

## **The Endocrine Physiology**

# **Posterior Pituitary**

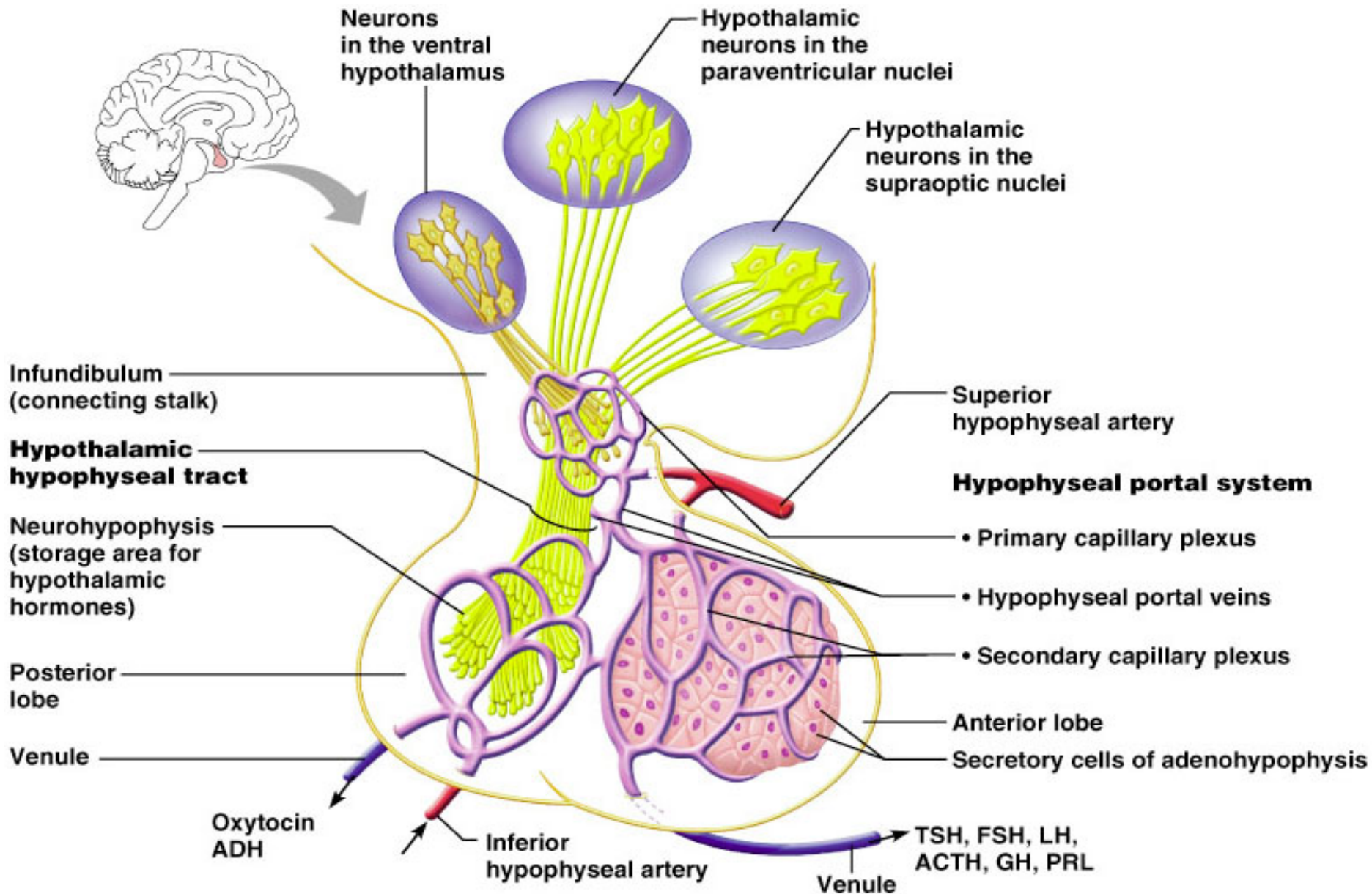
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# Learning Objectives

