

Chronic Venous and Arterial Ulcers

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THE STORY IN ONE SENTENCE

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“Leg sores from bad veins look wet and brown near the ankle, while sores from clogged arteries are dry, deep, and hurt even at rest—know which type you are treating because the fix is completely different.”

Normal anatomy — the body's job

Healthy leg circulation means arteries bring oxygen-rich blood down to feed your skin, veins with working one-way valves push old blood back up to your heart, and nerves let you feel pressure so you shift your weight and avoid injury—when all three jobs work, your skin stays pink, warm, and whole.

BODY'S JOB

Leg arteries deliver oxygen and nutrients to every skin cell. Leg veins collect the used blood and pump it back uphill against gravity using one-way valves that snap shut after each squeeze from your calf muscles (like a series of locks in a canal). Nerves sense pain and pressure, so you automatically move your foot before tissue gets crushed. Together these three systems keep your skin alive, dry, and protected.

1. Fresh blood flows DOWN through leg arteries, branching into thousands of tiny vessels that feed every patch of skin with oxygen and fuel.
2. After dropping off oxygen, the blood (now dark and low in oxygen) collects in tiny veins, then bigger veins, to travel back UP toward the heart.
3. Every time your calf muscle squeezes (walking, standing on tiptoes), it pushes a column of vein blood upward; one-way valves inside the vein snap shut to stop the blood from falling back down.

4. Nerves constantly send signals to your brain—'too much pressure here, this spot is getting sore'—so you shift your weight or change position without even thinking about it.
5. When all three jobs work, skin stays at the right pressure, gets enough oxygen, and avoids trauma, so it never breaks down into a sore.

What breaks

The upstream break is either a leaky valve in your leg vein (blood pools and pressure rises), a clog in your leg artery (oxygen delivery drops), or dead nerves that no longer warn you about pressure—each one causes a different pattern of skin damage.

CLINICAL TERM

Chronic venous insufficiency (CVI) from incompetent valves (post-thrombotic syndrome, primary valve failure, or deep vein reflux), peripheral artery disease (PAD) from atherosclerotic obstruction (usually superficial femoral or tibial arteries), or diabetic neuropathy with loss of protective sensation—each mechanism produces a distinct ulcer phenotype that guides treatment.

How the body compensates

The body tries to adapt—veins push harder, nearby vessels try to detour around a clog, thick skin forms to protect damaged areas—but these fixes only buy time and cannot reverse the upstream break, so the sore keeps growing unless you fix the root cause.

1. {'what': 'In venous disease, the body grows new tiny veins to try to drain the pooled blood, and skin thickens into a leathery shield (lipodermatosclerosis).', 'limit': 'These detour veins are too small to handle the load, and the thick skin is stiff and fragile—so swelling and ulcers still form.'}
2. {'what': 'In arterial disease, small side-branch arteries try to open wider (collateral circulation) to bring blood around the blockage.', 'limit': 'Collaterals grow slowly and may never deliver enough oxygen, especially when you walk or lie flat—so pain at rest and non-healing sores persist.'}
3. {'what': 'Skin grows calluses over pressure points to try to absorb repeated impact.', 'limit': 'Calluses actually concentrate pressure and hide the damage forming underneath—without pain signals, the tissue dies silently and infection tunnels deep before you notice.'}
4. {'what': 'Early on, the patient feels only mild aching (venous) or cramping when walking (arterial claudication), so they cut back activity and avoid noticing how bad the circulation really is.', 'limit': 'This hides the urgency—by the time a sore appears, the system has already failed for weeks or months, and the window to heal without surgery or major intervention is closing.'}

