

Chronic Hfref

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THE STORY IN ONE SENTENCE

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“Chronic HFrEF needs four-pillar GDMT (ARNI, beta-blocker, MRA, SGLT2i) started and pushed to target doses as fast as the patient can tolerate.”

Normal anatomy — the body's job

The left ventricle (the heart's main pumping chamber) fills with blood from the lungs, then squeezes hard and ejects 55–70% of that blood out through the aortic valve into the aorta to feed the whole body. This strong, rhythmic squeeze and the percentage pushed out—called ejection fraction or EF—keeps oxygen and nutrients flowing to every organ.

BODY'S JOB

The left ventricle's job is to be a powerful, elastic pump: it relaxes to fill with oxygen-rich blood from the lungs, then contracts forcefully to eject most of that blood forward through the aorta to the brain, kidneys, muscles, and every tissue. A normal EF of 55–70% means the heart empties more than half its chamber with every beat, delivering steady blood flow even when you stand, walk, or run.

1. Oxygen-rich blood flows from the lungs into the left atrium (the top left chamber).
2. The atrium squeezes gently and pushes blood through the mitral valve into the left ventricle (the bottom left chamber, the main pump).
3. The left ventricle fills to about 120 mL, then squeezes hard and ejects 55–70% (70–80 mL) out through the aortic valve into the aorta.
4. That ejected blood travels through arteries to every organ, delivering oxygen and fuel.
5. The ventricle relaxes again to refill, and the cycle repeats 60–100 times every minute.

What breaks

The left ventricle muscle becomes weak, stiff, scarred, or thin so it cannot squeeze hard enough. Instead of ejecting 55–70%, it ejects less than 40% of the blood in the chamber, so less oxygen and fuel reach the body and blood backs up into the lungs.

CLINICAL TERM

Systolic dysfunction of the left ventricle with ejection fraction $\leq 40\%$, caused by blood-starved (ischemic) cardiomyopathy (prior heart attack (myocardial infarction) killing heart muscle), non-blood-starved cardiomyopathy (idiopathic dilated, viral myocarditis, peripartum, alcohol-induced, chemotherapy-induced, infiltrative disease, genetic mutations, a fast heart rate (tachycardia)-induced), chronic pressure overload (long-standing high blood pressure (hypertension), aortic narrowing (stenosis)), or chronic volume overload (severe mitral or aortic regurgitation). The weakened myocardium cannot generate adequate stroke volume, leading to reduced cardiac output and activation of neurohormonal compensatory mechanisms.

How the body compensates

When the heart's squeeze weakens, the body immediately turns on survival systems—stress hormones, faster heart rate, squeezed blood vessels, and salt and water retention—to try to maintain blood pressure and flow. These moves keep you walking and talking for weeks or months, but they overwork and scar the heart, so the pump fails even more. Spotting these compensation clues early lets you stop the spiral before it tips into shock or sudden death.

1. {'step': 'Sympathetic nervous system fires (adrenaline and noradrenaline released)', 'why': 'Baroreceptors sense low cardiac output and signal the brainstem to dump adrenaline, which makes the heart beat faster and harder and squeezes peripheral vessels to keep blood pressure up short-term.'}
2. {'step': 'Renin-angiotensin-aldosterone system (RAAS) activates', 'why': 'Low renal blood flow (perfusion) triggers renin release → angiotensin II (a powerful vessel constrictor) → aldosterone (tells kidneys to hold salt and water) to expand blood volume and maintain pressure.'}
3. {'step': 'Heart rate rises and contractility tries to increase', 'why': 'Adrenaline pushes the heart to beat 90–110+ times per minute and squeeze harder to make up for low stroke volume, but this burns oxygen and energy the weak muscle does not have.'}
4. {'step': 'Blood vessels constrict (high systemic vascular resistance)', 'why': 'Angiotensin II and sympathetic tone squeeze arteries to keep pressure feeding the brain and heart, but this high resistance (afterload) makes it even harder for the weak ventricle to eject blood forward—a vicious cycle.'}

