

Cardiogenic Shock

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THE STORY IN ONE SENTENCE

The heart muscle is so badly damaged that it cannot pump enough blood forward to keep the body's organs alive.

“Cardiogenic shock means the pump itself has failed, and now every organ is starving for blood and oxygen.”

Normal anatomy — the body's job

The heart is a two-sided pump. The right side pumps blood through the lungs to load oxygen. The left side pumps that oxygen-rich blood out to the rest of the body. Every organ—brain, kidneys, liver, muscles—depends on steady blood flow to deliver oxygen and fuel. The amount of blood the heart pumps each minute (cardiac output) equals how much it pushes with each beat (stroke volume) times how many beats per minute (heart rate). When this pump works normally, every organ gets the blood and oxygen it needs, blood pressure stays steady, and waste is carried away.

BODY'S JOB

The heart's job is to push oxygen-rich blood forward to every organ, every second, so cells can make energy and stay alive. If the pump weakens or stops, organs begin to fail within minutes.

1. Blood returns from the body (low in oxygen) into the right atrium, then into the right ventricle.
2. The right ventricle squeezes and pushes blood through the lungs, where it picks up oxygen and releases carbon dioxide.
3. Oxygen-rich blood flows back to the left atrium, then into the left ventricle.
4. The left ventricle squeezes hard and pushes blood out through the aorta (the body's main artery) to every organ and tissue.

5. Organs take the oxygen and nutrients they need from the blood, then send waste-filled blood back to the right side of the heart to start the loop again.

What breaks

The heart muscle becomes so weak or damaged that it cannot squeeze hard enough to push blood forward, so organs begin to starve.

CLINICAL TERM

The left ventricle loses its ability to generate enough forward stroke volume to maintain systemic blood flow (perfusion) and blood pressure.

How the body compensates

The body fights hard to keep blood pressure and oxygen delivery steady, even as the heart is failing. For a while, stress hormones and squeezed blood vessels can hide how sick the patient really is—but that fight cannot last. Once the body's backup systems are exhausted, organs collapse quickly.

1. {'what': 'Sympathetic nervous system activates (fight-or-flight)', 'why': "Sensors in the carotid arteries (neck arteries) and the brainstem detect falling blood pressure and low oxygen; they trigger the release of epinephrine (adrenaline, the body's main fight-or-flight hormone) and norepinephrine (noradrenaline, a hormone that squeezes blood vessels and speeds the heart) to try to raise blood pressure and keep oxygen flowing to the brain."}
2. {'what': 'Heart rate goes up', 'why': 'A faster heart rate tries to make up for the weak squeeze by pumping more times per minute, hoping to keep total blood flow steady.'}
3. {'what': 'Blood vessels squeeze tight (vasoconstriction)', 'why': 'Tightening arteries and veins raises blood pressure and pushes more blood back to the heart, trying to keep the brain and heart supplied—even if it means stealing from skin, muscles, and kidneys.'}
4. {'what': 'Renin-angiotensin-aldosterone system (RAAS) turns on', 'why': 'The kidneys sense low blood flow and release renin (a kidney enzyme that starts a hormone chain), which forms angiotensin II (a powerful vessel-squeezer) and aldosterone (a hormone that tells the kidneys to hold onto salt and water), trying to keep blood volume and pressure up.'}
5. {'what': 'Blood is shunted away from skin, muscles, and gut toward the brain and heart', 'why': 'The body sacrifices less vital areas to keep oxygen flowing to the two organs that must not fail: the brain and the heart itself.'}
6. {'what': 'Cold, clammy, pale skin appears', 'why': 'Squeezed skin vessels reduce blood flow to the surface; sweat from stress hormones makes skin clammy.'}

