

Postpartum Cardiomyopathy

The heart muscle weakens suddenly in the last month of pregnancy or up to five months after delivery, for no known reason.

THE STORY IN ONE SENTENCE

The heart muscle weakens suddenly in the last month of pregnancy or up to five months after delivery, for no known reason.

“The left ventricle (the heart's main pumping chamber) gets weak in late pregnancy or up to five months after birth, and we do not know why. Treat it like any other heart failure with a weak pump: diuretics to drain extra fluid, beta-blockers to slow the racing heart, and blood-pressure drugs—but AVOID ACE inhibitors and ARBs while pregnant because they harm the baby; use hydralazine and nitrates instead. After delivery, switch to the full heart-failure toolkit including ACE inhibitors or ARBs, and consider bromocriptine to block prolactin, which may be part of the trigger.”

Normal anatomy — the body's job

During pregnancy, the heart has to work much harder than usual to support both mother and baby, pumping about fifty percent more blood every minute, holding more volume, and beating faster—all normal and healthy.

BODY'S JOB

The left ventricle (the heart's main pumping chamber, the thick muscular room on the bottom left) squeezes hard to push oxygen-rich blood out to the whole body. During pregnancy, blood volume swells by thirty to fifty percent, the heart rate climbs ten to twenty beats per minute, and the amount of blood the heart pumps every minute (called cardiac output) rises by fifty percent, all to feed the growing baby and the mother's bigger workload. The heart muscle stretches a bit to hold the extra volume and squeezes harder and faster to keep up. After delivery, these changes reverse over weeks to months as the body returns to its pre-pregnancy state.

1. Oxygen-poor blood flows from the body into the right atrium (top-right room of the heart).
2. The right atrium squeezes and pushes blood into the right ventricle (bottom-right pumping chamber).

3. The right ventricle squeezes and pushes blood through the pulmonary arteries to the lungs, where it picks up oxygen and drops off carbon dioxide.
4. Oxygen-rich blood flows back from the lungs into the left atrium (top-left room).
5. The left atrium squeezes and pushes blood into the left ventricle (the main pumping chamber, bottom-left, with the thickest muscle wall).
6. The left ventricle squeezes powerfully and pushes blood through the aortic valve into the aorta (the body's biggest artery), which branches out to deliver oxygen and fuel to every organ and tissue.
7. During pregnancy, the left ventricle handles fifty percent more volume and pumps fifty percent more blood every minute, stretching and working harder to meet the doubled demand.

What breaks

The left ventricle (the heart's main pumping chamber) suddenly weakens and cannot squeeze hard enough to push blood forward, so blood backs up into the lungs and the body does not get enough oxygen-rich blood.

CLINICAL TERM

Peripartum cardiomyopathy: the left ventricular ejection fraction (the percentage of blood the left ventricle pushes out with each beat) drops below forty-five percent, and often below thirty percent, in the last month of pregnancy or up to five months after delivery, in a woman with no prior heart disease and no other clear cause for heart failure.

How the body compensates

The body tries to keep blood pressure and oxygen delivery steady even as the heart weakens, so early on vital signs can look nearly normal and the mother may seem fine—this is the dangerous middle, because it hides how sick the heart really is until the system crashes.

1. {'what': "The sympathetic nervous system (the body's fight-or-flight system) revs up and releases epinephrine and norepinephrine (adrenaline-type hormones).", 'why': "These hormones speed up the heart rate and squeeze blood vessels tighter to keep blood pressure from falling, buying time while the pump is weak—but over days and weeks, the constant adrenaline overdrive wears the heart out even more."}

2. {'what': 'The kidneys release renin, which triggers a chain (the renin-angiotensin-aldosterone system, or RAAS) that makes the hormone aldosterone; aldosterone tells the kidneys to hold onto salt and water.', 'why': 'The kidneys sense less blood flow and think the body is dehydrated, so they hoard fluid to boost blood volume and pressure—but when the pump is already failing, the extra fluid just makes congestion and swelling worse.'}
3. {'what': 'The heart itself stretches (dilates) to hold more blood in each beat, trying to push out more volume even though each squeeze is weaker (this is called the Frank-Starling mechanism).', 'why': 'A stretched muscle fiber contracts a bit harder, so the heart grows bigger to compensate—but if it stretches too far, the muscle fibers slide past each other and lose all squeezing power, like an overstretched rubber band.'}
4. {'what': 'The heart muscle wall may thicken slightly (hypertrophy) to try to generate more force.', 'why': 'Thicker muscle can squeeze harder—but in a failing heart, the muscle is already sick, so thickening happens unevenly and worsens stiffness and electrical instability.'}
5. {'what': 'Blood gets shunted away from less critical organs (skin, gut, kidneys) and focused on the brain and heart.', 'why': 'The body sacrifices the periphery to protect the core—so you feel cold, clammy, tired, and nauseated, and kidney function starts to drop.'}
6. {'what': 'Breathing gets faster and deeper to pull in more oxygen and blow off carbon dioxide.', 'why': 'Backed-up lungs cannot exchange gas well, so the body speeds up breathing to compensate—but this only helps a little, and eventually the person gets too tired to keep breathing fast.'}
7. {'what': 'All these tricks keep blood pressure and oxygen levels nearly normal for a while, so labs and vital signs may look okay even though the heart is very sick—this is the dangerous window, because the mother, family, and even clinicians can miss how close she is to crashing.', 'why': 'Compensation hides the cliff edge until the body runs out of tricks; once it tips, the crash is sudden—blood pressure drops, oxygen plummets, the heart throws dangerous rhythms, and the window to reverse it closes fast.'}
8. {'what': 'Watch for the pattern over time, not just one snapshot: worsening shortness of breath, new swelling, faster heart rate, dropping oxygen saturation, rising jugular veins, new crackling sounds in the lungs (rales), or new confusion.', 'why': 'A stable-looking patient on a downhill slope is not truly stable—the trend tells you where she is headed, and early treatment (diuretics, beta-blockers, afterload reducers, possibly bromocriptine, sometimes a wearable defibrillator or even a temporary heart pump) can stop the fall before the crash.'}

