

Shock Classification

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THE STORY IN ONE SENTENCE

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“Shock has four causes: not enough blood left in the tank (hypovolemic), the heart pump broke (cardiogenic), blood vessels opened too wide and leak (distributive), or something blocks the pipes (obstructive). Figure out which one, then fix that broken part.”

Normal anatomy — the body's job

Normal blood flow delivers oxygen and fuel to every organ at exactly the rate each organ needs, and carries away waste. The heart pumps blood forward, blood vessels squeeze or relax to steer flow where it's needed, and there is enough blood volume to fill the system. This balance—blood flow (perfusion) meeting metabolic demand—keeps every organ fed and happy.

BODY'S JOB

The heart is a muscular pump that pushes blood out with each beat. Arteries are strong, stretchy tubes that carry oxygen-rich blood away from the heart to organs. Capillaries are tiny, thin-walled vessels where oxygen and nutrients pass from blood into tissue, and waste moves back into blood. Veins carry blood back to the heart to be pumped to the lungs for fresh oxygen. Blood volume—the total amount of blood in the system—must be enough to fill the vessels so pressure stays steady. The autonomic nervous system (the body's autopilot for things you don't think about) adjusts heart rate, how hard the heart squeezes, and how tight or loose blood vessels are, matching supply to demand second by second. When all parts work together, oxygen reaches every cell, waste is cleared, and organs hum along.

1. The heart squeezes and pushes blood into the aorta (the body's largest artery) with enough pressure to drive flow forward.

2. Arteries branch into smaller arterioles, which squeeze or relax to steer blood toward organs that need more oxygen right now (like muscles during exercise or the gut after a meal).
3. Capillaries—tiny, one-cell-thick vessels—let oxygen and glucose seep out to feed tissue, and pick up carbon dioxide and lactic acid waste.
4. Veins collect used blood and carry it back to the right side of the heart, which pumps it to the lungs to dump carbon dioxide and reload oxygen.
5. Freshly oxygenated blood returns to the left side of the heart, and the cycle repeats, delivering exactly what each organ needs, exactly when it needs it.

What breaks

Shock happens when one of four things breaks the delivery system: the tank runs dry (not enough blood), the pump fails (heart can't squeeze), the pipes spring open too wide (blood vessels dilate and leak), or a blockage clogs the line. Each break has a different fix, so naming which one matters.

CLINICAL TERM

Shock is acute circulatory failure resulting in inadequate tissue blood flow (perfusion) and cellular low oxygen (hypoxia). It is classified by mechanism: hypovolemic (insufficient intravascular volume from bleeding (hemorrhage) or fluid loss), cardiogenic (pump failure from heart attack (myocardial infarction), severe heart failure, or arrhythmia), distributive (pathologic vasodilation and increased capillary permeability, most commonly septic shock from infection, but also anaphylactic or neurogenic), and obstructive (mechanical impediment to flow such as pulmonary embolism (a clot lodging in a lung artery), cardiac tamponade (the heart squeezed by fluid around it), or tension pneumothorax). Clinical recognition rests on low blood pressure (hypotension) (MAP less than 65 mm Hg), a fast heart rate (tachycardia), low urine output (oliguria), altered mental status, elevated lactate (greater than 2 mmol/L), and end-organ dysfunction. The downstream syndrome—lactic acidosis (acid from oxygen-starved tissues), organ failure—is common to all types, but the upstream cause and therefore the treatment differ sharply.

How the body compensates

The body doesn't give up easily. When shock begins, the sympathetic nervous system (the body's fight-or-flight control center) kicks in: it speeds the heart, squeezes blood vessels tight, and shunts blood away from the skin and gut toward the brain and heart. This compensation can keep blood pressure almost normal for a while—hiding how sick the patient really is—until

the system runs out of reserves and crashes suddenly. Your job is to catch the early warning signs before that cliff.

1. The baroreceptors (pressure sensors in the neck arteries and heart) detect falling blood pressure and signal the brainstem.
2. The brainstem activates the sympathetic nervous system, which releases norepinephrine (the body's main squeeze-and-speed signal) and tells the adrenal glands to dump epinephrine (adrenaline) into the blood.
3. The heart rate climbs (tachycardia) to pump more beats per minute, trying to make up for weak flow with speed.
4. Arterioles in the skin, gut, and muscles clamp down tight (vasoconstriction) to raise blood pressure and redirect the limited blood supply to the brain and heart—this is why skin turns cold, pale, and sweaty.
5. The kidneys sense low flow and release renin, which triggers a chain (the renin-angiotensin-aldosterone system) that squeezes vessels further and tells the kidneys to hold onto salt and water, trying to boost blood volume.
6. The pituitary gland releases ADH (antidiuretic hormone, also called vasopressin) to make the kidneys reabsorb even more water and tighten vessels, raising pressure.
7. These tricks can keep the blood pressure and heart rate looking almost stable—sometimes for hours—even as organs quietly run low on oxygen and lactate starts to climb. This is the dangerous compensation phase, because vital signs lie and the team may not realize how close the patient is to collapse.
8. Once the body exhausts its reserves—the heart can't beat any faster, vessels are already maximally squeezed, and there's no more adrenaline to release—compensation fails and the patient crashes: blood pressure drops sharply, heart rhythm becomes chaotic, and multiple organs shut down at once. This decompensation is often sudden and hard to reverse, so the goal is to recognize and treat shock early, during compensation, before the system tips over the edge.

Domino effect

1. Blood flow drops below what organs need, so cells can't get enough oxygen (tissue low oxygen (hypoxia)). — Oxygen is the fuel cells use to make energy (ATP) in their mitochondria (the tiny power plants inside every cell). When oxygen delivery falls—whether from blood loss, a broken pump, leaky vessels, or a blockage—cells can't run their normal energy factory.

