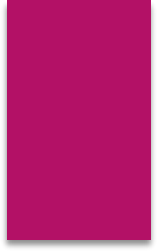


**We only live once,
Snoopy.**

**Wrong! We only
die once. We live
every day!**









Control of ECF Osmolarity & Sodium concentration

DR SARAH SHAHID

LECTURER PHYSIOLOGY

Learning objectives

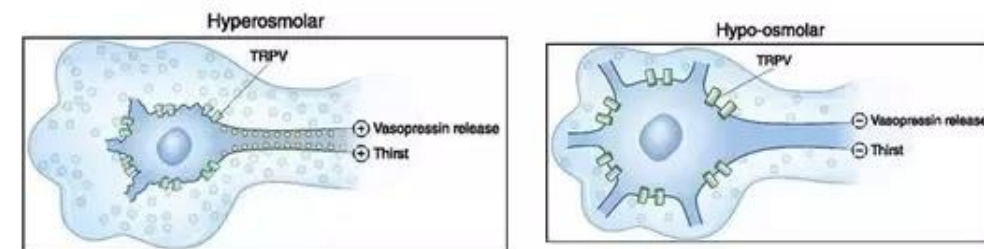
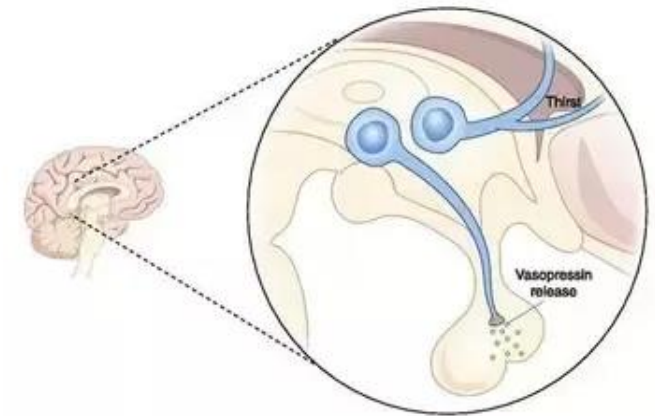
- ▶ Describe the OSMORECEPTOR-ADH FEEDBACK SYSTEM
- ▶ Enumerate the Factors affecting ADH Secretion
- ▶ Describe the Role of thirst in controlling extracellular fluid osmolarity and sodium concentration
- ▶ Discuss Role of Angiotensin II and Aldosterone in Controlling Extracellular Fluid Osmolarity and Sodium Concentration
- ▶ Describe Salt-appetite mechanism for controlling extracellular fluid sodium concentration and volume

Osmoregulation

- ▶ The process of regulating the concentration of water and mineral salts in the body fluid is called **osmoregulation**
- ▶ Osmoregulation of the extracellular fluid is achieved by regulated addition and subtraction of free water from the extracellular fluid, thus diluting or concentrating the already present electrolytes. via adding or removing free water from the ECF.

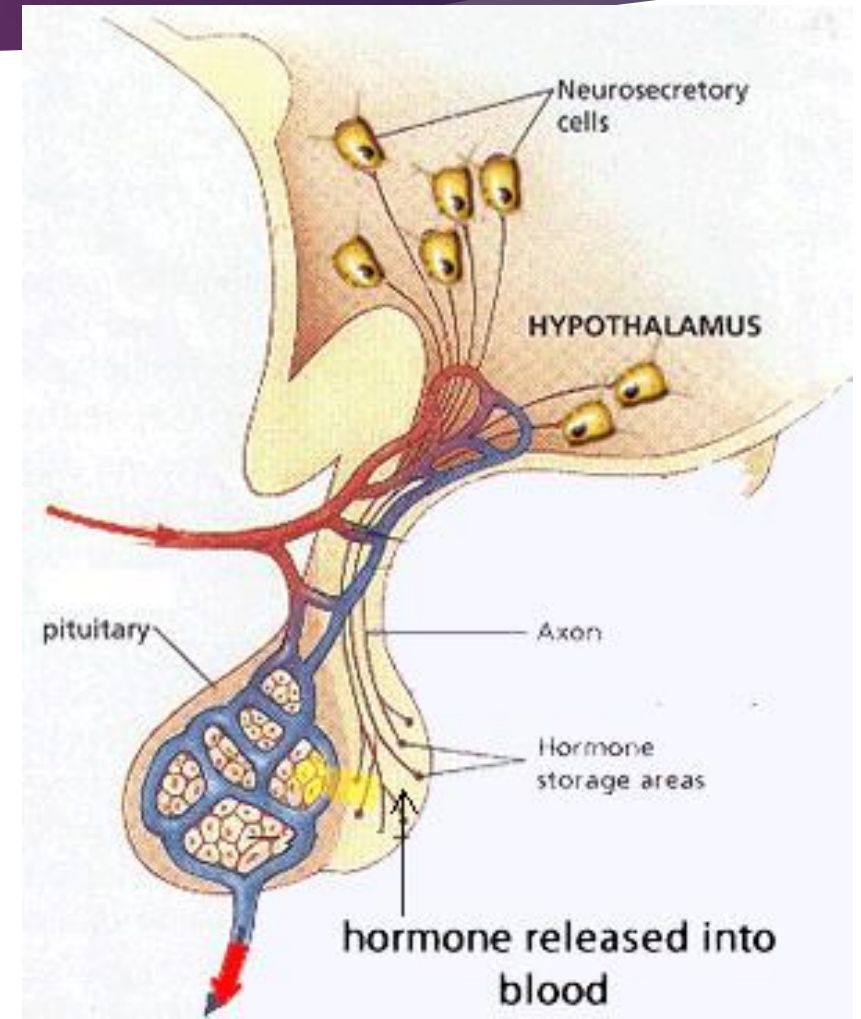
OSMORECEPTOR-ADH FEEDBACK SYSTEM

- ▶ An increase in extracellular fluid osmolarity causes the special nerve cells called osmoreceptor cells, located in the anterior hypothalamus near the supraoptic nuclei, to shrink.
- ▶ Shrinkage of the osmoreceptor cells causes them to fire, sending nerve signals to additional nerve cells in the supraoptic nuclei,
- ▶ which then relay these signals down the stalk of the pituitary gland to the posterior pituitary.

