

Post Cardiac Arrest Care

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THE STORY IN ONE SENTENCE

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“After the heart stops and is restarted, the brain and heart are injured from losing oxygen, so we cool the body to protect the brain, fix what caused the arrest, support the heart and blood pressure, and watch closely to see if the brain will recover.”

Normal anatomy — the body's job

Normally, the heart pumps blood nonstop to every part of the body, especially the brain, so both get a constant supply of oxygen and sugar to power their work. The brain uses that oxygen to run every thought, breath, and heartbeat; the heart itself needs oxygen-rich blood flowing through the coronary arteries (the small vessels that feed the heart muscle) to keep squeezing. As long as blood flows, cells stay alive and the body hums along smoothly.

BODY'S JOB

The heart's job is to pump blood; the blood's job is to carry oxygen; the brain and every organ use that oxygen to make energy and stay alive. If blood stops flowing even for a few minutes, brain cells and heart cells start to die because they have no backup fuel tank.

1. The heart squeezes about 60 to 100 times per minute, pushing blood out through the aorta (the body's main highway) to every organ.
2. At the same time, the coronary arteries branch off the aorta and loop back to feed the heart muscle itself with oxygen-rich blood.
3. The brain sits at the top of that highway and gets about 15% of all the blood the heart pumps, because brain cells burn oxygen faster than almost any other tissue.
4. Each heartbeat delivers a fresh wave of oxygen; each brain cell uses that oxygen within seconds to make ATP (the energy molecule cells run on).

5. If the heart stops, blood flow stops, oxygen delivery stops, and within 4 to 6 minutes brain cells begin to die; heart muscle cells start to die soon after.

What breaks

When the heart stops (cardiac arrest), blood stops flowing, so the brain and heart run out of oxygen within minutes. Brain cells start to swell and die; heart muscle cells get stunned and stop squeezing properly. Even after the heart is restarted, damage keeps happening: dying brain cells leak toxic chemicals, blood vessels become leaky, and the heart may be too weak to pump well, so blood pressure drops and organs do not get enough blood. On top of that, whatever caused the heart to stop in the first place (a blocked artery, a dangerous rhythm, low oxygen, severe infection) is often still there and must be found and fixed, or the heart will stop again.

CLINICAL TERM

Post-cardiac arrest syndrome: a combination of anoxic brain injury (brain damage from lack of oxygen), myocardial stunning (shocked, weak heart muscle), systemic starved blood flow (ischemia)-reperfusion injury (damage that happens when blood flow comes back and floods tissues with inflammatory chemicals), and the persistent precipitating pathology (whatever caused the arrest—most often acute coronary syndrome, arrhythmia, respiratory failure, or sepsis).

How the body compensates

The body has almost no way to compensate after cardiac arrest, because the brain and heart—the two master controllers—are both injured. Any vital signs that look stable are usually only holding because of CPR, medications, or machines, not because the body fixed itself. The real danger is that things can look calm for hours, then suddenly crash as the brain swells more or the heart rhythm collapses again.

1. During CPR, chest compressions and rescue breaths provide just enough blood flow and oxygen to keep some brain and heart cells alive, buying a few extra minutes for the heart to restart.
2. Once the heart restarts, machines (the ventilator, IV blood-pressure medications, the cooling blanket) do the work the body cannot do on its own: the ventilator breathes for the patient, vasopressors (drugs that squeeze blood vessels) like norepinephrine keep pressure up, and cooling slows the brain's metabolism so damaged cells do not die as fast.

3. The body cannot increase heart rate or blood pressure on its own if the brain's control centers (the brainstem, which runs breathing and heart rate, and the hypothalamus, which controls body temperature) are hurt, so every vital sign must be watched and supported from the outside.
4. Seemingly stable numbers—normal oxygen saturation, steady heart rate—can hide the fact that the brain is swelling or the heart is barely hanging on; stability is fragile and can vanish in minutes.
5. The dangerous middle is the first 24 to 72 hours after arrest: the brain injury is still evolving, the heart is still stunned, and any new stress (low oxygen, low blood pressure, fever, seizure) can tip the patient into another arrest or make brain damage permanent.
6. The crash point to watch for is: another cardiac arrest, worsening brain swelling that shuts off all brain function, or multi-organ failure as kidneys, liver, and lungs give out from starved blood flow (ischemia)–reperfusion injury.
7. There is no true compensation here—only bought time. If the underlying cause is not fixed and the brain and heart are not protected minute by minute, the patient will not survive or will survive with devastating injury.

Domino effect

1. The heart stops (ventricular fibrillation, pulseless ventricular a fast heart rate (tachycardia), asystole, or pulseless electrical activity), so blood flow stops instantly. — Without blood flow, oxygen delivery stops, and within 10 seconds the brain runs out of the oxygen it has stored; within 4 to 6 minutes, brain cells begin to die because they cannot make ATP (energy) without oxygen.
2. Brain cells swell, leak toxic chemicals (glutamate, an excitatory neurotransmitter, and calcium), and trigger inflammation; the longer the arrest, the more cells die. — When cells cannot make energy, their pumps fail, sodium and calcium flood in, the cell swells like a water balloon, and it leaks glutamate that poisons neighboring cells and spreads the damage like a wildfire.
3. The heart muscle itself is starved of oxygen (because the coronary arteries also lost flow), so even after it restarts, it is stunned, weak, and cannot pump well for hours to days. — Heart muscle cells that went without oxygen are damaged and cannot contract strongly, so cardiac output (the amount of blood pumped per minute) drops, blood pressure falls, and organs do not get enough blood—this is called myocardial stunning.