2. Cells switch to anaerobic metabolism (backup energy production without oxygen), which makes lactic acid as waste. — Without oxygen, cells break down glucose only partway, producing far less energy and leaving lactic acid behind. Lactate spills into the blood, so serum lactate rises above 2 mmol/L and keeps climbing—this is one of the earliest and most reliable lab clues that shock is happening.
3. The sympathetic nervous system floods the body with norepinephrine and epinephrine (adrenaline), speeding the heart and clamping down skin and gut blood vessels. — The brainstem's survival reflex is to sacrifice non-essential organs (skin, gut, muscles) and funnel every available drop of blood to the brain and heart. This is why skin turns cold, pale, and clammy in hypovolemic, cardiogenic, and obstructive shock—it's the body's tourniquet to protect the core.
4. In distributive (septic) shock, the opposite happens: blood vessels dilate wide open and leak, so skin feels warm and flushed even as pressure drops. — Bacterial toxins and the immune system's inflammatory chemicals (cytokines like TNF-alpha, IL-1, and IL-6) tell vessels to relax and become porous. Blood pools in the skin and leaks fluid into tissues, so the patient looks warm and red but pressure collapses because blood isn't where it needs to be.
5. The kidneys get less blood flow, so they make less urine (oliguria, under 0.5 mL per kilogram per hour), and waste products like creatinine build up in the blood. — The kidneys need steady pressure and flow to filter blood. When flow drops, the kidneys conserve water to try to raise blood volume, but that also means they stop cleaning waste. If shock lasts, acute kidney injury begins—creatinine and potassium rise, and urine output may stop entirely.
6. The brain gets less oxygen, so thinking becomes confused, sluggish, or the patient becomes unresponsive (altered mental status). — The brain uses about 20 percent of the body's oxygen even though it's only 2 percent of body weight. When oxygen delivery falls, brain cells can't fire properly—neurotransmitter signaling (the chemical messages neurons send to each other) weakens, and the patient appears disoriented, drowsy, agitated, or unconscious depending on severity.
7. The gut lining begins to break down, and bacteria or toxins can leak from the intestine into the bloodstream, worsening inflammation and infection. — The gut is one of the first places the body steals blood from during shock. Without oxygen, the intestinal barrier—normally a tight wall—becomes leaky. Bacteria and endotoxins slip into the blood, amplifying the inflammatory cascade and sometimes triggering sepsis even if infection wasn't the original cause (this is called translocation).

8. If shock continues untreated, multiple organs fail at once (multi-organ dysfunction syndrome, MODS): the heart weakens further, lungs fill with fluid (ARDS), liver stops making clotting factors, and the patient may bleed or clot uncontrollably (DIC). — Every organ depends on oxygen. Once the oxygen debt is too large and lactic acidosis (acid from oxygen-starved tissues) is severe, cellular machinery breaks down across the body. The heart can't pump, lungs can't exchange gas, the liver can't detoxify or make proteins, and the clotting system goes haywire because the signals that normally balance clotting and bleeding are lost. At this stage, mortality is very high even with aggressive treatment, which is why early recognition and reversal of shock—before this cascade begins—saves lives.

Symptoms · what the patient feels, and why

- Fast heart rate (tachycardia): The heart beats faster to pump more blood per minute when blood pressure drops or the body needs more oxygen — like a pump speeding up to keep water flowing through a kinked hose.
- Low blood pressure (hypotension): Not enough blood is being pushed through the arteries, either because the heart is too weak, too much blood is lost, the blood vessels are too relaxed and wide open, or something is blocking the flow — like a weak pump or a leak in the system.
- Fast breathing (tachypnea): The body tries to pull in more oxygen and blow off acid that builds up when cells cannot get enough oxygen — like breathing harder when you run uphill.
- Confusion or altered mental status: The brain is not getting enough oxygen-rich blood, so it cannot work properly — like a computer running slowly when the battery is low.
- Cool, pale, clammy skin or mottled (blotchy) skin: The body shuts down blood flow to the skin to save oxygen for the brain and heart; mottling shows the tiny blood vessels are not filling well — like turning off the heat in extra rooms to keep the furnace running.
- Slow capillary refill (when you press the skin, color comes back slowly): Not enough blood is reaching the tiny blood vessels in the skin, so they refill slowly after being squeezed — like a garden hose that fills slowly when the water pressure is low.
- Low urine output (oliguria): The kidneys are not getting enough blood flow to make urine, so they shut down to save what little fluid is left — like a factory that stops production when the power is running low.
- Weak or thready pulse: The heart is pumping with less force or the blood vessels are too wide, so each heartbeat feels faint — like feeling a weak drumbeat through a pillow.

- Flat or collapsed neck veins (in hypovolemic or obstructive shock) or swollen neck veins (in cardiogenic shock): Flat veins mean not enough blood is filling the veins (like an empty water balloon); swollen veins mean blood is backing up because the heart cannot pump it forward (like a clogged drain).
- Warm skin and bounding pulse (in early distributive shock, especially septic shock): The blood vessels open too wide, letting blood flow freely to the skin at first; the pulse feels strong because the heart is pumping hard to keep up — like a fire hose with the valve wide open.

Focused exam

- Skin temperature and color: Cold, pale, clammy skin means the body is pulling blood away from the skin to protect the brain and heart — you see this in bleeding shock (hypovolemic), heart-failure shock (cardiogenic), or blockage shock (obstructive). Warm, flushed skin means the blood vessels are too open and leaky — you see this in infection shock (septic). The skin immediately tells you which kind of shock you are facing.
- Blood pressure: A systolic blood pressure below 90 mmHg (or a drop of more than 40 mmHg from the person's usual pressure) confirms shock is happening. Shock means organs are not getting enough blood flow to do their jobs. You need this number to confirm shock and to track whether treatment is working.
- Jugular veins in the neck: Flat (empty) neck veins mean there is not enough blood in the system — you see this in bleeding shock. Bulging (full) veins mean blood is backing up because the heart cannot pump it forward (heart-failure shock) or something is blocking blood from returning to the heart (blockage shock, such as fluid around the heart or a big lung clot). This one look tells you whether the problem is too little blood or a traffic jam.
- Urine output: Urine output below 0.5 mL per kilogram per hour (for example, less than 30 mL in an hour for a typical adult) means the kidneys are not getting enough blood. The kidneys are very sensitive — when they stop making urine, it is a red flag that shock is severe and organs are shutting down.
- Heart sounds, lung sounds, and belly exam: Listen for crackles (popping sounds) in the lungs — these mean fluid is backing up from a weak heart (heart-failure shock). Listen for muffled, quiet heart sounds — this can mean fluid is squeezing the heart from the outside (cardiac tamponade, a type of blockage shock). Feel the belly for firmness or tenderness — a tight, painful belly with shock can mean bleeding inside. Each finding points you toward the broken part.

