Endocrine Disorders

Contents

- Overview of anatomy & physiology of endocrine glands
- Disorders of pituitary gland
  - Anterior pituitary gland disorder
    - Hyperpituitarism
    - Hypopituitarism
  - posterior pituitary gland disorder
    - Diabetes insipidus
    - SIADH
- Disorders of Thyroid gland
- Disorders of parathyroid gland
- Disorders of adrenal gland
- Diabetes Mellitus

Learning OBJECTIVEE

By the end of this lecture, you should be able to:

- **Describe** anatomic and physiologic overview of endocrine glands
- Discuss the pituitary functions and dysfunction
- Describe clinical manifestations of pituitary tumors, assessment and diagnostic findings, and an overview of medical management.
- Define diabetes insipidus, describe its manifestations, diagnostic evaluation, medical and nursing management.

Anatomic and Physiologic Overview

- **Humans rely on two systems for regulation:**

1. **Nervous System** :

- a system of nerve cells called neurons.
- It consists of a CNS (brain &spinal cord) & PNS.
- Typical responses are fast & short lasting.

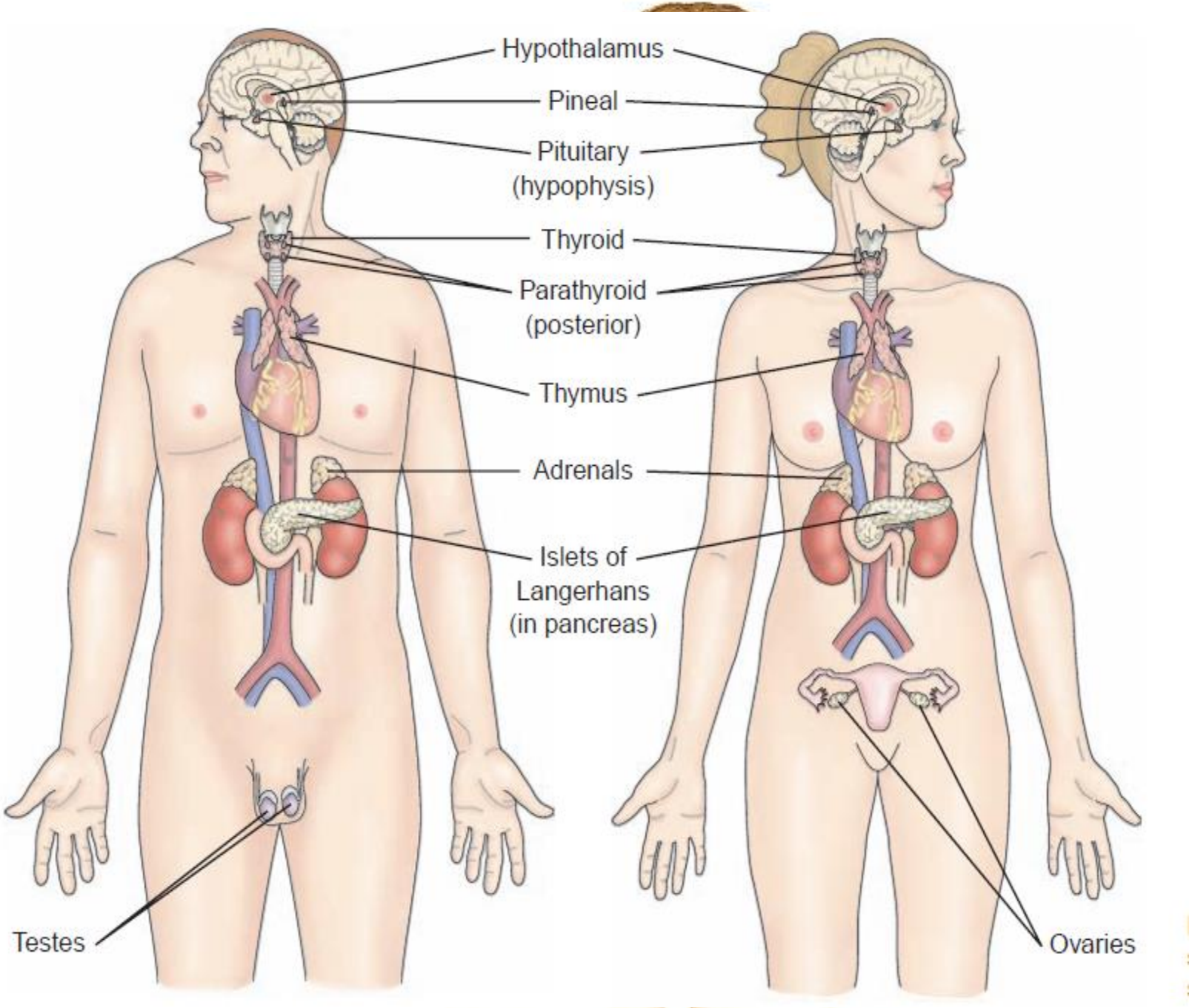
2. **Endocrine System** :

- a system of ductless glands that secrete hormone into the blood.
- Hormones travel to target tissues.

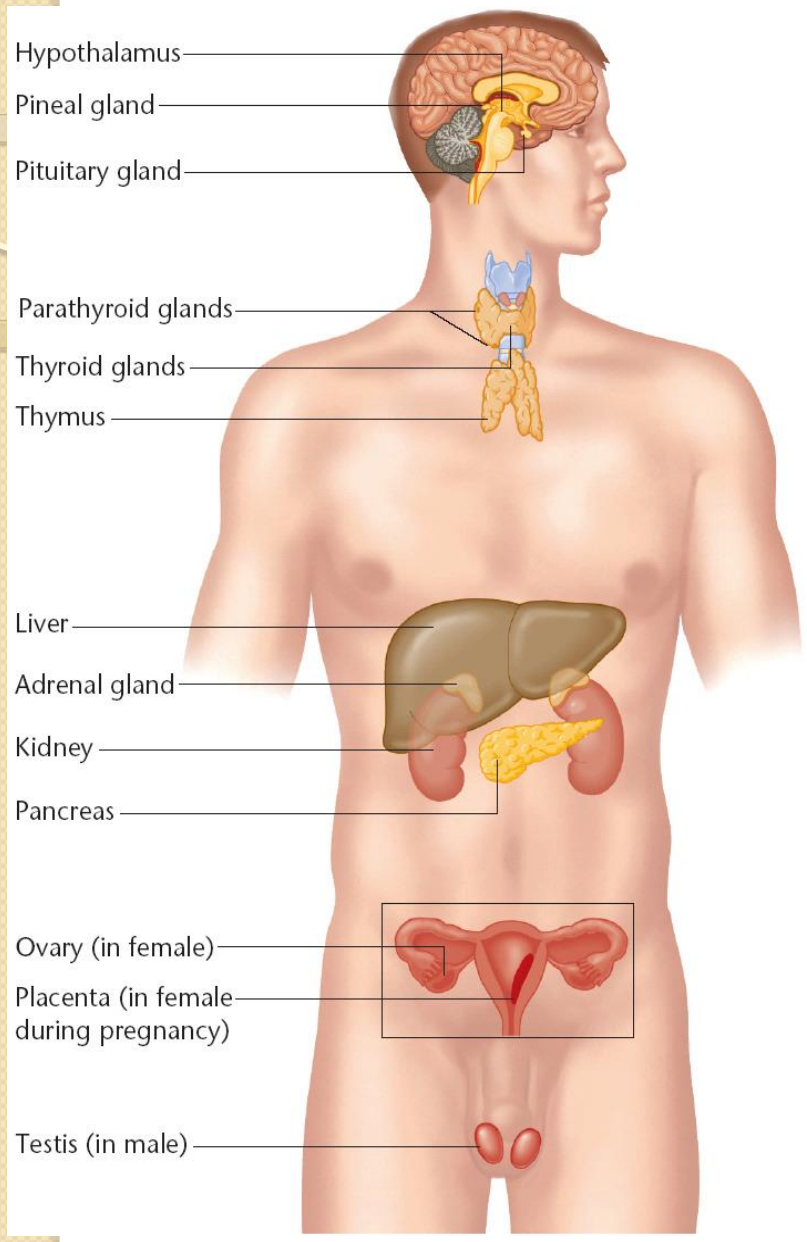
Anatomic and Physiologic Overview…

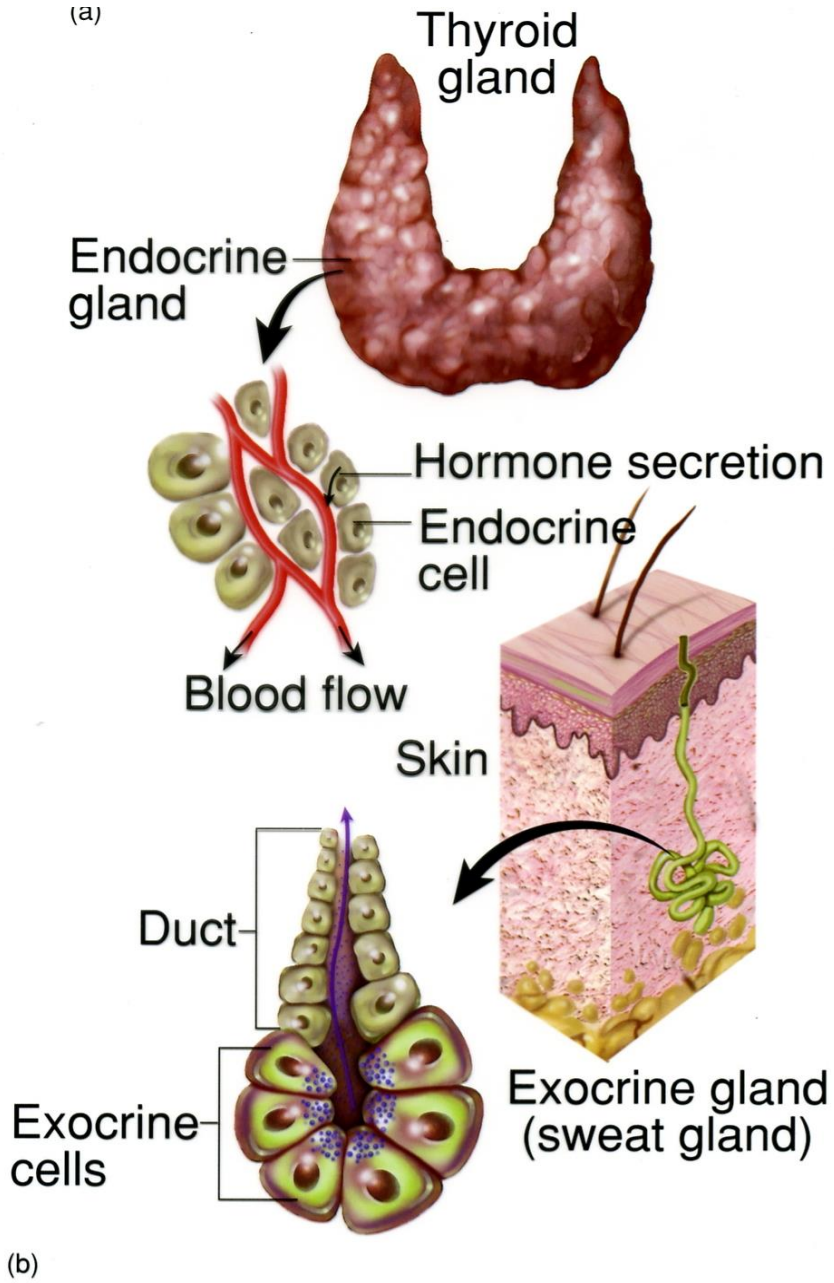
- The endocrine system release chemical substances (hormones) to regulate & integrate body functions.
- Major hormones are produced by the endocrine glands.
- some are also produced by other tissues. Like
  - **GI** mucosa produces hormones (gastrin, secretin…) that are important in the digestive process.
  - **kidneys** produce erythropoietin hormone that stimulates the bone marrow to produce RBCs; and the WBCs w/c actively participate in inflammatory & immune responses.

Identi


Hypothalamus
Pineal
Pituitary
(hypophysis)
Thyroid
Parathyroid
(posterior)
Thymus
Adrenals
Islets of
Langerhans
(in pancreas)
Testes
Ovaries

Slide 2 – Endocrine system





Anatomic and Physiologic Overview…

- endocrine system is composed of several glands:
  - ✓ the pituitary & parathyroid,
  - ✓ thyroid & adrenal glands,
  - ✓ pancreatic islets,
  - ✓ ovaries, and testes
- **endocrine** glands :**most hormones secreted from these** are released directly into the bloodstream.
- Exocrine glands: secrete their products through ducts onto their receptors site. Such as sweat, salivary, tears… glands

Anatomic and Physiologic Overview…

- **Function and Regulation of Hormones**

- Hormones help regulate organ function in concert with the nervous system(dual regulatory systems)

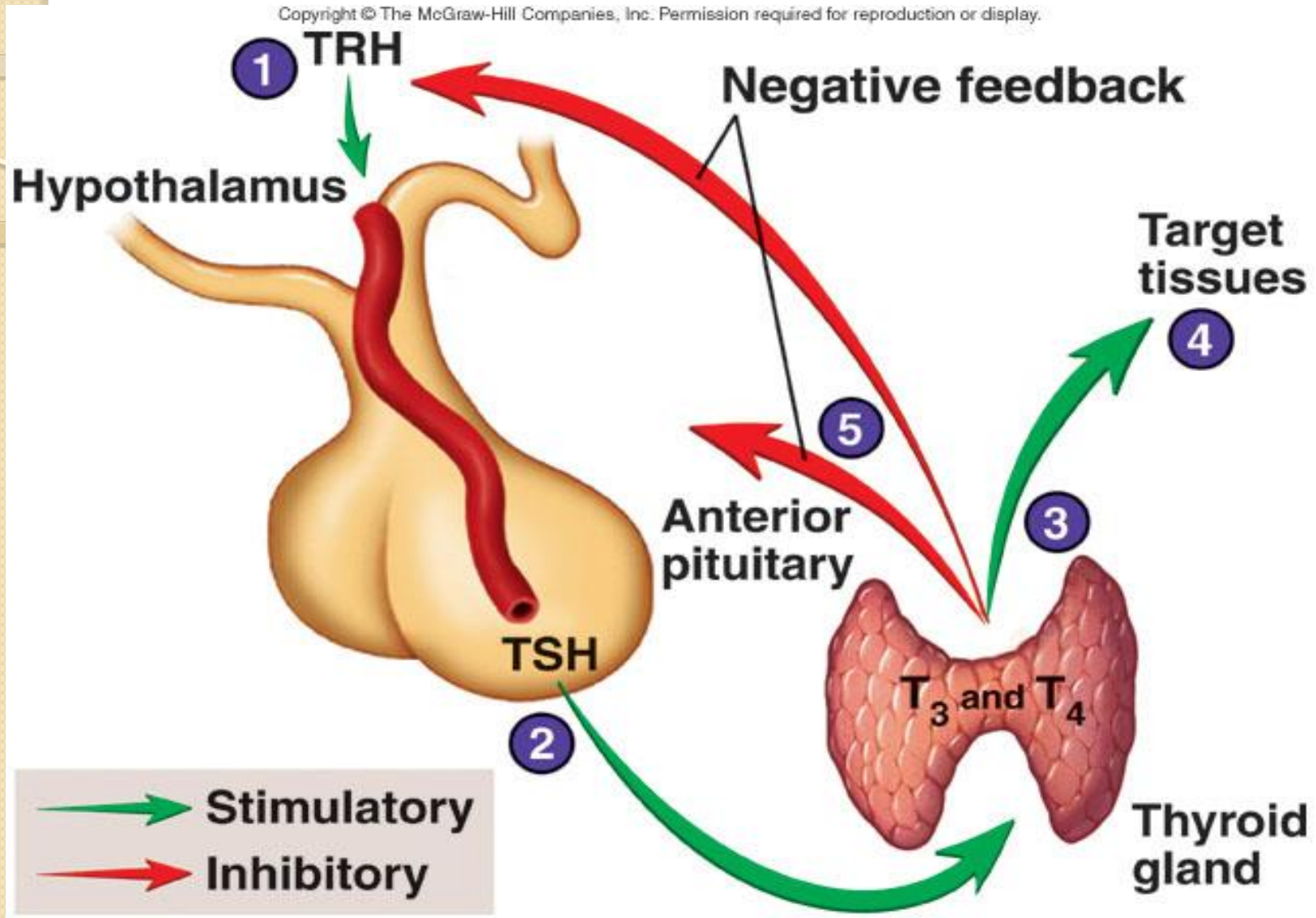
- Rapid action by the nervous system is balanced by slower hormonal action.

- This permits precise control of organ functions in response to varied changes within and outside the body.

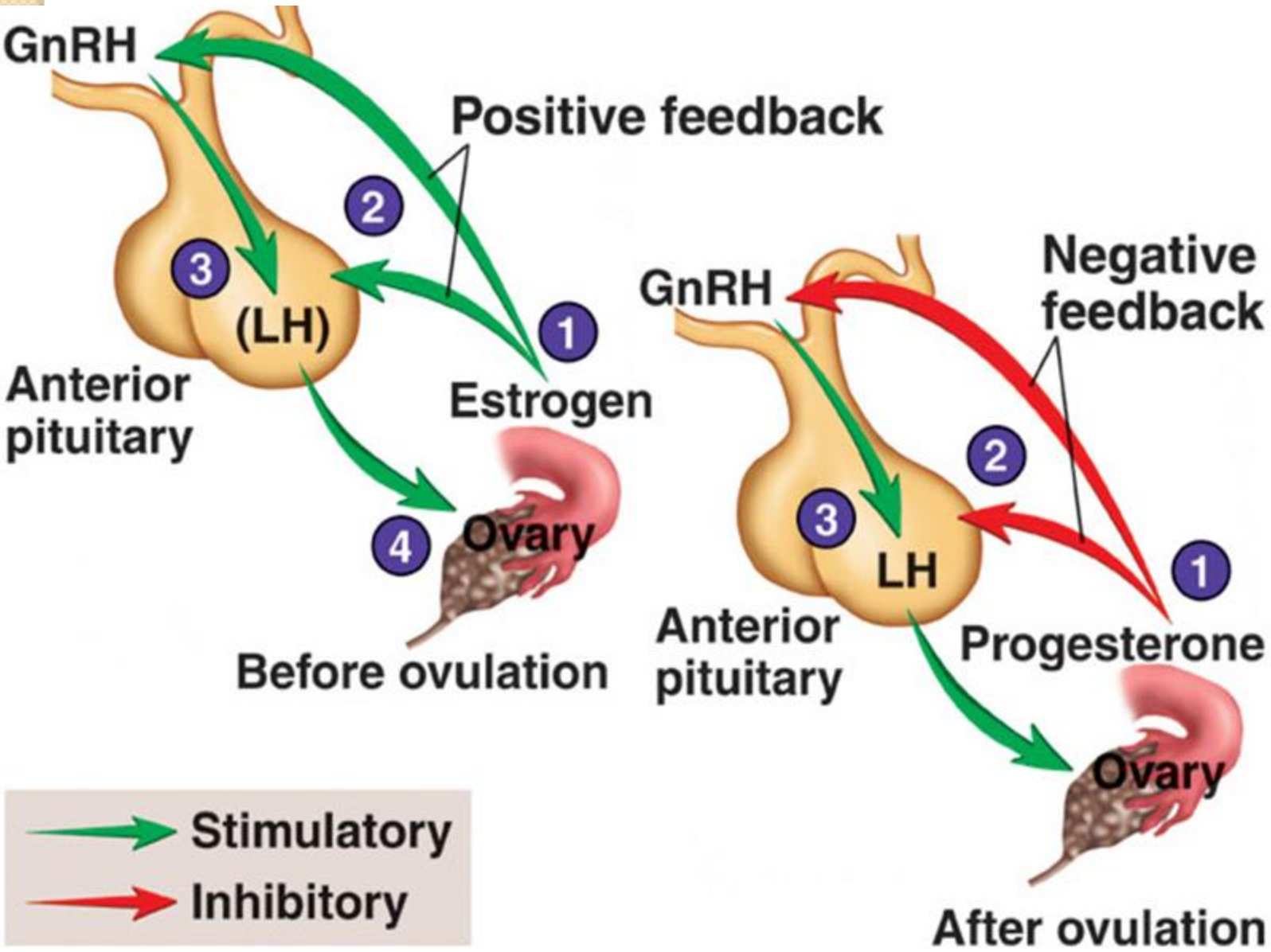
Anatomic and Physiologic Overview…

- mechanism for regulating hormone (Negative feedback):
  - ✓When the hormone concentration ↑ses, further production of that hormone is inhibited.
  - ✓ when the hormone concentration ↓ses, the rate of production of that hormone ↑ses.

