

Hyperthyroidism

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THE STORY IN ONE SENTENCE

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“Hyperthyroidism means the thyroid is making way too much hormone, so your whole body runs too fast — like a car stuck in high gear that cannot slow down.”

Normal anatomy — the body's job

The hypothalamus (a control center deep in your brain) tells the pituitary gland (a pea-sized gland just below it) to release TSH (thyroid-stimulating hormone) into your bloodstream. TSH travels to your thyroid gland (a butterfly-shaped gland in the front of your neck, just below your voice box). The thyroid makes two hormones: T4 (thyroxine, the storage form) and a little bit of T3 (triiodothyronine, the active form). T4 travels through your blood to your liver, kidneys, and other tissues, where an enzyme snips off one iodine atom and turns T4 into T3. T3 plugs into thyroid hormone receptors inside almost every cell in your body. Once plugged in, T3 tells the cell's DNA to make more energy-burning proteins, so your heart beats faster, your gut moves food along quicker, your brain fires faster, and your muscles burn more fuel. When enough T3 builds up, it sends a 'slow down' signal back to the hypothalamus and pituitary (negative feedback), so they make less TSH and the thyroid makes less hormone. That loop keeps your metabolism steady.

BODY'S JOB

Thyroid hormone is your body's throttle. It sets the baseline speed for your heart, how much heat you make, how fast you burn calories, how alert your brain is, and how quickly your gut moves. Without it, everything slows down. Too much, and everything races. The feedback loop keeps the speed just right for the work your body needs to do.

1. The hypothalamus releases TRH (thyrotropin-releasing hormone).
2. TRH tells the pituitary to release TSH into the blood.

3. TSH travels to the thyroid and tells it to make T4 and a little T3.
4. T4 travels through your blood to your liver, kidneys, and other tissues.
5. An enzyme (deiodinase) in those tissues cuts one iodine off T4, turning it into T3.
6. T3 enters cells and plugs into thyroid hormone receptors inside the cell nucleus.
7. The receptor-T3 pair tells the cell's DNA to make proteins that speed up energy use.
8. Your heart beats faster, your body makes more heat, your gut moves quicker, and your brain speeds up.
9. When T3 levels rise, they tell the hypothalamus and pituitary to make less TRH and TSH (negative feedback).
10. That feedback loop keeps thyroid hormone levels — and your body's speed — steady.

What breaks

Hyperthyroidism happens when your thyroid makes and releases way too much hormone, or when stored hormone leaks out all at once. The thyroid ignores the normal off switch, so T3 and T4 flood your bloodstream and tell every cell in your body to speed up. There are three main upstream breaks that cause this.

CLINICAL TERM

In Graves disease (the most common cause, about 60–80% of hyperthyroidism), your immune system makes antibodies called thyroid-stimulating immunoglobulins (TSI). TSI looks just like TSH to the thyroid, so it plugs into the TSH receptor and turns the gland on full blast. Because TSI does not respond to negative feedback, the thyroid keeps pumping out T4 and T3 even when levels are already sky-high. TSH from the pituitary drops to near zero (because high T3 shuts it off), but the thyroid does not care — it listens to the rogue antibody instead. In toxic nodular disease (toxic adenoma if it is one lump, toxic multinodular goiter if it's many), a clump of thyroid cells mutates and starts making hormone on its own without waiting for any signal. These nodules ignore TSH completely. The rest of the thyroid shuts down (because TSH is suppressed), but the hot nodule keeps churning out hormone. In thyroiditis (can be subacute/painful, silent/postpartum, or from certain drugs like amiodarone or immune checkpoint inhibitors), inflammation cracks open thyroid follicles and dumps pre-made T4 and T3 into the blood all at once. This is not true overproduction — it is a leak. The gland is actually damaged, so after a few weeks the stored hormone runs out and the patient often swings into hypothyroidism (low thyroid) for a while before recovering. Rare causes include taking too much thyroid hormone pills (factitious hyperthyroidism), a pituitary tumor that makes too much TSH (TSH will be high, not low), or a molar pregnancy/testicular cancer that makes hCG (which can weakly stimulate the thyroid). The common thread: too much T3 in the blood, no matter the source.