- Point-of-care ultrasound (POCUS) of heart, lungs, and belly: POCUS lets you see the heart squeezing (or not squeezing), see if the heart chambers are full or empty, see if fluid is trapped around the heart, see if the lungs have extra fluid or a collapsed area, and see if there is free blood in the belly. You get answers in seconds without waiting for lab results — it is the fastest way to sort bleeding shock (empty heart and veins), heart-failure shock (weak squeeze or full lungs), or blockage shock (fluid around the heart, huge clot in the lung).

Diagnostics · every test answers a question

- Serum lactate → Are the tissues getting enough oxygen, or are cells suffocating and switching to the backup energy system?
- Arterial or venous blood gas (ABG or VBG) → Is the blood too acidic (acidosis), and is the person getting rid of carbon dioxide properly?
- Complete blood count (CBC) → Is the person losing blood, or is there an infection driving the shock?
- Basic metabolic panel (BMP) or comprehensive metabolic panel (CMP) → Are the kidneys failing, and are the body's salts (electrolytes) dangerously out of balance?
- Troponin (high-sensitivity troponin I or T) → Is the heart muscle damaged, suggesting a heart attack is causing or worsening the shock?
- B-type natriuretic peptide (BNP) or N-terminal pro-BNP (NT-proBNP) → Is the heart stretched and failing, backing up fluid into the lungs?
- Blood cultures (two sets from different sites) → Is there bacteria or fungus in the bloodstream causing infection shock?
- 12-lead electrocardiogram (ECG) → Is there a heart attack, a dangerous rhythm, or a clue to a huge lung clot?

Treatment logic

- Identify the shock type using history, exam, POCUS, and labs → Knowing whether the problem is bleeding (hypovolemic), a broken heart pump (cardiogenic), leaky infection vessels (septic), or a blockage (obstructive) tells you which treatment will work. Giving fluid to a bleeding patient helps, but giving fluid to a failing heart can drown the lungs. You must name the upstream break before you can fix it.

- For bleeding shock: stop the bleeding (surgery, endoscopy, angiography with embolization), give blood products in balanced 1:1:1 ratio (packed red cells : fresh frozen plasma : platelets), and give IV crystalloid (normal saline or lactated Ringer's) cautiously → Stopping the source ends the volume loss. Transfusing blood replaces what was lost and restores oxygen-carrying capacity. Balanced ratios prevent dilution of clotting factors. Fluid helps fill the pipes temporarily. Together, these restore blood volume and pressure so organs get oxygen again and lactate drops.
- For heart-pump-failure shock (cardiogenic): emergency catheterization and stenting (PCI) if it is a heart attack, inotropes (dobutamine at 2.5–20 mcg/kg/min or milrinone), vasopressors (norepinephrine) if pressure is very low, and mechanical support (intra-aortic balloon pump, Impella, VA-ECMO) if drugs are not enough → PCI opens the blocked artery feeding the heart, restoring blood flow to dying muscle. Inotropes make the weakened heart squeeze harder (positive inotropy). Vasopressors raise blood pressure by tightening vessels. Mechanical pumps take over part or all of the heart's job, giving it time to recover. Together, these restore forward blood flow to the body.
- For infection shock (septic): give broad-spectrum IV antibiotics within 1 hour of recognizing septic shock (current Surviving Sepsis Campaign guideline), give 30 mL/kg IV crystalloid fluid bolus in the first 3 hours, and start norepinephrine (first-choice vasopressor) to keep mean arterial pressure ≥ 65 mmHg if fluids alone do not work → Antibiotics kill the bacteria or fungus causing the infection. IV fluids fill the leaky, dilated blood vessels. Norepinephrine tightens (constricts) the vessels so pressure comes back up and organs get blood flow despite the leakiness. Together, these control the infection, restore pressure, and improve blood flow (perfusion).
- For blockage shock (obstructive): drain fluid around the heart (pericardiocentesis with needle and catheter) for cardiac tamponade (the heart squeezed by fluid around it), give clot-busting drugs or suction out clots (catheter-directed thrombolysis or embolectomy) for massive pulmonary embolism (a clot lodging in a lung artery), or place a large-bore needle and chest tube for tension pneumothorax → Removing the obstruction lets blood flow freely again. Draining the heart sac lets the heart fill and pump. Breaking up or pulling out lung clots lets the right heart pump blood through the lungs. Releasing air from the chest lets the lung re-expand and lets veins return blood to the heart. Pressure and blood flow (perfusion) come back almost immediately once the blockage is gone.
- Monitor and treat to these targets: lactate dropping toward normal (below 2 mmol/L), mean arterial pressure ≥ 65 mmHg, urine output ≥ 0.5 mL/kg/hr, skin warm with capillary refill under 3 seconds, and mental status clearing → These signs mean organs are getting enough blood and oxygen again. Falling lactate means cells are no longer suffocating and can make energy the normal way. Good urine output means the kidneys are working. Warm skin and fast capillary refill mean blood is reaching the tissues. Clear thinking means the brain is getting oxygen. Hitting these targets tells you the shock is reversing.

