

Cardiomyopathy

A plain-English guide for you and your family

WHAT IS HAPPENING

Your heart is a muscular bag the size of your fist. Normally, the muscle walls squeeze hard and then relax completely to pump blood through your body. Cardiomyopathy means the heart muscle itself is sick. Sometimes the muscle stretches out and gets weak (dilated cardiomyopathy)—the heart balloons like an overstretched rubber band and cannot squeeze hard. Sometimes the muscle grows too thick (hypertrophic cardiomyopathy)—the walls are bulky and stiff, so they block blood from flowing out and cannot relax enough to let blood in. Sometimes the muscle or the sac around it turns stiff (restrictive cardiomyopathy)—the heart cannot stretch to fill, like a clenched fist that will not open. Sometimes scar tissue and fat replace the muscle in the right side (ARVC)—that side weakens and the electrical signals get scrambled. Sometimes sudden stress makes the tip of the heart balloon out and freeze (takotsubo)—it looks like a broken heart, literally. And sometimes proteins, iron, or other stuff piles up inside the muscle (infiltrative cardiomyopathy)—the heart gets heavy and stiff. The causes are many: genes you were born with, damage from a heart attack, alcohol, chemotherapy drugs (especially doxorubicin), viruses, pregnancy, or diseases like amyloidosis, sarcoidosis, and hemochromatosis. Sometimes we never find the cause (idiopathic). No matter the cause, the end result is heart failure—your heart cannot pump enough blood for your body. You feel short of breath, tired, dizzy, or notice your legs swelling. Some types also cause dangerous irregular heartbeats that can lead to sudden death. Each type looks different on tests and needs different treatment—medicines, devices, or sometimes a transplant—so figuring out which type you have is the first step.

What you might feel

- Shortness of breath, especially when lying flat or during activity
- Swelling in the legs, ankles, or belly
- Extreme tiredness and weakness
- Chest pain or pressure
- Fast, pounding, or fluttering heartbeat (palpitations)
- Fainting or feeling lightheaded

- Coughing, especially at night or when lying down
- Family history of sudden death or heart failure at a young age

Call the doctor right away if...

- Sudden fainting without warning, especially during exercise
- New or worsening chest pain that does not go away with rest
- Severe shortness of breath at rest or gasping for air
- Rapid weight gain (2-3 pounds in one day or 5 pounds in one week) from fluid buildup
- Rapid or very irregular heartbeat with dizziness or near-fainting

What the care team will do

- Monitor oxygen saturation continuously and keep SpO2 above 90% with supplemental oxygen if needed.
- Weigh the patient daily at the same time in similar clothing and report gains of 2-3 pounds in one day or 5 pounds in one week immediately.
- Place the patient in high Fowler position (sitting upright at 60-90 degrees) or a position of comfort that eases breathing.
- Monitor intake and output strictly, restricting fluids to 1.5-2 liters per day if ordered, and limit sodium to less than 2 grams daily.
- Administer prescribed diuretics (furosemide or bumetanide) on time, monitor potassium and magnesium levels, and watch urine output closely.
- Monitor continuous telemetry (heart rhythm monitor) for arrhythmias, especially atrial fibrillation (an irregular, quivering heartbeat), ventricular a fast heart rate (tachycardia), or heart block.
- Assess for signs of poor blood flow (perfusion) (circulation): cool extremities, delayed capillary refill longer than 3 seconds, low blood pressure, confusion, or decreased urine output below 30 mL per hour.
- Teach the patient to balance rest with light activity, avoid Valsalva maneuvers (straining during bowel movements), and recognize worsening symptoms that need immediate medical attention.

Medicines you may be given — and why

Sacubitril-valsartan (an ARNI)

This drug does two jobs at once. First, it blocks angiotensin receptors (stopping a hormone that squeezes blood vessels tight and makes the heart work harder). Second, it stops

neprilysin, an enzyme that normally breaks down helpful peptides—so now those helpful peptides (like BNP) can stay around longer and open blood vessels, help the kidneys get rid of salt and water, and ease the load on the heart. Together, these two actions lower blood pressure and reduce the strain on a weakened, stretched heart muscle.

Metoprolol or carvedilol or bisoprolol

These drugs block beta receptors on the heart, which are docking spots for adrenaline (the 'fight-or-flight' hormone). Normally, adrenaline makes the heart beat faster and squeeze harder. In a failing or thickened heart, that extra stress wears the muscle out over time. By blocking adrenaline, beta-blockers slow the heart rate, lower blood pressure, and give the heart more time to fill and rest between beats—protecting it from constant overdrive and helping it pump more efficiently in the long run.

Spirolactone or eplerenone

These drugs block aldosterone, a hormone that tells the kidneys to hold onto salt and water and push out potassium. In heart failure, the body makes too much aldosterone, which causes fluid to pile up (swelling, lung congestion) and scars the heart muscle over time. By blocking aldosterone receptors in the kidneys and heart, MRAs help the body get rid of extra salt and water (reducing swelling and lung fluid) and prevent harmful scarring and stiffening of the heart.

Dapagliflozin, empagliflozin, or sotagliflozin

These drugs block a transporter in the kidney that normally pulls sugar (glucose) and sodium back into the blood from the urine. By blocking it, the kidneys dump more sugar and sodium into the urine, which pulls water along with it. This gentle diuretic effect reduces fluid overload and blood pressure. Beyond that, SGLT2 inhibitors shift the heart's fuel use (the heart burns more ketones and less glucose, which is more efficient), reduce inflammation, and protect the kidneys—all of which help a failing heart work better and slow the progression of cardiomyopathy.