How the body compensates

At first, your body tries to cope with too much thyroid hormone by using the extra energy and dumping the extra heat. Your heart beats faster to move blood and cool you down. You sweat more. You eat more to replace the calories you are burning. For a while, you might just feel energized or jittery. But this is not truly stable — it is your body working overtime. If hormone levels keep climbing or if a stress (infection, surgery, trauma, suddenly stopping anti-thyroid medicine) hits, compensation fails and you crash into thyroid storm: extreme fever, heart rate over 140, confusion, seizures, vomiting, diarrhea, and heart failure. That is a medical emergency with a death rate of 10–30% even with treatment. The job is to spot hyperthyroidism early and treat it before the body runs out of runway.

1. The heart beats faster (tachycardia, often 90–140 bpm at rest) to move the extra heat and oxygen demand that high metabolism creates. Cardiac output goes up. For a while, blood pressure may stay normal or the top number (systolic) goes up while the bottom number (diastolic) drops (wide pulse pressure).
2. Blood vessels in the skin open wide (vasodilation) to dump heat, so your skin feels warm, flushed, and sweaty. You feel hot even in a cool room.

3. You eat more because your body is burning calories faster, but you still lose weight because the calorie burn outpaces intake. Muscle breaks down for fuel (catabolism).
4. Your gut speeds up, so you have frequent bowel movements or diarrhea. Food moves through faster.
5. You feel anxious, jittery, or cannot sleep because the sympathetic nervous system (your fight-or-flight system) is amplified. The amygdala (your brain's alarm center) and the locus coeruleus (a brainstem area that releases norepinephrine, the brain's adrenaline) are extra sensitive to signals.
6. Women stop having periods (oligomenorrhea or absent periods (amenorrhea)) because the hormonal axis shifts and the ovaries do not get the right signals.
7. Bones release calcium faster than they rebuild (increased bone turnover), so older adults are at higher risk for fractures. Blood calcium can go up a little.
8. The danger zone: if thyroid hormone keeps rising, or if infection, surgery, stopping methimazole suddenly, or trauma hits, the body cannot keep up. You tip into thyroid storm — fever above 104°F (40°C), heart rate above 140, atrial fibrillation (an irregular, quivering heartbeat) (chaotic top-chamber heart rhythm), heart failure (the heart cannot pump enough), confusion or agitation, seizures, severe vomiting and diarrhea leading to dehydration, and shock. Thyroid storm has a 10–30% death rate even with ICU care.
9. Once you are in thyroid storm, the window to reverse it shrinks fast. The heart can fail, a blood clot can throw a stroke, or multi-organ failure can set in. That is why we treat hyperthyroidism early — before compensation breaks.

Domino effect

1. Too much T3 floods your bloodstream. — The upstream break (Graves antibodies, a hot nodule, or thyroid inflammation) drives the thyroid to make or release way more T4 and T3 than normal. T3 is the active hormone that plugs into receptors in almost every cell.
2. Your heart races (tachycardia, often 90–140 bpm at rest) and beats harder. — T3 makes beta-1 adrenergic receptors on heart muscle extra sensitive to adrenaline (epinephrine) and norepinephrine. It also tells heart cells to make more receptors. The sinoatrial node (your heart's natural pacemaker in the right atrium) fires faster, and the ventricles squeeze harder. Cardiac output (how much blood your heart pumps per minute) goes up. Over time, the top chambers can start beating chaotically (atrial fibrillation), which raises stroke risk because blood can pool and clot in the atrium.
3. Your skin feels warm, flushed, and sweaty. — High T3 cranks up your basal metabolic rate, so every cell makes more heat. To dump that heat, blood vessels in your skin dilate (widen). Warm blood flows to the surface, your skin flushes, and sweat glands turn on to cool you by evaporation. You feel hot even in a cool room and you sweat a lot.

