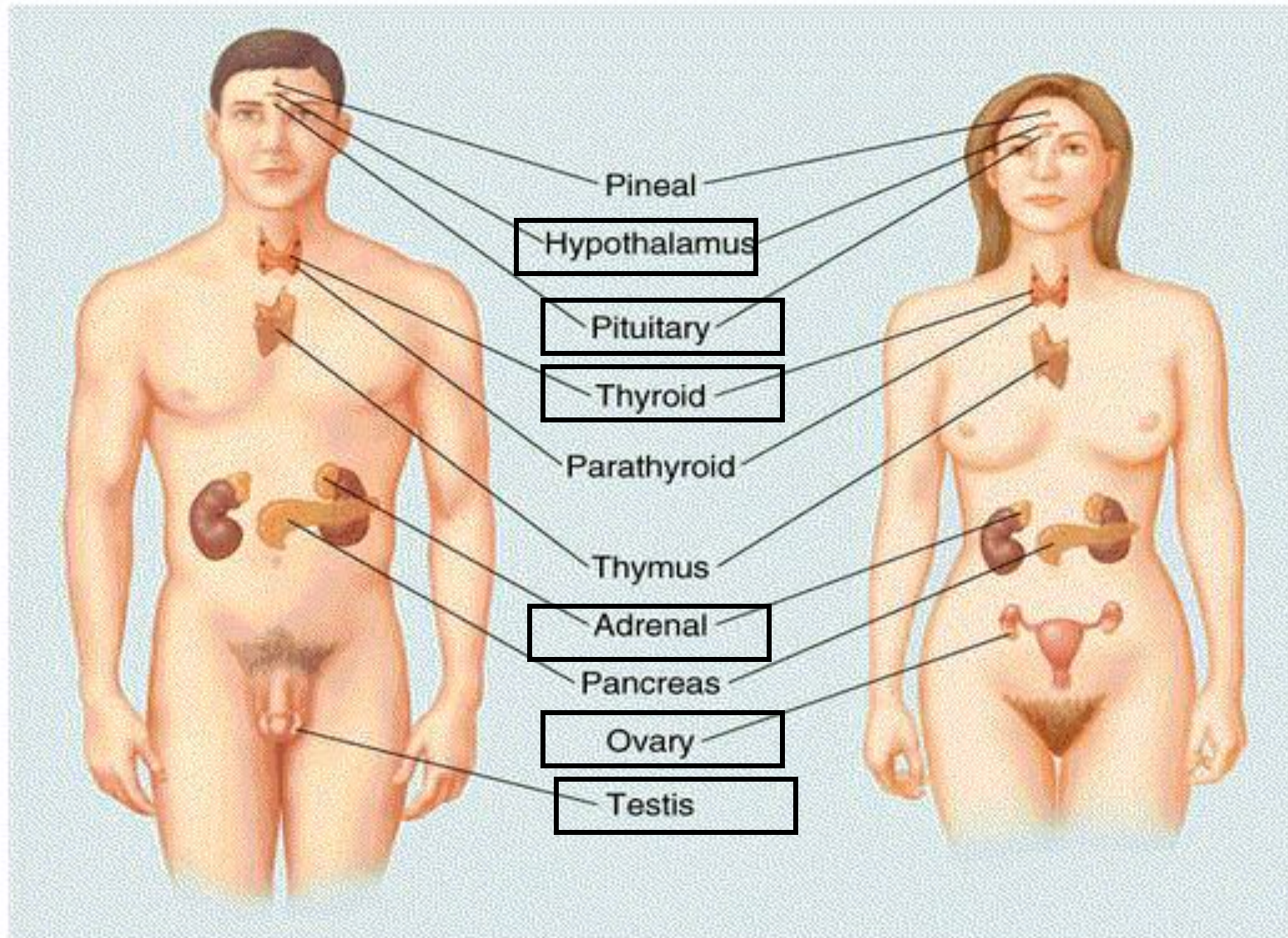


Karazin Kharkiv National University
Department of Pediatrics

Thyroid gland diseases

Assistant Tatyana Golovko

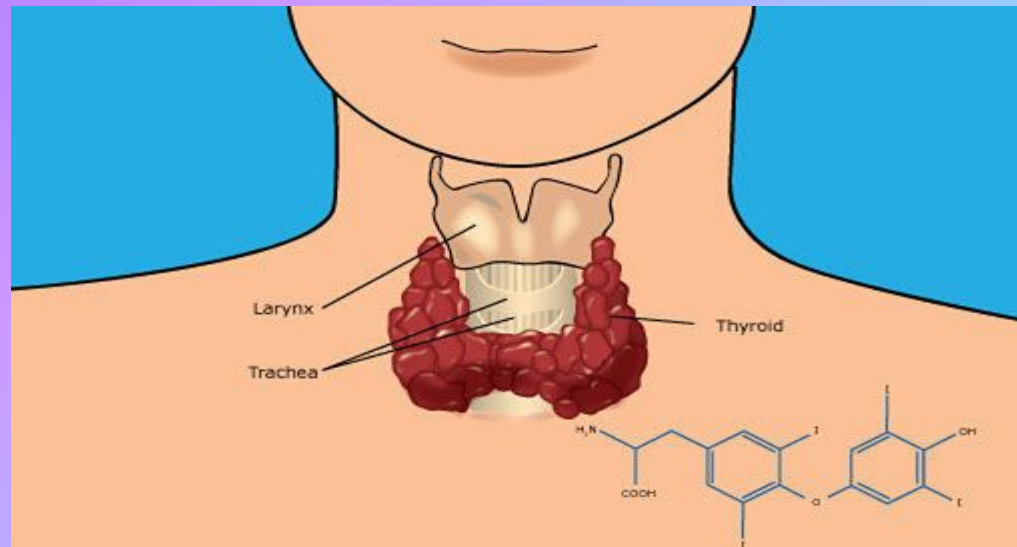
► Endocrine Glands



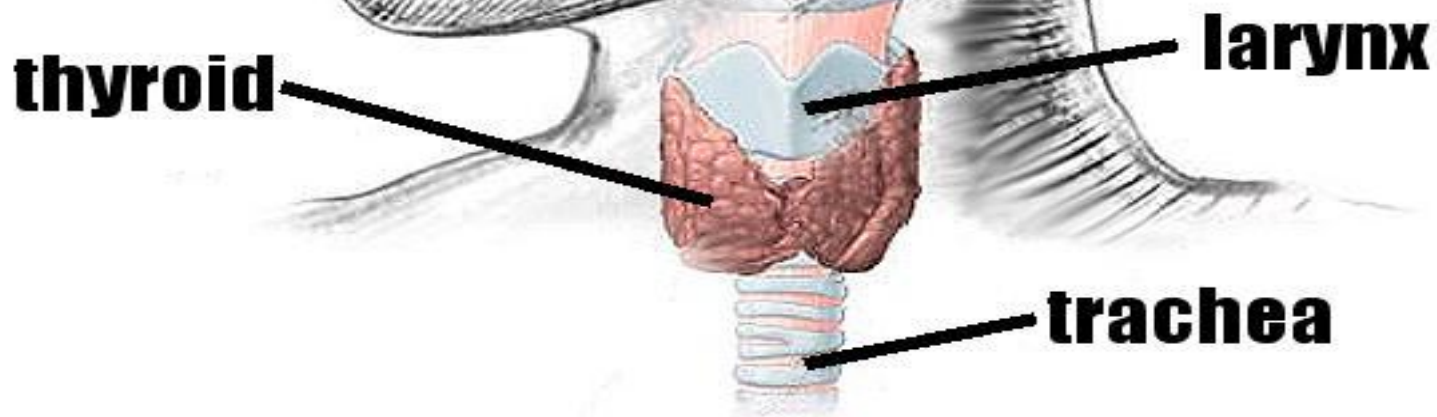
Introduction

Thyroid hormone is essential for the growth and maturation of many target tissues, including the brain and skeleton.

As a result, abnormalities of thyroid gland function in infancy and childhood is a result not only in the metabolic consequences of thyroid dysfunction seen in adult patients, but in unique effects on the growth and /or maturation of these thyroid hormone-dependent tissues as well.



The Thyroid Gland



Location: Located close to thyroid cartilage. Has two **lateral lobes** connected by **thyroid isthmus** medially. Weight range from **12 to 30g**

Development: Develops from endodermal floor of early pharynx

THYROID GLAND

Innervation: Vagus Nerve (X)

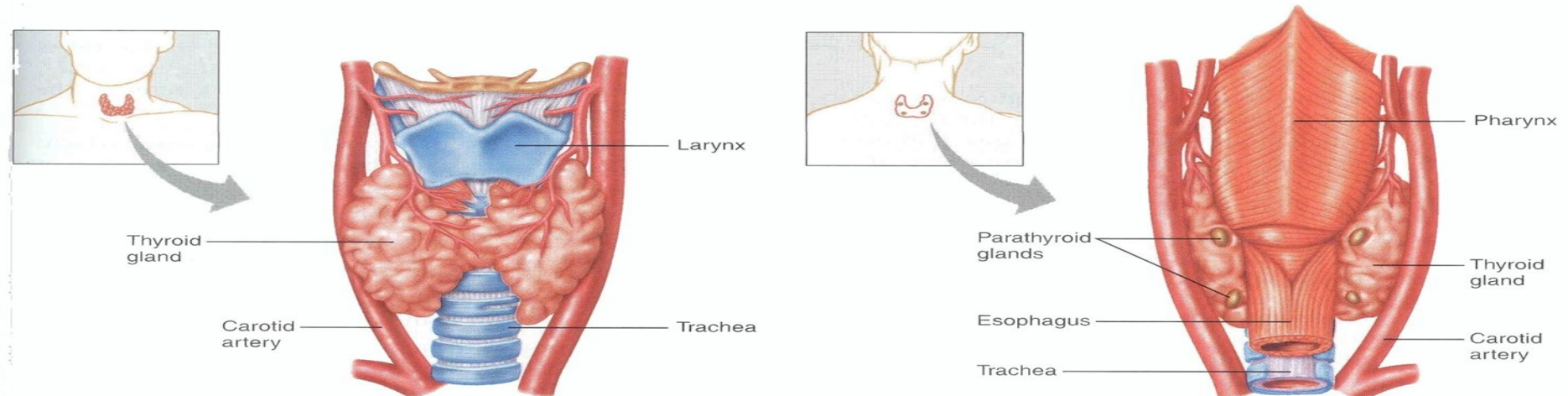
Arterial Supply: superior thyroid artery (branch of external carotid artery).

