

Skin damage

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THE STORY IN ONE SENTENCE

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“Pressure injury — prevent it by relieving pressure every two hours; describe the stage, cut away dead tissue, and treat any infection.”

Normal anatomy — the body's job

Skin stays healthy when pressure is relieved regularly, so blood can flow through the tiny vessels and deliver oxygen and food to every layer.

BODY'S JOB

The skin is a barrier that protects the body from germs and injury. Underneath the skin sit tiny blood vessels (capillaries) that bring oxygen and nutrients to skin cells so they can stay alive and repair themselves. When you sit or lie down, your body weight presses the skin against bone — like your tailbone pressing into a chair. Healthy people shift their weight without thinking (crossing legs, rolling over in sleep) because even a few minutes of pressure starts to feel uncomfortable. These small movements let blood flow back into the pressed spot, so the skin gets fresh oxygen and stays strong.

1. The outer skin (epidermis) is a tough layer of flat cells that block water and germs.
2. Just below that, the dermis holds blood vessels, nerve endings, sweat glands, and strong fibers (collagen) that give skin its stretch and toughness.
3. Tiny capillaries in the dermis are only one cell thick — they are delicate and easy to squash.
4. When you press skin against bone, the capillaries flatten and blood cannot squeeze through, like stepping on a garden hose and stopping the water.
5. As soon as you lift the pressure, blood rushes back in (that is why a pressed spot turns white, then pink again).

6. Fat and muscle under the skin act as cushions to spread out pressure, but over bony spots (heels, tailbone, hips, elbows) there is less cushion, so pressure goes straight to the capillaries.

What breaks

When someone cannot move, cannot feel pain, has wet skin, or eats poorly, pressure squashes the blood vessels under the skin and oxygen stops reaching that spot, so the skin starts to die.

CLINICAL TERM

Immobility, sensory loss, moisture, and malnutrition cause sustained external pressure over bony prominences that exceeds capillary blood flow (perfusion) pressure (normally 20–32 mm Hg), leading to starved blood flow (ischemia) (not enough blood reaching the tissue to feed it) and tissue death (necrosis) (death of skin and deeper layers).

How the body compensates

The body tries to save the pressed skin by sending extra blood to the area (causing redness), but if pressure stays on, that effort fails and tissue dies. Vital signs may look normal even as a pressure injury worsens, so you must inspect the skin directly and watch for progression.

1. At first, capillaries dilate (widen) nearby to push more blood toward the pressed spot — this is why you see redness (reactive hyperemia) when pressure is briefly relieved.
2. If pressure returns quickly, the red area does not turn white when you press it (non-blanchable redness (erythema)) — that means capillaries are congested with trapped blood and still not flowing well.
3. The body tries to clear away damaged cells by sending white blood cells (inflammation), which causes swelling, warmth, and more redness, but this cannot work if pressure keeps blocking blood flow.
4. Eventually, with no oxygen, cells switch to anaerobic metabolism (making energy without oxygen), which produces lactic acid and toxic byproducts that poison nearby cells.
5. Once cells die, the body walls off the dead tissue (eschar or slough) to protect deeper layers, but this dead material becomes a barrier to healing and a home for bacteria.
6. Vital signs (heart rate, blood pressure) stay stable during early stages because the injury is local; the systemic crash (sepsis, osteomyelitis) comes later if bacteria invade through the open wound.
7. The key danger is that a stable-looking patient can have a worsening pressure injury hidden under clothing or bedding, so regular skin checks (every shift) are the only way to catch it early.

8. Once stage 3 or 4 is reached, healing slows dramatically because the body must rebuild all layers from the bottom up — this can take months and often requires surgery.