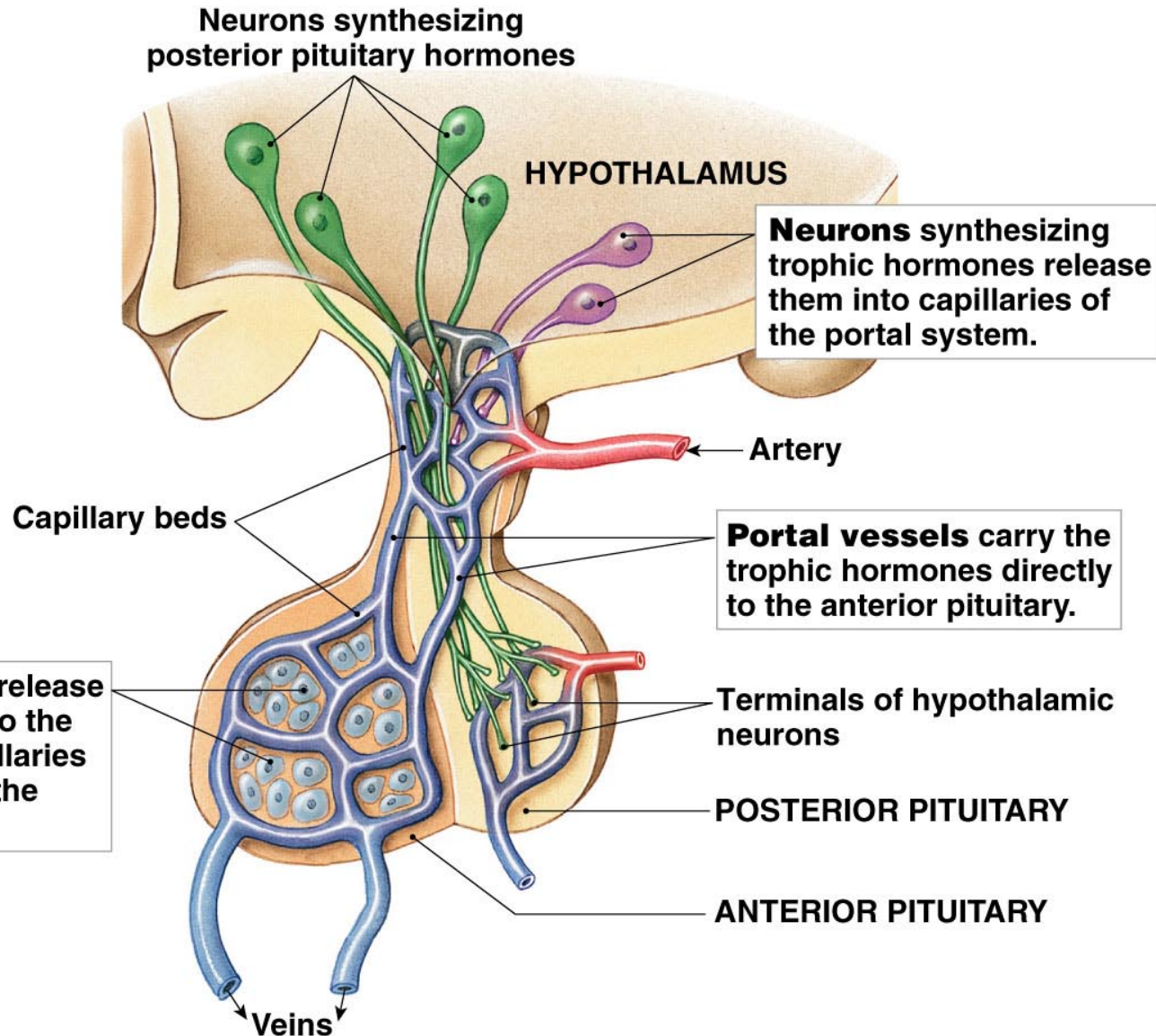
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- Describe the posterior pituitary relationship with the hypothalamus
- List the target organs and functional effects of oxytocin.
- Name the stimuli for oxytocin release in relation to its reproductive and lactation functions.
- List the target cells 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.

