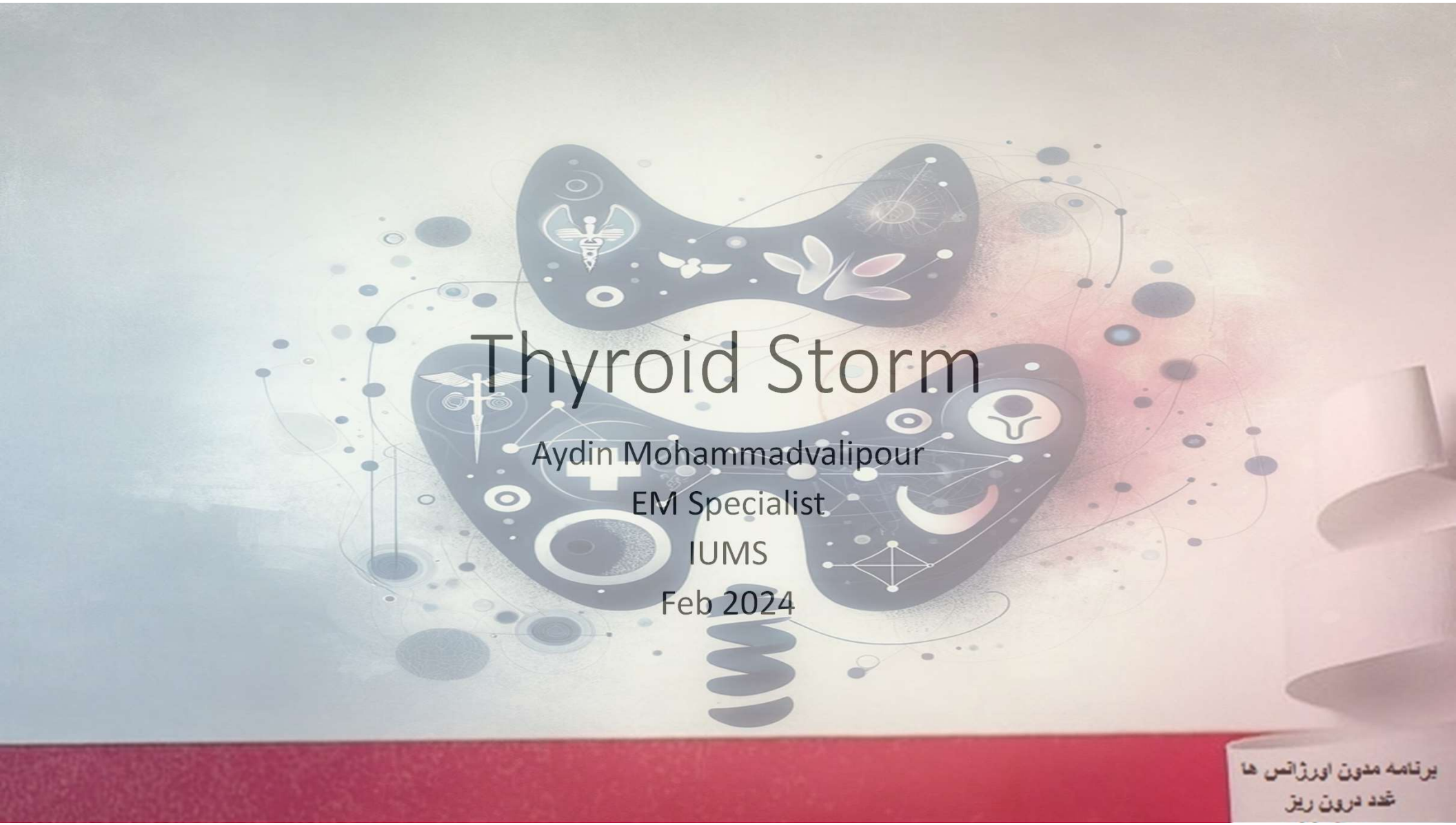

Thyroid Storm

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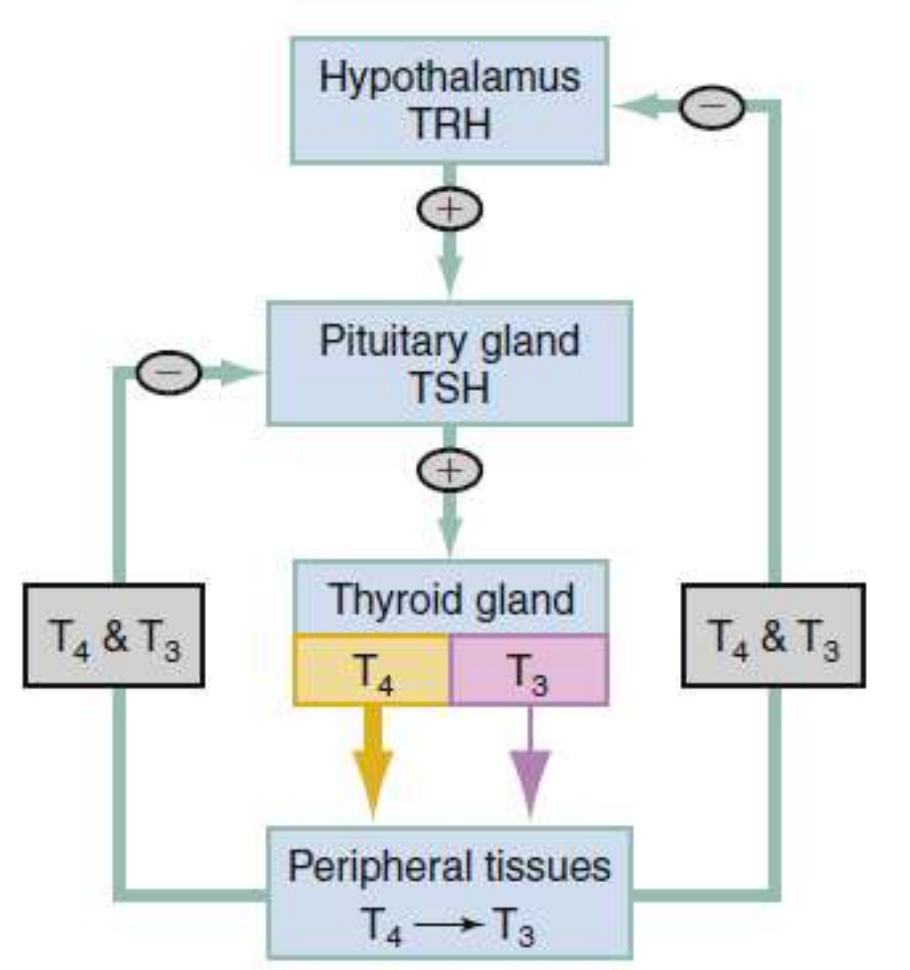
Introduction

- **Hyperthyroidism** is a condition caused by **overproduction** and **increased circulation** of **thyroid hormone**. The disorder runs the **spectrum** from **subclinical hyperthyroidism** to **thyrotoxicosis** and **thyroid storm**, a life-threatening disorder.
- This can occur from **hormone overproduction**, **increased thyroid hormone release** from an injured gland, or **exogenous thyroid hormone**.
- The **function** of thyroid hormone is to **influence** the **metabolism** of cells by **increasing** their **basal metabolic rate**. It has a **role** in **protein synthesis** and functions together with other hormones necessary for normal growth and development.
- **T3** and **T4** **increase** the expression and sensitivity of β-adrenergic receptors, increasing the response to endogenous catecholamines.