4. When blood flow returns (after CPR or defibrillation restarts the heart), the sudden flood of oxygen triggers a burst of free radicals (unstable oxygen molecules) and inflammatory chemicals that damage blood vessels throughout the body. — This is starved blood flow (ischemia)–reperfusion injury: the very act of restoring blood flow paradoxically causes more damage because oxygen reacts with damaged cell parts to make toxic molecules, vessel walls become leaky, and organs swell and fail (especially lungs, kidneys, and liver).
5. The brain loses control of breathing, heart rhythm, blood pressure, and temperature, so the patient stays unconscious (comatose), breathes irregularly or not at all, and cannot maintain stable vital signs without machines. — The brainstem (the part that controls automatic functions like breathing and heart rate) and the cerebral cortex (the outer layer of the brain that controls waking and thinking) are both injured, so the patient cannot wake up, breathe on their own, or regulate their body.
6. Over the next 24 to 72 hours, brain swelling peaks, and the team watches for signs of recovery (pupil reactions, movement, waking) or signs of irreversible injury (no pupil response, no movement, flat EEG). — Brain injury after cardiac arrest evolves slowly; some cells that were hurt but not dead may recover if protected (by cooling, good blood pressure, normal oxygen and sugar), but others will die no matter what, and it takes days to know which path the patient is on.
7. Meanwhile, the team hunts for and fixes the cause of the arrest: emergency cardiac catheterization (threading a tube into the heart arteries) if it was a heart attack, antiarrhythmic drugs (medicines that steady heart rhythm) or ablation (burning the spot causing the bad rhythm) if it was a deadly rhythm, antibiotics if it was sepsis (severe infection), surgery if it was bleeding. — If the root cause is not fixed, the patient will arrest again; for example, if a coronary artery is blocked, reopening it with a stent (a tiny metal tube) restores blood flow to the heart and prevents another arrest.
8. The final outcome is decided by two things: how long the heart was stopped (down time) and how well the brain and organs are supported afterward; some patients wake up fully, some stay in a persistent vegetative state, and some die from brain death or multi-organ failure. — Every minute without blood flow kills more brain cells; even perfect post-arrest care cannot bring dead cells back, so the length of the arrest and the quality of CPR are the biggest predictors of whether the person will survive with a working brain.

Symptoms · what the patient feels, and why

- No response to voice or touch: During the arrest the brain got almost no oxygen, and neurons (the nerve cells that send all signals) were starved. Many are still stunned or damaged, so the circuits that wake a person up—running from the brainstem through the thalamus (the brain's relay station) up to the cortex (the outer thinking layer)—cannot fire properly yet.

- **Weak or absent breathing:** The medulla (the lowest part of the brainstem that runs automatic breathing) may have been injured by lack of oxygen. Without steady signals from the medulla, the diaphragm (the big breathing muscle under the lungs) and chest muscles do not contract regularly, so breaths are shallow, slow, or stop completely.
- **Very low or very high blood pressure:** The heart muscle itself is often weak and stunned after an arrest—called post-cardiac-arrest myocardial dysfunction—so it cannot squeeze hard enough to push blood out. At the same time, the autonomic nervous system (the autopilot that tightens or relaxes blood vessels) is unstable: vessels may clamp down too tight (raising pressure) or relax too much (dropping pressure).
- **Fast or irregular heart rhythm:** The heart's electrical wiring may still be irritated or scarred from the original problem that caused the arrest (such as starved blood flow (ischemia)—not enough blood feeding the heart muscle—or scar tissue from an old heart attack). Injured heart cells can fire extra beats or chaotic signals, creating a fast or jumpy rhythm.
- **Seizures (shaking fits or stiffening):** Parts of the cortex (the brain's outer layer that controls movement and thought) were damaged by lack of oxygen. Injured neurons become hyperexcitable—they fire in wild, synchronized bursts instead of orderly patterns—and that uncontrolled electrical storm spreads across the brain, causing the muscles to shake or lock up.
- **Cool, pale, or bluish skin:** The heart is pumping weakly, so less blood reaches the skin. The body also redirects whatever blood it has away from the skin and toward vital organs—the brain, heart, and kidneys—leaving the skin cold, pale, or even blue (cyanotic) from low oxygen.
- **Low oxygen level on the monitor (low SpO₂):** The lungs may have fluid in them (pulmonary swelling (edema)) because the weak heart cannot pump blood forward, so it backs up into the lung vessels and leaks into the air sacs. Fluid blocks oxygen from crossing into the blood. Or the person is not breathing deeply enough, so fresh oxygen never reaches the blood in the first place.
- **Abnormal posturing (arms or legs stiff in odd positions):** Deep brain structures—the brainstem and midbrain—were damaged by lack of oxygen. These areas normally balance signals that flex and extend muscles. When one side is knocked out, unopposed signals lock the arms, legs, or whole body into rigid, strange postures (decorticate or decerebrate posturing).
- **No pupil reaction to light:** The brainstem controls the reflex that makes pupils shrink when light hits them. If the midbrain (the part of the brainstem that runs this reflex) was badly injured by lack of oxygen, the pupils stay wide open and fixed, even under a bright penlight.
- **Little or no urine output:** The kidneys need steady blood flow to filter waste and make urine. After cardiac arrest the heart is weak and blood pressure may be low, so the kidneys get too little blood (renal poor blood flow (hypoperfusion)). Without enough flow, the nephrons (the tiny filters inside the kidney) slow down or stop making urine.

