

Peripheral Neuropathy

Peripheral neuropathy: find what is hurting the nerve, stop it, and treat the symptoms while the nerve tries to heal.

THE ONE-LINE TRUTH

The long wires that carry messages from your brain to your toes and fingers are breaking down.

The chain — how it unfolds

1. The longest sensory axons — from your toes to your spinal cord — start dying from the tip backward, because they are farthest from the cell body and hardest to feed. — why: Diabetic damage to tiny blood vessels, B12 deficiency, alcohol toxicity, or chemotherapy poison all nerves, but the three-foot nerve to your big toe needs the most oxygen and building materials, so it fails first while shorter nerves to your knee are still fine.
2. You lose sensation in both feet in a symmetric stocking pattern — numbness, tingling, or a dead feeling that starts at the toes and creeps up toward the knee over months to years. — why: The tips of the longest sensory axons die first, so the skin of the toes stops sending touch, pain, and temperature signals to the brain; as more axon dies back, the numb zone climbs higher, like a stocking being pulled on.
3. You lose proprioception (position sense) from your feet, so you cannot feel where your feet are unless you look at them. — why: Specialized sensory axons from joint and muscle receptors carry the 'where-am-I' map to the brain; when these die, the brain loses the map, and you walk like you are on a boat in the dark — unsteady, wide-based, and at high risk for falls.
4. Neuropathic pain starts — burning, stabbing, electric-shock, or pins-and-needles sensations in the feet and hands, often worse at night. — why: Dying or demyelinated axons become hyperexcitable and fire randomly; the brain interprets this electrical noise as intense pain even though nothing is touching the skin. Bare nerve endings sprout pain receptors (nociceptors) to try to detect danger, worsening the pain.

5. Motor axons to foot and ankle muscles die, causing weakness — foot drop (cannot lift the front of the foot), trouble standing on tiptoes, and a slapping gait. — why: Motor neurons in the spinal cord send long axons to command the tibialis anterior (lifts the foot) and gastrocnemius (points the foot down); when those motor axons die from the tip backward, muscles stop receiving signals, weaken, and shrink (atrophy).
6. Deep tendon reflexes disappear — tapping the Achilles tendon or patellar tendon produces no ankle jerk or knee jerk. — why: The reflex arc requires a sensory axon to carry the stretch signal from the muscle spindle to the spinal cord and a motor axon to carry the 'contract' signal back to the muscle; if either axon is dead or demyelinated, the loop is broken and the reflex is gone (areflexia).
7. If the neuropathy is severe or affects autonomic nerves, you may get orthostatic low blood pressure (hypotension) (blood pressure drops when you stand, causing dizziness or fainting), gastroparesis (stomach empties slowly, causing nausea and bloating), or loss of sweating in the feet. — why: Autonomic axons control blood vessel constriction, gut movement, and sweat glands; when they die, blood pools in the legs when you stand (causing fainting), the stomach does not push food into the intestines (causing nausea), and feet stop sweating (causing dry, cracked skin that is prone to infection).
8. In demyelinating neuropathies like Guillain-Barré syndrome, weakness ascends rapidly from legs to arms to face and breathing muscles over days. — why: Antibodies strip myelin from nerves throughout the body, blocking signals; the longest nerves (legs) fail first, then arms, then cranial nerves (causing double vision, trouble swallowing) and phrenic nerves to the diaphragm, leading to respiratory failure and death if not treated with IVIG or plasmapheresis in the ICU.

Spot it — what you see, and why

- Tingling or burning in the hands and feet: The outer coating (myelin sheath) or the long wire part (axon) of the nerve is damaged, so the nerve sends mixed-up signals that the brain reads as tingling or burning even though nothing is actually hot or poking the skin.
- Numbness starting in the toes and fingers and slowly moving up: The longest nerves (the ones reaching the feet and hands) run out of fuel or get poisoned first, so they stop sending touch signals back to the brain. It creeps up because shorter nerves higher up the limb are healthier longer.
- Weakness in the feet or hands, such as trouble lifting the toes or gripping: The motor nerves (the wires that tell muscles to move) are damaged, so the signal from the brain doesn't reach the muscle strongly enough to make it contract.
- Sharp, shooting, or electric-shock pains: Damaged nerve fibers fire off spontaneous bursts of electricity like a frayed wire sparking, and the brain interprets those random bursts as sudden stabs of pain.

