



## Chapter 128 – Thyroid and Adrenal Disorders

### Episode overview

#### Core questions:

1. List 8 causes of thyrotoxicosis
2. List 5 ophthalmologic findings in hyperthyroidism / Graves'
3. List the diagnostic criteria for thyroid storm
4. List 4 precipitants of thyroid storm
5. What is the treatment of thyroid storm?
6. List 8 causes of hypothyroidism and 10 signs and symptoms
7. List 5 lab abnormalities, 2 ECG findings, and 1 CXR finding in hypothyroidism
8. Describe 5 key clinical features of myxedema coma and list 8 precipitating factors
9. Describe the treatment of myxedema coma
10. List the hormones produced by the adrenal glands
11. List 3 effects of aldosterone
12. List 5 causes of chronic primary adrenal insufficiency + 1 acute + 3 secondary
13. Describe the treatment of acute adrenal insufficiency

### WiseCracks

1. What is apathetic thyrotoxicosis?
2. What is the analgesic / antipyretic of choice in thyroid storm? Which should you avoid?
3. Explain the thyroid lab tests and their clinical significance
4. What are some special situations in the management of thyroid storm?

### Key Concepts

#### Hyperthyroidism

- ❑ Thyroid hormone exerts effects on nearly every organ system. A high degree of suspicion is needed to diagnose hyperthyroidism.
- ❑ The laboratory evaluation of choice is determination of the TSH concentration with free T4 and T3 levels. Total T4 and T3 levels are of limited value.
- ❑ Thyroid storm is a life-threatening thyrotoxic crisis that requires prompt recognition and therapy, as well as identification and treatment of any precipitating cause, such as infection.
- ❑ 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), PTU or methimazole, and then iodine (SSKI, Lugol's solution).



### Hypothyroidism

- ❑ Hypothyroidism results from lack of stimulation of the thyroid gland (central or secondary hypothyroidism) or intrinsic gland dysfunction limiting hormone production (primary hypothyroidism).
- ❑ Signs and symptoms of hypothyroidism range from asymptomatic to overt organ failure, which can lead to death.
- ❑ Determination of an elevated TSH level is the most sensitive and single best screening test to confirm the diagnosis of primary hypothyroidism.
- ❑ Replacement with levothyroxine (T4) remains the treatment of choice and resolves most physical and psychological signs and symptoms in most patients.
- ❑ Myxedema coma is a life-threatening event that is most often precipitated by some stressful event in patients with untreated or undertreated hypothyroidism. Treatment with thyroid hormone replacement must be initiated, often solely on clinical findings.

### Adrenal Insufficiency

- ❑ Clinical manifestations of primary and secondary adrenal insufficiency may be vague and nonspecific and require a high index of suspicion for diagnosis.
- ❑ Predominant complaints include fatigue, weakness, dizziness, nausea, vomiting, and other nonspecific GI symptoms.
- ❑ Patients with primary adrenal insufficiency characteristically have more pronounced clinical manifestations and skin hyperpigmentation. Measurement of cortisol in the ACTH stimulation test is the standard and most convenient method to assist in confirming the diagnosis.
- ❑ Refractory hypotension in the acutely ill patient may be the only clue to adrenal insufficiency and is readily treated with the IV administration of glucocorticoids (dexamethasone, 4 mg, or hydrocortisone, 100 mg).

## Rosen's In Perspective

*Here's the physiology of thyroid production - as a review:*

***The thyroid's function is to secrete two iodinated hormones, T3 and T4. Only about 20% of circulating T3 is directly secreted by the thyroid; the remainder is produced by peripheral conversion of T4 to the more biologically active T3.***

*The thyroid is the only endocrine gland that stores large quantities of hormone, with enough for a 100-day supply.*

### ***So how is production regulated?***

*Hormone production is regulated by a negative feedback loop involving the hypothalamic-pituitary-thyroid axis (Fig. 120.2).*



