

# Endocrinology

## Posterior Pituitary

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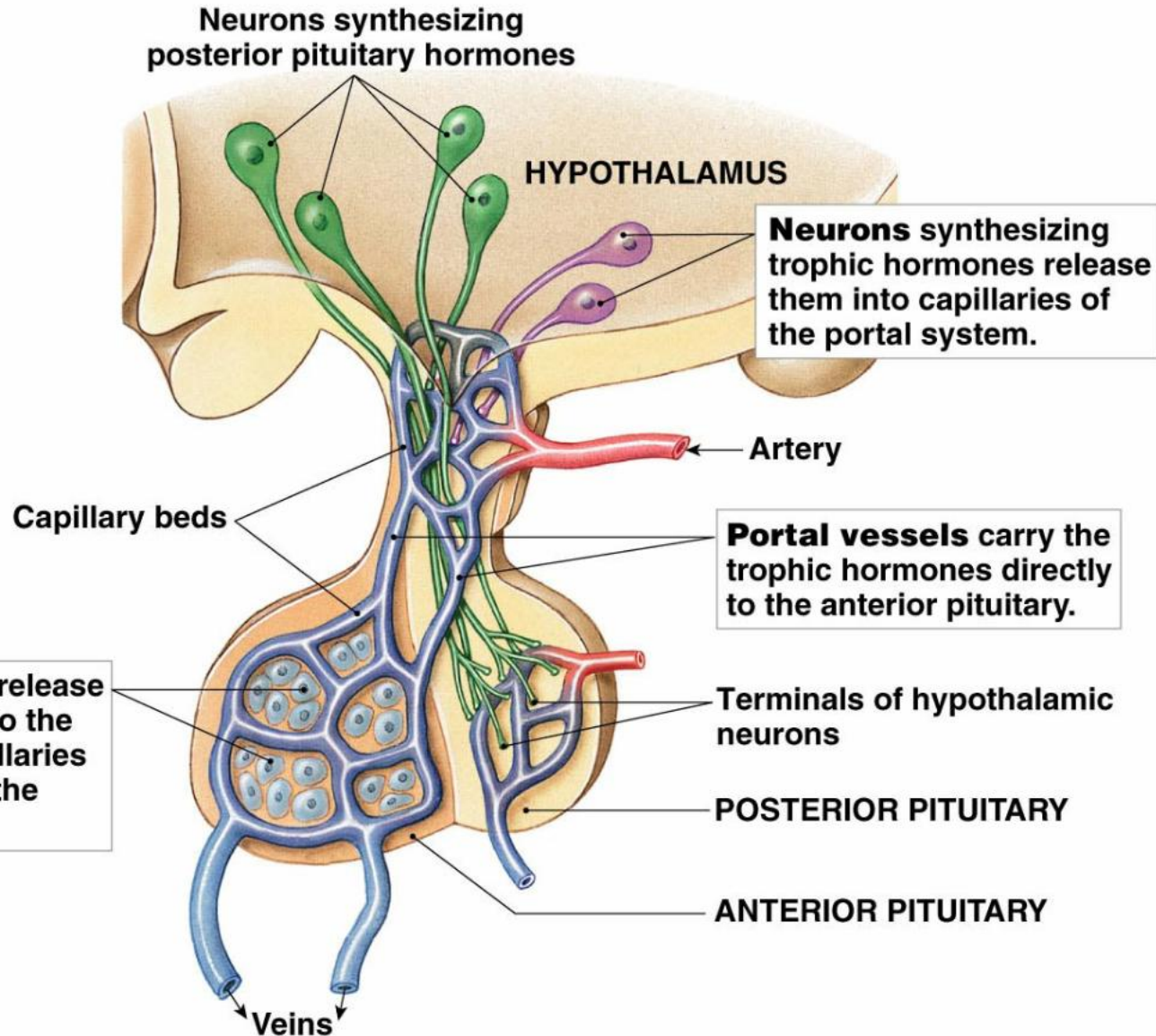
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# Learning Objectives

