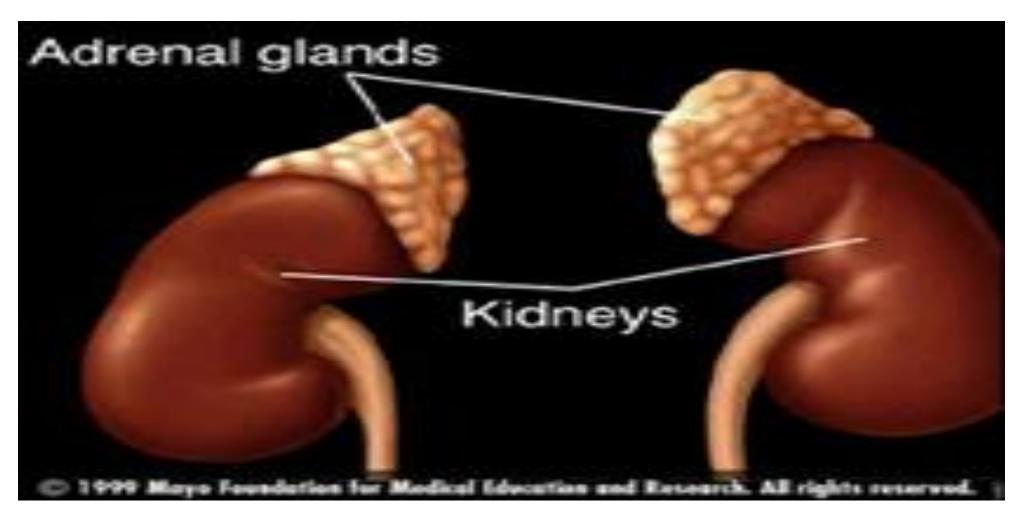
### **THE ADRENAL GLANDS**

The adrenal glands, each of which weight about 4 grams, lie at the superior poles of the two kidneys.

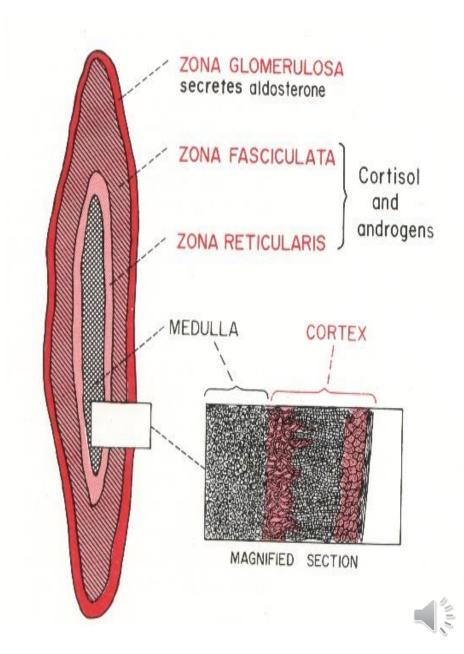




### THE ADRENOCORTICAL HORMONES

Each gland is composed of two distinct parts:

- 1. The inner adrenal medulla (20% of the gland) is functionally related to the sympathetic nervous system; it secretes catecholamines (adrenaline, nor adrenaline, and dopamine) in response to sympathetic stimulation.
- 2. The outer adrenal cortex (80% of the gland) secretes steroid hormones (corticosteroid) which are essential for the life and includes:



- 1. Mineralocorticoids (aldosterone): affect the electrolytes of ECF, Na<sup>+</sup> and K<sup>+</sup>
- 2. Glucocorticoids (cortisol): It is affect by increasing blood glucose concentration, an additional affect on both protein and fat metabolism.
- 3. Androgenic hormones: (small amounts): It is effects in the body as the male sex hormone testosterone.



# **Glucocorticoids:**

- **1. Cortisol** (very potent, account for 95% of all glucocorticoid activity).
- **2. Corticosterone** (4% of total glucocorticoid activity, less potent than cortisol).
- 3. Cortisone (synthetic, almost as potent as cortisol).
- **4. Prednisolone** (synthetic, 4 times as potent as cortisol).
- 5. Dexamethasone (synthetic, 30 times as potent as cortisol).



## **Effect of glucocorticoids (cortisol):**

- 1. Effects on CHO metabolism: Stimulation of gluconeogenesis and ↓Glucose utilization by the cells will lead to elevated blood glucose concentration
- 2. *Effect on protein metabolism*: cause increase protein catabolism (muscle weakness) AA concentration in the blood will increase.

## 3. Effect on fat metabolism:

- a. Cortisol causes mobilization of FA from the adipose tissue→ ↑ FFA in plasma→↑ utilization of FA for energy.
- b. Ketogenic effect

Obesity caused by cortisol: Excess cortisol secretion lead to excess deposition of fat in the chest and head regions of the body, giving to a *buffalo like torso* and a rounded face called *moon face*.