Domino effect

1. The left ventricle (the heart's main pumping chamber) weakens and cannot squeeze hard enough to push blood forward. — This is the upstream break—no one knows exactly why, but a toxic piece of prolactin, autoimmune attack, viral infection, or the extreme stress of pregnancy on a vulnerable heart are the leading suspects; the ejection fraction (percent of blood pumped out each beat) drops below forty-five percent, often into the twenties.
2. Blood backs up behind the weak pump, first into the left atrium, then into the pulmonary veins, and finally into the lung capillaries, where pressure rises and fluid leaks into the air sacs (pulmonary swelling (edema)). — A clogged pipe backs up—when the left ventricle cannot empty itself, the whole chain upstream fills and overfills; high pressure in the lung capillaries forces water out of the blood and into the air spaces, drowning the lungs from the inside.
3. You feel short of breath, especially when lying flat (orthopnea), and you may wake up gasping at night (paroxysmal nocturnal trouble breathing (dyspnea)). — Fluid in the air sacs blocks oxygen from reaching your blood, so every breath feels like work; lying flat lets gravity spread the fluid across both lungs, making it even worse, so you have to prop yourself up on pillows or sit upright to breathe.
4. Your legs, ankles, and feet swell with pitting swelling (edema) (when you press the skin, it leaves a dent). — The weak pump cannot move blood forward, so blood pools in the veins of your legs; high pressure in those veins pushes fluid out into the surrounding tissue, and gravity pulls it down to your feet and ankles.
5. Your heart races (tachycardia, often ninety to one hundred twenty beats per minute or higher). — The heart senses it is not pushing enough blood with each beat, so the sympathetic nervous system (fight-or-flight) revs the rate up to try to make up the difference—more beats per minute to move the same total volume—but this wears the heart out faster.
6. You feel exhausted, weak, dizzy, and your hands and feet may be cold and pale. — The weak pump cannot deliver enough oxygen-rich blood to your muscles, brain, and skin, so your body runs on fumes; blood flow is shunted away from the periphery to protect the brain and heart, leaving you cold, tired, and lightheaded.
7. Early on, you may look and feel like any pregnant or postpartum woman—a little swollen, a little tired, heart beating fast—so the heart failure hides inside normal pregnancy changes until it gets severe. — Pregnancy already swells your body, speeds your heart, and makes you tired, so the early warning signs of postpartum cardiomyopathy blend in; by the time breathlessness is obvious, the heart may already be very weak and close to crashing.

8. If untreated, the heart can go into shock (cardiogenic shock: blood pressure crashes, organs fail), throw dangerous rhythms (ventricular a fast heart rate (tachycardia) or fibrillation, which can cause sudden cardiac arrest), or form clots that break off and cause stroke or pulmonary embolism (a clot lodging in a lung artery). — A failing heart is a house of cards—once compensation runs out, everything falls at once; low blood flow starves the organs, the sick muscle misfires electrically, and stagnant blood clots, all of which can kill in minutes to hours if not caught and treated.

Symptoms · what the patient feels, and why

- Shortness of breath, especially when lying flat or during activity: The weakened heart muscle cannot pump blood forward well, so fluid backs up into the lungs (like water rising in a backed-up sink). This makes it hard to breathe because the air sacs in the lungs get wet and heavy.
- Swelling in the legs, ankles, and feet: Because the heart is not pumping strongly enough, blood pools in the veins of the legs, and fluid leaks out into the tissues (like water seeping out of a weak hose). This causes puffiness.
- Extreme tiredness and weakness: The heart cannot deliver enough oxygen-rich blood to the muscles and brain, so the body runs low on fuel. It feels like running on empty, even after rest.
- Fast or irregular heartbeat (palpitations): The heart tries to make up for being weak by beating faster or skipping beats. Sometimes the electrical signals in the heart go haywire because the muscle is stretched and stressed.
- Cough that may bring up pink, frothy mucus: Fluid that has backed up into the lungs can leak into the airways. The pink color comes from tiny blood vessels breaking under pressure, mixing with the fluid.
- Chest discomfort or pressure: The heart muscle itself is struggling and may not get enough oxygen (like a cramping muscle), or the fluid buildup puts pressure on the chest from the inside.
- Waking up at night gasping for air: When lying flat, extra fluid shifts into the lungs because gravity is no longer pulling it down into the legs. The person wakes up feeling like they are drowning.
- Trouble concentrating or feeling confused: The brain is not getting enough oxygen-rich blood because the heart is too weak to pump well. This makes thinking slow and foggy, like a computer running on low battery.