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

# Pituitary (Hypophysis)



# The Posterior Pituitary & 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.

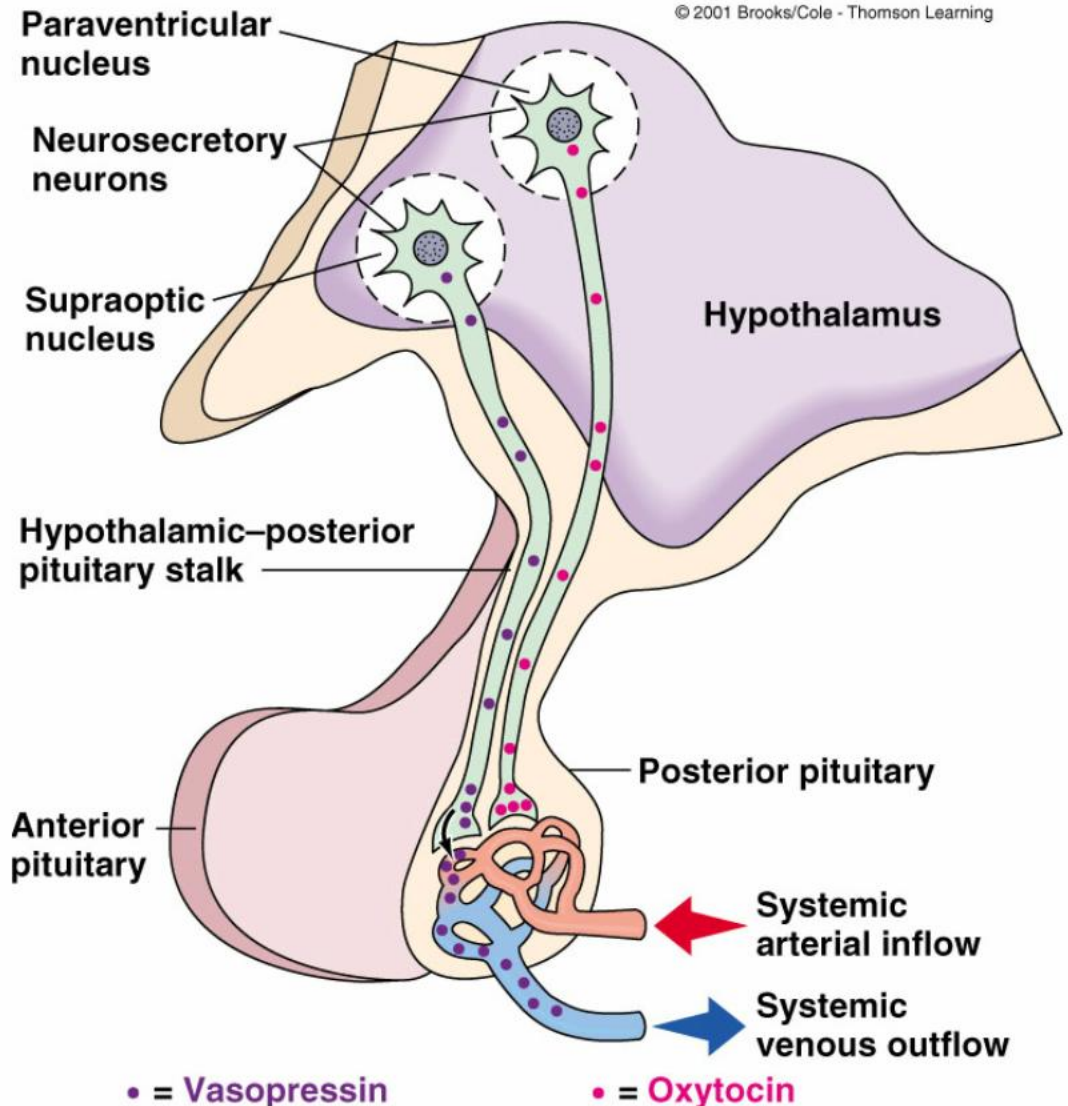
# Oxytocin and Vasopressin

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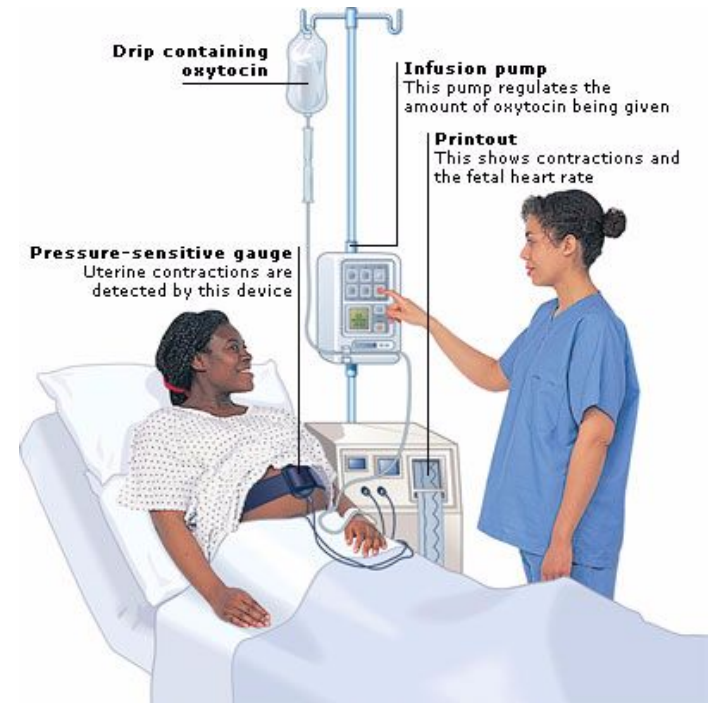
- **Vasopressin:** Cys-Tyr-Phe-Gln-Asn-Cys-Pro-Arg-GlyNH<sub>2</sub>
- **Oxytocin:** Cys-Tyr-Ile-Gln-Asn-Cys-Pro-Leu-GlyNH<sub>2</sub>

# Posterior Pituitary

- Does not synthesize Hormones.
- Consists of axon terminals of hypothalamic neurons.



