

Transfusion Reactions

The bag is guilty until proven innocent.

THE STORY IN ONE SENTENCE

The bag is guilty until proven innocent.

“Transfusion reaction — stop the bag first, ask questions second.”

Normal anatomy — the body's job

A matched bag of red blood cells is supposed to quietly restore oxygen-carrying capacity without triggering the receiver's immune system. The donor plasma is mostly removed before the bag is hung. The antigens (surface markers) on the donor red cells are compatible with the receiver's antibodies, so the immune system stays calm. Citrate in the bag keeps the blood from clotting during storage. That is the normal job. Keep that picture clear, because every weird finding you see during a transfusion reaction is a change from this quiet, compatible handoff.

BODY'S JOB

The bag's job is to deliver fresh, compatible red cells that slip into circulation, bind oxygen in the lungs, carry it to every tissue, and then return to pick up more — all without waking up the immune system. The spleen quietly filters out old donor cells over weeks. The receiver's bone marrow eventually takes over full red-cell production again. When this handoff works, nothing dramatic happens: hemoglobin goes up, oxygen delivery improves, and the patient feels stronger.

1. Donor blood is tested for ABO and Rh type, screened for unexpected antibodies, and matched to the receiver's type.
2. The red cells are separated from most of the plasma (which holds donor antibodies) and suspended in a preservative solution with citrate to prevent clotting.
3. At the bedside, the nurse double-checks the patient ID and blood-bank label, then hangs the bag and starts the transfusion slowly for the first 15 minutes while watching for any reaction.

4. Compatible donor red cells flow into the receiver's veins, travel to the lungs to pick up oxygen, then spread through arteries to every tissue.
5. The receiver's immune system checks the surface markers (ABO, Rh, and others) on the donor cells. If they match, the immune system stays quiet.
6. Over days to weeks, the spleen gently removes old donor red cells, and the receiver's bone marrow ramps up its own production to take over the job.

What breaks

Usually one upstream break — an immune mismatch, a contaminated bag, or too much volume — explains every weird finding together.

CLINICAL TERM

The receiver's immune system has antibodies (from prior transfusion, pregnancy, or transplant) that recognize donor red-cell antigens as foreign and attack them, bursting the cells (acute destroying red blood cells (hemolytic) transfusion reaction, AHTR). Or the donor plasma (not fully removed) has antibodies that bind the receiver's white cells in lung capillaries, making fluid leak into the air sacs (TRALI — transfusion-related acute lung injury). Or bacteria (often from skin during collection or cold-chain break) multiplied in the bag and now release endotoxin into the bloodstream (septic transfusion reaction). Or leftover donor white cells release cytokines (inflammatory signals) that trigger fever without killing red cells (febrile non-destroying red blood cells transfusion reaction, FNHTR). Or the receiver's IgE antibodies recognize harmless donor plasma proteins and trigger mast cells to dump histamine, causing hives (allergic reaction) or, rarely, full airway swelling and shock (anaphylaxis). Or the volume of the transfusion overloads a weak heart, raising pressure in the lungs (TACO — transfusion-associated circulatory overload). That break does not stay local. The downstream signals you see at the bedside are: red blood cell destruction (hemolysis) spills free hemoglobin that clogs kidney tubules, uses up clotting factors, and drops blood pressure (DIC and shock). TRALI floods the lungs and drops oxygen. Septic reaction triggers systemic inflammation and shock. Allergic reaction releases histamine that dilates vessels and leaks fluid. Read every weird finding backward from one of these breaks — they all point upstream.

How the body compensates

Vital signs can look stable for a short window while the body works hard to cope — raising heart rate to maintain blood pressure, shunting blood away from the gut and skin to protect the brain and heart, and ramping up breathing to blow off acid. That middle window is dangerous because it hides how close the patient is to crashing. The job is to spot the

compensation early and stop the transfusion before the system tips into shock, kidney failure, or respiratory failure.