*As the serum levels of T4 and T3 fall, the hypothalamus releases the tripeptide thyrotropin-releasing hormone (TRH), which in turn stimulates the anterior pituitary gland's release of the polypeptide thyroid-stimulating hormone (TSH) from its thyrotroph cells.*

*TSH then binds to epithelial cells on the thyroid gland, stimulating follicular cells to synthesize and secrete the thyroid hormones T4 and T3 (**T3 is the biologically active hormone we care most about!**).*

*TRH release may also result from exercise, stress, malnutrition, hypoglycemia, and sleep.*

*What does the thyroid hormone actually do?*

- *increases one's basal metabolic rate.*
- *increases protein synthesis and functions together with other hormones necessary for normal growth and development.*
- *increases the expression and sensitivity of  $\beta$ -adrenergic receptors, dramatically increasing response to endogenous catecholamines.*

**Refer to figure 120.2 in Rosen's 9<sup>th</sup> edition for an illustration of the negative feedback loop of thyroid hormone regulation, the hypothalamic-pituitary-thyroid axis**

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## **[1] List 8 causes of thyrotoxicosis**

**Thyrotoxicosis is a hypermetabolic condition that results from elevated levels of thyroid hormones—triiodothyronine (T3) and thyroxine (T4).**

This can occur from:

- Hormone overproduction (Graves' disease, toxic multinodular goiter),
- Increased thyroid hormone release from an injured gland (thyroiditis, trauma),
- Exogenous thyroid hormone (thyrotoxicosis factitia).

Most cases of thyrotoxicosis (>80%) are due to autoimmune disease. For the purpose of this discussion, the terms hyperthyroidism and thyrotoxicosis are used interchangeably.

Here's the list of 8 causes:

1. Graves' disease: Most common cause!
  - a. Autoimmune: autoantibodies bind to the TSH receptor and stimulate thyroid hormone production and release
2. Toxic multinodular goiter: second most common cause
  - a. Autonomously functioning nodules, usually in women older than 50 years
  - b. Milder than Graves' disease - but can present acutely in patients who are iodine deficient and receive an iodine load



3. Toxic adenoma
  - a. A single hyperfunctioning nodule within the thyroid
4. Thyroiditis: multiple possible causes causing thyroid inflammation - this leads to follicular cell breakdown and the release of preformed thyroid hormones
  - a. Trauma
  - b. Drug-induced (commonly amiodarone / lithium)
  - c. Infectious (suppurative) thyroiditis (bacterial or fungal infection of the thyroid, usually in patients with AIDS)
  - d. Autoimmune (including silent thyroiditis)
    - i. *The most common form of thyroiditis in the United States is Hashimoto's thyroiditis, an autoimmune disorder characterized by the presence of thyroid antibodies and lymphocytic infiltration of the thyroid gland. Typically, patients present with a painless goiter and hypothyroidism, but some have transient thyrotoxicosis (hashitoxicosis) that may last a few months.*
5. Postpartum thyroiditis
6. Subacute thyroiditis (viral inflammation of the thyroid)
7. Subclinical thyroiditis
8. Factitious thyroiditis (ingestion of excess thyroxine)

The categories of thyrotoxicosis:

- Autoimmune, infectious, drug induced, endocrine, silent/subclinical, factitious

## [2] List 5 ophthalmologic findings in hyperthyroidism / Graves'

*Ophthalmopathy is a classic finding in Graves' disease; it is thought to result in a proliferation of orbital fibroblasts differentiating into **adipocytes and orbital infiltration of inflammatory cells**.*

*Patients subsequently present with diplopia, photophobia, tearing, grittiness, and pain **because of corneal exposure**, as well as 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).*

- Thyroid Stare
- Proptosis / exophthalmos
  - AP distance from orbital ridge to ant cornea > 20mm
- Excessive tearing
- Diplopia
- FB sensation/irritation



Said another way:

- History
  - Ophthalmologic: Tearing, irritation, wind sensitivity, diplopia, foreign body sensation
- Physical:
  - Ophthalmologic: Widened palpebral fissures (stare), lid lag, globe lag, conjunctival injection, periorbital edema, proptosis, limitation of superior gaze