# Oxytocin



# Synthesis of Oxytocin

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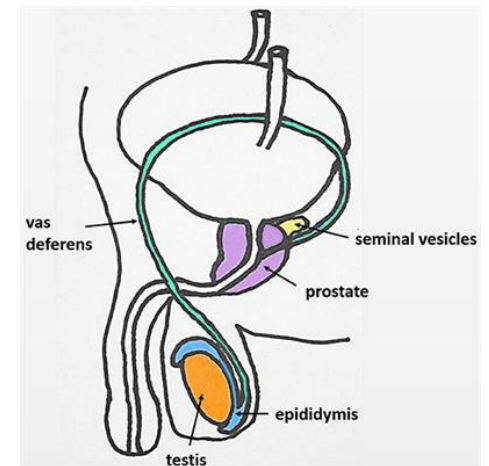
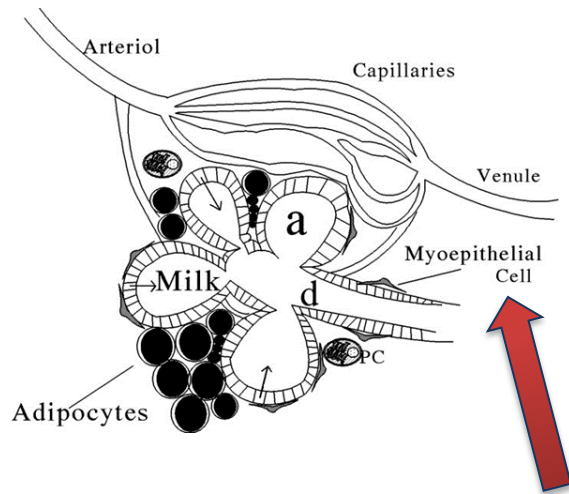
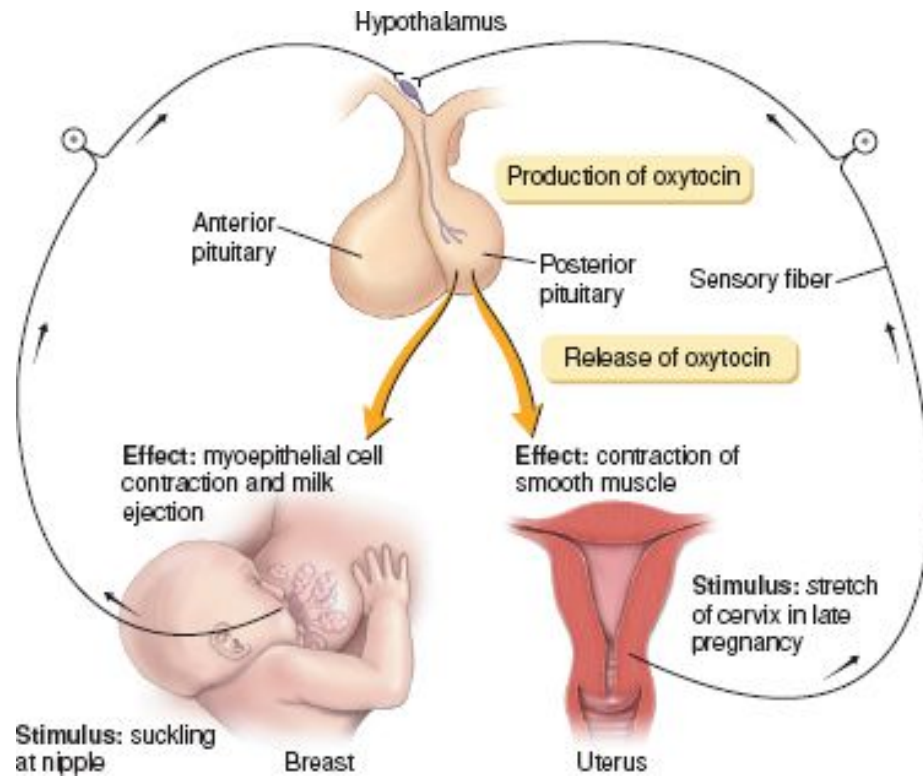
- Oxytocin is synthesized in the cell bodies of hypothalamic neurons ([paraventricular nucleus](#))
- Oxytocin is stored in the posterior pituitary



# Functions of oxytocin

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- Oxytocin is a strong stimulant of uterine contractions.
- 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

