

Compartment Syndrome

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THE STORY IN ONE SENTENCE

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“Compartment syndrome is when swelling or bleeding trapped inside a tight sleeve of tissue squeezes the blood vessels shut.”

Normal anatomy — the body's job

Muscles live inside sealed sleeves of fascia—tough tissue that does not stretch. Blood flows into the compartment through arteries and out through veins. Normally the pressure inside is much lower than the pressure pushing blood through the arteries, so oxygen-rich blood feeds the muscle and nerve without trouble.

BODY'S JOB

The fascia holds muscles in place and gives them a firm anchor to pull against when they contract. The low pressure inside the compartment keeps veins open so blood can leave and arteries open so fresh blood can arrive. Nerves run through the same space, carrying signals for movement and sensation.

1. Picture a forearm or lower leg wrapped in a snug, non-stretchy sleeve—that is the fascia.
2. Inside the sleeve sit muscles, nerves, and blood vessels all packed together.
3. Arteries pump blood in under pressure (like a garden hose); veins carry used blood out (like a soft drain pipe).
4. Because the compartment pressure is low—usually under 10 millimeters of mercury—the hose stays open and the drain stays open, so everything flows smoothly.
5. The muscle gets oxygen and glucose from the blood; the nerve stays alive and sends signals back and forth to the brain.

What breaks

Something makes the compartment swell or bleed, but the fascia cannot stretch, so pressure climbs until it squeezes the blood vessels shut—first the veins, then the arteries.

CLINICAL TERM

Increased intra-compartmental pressure exceeds capillary blood flow (perfusion) pressure, causing starved blood flow (ischemia) of muscle and nerve within a closed fascial space.

How the body compensates

The body has no good way to fix compartment syndrome on its own. Early on, blood pressure may rise a little to try forcing flow past the squeeze, and you may see normal vital signs even though muscle is dying—making it easy to miss the emergency until it is too late.

1. {'what': 'Blood pressure climbs slightly', 'why': 'The heart tries to push harder to force blood through the pinched vessels—but fascia does not stretch, so the effort fails and pressure keeps rising inside the compartment.'}
2. {'what': 'Pulse and oxygen numbers stay normal', 'why': 'Those measure the whole body or the finger; they do not show what is happening inside one sealed muscle compartment, so the monitor looks fine while muscle starves.'}
3. {'what': 'Pain escalates out of proportion', 'why': 'Nerve endings scream as oxygen runs out and acid builds up; this is the body's only reliable alarm, but it is often dismissed as normal post-injury pain.'}
4. {'what': 'No real compensation exists', 'why': 'Because the fascia cannot stretch and the body cannot dissolve it, the only fix is surgical—cutting the fascia open (fasciotomy) to drop the pressure instantly.'}

Domino effect

1. Injury causes bleeding or swelling inside a closed fascial compartment. — Broken bone, crush, burn, or restored blood flow after blockage dumps fluid or blood into a space wrapped by tough, non-stretchy fascia—volume goes up but the container cannot expand.
2. Compartment pressure rises above the pressure in the veins (usually above 20–30 mm Hg). — Veins are low-pressure tubes, so even a small pressure increase squeezes them flat and blocks blood from leaving—used blood and extra fluid get trapped, making swelling even worse.

3. Pressure climbs further and pinches capillaries, stopping oxygen delivery to muscle and nerve. — When pressure nears or exceeds capillary pressure (roughly 30–40 mm Hg), fresh blood cannot reach the tissue—muscle and nerve cells begin starving for oxygen within minutes.
4. Severe pain begins, much worse than the original injury, and stretching the muscle makes it sharper. — Dying muscle releases acid and toxins that fire nerve pain signals; stretching pulls on the blood-starved (ischemic) (oxygen-starved) tissue and squeezes out the last bit of blood, intensifying the pain.
5. Tingling or numbness (Paresthesias) (tingling, numbness) appear as nerves start to fail. — Nerves need constant oxygen; when supply drops, they misfire or stop firing—you feel pins and needles first, then numbness as the nerve shuts down completely.
6. Muscle weakness progresses to paralysis. — Without oxygen, muscle cells cannot contract; the nerve cannot tell the muscle what to do because the nerve itself is dying—voluntary movement disappears.
7. Pulse becomes weak or absent, and the limb feels tight and swollen. — Arterial flow finally stops when compartment pressure exceeds arterial diastolic pressure (roughly 60–80 mm Hg)—by this point, muscle and nerve have been dying for hours and permanent damage is likely.
8. Muscle dies (necrosis) after four to six hours of starved blood flow (ischemia), releasing myoglobin and potassium into the bloodstream (rhabdomyolysis). — Dead muscle cells burst open, dumping myoglobin (a protein that clogs kidney filters) and potassium (which disrupts heart rhythm)—this can cause kidney failure and fatal arrhythmia if not treated.

