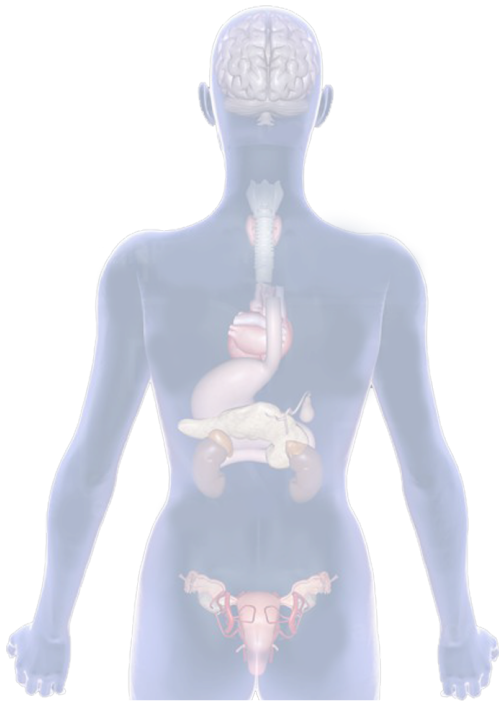
435 team

#6&7 Thyroid Gland & Hypo\Hyper Thyroidism

Objectives:

- LIST THYROID GLAND HORMONES.
- DESCRIBE THE SYNTHESIS OF THE THYROID HORMONES.
- DESCRIBE THE RELEASE AND ACTIONS OF THYROID HORMONES.
- DESCRIBE THE NEGATIVE FEEDBACK MECHANISM (CONTROL).
- DESCRIBE PATHOPHYSIOLOGY BEHIND THE CAUSES OF HYPER-HYPOTHYROIDISM.
- DESCRIBE PATHOPHYSIOLOGY BEHIND THE SIGNS AND SYMPTOMS OF THE HYPER-HYPOTHYROIDISM.
- LIST THE TREATMENT.



We recommend u to study histology of thyroid gland first.

Important
Male's notes
Female's notes
Extra

Resources: 435 male's & female's slides + guyton
Editing file: click Here

Revised by
خولة العماري & هشام الغفيلي

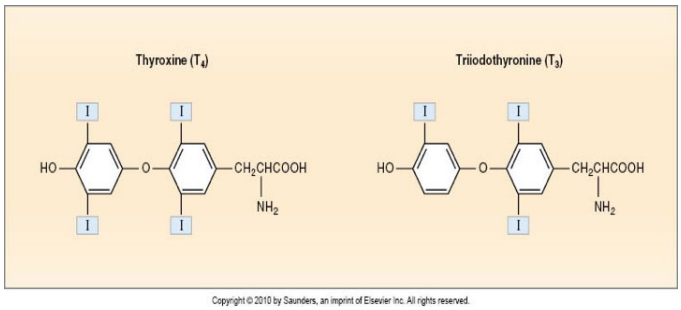
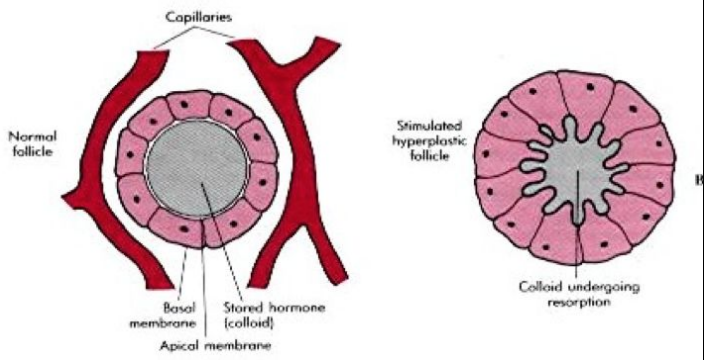
Overview of Thyroid Gland

Thyroid Gland - Thyroid Hormones (Duration: 12:46 min)HIGHLY RECOMMENDED

Located below the larynx on either sides and anterior to trachea.

The first recognized endocrine gland.

20g in adult.

Its Hormones	Synthesis sites
T4 (Tetraiodothyronine) (Thyroxine). 90% **T3 (Triiodothyronine). 10%** **Reverse T3**(inactive T3) **Calcitonin.**  	-follicular cells: T3 & T4 -Parafollicular cells: Calcitonin (there are follicles within the gland, the cells lining it are follicular cells, the ones adjacent to them are parafollicular cells) 

Unique Features of The Thyroid Gland: (3 factors)

The synthesis of thyroid hormones is more complex than that of most hormones. There are three unusual features of the synthetic process:

1- Contains a large amount of iodine. (supplied in diet; 1mg/week.) من السمك، او صاروا يحطونه بالملح

2- Synthesis is partially intracellular(inside the follicular cell) and partially extracellular(inside lumen of the follicular cell) .

3- T4 is the major product (Tetraiodothyronine).As noted, although T_4 is the major secretory product of the thyroid gland, it is not the most active form of the hormone. يعني بالعادة القلاند تفرز شي "اكثر شي اكتف" بس هنا زي ما نلاحظ اغلب افرازها شي اقل اكتف وهذه احد مميزات الثايروويد

Biosynthesis of Thyroid Hormones Done by follicular cells

1- Thyroglobulin formation & transport.
glycoprotein containing large quantities of ***tyrosine****, is synthesized on* the **rough endoplasmic reticulum**, and packaged into vesicles in the **Golgi apparatus**.

