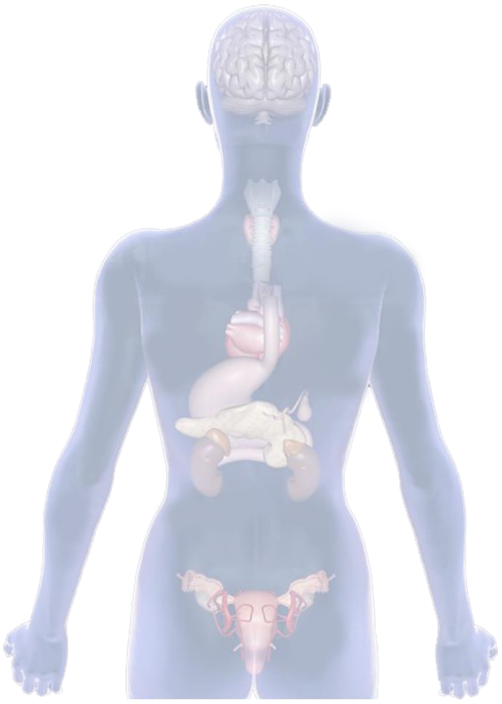
Physiology 435 team

#Summary & MCQs of adrenal gland



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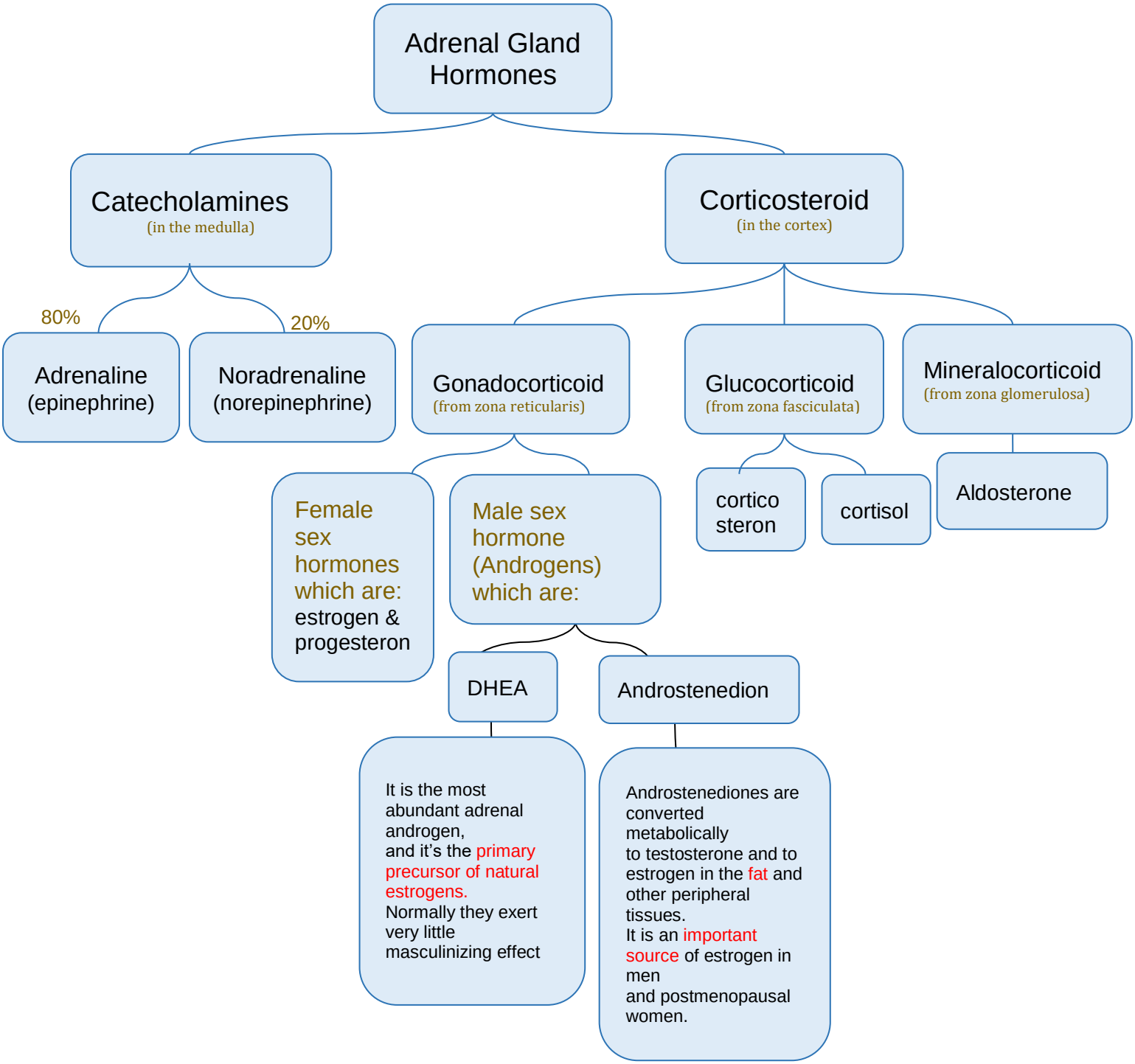
SUMMARY