Symptoms · what the patient feels, and why

- Very bad pain that gets much worse when you stretch the muscle: Pressure inside the muscle compartment (the tissue wrapping around the muscle) is crushing the muscle and nerves, so any stretch pulls on already-damaged tissue and sends sharp pain signals to your brain.
- The compartment feels firm and tight, like a blown-up balloon: Swelling or bleeding inside the compartment has nowhere to go, so pressure builds up and the tissue stretches tight against its wrapping.
- Numbness or tingling in the area past the swelling: High pressure squeezes the nerves that carry feeling signals, blocking them like pinching a phone cable so the message cannot get through.
- Weakness or trouble moving the fingers or toes beyond the injury: Pressure is crushing the nerves that tell muscles to move, so the brain's commands cannot reach those muscles anymore.

- Pale or shiny skin over the area: Swelling stretches the skin tight and squeezes tiny blood vessels near the surface, so less blood reaches the skin and it looks paler or shiny.
- Pain that does not get better with rest or usual pain medicine: The source of pain is ongoing pressure and tissue damage inside the compartment, not simple inflammation, so normal pain relievers cannot fix it.
- A pulse that is hard to feel or missing below the injury: Severe pressure can squeeze the main arteries (blood highways) enough to slow or block blood flow, so the pulse wave that normally travels through them weakens or disappears.

Focused exam

- Pain that feels way worse than the injury should cause: This is the earliest and most important warning sign. Rising pressure inside the muscle compartment squeezes nerves before anything else happens. If pain seems out of proportion, think compartment syndrome right away.
- Pain shoots up when you gently stretch the muscle (passive stretch): Stretching the muscle pushes against the tight fascia (the wrapping around the compartment) and makes the pressure spike even higher. This flash of pain tells you the compartment has no room left to expand.
- The compartment feels tight, swollen, and hard to the touch: Blood and swelling are trapped inside the fascia with no way out. The firm, tense feel means pressure is building. Compare the hurt limb to the other side — healthy muscle feels soft.
- Tingling, numbness, or a pins-and-needles feeling in the limb: Nerves are getting crushed by the rising pressure. Nerves fail before muscles do, so tingling appears early. If you wait until numbness is complete, permanent nerve damage may already be done.
- Pulses are still present early on (palpable or by Doppler): Arteries have thick, muscular walls and can stay open even when compartment pressure is dangerously high. A pulse does NOT rule out compartment syndrome. Muscle and nerves die before the artery collapses.
- Pale, cold skin and absent pulse appear very late: By the time the pulse disappears, muscle has already been dying for hours. These are end-stage signs. The window to save the limb has almost closed.

Diagnostics · every test answers a question

- Compartment pressure measurement (handheld manometer or needle probe) → Is the pressure inside the muscle compartment high enough to block blood flow?
- Creatine kinase (CK) level in blood → Is muscle breaking down (rhabdomyolysis) from prolonged starved blood flow (ischemia) (lack of oxygen)?

- Serum potassium → Is potassium leaking out of dying muscle cells and reaching dangerous levels in the blood?
- Urine myoglobin or urine color (dipstick for blood without RBCs under microscope) → Is myoglobin (a muscle protein) being dumped into the urine and threatening the kidneys?
- Serum creatinine and blood urea nitrogen (BUN) → Are the kidneys starting to fail from myoglobin overload?
- X-ray of the injured limb → Is there a fracture or other bone injury that caused the compartment syndrome?