Domino effect

1. Vein valves break and leak backward (from old clot damage, vein wall stretching, or inherited weakness). — The one-way door inside the vein cannot close, so every time your calf squeezes or you stand up, blood falls back down instead of climbing toward the heart—pressure in the lower leg veins shoots up (normal is 10–15, now climbs to 60–90 mmHg when standing).
2. High vein pressure forces fluid out through vessel walls into the surrounding tissue (edema and swelling around the ankle). — Pressure inside the tiny veins (capillaries) is now higher than the pressure holding fluid inside the vessel, so water, proteins, and even red blood cells leak out—the ankle puffs up and feels heavy.
3. Swelling compresses tiny arteries and starves the skin of oxygen; red blood cells trapped in tissue break open and stain the skin rust-brown (hemosiderin deposition). — The swollen, water-logged tissue squeezes shut the small arteries trying to deliver oxygen—skin cells start to die from lack of fuel, and the iron from broken red cells deposits permanently, creating the brown 'staining' you see above the ankle.
4. Chronic low oxygen and inflammation turn the skin thick, hard, and shiny (lipodermatosclerosis); the lower leg looks like an upside-down bowling pin. — The body lays down scar tissue to try to protect the area, but this makes the skin stiff, fragile, and unable to stretch—minor trauma (a bump, a scratch, even tight socks) now breaks the skin because it has no cushion left.
5. Skin finally opens into a shallow, weeping, irregular sore just above the inner ankle bone (medial malleolus)—the classic venous ulcer. — That spot sits at the deepest point of swelling and the lowest oxygen delivery in a standing leg; the skin there is under the most pressure and has been oxygen-starved the longest, so it is the first to die and ulcerate.
6. The open sore leaks fluid, gets infected easily, and grows larger because the high vein pressure and swelling never stop—healing cannot happen while the valve is still broken. — New skin cells need oxygen and a dry, low-pressure environment to knit together; as long as fluid keeps leaking and pressure stays high, every attempt to heal is washed away—the ulcer becomes chronic (lasting months to years).
7. Without compression therapy to squeeze the fluid out and push blood back up, the cycle repeats—swelling, staining, ulcer, infection—and the risk of losing independent mobility or developing sepsis (blood infection) climbs every month the sore stays open. — The only way to break the loop is to fix the broken valve (surgery or ablation) or externally compress the leg (wrap or stocking) to force the pooled blood out and restore normal pressure—medication and wound cream alone will never close a venous ulcer if the upstream leak is still pouring fluid in.

8. In arterial ulcers, the domino is different: a cholesterol plaque narrows or plugs the artery, blood flow drops, oxygen delivery falls, and any injury (even trimming a toenail) becomes a deep, punched-out sore that hurts terribly and will not heal until you reopen the artery with surgery or a stent. — Skin cells cannot survive or repair without oxygen—when arterial flow is cut by 50–70%, there is barely enough oxygen for rest but zero reserve for healing; the sore keeps digging deeper (into tendon, even bone) because the tissue is literally starving to death.

Symptoms · what the patient feels, and why

- Open sore on the lower leg or foot that won't heal after several weeks: When an artery is clogged (arterial ulcer), the tissue downstream doesn't get enough oxygen and nutrients to repair itself. When veins are weak and blood pools (venous ulcer), the pressure builds and damages the skin. In both cases, even a tiny scratch can't close up and becomes a lasting wound.
- Brown or rust-colored staining of the skin around the ankle (hemosiderin staining): In venous ulcers, blood backs up in the leg veins and red blood cells leak out into the tissue. When those cells break down, they leave behind iron that stains the skin like rust, especially around the ankle and lower shin.
- Swelling of the lower leg or ankle (edema): When the valves inside leg veins stop working, blood flows backward and pools in the lower leg instead of returning to the heart. The pressure forces fluid out of the blood vessels into the surrounding tissue, making the leg and ankle puff up.
- Pain that gets worse when the leg hangs down and better when it is raised (venous ulcer pattern): Gravity pulls more blood into already-swollen veins when the leg is down, cranking up the pressure and the ache. Propping the leg up helps blood drain back toward the heart, lowering the pressure and easing the pain.
- Pain that gets worse when the leg is elevated and better when it hangs down (arterial ulcer pattern): In arterial disease, the arteries are partly blocked so the heart struggles to push blood uphill. When the leg hangs down, gravity helps a trickle of blood reach the starving tissue and the pain eases a bit. Raising the leg cuts off that trickle and the pain spikes.
- Pale, cool skin with a shiny, tight look and loss of hair on the lower leg (arterial ulcer): When arteries are clogged, not enough oxygen-rich blood reaches the skin and hair follicles. Hair stops growing and falls out, sweat glands shrink, and the skin becomes thin, shiny, and waxy because the tissue is slowly starving.