5. {'step': 'Kidneys retain sodium and water (volume expansion)', 'why': 'Aldosterone and ADH tell the kidneys to hold onto salt and water, raising preload (the stretch of the ventricle before it contracts) to try to boost stroke volume via the Frank-Starling mechanism, but the overstretched, weak muscle cannot respond and instead the lungs and legs fill with fluid.'}
6. {'step': 'Ventricle remodels—dilates, walls thin, and fibrosis sets in', 'why': 'Chronic high wall stress, neurohormones, and inflammatory signals trigger myocyte apoptosis (muscle cell death), collagen deposition (scarring), and chamber dilation; the heart becomes a big, baggy, stiff balloon that squeezes poorly.'}
7. {'step': 'Electrical chaos: arrhythmias emerge', 'why': 'Scar tissue disrupts the normal conduction pathways, creating re-entry circuits for ventricular a fast heart rate (tachycardia) and fibrillation, and stretch triggers ectopic beats; sudden cardiac death risk jumps.'}
8. {'step': 'Compensation fails and decompensation begins', 'why': 'Eventually the heart cannot keep up even with maximum adrenaline and fluid; cardiac output drops, blood pressure falls, organs (kidneys, liver, gut) fail from low blood flow (perfusion), lungs flood with fluid (pulmonary swelling (edema)), and the patient tips into cardiogenic shock or arrests from a fatal arrhythmia.'}

Domino effect

1. The left ventricle squeezes weakly and ejects <40% of its blood (low EF, low cardiac output). — Dead, scarred, or sick muscle cannot generate enough force, so less blood leaves the heart with each beat and organs start to sense low flow and low oxygen delivery.
2. Baroreceptors and the kidneys sense low output and turn on the sympathetic nervous system and RAAS. — The body interprets low stroke volume as 'not enough blood,' so it releases adrenaline (to speed and strengthen the heart) and renin (starting the angiotensin-aldosterone cascade) to raise pressure and volume—an ancient survival reflex.
3. Heart rate climbs, vessels constrict, and kidneys hold salt and water. — Adrenaline makes the heart race (higher oxygen demand), angiotensin II clamps down arteries (higher afterload), and aldosterone expands blood volume (higher preload)—all trying to maintain blood pressure, but all making the weak pump work even harder.
4. The ventricle stretches, thins, scars, and balloons out (adverse remodeling). — Chronic high wall stress, constant neurohormone bathing, and inflammatory signals kill muscle cells, lay down stiff collagen scar, and dilate the chamber into a weak, baggy sac that cannot squeeze or relax properly.

5. Fluid backs up into the lungs (pulmonary congestion, shortness of breath) and body (leg swelling, liver congestion). — The weak left ventricle cannot empty forward, so pressure rises backward through the left atrium into the pulmonary veins and capillaries (causing lung crackles, pink frothy coughed-up mucus (sputum), breathlessness when lying flat (orthopnea)); if the right heart fails too, pressure backs into the body veins (causing jugular venous distension, an enlarged liver (hepatomegaly), pitting swelling (edema)).
6. Scar and stretch create dangerous heart rhythms (ventricular a fast heart rate (tachycardia), atrial fibrillation (an irregular, quivering heartbeat)). — Fibrotic tissue blocks normal electrical flow and creates loops where impulses spin in circles (re-entry); stretched atria fibrillate chaotically; either rhythm drops cardiac output further or degenerates into ventricular fibrillation and sudden death.
7. Organs begin to fail from low blood flow (perfusion): kidneys (rising creatinine, low urine), liver (rising bilirubin, INR), gut (nausea, poor absorption), brain (confusion, fatigue). — Cardiac output is now too low to feed vital organs; kidneys trigger more RAAS (worsening the cycle), liver becomes congested and leaks enzymes, gut wall swells and leaks bacteria, and brain gets less oxygen causing lethargy or delirium.
8. Without treatment, the cycle spirals to cardiogenic shock, multi-organ failure, fatal arrhythmia, or need for mechanical support or transplant. — The compensatory systems meant to buy time have now become the disease themselves—blocking them with GDMT (ARNI, beta-blocker, MRA, SGLT2i) interrupts the spiral, lets the heart rest and reverse remodeling, prevents arrhythmias, and saves lives; without these pillars, the heart exhausts itself and the patient dies or needs a pump or new heart.