4. You lose weight even though you are eating more. — T3 tells your cells to burn glucose and fat faster (increased oxygen consumption and heat production in mitochondria, the cell's power plants). Your body uses up calories faster than you can eat them. Muscle protein also breaks down (catabolism) to feed the revved-up engine, so you lose both fat and muscle mass.
5. You feel jittery, anxious, or cannot sleep. — T3 amplifies the sympathetic nervous system (your fight-or-flight response). The locus coeruleus (a tiny cluster in your brainstem that makes norepinephrine, the brain's version of adrenaline) becomes hyperactive. The amygdala (your brain's alarm center for detecting danger) fires more easily. The prefrontal cortex (the front part of your brain that usually puts the brakes on big emotions) has a harder time calming things down. You feel wired, on edge, and your mind races. You may have tremor (shaky hands) because motor neurons are extra excitable.
6. You have frequent bowel movements or diarrhea. — T3 speeds up gut motility (how fast food moves through your intestines). The enteric nervous system (the nerve network in your gut wall) fires faster, and smooth muscle in the intestines contracts more often, pushing food through quickly. Less water is absorbed, so stools are loose.
7. Your bones break down faster, and you can develop osteoporosis or high blood calcium. — T3 revs up bone remodeling. Osteoclasts (cells that dissolve old bone and release calcium into the blood) work faster than osteoblasts (cells that build new bone). Over months to years, bone density drops, raising fracture risk, especially in post-menopausal women. Blood calcium can tick up because bone is releasing it.
8. In thyroid storm, all these effects spiral out of control. — A trigger (infection, surgery, stopping anti-thyroid medicine suddenly, trauma, or childbirth) pushes T3 levels even higher or unmasks the body's inability to cope. Fever spikes above 104°F because the hypothalamus (your brain's thermostat) is overridden. Heart rate climbs above 140; atrial fibrillation (an irregular, quivering heartbeat) or heart failure sets in because the heart cannot keep up the pace. Confusion, agitation, or seizures happen because the brain is overheated and overstimulated. Severe vomiting and diarrhea cause dehydration and shock. Multi-organ failure follows. This is a medical emergency with 10–30% mortality even with ICU care (IV beta-blockers like esmolol or propranolol to slow the heart, IV hydrocortisone because stress can unmask adrenal insufficiency, methimazole or PTU to block new hormone production, iodine solution one hour later to block hormone release, cooling blankets, IV fluids, and treating the trigger infection or stress).

Symptoms · what the patient feels, and why

- Fast heart rate (often over 100 beats per minute, even at rest): Too much thyroid hormone (the body's main speed-control chemical) pushes the heart to pump faster and harder, like stepping on a car's gas pedal

- Fine shaking of the hands (tremor): Extra thyroid hormone revs up the nervous system, making muscles fire too quickly and causing a fine, rapid shake you can see when you hold your hands out
- Warm, sweaty skin: Thyroid hormone cranks up the body's metabolism (how fast cells burn fuel), generating extra heat—the body sweats to cool down, even when the room feels normal
- Weight loss despite eating normally or more: Thyroid hormone speeds up how fast the body burns calories, so even if you eat the same or more, you lose weight because you're burning fuel faster than you can replace it
- Feeling nervous, worried, or jittery: Too much thyroid hormone overactivates the amygdala and other brain circuits that control alertness and emotion, making you feel on edge or anxious, like you've had too much caffeine
- Missed or irregular periods in women: High thyroid hormone disrupts the normal signals between the hypothalamus (the brain's hormone control center) and the ovaries, throwing off the monthly cycle
- Bulging eyes (proptosis) and eyelid retraction (eyes look wide or staring): In Graves disease, the immune system attacks the muscles and fat behind the eyeball, causing swelling that pushes the eye forward; the eyelids also pull back because the muscles get overstimulated by thyroid hormone
- Enlarged, smooth thyroid gland (goiter) that you can feel in the neck: In Graves disease, autoantibodies (rogue immune proteins) constantly stimulate the thyroid, making it grow bigger and work overtime; in toxic nodular goiter, clumps of thyroid cells grow and pump out hormone on their own
- Painful, tender thyroid gland (in subacute thyroiditis): A viral infection triggers inflammation inside the thyroid gland, causing it to swell and hurt; damaged cells leak stored thyroid hormone into the blood all at once
- Confusion, fever over 104°F, or very fast heart rate (over 140) at rest: In thyroid storm (a rare emergency), a sudden surge of thyroid hormone overwhelms the body's ability to cope, causing the brain, heart, and other organs to go into overdrive and start to fail

