



Between the Extremes: A Tale of Two Thyroid Emergencies

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Disclosures

The planner(s) and speaker(s) have indicated that there are no relevant financial relationships with any ineligible companies to disclose.

Learning Objectives

At the end of this session, learners should be able to:

Recognize	the clinical features and diagnostic criteria of thyroid storm and myxedema coma
Outline	the recommended management strategies for thyroid storm and myxedema coma based on current guidelines
Identify	controversies with current diagnosis and management of thyroid storm and myxedema coma

Outline

Thyroid Hormone
Physiology

Thyroid Storm

- Epidemiology, Pathophysiology, Diagnosis
- Treatment & controversies

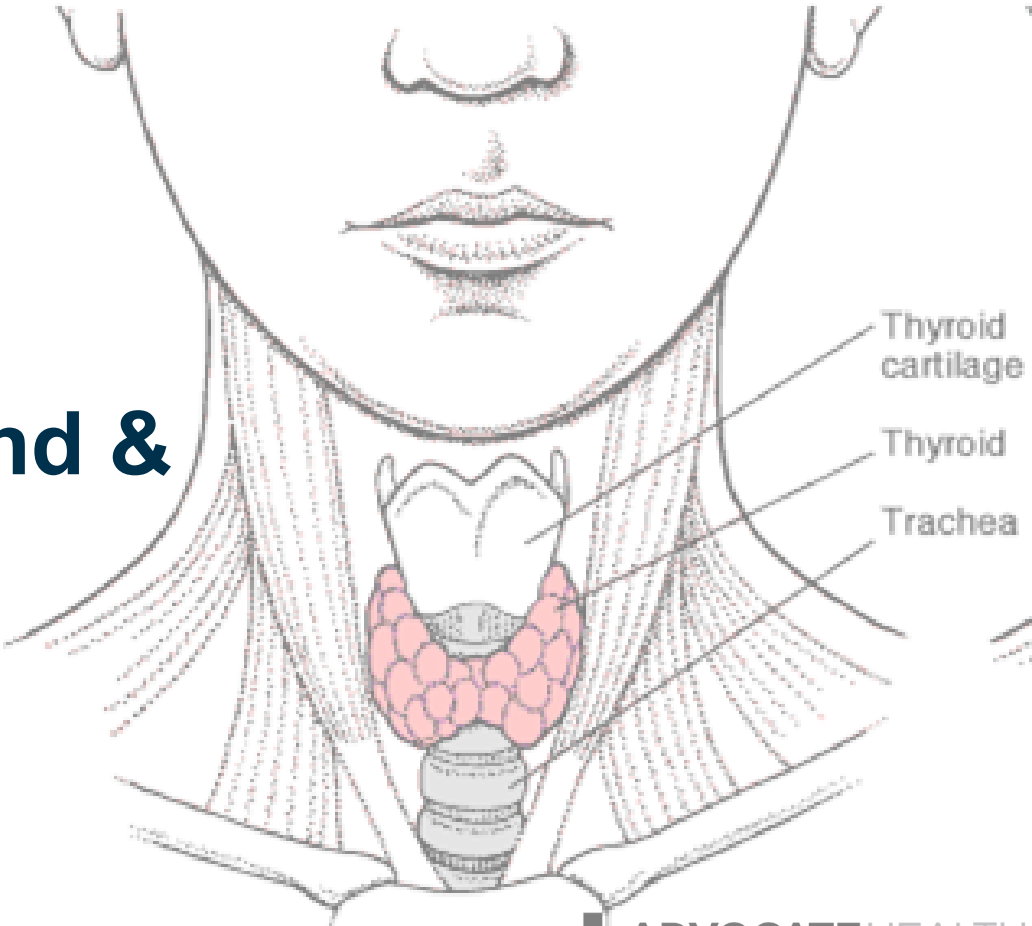
Myxedema coma

- Epidemiology, Pathophysiology, Diagnosis
- Treatment & controversies

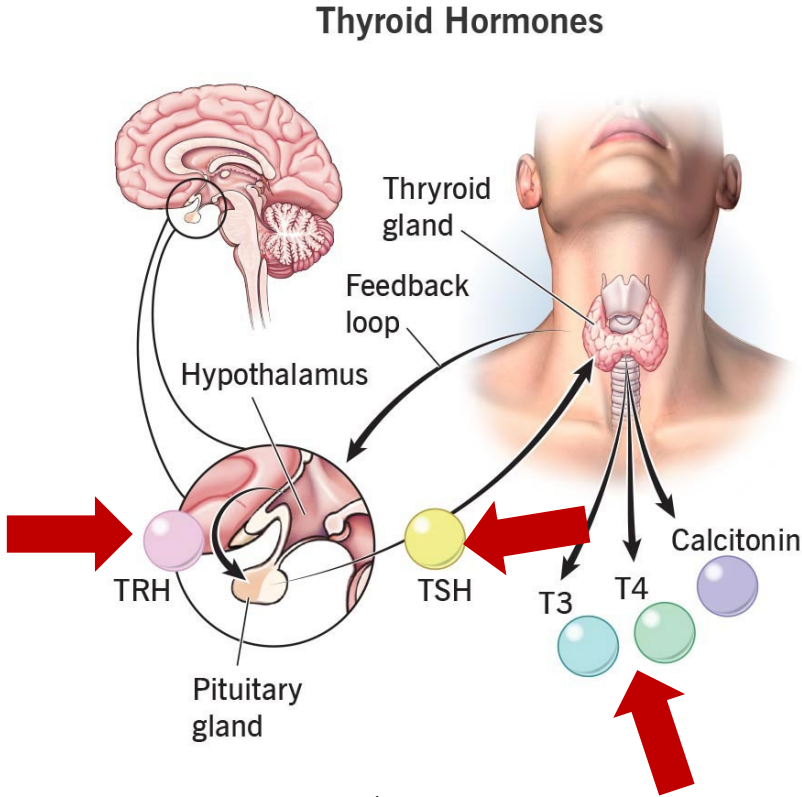
Abbreviation Key

- **ATA:** American Thyroid Association
 - **CAD:** coronary Artery Disease
 - **BWPS:** Burch-Wartofsky Point Scale
 - **CHF:** Congestive Heart Failure
 - **CNS:** Central nervous system
 - **DI:** deiodinase
 - **GI:** Gastrointestinal
 - **ICU:** Intensive care unit
 - **IV:** intravenous
 - **JTA:** Japanese Thyroid Association
 - **LD:** Loading dose
 - **LFT:** Liver function tests
 - **LT4:** Levothyroxine
 - **MMI:** Methimazole
 - **PTU:** propylthiouracil
 - **T3:** Triiodothyronine
 - **T4:** Thyroxine
 - **TG:** Thyroglobulin
 - **TH:** Thyroid hormones
 - **TPE:** therapeutic plasma exchange
 - **TPO:** Thyroid Peroxidase
 - **TRH:** Thyrotropin releasing hormone
 - **TS:** Thyroid storm
 - **TSH:** Thyroid stimulating hormone
-