Symptoms · what the patient feels, and why

- Shortness of breath (dyspnea) during activity or at rest: The weakened heart cannot pump enough oxygen-rich blood to meet the body's needs, so the lungs work harder and breathing feels difficult. Fluid may also back up into the lungs (called pulmonary congestion), making it harder for oxygen to get into the blood.
- Feeling very tired (fatigue) and weak muscles: Because the heart pumps less blood with each beat, muscles and organs do not get enough oxygen and nutrients to work well. This feels like having a low battery — everything takes more effort.
- Swelling in the ankles, legs, or belly (edema): When the heart cannot pump blood forward strongly, blood backs up in the veins. This increases pressure in the tiny blood vessels, pushing fluid out into the surrounding tissues. Gravity pulls this fluid down to the ankles and legs.

- Weight gain of more than 2 to 3 pounds in one day or 5 pounds in one week: The extra weight is not fat — it is fluid building up in the body because the kidneys hold onto salt and water when blood flow to them drops. This sudden fluid buildup means heart failure is getting worse.
- Hard to breathe when lying flat (orthopnea): When you lie down, fluid that has settled in your legs spreads back toward the chest and lungs. The weakened heart cannot handle this extra fluid load, so it pools in the lungs and makes breathing harder. Propping up with pillows helps gravity keep fluid down.
- Waking up at night gasping for air (paroxysmal nocturnal trouble breathing (dyspnea)): After lying flat for a while, fluid shifts into the lungs. As fluid builds up, oxygen levels drop and the brain sends a panic signal to wake you up and sit upright. Sitting up lets gravity pull fluid back down and relieves the feeling.
- Neck veins that bulge or stick out (jugular venous distension): The right side of the heart pumps blood returning from the body into the lungs. When the heart is weak, blood backs up into the large veins, including the jugular veins in the neck. The increased pressure makes these veins swell and become visible, especially when sitting upright.
- Extra heart sound (S3 gallop): In a failing heart, the left ventricle (main pumping chamber) becomes stiff and overfilled with blood. When blood rushes in during filling, it hits the stiff walls and makes a low-pitched thump — the third heart sound — which sounds like a galloping horse when heard with a stethoscope.
- Crackling or bubbling sounds in the lungs (rales or crackles): Fluid leaks from backed-up blood vessels into the air sacs (alveoli) of the lungs. When you breathe in, air pops through this fluid and makes fine crackling sounds, like the sound of crumpling tissue paper.
- Cough that may bring up white or pink frothy mucus: Fluid in the lungs irritates the airways and triggers a cough. If fluid pressure is very high, it mixes with air and mucus to form a frothy, sometimes blood-tinged coughed-up mucus (sputum) (pink because tiny amounts of blood leak into the fluid).

Focused exam

- Neck veins bulging out even when the patient sits up at a 45-degree angle: In chronic HFrEF, the right side of the heart cannot push blood forward, so blood backs up into the neck veins. When you see the jugular veins standing out above the collarbone while the patient sits up, it means the pressure in the heart is too high and the pump is failing.
- Crackles or wet popping sounds at the bottom of both lungs when you listen with a stethoscope: Fluid leaks into the tiny air sacs in the lungs because the left side of the heart cannot pump blood out of the lungs fast enough. This is called pulmonary edema (fluid flooding the lungs)—like the lungs are slowly drowning because the heart pump is too weak.

- Swelling in both legs and ankles that leaves a dent when you press it (called pitting swelling (edema)): Fluid pools in the legs because the heart pumps less blood forward and pressure builds up in the veins. In chronic HFrEF, this happens slowly over weeks or months as the heart gets weaker.
- Heart sounds that include an extra third sound right after the normal two beats, called an S3 gallop: This extra sound happens when blood rushes into a heart that is too stiff or stretched out to fill smoothly. An S3 gallop is a direct sign that the heart muscle is failing in chronic HFrEF.
- Liver edge you can feel below the rib cage on the right side, bigger and firmer than normal: Blood backs up from the right side of the failing heart into the liver, making it swell and become tender. In chronic HFrEF, this congestion happens over time and can damage the liver if not treated.
- Heart rate over 90 beats per minute even when the patient is resting and calm: The heart beats faster to try to pump more blood and oxygen to the body because each beat is weaker. In chronic HFrEF, this becomes the body's new normal way of keeping up.