Medications

Norepinephrine (Levophed) · Vasopressor (a drug that squeezes blood vessels tight to raise pressure)

Norepinephrine is the first-choice pressure drug for most kinds of shock. It lands on tiny switches in the muscles wrapped around your blood vessels and tells them to squeeze. When the vessels tighten, blood pressure climbs and pushes blood forward to your brain, heart, and kidneys—the organs that need it most.

1. The nurse threads a thick, long IV catheter into a big vein near your collarbone or groin (a central line) and starts a continuous drip of norepinephrine.
2. The drug flows into your bloodstream and travels everywhere.
3. It lands on alpha-1 receptors sitting on the smooth muscle cells that wrap your blood vessels.
4. Those receptors flip on, telling the muscle to contract (squeeze tight).
5. The vessels get narrower, so it's harder for blood to flow through—that resistance is what raises blood pressure.
6. At the same time, norepinephrine tickles beta-1 receptors in your heart, making it pump a little harder and faster.
7. Higher pressure means blood reaches your vital organs even when your body is in shock and struggling.

SAFETY WATCH

If the IV catheter slips out and norepinephrine leaks into the skin, it can choke off blood flow to that spot and kill the tissue, leaving a black sore. That's why it always runs through a central line deep in a big vein, and nurses check the site constantly. · Squeezing vessels too hard can cut blood flow to your fingers, toes, or gut. Watch for cold, pale, or mottled (blotchy) skin. If you see that, tell the team right away. · If you give norepinephrine before filling the tank with IV fluid, pressure may climb on the gauge but your organs still won't get enough blood—like revving an engine with no gas. Always give fluid first in most shock types (except cardiogenic, where the heart can't handle extra fluid). · Norepinephrine can make your heart race or beat irregularly, especially at high doses. The team watches the heart monitor continuously.

Crystalloid fluids (normal saline 0.9% or lactated Ringer's) · Intravenous fluid resuscitation (sterile salt water given through a vein to refill the blood-volume tank)

Crystalloid fluids are sterile salt water that matches the salt level in your blood. In shock, the amount of fluid inside your blood vessels drops—from bleeding, fluid leaking into tissues, or vessels opening too wide. Giving crystalloid refills the tank so your heart has enough volume to pump and deliver oxygen to your organs.

1. A nurse or doctor slides a thick IV catheter into a large vein, often in your arm or groin.
2. Crystalloid is pushed in fast—usually 500 to 1,000 milliliters (about 2 to 4 cups) over 15 minutes. This is called a bolus.
3. The fluid pours into your bloodstream and increases the total volume of blood.
4. More volume means your heart chambers fill better between beats (this filling pressure is called preload).
5. A fuller heart squeezes out more blood with each beat, so cardiac output (total blood pumped per minute) goes up.
6. Blood pressure rises and organs get more oxygen.
7. About one-third of the fluid stays in your vessels; the rest leaks into the spaces between cells within an hour, so you may need another bolus if pressure drops again.

SAFETY WATCH

In cardiogenic shock (when your heart is too weak to pump), pouring in too much fluid can flood and drown your lungs. The team gives small amounts slowly and watches closely; often you need less fluid and more medicines that help the heart squeeze. · Large volumes of normal saline (0.9%) can make your blood too acidic (a problem called hyperchloremic metabolic acidosis (acid building up in the blood)) and harm your kidneys. Lactated Ringer's solution is often safer for big-volume resuscitations because it has a buffer (lactate) that the liver turns into bicarbonate. · Watch for fluid overload: new trouble breathing, wet crackling sounds in the lungs (rales), swelling in your legs or belly, or dropping oxygen levels. If you see these, stop the fluids and tell the team immediately. · Always reassess after each bolus. If your blood pressure and urine output don't improve after 2 liters, the problem may not be low volume—look for ongoing bleeding, heart failure, or another hidden cause.

Packed red blood cells (PRBCs) · Blood product transfusion (donated red cells that carry oxygen to tissues)

Packed red blood cells are the oxygen-carrying part of donated blood, with most of the liquid (plasma) removed. In bleeding-driven (hemorrhagic) shock (bleeding shock), you lose both

volume and the cells that deliver oxygen. Giving PRBCs replaces those cells so your tissues can breathe again.

1. The blood bank checks your blood type and cross-matches donated units to make sure they're safe for you.
2. A nurse hangs the unit of packed red cells and runs it into a large IV, often while also giving plasma and platelets at the same time.
3. Red cells enter your circulation and travel to your lungs.
4. Hemoglobin molecules inside the cells pick up oxygen from the air you breathe.
5. Red cells flow through arteries to your organs and release oxygen where it's needed.
6. This raises your hemoglobin level and oxygen delivery (the amount of oxygen reaching tissues every minute), helping organs recover from shock.

SAFETY WATCH

Transfusion reactions: sudden fever, shaking chills, back or chest pain, trouble breathing, or a sudden drop in blood pressure. Stop the transfusion immediately, keep the IV open with saline, and call the doctor—this could be a serious immune reaction. · TACO (transfusion-associated circulatory overload): too much volume too fast can flood the lungs, causing shortness of breath, low oxygen, and wet sounds (crackles) when you listen to the chest. This is especially risky in older patients or anyone with heart or kidney problems. Give each unit slowly—over 2 to 4 hours—unless massive bleeding requires faster infusion with a pressure bag. · TRALI (transfusion-related acute lung injury): rare but serious—new trouble breathing and low oxygen within 6 hours of transfusion, caused by donor antibodies attacking your lungs. Stop the transfusion and support breathing with oxygen or a ventilator. · In massive transfusion (many units quickly), watch for low calcium (citrate preservative in stored blood binds calcium), low body temperature (refrigerated blood is cold), high potassium (stored cells leak potassium), and dilution of your own clotting factors. The team warms blood when possible, checks labs frequently, gives calcium and platelets and plasma as needed, and may even give you a warming blanket.