Adrenal Gland Hormones
Catecholamines
(in the medulla)
Corticosteroid
(in the cortex)
80%
20%
Adrenaline (epinephrine)
Noradrenaline (norepinephrine)
Gonadocorticoid
(from zona reticularis)
Glucocorticoid
(from zona fasciculata)
Mineralocorticoid
(from zona glomerulosa)
Female sex hormones which are:
estrogen & progesteron
Male sex hormone (Androgens) which are:
cortico steron
cortisol
Aldosterone
DHEA
Androstenedion
It is the most abundant adrenal androgen, and it's the primary precursor of natural estrogens. Normally they exert very little masculinizing effect
Androstenediones are converted metabolically to testosterone and to estrogen in the fat and other peripheral tissues. It is an important source of estrogen in men and postmenopausal women.

Hormone	aldosterone	cortisol	androgen	catecholamine
biochem	●60% bound to plasma protein…40% is free. ●Half life: 20 min ●metabolized by the liver and converted to tetrahydroglucuroind.	●90-95% Bound Mostly to transcortin ,Albumin…4% is free ●Half life =60-90 minutes ●Metabolized in liver by reductases &conjugated to glucuronides and excreted via kidney.	**Androgens sources:** 1-Testis (testosterone) : It's the major active androgen. 2-Adrenal gland (adrenal androgen).	**derived from tyrosine :** Tyrosine→Dopamine→Norepinephrine →Epinephrine. (PNMT is an enzyme found in the adrenal medulla that converts norepinephrine to epinephrine therefore epinephrine comes solely from chromaffin cells of the adrenal medulla whereas norepinephrine comes from both adrenal medulla &postganglionic sympathetic nerves).
functions	●Reabsorption of Na+ in the ECF. ●Secretion of K+ &H +in the urine. ●Increases ECF volume & Arterial Pressure.	●**Metabolism:** ***Carb:*** ↑ gloconeognesis in lliver & ↓ glucose uptake by extrahepatic cells>adrenal diabetes. ***Protein:*** anabolic effect on hepatic cells & catabolic effect in extrahepatic cells. ***Lipid:*** lipolysis. ●Reduce all aspects of inflammation. ●has very low mineral activity (Na,K regulation, water retention) ●Increases RBCs count ,platelets and neutrophils. ●Decreases lymphocyte ,eosinophils, basophils. ●Resist the stress by: increase the glocos & fat metabolism, enhances the capacity of glucagon and catecholamines (↑ PB) ●Maintains normal renal function. ●Anti-vitamin D effect	●**Male:** 1. Spermatogenesis 2. Inhibition of fat deposition. 3. Muscle mass. 4. Brain: Androgen levels have been implicated in the regulation of human aggression and libido. 5. Masculinization ●**Female:** 1. Growth of pubic and axillary hair 2. Pubertal growth spurt. 3. relaxation of myometrium. 4. Development and maintenance of female sex drive.	●**Epinephrine** is the more potent stimulator of the heart and metabolic activities ●**Norepinephrine** is more influential on peripheral vasoconstriction and blood pressure
Increase its secretion	● ↑ K or ↓ Na+ in the ECF ● ↑ ACTH causes small transient increases of aldosterone during stress. ●Decreasing blood volume(Hypovolemia) or pressure(Hypotension) →Increased activity of the renin angiotensin system →increased levels of angiotensin II(Ag II increases BP through :vasocontraction of the arterioles & ↑ aldosteron secretion)	stress		●Activation of sympathetic in response to stress (short term stress) ●Cortisol secation has permissive effect on catecholamine.
Inhibit its secretion	ANP			