Focused exam

- Pupil size and light reflex after rewarming is complete: During cooling, blown (wide-open) pupils mean nothing—the cold itself freezes the tiny muscle that shrinks the pupil. But after you warm the person back to normal temperature, if both pupils stay wide and do not tighten when you shine a light, it means the arrest starved the brain stem (the deep part of the brain that runs breathing, heart rate, and basic reflexes) so badly that it cannot control the eyes anymore. Absent light reflex at 72 hours post-rewarming is a sign of severe, likely permanent brain damage.
- Corneal reflex (blink when you touch the clear part of the eye) and gag reflex at 72 hours post-rewarming: These two reflexes come from the brain stem, the part below the thinking brain that keeps you breathing and awake. If both are gone 72 hours after you finish warming, it means the arrest killed the brain stem and the person will not wake up. Testing too early—before 72 hours or while still sedated or cooling—gives false answers because sedation and cold slow everything down.
- Motor response to pain—can the person pull away, push your hand, or only twitch or not move at all?: After cardiac arrest, the brain might be awake inside but unable to talk or open eyes, or the brain might be so damaged it cannot send signals to move at all. Watching how the person moves when you press hard on a knuckle or rub the chest tells you how much of the brain still works. Purposeful movement (pulling away, reaching toward the pain) is a good sign; no movement or only stiff posing (arms and legs rigid, toes pointing) is a very bad sign.
- Heart rhythm on the monitor—is it a steady organized beat or is it chaotic, too fast, or stopping again?: After you restart the heart, the rhythm tells you two things: is the heart muscle so damaged from the arrest that it cannot beat steadily, and is the original cause of the arrest (like a blocked artery or dangerous rhythm) still there? If the rhythm stays messy or keeps breaking into ventricular fibrillation (the chaotic quiver that stops the heart from pumping), the heart is either too hurt or the trigger is still firing.
- Blood pressure and whether you need medicines (vasopressors like norepinephrine) to keep it up: Cardiac arrest stuns the heart muscle—it is like the heart got punched and is too weak to squeeze hard enough to push blood through the body. If blood pressure stays low even after you give IV fluids, it means the heart cannot recover on its own yet and you need medicines (or even a mechanical pump) to do the squeezing for it while the heart heals.
- Oxygen level and how hard the person is working to breathe (or how much the ventilator is doing the work): The arrest stopped blood flow to the lungs too, and the CPR and the time without oxygen damaged the lung tissue. Many people need a breathing tube and a ventilator (breathing machine) after arrest because the lungs are swollen, stiff, and leaking fluid into the air sacs, and cannot get enough oxygen into the blood on their own.

Diagnostics · every test answers a question

- 12-lead electrocardiogram (ECG) immediately after return of spontaneous circulation → Is there a STEMI (ST-elevation heart attack (myocardial infarction), the kind of heart attack where a big coronary artery is completely blocked) that caused the arrest?
- Arterial blood gas (ABG) → Is the blood getting enough oxygen, and is carbon dioxide building up or being blown off too much?
- Serum lactate → Is the body still starving for oxygen and making acid because blood flow is not good enough?
- High-sensitivity troponin I or T → Did the arrest damage the heart muscle itself, and how badly?
- Chest X-ray (portable, at the bedside) → Is the breathing tube (endotracheal tube) in the right place, are the lungs filling with fluid, and did CPR break ribs or puncture a lung?
- Continuous electroencephalogram (EEG, brain wave monitoring) → Is the brain having seizures (even though the body is paralyzed with medicines and you cannot see shaking), and are the brain waves so flat or broken that recovery is impossible?
- Neuron-specific enolase (NSE) blood level at 48–72 hours after arrest → How much brain cell death happened during the arrest?
- Head CT scan (computed tomography of the brain without contrast dye) if exam worsens or does not improve as expected → Is there bleeding in the brain, massive swelling, or a stroke that caused the arrest or happened because of it?

Treatment logic

- Targeted Temperature Management (TTM): cool the body to 32–36°C for 24 hours, then rewarm slowly at 0.25–0.5°C per hour over 8–12 hours, then prevent fever >37.5°C for at least 72 hours after arrest → Cooling the brain slows down its need for oxygen and energy, like turning down a furnace. It also turns down inflammation (the immune system's repair response that can overshoot and cause more swelling and cell death). This gives injured-but-still-alive brain cells time to heal instead of swelling up and dying in the hours after blood flow comes back. Studies show TTM improves the chance of waking up with a working brain, especially after shockable rhythms (VF, pulseless VT).

- Restore and maintain blood flow (perfusion) pressure: keep mean arterial pressure (MAP) ≥ 65 mmHg with IV fluids first, then norepinephrine or epinephrine infusion if fluids are not enough; keep oxygen saturation 94–98% and PaCO₂ 35–45 mmHg on the ventilator → The brain and heart need oxygen and fuel (glucose) delivered by blood flow. Keeping blood pressure up (MAP ≥ 65 mmHg) pushes blood into the tiny vessels in the brain and heart muscle so the injured cells can try to recover instead of dying from a second round of starvation. Keeping oxygen and CO₂ in the normal range stops the brain blood vessels from clamping down (which happens when CO₂ is too low) or swelling (which happens when CO₂ or oxygen is abnormal).
- Emergency coronary angiography (heart catheterization) if the ECG shows STEMI or if the arrest rhythm was shockable (VF or pulseless VT) and a heart cause is likely, even if the person is still unconscious → If a blocked coronary artery (the vessel that feeds the heart muscle) caused the arrest, threading a catheter up to the blockage and opening it with a balloon and metal stent restores blood flow to the dying heart muscle, stops ongoing damage, and prevents the heart from arresting again. Current guidelines (AHA 2020) say do not wait for the person to wake up—go to the cath lab right away if STEMI is on the ECG or if a heart attack is the likely cause.
- Sedation (propofol or midazolam continuous infusion) and analgesia (fentanyl) during TTM; add neuromuscular blockade (paralytic like rocuronium or cisatracurium) if shivering despite sedation → Shivering is the body trying to warm itself up by shaking the muscles, which makes heat and burns oxygen and glucose. Sedation and paralysis stop shivering so the cooling can work. This also keeps the person comfortable (they cannot feel the breathing tube in the throat or the cold) and prevents muscle breakdown (rhabdomyolysis, where muscle breaks down and clogs the kidneys) from constant shaking.
- Neuroprognostication (figuring out if the brain can recover) using a multimodal approach at ≥ 72 hours after rewarming: clinical exam (pupil and corneal reflexes, motor response to pain), continuous EEG (looking for seizures and background brain activity), brain imaging (MRI preferred, or CT), and serum biomarkers (neuron-specific enolase) → By 72 hours after rewarming, the sedation is cleared and the true extent of brain damage is visible. Combining physical exam, EEG, imaging, and blood tests gives families and the medical team an honest, evidence-based answer: will this person wake up, and if so, will they be able to think, talk, and live independently? This guides shared decisions about continuing life support or transitioning to comfort-focused care. No single test is perfect; you must use all of them together.