- 4. Effects on blood cells and lymphatic organs.
- 5. Resistance to stress.
- 6. Anti-allergic effect.
- 7. Anti-inflammatory effect.
- **8. Other effects:** glucocorticoids in high doses lead to: \JGH secretion, \JTSH secretion and accelerate the maturation of surfactant in the lungs of fetus



## THE MINERALOCORTICOIDS

- 1. Aldosterone (very potent, account for 95% of mineralocorticoid activity).
- **2. Deoxycorticosterone** (one fifteenth as potent as aldosterone, very small quantities secreted).
- **3. Corticosterone** (slight activity).
- **4. Cortisol** (very slight activity).
- **5. Cortisone** (synthetic, slight activity).

## **Physiological effects**

- 1.  $\uparrow$  Reabsorption of Na<sup>+</sup> in exchange for (K<sup>+</sup>) and (H<sup>+</sup>) ions in the renal. Thus causing water retention, urine acidity, and  $\downarrow$  H<sup>+</sup> ion concentration in ECF.
- 1. ↑ Reabsorption of Na<sup>+</sup> ions from the sweat, saliva, gastric juice, and intestinal secretion.



### **Regulation of aldosterone secretion:**

- *K*<sup>+</sup> *ion concentration in the ECF*: increase 1meq/L in K<sup>+</sup> concentration in ECF can directly stimulate the zona glomerulosa cells to secrete aldosterone.
- *Renin-angiotensin system:* elevated values of renin and angiotensin lead to † aldosterone secretion.
- Quantity of body sodium: decreased Na<sup>+</sup> lead to → ↓ECF volume → ↑renin secretion → formation of angiotensin → stimulate aldosterone secretion.
- ACTH: ACTH. It also has effect on aldosterone secretion



## **Abnormalities of adrenocortical secretion**

Hypoadrenalism (Addison's disease): failure of adrenal cortices to produces adrenocortical hormones

<u>Causes</u>: (TB, autoimmune diseases, and invasion by cancer)



## **Features:**

# 1. Mineralocorticoid deficiency

- ↓Na<sup>+</sup> reabsorption(Na<sup>+</sup>, Cl<sup>+</sup>, and water lost in urine)→(↓ECF volume)
   → shock → death
- Hyperkalemia (↑ K<sup>+</sup>)
- Acidosis

# 2. Glucocorticoid deficiency

- Impossible to maintain normal glucose concentration between meals (impaired gluconeogenesis)
- $\downarrow$  of fat and protein  $\rightarrow$ ( $\downarrow$  metabolic function of the body)
- Melanin pigmentation in the skin and mucous membrane → (↓
  cortisol secretion) → (↑ACTH and MSH secretion) → melanin
  pigmentation.



## **Hyperadrenalism (Cushing's syndrome):** († increase cortisol secretion)

- 1. Tumor in the adrenal cortex  $\rightarrow \uparrow$  secrete cortisol
- 2. Hyperplasia of both adrenal cortices (\( \frac{ACTH}{ACTH} \) secretion)
  - a. ↑ACTH secretion from anterior pituitary (microadenoma)
  - b. Ectopic secretion of ACTH by tumor e.g. abdominal carcinoma
- 3. Iatrogenic (drug up use)





#### Features:

- Buffalo torso.
- Moon face.
- Acne and hirsutism.
- Hypertension (80%).
- Adrenal diabetes.
- Sever muscle weakness.
- Suppressed immunity→ infection (death )
- Purplish striae (abdomen).
- *Osteoporosis* (bone weakness)→ fracture.



#### **Diagnosis:**

- Clinical features.
- ↑ plasma cortisol level.
- \(\gamma\) secretion of 17 hydroxysteroid in urine.

**Treatment:**  $\rightarrow$  according to the cause





- Hypertension (80% of the patients)→ slight mineralocorticoid effect of cortisol
- ↑in blood glucose concentration (↑gluconeogenesis) →diabetes mellitus.
- Severe muscle weakness (protein catabolism).
- Suppressed immunity→ death(infection)
- Diminished collagen fibers in subcutaneous tissues (SC),
   SC tissues tears easily→ purplish striae (abdomen).
- Lack of protein deposition in the bones osteoporosis (bone weakness) fracture.



