



# ENDOCRINE BLOCK

## LECTURE 4

Physiology of Posterior Pituitary Gland



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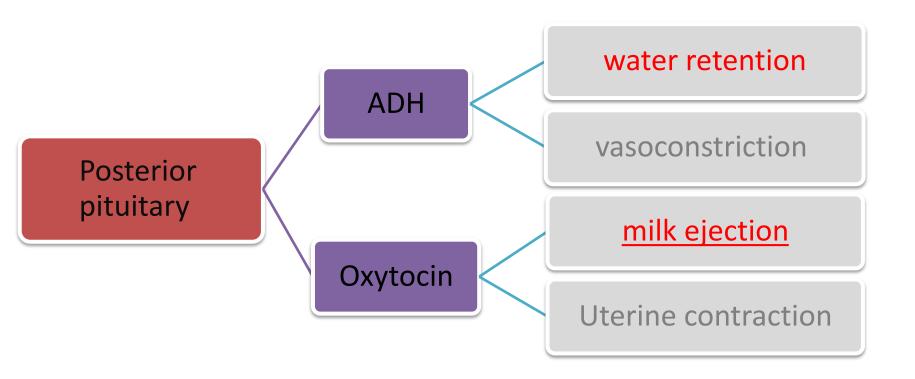




- Hypothalamic control
- Posterior pituitary hormones
  - ADH
    - Physiological functions
    - Control of secretion
      - Osmotic stimuli
      - Non-osmotic stimuli
  - Oxytocin
    - Physiological functions
    - Control of secretion





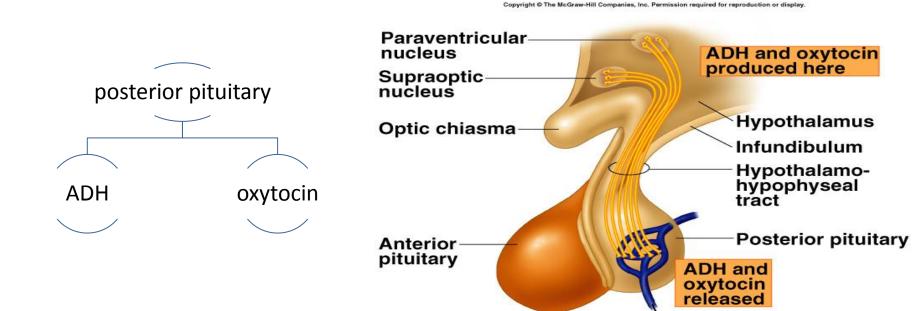






## **Posterior Pituitary Gland**





- The two hormones synthesized in **hypothalamus** from two nuclei (supraoptic nucleus & paraventricular nucleus), but released in **posterior pituitary**.
- Hypothalamus controls the Posterior Pituitary secretions by nervous signals delivered to Posterior pituitary by axons. (neurohypophysis)





- Synthesized as pre-prohormone a nonapeptide (nine amino acids) in the cell bodies of hypothalamic supraoptic nucleus.
- ADH stored in the neurohypophysis (posterior pituitary) <u>Forms the most readily</u> <u>released ADH pool.</u>
- Receptors for ADH (3 types):
- 1. V1A
- 2. V1B
- 3. V2
- V1A mediate vasoconstriction (blood vessels) also in liver glycogenolysis.
- V1B are unique to anterior pituitary mediate increased ACTH secretion.
- V2 are located in the principle cells of distal convoluted tubule and collecting ducts in kidney.

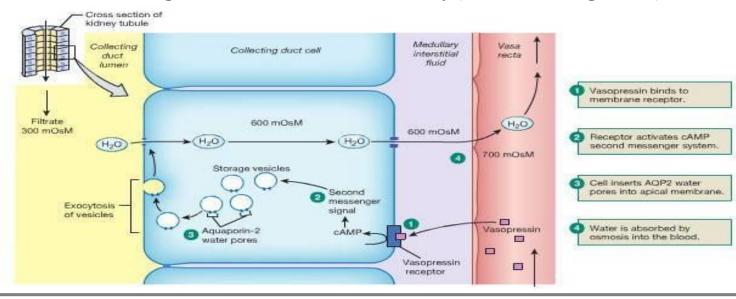
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#### Mechanism of action of ADH

- ADH binds to V2 receptors on peritubular surface (protien do not enter the cell)
  in principle cells of distal convoluted tubule medullary collecting ducts.
- Via adenylate cyclase/cAMP (2<sup>nd</sup> messenger) induces production and insertion of aquaporin2 (there are 7 types) into the luminal membrane and enhances permeability of cell to water.
- Increased membrane permeability to water permits back diffusion of solute –
   free water leading to increase urine osmolality (concentrating urine).



















#### Secretion of ADH (osmotic stimuli)

- Plasma osmolality is directly increased by administration of sodium(do not rapidly penetrate cell membrane), so <u>cause ADH release</u>.
- Urea (enter cells rapidly) do not change osmotic equilibrium and thus do not stimulate ADH release.
- ADH secretion is very sensitive to changes in osmolality by (osmoreceptors).
- Fasting or dehydration → increase plasma osmolality (salt > water) → osmoreceptors (in brain) sense that → shrinking → membrane action potential from hypothalamus → reach posterior pituitary and thirst center → ADH released and drinking water → decrease osmotic pressure.
- Changes of plasma osmolality 1-2% result in increased ADH secretion.