1. {'what': "The sympathetic nervous system (the body's fight-or-flight wiring) kicks in immediately.", 'how': 'The adrenal glands dump epinephrine and norepinephrine, which squeeze blood vessels to keep blood pressure up and speed the heart to push more blood per minute, buying time even as red cells are being destroyed or fluid is leaking into the lungs.'}
2. {'what': 'Blood flow is redirected to protect vital organs.', 'how': 'Vessels in the skin, gut, and muscles clamp down (causing cool, pale skin and slowed digestion) so more blood reaches the brain, heart, and kidneys — this keeps the patient awake and the heart beating, but it cannot last.'}
3. {'what': 'Breathing rate climbs to blow off acid and bring in more oxygen.', 'how': 'Chemoreceptors in the brainstem (the breathing control center) sense rising carbon dioxide and acid from shock or lung flooding, so the respiratory muscles work faster and deeper to keep pH and oxygen steady.'}
4. {'what': 'The renin-angiotensin-aldosterone system tries to hold onto fluid and salt.', 'how': 'The kidneys release renin, which makes angiotensin II (a powerful vessel constrictor) and aldosterone (a hormone that keeps sodium and water from leaving in urine), raising blood volume and pressure — but this backfires if the kidneys are clogged with hemoglobin or the lungs are already overloaded.'}
5. {'what': 'Clotting factors are consumed trying to seal damaged vessels.', 'how': 'Free hemoglobin, endotoxin, or immune complexes activate the clotting cascade everywhere at once (DIC — disseminated intravascular coagulation), forming tiny clots that use up platelets and fibrinogen, leaving the patient at risk for bleeding even while clotting.'}
6. {'what': 'That compensation window is the dangerous middle, because it lets the team miss where the patient really is on the curve.', 'how': "A patient with a heart rate of 110, blood pressure holding at 100/60, and oxygen saturation 92 percent looks 'stable' on the monitor, but those numbers mean the body is working at maximum to avoid collapse — any further hit (more red blood cell destruction (hemolysis), more fluid leak, more vasodilation) tips the system into shock, and the window to reverse it closes fast."}
7. {'what': 'The crash points to watch for are: acute kidney injury from free hemoglobin clogging the tubules, respiratory failure from TRALI or TACO flooding the lungs, or distributive shock from septic or anaphylactic vasodilation.', 'how': 'Once the kidneys stop making urine, potassium and acid pile up and can stop the heart. Once the lungs fill with fluid, oxygen drops and carbon dioxide rises, leading to respiratory arrest. Once blood pressure drops below the point where organs are fed, multi-organ failure begins — and survival depends on how fast the transfusion is stopped and supportive care is started.'}

8. {'what': 'Reactions in the first 15 minutes are the most dangerous.', 'how': 'ABO-mismatched red blood cell destruction (hemolysis), anaphylaxis, and septic reactions all hit fast because the immune response or toxin release starts the moment incompatible blood enters the vein — that is why the nurse watches closely and runs the transfusion slowly at first.'}
9. {'what': 'Look at the trend, not just one snapshot.', 'how': 'A patient whose heart rate climbed from 80 to 110 in ten minutes, even if blood pressure is still normal, is decompensating. A patient whose urine turned dark or whose oxygen dropped from 98 to 94 percent is showing early organ injury. Catch the downhill slide before the crash, because once vital signs collapse, the damage is often already done.'}

Domino effect

1. Acute destroying red blood cells (hemolytic) reaction: Receiver antibodies bind donor red cells and burst them open (hemolysis), spilling hemoglobin into the plasma. — The receiver's immune system (from prior transfusion, pregnancy, or transplant) has antibodies (usually anti-A, anti-B, or minor antigens like Kell or Duffy) that recognize donor cell-surface markers as foreign, bind them, activate complement proteins, and punch holes in the cell membrane — the red cell explodes, dumping hemoglobin and cell fragments into the bloodstream.
2. Free hemoglobin clogs the kidney filters and triggers acute kidney injury. — Free hemoglobin is toxic to the proximal tubule cells in the kidney (the part that reabsorbs water and salt). It also binds nitric oxide (a vessel relaxer), causing kidney blood vessels to clamp down. Together, hemoglobin physically blocks the tubules like mud in a drainpipe and starves the cells of oxygen, so the kidneys stop making urine, and waste, potassium, and acid pile up in the blood.
3. Shock develops as blood vessels dilate and blood pressure drops. — Red blood cell destruction (Hemolysis) and immune activation release cytokines (TNF-alpha, IL-1, IL-6) that tell blood vessels to relax and leak fluid into tissues. At the same time, the kidneys cannot balance fluid and electrolytes, and the heart struggles to pump enough blood to fill the widened vessels — blood pressure falls, organs are underfed, and the patient feels dizzy, weak, or confused.
4. DIC (disseminated intravascular coagulation) starts: clotting factors are used up everywhere, causing bleeding and clotting at the same time. — Free hemoglobin, cell fragments, and cytokines activate the clotting cascade all over the body at once, forming thousands of tiny clots in small vessels (using up platelets, fibrinogen, and clotting factors). The body then activates its clot-busting system (fibrinolysis), breaking clots into D-dimer fragments. The result: bleeding from the IV site, gums, or gut because clotting factors are gone, plus clots blocking blood flow to fingers, toes, kidneys, or lungs.