### [3] List the diagnostic criteria for thyroid storm

- Rare form of thyrotoxicosis. 100% mortality without treatment
  - *Although it can occur as the result of unrecognized or undertreated thyrotoxicosis, more often it is an acute reaction to thyroid or non-thyroid surgery, trauma, infection, iodine load (contrast media or amiodarone), or parturition in patients with preexisting hyperthyroidism. Other precipitants include acute myocardial infarction, pulmonary embolism, hyperemesis, toxemia of pregnancy, and diabetic ketoacidosis.*
  - *The typical clinical manifestations of thyroid storm include:*
    - *Marked pyrexia (104°–106°F [40°–41°C]),*
    - *Extreme tachycardia (often out of proportion to level of fever),*
    - *Altered mental status (agitation, delirium, or coma).*

*These findings, coupled with the clinical picture of a patient with hyperthyroidism, lid lag, stare, goiter, ophthalmopathy, and tremor...*

**\*\*\*No validated diagnostic criteria exist\*\*\***, a scoring system developed by Bahn Chair and colleagues can help distinguish among thyrotoxicosis, impending thyroid storm, and frank thyroid storm

FAT PIG!!!

- Fever
- Altered LOC
- Tachycardia
- Precipitating event
- Increased CO (high output CHF)
- GI / hepatic symptoms

**Refer to table 120.1 in Rosen's 9<sup>th</sup> edition for the detailed diagnostic criteria for thyroid storm including the scoring system**

**Elevation of free T4 and free T3 levels in conjunction with TSH suppression is diagnostic of thyrotoxicosis.**



#### [4] List 4 precipitants of thyroid storm

Well, it can really occur in anyone with hyperthyroidism (Graves' disease, toxic multinodular goiter/adenoma) OR be the first presentation of thyrotoxicosis,

**BUT usually the storm starts after one of these insults:**

- **Thyroid or nonthyroidal surgery,**
- **Trauma,**
- **Infection,**
- **An acute iodine load,**
- **Postpartum,**
- **In addition, irregular use or discontinuation of antithyroid drugs is a commonly reported precipitant of thyroid storm**

Source: Uptodate

#### [5] What is the treatment of thyroid storm

- ❑ **Step 1: Beta-blockade**
  - ❑ Propranolol 60mg PO q6hrs
  - ❑ Propranolol 1mg IV q15min PRN
- ❑ **Step 2: Inhibition of T3 & T4 Production**
  - ❑ Propylthiouracil (PTU) 600mg PO/NG/PR q6hrs
  - ❑ Methimazole 30mg PO/NG/PR q6hrs
- ❑ **Step 3: Inhibition of T3 & T4 release**
  - ❑ Potassium iodide 1-2 drops PO/NG/PR q6hrs OR
  - ❑ Lugol's solution 5 gtt PO/NG/PR q6hrs OR
  - ❑ Lithium 300mg PO/NG q6hrs (only if allergy to iodine/amio/contrast)
  - ❑ NOTE: must delay 1 hr post step 2 so as not to allow solution to act as substrate for more T3/T4 production
- ❑ **Step 4: Inhibition of peripheral T4 to T3 conversion**
  - ❑ Hydrocortisone 300 mg IV x 1 then
  - ❑ Hydrocortisone 100mg IV q8hrs
  - ❑ Note: PTU and propranolol also do this
- ❑ **Step 5: Supportive care**
  - ❑ ABCs
  - ❑ Antibiotics (given other entities on Ddx)
    - ❑ *Thyroid storm is often precipitated by a physiologic stressor, usually an infection. Empirical antibiotics are not necessary without an identified source of infection. Other common stressors include myocardial ischemia, pulmonary embolism, and stroke.*
  - ❑ Benzos (for agitation)
  - ❑ Cooling / warming (acetaminophen)



