

Skin damage

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

THE ONE-LINE TRUTH

Pressure squashes tiny blood tubes in the skin, so oxygen stops reaching the spot and the skin dies.

The chain — how it unfolds

1. Pressure on a bony spot squashes capillaries and blocks blood flow to the skin above it. — why: 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. — why: 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. — why: 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. — why: 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. — why: 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. — why: 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. — why: 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. — why: 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.

Spot it — what you see, 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.

Red flags — act now

- 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)

Treat it — in this order

1. 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.
2. 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.
3. 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.
4. 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.
5. 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.

Your top priorities at the bedside

1. Reposition the patient every 2 hours around the clock using a turn schedule and document each turn with time and position — why: 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 — why: 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 — why: 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