

Post Cardiac Arrest Care

A plain-English guide for you and your family

WHAT IS HAPPENING

When someone's heart stops and then is brought back to life with CPR or a shock, the biggest danger is not over yet. While the heart was stopped, the brain and heart ran out of oxygen, and that hurts both of them deeply. Even after the heart starts beating again, damaged brain cells swell and die, the heart muscle is stunned and weak, and blood vessels leak fluid everywhere. Doctors now race to protect what is left: they cool the body down to slow further brain damage, they fix whatever caused the heart to stop in the first place (a blocked artery, a dangerous rhythm, low oxygen), they keep blood pressure high enough so the brain gets fed, and they wait to see if the person will wake up. The next 24 to 72 hours are critical. Some people wake up fully; others stay asleep or wake only partly. The team watches heart rhythm, blood pressure, oxygen levels, and brain signs every minute, because one slip can tip the person back into another arrest or leave the brain too damaged to recover. Every treatment—the cooling blanket, the blood-pressure drips, the breathing tube, the heart monitor—buys time for the brain and heart to heal, if they can.

What you might feel

- No response to voice or touch
- Weak or absent breathing
- Very low or very high blood pressure
- Fast or irregular heart rhythm
- Seizures (shaking fits or stiffening)
- Cool, pale, or bluish skin
- Low oxygen level on the monitor (low SpO₂)
- Abnormal posturing (arms or legs stiff in odd positions)
- No pupil reaction to light
- Little or no urine output

Call the doctor right away if...

- 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

What the care team will do

- Keep mean arterial pressure at 65 mmHg or higher using IV fluids first, then add vasopressors like norepinephrine if needed
- 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
- 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
- Watch continuously for seizures by looking for jerking, eye twitching, or staring, and hook up continuous EEG monitoring if available
- Check neurologic status every hour including pupil size and response to light, movement of arms and legs, and any response to voice or pain
- 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
- Monitor and treat blood sugar to keep glucose between 140 and 180 mg/dL, checking every 2 to 4 hours
- 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

Medicines you may be given — and why

Targeted Temperature Management (TTM) at 32-36°C for 24 hours, then slow rewarming

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.

Norepinephrine or vasopressin

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.

Oxygen therapy (target saturation 94-98%)

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.

Ventilator adjustment to keep PaCO₂ at 35-45 mmHg

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).

Propofol or midazolam (sedation) and rocuronium or vecuronium (paralysis)

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.

Regular insulin or rapid-acting insulin (target glucose 140-180 mg/dL)

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.

Levetiracetam, fosphenytoin, or valproate (if seizures occur)

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.

Percutaneous coronary intervention (PCI) with aspirin, ticagrelor or clopidogrel, and heparin (if arrest was caused by heart attack)

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.

Continuous EEG monitoring and anticonvulsants (if nonconvulsive seizures are found)

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.

Multimodal neuroprognostication (clinical exam, EEG, MRI or CT, and biomarkers like NSE) at ≥ 72 hours after rewarming

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.

Structured goals-of-care discussions with family and surrogate decision-makers

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.

Where this information comes from

- 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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