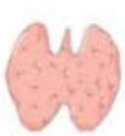


Introduction

- **T4** is a **prohormone** with only mild intrinsic activity; its deiodination produces **T3**, the **biologically active hormone**.
- More than 99.5% of thyroid hormones are protein-bound in the serum to thyroxine-binding globulin (TBG) and other proteins, rendering them metabolically inactive. As a result, **only free T4 and free T3 are clinically relevant**.
- Although iodide is a necessary substrate for thyroid hormone production, **excess iodide** can have two opposing effects.
- In the **Wolff-Chaikoff** effect, excess iodine inhibits the release of thyroid hormone from the gland by blocking iodide trapping and thyroglobulin iodination. This inhibition is transient, typically lasting only a matter of days.
- An iodine load can induce hyperthyroidism (**Jod-Basedow** effect) in some patients with multinodular goiter and occult Graves disease.

Wolff–Chaikoff effect

Drs. Jan Wolff and Israel Lyon Chaikoff



Seen with Thyroid gland under pituitary control

Normal Thyroid (Euthyroid)

Grave's disease (Hyperthyroid)

Ingest iodine

Diet | Contrast | Amiodarone

world.co.in

inhibits organification in the thyroid gland,
inhibits the formation of thyroid hormones inside the thyroid follicle
inhbits release of thyroid hormones into the bloodstream

Iodine-induced HYPOthyroidism

Normal Thyroid "Escapes" after 10 days

In autoimmune disease it persists

Escape phenomenon" is believed to occur because of decreased inorganic iodine concentration inside the thyroid follicle below a critical threshold secondary to down-regulation of sodium-iodide symporter (NIS) on the basolateral membrane of the thyroid follicular cell.Add a little bit of body text

Jod-Basedow effect

Jod (German) = Iodine

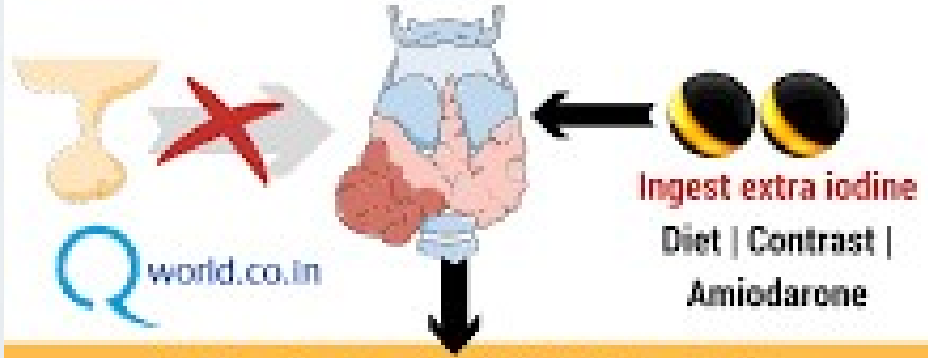
Basedow = Scientist who described effct

Seen with Abnormal Thyroid gland

free from pituitary control

Endemic goitre | Graves disease |

Toxic Multinodular Goitre | Thyroid adenoma



Iodine-induced HYPERthyroidism

Clinical Features

- **Altered mental status and coma** typify **thyroid storm**, the most severe manifestation of disease.
- **Hyperthyroidism** in **older adults** often manifests in **more subtle** ways, often **asymptomatic** or with **nonspecific symptoms** of weight loss, shortness of breath, and/or dementia.
- **Elders** are more prone to **cardiac manifestations** of hyperthyroidism and may present with **atrial fibrillation**.
- **Elders** who **smoke** or have **higher circulating** thyroid hormone levels appear to have **more severe symptoms**.
- **Thyrotoxic periodic paralysis** is a rare manifestation that presents as a **sudden** and **profound muscle weakness** progressing to **flaccid paralysis**, similar to familial hypokalemic periodic paralysis.

Clinical Features

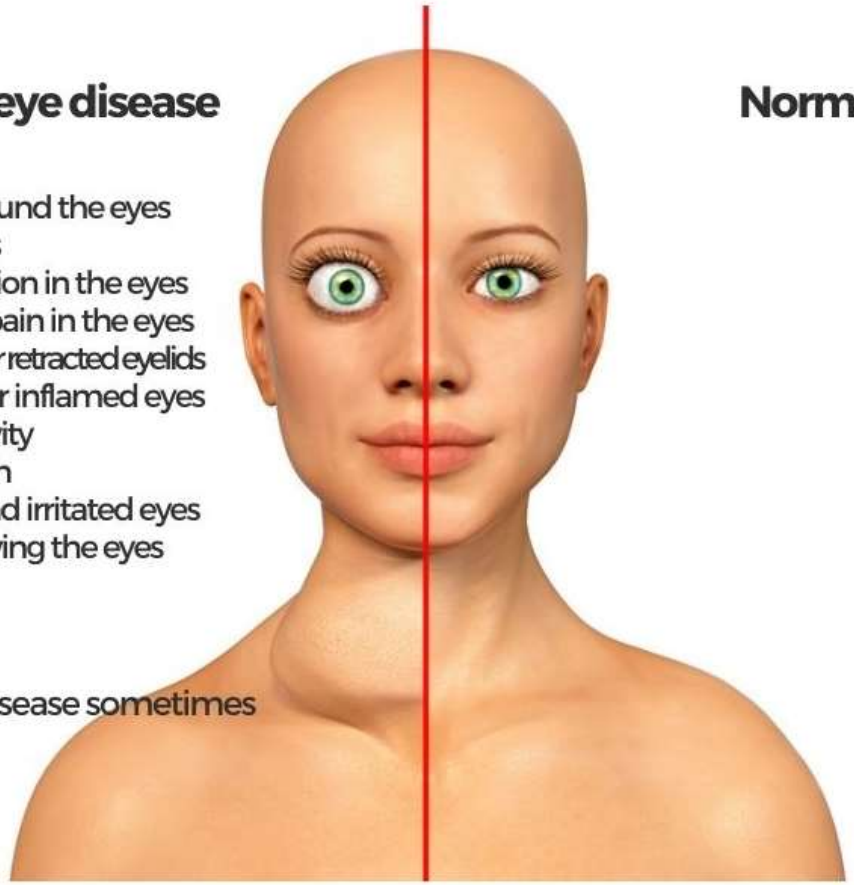
- **Ophthalmopathy** is a **classic finding in Graves disease**. Patients subsequently present with **eyelid edema**, **hyperemia**, **conjunctival hyperemia**, and **chemosis**. Graves ophthalmopathy is also associated with **restrictive extraocular myopathy**, and **exophthalmos**.
- As the disease progresses, patients may experience **restriction** of their **upward gaze** from infiltration of the inferior rectus muscle and **visual loss** from optic nerve involvement (compression by inflamed, enlarged orbital contents).
- Increased activity at the sympathetic innervation of the eyelids leads to **widening** of the **palpebral fissures**, resulting in the **characteristic stare and lid lag** of thyrotoxicosis.