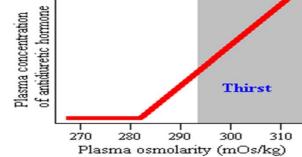
• Normal plasma osmolality (270-280) if it increases ADH and thirst center will be



Increase Solute(Na) not urea

Increase plasma osmolality

Increase ADH





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Females' Notes

Explanation

■ Males' Notes





Osmoreceptors in hypothalamus trigger

release of ADH.

- Dehydration
  - ADH released
- •Overhydration
  - ADH inhibited

In loop of Henley high urine conc.

→increase osmotic pressure  $\rightarrow$  ADH  $\rightarrow$  water

retention

Kidneys retain more water. urine output

High blood osmotic pressure stimulates hypothalamic osmoreceptors Osmoreceptors

Osmoreceptors activate the hypothalamic neurosecretory cells that synthesize and release ADH

Nerve impulses liberate ADH from axon terminals in the posterior pituitary gland into the

bloodstream

ADH :

Sudoriferous (sweat) glands decrease water loss by perspiration from the skin

Low blood osmotic pressure inhibits hypothalamic osmoreceptors

Inhibition of osmo-

receptors reduces or stops ADH secretion

Thirst Hypothalamus **Drinking reduces** blood osmolarity ADH to set point. Pituitary Increased gland permeability Distal tubule H,O reabsorption helps STIMULUS: Increase in blood prevent further osmolarity osmolarity increase. Collecting duct

which decreases

Arterioles constrict. which increases blood pressure

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Homeostasis:

**Blood osmolarity** 

(300 mOsm/L)







#### Secretion of ADH (non-osmotic stimuli)

- Stress, major surgery, hypovolemic shock and hemorrhage.
- Hypovolemia (hypotension) is sensed 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 the inhibiting impulses from pressure receptors on hypothalamic cells that secrete ADH.
- ADH released causing vasoconstriction and (water retention) by the kidney.
- Sensitivity of baroreceptors is less than osmoreceptors – it senses 5 to 10% change in volume.

# Other stimuli that affect ADH secretion

#### Stimuli that increase ADH secretion:

- Pain(hyponatremia(120),edema,oligouria)
- Nausea
- Surgical stress
- Emotional stress
- Sympathetic activation

# Stimuli that decrease ADH secretion:

Alcohol intake (polyuria)

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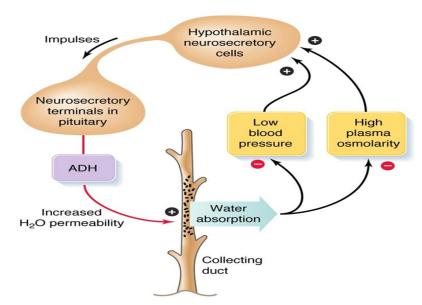
#### **Control of ADH Release**

#### **Osmotic pressure:**

- · Osmoreceptor mediated
- Hyper-osmolality → increase ADH secretion
- Hypo-osmolality → decrease ADH secretion

#### **Volume effects**

- Baroreceptor mediated (vagus nerve)
- hypertension → decrease ADH secretion
- Hypotension → increase ADH secretion



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 in volume
Resulting Amount of ADH	Small	Large (vasoconstriction)

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## Oxytocin



#### **Actions of Oxytocin:**

- Breast-feeding "milk ejection"
  - 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)

#### \*the target cells for oxytocin action are:

- 1- myoepithelial cells of alveoli of the breast. <u>The major action of oxytocin is to cause milk</u> letdown.
- 2- uterine smooth muscle (myometrium). To produce contraction of uterine wall











### Stimuli for the release of Oxytocin:



## A- Suckling "major stimulus".

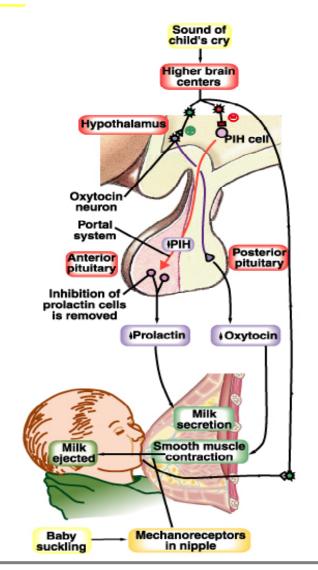
-Baby suckling lead to the stimulate of the mechanoreceptors "in nipple" which send impulses to the Hypothalamus.

- -then the hypothalamus send impulses to the posterior pituitary gland to release oxytocin.
- oxytocin cause myoepithelial cells contraction then milk flows out the nipple.

# B- Conditioned responses to sight, sound or smell of the baby

\*When the mother hears her baby's crying this stimulate the higher centers this which in turn stimulate the hypothalamus leading to the stimulation of both lobes of pituitary gland causing the release of both prolactin and oxytocin.

