

Peripheral Neuropathy

A plain-English guide for you and your family

WHAT IS HAPPENING

Think of your nerves as long wires running from your spinal cord out to your toes and fingers. Each wire has a center cable called the axon that carries the electrical message, wrapped in a fatty insulation called myelin (like the plastic coating on a phone charger) that speeds the signal and protects it. Cells called Schwann cells build and repair that myelin coat. Blood vessels feed the axon with oxygen and sugar. When everything works, you can feel a feather on your toe, know where your foot is without looking, and command your muscles to move. Peripheral neuropathy means those long wires are failing — either the axon itself is starving because of diabetes, low vitamin B12, too much alcohol, chemotherapy drugs, or kidney failure (uremia), or the myelin coat is being stripped away by the immune system attacking it (as in Guillain-Barré syndrome or CIDP) or by genes that build faulty myelin (hereditary neuropathies like Charcot-Marie-Tooth). The longest wires fail first because they are farthest from the spinal cord and hardest to feed, so numbness starts in the toes and slowly creeps up like a stocking, then into the fingertips like a glove. You might feel tingling or burning because damaged nerves misfire. You may lose balance because your brain no longer knows where your feet are. Muscles can weaken and shrink because the motor wires that command them are also dying. The goal is to find and stop whatever is poisoning or attacking the nerve, control pain and protect numb feet from injury, and give the nerve the best chance to heal — which can be slow, because nerves regrow only about one millimeter per day.

What you might feel

- Tingling or burning in the hands and feet
- Numbness starting in the toes and fingers and slowly moving up
- Weakness in the feet or hands, such as trouble lifting the toes or gripping
- Sharp, shooting, or electric-shock pains
- Loss of balance or feeling unsteady when walking
- Extreme sensitivity to light touch (allodynia)
- Sudden weakness in one limb or on one side of the body

- Rapid worsening over days to weeks
- Trouble breathing, swallowing, or speaking

Call the doctor right away if...

- 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

What the care team will do

- Assess respiratory effort, oxygen saturation, and ability to cough every four hours if patient has new or worsening weakness
- Inspect both feet daily for skin breaks, redness, blisters, or foreign objects; ensure patient or family demonstrates proper foot checks
- Implement fall precautions including non-skid footwear, bedside commode, call bell within reach, and physical therapy consult for gait training
- Administer duloxetine, gabapentin, or pregabalin as prescribed for neuropathic pain; assess pain using a numeric scale before and one hour after dosing
- Monitor blood glucose before meals and at bedtime if patient has diabetes; report sustained readings above 180 mg/dL or HbA1c above seven percent to provider
- Teach patient to test water temperature with elbow before bathing and to never use heating pads or hot water bottles on numb areas
- Coordinate with pharmacy to review all current medications for neurotoxic agents; question orders for metronidazole courses longer than fourteen days or new fluoroquinolones
- Ensure referral to physical and occupational therapy for ankle-foot orthotics fitting if patient has foot drop (inability to lift the front of the foot)

Medicines you may be given — and why

Gabapentin (Neurontin)

Gabapentin quiets pain signals traveling up damaged nerves by blocking voltage-gated calcium channels (gates on nerve endings that normally let calcium rush in and fire off pain messages). When these gates stay more closed, the nerve can't shout 'pain!' as loudly, even though the nerve itself is still damaged.

Pregabalin (Lyrica)

Pregabalin works almost identically to gabapentin—it binds to the same alpha-2-delta calcium-channel subunit on nerve terminals and blocks calcium from rushing in. This quiets the release of pain neurotransmitters. Pregabalin is absorbed more predictably and reaches steady levels faster, so it often works at lower doses.

Duloxetine (Cymbalta)

Duloxetine blocks the brain and spinal cord from sucking serotonin and norepinephrine (two chemical messengers) back into nerve cells after they're released. When these messengers stay in the gaps between nerves longer, they strengthen the body's natural pain-dampening pathways that run from the brainstem down the spinal cord, turning down the volume on pain signals climbing up from damaged peripheral nerves.

Amitriptyline (Elavil)

Amitriptyline blocks the reuptake of serotonin and norepinephrine (just like duloxetine, but less selectively), boosting the brain's descending pain-control pathways. It also blocks sodium channels on nerve membranes, which directly quiets overactive pain-signaling nerves. Finally, it has anticholinergic effects (blocks acetylcholine receptors), which contribute to side effects but may add a small pain-dampening effect.

Intravenous immunoglobulin (IVIG)

IVIG delivers a concentrated mixture of antibodies collected from thousands of healthy donors. These antibodies flood the immune system and shift it away from attacking the body's own myelin (the insulating coat around nerves). The exact mechanism is not fully understood, but IVIG blocks harmful autoantibodies, saturates Fc receptors on immune cells (so they can't latch onto myelin as easily), and releases anti-inflammatory signals that quiet the immune attack. Over weeks to months, this allows the myelin sheath to stop being stripped away and—in some cases—begin to repair.

Plasmapheresis (Plasma exchange, PLEX)

Plasmapheresis physically removes the liquid part of your blood (plasma), which contains the autoantibodies and inflammatory proteins attacking your nerves. The red and white blood cells are separated out, mixed with clean donor plasma or albumin (a replacement fluid), and pumped back into your body. Each session removes roughly 60–70% of circulating autoantibodies; after 5–6 sessions over 10–14 days, the level of harmful antibodies drops enough to stop or slow the immune assault on myelin, allowing the nerves to begin recovering.

Capsaicin 8% patch (Qutenza) or 0.025-0.075% cream

Capsaicin is the chemical that makes chili peppers hot. When applied to the skin, it binds to TRPV1 receptors (heat and pain sensors) on the tiny nerve endings in the skin. At first, this causes a burning sensation as the nerves fire intensely. But with repeated or high-dose exposure (as with the 8% patch), the nerve endings become exhausted—they release all their substance P (a pain chemical) and then go quiet for weeks to months. This creates a localized numbing effect, reducing burning, stabbing, or tingling pain in that one spot.

Lidocaine 5% patch (Lidoderm)

Lidocaine is a local anesthetic that blocks sodium channels on nerve membranes. When applied to the skin as a patch, it soaks into the top layers and prevents the small pain-sensing nerve endings (C-fibers and A-delta fibers) from firing. Sodium channels are the gates that let sodium rush into a nerve cell to create an electrical pain signal; when lidocaine locks these gates shut, the nerve can't send pain messages to the spinal cord. The effect is localized to the patch area and lasts as long as the patch is on (up to 12 hours per day).

Where this information comes from

- American Academy of Neurology (AAN) neuropathy guideline 2021
- ADA Standards of Care in Diabetes 2024
- Cochrane systematic reviews on neuropathy
- NEJM clinical practice reviews

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