The **Thyroglobulin** then enter the lumen via exocytosis

2- Iodide pump or Iodide trap.
Iodide *is actively transported from blood into the follicular epithelial cells by* **Iodide pump**:(it's Active transport stimulated by TSH).
***Wolff-chaikoff effect** (a reduction in Thyroid hormone levels caused by administration of a large amount of iodine قبل كانو يعالجون بهالطريقه كأنها تسبب نيقتف فيدباك "وداوها بالتي كانت هي الداء).

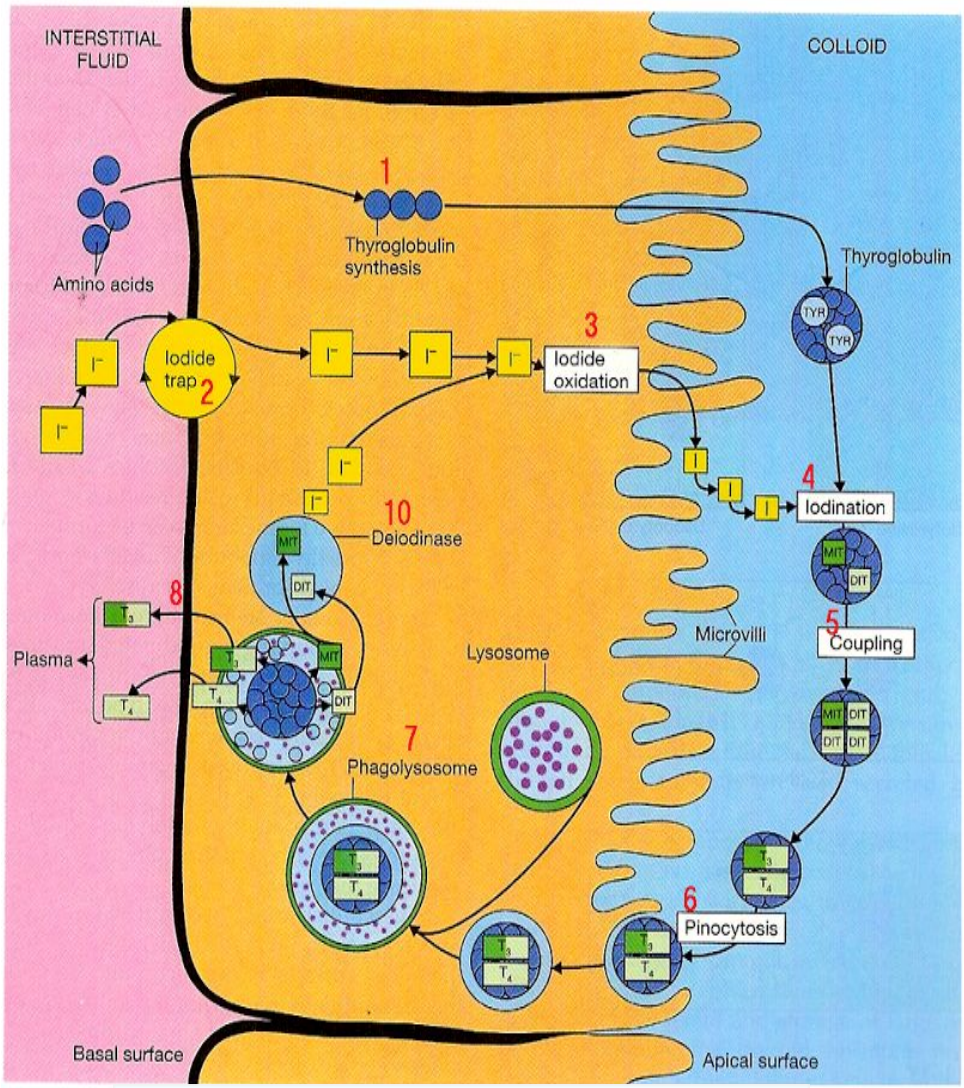
Wolff-chiakoff effect:
- High iodine in the blood = less uptake by the thyroid gland
and the vice versa
مثل لو كان فيه سلعة متوفرة بكثرة الإقبال عليها عادي أو قليل، لكن لو كانت بتقضي من السوق تشوف الأخذ لها عالي وكل الناس تتسابق عليها.

3- Oxidation of Iodide to Iodine.
By **Thyroid peroxidase** (It is located or attached to the apical membrane).

4- Iodination (Organification) of Thyroglobulin.
just inside the lumen, iodine *combines with thyroglobulin Then catalyzed by* ***thyroid peroxidase,*** *to form* ***monoiodotyrosine*** *(**MIT**) and* ***diiodotyrosine*** *(**DIT**). MIT and DIT remain attached to thyroglobulin in the follicular lumen until the thyroid gland is stimulated to secrete its hormones.*

5-coupling reaction.
DIT + DIT → T4 (faster)
2+2=4
DIT + MIT → T3
2+1=3
Done by Thyroid peroxidase>Then stored as colloid(sufficient for 2-3 months)عشان كذا الي عنده نقص مايبان عليه الا بعد ثلاث شهور

عشان كذا التيفور هو الأعلى كمية بالسكريشنز لأنه ينتج بسرعة :)



Propylthiouracil (**PTU**): A drug that blocks the production of **thyroid** hormone by the **thyroid** gland.
PTU is used to treat hyperthyroidism

6- Endocytosis of Thyroglobulin

7- Fusion of Lysosomes with vesicles.

8- Hydrolysis of the peptide bond
To release DIT + MIT + T4 + T3 from the thyroglobulin.