- Dexamethasone / steroids
- Fluids / electrolyte replacement
- Glucose

**Refer to box 120.3 in Rosen's 9<sup>th</sup> edition for a detailed description of the management of thyroid storm**

### **EMCRIT approach**

#### **Block New Production**

- The thionamides: Methimazole and PTU; the latter may be preferred as it also blocks peripheral T4 to T3 conversion
- PTU 500-1000 mg load then 250 mg Q4 hours (Guidelines from AACE (endo group))
- Methimazole 60-80 mg qday, divided into doses q4-6 hrs (20 mg Q6)

#### **Block Thyroid Hormone Release**

Wolf-Chaikoff effect blocks iodide binding to thyroglobulin once critical levels of iodide are reached

- SSKI 5 drops PO q6; OR
  - Lugol's Solution 8 drops PO q 6; OR
  - Sodium Iodide 0.5 mg IV Q 12 hours
- \*Don't give until 60 minutes after thionamides

Lithium can be substituted in patients who will undergo radioactive iodide treatment or patients allergic to iodides. Use 300 mg q 6-8, but personally, I would consult an endocrinologist before going this road. (J Inten Care Med 2015;30(3):131)

#### **Treat Volume Loss**

These patients have large insensible losses and diuresis. Even in the setting of seeming heart failure, they may need fluids as the heart failure is high-output.

#### **Treat Sympathetic Surge**

- Propranolol 1 mg IV (test dose) then Propranolol 1-2 mg q 15 minutes until HR of 100 bpm
- Then start Propranolol drip at whatever dose it took to get IV load control (Max 3-5 mg/hr)
- Propranolol also blocks T4 to T3 conversion
- Or titrate esmolol for HR of 100 bpm, but selective B1 means may be less effective

#### **Block Peripheral Conversion and Shield from Adrenal Insufficiency**

- Dexamethasone 4 mg IV Q 6 hours; OR



- ❑ Hydrocortisone 300 mg IV and then 100 mg q 8 hours

### **Not Available in the US?**

Oral cholecystographic agents (HIDA Scan Contrast) 2g loading dose followed by 1g q day

### **Temperature regulation**

Do not aggressively cool these patients; this is *contraindicated* because it can lead to further vasoconstriction

### **Fix Precipitating Event/Treat Infection**

Look carefully, treat aggressively

Chris' mantra:

“Supportive care & Bb-asic C-P-R”

Supportive: IV fluids, cooling, oxygen, benzo's for sedation

BBlock, corticosteroids, PTU/production inhibition, Release inhibition (iodine)

Or follow the letters of the alphabet: Production inhibition before Release inhibition!

## **[6] List 8 causes of hypothyroidism and 10 signs and symptoms**

**Central/secondary hypothyroidism:** lack of stimulation of the thyroid gland, usually due to pituitary disease - hemorrhage, adenoma, etc., VERY RARE.

OR

**Primary hypothyroidism:** intrinsic gland dysfunction limiting hormone production. Much more common.

>99% of cases are intrinsic gland failure due to any of:

- Autoimmune disorders - **#1 cause in developed world**
  - **Can occur with other autoimmune diseases, such as diabetes mellitus, pernicious anemia, Addison's disease, and hyperparathyroidism.**
  - **Hashimoto's thyroiditis, or chronic autoimmune lymphocytic thyroiditis, first described in 1912 by Hakaru Hashimoto, is one of the most common organ-specific autoimmune diseases and the most common cause of primary hypothyroidism. It is characterized by infiltration of the thyroid gland by lymphocytic inflammatory cells, which is then often followed by hypothyroidism as a result of destruction and eventual fibrous replacement of the gland's follicular tissue.**
- Infiltrative disorders,
- Congenital thyroid dysfunction,
- Pregnancy,



- Radiotherapy,
- Medications,
  - Lithium
  - Amiodarone
- Infection,
- Surgery,
- Inadequate dietary iodine intake, (#1 cause worldwide)
- Thyroid medication noncompliance,
- Previous treatment of thyrotoxicosis.