GRAVES' EYE DISEASE SYMPTOMS

GRAVES' OPHTHALMOPATHY OR GRAVES' ORBITOPATHY (GO)

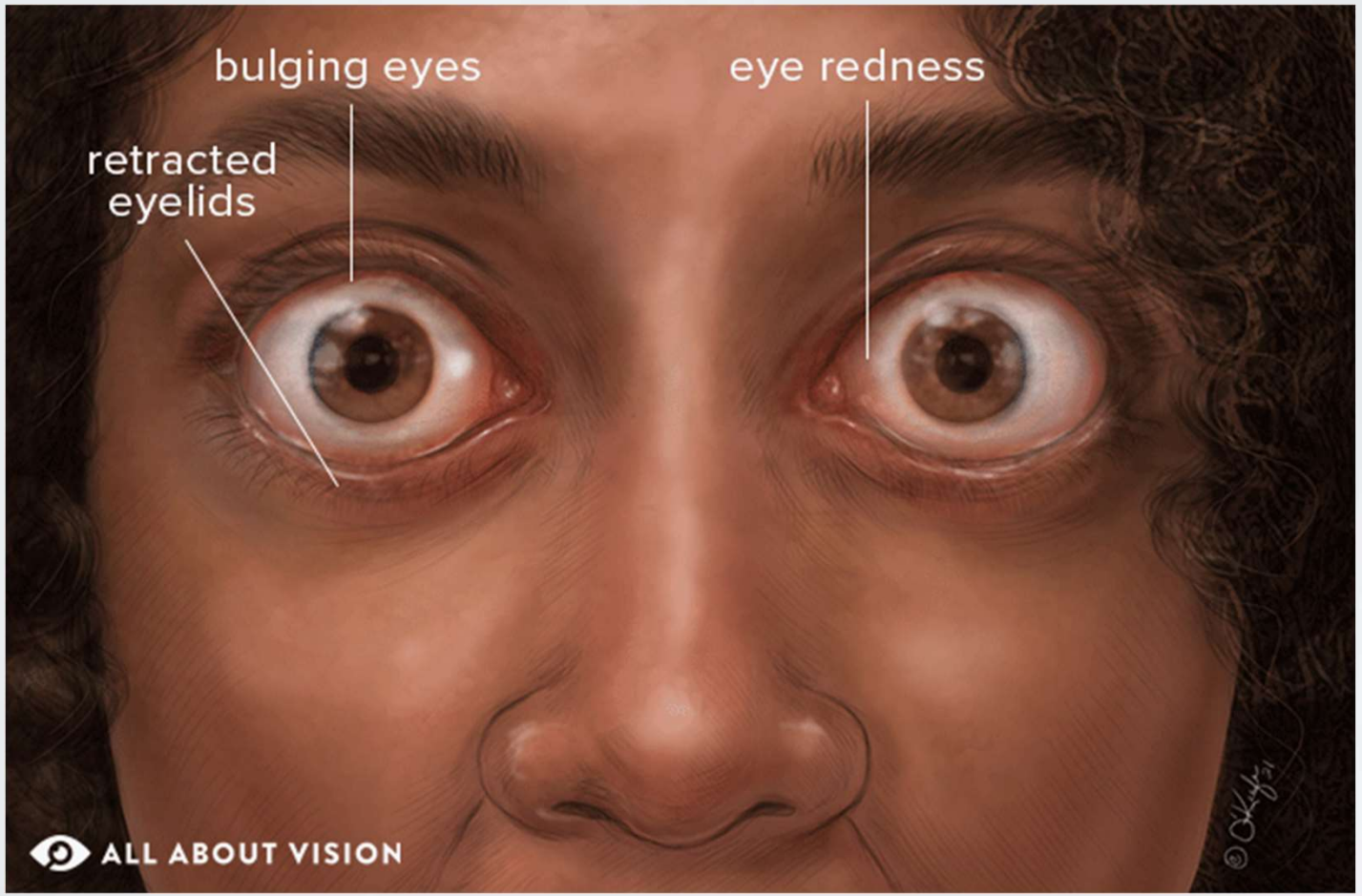
Graves' eye disease

Normal



- Swelling around the eyes
- Bulging eyes
- Gritty sensation in the eyes
- Pressure or pain in the eyes
- Puffy eyelids or retracted eyelids
- Reddened or inflamed eyes
- Light sensitivity
- Double vision
- Dry, gritty, and irritated eyes
- Trouble moving the eyes
- Vision loss

- In graves disease sometimes also goiter



Clinical Features

BOX 117.1 Symptoms of Thyrotoxicosis

Constitutional: Weight loss despite hyperphagia, fatigue, generalized weakness

Hypermetabolic: Heat intolerance, cold preference, excessive perspiration

Cardiorespiratory: Palpitations, dyspnea, dyspnea on exertion, chest pains, poor exercise tolerance

Gastrointestinal: Nausea, vomiting, diarrhea, dysphagia

Neuropsychiatric: Anxiety, restlessness, hyperkinesis, emotional lability, confusion, insomnia, poor attention

Neuromuscular: Myopathy, myalgias, tremor, proximal muscle weakness (difficulty getting out of a chair or combing hair)

Ophthalmologic: Tearing, irritation, wind sensitivity, diplopia, foreign body sensation

Thyroid gland: Neck fullness, dysphagia, dysphonia

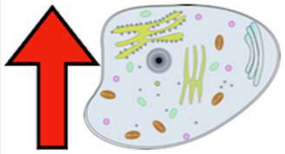
Dermatologic: Flushed feeling, hair loss, pretibial swelling

Reproductive: Oligomenorrhea, amenorrhea, menometrorrhagia, decreased libido, gynecomastia, erectile dysfunction, infertility

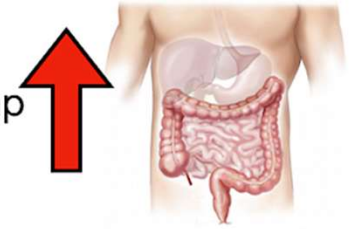
Hyperthyroidism Symptoms

Increased Levels of Thyroid Hormone in the Blood

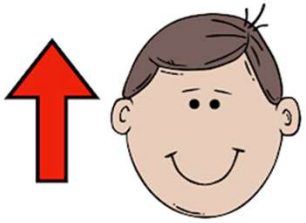
- *Symptoms:* ↑ Thyroid Hormone Effects **"Speeds Things Up"**