Produces: T_4 and T_3 (active hormone)

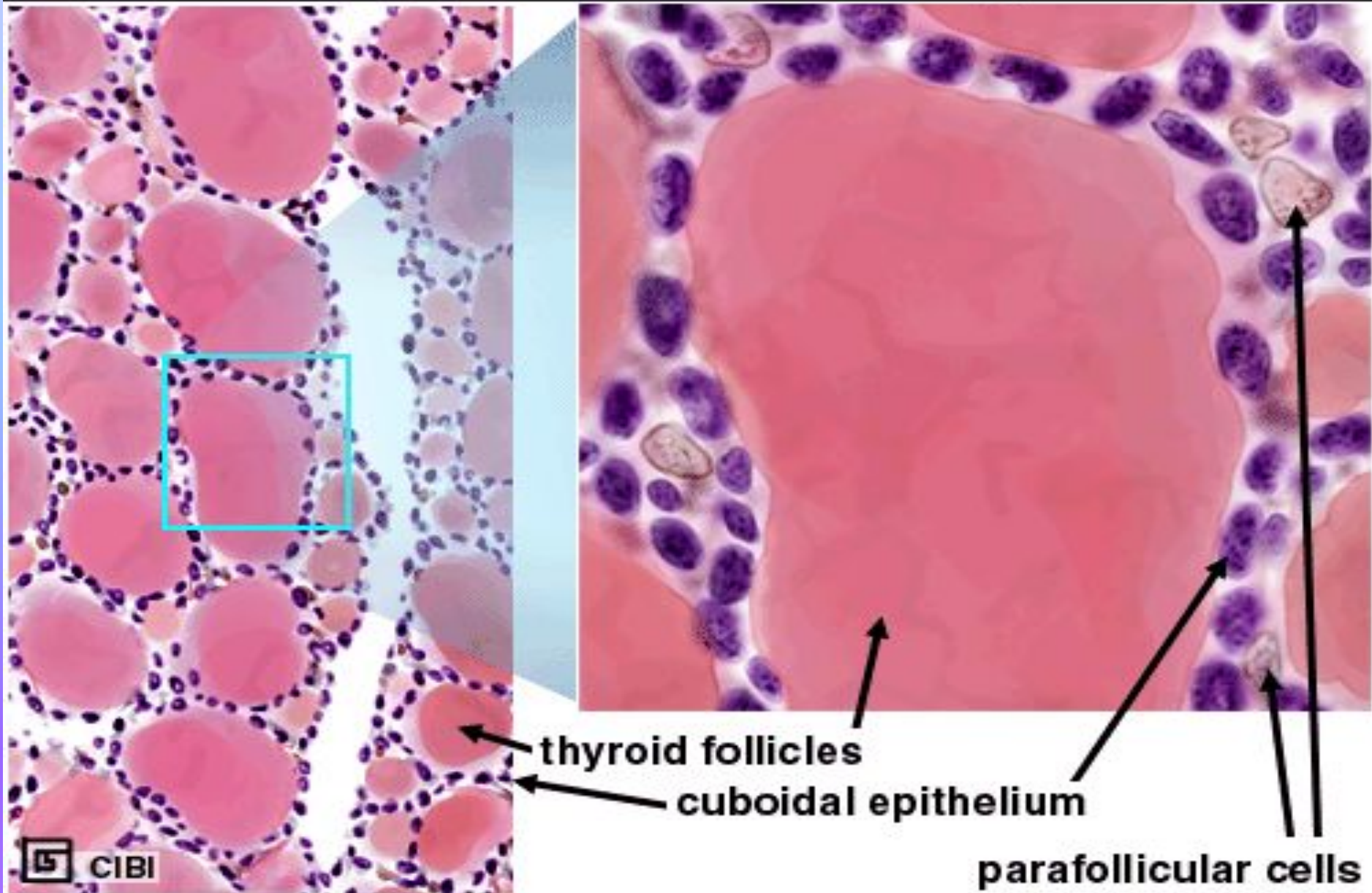
Regulation: “negative Feed-back” axis

Functions: **THYROXIN** – regulate rate of metabolism

CALCITONIN – decreases levels of calcium and phosphate in the blood (partially antagonistic to parathyroid hormone).



Thyroid Gland: Histology



Thyroid Physiology

THYROID GLAND

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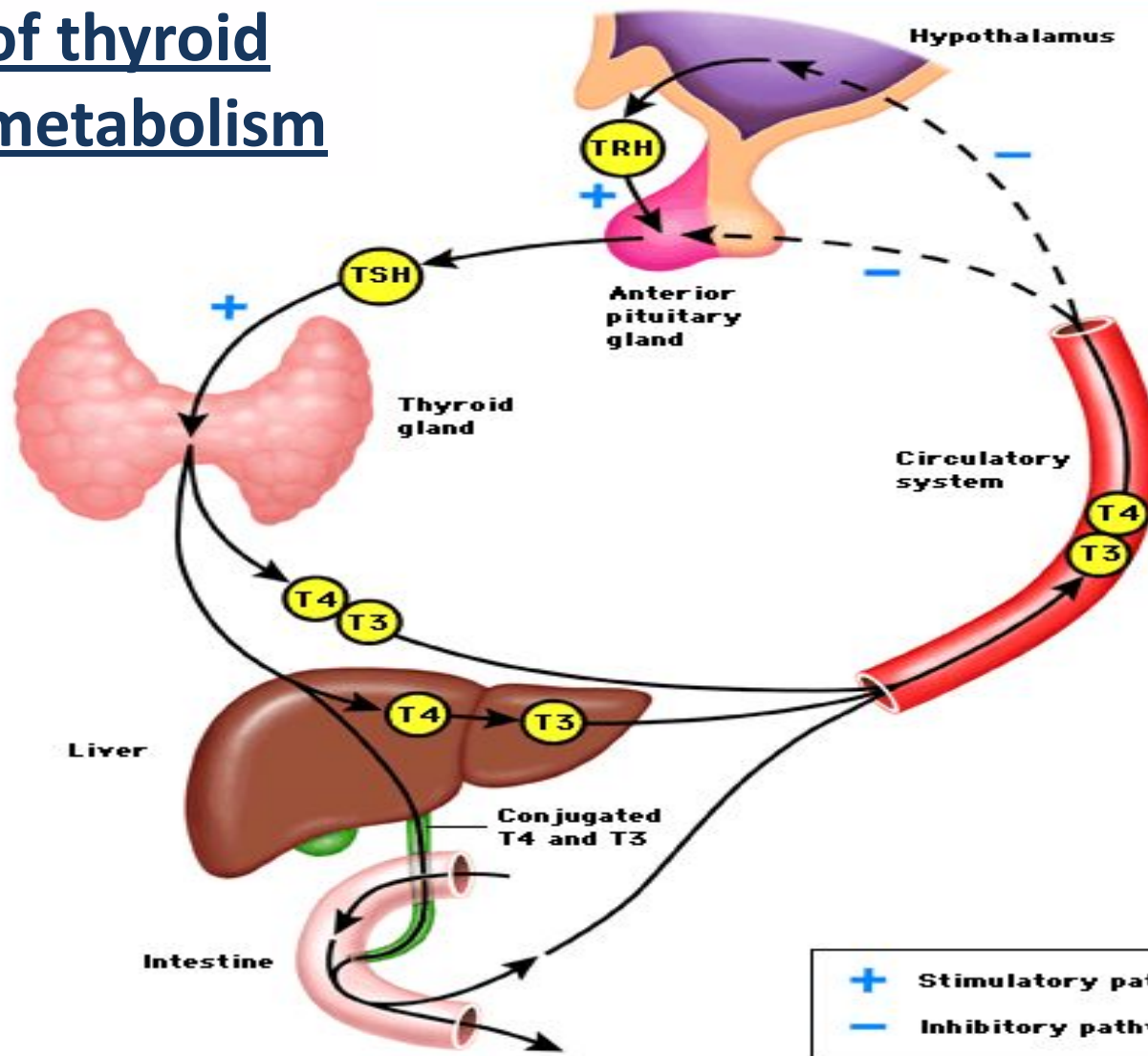
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Pathways of thyroid Hormone metabolism



Pathways of thyroid hormone metabolism Thyrotropin-releasing hormone (TRH) increases the secretion of thyrotropin (TSH), which stimulates the synthesis and secretion of triiodothyronine (T3) and thyroxine (T4) by the thyroid gland. T3 and T4 inhibit the secretion of TSH, both directly and indirectly by suppressing the release of TRH. T4 is converted to T3 in the liver and many other tissues by the action of T4 monodeiodinases. Some T4 and T3 is conjugated with glucuronide and sulfate in the liver, excreted in the bile, and partially hydrolyzed in the intestine. Some T4 and T3 formed in the intestine may be reabsorbed. Drug interactions may occur at any of these sites.

Feedback regulation Of TSH

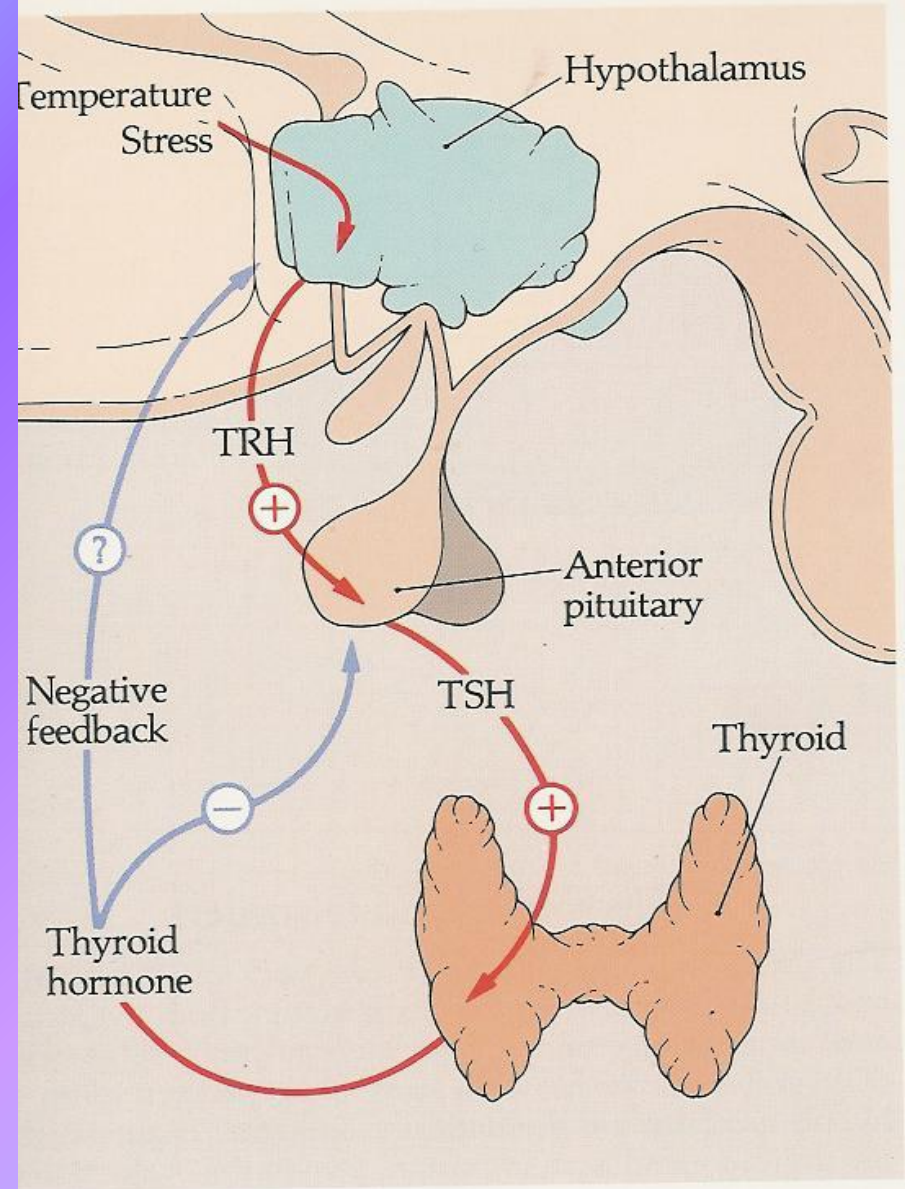
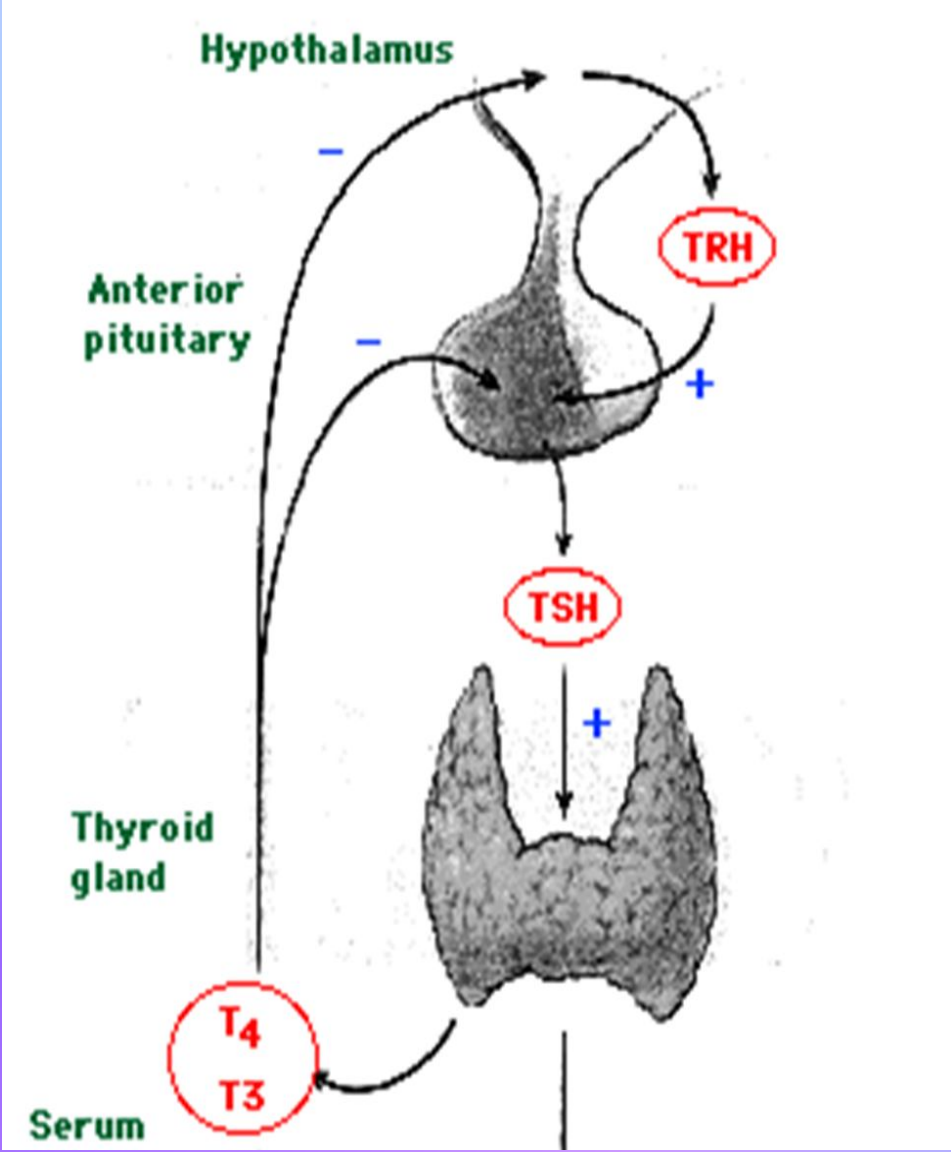