5. TRALI: Donor antibodies attack the receiver's lung capillaries, flooding the air sacs with fluid within six hours. — Donor plasma (not fully removed) has antibodies against the receiver's white-cell markers (HLA or neutrophil antigens). These antibodies bind white cells stuck to the lung capillary walls, activate them, and make them release enzymes and oxygen radicals that damage the thin vessel wall — fluid pours from blood into the air sacs (alveoli), blocking oxygen from crossing into the blood and causing sudden shortness of breath, low oxygen, and white patches on chest X-ray.
6. Septic reaction: Bacteria in the bag multiply and dump toxins that trigger systemic inflammation and shock. — Bacteria (skin flora from the needle stick or cold-tolerant Gram-negatives like *Yersinia* or *Pseudomonas*) grow in the stored blood and release endotoxin (from Gram-negative cell walls) or exotoxin. These toxins bind receptors on white cells and blood-vessel walls, triggering a cytokine storm (TNF-alpha, IL-1, IL-6) that dilates vessels, leaks fluid, drops blood pressure, and raises body temperature — the patient spikes a high fever, shakes with rigors, and can crash into septic shock within minutes.
7. Allergic reaction and anaphylaxis: Receiver antibodies recognize donor proteins, release histamine, and cause hives or, rarely, airway swelling and shock. — The receiver's IgE antibodies (from prior exposure to donor plasma proteins) bind those proteins, cross-link on mast cells and basophils, and trigger degranulation — the cells dump histamine, leukotrienes, and prostaglandins that dilate blood vessels (causing hives, flushing, and itching) and, in anaphylaxis, relax airway smooth muscle and leak fluid into the throat (causing wheezing, throat tightness, tongue swelling, and shock). IgA-deficient patients with anti-IgA antibodies are at highest risk.
8. TACO (volume overload): The heart cannot pump the extra volume, so pressure backs up into the lungs and fluid leaks into the air sacs. — The transfusion adds volume faster than a weak or stiff heart (from heart failure, kidney failure, or old age) can pump it forward. Pressure rises in the left atrium, then the pulmonary veins, then the lung capillaries — fluid is pushed across the thin capillary wall into the alveoli (air sacs), blocking oxygen exchange and causing shortness of breath, low oxygen, jugular-vein distension in the neck, leg swelling, and crackles heard with a stethoscope. Unlike TRALI, TACO improves with diuretics (water pills) and slowing the transfusion.

Symptoms · what the patient feels, and why

- Fever (temperature rises above 100.4°F / 38°C) during or right after the transfusion: When donor blood cells don't match yours, your immune system spots them as invaders and releases alarm chemicals called cytokines (messengers that crank up body heat to fight what looks like an infection), so your temperature spikes.

- Pain in your back, sides, or chest: Mismatched red cells burst apart inside your blood vessels (hemolysis), spilling their insides everywhere. This clogs tiny filters in your kidneys and irritates blood-vessel walls, triggering pain signals along your back and flanks where the kidneys sit.
- Trouble breathing or breathing faster than usual: Either fluid is leaking into the air sacs of your lungs (from donor white cells releasing chemicals that make lung capillaries leaky — called TRALI), or you got too much volume too fast and your heart can't keep up (volume overload), so oxygen can't cross into your blood easily and you gasp for air.
- Hives or itchy red welts on your skin: Your immune system's mast cells (guard cells in the skin) release histamine (a chemical that makes blood vessels widen and leak) when they recognize foreign proteins in the donor plasma, causing itchy raised bumps.
- Blood pressure drops suddenly (systolic falls below 90 or drops more than 30 points from baseline): Massive red blood cell destruction (hemolysis) or a severe allergic reaction floods your bloodstream with inflammatory chemicals (cytokines, histamine, bradykinin) that tell blood vessels to relax and widen; blood pools in the belly and legs, so not enough pressure pushes blood back to the heart and brain.
- Dark brown or cola-colored urine: When red cells explode (hemolysis), hemoglobin (the oxygen-carrying protein inside) spills into your plasma, travels to your kidneys, and gets filtered into the urine, turning it brown or red — a direct sign that cells are being destroyed.
- Chills and shaking (rigors): Cytokines released during an immune reaction reset your brain's internal thermostat (the hypothalamus) higher, so your body shivers hard to make heat and reach the new 'set point,' even though you already have a fever.
- Nausea or vomiting: Inflammatory chemicals and the stress response during a reaction activate the vomiting center in your brainstem (the area postrema) and slow your gut, making you feel sick to your stomach.
- Bleeding that won't stop or oozing from IV sites: Massive red blood cell destruction (hemolysis) can trigger disseminated intravascular coagulation (DIC) — your clotting factors and platelets get used up all at once trying to patch tiny clots everywhere, so you run out and can't stop bleeding anywhere else.
- Confusion, restlessness, or feeling like something is very wrong: Low blood pressure and poor oxygen delivery mean your brain isn't getting enough fuel; also, stress hormones (epinephrine, cortisol) surge during the reaction, making you feel anxious and foggy.