Focused exam

- **Hard to breathe when lying flat (orthopnea):** This is the signal that fluid is backing up into the lungs because the heart is too weak to pump blood forward. In postpartum cardiomyopathy, the heart muscle lost its squeeze power, so blood pools backward into the lungs.
- **Swollen legs and feet (edema):** When the heart cannot pump blood out efficiently in postpartum cardiomyopathy, blood backs up into the veins. Fluid leaks out of the vessels into the tissue spaces. Press your thumb on the shin—if the dent stays, fluid is there.
- **Heart racing fast (tachycardia):** The weak heart in postpartum cardiomyopathy is not pumping enough blood to the body. The body panics and tells the heart to beat faster to try to move enough oxygen around. This is the body buying time, but it is a red flag.
- **Crackling sounds at the bottom of the lungs (crackles or crackles in the lungs (rales)):** Fluid from the backed-up circulation has soaked into the lung tissue. When air moves through wet lungs, it makes a crackling sound. This tells you the weak heart in postpartum cardiomyopathy has caused fluid to fill the lungs.
- **Weak pulse and low blood pressure:** The heart muscle in postpartum cardiomyopathy has lost its pump strength. The body cannot push blood hard enough to reach the arms and legs with normal pressure. Low blood pressure means the body is not getting enough oxygen.
- **Enlarged heart on chest X-ray (cardiomegaly):** When the heart gets weak in postpartum cardiomyopathy, it stretches out like a worn-out balloon. The pump chambers get bigger because they cannot empty. The X-ray shows this enlarged silhouette.

Diagnostics · every test answers a question

- **Echocardiogram (ultrasound of the heart) →** Is the heart muscle weak, and how much blood is it squeezing out with each beat?
- **BNP (B-type natriuretic peptide) or NT-proBNP blood test →** Is the heart stretched and struggling under pressure?
- **Chest X-ray →** Is the heart bigger than normal, and is fluid leaking into the lungs?
- **Troponin blood test →** Is the heart muscle being damaged right now?
- **Complete blood count (CBC) →** Is anemia making the heart work even harder?
- **Thyroid-stimulating hormone (TSH) and free T4 →** Could thyroid disease be causing or worsening the heart symptoms?
- **Electrocardiogram (ECG or EKG) →** Is the heart rhythm normal, or is there a dangerous irregular beat?

Treatment logic

- Loop diuretic (furosemide) to remove excess fluid → Removes the extra water the body is holding onto by making the kidneys push more urine out. This shrinks the swelling in the legs and pulls fluid out of the lungs, so breathing when lying flat gets easier. Works fast, often within hours.
- ACE inhibitor (lisinopril) or ARB (losartan) or ARNI (sacubitril/valsartan) after delivery → Relaxes the blood vessels so the weak heart does not have to push as hard to move blood forward. ACE inhibitors and ARBs block signals (angiotensin II) that squeeze vessels tight. ARNI also boosts a natural peptide (BNP) that opens vessels and removes salt. This lowers the pressure inside the heart and stops it from stretching further. Over months, this can help the heart recover some strength.
- Beta-blocker (metoprolol or carvedilol) → Slows the heart rate and reduces how hard the heart contracts each beat by blocking adrenaline (epinephrine and norepinephrine). This gives the weak muscle more time to fill with blood between beats and less stress on each squeeze. Over time, this protects the heart from further damage and can improve ejection fraction.
- Mineralocorticoid receptor antagonist (spironolactone or eplerenone) → Blocks aldosterone (a hormone from the adrenal glands that tells the kidneys to hold onto salt and water). This reduces the total fluid load the heart has to pump and prevents scarring of the heart muscle. Helps the body pee out extra water without losing too much potassium.
- SGLT2 inhibitor (dapagliflozin or empagliflozin) → Helps the kidneys remove sugar and water in the urine, which lowers fluid overload. Also shifts the heart's fuel source to burn fat (ketones) instead of just sugar, which is more efficient. This reduces stress on the weak heart and improves survival in heart failure. Works even if the patient does not have diabetes.
- Anticoagulation (warfarin or a direct oral anticoagulant like apixaban) if ejection fraction is below 30% or if a blood clot is seen in the heart → Thins the blood to prevent clots from forming inside the sluggish heart chambers. When the heart is too weak to squeeze hard, blood sits still and can clot. Anticoagulation stops these clots from traveling to the lungs (pulmonary a traveling clot lodging in a vessel (embolism)) or brain (stroke).
- Bromocriptine (a dopamine agonist) started early, within the first weeks after diagnosis → Blocks prolactin (the hormone that triggers milk production). Some studies show that high prolactin after delivery may damage the heart muscle by breaking into harmful fragments. Bromocriptine may help the heart recover faster and improve ejection fraction. Also stops breastfeeding, which may reduce the heart's workload.

Medications

Metoprolol or carvedilol (beta-blockers) · Beta-adrenergic receptor blocker

These drugs block adrenaline (the body's 'go-faster' hormone) from locking onto special docking sites (beta receptors) on the heart muscle. When adrenaline can't dock, the heart slows down and pumps less forcefully, which gives the tired, stretched heart muscle a chance to rest and recover. Over weeks to months, the heart muscle can remodel itself—shrinking back toward normal size and pumping better. Beta-blockers also lower blood pressure and reduce dangerous irregular heartbeats.

1. Adrenaline (and noradrenaline) normally binds to beta receptors on the heart and tells it to beat harder and faster.
2. The beta-blocker sits in the receptor's docking site and blocks adrenaline from landing there.
3. Without adrenaline's 'speed up' signal, the heart rate slows and each beat is gentler.
4. The heart muscle uses less oxygen and energy, reducing strain on the weakened pump.
5. Over time, the heart's stretch signals (neurohormones) dial down, and the muscle can reverse some of the harmful overgrowth and stiffness.
6. Blood pressure drops because the heart pumps less forcefully and the kidneys release less renin (a hormone that raises blood pressure).