Focused exam

- Resting heart rate above 90, often 100–120 or higher: Too much thyroid hormone makes the heart's beta receptors (the switches that control heart speed) super-sensitive, so the heart races even when you are sitting still. A fast resting heart rate is the most reliable sign that the thyroid is running too hot.

- Fine tremor in outstretched hands: Thyroid hormone speeds up every nerve and muscle in the body. The tiny shaking in your hands tells you the body's engine is revving too fast, like a car idling at high RPM.
- Warm, sweaty skin even when the room is cool: Blood vessels open wide to dump extra heat because metabolism is burning fuel so fast. Sweating without fever is a clue hyperthyroidism is the culprit, not infection.
- Eyelid lag — upper eyelid stays open when you look down: Thyroid hormone makes the small muscles in the eyelid twitch and stay tight. When the eyelid does not follow the eye down smoothly, it points straight to the thyroid as the problem.
- In Graves disease: eye bulging (exophthalmos) and a soft, enlarged thyroid gland in the neck (goiter): Graves disease happens when your immune system makes antibodies that attack the thyroid and the tissue behind the eyes. Eye bulging and a big, soft goiter are the body's signature for Graves, not for other kinds of hyperthyroidism like nodules or thyroiditis.
- Weight loss despite eating more than usual: Every cell in the body burns fuel faster when thyroid hormone is high. Even if you eat extra food, your engine uses it quicker than it comes in, so you lose weight. This mismatch — eating more but weighing less — is classic for hyperthyroidism.

Diagnostics · every test answers a question

- TSH (thyroid-stimulating hormone) → Is the thyroid making too much hormone on its own, or is the brain telling it to make more?
- Free T4 (thyroxine) → How much active thyroid hormone is floating in the blood right now?
- Free T3 (triiodothyronine) → Is T3 — the strongest thyroid hormone — also high?
- Thyroid-stimulating immunoglobulin (TSI) or TSH receptor antibody → Is the immune system making antibodies that turn on the thyroid like a fake TSH signal?
- Radioactive iodine uptake scan (RAIU) → Is the thyroid gland actively making new hormone, or is it just leaking stored hormone because it is inflamed?
- Thyroid ultrasound → Are there lumps (nodules) in the thyroid that could be making hormone on their own?
- EKG (electrocardiogram) → Is the heart beating in a normal rhythm, or is it quivering instead?

Treatment logic

- Beta-blocker (propranolol 10–40 mg every 6–8 hours, or atenolol 25–100 mg daily) → Blocks beta receptors in the heart and nerves so your heart slows down, shaking stops, and you feel calmer within hours. This buys time while the real thyroid problem gets fixed.
- Methimazole (10–40 mg daily; or propylthiouracil 100–150 mg three times daily in first trimester pregnancy or thyroid storm) → Stops the enzyme thyroid peroxidase inside the thyroid gland so it cannot make new T4 and T3 hormone. Hormone levels drop over weeks, and the body slows back to normal speed.
- Radioactive iodine (I-131, single oral dose) → Destroys thyroid cells from the inside because iodine collects in the thyroid and the radiation kills the cells. Hormone levels drop and stay low, fixing hyperthyroidism permanently.
- Thyroid surgery (partial or total thyroidectomy) → Removes the overactive part of the gland physically, so no more hormone can be made. Fixes the problem immediately during the operation.
- Corticosteroid (prednisone 40 mg daily, tapered over weeks) for subacute or silent thyroiditis → Calms inflammation in the thyroid gland so it stops leaking stored hormone into the blood. Hormone levels drop as the swelling goes down.
- Iodine solution (saturated potassium iodide or Lugol solution) in thyroid storm → Floods the thyroid with so much iodine that it temporarily shuts off hormone release (Wolff-Chaikoff effect). Works within hours to drop hormone levels fast in a life-threatening crisis.