Thyroid Gland & Hormones

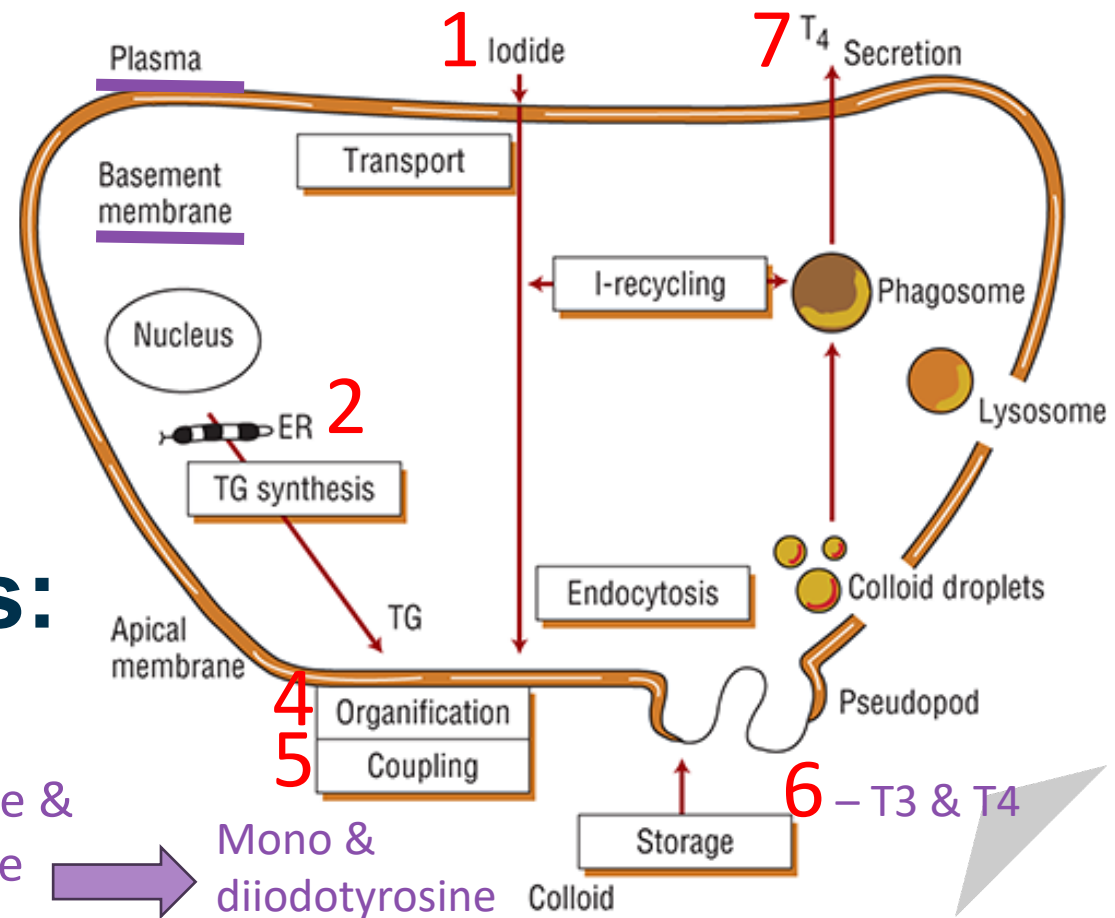


Hypothalamic-Pituitary-Thyroid Axis

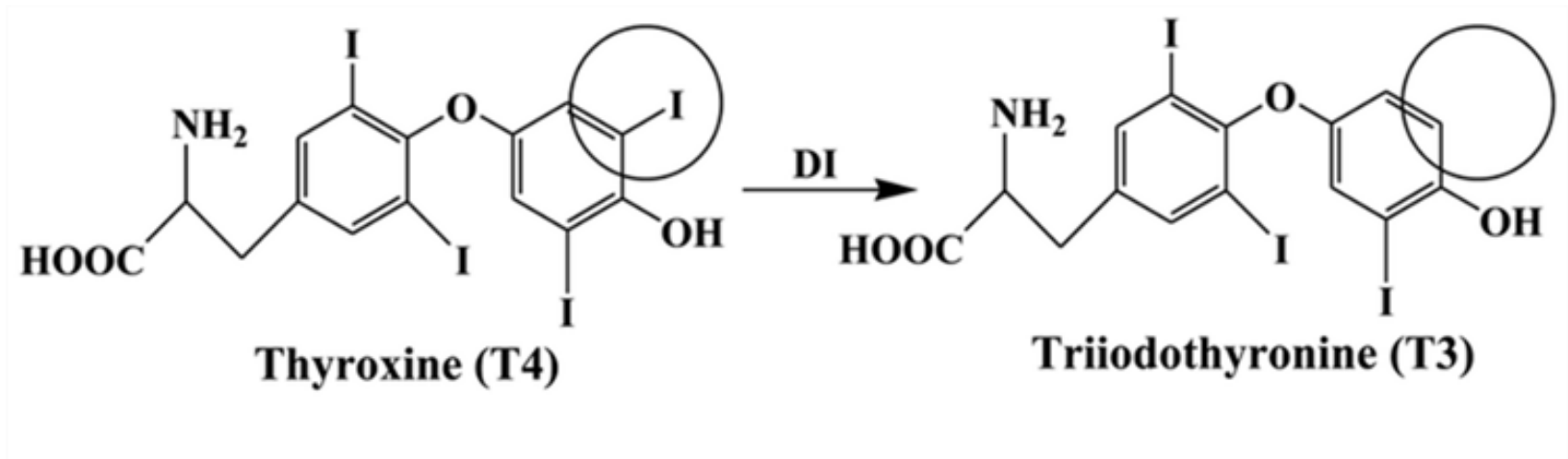


Thyroid hormone synthesis:

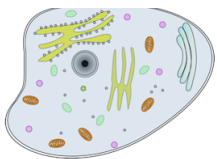
3 – activation of hydrogen peroxide & thyroid peroxidase (TPO)  Mono & diiodotyrosine



Thyroid Hormones (TH):



Physiologic Effect of TH:



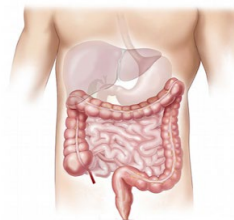
Protein Synthesis and Metabolic Activity in Cells



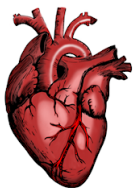
Body Metabolism
Body Heat



Neurogenesis
Myelination
Synaptogenesis
Dendrite Formation
Sympathetic Activity



GI Motility
GI Secretions



Beta-1 Receptors



Heart Rate
Stroke Volume

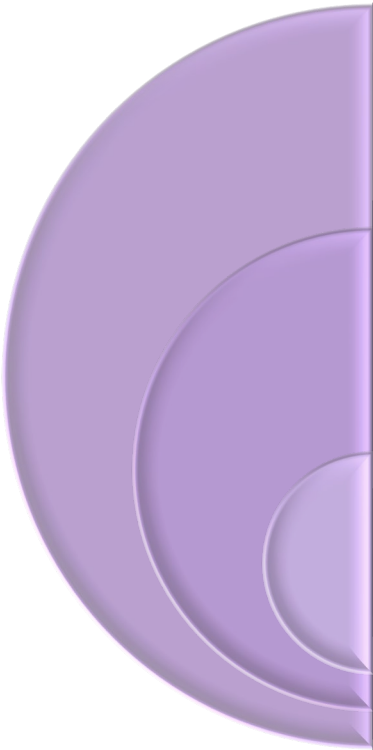


Cardiac Output
Blood Pressure



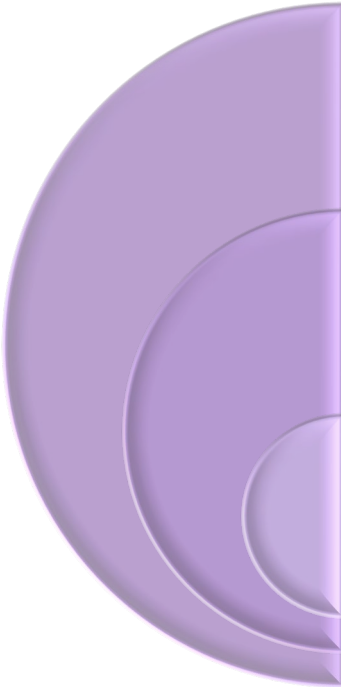
Proliferation of Skin Cells
Sweat & Skin Secretions
Hair Growth
Nail Growth