**Symptoms of hypothyroidism:**

- Sinus bradycardia
- Diastolic heart failure
- Dyspnea on exertion
- Decreased exercise capacity
- Constipation
- Menorrhagia
- Periorbital swelling
- Goiter
- Cold intolerance
- Coarse, brittle hair
- Alopecia
- Dry skin
- Lethargy
- Agitation
- Mononeuropathy
- Proximal myopathy

**Signs of hypothyroidism:**

- Vitals – normal or low and slow

**Refer to box 120.5 in Rosen's 9<sup>th</sup> edition for the symptoms and signs of hypothyroidism**

**[7] List 5 lab abnormalities, 2 ECG findings, and 1 CXR finding in hypothyroidism**

**An elevated TSH level with a low T4 level is indicative of primary hypothyroidism.**

**Other laboratory findings may include:**

- Mild anemia,
- Hypercholesterolemia,



- Elevated hepatic enzyme levels,
- Elevated prolactin level,
- Hyponatremia secondary to extracellular volume expansion produced by an elevated antidiuretic hormone level.
- Blood glucose levels may be normal to low as a result of decreased gluconeogenesis and reduced insulin clearance.

**The electrocardiogram is nonspecific in hypothyroidism. It might reveal sinus bradycardia with low-voltage complexes and nonspecific ST-T wave changes.**

**CXR - pulmonary edema, features of CHF.**

Central hypothyroidism is associated with a low or normal TSH level, with a low T4 level. An increased TSH concentration with a normal T4 level represents subclinical hypothyroidism

**[8] Describe 5 key clinical features of myxedema coma and list 8 precipitating factors**

**Refer to box 120.7 in Rosen's 9<sup>th</sup> edition for recognition of the clinical features of myxedema coma**

**Clinical features:**

- Hypothermia
- Hypotension - refractory
- Hypoventilation
- Bradycardia
- AMS
- Edematous face

**Refer to box 120.6 in Rosen's 9<sup>th</sup> edition for a list of the aggravating or precipitating factors in myxedema coma**

**Precipitating events include:**

- Myocardial infarction
- Infection
- Sepsis
- Stroke
- Pulmonary embolism
- Prolonged exposure to cold
- Exposure to drugs that suppress the central nervous system

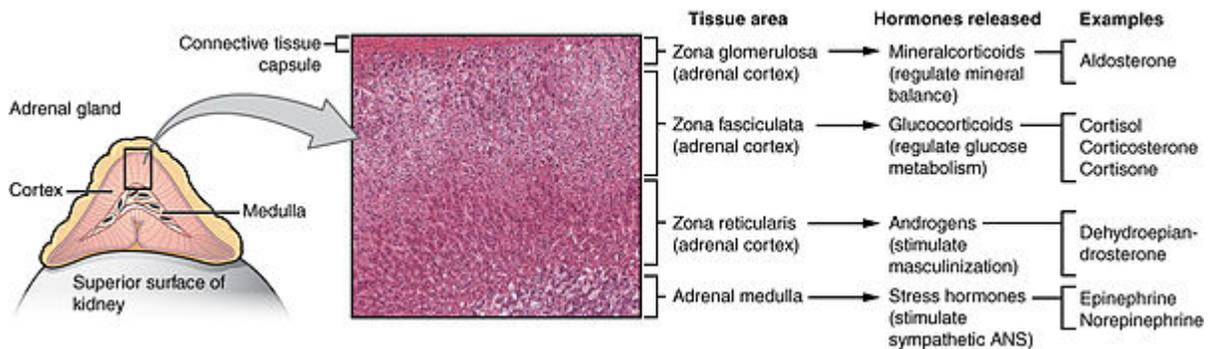
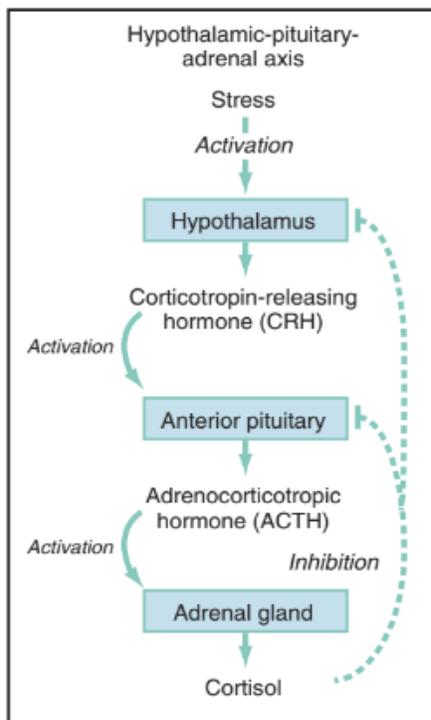