Focused exam

- Dark brown or red urine in the catheter bag or tubing: Free hemoglobin from destroyed red cells is spilling into the urine — this means red cells are breaking apart right now, the most dangerous kind of transfusion reaction.

- Blood pressure drops during or within minutes of starting the transfusion: The body is losing its ability to hold fluid inside blood vessels — either from red blood cell destruction (hemolysis) destroying cells, bacteria-contaminated blood causing septic shock, or anaphylaxis collapsing the vessels.
- Back pain or flank pain while the bag is running: The kidneys are being damaged because free hemoglobin is clogging the tiny filters inside them — this points directly to destroying red blood cells (hemolytic) transfusion reaction.
- Fever (temperature rise of 1°C or more during transfusion): Fever can mean the patient's antibodies are attacking donor cells, or bacteria are growing in the bag — either way, stop the transfusion and figure out which.
- Trouble breathing or chest tightness within 6 hours of transfusion start: TRALI (donor antibodies attacking the patient's lungs) or fluid overload is filling the air sacs — getting oxygen in is the top priority right now.
- Hives or itching on the skin: Allergic transfusion reaction means the patient's immune system sees something in the donor plasma as foreign — can stay mild or jump to life-threatening anaphylaxis in seconds.

Diagnostics · every test answers a question

- Direct antiglobulin test (DAT / direct Coombs) → Are antibodies stuck to the patient's red blood cells right now?
- Serum free hemoglobin and haptoglobin → Are red blood cells breaking apart inside the bloodstream?
- Urinalysis and urine hemoglobin → Is free hemoglobin spilling into the urine and damaging the kidneys?
- Blood culture from patient AND the donor bag → Is the blood bag contaminated with bacteria?
- Chest X-ray → Are the lungs filling with fluid, and is it from TRALI or fluid overload?
- Complete blood count (CBC) and coagulation panel (PT, PTT, fibrinogen, D-dimer) → Is the patient bleeding and clotting at the same time from DIC?
- Serum creatinine and urine output → Are the kidneys shutting down from hemoglobin clogging the filters?
- Send the donor bag and patient's post-transfusion sample to the blood bank immediately → Does the donor blood match the patient, and are there antibodies we missed?

Treatment logic

- Stop the transfusion immediately and keep IV line open with normal saline → Stops more donor cells or bacteria from entering the body. Keeps the vein open so you can give fluids and emergency medicines fast.
- Give large amounts of IV normal saline to keep urine flowing fast (goal urine output ≥ 100 mL/hr in adults) → Dilutes free hemoglobin in the blood and flushes it through the kidneys before it clogs the kidney tubules and shuts down the kidney.
- Give vasopressors (like norepinephrine) if blood pressure stays low despite fluids → Squeezes blood vessels to push blood to the heart, brain, and kidneys when the body cannot hold pressure on its own after red blood cell destruction (hemolysis) or septic shock.
- For anaphylaxis: give epinephrine 0.3-0.5 mg intramuscular (into the thigh muscle) right away → Blocks the allergic cascade by tightening blood vessels (raising blood pressure) and opening airways (stopping throat swelling). Works within 1-2 minutes.
- For suspected septic reaction: draw blood cultures from patient and the donor bag, then start broad-spectrum IV antibiotics immediately (e.g. ceftriaxone + vancomycin or piperacillin-tazobactam) → Kills bacteria before they multiply and release toxins that cause organ failure and septic shock.
- For TRALI: support breathing with supplemental oxygen or mechanical ventilation; do NOT give diuretics (water pills) → Oxygenates the blood when fluid leaking into the air sacs blocks gas exchange. Diuretics would make it worse because TRALI is lung injury from inflammation, not volume overload.
- For feverish (febrile) non-destroying red blood cells (hemolytic) reaction (fever only, no other signs): give acetaminophen and slow or stop transfusion → Lowers fever caused by antibodies to donor white blood cells or cytokines released during storage. Safe to restart transfusion slowly if fever is the only symptom.