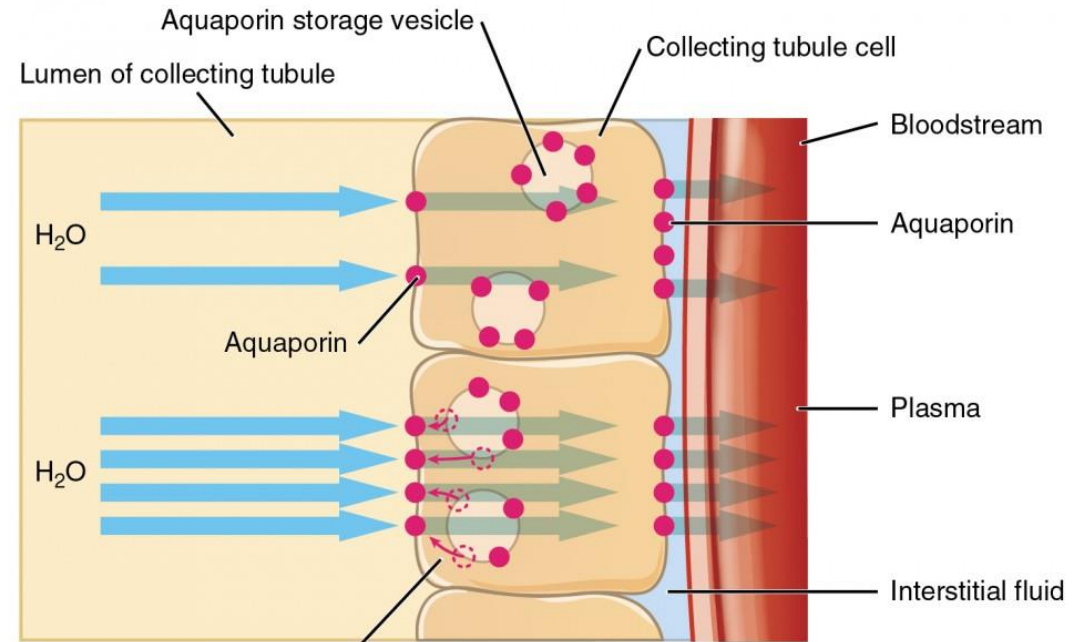


OSMORECEPTOR-ADH FEEDBACK SYSTEM

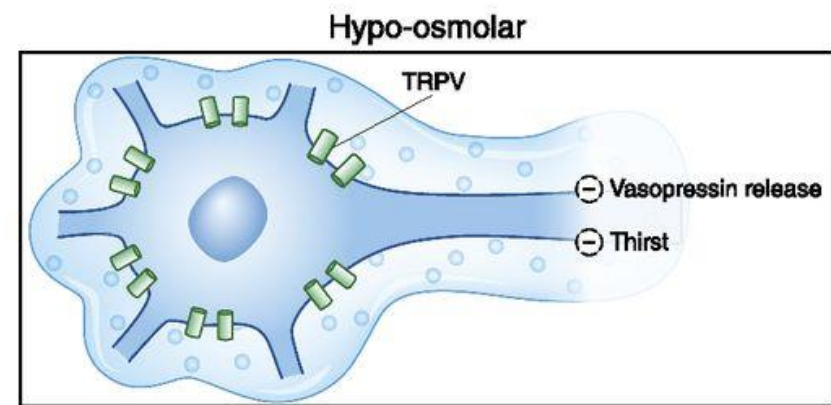
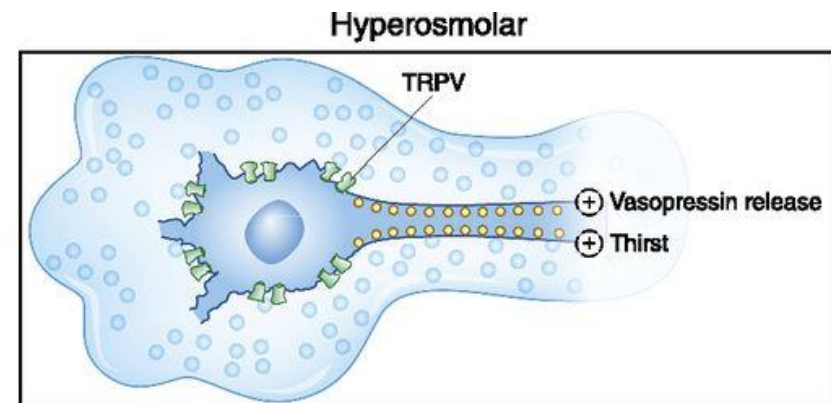
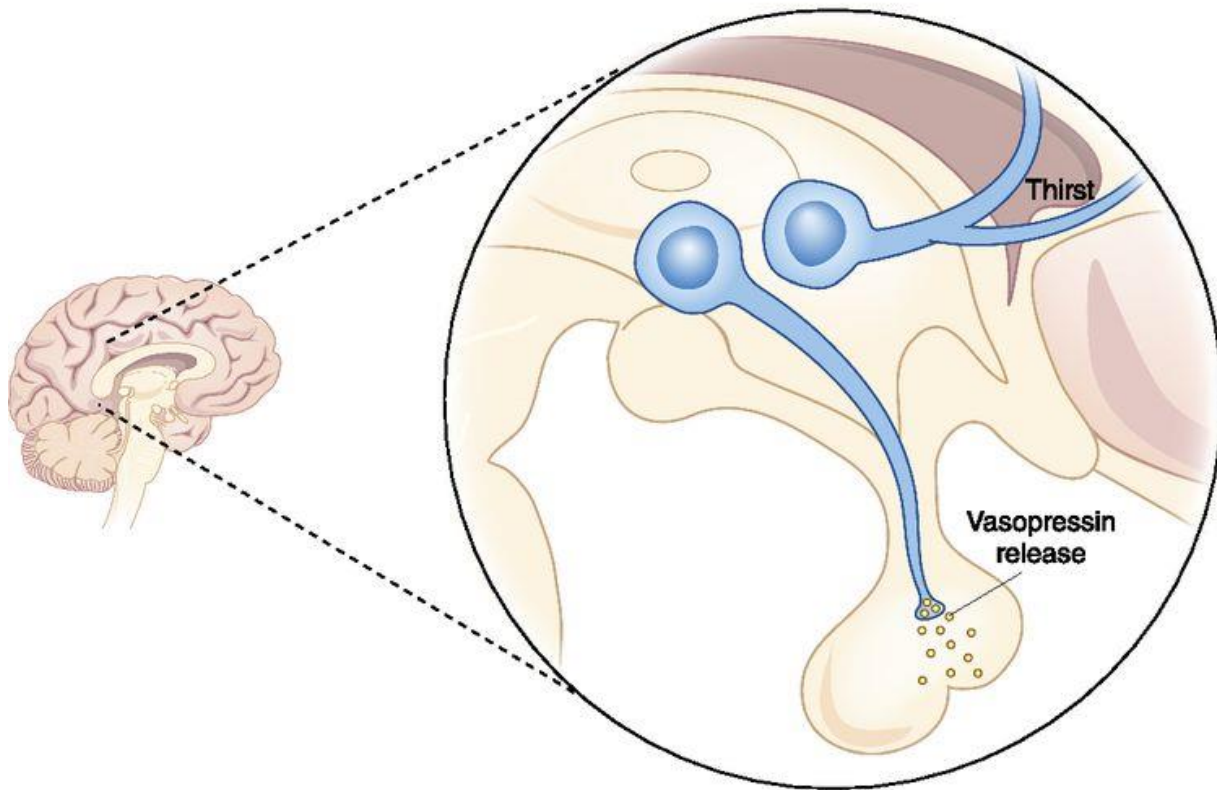
- ▶ These action potentials conducted to the posterior pituitary stimulate the release of ADH, which is stored in secretory granules (or vesicles) in the nerve endings.
- ▶ ADH enters the blood stream and is transported to the kidneys,
- ▶ where it increases the water permeability of the late distal tubules, cortical collecting tubules, and inner medullary collecting ducts.



- ▶ The increased water permeability in the distal nephron segments causes increased water reabsorption and excretion of a small volume of concentrated urine.

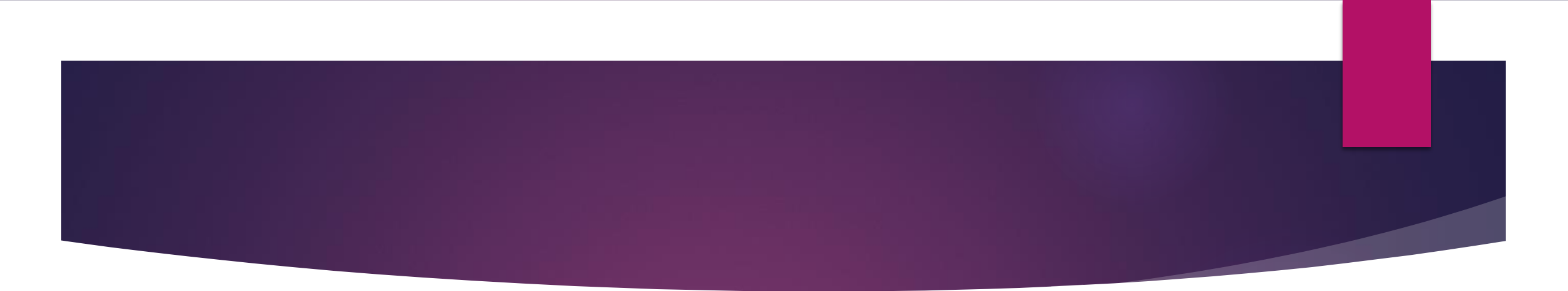


Aquaporin pores are inserted into cell membrane, increasing flow of H₂O out of tubule



ADH Synthesis in Supraoptic and Paraventricular Nuclei of the Hypothalamus and ADH Release from the Posterior Pituitary

- ▶ The hypothalamus contains two types of magnocellular (large) neurons that synthesize ADH in the
- ▶ **supraoptic nuclei:** about five sixths
- ▶ **paraventricular nuclei:** about one sixth
- ▶ Both of these nuclei have axonal extensions to the posterior pituitary.
- ▶ Once ADH is synthesized, it is transported down the axons of the neurons to their tips, terminating in the posterior pituitary gland.

- 
- ▶ When the supraoptic and paraventricular nuclei are stimulated by increased osmolarity or other factors, nerve impulses pass down these nerve endings, changing their membrane permeability and increasing calcium entry.
 - ▶ ADH stored in the secretory granules (also called vesicles) of the nerve endings is released in response to increased calcium entry.
 - ▶ The released ADH is then carried away in the capillary blood of the posterior pituitary into the systemic circulation.