- Thick, hard, leathery skin around the ankle that looks like an upside-down bowling pin (lipodermatosclerosis in venous disease): Chronic swelling and inflammation from backed-up blood cause the fat and connective tissue under the skin to scar and harden over months or years. The lower leg shrinks and tightens while the upper calf stays swollen, creating a bottle shape.
- Drainage or oozing from the ulcer, sometimes with a bad smell: Open wounds often leak clear or yellowish fluid as the tissue tries to heal. If bacteria invade the wound, they feed on dead tissue and produce pus and a foul odor. Heavy drainage or a sudden bad smell can mean infection is setting in.
- Very pale or bluish toes, or black, dead tissue at the ulcer edges (necrosis): When an artery is severely blocked or completely shut, tissue downstream gets zero oxygen and the cells die. Dead tissue turns black and leathery, like severe frostbite. This dead material must be removed or it can trigger life-threatening infection.
- Weak or absent pulse in the foot or behind the ankle: In arterial ulcers, plaque (a mix of fat, cholesterol, and calcium) narrows or blocks the arteries that supply the leg. Less blood flows through, so the pulse downstream becomes faint or disappears entirely—a key sign that the arteries are clogged.

Focused exam

- Brown or rust-colored stain (hemosiderin) on the skin above the ankle bone: Old blood leaked from damaged veins and settled in the skin. The brown pigment means vein valves have been failing for a long time and blood has been pooling backward.
- Swelling (edema) in the lower leg that leaves a dent when you press it (pitting swelling (edema)): Fluid is trapped in the tissues because broken vein valves cannot push it back to the heart. This swelling stretches the skin and makes it harder for wounds to heal.
- Open sore (ulcer) above the ankle bone, usually wet and shallow with irregular borders: The skin broke down because swelling and poor blood flow starved it of oxygen. The wet, weeping appearance is typical of venous ulcers.
- Ankle-brachial index (ABI) measured with a blood pressure cuff and Doppler probe: ABI < 0.9 means the arteries are blocked. You must know this before using tight compression bandages, which can harm a leg with blocked arteries.
- Shiny, hairless skin on the foot and toes, with pale or dusky color: Blocked arteries starve the skin of blood, so hair follicles die and the skin becomes thin and shiny. This points to an arterial ulcer or mixed ulcer.
- Pain at rest or pain that gets worse when the leg is raised (elevation worsens pain): Rest pain means the arteries are so blocked that even lying still does not bring enough oxygen to the tissues. This is a sign of critical limb starved blood flow (ischemia) (severely blocked arteries).

Diagnostics · every test answers a question

- Ankle-brachial index (ABI) → Are the arteries blocked, making this an arterial or mixed ulcer instead of a pure venous ulcer?
- Venous duplex ultrasound (vein sound-wave picture) → Are the vein valves broken, and is blood flowing backward (reflux)?
- Arterial duplex ultrasound or CT/MR angiography → If ABI is low, where exactly is the artery blocked, and can it be opened?
- Wound culture (if the ulcer smells bad, has pus, or the skin around it is hot and red) → Is there infection in the wound, and which bacteria are growing?
- X-ray of the foot or leg (if the ulcer is deep or you can see or feel bone) → Is the bone infected (osteomyelitis)?
- Hemoglobin A1c (HbA1c) → Is high blood sugar slowing healing and feeding infection?

Treatment logic

- Ankle-brachial index (ABI) test before starting treatment → Splits venous ulcers (normal ABI) from arterial or mixed ulcers (low ABI). This one test decides whether tight compression is safe or dangerous.
- Compression therapy (multilayer bandages or compression stockings, 30–40 mmHg) → Squeezes the leg to push pooled fluid back into the veins and toward the heart. Reduces swelling, speeds healing of venous ulcers, and prevents new ulcers. Only safe if $ABI \geq 0.8$.
- Leg elevation (raise leg above heart level for 30 minutes, 3–4 times per day) → Gravity drains fluid out of the leg, shrinking swelling and improving oxygen delivery to the wound. Most helpful for venous ulcers; less helpful for arterial ulcers.
- Wound debridement (removal of dead tissue) and moisture-retaining dressings (hydrocolloid, foam, or alginate) → Removes dead tissue and bacteria so healthy new skin can grow. Keeps the wound moist (not dry) to speed healing. Stops infection from tunneling into bone.
- Revascularization (angioplasty, stenting, or bypass surgery for arterial ulcers with $ABI < 0.9$) → Opens blocked arteries so oxygen-rich blood reaches the foot again. Pale skin turns pink, rest pain disappears, and deep ulcers start healing.
- Smoking cessation and blood sugar control (target HbA1c $< 7\%$) → Stopping smoking widens arteries and improves oxygen delivery. Lowering blood sugar protects nerves, strengthens white blood cells, and speeds healing.
- Systemic antibiotics (only if there is spreading infection, fever, or bone involvement) → Kills bacteria spreading into surrounding tissue or bone. Stops infection from becoming life-threatening or causing limb loss.