## **Primary aldosteronism:**

- 1. Tumor of the adrenal cortex $\rightarrow$  secrete aldosterone.
- 2. Hyperplasia of adrenal cortices secrete aldosterone rather than cortisol

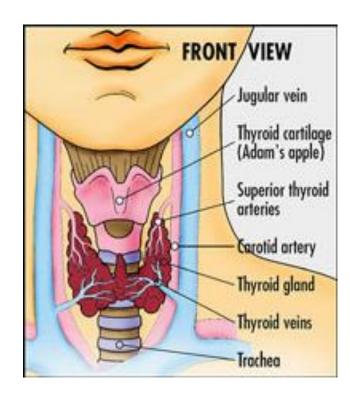
## **Features**:

- ↑Na+ reabsorption → ↑ Na+ concentration in ECF (slight) →
   ↑ECF volume → almost always hypertension
- $\downarrow K^+$  in ECF (hypokalemia)  $\rightarrow$  periods of muscle weakness



#### THE THYROID GLAND

- The thyroid gland is consists of 2 lobes (R and L lobes) connected by a bridge of tissue called *the thyroid isthmus*.
- Follicular cells  $\rightarrow$  thyroxine (90%) and triiodothyronine (10%) Parafollicular cells  $\rightarrow$  calcitonin
- T3 is 4 times as potent as T4 but its present in lower concentration and its duration of action is shorter  $\rightarrow$  (effect of T3 = effect of T4).
- Considerable portion of T4 is converted to T3 in blood & tissues





**Thyroglobulin:** is a large glycoprotein synthesized in the thyroid cells.

# **Transport of thyroid hormones:**

More than 99% of T4 and T3 are bound to plasma proteins.

## Functions of thyroid hormones in tissues:

- 1. An\(\gamma\) in the overall metabolic rate: thyroid hormones \(\gamma\) metabolic rate of almost all tissues of the body (except brain, retina, testes, spleen, and lungs).
- 2. In children, stimulation of growth.
  - Hypothyroidism → growth retardation
  - Promote growth and *development of brain* during fetal life and 1<sup>st</sup> few years of postnatal life (lack of thyroid hormones during this period → mental retardation)



### **Effects of thyroid hormones on specific body mechanisms:**

- **1.** Effect on CHO metabolism:
- $\uparrow$  Thyroid hormones  $\rightarrow$  decrease cholesterol, TG and  $\uparrow$ FFA.
- Thyroid hormones → increase in basal metabolic rate (60-100%).
   decrease body weight and increases the appetite.
- 2. Effect on the CVS:
  - a. Blood flow and cardiac output:(\(\extrm{ in COP}\)
  - b. Heart rate: increase in heart rate and strength of heart rate
  - c. Blood volume: increased (vasodilatation).
  - d. Arterial pressure: Diastolic blood pressure (↓), Systolic blood pressure (↑)



- 1. Effect on respiration:  $\uparrow$  metabolism  $\rightarrow$  ( $\uparrow$  in  $0_2$  utilization and  $CO_2$  production)
  - → increase rate and depth of respiration.
- 2. Effect on Gastrointestinal system:
  - a. ↑ in appetite and food intake
  - b. †secretion
  - c.  $\uparrow$  motility  $\rightarrow$  diarrhea
- 3. Effect on CNS:
  - ↑Thyroid hormone → extreme nervousness
- 11. Effect on the function of the muscle:
- ↑ in the hormone → muscle reacts with vigor
- Excessive ↑ in the hormones → muscle weakness (protein catabolism)
- Lack of hormone → muscle become sluggish and relaxes slowly after contraction
- Muscle tremor: fine muscle tremor is characteristic sign of hyperthyroidism.
- 12. Effect on sleep:
- Hyperthyroid→ difficult to sleep
- Hypothyroid → extreme somnolence.



# Causes: <u>Hyperthyroidism</u>:

- •(Toxic goiter, thyrotoxicosis, Graves, disease)
- Toxic adenoma

## **Graves**, disease:

- Gland hyperplastic, 2-3 times of normal size (goiter).
- Rate of thyroid hormone secretion increased 5-15 times of the normal (changes similar to that produced by TSH stimulation of the gland).
- TSH level low



## **Symptoms of hyperthyroidism:**

- intolerance to heat, ↑ sweating, weight loss, diarrhea, muscular weakness, nervousness, inability to sleep, ↑pulse pressure, and tremor of the hands.
- Exophthalmos: protrusion of the eyeball (occur in most hyperthyroid), usually disappear or  $\downarrow$  by treatment of hyperthyroidism







### **Hypothyroidism**

- **1.** Autoimmunity → destruction of the gland (inflammation → fibrosis) → hypothyroidism.
- 2. Endemic colloid goiter: Iodine deficiency → decrease thyroid hormone synthesis and secretion → increase TSH → stimulation of secretion of thyroglobulin (colloid) → goiter
- **3. Food** which has propylthyiouracil type of antithyroid activity e.g. of these goitrogenic substances are turnips and cabbage.

## **Characteristic of hypothyroidism:**

• Fatigue and extreme somnolence (sleeping 14-16 hours a day), extreme muscular sluggishness, bradycardia, †body weight, constipation, mental sluggishness, edematous swelling of the body (myxedema) and atherosclerosis († plasma cholesterol).