Furosemide or torsemide or bumetanide

These drugs block a transporter in the kidney's loop of Henle (a part of the kidney tubule) that normally pulls sodium, chloride, and water back into the blood. By blocking it, the kidneys dump a large amount of salt and water into the urine. This quickly reduces fluid overload—less swelling in the legs, less congestion in the lungs—and makes breathing easier. Loop diuretics are used when fluid buildup is severe or when other diuretics are not strong enough.

Mavacamten

This drug is used specifically for hypertrophic cardiomyopathy (HCM) when the heart muscle is too thick and squeezes too hard, blocking blood flow out of the heart. Mavacamten works by

binding to cardiac myosin (the motor protein that makes heart muscle contract) and reducing the number of myosin heads that can grab onto actin (the track the motor pulls on). With fewer myosin heads working, the heart's contraction becomes less forceful and the muscle relaxes better. This reduces the obstruction, improves blood flow, and relieves symptoms like chest pain and shortness of breath.

Disopyramide

This older drug is sometimes used in hypertrophic cardiomyopathy (HCM) when the heart squeezes too hard and blocks blood flow. Disopyramide blocks sodium channels in heart muscle cells, which slows down the electrical signal and reduces the force of contraction (negative inotropy). By making the heart squeeze less forcefully, it reduces the obstruction in the outflow tract and improves symptoms. It also has anticholinergic effects (blocks acetylcholine), which can speed up the heart rate slightly—balancing out the slowing effect.

Tafamidis

This drug is used for transthyretin amyloid cardiomyopathy (ATTR-CM), a type of restrictive cardiomyopathy where misfolded transthyretin protein clumps together and deposits in the heart, making it stiff. Tafamidis works by binding to transthyretin and stabilizing its normal shape, preventing it from falling apart and clumping into amyloid fibrils. By stopping new amyloid from forming, it slows the progression of the disease, reduces hospitalizations, and improves survival—though it does not remove amyloid that is already there.

Patisiran or inotersen

These drugs are used for hereditary transthyretin amyloidosis (hATTR) with nerve or heart involvement. They work by silencing the gene in the liver that makes transthyretin protein. Patisiran uses RNA interference (RNAi) to destroy the messenger RNA before it can make TTR protein; inotersen uses an antisense oligonucleotide to block the RNA. Either way, the liver makes much less transthyretin, so there is less protein available to misfold and form amyloid. This slows disease progression and can improve nerve and heart symptoms.

Deferoxamine, deferasirox, or deferiprone

These drugs are used when cardiomyopathy is caused by iron overload (hemochromatosis or repeated blood transfusions, like in thalassemia). Too much iron deposits in the heart muscle, causing damage, stiffness, and eventually heart failure. Iron chelators bind to excess iron in the blood and tissues and help the body get rid of it through urine or stool. By lowering iron levels, they prevent further heart damage and can improve heart function over time.

Prednisone or methylprednisolone

Steroids are used when cardiomyopathy is caused by inflammation—for example, in sarcoidosis (abnormal immune clumps called granulomas) or giant cell myocarditis (severe heart

inflammation). Steroids turn down the immune system, reducing inflammation, swelling, and damage to the heart muscle. By calming the immune attack, they can stop progression and sometimes improve heart function, especially if started early.

Digoxin

Digoxin is an older drug sometimes used in heart failure (including dilated cardiomyopathy) to help control heart rate and improve symptoms—though it does NOT reduce mortality. It works by blocking the sodium-potassium pump in heart cells, which indirectly raises calcium inside the cell. More calcium means a slightly stronger contraction. Digoxin also slows the heart rate by increasing vagal (parasympathetic) tone, which is helpful in atrial fibrillation (an irregular, quivering heartbeat). It is used mainly when other drugs are not enough or when rate control is needed.

Ivabradine

Ivabradine is used in heart failure (including some dilated cardiomyopathies) when the heart rate stays too fast despite a beta-blocker, or when a patient cannot tolerate a full dose of beta-blocker. It works by blocking the 'funny current' (If) in the sinus node—the heart's natural pacemaker. By slowing the firing rate of the pacemaker, it lowers heart rate without affecting the force of contraction or blood pressure. A slower heart rate gives the heart more time to fill and rest, which can improve symptoms and reduce hospitalizations.

Hydralazine and isosorbide dinitrate (used together)

This combination is used in heart failure (including dilated cardiomyopathy) when a patient cannot take an ACE inhibitor, ARB, or ARNI (because of kidney problems, high potassium, or allergies). Hydralazine relaxes arteries directly, lowering the resistance the heart has to pump against. Isosorbide dinitrate relaxes veins, reducing the volume of blood returning to the heart (preload). Together, they reduce the heart's workload. Studies show this combination improves survival and symptoms, especially in Black patients, who may respond better to this combination than other groups.

Verapamil or diltiazem

These drugs are sometimes used in hypertrophic cardiomyopathy (HCM) when the heart muscle is thick and stiff, especially if the patient cannot tolerate a beta-blocker or needs help with heart rate control in atrial fibrillation (an irregular, quivering heartbeat). They block calcium channels in heart muscle and the conduction system, which relaxes the muscle, slows the heart rate, and improves filling (diastolic function). However, they are NOT used in obstructive HCM because they can worsen the outflow obstruction by dilating blood vessels and lowering blood pressure too much.

Where this information comes from

- AHA/ACC 2020 Guideline for the Diagnosis and Treatment of Cardiomyopathy
- European Society of Cardiology (ESC) 2023 Cardiomyopathy Guidelines
- American College of Cardiology (ACC) Expert Consensus
- Cochrane Reviews on Heart Failure Management

Last reviewed: 2026-07-07