7. {'what': 'Cells switch to anaerobic metabolism (working without oxygen)', 'why': 'Starved of oxygen, muscles and organs burn fuel without oxygen, creating lactic acid as waste—lactate rises in the blood.'}
8. {'what': 'Urine output drops', 'why': 'Low blood flow to the kidneys forces them to save every drop of water; rising creatinine signals kidney injury.'}
9. {'what': 'Mental status changes—confusion, anxiety, or sleepiness', 'why': 'The brain is not getting enough oxygen and glucose; waste products build up.'}
10. {'what': 'All these defenses can keep blood pressure near-normal for a short time, masking how close the patient is to collapse', 'why': 'A patient may look stable on the monitor but is actually on the edge of multi-organ failure. Vital signs alone do not show how hard the body is working or how little reserve is left.'}
11. {'what': 'Once compensatory mechanisms fail, the patient crashes quickly—organs shut down, blood pressure plummets, and death follows within hours without emergency intervention', 'why': 'The body has exhausted every backup plan. Early revascularization (opening the blocked artery with a stent or bypass surgery) and mechanical circulatory support (devices like an intra-aortic balloon pump or Impella that help or replace the heart's pumping) are the only way to stop the spiral. The landmark SHOCK trial showed that emergency revascularization within 6–12 hours significantly improves survival.'}

Domino effect

1. The left ventricle muscle is badly damaged (usually by a large heart attack) and loses its squeeze strength. — Without enough living, contracting muscle, the heart cannot push blood forward—cardiac output drops sharply.
2. Blood pressure falls and organs begin to sense they are not getting enough oxygen. — Low cardiac output means less blood flow to every organ; sensors in the carotid arteries (neck arteries), kidneys, and brainstem detect the drop.
3. The sympathetic nervous system floods the body with epinephrine (adrenaline) and norepinephrine (noradrenaline); heart rate climbs and blood vessels squeeze tight. — The body tries to raise blood pressure and keep flow to the brain and heart by speeding the pump and tightening the pipes—even though the broken pump cannot keep up.
4. Blood is stolen from the skin, muscles, kidneys, and gut and sent to the brain and heart—hands and feet turn cold, pale, and sweaty. — Squeezed vessels in the arms, legs, and skin redirect the limited blood supply to the two organs that must not fail; cold, clammy skin is a visible sign of this redistribution.
5. Organs switch to working without oxygen (anaerobic metabolism), and lactic acid builds up in the blood. — Starved muscle and organ cells burn fuel without oxygen, producing lactate as waste—rising lactate is a chemical alarm that cells are suffocating.

6. The kidneys sense low flow, release renin (the enzyme that starts the kidney's emergency hormone chain) to hold onto salt and water, and stop making much urine; creatinine rises. — Low blood flow triggers the renin-angiotensin-aldosterone system to save every drop of fluid and keep blood volume up, but this also means the kidneys stop filtering waste—acute kidney injury begins.
7. The liver, gut, and lungs also begin to fail—liver enzymes rise, the gut may bleed or become blood-starved (ischemic) (not enough blood to feed it), and fluid backs up into the lungs causing pulmonary edema (fluid flooding the lungs) (water in the air sacs) and low oxygen. — Every organ depends on steady blood flow; as the pump weakens further, each organ shuts down in turn—this is multi-organ failure.
8. The brain, starved of oxygen and drowning in waste, causes confusion, agitation, or loss of consciousness. — Brain cells are extremely sensitive to low oxygen and low blood sugar—mental status change is often the first sign that shock is becoming irreversible.
9. The heart itself, already damaged, gets even less oxygen from its own coronary arteries (the arteries that feed the heart muscle), so it weakens further—a vicious spiral where shock worsens shock. — Low blood pressure means the heart muscle cannot feed itself; dying heart muscle cannot pump, which drops pressure further, which starves the heart more—the spiral accelerates toward death unless the blocked artery is opened or a pump-assist device is placed immediately.

