MEDICINE

432 Team

39 Thyroid Disorders

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COLOR GUIDE: • Females' Notes • Males' Notes • Important • Additional

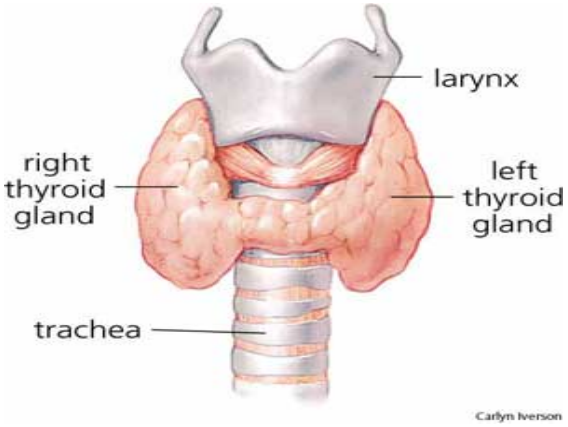
Objectives

1. Thyroid anatomy
2. Action of thyroid hormones
3. Thyroid function
4. Thyroid disorders
    - Goiter
    - Hyperthyroidism
    - Hypothyroidism

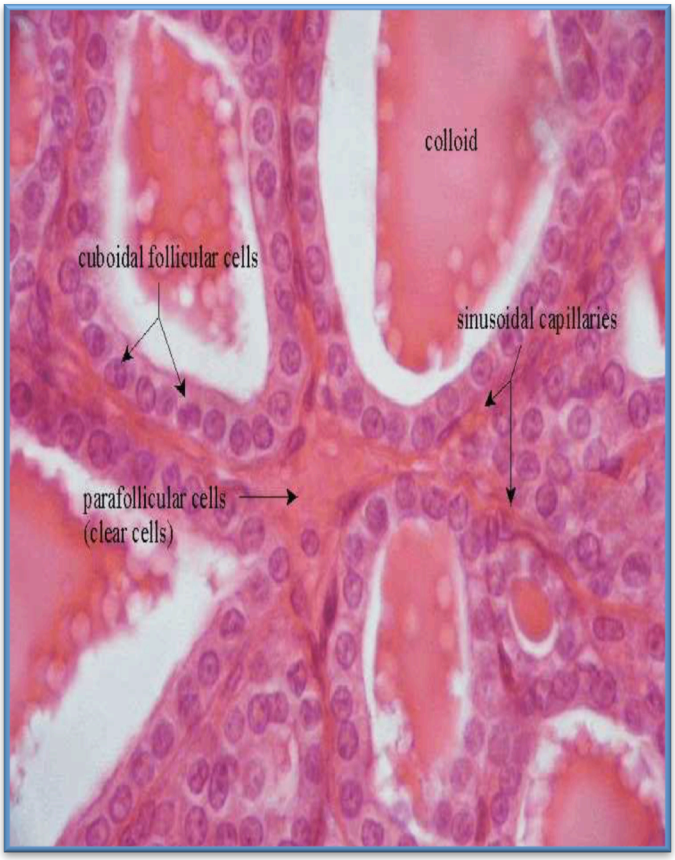
Let's review some basics!

Anatomy of the thyroid:

- The thyroid gland is made up of numerous thyroid follicles.
- It has 2 lobes that are connected by the isthmus.
- Weighs 20 g, has more volume in men, increases with age and body weight, and decreases with iodine intake.
- Located in front of the larynx



Histology of the thyroid:



Remember!

The thyroid gland consists of: stroma & parenchyma. The parenchyma contains the thyroid follicles, which are the structural and functional units of the thyroid. They are made up of simple cuboidal epithelium and colloids.

Simple cuboidal epithelium:
a- Follicular cells
b- Para-follicular cells

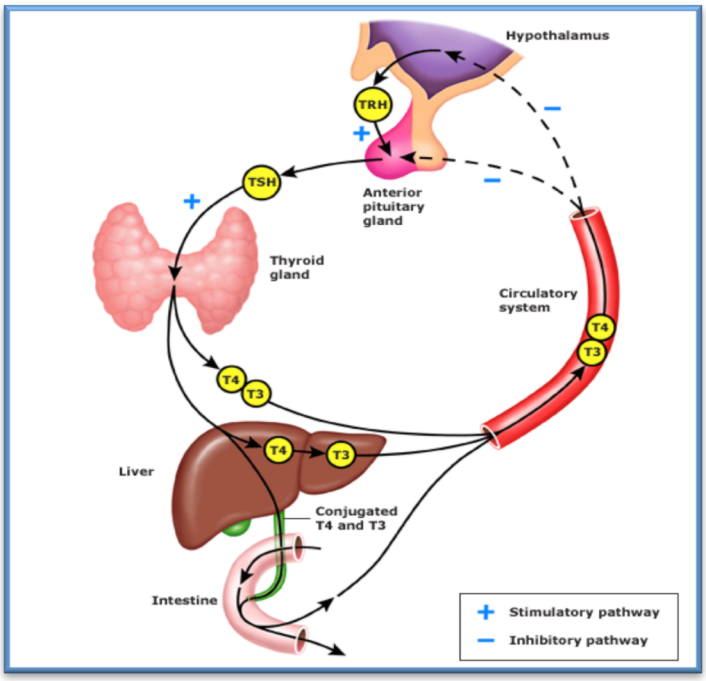
Colloid:
Central colloid-filled lumen. (Without any cells and rich in iodine and thyroglobulin & it's also the place of iodination)