SAFETY WATCH

Start at a very low dose and increase slowly every 1–2 weeks; jumping to a full dose too fast can make heart failure worse at first. · Watch for a heart rate that drops too low (below 50–55 beats per minute while awake), dizziness when standing, or extreme fatigue. · Do NOT stop suddenly—the heart can rebound with dangerous fast rhythms or worsening failure; taper off slowly under a doctor's watch. · Carvedilol also blocks alpha receptors (widening blood vessels), so dizziness and low blood pressure may be more common than with metoprolol. · Rarely, beta-blockers can worsen asthma by tightening airways; tell your doctor if you have lung disease. · Safe during breastfeeding in usual heart-failure doses; tiny amounts pass into milk but are not harmful to the baby.

Hydralazine plus isosorbide dinitrate (combination vasodilators, safe during pregnancy and breastfeeding) · Direct arterial vasodilator (hydralazine) plus nitric-oxide donor (isosorbide dinitrate)

These two drugs work as a team to relax and widen blood vessels—like opening more lanes on a highway so blood flows more easily. Hydralazine relaxes the muscle rings around arteries (the vessels that carry blood away from the heart), lowering the pressure the heart has to

pump against. Isosorbide dinitrate releases nitric oxide (a natural gas signal in the body) that relaxes veins (the vessels that return blood to the heart), reducing the amount of blood pooling in the lungs and body. Together they cut the heart's workload from both ends. This combination is especially important during pregnancy because the safer long-term drugs (ACE inhibitors and ARBs) can harm the developing baby.

1. Hydralazine enters the smooth muscle cells around arteries and triggers chemical changes that tell the muscle to relax and let the vessel widen.
2. Wider arteries mean lower resistance, so the heart can push blood forward more easily and blood pressure drops.
3. Isosorbide dinitrate breaks down in the body to release nitric oxide, a gas that the blood vessel lining normally makes to keep vessels relaxed.
4. Nitric oxide tells the muscle in veins to relax, so veins expand and hold more blood; this reduces the flood of blood returning to the heart.
5. With less blood rushing back, pressure in the lungs drops and fluid is less likely to leak into lung air sacs (reducing shortness of breath).
6. Together, the heart pumps against lower pressure (forward) and receives less volume (backward), so it works less hard and can recover.

SAFETY WATCH

Hydralazine can cause headache, fast heartbeat, and fluid retention (swelling); the nitrate and a diuretic usually balance the last two. · Isosorbide dinitrate often causes headache in the first few days; this usually fades as the body adjusts. Taking acetaminophen (Tylenol) can help. · Take nitrates on a schedule that includes an 8–12 hour nitrate-free window each day (often overnight) to prevent tolerance (the drug stops working). · Check blood pressure regularly; if it drops too low you may feel dizzy or faint when standing. · Very rarely, hydralazine can trigger a lupus-like syndrome (joint pain, rash, fever) with long-term high-dose use; tell your doctor if these appear. · Both drugs are safe during pregnancy and breastfeeding and are first-line therapy when ACE inhibitors cannot be used.

Furosemide or bumetanide (loop diuretics, 'water pills') · Loop diuretic

These drugs tell the kidneys to pull extra salt and water out of the blood and send it to the bladder as urine—like opening a drain to lower the water level in an overflowing sink. When the weakened heart can't pump well, fluid backs up into the lungs (causing shortness of breath) and into the legs, belly, and liver (causing swelling). Loop diuretics quickly remove that extra fluid, relieving congestion and making it easier to breathe and move. They act on a

specific part of the kidney's filtering tube (the loop of Henle) where most salt reabsorption happens.

1. The drug travels through the blood to the kidneys and enters the urine inside the filtering tubes.
2. It blocks the NKCC2 transporter, a tiny pump that normally pulls sodium, potassium, and chloride (salts) back into the blood.
3. With the pump blocked, those salts stay in the urine instead of being reabsorbed.
4. Water follows the salts (water moves toward higher salt concentration), so more water stays in the urine too.
5. The kidneys produce much more urine than usual, and the extra fluid leaves the body.
6. Fluid pressure in the veins and lungs drops, swelling shrinks, and breathing becomes easier.

SAFETY WATCH

Use the lowest dose that keeps you comfortable; too much can dehydrate you, drop blood pressure dangerously, and harm the kidneys. · Loop diuretics wash out potassium and magnesium in the urine; your doctor will check blood levels and may add supplements or a potassium-sparing diuretic. · Watch for dizziness, extreme thirst, muscle cramps, or confusion—signs of too much fluid loss or low electrolytes. · Weigh yourself every morning (after urinating, before eating) and call your doctor if you gain more than 2–3 pounds in a day or 5 pounds in a week (fluid is building up again). · If you are breastfeeding, high doses may slightly reduce milk supply; use the smallest effective dose and monitor the baby's weight gain. · Bumetanide is about 40 times stronger than furosemide, so doses look much smaller but work the same way.

Digoxin · Cardiac glycoside (positive inotrope and AV nodal blocker)

Digoxin makes each heartbeat a little stronger by shifting the balance of sodium and calcium inside heart muscle cells—like turning up the volume on a weak speaker. It blocks a pump (the sodium-potassium ATPase) on the cell surface, which causes sodium to build up inside the cell. Higher sodium forces another transporter to push out less calcium, so calcium stays in the cell longer. Extra calcium strengthens the squeeze of each heartbeat. Digoxin also slows electrical signals through the heart's relay station (the AV node), which helps control a fast or irregular heart rhythm (such as atrial fibrillation (an irregular, quivering heartbeat)) that often accompanies heart failure.

1. Digoxin binds to and blocks the sodium-potassium ATPase, a pump that normally pushes sodium out of the cell and pulls potassium in.
2. With the pump slowed, sodium builds up inside the heart muscle cell.