Diagnostics · every test answers a question

- Echocardiogram (heart ultrasound) → How much blood does the heart pump out with each beat, and what does the left ventricle look like inside?
- BNP or NT-proBNP blood test → Is the heart under stress and stretching out from trying too hard?
- Complete blood count (CBC) → Does the patient have enough red blood cells to carry oxygen to the body?
- Basic metabolic panel (kidney function and electrolytes) → Are the kidneys still filtering waste, and are sodium and potassium levels safe?
- Thyroid-stimulating hormone (TSH) → Is the thyroid gland making the heart work too hard or too slow?
- Chest X-ray → Is there fluid in the lungs, and is the heart bigger than normal?
- Electrocardiogram (ECG) → Is the heart beating in a normal rhythm, or is there damage to the heart muscle causing electrical problems?

Treatment logic

- Sacubitril-valsartan (or ACE inhibitor like lisinopril, or ARB like losartan if sacubitril-valsartan is not available) → Blocks the stress hormone system that makes the heart bigger and scarred over time, and opens blood vessels so the heart does not have to work as hard. This lets the weakened pump send blood around the body more easily and prevents the heart from stretching out further.

- Beta-blocker (carvedilol, metoprolol succinate, or bisoprolol) → Slows the heart rate and makes each beat less forceful, so the tired heart muscle does not burn out from working too hard. Lets the heart rest and slows down scarring and remodeling over months.
- Mineralocorticoid receptor antagonist (spironolactone or eplerenone) → Stops a stress hormone called aldosterone that makes the heart swell with fluid and scar tissue. This shrinks the heart slightly, reduces extra fluid in the body, and lets the pump work better.
- SGLT2 inhibitor (dapagliflozin or empagliflozin) → Tells the kidneys to dump extra salt and sugar in the urine, which shrinks the fluid burden on the heart and lets it pump more easily. Also protects the heart muscle from further damage by reducing inflammation and stress.
- Loop diuretic like furosemide (a water pill) for swelling and shortness of breath → Removes excess fluid from the body through urine so fluid does not back up into the lungs or legs. The heart has less volume to pump and breathing becomes easier within hours to days.
- Implantable cardioverter-defibrillator (ICD) if ejection fraction stays under 35% after at least 3 months of medicines, or cardiac resynchronization therapy (CRT) if the heart's electrical signals are out of sync → An ICD prevents sudden death from dangerous rhythms by shocking the heart back to normal. A CRT device coordinates the heart's contractions so the weakened pump squeezes both sides at the same time and pushes blood more efficiently.
- Restrict salt to less than 2 grams per day and limit fluids to about 1.5 to 2 liters per day if swelling is bad → Reduces the amount of water the body holds, so the heart does not have to pump as much volume. This lowers congestion in the lungs and legs and makes breathing easier.

Medications

Sacubitril-valsartan (Entresto) · ARNI — angiotensin receptor-neprilysin inhibitor

This drug does two jobs at once. First, it blocks a hormone called angiotensin II that squeezes blood vessels and makes the heart pump harder. Second, it stops the body from breaking down helpful chemicals called natriuretic peptides that relax blood vessels and help the kidneys get rid of extra salt and water. Together, these actions lower the workload on the weak heart and help it pump blood more easily.

1. Valsartan blocks angiotensin II receptors on blood vessel cells, stopping the squeeze signal
2. Sacubitril blocks neprilysin, the enzyme that breaks down natriuretic peptides
3. More natriuretic peptides stay in the blood, telling blood vessels to relax and kidneys to release salt and water
4. Blood pressure drops and the heart doesn't have to work as hard to push blood out

5. Over time, the heart chambers may shrink a bit and pump better

SAFETY WATCH

Can cause low blood pressure — stand up slowly and watch for dizziness · Can raise potassium levels — avoid potassium supplements and salt substitutes unless your doctor says it's safe · Do NOT use if you have had angioedema (sudden swelling of face or throat) with ACE inhibitors before · Do NOT take with ACE inhibitors; wait 36 hours after stopping an ACE inhibitor before starting this drug · Can harm a developing baby — use reliable birth control if you can become pregnant · Kidney function and potassium levels need regular blood tests

Beta-blockers — carvedilol, metoprolol succinate (Toprol-XL), or bisoprolol · Beta-adrenergic receptor blockers

These drugs block adrenaline (the 'fight or flight' hormone) from reaching the heart. In heart failure, the body pours out too much adrenaline trying to keep blood pressure up, but this overdrive wears out the heart muscle even faster. Beta-blockers slow the heart rate, lower blood pressure, and give the tired heart time to rest and recover between beats.

