

Cardiogenic Shock

A plain-English guide for you and your family

WHAT IS HAPPENING

Your heart is a pump made of muscle. The left side pumps oxygen-rich blood out to your whole body—your brain, kidneys, liver, arms, and legs. The right side pumps blood through your lungs to pick up oxygen. Every minute, your heart has to push enough blood forward so every organ gets the fuel it needs to work. In cardiogenic shock, something has badly damaged or broken the heart muscle, so it can no longer squeeze hard enough to push blood forward. The most common cause is a large heart attack that kills a big area of the left ventricle (the main pumping chamber). Other causes include a torn valve inside the heart (like a mitral valve that suddenly leaks backward), a hole that rips open between the two sides of the heart after a heart attack (ventricular septal rupture), severe infection of the heart muscle (fulminant myocarditis), or very advanced heart failure that suddenly gets much worse. Because the pump is failing, blood pressure drops and organs begin to starve. Your body tries to save itself: it squeezes blood vessels tight, speeds up your heart, and releases stress hormones to keep blood flowing to your brain and heart—even if that means stealing blood from your skin, arms, legs, and kidneys. That is why your hands and feet turn cold and sweaty. Your muscles and organs, not getting enough oxygen, switch to a backup energy system that produces a waste chemical called lactate, which builds up in your blood. One by one, your kidneys, liver, and brain start to fail because they are not getting the fuel they need. Every strange sign—cold skin, confusion, rising lactate, falling urine output—is your body fighting for survival. The only way to stop the spiral is to fix or support the broken pump, open any blocked arteries quickly, and give medicines or devices that help the heart push blood forward again. Without fast treatment, cardiogenic shock leads to death within hours.

What you might feel

- Blood pressure drops very low (top number under 90)
- Fast, weak pulse
- Skin feels cold, clammy, and looks pale or bluish (especially fingers, toes, lips)
- Breathing feels fast and hard, or the person is gasping for air
- Confusion, sluggish thinking, or loss of consciousness

- Urine output drops to almost nothing (less than half a cup per day for an adult)
- Lactate level in the blood is high (above 2 mmol/L on a blood test)
- Neck veins look swollen or bulging
- Heart sounds like it's racing or beating irregularly
- Legs or belly swell with fluid

Call the doctor right away if...

- Top blood pressure number stays below 90 even after giving fluids through an IV
- Person is confused, very drowsy, or unresponsive
- Skin is cold, sweaty, and gray or blue
- Urine output stops or drops to almost nothing for several hours
- Breathing is extremely fast, labored, or the person is gasping and can't speak in full sentences

What the care team will do

- Start continuous cardiac monitoring and measure blood pressure every 5 to 15 minutes using an automatic cuff or place an arterial line (a thin tube in the wrist artery for second-by-second pressure readings).
- Place two large-bore IV catheters (thick needles) in separate arm veins and start norepinephrine infusion immediately, titrating (adjusting) the rate to keep systolic blood pressure at least 80 to 90 mmHg.
- Measure urine output every hour using a Foley catheter (a tube placed into the bladder) and report immediately if output drops below 0.5 milliliters per kilogram per hour (about 30 milliliters per hour for an average adult).
- Give high-flow oxygen through a non-rebreather mask (a mask with a reservoir bag) or prepare for non-invasive positive pressure ventilation (BiPAP) or intubation (breathing tube) if oxygen saturation stays below 90 percent or the patient is gasping.
- Assess skin color, temperature, and capillary refill (press a fingernail and count seconds until pink returns—normal is under 2 seconds) every 15 to 30 minutes, and report cold, sweaty, gray, or blue skin immediately.
- Check mental status every 15 minutes using simple questions (name, date, location) and report any confusion, sleepiness, or failure to respond to voice.
- Restrict IV fluids to small boluses (250 to 500 milliliters) only if central venous pressure (CVP) is low (under 8 mmHg), and stop fluids immediately if lung sounds become wet or crackly or oxygen levels drop.