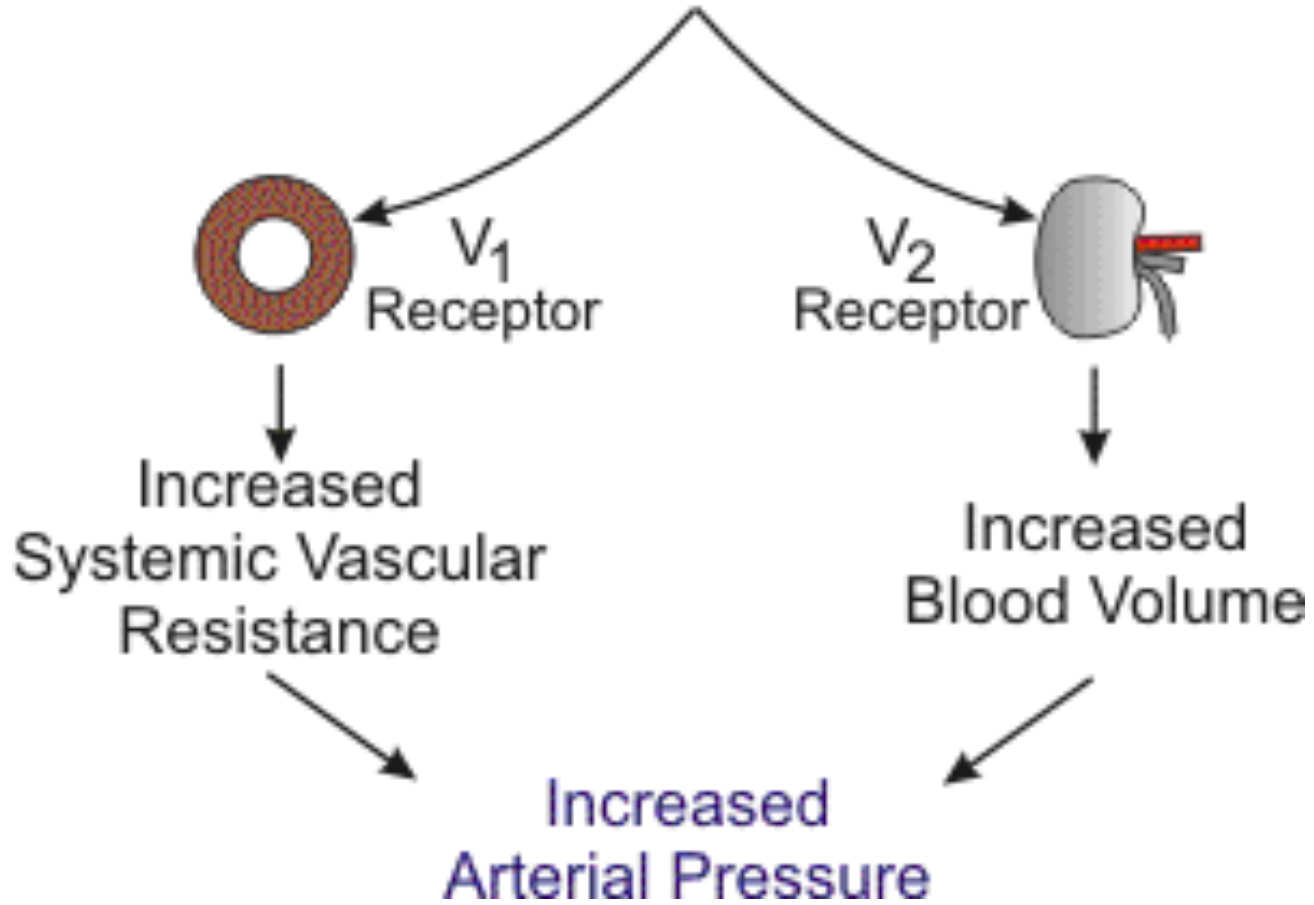
# Receptors of ADH (vasopressin)