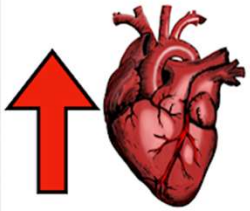
Weight Loss
Increased Appetite
Increased Body Temp
Heat Intolerance
Sensitivity to Heat



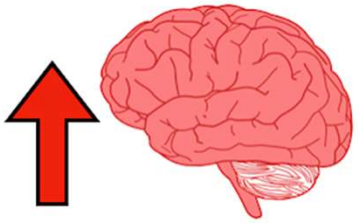
Diarrhea



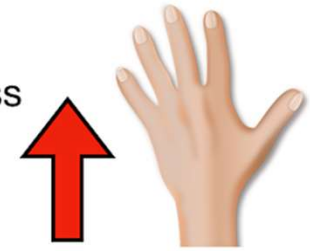
Sweating
Hair Growth
Fine, Soft Hair



Tachycardia
Palpitations
Arrhythmias
Hypertension



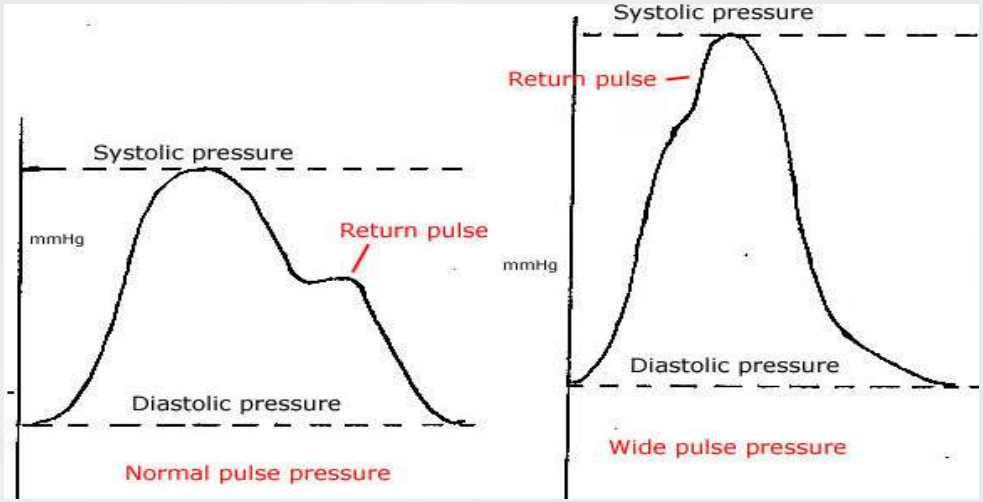
Anxiety
Nervousness
Irritability
Insomnia
Tremors

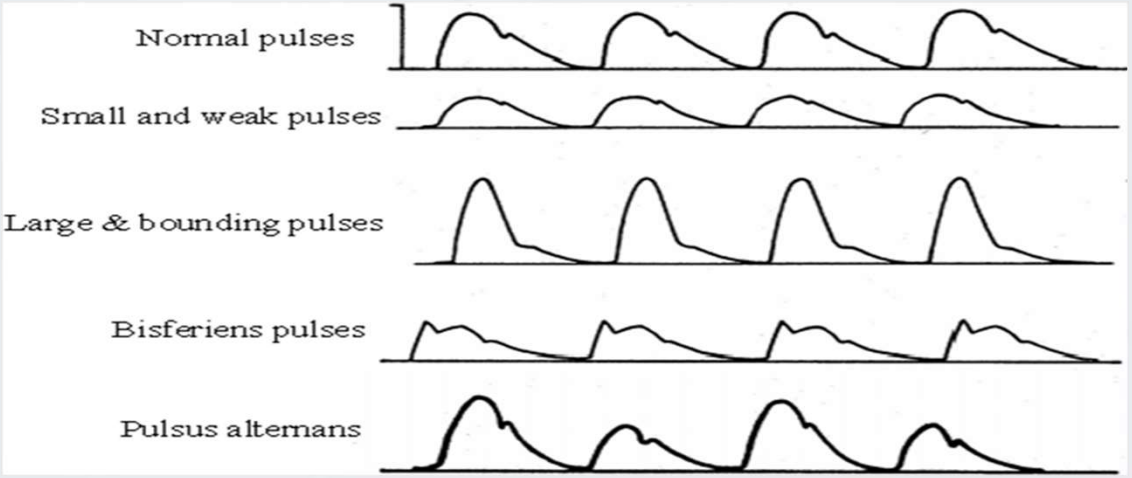


Nail Growth
Onycholysis

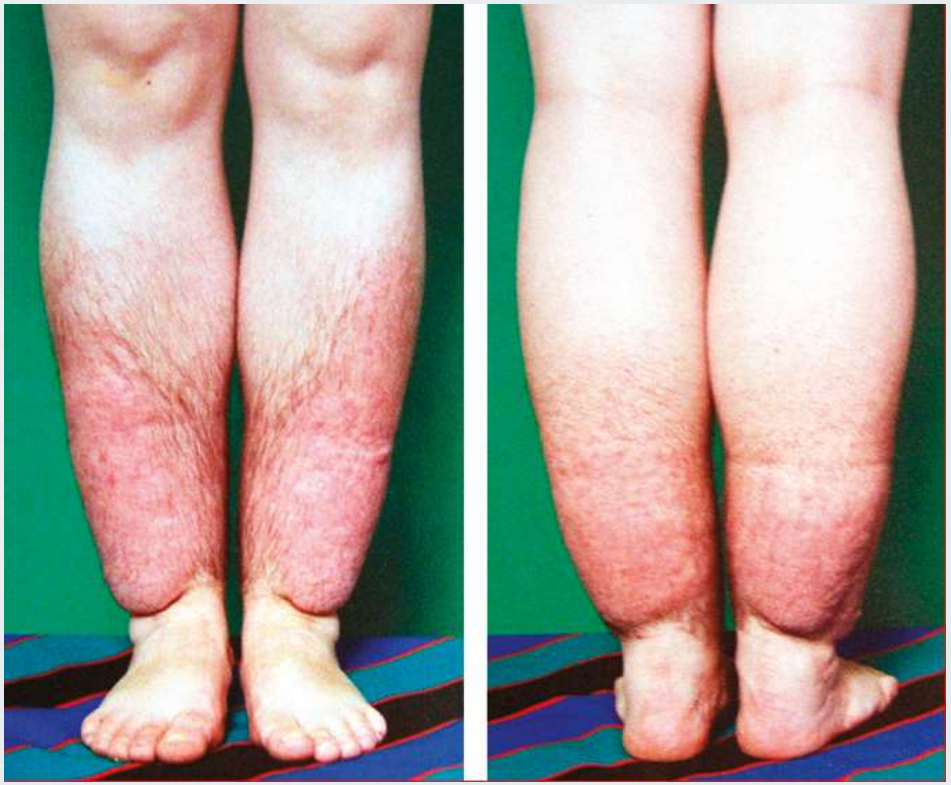
Physical examination

- Younger patients typically present with signs of sympathetic stimulation, whereas older adults often lack the same adrenergic response and present with weight loss and fatigue, more consistent with apathetic hyperthyroidism.
- In **Graves disease**, patients uncommonly have **classic pretibial myxedema**, which are confluent, painless, reddish raised nodules and plaques over the pretibial area and dorsum of the feet, often described as **orange skin**.
- Hyperpigmentation and induration are present, but pitting is absent. Pretibial myxedema is often associated with Graves ophthalmopathy.
- **Tachycardia is the most common cardiac finding.**
- Other findings include a **widened pulse pressure, bounding peripheral pulses** and, rarely, a friction rub heard along the **left sternal border** (**Means-Lerman scratch**).
- **Atrial fibrillation is more common in elders, especially those over 65 years of age**.
- **Dilated cardiomyopathy** may develop as a complication of a high cardiac output state.