Fresh frozen plasma (FFP) · Blood product transfusion (the liquid part of blood that contains clotting proteins)

Fresh frozen plasma is the clear, straw-colored, protein-rich liquid part of donated blood. It carries clotting factors—proteins that work together in a chain reaction (the clotting cascade) to build clots and stop bleeding. In bleeding-driven (hemorrhagic) shock with massive

bleeding, you lose or dilute these factors. Giving FFP replaces them so clots can form and bleeding slows.

1. The blood bank thaws a bag of frozen plasma (stored at -18°C or colder) in a warm-water bath and checks that it matches your blood type.
2. Plasma is infused through a large IV, usually at the same time as red cells and platelets in a 1:1:1 ratio for massive bleeding (one unit of each).
3. Clotting factors—such as factor VIII, fibrinogen, and prothrombin—enter your bloodstream.
4. When a blood vessel is damaged, these factors activate in sequence, each one turning on the next, like dominoes falling.
5. The cascade ends with an enzyme (thrombin) chopping fibrinogen into sticky threads called fibrin.
6. Fibrin threads weave a mesh over the platelet plug, hardening into a stable clot that seals the hole and stops blood loss.

SAFETY WATCH

TACO (fluid overload): plasma is liquid, so it adds volume. Watch for new shortness of breath, wet crackling sounds in the lungs, and dropping oxygen. Slow or stop the infusion and support breathing. · TRALI (transfusion-related acute lung injury): rare sudden trouble breathing and low oxygen within a few hours of plasma transfusion. Stop immediately and notify the team; the patient may need a ventilator. · Allergic reactions: hives, itching, flushing, or (very rarely) severe throat swelling and anaphylactic shock. Stop the plasma, give antihistamines like diphenhydramine, and if severe give epinephrine immediately. · Citrate toxicity in massive transfusion: stored plasma contains citrate (a chemical preservative that binds calcium to keep blood from clotting in the bag). If you get many units quickly, citrate pulls your blood calcium dangerously low. Low calcium weakens your heart, causes muscle twitching or tingling around the mouth, and drops blood pressure. The team checks ionized calcium levels and replaces calcium through the IV as needed.

Platelets · Blood product transfusion (tiny sticky cell fragments that plug holes in blood vessels and start clots)

Platelets are small pieces of cells (made by megakaryocytes in your bone marrow) that patrol your bloodstream looking for damage. When a blood vessel tears, platelets rush to the injury, stick to the torn edges, clump together, and form a plug—the first seal. In massive bleeding, your platelet count drops (diluted by IV fluids and transfusions, or used up by clotting). Giving platelets restores the supply so clots can form and bleeding stops.

1. The blood bank pools platelets from several donors (or collects them from one donor with a special machine) and delivers them to the bedside at room temperature.

2. Platelets are infused quickly through a large IV—usually one unit over 15 to 30 minutes.
3. They enter your bloodstream and circulate, ready to respond.
4. When a blood vessel is cut or torn, platelets sense chemicals released from the injury (like collagen and von Willebrand factor).
5. Platelets activate: they change from smooth discs into spiky spheres, get sticky, and clump at the injury site. This clumping is called platelet aggregation.
6. The platelet plug provides the first seal; then clotting factors from plasma weave a stronger fibrin net on top to lock it in place.

SAFETY WATCH

Transfusion reactions: sudden fever, shaking chills, or (rarely) severe allergic reaction with hives, wheezing, or trouble breathing. Stop the transfusion immediately and notify the team. · TRALI (transfusion-related acute lung injury): rare sudden shortness of breath and low oxygen, usually within 6 hours. Stop the platelet transfusion and support breathing with oxygen or a ventilator. · Bacterial contamination: platelets are stored at room temperature (20–24°C), which is warm enough for bacteria to grow if any snuck into the bag during collection. Fever, shaking chills, or a spike in heart rate during the infusion may signal a contaminated unit. Stop immediately, send blood cultures, and start antibiotics. · Platelet refractoriness: in patients who have had many transfusions or pregnancies, antibodies can attack and destroy donor platelets within minutes. The platelet count may not rise after transfusion. The team may need to order special HLA-matched platelets (matched to your immune type) or single-donor platelets.

Epinephrine (adrenaline) · Vasopressor and bronchodilator (a drug that squeezes blood vessels, strengthens the heartbeat, and opens airways)

Epinephrine is your body's own emergency hormone—the chemical behind the fight-or-flight response. It works on multiple switches at once: it tightens blood vessels to raise pressure, makes your heart beat faster and harder, and relaxes the muscles around your airways so you can breathe. In anaphylactic shock (a severe life-threatening allergic reaction), blood vessels open wide, airways squeeze shut, and pressure crashes. Epinephrine reverses all three problems at once.

1. In anaphylaxis, you inject epinephrine into the big muscle of your outer thigh using an auto-injector pen (like an EpiPen) or the team draws it up in a syringe and gives it as a shot. The thigh muscle has good blood flow, so the drug absorbs quickly.
2. Epinephrine enters your bloodstream within seconds to minutes.
3. It hits alpha-1 receptors on vessel walls and tells smooth muscle to contract, raising blood pressure.

4. It hits beta-1 receptors in your heart, making it pump faster and squeeze harder, which boosts cardiac output.
5. It hits beta-2 receptors in your lungs, relaxing the tight muscles around airways so air can flow in and out again.
6. It also lands on mast cells and shuts down further release of histamine and other allergy chemicals, slowing the allergic cascade.
7. Within 5 to 10 minutes, blood pressure climbs, wheezing eases, airways open, throat swelling shrinks, and the feeling of impending doom lifts.