Medications

Pentoxifylline · Hemorrheologic agent (blood-flow improver)

Makes red blood cells softer and bendier so they can squeeze through narrow, stiff, or partly blocked vessels more easily. Think of turning hard marbles into soft gummy bears—the flexible cells can now slip through tight spaces and deliver oxygen to starving leg tissue even when the pipes are damaged.

1. You swallow the pill and it enters your bloodstream.
2. Pentoxifylline changes the outer shell of red blood cells, making them less stiff.
3. The softer red cells can now bend and squeeze through narrow or partly clogged vessels.
4. More oxygen reaches the skin and muscle that were starving before.
5. Better oxygen delivery helps ulcers heal and cuts down pain when you walk (claudication).

SAFETY WATCH

Can cause upset stomach, nausea, or dizziness—take with food to help. · May increase bleeding risk slightly; tell your doctor if you take blood thinners like warfarin, apixaban, or aspirin. · Do NOT use if you recently had bleeding in the eye (retinal bleeding (hemorrhage)) or brain. · Tell your doctor if you have severe kidney or liver disease—the dose may need to be lower. · Call your doctor if you develop chest pain, irregular heartbeat, or signs of bleeding (black stools, easy bruising).

Aspirin (low-dose, 81 mg daily) · Antiplatelet agent (clot preventer)

Stops tiny blood-clotting pieces called platelets from sticking together inside arteries. Platelets normally clump to stop bleeding, but in damaged arteries they can build dangerous clots that block oxygen flow. Aspirin keeps them slippery so blood keeps moving smoothly.

1. You swallow the aspirin tablet.
2. Aspirin travels to your platelets and permanently shuts off an enzyme called cyclooxygenase-1 (COX-1).
3. Without working COX-1, platelets cannot make a sticky chemical called thromboxane A2.
4. Platelets stay smooth and slippery instead of clumping together.
5. Blood flows more easily through narrowed or rough arteries, lowering the chance of a new clot forming and blocking oxygen to your leg, heart, or brain.

SAFETY WATCH

Can cause stomach upset, heartburn, or bleeding (stomach ulcers, nosebleeds, blood in stool)—take with food and call your doctor if you see black, tarry, or bloody stools. · Do NOT take if you have active bleeding, a known allergy to aspirin, or severe liver disease. · Tell your doctor before any surgery or dental work—you may need to stop aspirin 5–7 days before to reduce bleeding risk. · Avoid other NSAIDs (ibuprofen, naproxen) at the same time unless your doctor approves—stacking them raises bleeding and kidney injury risk. · Rarely, aspirin can trigger severe allergic reactions (hives, wheezing, swelling)—call 911 if this happens.

Cilostazol · Phosphodiesterase-3 (PDE3) inhibitor (vasodilator and antiplatelet)

Relaxes the muscle layer inside artery walls so vessels open wider, and at the same time keeps platelets from sticking. Imagine loosening a tight belt around a garden hose—more blood flows through, oxygen reaches starving leg muscles and skin, and pain when you walk drops.

1. You swallow the pill twice a day, 30 minutes before or 2 hours after meals (food reduces absorption).
2. Cilostazol enters smooth muscle cells lining your arteries.
3. It blocks an enzyme called phosphodiesterase-3 (PDE3) that normally breaks down a chemical messenger named cyclic AMP (cAMP).
4. With PDE3 blocked, cAMP builds up inside muscle cells, telling them to relax.
5. Arteries widen, blood flow increases, and oxygen delivery improves to legs and skin.
6. At the same time, higher cAMP in platelets keeps them from clumping, so new clots are less likely.

SAFETY WATCH

ABSOLUTE CONTRAINDICATION: Do NOT use if you have heart failure of ANY severity (even mild)—cilostazol can make heart failure worse, increase dangerous heart rhythms, and raise the risk of death. · Common side effects include headache (often improves after a few weeks), diarrhea, and palpitations (feeling your heart race or skip). · May increase bleeding risk—tell your doctor if you take aspirin, clopidogrel, warfarin, or other blood thinners. · Tell your doctor if you have kidney or liver disease—the dose may need adjustment or the drug may not be safe. · Call your doctor if you develop chest pain, shortness of breath, swelling in your ankles, or signs of bleeding.