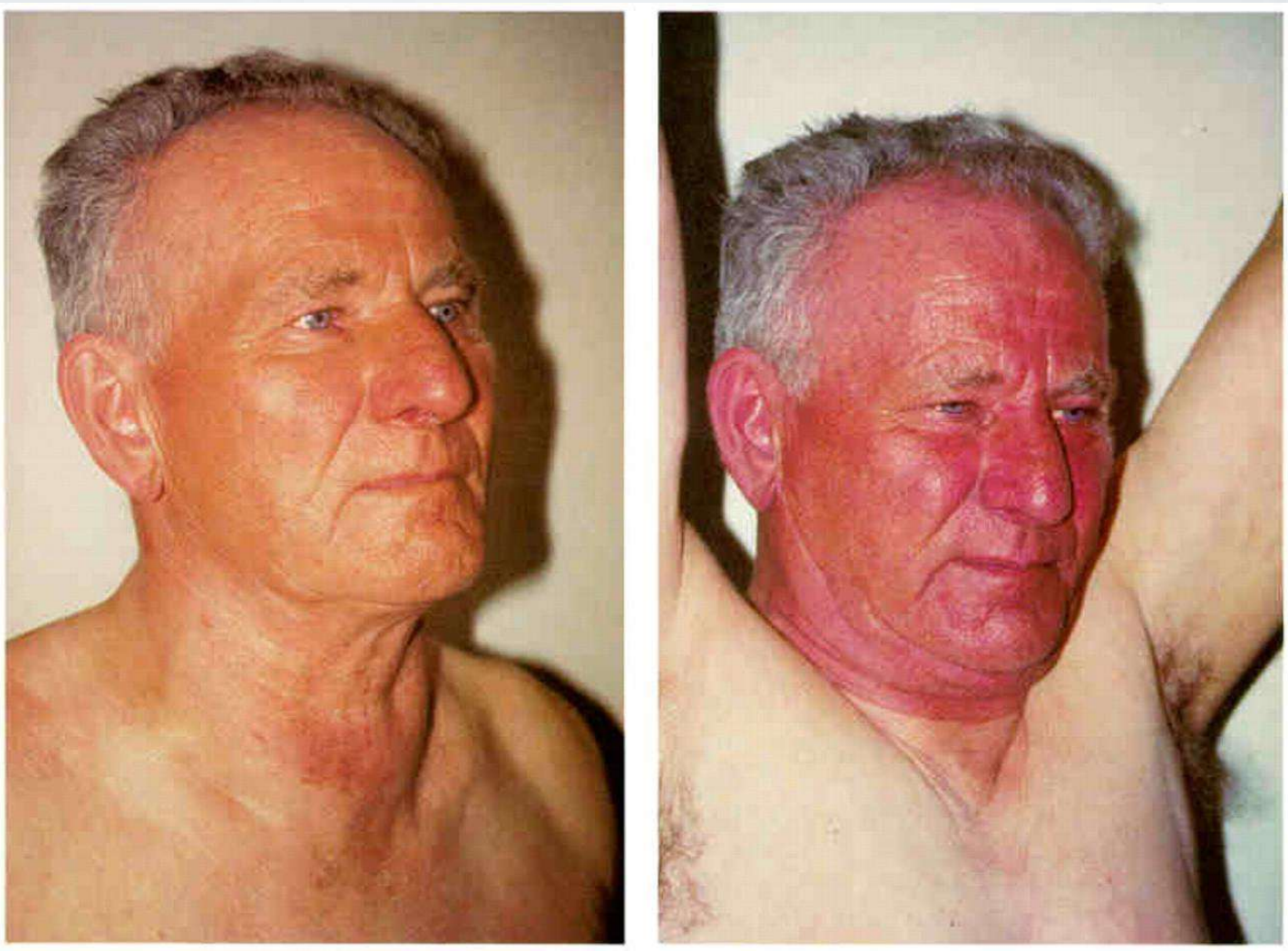
Physical examination



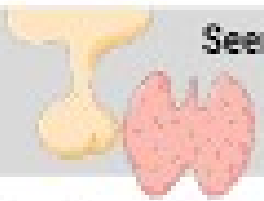
Physical examination

- Patients can also develop **primary pulmonary hypertension**, as well as increased chamber size and poor right ventricular function.
- Most hyperthyroid patients have an enlarged thyroid gland, especially in toxic multinodular goiter or Graves disease, although **many elders with Graves disease** have **nonpalpable thyroids**.
- **Retrosternal enlargement** can occur, making detection difficult while causing the **obstructive symptoms**.
- **Facial and neck vein engorgement** can be elicited when **arms are elevated** above the head (known as the **Pemberton sign**).
- The absence of thyroid enlargement should suggest exogenous (factitious) thyroiditis as well as ectopic thyroid hormone production, such as a hydatidiform mole or struma ovarii, an ovarian tumor composed of some thyroid tissue.

Pemberton sign



Thyrotoxicosis factitia



Seen with Normal Thyroid gland
under pituitary control

Ingestion of exogenous thyroid hormone (levothyroxine) or as a symptom of Munchausen syndrome

Levothyroxine induced HYPERthyroidism

Diagnosis

Thyrotoxicosis factitia:	True Thyrotoxicosis
Low Serum thyroglobulin	High Serum thyroglobulin
High fecal thyroxine	Low fecal thyroxine

Physical examination

BOX 117.2 Physical Findings in Thyrotoxicosis

Vital signs: Tachycardia, widened pulse pressure, bounding pulses, fever

Cardiac: Hyperdynamic precordium, systolic flow murmur, prominent heart sounds, systolic rub (Means-Lerman scratch), tricuspid regurgitation, atrial fibrillation, evidence of heart failure

Ophthalmologic: Widened palpebral fissures (stare), lid lag, globe lag, conjunctival injection, periorbital edema, proptosis, limitation of superior gaze