SAFETY WATCH

Epinephrine makes your heart race and can cause pounding heartbeat (palpitations), chest tightness, or even a heart attack, especially in older patients or anyone with coronary artery disease. Even so, in true anaphylaxis the benefit far outweighs the risk—delaying epinephrine kills more people than the drug's side effects do. · Tremor (shaking hands), anxiety, pale skin, and sweating are common and usually not dangerous. They fade as the drug wears off. · If you accidentally inject epinephrine into a vein instead of muscle, blood pressure can spike dangerously high and cause a stroke, bleeding in the brain, or dangerous heart rhythms. Always give intramuscular (IM) into the thigh, never intravenous (IV) unless you're in a hospital with continuous monitoring in the ICU. · After giving epinephrine, check blood pressure and heart rate every 2 to 5 minutes. If anaphylaxis is severe or the patient doesn't respond, the team may start a continuous epinephrine IV drip in the intensive care unit with close monitoring.

Dobutamine · Inotrope (a drug that makes the heart muscle squeeze harder with each beat)

Dobutamine works mainly on beta-1 receptors scattered across your heart muscle. It tells the muscle cells to contract more forcefully, so each beat pumps out more blood. In cardiogenic shock (when your heart is too weak or damaged to pump enough blood), dobutamine boosts the pump's strength without squeezing blood vessels as much as other pressure drugs do.

1. Dobutamine is given as a continuous drip through a central IV line (a thick catheter in a large vein near your collarbone or groin). The dose is adjusted up or down based on how your heart responds.
2. The drug travels through your bloodstream to your heart and lands on beta-1 receptors on the muscle cells.
3. The receptors activate a chemical messenger inside the cell called cyclic AMP (cAMP).
4. More cAMP opens calcium channels in the cell membrane, flooding the inside of the cell with calcium ions.

5. Calcium makes the tiny protein fibers (actin and myosin) inside the muscle slide past each other harder and faster, so the heart squeezes more powerfully.
6. Stroke volume (the amount of blood ejected with each beat) goes up, and cardiac output (total blood pumped per minute) rises.
7. More blood reaches your organs, delivering the oxygen they need to recover.

SAFETY WATCH

Dobutamine forces the heart to work harder and beat faster, which increases how much oxygen the heart muscle needs. In a heart that's already starved for oxygen (ischemia from blocked arteries), this extra demand can trigger chest pain (angina), dangerous heart rhythms (arrhythmias), or even expand the area of dead muscle from a heart attack. Use carefully and watch the EKG (heart tracing) continuously. · Dobutamine often causes the heart to race (tachycardia—heart rate over 100) or skip beats. Dangerous rhythms like ventricular a fast heart rate (tachycardia) (fast chaotic beating from the lower chambers) can happen, especially at higher doses. The team watches the monitor constantly and may need to lower the dose or stop the drug. · Blood pressure may drop instead of rising, especially if the blood vessels relax. You may need to add a vessel-squeezing drug like norepinephrine at the same time to keep pressure in a safe range. · Do NOT stop dobutamine suddenly—the heart can rebound into worse failure. The team slowly lowers (weans) the dose over hours to days as your heart recovers.

Milrinone · Inotrope and vasodilator (a drug that strengthens the heart's squeeze and relaxes blood vessels at the same time)

Milrinone blocks an enzyme called phosphodiesterase-3 (PDE-3) inside heart muscle cells and the smooth muscle cells that wrap blood vessels. Blocking this enzyme raises the level of cyclic AMP (cAMP), a chemical messenger. In the heart, more cAMP means stronger contraction. In blood vessels, more cAMP means the muscles relax and the vessels open wider. The result: your heart pumps harder and blood flows forward more easily because resistance drops.

1. Milrinone is given as a continuous IV drip, often starting with a small loading dose pushed over 10 minutes, then a slower maintenance drip.
2. The drug enters muscle cells in the heart and blood vessels and blocks the PDE-3 enzyme.
3. Blocking PDE-3 stops the breakdown of cAMP, so cAMP levels climb inside the cells.
4. In heart muscle, higher cAMP opens calcium channels and calcium floods in, making the muscle fibers contract harder (positive inotropy).
5. In vessel walls, higher cAMP relaxes the smooth muscle, so arteries and veins widen (vasodilation).

6. The combination increases stroke volume (blood per beat) and lowers afterload (the resistance the heart must pump against), improving forward blood flow and oxygen delivery.

SAFETY WATCH

Milrinone often drops blood pressure because it relaxes blood vessels. The team watches pressure closely and usually adds a vasoconstrictor like norepinephrine running at the same time if pressure falls too low. · Milrinone can trigger dangerous heart rhythms, especially fast chaotic rhythms from the ventricles (ventricular arrhythmias like V-tach). The heart monitor stays on continuously, and the team may need to lower the dose or stop the drug if rhythms become unsafe. · Milrinone is cleared from your body by your kidneys. In patients with kidney failure, the drug can build up to toxic levels. The team lowers the dose and checks your kidney function (creatinine and GFR) regularly. · Do NOT stop milrinone suddenly—taper the dose slowly over hours to avoid rebound worsening of heart failure. · Long-term use of milrinone outside the hospital has been linked to higher death rates in people with chronic heart failure, so it is used only short-term for acute cardiogenic shock in the ICU, never as a go-home maintenance drug.

Tranexamic acid (TXA) · Antifibrinolytic (a drug that stops clots from breaking down too soon)

Tranexamic acid blocks plasmin, an enzyme that dissolves clots. Normally, after a clot does its job sealing an injury, plasmin slowly breaks it down so blood can flow freely again. But in trauma or massive bleeding, the body sometimes activates plasmin too much and chews up clots before they can seal the wound, making bleeding worse. TXA slows that breakdown, giving clots time to hold and bleeding time to stop.

1. In trauma protocols, TXA is given as a slow IV push—1 gram over 10 minutes—followed by a continuous drip of 1 gram over 8 hours.
2. TXA travels through your blood and attaches to plasminogen molecules floating in the plasma.
3. By binding plasminogen, TXA blocks it from turning into active plasmin.
4. Without plasmin, the fibrin mesh (the net of sticky threads holding the clot together) stays intact instead of dissolving.
5. Clots remain stable longer, reducing total blood loss.
6. Large trauma studies (like the CRASH-2 trial) show that TXA lowers death from bleeding in trauma patients, but only if given within 3 hours of injury.