Clopidogrel · Antiplatelet agent (P2Y12 inhibitor)

Blocks a different clotting pathway than aspirin, keeping platelets from sticking when they get an activation signal called ADP (adenosine diphosphate, a chemical released by damaged vessel walls). Think of it as cutting a second wire in the clotting alarm system—platelets stay calm and slippery, blood flows smoothly, and the chance of a clot forming in a narrowed artery drops.

1. You swallow one 75 mg tablet each day.
2. Clopidogrel is converted in your liver into its active form.
3. The active drug permanently blocks a receptor on platelets called P2Y12.
4. When a platelet meets ADP (a chemical signal released by injured vessel walls), it cannot respond because the receptor is shut off.
5. Platelets stay smooth and do not clump, so blood keeps moving and oxygen reaches tissue.
6. The effect lasts the lifetime of each platelet (about 7–10 days).

SAFETY WATCH

Can cause bleeding (nosebleeds, bruising, blood in urine or stool)—call your doctor if bleeding is heavy or does not stop. · Do NOT stop suddenly without talking to your doctor—if you have a stent or recent heart attack or stroke, stopping can cause a life-threatening clot. · Tell your doctor before any surgery or dental work—you may need to stop clopidogrel 5–7 days before to reduce bleeding risk. · Rare but serious: thrombotic thrombocytopenic purple bleeding blotches (purpura) (TTP), a blood disorder causing fever, confusion, and purple spots on skin—seek emergency care if these appear. · Some people have genes that make clopidogrel less effective (reduced metabolism)—genetic testing can check this if your doctor suspects it. · Avoid proton-pump inhibitors like omeprazole (used for heartburn) if possible—they can reduce clopidogrel's effect; talk to your doctor about alternatives like famotidine.

Atorvastatin or other statin (e.g., simvastatin, rosuvastatin) · HMG-CoA reductase inhibitor (statin—cholesterol-lowering drug)

Blocks the liver enzyme that makes cholesterol, forcing the liver to pull LDL (bad cholesterol) out of the blood. Lower LDL slows plaque buildup in arteries, stabilizes existing plaques so they are less likely to rupture and cause clots, and slightly improves the lining of blood vessels so they work better.

1. You swallow one tablet each evening (cholesterol production peaks at night).
2. Atorvastatin enters liver cells and blocks the enzyme HMG-CoA reductase, which is the first step in making cholesterol.

3. The liver cannot make as much cholesterol, so it pulls LDL cholesterol particles out of your bloodstream to meet its needs.
4. LDL cholesterol in your blood drops, slowing new plaque formation in artery walls.
5. Existing plaques become more stable (less likely to crack open and trigger a clot).
6. The inner lining of arteries (endothelium, the smooth layer that touches the blood) works better, slightly improving blood flow and reducing inflammation.

SAFETY WATCH

Can cause muscle aches or weakness—tell your doctor if this happens; rarely, severe muscle breakdown (rhabdomyolysis) can damage kidneys. · Your doctor will check liver enzymes and sometimes muscle enzymes (CK) with blood tests before starting and during treatment. · Do NOT take if you are pregnant or breastfeeding—statins can harm a developing baby. · Avoid grapefruit juice—it raises statin levels in your blood and increases the risk of side effects. · Rare side effects include memory problems, high blood sugar (slightly increased diabetes risk), and liver enzyme elevation. · Call your doctor if you develop dark urine, severe muscle pain, yellowing of skin or eyes, or unexplained fatigue.

Compression therapy (multilayer bandages or graduated compression stockings) · Mechanical treatment (external pressure device)

Squeezes the leg from the outside to push pooled blood and leaked fluid up toward the heart, overcoming broken valves in leg veins that let blood fall backward and collect in the ankle. Think of wringing out a soaked sponge—the steady squeeze moves stuck fluid, reduces swelling, and helps oxygen-rich blood reach the skin so venous ulcers can heal.