Medications

Propranolol or atenolol (beta-blockers) · Beta-adrenergic antagonist (beta-blocker)

These drugs block the body's response to too much thyroid hormone, especially in the heart and nerves. They slow the racing heart, stop the shaking, and ease the sweaty, panicky feelings. They do NOT lower thyroid hormone itself—they just turn down the alarm bells while the real treatment works.

1. Thyroid hormone makes the heart and nerves extra sensitive to adrenaline (the body's 'go' signal).
2. The beta-blocker sits on the adrenaline docking spots (beta receptors) in the heart and nerves and blocks adrenaline from landing there.
3. Without adrenaline's signal, the heart slows down, the shaking stops, and the sweating and anxiety ease.

4. The thyroid hormone level stays high—this drug just shields the body from the worst symptoms while you wait for antithyroid drugs, radioactive iodine, or surgery to actually lower the hormone.

SAFETY WATCH

Can make asthma worse—propranolol blocks receptors in the lungs and can tighten airways. Use a heart-selective blocker like atenolol or metoprolol in people with asthma or COPD. · Can hide low blood sugar warning signs (shaking, fast heart) in people with diabetes. · Can slow the heart too much—watch for dizziness or fainting. · Do NOT stop suddenly after long use; taper slowly to avoid rebound fast heart rate.

Methimazole · Thionamide antithyroid drug

Methimazole blocks the thyroid gland from making new thyroid hormone. It stops an enzyme (thyroid peroxidase) that glues iodine onto thyroglobulin—the step that builds T3 and T4. The gland keeps running, but it can't make hormone, so levels in the blood drop over weeks.

1. Normally, thyroid peroxidase takes iodine and sticks it onto a protein called thyroglobulin inside the thyroid gland.
2. That iodine-tagged protein is then clipped into T3 and T4 (the active thyroid hormones).
3. Methimazole jams the thyroid peroxidase enzyme so it can't attach iodine.
4. Without iodine added, no new T3 or T4 can be made.
5. Old hormone in the blood keeps circulating for a few weeks (half-life ~7 days for T4), so it takes 4–6 weeks to see the full drop in thyroid hormone levels.

SAFETY WATCH

Rare but serious: agranulocytosis (white blood cells drop to zero). Tell patients to stop the drug and call immediately if they get fever, severe sore throat, or mouth sores—usually happens in the first 3 months. · Liver injury (rare)—watch for yellow skin, dark urine, or belly pain. · Rash and itching (common, usually mild). · Can cross the placenta—safe in pregnancy EXCEPT the first trimester, when it rarely causes scalp defects (aplasia cutis) and a pattern of birth defects (methimazole embryopathy). Switch to PTU in the first 12 weeks if pregnancy is planned or discovered. · Check CBC and liver enzymes at baseline and if symptoms appear; routine monitoring is not required by guidelines but many clinicians recheck in the first 3 months.

Propylthiouracil (PTU) · Thionamide antithyroid drug

PTU works like methimazole—it blocks thyroid peroxidase so the gland can't make new thyroid hormone. PTU also blocks the enzyme (deiodinase) that converts T4 into T3 (the stronger form)

in the liver and other tissues, so it lowers active hormone a bit faster. It is used in the first trimester of pregnancy and in thyroid storm because of that extra effect.

1. PTU jams thyroid peroxidase in the gland, stopping iodine from being added to thyroglobulin (same as methimazole).
2. It also blocks the enzyme deiodinase outside the thyroid, which normally snips one iodine off T4 to make T3 (the form that actually flips the switches in cells).
3. By blocking that conversion, PTU lowers active T3 a little faster than methimazole does.
4. Total effect takes weeks because old hormone in the blood still needs to break down.