Neurologic: Fine tremor, hyperreflexia, proximal muscle weakness

Psychiatric: Fidgety, emotionally labile, poor concentration

Dermatologic: Warm, moist, smooth skin; rosy cheeks, blushing face; fine brittle hair; alopecia, flushed facies; palmar erythema; hyperpigmented pretibial plaques, nodules, or induration that is nonpitting; onycholysis (Plummer nails, separation of the distal portion of the fingernail from the nail bed)

Neck: Diffuse symmetric thyroid enlargement, sometimes with a bruit and palpable thrill; thyroid with multiple irregular nodules or a prominent single nodule; tracheal deviation, venous prominence with arm elevation (Pemberton sign)

Thyroid Storm

- Thyroid storm is a rare, **life-threatening** form of severe thyrotoxicosis, with multiorgan dysfunction and significantly higher mortality rates than thyrotoxicosis without storm.
- Although it can occur as the result of unrecognized or undertreated thyrotoxicosis, more **often**, it is an **acute reaction** to surgery, trauma, infection, iodine load or parturition in patients with preexisting hyperthyroidism.
- Other precipitants include acute myocardial infarction, pulmonary embolism, hyperemesis gravidarum, preeclampsia, and diabetic ketoacidosis.
- **Untreated, mortality approaches 100%**, but prompt recognition and therapy have lowered mortality to 10% to 30%.
- Death in thyroid storm is caused by multiorgan dysfunction, congestive heart failure, respiratory failure, arrhythmias, disseminated intravascular coagulation, hypoxic brain insult, or sepsis.

Thyroid Storm

- The **typical clinical manifestations** of thyroid storm include **marked pyrexia** (**40°–41°C**), **extreme tachycardia** (often **out of proportion** to level of **fever**), and **altered mental status**.
- These findings, coupled with the clinical picture of a patient with hyperthyroidism, lid lag, stare, goiter, ophthalmopathy, and tremor, should alert the emergency clinician to the diagnosis.
- **Cardiovascular collapse** can result in congestive heart failure, hypotension, and cardiac arrhythmias.
- **Hypotension** can also result from volume depletion secondary to nausea, vomiting, and diarrhea.
- **Abdominal pain** is **common**; **hepatic failure** with **cholestatic jaundice** is **less common** but carries a **poor prognosis**.
- **Thyroid storm is a clinical diagnosis**, and **no validated diagnostic criteria** yet exist. However, a scoring system developed by Burch and Wartofsky in 1993 is repeatedly cited and utilized by practicing endocrinologists.
- Although the test characteristics (e.g., sensitivity and specificity) **have not been published**, it may help distinguish among thyrotoxicosis, impending thyroid storm, and frank thyroid storm .

TABLE 117.1 Diagnostic Criteria for Thyroid Storm

Criteria	Score[a]
Fever (°F)	
99–99.9	5
100–100.9	10
101–101.9	15
102–102.9	20
103–103.9	25
≥104	30
Tachycardia (beats/min)	
90–109	5
110–119	10
120–129	15
130–139	20
≥140	25
Mental Status	
Normal	0
Mild agitation	10
Delirium, psychosis	
Extreme lethargy	20
Coma, seizures	30
Congestive Heart Failure	
Absent	0
Mild (edema)	5
Moderate (rales)	10
Pulmonary edema	15
Atrial fibrillation	10
Gastrointestinal and Hepatic Symptoms	
None	0
Nausea, vomiting	10
Diarrhea, abdominal pain	
Unexplained jaundice	20
Precipitating Event	
None	0
Present	10

[a]Tally the maximum score from each category. A score of 45 or higher suggests thyroid storm, or impending storm, and a score below 25 is unlikely to represent thyroid storm.

Differential Diagnoses

- The differential diagnosis for the thyrotoxic patient is **broad**.
- An **anxious** patient may be interpreted to be manic or experiencing a **panic attack**.
- The **hyperadrenergic state** may be confused with that seen in patients with sympathomimetic intoxication, suffering from anticholinergic crisis, or experiencing withdrawal from alcohol or sedative-hypnotics.
- The **hyperpyrexia** and **altered mental status** seen in thyroid storm may mimic other hyperthermic disorders such as heatstroke, neuroleptic malignant syndrome, serotonin syndrome, bacterial meningitis, and sepsis.
- **Elders** with apathetic hyperthyroidism may be **mistakenly diagnosed** with **psychiatric illness**.

Diagnostic Testing

- The **initial diagnosis** of thyrotoxicosis is **based on the clinical picture** and confirmed with laboratory values.
- Measurement of the **serum TSH level** is the **most sensitive test** for hyperthyroidism.
- In thyrotoxicosis, the serum TSH concentration is depressed or undetectable (<0.01 μU/mL in third-generation assays), and a normal TSH level excludes hyperthyroidism.
- **Accuracy of the TSH determination is improved when the free T4 test is added.**
- Assessment of thyroid function during acute nonthyroidal illness is difficult, especially in critically ill patients.
- **Severe systemic illness** depresses TSH production, leading to low levels of TSH, free T3, and free T4.

Diagnostic Testing

TABLE 117.2 Thyroid Function Test Interpretation

Tsh	Free T_4	Free T_3	Disease
Normal	Normal	Normal	None
Low	High	High	Hyperthyroidism
Low	Normal	Normal	Subclinical hyperthyroidism
Low	Normal	High	T_3 toxicosis
Low	High	Normal	Thyroiditis, T_4 ingestion, hyperthyroidism in older adults or those with comorbid illness
Low	Low	Low	Euthyroid sick syndrome; central hypothyroidism
High	Normal	Normal	Subclinical hypothyroidism; recovery from euthyroid sick syndrome

T_3, Triiodothyronine; *T_4*, thyroxine; *TSH*, thyroid-stimulating hormone.

Diagnostic Testing

- **Elevation of free T4 and free T3 levels in conjunction with TSH suppression is diagnostic of thyrotoxicosis.**
- Because nearly all T3 and T4 is bound to TBG, assays measuring total T3 or T4 are influenced by changes in TBG; they are therefore unreliable and should not be used.
- Subclinical hyperthyroidism is likely if TSH is suppressed and the free T4 level is normal.
- **T3 toxicosis** occurs in about 5% of patients with thyrotoxicosis. These patients have an **elevated free T3 level and a normal free T4 level.**
- When the **reverse pattern is present**—normal free T3 level and elevated free T4 level—the **differential includes** thyroiditis, exogenous levothyroxine ingestion, and hyperthyroidism in elders, often with suppressed T4 to T3 conversion due to comorbid illness.
- Since Graves is caused by autoantibodies to the TSH receptor, the presence of thyroid receptor antibodies in the serum can help distinguish it from other causes. Additionally, a radioiodine scan can help discern Graves from exogenous intake or hyperthyroidism due to struma ovarii.
- A magnetic resonance imaging (MRI) test of the brain and an ultrasound of the thyroid may help differentiate whether excess levels of thyroid hormone are emanating from either the pituitary or the thyroid gland.