3. The sodium-calcium exchanger (another pump) tries to push the extra sodium out, but to do that it must bring calcium back in instead of pumping it out.
4. Calcium accumulates in the cell, and more calcium is available when the cell contracts.
5. The stronger calcium signal makes the muscle fibers squeeze harder, so each heartbeat ejects a bit more blood.
6. In the AV node, digoxin increases vagal tone (the calming brake nerve from the brain) and directly slows electrical conduction, which keeps a fast atrial rhythm from racing the ventricles.

SAFETY WATCH

Digoxin has a narrow margin between a helpful dose and a toxic dose; blood levels must be checked regularly (target 0.5–0.9 ng/mL in heart failure). · Toxic levels cause nausea, vomiting, confusion, seeing yellow or green halos around lights, and dangerous heart rhythms (including very slow or very fast irregular beats). · Low potassium or low magnesium makes digoxin toxicity much more likely, so electrolytes must be monitored closely—especially if you are on a diuretic. · Kidney function affects how the body clears digoxin; dose must be lowered if kidney numbers worsen. · Many drugs interact with digoxin (raising or lowering its level); tell every prescriber and pharmacist you are taking it. · Digoxin passes into breast milk in small amounts; it is usually considered safe during breastfeeding at standard doses, but the baby should be monitored.

Enalapril, lisinopril, or other ACE inhibitor (AFTER delivery only—never during pregnancy) · Angiotensin-converting enzyme (ACE) inhibitor

ACE inhibitors block an enzyme (ACE) that turns a weak hormone (angiotensin I) into a powerful one (angiotensin II). Angiotensin II squeezes blood vessels tight, raises blood pressure, tells the adrenal glands to release aldosterone (a hormone that makes the kidneys hold onto salt and water), and over time causes the heart muscle to thicken and scar. By blocking ACE, these drugs let blood vessels stay relaxed, reduce fluid retention, and slow harmful remodeling of the heart. In large studies, ACE inhibitors help people with heart failure live longer, stay out of the hospital, and feel better. However, they can cause severe birth defects and must NEVER be used during pregnancy.

1. The kidneys sense low blood flow and release renin, an enzyme that starts a hormone chain.
2. Renin clips a liver protein to make angiotensin I, a weak molecule.
3. ACE (mostly in the lungs) converts angiotensin I into angiotensin II, a very strong hormone.
4. Angiotensin II tightens blood vessels, raises blood pressure, and triggers aldosterone release from the adrenal glands; aldosterone makes the kidneys save salt and water.
5. The ACE inhibitor blocks step 3, so angiotensin II levels drop.

6. With less angiotensin II, blood vessels relax, blood pressure falls, the kidneys release more salt and water, and the heart muscle receives fewer 'grow and scar' signals.
7. Over months, the heart can shrink back toward normal size and pump more efficiently.

SAFETY WATCH

NEVER use during pregnancy—ACE inhibitors cause kidney failure, skull defects, and death in the developing baby, especially in the second and third trimesters. If you become pregnant, stop immediately and call your doctor. · Safe during breastfeeding; only tiny amounts enter breast milk, and enalapril and captopril are preferred ACE inhibitors for nursing mothers. · Watch for a dry, tickling cough (happens in 5–10% of people); it is harmless but annoying and may require switching to an ARB. · Check kidney function and potassium within 1–2 weeks of starting; ACE inhibitors can raise potassium and may temporarily reduce kidney filtration (usually acceptable if mild). · Rarely causes angioedema (sudden swelling of the lips, tongue, or throat that can block breathing); if this happens, stop the drug and go to the emergency room immediately. · Do NOT use potassium supplements or salt substitutes (which contain potassium) without asking your doctor—ACE inhibitors already raise potassium, and adding more can cause dangerous heart rhythms.

Losartan, valsartan, or other ARB (angiotensin receptor blocker; AFTER delivery only—never during pregnancy) · Angiotensin II receptor blocker (ARB)

ARBs block the docking site (the AT1 receptor) where angiotensin II lands to do its work. Even though angiotensin II is still made, it cannot squeeze blood vessels, release aldosterone, or send 'grow and scar' signals to the heart because it is locked out of its receptor. The effect is very similar to an ACE inhibitor—relaxed vessels, lower blood pressure, less fluid retention, and better heart remodeling—but ARBs rarely cause the dry cough seen with ACE inhibitors. They are used when someone cannot tolerate an ACE inhibitor or sometimes combined with one (though combination therapy is less common now that newer drugs are available). Like ACE inhibitors, ARBs improve survival and reduce hospitalizations in heart failure, but they must NEVER be used during pregnancy.

1. The body makes angiotensin II (either through ACE or backup enzymes like chymase).
2. Angiotensin II travels to its target cells and tries to bind to the AT1 receptor.
3. The ARB sits in the receptor and blocks angiotensin II from docking.
4. Without the signal, blood vessels stay relaxed instead of squeezing tight.
5. The adrenal glands release less aldosterone, so the kidneys let go of more salt and water.
6. Heart muscle cells receive fewer signals to grow abnormally or lay down scar tissue.

7. Blood pressure drops, fluid overload eases, and the heart's workload shrinks.

SAFETY WATCH

NEVER use during pregnancy—ARBs cause the same severe birth defects as ACE inhibitors (kidney failure, skull malformations, fetal death). Stop immediately if pregnancy is detected. · Safe during breastfeeding; very low levels appear in milk, and many ARBs are compatible with nursing. · Check kidney function and potassium 1–2 weeks after starting, just like with ACE inhibitors. · Do NOT combine with ACE inhibitors unless a specialist recommends it; the combination raises the risk of kidney injury and high potassium without extra benefit in most people. · Rarely causes angioedema (swelling of lips, tongue, throat), though much less often than ACE inhibitors. · Avoid potassium supplements and salt substitutes unless your doctor approves.