Treatment logic

- Remove or loosen any tight cast, dressing, splint, or wrap immediately → Cuts external pressure on the limb right away so blood can flow back into the compartment. This buys time before tissue dies and may prevent the need for surgery if caught very early.
- Emergency fasciotomy (surgical cuts through the fascia to open all compartments) → Opens the tight compartment so pressure drops instantly below the blood pressure level. Oxygen and nutrients can reach the muscle again, stopping the chain of cell death. This is the only treatment that fixes the core problem.
- Keep the limb at heart level (NOT elevated) → Keeps blood pressure inside the limb as high as possible so blood can still push into the compartment against the rising pressure. Elevating the limb lowers the driving pressure and worsens starved blood flow (ischemia).
- Give aggressive IV fluids (normal saline or lactated Ringer's at 200–300 mL/hour) and monitor urine output closely → Flushes myoglobin through the kidneys before it clogs the tubules and causes kidney failure. Keeps urine output above 200–300 mL/hour to prevent myoglobin from crystallizing in the kidney.
- Treat high potassium (hyperkalemia) if potassium is dangerously high (calcium gluconate 1 g IV, insulin 10 units + dextrose 25 g IV, sodium bicarbonate if acidotic) → Calcium stabilizes the heart muscle so high potassium cannot trigger a fatal arrhythmia. Insulin and dextrose drive potassium back into cells temporarily. Bicarbonate shifts potassium intracellularly if the blood is acidic.
- Plan delayed wound closure or skin graft days later, after swelling resolves → Once the compartment is decompressed and swelling goes down, the surgeon can close the fasciotomy wounds or cover them with a skin graft without crushing the healing tissue again.
- Avoid giving opioids in escalating doses without re-examining the limb → Pain out of proportion is the key diagnostic clue. Masking the pain with more and more opioids can hide the worsening compartment syndrome until it is too late to save the limb.

Medications

Fasciotomy (surgical procedure, not a drug) · Emergency surgical decompression

The surgeon cuts open the tight tissue wrapper (fascia) around the muscle compartment so the swelling can escape and blood can flow again. This lowers the pressure inside the compartment immediately, which saves the muscle and nerves from dying.

1. The surgeon makes a long cut through the skin and the fascia covering the swollen muscle compartment.
2. The muscle bulges out through the opening because it was so tightly squeezed before.
3. Pressure inside the compartment drops right away, usually from dangerously high levels (above 30 mmHg) down to normal (under 10 mmHg).
4. Blood flow returns to the muscle and nerves, delivering oxygen and nutrients again.
5. The wound is left open or loosely covered for several days until swelling goes down, then closed or covered with a skin graft in a second surgery.

SAFETY WATCH

Infection risk is high because the wound is left open for days; watch for redness, warmth, pus, or fever. · Bleeding can happen during or after surgery; the team watches clotting labs and blood pressure closely. · The limb may lose some function permanently if fasciotomy was delayed and muscle or nerve tissue already died. · Scarring and skin grafts may be needed, which can limit movement or look different from normal skin.

Intravenous normal saline · Isotonic crystalloid fluid

Saline is salt water given through an IV to flush damaged muscle proteins (myoglobin) out of the blood and through the kidneys before those proteins clog the kidney filters. It also keeps blood pressure stable and dilutes dangerous potassium that leaks from crushed muscle cells.

1. Saline is infused rapidly through an IV, often 200–400 mL per hour or more in adults (adjusted for body size in children).
2. The extra fluid increases blood volume, which raises blood pressure inside the kidney's filters (glomeruli).
3. Higher pressure pushes more water and waste through the filters, making more urine.
4. Myoglobin (a reddish-brown muscle protein released when muscle cells break open) gets diluted and flushed into the urine before it clumps and blocks kidney tubules.
5. Potassium (which leaks from dying muscle) is also diluted and excreted, lowering the dangerously high blood potassium level (hyperkalemia).

SAFETY WATCH

Giving too much saline can overload the lungs with fluid, causing trouble breathing (pulmonary swelling (edema)); the team listens to lung sounds and watches oxygen levels. · Monitor urine output closely—goal is at least 200–300 mL per hour in adults to keep myoglobin moving out; a catheter is usually placed to measure every hour. · Watch labs for rising potassium, creatinine (kidney function), and creatine kinase (CK, a marker of muscle breakdown); these guide how much fluid is needed. · In patients with heart failure or kidney disease, saline must be given more slowly and carefully to avoid fluid overload.

Sodium bicarbonate (baking soda solution given IV) · Alkalinizing agent

Bicarbonate makes the blood and urine less acidic (more alkaline). When urine is alkaline, myoglobin is much less likely to clump together and clog the kidney's tiny tubes, so it flows out safely in the urine instead of causing kidney failure.

1. Sodium bicarbonate is added to the IV saline or given separately through the IV line.
2. Bicarbonate ions (HCO_3^-) soak up extra hydrogen ions (H^+) in the blood, raising the blood pH from acidic toward normal (goal pH around 7.4).
3. The kidneys filter bicarbonate into the urine, raising urine pH to 6.5 or higher.
4. At higher pH, myoglobin stays dissolved and does not precipitate (clump into crystals) in the kidney tubules.
5. Myoglobin flows safely out in the urine, reducing the risk of acute kidney injury.