Hyperthyroidism/Thyrotoxicosis:



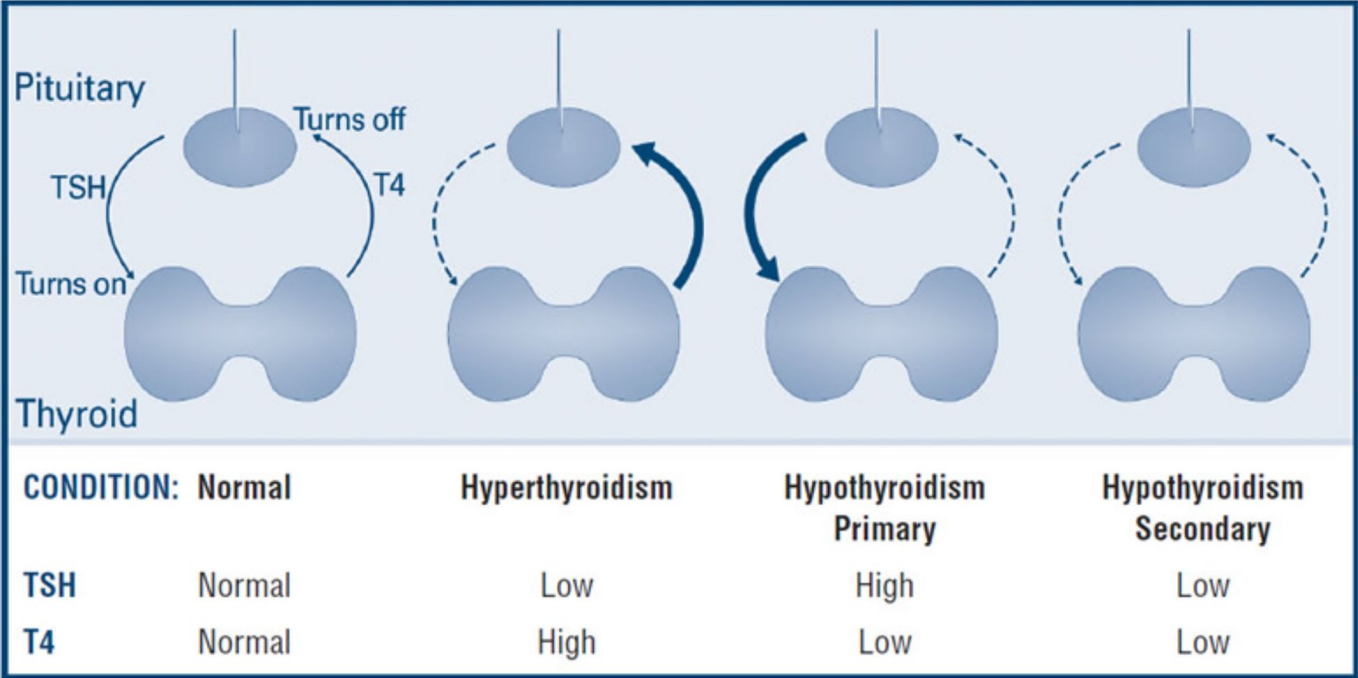
State of thyroid hormone excess
<p>Etiology:</p> <p>Most common: Graves' disease and toxic multinodular goiter</p> <p>Other causes: toxic adenoma, overproduction of TSH from a pituitary adenoma, thyroiditis, exogenous thyroid hormone ingestion, thyroid cancer, or human chorionic gonadotropin-mediated hyperthyroidism</p>
<p>Signs and symptoms: anxiety, fatigue, diaphoresis, heat intolerance, tremors, palpitations, tachycardia, weight loss, hyperreflexia, warm and moist skin</p>

Hypothyroidism:



State of low circulating thyroid hormone
Etiology: Hashimoto's thyroiditis, nutritional iodine deficiency, history of thyroidectomy, radioactive iodine therapy, or decreased TSH production by the pituitary
Signs and symptoms: fatigue, malaise, weight gain, dry and puffy skin, constipation, cold intolerance, altered cognition, and hyporeflexia

Thyroid Function Tests



Thyroid Storm



What is it?

- ❖ A life-threatening medical emergency
- ❖ State of extreme hyperthyroidism with multiorgan system involvement

Clinical Presentation Thyroid Storm:

Neuropsychiatric

Agitation/restlessness
Confusion, delirium, somnolence,
lethargy, coma
Hyperreflexia
Periodic paralysis
Psychosis
Apathy

Cardiovascular

Tachyarrhythmias

Dilated cardiomyopathy
Congestive heart failure
Chest discomfort

Respiratory

Dyspnea on exertion

Reproductive

Oligomenorrhea
Anovulation

Gastrointestinal

Nausea & vomiting
Diarrhea
Liver dysfunction and hepatomegaly
Jaundice

Thermoregulatory

Hyperthermia

pyrexia
Diaphoresis
Insensible fluid loss

Metabolic

Hyperglycemia
Hypercalcemia

Epidemiology

0.57-0.76 cases per 100,000 persons per year

16-17% of patients hospitalized with thyrotoxicosis

Predominantly seen in females given common cause is Graves disease which exhibits a female-to-male ratio between 4-10:1

1.2-3.6% mortality

Triggers

Disease state

- Cardiovascular: myocardial infarction, chronic heart failure, pulmonary embolism
- Infection
- Diabetic ketoacidosis