Medications

Epinephrine (intramuscular, 0.3-0.5 mg for adults) · Direct sympathomimetic (adrenaline)

Epinephrine is the body's emergency hormone that quickly reverses life-threatening allergic reactions (anaphylaxis). It squeezes blood vessels tight to lift blood pressure, opens up the breathing tubes, and stops mast cells (the immune cells that explode and release histamine) from dumping more chemicals. Think of it as hitting the emergency brake and the gas pedal at once: it stops the allergic avalanche and restarts the organs.

1. Patient gets transfusion; immune system mistakes donor protein as dangerous invader and launches anaphylaxis (full-body allergic attack).

2. Mast cells dump histamine and other chemicals; blood vessels leak and widen, blood pressure drops, airways squeeze shut.
3. Epinephrine binds to alpha receptors on vessel walls → vessels clamp down → blood pressure climbs back up.
4. Epinephrine binds to beta-2 receptors in airway muscle → muscle relaxes → breathing tubes open.
5. Epinephrine shuts off mast cells → stops the chemical flood → allergic cascade halts.
6. Heart rate and squeeze strength rise (beta-1 effect) → pumps harder to keep organs fed.

SAFETY WATCH

Must give in the outer thigh muscle (vastus lateralis), NOT into a vein (IV epinephrine needs intensive care and a pump). · Can cause fast heart rate, shaking, chest tightness, or a spike in blood pressure—necessary trade-off to save life. · Repeat every 5–15 minutes if symptoms do not reverse; do NOT delay the first dose. · Older adults or anyone with heart disease may feel chest discomfort; still give it—anaphylaxis is more dangerous than the side effects.

Normal saline (0.9 % sodium chloride IV) · Isotonic crystalloid (IV fluid)

Normal saline is salt water that matches the salt level of your blood. When you pour it into a vein, it fills up the blood-vessel 'pipes' without shifting water in or out of cells. In destroying red blood cells (hemolytic) reactions (donor red cells bursting) and in anaphylaxis (blood vessels leaking), the circulation loses volume fast. Saline refills the tank so blood pressure stays up and kidneys get enough flow to wash out the red-cell debris (hemoglobin) before it clogs the kidney filters.

1. Transfusion reaction happens: blood vessels leak (anaphylaxis) or red cells explode (hemolytic), dropping effective volume.
2. Saline flows into the vein → raises the amount of liquid in circulation → blood pressure climbs.
3. More volume reaches the kidneys → kidneys filter faster → urine output goes up.
4. Free hemoglobin (toxic red-cell wreckage) gets flushed out in urine before it can jam kidney tubules and cause acute kidney injury.
5. Keeps the IV line open after you stop the transfusion so you still have vein access for emergency drugs.

SAFETY WATCH

Give fast (wide-open or bolus) in destroying red blood cells (hemolytic) and anaphylactic reactions to prevent shock and kidney failure. · Watch for fluid overload if you give many liters, especially in older adults or people with heart failure (listen for crackling lung sounds). · Do NOT use saline to treat TRALI (transfusion-related acute lung injury)—extra fluid makes that worse; TRALI needs oxygen and a breathing machine, not more IV fluid.

Acetaminophen (paracetamol, 650-1 000 mg by mouth or IV) · Antipyretic analgesic (fever and pain reducer)

Acetaminophen lowers the brain's thermostat set-point and blocks pain signals. In feverish (febrile) non-destroying red blood cells (hemolytic) transfusion reactions (FNHTR), the patient's white-cell antibodies attack donor white cells, releasing cytokines (immune alarm chemicals) that tell the hypothalamus (the brain's temperature control center) to crank up body heat. Acetaminophen turns that alarm down, bringing fever and chills under control. It does NOT stop the immune reaction—it just makes the patient comfortable.

