

Primary Aldosteronism

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Primary Hyperaldosteronism.

- By the end of this section of the lecture, Medical student will be able to
 - Knowledge on important causes of primary hyperaldosteronism.
 - Describe the clinical and biochemical abnormalities including sign and symptoms of primary hyperaldosteronism.
 - Knowledge on how to investigate clinical case of hyperaldosteronism

Adrenogenital syndrome

- Basic knowledge on steroid genesis pathway and common enzyme involved in the process of steroid biosynthesis
- Knowledge on clinical presentation of both classical and non-classical forms of Adrenogenital syndrome.
- Skills of investigation needed to diagnose adrenogenital syndrome and important issues on acute and long-term therapies

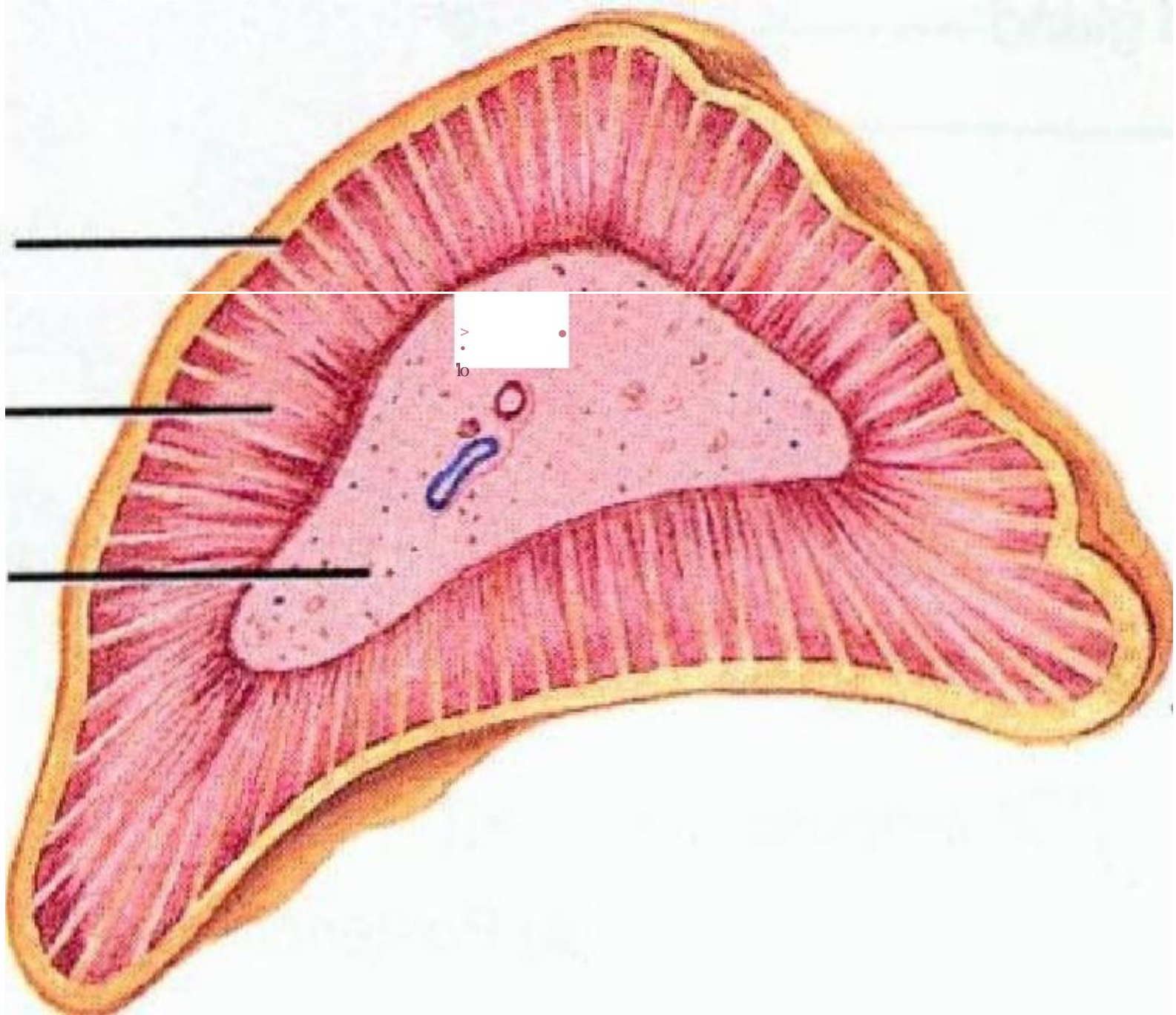
Capsule



Cortex



Medulla



- Adrenal Cortex

- Zona Glomerulosa

- mineralocorticoid production,

- aldosterone

- Zona Fasciculata