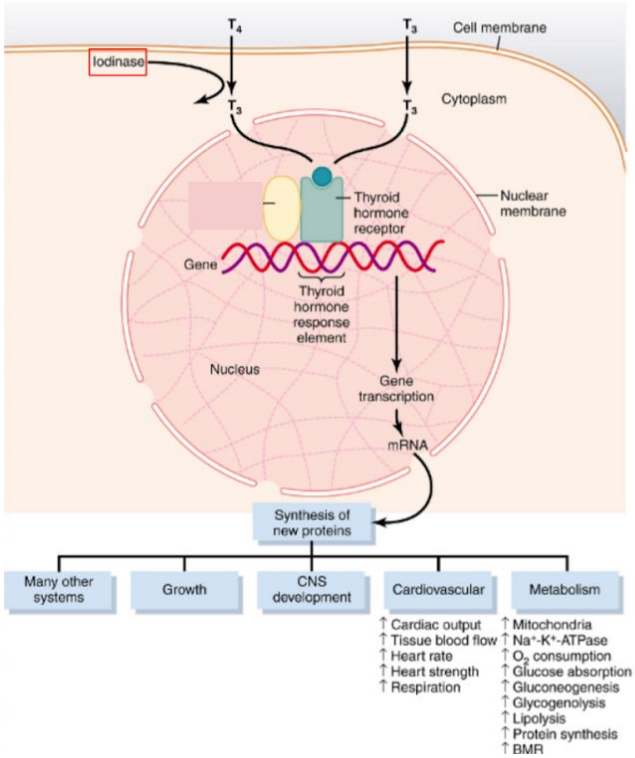
9- Delivery of T4 & T3 to systemic circu.

10- Deiodination of DIT & MIT (recycling) By **Thyroid deiodinase**

Thyroid Hormones in the circulation

Bound	Unbound (free)
70-80% bound to **Thyroxine binding globulin (TBG)** which is synthesized in the liver. The remainder is bound to Albumin.	0.03% of T4 0.3% of T3(more, cuz it's the needed form) Free hormone it's the only type that triggers negative Feedback and it is the active form.
In liver disease	**In pregnancy**
Low TBG → increased Free T3/T4 → inhibits thyroid secretion(until normal plasma levels of free T3/T4 are achieved) في حال مالقا الثايروكسين بروتين يرتبط معه يصير مصيره انه يسبح حرا بالدم فنتيجه لذلك تزيد نسبة الثايروكسين بالدم فالجسم يحسب زيادة مستويات الثايروكسين بالدم ناتجه عن كثره افرازه (مادرا ان البلى من قله البروتين باوند)فيثبط افراز الثايروكسين من الثايروويد الى ان يرجع الى مستواه الطبيعي	High estrogen → high TBG → low Free T3/T4 → stimulation of thyroid secretion(until normal plasma levels of free T3/T4 are achieved). -further explanation: during pregnancy, a fall in free thyroid hormone concentration induced by increased TBG levels in the plasma causes a compensatory rise in TSH secretion, which in turn increases the production of free thyroid hormones until normal plasma levels of free hormone are achieved.

The effect of T3 & T4 at the cellular level



1- The release T4 & T3 to the Tissues is **slow** because of the high affinity to plasma proteins.

- غير مهم½ of T4 in blood is released every 6 days.
- غير مهم½ of T3 in blood is released every 1 day.

2- T4 & T3 readily diffuse through the cell membrane.

3- Stored in the targeted tissues (days to weeks)

4- Most of T4 is deionized to T3 by iodinase enzyme inside the target cell.(in other words Before binding to the nuclear receptors 90% of T4 is converted to T3.)

5- In the nucleus, T3 mainly binds to "thyroid hormone receptor" to influence transcription of genes.

Not imp[T3 + nuclear receptor → activation of thyroid regulating element on DNA → DNA transcription → formation of mRNA → translation of mRNA → specific **protein synthesis** → target tissue specific]

PHYSIOLOGIC ACTIONS OF THYROID HORMONES

Effects on Autonomic nervous system:

Produce the same action as catecholamines via β -adrenergic receptors including:

- increase BMR.your resting metabolic rate
- increase heat production.
- increase heart rate.
- increase stroke volume.

Therefore β -blocker (propranolol) is used in treatment of hyperthyroidism.

Effects on Metabolism

The thyroid hormones increase the metabolic activities of almost all the tissues of the body

The metabolic effects are due to the induction of *metabolic enzymes*

Carbohydrate Metabolism:

الثايروويد مستعد يسوي اي شي عشان يزيد مستوى الجلوكوز بالخليه:

1- increase glucose uptake by cells.
2- increase glycogenolysis.يكسر الجلايكوجين عشان يعطينا جلوكوز
3- increase gluconeogenesis.
4- increase Glucose absorption from the GIT.

Protein Metabolism:

overall effect is *catabolic* leading to decrease in muscle mass.

Fat Metabolism:

زي ماعارفين الثايروويد يحرق الدهون ,عشان كذا بعض الستات ياخذون ثايروكسين عشان يخسو , بس اوعكم تعملوها اللعب بالهرمون زي اللعب بالنار

1- increase lipolysis.
2- decrease plasma cholesterol by increase loss in feces.
3- increase oxidation of free fatty acids.

Effects on the GIT

Increase:

- Appetite,
- digestive juices secretion and
- G.I tract motility:

- excess secretion → diarrhea.
- lack of secretion → constipation.

Effect on bone

promote :

- Bone maturation
- Bone formation,
- ossification(fusion of bone plate).