1. Patient's anti-white blood cell (leukocyte) (anti-white-cell) antibodies bind donor white cells in the transfused blood.
2. White cells release cytokines (IL-1, IL-6, TNF- α) into the bloodstream.
3. Cytokines reach the hypothalamus → thermostat cranks up → fever, chills, shaking.
4. Acetaminophen inhibits cyclooxygenase (COX) in the brain → less prostaglandin E₂ (the chemical that raises the set-point) → thermostat dials back down.
5. Body temperature falls; chills and aches ease.

SAFETY WATCH

Safe ceiling is 3–4 grams per day in healthy adults; above that risks liver damage. · Many over-the-counter cold and pain pills contain acetaminophen—ask about other meds to avoid accidental double-dosing. · Use cautiously (lower dose) in liver disease or heavy alcohol use. · IV form works faster but costs more; oral is fine if patient can swallow and reaction is mild.

Diphenhydramine (25-50 mg IV or by mouth) · First-generation H₁ antihistamine

Diphenhydramine blocks histamine receptors (the docking ports for histamine, an itch-and-swallow chemical) on blood vessels and skin. In mild allergic transfusion reactions, mast cells release histamine, which makes vessels leak (hives, swelling) and itch. Diphenhydramine plugs

the receptors so histamine cannot land and trigger those symptoms. It calms hives and itch but does NOT reverse severe reactions like anaphylaxis—epinephrine is still first for that.

1. Donor plasma protein or antibody triggers mast cells to dump histamine.
2. Histamine binds H₁ receptors on small blood vessels → vessels leak → raised, red, itchy welts (hives).
3. Diphenhydramine occupies the H₁ receptor → histamine cannot dock → leak and itch signals stop.
4. Existing hives fade; new ones do not form.
5. Crosses into brain → blocks wake-promoting histamine → patient feels sleepy.

SAFETY WATCH

Causes significant drowsiness—warn the patient not to drive or operate machines. · Can cause dry mouth, blurry vision, urinary retention (trouble peeing), especially in older adults. · Avoid in narrow-angle glaucoma and severe prostate enlargement. · IV push can cause a brief blood-pressure drop if given too fast—push over 2 minutes.

Furosemide (20–40 mg IV) — used ONLY in TACO, NOT in TRALI · Loop diuretic (water pill)

Furosemide tells the kidneys to dump salt and water into the urine by blocking the pump (the sodium-potassium-chloride cotransporter) in the loop of Henle (the kidney's recycling tube). In transfusion-associated circulatory overload (TACO), you gave blood faster than the heart could handle, so fluid backs up into the lungs. Furosemide pulls that extra fluid out through the kidneys, shrinking lung congestion and easing breathing. You do NOT use it in TRALI (lung injury from immune attack), because TRALI is NOT a fluid problem—dumping fluid makes TRALI worse.

1. Transfusion given too fast or to a patient with weak heart → blood volume expands beyond what the heart can pump forward.
2. Pressure rises in the lung vessels → fluid squeezes out into air sacs (pulmonary swelling (edema)) → patient cannot breathe, oxygen drops.
3. Furosemide blocks the loop pump → kidneys stop reabsorbing salt → salt and water pour into urine.
4. Urine output jumps → circulating volume shrinks → pressure in lung vessels falls.
5. Fluid reabsorbs out of the air sacs → lungs clear → breathing improves, oxygen climbs.

SAFETY WATCH

Watch potassium—loop diuretics flush potassium out; low potassium can cause dangerous heart rhythms. Check level and replace if needed. · Can drop blood pressure too much if patient is already dry or in shock—use only when you are sure the problem is volume overload. · May worsen kidney function if pushed too hard in someone already dehydrated. · Do NOT give in TRALI—confirming the diagnosis (chest X-ray, timing, BNP level) is critical before giving any diuretic.

Broad-spectrum antibiotics (e.g. vancomycin + ceftriaxone or piperacillin-tazobactam) · Antibacterial agents

If bacteria contaminated the blood unit (septic transfusion reaction), they multiply in the patient's bloodstream and release endotoxin (a poison from bacterial walls) that triggers massive inflammation, fever, and shock. Broad-spectrum antibiotics kill the bacteria by breaking their cell walls (beta-lactams like ceftriaxone, piperacillin-tazobactam) or blocking their wall assembly (vancomycin). You start antibiotics immediately—within the first hour—because septic shock can kill fast, and you choose drugs that cover both Gram-positive (like *Staphylococcus*, common in platelet units stored at room temperature) and Gram-negative bacteria (like *Yersinia*, *Serratia*, which grow in refrigerated red cells).

