

Wernicke Encephalopathy

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THE STORY IN ONE SENTENCE

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“Always give thiamine BEFORE giving sugar (dextrose or IV fluids with glucose) to any patient at risk—alcoholism, malnutrition, vomiting, or refeeding—because sugar without thiamine can trigger or worsen Wernicke encephalopathy.”

Normal anatomy — the body's job

Thiamine (vitamin B1) is the essential helper molecule that lets every cell—especially brain cells—turn sugar (glucose) into usable energy (ATP). The brain runs on a tight energy budget and has almost no backup fuel, so it depends on a steady thiamine supply every single day.

BODY'S JOB

Thiamine works inside the mitochondria (the cell's power plant) as a partner to key enzymes—alpha-ketoglutarate dehydrogenase, pyruvate dehydrogenase, and transketolase—that break down glucose step by step and release energy. Think of thiamine as the gear oil that keeps the energy engine turning. Without it, glucose piles up but cannot be burned, and the cell starves even when sugar is everywhere.

1. You eat food containing carbohydrates (bread, rice, fruit, alcohol).
2. Your small intestine absorbs thiamine from food and sends it into the bloodstream.
3. Thiamine travels to every cell, especially brain neurons and heart muscle, which burn the most energy.
4. Inside the mitochondria, thiamine activates enzymes that crack open glucose and pull out ATP (the energy molecule).

5. The brain's memory center (mammillary bodies and thalamus) and balance center (cerebellum and brainstem) need the most thiamine because they fire constantly.
6. Any leftover thiamine is stored in the liver and muscles, but those stores only last about 18 days if you stop taking in thiamine.

What breaks

Wernicke encephalopathy happens when thiamine runs out and the brain cannot make energy from glucose anymore. The break is not in one organ—it is in every cell's power plant. The brain regions that burn the most energy (the mammillary bodies and thalamus for memory, the cerebellum and brainstem for balance and eye movement) die first.

CLINICAL TERM

Thiamine depletion halts the Krebs cycle (the cell's main energy loop) because the enzymes that depend on thiamine—pyruvate dehydrogenase, alpha-ketoglutarate dehydrogenase, and transketolase—cannot work. Glucose and pyruvate pile up but cannot be burned, so neurons switch to less efficient pathways that make lactic acid and free radicals, which poison the cell. The mammillary bodies, medial thalamus, periaqueductal gray matter, and cerebellar vermis are especially vulnerable because they have high metabolic demand and dense capillary networks. Giving a glucose load (IV dextrose or high-carb refeeding) without thiamine floods these cells with substrate they cannot process, accelerates energy failure, and triggers excitotoxicity (too much glutamate, the brain's main excitatory signal, which overstimulates neurons until they die). That is why you see the triad of confusion (thalamus and mammillary body injury), loss of coordination (ataxia) (cerebellar injury), and ophthalmoplegia or involuntary eye jerking (nystagmus) (brainstem cranial nerve nuclei injury)—but only about 10–16% of patients show all three. The rest show altered mental status alone, or loss of coordination alone, or hypothermia and low blood pressure (hypotension) from autonomic dysregulation.

How the body compensates

The body has almost no way to compensate for thiamine deficiency because thiamine is not made in the body—it must come from food. The liver and muscles store a small reserve, but it runs out in 2–3 weeks. Once stores are gone, the brain and heart begin to fail, and vital signs can look deceptively normal until sudden cardiovascular collapse or irreversible brain injury occurs.

1. The liver and muscle release stored thiamine to keep blood levels steady for the first 2–3 weeks of poor intake—this is the silent window where the patient may have no symptoms but stores are draining.

2. The brain tries to lower its energy demand by slowing non-essential functions—early confusion, apathy, and slowed thinking are the brain rationing power to survival circuits.
3. The autonomic nervous system becomes unstable because the brainstem nuclei that control heart rate, blood pressure, and temperature need thiamine—this causes hypothermia (body temperature often 35–36°C / 95–96.8°F) and low blood pressure (hypotension) that looks like sepsis or shock.
4. The heart muscle, which also burns huge amounts of energy, begins to fail if thiamine is not replaced—this is called wet beriberi (high-output heart failure with warm extremities, bounding pulses, swelling (edema), and eventually shock).
5. The danger point: vital signs may look stable or only mildly abnormal (slight confusion, slight low BP, slight hypothermia) while irreversible brain damage is happening—the triad (confusion, loss of coordination (ataxia), eye findings) is present in fewer than 20% of cases, so you must think of Wernicke encephalopathy based on risk factors and subtle signs, not wait for the textbook picture.
6. Once neurons die in the mammillary bodies and thalamus, memory cannot be fixed—the patient develops Korsakoff syndrome (permanent anterograde amnesia, confabulation, apathy) even if you give thiamine late.
7. The window to reverse symptoms is hours to days—giving thiamine early (within 24–48 hours of symptom onset) can reverse confusion, eye findings, and loss of coordination (ataxia) in most patients; waiting longer makes recovery incomplete or impossible.