- Glycemic control: keep blood glucose 140–180 mg/dL with IV insulin infusion; check glucose every 1–2 hours initially and avoid low blood sugar (hypoglycemia) <80 mg/dL → The stressed, injured brain needs glucose for fuel, but very high glucose (>180 mg/dL) worsens inflammation, brain swelling, and cell death. Very low glucose (<80 mg/dL) starves the brain and can trigger seizures and cause more injury. Keeping glucose in the middle zone (140–180 mg/dL) gives the brain steady fuel without the extra harm from extremes.
- Seizure detection and treatment: if continuous EEG shows seizures or status epilepticus (a seizure that will not stop), give levetiracetam 1000–3000 mg IV load (or valproic acid or fosphenytoin if levetiracetam is not available), then start continuous infusion if seizures keep firing → Seizures after cardiac arrest burn huge amounts of oxygen and glucose in the brain and can cause more neuron death. Stopping seizures quickly reduces that extra injury. Myoclonus (repetitive muscle jerking) in the first 24–48 hours is common and does not always mean bad outcome, but true electrical seizures on EEG (especially status epilepticus (a seizure that will not stop), seizures that do not stop) need treatment right away.

Medications

Targeted Temperature Management (TTM) at 32–36°C for 24 hours, then slow rewarming · Therapeutic hypothermia (cooling the body on purpose)

After the heart stops and then starts again, brain cells are hurt and swollen from the time they went without oxygen. Cooling the whole body down to about 90–97°F slows the brain's energy burn and quiets the chemical storms inside the cells that can cause more damage. Think of it like turning down the heat on a pot that's boiling over—it gives the brain time to calm down and heal instead of spiraling into more injury. After 24 hours, you warm the person up very slowly (about 0.5°F every hour) so the body doesn't get shocked by a sudden temperature jump.

1. The brain cells that were starved of oxygen start releasing harmful chemicals like glutamate (an excitatory messenger) and calcium floods into the cells, which can kill them.
2. Cooling slows down the cells' metabolism (how fast they burn energy), so they release less glutamate and take in less calcium.
3. It also calms inflammation—white blood cells that rush in and can accidentally hurt healthy tissue are less active when the body is cooler.
4. Finally, it reduces swelling (cerebral swelling (edema)) by tightening blood vessels slightly and lowering the pressure inside the skull.
5. After 24 hours, slow rewarming prevents rebound swelling and sudden shifts in potassium and other electrolytes that can stop the heart again.

SAFETY WATCH

Shivering: The body fights the cooling and shivers, which burns energy and raises the temperature back up. Sedation (calming medicine like propofol or midazolam) and sometimes paralysis medicine (like rocuronium or vecuronium) are given to stop shivering.

- Infection risk: Cooling weakens the immune system, so pneumonia and bloodstream infections are more common. Careful handwashing and sterile technique are key.
- Heart rhythm problems: Cold slows the heart and can cause irregular beats (arrhythmias). The monitor watches the heart constantly.
- Blood sugar swings: Cooling makes insulin work poorly, so glucose can climb. Insulin drips keep it between 140–180 mg/dL.
- Bleeding: Cold affects clotting factors, so small bleeds (like from IV sites) can happen. Watch for any unusual bruising or blood.
- Electrolyte shifts during rewarming: Potassium can drop or spike suddenly, so labs are checked every few hours.

Norepinephrine or vasopressin · Vasopressors (medicines that squeeze blood vessels to raise blood pressure)

After the heart restarts, it's often weak and the blood vessels are floppy, so blood pressure drops. The brain, kidneys, and heart need steady pressure to get enough blood. A mean arterial pressure (MAP) above 65 mmHg keeps blood flowing to all the organs. Norepinephrine tightens the blood vessels by acting on alpha receptors (tiny switches on the vessel walls) and also gives the heart a small squeeze boost via beta receptors. Vasopressin (also called ADH, or antidiuretic hormone) works on different receptors (V1) to tighten vessels without speeding the heart. Both push the blood forward so oxygen reaches the brain and other tissues.

1. Norepinephrine binds to alpha-1 receptors on the smooth muscle around arteries, telling them to contract (squeeze). This narrows the pipe and raises the pressure.
2. It also hits beta-1 receptors on the heart, making it beat a bit harder and faster, which pumps more blood per beat.
3. Vasopressin binds to V1 receptors on vessel walls and causes constriction without making the heart race, so it's often added if norepinephrine alone isn't enough or if the heart rhythm is fragile.
4. The combined effect lifts MAP above 65, so blood flow to the brain and kidneys is restored.

SAFETY WATCH

Finger and toe starved blood flow (ischemia): If the dose is very high or given for a long time, the extreme vessel squeezing can starve fingers, toes, or skin of blood. Watch for pale, cold, or purple extremities. · Arrhythmias: Norepinephrine can trigger fast or irregular heartbeats because of the beta effect. The monitor tracks every beat. · Extravasation injury: If the IV leaks into the skin, these medicines can kill tissue. Central lines are safer than wrist or hand IVs. · Organ starved blood flow (ischemia): Too much squeezing can reduce blood flow to the gut or kidneys despite a higher MAP number. Labs (creatinine for kidneys, lactate for tissue oxygen) are checked.

Oxygen therapy (target saturation 94-98%) · Supportive gas therapy

The brain was starved of oxygen during the arrest, but flooding it with too much oxygen after the heart restarts can create harmful molecules called free radicals (like rust forming inside cells), which damage the mitochondria (the cell's power plants). Keeping oxygen saturation in the normal range (94-98%) ensures the brain and heart get enough oxygen to make energy without the toxic extra. A ventilator (breathing machine) or oxygen mask is adjusted to hit this sweet spot, guided by a pulse oximeter (the clip on the finger) and blood gas tests.