Symptoms · what the patient feels, and why

- Blood pressure drops very low (top number under 90): The left ventricle (the heart's main pumping chamber) is too weak or damaged to squeeze hard enough to push blood out with normal force, so pressure in the arteries falls.
- Fast, weak pulse: The heart tries to beat faster to make up for the low amount of blood pumped with each beat, but each beat is weak because the heart muscle isn't squeezing well.
- Skin feels cold, clammy, and looks pale or bluish (especially fingers, toes, lips): Blood vessels in the skin tighten up (vasoconstriction) to send blood first to the brain and vital organs, so less blood reaches the skin; without enough oxygen-rich blood, skin turns cool and may look pale or blue.
- Breathing feels fast and hard, or the person is gasping for air: Blood backs up in the lungs because the weak left ventricle can't pump it forward, causing fluid to leak into the tiny air sacs (pulmonary swelling (edema) — water in the lungs), which makes it hard to breathe.
- Confusion, sluggish thinking, or loss of consciousness: The brain isn't getting enough oxygen-rich blood because the heart's pump is too weak, so thinking becomes foggy or the person may pass out.

- Urine output drops to almost nothing (less than half a cup per day for an adult): The kidneys need good blood flow to filter waste and make urine; when the heart can't pump enough blood, the kidneys slow down or stop working.
- Lactate level in the blood is high (above 2 mmol/L on a blood test): Tissues aren't getting enough oxygen, so cells switch to a backup energy process that makes lactate (a waste product); rising lactate means cells are starving for oxygen.
- Neck veins look swollen or bulging: Blood is backing up into the jugular veins (the big veins in the neck) because the right side of the heart can't pump it forward efficiently, so pressure rises in the veins and they swell.
- Heart sounds like it's racing or beating irregularly: The damaged heart may develop abnormal electrical signals (arrhythmias — bad heart rhythms) as it struggles to pump, causing an uneven or very fast rhythm.
- Legs or belly swell with fluid: Fluid leaks out of blood vessels and collects in tissues (edema) because pressure backs up in the veins when the heart can't move blood forward.

Focused exam

- Systolic blood pressure below 90 mmHg despite the person being awake: The heart is so weak it cannot push enough blood forward. Low pressure means the brain, kidneys, and other organs are not getting the oxygen and fuel they need right now.
- Skin cold, clammy, and pale — especially the hands and feet: The body has squeezed all the tiny blood vessels in the skin to keep blood flowing to the brain and heart. This is the body trying to survive, and it tells you blood flow everywhere else is failing.
- Jugular veins bulging high in the neck, even when sitting up at 45 degrees: Blood is backing up because the right side of the heart cannot push it into the lungs. In cardiogenic shock, this bulge shows the pump on the right side is too weak, or the left side is so backed up it is blocking the right.
- Crackles (rales) at the lung bases when you listen with a stethoscope: Fluid is leaking backward from the weak left ventricle (the heart's main pumping chamber) into the tiny air sacs in the lungs. This makes it harder to breathe and tells you the left side of the heart is failing.
- Urine output less than 0.5 milliliters per kilogram per hour (for example, less than 35 mL per hour in a 70-kg adult): The kidneys shut down when blood pressure drops and blood flow is poor. Low urine is one of the earliest signs that organs are starting to fail.
- Confusion, slow thinking, or unusual restlessness: The brain is not getting enough oxygen because the heart cannot pump enough blood. Any change in thinking means the shock is harming vital organs right now.

Diagnostics · every test answers a question

- Arterial blood gas with lactate → Is the body getting enough oxygen, and are tissues switching to the backup fuel system?
- Troponin I or T → Is heart muscle dying right now?
- B-type natriuretic peptide (BNP) or NT-proBNP → Is the heart stretched and failing?
- Bedside echocardiogram (ultrasound of the heart) → What part of the pump is broken?
- 12-lead electrocardiogram (ECG) → Is a blocked artery causing a heart attack right now?
- Chest X-ray → Is fluid backing up into the lungs?
- Basic metabolic panel (sodium, potassium, creatinine, blood urea nitrogen) → Are the kidneys shutting down from low blood flow?
- Complete blood count and liver enzymes (AST, ALT) → Are other organs failing because of low blood flow?