- This explain why sometimes the mother fond the milk going out even before baby suckling.











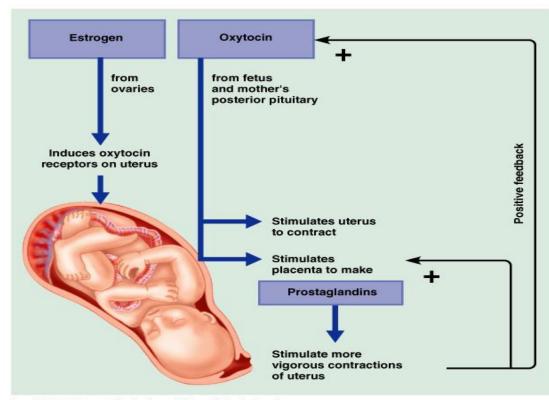






#### C- dilation of cervix during labor

- Stretched cervix will send neural impulses to the hypothalamus which in turn stimulate the posterior pituitary gland to release oxytocin.
- -Oxytocin act on uterine smooth muscle causing uterine wall contraction.
- •Oxytocin is also important after delivery in reducing the size of the uterus back to the normal. How?? Nursing the baby keep good level of oxytocin which help in involution of uterus.



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\*Pitocin is giving I.V to induce labor if the contraction of the uterine wall is weak.







### Other Stimuli that Control release of Oxytocin



**Not Important** 

- 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:**

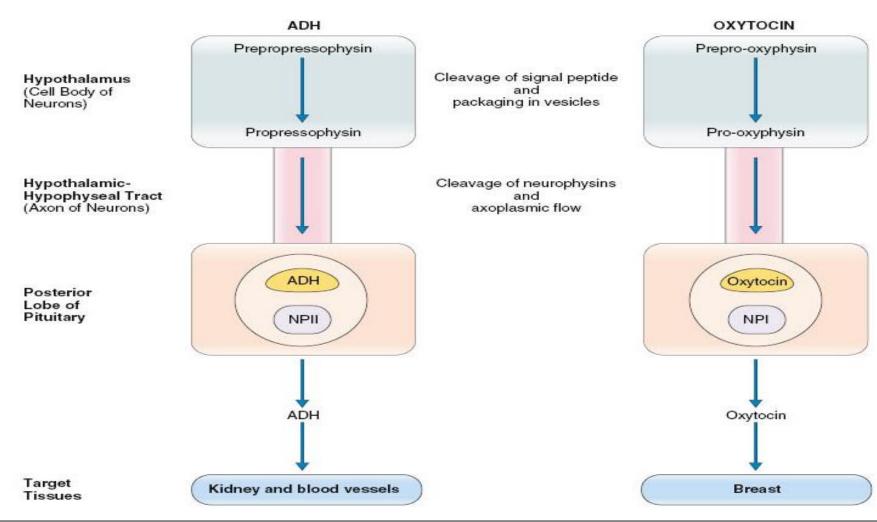
- Autistic group had significantly lower plasma oxytocin levels than in the non-autism group
- Elevated oxytocin was associated with higher scores on social and developmental measures for the non-autistic children

### **Posterior pituitary hormones**





#### POSTERIOR PITUITARY HORMONES



<u>Important</u>

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Explanation

**■ Females' Notes** 





- Oxytocin and Vasopressin are manufactured in the hypothalamus, but released in the posterior pituitary gland.
- Secretions of the posterior pituitary are controlled by Nervous signals from hypothalamus.
- ADH synthesized in the cell bodies of hypothalamic neurons(supraoptic nucleus) and stored in the neurohypophysis (posterior pituitary)
- There are 3 types of receptors for ADH: V1A V1B V2
- The single most important function of ADH is to conserve body water by reducing urine output.

#### Control of ADH secretion by:

- 1- osmotic stimuli: increasing in the osmolality leads to increase its secretion.
- 2- Non-osmotic stimuli.
- Actions of oxytoocin include milk ejection and uterine contraction.
- Stimulatory factors causing oxytocin secretion include: Suckling, sight, smile, or smell of baby, and dilation of the cervix.

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## 1. which of the following stimulate ADH hormone secretion:

- A) hypervolemia.
- B) decrease ECF.
- C) high blood pressure.
- D) decrease plasma osmolarty.

#### 2. ADH is synthesized in:

- A) Posterior pituitary gland.
- B) Anterior pituitary gland.
- C) Supraoptic nuclei of hypothalamus
- D) Paraventricular nuclei of hypothalamus.

#### 3. The major stimulus for oxytocin secretion is:

- A) Suckling.
- B) Cnditioned responses to sound, smell, or sight of baby.
- C) Stretched cervix.

#### 4. The major action of oxytocin is:

- A) Milk production.
- B) Milk ejection.
- C) A and B.

#### 5. The target cells of oxytocin:

- A) Myoepithelial cells of alveoli.
- B) Myometrium.
- C) A and B

2 C 3 A 4 B

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## HEAD FLO



# If there are any Problems or Suggestions, Feel free to contact us:

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