Function of cells:
Follicular cells (*Principal cells*): Synthesis of thyroid hormones (T3 and T4)
Para-follicular cells (*Clear cells or C cells*): secretion of calcitonin

Thyroid hormone:

Some important facts:

1. Follicular cells of the thyroid are the main sites of hormone synthesis.
2. Iodine is needed to produce the thyroid hormones. The average requirement of iodine is 150 mcg a day for adults, 220 mcg for pregnant women, and 290 mcg for lactating women. Iodine sources include: dairy products, seafood, and table salt.
3. 99.9% of T4 and T3 are bound to protein in the blood: TBG (thyroxine binding globulin), albumin, and lipoprotein.
4. T4 and T3 synthesis and secretion is regulated by pituitary and thyroid stimulating hormone (TSH).
5. TSH is inhibited by T4 and T3 (through the negative feedback mechanism) and is stimulated by the thyroid releasing hormone (TRH, produced by the hypothalamus)
6. The main action of the thyroid hormone is by **T3**, however the gland mainly synthesizes T4, which is then converted into T3. (80% of T3 is derived from peripheral conversion and only 20% is produced by the thyroid gland itself)
7. Extra-thyroidal conversion of T4 to T3 is regulated by nutrition, illness and other hormonal factors.
8. Thyroid hormones are important for somatic development in adults and brain development in infants. (**Development**)
9. Maternal T4 reaches the fetus during development.
10. Fetal thyroid functions at 10 -12 weeks of gestation
11. If a mother has hypothyroidism → pre-term delivery, miscarriage, and cognitive impairment of infant.



Action of the thyroid hormone:

1. It acts on the bone and bone development. (**Growth**)
   - In children impaired levels of thyroid hormone → delayed growth and delayed epiphyseal growth. On the brain→ cognitive impairment
2. Acts on the cardiac muscle → Tachycardia or bradycardia.
3. Regulates metabolic rates and little changes in bodyweight.
4. Affects the reproductive system, as normal thyroid hormone levels are needed for the normal function of the reproductive system. Hypothyroidism is specifically associated with infertility.

Evaluation of the Thyroid Function:

- TSH (The one that is usually measured)
- Free T4 and free T3

} These are the tests done for diagnostic reasons

- TRH → Mostly done for the purpose of research
- TBG (thyroxin binding globulin)
- Thyroid antibodies: Microsomal antibodies and TSH receptor antibodies (present with Graves Disease) and thyroglobulin antibodies and anti-thyroid peroxidase (TPO antibodies) (Hashimoto's Disease)

Radiological Imaging of Thyroid Function:

- Ultrasound of the neck
  +
- Radioactive uptake scan

} (Best modalities)

- CT scan of the neck if suspecting retrosternal goiter.

History and Physical examination:

History:

When facing a suspected case of thyroid disease, make sure you ask about the following topics:

1. Exposure to ionizing radiation. (Do you work in radiology labs or in areas that have radiation fields?)
2. Lithium carbonate (used by psychiatric patients)
3. Residence in areas of low dietary iodide
4. Iodine ingestion:
    a. Kelp: herbs full of iodine.
    b. Iodide containing cough preparations
    c. IV iodide-containing contrast media. (Full of iodine)
5. Family history
6. Immunological disorders including: Diabetes, rheumatoid disease, pernicious anemia, alopecia, vitiligo, myasthenia gravis and MEN 2A. (These were found to coexist with thyroid diseases)

> ***Note!***
> Excessive iodine intake can give you both (hypo & hyper)-thyroidism

Physical examination:

Geeky Medicine: https://www.youtube.com/watch?v=ziaYBkgEZNU

1. Observe the neck, especially as the patient swallows
2. Examine from the front, rotating the gland slightly with one thumb while palpating the other lobe with the other thumb
3. Examine from behind, using three fingers and the same technique
4. Determine the size of the thyroid lobes, consistency, and presence of nodules.

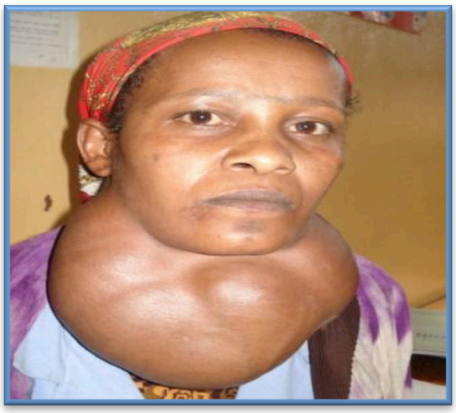
> ***To locate the thyroid***
> Touch the neck from top to bottom: Start at the hyoid bone, then the thyroid cartilage and finally the cricoid. The isthmus of the thyroid is just below the cricoid

So after taking history & PE, the next step is to determine the function (whether it's hyper or hypo), determine the size, and determine whether it's benign or malignant.

Goiter:

- **Goiter**: chronic enlargement of the thyroid gland.
- **Endemic Goiter**: common in China and Central Africa (or in regions where there lack of iodine). Defined as thyroid enlargement that occurs in more than 10% of the population. (Medscape)
- **Sporadic Goiter**: Multi-nodular goiter that is a result of environmental or genetic factors that don't affect the general population. (Medscape)
- **Familial**

When is goiter present?



- Hashimoto's thyroiditis: Early stage
- Graves' disease: due to chronic stimulation of TSH receptor
- Diet: Cabbage and cauliflower
- Chronic and excessive, iodine consumption
- Medication: lithium in 6%
- Neoplasms.