SAFETY WATCH

Higher risk of severe liver injury than methimazole—rare cases of acute liver failure requiring transplant. Watch for yellowing of the skin and eyes (jaundice), dark urine, nausea, belly pain. · Agranulocytosis (same risk as methimazole)—fever, sore throat → stop drug and check CBC immediately. · Rash, joint pain, vasculitis (rare). · Safer than methimazole in the FIRST TRIMESTER of pregnancy (no scalp or organ defects), so it is first-line weeks 0–12. After week 12, switch back to methimazole because PTU liver risk outweighs methimazole's small risk. · Preferred in thyroid storm because it blocks T4→T3 conversion. · Requires dosing 2–3 times daily (methimazole is once daily), so adherence can be harder.

Radioactive iodine-131 (RAI) · Radioisotope ablation

You swallow a capsule or liquid containing radioactive iodine. The thyroid gland soaks up iodine to make hormone, so it pulls in the radioactive version. The radiation destroys the overactive thyroid cells from the inside. Over 2–3 months, the gland shrinks and hormone levels drop—usually all the way to low (hypothyroidism), which you then treat with thyroid hormone pills for life.

1. You swallow iodine-131 (a radioactive form of iodine).
2. The thyroid gland has sodium-iodide transporters that pull iodine out of the blood to use for making T3 and T4.
3. The radioactive iodine gets trapped inside thyroid cells.
4. Over days to weeks, the radiation (beta particles) damages the DNA and cell machinery of those thyroid cells, killing them.
5. Dead cells can't make hormone, so thyroid levels fall.
6. Most people become hypothyroid (low thyroid) within 3–6 months and need lifelong levothyroxine replacement.

SAFETY WATCH

Absolutely contraindicated in pregnancy and breastfeeding—radiation destroys the baby's thyroid. · Women must avoid pregnancy for 6 months after RAI; men should avoid fathering a child for 3–4 months. · Can worsen thyroid eye disease (Graves ophthalmopathy)—give prednisone cover if eye symptoms are present. · Radiation thyroiditis: the dying gland can dump stored hormone into the blood in the first 1–2 weeks, briefly worsening hyperthyroidism. Use beta-blocker and sometimes a short course of antithyroid drug before RAI if thyroid hormone is very high. · Most patients become hypothyroid and need levothyroxine for life—this is expected, not a complication. · Small, temporary radiation precautions for 3–7 days: sleep alone, avoid close contact with pregnant women and young children, flush toilet twice, wash hands well.

Thyroid surgery (thyroidectomy) · Surgical treatment (not a medication, but listed here as a definitive therapy option)

A surgeon removes most or all of the thyroid gland. With the gland gone, thyroid hormone production stops. This is permanent and works immediately (once you're healed). You will need thyroid hormone pills (levothyroxine) for life afterward. Surgery is chosen when the gland is very large (causing trouble swallowing or breathing), when there are suspicious nodules that might be cancer, when eye disease is severe, or when the patient prefers surgery over radioactive iodine.

1. The surgeon makes a small cut in the lower neck and lifts out most (subtotal) or all (total) of the thyroid gland.
2. With the gland gone, there are no cells left to make thyroid hormone.
3. Thyroid hormone levels drop to zero within days (half-life ~7 days).
4. You start levothyroxine right away to replace what the body needs.

SAFETY WATCH

Complications (rare but important): damage to the recurrent laryngeal nerve (the nerve to the vocal cords) causing hoarse voice or trouble breathing; injury to the parathyroid glands (sit behind the thyroid) causing low calcium (hypoparathyroidism), which needs calcium and vitamin D supplements. · Surgical risks: bleeding, infection, anesthesia reactions. · Best outcomes with a high-volume thyroid surgeon (>25 thyroidectomies/year). · Need lifelong levothyroxine and regular TSH monitoring.