Domino effect

1. Pressure on a bony spot squashes capillaries and blocks blood flow to the skin above it. — Body weight pressing skin against bone (tailbone, heels, hips) creates force that flattens the tiny blood vessels, like pinching a straw shut — blood cannot get through, so oxygen and nutrients stop reaching skin cells.
2. Within 2-6 hours, oxygen-starved cells start to die (ischemia), and the skin turns red as nearby vessels dilate to try to rescue the area. — Cells need a constant oxygen supply; when it is cut off, they begin to fail. The body responds by opening nearby blood vessels wide to push more blood toward the danger zone, which causes the red color.
3. Stage 1: The red spot does not turn white when you press on it (non-blanchable redness (erythema)) — this is the earliest warning sign. — Normally, pressing skin pushes blood out of capillaries and the spot turns white briefly, then pink again. When the red does not blanch, it means capillaries are jammed with stagnant blood and still not flowing — damage has already started.
4. Stage 2: The outer layer of skin peels or blisters away, creating a shallow open sore. — With continued pressure, the epidermis (top skin layer) dies and separates from the dermis below, forming a blister or raw patch — like a friction burn, but caused by crushed blood vessels instead of rubbing.
5. Stage 3: The sore goes through all layers of skin, exposing the fat layer underneath; edges may be rolled and thick dead tissue (slough) fills the crater. — Once the dermis dies, the full thickness of skin is gone. Fat has even less blood supply than skin, so it dies quickly when exposed, turning yellow or gray (slough). The wound edges thicken as the body tries to wall off the dead zone.
6. Stage 4: Muscle, tendon, or bone becomes visible at the bottom of the wound. — The same pressure and blocked blood flow that killed skin now kill the deeper soft tissue (muscle, ligament). Bone may show through because all the cushioning layers above it are dead and gone.
7. Bacteria invade the dead tissue and open wound, leading to local infection, and if they reach bone, osteomyelitis (bone infection) develops. — Dead tissue has no blood flow, so the immune system cannot reach it — bacteria thrive there. If bacteria tunnel down to bone (especially in stage 4 ulcers), they cause a deep, hard-to-treat bone infection that may require months of IV antibiotics or surgery.

8. If infection spreads into the bloodstream, sepsis occurs — a life-threatening systemic response with fever, low blood pressure, and organ failure. — Once bacteria cross from the wound into blood vessels, the immune system floods the body with inflammatory signals (cytokines) to fight the infection, but this can overshoot and damage organs (kidneys, lungs, heart), leading to shock and death if not treated immediately.

Symptoms · what the patient feels, and why

- Red or purple patches that do not turn white when you press them (stage 1 pressure injury): Constant pressure squeezes the tiny blood vessels shut so oxygen cannot reach the skin. The skin gets inflamed and changes color, but it is still intact with no break or opening yet.
- Shallow open sore or blister with pink or red base (stage 2 pressure injury): Prolonged pressure kills the top layers of skin (the epidermis and part of the dermis), causing them to peel away and leave an open wound that looks like a scrape or burst blister.
- Deep crater or hole in the skin showing yellow fat underneath (stage 3 pressure injury): Pressure continues long enough that the full thickness of skin dies and sloughs off, exposing the fat layer below the skin and creating a deep wound.
- Very deep wound exposing muscle, tendon, or bone (stage 4 pressure injury): Unrelieved pressure destroys skin, fat, and deeper tissues all the way down to muscle or bone, often with dead black tissue (eschar) or yellow-tan slough covering parts of the wound.
- Dark purple or maroon skin, or blood-filled blister (deep tissue injury): Pressure and shear forces damage the muscle and fat layers deep under the skin before the surface breaks, trapping blood and causing a bruised appearance that can quickly open into a deep wound.
- Wound covered by thick yellow, tan, or black dead tissue so you cannot see the bottom (unstageable pressure injury): Dead tissue (slough or eschar) hides the wound bed, making it impossible to measure depth until the dead tissue is removed by the body or by a clinician.
- Pain, tenderness, or warmth over a bony area: Damaged tissue releases inflammatory chemicals (like bradykinin and prostaglandins) that activate pain nerves; warmth comes from increased blood flow as the body tries to repair the injury.
- Skin feels softer, firmer, or warmer than surrounding tissue: Early pressure damage disrupts the normal structure of skin and triggers local inflammation, changing the texture and temperature before a visible wound appears.

Focused exam

- Non-blanching redness (erythema) (red skin that stays red when you press it) over bony spots like the sacrum (tailbone), heels, elbows, or hips: This is Stage 1 pressure injury — the earliest sign that pressure has squeezed blood away from the skin. The skin cells are hungry for oxygen and starting to die. Catching it here can stop deeper damage.
- Partial-thickness skin loss with exposed dermis (the pink layer under the top skin), or an intact or ruptured blister filled with clear or blood-tinged fluid: This is Stage 2 pressure injury — the top layer of skin has broken down because blood could not feed it. The body cannot heal this without pressure relief and good nutrition.
- Full-thickness skin loss showing fat underneath, sometimes with yellow or gray slough (dead tissue) or dark brown or black eschar (hardened dead tissue): This is Stage 3 or Stage 4 pressure injury — the wound goes deep into fat, and if you see muscle, tendon, or bone, it is Stage 4. Infection can spread into bone (osteomyelitis) or blood (sepsis) from here.
- Braden Scale score under 16, or patient cannot move on their own, has poor nutrition (albumin under 3.0 g/dL or eats less than half of meals), or has incontinence (wet skin from urine or stool): These are the upstream causes of pressure injury. A low Braden score means the patient is at high risk and needs prevention now — turning every 2 hours, pressure-relief mattress, heel protection, and nutrition support.
- Warmth, swelling, pus, foul smell, or fever (temperature over 100.4°F or 38°C) in or around a pressure wound: This means bacteria have invaded the wound and infection is spreading. In deep pressure injuries, infection can tunnel into bone or spill into blood, causing sepsis — a life-threatening emergency.
- Thick callus, hard dry eschar (black or brown dead skin that does not blanch or move), or deep undermining (the wound tunnels sideways under the skin edge): Dead tissue blocks healing and hides how deep the wound really goes. If blood flow is poor, removing the eschar can let infection in. If blood flow is good, the dead tissue must be cut away (debridement) so new skin can grow.