Effect on respiration

- increase ventilation rate.
- increase dissociation of oxygen from Hb by increasing RBC 2,3-DPG[1]

Basal Metabolic Rate (BMR)

It is the energy requirement under the basal(resting) condition (mental and physical rest 12-18 hours after a meal).

- Complete lack of thyroid hormones → 40-50% decrease in BMR.
- Extreme increase of thyroid hormones → 60-100% increase in BMR.

[1] 2,3 Diphosphoglycerate: a highly anionic organic phosphate which is present in human red blood cells at about the same molar ratio as hemoglobin. It binds to deoxyhemoglobin but not the oxygenated form, therefore diminishing the oxygen affinity of hemoglobin.

Effects on Cardiovascular system

What are the effects of thyroid hormone on CVS:

الزبده انه يزيد انقباض عضلة القلب عشان يوصل الدم المؤكد للانسجه :)

- **increase heart rate and stroke volume** (increased contractility)

→ which raises the **cardiac output up to 60%** → The end result is increased delivery of oxygenated blood to the tissues.

- **decrease peripheral resistance** → The end result is increased delivery of oxygenated blood to the tissues. **why there is ↓ peripheral resistance??** Increased metabolism in the tissues causes more rapid utilization of oxygen than normal and release of greater than normal quantities of metabolic end products from the tissues. These effects cause vasodilation in most body tissues, thus increasing blood flow.

How do the thyroid hormones perform their effects on CVS: The increased contractility is partly direct and partly indirect:

1- **indirect:** Thyroid hormones → ↑activation of β -adrenergic receptors → potentiate the effect of catecholamines in the circulation.

2- **Direct** induction of: دايركت يشتغل على عضلة القلب والانزيمات تبعتها.

a) myocardial β -adrenergic receptors.
b) sarcoplasmic reticulum.
c) Ca+2 ATPase.
d) myosine

Effects on CNS

Perinatal Period: مهم جدا

Thyroid hormones are essential for **maturation of the CNS**.

- decrease of hormones secretion → irreversible mental retardation.
- Screening is necessary to introduce hormone replacement. لو عندك بيبي هادي وخامل طول الوقت مايصيح ولا حاجه هنا لازم لازم تشيكي على الثايرود وتلحقينه من بدري قبل مايصير له تخلف عقلي وتقزم.

In Adults:

الثايروويد هورمون يزيد سرعة التفكير العقلي يعني بحالة الهايبوثايرودزم يفكرون ببطء وحتى اجاباتهم متاخرة وبطيئة

- Increase in thyroid hormone secretion:

1-hyperexcitability.
2- irritability.

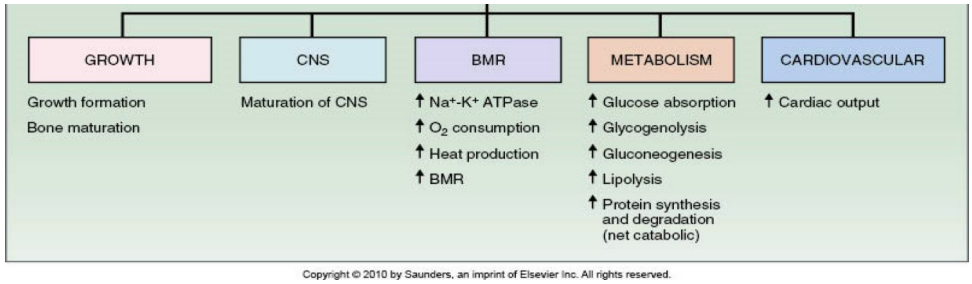
- Decrease in thyroid hormones secretion:

1- slow movement.
2- impaired memory.
3- decreased mental capacity.

Effect on Sexual Function

In men, lack of thyroid hormone is likely to cause loss of libido; In women, lack of thyroid hormone often causes *menorrhagia* and *polymenorrhea* (that is, respectively, excessive and frequent menstrual bleeding). Yet, strangely enough, in other women thyroid lack may cause irregular periods and occasionally even *amenorrhea*

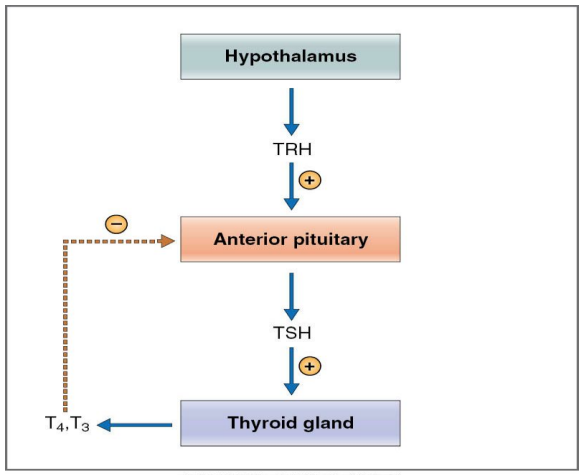
Summary:



Regulation Of Hormone Secretion

It is regulated by the hypothalamic-pituitary axis:

- Hypothalamus releases TRH which stimulates the anterior pituitary gland to release TSH which stimulates the release of T3 and T4 from the thyroid gland. T3 and T4 have a negative feedback effect on the anterior pituitary
- Some factors like environmental influence (temperature) affect the release of hypothalamic hormone TRH.



1- Thyrotropin-releasing hormone (TRH):

غير مهم it's Tripeptide. secreted from Paraventricular nuclei of the hypothalamus → Act on the thyrotrophs of the anterior pituitary to release TSH.