1. Oxygen enters the lungs and diffuses across the thin alveolar wall into capillaries (the smallest blood vessels).
2. Hemoglobin in red blood cells binds the oxygen and carries it through arteries to the tissues.
3. In the brain and other organs, oxygen is released and enters the mitochondria, where it combines with glucose to make ATP through a process called oxidative phosphorylation.
4. If oxygen is too low (hypoxemia, saturation below 90%), cells can't make enough ATP and start to die. If it's too high (hyperoxemia, saturation near 100% on high oxygen), excess oxygen generates superoxide and hydrogen peroxide, which punch holes in cell membranes and DNA.

SAFETY WATCH

Hyperoxia toxicity: Oxygen saturation above 98-100% for hours can worsen brain and lung injury. The team adjusts the ventilator or oxygen flow to stay in the safe zone. · Low oxygen (Hypoxia): If the saturation drops below 90%, the brain and heart suffer. Alarms on the monitor alert the team immediately. · Ventilator-associated pneumonia: Being on a breathing tube raises infection risk. Mouth care, head-of-bed elevation, and sterile suctioning help prevent it. · Barotrauma: Too much pressure from the ventilator can pop an air sac and cause a collapsed lung (pneumothorax). Pressure settings are kept as gentle as possible.

Ventilator adjustment to keep PaCO₂ at 35–45 mmHg · Ventilator management (not a drug, but a key part of post-arrest care)

Carbon dioxide (CO₂) is a waste gas made when cells burn glucose for energy. It dissolves in the blood and controls how wide or narrow the brain's blood vessels are. If CO₂ is too low (hypocapnia, below 35 mmHg), the brain's arteries squeeze tight and starve the brain of blood, which can worsen injury. If CO₂ is too high (hypercapnia, above 45 mmHg), the vessels open too wide, pressure inside the skull can rise, and the blood becomes acidic, which can stop the heart again. The ventilator is adjusted—breathing faster or slower, or changing the size of each breath—to keep CO₂ in the normal range, guided by blood gas tests (a sample from an artery, usually the wrist).

1. Cells in the body make CO₂ as they burn glucose and oxygen. CO₂ enters the bloodstream and is carried to the lungs.
2. In the lungs, CO₂ diffuses out of the blood into the alveoli and is exhaled.
3. The ventilator adjusts the respiratory rate (breaths per minute) and tidal volume (size of each breath) to control how much CO₂ is removed.
4. If you blow off too much CO₂ (by breathing too fast or too deeply), PaCO₂ drops, brain vessels constrict, and blood flow to the brain falls (cerebral vasoconstriction).
5. If you don't blow off enough CO₂ (breathing too slowly or shallowly), PaCO₂ rises, vessels dilate, and the brain can swell; plus the blood becomes acidic, which weakens the heart.

SAFETY WATCH

Hypocapnia (low CO₂): Can cause severe cerebral vasoconstriction and starved blood flow (ischemia) (not enough blood reaching the brain to feed it). Blood gases are checked every few hours to catch this. · Hypercapnia (high CO₂): Can raise intracranial pressure, cause too much acid in the blood (acidosis), and trigger arrhythmias. Ventilator settings are adjusted to prevent it. · Rapid changes: Swinging CO₂ up or down too fast can destabilize the brain and heart. Changes are made gradually.

Propofol or midazolam (sedation) and rocuronium or vecuronium (paralysis) · Sedatives (GABA agonists) and neuromuscular blockers

During cooling (TTM), the body shivers to warm itself back up, which fights the treatment and burns energy. Sedatives like propofol or midazolam boost GABA (gamma-aminobutyric acid, the brain's main calming signal) in the brain, which turns down consciousness and stops the shivering reflex. If sedation alone doesn't stop the shivering, a paralytic medicine like rocuronium or vecuronium is added—it blocks the signal from nerves to muscles at the neuromuscular junction (the spot where the nerve plugs into the muscle), so the muscles can't

contract even if the brain sends the shiver command. This keeps the person still and cool. The person is on a ventilator because they can't breathe on their own while paralyzed.

1. Propofol or midazolam binds to GABA-A receptors and opens chloride channels, letting negatively charged chloride ions flood into neurons. This makes the neurons less likely to fire, so the brain activity slows and consciousness fades.
2. The reticular activating system, which keeps you awake, is turned off, so the person is deeply asleep and doesn't feel the cold or the tube in their throat.
3. If shivering still breaks through, rocuronium or vecuronium is given. It blocks acetylcholine (the signal that tells muscles to contract) from binding at the neuromuscular junction.
4. Without the acetylcholine signal, the muscle can't contract, so shivering stops completely. The brain may still send the command, but the muscle never gets the message.

SAFETY WATCH

Awareness under paralysis: If sedation wears off but paralysis is still working, the person could be awake and terrified but unable to move or communicate. Sedation levels are checked with a monitor (BIS or entropy) or by watching for signs like tears or elevated heart rate. · Propofol infusion syndrome: Rare but serious—high doses of propofol for days can cause heart failure, severe too much acid in the blood (acidosis), and muscle breakdown (rhabdomyolysis). The dose and duration are limited, and triglycerides (a type of fat) are monitored because propofol is dissolved in fat. · Low blood pressure (Hypotension): Propofol relaxes blood vessels and can drop blood pressure. Vasopressors may be needed. · Prolonged paralysis: After stopping rocuronium or vecuronium, it can take hours for muscle strength to return, especially in people with kidney problems. A nerve stimulator checks if the drug has worn off. · Ventilator dependence: The person can't breathe on their own while paralyzed, so the ventilator must be working perfectly and monitored constantly.

Regular insulin or rapid-acting insulin (target glucose 140-180 mg/dL) · Hormone replacement (insulin is the body's main blood-sugar-lowering hormone)

After cardiac arrest and during cooling, the body is under enormous stress. Stress hormones like cortisol (the body's main stress hormone) and epinephrine (adrenaline) flood the bloodstream, which tells the liver to dump glucose into the blood and makes cells resistant to insulin (the hormone that pushes glucose from blood into cells). Glucose can climb dangerously high (hyperglycemia, often above 200 mg/dL), which is toxic to the already-injured brain—it increases inflammation, free radicals, and swelling. Giving insulin through an IV drip lowers glucose by telling muscle, fat, and liver cells to take glucose back in. The target is 140-180 mg/

dL, which is high enough to avoid dangerous low sugar (hypoglycemia, below 70) but low enough to protect the brain.