1. Adrenaline normally binds to beta receptors on heart cells and speeds up the heart rate and contraction strength
2. Beta-blocker sits in the receptor and blocks adrenaline from attaching
3. Heart rate slows down, giving the heart more time to fill with blood between beats
4. The heart uses less oxygen because it's not working in overdrive
5. Over months, the heart muscle may get stronger and pump more blood with each beat

SAFETY WATCH

Start at a very low dose and increase slowly over weeks — too much too fast can make heart failure worse · Can make you feel more tired, dizzy, or short of breath at first — this usually gets better after a few weeks · Can cause slow heart rate — let your doctor know if you feel very dizzy or faint · Do NOT stop suddenly — this can trigger dangerous heart rhythms or a heart attack · Can mask low blood sugar symptoms in people with diabetes · May worsen asthma or severe lung disease — use with caution and close monitoring

Mineralocorticoid receptor antagonists (MRAs) — spironolactone or eplerenone · Aldosterone blockers (potassium-sparing diuretics)

These drugs block aldosterone, a hormone that makes the kidneys hold onto salt and water and helps scar tissue build up in the heart. By blocking aldosterone, these medicines help the body

release extra fluid, lower blood pressure, and slow down harmful remodeling (thickening and stiffening) of the heart muscle.

1. In heart failure, the body makes too much aldosterone, which tells the kidneys to hold onto sodium and water
2. MRAs block aldosterone receptors in the kidneys
3. Kidneys release more sodium and water into the urine, lowering fluid overload
4. Potassium is kept in the body instead of being lost in the urine
5. Less aldosterone signal also means less scarring and stiffening of the heart over time

SAFETY WATCH

Can raise potassium to dangerous levels — get regular blood tests and avoid potassium supplements and salt substitutes · Can worsen kidney function — blood tests are needed to check creatinine and potassium · Spironolactone can cause breast tenderness or enlargement in men (gynecomastia) — eplerenone has less of this effect · Can cause low blood pressure and dizziness · Do NOT use if potassium is already high or kidney function is very poor

SGLT2 inhibitors — dapagliflozin (Farxiga) or empagliflozin (Jardiance) · Sodium-glucose cotransporter-2 inhibitors

These drugs were first made for diabetes, but they also help the failing heart. They block a protein in the kidneys that normally pulls sugar and sodium back into the blood. When this protein is blocked, the kidneys dump more sugar, sodium, and water into the urine. This lowers fluid overload, blood pressure, and stress on the heart. They may also protect the heart muscle and kidneys through other pathways scientists are still studying.

1. Kidneys filter blood and then normally reabsorb most of the sugar and some sodium back into the bloodstream through SGLT2 proteins
2. SGLT2 inhibitor blocks these proteins
3. Sugar and sodium stay in the urine and are flushed out, taking extra water with them
4. Blood volume drops, lowering the pressure the heart has to pump against
5. The heart and kidneys may also benefit from reduced inflammation and better energy metabolism

SAFETY WATCH

Can cause yeast infections in the genital area because of extra sugar in the urine — keep the area clean and dry · Rarely can cause a serious infection called Fournier's gangrene (infection of the skin between the genitals and anus) — seek help immediately for pain, redness, swelling, or fever in that area · Can cause urinating more often, especially in the first few weeks · Rarely can cause ketoacidosis (dangerous acid buildup) even with normal blood sugar — watch for nausea, vomiting, stomach pain, trouble breathing, or confusion · Can cause low blood pressure and dizziness, especially when combined with other blood pressure medicines · Drink enough water to avoid dehydration

Loop diuretics — furosemide (Lasix), bumetanide, or torsemide · Loop diuretics (water pills)

These are strong water pills that help the kidneys release large amounts of salt and water into the urine. They work in the loop of Henle, a key part of the kidney's filtering system, to stop sodium from being pulled back into the blood. When sodium leaves, water follows, reducing swelling in the legs, belly, and lungs and making it easier to breathe.