Treatment logic

- Norepinephrine intravenous (IV) infusion → Squeezes blood vessels to raise blood pressure and also strengthens each heartbeat so more blood reaches the brain, kidneys, and other organs. Buys time while you find and fix the broken part of the heart.
- Emergency cardiac catheterization (threading a thin tube into the heart arteries through the wrist or groin) → Shows exactly which coronary artery is blocked. The team can open the artery immediately with a balloon and stent (percutaneous coronary intervention, PCI), restoring blood flow to dying muscle and often reversing the shock.
- Inotropic drugs: dobutamine or milrinone IV (only if systolic BP is at least 80–85 mmHg) → Makes the weakened heart muscle squeeze harder and relax faster, so more blood moves forward with each beat. This improves organ blood flow without squeezing vessels as tightly as norepinephrine.
- Mechanical circulatory support: intra-aortic balloon pump (IABP), Impella, or veno-arterial extracorporeal membrane oxygenation (VA-ECMO) → A device physically does some or all of the heart's pumping work. The balloon inflates between heartbeats to push blood forward; Impella is a tiny pump inside the heart; ECMO is a machine outside the body that pumps and adds oxygen. All of these give organs blood flow while you fix the heart or wait for it to recover.
- Careful fluid management: give very little IV fluid, and remove extra fluid with diuretics (furosemide IV) if lungs are wet → Keeps the lungs from filling with fluid (which makes breathing harder and lowers oxygen) and prevents the weak heart from being flooded with extra volume it cannot pump.

- Urgent surgery if echocardiogram shows a torn valve (mitral regurgitation) or a hole in the wall between ventricles (ventricular septal rupture) → Surgically repairs the structural break so blood flows the right direction again and the pump can work. Without surgery, these mechanical complications are almost always fatal.

Medications

Norepinephrine · Vasopressor (a medicine that squeezes blood vessels tighter to raise blood pressure)

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.

1. Norepinephrine locks onto alpha-1 receptors on the smooth muscle wrapping around arteries and veins.
2. A signal cascade inside those muscle cells causes calcium to flood in.
3. Calcium makes the muscle fibers slide together and squeeze—the vessel diameter shrinks.
4. Blood pressure climbs because the same volume of blood is now forced through narrower pipes.
5. At the same time, norepinephrine binds beta-1 receptors in the heart, raising the force and speed of each contraction.
6. More blood reaches vital organs (brain, kidneys, heart itself), keeping cells alive while the team fixes the root problem.

SAFETY WATCH

Can starve fingers, toes, or bowel of blood if the dose is too high or runs too long—extreme squeezing can cut off circulation and kill tissue (gangrene). · May trigger dangerous fast heart rhythms (ventricular a fast heart rate (tachycardia), atrial fibrillation (an irregular, quivering heartbeat)) because it revs the heart. · Must be given through a central line (large IV in the chest or neck)—if it leaks into skin, it kills that patch of tissue (necrosis). · Requires continuous blood-pressure monitoring (arterial line) because too much can spike pressure so high it causes stroke or bursts vessels.

Dobutamine · Inotrope (a medicine that makes the heart squeeze harder)

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.

1. Dobutamine binds to beta-1 receptors on the surface of heart muscle cells.
2. This activates a chain reaction inside the cell (cAMP pathway) that raises calcium levels.
3. More calcium floods the contractile machinery—the fibers that slide past each other to squeeze.
4. Each heartbeat becomes stronger; the ventricle ejects a bigger volume of blood (stroke volume goes up).
5. Beta-2 receptors in vessel walls also respond, relaxing the smooth muscle slightly and lowering resistance.
6. The heart pumps more blood forward, and because resistance is lower, it doesn't have to work quite as hard against the pipes.