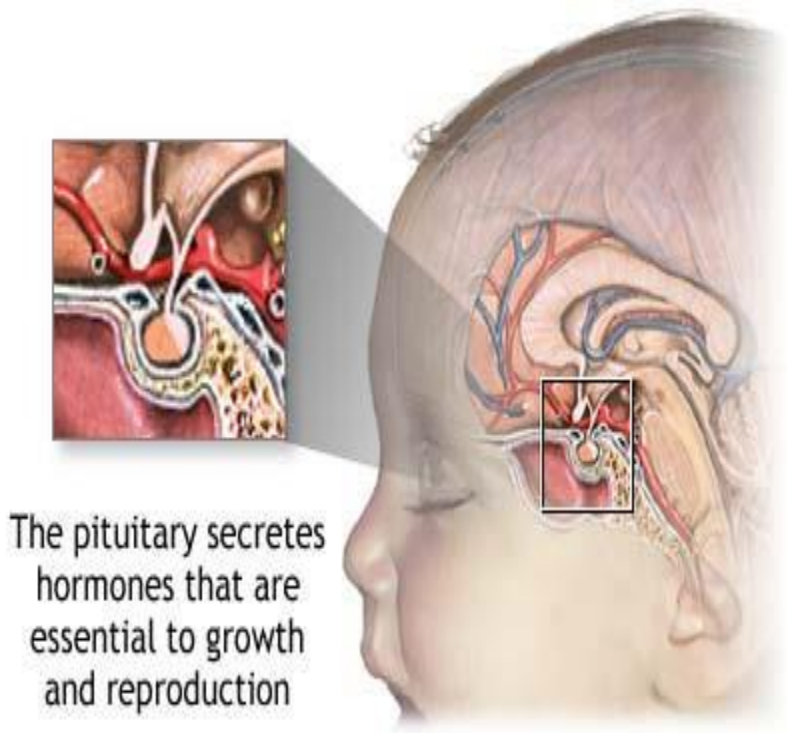
Anatomic and Physiologic Overview…



Anatomic and Physiologic Overview…



Pituitary Gland.



- The pituitary is located at the base of the brain, in a small depression of the sphenoid bone (sella turcica).
- Purpose: control the activity of many other endocrine glands.

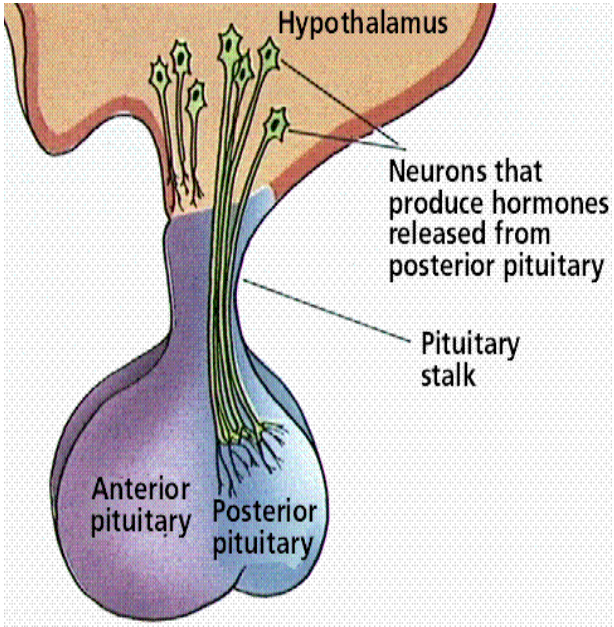
" Master gland"

- Has two lobes, the anterior & posterior lobes.

Anatomy

- Anterior lobe: glandular tissue, accounts for 75% of total weight. Hormones in this lobe are controlled by regulating hormones from the hypothalmus (stimulate or inhibit)

- Posterior lobe: nerve tissue & contains axons that originate in the hypothalmus. Therefore this lobe does not produce hormones but stores those produced by the neurosecretory cells in the hypothalmus.
- Release of hormone is triggered by receptors in the hypothalmus.



Terms

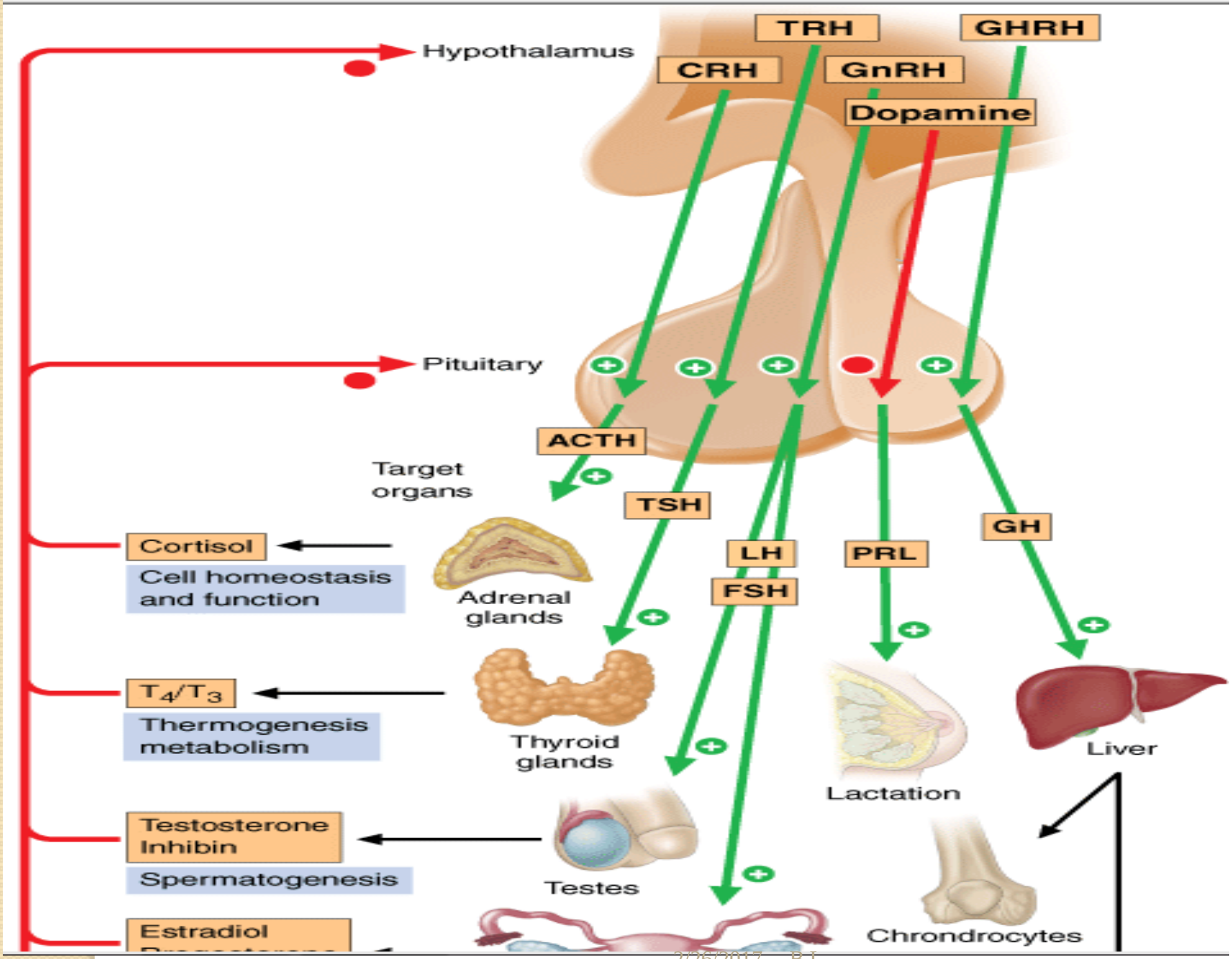
- **Trophic hormones**: hormones that control the secretion of hormones by other glands. Example: TSH stimulates the thyroid to secrete hormones.
- **Effector hormones**: produce an effect directly when secreted. Example ADH stimulates kidneys

Anterior Pituitary

- GH: growth hormone
- ACTH: adrenocorticotropic hormone
- TSH: thyroid-stimulating hormone
- PRL: prolactin
- FSH: follicle-stimulating hormone
- LH: luteinizing hormone
- MSH: melanocyte stimulating hormone

Posterior Pituitary

- ADH: anti-diuretic hormone (vasopressin)
- OT: oxytocin


Hypothalamus
TRH
GHRH
CRH
GnRH
Dopamine
Pituitary
ACTH
Target organs
TSH
GH
LH
PRL
FSH
Cortisol
Cell homeostasis and function
Adrenal glands
T_4/T_3
Thermogenesis metabolism
Thyroid glands
Lactation
Liver
Testosterone
Inhibin
Spermatogenesis
Testes
Chrondrocytes
Estradiol

Anterior Pituitary Secretes:

- **GH:** stimulates growth of bone and muscle , promotes protein synthesis and fat metabolism.
- **ACTH** (Adrenocorticotropin): stimulates adrenal gland cortex secretion of mineralcorticoids (aldosterone) & glucocorticoids (cortisol).
- **TSH:** stimulates thyroid to increase secretion of thyroxine, its control is from regulating hormones in the hypothalmus.

Anterior Pituitary Cont'd

- **Prolactin:** stimulates milk production from the breasts after childbirth to enable nursing. Oxytoxin from posterior lobe controls milk ejection.

- **FSH:** promotes sperm production in men and stimulates the ovaries to enable ovulation in women. LH and FSH work together to cause normal function of the ovaries and testes.

- **LH:** regulates testosterone in men and estrogen, progesterone in women.

Posterior Pituitary

- **Antidiuretic hormone or ADH** - also called vasopressin, vasoconstricts arterioles to increase arterial pressure; increases water reabsorption in distal tubules.

- **Oxytocin**: stimulates uterus to contract at childbirth; stimulates mammary ducts to contract (milk ejection in lactation).

Anterior Pituitary disorder

- What would happen if you had too much growth hormone secretion???

Anterior Pituitary Disorders

Hormone	Increased level	Decreased level
GH	Gigantism (child) Acromegaly (adult)	Dwarfism (child) Lethargy, premature aging
ACTH	Cushing's Disease	Addison's Disease
TSH	Goiter, increased BMR, HR, BP Graves disease	Decreased BMR, HR, CO, BP, non-toxic goiter Cretinism (children)
Prolactin	amenorrhea	Too little milk
FSH		Late puberty, infertility
LH	Menstrual cycle	Amenorrhea,

Posterior Pituitary Disorders

Hormone	Increased	Decreased
Oxytocin	Precipitates childbirth, excess milk	Prolonged childbirth, diminished milk
ADH (vassopressin)	Increased BP, decreased urinary output, edema. SIADH	Diabetes insipidus, dilute urine & increased urine output

Hyperpituitarism

Hyperfunction

- Results in excess production and secretion of one or more hormones such as :
  - GH,
  - PRL,
  - ACTH,
  - TSH,
  - GnTH.
- Most common cause is a benign adenoma.

Increased GH

- Increased GH: Gigantism & Acromegaly
- a statue of Robert Wadlow, the "Alton Giant," who measured 8 feet 11 inches at the time of his death.
- Young 12 yrs male standing with his mother

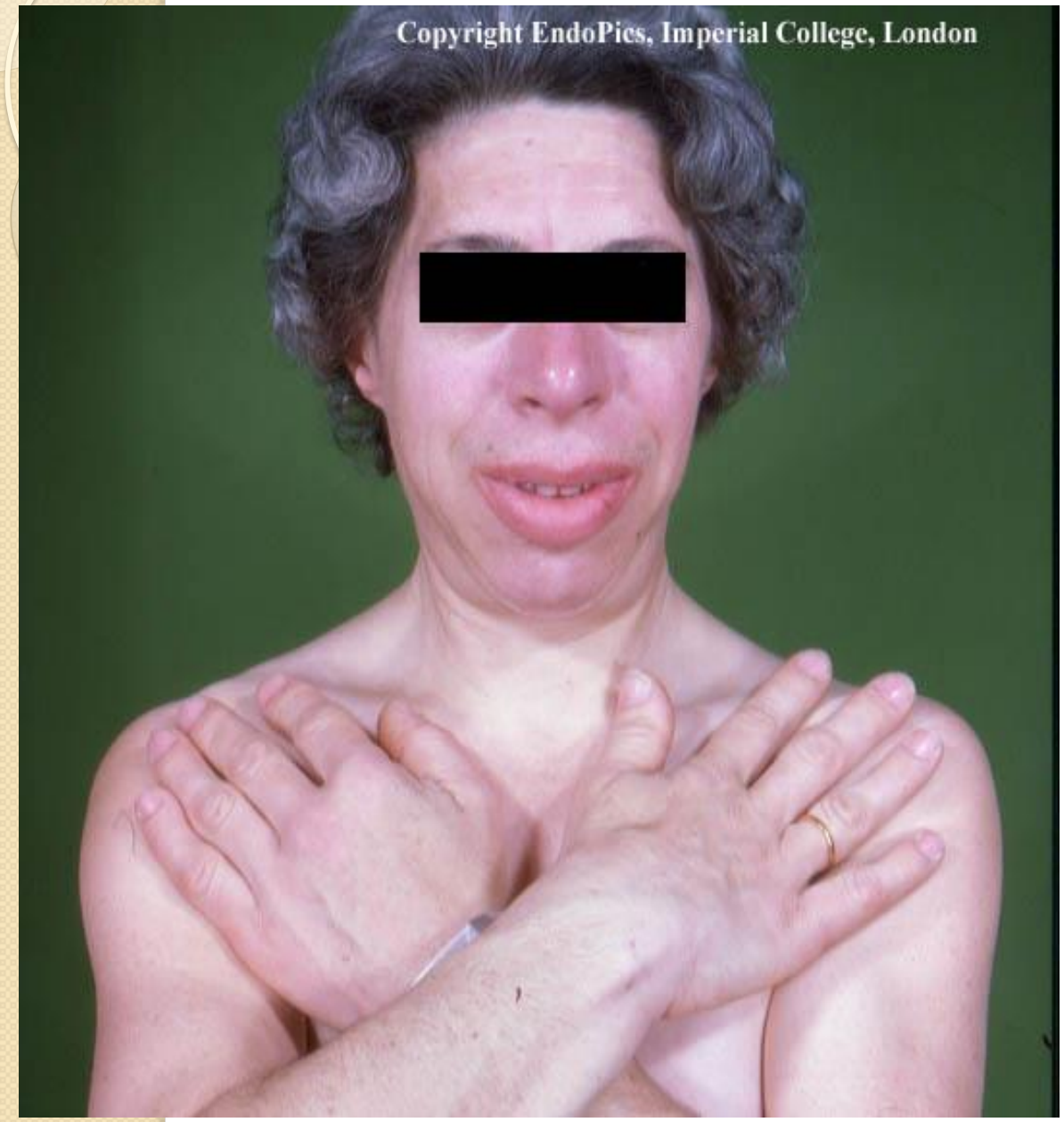
- **Gigantism** is the result of GH hypersecretion before the closure of the epiphyseal plates (childhood).
  - Abnormally tall but body proportions are normal
- **Acromegaly** is over secretion of GH in adulthood
  - Continued growth of boney, connective tissue leads to disproportionate enlargement of tissue..

Acromegaly

Rare condition – develops between ages 30-50

Symptoms:

- Coarsening of facial features
- Enlarged hands & feet
- Carpel turnnel syndrome
- Excessive sweating & oily skin
- Headaches
- Vision disturbance
- Sleep apnea
- General tiredness
- Oligomenorrhea or amenorrhea
- Impotence (adult males)
- Decreased libido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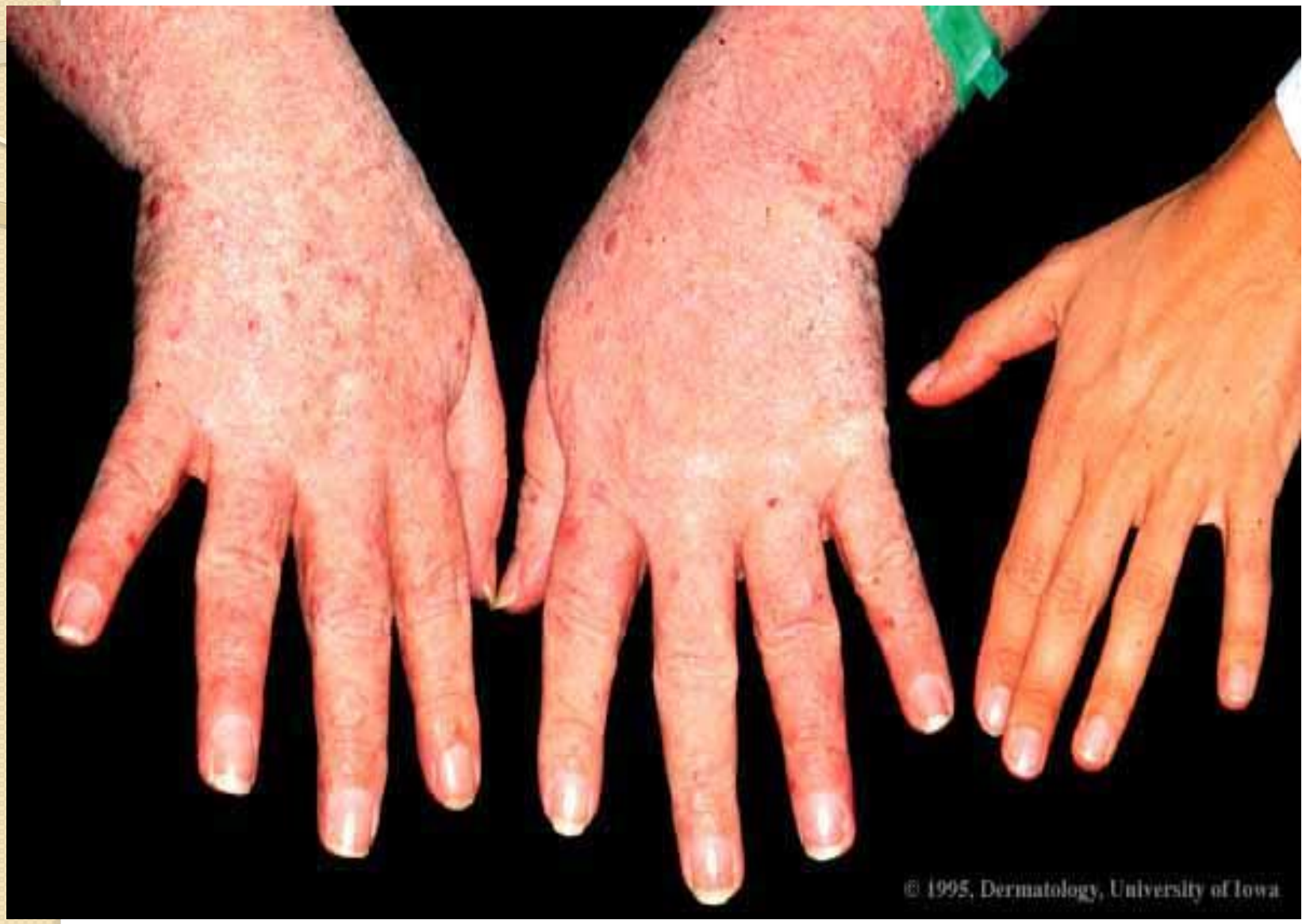
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Diagnosis

- History & physical exam
- Investigation includes:
  - GH analysis (glucose tolerance) Normally GH concentarion falls with oral glucose; in acromegaly it does not.
  - Prolactin levels as well as other pituitary function tests
  - MRI or CT & visual field tests to determine size and position of the adenom