1. Kidneys filter blood and try to reabsorb most of the sodium and water back into the bloodstream
2. Loop diuretic blocks the sodium-potassium-chloride transporter in the loop of Henle
3. Sodium, chloride, and water stay in the urine instead of returning to the blood
4. Large amounts of urine are made, pulling fluid out of swollen legs, belly, and lungs
5. Blood volume drops, reducing the fluid backup into the lungs and body tissues

SAFETY WATCH

Can cause low potassium, magnesium, and sodium — blood tests are needed to check these levels · Can cause dehydration if too much fluid is removed — watch for dry mouth, dizziness, or feeling very thirsty · Can make kidney function worse if you become too dehydrated · Can cause ringing in the ears or hearing loss at very high doses · Can raise blood sugar and uric acid (which can trigger gout) · The dose often needs to be adjusted — work with your doctor to find the right amount

Hydralazine and isosorbide dinitrate (combination) · Vasodilators — arterial dilator (hydralazine) and venous dilator (nitrate)

This combination relaxes blood vessels in two different ways. Hydralazine relaxes the arteries (the vessels that carry blood away from the heart), making it easier for the heart to push blood out. Isosorbide dinitrate relaxes the veins (the vessels that carry blood back to the heart),

reducing the amount of blood returning to the heart and easing the backup into the lungs. Together, they lower the heart's workload.

1. Hydralazine acts directly on smooth muscle cells in artery walls, causing them to relax and widen
2. Arteries dilate, lowering the resistance the heart has to pump against
3. Isosorbide dinitrate breaks down into nitric oxide, a chemical signal that relaxes vein walls
4. Veins dilate, so less blood pools and backs up into the lungs
5. The heart pumps more easily and the lungs clear of excess fluid

SAFETY WATCH

Can cause low blood pressure and dizziness — stand up slowly · Hydralazine can cause a lupus-like syndrome (joint pain, fever, rash) at high doses or with long-term use — stop the drug and see your doctor if these symptoms appear · Isosorbide dinitrate can cause headaches, especially at first — these usually improve with time · Nitrates can interact dangerously with erectile dysfunction medicines like sildenafil (Viagra) — do NOT use them together · You may develop tolerance to nitrates, making them less effective over time · This combination is often recommended for self-identified Black patients because studies show it provides extra benefit in this group

ACE inhibitors — enalapril, lisinopril, or ramipril · Angiotensin-converting enzyme inhibitors

These drugs block an enzyme that makes angiotensin II, a hormone that squeezes blood vessels tight and raises blood pressure. By blocking this enzyme, ACE inhibitors relax blood vessels, lower blood pressure, and reduce the strain on the weak heart. They also slow down harmful remodeling (the heart getting stiffer and bigger in unhealthy ways).

1. The body makes a chain of hormones: renin → angiotensin I → (ACE enzyme) → angiotensin II
2. Angiotensin II squeezes blood vessels, raises blood pressure, and triggers scarring in the heart
3. ACE inhibitor blocks the ACE enzyme so angiotensin II can't be made
4. Blood vessels relax, blood pressure drops, and the heart doesn't have to work as hard
5. Less angiotensin II also means less aldosterone, so the kidneys release more salt and water

SAFETY WATCH

Can cause a dry, persistent cough in up to 10-20% of people — switch to an ARB if this happens · Can raise potassium levels — avoid potassium supplements and salt substitutes unless your doctor says it's safe · Can cause angioedema (sudden swelling of the face, lips, tongue, or throat) — this is rare but dangerous; stop the drug and get emergency help immediately · Can worsen kidney function, especially if you are dehydrated or have kidney disease — regular blood tests are needed · Can cause low blood pressure and dizziness · Can harm a developing baby — use reliable birth control if you can become pregnant

Angiotensin receptor blockers (ARBs) — losartan, valsartan (alone), or candesartan · Angiotensin II receptor blockers

These drugs block the receptor where angiotensin II (a blood vessel-squeezing hormone) attaches. Even if your body makes angiotensin II, it can't do its job because the receptor is blocked. This relaxes blood vessels, lowers blood pressure, and reduces strain on the weak heart. ARBs work like ACE inhibitors but do NOT cause a cough.