1. A nurse or trained caregiver wraps your leg snugly with special elastic bandages (e.g., Unna boot, four-layer bandage) or you put on graduated compression stockings (tightest at the ankle, looser higher up).
2. The external pressure squeezes veins and tissue, forcing pooled blood and fluid upward toward the heart.
3. Broken valves cannot stop the upward flow when external pressure is applied.
4. Swelling goes down, pressure inside the veins drops, and less fluid leaks into the skin.
5. Better circulation and lower tissue pressure allow oxygen and nutrients to reach the ulcer, and waste products are carried away, speeding healing.

SAFETY WATCH

MANDATORY: Measure the ankle-brachial index (ABI) BEFORE applying compression. If ABI is below 0.5 (severe peripheral artery disease), do NOT use compression—it can worsen starved blood flow (ischemia) (lack of oxygen) and cause tissue death. If ABI is 0.5–0.8, use reduced (modified) compression (15–25 mmHg) only under doctor supervision. · Compression should feel snug but NEVER painful. If toes turn blue, cold, numb, or you feel severe pain, remove the bandage immediately and call your doctor. · Wash skin gently and reapply bandages daily or as directed; check for new sores, blisters, or skin breakdown. · Do NOT use on legs with active soft-tissue infection (cellulitis) or untreated a clot in a deep leg vein (deep vein thrombosis) (DVT) until cleared by your doctor. · Elevate your legs above heart level when resting to help fluid drain. · Once the ulcer heals, you will need to wear compression stockings long-term (usually 30–40 mmHg) every day to prevent recurrence—venous insufficiency does not go away.

Topical antibiotics (mupirocin ointment, silver sulfadiazine cream, or other antimicrobial dressings) · Topical antimicrobial agents (germ fighters applied to the wound)

Kill or stop bacteria from multiplying directly on the wound surface, preventing infection from taking hold and interfering with healing. Think of spraying disinfectant on a counter—the medicine stays on top of the ulcer and attacks germs before they can invade deeper tissue or enter the bloodstream.

1. Clean the ulcer gently with saline or clean water to remove loose debris and old ointment.
2. Apply a thin layer of antibiotic ointment or cream (e.g., mupirocin, silver sulfadiazine) directly onto the wound, or apply an antimicrobial dressing (e.g., silver foam, iodine dressing).
3. The active ingredient enters bacterial cells on the wound surface.
4. Mupirocin stops bacteria from making proteins they need to survive; silver damages bacterial cell walls and DNA; iodine disrupts bacterial metabolism (the chemical reactions they need to live).
5. Bacteria die or stop multiplying, keeping the wound cleaner so your body's repair cells (fibroblasts that make collagen, keratinocytes that rebuild skin) can work without interference.

SAFETY WATCH

Stop and call your doctor if you see spreading redness, warmth, swelling, pus, red streaks going up the leg, fever, or worsening pain—these signs mean infection is spreading beyond the surface and topical treatment is not enough. · Some people develop allergy or irritation (itching, rash, burning)—switch products if this happens. · Do NOT use silver sulfadiazine if you are allergic to sulfa drugs or if you are pregnant near delivery (can cause yellowing of the skin and eyes (jaundice) in the newborn). · Use only as long as your doctor recommends—prolonged use can select for resistant bacteria, cause toxicity (silver can rarely build up and turn skin gray—argyria), or delay healing by irritating healthy tissue. · Most venous and arterial ulcers are NOT infected and do NOT need routine topical antibiotics—use them only for signs of infection or heavy bacterial burden (critical colonization).

Oral or IV antibiotics (e.g., cephalexin, doxycycline, clindamycin, vancomycin—choice depends on culture and severity) · Systemic antimicrobial agents (infection fighters taken by mouth or vein)

Travel through your bloodstream to reach bacteria that have invaded tissue around or under the ulcer, killing or stopping them from multiplying. Unlike creams that stay on the surface, pills or IV antibiotics attack germs deep in swollen tissue, bone, or blood, preventing infection from spreading to distant organs and saving limbs and lives.

1. You swallow the antibiotic pill (or it is given through an IV line if infection is severe, you cannot swallow, or the germ is resistant).
2. The drug enters your bloodstream and travels to the infected area.
3. It enters bacterial cells or disrupts their outer wall.
4. Different antibiotics work in different ways: beta-lactams (cephalexin, amoxicillin-clavulanate) punch holes in bacterial cell walls; tetracyclines (doxycycline) stop bacteria from making proteins; clindamycin and vancomycin also block protein or cell-wall building.
5. Bacteria die or stop growing, and your immune system (white blood cells) cleans up the debris, reducing redness, warmth, swelling, and pus.