SAFETY WATCH

Can cause blood pressure to fall if vessel relaxation outweighs the stronger heartbeat—only use when pressure is stable enough (usually after norepinephrine is already running).
· May trigger dangerous fast rhythms (atrial fibrillation, ventricular a fast heart rate (tachycardia)) because it speeds and strengthens the heart. · Increases how much oxygen the heart muscle needs—risky if coronary arteries are blocked, because the heart may outrun its fuel supply and die faster. · Requires continuous heart-rhythm monitoring (telemetry) and frequent checks for chest pain or new EKG changes showing more muscle damage.

Milrinone · Phosphodiesterase-3 inhibitor (inotrope and vasodilator—makes the heart squeeze harder and relaxes blood vessels)

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.

1. Milrinone enters cells and blocks phosphodiesterase-3, the enzyme that chews up cAMP.
2. cAMP levels rise in heart muscle, boosting calcium flow during contraction—the heart squeezes harder.
3. cAMP also rises in vessel walls, relaxing the smooth muscle—arteries and veins widen.

4. The stronger heartbeat ejects more blood per stroke (cardiac output goes up).
5. Lower vascular resistance means the heart faces less back-pressure, reducing its workload.
6. Better kidney blood flow (perfusion) may increase urine output, helping clear some extra fluid.

SAFETY WATCH

Can drop blood pressure dangerously low—only use when pressure is already supported (typically with norepinephrine running). · Carries high risk of life-threatening irregular rhythms (ventricular a fast heart rate (tachycardia), ventricular fibrillation). · Has a long half-life (hours)—once started, effects can't be turned off quickly; the drug lingers in the body. · May worsen outcomes in acute heart attack (myocardial infarction) (heart attack)—reserve for refractory cases under intensive monitoring. · Requires continuous telemetry and arterial-line blood-pressure monitoring.

Epinephrine · Vasopressor and inotrope (squeezes blood vessels and strengthens the heart)

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.

1. Epinephrine locks onto alpha-1 receptors in vessel walls, causing intense vasoconstriction—blood pressure shoots up.
2. It also grabs beta-1 receptors in the heart, cranking up both the force (inotropy) and speed (chronotropy) of each beat.
3. Beta-2 receptors in the lungs relax airway smooth muscle, opening breathing passages (helpful if there is also pulmonary edema (fluid flooding the lungs) or bronchospasm).
4. Blood pressure climbs steeply, forcing flow to the brain, kidneys, and coronary arteries.
5. Heart rate and contractility skyrocket—the heart works at maximum capacity, buying time but burning far more oxygen.

SAFETY WATCH

Dramatically increases myocardial oxygen demand—can worsen heart attack damage and expand the a patch of dead tissue (infarct) zone. · Very high risk of dangerous arrhythmias (ventricular a fast heart rate (tachycardia), ventricular fibrillation, severe a fast heart rate). · Can cause severe high blood pressure (hypertension), risking intracranial bleeding (hemorrhage) (bleeding in the brain) or aortic dissection. · Used only when norepinephrine alone fails to maintain adequate blood flow (perfusion)—never a first-line choice. · Requires continuous telemetry, arterial-line monitoring, and immediate readiness to defibrillate.

Furosemide (Lasix) · Loop diuretic (a water pill that makes the kidneys dump salt and water into the urine)

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.

1. Furosemide enters the bloodstream and is secreted into the kidney tubule.
2. It reaches the thick ascending limb of the loop of Henle and blocks the sodium-potassium-chloride cotransporter (NKCC2).
3. Sodium, potassium, and chloride stay in the tubule instead of being reabsorbed into the blood.
4. Water is pulled along with the solutes (osmosis)—urine volume jumps.
5. Fluid leaves the lungs (reducing pulmonary edema (fluid flooding the lungs) and breathlessness) and tissues, lowering the volume load on the failing heart.
6. Venous return (preload) drops, easing the stretch on the overfilled ventricles.