NSAIDs (ibuprofen, naproxen) or aspirin for thyroiditis · Nonsteroidal anti-inflammatory drug (NSAID) or salicylate

In subacute (painful) thyroiditis, the thyroid gland is inflamed and tender—often after a viral infection. NSAIDs block the enzyme cyclooxygenase (COX), which makes prostaglandins that cause pain, fever, and swelling. Lowering prostaglandins eases the neck pain and fever. The thyroid will heal on its own in weeks to months; NSAIDs just make you comfortable while you wait.

1. A virus or immune trigger damages thyroid cells.
2. Damaged cells release their stored thyroid hormone into the blood (causing temporary hyperthyroidism) and also release chemical signals (cytokines, prostaglandins) that cause pain and inflammation.
3. NSAIDs block COX enzymes, so fewer prostaglandins are made.
4. Less prostaglandin means less pain, less fever, and less swelling in the gland.
5. The gland heals over 2–6 months. Thyroid hormone levels often swing low (hypothyroid phase) before returning to normal.

SAFETY WATCH

Stomach upset, ulcers, bleeding—take with food; avoid in people with ulcer history or on blood thinners. · Kidney injury with long-term use or in people with existing kidney disease, heart failure, or dehydration. · Can raise blood pressure a bit. · Aspirin: higher risk of stomach bleeding and Reye syndrome in children with viral illness—avoid aspirin in kids; use ibuprofen instead. · If NSAIDs do not control pain, a short course of prednisone (steroid) often works very well in subacute thyroiditis.

Prednisone (corticosteroid, for severe thyroiditis pain) · Glucocorticoid (steroid anti-inflammatory)

Prednisone is a powerful anti-inflammatory. It turns down the immune system and blocks many inflammation signals at once. In subacute thyroiditis, when the gland is very swollen and painful and NSAIDs are not enough, a short course of prednisone (2–4 weeks, tapered down) quickly eases pain and shrinks the swelling. It does NOT lower thyroid hormone; it just calms the inflammation.

1. Prednisone enters cells and binds to glucocorticoid receptors.
2. This receptor complex moves into the cell nucleus and changes which genes are turned on or off.
3. It shuts down genes that make inflammatory cytokines (like IL-1, TNF-alpha) and blocks immune cells (T cells, macrophages) from swarming into the thyroid.
4. It also stabilizes cell membranes so less stored thyroid hormone leaks out (minor effect).

5. Pain, swelling, and fever drop within 24–48 hours.
6. The drug is tapered over 2–6 weeks to avoid rebound inflammation when you stop.

SAFETY WATCH

Short-term risks (2–4 week course): high blood sugar (watch in diabetes), mood swings, trouble sleeping, increased appetite, fluid retention. · Do NOT stop suddenly after >2 weeks of use—taper the dose to let the adrenal glands wake back up. · Can raise blood pressure and unmask infections (steroids suppress immune defense). · Long-term use (not typical in thyroiditis) risks osteoporosis, weight gain, cataracts, adrenal suppression—but thyroiditis treatment is short, so these are not major concerns.

Levothyroxine (for the hypothyroid phase after thyroiditis or RAI/surgery) · Thyroid hormone replacement (synthetic T4)

Levothyroxine is a pill form of T4 (the main thyroid hormone). After radioactive iodine, surgery, or the late phase of thyroiditis, your thyroid gland can't make enough hormone on its own. Taking levothyroxine every morning replaces what's missing. Your body converts T4 into T3 (the active form) as needed, so one daily pill keeps your metabolism, heart, and brain running normally.

1. You swallow levothyroxine on an empty stomach (works best 30–60 minutes before breakfast).
2. The pill dissolves and T4 is absorbed in the small intestine.
3. T4 travels in the blood, mostly stuck to binding proteins.
4. In cells, an enzyme (deiodinase) clips one iodine off T4 to make T3.
5. T3 enters the cell nucleus and binds to thyroid hormone receptors, turning on genes that control metabolism, heart rate, heat production, brain function, and growth.