Domino effect

1. Thiamine stores run out (from alcohol use disorder, malnutrition, hyperemesis, or malabsorption). — Chronic alcohol blocks thiamine absorption in the intestine, increases urinary loss, and impairs conversion of thiamine to its active form (thiamine pyrophosphate). Poor diet, repeated vomiting, or gastrointestinal surgery (bariatric surgery, gastrectomy) also prevent thiamine from getting into the body. Stores last only 18 days, so depletion happens fast.
2. Mitochondria cannot make ATP, so brain cells starve for energy. — Thiamine is the required co-factor for pyruvate dehydrogenase (converts pyruvate to acetyl-CoA), alpha-ketoglutarate dehydrogenase (runs the Krebs cycle), and transketolase (runs the pentose phosphate pathway). Without thiamine, all three pathways stop, ATP production drops by 60–80%, and neurons cannot power ion pumps, neurotransmitter synthesis, or membrane repair.

3. Giving IV dextrose or starting refeeding WITHOUT giving thiamine first triggers acute Wernicke encephalopathy. — A glucose load forces the few remaining thiamine molecules to be consumed instantly trying to process the flood of sugar. This sudden depletion crashes energy production in the most vulnerable brain regions (mammillary bodies, thalamus, periaqueductal gray, cerebellar vermis), and excitotoxicity and free-radical damage kill neurons within hours.
4. The patient develops the classic triad: confusion (encephalopathy), loss of coordination (ataxia) (wide-based, unsteady gait), and ophthalmoplegia or involuntary eye jerking (nystagmus) (eyes not moving together or jerking). — The medial thalamus and mammillary bodies (memory and alertness centers) die, causing confusion and disorientation. The cerebellar vermis (balance and coordination center) dies, causing loss of coordination (ataxia). The brainstem nuclei controlling cranial nerves III (oculomotor), IV (trochlear), and VI (abducens) die, causing eye movement problems—involuntary eye jerking (nystagmus) (rhythmic eye jerking, often horizontal) or ophthalmoplegia (paralysis of eye muscles, causing double vision or inability to look in certain directions).
5. Autonomic dysregulation causes hypothermia and low blood pressure (hypotension). — The brainstem autonomic centers that regulate body temperature and blood pressure need thiamine to function. When they fail, core temperature drops (often 35–36°C) and blood pressure falls, mimicking sepsis or shock—but fluids and pressors do not fix it; only thiamine does.
6. Peripheral nerves lose their myelin coating, causing neuropathy. — Schwann cells need thiamine to maintain the myelin sheath around peripheral nerves. Thiamine deficiency causes distal symmetric polyneuropathy—numbness, tingling, burning pain, and loss of ankle reflexes in a stocking-glove distribution. This is often called dry beriberi.
7. If thiamine is not given in time, confusion becomes permanent (Korsakoff syndrome). — Neurons in the mammillary bodies and medial thalamus die permanently, leaving the patient with severe anterograde amnesia (cannot form new memories), confabulation (makes up stories to fill memory gaps), apathy, and lack of insight. About 80% of untreated Wernicke patients progress to Korsakoff, and only 20% of those recover meaningful memory function even with thiamine.
8. The heart can fail (wet beriberi) or the patient can die from cardiovascular collapse. — The heart muscle has extremely high energy demand. Thiamine deficiency causes high-output heart failure (the heart pumps fast but weakly, causing warm skin, bounding pulses, wide pulse pressure, pulmonary edema (fluid flooding the lungs), and eventual cardiogenic shock). This is called wet beriberi, and it can be fatal within hours if thiamine is not given.