Fig. 9.3 Feedback regulation of thyroid-stimulating hormone (TSH) at the level of the hypothalamus, pituitary, and thyroid gland.

Thyroid physiology, continuation

THYROID GLAND

Innervation: Vagus Nerve (X)

Arterial Supply: superior thyroid artery (branch of external carotid artery).

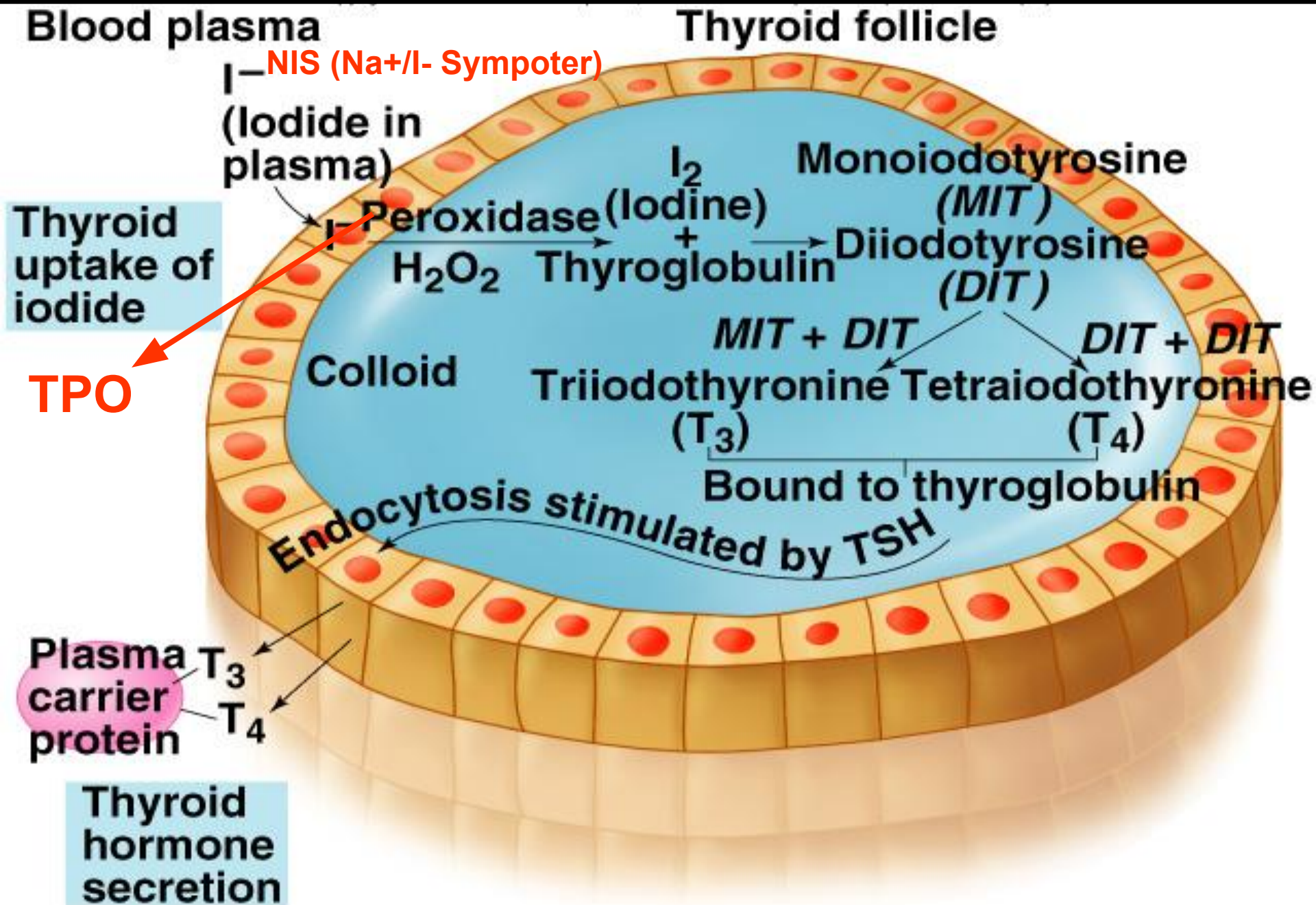
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Regulation: “negative Feed-back” axis

Functions: **THYROXIN** – regulate rate of metabolism

CALCITONIN – decreases levels of calcium and phosphate in the blood (partially antagonistic to parathyroid hormone).

Production of Thyroid Hormones



Thyroid Hormone

- Majority of circulating hormone is T_4 (98,5% T_4 , 1,5% T_3)
- Total Hormone load is influenced by serum binding proteins:
 - Albumin 15%;
 - Thyroid Binding Globulin 70%;
 - Transthyretin 10%.
- Regulation is based on the free component of thyroid hormone.

Effects of thyroid hormones

- Fetal brain and skeletal maturation;
- Increase in basal metabolic rate;
- Inotropic and chronotropic effects on heart;
- Stimulates gut motility;
- Increase bone growth;
- Increase in serum glucose, decrease in serum cholesterol;
- Play role in thermal regulation.

Thyroid hormones

THYROID GLAND

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Comparative analysis of thyroid hormones

	T_4	T_3
Potency	1	10
Protein Bound	10-20	1
Half-Life	5-7d	< 24h
Secreted by thyroid	100 ug/d	6 ug/d

THYROID GLAND DISORDERS DIVIDED INTO:

- **THYROTOXICOSIS** (Hyperthyroidism)
 - Overproduction of thyroid hormones;
- **HYPOTHYROIDISM** (Gland destruction)
 - Underproduction of thyroid hormones;
- **NEOPLASTIC PROCESSES**
 - Benign;
 - Malignant.

Diagnostic of Thyroid gland disease

THYROID GLAND

Innervation: Vagus Nerve (X)

Arterial Supply: superior thyroid artery (branch of external carotid artery).

Produces: T_4 and T_3 (active hormone)

Regulation: “negative Feed-back” axis

Functions: **THYROXIN** – regulate rate of metabolism

CALCITONIN – decreases levels of calcium and phosphate in the blood (partially antagonistic to parathyroid hormone).

LABORATORY EVALUATION

TSH (thyroid-stimulating hormone) normal, practically excludes abnormality

- ## THYROID GLAND

Innervation: Vagus Nerve (X)

Arterial Supply: superior thyroid artery (branch of external carotid artery).

Produces: T_4 and T_3 (active hormone)

Regulation: “negative Feed-back” axis

Functions: **THYROXIN** – regulate rate of metabolism

CALCITONIN – decreases levels of calcium and phosphate in the blood (partially antagonistic to parathyroid hormone).

- **High TSH usually means Hypothyroidism**

- Rare causes:

- TSH-secreting pituitary tumor;
 - Thyroid hormone resistance;
 - Assay artifact.

- **Low TSH usually indicates Thyrotoxicosis**

- Other causes:

- First trimester of pregnancy;
 - After treatment of hyperthyroidism;
 - Some medications (Esteroids-dopamine).