- glucocorticoid production,

- cortisol

- Zona Reticularis

- androgen production, including

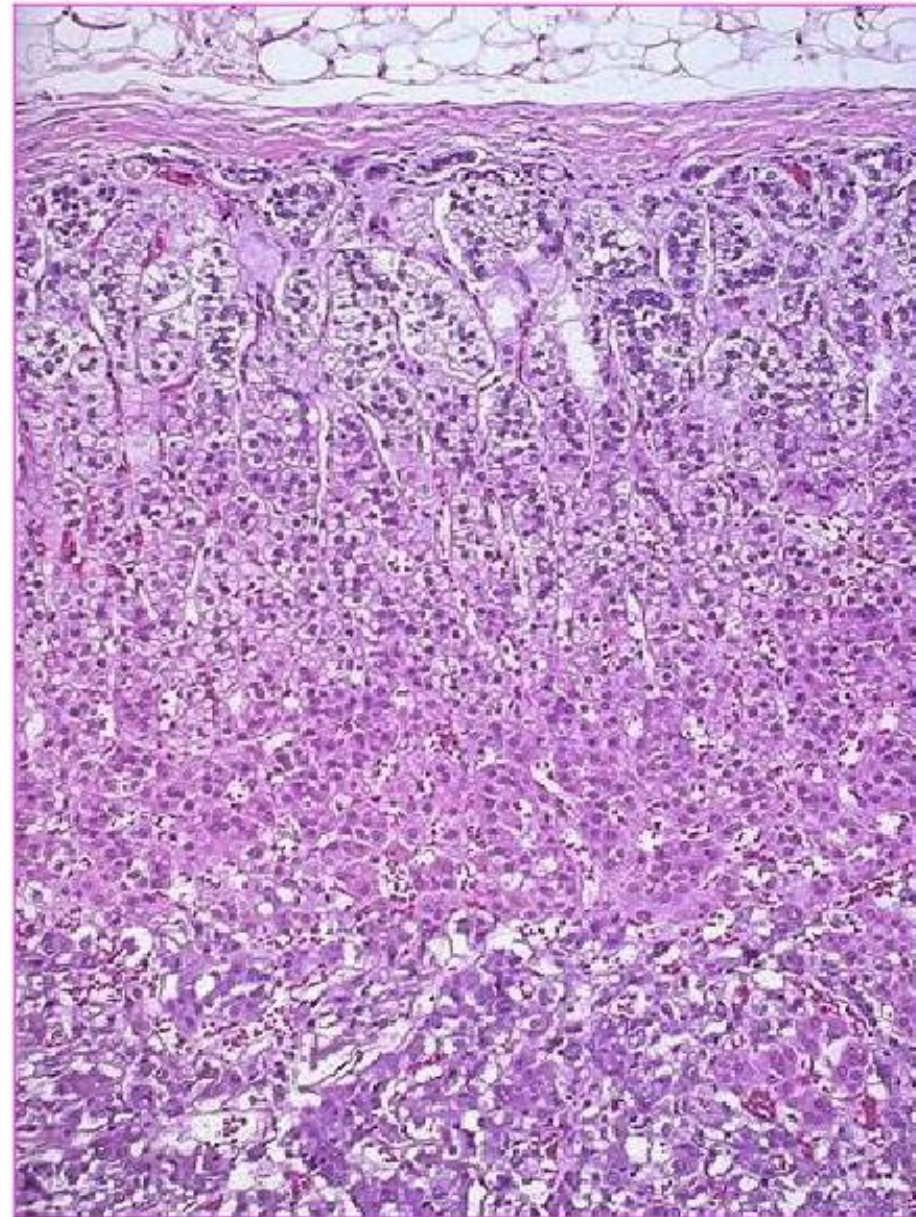
- testosterone

- Medulla

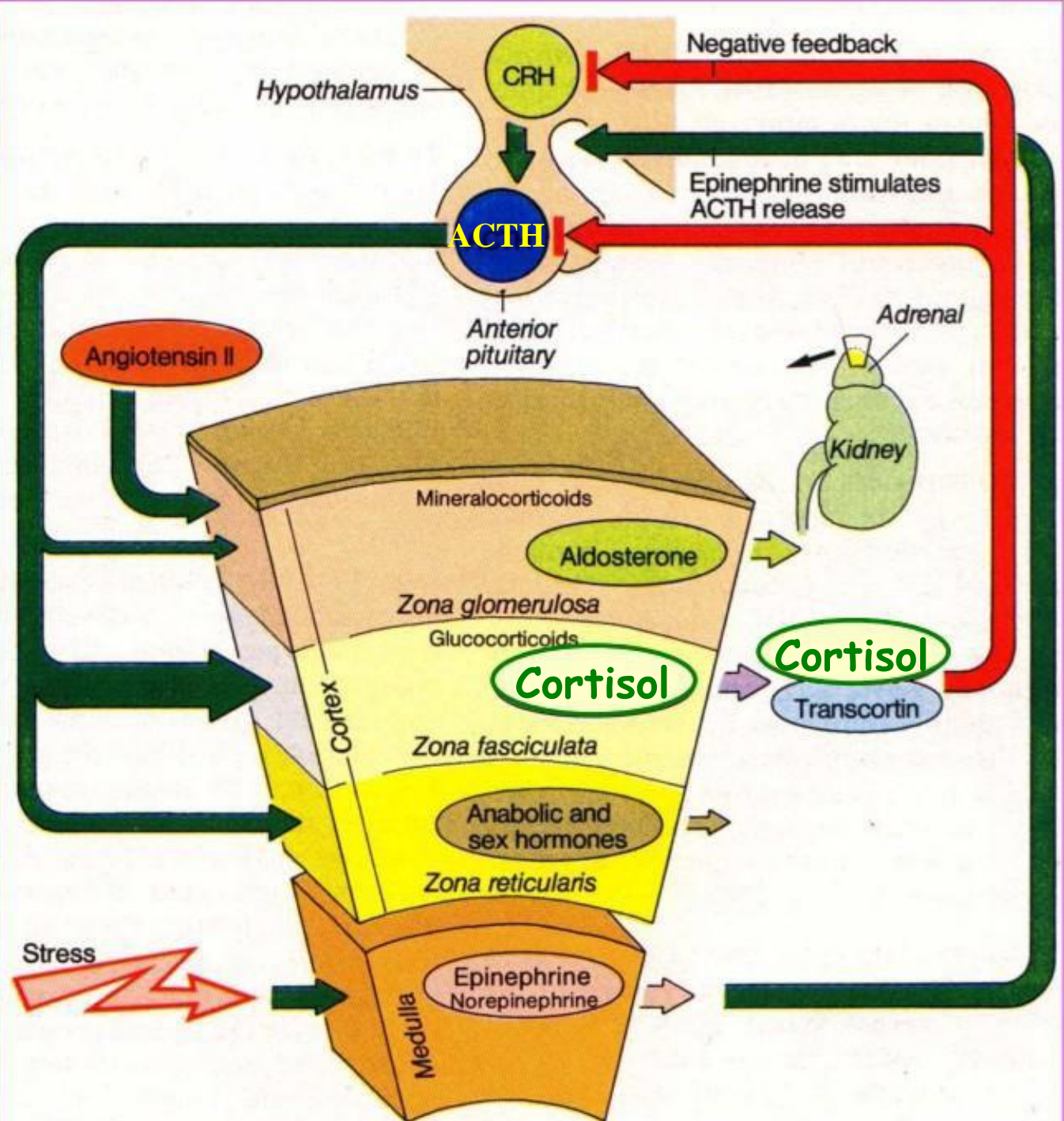
- catecholamines epinephrine

- and

- norepinephrine



Regulation of adrenal gland secretion

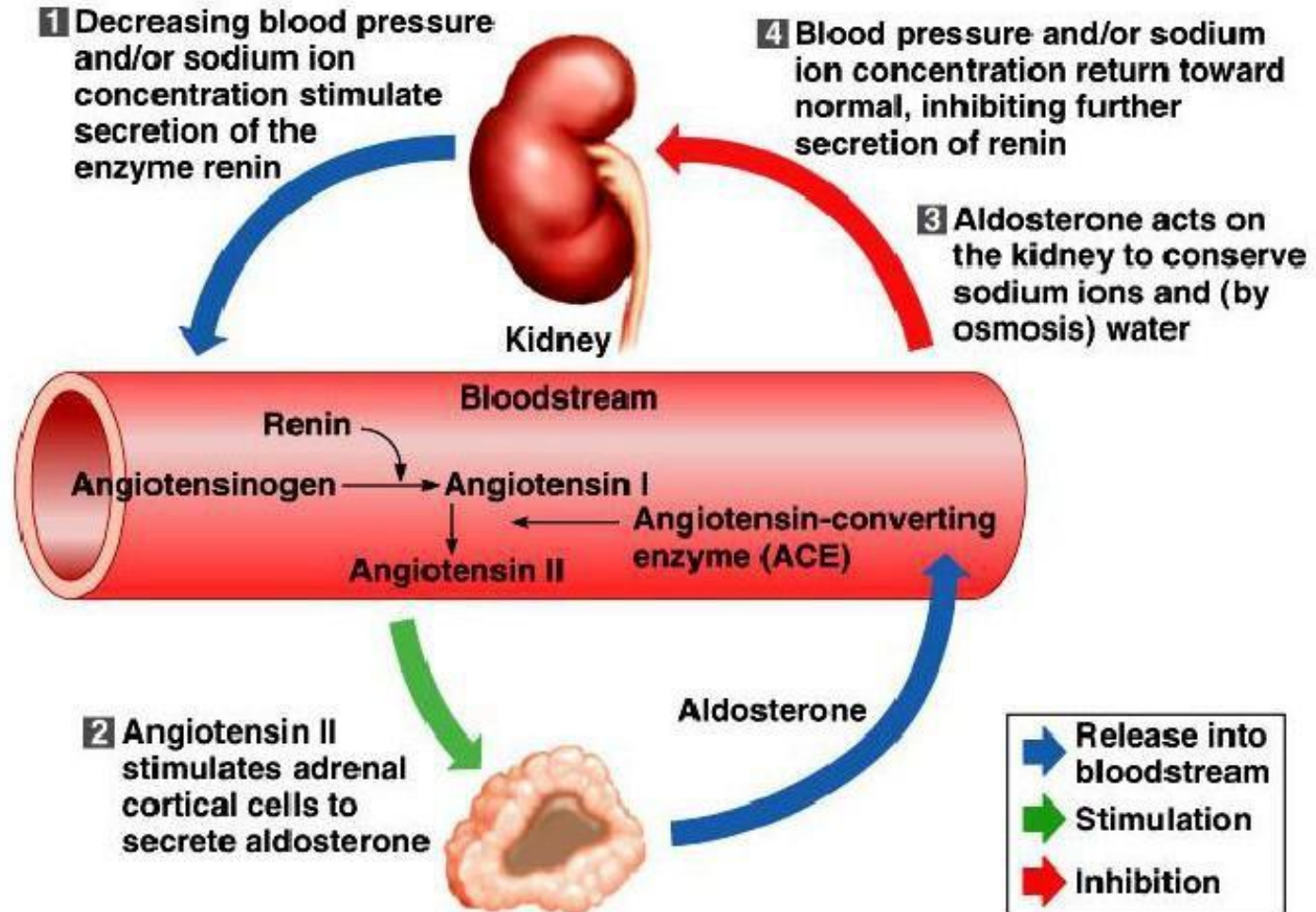


Aldosterone

- Mineralocorticoid
- Regulates concentration of Na^+ and K^+ .
 - Kidney conserves Na^+ .
 - Kidney excretes K^+ .
- Responds to changes in composition of plasma.
- Linked to renin-angiotensin system of kidney

Aldosterone Functions

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Functions of the aldosterone : ANP ↑ → “escape” phenomenon

- (1) Tubular reabsorption of Na^+
Tubular secretion of K^+ or H^+
(Kaliuresis)
- (2) Hypokalemia & muscle paralysis
Hyperkalemia & cardiac toxicity
- (3) Increase tubular H^+ secretion
“alkalosis”
- (4) Circulatory function
absence of aldosterone secretion