2- Thyroid-stimulating hormone (TSH):

غير مهم Glycoprotein secreted Anterior pituitary to Regulate metabolism, secretion and growth of thyroid gland (trophic effect).

- TSH secretion starts at 11-12 of gestational weeks. غير مهم
- TSH + receptor → activation of adenylyl cyclase via Gs protein → cAMP → activation of protein kinase → multiple phosphorylation → secretion and thyroid growth. غير مهم

- **Actions of TSH:** TSH tends to rapidly **increase** (within minutes or an hour) **ALL steps** in the synthesis and degradation of thyroid hormones, including:

1- Increase proteolysis of the thyroglobulin.
2- Increase pump activity.
3- Increase iodination of tyrosine.
4- Increase coupling reaction.
5- Trophic effect.

Table 9-8 Factors Affecting Thyroid Hormone Secretion

Stimulatory Factors	Inhibitory Factors
TSH	I^- deficiency
Thyroid-stimulating immunoglobulins	Deiodinase deficiency
Increased TBG levels (e.g., pregnancy)	Excessive I^- intake (Wolff-Chaikoff effect)
	Perchlorate; thiocyanate (inhibit Na^+-I^- cotransport)
	Propylthiouracil (inhibits peroxidase enzyme)
	Decreased TBG levels (e.g., liver disease)

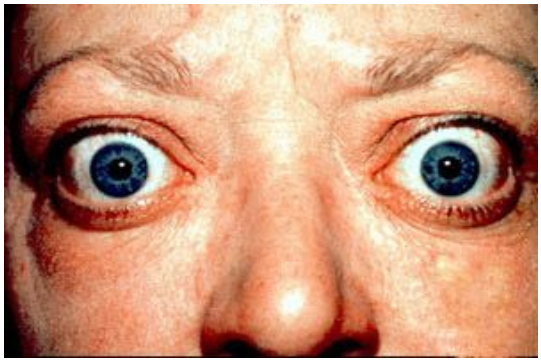
DISEASES OF THE THYROID GLAND

Hyperthyroidism vs Hyothyroidsm:

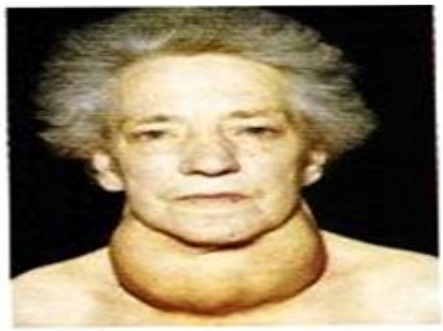
Disease	Hyperthyroidism	Hyothyroidsm
definition	its an over activity of the thyroid gland. • Women : men ratio (8:1). •activity of gland : what will happen ? a)- 5- 10 times increase in secretion. b)- 2-3 times increase in size.	Under activity of the thyroid gland more in woman (30- 60 years).
causes	**1-Graves' disease :** •an autoimmune disorder increased circulating level of thyroid- stimulating immunoglobulins[2] (TSI) 95%. •4 – 8 times more common in women than men. **2- Thyroid gland tumor :** ورم => تضخم => زيادة افراز •95% is benign. •5% is malignant. •history of head and neck irradiation and family history. **3- Exogenous T3 and T4**(rarely cause) يأخذ هرمونات **4- Excess TSH secretion:** could be due to: •diseases of the hypothalamus (TRH) •diseases of the pituitary (TSH).	**1- inherited abnormalities of thyroid hormone synthesis :** • peroxidase defect. • Iodide trapping defect. •thyroglobulin defect. **2- Endemic Colloid Goiter:** (before table salt) decrease iodide → decrease hormone formation→increase TSH→ increase Thyroglobulin → ↑ size (> 10 times) **3- Idiopathic Nontoxic Colloid Goiter:** • I intake is normal. •Could be due to thyroiditis???? inflammation→increase cell damage →decrease hormone secretion→ increase TSH →increase of activity of normal cells →increase size **4- Gland destruction (surgery).** **5- Pituitary diseases or tumor.** **6- Hypothalamus diseases or tumor.**

[2] The most common form of hyperthyroidism is **Graves disease**, an autoimmune disorder characterized by increased circulating levels of thyroid-stimulating immunoglobulins.
these immunoglobulins are anti- bodies to TSH receptors on thyroid follicular cells. **When present, the antibodies intensely stimulate the thyroid gland, resulting in increased secretion of thyroid hormones and hypertrophy of the gland.**