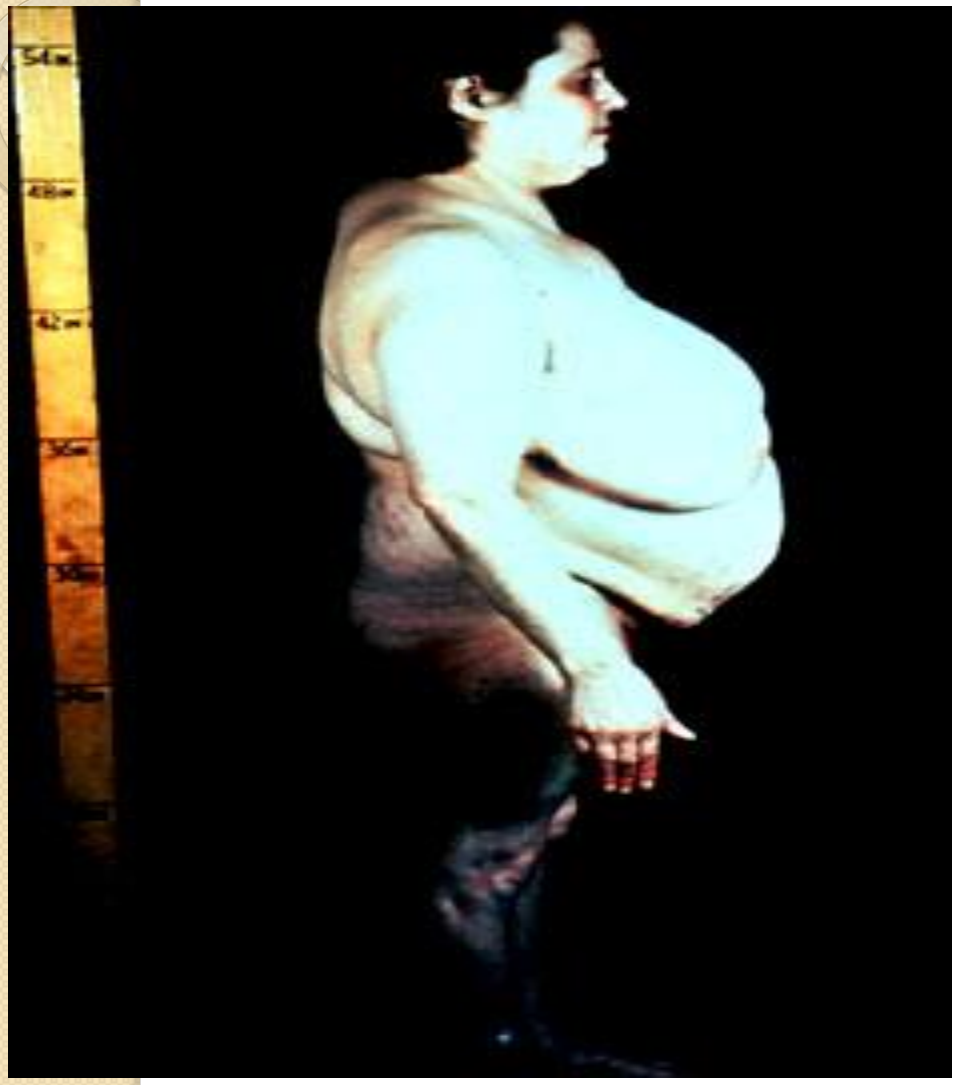
Medical Interventions for Pituitary Tumors

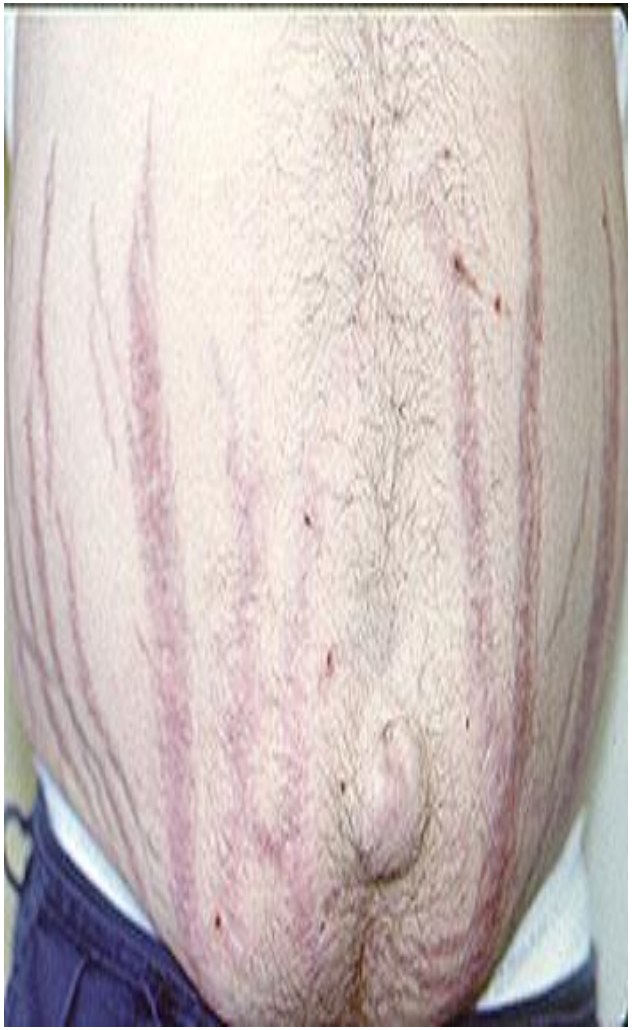
- **Medications**
- Dopamine agonists: Dopamine agonists work on specialist markers (dopamine receptors) on the surface of the tumor to inhibit GH release from the tumour (Parlodel).
- Somatostatin: growth hormone receptor antagonist decreases the action of GH on target tissues. (octreocide acetate)
- **Radiation therapy**
  - **external radiation will bring down GH levels 80% of time**

Increased ACTH: Cushing's Disease

- Cushing's is a disorder in which the adrenal glands are producing too much cortisol (hypercotisolism).
- If the source of the problem is the pituitary gland, then the correct name is **Cushing's Disease** whereas, if it originates anywhere else (adrenal tumors, long term steroid administration) then the correct name is **Cushing's Syndrome**.
- Cushing's Syndrome, a group of similar disorders caused by excessive levels of hormones called glucocorticoids.
- .

Advanced Cushing's



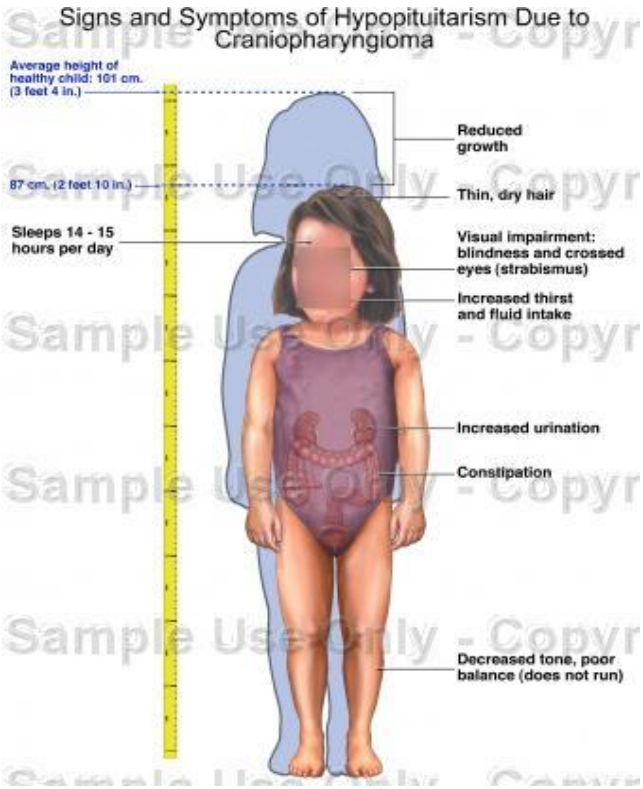


Hypopituitarism

Hypopituitarism- Anterior Pituitary

Decreased GH in child: Dwarfism

- Condition of being undersized
- There are many forms of dwarfism
- Dwarfism related to pituitary gland is the result of insufficient GH
- Pituitary dwarfism is successfully treated by administering human growth hormone

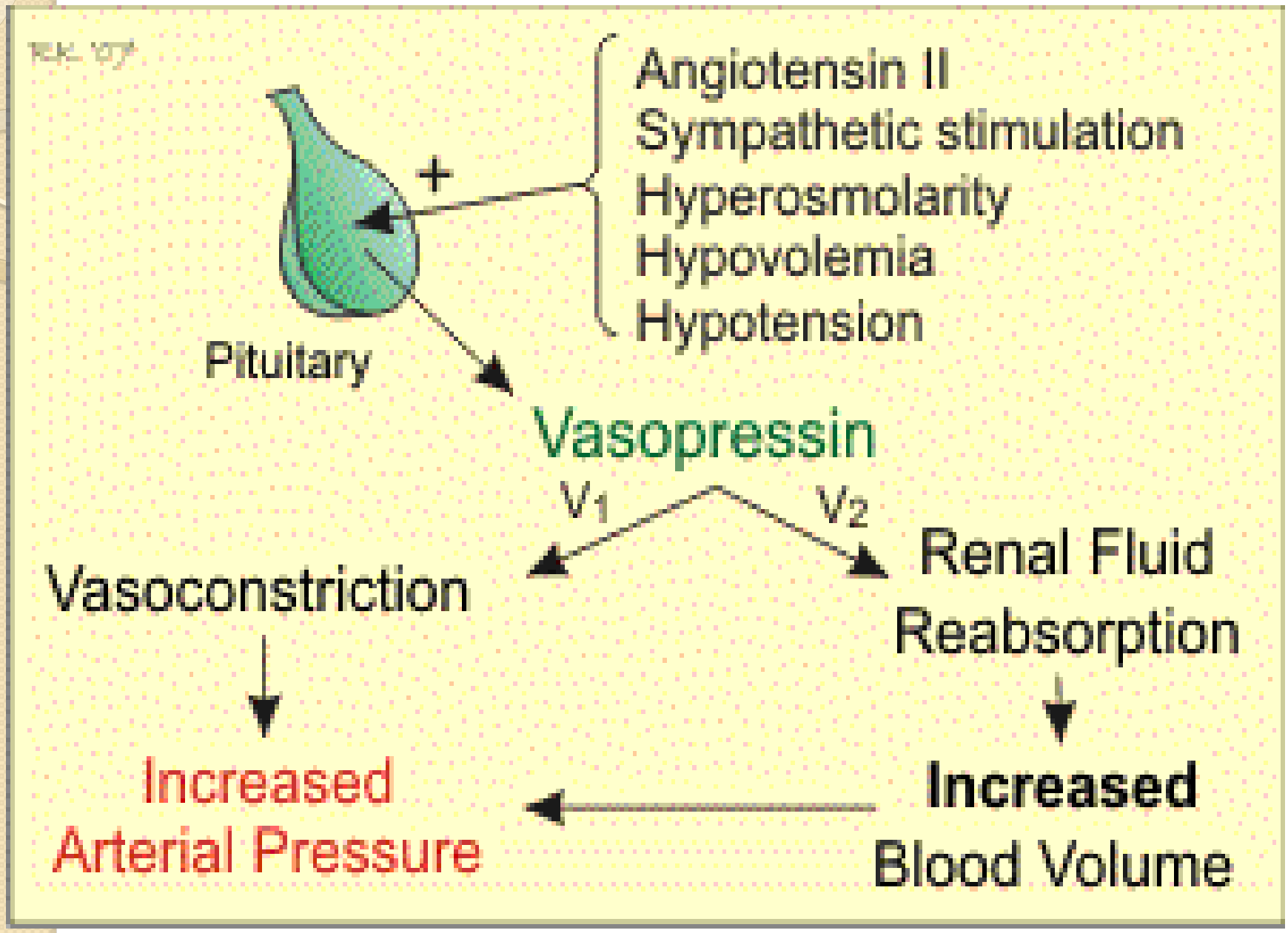


ADH
(Vasopressin/AVP)

- **secreted by cells in the hypothalmus and stored in posterior pituitary**

- **acts on distal & collecting tubules of the kidneys making more permeable to H20 -- or volume excreted?**

- **Under what conditions is ADH released?**
- **ADH has vasoconstrictive or vasodilation action???**


Angiotensin II
Sympathetic stimulation
Hyperosmolarity
Hypovolemia
Hypotension
+
Pituitary
Vasopressin
V1
V2
Vasoconstriction
Renal Fluid Reabsorption
Increased Arterial Pressure
Increased Blood Volume

Oxytocin

- Controls lactation & stimulates uterine contractions

Deficiency or excess of ADH

1. Diabetes insipidus(Deficincy)
2. SIADH(excess)

Posterior Lobe Disorders

Hyper – Posterior Pituitary

SIADH
(Syndrome of Inappropriate Anti-Diuretic Hormone)

- Too much ADH produced or secreted.
- SIADH commonly results from malignancies, CHF, & CVA - resulting in damage to the hypothalamus or pituitary which causes failure of the feedback loop that regulates ADH.
- Client retains water causing dilutional hyponaetremia & decreased osmolality.
- Decreased serum osmolality cause water to move into cells

Signs and Symptoms

- Lethargy & weakness
- Confusion or changes in neurological status
- Cerebral edema
- Muscle cramps
- Decreased urine output
- Weight gain without edema
- Hypertension

(Note: b/c of the low Na, edema will not accompany the FVE)

Assessment

- Serum sodium low
- Serum osmolality low
- Urine osmolality disproportionately elevated in relation to the serum osmolality
- Urine specific gravity elevated
- Plasma ADH elevated

Water intoxication, cerebral edema, severe hyponatremia cause altered neurological status, which untreated may cause death!

!!!!!

Treatment of SIADH

- Treat underlying cause
- Hypertonic or isotonic IV solution
- Monitor for signs of fluid and electrolyte imbalance
- Monitor for neurological effects
- Monitor in and out
- Weigh
- Restrict fluid intake
- Medic Alert
- Lithium inhibits action of ADH to promote water excretion.

Hypofunction – Posterior pituitary

Diabetes Insipitus (DI)

- Decreased secretion or action of AVP(action of vasopressin) usually manifests as DI, a syndrome characterized by the production of abnormally large volumes of dilute urine. The 24-h urine volume is >50 mL/kg body weight and the osmolarity is <300 mosmol/L.
- DI is usually insidious but can occur with damage to the hypothalamus or the pituitary. (neurogenic DI)

- May be a result of defect in renal tubules, do not respond to ADH (nephrogenic DI)
- Decreased production or release of ADH results in massive water loss
- Leads to hypovolemic & dehydration.

Clinical Manifestations

- Polyuria of more than 3 litres per 24 hours in adults (may be up to 20!)
- Urine specific gravity low
- Polydipsia (excessive drinking)
- Weight loss
- Dry skin & mucous membranes
- Possible hypovolemia, hypotension, electrolyte imbalance

Diagnostic Tests

- Serum sodium
- Urine specific gravity
- Serum osmolality
- Urine osmolality
- Serum ADH levels
- Vasopressin test and water deprivation test: increased hyperosmolality is diagnostic for DI.

Management

- Medical management includes
  - Rehydration IV fluids (hypotonic)
  - Symptom management
  - ADH replacement (vasopressin)

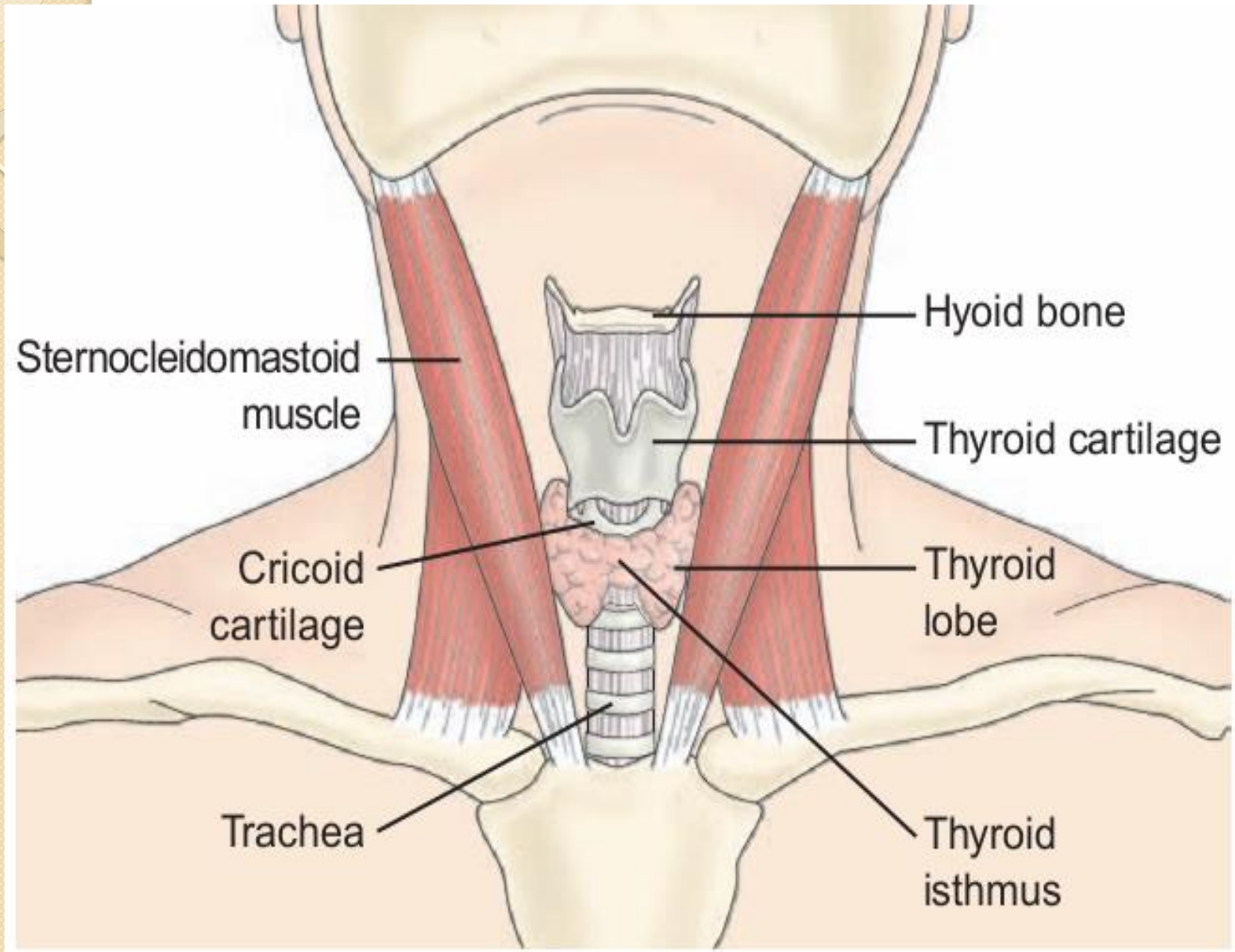
Nursing Responsibility

1. Monitor for signs of fluid and electrolyte imbalance
2. Monitor in and out
3. Daily weight
4. Monitor for excessive thirst or output
5. Assess serum and urine values (decreased SG, decreased urine osmolality, high serum osmolality are early indicators

Panhypopituitarism

- When both the anterior and posterior fail to secrete hormones, the condition is called ***panhypopituitarism.***
- Causes include tumors, infection, injury, iatrogenic (radiation, surgery), infarction
- Manifestations don't occur until 75% of pituitary has been obliterated.
- Treatment involves removal of cause and hormone replacement (adrenaocortical insufficiency, thyroid hormone, sex hormones)

Disorders of Thyroid and Parathyroid Glands


Hyoid bone
Sternocleidomastoid muscle
Thyroid cartilage
Cricoid cartilage
Thyroid lobe
Trachea
Thyroid isthmus

Introduction….

- The blood flow is very high-- approximately five times the blood flow to the liver.
- This reflects the high metabolic activity of the thyroid gland.
- Produces three hormones:
  - ✓thyroxine (T4),
  - ✓triiodothyronine (T3), and
  - ✓calcitonin.

Introduction….

- Various hormones and chemicals are responsible for normal thyroid function.
- Key among them are:
  - ✓thyroid hormone
  - ✓calcitonin and
  - ✓iodine.

Introduction….

Thyroid Hormone

- T4 and T3 (thyroid hormone), are two separate hormones produced by the thyroid gland.
- Both are amino acids that contain iodine molecules bound to the amino acid structure
- T4 contains four iodine atoms in each molecule &
- T3 contains three.

Introduction….