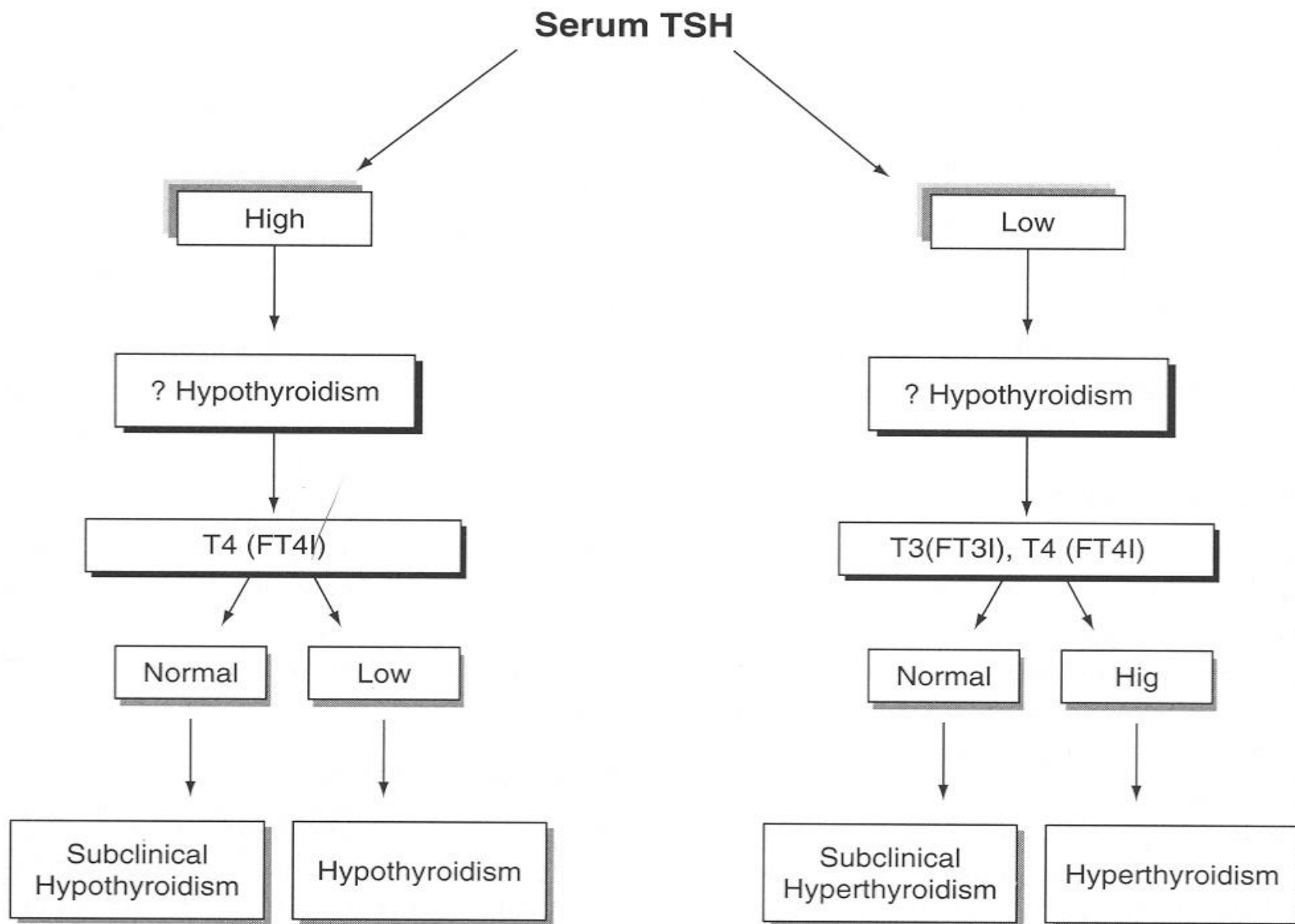
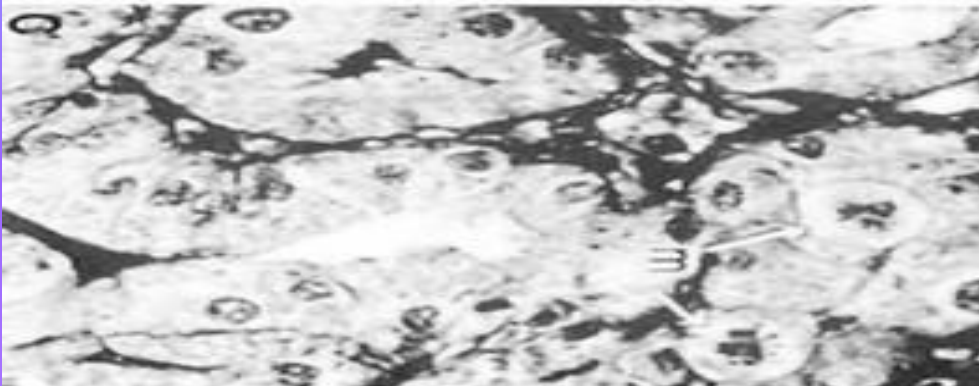
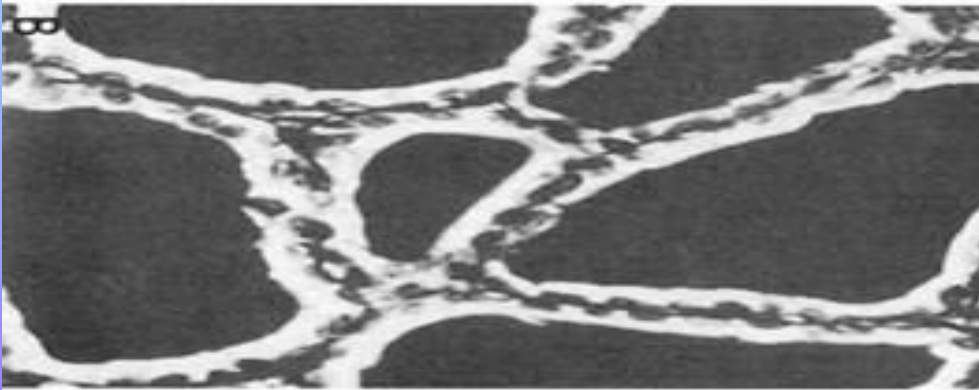
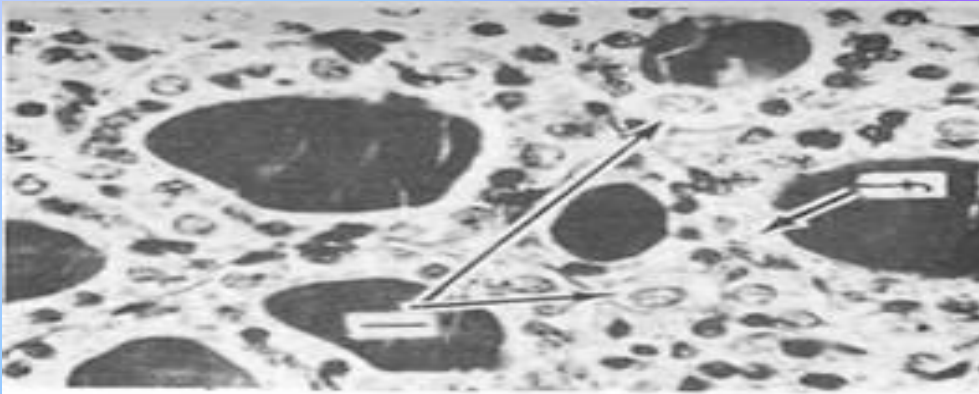


Figure 118-2. Algorithm for using the TSH level in the evaluation of thyroid function.

RAIU (Radioactive iodine uptake)

- Scintillation counter measures radioactivity after I^{123} or I^{131} administration (per os or IV). Radioactivity of TG measures between 4 h to 24 h. In children is limited in use.
- Uptake varies greatly by iodine status:
 - Indigenous diet (normal uptake 10% vs. 90%);
 - Amiodarone, Contrast study, Topical betadine.
- High RAIU with hyperthyroid symptoms
 - Graves’;
 - Toxic goitre.
- Low RAIU with hyperthyroid symptoms:
 - Thyroiditis (Subacute, Active Hashimoto’s);
 - Hormone ingestion (Thyrotoxicosis factitia, Hamburger Thyrotoxicosis);
 - Excess I^- intake in Graves’ (Jod-Basedow effect);
 - Ectopic thyroid carcinoma (Struma ovarii).

Iodine states



- Normal Thyroid
- Inactive Thyroid
- Hyperactive Thyroid

Thyrotoxicosis

- Primary;
- Secondary;
- Without Hyperthyroidism;
- Exogenous or factitious.

Hypothyroidism

- Primary;
- Secondary;
- Peripheral.

- **HYPERTHYROIDISM or THYROTOXICOSIS:**

- is the result of excessive thyroid gland function because is defined as the state of thyroid hormone excess.

- **GOITRE**

- is a chronic enlargement of the thyroid gland, that is not due to malignant neoplasm.

Causes of Thyrotoxicosis:

– Primary Hyperthyroidism:

- Graves';
- Toxic Multinodular Goitre;
- Toxic adenoma;
- Functioning thyroid carcinoma metastases;
- Activating mutation of TSH receptor;
- Struma ovarii;
- Drugs: Iodine excess.

Causes of Thyrotoxicosis:

– Thyrotoxicosis without hyperthyroidism:

- Subacute thyroiditis;
- Silent thyroiditis;
- Other causes of thyroid destruction:
 - Amiodarone, radiation, infarction of an adenoma;
- Exogenous/Factitious.

– Secondary Hyperthyroidism:

- TSH-secreting pituitary adenoma;
- Thyroid hormone resistance syndrome;
- Chorionic Gonadotropin-secreting tumor;
- Gestational thyrotoxicosis.

Symptoms of Hyperthyroidism

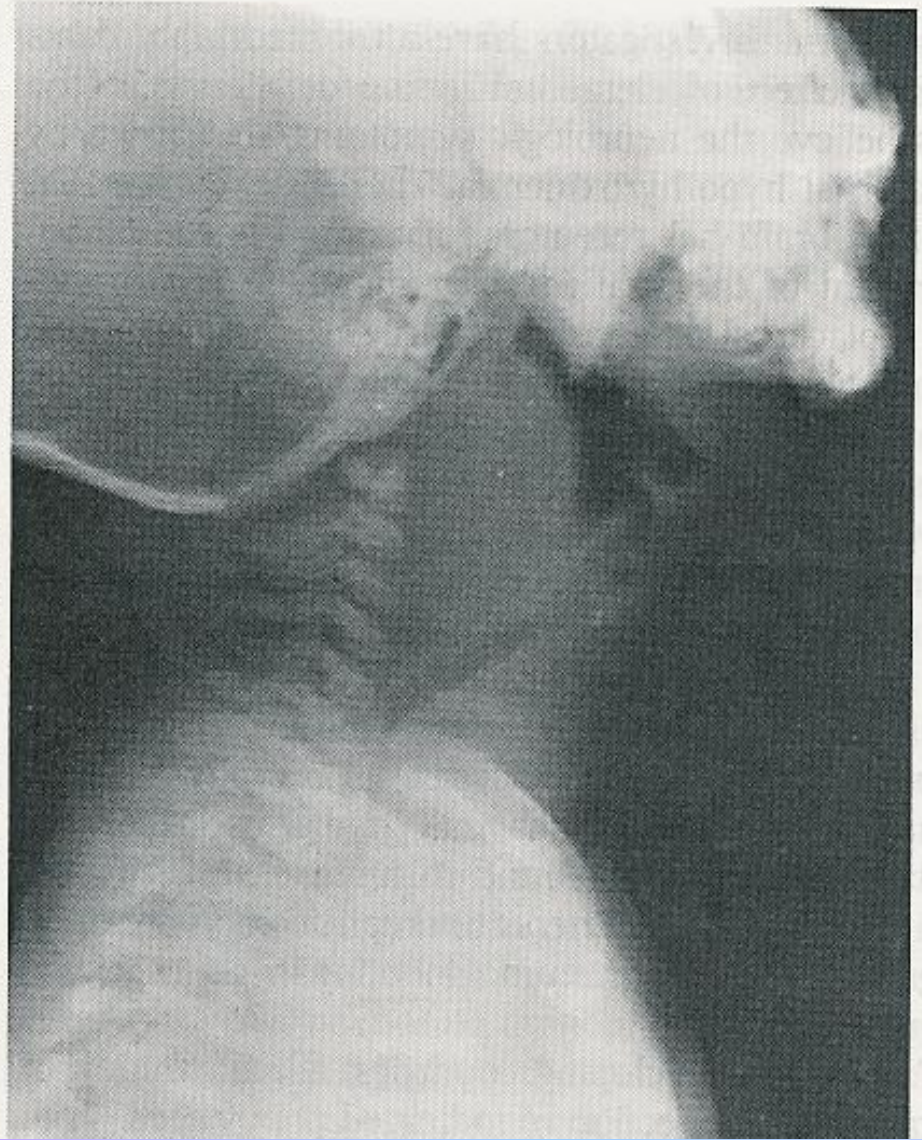
- Heat intolerance, dislike of hot weather;
- Hyperactivity, irritability, nervousness, fatigue;
- Weight loss (normal to increased appetite);
- Diarrhea;
- Tremor, palpitations;
- Diaphoresis (sweating);
- Lid retraction, thyroid stare;
- Pretibial myxedema and Graves ophthalmopathy (chemosis, diplopia, and exophthalmos);
- Menstrual irregularity;
- Goitre;
- Tachycardia;
- Females are more commonly affected(F:M = 5:1).

Causes of Transient Neonatal Hyperthyroidism

- Neonatal hyperthyroidism is almost always transient and results from the transplacental passage of maternal TSH, receptor stimulating antibodies.
- Hyperthyroidism develops only in babies born to mothers with the most potent stimulatory activity in serum.
- This corresponds to 1-2% of mothers with Graves ' disease, or 1 in 50,000 newborns.