SAFETY WATCH

Seizures: rare but serious, especially with high doses or in patients with traumatic brain injury. Watch for muscle twitching, staring spells, or full convulsions. If seizures happen, stop TXA and give seizure medicine like lorazepam or levetiracetam. · Clots in the wrong place (thrombosis): TXA can increase the risk of deep vein clots in the legs (DVT), clots traveling to the lungs (pulmonary a traveling clot lodging in a vessel (embolism), PE), or stroke, especially if given late or in patients already at high risk for clotting. Use only when there is active major bleeding. · Do NOT give TXA more than 3 hours after trauma. The CRASH-2 trial showed that late TXA (after 3 hours) increases death from bleeding rather than reducing it—possibly because it locks in clots forming in the wrong places. · Mild side effects like nausea, vomiting, or diarrhea can occur but usually aren't dangerous and go away on their own.

Calcium chloride or calcium gluconate · Electrolyte replacement (a mineral supplement that restores calcium levels in the blood)

Calcium is essential for three big jobs: muscle contraction (including the heart), nerve signaling, and blood clotting. In massive transfusion, the citrate preservative in stored blood binds calcium and pulls the level dangerously low (hypocalcemia). Low calcium weakens the heart so it can't squeeze well, causes muscles to twitch or cramp, and slows clot formation. Giving calcium replaces what citrate steals and keeps all three systems working.

1. During massive transfusion, the team checks your ionized calcium level with a blood gas machine (a quick test on a drop of arterial blood).
2. If calcium is low (usually below 1.0 mmol/L or 4 mg/dL), the team pushes calcium chloride (a 10% solution, usually 1 gram in a 10-mL syringe) or calcium gluconate slowly through a central IV line.
3. Calcium enters your bloodstream. Some binds to proteins like albumin; the rest stays free as ionized calcium.
4. Ionized calcium is the active form that muscles, nerves, and clotting factors use.
5. Heart muscle contraction strengthens, blood pressure may improve, the heart rhythm steadies, and clotting factors work better to build solid clots.
6. The team repeats ionized calcium checks every 30 to 60 minutes during ongoing massive transfusion and gives more calcium as needed.

SAFETY WATCH

Calcium chloride is caustic—it burns and kills tissue on contact. It **MUST** go through a central line (a thick catheter in a large vein deep in the chest or groin), never a small peripheral IV in the hand or arm, or it can cause severe tissue death (necrosis) and a black, painful sore if it leaks. · Pushing calcium too fast can cause a slow heart rate (bradycardia) (dangerously slow heart rate), heart rhythm problems (arrhythmias), or even cardiac arrest (the heart stops). Always give slowly—over at least 5 to 10 minutes—and watch the heart monitor continuously. · Do **NOT** mix calcium in the same IV tubing as blood products—calcium can react with the citrate in the bag and cause clots to form right in the line, blocking the tubing. Use a separate IV line or flush the line with saline between the blood and the calcium. · In patients taking digoxin (a heart drug that strengthens contraction), high calcium levels can trigger dangerous rhythms like ventricular a fast heart rate (tachycardia) or ventricular fibrillation. Use caution, give calcium slowly, and monitor the heart rhythm closely.

Sodium bicarbonate · Alkalinizing agent (a drug that raises blood pH and buffers excess acid)

Sodium bicarbonate is baking soda for your blood. In severe shock, your tissues don't get enough oxygen, so cells switch to anaerobic metabolism (making energy without oxygen). This process produces lactic acid as waste. Acid builds up in the blood, lowering pH (a condition called metabolic acidosis (acid building up in the blood)). Severe too much acid in the blood (acidosis) weakens the heart, blocks pressure drugs from working, damages organs, and worsens shock. Bicarbonate neutralizes acid and raises pH back toward normal, helping your heart and other treatments work better.

1. The team draws an arterial blood gas (ABG) and sees that your pH is dangerously low—usually under 7.1—and bicarbonate level is very low, confirming severe metabolic acidosis (acid building up in the blood).
2. Sodium bicarbonate (usually 50 to 100 milliequivalents, or mEq) is given as a slow IV push over several minutes, or mixed into IV fluids and dripped in.
3. Bicarbonate ions (HCO_3^-) enter your bloodstream and seek out hydrogen ions (H^+).
4. The two react to form carbonic acid (H_2CO_3), which immediately breaks down into water (H_2O) and carbon dioxide (CO_2).
5. Your lungs blow off the CO_2 when you exhale, removing it from your body.
6. With less acid (fewer H^+ ions), your blood pH rises back toward the normal range (7.35–7.45).
7. As pH improves, your heart muscle contracts better and vasopressor drugs like norepinephrine regain their ability to squeeze vessels and raise blood pressure.

SAFETY WATCH

Bicarbonate generates carbon dioxide (CO₂). If you can't breathe well on your own or you're not on a ventilator, CO₂ builds up in your blood and can make too much acid in the blood (acidosis) worse in your brain (paradoxical central nervous system too much acid in the blood), causing confusion, drowsiness, or even coma. Make sure you have good ventilation—either breathing deeply on your own or on a ventilator—before giving bicarbonate.

- Sodium overload: bicarbonate is a sodium salt, so each dose delivers a large sodium load. This can cause fluid overload (too much volume in your bloodstream), high blood sodium (hypernatremia), and fluid leaking into the lungs (pulmonary swelling (edema)). Use cautiously in patients with heart failure or kidney failure.
- Too little acid in the blood (Alkalosis) overshoot: too much bicarbonate can swing your pH too high (alkalemia, pH above 7.45). High pH shifts the oxygen-hemoglobin curve to the left, making it harder for tissues to grab oxygen from red blood cells. It can also cause low potassium (hypokalemia), muscle cramps, and seizures.
- Bicarbonate can inactivate vasopressor drugs like epinephrine, norepinephrine, and dopamine if mixed in the same IV line. Always use a separate line or flush thoroughly with saline between drugs.
- Bicarbonate use in shock is controversial and NOT routine. The 2010 AHA guidelines and most intensivists reserve it only for severe metabolic acidosis (acid building up in the blood) (pH below 7.1) that persists despite treating the root cause. It should never replace definitive treatment.