**[9] Describe the treatment of myxedema coma**

Refer to box 120.8 in Rosen’s 9<sup>th</sup> edition for the treatment of myxedema coma  
 Stress doses of an IV glucocorticoid are recommended due to possible concomitant adrenal insufficiency. **Hydrocortisone, 100 mg IV**, is the drug of choice because it has mineralocorticoid and glucocorticoid effects.

**[10] List the hormones produced by the adrenal glands**

The adrenal glands are responsible for the release of following hormones: **aldosterone, corticosteroids, androgens, and catecholamines.**



\*Images from Wikipedia.



## [11] List 3 effects of aldosterone

Aldosterone tends to promote  $\text{Na}^+$  and water retention...

- $\text{Na}^+$  and water retention,
- Lower plasma  $\text{K}^+$  concentration (via renal secretion of  $\text{K}^+$ )
- Increased Blood pressure

It has the opposite effects of Atrial natriuretic peptide

Here is the full list of aldosterone effects from Wikipedia

1. Acting on the nuclear mineralocorticoid receptors (MR) within the principal cells of the distal tubule and the collecting duct of the kidney nephron, it upregulates and activates the basolateral  $\text{Na}^+/\text{K}^+$  pumps, which pumps three sodium ions out of the cell, into the interstitial fluid and two potassium ions into the cell from the interstitial fluid. This creates a concentration gradient which results in reabsorption of sodium ( $\text{Na}^+$ ) ions and water (which follows sodium) into the blood, and secreting potassium ( $\text{K}^+$ ) ions into the urine (lumen of collecting duct).
2. Aldosterone upregulates epithelial sodium channels (ENaCs) in the collecting duct and the colon, increasing apical membrane permeability for  $\text{Na}^+$  and thus absorption.
3.  $\text{Cl}^-$  is reabsorbed in conjunction with sodium cations to maintain the system's electrochemical balance.
4. Aldosterone stimulates the secretion of  $\text{K}^+$  into the tubular lumen.
5. Aldosterone stimulates  $\text{Na}^+$  and water reabsorption from the gut, salivary and sweat glands in exchange for  $\text{K}^+$ .
6. Aldosterone stimulates secretion of  $\text{H}^+$  via the  $\text{H}^+/\text{ATPase}$  in the intercalated cells of the cortical collecting tubules
7. Aldosterone upregulates expression of NCC in the distal convoluted tubule chronically and its activity acutely.

## [12] List 5 causes of chronic primary adrenal insufficiency + 1 acute + 3 secondary

*The clinical manifestations are the result of primary adrenal failure or secondary adrenal disease from malfunction of the hypothalamic-pituitary-adrenal (HPA) axis in its production of adrenocorticotrophic hormone (ACTH).*

**Secondary causes are much more common than primary causes.**

**Primary = high ACTH and low cortisol (get subsequent hyperpigmentation)**



*Mild to moderate hyponatremia, with levels typically above 120 mEq/L, is seen in primary adrenal insufficiency; hyperkalemia is common as well.*

**Acute:**

- Acute hemorrhage / infarction

**Chronic:**

- **Autoimmune:**
  - Due to idiopathy or post infectious - viral (CMV, HIV), bacterial (MAC, TB),
- **Infectious - AIDS related**
- **Cancer - mets, lymphoma**
- **Congenital adrenal hyperplasia**
- **Medication:**
  - Multiple doses of etomidate