Sacubitril/valsartan (ARNI: angiotensin receptor-neprilysin inhibitor; AFTER delivery only) · Angiotensin receptor-neprilysin inhibitor (ARNI)

This drug combines an ARB (valsartan) with a neprilysin inhibitor (sacubitril) in one pill. Neprilysin is an enzyme that breaks down helpful natural peptides—BNP, ANP, and bradykinin—which relax blood vessels, help the kidneys release salt and water, and protect the heart. By blocking neprilysin, sacubitril lets these good peptides hang around longer and keep working. Valsartan blocks angiotensin II (the harmful hormone), while sacubitril boosts the helpful ones. The result is stronger blood-vessel relaxation, better fluid removal, and improved heart remodeling compared to an ACE inhibitor or ARB alone. Large trials show sacubitril/valsartan reduces death and hospitalization more than enalapril in people with heart failure and low ejection fraction, so current guidelines recommend it as first-line therapy after delivery.

1. Valsartan blocks the AT1 receptor so angiotensin II cannot tighten blood vessels or release aldosterone.
2. Sacubitril is converted in the body to sacubitrilat, which blocks the neprilysin enzyme.
3. Neprilysin normally chops up BNP, ANP, and bradykinin (natural hormones that relax vessels and promote urination).
4. With neprilysin blocked, these peptides stay active longer.
5. BNP and ANP bind to receptors on blood vessels and kidney cells, telling them to relax and release salt and water.
6. Bradykinin also relaxes blood vessels and has anti-inflammatory effects.
7. The combined effect—blocking the bad hormone (angiotensin II) and preserving the good ones—leads to lower blood pressure, less fluid buildup, and better heart recovery.

SAFETY WATCH

NEVER use during pregnancy—valsartan (the ARB component) causes severe fetal harm, just like other ARBs and ACE inhibitors. · Do NOT start sacubitril/valsartan within 36 hours of taking an ACE inhibitor; the combination sharply raises the risk of angioedema (dangerous throat swelling). · Safe during breastfeeding according to expert consensus, though data are limited; discuss with your cardiologist and pediatrician. · Watch for low blood pressure, dizziness, and high potassium, just as with ARBs. · Angioedema is rare but more common in Black patients and anyone with a history of ACE-inhibitor-related swelling. · Check kidney function and potassium 1–2 weeks after starting and whenever the dose is increased. · The pill is more expensive than generic ACE inhibitors or ARBs; insurance prior authorization may be needed.

Spirolactone or eplerenone (mineralocorticoid receptor antagonists, MRAs)**· Mineralocorticoid receptor antagonist (MRA, aldosterone blocker)**

These drugs block aldosterone, a hormone made by the adrenal glands that tells the kidneys to hold onto salt and water and dump potassium. In heart failure, aldosterone levels stay high even when they should not be, driving fluid retention, high blood pressure, and scarring (fibrosis) of the heart muscle. MRAs sit on the aldosterone receptor in the kidney and heart and prevent aldosterone from landing. This lets the kidneys release more salt and water (acting like a mild diuretic), keeps potassium in the body, and—most importantly—reduces harmful remodeling and scarring in the heart. Studies show MRAs improve survival in heart failure when added to ACE inhibitors (or ARBs) and beta-blockers. Spirolactone is older and cheaper but can cause breast tenderness and enlargement in men and women; eplerenone is more selective and causes fewer hormone-related side effects.

1. The adrenal glands release aldosterone in response to angiotensin II, high potassium, or low sodium.
2. Aldosterone binds to mineralocorticoid receptors in kidney cells and tells them to reabsorb sodium and water and dump potassium into the urine.
3. The MRA blocks the receptor so aldosterone cannot deliver its message.
4. The kidneys let go of more sodium and water (reducing swelling and blood pressure) and keep more potassium.
5. In the heart and blood vessels, blocking aldosterone reduces signals that cause collagen buildup (fibrosis) and inflammation.
6. Over time, the heart muscle becomes less stiff and scarred, and the pump can work more efficiently.

SAFETY WATCH

MRAs raise potassium, which is helpful if you are on a loop diuretic (which lowers potassium), but dangerous if potassium climbs too high (above 5.5 mEq/L). High potassium can cause life-threatening heart rhythms. · Check potassium and kidney function 3–7 days after starting, then regularly (at least every few months). · Do NOT use potassium supplements or salt substitutes (which are potassium chloride) while on an MRA unless your doctor specifically tells you to. · Do NOT combine with other potassium-sparing diuretics (amiloride, triamterene) or very high doses of ACE inhibitors or ARBs without close monitoring. · Spironolactone can cause breast swelling and tenderness (gynecomastia) in men and irregular periods in women because it weakly blocks other hormone receptors; eplerenone does not do this. · Safe to use during breastfeeding in low doses; spironolactone passes into milk in tiny amounts and is considered compatible by most experts. · Use with caution if kidney function is reduced; MRAs can make it worse or push potassium dangerously high.

Dapagliflozin, empagliflozin, or other SGLT2 inhibitor · Sodium-glucose cotransporter-2 (SGLT2) inhibitor

SGLT2 inhibitors were first designed to lower blood sugar in diabetes, but researchers discovered they also powerfully protect the heart and kidneys in people with or without diabetes. These drugs block a transporter (SGLT2) in the kidney that reabsorbs glucose and sodium from the urine back into the blood. When SGLT2 is blocked, glucose and sodium spill into the urine and are lost, which lowers blood sugar (if it is high) and causes a gentle, sustained loss of salt and water—like a mild, steady diuretic. This reduces blood pressure and fluid overload. Beyond that, SGLT2 inhibitors shift the heart's fuel use toward more efficient pathways (burning ketones and fat instead of only glucose), reduce inflammation, protect kidney filtering units, and improve the stiffness of the heart muscle. In major trials, they cut heart-failure hospitalizations and death even when added to all the standard drugs. Current guidelines recommend them for everyone with heart failure and low ejection fraction, regardless of whether they have diabetes.