- Prepare the patient for emergency cardiac catheterization by ensuring nothing by mouth (NPO), checking allergies to contrast dye and shellfish, and confirming IV access for sedation and dye injection.

Medicines you may be given — and why

Norepinephrine

Norepinephrine grabs onto alpha-1 receptors (lock-and-key spots on the outside of smooth muscle cells that wrap around blood vessels). When it locks on, those muscle cells squeeze tight—like clenching a fist around a garden hose—and the vessel gets narrower. Narrower pipes mean higher pressure, so blood reaches the brain, heart, and kidneys even when the heart itself is too weak to pump well. Norepinephrine also tickles beta-1 receptors on the heart muscle, making it contract a bit harder and faster.

Dobutamine

Dobutamine tickles beta-1 receptors on heart muscle cells, telling them to contract with more force. It also gently nudges beta-2 receptors in blood vessel walls, making vessels relax a little. The net effect is a stronger heartbeat and slightly wider pipes, so more blood moves forward with each pump—helpful when the heart is too weak but blood pressure is not dangerously low.

Milrinone

Milrinone blocks phosphodiesterase-3 (an enzyme that normally breaks down cAMP, a messenger molecule inside cells). When cAMP builds up, heart muscle contracts harder and blood vessels relax. The heart ejects more blood per beat, and resistance drops, so the heart doesn't have to push as hard. This combination can help when other inotropes aren't enough—but only if blood pressure is supported.

Epinephrine

Epinephrine hits alpha-1, beta-1, and beta-2 receptors all over the body. It tightens blood vessels hard (raising pressure), makes the heart beat much harder and faster, and opens airways in the lungs. It is a last-resort drug—used only when norepinephrine alone isn't keeping the patient alive—because it dramatically increases the heart's oxygen demand and arrhythmia risk.

Furosemide (Lasix)

Furosemide blocks the sodium-potassium-chloride cotransporter (a pump that normally pulls salt back into the blood) in the thick ascending limb of the loop of Henle (a hairpin turn deep in the kidney tubule). When salt stays in the urine, water follows it out by osmosis. The patient pees more, removing extra fluid from the lungs and body. In cardiogenic shock, only small,

careful doses are used—too much diuresis drops blood pressure further and can shut down the kidneys.

Aspirin

Aspirin permanently blocks cyclooxygenase-1 (COX-1), an enzyme inside platelets that makes thromboxane A2—a powerful chemical signal that tells platelets to clump and stick together. Without thromboxane A2, platelets become much less sticky. This prevents new clots from forming or growing in coronary arteries, which is critical if the cardiogenic shock is caused by acute heart attack (myocardial infarction) (heart attack).

Heparin (unfractionated) or Enoxaparin (low-molecular-weight heparin)

Heparin binds to antithrombin III (a natural protein in the blood that shuts down clotting factors) and changes its shape, making it work thousands of times faster. Supercharged antithrombin inactivates thrombin (the enzyme that converts fibrinogen into fibrin clot strands) and factor Xa (a key enzyme earlier in the clotting cascade). This prevents new clots from forming in coronary arteries, in mechanical support devices, and elsewhere—buying time for stents, surgery, or other definitive therapy.

Morphine

Morphine binds to mu-opioid receptors in the brain (periaqueductal gray, limbic system) and spinal cord (dorsal horn), blocking pain signals and calming fear and air hunger (the terrifying feeling of suffocating). It also causes mild venodilation (widening of veins), which pools blood in the periphery, reducing venous return (preload) to the heart and slightly lowering lung congestion. In severe pulmonary edema (fluid flooding the lungs) with cardiogenic shock, tiny doses can ease distress—but it must be used extremely cautiously because it can drop blood pressure and suppress breathing.

Where this information comes from

- AHA/ACC 2022 Heart Failure Guideline
- AHA 2020 Advanced Cardiovascular Life Support (ACLS)
- NEJM Cardiogenic Shock Review
- SHOCK Trial and CULPRIT-SHOCK Trial

Last reviewed: 2026-07-07