Osmoreceptors
(circumventricular
organs)

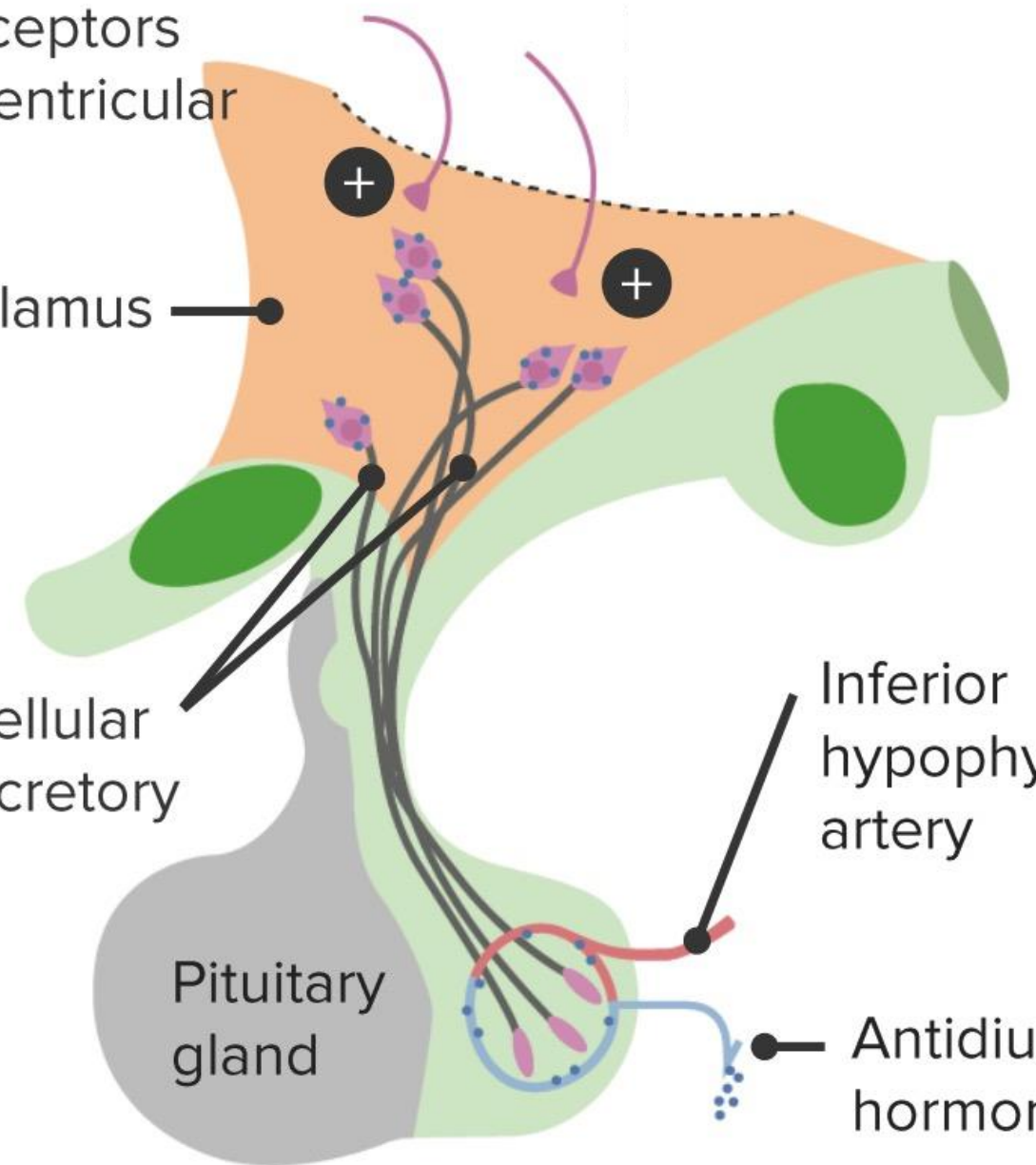
Hypothalamus

Magnocellular
neurosecretory
cells

Pituitary
gland

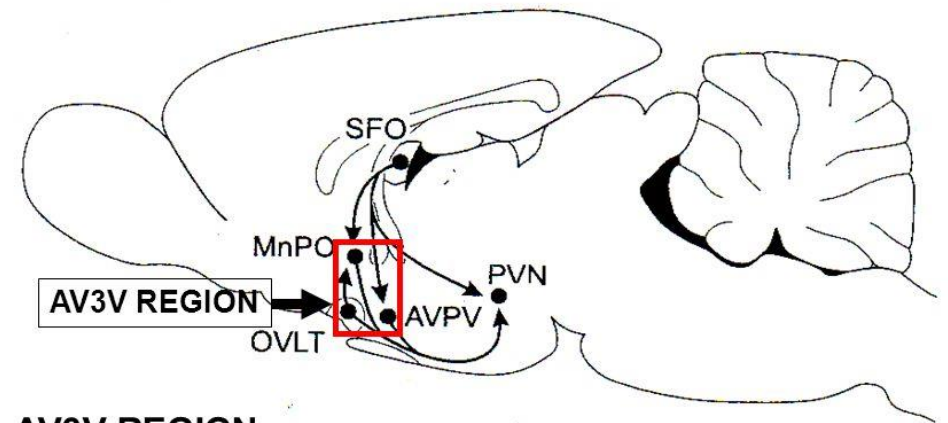
Inferior
hypophyseal
artery

Antidiuretic
hormone (ADH)



- ▶ A second neuronal area important in controlling osmolarity and ADH secretion is located along the anteroventral region of the third ventricle, called the **AV 3V region**.
- ▶ Lesions of the AV 3 V region cause multiple deficits in the control of ADH secretion, thirst, sodium appetite, and blood pressure.
- ▶ Electrical stimulation of this region or stimulation by angiotensin II can alter ADH secretion, thirst, and sodium appetite.

AV3V REGION
(anteroventral 3rd ventricle region)



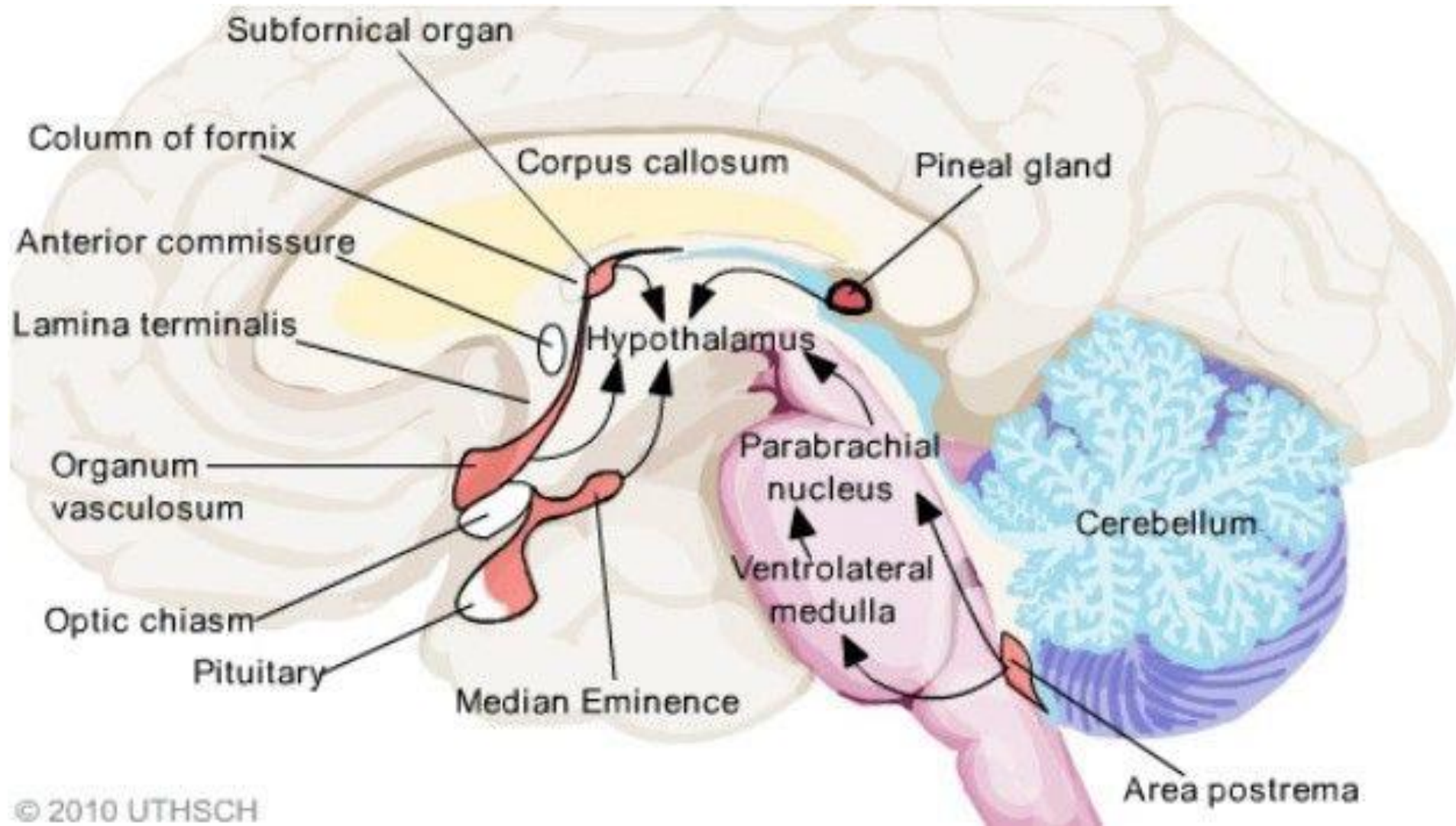
AV3V REGION

MnPO = median preoptic nucleus

AVPV = anteroventral periventricular nuclei

OVLT = organum vasculosum of the lamina terminalis

(Brody and Johnson, 1978)