Synthesis of Thyroid Hormone

- Iodine is essential to the thyroid gland for synthesis of its hormones.
- Iodide is ingested in the diet and absorbed into the blood from the GI tract.
- The thyroid gland takes iodide from the blood and concentrate it within the cells.
- In the cells iodide ions are converted to iodine molecules, which react with tyrosine (an amino acid) to form the thyroid

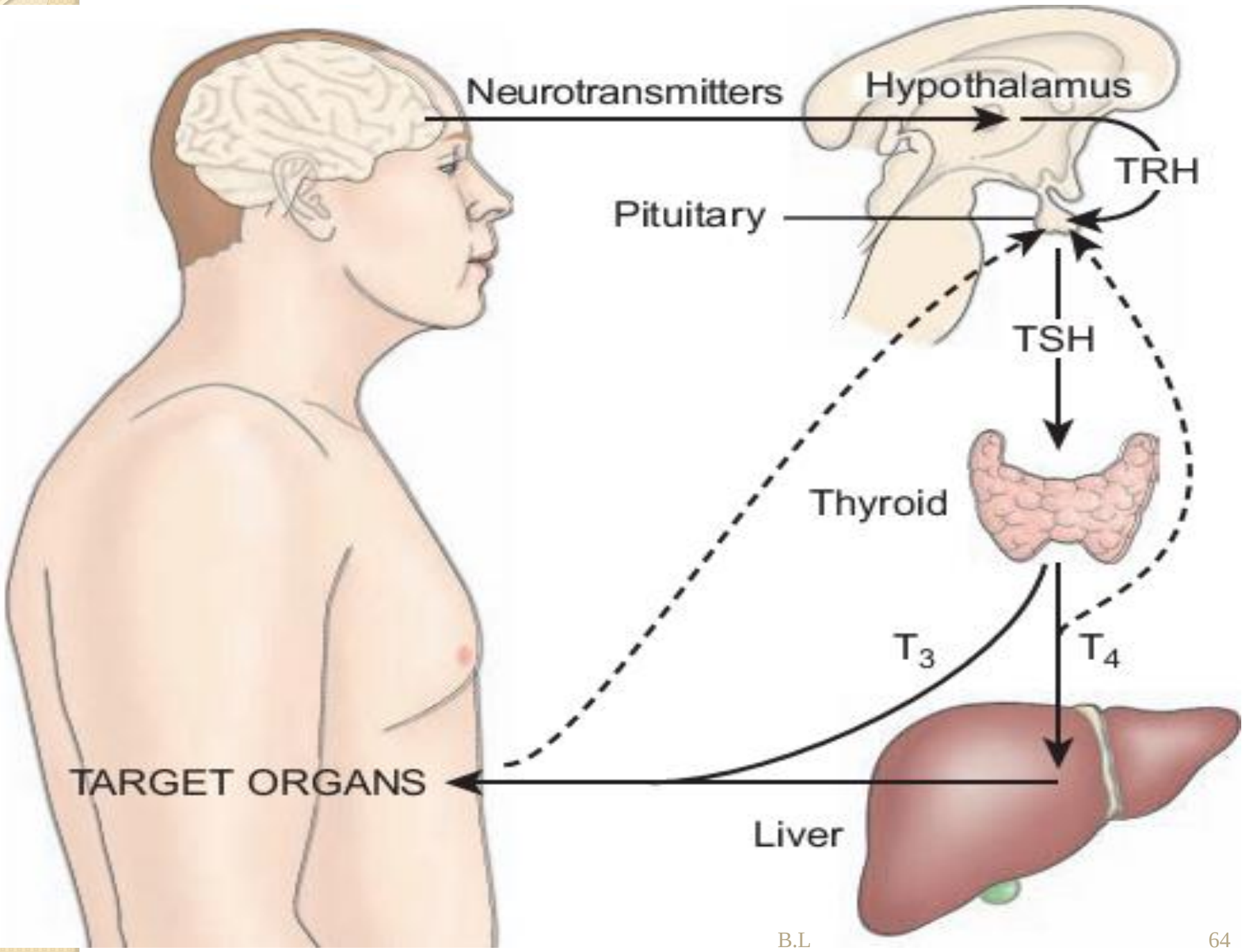
Introduction….

Regulation of Thyroid Hormone

- The secretion of T3 and T4 by the thyroid gland is controlled by TSH from the anterior pituitary gland.
- TSH controls the rate of thyroid hormone release through a negative feedback mechanism.
- In turn, the level of thyroid hormone in the blood determines the release of TSH.
- If the thyroid hormone concentration in the blood decreases, the release of TSH increases, which causes increased output of T3 and T4.

Introduction….

- Thyrotropin-releasing hormone (TRH), secreted by the hypothalamus, exerts a modulating influence on the release of TSH from the pituitary.
- Environmental factors, such as a decrease in temperature, may lead to increased secretion of TRH, resulting in elevated secretion of thyroid hormones.


Neurotransmitters
Hypothalamus
TRH
Pituitary
TSH
Thyroid
T_3
T_4
TARGET ORGANS
Liver

Function of Thyroid Hormone

- The primary function of thyroid hormone is to control cellular metabolic activity.
- T4 maintains body metabolism in a steady state.
- T3 is about five times as potent as T4 and has a more rapid metabolic action.
- Important in brain development.
- Necessary for normal growth.
- Influence every major organ system.

Specific Disorders of the Thyroid Gland

HYPOTHYROIDISM

- Results from suboptimal levels of thyroid hormone.
- Thyroid deficiency can affect all body functions and can range from mild, subclinical forms to myxedema, an advanced form.
- The most common cause of hypothyroidism in adults is autoimmune thyroiditis (Hashimoto's disease), in which the immune system attacks the thyroid gland.
- Hypothyroidism also commonly occurs in patients with previous hyperthyroidism that has been treated with radioiodine or anti-thyroid medications or thyroidectomy.

HYPOTHYROIDISM….

- Occurs most frequently in older women.
- In addition, there is an increased incidence of thyroid cancer in men who have undergone radiation therapy for head and neck cancer.
- Therefore, testing of thyroid function is recommended for all patients who receive such treatment.
- More than 95% of patients with hypothyroidism have primary or thyroidal hypothyroidism, which refers to dysfunction of the thyroid gland itself.

HYPOTHYROIDISM….

- ***Central Hypothyroidism***:- failure of the pituitary gland, the hypothalamus, or both.
- ***Pituitary or secondary hypothyroidism***:-If the cause is entirely a pituitary disorder.
- ***Hypothalamic or tertiary hypothyroidism*:-** a disorder of the hypothalamus resulting in inadequate secretion of TSH due to decreased stimulation of TRH.
- ***Cretinism***:-If thyroid deficiency is present at birth.

Causes of hypothyroidism

- ✓ Autoimmune disease (Hashimoto's thyroiditis, post- Graves' disease)
- ✓ Atrophy of thyroid gland with aging
- ✓ Therapy for hyperthyroidism
- ✓ Radioactive iodine (1311)
- ✓ Thyroidectomy

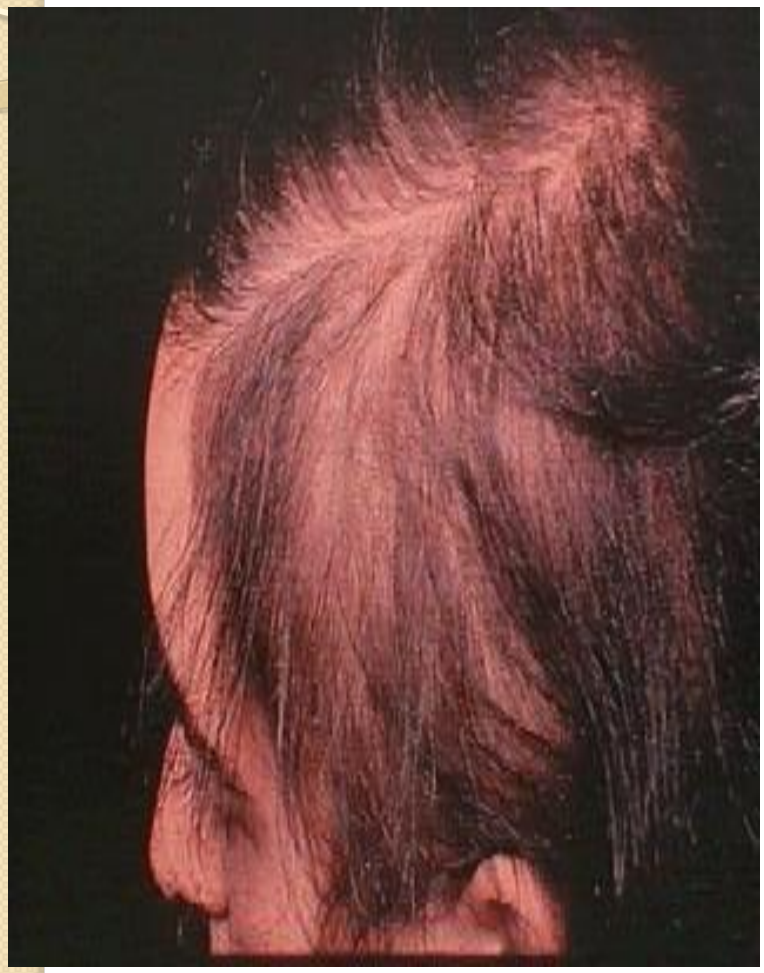
Causes….

- **Medications**
  - ✓ Lithium
  - ✓ Iodine compounds
  - ✓ Anti-thyroid medications
  - ✓ Radiation to head and neck for treatment of head and neck cancers, lymphoma.
  - ✓ Iodine deficiency and iodine excess.

Hypothyroidism Symptoms

- ✓ Tiredness and weakness
- ✓ Dry skin
- ✓ Feeling cold
- ✓ Hair loss
- ✓ Difficulty in concentrating and poor memory
- ✓ Constipation
- ✓ Weight gain with poor appetite
- ✓ Hoarse voice
- ✓ Menorrhagia, later oligo and amenorrhoea
- ✓ Paresthesias
- ✓ Impaired hearing

Hypothyroidism Signs



- ✓ Dry skin, cool extremities
- ✓ Puffy face, hands and feet
- ✓ Delayed tendon reflex relaxation
- ✓ Carpal turnnel syndrome
- ✓ Bradycardia
- ✓ Diffuse alopecia
- ✓ Serous cavity effusions

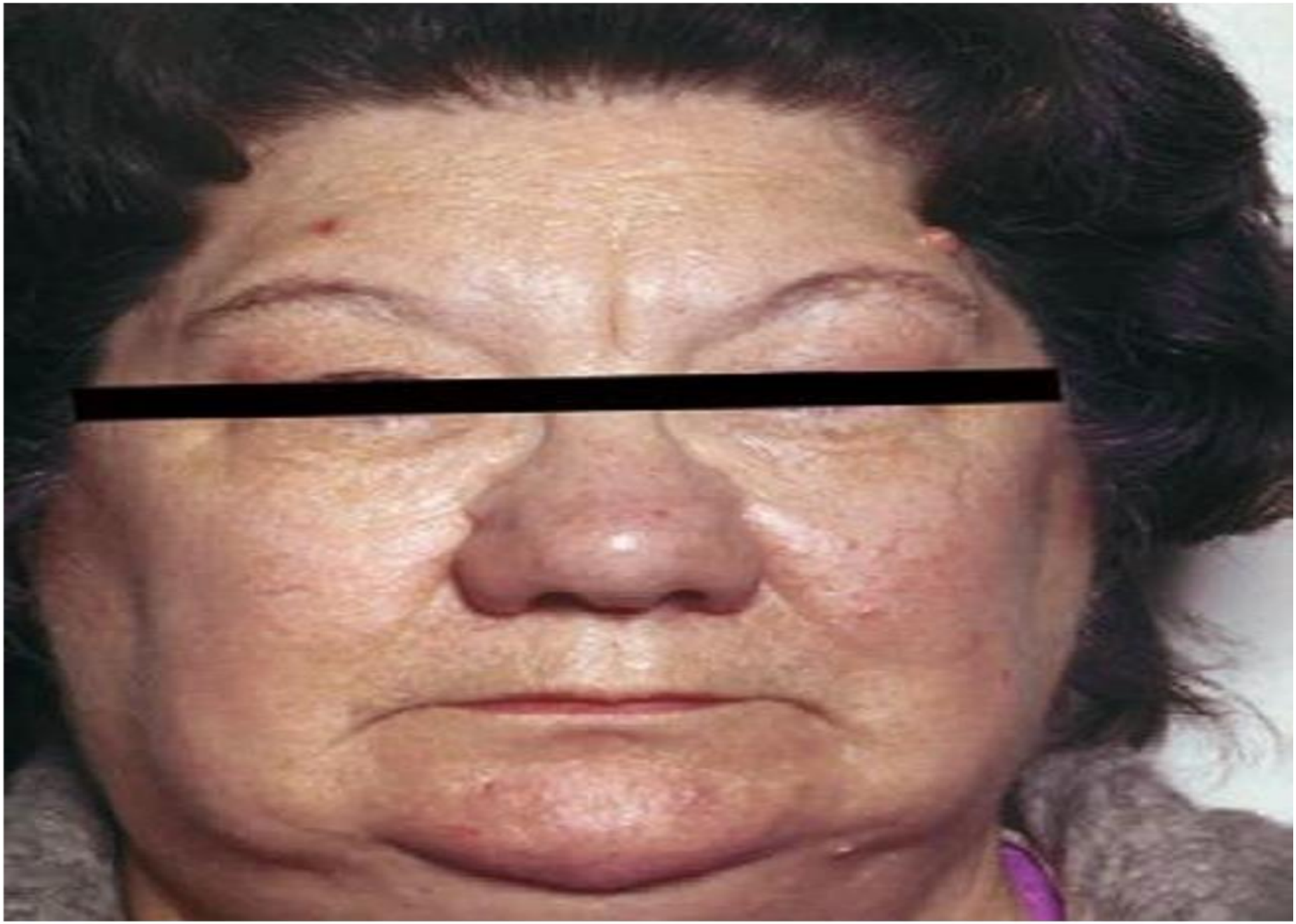


FIGURE 405-6 **Facial appearance in hypothyroidism.** Note puffy eyes and thickened skin.

THYROTOXICOSIS

- The state of thyroid hormone excess and is not synonymous with hyperthyroidism, which is the result of excessive thyroid function.

Causes of thyrotoxicosis

Primary Hyperthyroidism
Graves' disease
Toxic multinodular goiter
Toxic adenoma
Functioning thyroid carcinoma metastases
Activating mutation of the TSH receptor
Activating mutation of $G_{s\alpha}$ (McCune-Albright syndrome)
Struma ovarii
Drugs: iodine excess (Jod-Basedow phenomenon)
Thyrotoxicosis Without Hyperthyroidism
Subacute thyroiditis
Silent thyroiditis
Other causes of thyroid destruction: amiodarone, radiation, infarction of adenoma
Ingestion of excess thyroid hormone (thyrotoxicosis factitia) or thyroid tissue

Cont.....

Secondary Hyperthyroidism

TSH-secreting pituitary adenoma

Thyroid hormone resistance syndrome: occasional patients may have features of thyrotoxicosis

Chorionic gonadotropin-secreting tumors[a]

Gestational thyrotoxicosis[a]

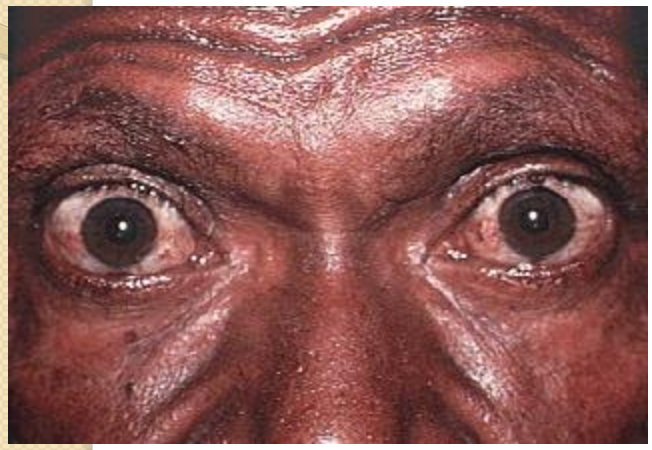
S/s of Thyrotoxicosis

Symptoms	Signs[a]
Hyperactivity, irritability, dysphoria	Tachycardia; atrial fibrillation in the elderly
Heat intolerance and sweating	Tremor
Palpitations	Goiter
Fatigue and weakness	Warm, moist skin
Weight loss with increased appetite	Muscle weakness, proximal myopathy
Diarrhea	Lid retraction or lag
Polyuria	Gynecomastia
Oligomenorrhea, loss of libido	

Graves' Disease

- Autoimmune disorder
- Ab^{s} directed against TSH receptor with intrinsic activity. Thyroid and fibroblasts
- Responsible for 60-80% of Thyrotoxicosis
- More common in women

Graves' ophthalmopathy



N(0)- no signs or symptoms

O(1)– only signs (lid retraction or lag) no symptoms

S(2) – soft tissue involvement (periorbital oedema)

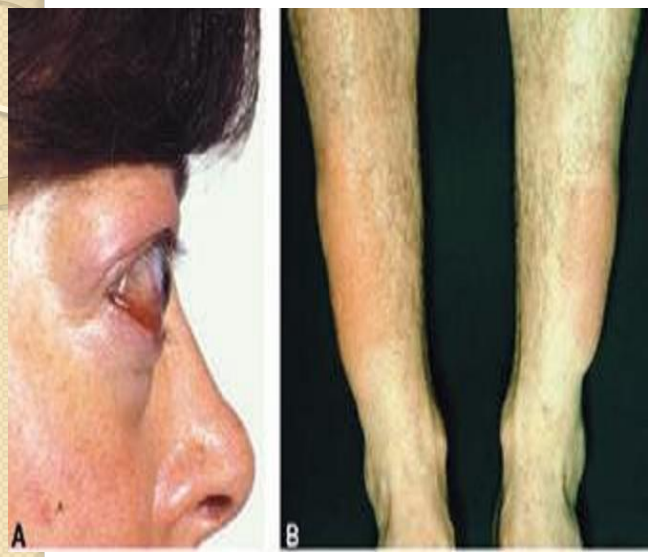
P(3) – proptosis (>22 mm)

E(4) – extra ocular muscle involvement (diplopia)

C(5) – corneal involvement (keratitis)

S(6)– sight loss (compression of the optic nerve)

Graves' Disease Other Manifestations



- Pretibial myxoedema
- Thyroid acropachy
- Onycholysis(nail damage)
- Thyroid enlargement with a bruit frequently audible over the thyroid

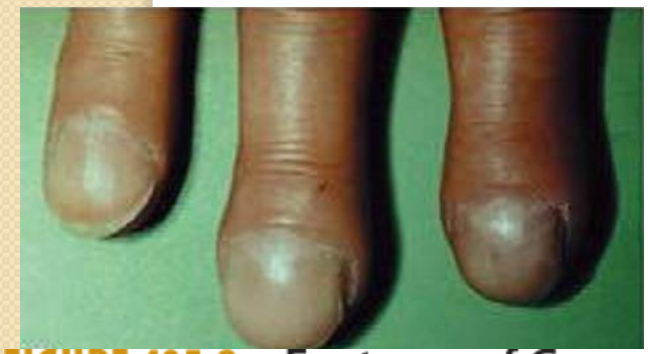


FIGURE 405-8 **Features of Graves' disease.** ***A.*** Ophthalmopathy in Graves' disease; lid retraction, periorbital edema, conjunctival injection, and proptosis are marked. ***B.*** Thyroid dermopathy over the lateral aspects of the shins. ***C.*** Thyroid acropachy.

Diagnostic Findings

- TSH ↓, free T4 ↑
- Thyroid auto antibodies
- Nuclear thyroid scintigraphy (I_{123}, Te_{99})

Treatment of Graves' Disease

- Reduce thyroid hormone production or reduce the amount of thyroid tissue
  - ✓ Antithyroid drugs: propylthiouracil, carbimazole
  - ✓ Radioiodine
  - ✓ Subtotal thyroidectomy
- Symptomatic treatment
  - ✓ Propranolol

HYPERTHYROIDISM

- The second most prevalent endocrine disorder, after **diabetes mellitus.**
- Graves' disease, the most common type of hyperthyroidism, results from an **excessive output of thyroid hormones.**
- It affects women eight times more frequently than men, with onset usually between the second and fourth decades.
- The disorder may appear after an emotional shock, stress, or an infection, but the exact significance of these relationships is not understood.
- Other common causes of hyperthyroidism include thyroiditis and excessive ingestion of thyroid hormone.

Clinical Manifestations

- ✓ Hyperactivity
- ✓ Irritability
- ✓ Dysphoria
- ✓ Heat intolerance & sweating
- ✓ Palpitations
- ✓ Fatigue & weakness
- ✓ Weight loss with increased appetite
- ✓ Diarrhea
- ✓ Polyuria
- ✓ Sexual dysfunction
- ✓ Tachycardia
- ✓ Atrial fibrillation
- ✓ Tremor
- ✓ Toxic Goiter
- ✓ Warm, moist skin
- ✓ Muscle weakness, myopathy
- ✓ Gynecomastia

Diagnostic Findings

- A decrease in serum TSH,
- Increased free T4, and
- Increase in radioactive iodine uptake.

Medical Management

- No treatment for thyrotoxicosis is without side effects.
- Relapse or recurrent hyperthyroidism and permanent hypothyroidism.

Pharmacologic Therapy

- Two forms of pharmacotherapy are available:
  - ✓ use of irradiation by administration of the radioisotope iodine 131 (1311)
  - ✓ anti-thyroid medications

❑ Radioactive Iodine Therapy

- The goal of radioactive iodine therapy (1311) is to destroy the overactive thyroid cells.
- contraindicated during pregnancy and while breast-feeding.
- A major advantage of treatment with radioactive iodine is that it avoids many of the side effects associated with anti-thyroid medications.

Anti-thyroid Medications

- Most commonly, propylthiouracil (PTU) or methimazole is used until the patient is euthyroid.
- These medications block extrathyroidal conversion of T4 to T3.
- Because antithyroid medications do not interfere with release or activity of previously formed thyroid hormones, it may take several weeks until relief of symptoms occurs.
- PTU is the treatment of choice during pregnancy

Adjunctive Therapy

- Iodine or iodide compounds, once the only therapy available for patients with hyperthyroidism, are no longer used as the sole method of treatment.
- Compounds such as potassium iodide (KI), Lugol's solution, and saturated solution of potassium iodide (SSKI) may be used in combination with antithyroid agents or beta-adrenergic blockers to prepare the patient with hyperthyroidism for surgery.
- Beta-adrenergic blocking agents are important in controlling the sympathetic nervous system effects of hyperthyroidism.
- For example, propranolol is used to control nervousness, tachycardia, tremor, anxiety, and heat intolerance.