1. Angiotensin II is made in the blood and normally binds to AT1 receptors on blood vessel cells
2. This binding squeezes blood vessels, raises blood pressure, and triggers heart muscle scarring
3. ARB sits in the AT1 receptor and blocks angiotensin II from attaching
4. Blood vessels stay relaxed, blood pressure drops, and the heart's workload decreases
5. Less aldosterone is released, so the kidneys get rid of more salt and water

SAFETY WATCH

Can raise potassium levels — avoid potassium supplements and salt substitutes unless your doctor says it's safe · Can worsen kidney function — regular blood tests are needed · Can cause low blood pressure and dizziness · Can harm a developing baby — use reliable birth control if you can become pregnant · Angioedema (sudden swelling) is much less common than with ACE inhibitors but can still happen · Usually used only if you can't take sacubitril-valsartan or an ACE inhibitor

Ivabradine (Corlanor) · If channel blocker (heart rate-lowering agent)

This drug slows the heart rate without lowering blood pressure or weakening the heart's squeeze. It works on the heart's natural pacemaker cells by blocking a special channel (called the If channel) that controls how fast the pacemaker fires. A slower heart rate gives the heart

more time to fill with blood and rest between beats, which can improve symptoms and reduce hospitalizations.

1. The SA node generates electrical signals that set the heart rate
2. If channels let positive ions flow into pacemaker cells, speeding up the rate they fire
3. Ivabradine blocks If channels, slowing the flow of ions
4. The SA node fires more slowly, lowering the heart rate
5. The heart has more time to fill and eject blood with each beat

SAFETY WATCH

Only works in normal sinus rhythm — do NOT use if you have atrial fibrillation (an irregular, quivering heartbeat) or other irregular rhythms · Can cause visual side effects like seeing bright lights or flashes (phosphenes) — usually harmless and temporary · Can slow the heart rate too much — watch for dizziness, extreme fatigue, or fainting · Only use if your resting heart rate is 70 beats per minute or higher despite being on a beta-blocker · Not a first-line drug — only added when other medicines aren't enough

Digoxin (Lanoxin) · Cardiac glycoside

This old drug makes the heart squeeze a bit harder by increasing the amount of calcium available inside heart muscle cells. It also slows the heart rate by acting on the vagus nerve, which puts a brake on the heart's electrical system. Digoxin can help reduce hospitalizations and improve symptoms, but it does NOT help you live longer.

1. Digoxin blocks the sodium-potassium pump on heart cell membranes
2. Sodium builds up slightly inside the cell
3. The sodium-calcium exchanger can't work as well, so calcium also builds up inside
4. More calcium means a stronger squeeze when the heart contracts
5. Digoxin also boosts vagus nerve activity, slowing the heart rate and the conduction of signals through the AV node

SAFETY WATCH

Very narrow safety window — too much can cause dangerous toxicity · Toxicity causes nausea, vomiting, confusion, vision changes (seeing yellow or green halos), and dangerous heart rhythms · Blood levels need to be checked regularly, especially if kidney function changes or if you take other medicines that interact · Low potassium or magnesium increases the risk of toxicity — these levels must be kept normal · Many drug interactions — tell your doctor about all medicines you take · Older adults and people with kidney disease are at higher risk for toxicity

Vericiguat (Verquvo) · Soluble guanylate cyclase stimulator

This newer drug boosts a natural signaling pathway inside cells that helps blood vessels relax and protects the heart muscle. It works by stimulating an enzyme called soluble guanylate cyclase (sGC), which makes more cyclic GMP (a messenger molecule). More cyclic GMP helps blood vessels stay open and may slow harmful changes in the heart muscle.

1. Normally, nitric oxide (a natural relaxing signal) binds to sGC inside cells
2. sGC then makes cyclic GMP, which relaxes blood vessels and protects cells
3. In heart failure, nitric oxide levels are low and sGC doesn't work as well
4. Vericiguat directly stimulates sGC even when nitric oxide is low
5. Cyclic GMP levels rise, blood vessels relax, and cellular stress decreases

SAFETY WATCH

Can cause low blood pressure — stand up slowly and watch for dizziness · Not recommended if your blood pressure is already very low · Take with food to help your body absorb it properly · Do NOT crush or split the tablets · Can harm a developing baby — use reliable birth control if you can become pregnant · Only prescribed after you've been hospitalized or had worsening heart failure despite being on other guideline-directed medicines