Situations That Should Prompt Consideration of Neonatal Hyperthyroidism:

- Unexplained tachycardia, goitre or stare;
- Unexplained petechiae, hyperbilirubinemia, or hepatosplenomegaly;
- There is a persistently high TSH receptor antibody titer in mother during pregnancy in history;
- There is a persistently high requirement for antithyroid medication in mother during pregnancy in history;
- There is a thyroid ablation for hyperthyroidism in mother in history;
- There are previously affected sibling in history.



Congenital thyrotoxic goiter of and infant born to a mother with thyrotoxicosis

Therapy of Transient neonatal hyperthyroidism

- Treatment is accomplished by maternal administration of antithyroid medication in fetus.
- Till nowadays propylthiouracil (PTU) was the preferred drug for pregnant women in North America, but current recommendations suggest the use of mercazolilum rather than PTU after the first trimester because of concerns about potential PTU-induced hepatotoxicity.
- The goals of therapy are to utilize the minimal dosage which is necessary to normalize the fetal heart rate and render the mother euthyroid or slightly hyperthyroid.

Therapy of Transient neonatal hyperthyroidism

- In the neonate, treatment is the follows: either PTU (5 to 10 mg/kg/day) or mercazolilum (0.5 to 0.7 mg/kg/day) has been used initially in 3 divided doses.
- If the hyperthyroidism is severe, a strong iodine solution (Lugol's solution or SSKI, 1 drop every 8 hours) is added to block the release of thyroid hormone immediately.
- Often the effect of PTU and mercazolilum is not as delayed in infants as it is in older children or adults.

Therapy of Transient neonatal hyperthyroidism

- Propranolol (2 mg/kg/day in 2 or 3 divided doses) is added if sympathetic overstimulation is severe, particularly in the presence of pronounced tachycardia.
- If cardiac failure develops, treatment with digoxin should be initiated, and propranolol should be discontinued.
- Rarely, prednisone (2 mg/kg/day) is added for immediate inhibition of thyroid hormone secretion.
- Measurement of TSH receptor antibodies in treated babies may be helpful in predicting when antithyroid medication can be safely discontinued.
- Lactating mothers on antithyroid medication must continue nursing as long as the dosage of PTU or mercazolilum does not exceed 400 mg or 40 mg respectively.

Permanent neonatal hyperthyroidism

- Rarely, neonatal hyperthyroidism is inconvertible and is due to a germline mutation in the TSH receptor resulting in its constitutive activation.
- Function mutation of the TSH receptor should be suspected if persistent neonatal hyperthyroidism occurs in the absence of detectable TSH receptor antibodies in the maternal circulation.
- An autosomal dominant inheritance has been noted in many of these infants. Other cases have been sporadic, arising from new mutation.

Permanent neonatal hyperthyroidism

- Early recognition is important because the thyroid function of affected infants is frequently difficult to manage medically, and, when diagnosis and therapy is delayed, irreversible sequelae, such as cranial synostosis and developmental delay may result.
- For this reason early, aggressive therapy with either thyroidectomy or even radioablation has been recommended.

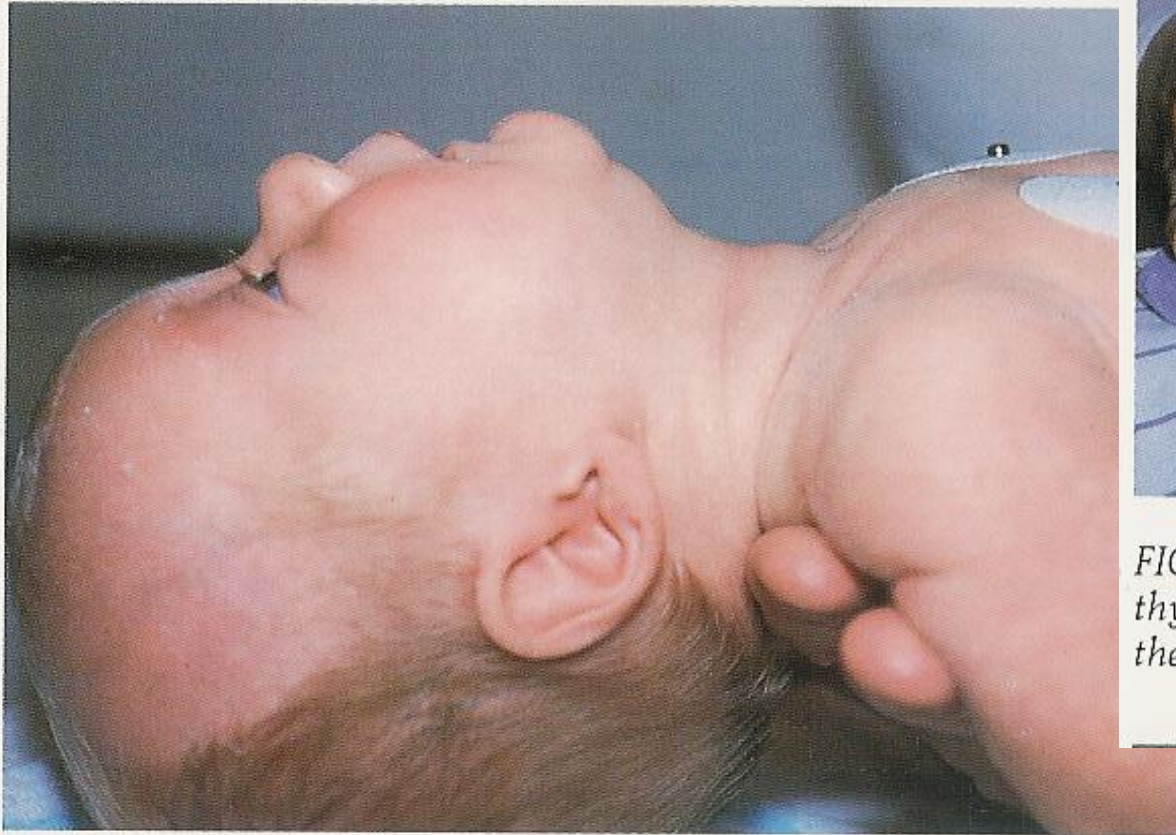


FIG. 9.11 Examination of the neonatal thyroid gland may be effectively performed by elevating the infant's trunk and allowing the head to drop back gently, as shown.



FIG. 9.9 Correct palpation of the thyroid gland is performed from behind the child.

Goitre

- Endemic goitre:
 - Areas where $> 5\%$ of children 6-12 years old have goiter
 - Common in China and central Africa
- Sporadic goitre:
 - Areas where $< 5\%$ of children 6-12 years old have goiter
 - *Multinodular goiter* in sporadic areas often denotes the presence of multiple nodules rather than gross gland enlargement
- Familial.

Goitre

• Etiology

- Hashimoto's thyroiditis:
 - Early stages only, late stages show atrophic changes;
 - May present with hypo, hyper or euthyroid states;
- Graves':
 - Due to chronic stimulation of TSH receptor;
- Diet: vegetarian with mainly usage of various types cabbage;
- Chronic iodine excess:
 - iodine excess leads to increased colloid formation and can prevent hormone release;
 - If a patient does not develop iodine leak, excess iodine can lead to goiter;
- Medications:
 - Lithium prevents release of hormone, causes goiter in 6% of chronic users;
- Neoplasm.

Goitre

-

THYROID GLAND

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Produces: T_4 and T_3 (active hormone)

Regulation: “negative Feed-back” axis

Functions: **THYROXIN** – regulate rate of metabolism

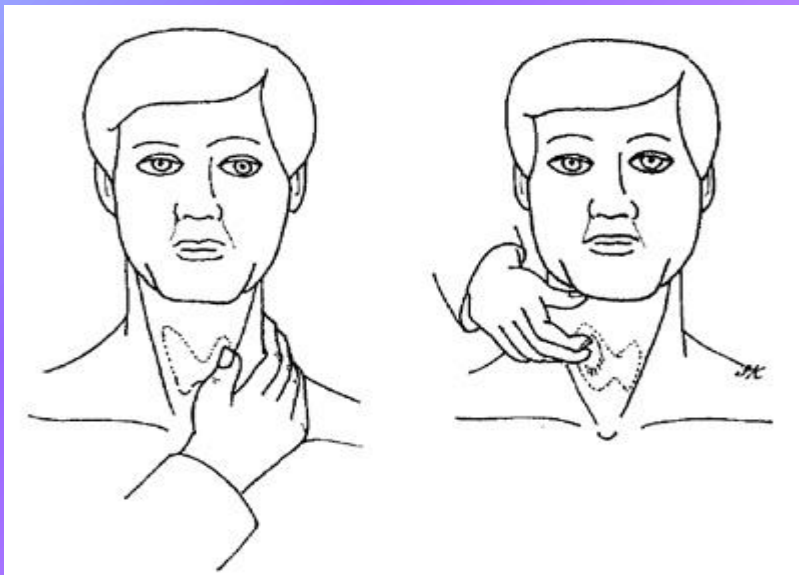
CALCITONIN – decreases levels of calcium and phosphate in the blood (partially antagonistic to parathyroid hormone).

Classification of Goitre WHO (1994)

0 – goitre is absent;

I – goitre isn't visualized, but it's size less than distal phalanx of thumb;

II – goitre is palpated and visualized.



Functional condition of Thyroid influence may be as

- Euthyroidism;
- Hypothyroidism;
- Hyperthyroidism.

Non-Toxic Goitre

- Cancer screening in non-toxic MNG (*Multinodular goitre*)
 - Longstanding MNG has a risk of malignancy of solitary nodules (<5%);
 - MNG with nodules < 1.5 cm may be followed clinically;
 - MNG with non-functioning nodules > 4cm should be excised:
 - No FNA needed due to poor sensitivity;
 - Incidence of cancer (up to 40%);
 - Fine-needle aspiration (FNA) in MNG:
 - Sensitivity 85% - 95%;
 - Specificity 95%;
 - Negative FNA can be followed with annual US (ultrasound);
 - Insufficient FNA's should be repeated;
 - While FNA hyperfunctioning nodules may mimic follicular neoplasm.

Non-Toxic Goitre

- **Treatment options** (no compressive symptoms):
 - Use follow-up to monitor for progression;
 - Thyroid suppression therapy:
 - May be used for progressive growth;
 - May reduce gland volume up to 50%;
 - Goitre regrowth occurs rapidly following therapy cessation.
 - Surgery indications:
 - Suspicious neck lymphadenopathy;
 - There is a radiation to the cervical region in history;
 - Rapid enlargement of nodules;
 - Papillary histology;
 - Microfollicular histology.

Non-Toxic Goitre

- **Treatment options (compressive symptoms):**
 - Radioactive iodine (RAI) ablation:
 - Volume reduction 33% - 66% in 80% of patients
 - Improvement of dysphagia or dyspnea in 70% - 90%
 - Post RAI hypothyroidism 60% of patients during 8 years
 - Post RAI Graves' disease 10% of patients
 - Post RAI lifetime cancer risk 1.6% of patients
 - Surgery treatment apply rare:
 - Most commonly recommended treatment if conservative treatment noneffective.