Symptoms · what the patient feels, and why

- **Confusion or trouble thinking clearly:** The thalamus (the brain's relay station that sorts incoming messages) and the mammillary bodies (memory hubs near the base of the brain) are starving for thiamine (vitamin B1), so they cannot process information or form memories properly.
- **Unsteady walk or trouble balancing (ataxia):** The cerebellum (the brain's balance and coordination center at the back) is damaged by thiamine shortage, so signals to the legs and trunk get scrambled and muscles lose smooth teamwork.
- **Abnormal eye movements — eyes may drift, shake, or not move together (nystagmus or ophthalmoplegia):** The brainstem nuclei (tiny control centers in the brainstem that steer the eye muscles) need thiamine to fire properly; without it, the eyes lose their coordinated aim and may jiggle or freeze in certain directions.
- **Double vision:** Damage to the nerves controlling eye muscles — especially the sixth cranial nerve (the abducens nerve, which pulls the eye outward) — means the two eyes cannot point at the same spot, so the brain sees two images.
- **Droopy eyelid (ptosis):** The third cranial nerve (the oculomotor nerve, which lifts the upper eyelid and moves the eye in most directions) is weakened by thiamine deficiency, so the lid falls and the eye cannot open fully.
- **Low blood pressure when standing (orthostatic low blood pressure (hypotension)) or fast heart rate:** The autonomic nervous system (the automatic pilot that adjusts blood vessel tightness and heart rate) is damaged, so blood pools in the legs when you stand and the heart races to try to keep enough blood reaching the brain.
- **Low body temperature (hypothermia):** The hypothalamus (the brain's thermostat, deep in the center) loses control of temperature regulation when thiamine runs out, so the body cannot hold its normal 98.6 °F and may drop dangerously low.
- **Memory loss, especially for new events, and tendency to make up stories to fill gaps (confabulation):** Severe damage to the mammillary bodies and parts of the thalamus prevents the brain from recording new memories, so the person cannot remember what just happened and may unknowingly invent details to cover the blanks.

Focused exam

- **Confusion or memory loss that gets worse fast:** Wernicke encephalopathy damages the mammillary bodies and thalamus (the brain's memory-making centers). When thiamine runs out, these parts cannot make energy from sugar, so they misfire and stop forming new memories. This is the biggest warning sign.

- Eyes that do not move side-to-side the right way, or eyes that bounce when looking at something (nystagmus): Thiamine shortage damages the abducens and oculomotor nuclei (the brain stems' eye-control centers) and the nerves that run the eye muscles. This causes double vision, gaze palsy, or rhythmic bouncing. It is one of the three classic signs.
- Walking that looks drunk or wobbly (ataxia), even when the patient did not drink today: The cerebellum and vestibular nuclei (the brain's balance centers) need thiamine to coordinate movement. Without it, the body cannot walk a straight line, stand steady, or keep balance.
- Body temperature below 96°F (35.5°C), or the patient feels cold when the room is warm: Wernicke encephalopathy damages the hypothalamus (the brain's thermostat). The body loses its ability to sense cold or turn up the heat, so core temperature drops.
- Tingling or numbness in the hands and feet, or weakness that came on over weeks: Long-term thiamine shortage damages the peripheral nerves in the arms and legs. This is a clue the body has been missing thiamine for a long time, not just today.
- Patient just drank a lot of alcohol, threw up for days, had stomach surgery, or ate almost nothing for weeks: All of these wash away thiamine or block the gut from absorbing it. This history tells you to suspect Wernicke encephalopathy even before the full triad shows up.

Diagnostics · every test answers a question

- Serum thiamine level (whole blood or plasma) → Is the body running low on thiamine right now?
- Red blood cell transketolase activity (functional thiamine assay) → Is the enzyme that uses thiamine working inside the cells?
- Serum magnesium → Is there enough magnesium for thiamine to work inside the cells?
- Brain MRI with FLAIR and diffusion-weighted imaging (DWI) → Is there swelling or damage in the brain's thiamine-sensitive memory and balance centers?
- Serum glucose (or fingerstick blood sugar) → Is blood sugar low enough to cause confusion on its own, or high enough that giving more sugar without thiamine will crash the brain?
- Liver function tests (AST, ALT, GGT, bilirubin) → Is chronic alcohol use or liver disease blocking thiamine absorption or storage?
- Complete blood count (CBC) with mean corpuscular volume (MCV) → Is there anemia or other vitamin deficiency from malnutrition or alcohol?