Red flags

- Sudden, severe shortness of breath or gasping for air (may signal acute pulmonary edema (fluid flooding the lungs) — fluid flooding the lungs)
- Chest pain or pressure (could mean a heart attack is causing or worsening the heart failure)
- Confusion, extreme fatigue, or fainting (may mean the brain is not getting enough oxygen because the heart is failing badly)
- Fast weight gain (more than 2–3 pounds overnight or 5 pounds in a week) or worsening leg swelling despite taking medicines
- Coughing up pink, frothy coughed-up mucus (sputum) (sign of severe fluid backup in the lungs)

Nursing priorities

1. Assess breathing rate, oxygen saturation with pulse oximeter, and lung sounds every 4 hours or more often if the patient is short of breath. — A weak heart (low ejection fraction) cannot pump blood forward well, so blood backs up into the lungs and causes fluid leakage (pulmonary congestion); crackles in the lungs, fast breathing, or oxygen saturation below 90% means pulmonary edema (fluid flooding the lungs) is worsening and the patient may need more oxygen or IV diuretics.
2. Weigh the patient at the same time every morning before breakfast using the same scale, and report any gain of more than 2 pounds overnight or 5 pounds in one week. — Daily weight is the most sensitive early warning that the body is retaining extra fluid (volume overload) before swelling or lung congestion become severe; catching a 2-pound gain lets the care team increase diuretics before the patient ends up in the hospital with acute decompensation.
3. Monitor strict intake and output every shift, and watch for urine output less than 0.5 milliliters per kilogram per hour or 30 milliliters per hour. — The failing heart pumps less blood to the kidneys (reduced renal blood flow (perfusion)), so the kidneys make less urine and the body holds onto salt and water; low urine output means either the diuretic dose is too low or the kidneys are failing (cardiorenal syndrome), and the doctor may need to increase diuretics or add inotropes (medicines that make the heart squeeze harder).
4. Check blood pressure and heart rate before giving each dose of beta-blocker, ARNI, or other blood-pressure medicine, and hold the dose if systolic pressure is below 90 or heart rate is below 50. — Beta-blockers and ARNI (sacubitril-valsartan) lower blood pressure and slow the heart rate as part of how they help the heart remodel and live longer; but if pressure drops too low (hypotension) or the heart beats too slow (bradycardia), organs will not get enough blood and the patient can faint or go into shock.
5. Teach the patient to limit sodium (salt) to less than 2 grams per day and fluids to 1.5 to 2 liters per day if the doctor ordered fluid restriction. — Salt makes the body hold onto water (sodium attracts fluid), which increases the volume of blood the weak heart must pump; limiting salt and fluids reduces the workload on the failing heart and prevents fluid from building up in the lungs and tissues.
6. Inspect ankles, legs, sacrum (lower back when lying down), and belly every shift for pitting swelling (edema), and measure the swelling by pressing your finger into the skin for 5 seconds. — When the right side of the heart is too weak to pump blood back from the body, pressure builds up in the veins and forces fluid into the tissues (peripheral swelling (edema)); tracking where swelling is and how deep the pit stays after you press tells you if diuretics are working or if heart failure is getting worse.

7. Check potassium and creatinine (a kidney test) levels at least weekly or as ordered, especially when the patient is on spironolactone, eplerenone, or ARNI. — MRA medicines (spironolactone and eplerenone) and ARNI can cause the kidneys to hold onto potassium (hyperkalemia), which can stop the heart or cause dangerous rhythms; loop diuretics (furosemide) can cause low potassium (hypokalemia), which also triggers deadly rhythms; creatinine rising means the kidneys are failing from low blood flow or medicine toxicity.
8. Encourage the patient to sit up in a chair or elevate the head of the bed at least 30 to 45 degrees if they are short of breath when lying flat. — Sitting upright uses gravity to pull fluid down and out of the lungs (reduces pulmonary venous pressure), making it easier to breathe; lying flat lets fluid shift back into the chest, worsening breathlessness when lying flat (orthopnea) and work of breathing.

Sources

- AHA/ACC/HFSA 2022 Guideline for the Management of Heart Failure
- Cochrane Heart Failure Reviews
- ESC 2021 Heart Failure Guidelines (European Society of Cardiology)
- NEJM Heart Failure Clinical Reviews

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