Surgical Management

- In pregnant women who are allergic to anti-thyroid medications, in patients with large goiters, or in patients who are unable to take anti-thyroid agents.
- Surgery is performed soon after the thyroid function has re-turned to normal (4 to 6 weeks).
- Its use today is reserved for patients with obstructive symptoms, for pregnant women in the second trimester, and for patients with a need for rapid normalization of thyroid function.
- Before surgery, PTU is administered until signs of hyperthyroidism have disappeared.
- A beta-adrenergic blocking agent (e.g, propranolol) may be used to reduce the heart rate and other signs and symptoms of hyperthyroidism

Nursing Intervention

- Improving Nutritional Status
- Enhancing Coping Measures
- Improving Self-Esteem
- Maintaining Normal Body Temperature
- Monitoring and Managing Potential Complications

Endemic (Iodine-Deficient) Goiter

- The most common type of goiter, once encountered chiefly in geographic regions where the natural supply of iodine is deficient is the so-called simple or colloid goiter.
- simple goiter may also be caused by an intake of large quantities of goitrogenic substances in patients with unusually susceptible glands.
- These substances include excessive amounts of iodine or lithium, which is used in treating bipolar disorders.

Endemic Goiter….

- Simple goiter represents a compensatory hypertrophy of the thyroid gland, caused by stimulation by the pituitary gland.
- The pituitary gland produces TSH.
- Its production increases if there is sub-normal thyroid activity, as when insufficient iodine is available for production of the thyroid hormone.
- Such goiters usually cause no symptoms, except for the swelling in the neck, which may result in tracheal compression when excessive.

Endemic Goiter….

- Many goiters of this type recede after the iodine imbalance is corrected.
- Providing children in iodine-poor regions with iodine compounds can prevent simple or endemic goiter.

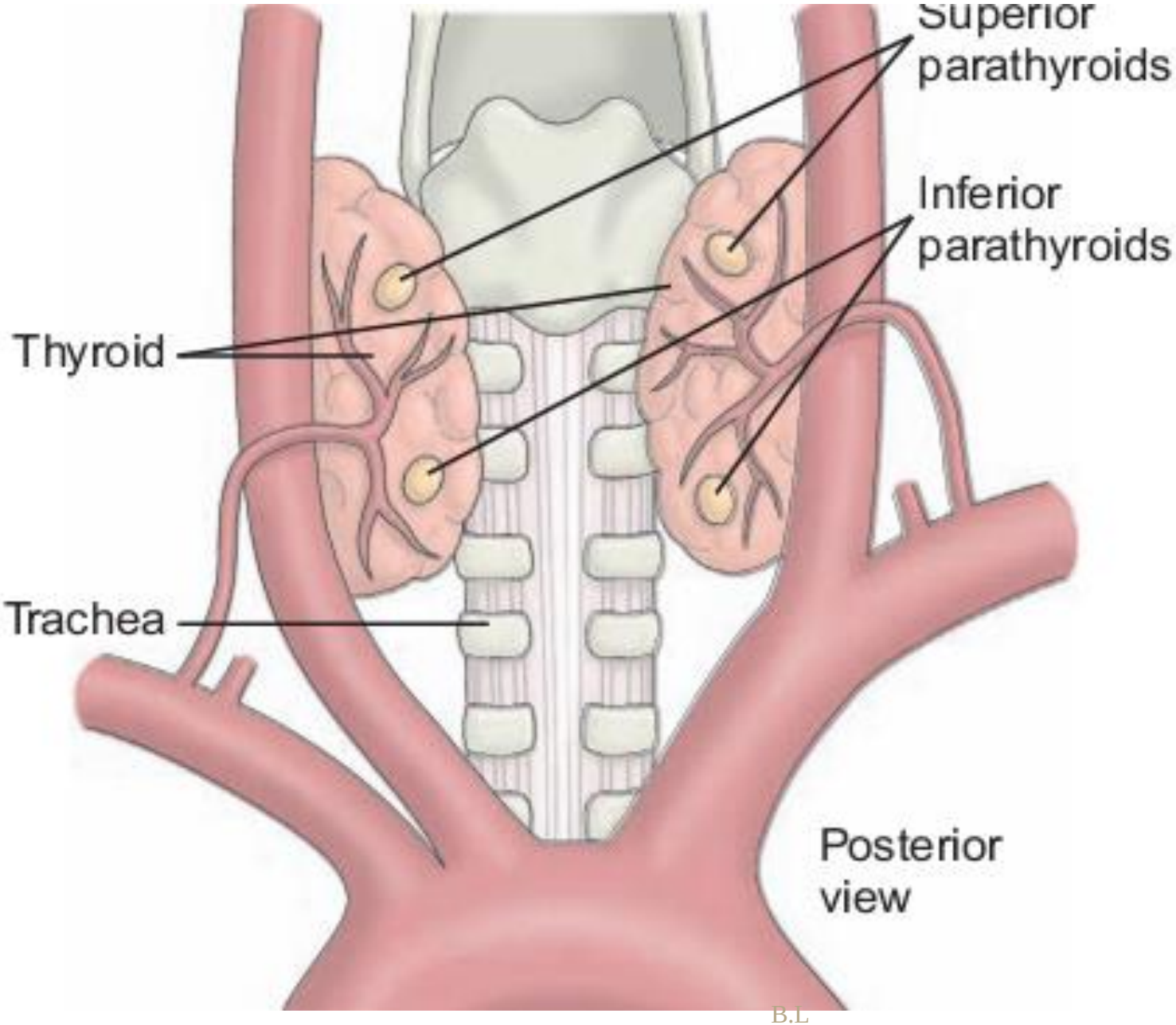
Nodular Goiter

- Some thyroid glands are nodular because of areas of hyperplasia (overgrowth).
- No symptoms may arise as a result of this condition, these nodules slowly increase in size, with some descending into the thorax, where they cause local pressure symptoms.
- Some nodules become malignant, and some are associated with a hyperthyroid state.
- Therefore, the patient with many thyroid nodules may eventually require surgery.

THE PARATHYROID GLANDS

Anatomic and Physiologic Overview

- The parathyroid glands (normally four) are situated in the neck and embedded in the posterior aspect of the thyroid gland.
- Parathormone (parathyroid hormone) regulates calcium and phosphorus metabolism.
- Increased secretion results in increased calcium absorption from the kidney, intestine, and bones, which raises the blood calcium level.


Superior parathyroids
Inferior parathyroids
Thyroid
Trachea
Posterior view

Specific Disorders of the Parathyroid Glands

HYPERPARATHYROIDISM

- Overproduction of parathormone
- Characterized by bone decalcification and the development of renal calculi (kidney stones) containing calcium.
- Primary hyperparathyroidism occurs 2-4 times more often in women than in men and is most common in people between 60 and 70 years of age.
- Rare in children younger than 15 years of age.
- Half of the people do not have symptoms.

HYPERPARATHYROIDISM….

- Secondary hyperparathyroidism, occurs in patients who have chronic renal failure and so-called renal rickets as a result of
  - ✓Phosphorus retention,
  - ✓Increased stimulation of the parathyroid glands, and
  - ✓Increased parathormone secretion.

Clinical Manifestations

- The patient may have no symptoms or may experience s/s resulting from involvement of several body systems.
  - ✓ Apathy, fatigue, muscle weakness, nausea, vomiting, constipation, hypertension, and cardiac dysrhythmias may occur.
  - ✓ Psychological effects may vary from irritability and neurosis to psychoses caused by the direct action of calcium on the brain and nervous system.

Manifestations….

- Renal calculi (kidney stones), obstruction, pyelonephritis, and renal failure.
- Skeletal pain and tenderness
- Pain on weight bearing; pathologic fractures; deformities; and shortening of body stature.

Assessment and Diagnostic Findings

- Primary hyperparathyroidism is diagnosed by persistent elevation of serum calcium levels and an elevated concentration of parathormone.
- The double-antibody parathyroid hormone test is used to distinguish between primary hyperparathyroidism and malignancy as a cause of hypercalcemia.
- US, MRI, thallium scan, and fine-needle biopsy have been used to evaluate the function of the parathyroid and to localize parathyroid cysts, adenomas, or hyperplasia.

Management

Surgical Management

- The recommended treatment for primary hyperparathyroidism is the surgical removal of abnormal parathyroid tissue
- In some cases only the removal of a single diseased gland is necessary, reducing morbidity rates associated with surgery.
- For asymptomatic patients who have only mildly elevated serum calcium concentrations and normal renal function, surgery may be delayed and the patient monitored closely for worsening of hypercalcemia, bone deterioration, renal impairment, or the development of kidney stones.

Hydration Therapy

- Because kidney involvement is possible, patients with hyperparathyroidism are at risk for renal calculi.
- Therefore, a daily fluid intake of 2000 mL or more is encouraged to help prevent calculus formation.
- The patient is instructed to report other manifestations of renal calculi, such as abdominal pain and hematuria.
- Thiazide diuretics are avoided, because they decrease the renal excretion of calcium and further elevate serum calcium levels.

Diet and Medications

- Nutritional needs are met, but the patient is advised to avoid a diet with restricted or excess calcium.
- If the patient has a coexisting peptic ulcer, prescribed antacids and protein feedings are necessary.
- Because anorexia is common, efforts are made to improve the appetite.
- Prune juice, stool softeners, and physical activity, along with increased fluid intake, help offset constipation, which is common postoperatively.

Nursing Management

- An awareness of the course of the disorder and an understanding approach by the nurse help the patient and family deal with their reactions and feelings.
- Although not all parathyroid tissue is removed during surgery in an effort to control the calcium–phosphorus balance, the nurse closely monitors the patient to detect symptoms of tetany.
- The nurse reminds the patient and family about the importance of follow-up to ensure return of serum calcium levels to normal.

Complications: Hypercalcemic Crisis

- Occur with extreme elevation of serum calcium levels.
- Serum calcium levels greater than 15 mg/dL result in neurologic, cardio-vascular, and renal symptoms that can be life-threatening.
- Treatment includes rehydration with large volumes of IV fluids, diuretic agents to promote renal excretion of excess calcium, and phosphate therapy to correct hypophosphatemia and decrease serum calcium levels by promoting calcium deposition in bone and reducing the gastrointestinal absorption of calcium.

Complications….

- Cytotoxic agents (eg, mithramycin), calcitonin, and dialysis may be used in emergency situations to decrease serum calcium levels quickly.
- A combination of calcitonin and corticosteroids has been administered in emergencies to reduce the serum calcium level by increasing calcium deposition in bone.
- Other agents that may be administered to decrease serum calcium levels include bisphosphonates (eg, etidronate, pamidronate)

HYPOPARATHYROIDISM

- The most common cause is inadequate secretion of parathormone.
- Atrophy of the parathyroid glands of unknown cause is a less common cause.
- Deficiency of parathormone results in increased blood phosphate and decreased blood calcium levels.
- In the absence of parathormone, there is decreased intestinal absorption of dietary calcium and decreased resorption of calcium from bone and through the renal tubules.
- Decreased renal excretion of phosphate causes hypophosphaturia, and low serum calcium levels result in hypocalciuria.

Clinical Manifestations

- Numbness, tingling, and cramps in the extremities, and stiffness in the hands and feet.
- Bronchospasm, laryngeal spasm, carpopedal spasm dysphagia, photophobia, cardiac dysrhythmias, and seizures.
- Anxiety, irritability, depression, and even delirium.
- ECG changes and hypotension also may occur.

Assessment and Diagnostic Findings

- A positive Trousseau's sign or a positive Chvostek's sign suggests latent tetany.
- The diagnosis of hypoparathyroidism often is difficult because of the vague symptoms, such as aches and pains.
- Therefore, laboratory studies are especially helpful.
- Tetany develops at serum calcium levels of 5 to 6 mg/dL (1.2 to 1.5 mmol/L) or lower.
- Serum phosphate levels are increased, and x-rays of bone show increased density.
- Calcification is detected on x-rays of the subcutaneous or paraspinal basal ganglia of the brain.

Medical Management

- The goal of therapy is to increase the serum calcium level to 9 to 10 mg/dL and to eliminate the symptoms.
- When hypocalcemia and tetany occur after a thyroidectomy, the immediate treatment is administration of IV calcium gluconate.
- If this does not decrease neuromuscular irritability and seizure activity immediately, sedative agents such as pentobarbital may be administered.
- Parenteral parathormone can be

Management

- Tracheostomy or mechanical ventilation may become necessary, along with bronchodilating medications, if the patient develops respiratory distress.
- A diet high in calcium and low in phosphorus is prescribed.
- Although milk, milk products, and egg yolk are high in calcium, they are restricted because they also contain high levels of phosphorus.
- Oral tablets of calcium salts, such as calcium

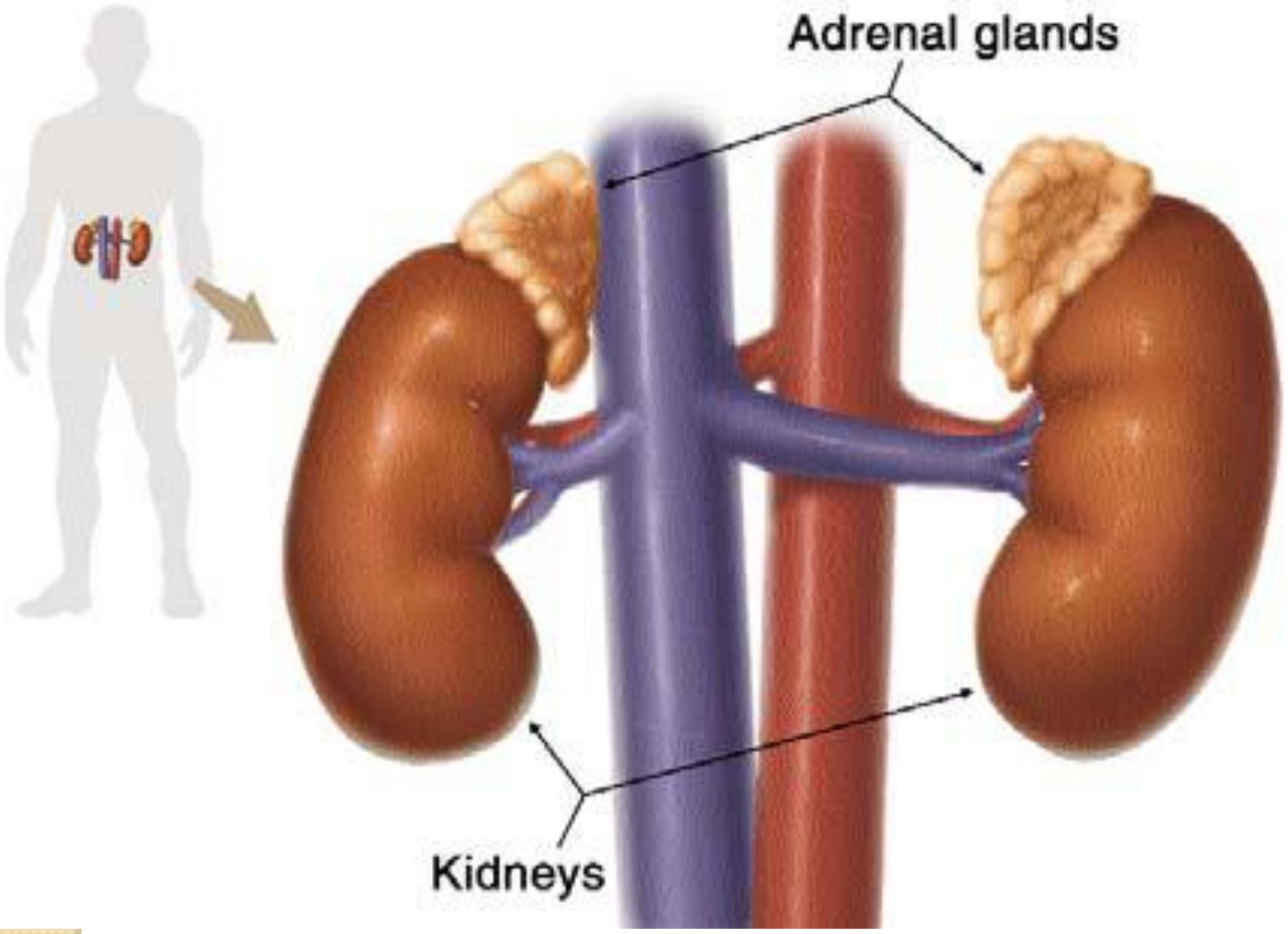
Nursing Management

- Care of postoperative patients who have undergone thyroidectomy, parathyroidectomy, or radical neck dissection is directed toward detecting early signs of hypocalcemia and anticipating signs of tetany, seizures, and respiratory difficulties.
- Calcium gluconate is kept at the bedside with equipment necessary for emergency IV administration.
- If the patient requiring administration of calcium gluconate has a cardiac disorder, is subject to

Cont….

- Consequently, the cardiac patient requires continuous cardiac monitoring and careful assessment.
- An important aspect of nursing care is teaching about medications and diet therapy.
- The patient needs to know the reason for high calcium and low phosphate intake and the symptoms of hypocalcemia and hypercalcemia.
- He or she should know to contact the physician immediately if these symptoms occur.

Disorder of adrenal gland


Adrenal glands
Kidneys

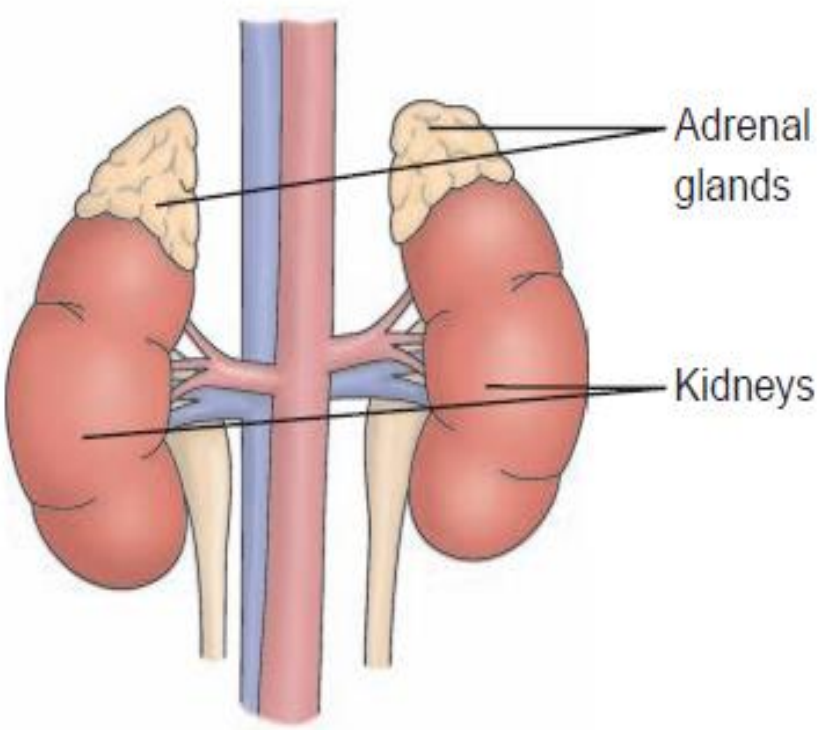
Anatomic and Physiologic Overview 0f adrenal…

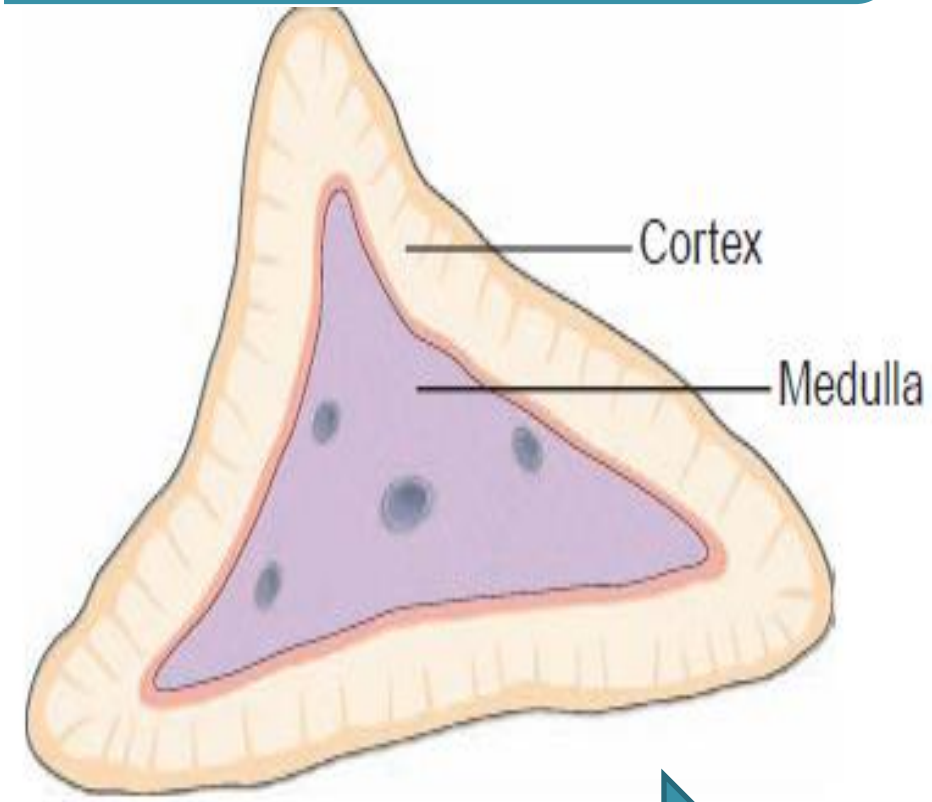
They are small, bilateral structures, wt ~5 g each.