1. The kidney filters blood and makes a preliminary urine, which contains glucose and sodium that the body wants to keep.
2. The SGLT2 transporter in the kidney tube normally pulls glucose and sodium back into the blood.
3. The SGLT2 inhibitor blocks this transporter, so glucose and sodium stay in the urine.
4. The kidneys excrete extra glucose (lowering blood sugar if it is elevated) and extra sodium and water (acting like a mild diuretic).
5. Lower sodium and water levels reduce blood volume, blood pressure, and the heart's workload.

6. The heart muscle shifts to burning ketones (a cleaner fuel made from fat) instead of relying only on glucose, which improves energy efficiency and reduces oxidative stress (harmful chemical reactions).
7. Inflammation, fibrosis, and stiffness in the heart and kidneys decrease over time, and the organs function better.

SAFETY WATCH

Safe during breastfeeding according to limited data; very low levels reach breast milk, and the drug is not absorbed well by infants. · Because these drugs cause mild fluid loss, start them when you are well-hydrated and not on very high diuretic doses (risk of dehydration and low blood pressure). · Watch for yeast infections of the genitals (vaginal yeast or balanitis in men) because sugar in the urine feeds yeast; treat with standard antifungal creams and keep the area clean and dry. · Rarely (about 1 in 1,000 to 1 in 5,000 people) SGLT2 inhibitors cause euglycemic diabetic ketoacidosis (the acid crisis of missing insulin)—a dangerous acid buildup in the blood even when blood sugar is normal. Stop the drug and seek emergency care if you have nausea, vomiting, extreme fatigue, or trouble breathing. · Check kidney function before starting; SGLT2 inhibitors work best when kidney filtration (eGFR) is above 20–25 mL/min. They can be continued even as kidney function declines because they protect the kidneys long-term. · Do NOT stop suddenly if you feel better—heart and kidney protection builds over months and disappears if the drug is stopped.

Bromocriptine (sometimes used, especially if prolactin is elevated or breastfeeding is not planned) · Dopamine agonist (prolactin inhibitor)

Bromocriptine lowers prolactin, the hormone that drives milk production. Some data suggest that high prolactin may contribute to postpartum cardiomyopathy in certain women by affecting heart metabolism or blood vessel function, though the mechanism is not fully understood and remains debated. Bromocriptine binds to dopamine receptors in the pituitary gland (the pea-sized hormone control center at the base of the brain) and mimics dopamine, the brain chemical that naturally suppresses prolactin. When prolactin drops, milk production stops. Some small studies and case reports suggest bromocriptine may speed heart recovery in women with postpartum cardiomyopathy, especially if started early. However, large randomized trials are lacking, and guidelines do not universally recommend it. Bromocriptine is most often considered when the heart is severely weakened, prolactin is unusually high, or the woman chooses not to breastfeed. It is NOT standard for all cases.

1. Lactotroph cells in the pituitary normally release prolactin into the blood.
2. Dopamine from the hypothalamus (the brain region above the pituitary) binds to D2 receptors on lactotrophs and tells them to stop releasing prolactin.

3. Bromocriptine mimics dopamine and locks onto the same receptors, amplifying the 'stop' signal.
4. Prolactin levels drop sharply, and milk production decreases or stops within days.
5. Some researchers believe high prolactin may worsen heart function by affecting calcium handling in heart cells or promoting blood vessel dysfunction; lowering it may reverse those effects, though evidence is not definitive.
6. In small studies, women treated with bromocriptine had faster improvement in ejection fraction (the heart's pumping strength), but whether bromocriptine directly heals the heart or simply correlates with recovery is unclear.

SAFETY WATCH

Bromocriptine stops milk production, so it is NOT compatible with breastfeeding. If you want to nurse, do not take this drug. · Common side effects include nausea, dizziness, headache, and fatigue, especially in the first few days; taking the dose at bedtime with food helps. · Rarely causes low blood pressure (especially when standing), fainting, hallucinations, or compulsive behaviors (gambling, shopping, hypersexuality) at high doses used for other conditions (doses for lactation suppression are much lower). · Very rarely linked to serious cardiovascular events (heart attack, stroke) when used for lactation suppression in women with pre-existing heart or vessel disease; the FDA withdrew approval for routine lactation suppression in the 1990s, but some specialists still use it off-label in postpartum cardiomyopathy after careful counseling. · Discuss risks and benefits with both your cardiologist and obstetrician; shared decision-making is essential.

Anticoagulation (apixaban, rivaroxaban, warfarin, or enoxaparin) if ejection fraction is very low (below 30%) or if a clot or atrial fibrillation (an irregular, quivering heartbeat) is present · Anticoagulant (blood thinner)

When the heart muscle is very weak and pumps poorly (ejection fraction below 30–35%), blood sloshes slowly in the heart chambers instead of being pushed out efficiently—like water sitting still in a bucket instead of flowing through a hose. Slow-moving blood can clot, forming a blood clot (thrombus) (clot) inside the heart. If a piece breaks off, it can travel to the brain (causing a stroke), lungs (pulmonary a traveling clot lodging in a vessel (embolism)), kidneys, or legs. Anticoagulants interfere with the clotting cascade—a chain of proteins in the blood that normally form clots to stop bleeding. By blocking one or more steps in that chain, these drugs keep the blood thinner and prevent clots from forming. They do NOT dissolve clots that are already there (thrombolytics do that), but they stop new ones. Warfarin blocks vitamin K, which several clotting factors need to be made. Direct oral anticoagulants (DOACs) like apixaban and rivaroxaban directly block specific clotting proteins (factor Xa or thrombin).