Diagnostic Testing

- **Many thyrotoxic patients have hyperglycemia**. This is likely to be the result of increased glycogenolysis and catecholamine-mediated antagonism of insulin.
- Mild hypercalcemia can be seen, is related to hormone-mediated bone resorption, and is associated with osteoporosis and increased fracture risk.
- Other frequent laboratory abnormalities include abnormal liver function tests, leukocytosis, mild anemia, and low serum cholesterol levels.
- In **thyroiditis**, the diagnostic evaluation is more difficult. An **exquisitely tender gland** and **elevated erythrocyte sedimentation** rate (**ESR**) or **C-reactive protein** make the diagnosis of subacute thyroiditis likely. The other forms of thyroiditis lack these findings.
- **Factitious thyrotoxicosis** can often be diagnosed by **history**. Laboratory testing will demonstrate **low thyroglobulin levels and a low T3/T4 ratio** (<20 ng/microgram). Furthermore, **radioactive iodine uptake is depressed in thyroiditis and factitious thyrotoxicosis** but **increased** in **hyperthyroidism**.

Management

- **Management** of thyrotoxicosis is **based on etiology and symptom severity**. For those with **mild** symptoms, **outpatient** referral and management are appropriate.
- Patients with **moderate to severe** symptoms are best managed in the emergency department (ED) setting.
- **Treatment** is divided into **supportive, symptomatic, and thyroid-directed therapy**.
- The **order of medication administration** in thyroid storm is **critical**.
- **Iodine** can **precipitate thyroid storm** and must be **given a minimum of 1 hour after thionamide therapy** (PTU or methimazole).
- As such, the **typical order is beta blocker (propranolol), propylthiouracil (PTU), or methimazole, and then iodine (saturated solution of potassium iodide [SSKI], Lugol solution).**
- In addition, it is **important to identify and treat the precipitating cause of thyroid storm**.

1. Supportive Treatment

- **Supportive therapy** for thyroid storm patients should include **management** of **hyperthermia** with **cooling and acetaminophen**.
- **Aspirin** should be **avoided** in thyrotoxic patients because it decreases the protein binding of T4 and T3.
- **Agitation** is controlled with **benzodiazepines**.
- **Fluid resuscitation** is needed to compensate for insensible and gastrointestinal (GI) losses; **dextrose-containing solutions** are **helpful** because glycogen stores are often depleted.
- **Electrolyte replacement** is guided by laboratory values.

2. Symptomatic Treatment

- **Symptomatic treatment** consists primarily of **beta blockade** to diminish the adrenergic response.
- **Propranolol** is the beta blocker of **choice** because it has the **added benefit** of **blocking conversion** of T4 to T3; its **nonselective effects** also improve **tremor**, **weakness**, **hyperpyrexia**, **restlessness**, **irritability**, and **emotional lability**. The **onset of action** after **oral dosing is about 1 hour**.
- For **more rapid beta blockade**, propranolol can be administered intravenously (IV). A short-acting agent such as esmolol may be used when concerns about beta blockade exist, due to underlying asthma or COPD, or pulmonary edema from heart failure.
- In **asthmatics, a β1-selective drug such as esmolol or metoprolol may be considered**.
- If **beta blockers are contraindicated**, **reserpine, 2.5 to 5 mg intramuscularly** (IM) every 4 hours, is also an option.
- Patients should be **closely monitored for hypotension, regardless of the agent used**, because thyrotoxicosis can lower systemic vascular resistance and cause congestive heart failure.
- Patients who develop **clinically significant heart failure should be treated** with the usual medications for heart failure, including **diuretics and angiotensin-converting enzyme inhibitors**.
- **Atrial fibrillation is often refractory to rate control until antithyroid therapy is instituted.**

3. Thyroid-Directed Treatment

- Thyroid-directed therapy has three goals:
- 1/Reduce thyroid hormone production
- 2/Prevent thyroid hormone release
- 3/Block peripheral conversion of T4 to T3.
- In conjunction with this treatment, an additional goal is the avoidance of therapeutic interventions that may worsen thyrotoxicosis. As such, certain drugs should be avoided in the thyrotoxic patient.
- Amiodarone and iodinated contrast material both contain iodine and can increase thyroid hormone production.
- Aspirin can increase free thyroid hormone concentrations through its effect on protein binding.
- Pseudoephedrine, ketamine, and albuterol increase sympathetic tone and can exacerbate the adrenergic effects of thyrotoxicosis; when indicated, they **should be used cautiously**.

1/Reducing Thyroid Hormone Production

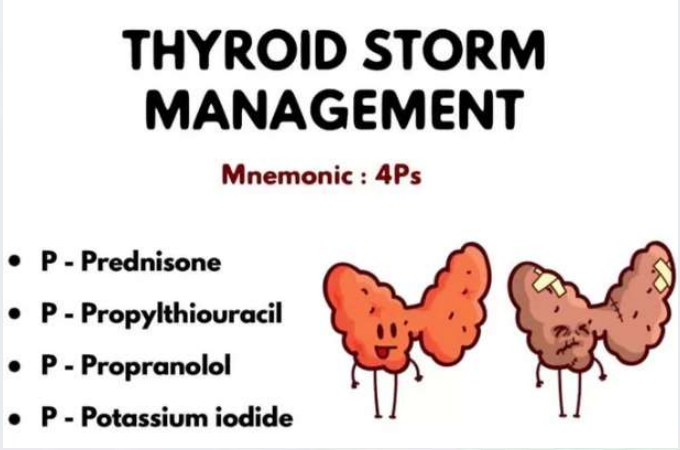
- **Thionamides** inhibit oxidation and organic binding of iodine to thyroglobulin, thereby blocking the synthesis of thyroid hormone.
- **PTU** or **methimazole** can be used.
- **PTU** has the **additional effect of impairing the conversion of T4 to T3**; **methimazole** has a **longer duration** of **action**.
- **Both** PTU and methimazole may be given by **nasogastric tube** or **rectum** as **needed**.
- **PTU is preferred in the first trimester** of **pregnancy**, and **methimazole** is **preferred in the second and third trimesters.**
- Methimazole is available in IV form outside the United States, but its use is not recommended by the most recent American Thyroid Association guidelines.
- **Side effects** of thionamide therapy vary from mild to life-threatening. These can range from urticarial, rash, arthralgia, GI upset, to agranulocytosis, hepatotoxicity and vasculitis.

2/Inhibiting Thyroid Hormone Release

- **Inorganic iodine blocks** the **release of stored thyroid hormone**.
- Because an iodine load can increase the synthesis of thyroid hormone, these agents **should not be administered until 1 hour after the initiation of PTU or methimazole therapy**.
- Traditionally, **oral iodine** in the form of **potassium iodide (SSKI), or Lugol solution**, is **administered**.
- Like the thionamides, these agents may be given via **nasogastric tube** or **retention enema**, as needed.
- **Lithium** may be considered an **alternative therapy for iodine-allergic patients**. **Lithium** is also the **agent of choice for iodine-induced hyperthyroidism**, which is usually the result of the administration of **amiodarone or iodinated contrast material**.