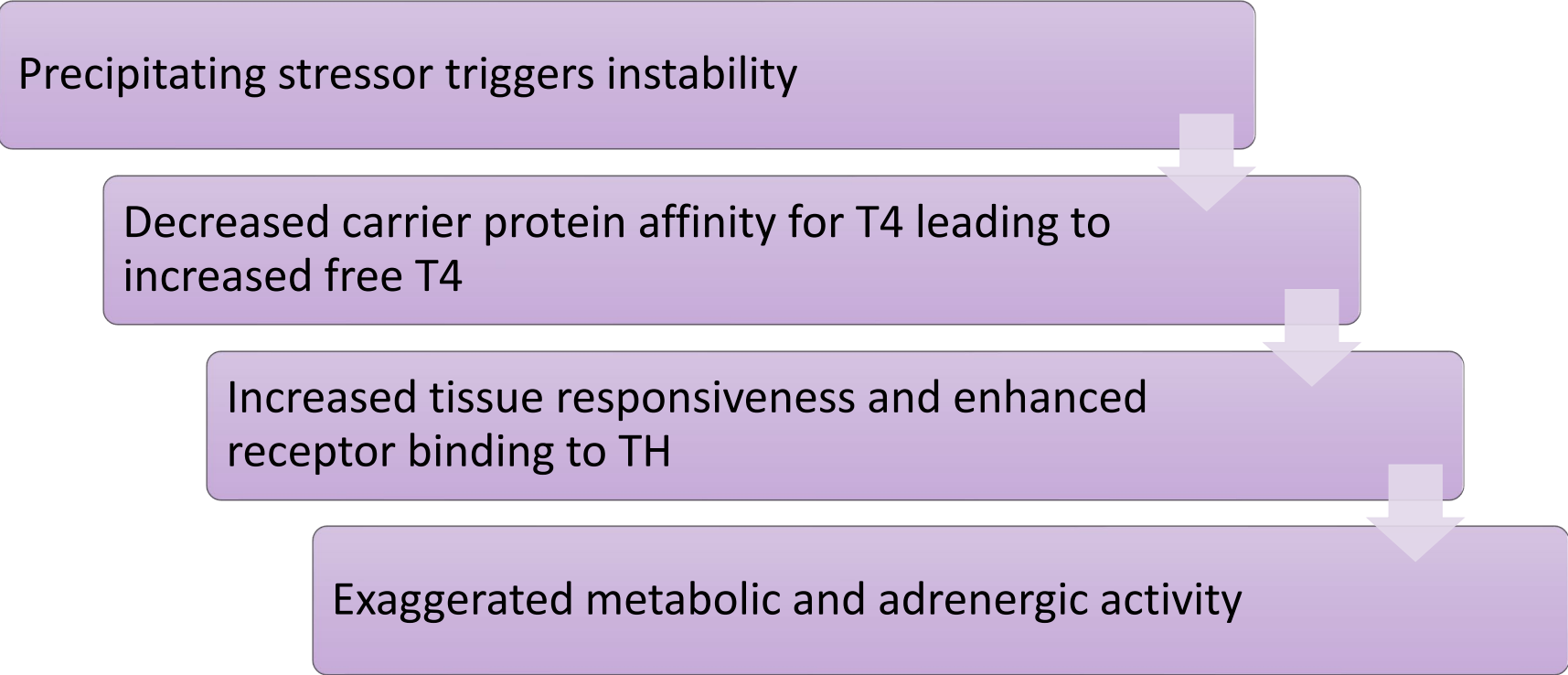
Medication related

- Stopping antithyroid drugs
- Iodinated contrast dye
- Salicylates, pseudoephedrine, and anesthetics

High stress

- Trauma
- Surgery
- Giving birth

Pathophysiology:



Diagnosis:

- Suspected in patients with thyrotoxicosis and evidence of multi-organ decompensation following a precipitating event
- Diagnostic criteria:
 - Burch-Wartofsky Point Scale (BWPS)
 - Japanese Thyroid Association (JTA) framework

BWPS

Criteria	Points	Criteria	Points
Thermoregulatory dysfunction		Gastrointestinal-hepatic dysfunction	
99.0-99.9 F	5	Absent	0
100.0-100.9 F	10	Moderate (diarrhea, abdominal pain, nausea/vomiting)	10
101.0-101.9 F	15	Severe (jaundice)	20
102.0-102.9 F	20		
103.0-103.9 F	25		
104 F	30		
Tachycardia		Central nervous system disturbance	
90-109 bpm	5	Absent	0
110-119 bpm	10	Mild (agitation)	10
120-129 bpm	15	Moderate (delirium, psychosis, extreme lethargy)	20
130-139 bpm	20	Severe (seizure, coma)	30
140 bpm	25		

BWPS

Criteria	Points
Congestive heart failure	
Absent	0
Mild (pedal edema)	5
Moderate (bibasilar edema)	10
Severe (pulmonary edema)	15
Atrial fibrillation	
Absent	0
Present	10
Precipitant history	
Negative	0
Positive	10

Total Score	Category
≥45	Thyroid storm
25-44	Impending storm
<25	Storm unlikely

JTA Definition and Diagnostic Criteria for Thyroid Storm

Prerequisite for diagnosis	Presence of thyrotoxicosis with elevated levels of free T3 or T4
Symptoms	<div><div>1.</div><div>Central nervous system manifestations (CNS): Restlessness, delirium, mental aberration or psychosis, somnolence or lethargy, coma</div><div>2.</div><div>Fever: ≥ 38 degrees Celsius</div><div>3.</div><div>Tachycardia: ≥ 130 beats per minute with or without atrial fibrillation</div><div>4.</div><div>Congestive heart failure (CHF): pulmonary edema, moist rales over more than half of the lung field, cardiogenic shock, or New York Heart Association Class IV status or \geq Class III in the Killip classification</div><div>5.</div><div>Gastrointestinal (GI)/hepatic manifestations: nausea, vomiting, diarrhea, or a total bilirubin level ≥ 3.0 mg/dL</div></div>
Exclusion and provisions	Cases are excluded if other underlying diseases clearly causing any of the following symptoms: fever, impaired consciousness, heart failure, and liver disorder





JTA Definition and Diagnostic Criteria for Thyroid Storm

Grade of TS	Combinations of Features	Requirements for Diagnosis
TS1	First combination	Thyrotoxicosis plus at least one CNS manifestation and one of the following: fever, tachycardia, CHF, or GI/hepatic manifestation
TS1	Alternate combination	Thyrotoxicosis and at least three of the following: fever, tachycardia, CHF, or GI/hepatic manifestation
TS2	First combination	Thyrotoxicosis and a combination of two of the following: fever, tachycardia, CHF, or GI/hepatic manifestations
TS2	Alternate combination	Meets the diagnostic criteria for TS1, except that serum free T3 or T4 levels are not available

Controversy with Use of Diagnostic Scores

JTA tends to underdiagnose cases compared to a BWPS ≥ 45

BWPS has been associated with overtreatment

	Sensitivity	Specificity	Interpretation
BWPS			Low risk of false negative, high risk of false positives
JTA			Low risk of false positive, high risk of false negatives

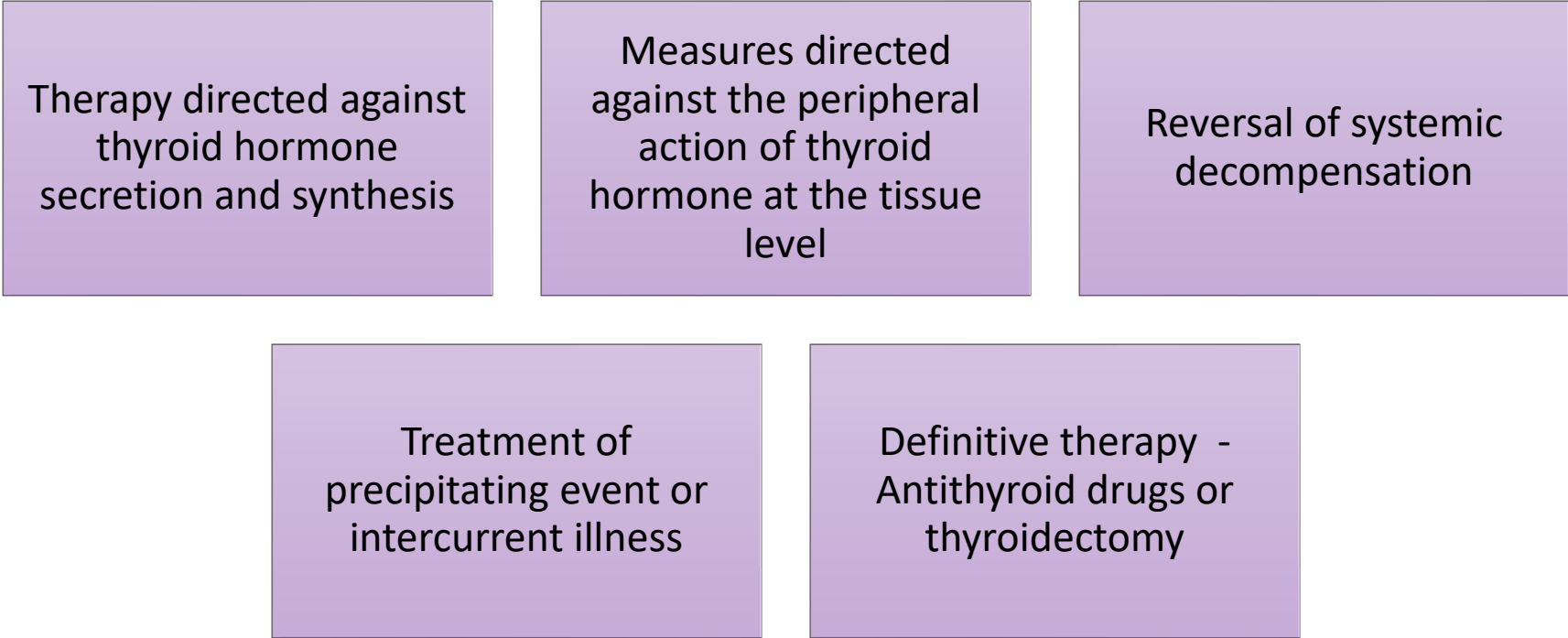
Treatment Guidelines for Thyroid Storm

2016 American Thyroid Association Guidelines for Diagnosis and Management of Hyperthyroidism and Other Causes of Thyrotoxicosis