Non-Toxic Goiter: A non-toxic goiter is a diffuse or nodular enlargement of the thyroid gland that does not result from an inflammatory or neoplastic process and is not associated with abnormal thyroid function (Medscape)

Assessing goiter: Assess thyroid function by:

1. Measuring free T4 and T3
2. Measuring TSH
3. Ultrasound of the neck

Treatment:

1. Thyroxine suppression therapy: not used anymore.
2. **Surgery**:
   a) If pressure symptoms are present (e.g. dysphagia and dyspnea)
   b) Malignancy
   c) Lymphadenopathy
3. **Radioactive Iodine therapy** → help shrink it in size.

Hyperthyroidism & Thyrotoxicosis

Definitions

Differentiate between:

Thyrotoxicosis: A clinical syndrome that results when tissues are exposed to high levels of circulating thyroid hormone.

&

Hyperthyroidism: It's the hyperactivity of the thyroid gland itself.

(In other words, thyrotoxicosis can occur because of various causes other than hyperthyroidism, including: excessive intake of thyroid hormones, thyroiditis, and neoplasms)

Causes of thyrotoxicosis:

1. **Diffuse toxic goiter** (Graves' Disease)
2. Subacute Thyroiditis
3. Toxic Multinodular Goiter
4. Toxic adenoma (Plummer's disease)
5. Hyperthyroid phase of Hashimoto's thyroiditis
6. Thyrotoxicosis factitia
7. Rare: Struma ovarii*, metastatic thyroid carcinoma (follicular)*, hydatiform mole*, TSH secreting pituitary tumor, pituitary resistance to T3 and T4.

Note! Medscape

"Subacute thyroiditis is a self-limited thyroid condition associated with a triphasic clinical course of hyperthyroidism, hypothyroidism, and return to normal thyroid function"

* For your own knowledge (Medscape)

- **Struma ovarii** is ectopic thyroid tissue associated with dermoid tumors or ovarian teratoma that can secrete excessive amounts of thyroid hormone and produce thyrotoxicosis.

- Patients with a **molar hydatidiform** pregnancy or choriocarcinoma have extremely high levels of beta human chorionic gonadotropin (β-hCG), which can weakly activate the TSH receptor. At very high levels of β-hCG, activation of the TSH receptors is sufficient to cause thyrotoxicosis

- **Metastatic follicular thyroid carcinoma** may also result in thyrotoxicosis. These lesions maintain the ability to make thyroid hormone, and in patients with bulky tumors, production may be high enough to cause thyrotoxicosis.

Diffuse Toxic Goiter (Grave's disease)

- Most common form of thyrotoxicosis
- Females > Males
- Features:
    a. Thyrotoxicosis
    b. Goiter
    c. Orbitopathy (exophthalmos)
    d. Dermopathy (pretibial myxedema)

Etiology:

- Autoimmune disease of unknown cause
- There is a strong familial predisposition
- Peak incidence in the 20- to 40- year age group

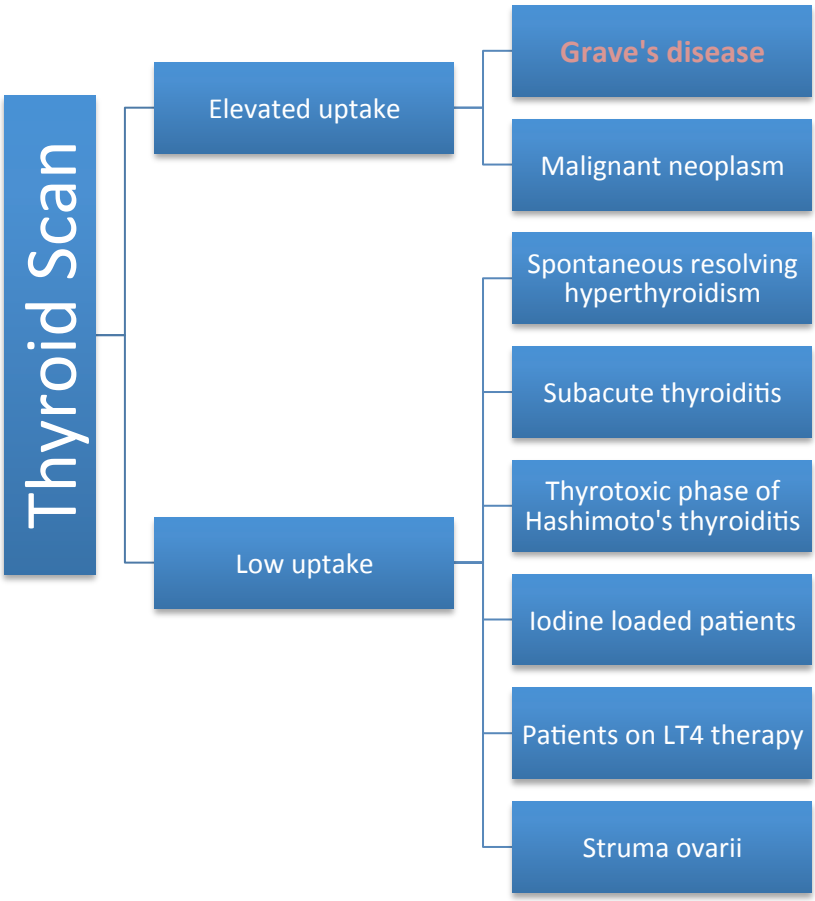
Pathogenesis:

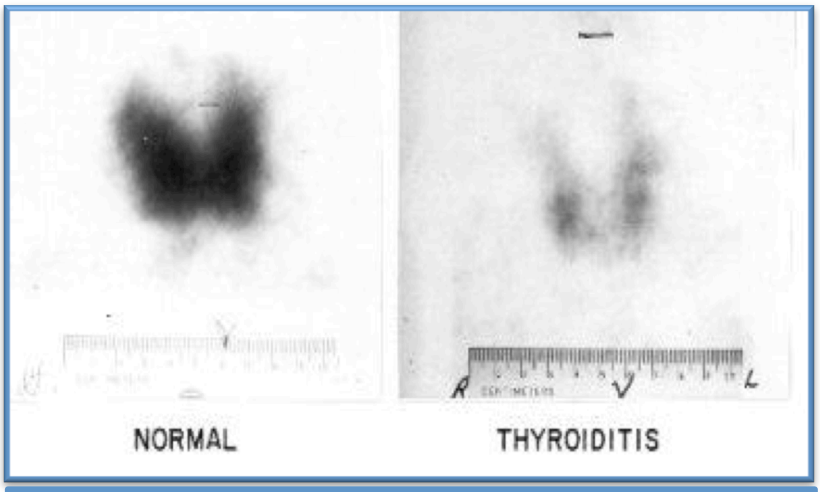
Local viral infection ➔ inflammatory reaction leading to production of IFN-g and other cytokines by non-thyroid specific infiltrating immune cells ➔ This will induce the expression of HLA class II molecules on the surface of thyroid follicular cells ➔ Subsequently, thyroid specific T-cells will recognize the antigen presented on the HLA class II molecules and will be activated. ➔ Activated thyroid-specific T-cells stimulate B cells to produce ➔ TSH receptor-stimulating antibodies ➔Hyperthyroidism

Diagnosis:

Elevated Free T4 & suppressed TSH *with* positive eye signs give you a certain diagnosis. But if there were elevated Free T4 levels & suppressed TSH levels *without* eye signs, we have to do the **thyroid scan**. (Radioiodine uptake scan – see next page).

Thyroid Scan:

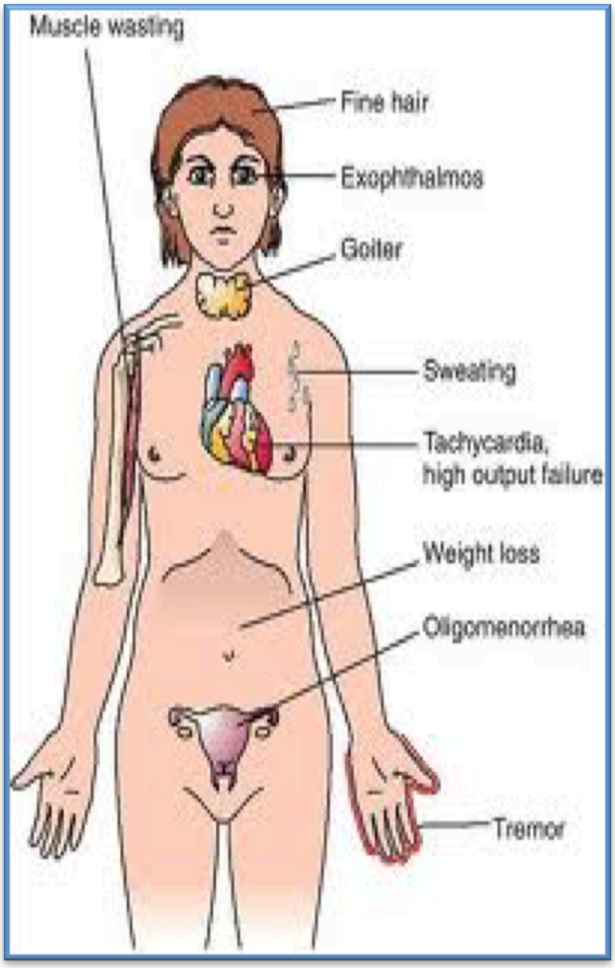




Notice how there's low uptake in the picture on the right.

Clinical features of thyrotoxicosis:

1. **Skin**: Warm and excessive sweating. (Heat intolerance)
2. Onycholysis (separation of the nail from it's bed – Talley) and hyperpigmentation.
3. Pruritis, vitiligo (loss of skin color), alopecia (hair loss), thinning of the hair.
4. Pretibial myxedema.
5. **Eyes**: Sympathetic over activity (common in Graves'). Extra-ocular muscles dysfunction: diplopia, lid lag, lid retraction*, exophthalmos, corneal ulceration*, optic neuropathy and blindness (due to optic nerve stretching – Talley).
6. **Cardiac**: Palpitations, atrial fibrillation in 10-20% of cases, high output cardiac failure, wide pulse pressure, and hypertension
7. **Respiratory**: Dyspnea
8. **Gastrointestinal**: Weight loss, diarrhea and increase liver enzymes
9. **Bone**: bone turnover increased → osteoporosis.
10. **Neuropsychiatry**: Behavioral and personality changes, tremors, irritability and depression.
11. Hyperactivity increases.



* lid retraction: appearance of the sclera above the iris

* corneal ulceration: due to inability to close the eyelids (all from Talley)

Note!

If exophthalmos and/or pretibial myxedema is seen, it's graves disease.

Keratopathy and ulcerations that occur in the eye secondary to hyperthyroidism are **irreversible**.