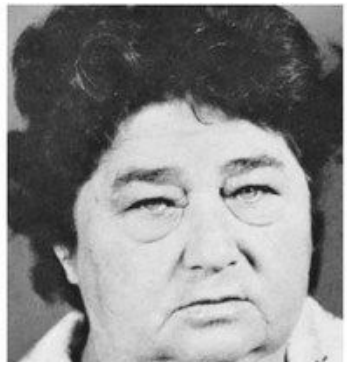
Disease	Hyperthyroidism	Hyothyroidsm
Symptoms مو كل شخص تصير له كل هالاعراض اذا لقيتي 2 او 3 لازم تشكي عشان كذا في كثير من الاحيان we missed the diagnosis	**Goiter:** A goiter is simply an enlarged thyroid and does not designate functional status. A goiter can be present in hypo-, and hyper- **ليش تجي مع الهايبو و الهايبر ماكنه تناقض؟؟؟؟** There is no correlation between thyroid size and function.مافهمتو لسا القاوتر شكل و الهايبر و الهايبو بالوظيفه مافي علاقه بينهم مثلا هل الطول له علاقه بالوظائف العقليه ؟لاااا الشكل ماله علاقه بالوظيفه **skin:** • smooth, warm and moist skin. • heat intolerance, night sweating.due to sympathetic effect. **musculo skeletal:** •Muscle atrophy. **Neurological:** due to excitability effect of thyroid hormone in CNS • tremor. • enhanced reflexes. • irritability **Cardiovascular:**due to up-regulation of β 1 receptors in the heart •increase heart rate and stroke volume. •arrhythmias. •hypertension mild cuz there's peripheral resistance. **G.I tract:** •weight loss due to the increased metabolic rate . •diarrhea. **Renal function:** •increase glomerular filtration rate. **Exophthalmos:**because of sympathetic overstimulation of the levator palpebrae superioris •anxious staring expression & protrusion of eye balls. **Others:** •menstrual cycle disturbance.	**skin:** •dry skin. •cold intolerance. **Musculo skeletal:** • muscle bulk. • muscle sluggishness(slowness) • slow relaxation after contraction **Neurological:** ^^ الحين كل واحد بيشك بنفسه • slow movement. • impaired memory. •decrease mental capacity. **Cardiovascular:** •decrease blood volume. • decrease heart rate • dcrease stroke volume. **G.I tract:** •constipation • increase weight. **Renal function:** •decrease glomerular filtration rate. **Myxoedema:** •An edematous appearance throughout body. **others:** • loss of libido. • menstrual cycle disturbance.
investigation	•**In primary hyperthyroidism:** المشكله من الغده high T3, T4 and low TSH . •**In secondary hyperthyroidism:**the problem from the upper axis high T3, T4 and high TSH.	•**In primary hypothyroidism:** T3,T4 are low and TSH is elevated •**In secondary hypothyroidism:** T3,T4 are low and TSH is low.
treatment	**1- Medical therapy:** e.g. propylthiourcal **2- Surgery:**Subtotal thyroidectomy. **- Indication for surgery:** a)Relapse after medical treatment. b)Drug intolerance. c)Cosmetic.تجميلي d)Suspected malignancy.	**1- Medical therapy:L- thyroxine** العلاج انا نعطيه الهرمون الي ناقصه The first response seen is the weight loss.



A wide-eyed, staring gaze



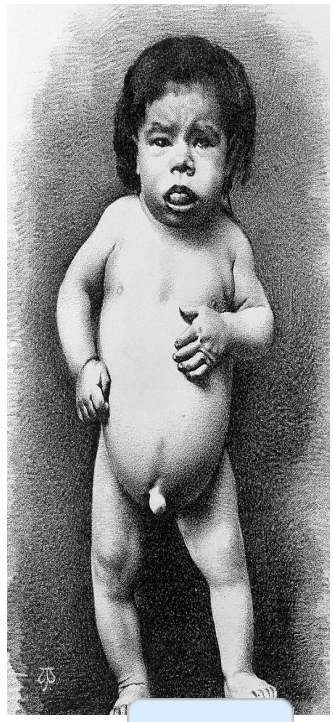
Goiter



Myxoedema

CRETINISM:

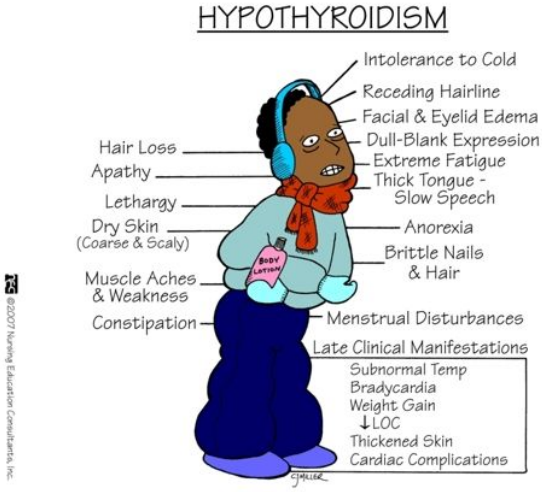
Definition	Extreme hypothyroidism during infancy and childhood (failure of growth).
Causes واحد منهم	1- Congenital lack of thyroid gland (congenital cretinism). 2- Genetic deficiency leading to failure to produce hormone. 3- Iodine lack in the diet (endemic cretinism).
Symptoms زي ماعرفنا دور الثايروید هورمون لنمو الجسم ونمو العقل و الظائف العقليه وتكوين العظام و انسجة الجسم فالي عنده نقص بتتأثر	1- Infant is normal at birth but abnormality appears within weeks. 2- Protruding tongue. 3- Dwarf with short limbs. 4- Mental retardation. 5- Often umbilical hernia. 6- abnormal teeth.
trearment	Changes are irreversible unless treatment is given early.

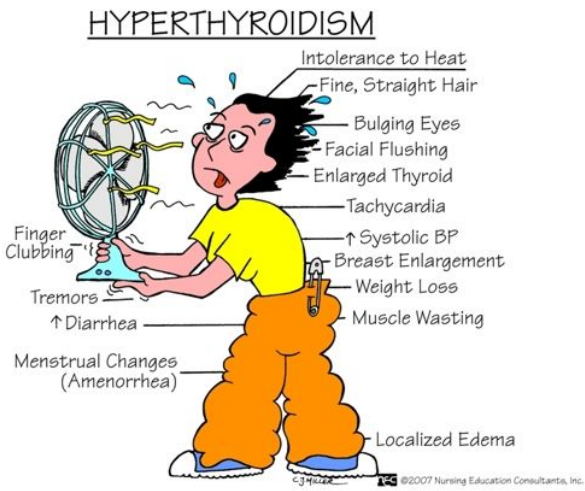


Cretinism

* in hypothyroidism there will be dwarfism and mental retardation , in GH deficiency there will be dwarfism with normal mentality.