Toxic Goitre

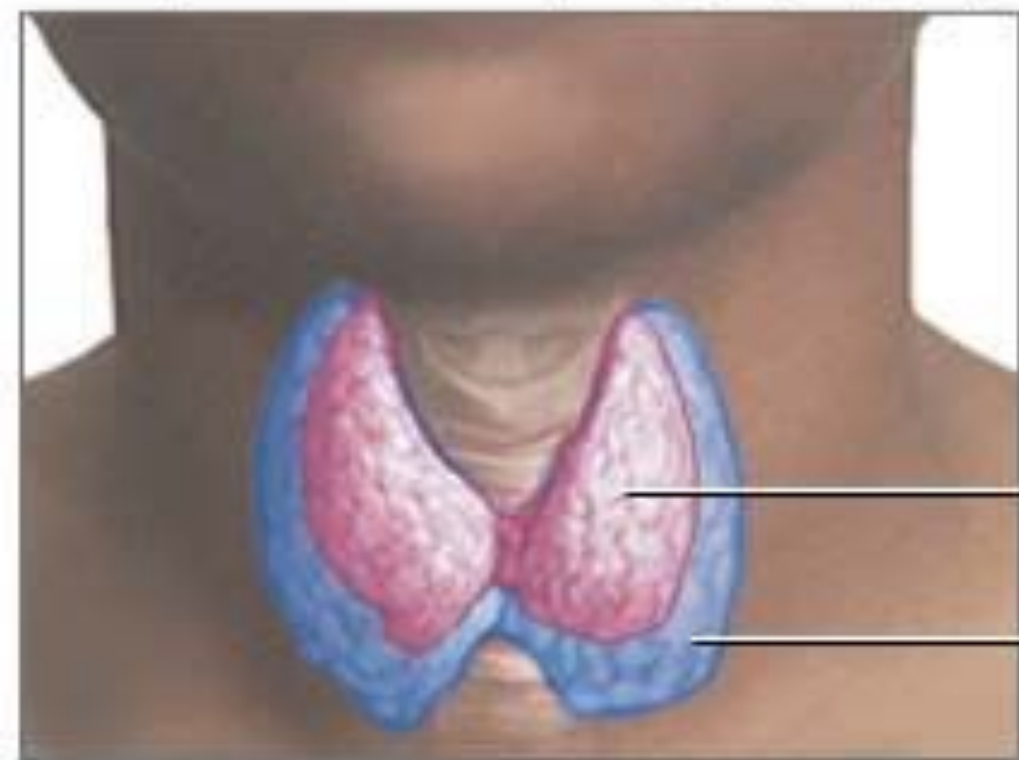
- **Treatment for Toxic MNG:**
 - Thionamide medications:
 - Not indicated for long-term use due to complications;
 - May be used for symptomatic individuals until definitive treatment.
 - Radioiodine:
 - Primary treatment for toxic MNG;
 - Large I¹³¹ dose required due to gland size;
 - Goitre size reduction by 40% within 1 year;
 - Risk of hypothyroidism 11% - 24%;
 - May require second dose.
 - Surgery:
 - Used for compressive symptoms;
 - Hypothyroidism occurs in up to 70% of subtotal thyroidectomy patients;
 - Pre-surgical stabilization with thionamide medications;
 - Avoid SSKI (Saturated Solution Of Potassium Iodine) due to risk for acute toxic symptoms.

Graves' Disease

- Diffuse toxic goitre is an autoimmune pathology with prolonged elevation T_3 and T_4 and enlargement of thyroid gland. In 70% cases with ophthalmopathy.
- Most common cause of thyrotoxicosis is in the industrialized world.
- Autoimmune condition with anti-TSHreceptor antibodies.
- Onset of disease may be related to severe stress which changes the immune response.
- Diagnosis
 - TSH, T_4 , T_3 to establish toxicosis;
 - Radioactive iodine uptake (RAIU) scan to differentiate toxic conditions;
 - Anti-TPO, Anti-TSAb, fT_3 as certain markers.



Exophthalmos (bulging eyes)



Diffuse goiter

Graves' disease is a common cause of hyperthyroidism, an over-production of thyroid hormone, which causes enlargement of the thyroid and other symptoms such as exophthalmos, heat intolerance and anxiety

Normal thyroid

Enlarged thyroid

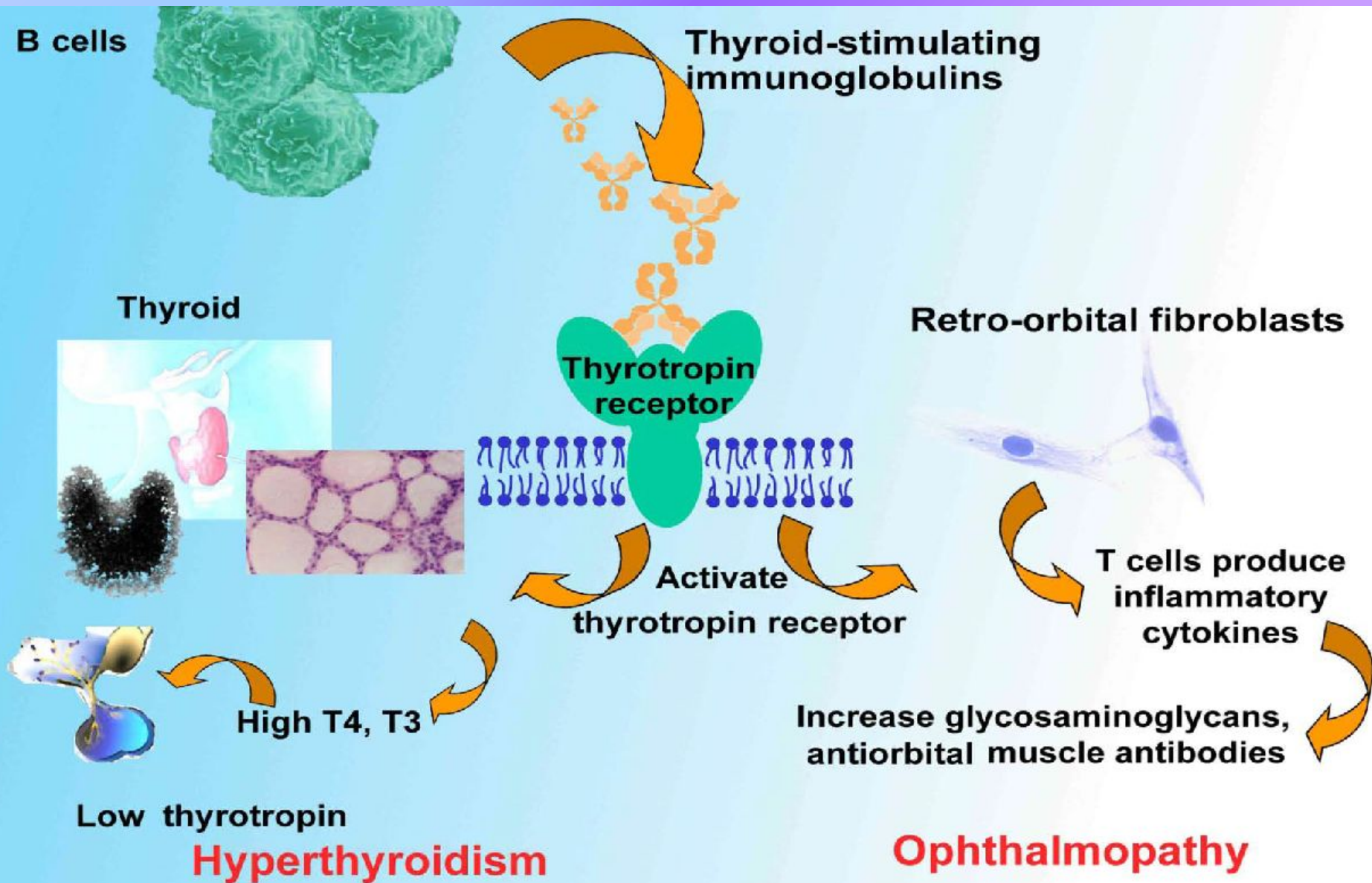
RAIU in Hyperthyroid States

High Uptake	Low Uptake
Graves'	Subacute Thyroiditis
Toxic MNG	Iodine Toxicosis
Toxic Adenoma	Thyrotoxicosis factitia

Differentiating Causes of Hyperthyroidism

	Graves Disease	"Hot" nodule	Hashitoxicosis	Subacute Thyroiditis	TSH adenoma
Thyroid hormone	□↑↑	↑↑	↑↑	↑↑	↑↑
TSH	↓↓	↓↓	↓↓	↓↓	
TSI	↑↑				
TPO	↑↑		↑↑		
Radionuclide scans	Overall ↑↑	Localized ↑↑	↓↓	↓↓	Overall ↑↑

Pathophysiology of ophthalmopathy



Graves disease Ophthalmopathy

- A feeling of "sandpaper" in the eyes and discomfort in the eyes;
- Retrobulbar pressure or pain;
- Eyelid retraction;
- Periorbital edema, chemosis, scleral injection;
- Proptosis of eyebulb;
- Extraocular muscle dysfunction;
- Exposure keratitis;
- Optic neuropathy.

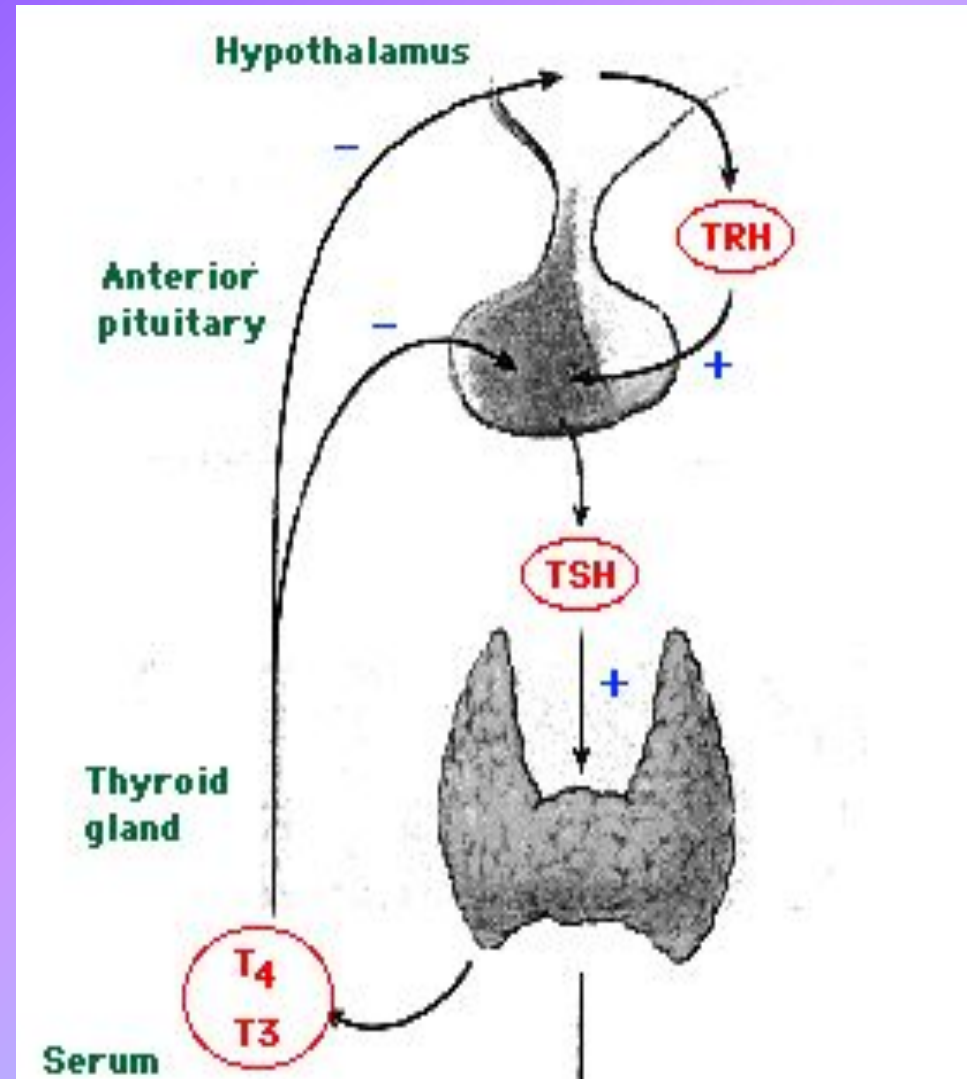


Graves Disease: Treatment

- Medications:
 - Beta-blockers for symptoms – can be discontinued as thyroid function tests normalize;
 - Methimazole (mercazolilum): block and replace the thyroid hormones;
- Surgery;
- Radioactive iodine administer in patients on the shady side of forty;
- Concurrent treatment of eye disease.