# Pituitary (Hypophysis)



# Pituitary (Hypophysis)



# 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)
- Their axons terminate in the posterior pituitary

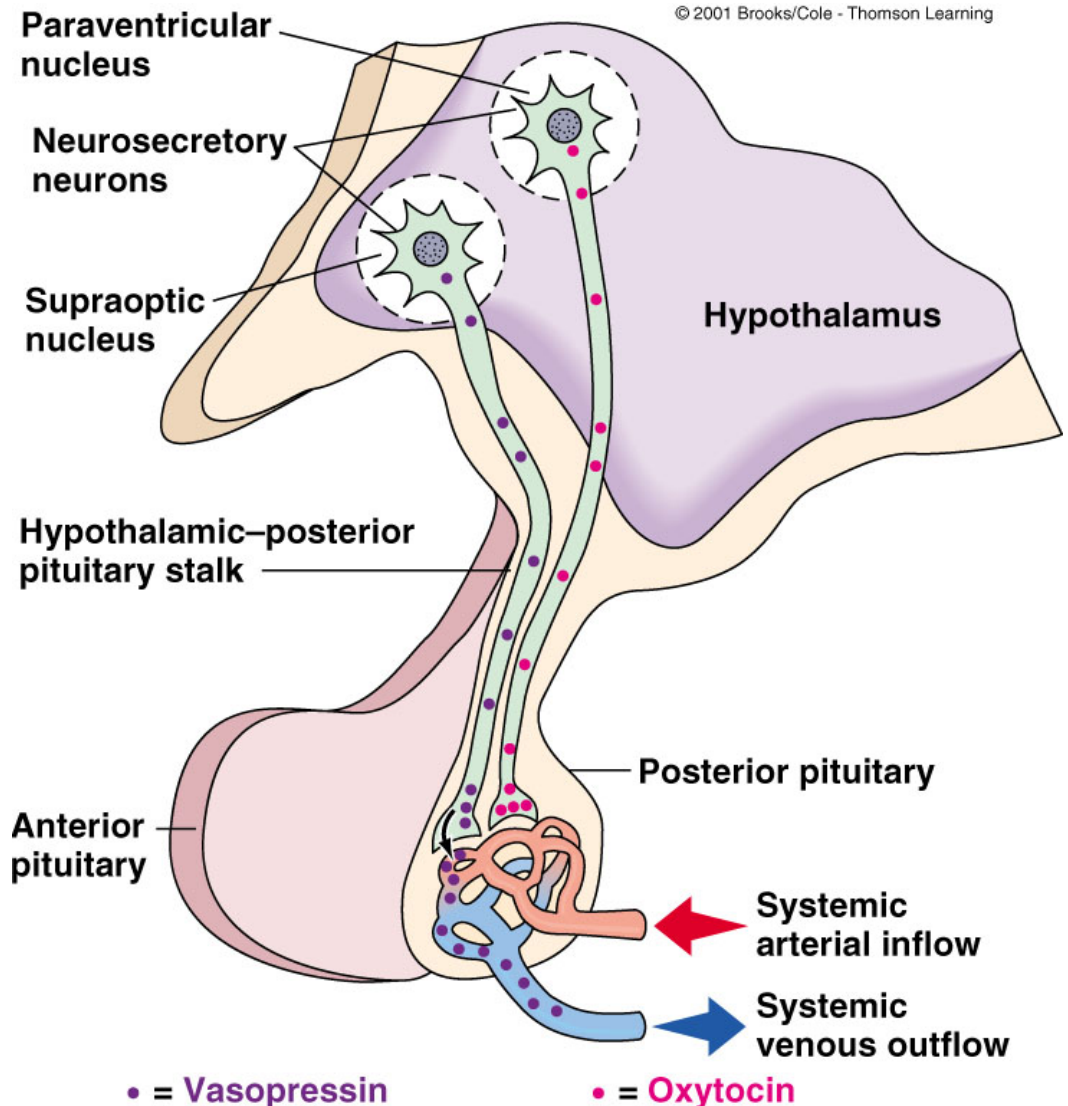
# Oxytocin and Vasopressin

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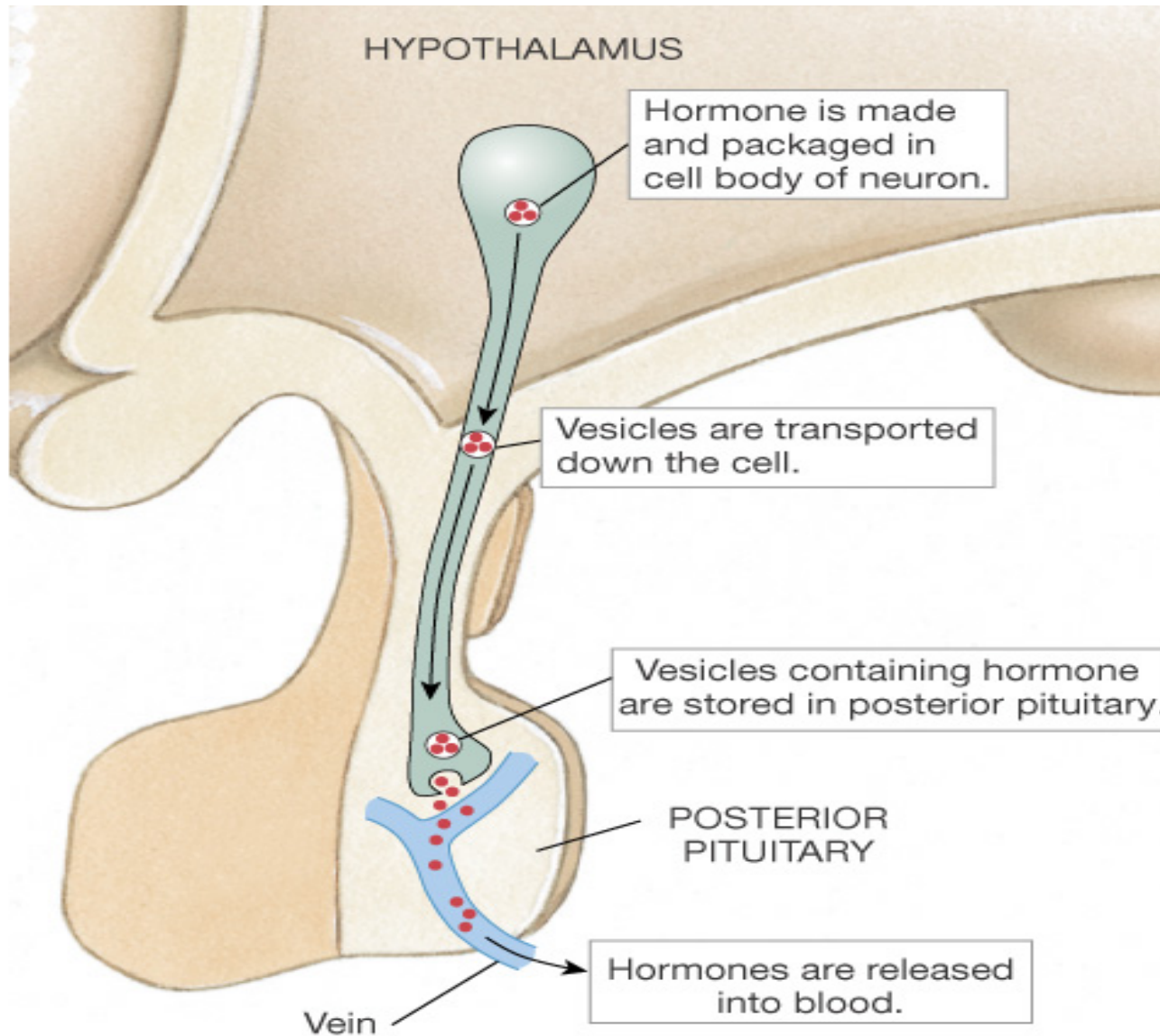
- **Vasopressin:** Cys-Tyr-Phe-Gln-Asn-Cys-Pro-Arg-GlyNH<sub>2</sub>
- **Oxytocin:** Cys-Tyr-Ile-Gln-Asn-Cys-Pro-Leu-GlyNH<sub>2</sub>

# Posterior pituitary

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



# Secretion of Posterior Pituitary Hormones





# Oxytocin



# Synthesis of Oxytocin

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

# Functions of oxytocin

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- 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
- Increases contraction of smooth muscle of the vas deferens, helping in the ejaculation process.

# **Antidiuretic Hormone (ADH)**

(vasopressin)