- Loss of balance or feeling unsteady when walking: The sensory nerves in the feet normally tell the brain where the ground is and how the body is positioned (proprioception). When those signals are lost, the brain can't adjust balance well, especially in the dark or on uneven ground.
- Extreme sensitivity to light touch (allodynia): Injured nerves can become hyperexcitable, meaning even a gentle touch (like bedsheets) is read by the damaged nerve as a much stronger, painful signal.

Red flags — act now

- Sudden weakness in one arm or leg, or weakness that affects only one side of the body
- Trouble breathing, swallowing, or talking
- Symptoms that get much worse over days to weeks instead of months to years
- Loss of bowel or bladder control
- Severe pain that does not respond to usual pain medicines

Treat it — in this order

1. Identify and treat the root cause: control blood sugar in diabetes (target A1c < 7%), replace vitamin B12 (1000 mcg intramuscular weekly for deficiency), stop alcohol, adjust or stop neurotoxic chemotherapy, treat hypothyroidism, or manage severe waste buildup in the blood (uremia) with dialysis → Stops the upstream break. If the axon is starving for glucose control or B12, fixing that halts the damage so nerves stop dying. If a toxin (alcohol, chemo drug, uremic waste) is poisoning the nerve, removing it stops further injury. This is the only intervention that can prevent progression.
2. Duloxetine (30–60 mg daily) or gabapentin (300–3600 mg daily in divided doses) or pregabalin (150–300 mg daily in divided doses) for neuropathic pain → These drugs quiet the pain signals in the dorsal horn of the spinal cord and in the brain. Damaged nerves misfire and send pain messages even when nothing is hurting you. Duloxetine raises serotonin and norepinephrine (brain chemicals that turn down pain volume). Gabapentin and pregabalin block calcium channels on nerve endings so they stop firing randomly. This makes the burning, tingling, and stabbing pain feel less loud.

3. Intravenous immunoglobulin (IVIG, 2 g/kg over 2–5 days) or plasmapheresis (plasma exchange, 5 sessions over 10–14 days) for acute inflammatory demyelinating polyneuropathy (Guillain-Barré syndrome) or chronic inflammatory demyelinating polyneuropathy (CIDP) → IVIG floods the bloodstream with normal antibodies that distract and block the rogue antibodies attacking myelin. Plasmapheresis physically filters out the bad antibodies from the blood. Both stop the immune attack on the myelin coat so the nerve signal highway stays intact. This prevents paralysis and can reverse weakness if started early (within 2 weeks of symptom onset for Guillain-Barré).
4. Daily foot inspection, properly fitted shoes, fall prevention (remove rugs, use a cane or walker, install grab bars), balance training, and physical therapy to keep muscles strong → Prevents the catastrophic complications of neuropathy: foot ulcers, infections, amputations, and fractures from falls. Physical therapy teaches you to use vision and upper-body strength to compensate for lost position sense and keeps leg muscles from shrinking even when the nerve signal is weak.
5. Topical lidocaine 5% patches or capsaicin 8% patches applied to painful skin areas → Lidocaine numbs the nerve endings in the skin so pain signals from that spot stop reaching the brain. Capsaicin (the chemical in hot peppers) exhausts substance P (the pain messenger stored in nerve endings) so the nerve runs out of ammunition to send pain signals. Both work only on the skin surface where you apply them.

Your top priorities at the bedside

1. Assess respiratory effort, oxygen saturation, and ability to cough every four hours if patient has new or worsening weakness — why: GBS and CIDP (chronic inflammatory demyelinating polyneuropathy) can ascend to the phrenic nerve (nerve controlling the diaphragm). Fifteen to thirty percent of GBS patients need mechanical ventilation (breathing machine). Early detection of shallow breathing or falling vital capacity (lung volume) prevents respiratory arrest.
2. Inspect both feet daily for skin breaks, redness, blisters, or foreign objects; ensure patient or family demonstrates proper foot checks — why: Sensory loss means patients cannot feel cuts, pressure sores, or penetrating objects. Minor trauma becomes a portal for bacteria. Daily inspection catches early cellulitis (skin infection) or ulcers before they extend to bone or require amputation.
3. Implement fall precautions including non-skid footwear, bedside commode, call bell within reach, and physical therapy consult for gait training — why: Loss of proprioception (position sense) and muscle weakness impair balance. Peripheral neuropathy triples fall risk. Hip fractures and head injuries increase mortality in older adults. Strengthening exercises and assistive devices reduce falls by forty percent.