3/Inhibiting Conversion of T4 to T3

- **Corticosteroids** are capable of **inhibiting the peripheral conversion of T4 to T3** and **blocking** the **release of hormone from the thyroid gland**.
- When steroids are used in conjunction with PTU and iodide, the concentration of T3 can return to normal within 48 hours.
- **Hydrocortisone** may be **administered**, and **dexamethasone** is an **alternative**.



BOX 117.3 Management of Thyrotoxicosis

β-Adrenergic Blockade

Propranolol 60–80 mg PO every 4 hours

or

Metoprolol, 25–50 mg PO every 6 hrs

If IV route is required, propranolol, 0.5–1.0 mg IV slow push test dose, then repeat 1–2 mg every 15 min as tolerated to desired effect, then 1–2 mg every 3 hr

or

Esmolol, 50–100 μg/kg/min infusion

Strict contraindication to beta blocker—reserpine 2.5–5 mg IM every 4 hr

Inhibition of Thyroid Hormone Synthesis

Propylthiouracil, 500–1000 mg loading dose, then 250 mg every 4 hr

or

Methimazole, 60–80 mg/day in divided doses

Preferred route, PO or nasogastric (NG); alternative route: PR (in rectum), enema prepared by pharmacy; same dose for all routes

Inhibition of Thyroid Hormone Release

Saturated solution of potassium iodide (SSKI, 50 mg iodide/drop), 1–2 drops PO or PR tid

or

Lugol solution (8 mg iodide/drop), 5–7 drops PO or PR tid

or

Sodium iodide, dosing as per Endocrinology recommendations

or

If allergic to iodine, lithium carbonate, 300 mg PO or NG qid

Administration of Corticosteroids

Inhibit T_4 to T_3 conversion; treat relative adrenal insufficiency.

Hydrocortisone, 300 mg IV, followed by 100 mg tid

or

Dexamethasone, 2–4 mg IV qid

Miscellaneous Therapies

- **Cholestyramine**, an **anion exchange resin**, interrupts the **enterohepatic recirculation** of thyroid hormone by binding it in the bowel lumen. Because it may help to **decrease the level of circulating hormone more rapidly**, its use is recommended for **severe or refractory thyroid storm**.
- Although it has been shown to result in a more rapid decline in hormone levels compared with thionamides alone, it **requires weeks of therapy** and, as such, is reserved for **outpatient management**.
- **Plasmapheresis** has been used in thyroid storm as an attempt to remove circulating thyroid hormone.
- Extracorporeal membrane oxygenation (**ECMO**) has been recently described as successful in supporting patients with severe thyroid storm and "rapid clinical deterioration."
- **Neither radioactive iodine nor surgery plays a role in the management of thyroid storm** or **thyrotoxicosis** until a sustained euthyroid state has been established because these interventions can **precipitate thyroid storm**.

Miscellaneous Therapies

- **First-line treatment of subacute thyroiditis is the use of nonsteroidal antiinflammatory drugs (NSAIDs).**
- **Corticosteroids** are used in **refractory cases**.
- Symptomatic patients are treated with beta blockers.
- **AIT** is classified as type 1 or type 2. Type 1 AIT occurs in patients with underlying thyroid pathology such as autonomous nodular goiter or Graves' disease. Type 2 AIT is a result of amiodarone causing a subacute thyroiditis with release of preformed thyroid hormones into the circulation.
- In the case of **AIT-1**, treatment typically consists of **stopping amiodarone and initiating antithyroid therapy**. **AIT-2** is managed with **corticosteroids**, and the decision to stop amiodarone is made on a case-by-case basis.
- Management of drug-induced hyperthyroidism related to lithium involves stopping lithium. When related to immunomodulator therapy, discontinuation of the offending drug is not always mandatory.

Identification and Treatment of the Precipitating Event

- Thyroid storm is often precipitated by a physiologic stressor, such as infection, myocardial ischemia, pulmonary embolism, and stroke.
- Management of thyroid storm with PTU, followed by iodine, beta blockers, corticosteroids, fluid resuscitation, rapid cooling, and treatment of the precipitating illness, can resolve acute thyroid storm within 24 hours.

Diagnosis and Treatment of Underlying Precipitant

Consider empirical antibiotics if critical.

Supportive Measures

Volume resuscitation and replacement of glycogen stores with D_5/0.9 NS (dose varies, depending on volume status and CHF)

Acetaminophen

Cooling blanket, fans, ice packs, ice lavage

Miscellaneous

Lorazepam or diazepam as anxiolytic and to decrease central sympathetic outflow

Cholestyramine (blocks enterohepatic recirculation of thyroid hormone), 1–4 g PO twice daily for severe or refractory thyrotoxicosis

CHF, Congestive heart failure; *D_5W/0.9 NS,* 5% dextrose in 0.9% normal saline; *IV,* intravenously; *NG,* by nasogastric tube; *PO,* by mouth; *PR,* by rectum; *T_3,* triiodothyronine; *T_4,* thyroxine.

Special Situations

BOX 117.4 Thyrotoxicosis and Thyroid Storm Special Situations

Congestive Heart Failure

If rate-related, high-output failure:
- Beta blockade is first-line therapy (dose as in Box 117.3)
- ACEI, digoxin, diuretics as needed

If depressed ejection fraction:
- Avoid beta blocker or one-quarter dose
- ACEI if blood pressure adequate
- Digoxin and furosemide as needed

If pulmonary hypertension:
- Oxygen
- Sildenafil

Atrial Fibrillation

Beta blocker preferred for rate control (dose as in Box 117.3)

Calcium channel blockers prone to hypotension; diltiazem, 10-mg test dose.
- Avoid verapamil.

Digoxin less effective but may be tried

Amiodarone should be avoided because of iodine load

Refractory to conversion to sinus rhythm unless euthyroid first

Thyroiditis (Subacute)

NSAIDs for inflammation and pain control

Prednisone, 40 mg/day, if refractory to NSAIDs

Beta blockade to control thyrotoxic symptoms

No role for PTU, methimazole, or iodides

Factitious Thyrotoxicosis

Beta blockade for thyrotoxic symptoms

Cholestyramine to block absorption of ingested thyroid hormone

No role for PTU, methimazole, or iodides

ACEI, Angiotensin-converting enzyme inhibitor; *NSAIDs,* nonsteroidal antiinflammatory drugs; *PTU,* propylthiouracil.

Disposition

- The disposition of patients with hyperthyroidism is guided by symptom severity, with intensive care unit (ICU) admission for patients in thyroid storm.
- Patients with mild thyrotoxicosis controlled with a first-line medication who are otherwise stable can be managed as outpatients.
- Admission to the hospital may be appropriate for patients who require more than beta blockers for symptom control or whose symptoms persist despite therapy.
- Patients with rapid atrial fibrillation should be admitted to a monitored setting.
- Patients managed as outpatients can be sent to a primary care physician or referred to an endocrinologist.
- Lack of insurance and other socioeconomic factors have been linked to higher admission rates in patients with thyrotoxicosis.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Q&a