SAFETY WATCH

Too much causes hyperthyroid symptoms: fast heart, shaking, weight loss, anxiety, bone loss over time. Too little causes hypothyroid symptoms: fatigue, weight gain, slow heart, depression. · Check TSH every 6–8 weeks when starting or adjusting dose, then yearly once stable. · Take on an empty stomach, same time every day. Wait 4 hours after levothyroxine before taking iron, calcium, antacids (they block absorption). · In elderly or people with heart disease, start low and go slow—sudden high thyroid hormone can trigger chest pain or arrhythmia. · Pregnancy increases levothyroxine need by ~30%; check TSH every trimester and adjust dose.

Red flags

- Fever over 104°F with confusion or agitation (possible thyroid storm)
- Heart rate above 140 at rest, chest pain, or trouble breathing (heart strain or failure)
- Sudden severe vision loss or eye pain (rare but urgent eye complication in Graves disease)
- New weakness in arms or legs, especially with low potassium on labs (thyrotoxic periodic paralysis)

Nursing priorities

1. Monitor heart rate and rhythm every 2 to 4 hours and report rate above 120 or new irregularity immediately — Excess thyroid hormone increases cardiac output (amount of blood the heart pumps) and can trigger atrial fibrillation (an irregular, quivering heartbeat); rapid rates above 150 can cause demand starved blood flow (ischemia) (heart muscle not getting enough oxygen) even without blocked arteries
2. Check temperature every 4 hours and notify provider if it rises above 100.4°F — Thyroid hormone increases basal metabolic rate (baseline energy burn), so patients already run warm; fever above 100.4°F may signal infection or early thyroid storm, which has 20 to 30 percent mortality even with treatment
3. Administer beta-blocker medication as prescribed and hold if heart rate is below 60 or systolic blood pressure is below 100 — Beta-blockers like propranolol block adrenaline receptors to slow heart rate and reduce tremor and anxiety within hours; they do not lower thyroid hormone but control dangerous cardiac effects while waiting for anti-thyroid drugs to work over weeks
4. Give methimazole or PTU at the same time every day and teach patient never to stop suddenly without provider guidance — These thionamide drugs block thyroid peroxidase (enzyme that makes thyroid hormone) and take 4 to 6 weeks to normalize levels; stopping abruptly can cause rebound hyperthyroidism or precipitate thyroid storm
5. Provide a cool, quiet, low-stimulation environment and offer frequent small high-calorie meals — Hypermetabolism increases calorie needs by 10 to 20 percent and heat production; patients are heat intolerant and irritable; a calm cool room reduces cardiac workload and agitation while extra calories prevent muscle wasting
6. Assess eyes for redness, dryness, and ability to close lids completely; provide lubricating eye drops if lids do not close fully — Graves ophthalmopathy (eye disease) causes lid retraction (upper lid pulls back) and proptosis (eyeball pushes forward); incomplete lid closure during sleep leads to corneal ulcers (open sores on the clear eye surface) and infection risk

7. Monitor for signs of agranulocytosis (severe white blood cell drop): fever, sore throat, mouth sores, or infection; hold anti-thyroid drug and notify provider immediately if these appear — Methimazole and PTU can destroy neutrophils (infection-fighting white cells) in 0.3 percent of patients, usually in the first 90 days; without neutrophils, bacterial infections become life-threatening sepsis within hours
8. Teach patient to avoid iodine-rich foods like kelp and seaweed, and to tell all providers about thyroid diagnosis before any imaging with contrast dye — Excess iodine provides raw material for more thyroid hormone production and can worsen hyperthyroidism or interfere with upcoming radioactive iodine treatment, which relies on the gland being iodine-hungry to take up the therapy

Sources

- American Thyroid Association 2016 Hyperthyroidism Guideline
- Endocrine Society Clinical Practice Guideline 2011 (updated review 2016)
- UpToDate Clinical Reference (Thyrotoxicosis)
- NEJM Clinical Practice Review on Hyperthyroidism

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