SAFETY WATCH

Too much bicarbonate can make the blood too alkaline (alkalosis), causing muscle twitching, confusion, or dangerous heart rhythms; check blood pH and bicarbonate levels regularly. · Sodium bicarbonate adds extra sodium, which can worsen fluid overload in patients with heart or kidney problems; monitor closely. · Watch serum calcium—too little acid in the blood (alkalosis) can lower ionized (active) calcium and cause muscle cramps or, rarely, seizures. · Not all hospitals routinely use bicarbonate for rhabdomyolysis; large IV saline alone is often enough. Bicarbonate is reserved for severe cases with very high CK or acidic blood.

Calcium gluconate (IV) · Calcium salt, membrane stabilizer

Calcium gluconate does not lower blood potassium, but it protects the heart muscle from dangerous rhythms caused by high potassium. It works by stabilizing the heart cell membranes so they are less likely to fire incorrectly, even while potassium is still high.

1. Calcium gluconate is pushed slowly through an IV over 2–5 minutes.

2. Calcium ions quickly bind to the outside of heart muscle cells.
3. High potassium normally makes the cell membrane too excitable (easier to trigger chaotic beats); calcium counteracts that by raising the threshold needed to fire an electrical signal.
4. Within 1-3 minutes, the heart rhythm becomes more stable and the risk of sudden cardiac arrest drops.
5. The effect lasts about 30-60 minutes, so other treatments (insulin, albuterol, dialysis) must be started right away to actually lower the potassium level.

SAFETY WATCH

Must be given slowly (over several minutes); pushing it too fast can cause a dangerously slow heart rate or even cardiac arrest. · Watch the EKG (heart monitor) during and after infusion for any rhythm changes. · Repeat the dose if EKG changes from high potassium return before potassium level is controlled. · Use cautiously in patients on digoxin (a heart medicine) because extra calcium can increase digoxin toxicity.

Regular insulin (with dextrose/glucose) · Hormone, potassium-lowering agent

Insulin pushes potassium from the bloodstream into cells, which lowers the blood potassium level temporarily. Glucose is given at the same time to prevent blood sugar from dropping dangerously low.

1. Regular insulin (usually 10 units) is given through an IV along with 25-50 grams of dextrose (sugar water).
2. Insulin binds to receptors on cell surfaces and activates the sodium-potassium pump inside the membrane.
3. The pump pushes potassium ions from blood into cells in exchange for sodium moving out.
4. Blood potassium drops by 0.5-1.2 mEq/L within 15-30 minutes.
5. The effect lasts 4-6 hours, so other treatments (dialysis if needed) must continue to keep potassium down long-term.

SAFETY WATCH

Monitor blood sugar closely every 15-30 minutes for the first hour; even with dextrose, blood sugar can drop dangerously low (hypoglycemia), causing confusion, sweating, or seizures. · Recheck potassium levels every 1-2 hours because potassium will eventually leak back out of cells if the underlying problem is not fixed. · Watch for signs of low blood sugar: shakiness, sweating, fast heartbeat, confusion, or loss of consciousness; treat immediately with more dextrose. · Not safe to use without dextrose except in patients with very high blood sugar (over 250 mg/dL).

Albuterol (inhaled, nebulized) · Beta-2 adrenergic agonist, potassium-lowering agent

Albuterol activates beta-2 receptors on cell surfaces, which turns on the sodium-potassium pump and drives potassium from the blood into cells. This is the same drug used for asthma, but here it is given in higher doses to shift potassium.

1. Albuterol is given through a nebulizer (breathing treatment) at a dose of 10–20 mg (much higher than asthma doses).
2. Albuterol binds to beta-2 receptors on cells and activates a signaling pathway inside the cell.
3. The sodium-potassium pump ramps up, pulling potassium from the blood into the cell interior.
4. Blood potassium drops by 0.5–1.0 mEq/L within 30–90 minutes.
5. The effect is temporary (2–4 hours), so it is used as a bridge while arranging definitive treatment like dialysis if needed.

SAFETY WATCH

High doses can cause fast heart rate (tachycardia), tremor (shaking), anxiety, or chest discomfort. · Watch the heart monitor because rapid heartbeats can be dangerous in patients with existing heart disease. · Recheck potassium levels 1–2 hours after treatment to confirm it worked and to see when potassium starts rising again. · May not work as well in patients already on beta-blocker medications (like metoprolol), which block the same receptors.