- lie retroperitoneally at the apex of each kidney.
- has two endocrine glands with separate, independent functions.

- AM at the center of the gland secretes catecholamines,
- AC at outer portion of the gland secretes steroid hormones .

Anatomic and Physiologic Overview 0f adrenal…





Fight OR flight

Adrenal Cortex…

Biosynthesis, Transport, and Metabolism

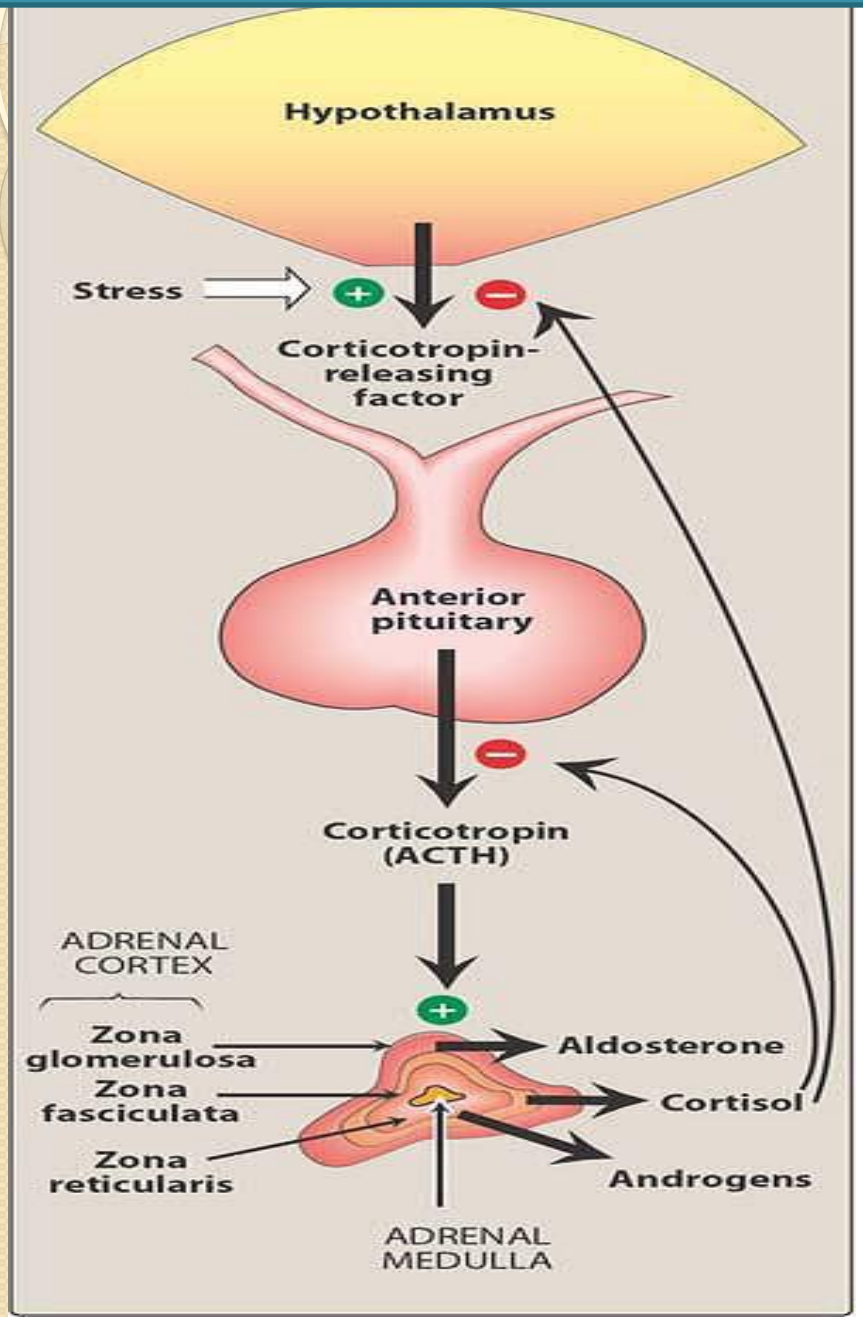
The adrenal cortex produces 3 classes of corticosteroid hormones:

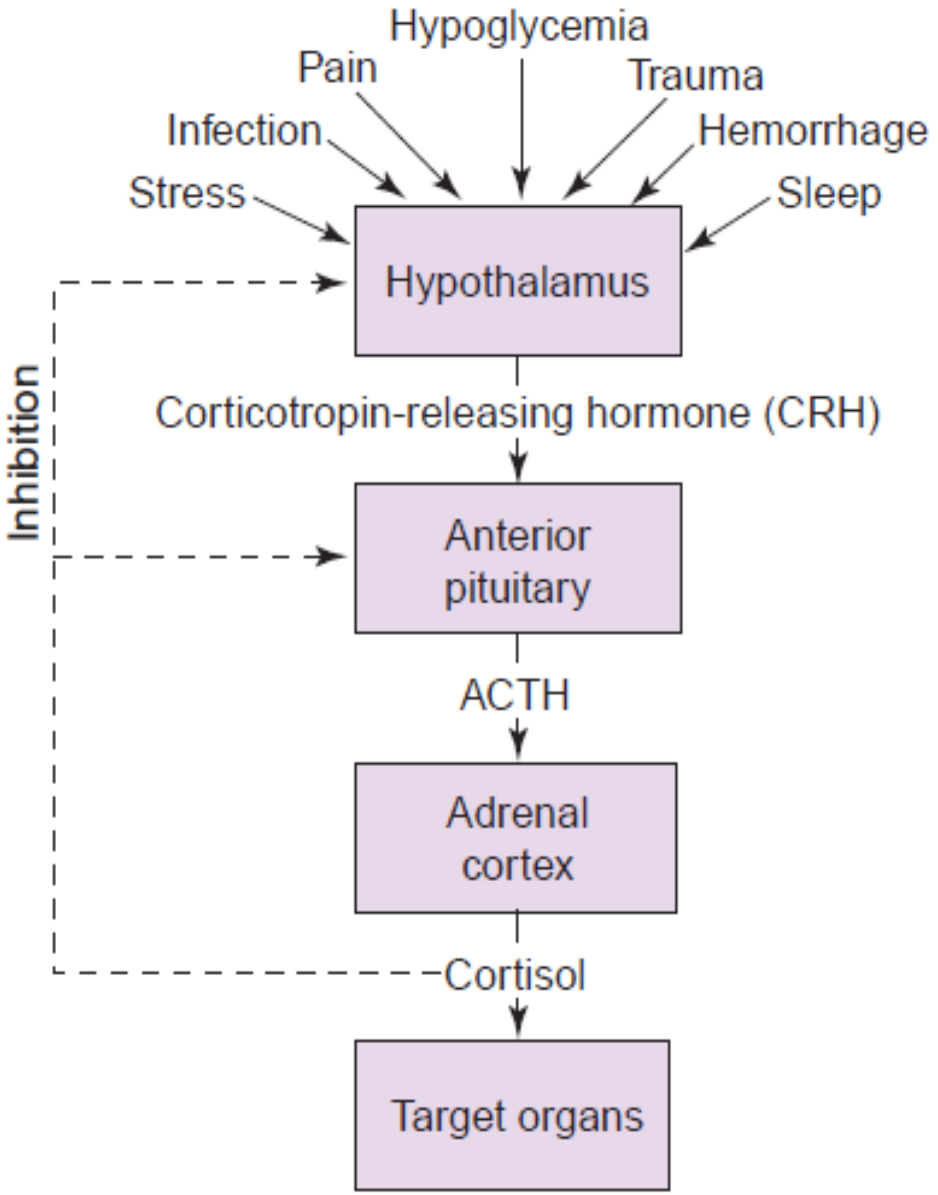
1. **Glucocorticoids(**hydrocortisone) → your cortisol
2. **Mineralocorticoids,** mainly → your aldosterone
3. **Sex hormones**, mainly **androgens** → your sex

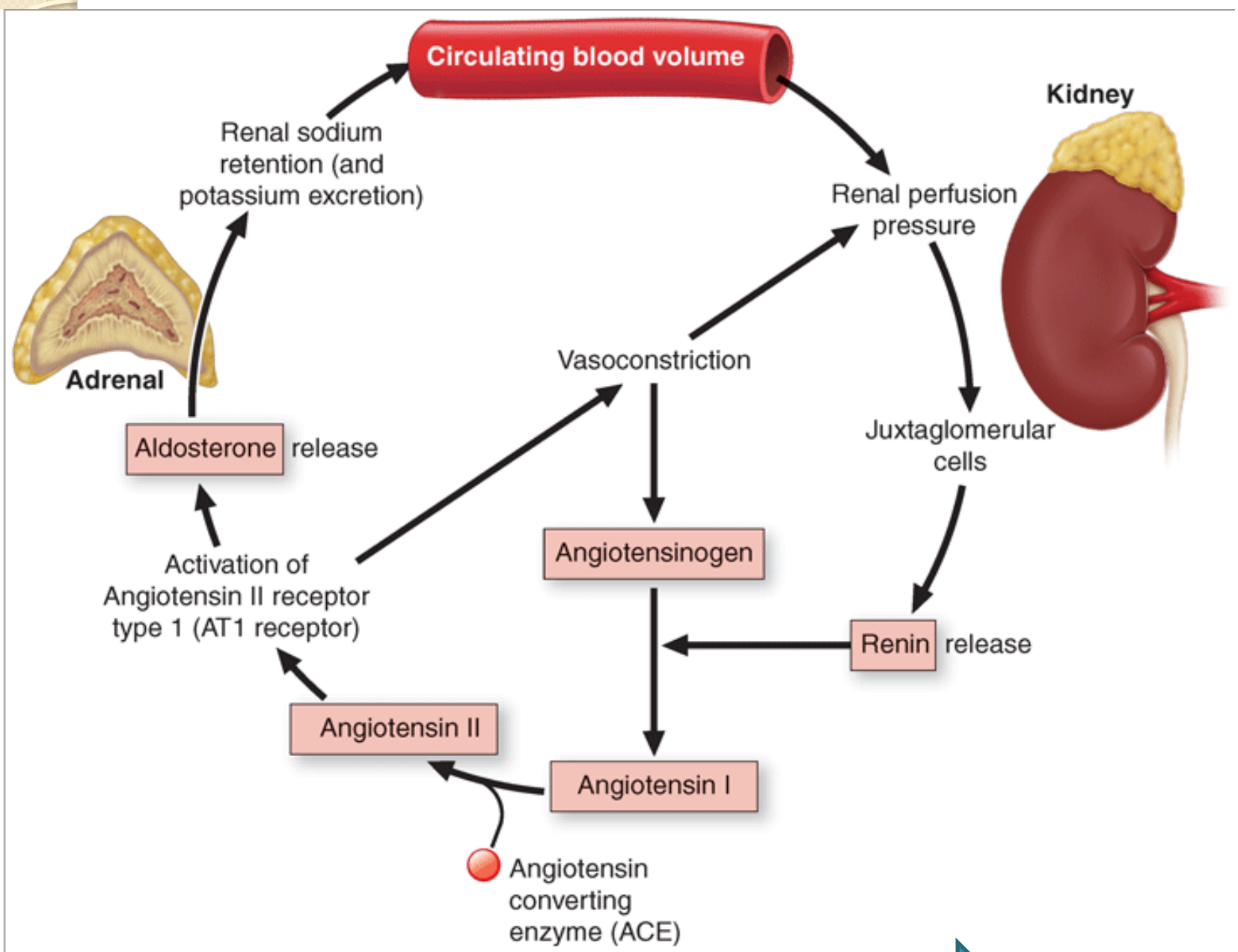
- The secretion of the glucocorticoids hormone is controlled by the ACTH.

- The main site for metabolism of the adrenal cortical hormones is the liver.
- eliminated in either the urine or the bile.

Regulation of corticosteroid secretion







Regulation of the renin-angiotensin-aldosterone (RAA) system

Hormones secreted by cortex

CORTICOSTEROIDS

Glucocorticoids

CORTISOL

- immune system
- glucose
- stress

Mineralocorticoids

ALDOSTERON

- blood pressure
- water and salt balance

Specific Disorders of the Adrenal Glands

1. **Disorders of the Adrenal medulla**
2. **Hyper vs hypo dysfunction of adrenal cortex**

1. Pheochromocytoma:

Introduction

Pheochromocytomas and paragangliomas are:

❖catecholamine- producing tumors derived from the sympathetic or parasympathetic NS.

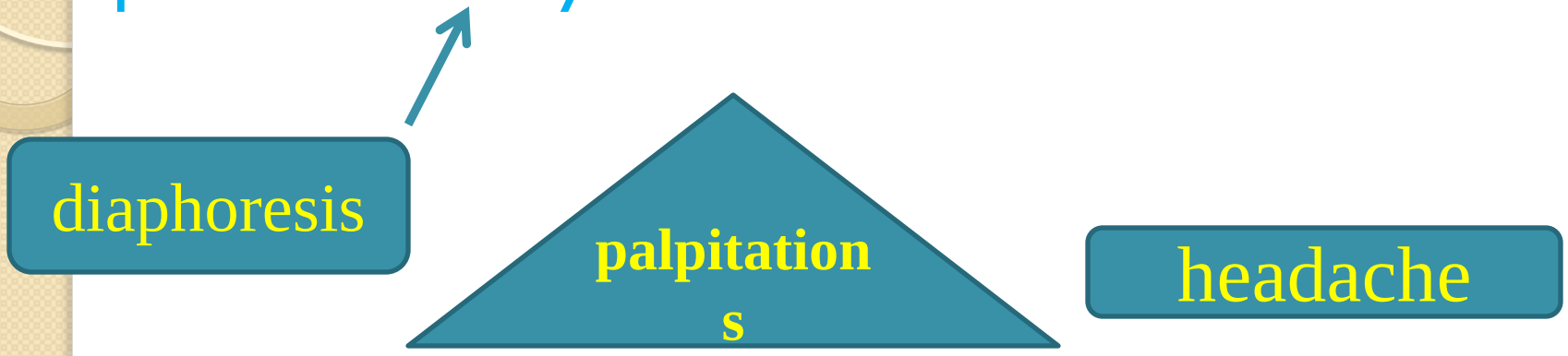
❖may arise sporadically or be inherited as features of multiple endocrine

❖ Dx: potentially correctable cause of HTN, and their removal can prevent hypertensive crises that can be lethal.

❖The clinical presentation is variable from adrenal disorder to HTN crisis with associated cerebrovascular or cardiac complications.

Clinical Manifestations

- typical **triad** symptoms of pheochromocytoma :



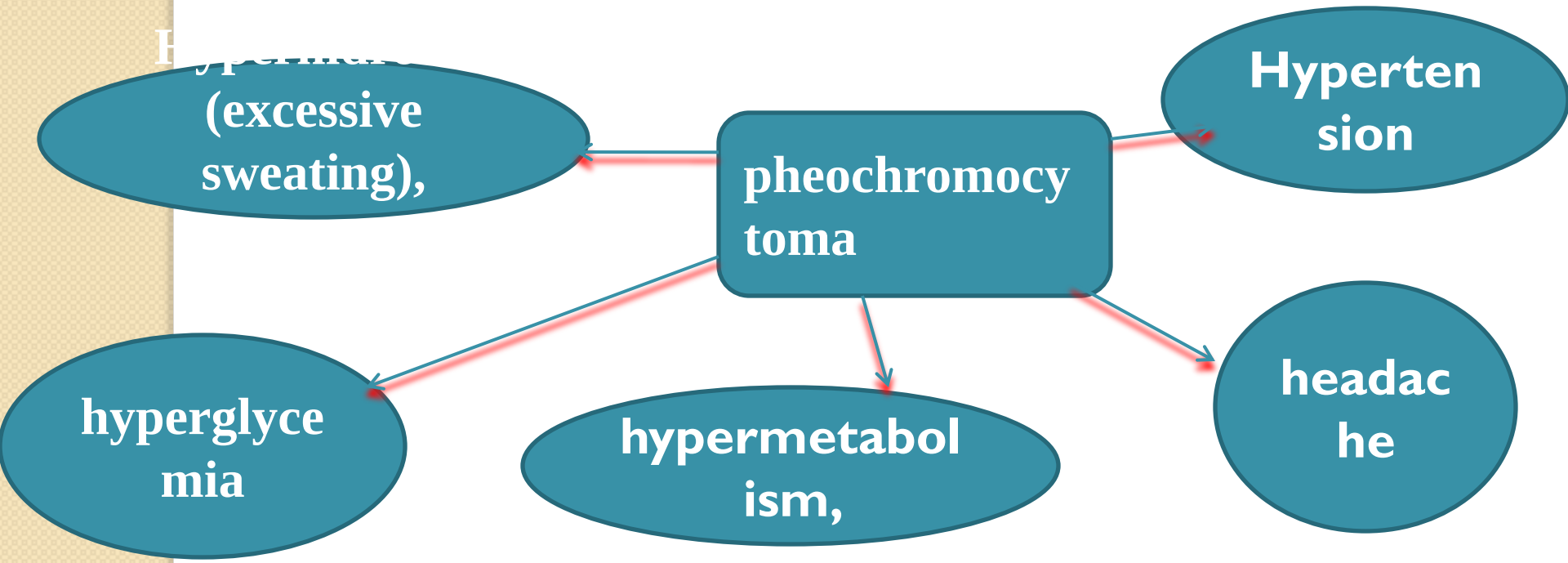
- Sx usually begin abruptly & subside slowly.

Other Sx:

- ✓ tremor, flushing, anxiety, hyperglycemia,
- ✓ polyuria, N/V & diarrhea, abdominal pain…

Assessment and Diagnostic Findings

1. S/S: It is suspected if signs of sympa over activity occur in association with marked elevation of BP. These signs can be associated with the "five H's":



- The presence of these signs is highly predictive of pheochromocytoma.

Diagnostic Method	Sensitivity	Specificity
24-h urinary tests		
Vanillylmandelic acid	++	++++
Catecholamines	+++	+++
Fractionated metanephrines	++++	++
Total metanephrines	+++	++++
Plasma tests		
Catecholamines	+++	++
Free metanephrines	++++	+++
CT	++++	+++
MRI	++++	+++
MIBG scintigraphy	+++	++++
Somatostatin receptor scintigraphy*	++	++
Dopa (dopamine) PET	+++	++++

*Particularly high in head and neck paragangliomas.

Assessment and Diagnostic Findings….

3. Determine plasma level of catecholamine

Catecholamine	Normal plasma values	Diagnostic values	Further evaluation
Epinephrine	<100 pg/ml (590 pmol/L)	>400 pg/ml (2180 pmol/L)	Values that fall b/n normal & diagnostic levels
Norepinephrine	>100 -550 pg/ml (590- 3240 pmol/L).	>2000 pg/ml (11,800 pmol/l)	

Assessment and Diagnostic Findings….

4. **Imaging studies:**

- CT, MRI & ultrasonography, carried out to localize & to determine whether more than one tumor is present.
- MIBG scintigraphy is a noninvasive, safe procedure that ↑sed the accuracy of diagnosis of adrenal tumors.

5. **Other studies :** focus on evaluating the function of other endocrine glands b/se of association factors.

Management of pheochromocytoma…

A. **Pharmacological Management**

I. Alpha-adrenergic blocking agents (phentolamine or smooth muscle relaxants) to ↓BP quickly.

II. CCBs (nifedipine) are:

- Usually well tolerated by pts
- reduced perioperative fluid requirements,
- prevent cardiovascular complications by prevent catecholamine-induced coronary vasospasm & myocarditis.

III. β-adrenergic blocking agents(propranolol)

- Used in pts with cardiac dysrhythmias and in those not responsive to alpha-blockers.

Surgical Management -adrenalectomy.

- The definitive Rx is surgical removal of the tumor,(**Bilateral or unilateral)**

Nursing role:

- Pt preparation, control of BP and blood volumes(over 4 to 7 d)
- Prevent the risk of postoperative HTN since the pt is subject to the stress & effects of a long surgical procedure may ↑se BP.
- Administer CCB as ordered; used safely without causing undue hypotension.
- For episodes of severe HTN, nifedipine is a fast and effective.