Complications of thyrotoxicosis:

Thyrotoxic crisis (thyroid storm): This happens when the patient is not treated. This hyper-metabolic state is induced by excessive release of thyroid hormones.

Clinical features include:

- Fever / Agitation
- Altered mental status
- Atrial fibrillation / Heart failure

Investigations:

Lab findings in hyperthyroidism

Increased T4	These results are expected in primary hyperthyroidism only. In Secondary hyperthyroidism even TSH will be elevated
Increased free T3	
Low TSH	
TSH receptor antibodies	
Increased radioactive uptake	

Treatment of hyperthyroidism: (Important)

1. Treat sympathetic over activity by giving **Beta blockers**.
2. **Radioactive Iodine (^{131}I)** (however it might cause irreversible hypothyroidism)
3. **Anti-thyroid drugs (*propylthiouracil* or *methimazole*)**
4. **Surgery**: as a last resort, or if there are obstructive symptoms or severe side effects to the treatment or patient refuses other methods of treatment. However, complications may occur. These include: hypothyroidism or hypoparathyroidism (accidental removal of the parathyroid glands). Also, the surgeon might accidentally damage the recurrent laryngeal nerve.

Treatment of certain conditions:

1. **Orbitopathy** ➔ Surgery. But, it doesn't have a "fully" reversible effect.
2. **Toxic adenoma** ➔ Start with radioactive iodine (best method). If it doesn't work, go for surgery.
3. **Subacute thyroiditis**: This is a disease caused by a virus. So, we don't give any medications related to the gland because there's no problem with it! We only treat the symptoms with B-Blockers, aspirin & analgesics.

Hypothyroidism:

Hypothyroidism is a common endocrine disorder resulting from deficiency of thyroid hormone. In areas of adequate iodine intake, autoimmune thyroid disease (Hashimoto disease) is the most common cause of hypothyroidism. Worldwide, iodine deficiency remains the foremost cause.

Causes

I. **Primary**:

1. *Hashimoto's thyroiditis*:
- With goiter

2."Idiopathic" thyroid atrophy, presumably end-stage autoimmune thyroid disease, following either Hashimoto's thyroiditis or Graves' disease

3. Neonatal hypothyroidism due to placental transmission of TSH-R blocking antibodies

4. Radioactive iodine therapy for Graves' disease

5. Subtotal thyroidectomy for Graves' disease or nodular goiter

6. Excessive iodine intake (kelp, radiocontrast dyes)

7. Subacute thyroiditis

8. Iodide deficiency

9. Medications such as lithium, amiodarone, PPIs, and antithyroid drug therapy

10. Inborn errors of thyroid hormone synthesis or congenital agenesis of the thyroid gland.

From Kumar

Table 18.11 Causes of hypothyroidism

PRIMARY DISEASE OF THYROID	**Infective**
Congenital	Post-subacute thyroiditis
Agenesis	**Post-surgery**
Ectopic thyroid remnants	**Post-irradiation**
Defects of hormone synthesis	Radioactive iodine therapy
Iodine deficiency	External neck irradiation
Dyshormonogenesis	**Infiltration**
Antithyroid drugs	Tumour
Other drugs (e.g. lithium, amiodarone, interferon)	**SECONDARY (TO HYPOTHALAMIC–PITUITARY DISEASE)**
Autoimmune	**Hypopituitarism**
Atrophic thyroiditis	Isolated TSH deficiency
Hashimoto's thyroiditis	**PERIPHERAL RESISTANCE TO THYROID HORMONE**
Postpartum thyroiditis	

II. **Secondary:**

Hypopituitarism due to:

a- Pituitary adenoma

b- Pituitary ablative therapy

c- Pituitary destruction

III. **Tertiary:**

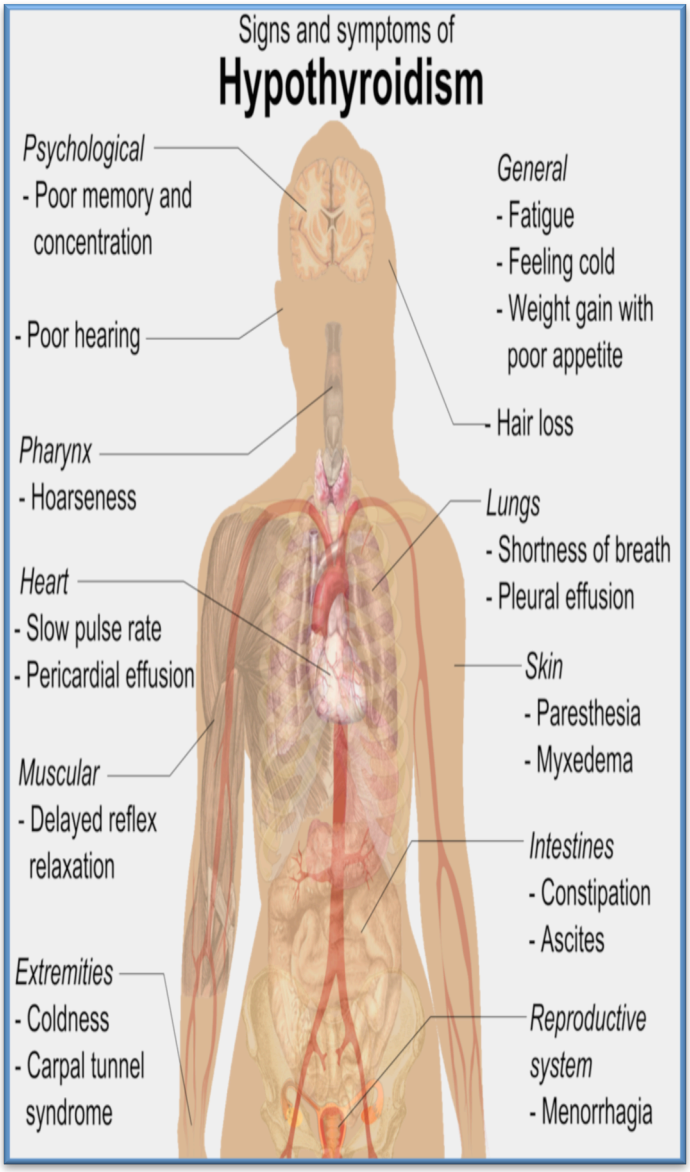
Hypothalamic dysfunction (rare).