# Synthesis of ADH

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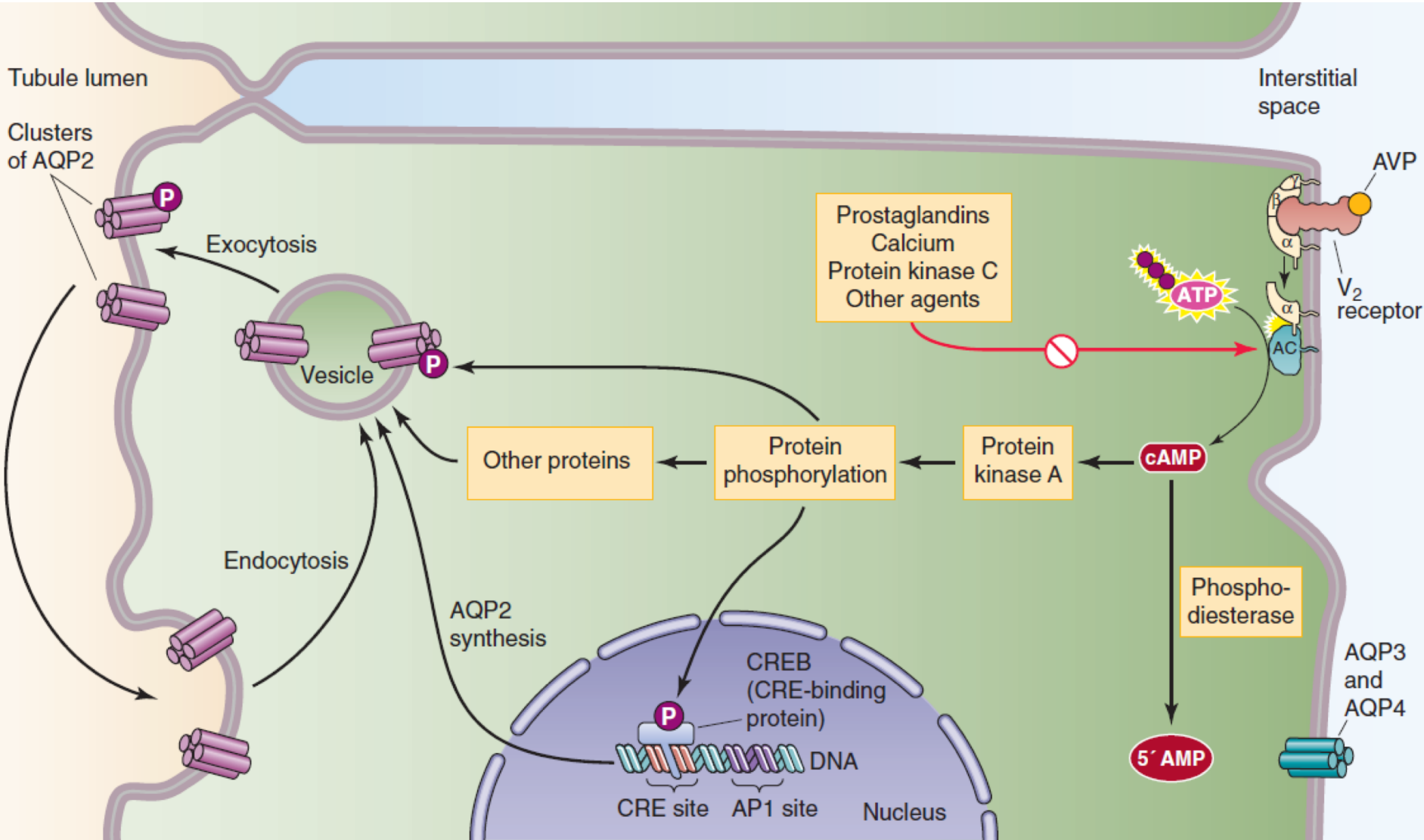
- It is synthesized as pre-prohormone and processed into a nonapeptide (9 amino acids)
- ADH synthesized in the cell bodies of hypothalamic neurons([supraoptic nucleus](#))
- ADH is stored in the posterior pituitary

# Receptors of ADH (vasopressin)

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- 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 left atrium:
  - $\uparrow$ blood pressure  $\rightarrow$   $\downarrow$  ADH secretion
  - $\downarrow$ blood pressure  $\rightarrow$   $\uparrow$  ADH secretion
- Pain, fear, trauma, and stress