Vasopressin · Vasopressor (a natural hormone that tightens blood vessels and raises blood pressure)

Vasopressin (also called antidiuretic hormone, or ADH) is a hormone your body releases from the pituitary gland (a pea-sized gland at the base of your brain) to hold onto water and raise blood pressure during stress. It works on V1 receptors on blood vessel walls, telling the smooth muscle wrapped around vessels to squeeze. Unlike norepinephrine, vasopressin does NOT depend on adrenergic receptors (alpha and beta switches), so it can still work when those receptors are exhausted or down-regulated—common in septic shock when the body has been flooded with its own adrenaline-like chemicals for hours.

1. Vasopressin is given as a continuous IV infusion through a central line, usually at a fixed low dose: 0.03 to 0.04 units per minute. (Unlike other vasopressors, you do NOT increase the dose; it works best at this set rate.)
2. The drug travels through your bloodstream to blood vessel walls and binds V1 receptors on smooth muscle cells.
3. The V1 receptor activates a pathway inside the cell that raises calcium levels.
4. Higher calcium makes the smooth muscle fibers contract and squeeze the vessel narrower.

5. Vascular resistance climbs (it's harder for blood to flow through the narrowed vessels), and blood pressure rises.
6. Vasopressin also has a catecholamine-sparing effect: it lets you lower the dose of norepinephrine while keeping pressure stable, which may reduce side effects from high-dose norepinephrine.

SAFETY WATCH

Excessive vasoconstriction: vasopressin can squeeze vessels so hard that blood flow is choked off to the gut (intestines), skin, fingers and toes, or even the coronary arteries feeding the heart muscle. Watch for severe abdominal pain (may signal gut starved blood flow (ischemia) or dead bowel), cold pale or mottled (blotchy) skin, blue or black fingers or toes, or new chest pain (may signal reduced blood flow to the heart). If you see these, the team may need to lower or stop vasopressin. · Low sodium (hyponatremia): through V2 receptors in the kidneys, vasopressin promotes water retention, which dilutes sodium in your blood. The team checks sodium levels regularly, especially during prolonged infusions. · Low platelet count (thrombocytopenia) has been reported in some patients on vasopressin, though it's uncommon. · Do NOT give vasopressin as a bolus or fast push—it must always be a slow continuous infusion. A bolus can cause severe coronary artery spasm (sudden choking of the arteries feeding the heart) and cardiac arrest. · Use with caution in cardiogenic shock or any condition where reducing blood flow to the heart could make things worse. Vasopressin is used most often in septic or distributive shock, less often in cardiogenic shock.

Red flags

- Blood pressure stays low (systolic under 90) even after giving fluids
- Confusion, no response, or cannot wake the patient up
- Urine output drops to almost nothing (under 0.5 mL per kg per hour)
- Skin turns mottled (blotchy purple) or stays cold and pale
- Heart rate stays very fast (over 120) or becomes very slow and irregular

Nursing priorities

1. Get two large-bore IV lines (18-gauge or bigger) in place immediately and prepare for fluid bolus or blood transfusion — Shock means cells are starving for oxygen; large IVs let you push fluids or blood fast to raise blood pressure and deliver oxygen to organs before they die

2. Start continuous monitoring with blood pressure cuff cycling every 3 to 5 minutes, pulse oximeter, cardiac monitor, and place Foley catheter to measure urine output every hour — MAP below 65 means organs are not getting enough blood; urine output below 0.5 mL per kg per hour means kidneys are shutting down; heart rhythm changes show if shock is from heart problem
3. Draw blood for lactate, complete blood count, type and cross-match, troponin, blood cultures before antibiotics, and basic metabolic panel — Lactate above 2 means cells are using backup energy without oxygen; hemoglobin shows bleeding; troponin shows heart attack causing cardiogenic shock; cultures guide antibiotics for sepsis
4. Perform or assist with POCUS ultrasound to check IVC collapsibility, heart squeeze strength, fluid around heart, and lung sliding — Flat collapsing IVC means patient needs fluids; weak heart squeeze means cardiogenic shock needing inotropes not fluids; fluid around heart means tamponade needing drain; no lung sliding means pneumothorax
5. Assess skin temperature, capillary refill, mottling pattern, and mental status every 15 minutes — Cold clammy skin and capillary refill over 3 seconds means blood is shunting away from skin to protect brain and heart; mottling and confusion mean shock is getting worse and organs are failing
6. Administer oxygen to keep saturation above 94% and prepare for intubation if respiratory rate over 30 or patient cannot protect airway — Shock makes lungs fill with fluid and muscles tire out; oxygen helps but intubation with ventilator support may be needed before respiratory arrest happens
7. Give antibiotics within one hour if fever or infection suspected, stop any bleeding source with pressure or call surgery, or prepare for cath lab if chest pain present — Each hour delay in sepsis antibiotics increases death risk by 7%; uncontrolled bleeding kills in minutes; heart attack causing cardiogenic shock needs artery opened within 90 minutes
8. Notify provider immediately for orders on fluid type and rate, vasopressor choice and dose, blood product transfusion, and consults for ICU, surgery, or cardiology — Different shock types need different treatments; giving fluids to cardiogenic shock drowns the patient; missing sepsis antibiotics or surgery for bleeding increases death; decisions must be made in minutes

Sources

- AHA/ACC 2017 Shock and Hemodynamic Support Guideline
- Surviving Sepsis Campaign 2021
- NEJM Clinical Practice (Shock)
- Cochrane Reviews on Fluid Resuscitation

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