SAFETY WATCH

Take the FULL course exactly as prescribed (usually 1–2 weeks for soft tissue, 4–6 weeks or longer for bone infection)—stopping early lets resistant bacteria survive and come back stronger. · Watch for allergic reactions: rash, hives, trouble breathing, swelling of face or throat—call 911 if severe (anaphylaxis). · Common side effects include upset stomach, nausea, diarrhea, and yeast infections (oral thrush, vaginal yeast). · Tell your doctor immediately if diarrhea becomes severe, watery, or bloody—antibiotics can cause a dangerous colon infection called *Clostridioides difficile* (C. diff). · If the ulcer gets worse, fever persists after 48–72 hours on antibiotics, or you develop confusion, low blood pressure, or rapid heart rate, return to the emergency department—you may need IV antibiotics, imaging (X-ray, MRI) to check for bone infection or abscess (pus pocket), surgical drainage, or intensive care. · Blood tests (CBC, CRP, ESR) and wound cultures guide antibiotic choice and duration—always follow up as directed.

Wound debridement (sharp, enzymatic, autolytic, or biosurgical) · Mechanical, enzymatic, or biological wound preparation

Removes dead, infected, or damaged tissue and thick scab (eschar, slough, biofilm) from the ulcer so healthy tissue underneath can breathe, grow, and receive oxygen. Think of clearing weeds and dead leaves from a garden bed—once the debris is gone, new skin cells can sprout and the wound shrinks faster.

1. A doctor, nurse, or wound specialist uses one of several methods: sharp debridement (scalpel, scissors, or curette to cut away dead tissue), enzymatic debridement (ointment with enzymes like collagenase to dissolve dead tissue), autolytic debridement (moist dressings that let your body's own enzymes digest debris), or biosurgical debridement (medical-grade maggots that eat only dead tissue).
2. Dead tissue and biofilm are removed, exposing the clean wound bed underneath.
3. Blood vessels can now reach the surface, delivering oxygen, nutrients, and immune cells.
4. Your body's repair cells (fibroblasts make collagen, keratinocytes rebuild skin) move in and start building new tissue.
5. Bacteria and toxins trapped in dead tissue are cleared, lowering infection risk and allowing healing to proceed.

SAFETY WATCH

Sharp debridement can cause bleeding—light bleeding is normal and often healthy (shows blood supply), but tell your doctor if bleeding is heavy or does not stop with gentle pressure. · Do NOT perform aggressive sharp debridement if blood flow is severely impaired (ABI less than 0.5, toe pressure less than 30 mmHg, or visible dry gangrene) until revascularization improves flow—cutting tissue without oxygen can enlarge the wound or cause infection. · Debridement may hurt—ask for local anesthetic (lidocaine gel, injection) or pain medicine before the procedure if needed. · Keep the wound clean and moist with appropriate dressings after debridement to support healing and prevent new debris buildup. · Repeat debridement may be needed weekly or as often as dead tissue returns—this is normal for chronic ulcers. · If you use enzymatic debridement (collagenase ointment), avoid combining it with silver or iodine dressings in the same application—they can inactivate the enzyme.

Hydrocolloid, foam, alginate, or hydrogel dressings (advanced wound dressings) · Moisture-balancing wound dressings (not drugs, but essential tools)

Keep the wound bed moist but not soaked, absorb excess fluid (exudate), protect new tissue from drying out or getting injured, and create an environment where your body's repair cells can work best. Think of it like putting a greenhouse dome over a seedling—the right moisture and protection help growth without letting things rot or dry up.

1. After cleaning and (if needed) debriding the ulcer, you or a nurse apply the appropriate dressing based on how much fluid the wound is making.
2. Hydrocolloid dressings (e.g., DuoDERM) absorb light to moderate fluid and form a gel that keeps the wound moist—good for dry to lightly draining wounds.
3. Foam dressings (e.g., Mepilex) absorb moderate to heavy fluid while cushioning the wound—good for pressure relief and heavy drainage.
4. Alginate dressings (made from seaweed fibers) absorb very heavy fluid and can pack deep wounds—they turn into a gel when wet.
5. Hydrogel dressings donate moisture to dry wounds and help soften hard eschar (thick black or brown scab) for autolytic debridement.
6. The dressing maintains a moist environment that allows enzymes, growth factors, and repair cells to work efficiently, speeds re-epithelialization (new skin growth), and reduces pain.