1. Contaminated unit enters vein → bacteria flood bloodstream → immune system detects endotoxin.
2. Massive cytokine release (TNF- α , IL-1, IL-6) → fever spikes, blood vessels leak, blood pressure crashes (septic shock).
3. Broad-spectrum antibiotics reach bacteria → vancomycin blocks cell-wall cross-linking in Gram-positives; beta-lactams punch holes in Gram-negative walls.
4. Bacteria die → endotoxin release stops → immune storm begins to settle (though damage may still evolve).
5. Supportive care (fluids, vasopressors, oxygen) keeps organs alive while antibiotics clear infection.

SAFETY WATCH

Draw blood cultures from the patient AND culture the implicated unit before starting antibiotics—you need to identify the bug. · Vancomycin requires monitoring of blood levels (trough) and kidney function (can cause nephrotoxicity). · Beta-lactams can cause allergic reactions; ask about penicillin allergy before giving. · Adjust doses in kidney or liver impairment. · If the patient has risk factors or local resistance patterns, infectious-disease consultation helps tailor the regimen.

Norepinephrine (0.05–2 mcg/kg/min IV infusion) · Vasopressor (alpha-1 and beta-1 adrenergic agonist)

Norepinephrine is a powerful vessel-squeezing hormone that raises blood pressure when it has crashed. In severe destroying red blood cells (hemolytic) reactions, anaphylaxis, or septic transfusion reactions, blood vessels relax too much or leak, so blood pressure falls dangerously low and organs (brain, kidneys, heart) do not get enough blood flow. Norepinephrine binds alpha-1 receptors on vessel walls and clamps them tight, lifting pressure. It also nudges the heart to squeeze a bit harder (beta-1 effect). You give it through a central line or large IV because it is so strong that a small peripheral IV can cause tissue damage if it leaks out.

1. Transfusion reaction (hemolysis, anaphylaxis, septic) triggers blood-vessel dilation or leak → blood pressure drops → organs starve for blood (shock).
2. Norepinephrine infusion starts → binds alpha-1 receptors → vessel walls contract → resistance (afterload) rises → blood pressure climbs.
3. Beta-1 stimulation → heart beats harder and a bit faster → cardiac output improves (if heart is not already failing).
4. Blood flow to brain, kidneys, and heart muscle increases → organs begin to recover if you also fix the underlying problem (stop transfusion, give fluids, antibiotics, etc.).

SAFETY WATCH

Must run through a central line or very secure large-bore IV—if it leaks into tissue (extravasation), it can kill the skin and muscle. · Can cause dangerous high blood pressure if dose is too high; monitor blood pressure continuously (arterial line preferred). · Squeezes vessels so tight that fingers, toes, gut, and kidneys can suffer low blood flow—watch urine output and skin color. · Requires intensive-care monitoring; never give on a regular hospital floor without ICU-level oversight. · Wean slowly once blood pressure stabilizes; stopping abruptly can cause rebound crash.

Supplemental oxygen (nasal cannula, high-flow, or mechanical ventilation) · Supportive respiratory therapy (not a drug, but essential medical intervention)

Oxygen is the gas your red blood cells carry to feed every cell. In TRALI (transfusion-related acute lung injury), anaphylaxis, or severe TACO, the lungs fill with fluid or the airways close, so oxygen cannot cross into the blood—your oxygen saturation (O₂ sat) drops. Giving extra oxygen through a mask, nasal prongs, or a breathing tube raises the amount available in the air sacs so more can push across into the bloodstream despite the damage. If that is not enough, a

ventilator (breathing machine) can push air in under pressure to open collapsed sacs and hold them open. Oxygen does NOT fix the lung injury—it buys time while you treat the cause.

1. Transfusion reaction damages lungs (TRALI immune attack, TACO fluid overload, anaphylaxis airway squeeze) → oxygen transfer fails → blood O₂ sat falls → organs starve.
2. Supplemental oxygen flows into nose, mask, or tube → fraction of inspired oxygen (FiO₂) rises from room air (21 %) to 30–100 %.
3. Higher oxygen pressure in alveoli → more oxygen molecules push across the thin membrane into blood → O₂ sat climbs.
4. If lungs are severely injured, positive-pressure ventilation splints airways open and pushes oxygen into harder-to-reach sacs → improves gas exchange.
5. Organs regain oxygen supply → brain clears, heart stabilizes, kidneys keep filtering—while you treat the root problem.