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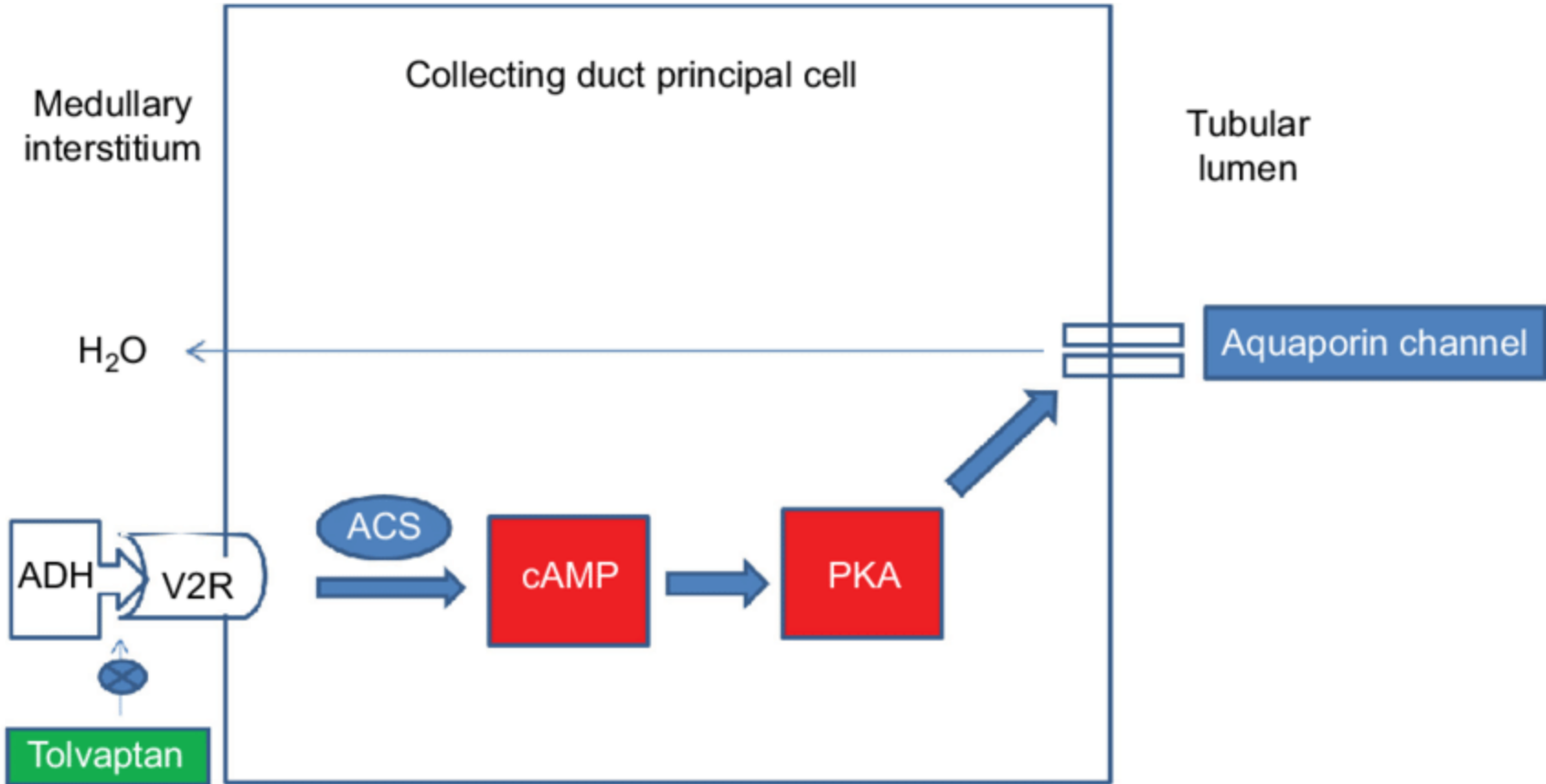
- There are 2 types of receptors for ADH:
  - $V_1$
  - $V_2$
- ⊙  **$V_1$**  receptors mediate vasoconstriction.
- ⊙  **$V_2$**  receptors are located in the *principle cells* in distal convoluted tubule & collecting ducts in the kidneys.

# Actions of ADH

## Arginine Vasopressin



# Mechanism of action of ADH



# Control of ADH Release

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## 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, aortic arch and left atrium:

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

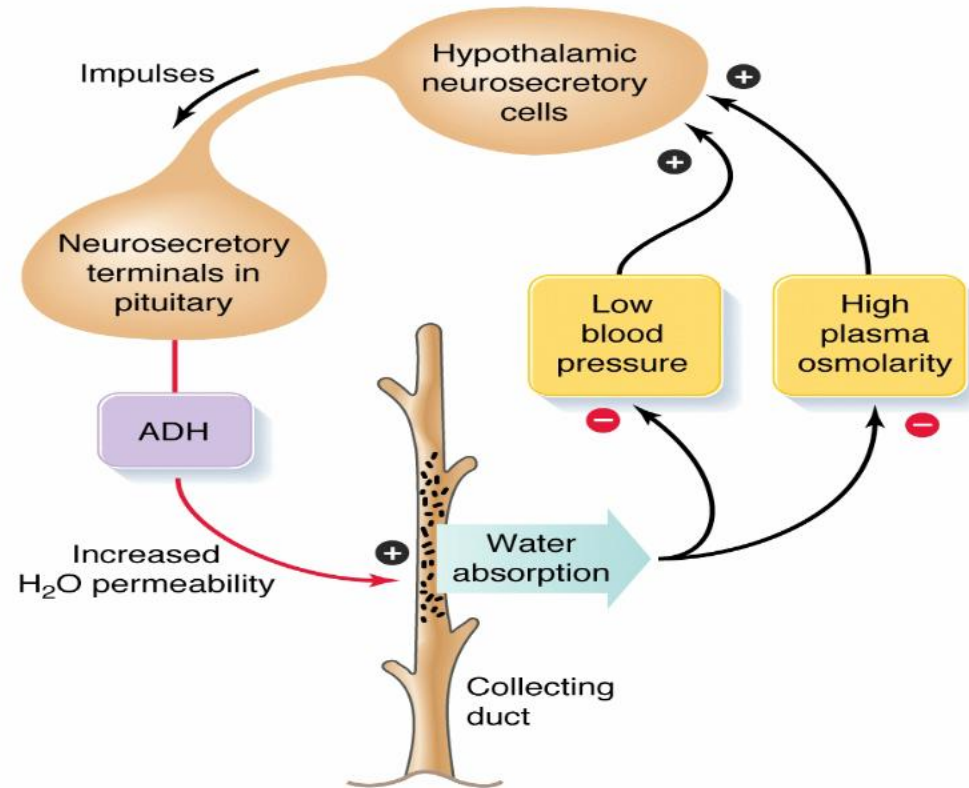


# Regulation of ADH

## Hypothalamus receives feedback from:

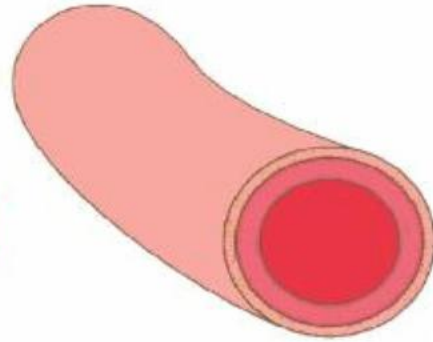
- Osmoreceptors
- Aortic arch baroreceptors
- Carotid baroreceptors
- Atrial stretch receptors

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



# Effects on Blood Vessels

Normal



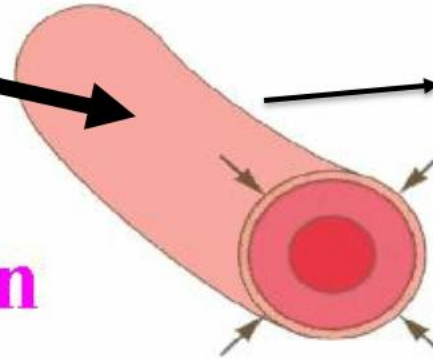
*ADH*

*V1 receptor*

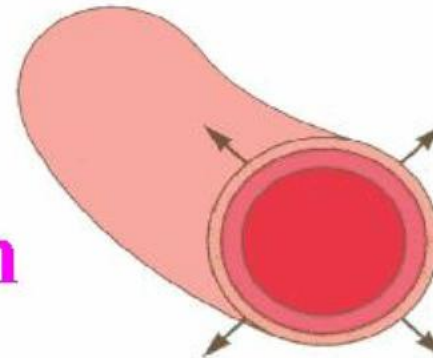


*Increased  
Blood  
Pressure*

Vasoconstriction



Vasodilation



# ADH Disorders

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- **Diabetes Insipidus (DI):**
  - **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**)

## DI

- Low ADH, Low water in body
- **High UO**, Polyuria
- **High sodium**
- High H&H and serum osmolality from dehydration
- Risk: **Hypovolemic shock**
- TX: DDAVP (ADH)

## SIADH

- High ADH, water intoxication
- **Low UO**, Oliguria
- **Low sodium** (dilutional)
- Low serum osmolality
- Weight gain
- Risk: **Seizures**
- TX: Hypertonic saline

# 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 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

- **DESMOPRESSIN (DDAVP)** ANALOG IS SUPERIOR BECAUSE:
- IT HAS LONGER HALF-LIFE (10 h vs 2-3 h)
- MORE POTENT
- ITS ANTIDIURETIC ACTIVITY IS 3000 TIMES GREATER THAN DESMOPRESSIN



ACTIC  
VP

ON OF ACTION (8-

ACTIVITY IS 3000 TIMES  
GREATER THAN DESMOPRESSIN

# Treatment of Nephrogenic DI

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