Diagnostics · every test answers a question

- Wound culture (bacterial swab or tissue biopsy from deep in the wound) → Is the wound infected, and which bacteria are causing it?
- Serum albumin → Does the patient have enough protein in their blood to heal wounds?
- Hemoglobin and hematocrit (red blood cell counts) → Is the patient anemic, so their blood cannot carry enough oxygen to feed healing tissue?
- X-ray of the bone under the wound → Has the infection spread into the bone (osteomyelitis)?

- Red blood cell (Erythrocyte) sedimentation rate (ESR) and C-reactive protein (CRP) → Is there deep infection or inflammation that is not obvious on exam?
- Blood cultures (two sets from different sites) → Has the infection spilled into the bloodstream (bacteremia or sepsis)?
- MRI of the affected area (if bone or deep soft-tissue infection is suspected) → How deep does the infection go, and are bone, muscle, or fascia involved?

Treatment logic

- Turn the patient every 2 hours and use a pressure-redistribution mattress (air, foam, or gel overlay) → Shifts pressure away from bony spots so blood can flow back into the skin and deliver oxygen. This stops new pressure injuries from starting and prevents Stage 1 injuries from getting worse.
- Elevate heels off the bed surface using pillows under the calves, or use heel-suspension boots → Completely removes pressure from the heel bone, which has almost no cushioning and gets pressure injuries faster than any other spot. Blood flow returns and Stage 1 injuries can heal.
- Keep skin clean and dry, change wet linens or diapers immediately, and apply moisture-barrier cream (like zinc oxide or dimethicone) to at-risk areas → Wet skin breaks down five times faster under pressure because moisture weakens the bonds between skin cells. Keeping skin dry and protected stops the top layer from peeling away.
- Give high-protein diet (1.25 to 1.5 grams per kilogram per day), plus vitamin C (500 mg twice daily) and zinc (15 to 30 mg daily if deficient) → Protein is the raw material for collagen, the glue that holds new skin together. Vitamin C is the cofactor that lets the body make collagen, and zinc helps skin cells multiply and fight infection. Without these, healing stops.
- Debride (cut away) dead tissue using sharp instruments, enzymes (like collagenase), or autolytic methods (moisture-retaining dressings that let the body's own enzymes dissolve dead tissue) → Removes the dead tissue that blocks healing and hides infection. Once the dead stuff is gone, blood vessels can grow into the wound and new skin can form. Dead tissue is also a feast for bacteria.
- Keep the wound bed moist with hydrocolloid, foam, hydrogel, or alginate dressings (not wet-to-dry gauze) → A moist wound lets skin cells (keratinocytes and fibroblasts) crawl across the wound and multiply to patch the hole. Dry wounds trap dead cells and stop migration. Wet-to-dry gauze tears out new tissue and is no longer recommended.
- Start IV antibiotics (vancomycin for MRSA, piperacillin-tazobactam or cefepime for Pseudomonas or mixed infection) if there is cellulitis, osteomyelitis, or sepsis → Kills the bacteria that are destroying tissue and spreading through the body. Antibiotics stop infection from getting worse and give the body a chance to heal.

Medications

Silver sulfadiazine (Silvadene) cream 1% · Topical antimicrobial

Silver ions damage the cell walls and DNA of bacteria (the germs that cause infection), which kills them or stops them from multiplying in the wound.

1. You spread a thin layer of the cream directly on the open wound (after cleaning it).
2. Silver ions release slowly from the cream into the wound tissue.
3. The silver enters bacteria and damages their cell walls and the DNA inside, so the bacteria die.
4. This lowers the germ count in the wound, reducing the chance of infection and helping the body repair the tissue.