EXTRA SKETCHES:



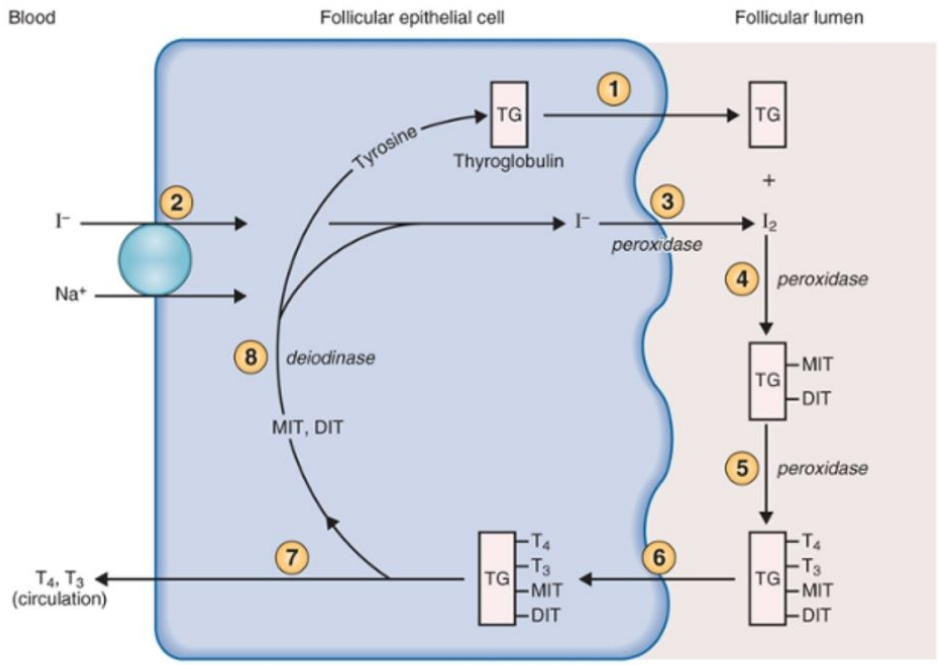


SUMMARY

Thyroid gland	
Hormones	-T3 -T4 -reversed T3(inactive T3) -Calcitonin
Synthesis site	-follicular cells: T3 & T4 -Parafollicular cells: Calcitonin
Features	1- Contains a large amount of iodine. (supplied in diet) 2- Synthesis is partially intracellular and partially extracellular. 3- T4 is the major product (Tetraiodothyronine).
Synthetic steps of its hormones	**1- Thyroglobulin formation & transport.**glycoprotein-Tyrosine-Rough endoplasmic reticulum & Golgi apparatus. **2- Iodide pump or Iodide trap.-**Active transport stimulated by TSH. - Wolff-chaikoff effect (a reduction in Thyroid hormone levels caused by administration of a large amount of iodine). **3- Oxidation of Iodide to Iodine:**By **Thyroid peroxidase** located or attached to the apical membrane. **4- Organification of Thyroglobulin:**(binding of iodine w/ Thyroglobulin). **Catalyzed by Thyroid peroxidase to form MIT/DIT** Remain attached to thyroglobulin until the gland is stimulated to secrete **5- Coupling Reaction:** **DIT + DIT → T4 (faster)** **DIT + MIT → T3** - catalyzed by Thyroid peroxidase then stored as colloid it sufficient for 2-3 months. **6- Endocytosis of Thyroglobulin.** **7- Fusion of Lysosomes with vesicles.** **8- Hydrolysis of the peptide bond:**To release DIT + MIT + T4 + T3 from the thyroglobulin. **9- Delivery of T4 & T3 to systemic circu.** **10- Deiodination of DIT & MIT (recycling)By Thyroid deiodinase**
Hormones in blood	**80%Bound:**-inactive-binds to thyroxin-boundable-globulin (made my liver)-small portion binds to albumen. **Free:**(small amount: T3)(smaller amount: T4)
Blood hormones in	**Liver failure:**Low TBG → increased Free T3/T4 → inhibits thyroid secretion **Pregnancy:**High estrogen → high TBG → low Free T3/T4 → stimulation of thyroid secretion.
Thyroids effects	**BMR:**Extreme increase of thyroid hormones → 60-100% increase in BMR. **Metabolics:** -Carbs: more glucose GIT absorbtion & cells uptake glycogenolysis & gluconeogenesis -fat: lipolysis & free fatty acids oxidization.Loosing cholesterol in feces (hypocholestrolemia) -Protein: very high catabolism (muscles atrophy) **CVS:**inc HR & SV (thus inc CO) (thus more blood to tissues)&dec perephral resistance. **Bone**:bone formation, maturation, ossification & plates fusion **GIT:** -inc appetite, GIT juices, motility -hyperthyroidism: dia. -hypothyrodism: constipation **CNS:** -in infants, its essential for CNS maturation so def in infants causes dwarfism & irreversible mental retardation. -adults hypertsm: hyperexcitability & irritability (disturbance) **Resp**:hyperventilation & inc RBCs 2,3-DPG faster O2 disassociation