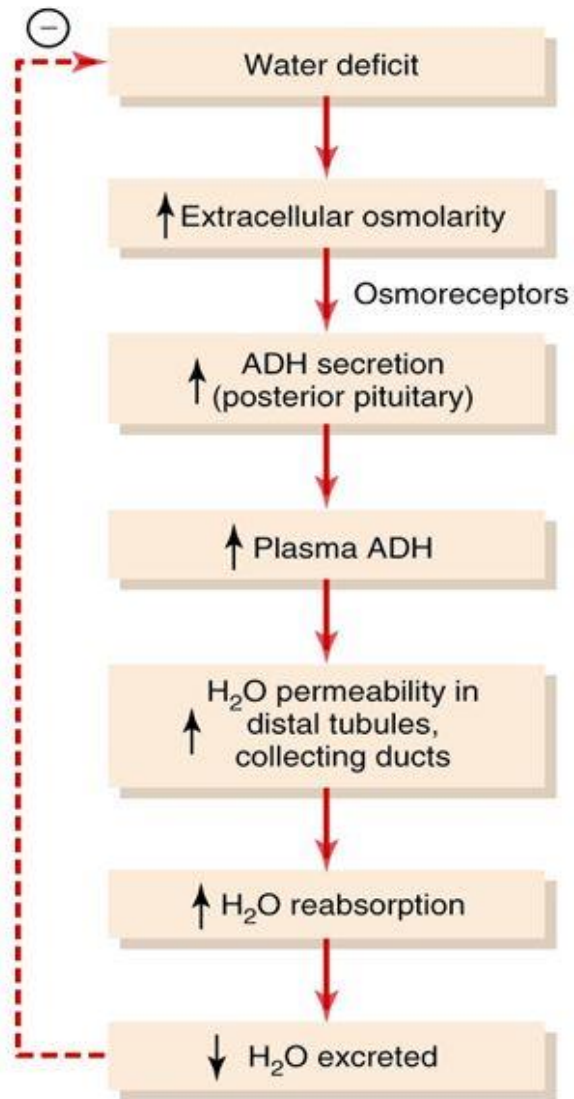


Stimuli for ADH Secretion

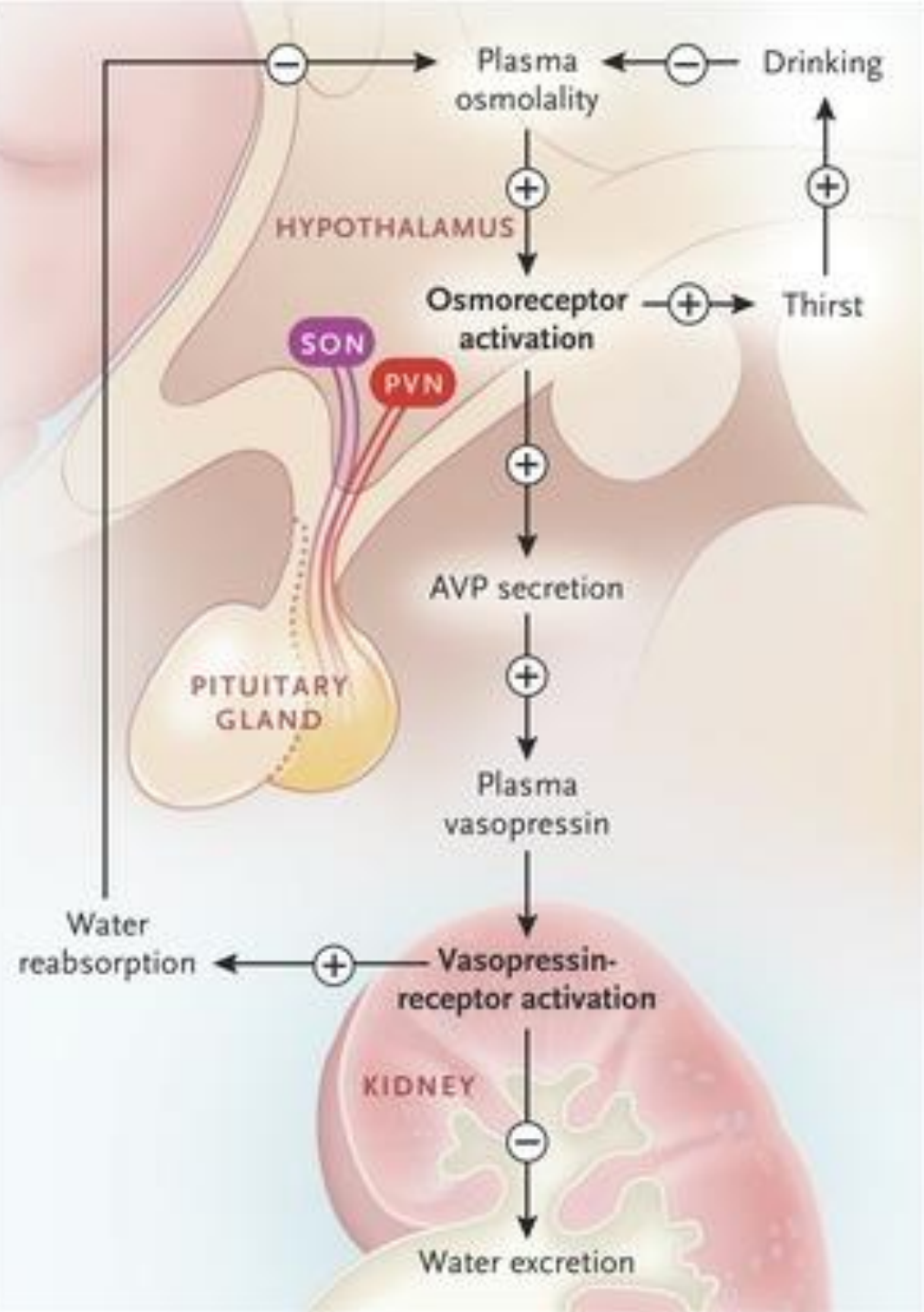
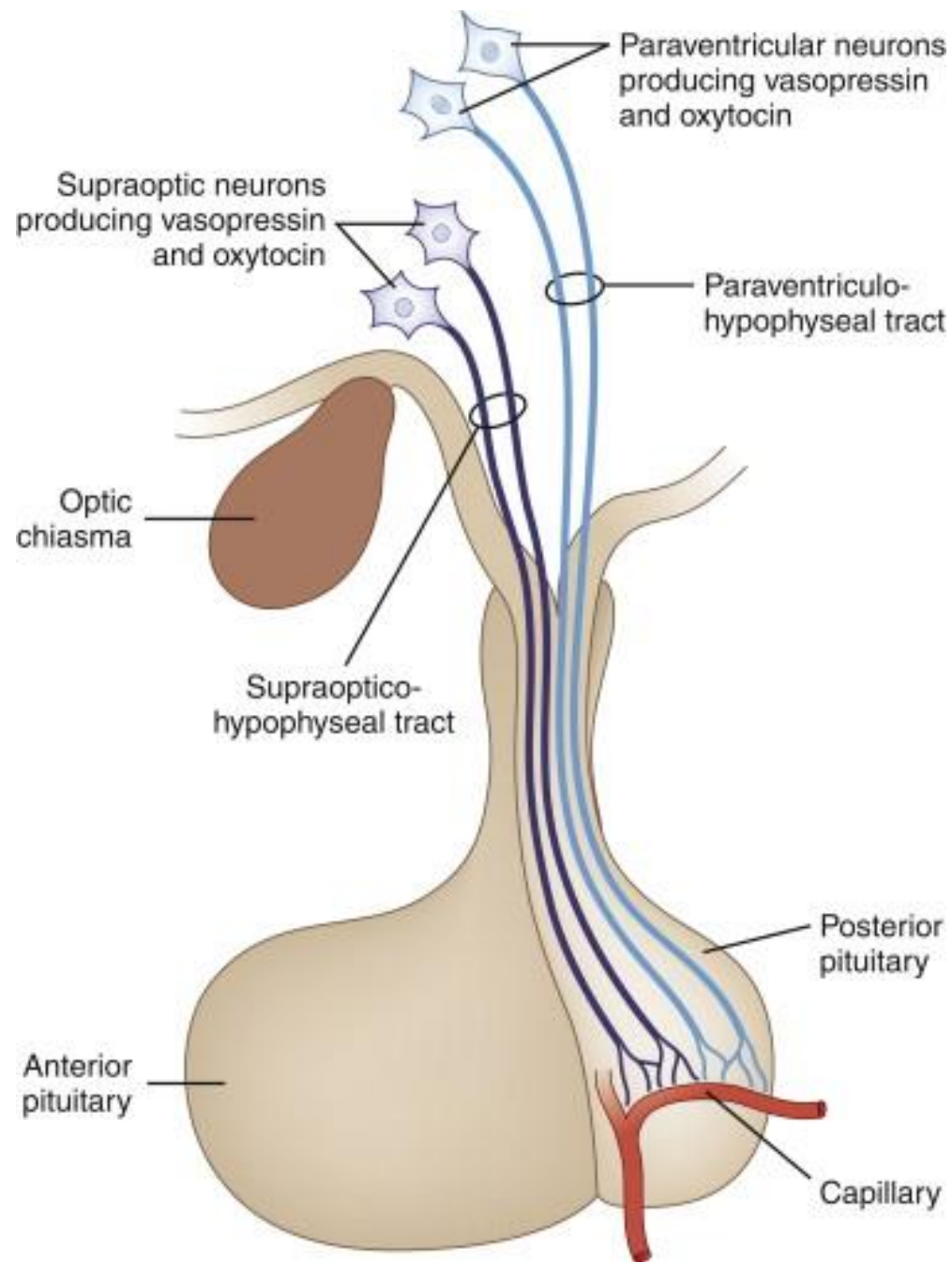
- ▶ Increased osmolarity
- ▶ Decreased blood volume
- ▶ Decreased blood pressure (arterial baroreceptors)
- ▶ input from cerebral cortex (e.g. fear)
- ▶ angiotensin II
- ▶ Nausea
- ▶ hypoxia
- ▶ nicotine
- ▶ morphine