1. Insulin binds to insulin receptors on the cell surface, which activates a chain of signals inside the cell.
2. GLUT4 transporters (glucose doors) move from inside the cell to the membrane and open, letting glucose flow in from the blood.
3. Muscle and fat cells take up glucose and either burn it for energy or store it as glycogen (in muscle) or fat.
4. The liver stops making new glucose (gluconeogenesis) and starts storing glucose as glycogen.
5. Blood glucose drops into the safe range, reducing toxic effects on the brain.

SAFETY WATCH

Low blood sugar (Hypoglycemia): If too much insulin is given or the person isn't eating, glucose can crash below 70 mg/dL, which starves the brain of fuel and can cause seizures, coma, or death. Glucose is checked every 1–2 hours, and the IV has a dextrose (sugar) line ready to push if it drops. · Low potassium (Hypokalemia): Insulin pushes potassium into cells along with glucose, so blood potassium can fall. Low potassium can cause dangerous heart rhythms. Potassium is checked with every glucose check and replaced if it drops below 3.5 mEq/L. · Rebound high blood sugar (hyperglycemia): When the insulin drip is turned down or stopped too quickly, glucose can spike again. Transitions are done slowly.

Levetiracetam, fosphenytoin, or valproate (if seizures occur) · Antiepileptic drugs (medicines that quiet abnormal electrical storms in the brain)

After the brain is starved of oxygen, injured neurons can fire in wild, uncontrolled bursts—this is a seizure. If the seizure doesn't stop or keeps coming back without the person waking up in between (status epilepticus), it burns massive energy, raises brain temperature, and causes more injury. Anticonvulsants calm the brain's electrical activity. Levetiracetam works by binding to SV2A (a protein on synaptic vesicles, the bubbles that hold neurotransmitters), which slows the release of excitatory signals. Fosphenytoin blocks sodium channels on neurons, stopping the rapid firing. Valproate boosts GABA (the brain's main calming neurotransmitter) and also blocks sodium and calcium channels. All three quiet the storm and stop the seizure.

1. Levetiracetam binds to SV2A on the neuron's axon terminals (the sending ends). This reduces how many neurotransmitter bubbles are released, so fewer excitatory signals (like glutamate) jump to the next neuron.

2. Fosphenytoin (which is converted to phenytoin in the body) plugs the sodium channels that neurons use to fire. Without sodium rushing in, the neuron can't generate an action potential (the electrical spike), so the seizure wave stops spreading.
3. Valproate increases GABA by blocking the enzyme that breaks GABA down and by opening GABA receptors. It also blocks sodium and T-type calcium channels, which further stabilizes neurons.
4. EEG (electroencephalogram, a test that reads brain waves through stickers on the scalp) is watched continuously to see if the seizure stops.

SAFETY WATCH

Sedation and confusion: All anticonvulsants can make the person drowsy or confused once they start to wake up. This can make it hard to assess brain recovery. · Low blood pressure (Hypotension): Fosphenytoin, especially if pushed too fast, can drop blood pressure. It's given slowly through an IV, and pressure is monitored. · Liver toxicity: Valproate can raise liver enzymes (ALT, AST). Labs are checked regularly. · Blood count changes: Valproate can lower platelets (the blood cells that clot), and levetiracetam can rarely lower white blood cells. Complete blood counts are monitored. · Allergic reactions: Fosphenytoin can cause rash or, very rarely, a severe skin reaction (Stevens-Johnson syndrome). Any new rash is reported immediately. · Drug interactions: Fosphenytoin affects many other medicines by speeding up how the liver breaks them down.

Percutaneous coronary intervention (PCI) with aspirin, ticagrelor or clopidogrel, and heparin (if arrest was caused by heart attack) · Procedure (emergency cardiac catheterization) plus antiplatelet and anticoagulant drugs

Many cardiac arrests happen because a coronary artery (one of the arteries feeding the heart muscle) is suddenly blocked by a clot, causing a heart attack (myocardial tissue death from blocked blood flow (infarction)). The heart muscle downstream from the blockage dies quickly without oxygen, and the dying muscle can trigger lethal rhythms like ventricular fibrillation. If the arrest was caused by a heart attack—especially a STEMI (ST-elevation MI, seen on the ECG as tall ST segments)—or if the rhythm was shockable (VF or pulseless VT) and a heart attack is suspected, the person goes to the cath lab even if still unconscious. A cardiologist threads a thin tube (catheter) through an artery in the wrist or groin up to the heart, finds the blockage, inflates a balloon to squash it open, and usually places a stent (a tiny metal scaffold) to keep it open. This restores blood flow to the heart muscle and can prevent another arrest. Antiplatelet drugs (aspirin, ticagrelor, or clopidogrel) and anticoagulants (heparin) are given during and after the procedure to keep new clots from forming.

1. A catheter is advanced from the wrist or groin artery, through the aorta, and into the coronary arteries. Dye is injected and X-rays (angiography) show where the blockage is.

2. A balloon catheter is positioned at the blockage and inflated, crushing the clot and plaque against the artery wall.
3. A stent is expanded in the artery to hold it open permanently.
4. Blood flow is restored, oxygen reaches the heart muscle, and the muscle can start to recover if it's not already dead.
5. Aspirin irreversibly blocks COX-1 (an enzyme in platelets), so platelets can't make thromboxane (a signal that tells them to clump). Ticagrelor or clopidogrel blocks the P2Y₁₂ receptor on platelets, which is another clumping signal.
6. Heparin (an anticoagulant) blocks thrombin and Factor Xa in the clotting cascade, preventing new clots from forming on the catheter or stent.