Hypothyroidism

Hypothyroidism - syndrome with particular or total deficiency of T₃, T₄ or their acts to target cells.



Classification of hypothyroidism

PRIMARY - defects of biosynthesis of T₃, T₄ due to pathology of thyroid gland.

SECONDARY - decreasing T₃, T₄ level due to deficiency of TSH (pituitary) or TRH (hypothalamus) or resistance of receptors for T₃, T₄ .

Primary:

- Autoimmune (Hashimoto's);
- Iatrogenic Surgery or ^{131}I administration;
- Drugs: amiodarone, lithium;
- Congenital (1 in 3000 to 4000);
- Iodine deficiency;
- Infiltrative disorders.

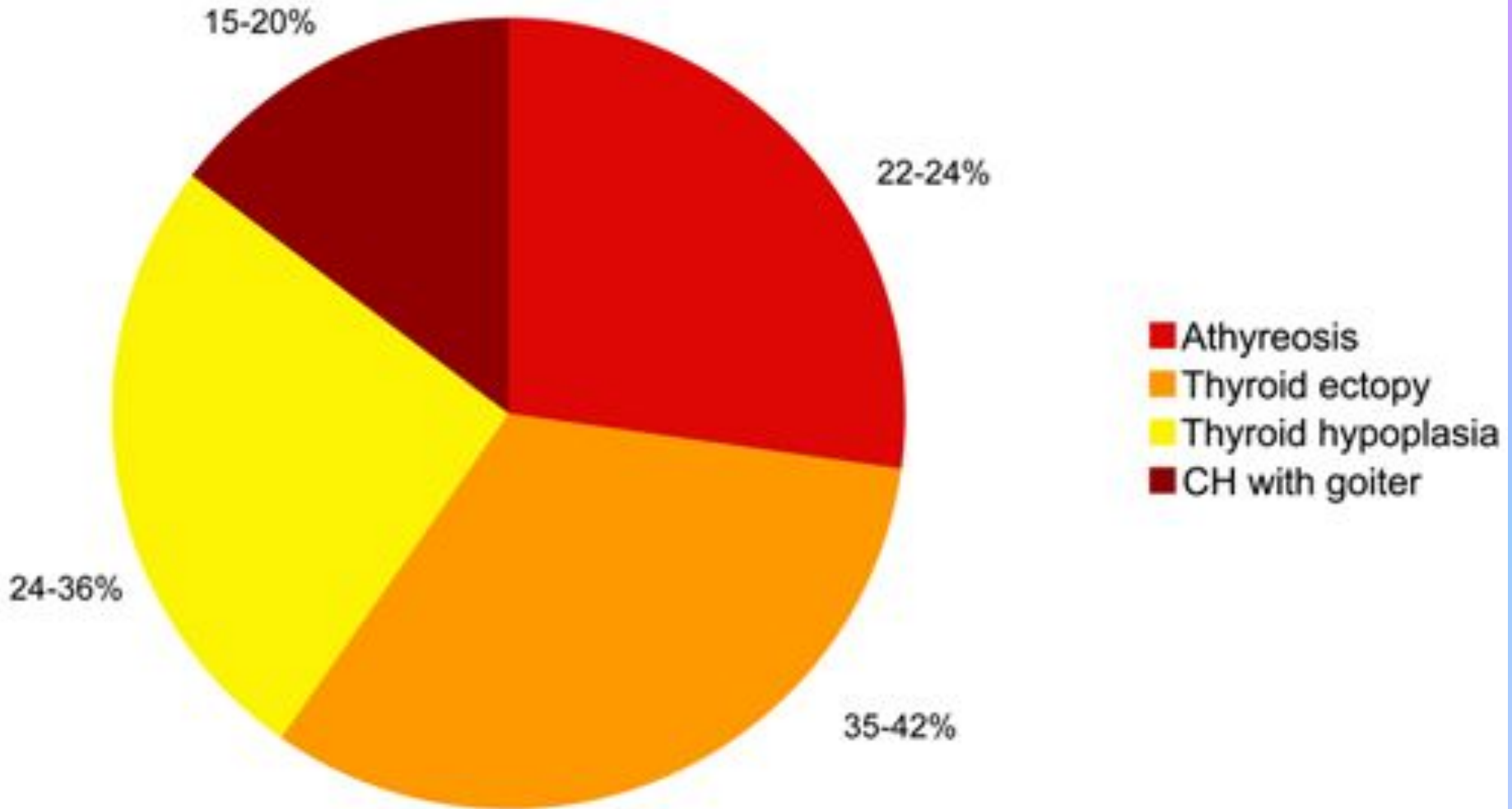
Secondary:

- Pituitary gland destruction;
- Isolated TSH deficiency;
- Bexarotene treatment;
- Hypothalamic disorders.

Peripheral:

- Rare, familial tendency.

Ethyology of congenital hypothyroidism



Expected Findings in Congenital Hypothyroidism

Cause	TSH	T ₄	Goiter	^{99m} Tc Scan Results	Ultrasonography Results
Thyroid dysgenesis					
Athyreosis	Increased	Decreased	No	No uptake	No gland
Ectopic thyroid	Increased	Decreased	No	Positive uptake outside of the thyroid bed, usually at the base of the tongue	No gland or Small gland
TSH receptor defect	Increased	Decreased	No	No uptake	Normal or small gland
Na-I symporter defect	Increased	Decreased	Yes	No uptake	Enlarged gland

Expected Findings in Congenital Hypothyroidism

Cause	TSH	T ₄	Goiter	^{99m} Tc Scan Results	Ultrasonography Results
Organification defects	Increased	Decreased	Yes	Positive uptake	Enlarged gland
Thyroglobulin synthesis defect	Increased	Decreased	Yes	Positive uptake	Enlarged gland
Deiodination defect	Increased	Decreased	Yes	Positive uptake	Enlarged gland
Central hypothyroidism	Low to normal	Decreased	No	Positive uptake	Normal-to-small gland
Transient	Increased	Decreased or normal	If iodine-deficient or exposed to goitrogen	Positive uptake usually; no uptake if maternal TSH receptor-blocking antibodies	Enlarged gland

Congenital hypothyroidism

- Agenesis (no goiter) or dysgenesis (aplasia, hypoplasia, ectopic gland) are the most common causes 85%.
- Dyshormonogenesis (10%) and a goiter will be present. Pendred syndrome with sensorineural deafness is the most common (often euthyroid).
- Transplacental maternal TSH receptor blocking Abs (TRBAbs) in 5% of cases.
- Pituitary failure and maternal administration of toxic substitute for thyroid gland.

Congenital hypothyroidism

Clinical features

- Coarse facial features, dry skin, prolonged jaundice, large fontanelles, posterior fontanell $> 1\text{cm}$, cutis marmorata, bradycardia, hypothermia, hoarse cry, cold extremities, short stature, possible deafness.
- Hypotonia, lethargy, poor feeding, constipation, macroglossia, umbilical hernia and edema.
- The brain is extremely sensitive to the presence of thyroid hormones from end of pregnancy until the 1st week of life, and if left untreated may result in irreversible mental retardation.



*FIG. 9.12 A child with cretinism.
(Courtesy of Dr. T.P. Foley, Jr.)*





Hypothyroidism Screening in the Newborn

More often the heel stick dried blood spot on 4th day in term and on 7th day in preterm is used to examine the T₄ level and TSH.

Because of the rapid changes in T₄ and TSH in the first few days of life, it is important to take into account when the sample is obtained. Some programs check in follow up in 2-4 weeks.

Different criteria must be employed for LBW babies.

A



B



TSH less than 20 IU/l	norma
TSH more than 50 IU/l	Congenital hypothyroidism, thyroxine must be prescribe
TSH more than 20 IU/l but less than 50 IU/l	Repeat test
If the repeated examination show the TSH level more than 20 IU/l and T ₄ level less than 120 nmol/l	Congenital hypothyroidism, thyroxine must be prescribe
If the repeated examination show the normal TSH level and T ₄ level	Transient hypothyroidism

DIAGNOSTIC STUDIES IN HYPOTHYROIDISM

Thyroid scan – ^{99}Tc or I^{123} uptake;

Bone age;

TSH level!!!

Free T₄ level – if hypothalamic-pituitary hypothyroidism suspected;

TBG (Thyroid Binding Globulin) – if TBG deficiency is suspected;

Anti-thyroid antibodies – if there is thyroiditis in maternal history.

Biochemical markers of CH

- Low serum T₄ level and T₃ level with evaluated **TSH** (primary) level;
- T₃ –normal, T₄ ↓- severe or longstanding;
- T₄ –normal but **TSH** is elevated – compensative CH, transient or subclinical hyperthyroidism;
- T₄ ↓ but TSH normal- congenital TBG-deficiency or hypothalamic-pituitary hypothyroidism.

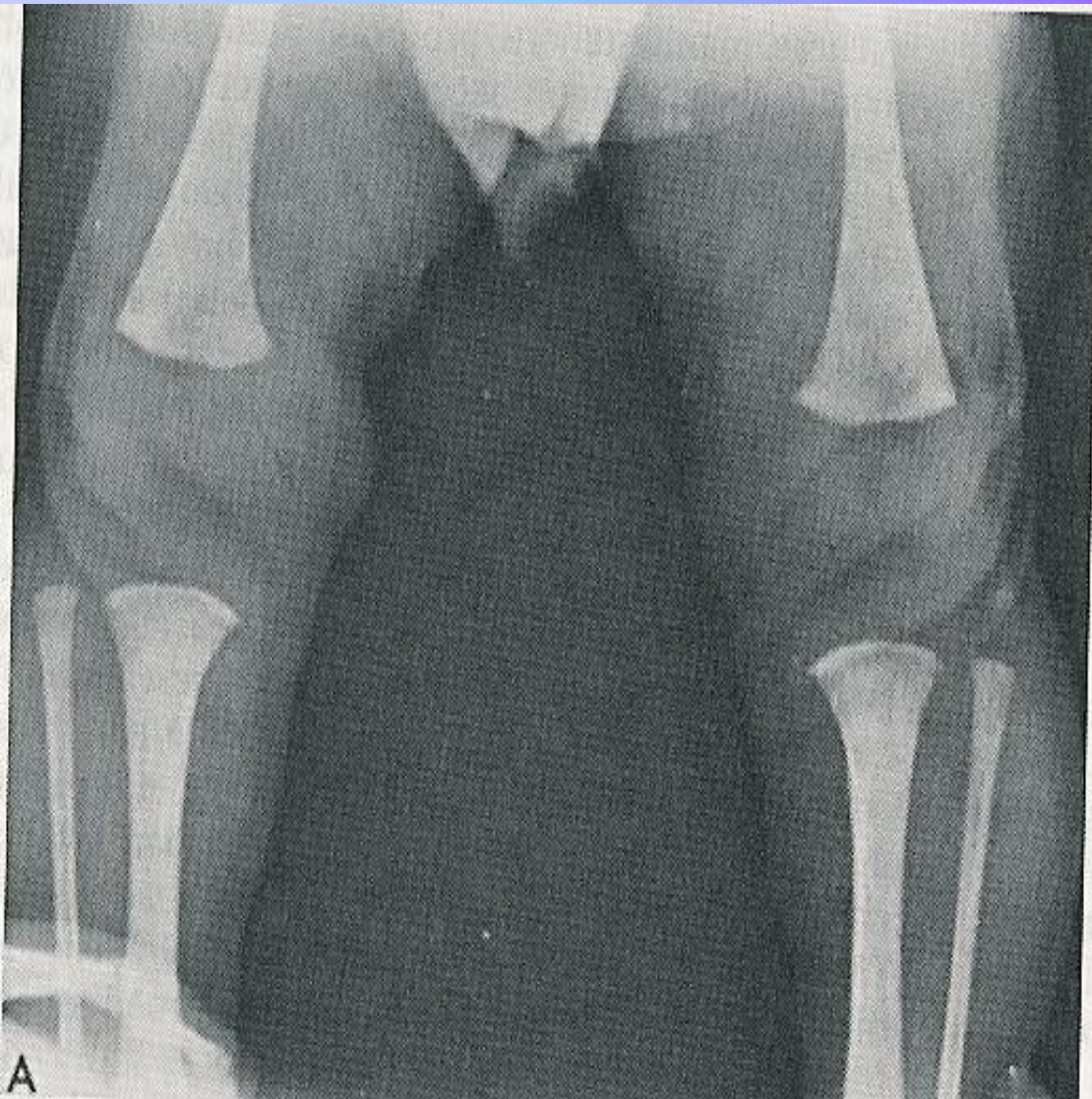
Biochemical markers of CH

Other:

- Elevated serum cholesterol;
- Elevated creatinphosphokinase;
- Hyponatremia.