SAFETY WATCH

Can drop blood pressure dangerously if too much fluid is removed too fast—kidneys and brain need adequate blood flow (perfusion) pressure. · May cause severe electrolyte imbalances: low potassium (hypokalemia), low magnesium (hypomagnesemia), low sodium (hyponatremia)—low potassium (hypokalemia) can trigger deadly arrhythmias. · Can worsen acute kidney injury if blood pressure is already low—kidneys need enough pressure to filter blood. · In cardiogenic shock, use the lowest dose that relieves pulmonary congestion; aggressive diuresis can be fatal. · Monitor electrolytes, kidney function (creatinine, urine output), and blood pressure closely.

Aspirin · Antiplatelet agent (stops platelets from sticking together to form clots)

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).

1. Aspirin enters the bloodstream and diffuses into platelets.
2. It binds irreversibly to cyclooxygenase-1 (COX-1), blocking the active site permanently.
3. COX-1 can no longer convert arachidonic acid into thromboxane A2.
4. Platelets lose their main signal to clump and aggregate—they become far less sticky for their entire 7–10 day lifespan.
5. New clots in coronary arteries stop forming or growing; existing small clots may stabilize.
6. Blood flow to blood-starved (ischemic) (starving) heart muscle improves, limiting further damage and a patch of dead tissue (infarct) expansion.

SAFETY WATCH

Increases bleeding risk—watch for gastrointestinal bleeding (black tarry stools, vomit that looks like coffee grounds), blood in urine, or bleeding in the brain. · Can cause or worsen stomach ulcers or gastritis, especially with chronic use or high doses. · Rare but serious: aspirin allergy (urticaria, angioedema, bronchospasm)—do not give if known hypersensitivity. · In acute coronary syndrome with cardiogenic shock, benefits far outweigh bleeding risk—give 162–325 mg chewed immediately unless contraindicated.

Heparin (unfractionated) or Enoxaparin (low-molecular-weight heparin) · Anticoagulant (blood thinner that stops clotting proteins from working)

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.

1. Heparin binds to antithrombin III, inducing a conformational change that activates it.
2. Activated antithrombin rapidly locks onto and inactivates thrombin (factor IIa), stopping it from building fibrin clots.
3. It also blocks factor Xa upstream, cutting off the clotting cascade before thrombin is even made.

4. The blood's ability to form new clots drops sharply.
5. Existing small blood clots (thrombi) stop growing, and vessels stay patent (open) long enough for percutaneous coronary intervention (PCI), coronary artery bypass grafting (CABG), or mechanical circulatory support (e.g., intra-aortic balloon pump, Impella, ECMO).

SAFETY WATCH

Major bleeding risk—intracranial bleeding (hemorrhage) (stroke symptoms: sudden severe headache, confusion, weakness), gastrointestinal bleeding, retroperitoneal bleeding (severe back or flank pain, dropping blood pressure). · Heparin-induced a low platelet count (thrombocytopenia) (HIT)—rare immune reaction where platelets drop and paradoxical NEW clots form (deep vein clot formation (thrombosis), pulmonary embolism (a clot lodging in a lung artery), limb starved blood flow (ischemia)); if suspected, stop heparin immediately and switch to a direct thrombin inhibitor (e.g., argatroban, bivalirudin). · Unfractionated heparin requires monitoring with activated partial thromboplastin time (aPTT) every 6 hours; target 1.5–2.5 times control to balance clot prevention and bleeding risk. · Enoxaparin (low-molecular-weight heparin) is dosed by weight, easier to use, does not require aPTT monitoring—but harder to reverse if major bleeding occurs (protamine only partially reverses it). · In patients undergoing PCI, unfractionated heparin is preferred because its effect can be monitored in real time and reversed rapidly with protamine sulfate.