SAFETY WATCH

Bleeding: Antiplatelet drugs and heparin raise bleeding risk. The catheter site (wrist or groin) is watched closely for a pooled collection of blood (hematoma) (a blood-filled swelling) or active bleeding. · Stroke: Rarely, the catheter or dye can dislodge a clot or plaque that travels to the brain. Neuro checks are done frequently. · Contrast-induced nephropathy: The dye can hurt the kidneys, especially if they're already weak. IV fluids are given before and after, and kidney function (creatinine) is monitored. · Arrhythmias during the procedure: Opening the artery or manipulating the heart can trigger new abnormal rhythms. The person is on a monitor the whole time. · Re-blockage (stent clot formation (thrombosis)): If antiplatelet drugs are stopped too soon, a clot can form on the stent and block the artery again. Aspirin is continued lifelong, and ticagrelor or clopidogrel for at least 12 months.

Continuous EEG monitoring and anticonvulsants (if nonconvulsive seizures are found) · Diagnostic monitoring (continuous EEG) and anticonvulsant therapy

After cardiac arrest, up to 1 in 3 people have seizures that don't cause visible shaking—these are called nonconvulsive seizures. The brain is firing in wild electrical storms, but because the person is already unconscious and often sedated or paralyzed, you can't see it. The only way to catch it is with continuous EEG (electroencephalogram), which reads brain waves through stickers on the scalp 24/7. If seizures or status epilepticus (a seizure that will not stop) (continuous seizure activity) is seen on the EEG, anticonvulsants are started immediately because ongoing seizures burn energy, raise brain temperature, and worsen injury. Treating the seizures gives the brain a better chance to recover.

1. Continuous EEG is started as soon as possible after the person is admitted. Technicians place electrodes on the scalp using paste, and the monitor records brain waves continuously for at least 24–48 hours (sometimes longer).

2. A neurologist or specialized technician reads the EEG and looks for patterns like spikes, sharp waves, or rhythmic discharges that indicate seizure activity.
3. If seizures are found, anticonvulsants are given IV and the EEG is watched to see if the abnormal activity stops.
4. The EEG also helps predict outcome—certain patterns (like burst suppression with identical bursts, or a completely flat line) can suggest severe brain injury.

SAFETY WATCH

Skin irritation: The electrode paste and stickers can irritate the scalp, especially after days of monitoring. The skin is checked and electrodes repositioned gently. · **Misinterpretation:** Muscle twitches, shivering, or electrical interference (from ventilators, IV pumps, or even the hospital bed) can look like seizures on the EEG. Trained readers distinguish artifact from real seizures. · **Sedation interference:** Heavy sedation can suppress brain waves and make the EEG look worse than it is, so medication levels are considered when interpreting the test.

Multimodal neuroprognostication (clinical exam, EEG, MRI or CT, and biomarkers like NSE) at ≥ 72 hours after rewarming · Structured assessment strategy (not a treatment, but a systematic approach to predict brain recovery)

After cardiac arrest, families and the medical team need to know whether the person's brain can recover enough to live a meaningful life, or whether the injury is so severe that continuing life support would only prolong dying. This assessment is called neuroprognostication. It must wait at least 72 hours after rewarming (finishing TTM) because sedation, paralytics, and the cooling itself can make the exam look worse than it really is. The team uses multiple tools together: (1) Neurologic exam—testing pupil responses (do the pupils shrink when a light is shined, via cranial nerve III?), corneal reflex (does the eye blink when the clear part is touched, via cranial nerves V and VII?), and motor response (does the person move or withdraw from pain, testing the motor cortex and spinal pathways?). Absent pupil and corneal reflexes, or no motor response better than extensor posturing (stiff straightening of the arms and legs) at 72+ hours is a very bad sign. (2) EEG—looking for patterns like burst suppression (flat line interrupted by brief bursts), absence of brain wave reactivity (no change when the person is stimulated), or status epilepticus (a seizure that will not stop). (3) Brain imaging—CT or MRI showing severe global swelling (edema) (swelling), loss of gray-white differentiation (the normal boundary between the cortex and deeper white matter is erased), or widespread restricted diffusion (a sign on MRI that cells are dying). (4) Biomarkers—blood tests for neuron-specific enolase (NSE, a protein released when brain cells die); very high levels suggest massive injury. No single test is perfect, so all findings are combined. If multiple bad signs are present and remain after 72 hours, the prognosis is very poor (likely death or permanent

vegetative state). If some signs are good (pupils react, some purposeful movement, normal EEG background), there is hope for recovery, though it may take weeks.

1. At 72 hours after rewarming, sedation and paralytics are stopped completely and given time to clear from the body (checked with blood levels or a nerve stimulator).
2. The physician performs a detailed neuro exam: shining a light in the eyes to see if the pupils constrict, touching the cornea gently with a swab to check for a blink, and applying firm pressure (like rubbing the sternum or nail beds) to see if the person moves or pulls away.
3. EEG is reviewed for background activity, reactivity (does the pattern change when the person is stimulated?), and seizure patterns.
4. MRI or CT is done to look at brain structure. MRI is better for seeing subtle injury (restricted diffusion on DWI sequences means cells are swelling and dying; T2/FLAIR images show swelling (edema)).
5. Blood is drawn to measure NSE and sometimes S100B (another brain injury marker). Very high NSE (above 60–90 µg/L, depending on the lab) suggests widespread neuron death.
6. All findings are combined. If multiple poor prognostic signs are present (absent pupil/corneal reflexes, no motor response, malignant EEG, severe MRI changes, very high NSE), the chance of meaningful recovery is extremely low. If findings are mixed or improving, prognostication is deferred and repeated in a few days.

SAFETY WATCH

Premature withdrawal of life support: If prognostication is done too early (before 72 hours, or while sedation is still present), it can falsely suggest no hope and lead to stopping care in someone who might have recovered. Guidelines stress waiting and using multiple tests.