ABNORMALITIES					
	Primary hyperaldosteronism (Conn's syndrome)	**Cushing syndrome**	**Adrenocortical insufficiency**	**Adrenogenital syndrome**	**Pheochromocytoma**
Cause:	adenoma or nodular hyperplasia of zona glomerulosa → secretes large amounts of aldosterone.	**ACTH independent:** ●adenomas of the adrenal cortex. **ACTH dependent:** ●a denomas of the anterior pituitary → increase ACTH(Cushing's disease). ●abnormal function of the hypothalamus → ↑CRH. ●ectopic secretion	**Primary causes, ie. Addison's disease:** Autoimmune disease, tumors, infection (e.g TB), hemorrhage, metabolic failure, ketoconazole(glucocorticoid antagonist activity) **Secondary causes:** ●Hypopituitarism ●Suppression by exogenous steroids.	**Adrenocortical tumors:** secretes excessive quantities. **Congenital adrenal hyperplasia:** lack of an enzyme (21-hydroxylase)	is a tumor of adrenal medulla :derived from **chromaffin cells** (arise from embryogenic neural crest) → Most tumors secrete epinephrine, NE, and dopamine and can cause episodic hypertension → ↑urinary vanillylmandelic acid(vanillylmandelic acid is a breakdown product of norepinephrine) and plasma catecholamines are elevated , also lt's Associated with neurofibromatosis
Effects:	● ↑ Na →increase in ECF volume and blood volume> hypertension. ● ↓ K →Neuromuscular manifestations(weakness, paresthesia, intermittent paralysis) ● ↓ H →alkalosis. ● ↓ plasma renin concentration .	**On Carbohydrate Metabolism:** ● ↑blood glucose. **On Protein Metabolism:** ● Decreases tissue proteins. Almost everywhere in the body (except liver). ● Severe weakness, ● Thinning of the skin(STRIAE). ● osteoporosis. ● Suppressed immune. **Abnormal Fat Redistribution:** Truncal obesity. buffalo torso ,Moon face. **80% of patients have hypertension, because of the mineralocorticoid**	**Due to Mineralocorticoid Deficiency, absence of aldosterone:** ●the volume depletion ●Increased excretion of sodium and water. ●low blood pressure, **Due to Glucocorticoid Deficiency:** ●Hypoglycemia. weakness , Feeling sick and vomiting & weight loss(diarrhea or constipation). **Due to ↑ ACTH :** ●Darkened pigmentation	**FEMALE:** **Before birth:** Pseudohermaphroditis mXX true female with external male genitalia **after birth:** she develops virile characteristics **MALE:** **Before puberty:** rapid development of the male sexual Organ but No spermatogenesis. **After puberty:** the virilizing characteristics of adrenogenital syndrome are usually obscured by the normal virilizing characteristics However, the excretion of 17-ketosteroids (derived from androgens) in urine may be 10 to 15 times normal, used in diagnosing the disease	classic triad **(palpitations ,headache & sweating)**
treatment	usually surgical removal,Spironolactone (K sparing diuretic)	Reducing corticosteroid use - Surgery - Radiation therapy - Medications			**DX:** High plasma catecholamine,Increased metabolites [VMA5] in urine **Treatment:** surgical resection.

Abnormalities of adrenocortical function:

Cushing's Syndrome

Increase secretion of corticosteroid (cortisol)
Increase or decrease ACTH (depending on etiology)

Altered appearance, bruising, muscle wasting, Hypertension, hypokalemia, diabetes.

Addison's disease

Decrease secretion of glucocorticoids (cortisol) and mineralocorticoids (aldosterone)

Hypoglycemia, hyponatremia, Hyperkalemia, raised urea.

Acute: abdominal pain, vomiting, dehydration and hypotension.

Chronic: fatigue, pigmentation, anorexia, weight loss and postural hypotension.

Conn's (primary hyperaldosteronism)