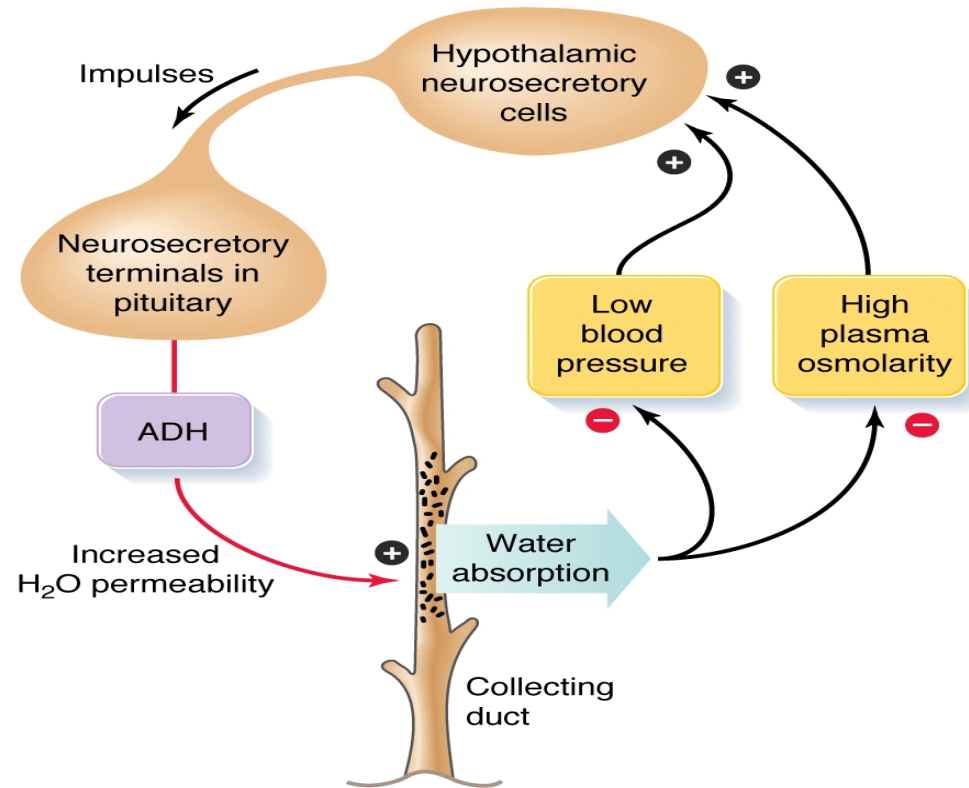


# Regulation of ADH

Hypothalamus receives feedback from:

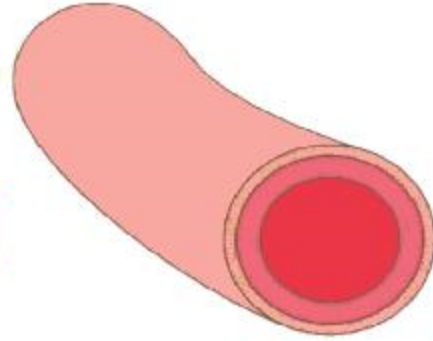
- Osmoreceptors
- Aortic arch baroreceptors
- Carotid baroreceptors
- Atrial stretch receptors

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



# Effects on Blood Vessels

Normal



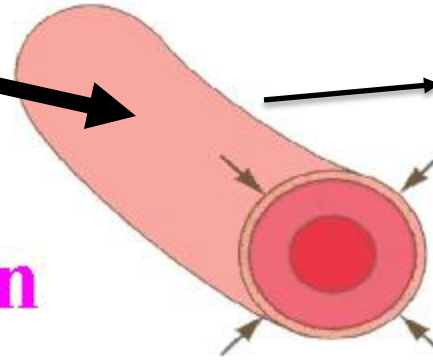
*ADH*

*V1 receptor*

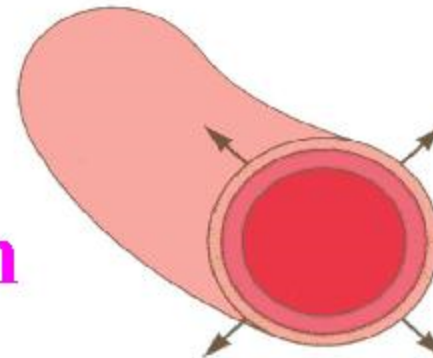


*Increased  
Blood  
Pressure*

Vasoconstriction



Vasodilation



# ADH Disorders

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- **Diabetes Insipidus:**

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

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

- Syndrome of Inappropriate Antidiuretic Hormone (**SIADH**)

# DIABETES INSIPIDUS

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- 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 producing adequate ADH.

# DIABETES INSIPIDUS

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- 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.

# Treatment

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- 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

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

# Summary of posterior pituitary hormones actions