Diagnosis

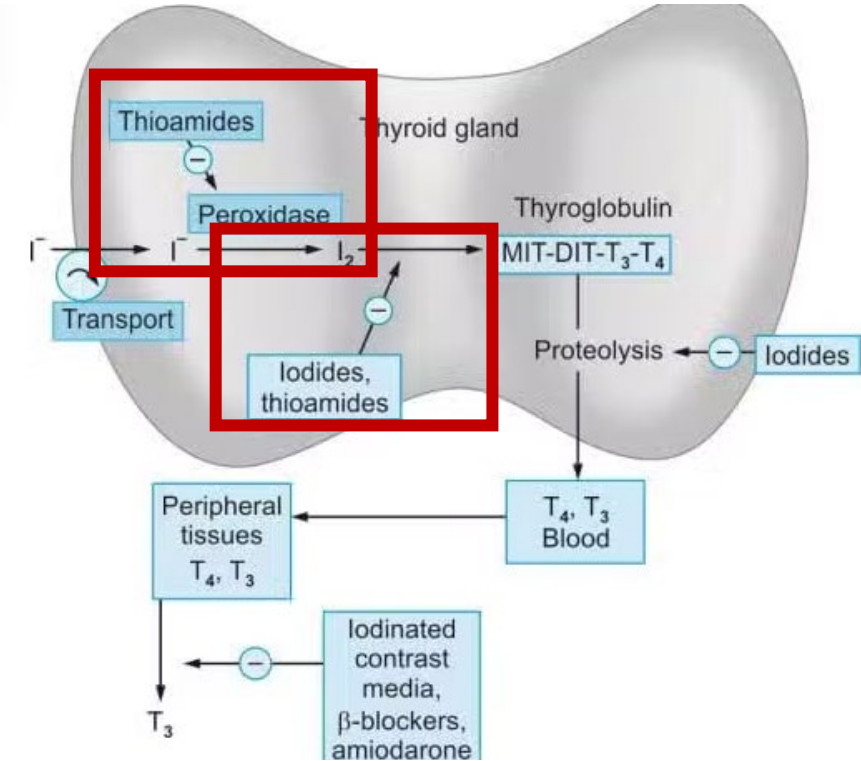
- Diagnosis should be made clinically, adjunctive use of sensitive diagnostic systems (BWPS & JTA) should be considered
- A BWPS of ≥ 45 or JTA categories of thyroid storm 1 or 2 with evidence of systemic decompensation require aggressive therapy
- The decision to use aggressive therapy in patients with BWPS of 25-44 should be based on clinical judgment

2016 American Thyroid Association Guidelines for Diagnosis and Management of Hyperthyroidism and Other Causes of Thyrotoxicosis



Thioamides

- Propylthiouracil, methimazole, carbimazole
- MOA: suppresses Thyroid Peroxidase (TPO) mediated steps in thyroid hormone synthesis
- Propylthiouracil also inhibits peripheral conversion of T4 to T3



Methimazole (MMI)

Role	<ul style="list-style-type: none">• Inhibit synthesis of new thyroid hormone
Dose	<ul style="list-style-type: none">• PO: 20 mg every 4 to 6 h, then 20 mg 1-2 per day once stable• *May be given via nasogastric tube or rectally
Pharmacokinetics	<ul style="list-style-type: none">• Time to peak 1-2 h, half-life 4-6 h, metabolized hepatically
Dosage form	<ul style="list-style-type: none">• Oral: tablet
Adverse reactions	<ul style="list-style-type: none">• Nausea, vomiting, headache, fever, joint pain, pruritus, edema, aplastic anemia, agranulocytosis, reversible hepatotoxicity
Monitoring	<ul style="list-style-type: none">• LFTs at baseline; thyroid function tests every 4-6 wks, then every 2-3 months once in range

Propylthiouracil (PTU)

Role	<ul style="list-style-type: none">• Inhibit synthesis of new thyroid hormone & peripheral conversion of T4 to T3
Dose	<ul style="list-style-type: none">• PO: 500 to 1000 mg loading dose, followed by 200 to 250 mg every 4 h• *May be given via nasogastric tube or rectally
Pharmacokinetics	<ul style="list-style-type: none">• Time to peak: 1-2 h, half-life ~1 h, metabolized in the liver
Dosage form	<ul style="list-style-type: none">• Oral: tablet
Adverse reactions	<ul style="list-style-type: none">• <u>Boxed warning for hepatotoxicity</u>; inhibition of myelopoiesis, fever, lupus-like syndrome
Monitoring	<ul style="list-style-type: none">• LFTs at baseline; thyroid function tests every 4-6 wks, then every 2-3 months once in range
Pearl	<ul style="list-style-type: none">• Preferred by the ATA guidelines

T3 & T4 in Hyperthyroidism. Comparison of Acute Changes During Therapy with Antithyroid Agents

PTU
(±iodide)

- Rapid drop in T3 within 24-48h
- Inhibits T4 → T3 peripheral conversion

MMI
(±iodide)

- Gradual T3 decline (by day 3-5)
- Inhibits thyroid hormone synthesis but has no effect on peripheral conversion of TH

Abuid J, et al. T3 & T4 in hyperthyroidism. *The Journal of clinical investigation*. 1974.¹⁹

Comparison of PTU vs MMI for Thyroid Storm in Critically Ill patients

Study Desing

- Multicenter, retrospective, comparative effectiveness

Intervention

- Patients were assigned to the exposure group based on the first thioamide therapy received

Primary Endpoint

- Composite of in-hospital death or discharge to hospice

Comparison of PTU vs MMI for Thyroid Storm in Critically Ill patients

Methods

- Looked at Premier Healthcare Database 1/1/2016 - 12/31/2020
- Inclusion criteria:
 - 18 years or older
 - Admitted to an intermediate care unit or ICU on first or second day of hospitalization
 - Had an ICD-10 code for thyroid storm
 - Received either PTU or MMI (but not both) on the first or second day of hospitalization

Comparison of PTU vs MMI for Thyroid Storm in Critically Ill patients

Results

- Analysis included 1383 patients (656 PTU, 727 MMI)
- Mean age 45 years, 71.8% women
- Composite of in-hospital death or discharge to hospice
 - MMI 6.3% (95% CI 4.6-8.1)
 - PTU 8.5% (95% CI 6.4-10.7)
 - Adjusted difference 0.6 (95% CI -1.8 – 3.0), $p = 0.64$