Morphine · Opioid analgesic (pain reliever that also reduces anxiety and work of breathing)

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.

1. Morphine locks onto mu-opioid receptors in the central nervous system.
2. Pain and trouble breathing (dyspnea) (breathlessness) signals are dampened; the sensation of suffocation eases.
3. Anxiety and sympathetic overdrive (fight-or-flight response) decrease—the patient breathes more slowly and uses less oxygen.
4. Veins relax and dilate slightly, shifting blood volume away from the central circulation and lungs.

5. Preload (the amount of blood returning to the heart) falls, reducing stretch on the failing ventricles and easing pulmonary congestion.
6. The patient feels calmer and less panicked, which may lower oxygen demand.

SAFETY WATCH

Can drop blood pressure dangerously, especially in shock—use tiny doses (1–2 mg IV, may repeat cautiously) and only if absolutely necessary. · Suppresses respiratory drive—can cause respiratory arrest (stopped breathing) if dose is too high or patient is very sick; have naloxone (opioid reversal drug) and airway equipment ready. · May mask worsening shock because the patient appears calmer even as blood flow (perfusion) deteriorates—do not rely on subjective comfort alone; monitor blood pressure, lactate, and urine output. · Modern guidelines (2017 ACC/AHA heart failure, 2021 ESC acute heart failure) recommend using morphine sparingly or avoiding it in cardiogenic shock—non-invasive ventilation (BiPAP, CPAP) and diuretics are safer for managing trouble breathing (dyspnea). · If respiratory distress is severe despite other treatments, early intubation and mechanical ventilation are preferred over escalating morphine doses.

Red flags

- 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

Nursing priorities

1. 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). — The heart is failing and blood pressure can drop suddenly to dangerously low levels; catching a pressure drop within minutes lets us increase norepinephrine or add mechanical support before organs die from lack of oxygen.
2. 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. — Norepinephrine squeezes blood vessels tight (vasoconstriction) to raise blood pressure quickly and keeps oxygen-rich blood flowing to the brain, heart, and kidneys; without it, organs shut down within minutes to hours.

3. 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). — The kidneys need steady blood flow at pressures above 65 mmHg to filter waste and make urine; when urine stops, it means blood pressure is too low and kidneys are failing (acute kidney injury), requiring stronger medicines or mechanical pumps.
4. 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. — The failing heart cannot pump oxygen-rich blood forward, so fluid backs up into the lungs (pulmonary swelling (edema)), making it hard to breathe; extra oxygen pressure forces oxygen across swollen lung tissue into the bloodstream to keep organs alive.
5. 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. — When the heart cannot pump enough blood, the body shuts off flow to the skin and extremities (peripheral vasoconstriction) to save the brain and heart; cold, blue skin signals severe shock and tells us mechanical support is needed urgently.
6. Check mental status every 15 minutes using simple questions (name, date, location) and report any confusion, sleepiness, or failure to respond to voice. — The brain needs constant oxygen and glucose delivered by blood flow; confusion or drowsiness means cardiac output has dropped so low that the brain is starving, signaling life-threatening shock that needs immediate escalation for IABP, Impella, or VA-ECMO.
7. 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. — The damaged heart cannot pump extra fluid forward, so giving too much IV fluid backs up into the lungs (flash pulmonary edema (fluid flooding the lungs)) and drowns the patient; careful fluid balance prevents worsening breathing while maintaining just enough blood volume for the weak heart to pump.
8. 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. — Most cardiogenic shock comes from a blocked coronary artery during a heart attack; opening the artery with a stent within 90 minutes restores blood flow to dying heart muscle, improves pumping strength, and is the only treatment that can reverse shock and save the patient's life.

Sources

- AHA/ACC 2022 Heart Failure Guideline

- AHA 2020 Advanced Cardiovascular Life Support (ACLS)
- NEJM Cardiogenic Shock Review
- SHOCK Trial and CULPRIT-SHOCK Trial

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