- False hope: Rarely, all tests look favorable but the person still does not wake up. Families need clear, compassionate communication that uncertainty remains.
- Confounders: Drugs (sedatives, opioids, anticonvulsants), metabolic derangements (liver failure, kidney failure), and severe infections can all make the exam worse than the brain injury itself. These must be corrected or accounted for.
- Emotional burden: This process is heartbreaking for families. The team must communicate with empathy, avoid medical jargon, and give families time to process.

Structured goals-of-care discussions with family and surrogate decision-makers · Communication and shared decision-making (not a medicine, but a critical component of post-arrest care)

Many people who survive cardiac arrest initially do not wake up, and some will never recover meaningful brain function. Families face impossible questions: Should we keep going? What would the person have wanted? Goals-of-care discussions are structured, compassionate

conversations between the medical team and the patient's family or designated decision-maker. The team explains what happened, what the current condition is, what treatments are being done, what the chances of recovery are (based on neuroprognostication), and what the possible outcomes range from (full recovery, survival with severe disability, death). The family shares what the person valued in life, what they said about being on life support, and what quality of life would be acceptable to them. Together, they decide whether the goal is to continue all aggressive treatments in hope of recovery, to shift toward comfort-focused care (stopping the ventilator, medications, and other invasive treatments and focusing only on keeping the person comfortable and pain-free), or something in between. This is not giving up—it is honoring what the person would have wanted.

1. The team gathers all medical information (exam, EEG, imaging, labs) and formulates a prognosis.
2. A meeting is scheduled with the family. The physician sits down (not standing), makes eye contact, and asks the family to share what they know and what questions they have.
3. The physician explains the situation in plain language. For example: 'After the heart stopped, the brain went without oxygen for several minutes. Even though we got the heart beating again, the brain was injured. We have been doing everything we can—cooling, medicine, breathing support—to give the brain time to heal. We have waited 72 hours and done several tests to see if the brain is recovering. Right now, the tests show [specific findings]. Based on that, we think the chance of a meaningful recovery is [very low / uncertain / fair].'
4. The team asks: 'What did [patient's name] value most in life? Did they ever talk about being on machines or what kind of life would be acceptable to them?'
5. The family shares the person's wishes (sometimes there is a written advance directive or designated healthcare proxy).
6. Together, they explore goals: Is the goal to do everything possible to try for recovery, even if the chance is very small? Is the goal to avoid prolonged suffering and allow a natural death if recovery is not happening? Or is the goal to give it a bit more time and reassess?
7. If the decision is made to shift toward comfort care, the team explains what that means: the ventilator will be removed, medications to support blood pressure will be stopped, and the focus will shift to keeping the person comfortable with pain medicine and sedation. The person will not be left alone; family can be present.
8. If the decision is to continue intensive care, the team supports that and continues aggressive treatment, with plans to reassess in a few days.

SAFETY WATCH

Emotional trauma: These conversations are among the hardest moments of a family's life. The team must be gentle, unhurried, and ready to repeat information because people in shock and grief often cannot absorb it all at once. · Cultural and religious sensitivity: Different cultures and religions have different views on life support, death, and decision-making. The team must ask and respect these values, and involve chaplains or cultural liaisons as needed. · Conflict within the family: Sometimes family members disagree. The team helps mediate and reminds everyone to focus on what the patient would have wanted, not what each family member wants for themselves. · Guilt and second-guessing: Families may feel guilt no matter what they decide. The team reassures them that they are making the best decision they can with the information available, and that they are honoring their loved one.

Red flags

- No breathing or very weak breathing after the heart starts beating again
- Seizures that keep happening or do not stop (status epilepticus)
- Pupils stay wide open and do not shrink when a light is shined in the eyes, lasting more than 72 hours
- Blood pressure drops very low despite medicines and fluids to raise it (refractory shock)
- Body temperature rises above 102°F (38.9°C) or drops below 93°F (34°C) without targeted temperature management

Nursing priorities

1. Keep mean arterial pressure at 65 mmHg or higher using IV fluids first, then add vasopressors like norepinephrine if needed — The brain and heart just survived being starved of oxygen and need strong blood flow now to recover and prevent more cell death
2. Start Targeted Temperature Management by cooling the patient to 32 to 36 degrees Celsius using cooling blankets or ice packs and check core temperature every hour — Cooling slows down the chemical reactions that cause brain cells to die after they have been without oxygen, giving the brain time to heal
3. Adjust the ventilator to keep oxygen saturation between 94 and 98 percent and carbon dioxide between 40 and 45 mmHg, checking blood gases every 4 hours — Too much oxygen creates harmful free radicals that damage recovering brain cells, and wrong carbon dioxide levels change blood flow to the injured brain

4. Watch continuously for seizures by looking for jerking, eye twitching, or staring, and hook up continuous EEG monitoring if available — Seizures after cardiac arrest can be silent or subtle but they increase brain oxygen demand and cause more injury when the brain is already hurt
5. Check neurologic status every hour including pupil size and response to light, movement of arms and legs, and any response to voice or pain — Changes in exam tell us if the brain is getting better or worse and help predict which patients will recover enough to live independently
6. Prepare for urgent cardiac catheterization if the ECG shows ST elevation or if the patient arrested from a shockable rhythm and no other clear cause is found — Opening a blocked heart artery within hours improves survival and protects the heart muscle from permanent damage
7. Monitor and treat blood sugar to keep glucose between 140 and 180 mg/dL, checking every 2 to 4 hours — High blood sugar is toxic to injured brain cells and worsens brain swelling, but low blood sugar starves the brain of fuel it needs to heal
8. After 24 hours of cooling, rewarm the patient slowly at 0.25 to 0.5 degrees per hour and watch for sudden drops in blood pressure or dangerous potassium levels — Fast rewarming causes blood vessels to open up suddenly, dropping blood pressure and releasing potassium from cells which can stop the heart again

Sources

- AHA/ILCOR Guidelines for Post-Cardiac Arrest Care 2020
- Cochrane Systematic Review: Targeted Temperature Management After Cardiac Arrest
- NEJM: Neurologic Prognostication and Outcomes After Cardiac Arrest
- AHA/ACC Advanced Cardiovascular Life Support (ACLS) 2020

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