IV. **Peripheral resistance of the action of thyroid hormone.**

Note!

- Amiodarone causes both hypo and hyperthyroidism.
- PPI inhibit the absorption of thyroxine
- Ca and Iron decrease hormone absorption

Clinical Features:



1. **Skin**: Cool, rough and dry skin. Puffy face and hands. Yellowish skin discoloration. Coldness.
2. **Edema**
3. Hoarse husky voice and slow reflexes
4. Loss or thinning of lateral third of eyebrows. (Known as Queen Anne's sign)
5. **Cardiac**: Bradycardia, decreased cardiac output, low voltage ECG (because of pericardial edema), cardiomegaly, pericardial effusion.
6. **Respiratory**: Shallow and slow respiration, respiratory failure.
7. **Gastrointestinal**: Weight gain, constipation and ileus (intestinal obstruction).
8. **Renal**: Impaired GFR that leads to water intoxication.
9. **Anemia**: Impaired hemoglobin synthesis, iron deficiency, folate deficiency, and pernicious anemia with B12 deficient megaloblastic anemia.
10. **Neuromuscular**: Severe muscle cramps, paresthesia, muscle weakness, and carpal tunnel syndrome (because of edema).
11. **CNS**: Chronic fatigue, lethargy, decreased concentration, anovulatory cycles and infertility, menorrhagia, depression and agitation.

Pathogenesis of edema

Accumulation of glycosaminoglycans-mostly **hyaluronic acid**- in interstitial tissues & increased capillary permeability to albumin will lead to interstitial edema (heart muscle, striated muscle)

Complications:

1- **Myxedema coma** (When a patient presents with hypothermia and hyponatremia, think of myxedema coma and don't wait for lab results. Ask about history and start treating the patient).

-The end stage of untreated hypothyroidism

-Progressive weakness, stupor, hypothermia, hypoventilation, hypoglycemia, hyponatremia, water intoxication, shock, and death.

-Associated illnesses and precipitating factors: pneumonia, MI, cerebral thrombosis, GI bleeding, ileus, excessive fluid administration, and administration of sedatives and narcotics.

-Three main issues: CO2 retention and hypoxia, fluid and electrolyte imbalance, and hypothermia.

2- **Myxedema** and **heart disease**

3- Hypothyroidism and neuropsychiatric disease

Investigations

Lab findings in hypothyroidism

Decreased T4	These results are expected in primary hyporthyroidism only. In Secondary hyporthyroidism even TSH will be decreased
Decreased free T3	
High TSH	
Positive TPO antibodies	
Low Na, Anemia, High cholesterol	

Treatment of hypothyroidism:

Levothyroxine (T4)

- Follow serum Free T4 and TSH.

- Morning dose

- Increase dose of T4 in malabsorptive states or concurrent administration of aluminum preparations, cholestyramine, calcium, or iron compounds

- Increase dose of T4 in pregnancy and lactation

- The t1/2 of levothyroxine is 7 days

Side effects:

No allergy has been reported to pure levothyroxine

-If FT4 and TSH are followed and T4 dose is adjusted, no side effects are reported

-If FT4 is higher than normal: hyperthyroidism symptoms may occur:

Including: cardiac symptoms, osteopenia and osteoporosis .

