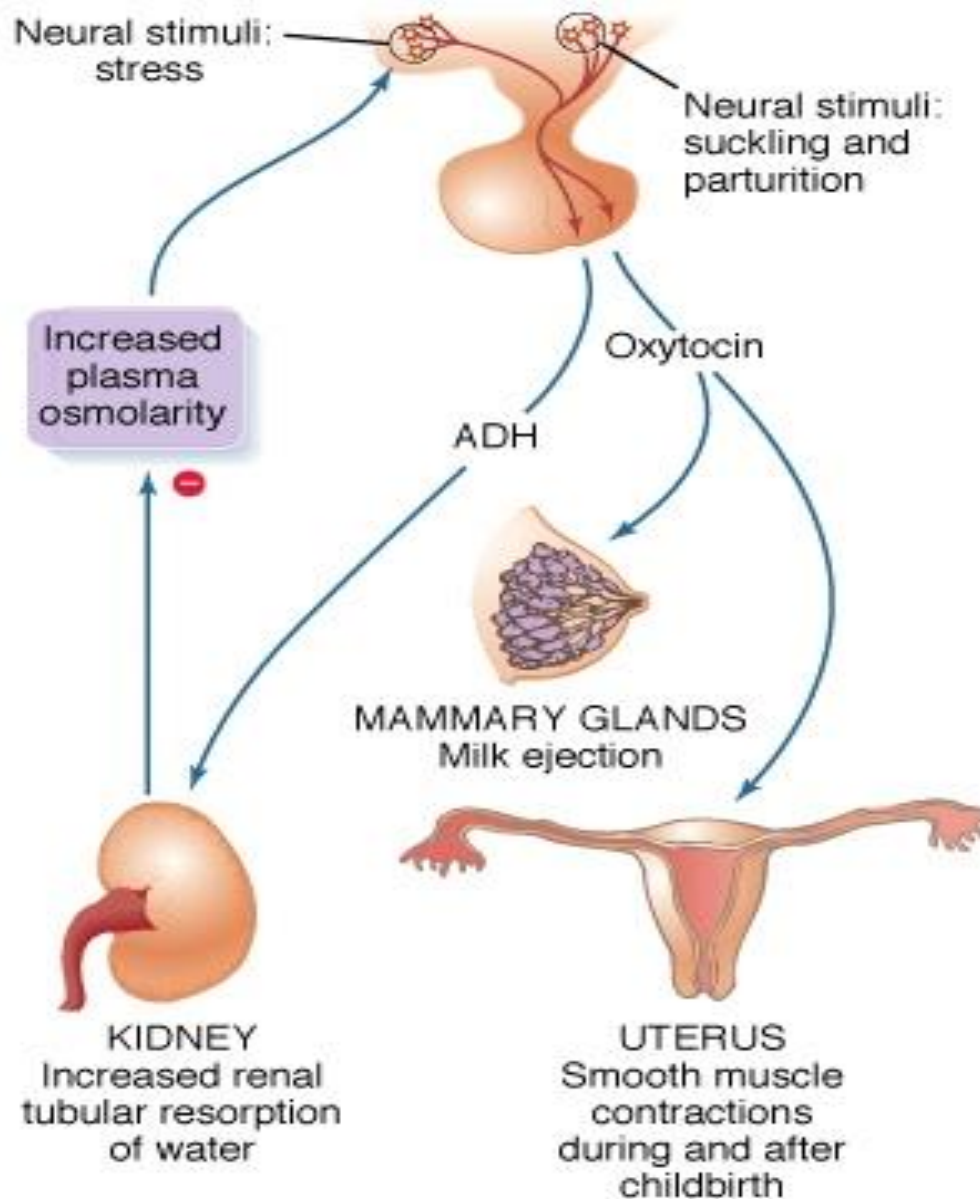
Synthesis of Oxytocin

- Oxytocin is synthesized in the cell bodies of hypothalamic neurons (paraventricular nucleus)
- Oxytocin is stored in the posterior pituitary

Functions of oxytocin

- Oxytocin is a strong stimulant of uterine contraction
- Regulated by a positive feedback mechanism
- This leads to increased intensity of uterine contractions, ending in birth
- Oxytocin triggers milk ejection (“letdown” reflex)
Contracts the *myoepithelial cells* of the alveoli

Summary of posterior pituitary hormones actions



ADH Disorders

- **Diabetes Insipidus:**

Neurogenic (central): (failure of neurohypophysis to synthesize or secrete ADH)

Nephrogenic: (failure of the kidney to respond appropriately to ADH)

- Syndrome of Inappropriate Antidiuretic Hormone
(SIADH)

DIABETES INSIPIDUS

- **DI is a disorder resulting from deficiency of anti-diuretic hormone (ADH) or its action and is characterized by the passage of copious amounts of dilute urine.**
- **It must be differentiated from other polyuric states such as primary polydipsia & osmotic diuresis. Central DI is due to failure of the pituitary gland to secrete adequate ADH.**

DIABETES INSIPIDUS /2

- **Nephrogenic DI results when the renal tubules of the kidneys fail to respond to circulating ADH.**
- **The resulting renal concentration defect leads to the loss of large volumes of dilute urine. This causes cellular and extracellular dehydration and hypernatremia.**

CAUSES OF CENTRAL DI

- **IDIOPATHIC (30% OF CASES)**
- **Benign or malignant tumors 25%**
- **INFECTIONS (ENCEPHALITIS, TB, etc)**
- **SKULL SURGERY**
- **TRAUMA**

CAUSES OF CENTRAL DI (2)

- **AUTOIMMUNE ASSOCIATED WITH THYROIDITIS**
- **FAMILIAL: 2 TYPES AD & X-LINKED INHERITANCE**
- **WOLFRAM SYNDROME (ALSO KNOWN AS DIDMOAD SYNDROME) CHARACTERIZED BY DI, DM, NERVE DEAFNESS AND OPTIC ATROPHY.**

CAUSES OF NEPHROGENIC DI

- **PRIMARY FAMILIAL: X-LINKED RECESSIVE THAT IS SEVERE IN BOYS & MILD IN GIRLS**
- **SECONDARY TO:**
- **CHRONIC PYELONEPHRITIS**
- **HYPOKALEMIA**
- **HYPERCALCEMIA**
- **SICKLE CELL DISEASE**
- **PROTEIN DEPRIVATION**

CLINICAL FEATURES

- **POLYURIA, POLYDIPSIA & THIRST**
- **NOCTURIA OR NOCTURNAL ENURESIS**
- **HYPERNATREMIC DEHYDRATION**
- **ANOREXIA, CONSTIPATION**
- **HYPERTHERMIA & LACK OF SWEATING**

TREATMENT

- **DESMOPRESSIN (DDAVP) A SYNTHETIC ANALOG IS SUPERIOR TO NATIVE AVP BECAUSE:**
- **IT HAS LONGER DURATION OF ACTION (8-10 h vs 2-3 h)**
- **MORE POTENT**
- **ITS ANTIDIURETIC ACTIVITY IS 3000 TIMES GREATER THAN ITS PRESSOR ACTIVITY**

TREATMENT OF NEPHROGENIC DI

- **PROVISION OF ADEQUATE FLUIDS & CALORIE**
- **LOW SODIUM DIET**
- **DIURETICS**
- **HIGH DOSE OF DDAVP**
- **CORRECTION OF UNDERLYING CAUSE**