Factors that Decrease ADH Secretion

- ▶ Decreased osmolarity
- ▶ Increased blood volume (cardiopulmonary reflexes)
- ▶ Increased blood pressure (arterial baroreceptors)
- ▶ Other factors:- alcohol- clonidine (-2 adrenergic agonist)
- ▶ haloperidol (antipsychotic)



Osmoreceptor– antidiuretic hormone (ADH) feedback mechanism for regulating extracellular fluid osmolarity

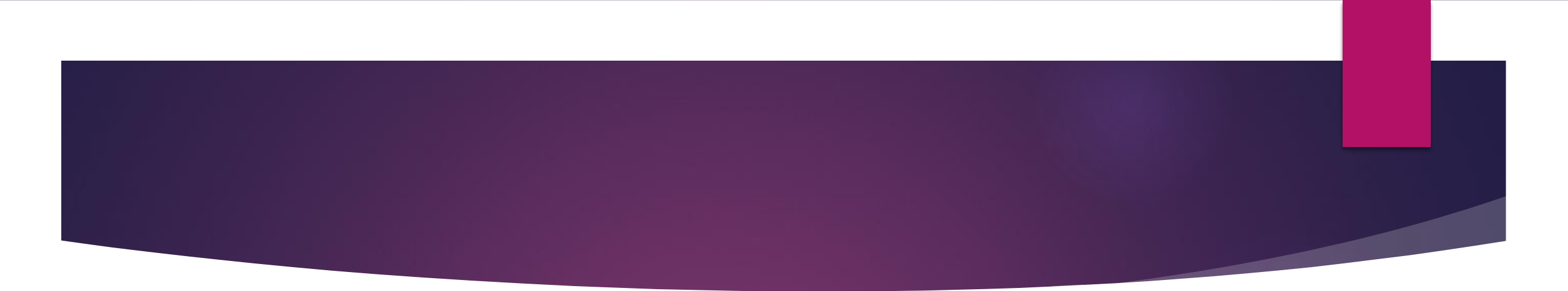


ROLE OF THIRST IN CONTROLLING EXTRACELLULAR FLUID OSMOLARITY AND SODIUM CONCENTRATION

- ▶ It is defined as the conscious desire for water.
- ▶ The kidneys minimize fluid loss during water deficits through the osmoreceptor-ADH feedback system.
- ▶ Adequate fluid intake, however, is necessary to counterbalance whatever fluid loss does occur through sweating and breathing and through the gastrointestinal tract.
- ▶ Fluid intake is regulated by the thirst mechanism, which, together with the osmoreceptor-ADH mechanism, maintains precise control of extracellular fluid osmolarity and sodium concentration.

Central Nervous System Centers for Thirst

- ▶ Located anterolaterally in the preoptic nucleus is another small area that, when stimulated electrically, causes immediate drinking that continues as long as the stimulation lasts.
- ▶ All these areas together are called the thirst center.
- ▶ The neurons of the thirst center respond to injections of hypertonic salt solutions by stimulating drinking behavior.

- 
- ▶ Increased osmolarity of the cerebrospinal fluid in the third ventricle has essentially the same effect to promote drinking.
 - ▶ It is likely that the organum vasculosum of the lamina terminalis, which lies immediately beneath the ventricular surface at the inferior end of the AV 3 V region, is intimately involved in mediating this response.

Stimuli for Thirst

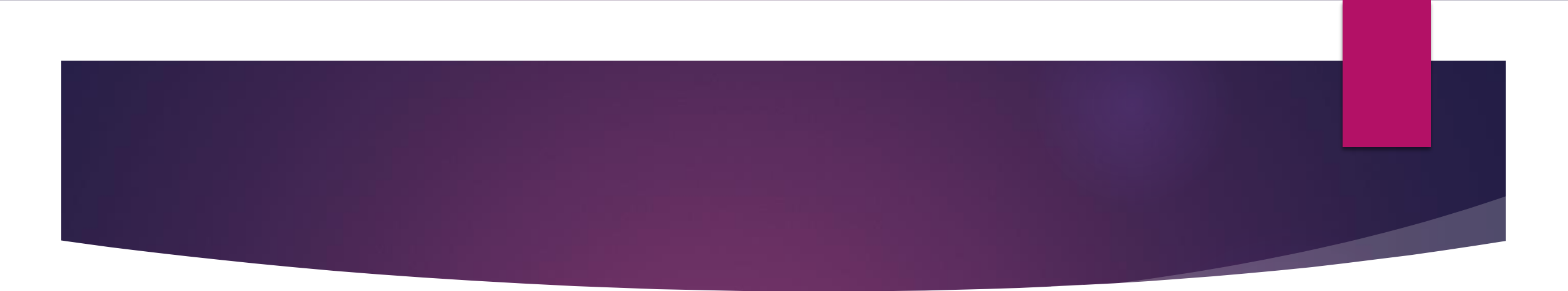
- ▶ Increased osmolarity
- ▶ Decreased blood volume
(cardiopulmonary reflexes)
- ▶ Decreased blood pressure (arterial baroreceptors)
- ▶ Increased angiotensin II
- ▶ dryness of mouth

Factors that Decrease Thirst

- ▶ Decreased osmolarity
- ▶ Increased blood volume
- ▶ Increased blood pressure (arterial baroreceptors)
- ▶ Decreased angiotensin II
- ▶ Gastric distention