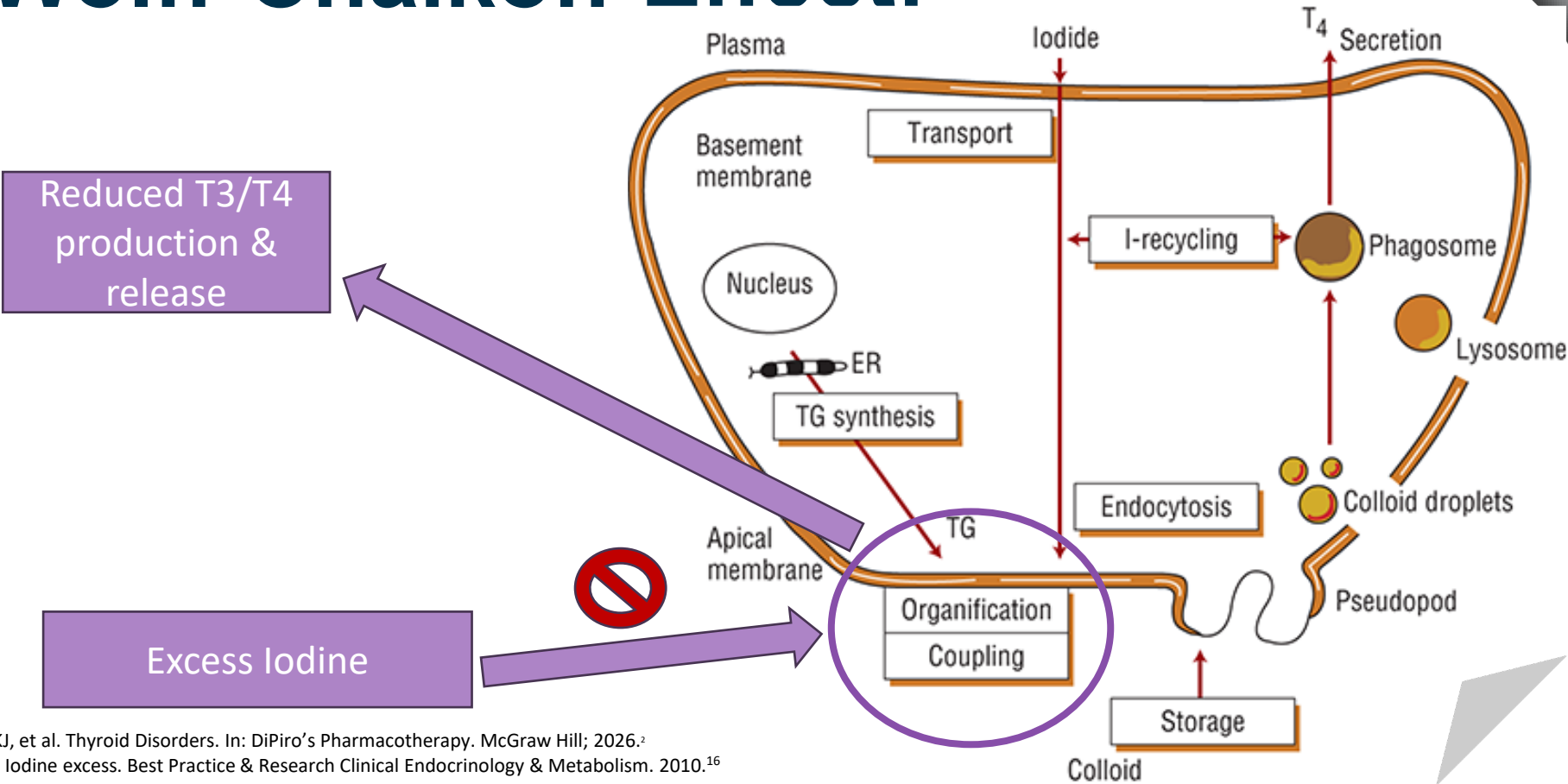
Conclusion

- PTU and MMI can be used interchangeably in the management of thyroid storm

Potassium Iodide

MOA	<ul style="list-style-type: none">Temporarily inhibits thyroid hormone synthesis and secretion into circulation
Dose	<ul style="list-style-type: none">solution: 5 drops (250 mg or 0.25 mL) every 6 h
Dosage form	<ul style="list-style-type: none">Oral: tablet, solution
Adverse reactions	<ul style="list-style-type: none">Cardiac arrhythmias, skin rash, diarrhea, vomiting, abdominal pain
Monitoring	<ul style="list-style-type: none">Thyroid function tests
Pearl	<ul style="list-style-type: none">Administer 1 h after antithyroid agent

Wolff-Chaikoff Effect:



Propranolol

MOA	<ul style="list-style-type: none">• Non-selective beta-adrenergic blocker
Role	<ul style="list-style-type: none">• Symptomatic support: tachycardia, palpitations, tremor and/or nervousness• May prevent peripheral conversion of T4 to T3
Dose	<ul style="list-style-type: none">• Oral: 60 to 80 mg every 4 to 6 h adjusted based on heart rate and blood pressure• IV: 0.5-1 mg over 10 min; may repeat dose of 1-3 mg over 10 to 15 min every few hours until able to switch to oral
Dosage form	<ul style="list-style-type: none">• Oral: IR tablet, ER capsule, solution• IV: push
Adverse reactions	<ul style="list-style-type: none">• Bradyarrhythmias, bronchospasm, fatigue, insomnia
Monitoring	<ul style="list-style-type: none">• Heart rate and blood pressure

Hydrocortisone

MOA	<ul style="list-style-type: none">• Short-acting corticosteroid with minimal sodium-retaining potential
Role	<ul style="list-style-type: none">• May block T4 to T3 conversion• Prophylaxis against relative adrenal insufficiency
Dose	<ul style="list-style-type: none">• 300 mg loading dose, followed by 100 mg every 8 h
Dosage form	<ul style="list-style-type: none">• Oral: capsule, tablet, and solution• IV: push
Adverse reactions	<ul style="list-style-type: none">• High blood sugar, increased blood pressure, mood and behavior changes
Monitoring	<ul style="list-style-type: none">• Serum glucose, blood pressure

Plasmapheresis – How it Works

- Removes potential pathological substances from the thyroid storm: hormones, autoantibodies, catecholamines, cytokines, and toxins
- Removes free thyroid hormones
- Replaces carrier proteins by unsaturated bound proteins of the replacement solution
- Removes 5'-monodesiodase which converts T4 to T3 hence lowering T3 production

Case Reports on Plasmapheresis (TPE):

	Case 1	Case 2	Case 3
Past medical history	Ventricular tachycardia (on amiodarone)	Hypertension, atrial fibrillation (on amiodarone)	Multinodular goiter (radioactive iodine 10 yrs prior), recent valve surgery
Presentation	Cardiac symptoms	Confusion, fever, tachyarrhythmias, dyspnea, loss of consciousness	Tachycardia, tachypnea, confusion, atrial fibrillation, respiratory failure
BWPS score (≥45: thyroid storm)	20-140	100-140	75-140
Initial treatment	Carbimazole, propranolol, potassium perchlorate, corticosteroids	Carbimazole, prednisone, propranolol, potassium perchlorate	Carbimazole, propranolol

Case Reports on Plasmapheresis (TPE):

	Case 1	Case 2	Case 3
Plasmapheresis	Started on day 16; 4 sessions done in 8 days	Started on day 2; completed 6 sessions	Started for worsening respiratory status; completed 4 sessions
Effect on thyroid hormone	Free T4 was reduced by 65% after 4 sessions	Rapid T3/T4 reduction after each session	Hormone levels normalized
Clinical outcome	Stable 2 weeks later, arrhythmias resolved, thyroidectomy performed	Regained consciousness in 3 weeks & muscle tone in 1 month; required tracheostomy	Clinical improvement and respiratory recovery

Discussion of Findings

- In all three cases plasmapheresis decreased both thyroid hormone level and controlled the cardiac symptoms within a few days when conventional therapy failed
- Severe neurological impairments may require a longer time after TPE session to improve
- Variable findings in other reports, some showing success and others finding no benefit
- Can be used as rescue therapy if other treatments are contraindicated or ineffective