Treatment logic

- IV thiamine 500 mg three times daily for 2–3 days, then 250 mg daily for 3–5 days, then 100 mg by mouth daily → Thiamine is the cofactor that lets the enzyme alpha-ketoglutarate dehydrogenase turn sugar into energy inside the mitochondria (the cell's power plants). Without thiamine, the brain cells in the mammillary bodies, thalamus, and cerebellum starve and misfire, causing confusion, eye jerking, and stumbling. High-dose IV thiamine floods the cells with the missing cofactor so energy production restarts and the misfiring stops.
- IV magnesium sulfate 2 grams over 15–30 minutes, then 1–2 grams daily while giving thiamine → Magnesium is the mineral cofactor that thiamine-dependent enzymes (transketolase and alpha-ketoglutarate dehydrogenase) need to actually work inside the cell. Without magnesium, thiamine sits unused even if you give plenty of it. Replacing magnesium fixes the broken enzyme system so thiamine can do its job.
- Do NOT give IV dextrose (sugar water) or oral glucose before thiamine is on board → Sugar floods the brain with demand for energy at the exact moment there is no thiamine to make that energy. The mitochondria try to burn the sugar, fail, and produce toxic lactate instead, which damages neurons faster. Giving thiamine first prevents this metabolic crash.
- Treat alcohol withdrawal with benzodiazepines (lorazepam 1–2 mg IV every 6 hours or diazepam 5–10 mg IV every 6 hours, adjusted to CIWA score) and address the underlying alcohol use disorder with counseling and medication-assisted treatment → Stopping alcohol removes the root cause that depleted thiamine (alcohol blocks gut absorption and increases urinary loss). Benzodiazepines boost GABA (the brain's main calming signal) to prevent seizures and delirium tremens, which would add more metabolic stress and worsen brain injury.
- Give balanced nutrition with thiamine 100 mg by mouth daily, plus B-complex vitamins (folate 1 mg, B12 1000 mcg, B6 50 mg), plus regular meals with adequate carbohydrate, protein, and fat → Normal food and other B vitamins support the brain's recovery by keeping all the fuel-making enzymes (not just the thiamine-dependent ones) running smoothly. This prevents relapse and helps repair other malnutrition damage (peripheral neuropathy, anemia).
- Monitor core body temperature, eye movements (nystagmus, gaze palsy), mental status (orientation, memory), and gait every 4–6 hours during the first 2–3 days → Early detection of worsening confusion, eye findings, loss of coordination (ataxia), or hypothermia tells you if thiamine is working or if the brain is still in trouble. This lets you escalate IV thiamine dosing or add other support (warming blankets, ICU monitoring) before permanent damage locks in.

- Physical and occupational therapy starting in the hospital and continuing after discharge → Retraining balance and coordination helps the cerebellum and vestibular system relearn old movement patterns after thiamine restarts energy production. This speeds functional recovery and reduces fall risk.

Medications

Thiamine (vitamin B1) · Essential vitamin replacement

Thiamine is a vitamin the brain must have to turn sugar into energy. When the body runs low on thiamine for weeks (usually because of heavy alcohol use or poor nutrition), brain cells start to starve even when blood sugar is normal. The parts of the brain that handle eye movement, balance, and forming new memories need the most energy, so they fail first. Giving thiamine refills the empty tank so the brain can make energy again and stop the damage. We give it through an IV (directly into a vein) at first because the gut may not absorb it well in someone who is very sick or has been drinking heavily for a long time.

1. Thiamine enters the bloodstream and crosses into the brain.
2. Inside each brain cell, thiamine is turned into thiamine pyrophosphate (the active form the cell can use).
3. Thiamine pyrophosphate acts as a helper (a cofactor) for three enzymes that break down sugar in the mitochondria: transketolase, pyruvate dehydrogenase, and alpha-ketoglutarate dehydrogenase.
4. With these enzymes working again, the cell can turn glucose (blood sugar) into ATP (the energy currency cells need to stay alive and send signals).
5. Brain cells that were starving for energy start to recover, and swelling and inflammation in the thalamus, mammillary bodies, and brainstem begin to reverse.
6. Eye movements, balance, and alertness often improve within hours to days; memory problems take longer and may not fully reverse if treatment is delayed.

SAFETY WATCH

NEVER give IV dextrose (sugar water) BEFORE giving thiamine in anyone at risk for Wernicke encephalopathy (chronic alcohol use, severe vomiting, eating disorder, recent weight-loss surgery). A sudden sugar load when thiamine is already very low can push the brain over the edge and trigger or worsen the encephalopathy. · Give 500 mg of thiamine IV three times a day for 2–3 days, then 250 mg IV or intramuscular once a day for another 3–5 days, then 100 mg by mouth daily for as long as risk factors continue (often lifelong in people with alcohol use disorder). · Check magnesium and replace it (usually 2 grams IV over 15–30 minutes, then 1–2 grams per day) because low magnesium blocks thiamine from working. · Watch for a rare allergic reaction during the first IV dose (flushing, itching, trouble breathing); have diphenhydramine and epinephrine ready, and give the first dose slowly. · Provide all the other B vitamins (a multivitamin or B-complex) because people with thiamine deficiency are often low in several vitamins. · Treat alcohol withdrawal separately with a benzodiazepine (such as lorazepam or diazepam) on a symptom-triggered or fixed-dose schedule to prevent seizures and delirium tremens. · Give balanced nutrition (food or tube feeding) as soon as it is safe, because thiamine and other vitamins come from food in the long run. · Address the root cause: refer for alcohol use disorder treatment, treat persistent vomiting or malabsorption, and monitor anyone at high risk (eating disorders, cancer, dialysis, hyperemesis gravidarum) for early signs.