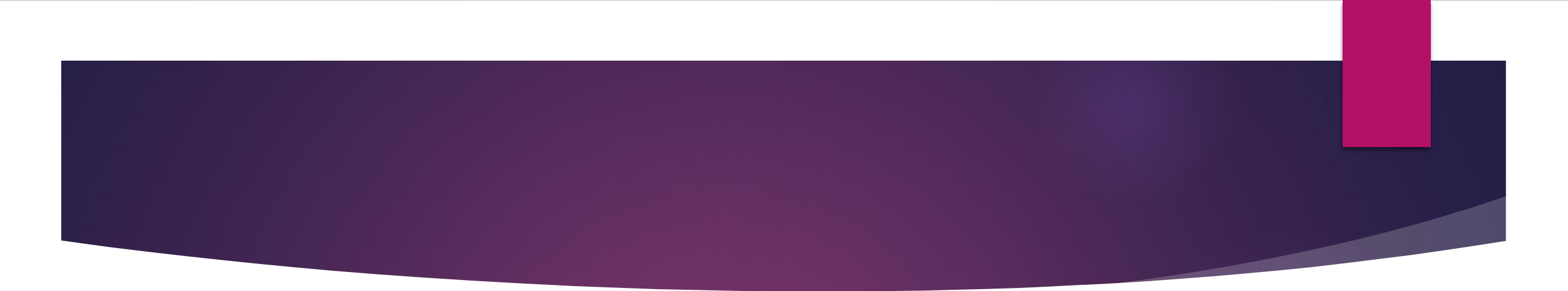
Stimuli for Thirst

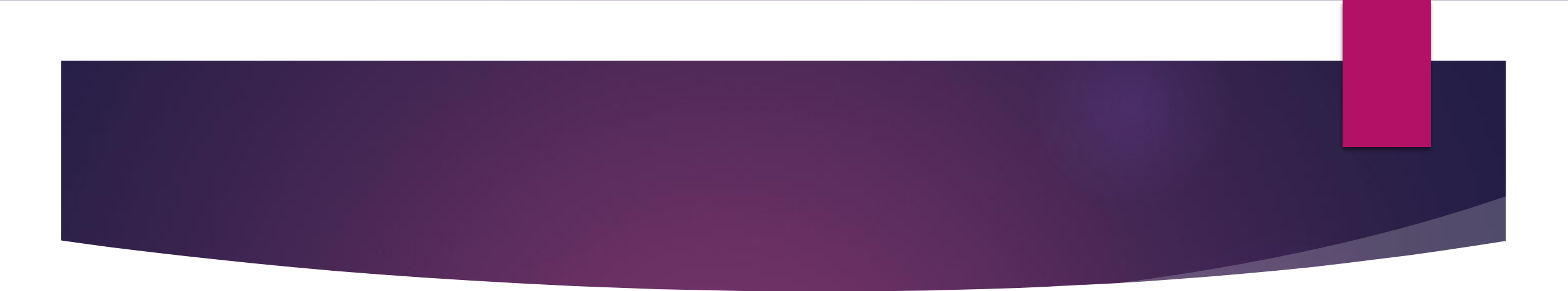
- ▶ One of the most important is increased extracellular fluid osmolarity, which causes intracellular dehydration in the thirst centers, thereby stimulating the sensation of thirst.
- ▶ it helps to dilute extracellular fluids and returns osmolarity toward normal.
- ▶ Decreases in extracellular fluid volume and arterial pressure also stimulate thirst

- 
- ▶ blood volume loss by hemorrhage stimulates thirst even though there might be no change in plasma osmolarity.
 - ▶ This probably occurs because of neutral input from cardiopulmonary and systemic arterial baroreceptors in the circulation.
 - ▶ A third important stimulus for thirst is angiotensin II. Studies in animals have shown that angiotensin II acts on the subfornical organ and on the organum vasculosum of the lamina terminalis.

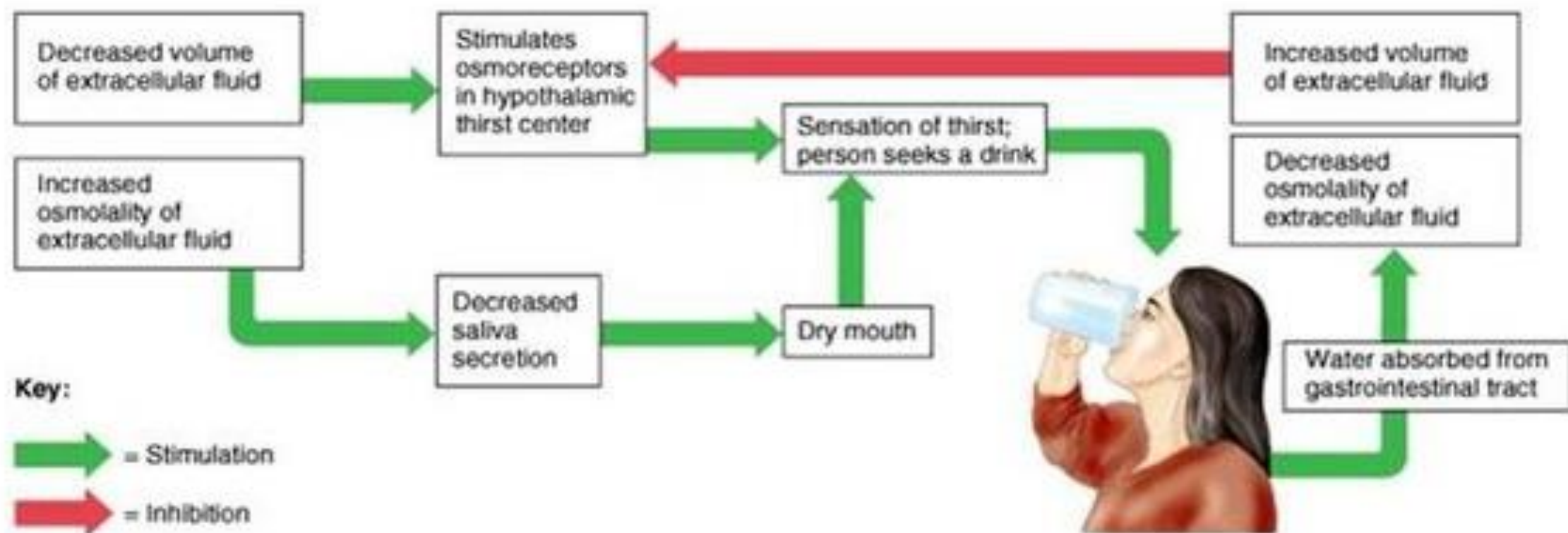
Stimuli for Thirst cont:

- ▶ These regions are outside the blood-brain barrier, and peptides such as angiotensin II diffuse into the tissues.
- ▶ Because angiotensin II is also stimulated by factors associated with hypovolemia and low blood pressure, its effect on thirst helps to restore blood volume and blood pressure toward normal, along with the other actions of angiotensin II on the kidneys to decrease fluid excretion.

- 
- ▶ Dryness of the mouth and mucous membranes of the esophagus can elicit the sensation of thirst.
 - ▶ As a result, a thirsty person may receive relief from thirst almost immediately after drinking water, even though the water has not been absorbed from the gastrointestinal tract and has not yet had an effect on extracellular fluid osmolarity.

- 
- ▶ Gastrointestinal and pharyngeal stimuli influence thirst. For example, in animals that have an esophageal opening to the exterior so that water is never absorbed into the blood, partial relief of thirst occurs after drinking, although the relief is only temporary

Thirst mechanism



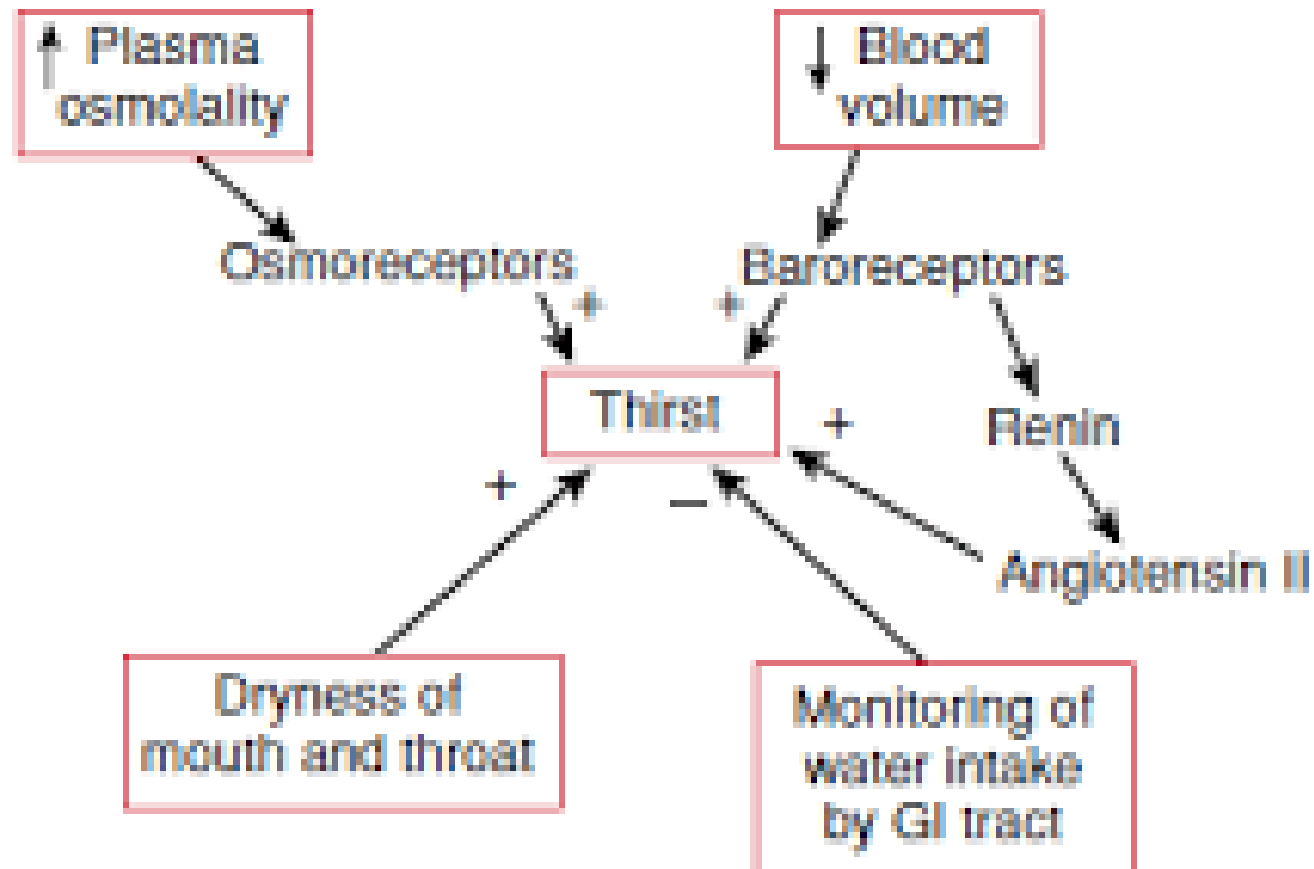
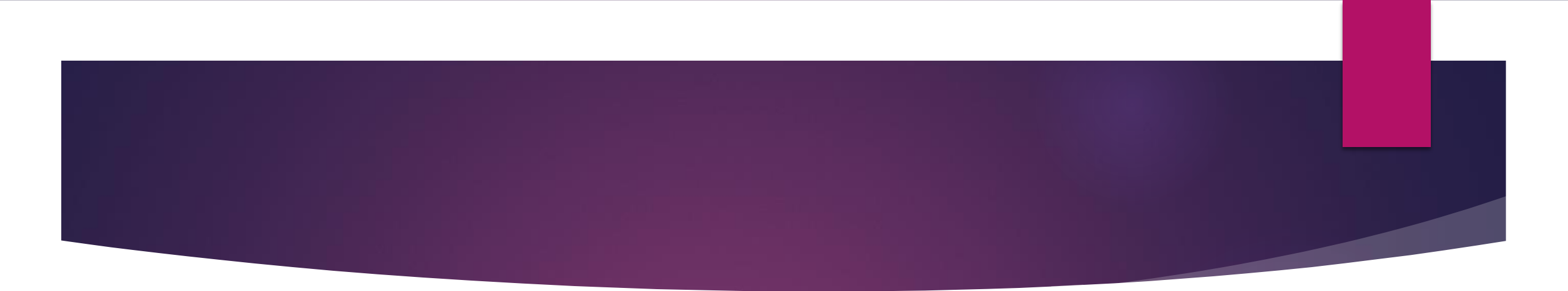