Nursing role…

- Manage a stressful preoperative and postoperative course.
- Monitored for several days in the ICU with special attention:
  - ✓ given to ECG changes,
  - ✓ arterial pressures,
  - ✓ fluid and electrolyte balance,
  - ✓ blood glucose levels.

1. Teaching Patients Self-Care

- Informs the pt about the importance of follow-up monitoring.

- Instructs the pt about the purpose, the medication schedule, & the risks of skipping doses or stopping their administration abruptly (corticosteroid).

- Teach pt & family how to measure the pt's BP & when to notify.

- Provides verbal & written instructions about the procedure for

collecting 24h urine specimens to monitor urine

Promoting Home and Community-Based Care…

2. Continuing Care

- A follow-up visit a home care nurse:

- assess the pt's postoperative recovery, surgical incision, and compliance with the medication schedule.

- Periodic checkups for BP especially in young pt since the risk of recurrence HTN.

- Scheduled the pt for periodic follow-up appointments to observe normal BP, plasma and urine levels of catecholamines.

Hyper vs hypo dysfunction of adrenal cortex

I. Adrenal Gland Hypofunction (Adrenal Insufficiency)

prevalence :

- 5 in 10,000 in the general population.
- Hypothalamic-pituitary origin most frequent, 3 in 10,000,
- primary adrenal insufficiency 2 in 10,000.
- one-half are acquired, mostly caused by autoimmune destruction of the adrenal glands;
- one-half are genetic, most commonly caused by distinct enzymatic blocks in adrenal steroidogenesis affecting glucocorticoid synthesis (i.e. congenital adrenal hyperplasia.)

Adrenal Insufficiency

Adrenocortical steroids may ↓**se** as a result of:

- inadequate secretion of ACTH
- Dysfunction of the hypothalamic-pituitary control mechanism
- Direct dysfunction of adrenal tissue

Adrenal Insufficiency…

- Primary insufficiency :
  - ✓ Occurs when adrenal cortex function is inadequate to meet the pt's need for cortical hormones. Such as Addison's Disease
- Secondary insufficiency:
  - ✓ occur as the result of hypopituitarism or because the pituitary gland has been surgically removed.
  - ✓ Sudden cessation of long-term high-dose glucocorticoid therapy
- Tertiary adrenal insufficiency: results from a hypothalamic defect.

Effect of Insufficiency

- Loss of aldosterone and cortical action
- Decreased gluconeogenesis →Hypoglycemia
- Depletion of liver and muscle glycogen
- Reduced urea nitrogen excretion
- Anorexia and weight loss
- Potassium, sodium, and water imbalances

Clinical Findings of Adrenal Insufficiency

Finding	Primary	Secondary/Tertiary
Anorexia and weight loss	Yes (100%)	Yes (100%)
Fatigue and weakness	Yes (100%)	Yes (100%)
Gastrointestinal symptoms, nausea, diarrhea	Yes (50%)	Yes (50%)
Myalgia, arthralgia, abdominal pain	Yes (10%)	Yes (10%)
Orthostatic hypotension	Yes	Yes
Hyponatremia	Yes (85%–90%)	Yes (60%)
Hyperkalemia	Yes (60%–65%)	No
Hyperpigmentation	Yes (>90%)	No
Secondary deficiencies of testosterone, growth hormone, thyroxine, antidiuretic hormone	No	Yes
Associated autoimmune conditions	Yes	No

ADDISON'S DISEASE (cortical insufficiency)

- It is adrenal cortex function is inadequate to meet the pt's need for cortical hormones

Cause:

- **Autoimmune** or idiopathic atrophy of the adrenal glands.
- **Infection**: TB & histoplasmosis are the most common.
- **surgical removal** of both adrenal glands .
- **Inadequate secretion** of ACTH from the pituitary gland.
- Therapeutic use of corticosteroids (Porth & Matfin, 2009).

Addison's disease

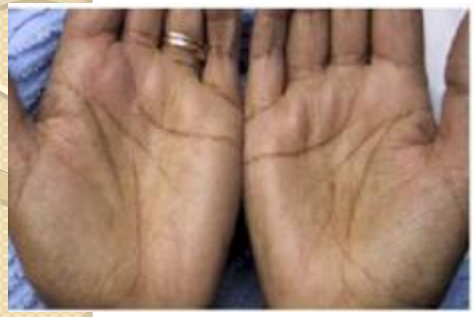
- PRIMARY cause
- destruction of adrenal cortex
  - autoimmune disorders
  - tuberculosis
  - chronic infection

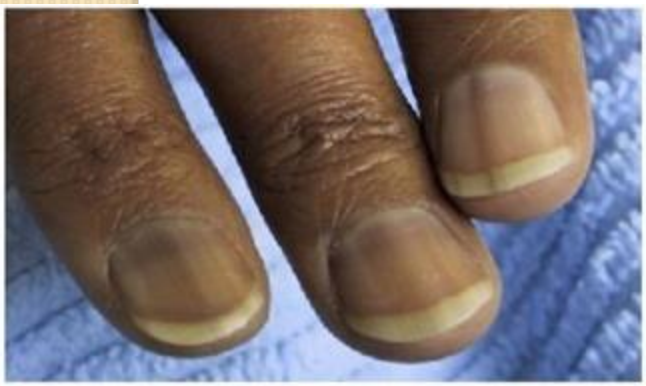
CORTISOL
ALDOSTERONE

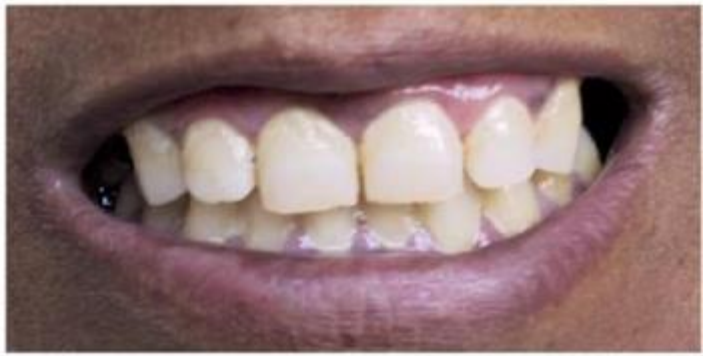
- SECONDARY cause
- Lack of ACTH
  - drugs
  - tumors and infections of pituitary gland

CORTISOL
ALDOSTERONE

Clinical Manifestations -Skin pigmentation



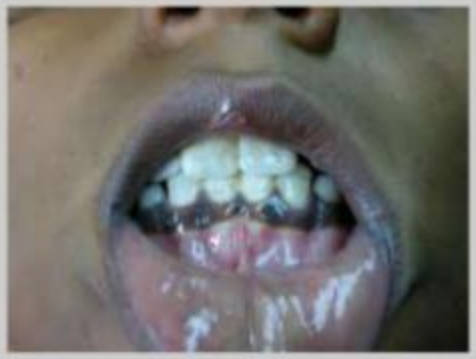


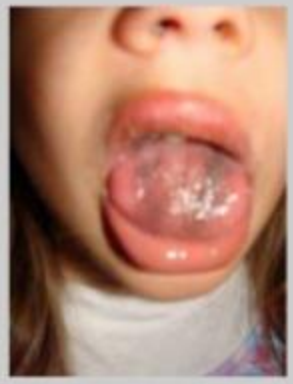


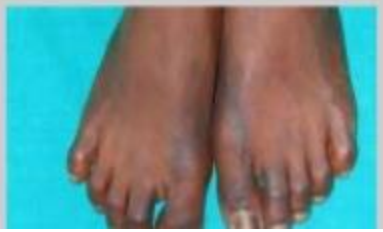
- Weight loss
- Muscle weakness
- Fatigue
- Low blood pressure
- Darkening of skin

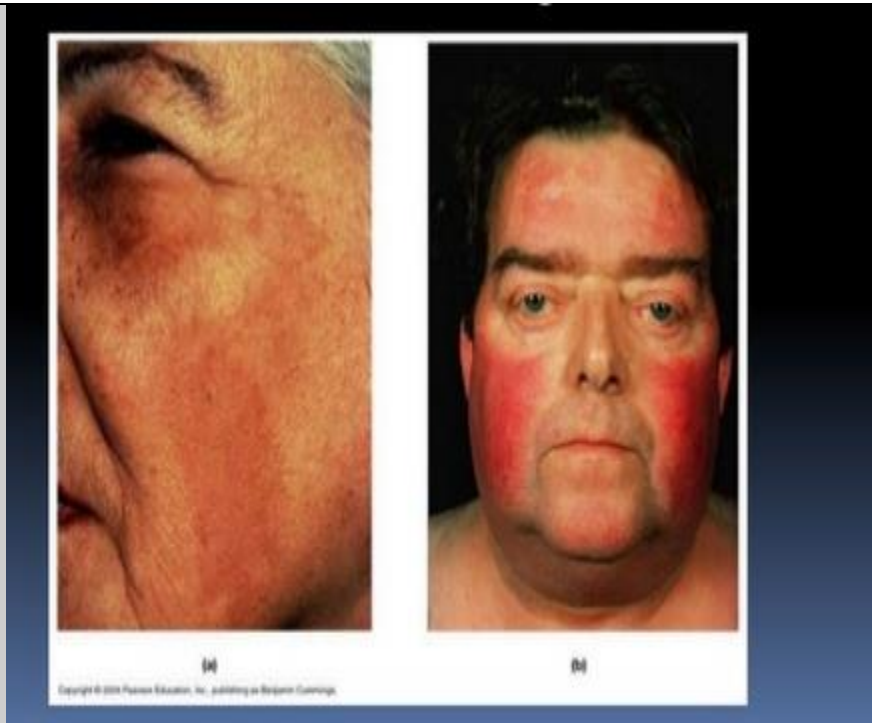
Symptoms include: changes in blood pressure or heart rate, chronic diarrhea, patchy skin color, unnaturally dark colors in some places, paleness, extreme weakness, fatigue, loss of appetite, mouth lesions on the inside of the cheek, nausea and vomiting, salt cravings, slow movements, and unintentional weight loss.

Addison's Pictures









Assessment &DIAGNOSIS

1. Psychosocial assessment

2. Laboratory tests: to differentiate

- ✓ primary from secondary adrenal insufficiency and
- ✓ adrenal insufficiency from normal adrenal function

➢ Pts with primary insufficiency have:

- ✓ A greatly ↑sed plasma ACTH level.
- ✓ A lower serum cortisol concentration

3. ACTH STIMULATION TEST

4. Imaging assessment (location & number of tumor)

Medical MGT & nurse responsibility

- ✓Immediate treat shock: restoring blood circulation by fluids
- ✓corticosteroids, hydrocortisone IV followed by D5W in NS
- ✓monitoring V/S,
- ✓ position pt =recumbent
- ✓Vasopressor if hypotension persists &
- ✓Give antibiotics pt with chronic adrenal insufficiency

Replacement therapy

Cortisol → **Hydrocortisone**

Aldosterone → **Fludrocortisone**

Medical MGT & nurse responsibility…

1. Assessing the Patient :-

Hx and P/E focus on :

- symptoms of fluid imbalance and on the pt's level of stress
- changes in color & turgor(for chronic insufficiency & hypovolemia).
- change in wt, muscle weakness, fatigue.
- any illness or stress that precipitated the acute crisis

- Monitor the BP and PR as the pt moves from a lying, sitting, and standing position to assess for inadequate fluid volume.
- A ↓se in SBP(≥20 mm Hg) may indicate depletion of fluid volume, especially if accompanied by symptoms.

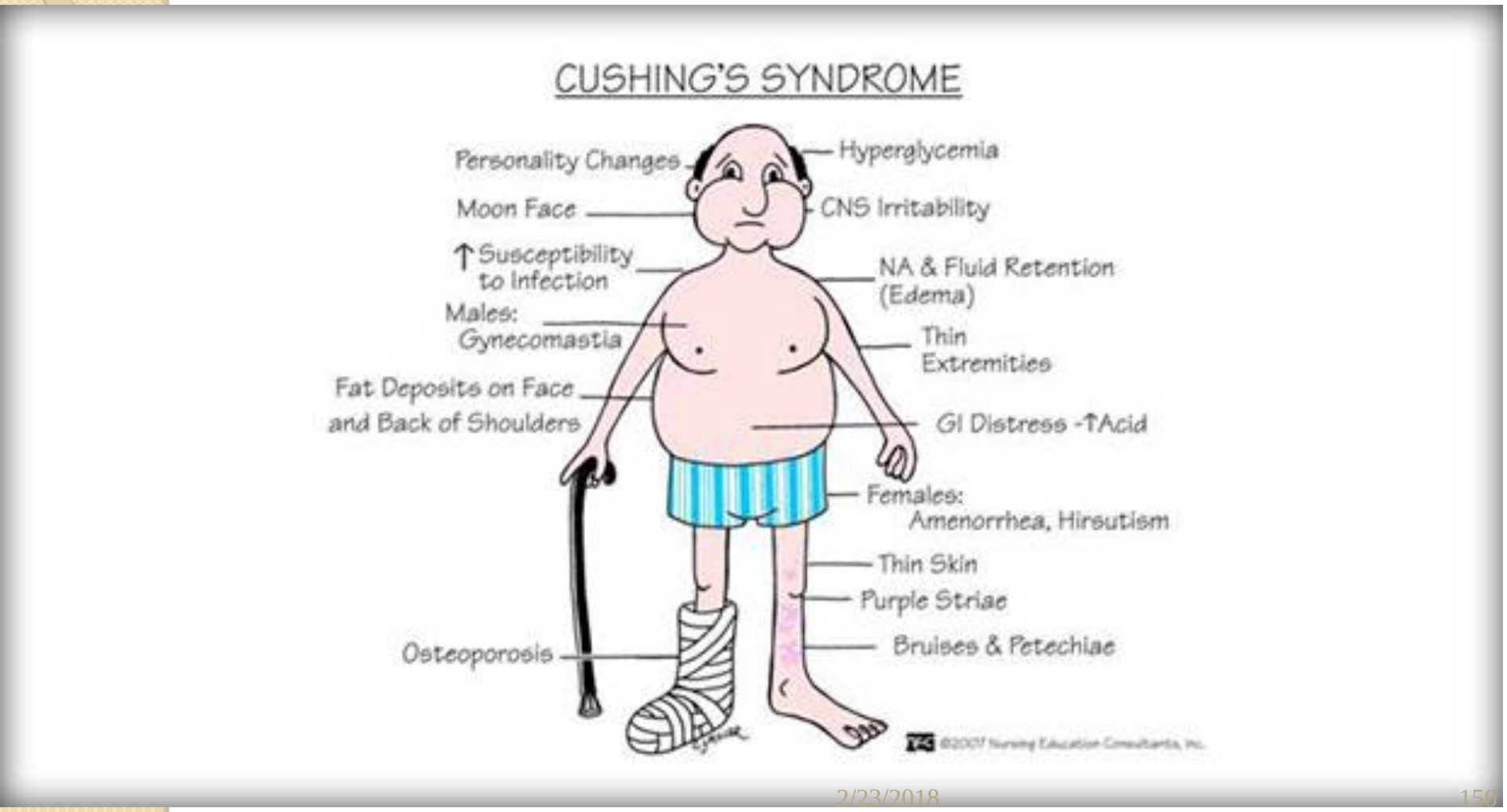
Nursing Management…

2. Monitoring and Managing Addisonian Crisis

- monitored for S/S indicative of addisonian crisis:
  - shock; hypotension; rapid, weak pulse; rapid RR, pallor and extreme weakness
- Avoided Physical and psychological stressors:
  - cold exposure, overexertion, infection, and emotional distress.
- The pt with addisonian crisis requires immediately:
  - Rx with IV fluid, glucose & electrolytes(esp. Na),
  - vasopressors & replacement of missing steroid hormones;

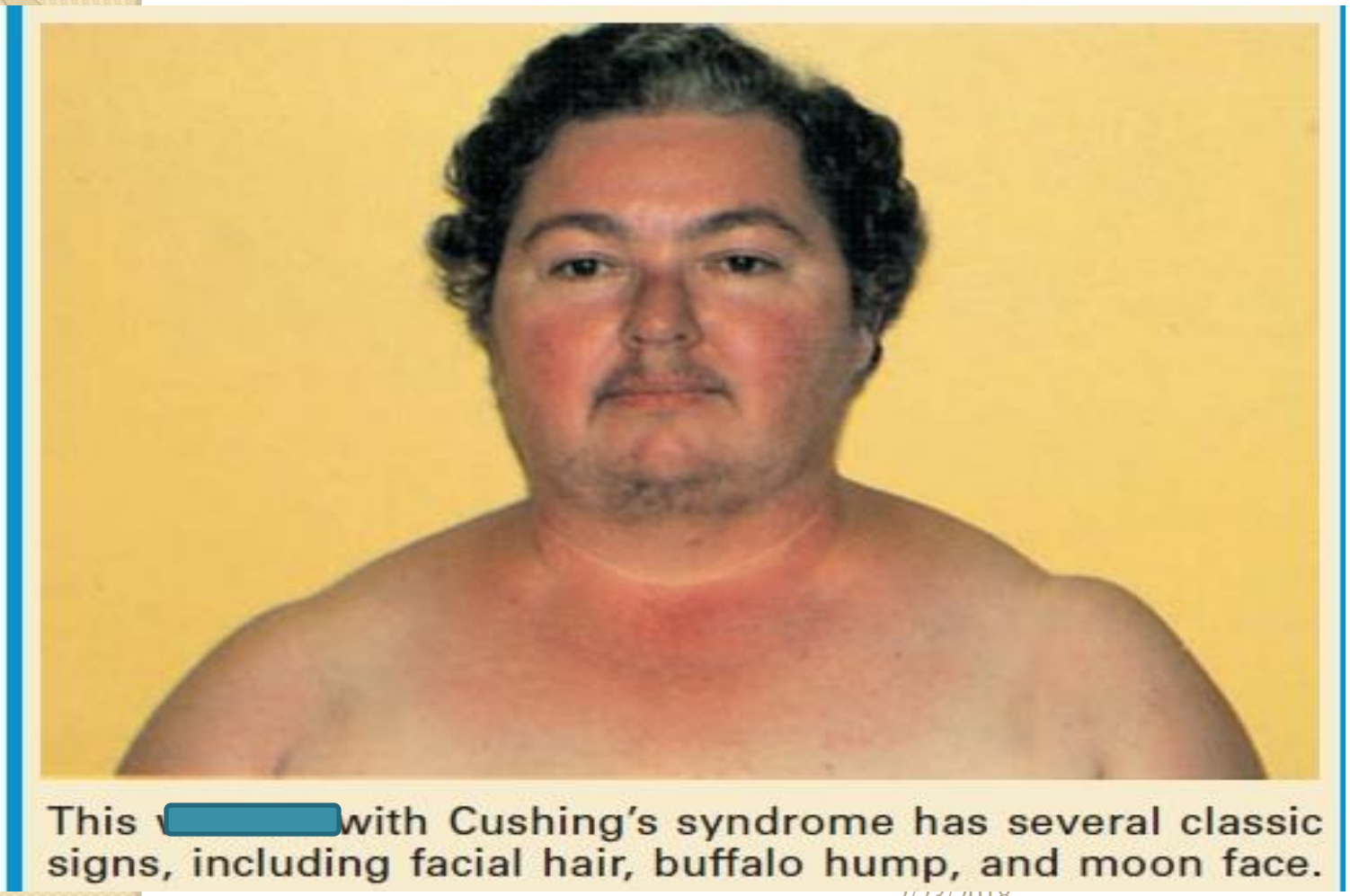
II. Adrenal Gland Hyper function

Cushing's syndrome reflects a constellation of clinical features that result from chronic exposure to excess glucocorticoids of any etiology.



II. Adrenal Gland Hyper function

CUSHING'S SYNDROME



This [redacted] with Cushing's syndrome has several classic signs, including facial hair, buffalo hump, and moon face.

CUSHING'S SYNDROME….

- It occurs with an incidence of 1–2 /100,000 population/ y.
- mild cortisol excess may be more prevalent among pts with several features.
- more frequently affects women, with the exception of prepubertal cases, where it is more common in boys.
- ectopic ACTH syndrome is more frequently identified in men.

CUSHING'S SYNDROME…

- It is the manifestations of hypercortisolism from any cause.
- 3 important forms of Cushing's syndrome result from excess glucocorticoid production by the body.

1. **pituitary form**, excessive production of ACTH by a tumor of the pituitary gland
2. ***Adrenal* form**, caused by a benign or malignant adrenal tumor.
3. **Ectopic form** ,caused by a non pituitary ACTH-secreting tumor.