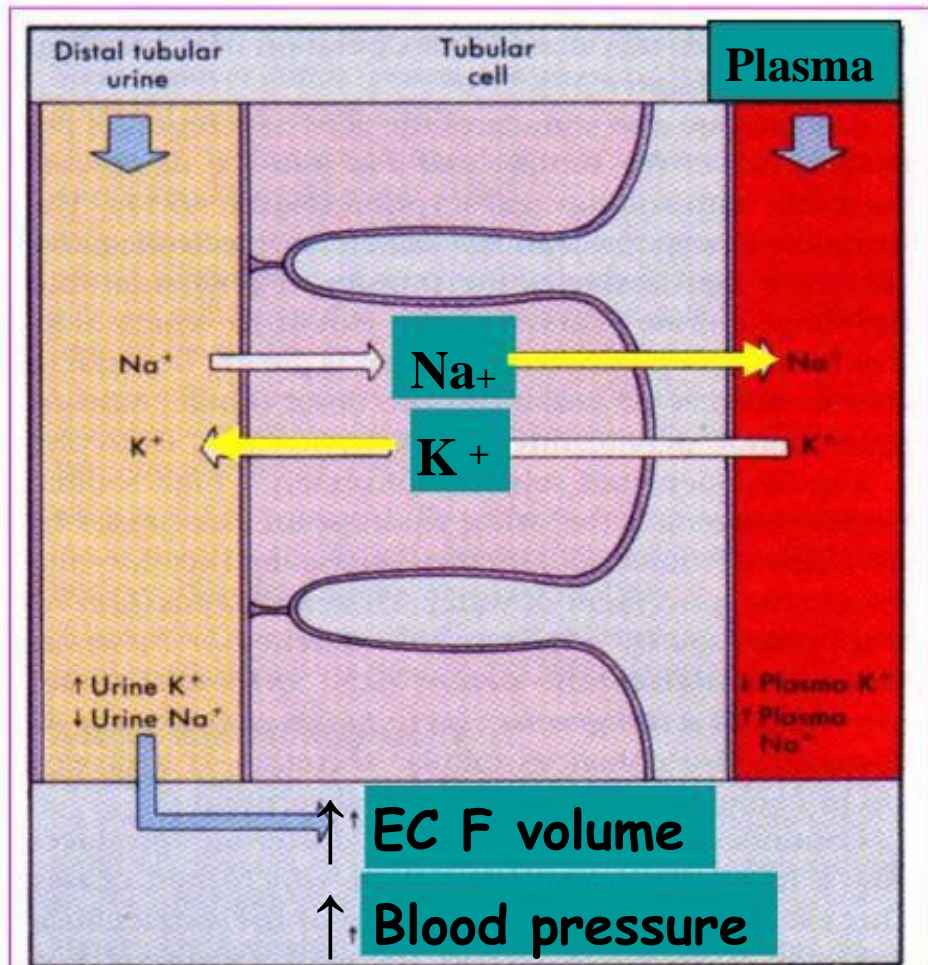


FIGURE 46-11 The action of aldosterone on the renal tubule. Sodium reabsorption from tubular urine is stimulated. Simultaneously, potassium secretion into the tubular urine is increased by responses to the electronegative gradient created by sodium movement. The net result is expansion of extracellular fluid and kaliuresis.

Primary Aldosteronism

Conn's Syndrome

Mineralocorticoids excess

- Conn syndrome is characterized by increased Aldosterone secretion from the adrenal glands
- It was first described in 1955 by J. W. Conn in a patient who had an Aldosterone-producing adenoma

Cause of Hyperaldosteronism

Primary Aldosteronism (Conn's Syndrome)

- | | |
|--|--------|
| -Aldosteron-producing adenoma : Conn's disease | 65-70% |
| -Idiopathic bilateral adrenal hyperplasia | 30% |
| -Adrenal carcinoma | < 1% |

- Aldosterone, by inducing renal distal tubular reabsorption of sodium, enhances secretion of potassium and hydrogen ions, causing hyponatremia, hypokalaemia, and metabolic alkalosis
- Patients with severe hypokalaemia report fatigue, muscle weakness, cramping, headaches, and palpitations.
- They can also have polydipsia and polyuria from hypokalaemia-induced Nephrogenic diabetes insipidus
- Long-standing HTN may lead to cardiac, retinal, renal, and neurologic problems, with all the associated symptoms and signs

Symptoms and Signs

- Hypersecretion of aldosterone may result in:
 - Hyponatremia
 - Hyperchlorhydria
 - Hypervolemia
 - Hypokalemic alkalosis manifested by:
 - episodic weakness
 - Paresthesias
 - transient paralysis
 - tetany
 - Diastolic hypertension with headache
 - Hypokalemic nephropathy with polyuria & polydipsia

Signs, Symptoms, and Laboratory Data in Primary Hyperaldosteronism

Summary slide

- Hypertension
- Headache
- Weakness/ Fatigue
- Paresthesia
- Muscle Cramps
- Polyuria/ Polydipsia
- Arrhythmias
- Hypokalemia
- No Other Cause For Hypertension Or Hypokalemia
- Metabolic Alkalosis
- **Hyperaldosteronism**
- **Hyporeninemia**

Aldosterone / Renin Ratio

- Normal & Patients With Essential Hypertension • < 20
- Primary Aldosteronism • > 30
- $> 90\%$ Sensitivity & Specificity

Overproduction of aldosterone

- Treatment
 - surgical for adenoma
 - medical for hyperplasia with spironolactone



Aldosterone-
producing



Tx : Unilateral adrenalectomy