SAFETY WATCH

Do NOT use if the patient is allergic to sulfa drugs (sulfonamides). · Do NOT use on the face near the eyes or on deep wounds that go down to bone or tendon without a doctor's OK. · Can cause a temporary gray stain on the skin that washes off. · Rarely causes low white blood cell count (leukopenia) if used over very large areas for weeks—lab checks may be needed. · Do NOT use on a pregnant woman at term or a newborn baby (risk of yellowing of the skin and eyes (jaundice)).

Collagenase (Santyl) ointment · Enzymatic debriding agent

Collagenase is an enzyme (a protein that speeds up a chemical reaction) that breaks down collagen, the main protein in dead tissue, into small pieces the body can clear away.

1. You apply the ointment directly to the dead tissue in the wound (after cleaning).
2. The collagenase enzyme chews through the collagen fibers holding the dead tissue together.
3. The dead tissue softens and dissolves into tiny fragments.
4. The body's white blood cells and wound fluid carry the fragments away, leaving clean, pink tissue ready to heal.

SAFETY WATCH

Do NOT use with silver-containing creams, iodine, or hydrogen peroxide at the same time—they destroy the enzyme and it won't work. · Do NOT use on dry, black, hard eschar (especially on heels with poor blood flow) without scoring or moistening it first, or the enzyme can't reach the tissue underneath. · Stop if the wound edges turn red, swollen, hot, or painful—those are signs of infection, and you need a different treatment. · Keep the ointment refrigerated; check the expiration date.

Hydrocolloid dressing (e.g. DuoDERM, Tegaderm Hydrocolloid) · Moisture-retentive wound dressing (not a drug, but a medical device that supports healing)

The dressing is a sticky, gel-forming pad that absorbs fluid from the wound and keeps the wound bed moist, which helps skin cells migrate and multiply faster to close the gap.

1. You clean the wound, then stick the hydrocolloid pad over it, covering the edges of healthy skin.
2. Wound fluid seeps into the pad and turns the gel layer into a soft, moist cushion.
3. The moist environment keeps skin cells (keratinocytes) and repair cells (fibroblasts) active, so they crawl across the wound faster and lay down new tissue.
4. The outer layer of the dressing blocks air and bacteria, protecting the wound while it heals.

SAFETY WATCH

Do NOT use on an infected wound (red, hot, pus, foul smell)—moisture will make infection worse. · Do NOT use on a deep wound (stage 3 or 4) or one with heavy drainage—the dressing will leak and peel off. · Change the dressing when it starts to leak, smell, or peel (usually every 3–7 days). · When you peel it off, you may see a gel or yellow fluid under the pad—that is normal liquefied dressing, NOT pus, unless it smells bad.

Foam dressing (e.g. Mepilex, Allevyn) · Absorbent wound dressing (not a drug, but a medical device)

The foam is a spongy pad that soaks up wound fluid (exudate) while keeping the wound bed moist, cushions the sore from pressure, and lets oxygen reach the tissue to help repair.

1. You clean the wound and place the foam pad over it, securing the edges.
2. The foam pulls fluid away from the wound into its sponge layer, keeping the skin around the wound dry.
3. The moist (but not soaked) surface helps skin and repair cells grow and move.

4. The soft foam spreads pressure away from the sore, lowering the squeeze on blood vessels underneath.

SAFETY WATCH

Do NOT use on a dry wound or one with hard black eschar—foam needs some moisture to work. · Do NOT use on an infected wound without antibiotic treatment—the dressing will trap bacteria. · Change when the foam is soaked through or starts to smell (usually every 2–7 days, depending on drainage). · Check the skin around the wound for wetness (maceration) or breakdown—if it is too wet, change the dressing more often or switch to a more absorbent type.

Zinc (oral supplement, typically 15-50 mg elemental zinc per day) · Mineral supplement

Zinc is a mineral that cells need to multiply and build collagen (the scaffold protein in new skin), and it also helps enzymes that fight infection and repair tissue work properly.

1. You swallow the zinc pill, and it is absorbed in the small intestine into the bloodstream.
2. Blood carries zinc to the wound site and to cells all over the body.
3. Cells use zinc to turn on genes for collagen production and to power enzymes (like matrix metalloproteinases and superoxide dismutase) that remodel tissue and fight free radicals.
4. With enough zinc, cells multiply faster and lay down stronger collagen, so the wound closes and strengthens.

SAFETY WATCH

Do NOT give high doses (over 50 mg per day) long-term without checking a zinc level—it can block copper absorption and cause anemia or nerve problems. · Can cause nausea or stomach upset; take with food if that happens. · Most people with normal nutrition do NOT need extra zinc—only give if a blood test shows deficiency or the patient is malnourished. · Do NOT replace a full, balanced diet with zinc alone—wound healing needs protein, vitamin C, calories, and other nutrients too.