CUSHING'S SYNDROME

- Result from:

ACTH-dependent causes

ACTH-secreting pituitary tumor (Cushing' s disease)

Pituitary CRH-secreting neoplasm (ectopic CRP syndrome)

Nonpituitary ACTH-secreting neoplasm (ectopic ACTH syndrome)

ACTH-independent causes

Adrenal adenoma

Adrenal carcinoma

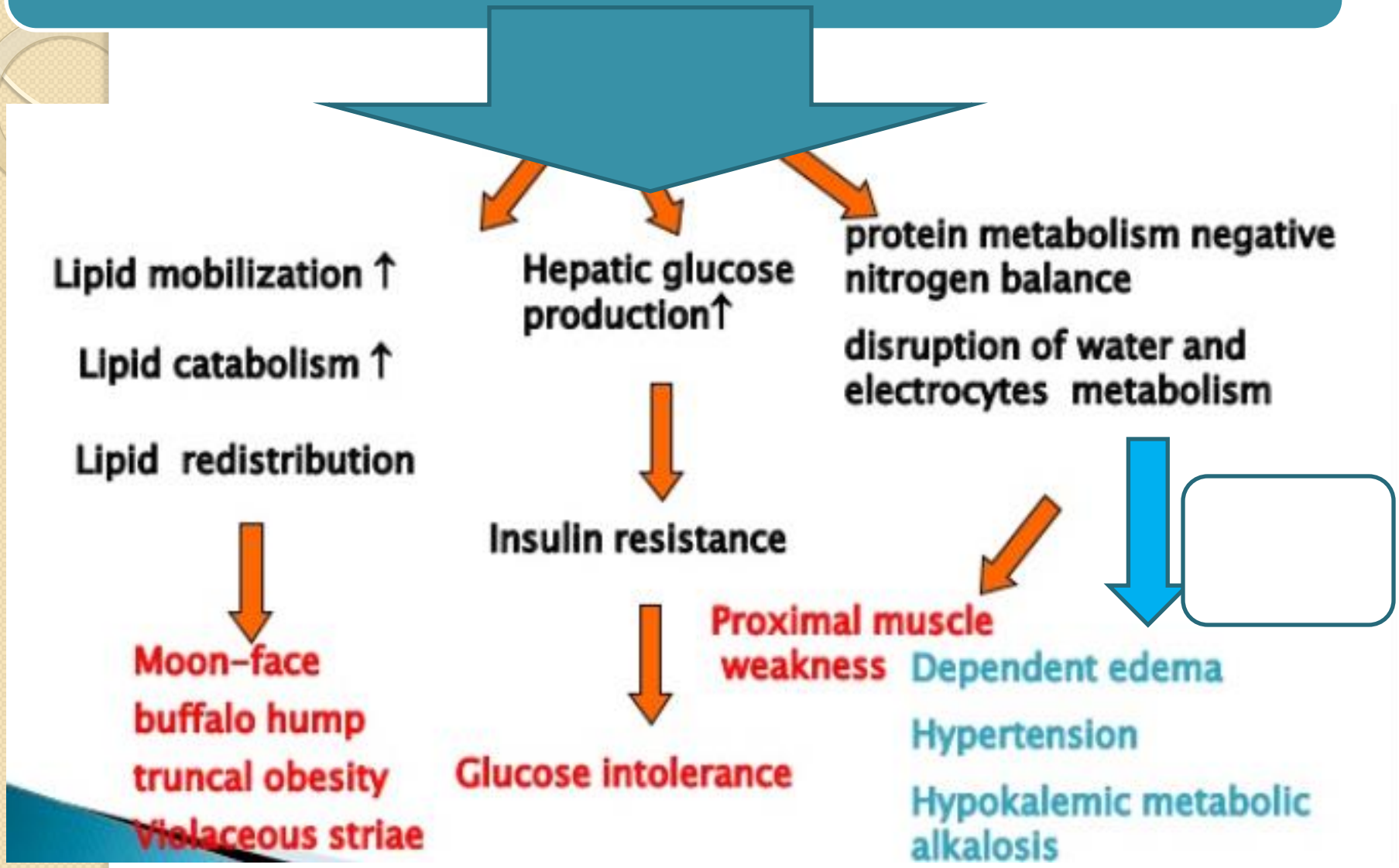
Micronodular adrenal disease

McCune-Albright syndrome

Massive macronodular adrenal diease

Pseudo-cushing Syndrome

Clinical feature



Systemic manifestation

Ophthalmic

- Cataracts & Glaucoma

Cardiovascular

- Hypertension & Heart failure

Endocrine/Metabolic

- Truncal obesity
- Moon face & buffalo hump
- Sodium retention & hypokalemia
- Hyperglycemia &metabolic alkalosis
- Menstrual irregularities & impotence
- Negative nitrogen balance
- Altered calcium metabolism
- Adrenal suppression

Immune Function

- ↓sed inflammatory responses
- Impaired wound healing
- ↑sed susceptibility to infections

Skeletal

- Osteoporosis & spontaneous fractures
- Aseptic necrosis of femur

Gastrointestinal

- Peptic ulcer &Pancreatitis

Muscular

- Myopathy & muscle weakness

Dermatologic

- Thinning of skin, Petechiae &
- Ecchymoses, striae & Acne

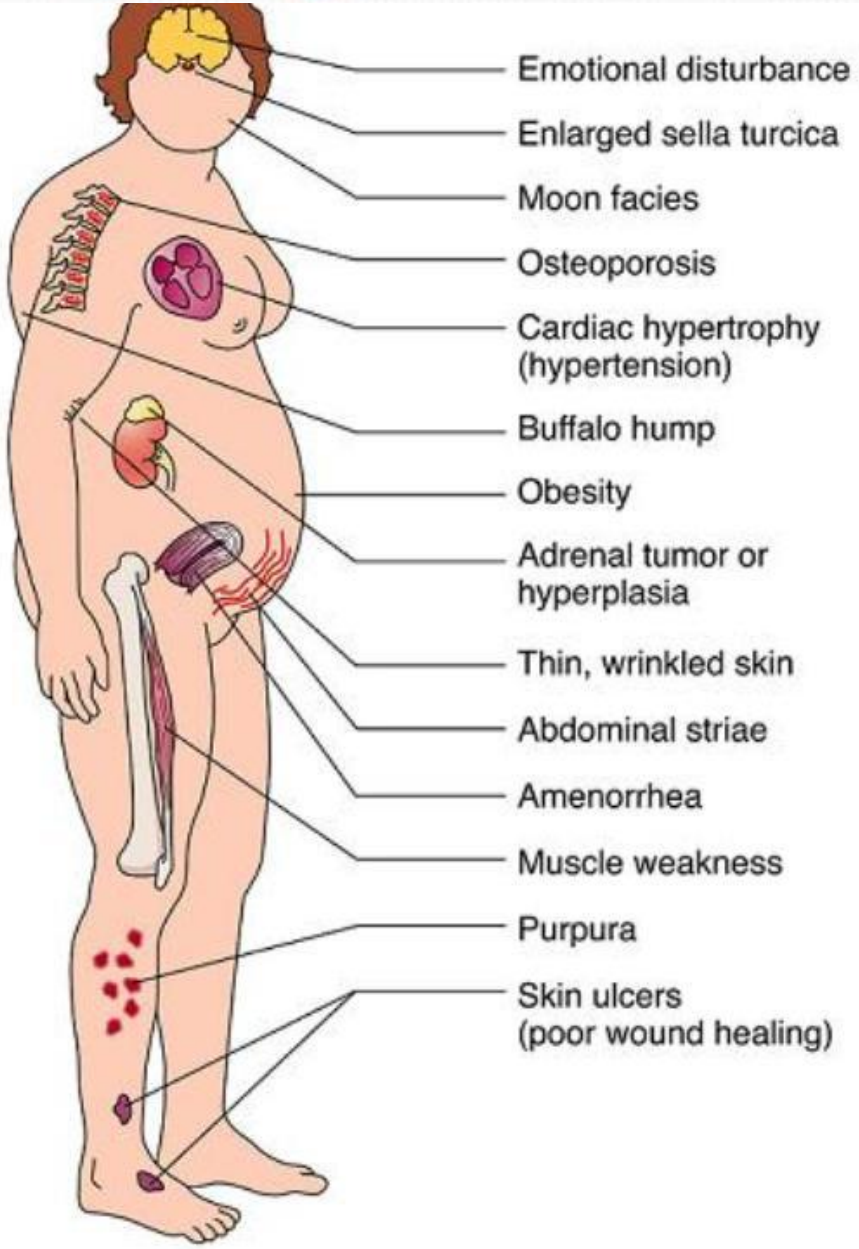
Psychiatric

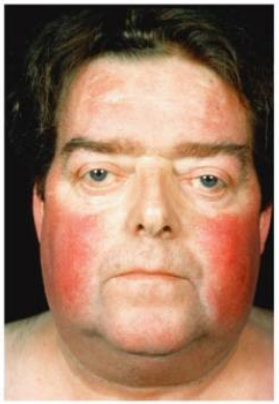
- Mood alterations &psychoses

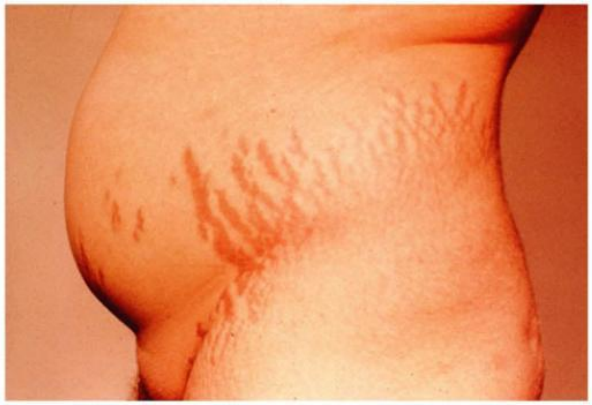
Symptoms of Cushing's

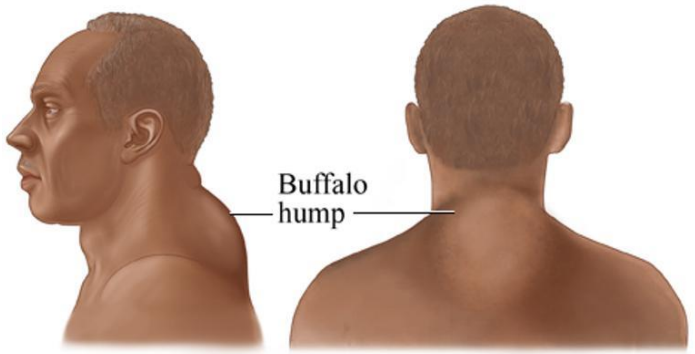
- A person can have more of less of these symptoms:
  - Upper body obesity with thin arms and legs
  - Buffalo Hump
  - Red, Round Face
  - High Blood Sugar
  - High Blood Pressure
  - Vertigo
  - Blurry Vision
  - Acne
  - Female Balding
  - Water Retention
  - Menstrual Irregularities
  - Thin Skin and Bruising
  - Purple Striae
  - Poor Wound Healing
  - Hirsutism
  - Severe Depression
  - Cognitive Difficulties
  - Emotional Instability
  - Sleep Disorders
  - Fatigue

Symptoms of Cushing's









Assessment and Diagnostic Findings

- **Overnight dexamethasone suppression test:**
  - Most widely used & most sensitive screening test for diagnosis of pituitary & adrenal causes.
  - Suppression of cortisol <5 mg/dL indicates the hypothalamic–pituitary–adrenal axis is functioning properly.
  - **falsely** elevate cortisol levels due to:
    - ✓ Stress, obesity, depression,
    - ✓ medications (antiseizure, estrogen &

Assessment and Diagnostic Findings

- **Nighttime salivary cortisol levels**:
  - ✓ show promise in screening for Cushing's syndrome (Gross, Mindea, Pick, et al., 2007).
  - ✓ **Serum analysis/measurement:**
    - ➢ to determine normal diurnal variation in plasma levels is frequently **absent** in adrenal dysfunction
    - ➢ ↑se in serum Na+, blood glucose levels & ↓se serum K+ , a reduction in the number of b/d eosinophils & disappearance of lymphoid tissue.

Assessment and Diagnostic Findings

- **plasma ACTH by radioimmunoassay in conjunction with the high-dose suppression test** to:

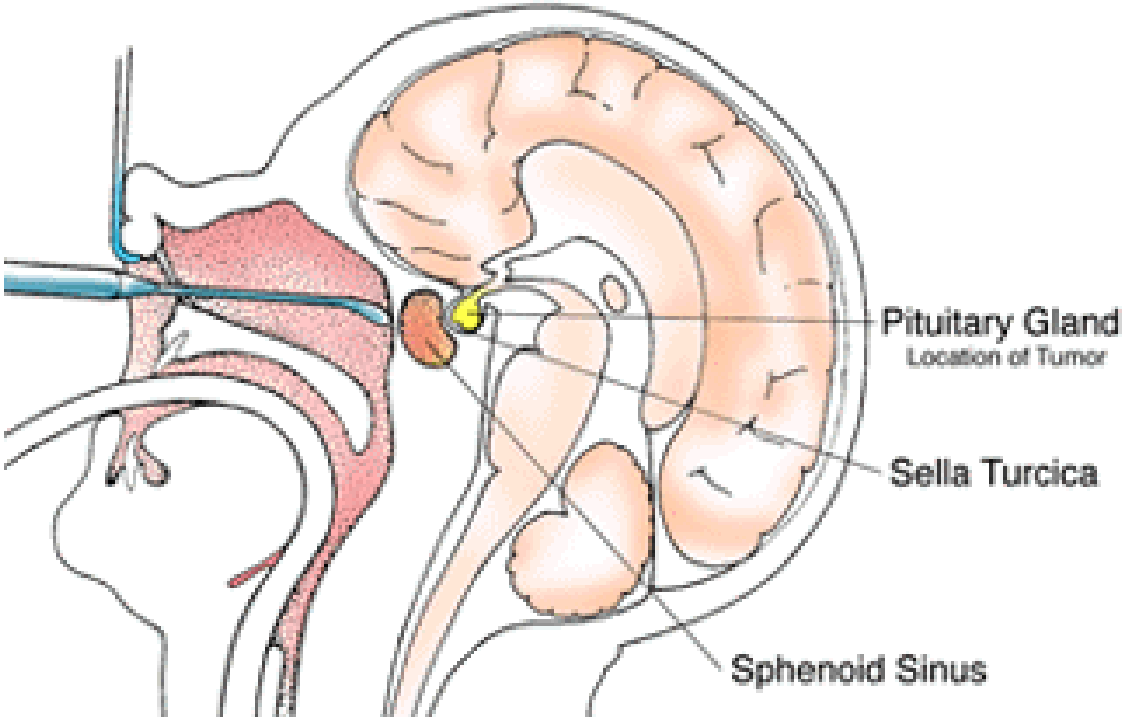
- distinguish pituitary tumors from ectopic sites of ACTH as the cause of Cushing's syndrome.
- ↑se both ACTH & cortisol → pituitary/ hypothalamic d/se.
- ↓se ACTH with ↑cortisol level → adrenal d/se.

CT, ultrasound, or MRI

- to localize adrenal tissue and detect tumors of the adrenal gland.

Management of Cushing's

- **SURGERY**
- **RADIATION**
- **CHEMOTHERAPY**
- **CORTISOL-INHIBITING DRUGS**



Management Cushing's syndrome

Cause dependent: If the is caused by

1. pituitary tumors:

- removal of the tumor is the treatment of choice
- has an 80% success rate.
- Radiation of the pituitary gland also successful.

2. Primary adrenal hypertrophy:

- Adrenalectomy is the treatment of choice

3. Ectopic ACTH secretion:

- Adrenal enzyme inhibitors to reduce hyperadrenalism.
- like metyrapone, aminoglutethimide, mitotane &

Nursing responsibilities

1. Assessment

- overall health history and examination focus on the effects adrenal cortex hormones
- The Hx includes:
  - pt's level of activity & ability to carry out routine and self care activities.
  - Assesses the skin: trauma, infection, breakdown, bruising & edema.
  - Changes in physical appearance & responses to the changes.
  - mental function: mood, responses to questions, awareness of environment & level of depression.

Nursing responsibilities….

2. Diagnosis :

Major nursing diagnoses of the pt with Cushing's syndrome :

- ✓ Risk for injury related to weakness
- ✓ Risk for infection related to altered protein metabolism & inflammatory response
- ✓ Self-care deficit related to weakness, fatigue, muscle wasting,
- ✓ Impaired skin integrity related to edema, impaired

Nursing responsibilities….

Planning and Goals

The major goals for the patient:

- decreased risk of injury & infection,
- increased ability to carry out self-care activities,
- improved skin integrity, body image & mental function,
- absence of complications.

Nursing responsibilities….

- ***Decreasing Risk of Injury***

- Establishing a protective environment helps prevent falls, fractures, and other injuries to bones and soft tissues.
- Foods high in protein, calcium, and vitamin D; low in Na & calories are recommended to minimize muscle wasting and osteoporosis.

Decreasing Risk of Infection

- avoid unnecessary exposure to others infections.
- frequently assesses for subtle signs of infection, b/se the anti-inflammatory effects of corticosteroids may mask the common signs of inflammation and infection.

Nursing responsibilities….

Preparing the Patient for Surgery

- Since DM and peptic ulcer are common in pts with Cushing's syndrome, insulin therapy and medication to treat peptic ulcer are initiated if needed.

- Before, during, and after surgery, BGL monitoring and assessment of stools for b/d are carried out to monitor for these complications.

- If the pt has experienced Wt gain, special instruction is given about postoperative breathing exercises.

Nursing responsibilities….

Encouraging Rest and Activity

- Although the pt with Cushing's syndrome experiences insomnia, weakness, fatigue, and muscle wasting,
- The nurse should encourage moderate activity to prevent complications of immobility and promote ↑ed self-esteem.
- It is important to help the pt plan and space rest periods throughout the day and promote.

Nursing responsibilities….

Promoting Skin Integrity

- Meticulous skin care to avoid traumatizing the pt's fragile skin.
- Avoid use of adhesive tape b/se it can irritate the skin and tear the fragile skin
- Frequently assesses the skin and bony prominences
- assists the pt to change positions frequently.

Improving Body Image

- discuss the effect of changes about his or her self-concept and r/hips with others.
- Modified Wt gain and edema by a low-carbohydrate, low-sodium diet, and a high protein intake to reduce bothersome symptoms.
- Support psychotic behavior & encourages the pt and family members to verbalize their feelings and concerns.

Nursing responsibilities….

Monitoring & Managing Potential complications(addisonian crisis)

- The pt with Cushing's syndrome, treated by withdrawal of corticosteroids (adrenalectomy, removal of a pituitary tumor)→ risk for adrenal hypofunction and addisonian crisis.

- If the hormone level is ↓sed rapidly b/se of surgery or abrupt cessation of corticosteroid agents, →hypofunction & addisonian crisis.

- pt with Cushing's syndrome should be assessed for signs & sx of addisonian crisis.

- If addisonian crisis occurs, the pt is treated for circulatory collapse & shock

Disorders of Adrenocorticosteroid Hormones

PRIMARY ALDOSTERONISM

- Excess production of aldosterone due to
  - Adrenocortical hyperplasia or adenoma
  - Excess levels of glucocorticoid(Cushing's syndrome)
  - Tend to raise the blood pressure
  - Facilitate salt and water retention by the kidney.

Clinical Manifestations

- profound ↓ K+ serum levels of (hypokalemia) &H+ (alkalosis)
- ↑se in pH and serum bicarbonate concentration.
- serum Na+ level is normal or elevated, depending on the amount of water reabsorbed with the sodium.
- HTN is the most prominent and almost **universal sign**

Assessment and Diagnostic Findings

- high or normal serum sodium level & high serum aldosterone
- low serum potassium & renin levels
- The measurement of the aldosterone excretion rate after salt loading is a useful diagnostic test.
- The renin–aldosterone stimulation test and bilateral adrenal venous sampling are useful in differentiating the cause.
- Antihypertensive medication may be D/C up to 2 wks before testing.

Medical & Nursing Management

- Surgical removal of the adrenal tumor through adrenalectomy.
- Hypokalemia resolves for all pts after surgery,
- Spironolactone to control HTN since it may persist.
- Bilateral adrenalectomy, replacement of corticosteroids will be lifelong;
- unilateral adrenalectomy , replacement therapy may be temporarily/short course.

Medical & Nursing Management…

- A normal serum glucose level is maintained with insulin, appropriate IV fluids, and dietary modifications to prevent acute complications
- in the postoperative period:
  - frequent assessment of V/S to detect early S/S of adrenal insufficiency and crisis or hemorrhage.
  - Explaining all treatments and procedures, providing comfort measures, and providing rest.

summary

A. What are the main function of adrenal glands?

B. What are the specific disorder of adrenal glands?

C. Can you explain the C/Ms pt with adrenal cortex disorder?

D. List the typical clinical manifestations of adrenal medulla dysfunction(triad Sx).

E. Can you explain the purpose of monitoring Addisonian crisis?

F. List down major nursing goals for pt with Cushing's syndrome.

G. List signs those are highly predictive of pheochromocytoma(5H).