Red flags

- Confusion that gets worse quickly — brain swelling or bleeding may be starting
- Eyes that cannot move in all directions or pupils that are unequal — pressure on brainstem nerves is a medical emergency
- Body temperature below 95 °F (35 °C) or very slow heart rate — the brain's automatic controls are failing and the person may collapse
- Seizures or sudden loss of consciousness — the brain is in crisis and needs thiamine and support immediately
- Signs of bleeding in the brain (severe headache, vomiting, neck stiffness, vision changes) — rare but life-threatening complication of Wernicke encephalopathy

Nursing priorities

1. Check if IV dextrose or oral glucose has been given or ordered, and hold it until after the first dose of thiamine 500 mg IV is infused — The brain needs thiamine to turn glucose into energy; giving sugar without thiamine forces the brain to use its last thiamine reserves and causes brain cells in the thalamus (memory center) and mammillary bodies (balance center) to swell and die within hours

2. Give thiamine 500 mg IV diluted in 100 mL normal saline over 30 minutes three times daily for at least 2 days before switching to lower doses — Oral thiamine absorbs poorly, especially in people who drink alcohol or vomit, and the blood level stays too low to reverse brain damage; IV thiamine at 500 mg reaches the brain quickly and saturates the damaged cells so they can start making energy again and stop dying
3. Give magnesium sulfate 2 grams IV over 15 to 30 minutes, then continue 1 to 2 grams IV daily while the patient receives thiamine — Thiamine cannot work inside cells without magnesium because magnesium activates the enzyme thiamine pyrophosphokinase that turns thiamine into its active form; alcohol use and malnutrition deplete magnesium, so replacing both together prevents treatment failure
4. Assess mental status every 2 to 4 hours using a simple question like 'What is your name and where are you?' and document any worsening confusion or new inability to stay awake — Confusion that gets worse despite thiamine may mean brain swelling, bleeding, or progression to Korsakoff syndrome (permanent memory damage); catching this early allows the team to order a head CT scan and increase thiamine frequency or add other treatments
5. Check eye movements every 4 hours by asking the patient to follow your finger up, down, left, and right, and report if the eyes do not move together or if one eye drifts — Eye movement problems come from damage to cranial nerves III, IV, and VI in the brainstem; if the eyes get worse or pupils become unequal, pressure on the brainstem is increasing and the patient may stop breathing without immediate intervention
6. Monitor the patient on continuous pulse oximetry and telemetry (heart monitor), and measure blood pressure and heart rate every 2 hours, reporting heart rate below 50 or systolic blood pressure below 90 mm Hg — Wernicke encephalopathy damages the hypothalamus and medulla (automatic control centers), causing dysautonomia (loss of automatic functions) with dangerous drops in blood pressure, body temperature, and heart rate that can lead to sudden cardiac arrest
7. Keep the bed in the lowest position with all four side rails up, assist with every transfer, and use a gait belt when the patient walks — Loss of coordination (Ataxia) (unsteady walk) comes from damage to the cerebellum (balance center) and often does not improve even after thiamine treatment; falls cause head injury and hip fractures that worsen outcomes, especially in older adults and people with osteoporosis
8. If the patient drinks alcohol regularly, start a CIWA (Clinical Institute Withdrawal Assessment) protocol and give scheduled benzodiazepines like lorazepam 1 to 2 mg IV every 6 hours to prevent withdrawal seizures — Alcohol withdrawal and Wernicke encephalopathy often happen at the same time, and withdrawal seizures increase brain oxygen demand when the brain already cannot make energy; treating both conditions together prevents seizures from causing more permanent brain damage

Sources

- EFNS Guidelines on Wernicke Encephalopathy 2010
- Royal College of Physicians (RCP) Alcohol Guidelines
- Cochrane Database Systematic Reviews
- American Academy of Neurology (AAN)

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