Enoxaparin (low-molecular-weight heparin) is injected and used short-term or when pills cannot be taken.

1. Blood clotting involves a cascade: inactive clotting factors are activated one by one, eventually forming fibrin threads that trap blood cells into a solid clot.
2. Warfarin enters the liver and blocks vitamin K epoxide reductase, an enzyme that recycles vitamin K. Without active vitamin K, factors II, VII, IX, and X cannot be made, so the clotting cascade stalls.
3. DOACs (apixaban, rivaroxaban) bind directly to factor Xa, a key protein late in the cascade, and prevent it from converting prothrombin to thrombin (the enzyme that makes fibrin).
4. Enoxaparin binds to antithrombin (a natural brake on clotting) and makes it work much faster, which shuts down factor Xa and thrombin.
5. With fewer active clotting factors, the blood stays fluid and clots do not form easily.
6. Existing slow flow or damaged heart lining cannot trigger a clot, reducing the risk of stroke and a traveling clot lodging in a vessel (embolism).

SAFETY WATCH

The main risk is bleeding—from minor (nosebleeds, heavy periods, bruising) to major (brain bleed, gut bleed). Report any unusual bleeding, black or bloody stools, severe headache, or uncontrolled bleeding immediately. · Warfarin requires frequent blood tests (INR) to keep the dose in a narrow safe range (usually 2.0–3.0); too low and you are not protected, too high and you bleed. Diet (especially leafy greens high in vitamin K), other drugs, and illness affect warfarin levels. · DOACs do NOT require routine blood monitoring and have fewer food and drug interactions, but they are more expensive and cannot be easily reversed if major bleeding occurs (though reversal agents now exist for some DOACs). · Warfarin is generally avoided during breastfeeding in the first few weeks (low levels in milk, but theoretical risk), though some sources consider it compatible after the newborn period; DOACs are NOT recommended during breastfeeding because data are insufficient. Enoxaparin does NOT enter breast milk and is safe while nursing. · If you are on an anticoagulant and planning a procedure, spinal/epidural anesthesia, or if you become injured, tell all providers—anticoagulation must be managed carefully to avoid dangerous bleeding. · Do NOT stop an anticoagulant suddenly without your doctor's instruction; clot risk can rebound. · Pregnancy and postpartum status increase clotting risk on their own, so careful balancing of clot prevention and bleeding risk is critical—your care team will choose the safest agent and monitor closely.

Red flags

- Sudden, severe shortness of breath or gasping for air (may mean fluid is flooding the lungs fast)
- Chest pain or pressure that does not go away (could mean the heart itself is starving for oxygen or another emergency)
- Fainting or near-fainting (the brain is not getting enough blood, which can be dangerous)
- Coughing up pink or bloody foam (sign of severe fluid backup in the lungs)
- New confusion, slurred speech, or one-sided weakness (could mean a stroke from a blood clot that formed in the weak heart)

Nursing priorities

1. Monitor continuous pulse oximetry and keep oxygen saturation above 92 percent with supplemental oxygen if needed — The weak heart cannot pump enough oxygen-rich blood to the body, and low oxygen levels mean organs are not getting enough blood flow
2. Place patient in high Fowler position (sitting up at 60 to 90 degrees) or position of comfort — Sitting upright uses gravity to pull fluid down from the lungs into the legs, making breathing easier when the heart is too weak to clear lung fluid
3. Measure strict intake and output every shift and weigh patient daily at the same time with same scale — Tracking fluid balance shows if diuretics (water pills) are working to remove extra fluid, and sudden weight gain means fluid is building up before symptoms start
4. Place patient on continuous cardiac monitoring and watch for ventricular arrhythmias or atrial fibrillation (an irregular, quivering heartbeat) — Stretched weak heart muscle becomes electrically unstable and can develop life-threatening irregular rhythms like ventricular a fast heart rate (tachycardia)
5. Check blood pressure before giving each dose of ACE inhibitor, ARB, ARNI, or beta-blocker and hold if systolic below 90 — These medicines lower blood pressure to reduce heart workload, but if pressure drops too low the brain and kidneys will not get enough blood
6. Assess for signs of blood clots including sudden chest pain, one-sided leg swelling, or new confusion — Blood pools in the weak heart chambers and forms clots that can travel to lungs causing pulmonary embolism (a clot lodging in a lung artery) or to brain causing stroke
7. Monitor potassium and creatinine labs before and during treatment with diuretics and spironolactone — Loop diuretics wash out potassium causing dangerous low levels, while spironolactone holds potassium causing dangerous high levels, and both can damage kidneys

8. Educate patient to avoid NSAIDs like ibuprofen for postpartum pain and use acetaminophen instead — NSAIDs (non-steroidal anti-inflammatory drugs) make the body hold salt and water which worsens heart failure fluid overload and blocks the helpful effects of ACE inhibitors

Sources

- ESC Heart Failure Guidelines 2021 (European Society of Cardiology)
- AHA/ACC Heart Failure Guideline 2022
- Cochrane Review on Peripartum Cardiomyopathy
- ACOG Practice Bulletin on Cardiac Disease in Pregnancy

Last reviewed: 2026-07-07