FIGURE 24.7 Factors affecting the thirst sensation. A plus sign indicates stimulation of thirst, the minus sign indicates an inhibitory influence.

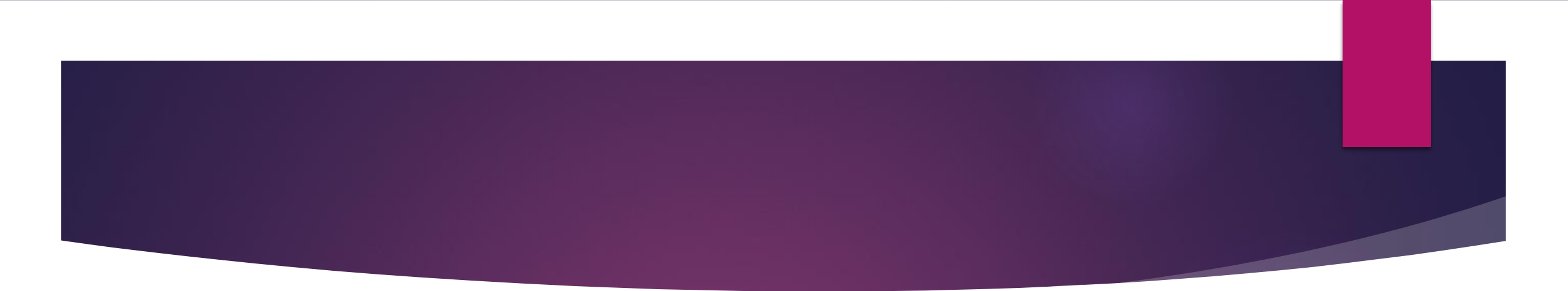
SALT-APPETITE MECHANISM FOR CONTROLLING EXTRACELLULAR FLUID SODIUM CONCENTRATION AND VOLUME

- ▶ Maintenance of normal extracellular fluid volume and sodium concentration requires a balance between sodium excretion and sodium intake.
- ▶ In modern civilizations, sodium intake is almost always greater than necessary for homeostasis.
- ▶ Salt appetite is due in part to the fact that animals and humans like salt and eat it regardless of whether they are salt deficient.

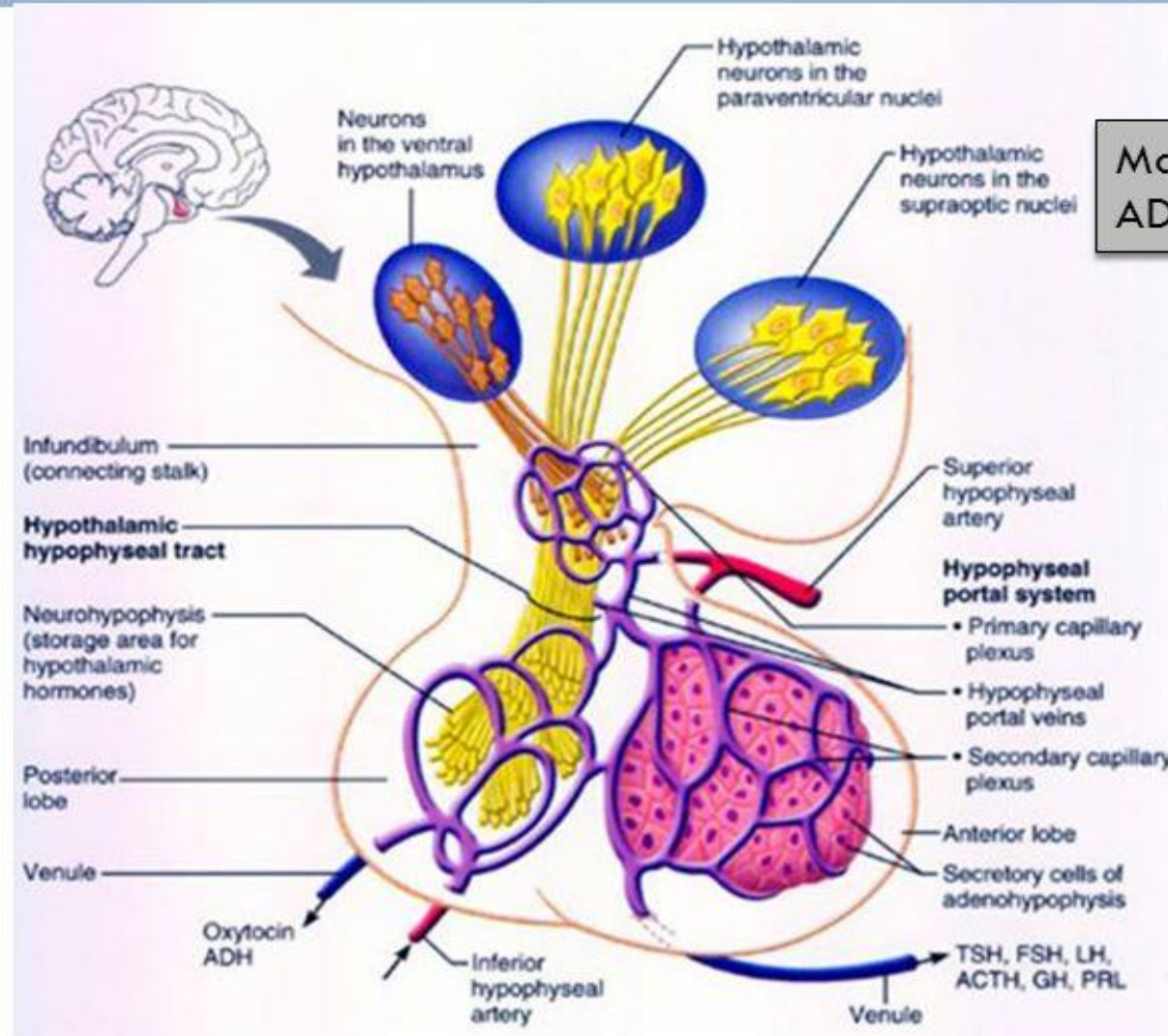
- 
- ▶ There is also a regulatory component to salt appetite in which there is a behavioral drive to obtain salt when there is sodium deficiency in the body.
 - ▶ In general, the two primary stimuli that are believed to excite salt appetite are
 - ▶ (1) decreased extracellular fluid sodium concentration and
 - ▶ (2) decreased blood volume or blood pressure, associated with circulatory insufficiency.
 - ▶ The neuronal mechanism for salt appetite are analogous to thirst mechanism.

Role of Angiotensin II and Aldosterone in Controlling Extracellular Fluid Osmolarity and Sodium Concentration

- ▶ Both angiotensin II and aldosterone play an important role in regulating sodium reabsorption by the renal tubules.
- ▶ When sodium intake is low, increased levels of these hormones stimulate sodium reabsorption by the kidneys and, therefore, prevent large sodium losses, even though sodium intake may be reduced to as low as 10 per cent of normal.