Thyroid Storm Treatment Summary:

Drug	Dosing	Role in therapy
Thioamides:		
Propylthiouracil	PO: 500-1000 mg load, then 250 mg every 4 h	Blocks T4 to T3 conversion
Methimazole	PO: 20 mg every 4 to 6 h	Blocks new hormone synthesis
Propranolol	60-80 mg orally every 4h or 1 mg IV over 10 min	Blocks T4 to T3 conversion in high doses
Iodide (saturated solution of potassium iodide)	5 drops (0.25 mL or 250 mg) orally every 6 h; started 1 h after antithyroid drug	Blocks new thyroid hormone synthesis and release
Hydrocortisone	300 mg IV load, then 100 mg every 8 h	May block T4 to T3 conversion Prophylaxis against relative adrenal insufficiency

Treatment Considerations in Pregnancy:

Most common causes: Grave's disease and human chorionic gonadotropin (hCG)-mediated hyperthyroidism

PTU is the drug of choice for initial management

Hydrocortisone is preferred over dexamethasone

Esmolol is 1st line beta-blocker, propranolol is 2nd line; avoid atenolol

After resolution, PTU if in 1st trimester or switch to MMI if in 2nd trimester

Assessment Question #1

Which of the following is the most appropriate initial management strategy for a patient presenting with thyroid storm?

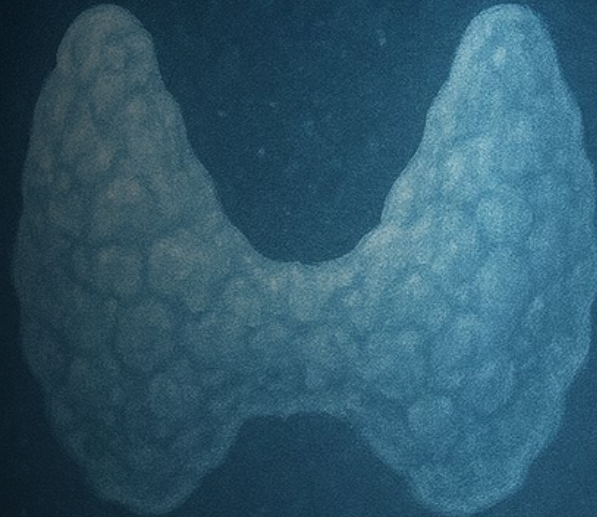
- A. Immediate administration of levothyroxine and beta-blockers
- B. Delay treatment until T3 and T4 levels are confirmed
- C. Send the patient for immediate thyroidectomy
- D. Administer therapy directed against thyroid hormone secretion and synthesis

Assessment Question #2

Which of the following is a key clinical consideration when choosing MMI over PTU in thyroid storm?

- A. Methimazole can be given IV in critically ill patients
- B. Methimazole has a lower risk of hepatotoxicity and may be preferred if propylthiouracil toxicity is a concern
- C. Methimazole uniquely blocks peripheral T4 to T3 conversion
- D. Methimazole is faster-acting than propylthiouracil, making it superior in emergent settings

Myxedema Coma



What is it?

A state of decompensated hypothyroidism resulting in widespread multiorgan dysfunction occurring after prolonged thyroid hypofunction

Clinical Presentation Myxedema Coma:

Neuropsychiatric

Confusion, obtundation, stupor, coma

Poor memory
Cognitive dysfunction
Depression
Psychosis

Cardiovascular

Bradycardia
Hypotension
Cardiomegaly
Pericardial effusion
Nonspecific S-T changes
Bundle branch blocks

Respiratory

Hypoxia
Hypercapnea
Pleural effusion
Laryngeal myxedema

Renal

Hyponatremia

Anasarca
Acute kidney injury
due to rhabdomyolysis

Gastrointestinal

Abdominal pain
Nausea
Constipation
Paralytic ileus

Thermoregulatory

Hypothermia

Hematologic

Normocytic anemia
Leukopenia
Acquired von Willebrand's disease

Metabolic

Hypoglycemia
Hypocholesterolemia

Epidemiology

- 2.56 cases per 1 million people per year
- Most common in elderly women with long-standing preexisting hypothyroidism
- Mortality rate: 6.8% - 29.5%

Triggers

Disease state

- Cardiovascular: myocardial infarction, chronic heart failure, pulmonary embolism
- Cerebrovascular accident
- Infection
- Diabetic ketoacidosis

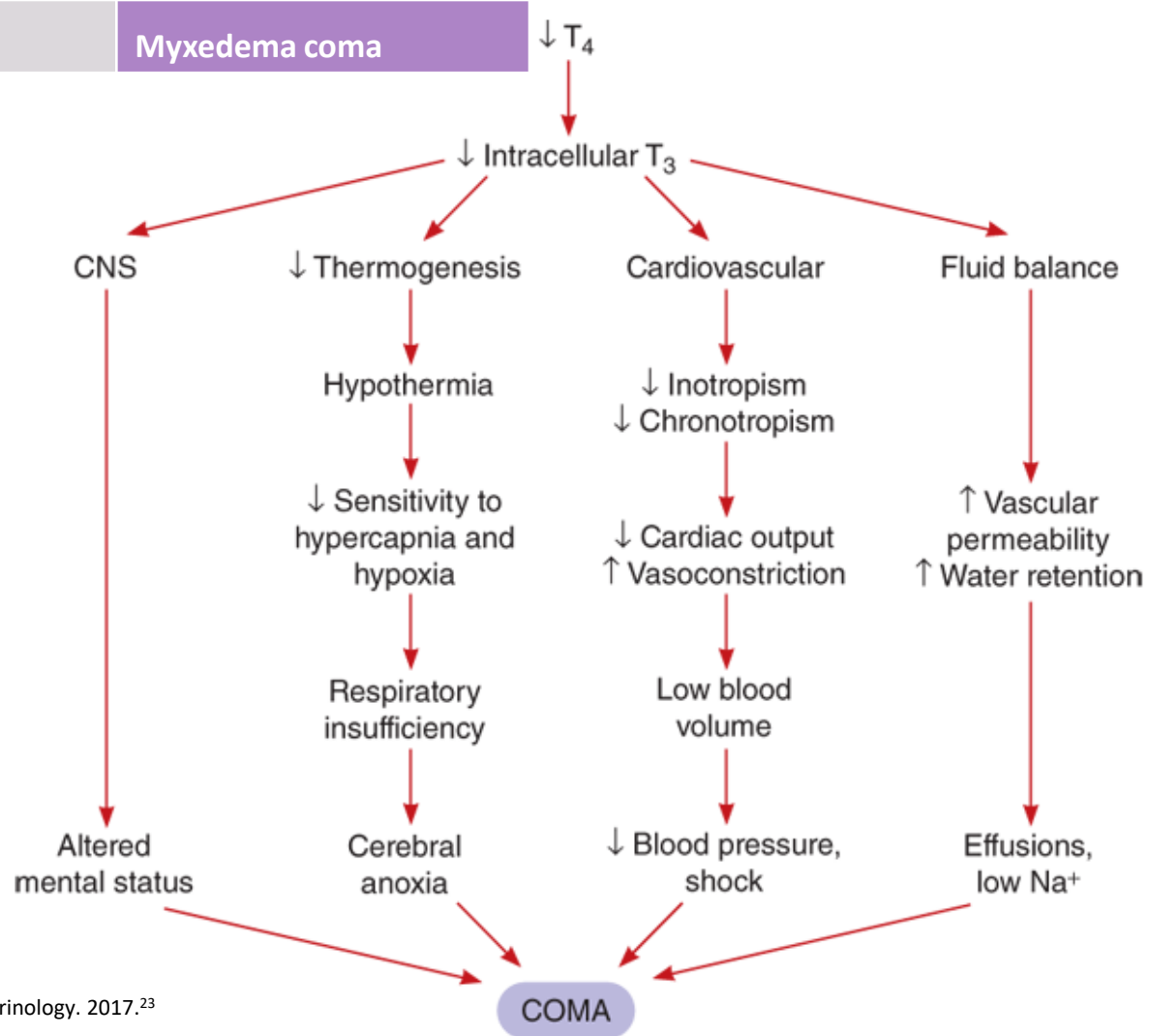
Medication related

- Sedatives
- Narcotics
- Anesthetics
- Amiodarone
- Lithium
- Radioactive iodine

