

Cardiomyopathy

Cardiomyopathy is disease of the heart muscle itself. The type of disease decides how we treat it.

THE ONE-LINE TRUTH

Cardiomyopathy means the heart muscle is sick—it might squeeze too weakly, get too thick, turn stiff, or fill with scar—and each type breaks the pump in a different way.

The chain — how it unfolds

1. The heart muscle is damaged or changed—stretched, thickened, stiffened, or replaced by scar and fat. — why: This is the upstream break. Genes, toxins, viruses, starved blood flow (ischemia), infiltration, or stress injure the muscle. Everything that follows flows from this one broken thing.
2. The damaged muscle cannot pump blood forward effectively (systolic failure) or cannot relax and fill properly (diastolic failure). — why: In dilated cardiomyopathy, the weak, stretched muscle squeezes poorly. In hypertrophic and restrictive types, the thick or stiff muscle cannot relax, so the heart cannot fill. In ARVC, the scarred right ventricle is both weak and electrically unstable.
3. Blood backs up behind the failing heart, causing congestion. — why: If the left side fails, blood backs up into the lungs—fluid leaks into the air sacs, causing shortness of breath, cough, and crackles on exam (pulmonary swelling (edema)). If the right side fails, blood backs up into the body—the liver swells, the belly fills with fluid (ascites), and the legs and ankles swell (peripheral swelling).
4. Not enough blood flows forward to the body and brain, causing fatigue, dizziness, fainting, and organ damage. — why: Low cardiac output means your muscles do not get enough oxygen (you feel weak and tired), your brain gets less blood (you feel lightheaded or faint), and your kidneys and liver start to fail from poor blood flow (perfusion).
5. The sick heart muscle and scarring disrupt electrical signals, leading to arrhythmias. — why: Scar tissue and stretched or thickened muscle interfere with the normal electrical pathway. You see atrial fibrillation (an irregular, quivering heartbeat) (fast, chaotic rhythm in the top chambers), ventricular a fast heart rate (tachycardia) (fast, dangerous rhythm in the bottom chambers), or heart block. Arrhythmias reduce pumping efficiency and can cause sudden cardiac arrest.

6. Blood pools in the dilated, weakly squeezing chambers, forming clots. — why: Slow-moving or stagnant blood clots easily. Clots can break off and travel—to the lungs (pulmonary a traveling clot lodging in a vessel (embolism)), brain (stroke), or limbs (arterial a traveling clot lodging in a vessel).
7. In hypertrophic cardiomyopathy, the thick septum can block blood flow out of the heart (left ventricular outflow tract obstruction). — why: The bulky muscle and abnormal valve motion create a narrow exit. Blood has to squeeze through a tight space, causing a harsh murmur, chest pain, fainting with exercise, and sudden death risk.
8. Over time, the heart remodels—it stretches more, scars more, and becomes even weaker. — why: The ongoing stress, inflammation, and cell death create a vicious cycle. The heart tries to compensate, but those very responses (adrenaline, stretching, thickening) damage the muscle further. Without treatment, the patient spirals toward end-stage heart failure, needing advanced therapies like a ventricular assist device or heart transplant.

Spot it — what you see, and why

- Shortness of breath, especially when lying flat or during activity: The heart muscle is too weak or too stiff to pump blood forward well, so blood backs up into the lungs. This extra fluid in the lungs makes it hard to breathe.
- Swelling in the legs, ankles, or belly: When the heart cannot pump strongly enough, blood pools in the veins. Fluid leaks out of the blood vessels into the tissues, causing puffiness and swelling (edema).
- Extreme tiredness and weakness: The weakened heart cannot deliver enough oxygen-rich blood to the muscles and organs, so they run low on fuel. This makes even small tasks feel exhausting.
- Chest pain or pressure: In some types of cardiomyopathy, the thick or stiff heart muscle needs more oxygen than the blood can deliver, causing the heart muscle to hurt (similar to angina).
- Fast, pounding, or fluttering heartbeat (palpitations): The damaged heart muscle can create abnormal electrical signals, causing the heart to beat too fast, skip beats, or quiver instead of pumping smoothly.
- Fainting or feeling lightheaded: Dangerous irregular heartbeats (arrhythmias) or a sudden drop in blood pressure can briefly cut oxygen to the brain, making a person feel dizzy or pass out.

Red flags — act now

- 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

Treat it — in this order

1. Identify the TYPE first: dilated vs. hypertrophic vs. restrictive vs. ARVC vs. takotsubo vs. infiltrative → You cannot pick the right medicine until you know which broken thing you are fixing. The echo and ECG pattern tell you which type.
2. For dilated cardiomyopathy with reduced ejection fraction (HFrEF): start ARNI (sacubitril/valsartan), beta-blocker (carvedilol, metoprolol succinate, or bisoprolol), MRA (spironolactone or eplerenone), and SGLT2 inhibitor (dapagliflozin or empagliflozin) → These four drugs together reduce the stress on the weak squeezing heart, block harmful hormones (like aldosterone and angiotensin), help the kidneys dump extra fluid, and help the heart muscle use sugar and fat better. Together they cut the risk of dying or ending up in the hospital by about 50 percent.
3. For hypertrophic cardiomyopathy: start a beta-blocker (metoprolol or atenolol) or a calcium-channel blocker (verapamil or diltiazem); consider mavacamten if symptoms persist → Beta-blockers and calcium blockers slow the heart rate and relax the thick muscle so the heart has more time to fill with blood. Mavacamten is a new drug that loosens the grip of the muscle fibers so blood flows better out of the heart. It can reduce obstruction and improve symptoms.
4. For infiltrative or restrictive cardiomyopathy: treat the underlying cause — tafamidis or diflunisal for ATTR cardiac amyloidosis, chemotherapy and stem-cell transplant for AL amyloidosis, corticosteroids or methotrexate for cardiac sarcoidosis, phlebotomy or chelation for hemochromatosis → If you stop amyloid protein from building up, shrink the inflammatory granulomas in sarcoid, or remove poison iron, the stiff heart stops getting worse. Tafamidis cuts the risk of death and hospitalization in ATTR amyloid by about 30 percent. The domino halts upstream.
5. For arrhythmogenic right ventricular cardiomyopathy (ARVC): start a beta-blocker, enforce strict exercise restriction (no competitive or intense sports), and place an implantable cardioverter-defibrillator (ICD) if high sudden-death risk → Beta-blockers calm the irritable right-side muscle and reduce dangerous irregular beats. Exercise makes ARVC worse, so stopping high-intensity activity slows the scarring. The ICD catches the fatal irregular beat (ventricular fibrillation) and shocks the heart back before it kills you.

Your top priorities at the bedside

1. Monitor oxygen saturation continuously and keep SpO₂ above 90% with supplemental oxygen if needed. — why: The weakened heart muscle cannot pump enough oxygen-rich blood to the body, and low oxygen worsens shortness of breath and forces the heart to work even harder, creating a dangerous cycle.
2. 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. — why: Daily weights catch fluid retention early before severe pulmonary edema (fluid flooding the lungs) (fluid in the lungs) develops, because each pound of weight gain equals about 500 mL of retained fluid that the failing heart cannot clear.
3. Place the patient in high Fowler position (sitting upright at 60-90 degrees) or a position of comfort that eases breathing. — why: Sitting upright uses gravity to shift fluid out of the lungs and down into the legs, reducing pulmonary congestion (fluid backup in lung blood vessels) and decreasing the work of breathing.