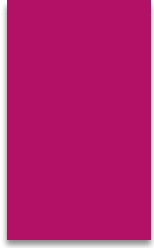
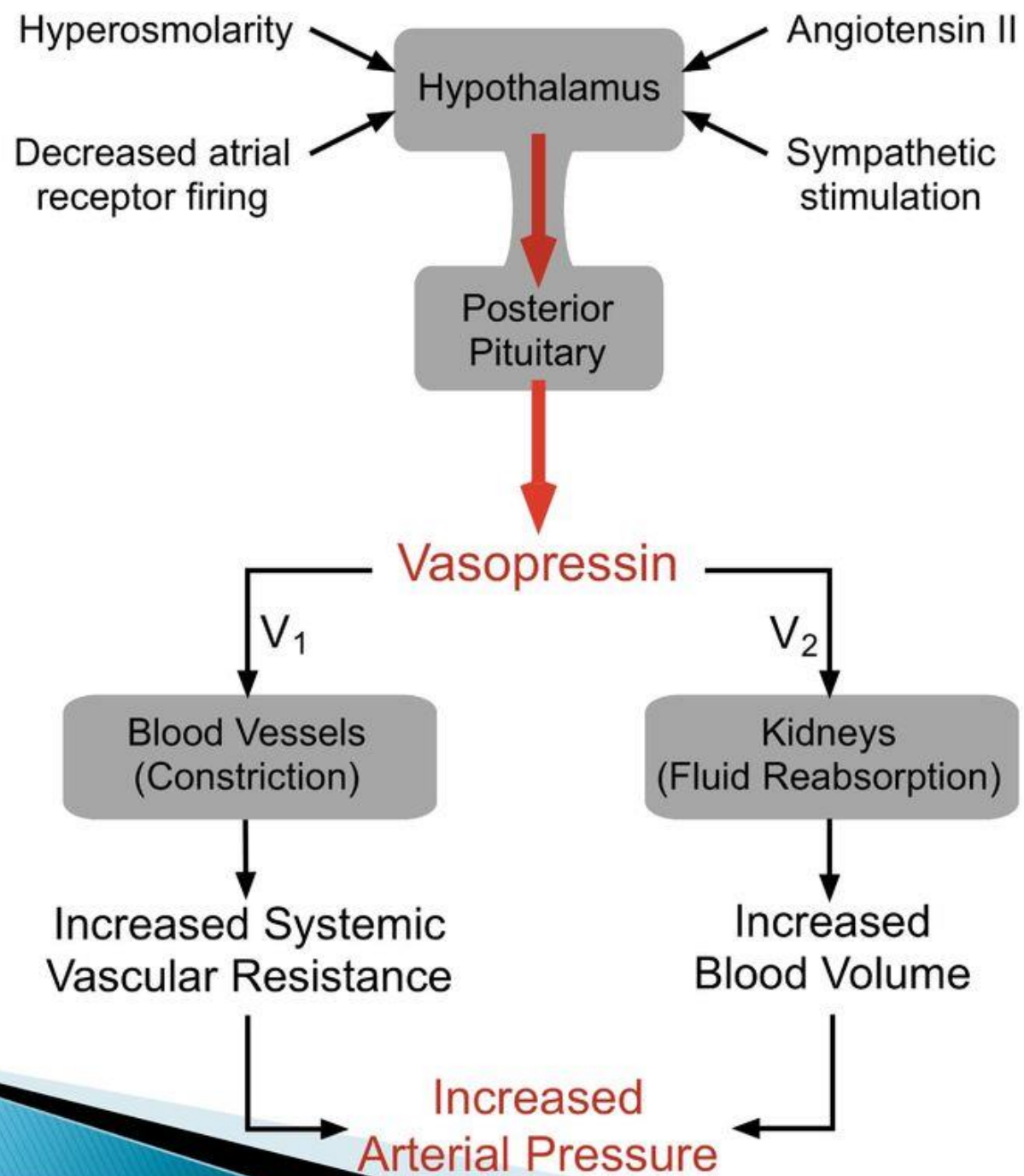
- 
- ▶ Because of the importance of angiotensin II and aldosterone in regulating sodium excretion by the kidneys, one might mistakenly infer that they also play an important role in regulating extracellular fluid sodium concentration.
 - ▶ Although these hormones increase the amount of sodium in the extracellular fluid, they also increase the extracellular fluid volume by increasing reabsorption of water along with the sodium.
 - ▶ Therefore. Angiotensin II and aldosterone have little effect on sodium concentration, except under extreme conditions.

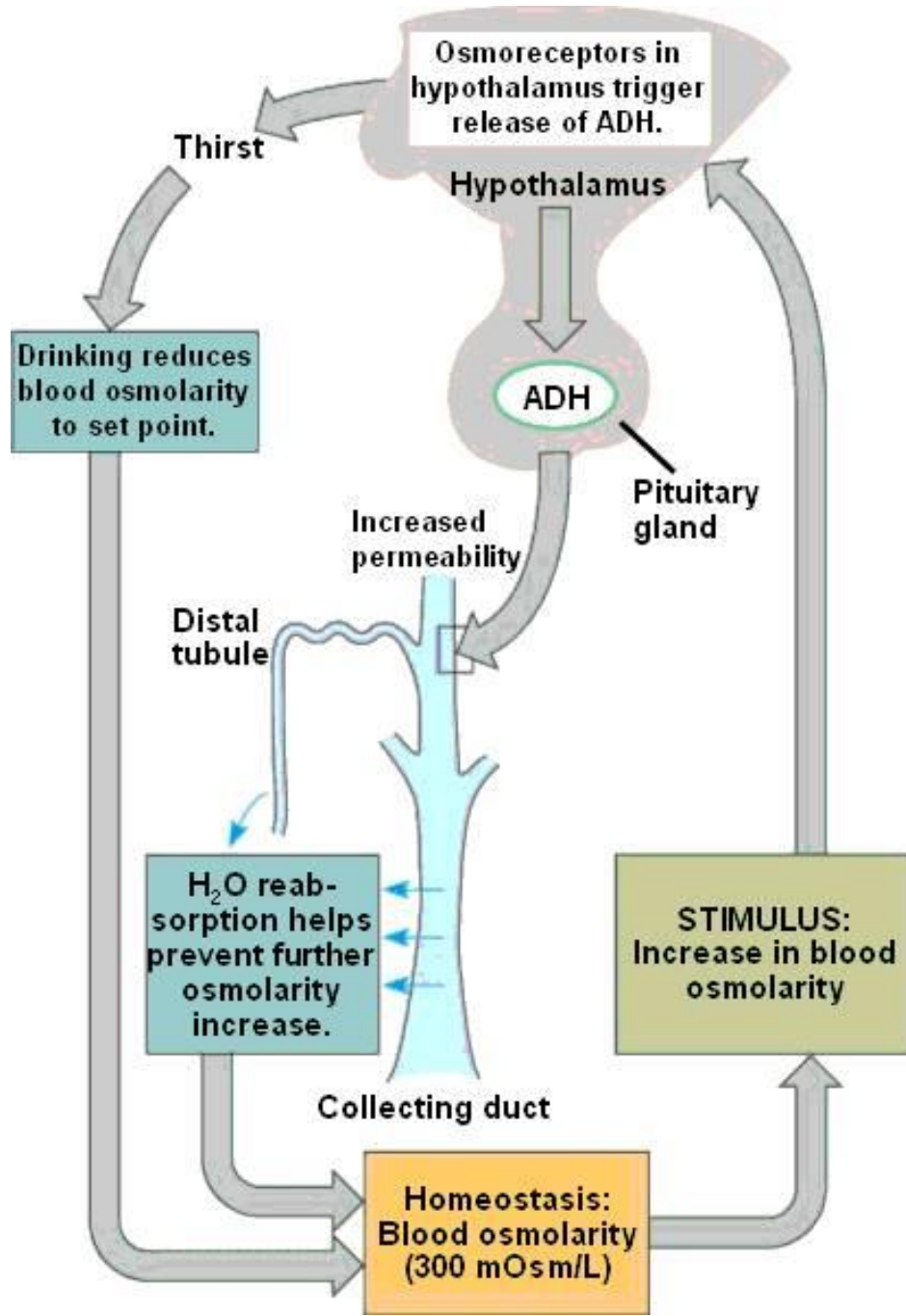
Where in the brain ADH is formed?



Main site for ADH synthesis

ADH is stored in posterior pituitary





**SO ANTIDIURETIC HORMONE (ADH)
JUST MAKES THE**



KIDNEYS DO THE SAME THING I DO

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Recommended books & learning resources

- ▶ Text book of Medical Physiology- Guyton and Hall
- ▶ Principles of human Physiology- Lauralee Sherwood
- ▶ Color Atlas of human Physiology



“Because
answers
exist only
to *questions...*”

Mungara Tarou Krishnamurti
26 Jan 2013 2:17 am

Any Questions?

Questions ,
Comments,
Feedback?

drsarahshahid@gmail.com

That's all Folks!