Hemodialysis · Renal replacement therapy (mechanical procedure, not a drug)

A dialysis machine removes blood from the body, runs it through a filter that takes out potassium, myoglobin, and other waste, and returns clean blood to the body. This is the only treatment that actually removes potassium and toxins permanently when the kidneys cannot do the job.

1. A large IV catheter (dialysis catheter) is placed into a central vein, usually in the neck or groin.
2. Blood is pumped out of the body through one line, into the dialyzer.
3. Inside the dialyzer, blood flows on one side of a membrane and dialysis fluid (dialysate) flows on the other side.
4. Potassium, myoglobin, urea, creatinine, and excess fluid pass from blood through the membrane into the dialysate because of differences in concentration (diffusion) and pressure (ultrafiltration).

5. Cleaned blood flows back into the body through the return line.
6. A typical session lasts 3–4 hours and can lower potassium by 1.5–2.0 mEq/L or more.

SAFETY WATCH

Placing the dialysis catheter carries risks: bleeding, infection, collapsed lung (pneumothorax if placed in the neck), or blood clots. · During dialysis, blood pressure can drop suddenly, causing dizziness, nausea, or fainting; fluids or medications may be needed to keep pressure stable. · Infection of the catheter site or bloodstream (sepsis) is a serious risk; watch for fever, redness, or pus at the catheter. · Dialysis removes potassium quickly, but levels can rebound after the session ends as potassium leaks back out of cells; repeat labs and possibly repeat dialysis may be needed. · Patients may feel exhausted or have muscle cramps after dialysis because of rapid fluid and electrolyte shifts.

Red flags

- Pain that keeps getting worse even after rest, ice, or pain medicine
- Numbness, tingling, or cannot feel light touch in the fingers or toes beyond the swelling
- Cannot move fingers or toes, or movement is very weak
- Skin looks very pale, blue, or feels cold past the injury
- Pulse is weak or cannot be felt below the injured area

Nursing priorities

1. Remove or loosen any cast, splint, tight bandage, or wrap down to the skin immediately and notify the provider right away. — External pressure from tight dressings adds to the pressure inside the compartment, so removing them may lower the pressure enough to restore some blood flow and buy time before surgery.
2. Assess and document pain level, firmness of the compartment, ability to move fingers or toes, sensation to light touch, skin color, skin temperature, and pulses distal to the injury every 15 to 30 minutes. — Compartment syndrome gets worse quickly, and frequent neurovascular checks (nerve and blood vessel checks) catch changes early so the surgeon can operate before permanent muscle and nerve death happen.
3. Keep the affected limb at heart level—do NOT elevate it above the heart. — Elevating the limb lowers the blood pressure going into the compartment and makes starved blood flow (ischemia) (lack of oxygen) worse, while keeping it at heart level maintains the best possible blood flow into the damaged area.

4. Start or maintain IV access with a large-bore IV catheter and give aggressive IV fluids such as normal saline or lactated Ringer's solution at 200 to 300 milliliters per hour as ordered. — High IV fluid rates help flush myoglobin (a muscle protein released when muscle dies) out of the kidneys and prevent acute kidney injury, which is a common complication after fasciotomy when dead muscle releases toxins into the blood.
5. Insert a urinary catheter and monitor urine output every hour, watching for dark brown or tea-colored urine. — Dark urine means myoglobin is being released from dying muscle and can clog the kidney tubules and cause kidney failure, so close urine monitoring helps guide fluid therapy and alerts the team to kidney damage.
6. Prepare the patient for emergency fasciotomy by keeping them NPO (nothing by mouth), obtaining informed consent, and coordinating with the operating room. — Fasciotomy is the only treatment that relieves the pressure and restores blood flow, and surgery must happen within 6 hours of symptom onset to save the muscle and nerve from permanent damage or death.
7. Monitor serum potassium, creatinine, and creatine kinase (CK) levels closely and report dangerous values to the provider immediately. — Dying muscle releases potassium into the blood (hyperkalemia), which can stop the heart, and releases creatine kinase and myoglobin, which damage the kidneys, so lab monitoring catches life-threatening complications early.
8. Have emergency high potassium (hyperkalemia) treatment at the bedside: calcium gluconate, insulin with dextrose, and sodium bicarbonate, ready to give if potassium rises above 6.0 milliequivalents per liter. — High potassium levels can cause deadly heart arrhythmias (irregular heartbeats), and calcium gluconate protects the heart muscle while insulin and dextrose move potassium back into cells to lower the blood level quickly.

Sources

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