Instrumental data

- Slightly decrease heart rate and amplitude of R wave (ECG);
- Increased left ventricular wall thickness, decrease LV chamber size and decrease cardiac output (EchoCG);
- Low-amplitude diffuse slowing (EEG).



A. Delayed epiphyseal appearance



B. Epiphyseal dysgenic

Management

Primary Congenital Hypothyroidism

High TSH and Low T4

Thyroxine

Form

Tablets

25-50-75 ug
Crush it, add to
5-10 ml of
water
or milk

Goals

Normal T4
In 2 wks
(upper ½ of N)

Normal TSH
In one month
(lower ½ of N)

Treatment prevents bone loss, cardiomyopathy, myxedema

Myxedema coma

- Reduced level of consciousness, seizures;
- Hypotension/shock;
- Hypothermia;
- Hyponatremia.

Treatment L-thyroxin (Levothyroxine)

Preterm	8 – 10 $\mu\text{g}/\text{kg}/\text{day}$
0-3 mo	10 – 15 $\mu\text{g}/\text{kg}/\text{day}$
3-6 mo	8 – 10 $\mu\text{g}/\text{kg}/\text{day}$
6-12 mo	6 – 8 $\mu\text{g}/\text{kg}/\text{day}$
1-3 years	4 – 6 $\mu\text{g}/\text{kg}/\text{day}$
3-10 years	3 – 4 $\mu\text{g}/\text{kg}/\text{day}$
10-15 years	2 – 4 $\mu\text{g}/\text{kg}/\text{day}$
> 15 years	2 – 3 $\mu\text{g}/\text{kg}/\text{day}$

PROGNOSIS

- ❑ If treatment is delayed, physical development can be hurt slightly. Early treatment is crucial to prevent mental and intellectual retardation.
- ❑ Early treatment of pregnant woman with thyroiditis may prevent mental subnormality in her child.

Juvenile hypothyroidism

- A child with growth retardation, constipation, becomes less sociable, gain weight; his school performance is deteriorating and he is intolerant to cold. He may also has goiter.
- Typical face with dry pale skin and periorbital edema.
- Typically no effect on intellect.

Causes of juvenile hypothyroidism

- Hashimoto thyroiditis. More common in girls who may have initial thyrotoxicosis or be euthyroid or hypothyroid.
- Hashimoto may be associated with Down, Turner and Klinefelter syndromes as well as SLE (systemic lupus erythematosus) and other autoimmune disorders.
- A goiter may be present initially with no clinical features of disturbed thyroid function.
- Other causes of JH include administration of goitrogens, iodine deficiency, hypothalamic/pituitary disorders and post thyroidectomy.

JH investigations

- Antithyroglobulin and antimicrosomal antibodies are found.
- Serum T4 is low (earlier than T3).
- Bone age is delayed.
- Treatment is with thyroxine.

ETIOLOGY OF ACQUIRED HYPOTHYROIDISM

- Chronic lymphocytic (Hashimoto`s) thyroiditis (CLT);
- Subacute thyroiditis (De Quervain`s);
- Goitrogens (iodide, thiouracil, etc.);
- Thyroidectomy or ablation following radioactive iodine;
- Infiltrative disease (e.g., cystinosis, histiocytosis X);
- Systemic disease.
- Hypothalamic or pituitary disease;
- Congenital thyroid disorders, e.g., ectopia, may not decompensate until later childhood and thus may appear acquired;
- Peripheral resistance to thyroid hormones, including receptor defects;
- Iatrogenic (propylthiouracil, methimazole, iodides, lithium, amiodarone);
- Hemangiomas of the liver.

SYMPTOMS OF ACQUIRED HYPOTHYROIDISM

- Slow growth;
- Edema;
- Decreased appetite;
- Constipation;
- Swollen thyroid gland;
- Lethargy;
- Drop in school performance;
- Cold Intolerance;
- Short stature;
- Delayed dentition;
- Myxedema or mildly overweight;
- Goiter;
- Galactorrhea;
- Menometrorrhagia.

SIGNS OF ACQUIRED HYPOTHYROIDISM

- Delayed reflex return;
- Mental depression;
- Pale, thick, or cool skin;
- Muscle pseudohypertrophy;
- Delayed puberty or precocious puberty;
- Treatment – same CH.

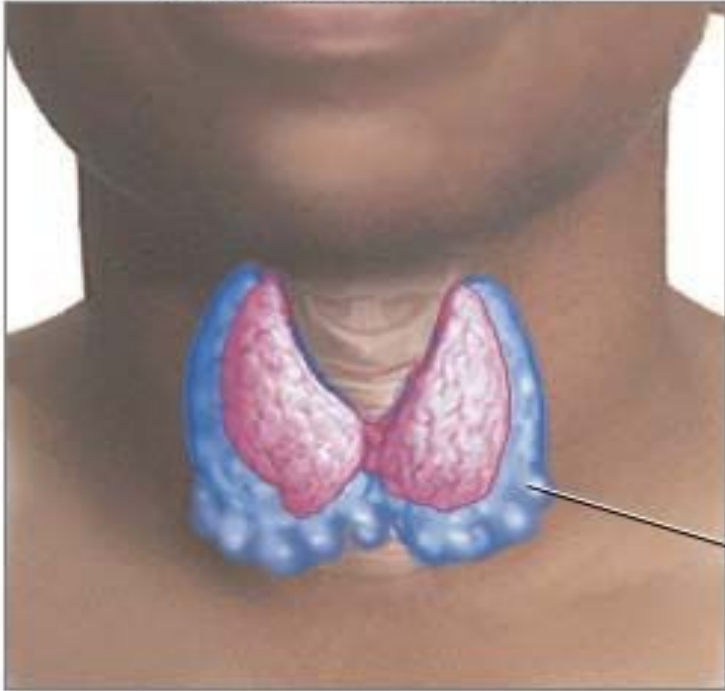
Chronic thyroiditis Hashimoto disease

Clinical presentation:

- Painless diffuse goiter;
- Goiter with euthyroidism;
- Toxic thyroiditis;
- Hypothyroidism with or without thyromegaly;
- Dysphagia, pain or pressure sensation in the neck, cough and headache.

Autoimmune hypothyroidism

Hashimoto's disease



Enlarged, inflamed hypofunctioning thyroid (goiter)

ADAM.



Diagnosis Hashimoto disease

- T4 total and free, serum TSH;
- Biopsy;
- Antibodies test: antithyroglobulin antibodies to thyroperoxidase, antimicrosomal test.

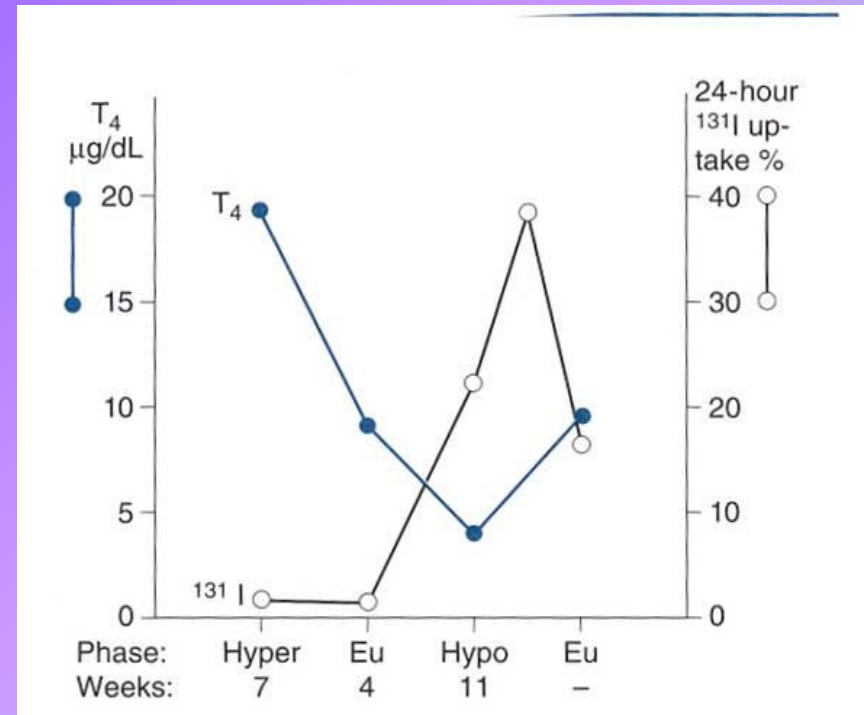
Treatment

- Levothyroxine if hypothyroid;
- Triiodothyronine (for myxedema coma);
- Thyroid suppression (levothyroxine) to decrease goiter size;
- Surgery treatment if compression or pain take place.

Subacute Thyroiditis

DeQuervain's, Granulomatous

- Most common cause of painful thyroiditis.
- Often follows upper respiratory infection.
- FNA may reveal multinucleated giant cells or granulomatous change.
- Disease running:
 - Pain and thyrotoxicosis (3-6 weeks);
 - Asymptomatic euthyroidism;
 - Hypothyroid period (weeks to months);
 - Recovery (complete in 95% after 4-6 months).



Subacute Thyroiditis

DeQuervain's, Granulomatous

- ## THYROID GLAND

Innervation: Vagus Nerve (X)

Arterial Supply: superior thyroid artery (branch of external carotid artery).

Produces: T_4 and T_3 (active hormone)

Regulation: “negative Feed-back” axis

Functions: **THYROXIN** – regulate rate of metabolism

CALCITONIN – decreases levels of calcium and phosphate in the blood (partially antagonistic to parathyroid hormone).

Acute Thyroiditis

- Causes:
 - 68% Bacterial (*S. aureus*, *S. pyogenes*);
 - 15% Fungal;
 - 9% Mycobacterial.
- May occur secondary to:
 - Pyriform sinus fistulae;
 - Pharyngeal space infections;
 - Persistent Thyroglossal Duct Cyst;
 - Thyroid surgery wound infections (rare).
- More common in HIV.

Acute Thyroiditis

- Diagnosis:
 - Warm, painful, enlarged thyroid;
 - FNA to drain abscess;
 - RAIU normal (versus decreased in DeQuervain's);
 - CT or US if infected Thyroglossal Duct Cyst suspected.
- Treatment:
 - High mortality without prompt treatment;
 - Antibiotics intravenous:
 - Nafcillin / Gentamycin or Rocephin for empiric therapy;
 - Search for pyriform fistulae (X-ray examination with barium meal, endoscopy);
 - Recovery is usually complete.



**Thank you for
listening!!!**