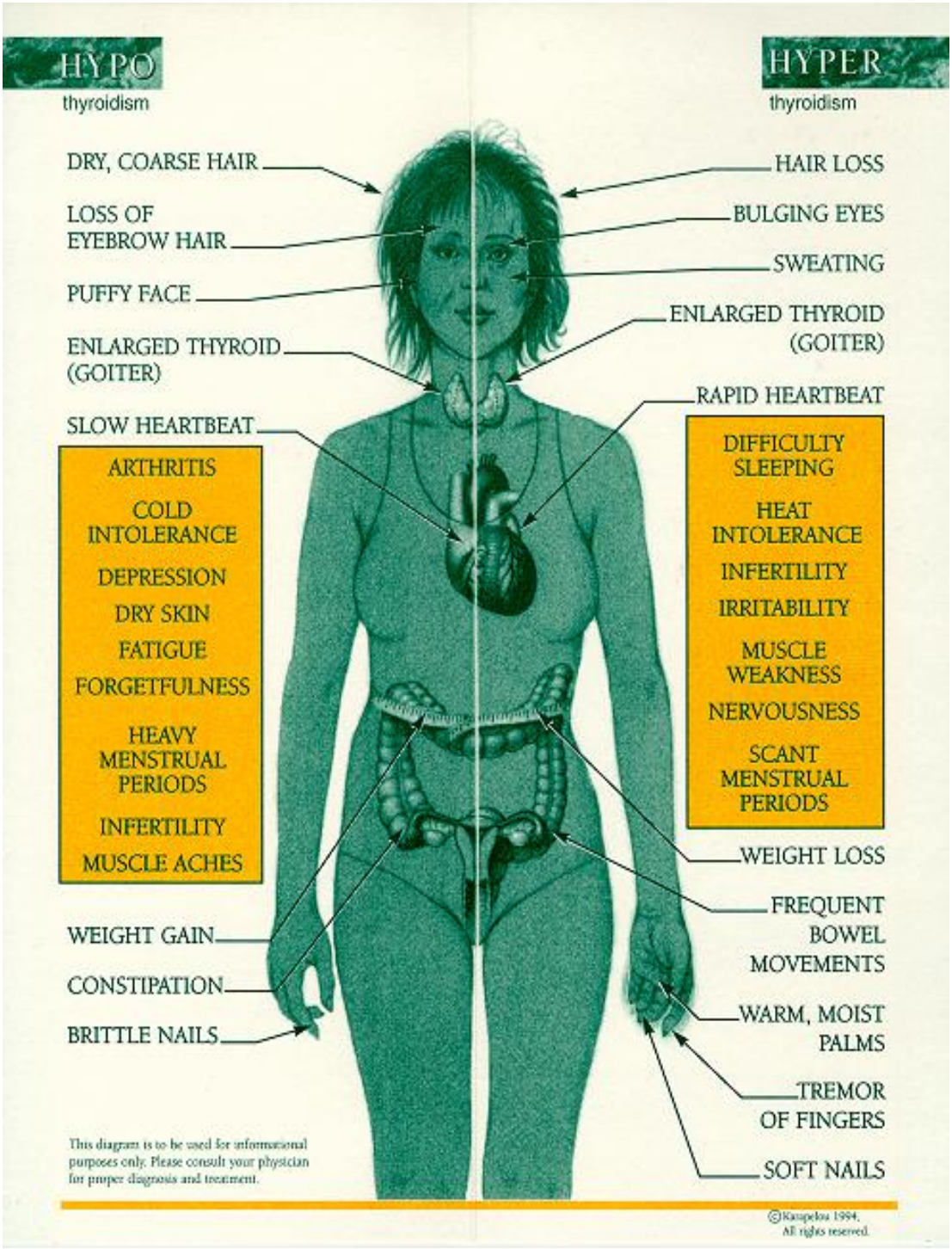
Treatment of certain conditions:

Myxedema coma

- Acute medical emergency
- Monitor blood gases
- Avoid excessive hydration
- Assess adrenal function and treat if needed
- In pituitary myxedema, glucocorticoid replacement is essential
- Be cautious in patients with coronary artery disease

Hypothyroidism	Large initial intravenous dose of 300 – 500 ug T4. If no response within 48 hours, add T3
Hypocortisolemia	Intravenous hydrocortisone 200-400 mg daily
Hypoventilation	Don't delay intubation and mechanical ventilation too long
Hypothermia	Blankets, no active rewarming
Hyponatremia	Mild fluid restriction
Hypotension	Cautious volume expansion with crystalloid or whole blood
Hypoglycemia	Glucose administration
Precipitating event	Identification and elimination by specific treatment (liberal use of antibiotics)

Summary


HYPO
thyroidism
HYPER
thyroidism
DRY, COARSE HAIR
LOSS OF EYEBROW HAIR
PUFFY FACE
ENLARGED THYROID (GOITER)
SLOW HEARTBEAT
ARTHRITIS
COLD INTOLERANCE
DEPRESSION
DRY SKIN
FATIGUE
FORGETFULNESS
HEAVY MENSTRUAL PERIODS
INFERTILITY
MUSCLE ACHES
WEIGHT GAIN
CONSTIPATION
BRITTLE NAILS
HAIR LOSS
BULGING EYES
SWEATING
ENLARGED THYROID (GOITER)
RAPID HEARTBEAT
DIFFICULTY SLEEPING
HEAT INTOLERANCE
INFERTILITY
IRRITABILITY
MUSCLE WEAKNESS
NERVOUSNESS
SCANT MENSTRUAL PERIODS
WEIGHT LOSS
FREQUENT BOWEL MOVEMENTS
WARM, MOIST PALMS
TREMOR OF FINGERS
SOFT NAILS
This diagram is to be used for informational purposes only. Please consult your physician for proper diagnosis and treatment.
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Approach to Hypothyroidism and Hyperthyroidism