SUMMARY

Steps involved in the synthesis of thyroid hormones in thyroid follicular cells: taken from costanzo book



Event	Site	Enzyme	Inhibitor
1 Synthesis of TG; extrusion into follicular lumen	Rough ER, Golgi apparatus		
2 Na^+ - I^- cotransport	Basal membrane		Perchlorate, thiocyanate
3 Oxidation of $I^- \rightarrow I_2$	Apical (luminal) membrane	Peroxidase	PTU
4 Organification of I_2 into MIT and DIT	Apical membrane	Peroxidase	PTU
5 Coupling reaction of MIT and DIT into T_3 and T_4	Apical membrane	Peroxidase	PTU
6 Endocytosis of TG	Apical membrane		
7 Hydrolysis of T_4 and T_3; T_4 and T_3 enter circulation	Lysosomes	Proteases	
8 Deiodination of residual MIT and DIT Recycling of I^- and tyrosine	Intracellular	Deiodinase	

SUMMARY

	Hyperthyroidism	Hypothyroidism
definition	•Over activity of the thyroid gland •Women:men ratio (8:1)	•Under activity of the thyroid gland •More in women (30-60 years)
Causes	1. Graves` disease (increased thyroid-stimulating immunoglobulins) 2. Thyroid neoplasm 3. Excess TSH secretion 4. Exogenous T3 or T4 (factitious)	1. Thyroiditis (autoimmune or Hashimoto's thyroiditis) 2. Surgery for hyperthyroidism 3. Iodine deficiency 4. Congenital (cretinism) 5. Decreased TRH or TSH
Symptoms	•Increased basal metabolic rate •Weight loss •Negative nitrogen balance •Increased heat production •Sweating •Increased cardiac output •Dyspnea •Tremor, muscle weakness •Exophthalmos •Goiter	•Decreased basal metabolic rate •Weight gain •Positive nitrogen balance •Decreased heat production •Cold sensitivity •Decreased cardiac output •Hypoventilation •Lethargy, mental slowness •Drooping eyelids •Myxedema •Growth retardation •Mental retardation (perinatal) •Goiter
Investigations	•**In primary hyperthyroidism:** high T3, T4 and low TSH . •**In secondary hyperthyroidism** high T3, T4 and high TSH.	•**In primary hypothyroidism:** T3,T4 are low and TSH is elevated •**In secondary hypothyroidism:** T3,T4 are low and TSH is low.
Treatment	1. Medical therapy:E.g. propylthiourcal 2. Surgery:Subtotal thyroidectomy	Thyroid hormone replacement therapy

CRETINISM	
Definition	Extreme hypothyroidism during infancy and childhood (failure of growth).
Causes	1- Congenital lack of thyroid gland (congenital cretinism). 2- Genetic deficiency leading to failure to produce hormone. 3- Iodine lack in the diet (endemic cretinism).
Symptoms	1- Infant is normal at birth but abnormality appears within weeks. 2- Protruding tongue. 3- Dwarf with short limbs. 4- Mental retardation. 5- Often umbilical hernia. 6- abnormal teeth.
trearment	Changes are irreversible unless treatment is given early.

MCQs

1. Thyroglobulin contains:
A. 140 Tyrosine
B. 140 Tryptophan
C. 130 Tyrosine

2. Organification of Thyroglobulin includes:
A. The oxidation of iodide
B. An active transport of iodide
C. Binding of iodine to Thyroglobulin

3. Oxidation of iodide to iodine:
A. Thyroid peroxidase
B. Thyroid oxidase
C. Thyroid deiodinase

4. Thyroid hormones are mostly circulating in the blood:
A. Bound to Albumin
B. Bound to TBG
C. Unbound

5. Sodium-iodide cotransport occurs at :
A. The apical membrane
B. Golgi apparatus
C. Basal membrane

6. In liver disease:
A. TBG will be decreased and free T3 + T4 will be increased
B. TBG will be decreased and free T3 + T4 will be normal
C. TBG will be increased and free T3 + T4 will be increased

7.Hypothyroidism in Infant called:
A. Myxoedema
B. Cretinism
C. Gull

8. which of the following is the first recognized endocrine gland:
A. Pancrease
B. Hypothalamus
C. Thyroid
D. Parathyroid

9.which of the following is not feature of thyroid gland:
A. Contains a large amount of iodine.
B. T3 is the major product.
C. Thyroxin is the major product

10.In pregnancy:
A. Decrease TBG , increase free TH
B. Increase TBG , decrease free TH
C. Increase TBG , normal free TH

11.Choose the correct answer regarding primary hyperthyroidism:
A. high T3, T4 and low TSH
B. High T3,T4 and TSH
C. Low T3,T4 and TSH
D. High TRH

12.Actions of Thyroid Hormone:
A. Increase glycogenolysis
B. Increase gluconeogenesis.
C. Increase lipolysis.
D. All of them

13.Which one of the following is stimulatory Factor Affecting Thyroid Hormone Secretion:
A. Iodide deficiency
B. Decreased TGB level
C. Excessive Iodide intake
D. None of them

Answer key:

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

Physiology 435 team

Thanks to this amazing team!

روان الضويحي
دانه فوزي
ريم العقيل
ريم البهلال
منيرة السلولي
مروج الحربي
لميس التميمي

عمر آل سليمان
عبد العزيز الحماد
محمد البشر

"Why do you call it a thyroid 'problem' when it's giving me an excuse for the 20 pounds I gained this year???"