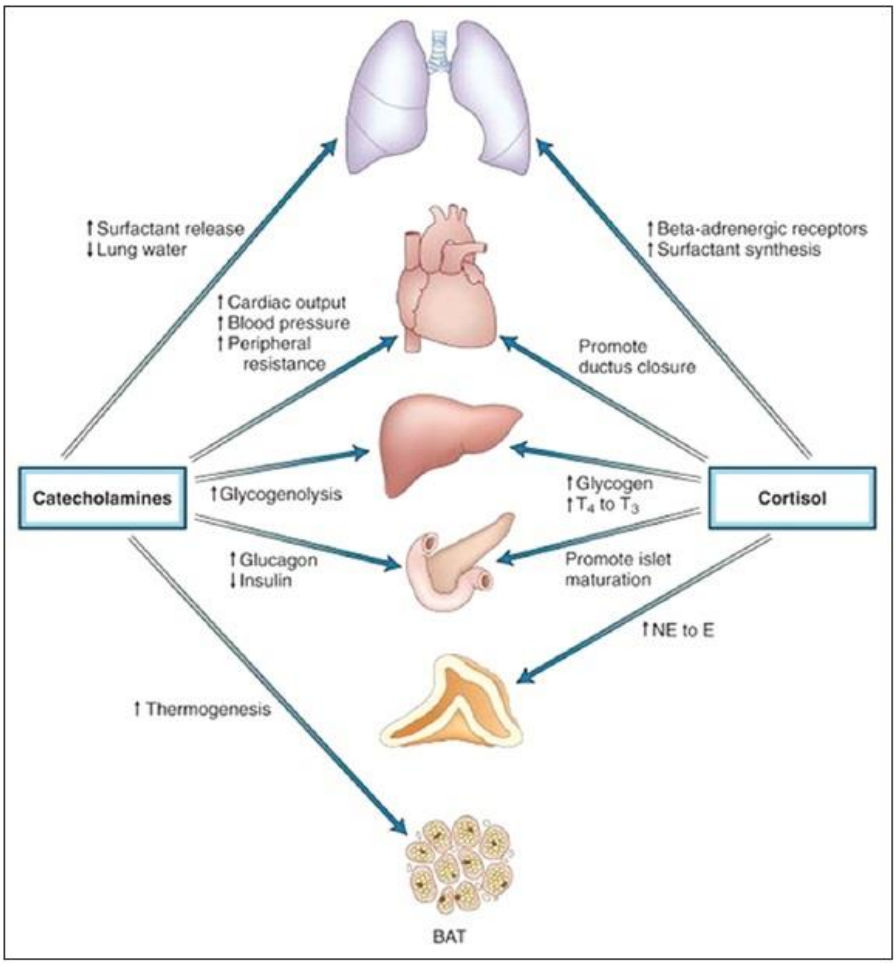
Increase aldosterone

Hypertension, hypokalemia, alkalosis.

Secondary hyperaldosteronism

Increase aldosterone and Renin

Hypertension, hypokalemia, alkalosis.



MCQs

1. Glucocorticoids is produced by which area?

a. Zona Glomerulosa
b. Zona Fasciculata
c. Zona Reticularis
d. B&C

2.Other than CRH, what can stimulate the secretion of ACTH?:

a. GH
b. ADH
c. TRH
d. Dopamine

3. Noradrenaline involved in which of the following:

a. Inhibition of the heart
b. Increase the blood pressure
c. stimulation of the metabolism.
d. Peripheral vasodilation

4. The rate-limiting step in the synthesis of all steroid is done by which enzyme?

a. 21-hydroxylase
b. 17- hydroxylase
c. Side chain cleavage enzyme
d. Anhydrase

5. Catecholamines can lead to <u>hyperglycemia</u> due to:

a. lipolysis
b. Glycolysis
c. Glycogenesis
d. Glycogenolysis

6.which cells Aldosterone act on?

a. Parietal cells
b. Alpha-intercalated cells
c. Principal cells
d. Tubular cells

7. Hyperpigmentation is a sign of:

a. Addison disease
b. cushing disease
c. Ectopic ACTH
d. A&C

8. What can stimulate aldosterone secretion:

a. Increase K+ in blood
b. Increase Na+ in blood
c. CRH
d. ANP

9. One of the manifestation of Conn's syndrome is:

a. hypernatremia
b. hypotension
c. Increased Renin level in blood
d. hypokalemia

10. One of the metabolic effect of cortisol is:

a. Proteoanabolic on extra hepatic tissue
b. Increase glycogen synthesis in the liver
c. Proteocatabolic in the liver
d. Decrease utilization of fat for energy

11. What is the enzyme found in adrenal medulla that converts Norepinephrine to Epinephrine:

a. Tyrosine hydroxylase
b. Nuclease
c. Phenylethanolamine N-methyltransferase (PNMT)
d. Phenylalanine hydroxylase.

12. The most amount of secretion of the adrenal medulla is …….. and the least amount of secretion is ……... .

a. Adrenaline, Noradrenaline
b. Dopamine, Adrenaline
c. Adrenaline, Dopamine
d. Dopamine. Noradrenaline

13. Adrenaline involved in which of the following:

a. Inhibition of the heart.
b. Decrease the blood pressure.
c. stimulation of the heart.
d. Peripheral vasoconstriction.

Answer key:

1 (d) | 2 (b) | 3 (b) | 4 (c) | 5 (d) | 6 (c) | 7 (d) | 8 (a) | 9 (d) | 10 (b) | 11 (c) | 12 (c) | 13 (c) |

14. Cortisol has a permissive effect on:

a. ADH
b. Norepinephrine
c. GH
d. ANP

15. Cortisol can increase the the count of which of the following:

a. RBC
b. Lymphocytes
c. Eosinophils
d. basophils

16. The Primary precursor of natural estrogen is:

a. DHEA
b. Corticosterone
c. Androstenedione
d. Cortisol

18. In men, small amount of testosterone can be converted into estrogen, where does it happen?

a. Liver
b. Skeletal muscle
c. Adipose tissue
d. testis

17. Main source of estrogen in men & postmenopausal women is:

a. Corticosterone
b. DHEA
c. Cortisol
d. Androstenedione

18. The precursor of all adrenal medulla secretion is:

a. Tyrosine
b. Tryptophan
c. MSH
d. PNMT

Answer key:

14 (b) | 15 (a) | 16 (a) | 17 (d) | 18 (a)