SAFETY WATCH

In COPD (chronic lung disease), very high oxygen can rarely suppress the drive to breathe—monitor closely, but do NOT withhold oxygen in a true emergency. · Prolonged very high FiO₂ (near 100 % for days) can injure lung tissue (oxygen toxicity)—wean to the lowest level that keeps O₂ sat safe (typically ≥ 90–92 %). · Mechanical ventilation carries risks (lung stretch injury, infection, sedation needs)—use only when non-invasive support fails. · Always pair with treatment of the underlying cause; oxygen alone never cures a transfusion reaction.

Red flags

- Blood pressure drops below 90 systolic or falls more than 30 points suddenly — sign of shock
- Trouble breathing, gasping, or oxygen saturation below 90% — may be fluid in the lungs (TRALI) or volume overload
- Dark brown or red urine during or right after transfusion — means red cells are bursting (hemolysis)
- Chest or back pain with fever — can mean acute destroying red blood cells (hemolytic) reaction, which can damage kidneys fast
- Bleeding that won't stop from gums, IV sites, or anywhere — may be DIC from massive cell destruction

Nursing priorities

1. Stop the transfusion immediately at the first sign of reaction and keep the IV line open with new normal saline tubing — Every additional drop of the suspect blood can worsen red blood cell destruction (hemolysis) (red cell destruction), introduce more allergen causing anaphylaxis, or pump more bacteria into the bloodstream; new tubing prevents any remaining donor blood from entering the patient
2. Keep the blood bag, all tubing, and attached labels and send them to the blood bank with a fresh patient blood sample — The lab must compare patient antibodies against donor cells to confirm red blood cell destruction (hemolysis), culture the bag for bacteria, and verify the right unit went to the right patient; this evidence determines if we report to the FDA
3. Monitor vital signs every 5 to 15 minutes including blood pressure, heart rate, oxygen saturation, temperature, and respiratory rate — Falling blood pressure signals shock needing vasopressors; rising fever may mean infection; dropping oxygen saturation shows lung injury or anaphylaxis; rapid changes require immediate escalation to the provider
4. Start or increase IV normal saline to keep urine output at least 1 milliliter per kilogram per hour (roughly 75 to 100 milliliters every hour for an average adult) — Hemolyzed red cells release hemoglobin (the oxygen-carrying protein) that clogs kidney tubules and causes acute kidney injury; fast urine flow washes hemoglobin through before it crystallizes and blocks the kidney filters
5. Measure urine color and send first urine sample to lab for hemoglobin and test blood for signs of red blood cell destruction (hemolysis) including free hemoglobin, haptoglobin (a protein that binds loose hemoglobin), bilirubin (breakdown product), and direct Coombs test (shows antibodies stuck to red cells) — Pink, red, or cola-colored urine proves hemoglobin is spilling into urine from destroyed red cells; lab tests confirm red blood cell destruction (hemolysis) versus other causes of fever and guide whether we continue this blood type in future
6. Draw blood cultures from two different sites on the patient (not from the IV line) and notify provider to culture the blood bag if patient has fever above 38.5 degrees Celsius or rigors — Bacterial contamination of platelets or red cells can cause septic shock; cultures identify the germ so we start the right antibiotic; bag culture proves contamination versus patient infection from another source like a catheter
7. Prepare emergency medications at bedside: epinephrine 0.3 milligrams intramuscular for anaphylaxis, diphenhydramine (an antihistamine) 25 to 50 milligrams IV for allergic symptoms, and acetaminophen for fever after destroying red blood cells (hemolytic) and septic causes are ruled out — Anaphylaxis can obstruct the airway within minutes; having epinephrine ready saves time; antihistamines reduce hives and itching but do not treat anaphylaxis; acetaminophen masks fever so we only give it after we know the reaction is not dangerous

- 8.** Watch for fluid overload signs (crackles in lungs, neck vein swelling, new oxygen need) especially in patients with heart or kidney failure — We give large IV fluid volumes to protect kidneys but too much fluid causes TACO where the lungs fill with water; elderly and heart failure patients cannot pump extra volume so fluid backs up into lungs and blocks oxygen exchange

Sources

- AABB (American Association of Blood Banks) Technical Manual, 21st edition
- SHOT (Serious Hazards of Transfusion) Annual Report 2023
- FDA Blood Safety Guidance 2022
- American Red Cross Transfusion Medicine Guidelines

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