Hypothyroidism	Hypercalcemia
DIFFERENTIAL DIAGNOSIS	
PRIMARY HYPOTHYROIDISM • **THYROIDITIS:** Hashimoto's, subacute, postpartum, irradiation • **IATROGENIC:** radioactive I131, thyroidectomy • **DRUGS** methimazole, propylthiouracil, iodide (kelp, radiocontrast dyes), lithium, amiodarone • **CONGENITAL:** thyroid agenesis, **thyroid dysgenesis** • **OTHERS:** iodine deficiency, idiopathic **SECONDARY HYPOTHYROIDISM:** diseases of the pituitary or hypothalamus (tumor, surgery, infarction, infection, infiltration, irradiation)	**PRIMARY HYPERTHYROIDISM** • **GRAVES' DISEASE:** (diffuse toxic goiter) most common cause of hyperthyroidism • **TOXIC NODULAR GOITER/TOXIC MULTINODULAR GOITER:** most common in elderly • **THYROIDITIS:** subacute thyroiditis, silent thyroiditis, Hashimoto's thyroiditis (''Hashitoxicosis ''), postpartum thyroiditis, radiation induced thyroiditis, drug induced thyroiditis (lithium, amiodarone, interferon) • **IODINE EXPOSURE:** kelp, seaweed, radio contrast dye • **EXOGENOUS:** L thyroxine ingestion, hamburger thyrotoxicosis • **ECTOPIC:** Struma ovarii (thyroid tissue present in an ovarian tumor), hydatiform mole (b hCG similar to TSH) **SECONDARY HYPERTHYROIDISM:** pituitary adenoma
CLINICAL FEATURES	
HISTORY: fatigue, dry skin, cold intolerance, depression, confusion, memory loss, goiter, constipation, weakness, carpel tunnel syndrome, menorrhagia, amenorrhea, weight gain, medications, family history of thyroid disease **PHYSICAL:** bradycardia, bradypnea, diastolic hypertension, hypothermia, cool and dry skin, vitiligo, orange skin (from carotonemia), carpel tunnel syndrome, hair thinning, periorbital edema, anemia, goiter, pleural effusion, pericardial effusion, proximal myopathy, pseudo myotonia, delayed relaxation phase of the reflexes, edema (non pitting)	**HISTORY:** fatigue, sweating, heat intolerance, psychosis, agitation, confusion, anxiety, goiter, dyspnea, palpitations, diarrhea, amenorrhea, weight loss, medications, family history **PHYSICAL:** vitals (tachycardia, atrial fibrillation, tachypnea, systolic hypertension, fever), systolic flow murmur, systolic pleuro pericardial scratch (Means Lerman scratch), thyroid acropachy (clubbing, Graves' only), onycholysis (Plummer's nails), palmar erythema, tremor, warm and moist skin ('velvet skin''), stare, exophthalmos (Graves' only), proximal myopathy, hyperreflexia, pretibial myxedema (Graves' only), splenomegaly

INVESTIGATIONS	
BASIC: LABS TSH **SPECIAL:** • ANTI TPO ANTIBODIES AND ANTITHYRO-GLOBULIN • ANTIBODIES Hashimoto's	**BASIC:** LABS TSH, fT4, fT3, TSH receptor antibody (Graves'), anti TPO antibody (Hashimoto's, Graves'), thyroglobulin (# if factitious), ESR (" in thyroiditis), CBCD, ALT, AST, ALP, bili **SPECIAL:** • THYROID SCAN • RADIOACTIVE IODINE UPTAKE
MANAGEMENT	
SYMPTOM CONTROL: MYXEDEMA COMA ABC, O2, IV. Levothyroxine 200 500 mg IV, then 100 mg IV daily. Hydrocortisone 100 mg IV q6h. Warming blankets. **TREAT UNDERLYING CAUSE**	**SYMPTOM CONTROL**: THYROID STORM ABC, O2, IV. Propylthiouracil 1000 mg PO/NG stat, then 300 mg PO q6h. Iodide drops 2 3 PO q6h to be given 1 h after each dose of PTU. Dexamethasone 2 mg IV q6h, propranolol 20 mg PO q6h. Cooling blankets **TREAT UNDERLYING CAUSE**

Questions

1- Thyrotoxicosis and uniformly increased radioactive iodine uptake in the thyroid can occur without any thyrotropin receptor antibodies or any thyroid autoimmunity in:

A. Graves' disease
B. Choriocarcinoma
C. Struma ovarii
D. Toxic multinodular goiter

2- Pretibial myxedema is associated with:

A. Graves' disease
B. Jod-Basedow phenomenon
C. Choriocarcinoma
D. Struma ovarii
E. Toxic multinodular goiter

3- Infiltration of orbital soft tissue and extraocular muscles with lymphocytes, mucopolysaccharides, and fluid is seen with:

A. Graves' disease
B. b.Jod-Basedow phenomenon
C. Choriocarcinoma
D. Struma ovarii
E. Toxic multinodular goiter

4- A low TSH, high T4, and high T3 suggests:

A. Hyperthyroidism
B. Nonthyroidal illness (sick euthyroidism)
C. Estrogen therapy
D. Subclinical hypothyroidism
E. Familial (euthyroid) dysalbuminenic hyperthyroxinemia

5- A 40-year-old patient with a recent viral infection presents with a significantly tender gland, low radioiodine uptake, and signs and symptoms of thyrotoxicosis. This presentation is most likely:

A. Graves' disease
B. Subacute thyroiditis
C. Toxic multinodular goiter
D. Hashimoto's thyroiditis
E. Toxic adenoma

6- A 65-year-old man presents with signs and symptoms of thyrotoxicosis. His radioiodine scan and 24-h uptake show a patchy pattern but normal amount of radioiodine uptake. This presentation is most consistent with:

A. Graves' disease
B. Subacute thyroiditis
C. Toxic multinodular goiter
D. Hashimoto's thyroiditis
E. Toxic adenoma

7- A 30-year-old woman with thyrotoxicosis has a diffusely enlarged gland on palpation of the neck. Her thyroid scan and 24-h uptake show uniformity of uptake and an increased percentage uptake. This patient has:

A. Graves' disease
B. Subacute thyroiditis
C. Toxic multinodular goiter
D. Hashimoto's thyroiditis
E. Toxic adenoma

Answers:

1st Question: A

2nd Question: A

3rd Question: A

4th Question: A

5th Question: B

6th Question: C

7th Question: A