**Secondary AI:**

- **Occurs post precipitant - severe physiologic stress (MI, sepsis, hypoglycemia, pain, etc.).**
  - **Acute:**
    - Pituitary surgery / TBI
    - SAH
    - Due to sudden removal of exogenous glucocorticoids = suppressed HPA axis (usually get this after several weeks)
  - **Chronic:**
    - Autoimmune - sarcoidosis
    - TB
    - Cancer
    - Radiation

**Refer to box 120.9 in Rosen's 9<sup>th</sup> edition for causes of primary and secondary adrenal insufficiency**

**[13] Describe the treatment of acute adrenal insufficiency**

Clinical features = think of the hormones that are missing...

NO cortisol = fatigue, weakness, depression, free water retention;

NO aldosterone = salt wasting (salt craving), hyperkalemia, hypovolemia;

NO catecholamines = hypotension.

But you can break it down more elegantly using Box 120.10

**Refer to box 120.10 in Rosen's 9<sup>th</sup> edition for the clinical features of adrenal insufficiency**



***Adrenal crisis presents with hypotension and shock that does not respond to fluid resuscitation and pressors. Patients may have many other nonspecific symptoms, as listed above, but shock is the hallmark. The constellation of symptoms seen in acute adrenal insufficiency—weakness, malaise, fatigue, nausea, dizziness, and arthralgias—is also present in steroid withdrawal syndrome.***

**Refer to box 120.11 in Rosen's 9<sup>th</sup> edition for the treatment of hypoadrenalism**

**Treatment of hypoadrenalism:**

General maintenance of hypoadrenalism:

- Hydrocortisone, 20 mg AM, 10 mg, PM
- Fludrocortisone, 50-100 mcg/day

Maintenance during minor illness:

- Hydrocortisone, 40 mg AM, 20 mg PM
- Fludrocortisone, 50-200 mcg/day

Coverage during procedural stress:

- Hydrocortisone, 100 mg IV (one time only)

Adrenal crisis or relative adrenal insufficiency of critical illness:

- Dexamethasone, 4 mg IV bolus OR
- Hydrocortisone, 100 mg IV bolus 0.9 NS, 2-3 L in the first few hours
- Switch to D<sub>5</sub>/NS (5% dextrose in normal saline) if hypoglycemia
- Treat precipitating illness

## **WiseCracks**

### **1) What is apathetic thyrotoxicosis?**

Atypical manifestation of hyperthyroidism. Presents with apathy and depression instead of hyperkinesia and mental alertness.

Older adults often lack the same adrenergic response and present with weight loss and fatigue, more consistent with apathetic hyperthyroidism.

### **2) What is the analgesic / antipyretic of choice in thyroid storm? Which should you avoid?**

- Acetaminophen is best!**
- Watch for underlying hepatic dysfunction
- Avoid ASA / NSAIDs as they decrease protein binding of T<sub>4</sub> and T<sub>3</sub> and therefore increase free serum concentrations



### 3) Explain the thyroid lab tests and their clinical significance

See table 120.2 in Rosen's 9<sup>th</sup> edition for thyroid test interpretation

TSH	Free T <sub>4</sub>	Free T <sub>3</sub>	Disease
Normal	Normal	Normal	None
Low	High	High	Hyperthyroidism
Low	Normal	Normal	Subclinical hyperthyroidism
Low	Normal	High	T <sub>3</sub> toxicosis
Low	High	Normal	Thyroiditis, T <sub>4</sub> 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

### 4) What are some special situations in the management of thyroid storm?

See box 120.4 in Rosen's 9<sup>th</sup> edition for thyrotoxicosis and thyroid storm special situations

- 1) Congestive heart failure
- 2) Atrial fibrillation
- 3) Thyroiditis (subacute)
- 4) Factitious thyrotoxicosis