Other

- Trauma
- Surgery
- Low temperatures
- Cruciferous vegetables

Pathophysiology



Diagnosis:

- Diagnosis of exclusion: Investigate non-thyroidal precipitating causes
- Suspected in patients with hypothyroidism presenting with altered mental status and hypothermia
- Scoring systems more sensitive than specific
- No biochemical pattern that reliably distinguished compensated severe hypothyroidism from myxedema coma

Diagnostic Scoring System:

Criteria	Score	Criteria	Score
Temperature (Fahrenheit) <div> >95 89.6-95 <89.6 </div>	<div>0</div> <div>10</div> <div>20</div>	Central Nervous System Effects <div> Absent Somnolent/Lethargy Obtunded Stupor Coma/seizure </div>	<div>0</div> <div>10</div> <div>15</div> <div>20</div> <div>30</div>
Gastrointestinal Findings <div> Anorexia/abdominal pain/constipation Decreased intestinal motility Paralytic ileus </div>	<div>5</div> <div>15</div> <div>20</div>	Precipitating Event <div> Absent Present </div>	<div>0</div> <div>10</div>

Diagnostic Scoring System:

Criteria	Score
Cardiovascular Dysfunction	
Bradycardia/Heart rate	
Absent	0
50-59	10
40-49	20
<40	30
EKG changes	10
Pericardial/pleural effusion	10
Pulmonary edema	15
Cardiomegaly	15
Hypotension	20
Metabolic Disorders	
Hyponatremia	10
Hypoglycemia	10
Hypoxemia	10
Hypercarbia	10
Decrease in GFR	10

Total Score	Category
>60	Highly suggestive/diagnostic of myxedema coma
25-59	Supportive of diagnosis of myxedema coma
<25	Myxedema coma unlikely

Popoveniuc G, et al. Diagnostic Scoring System. Endocrine Practice. 2014.²⁴

ATA Guidelines for Treatment of Hypothyroidism

IV glucocorticoids at stress doses prior to levothyroxine administration

IV levothyroxine

Liothyronine adjunct therapy for decreased T3 activation

Supportive Treatment:

Electrolyte and glucose replenishment

Passive rewarming with a blanket

Treatment of underlying comorbidities, including infection

Hydrocortisone:

MOA	<ul style="list-style-type: none">• Short-acting corticosteroid
Role	<ul style="list-style-type: none">• Prevent adrenal insufficiency
Dose	<ul style="list-style-type: none">• IV: 100 mg every 8 h until exclusion of possible adrenal insufficiency
Dosage form	<ul style="list-style-type: none">• Oral: capsule, tablet, and solution• IV: push
Adverse reactions	<ul style="list-style-type: none">• High blood sugar, increased blood pressure, mood and behavior changes
Monitoring	<ul style="list-style-type: none">• Serum glucose, blood pressure

Levothyroxine

MOA	<ul style="list-style-type: none">• Synthetic T4
Role	<ul style="list-style-type: none">• Replete T4
Dose	<ul style="list-style-type: none">• IV: initial loading dose 200 to 400 mcg as a slow bolus; followed by a daily replacement dose of 50 to 100 mcg
Dosage form	<ul style="list-style-type: none">• Oral: capsule, tablet, solution• IV: push
Adverse reactions	<ul style="list-style-type: none">• Palpitations, tachycardia, dyspnea, anxiety
Monitoring	<ul style="list-style-type: none">• Heart rate, blood pressure, thyroid function tests every 1-2 days
Pearl	<ul style="list-style-type: none">• Safe to give prior to thyroid function test results

Oral Levothyroxine as an Effective Option for Myxedema Coma

Study Design

- Retrospective, observational
- Included patients from 01/2010 to 12/2019

Intervention

- Oral LT4 given through nasogastric tube
- LD was stratified based on cardiac status
 - No CAD: LD 500 mcg followed by 200 mcg per day for 2 days, then 150 mcg daily for 2 days, then a MD of 1.6-2 mcg/kg at discharge
 - CAD with normal LVEF: LD 300-400 mcg, followed by the same taper as above
 - CAD with low LVEF (<60%): LD of 250-300 mcg, followed by 150 mcg daily for 2 days, then 100 mcg daily for 2 days, then MD

Primary Endpoint

- Survival rate related to effectiveness of oral LT4

Oral Levothyroxine as an Effective Option for Myxedema Coma

Results

- A total of 14 patients were included (10 males, 3 females, and one eleven-year-old boy) were included in the analysis
- Median age 67.5 years
- Survival rate: 13 out of 14 patients survived

Conclusion

- Oral LT4 is an effective treatment option for myxedema coma when intravenous LT4 is unavailable

Liothyronine

MOA	• Synthetic T3
Role	• Repletes active thyroid hormone
Dose	<ul style="list-style-type: none">• IV: Initial loading dose of 5 to 20 mcg as a slow bolus, followed by 2.5 to 10 mcg every 8 hours• Lower dosing used in smaller or older adults and those with a history of or at risk for coronary artery disease or arrhythmia
Dosage form	<ul style="list-style-type: none">• Oral: tablet• IV: push
Adverse reactions	• Acute myocardial infarction, angina pectoris, cardiac arrhythmias, tachycardia
Monitoring	• Heart rate, blood pressure, thyroid function tests every 1-2 days
Precaution	• Avoid use, or use with caution in patients with a history of heart disease or arrhythmia, or compromised cardiac dysfunction

Levothyroxine vs Liothyronine

No studies to compare given rarity and high mortality risk

Consider patients cardiac history

Liothyronine should not be used empirically, wait for thyroid labs

Consider as an adjunct in more severe cases and discontinue once stable

Management of Myxedema Coma Summary:

Drug	Dosing	Role in Therapy
Hydrocortisone	100 mg IV every 8 hours	Prevent adrenal insufficiency
Levothyroxine	IV: initial loading dose 200 to 400 mcg as a slow bolus; followed by a daily replacement dose of 50 to 100 mcg	Replete T4
Liothyronine	Initial loading dose of 5 to 20 mcg as a slow bolus, followed by 2.5 to 10 mcg every 8 hours *Lower end of dosing used in smaller or older adults and those with a history of or at risk for coronary artery disease or arrhythmia	Replete T3

Assessment Question #3

What type of electrolyte abnormality do patients with myxedema coma typically have?

- A. Hyperkalemia
- B. Hyponatremia
- C. Hypokalemia
- D. Hypernatremia

Assessment Question #4

Which of the following is the most appropriate initial treatment for a patient with myxedema coma?

- A. Administer methimazole followed by a beta-blocker
- B. Administer IV glucocorticoids followed by levothyroxine
- C. Send the patient for immediate thyroidectomy
- D. Delay treatment until T3 and T4 levels are confirmed

Key Takeaways:

- Thyroid hormone is vital for the function of all organ systems
- Thyroid storm and myxedema coma are diagnoses of exclusion
- In thyroid storm the goal is to stop excess thyroid hormone synthesis and block its effect on the body
- In myxedema coma the goal is to replete thyroid hormone

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Questions?

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