Vitamin C (ascorbic acid, oral supplement, typically 500-1000 mg per day in divided doses) · Vitamin supplement

Vitamin C is a helper molecule (cofactor) that enzymes need to build collagen, the main protein in skin and connective tissue. Without enough vitamin C, collagen is weak and wounds do not heal.

1. You swallow the vitamin C pill, and it is absorbed in the small intestine into the bloodstream.

2. Blood delivers vitamin C to fibroblasts (the cells that make collagen) at the wound.
3. Fibroblasts use vitamin C to activate enzymes (prolyl hydroxylase and lysyl hydroxylase) that stitch together collagen strands into strong, stable fibers.
4. With enough vitamin C, the collagen holds the new skin together firmly, and the wound closes faster and stronger.

SAFETY WATCH

Very high doses (over 2000 mg per day) can cause diarrhea, stomach cramps, or kidney stones in some people. · People with kidney disease or a history of kidney stones should ask their doctor before taking high doses. · Vitamin C is water-soluble—the body flushes out extra in urine, so it is hard to overdose, but very high doses are usually not more helpful. · Most people with a normal diet get enough vitamin C from food—only supplement if the patient is malnourished or has scurvy (very rare).

Red flags

- Fever, foul-smelling drainage, or spreading redness around the wound (signs of infection that can lead to sepsis)
- Bone visible in the wound or suspected bone infection (osteomyelitis requires weeks of intravenous antibiotics)
- Rapid expansion of a pressure injury despite treatment (suggests continued unrelieved pressure or serious underlying disease)
- Black or very dark brown dry dead tissue covering a large area (eschar may hide deep infection underneath)

Nursing priorities

1. Reposition the patient every 2 hours around the clock using a turn schedule and document each turn with time and position — Capillaries (tiny blood vessels) collapse under pressure above 32 millimeters of mercury cutting off oxygen and glucose delivery to skin cells which die after 2 hours of continuous pressure
2. Place patient on a pressure-redistribution mattress such as low-air-loss or alternating-pressure overlay within 1 hour of admission — These specialized surfaces spread body weight over larger area reducing peak pressure at bony prominences (hip bones, tailbone, heels) below the critical 32 millimeters of mercury threshold that causes starved blood flow (ischemia)

3. Elevate both heels completely off the bed surface using pillow under calves so knee is slightly bent at 5 to 10 degrees — Heels have thin tissue over bone with poor blood supply and cannot tolerate any pressure so must float free to prevent Category 3 or 4 injury that exposes tendon or bone
4. Inspect all skin over bony areas every 8 hours during bath and repositioning looking for non-blanchable redness and document findings with Braden Scale score — Stage 1 pressure injury appears as persistent red area that does not turn white with finger pressure indicating damaged capillaries leaking red blood cells into dermis (deep skin layer) requiring immediate pressure relief before advancing to open ulcer
5. Clean skin gently with pH-balanced no-rinse cleanser after each incontinence episode and apply moisture-barrier cream containing dimethicone or zinc oxide to buttocks and groin — Urine and stool contain enzymes and bacteria that digest the stratum corneum (outer protective skin layer) raising skin pH above normal 5.5 and causing maceration (waterlogged breakdown) plus chemical irritant dermatitis
6. Give high-protein oral supplement with 30 grams protein plus 500 milligrams vitamin C and 15 milligrams zinc twice daily with meals — Fibroblasts (cells that make new tissue) need protein building blocks called amino acids plus vitamin C cofactor to produce collagen and zinc mineral to activate enzymes for epithelial cell migration across wound bed
7. Cover Stage 2 or higher wounds with hydrocolloid dressing for shallow wounds or foam dressing for deeper wounds changing every 3 to 7 days or when saturated — Moist wound environment keeps growth factors and enzymes active at the wound surface allowing keratinocytes (skin cells) to migrate 3 times faster than dry wounds while autolytic debridement (self-cleaning) removes dead tissue
8. Consult wound care specialist or provider for sharp debridement if black eschar or yellow slough covers more than 25 percent of wound bed — Dead tissue called dead (necrotic) debris blocks oxygen diffusion and provides food source for bacteria preventing granulation tissue (new pink healing tissue with capillaries) formation until removed

Sources

- National Pressure Injury Advisory Panel (NPIAP) 2016 staging system
- Agency for Healthcare Research and Quality (AHRQ) pressure ulcer prevention guideline
- Wound Ostomy and Continence Nurses Society (WOCN) 2016 guideline
- Cochrane systematic reviews on pressure ulcer prevention and treatment

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