SAFETY WATCH

Change dressings as often as directed (daily to weekly depending on type and drainage)—leaving a saturated dressing on too long can macerate (soften and damage) healthy skin around the wound. · Check the skin around the wound (peri-wound skin) for redness, irritation, or breakdown—some people are sensitive to adhesives. · Do NOT use occlusive (airtight) dressings like hydrocolloid on infected wounds unless infection is controlled—trapping bacteria can worsen infection. · If the wound suddenly produces more fluid, smells bad, or the dressing sticks and is hard to remove, call your doctor—these can be signs of infection. · Advanced dressings can be expensive—check with your insurance and ask your doctor or wound clinic about samples or assistance programs.

Red flags

- Sudden increase in pain, redness, warmth, or pus around the ulcer (suggests infection spreading into deeper tissue or the bloodstream)
- Black or gray dead tissue, or foul-smelling drainage (suggests gangrene or severe infection that needs urgent care to prevent limb loss)
- New numbness, coldness, or complete loss of pulse in the foot (suggests a blood clot or sudden artery blockage that can lead to limb loss within hours)
- Fever or chills along with the ulcer (suggests the infection has spread into the blood—a life-threatening emergency called sepsis)

Nursing priorities

1. Check pedal pulses (dorsalis pedis and posterior tibial) and capillary refill in both feet every shift, and report absent pulses or refill longer than 3 seconds immediately — Arterial ulcers need oxygen-rich blood to heal; a missing pulse means the artery is blocked and tissue will die (necrosis) without emergency revascularization within hours
2. Measure ankle-brachial index (ABI) before applying any compression bandages, and never compress if ABI is below 0.8 — Compression squeezes veins to push blood up but can choke off an already weak artery; ABI below 0.8 means peripheral artery disease, and compression will cause gangrene
3. Elevate the leg above heart level for 30 minutes every 2 to 3 hours during the day for venous ulcers, but keep arterial ulcer legs dependent (hanging down) — Gravity helps drain pooled blood and reduce swelling (edema) (swelling) in venous disease, but arterial ulcers need gravity to pull blood down into starving tissue

4. Assess ulcer size, depth, drainage color and smell, and surrounding skin for warmth, spreading redness, or a crackling feel or sound (crepitus) (crackling under skin) every shift — Increasing size, pus-filled (purulent) (pus-filled) or foul drainage, and a crackling feel or sound (crepitus) signal necrotizing fasciitis (flesh-eating infection) or gas gangrene that spreads in hours and requires emergency surgery
5. Apply moisture-retaining dressings (hydrocolloid, foam, or alginate) and change per protocol, keeping venous ulcers moist but arterial ulcers dry until blood flow improves — Moist wound beds speed epithelialization (new skin growth) in venous ulcers, but moisture on blood-starved (ischemic) (low oxygen) tissue breeds infection before revascularization restores blood flow
6. Apply graduated compression stockings (30 to 40 mmHg) or multilayer bandages for confirmed venous ulcers after ABI is 0.8 or higher, and teach proper donning technique — Compression pushes blood out of leaky veins and reduces venous high blood pressure (hypertension) (high pressure) that caused the ulcer; wrong pressure or technique causes skin breakdown or tourniquet effect
7. Monitor blood glucose every 6 hours in diabetic patients and keep levels between 140 and 180 mg/dL during wound healing — High blood sugar (Hyperglycemia) impairs neutrophil function (white blood cells that fight infection) and slows fibroblast activity (cells that build new tissue), doubling infection risk and healing time
8. Teach patient to inspect both feet daily with a mirror, wear cushioned shoes, and never walk barefoot, even at home — Neuropathy (nerve damage) hides pain from new ulcers or foreign objects; early detection and offloading (removing pressure) prevents small wounds from becoming limb-threatening infections

Sources

- Society for Vascular Surgery (SVS) 2014 Lower Extremity Venous Ulcer Guideline
- American Heart Association / American College of Cardiology (AHA/ACC) Peripheral Artery Disease Guideline
- Wound Healing Society
- Cochrane Reviews on Chronic Wound Management

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