ER BASICS · ONE-PAGER

Heart Failure

HFrEF vs HFpEF, stages, GDMT pillars, warm/cold profiles, shock and diuretics

26 sources + more detail
erbasics.com/sheets/heart-failure.html
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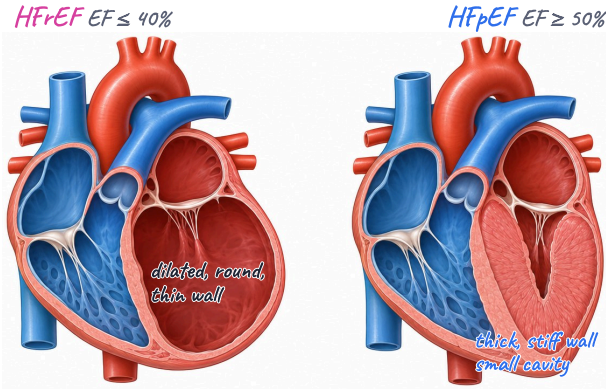
TYPES Two ways a left ventricle fails



1 Types, stages and NYHA class

2022 AHA/ACC/HFSA cut-offs. Finerenone, a nonsteroidal MRA, is FDA-approved (2025) for LVEF ≥ 40%, not HFrEF.

TYPE	LVEF	EXAM POINT
HFrEF	≤40%	Four pillars cut deaths
HFmrEF	41–49%	SGLT2i; MRA or ARNi may help
HFpEF	≥50%	SGLT2i; treat BP, AF, congestion
HFimpEF	Was ≤40%, now >40%	Keep GDMT

STAGE	MEANING
A	At risk (HTN, diabetes, obesity, CAD, cardiotoxins); no structural disease
B	Pre-HF: structural change or high BNP; never symptomatic
C	Structural disease with current or past symptoms
D	Advanced: marked symptoms, repeat admissions despite optimized GDMT

- NYHA I: no limits from HF
- NYHA II: symptoms with ordinary activity
- NYHA III: symptoms with less than ordinary activity
- NYHA IV: symptoms at rest or with any activity

EXAM TIP Stages never move backward: stage C stays C even when symptoms resolve. NYHA class can improve or worsen.

2 Left vs right failure

LEFT-SIDED (LUNGS)	RIGHT-SIDED (BODY)
Dyspnea, orthopnea, PND, fatigue	JVD, hepatojugular reflux
Crackles, cough	Dependent pitting edema
S3 gallop	Hepatomegaly, ascites, early satiety
High PCWP	High CVP

Left-sided failure is the most common cause of right-sided failure. Obesity lowers BNP and NT-proBNP.

3 HFrEF: the four pillars

Start all four unless contraindicated; titrate per your institution's protocols. Diuretics ease congestion but are not a pillar.

PILLAR	EXAMPLES	WATCH FOR
ARNi (preferred), ACEi or ARB	Sacubitril-valsartan, lisinopril, losartan	Low BP, high K; 36 h ACEi washout before ARNi; no ARNi or ACEi after angioedema
Beta-blocker	Carvedilol, metoprolol succinate, bisoprolol	Bradycardia; not started on IV inotropes; never stop abruptly
MRA	Spironolactone, eplerenone	Start if eGFR >30 and K <5.0; stop if K stays ≥5.5
SGLT2i	Dapagliflozin, empagliflozin	No diabetes needed; genital infection, volume loss, euglycemic DKA; hold ≥3 days before surgery

ACEi dry cough: switch to an ARNi or ARB. Add hydralazine-isosorbide dinitrate for self-identified African American patients with NYHA III–IV symptoms despite optimal therapy.

EXAM TIP Only carvedilol, bisoprolol and metoprolol succinate are proven in HFrEF; metoprolol tartrate is not. Sacubitril-valsartan raises BNP but not NT-proBNP, so follow NT-proBNP.

4 Decompensated HF and shock

Wet = congested (crackles, JVD, edema, weight gain). Cold = poorly perfused (cool skin, narrow pulse pressure, confusion, low urine output, rising lactate).

PROFILE	PICTURE	TREATMENT FOCUS
Warm-dry	Compensated	Titrate oral GDMT
Warm-wet	Most common	IV loop diuretic; vasodilator (nitroglycerin, nitroprusside) if BP allows
Cold-wet	Worst outlook	Inotrope plus diuretic; MCS if failing
Cold-dry	Uncommon	Cautious fluids; inotrope if needed

Pulmonary edema: sit upright, give oxygen and an IV loop diuretic; track urine output, K, BP. Keep GDMT unless contraindicated (e.g., shock); hold or reduce the beta-blocker for marked overload or low output. Refractory congestion: ultrafiltration or CRRT.

EXAM TIP Milrinone (a PDE3 inhibitor) works despite beta-blockers. It vasodilates (watch BP) and needs a lower rate in renal impairment.

SAFETY Cardiogenic shock: SBP <90 mm Hg for >30 min or pressors needed, plus hypoperfusion (urine <30 mL/h, lactate >2 mmol/L); typically CI <2.2 and PCWP >15. Revascularize if MI. Norepinephrine for hypotension, plus an inotrope (dobutamine or milrinone) for low output. Temporary MCS (e.g., Impella, VA-ECMO) if drugs fail; routine IABP or ECMO is not advised in MI shock.

5 Diuretic monitoring

- Daily standing weight, same time; strict intake and output
- Daily K, Mg, Na, BUN and creatinine while titrating
- BP and orthostatic symptoms; avoid over-diuresis
- Push usual IV furosemide doses (20–80 mg) over 1–2 min; infuse high doses no faster than 4 mg/min
- Tinnitus or hearing loss: ototoxicity (rapid or high doses, renal failure, aminoglycosides)
- Poor response: higher IV loop dose, or add metolazone (watch K, Na)
- Small creatinine rise with good decongestion: not a reason to stop

IV loop doses often start at ≥2× the home oral dose, per your institution's protocols.

SAFETY Low K or Mg raises the risk of digoxin toxicity. MRAs and ACEi/ARB/ARNi raise K; check labs before giving.

6 Patient teaching

- Weigh each morning after voiding, before eating, same scale
- Report a gain of 2–3 lb in a day or 5 lb in a week, or new dyspnea or swelling
- Limit sodium (processed, canned, restaurant food); often 1,500–2,300 mg/day
- Restrict fluids only if prescribed
- Take GDMT daily even when well; never stop a beta-blocker suddenly
- Avoid NSAIDs and potassium salt substitutes
- Take the diuretic early in the day; rise slowly; stay active

MORE Pathophysiology: neurohormones, remodeling and phenotypes

An injury (infarction, pressure or volume overload, cardiomyopathy, toxins) lowers stroke volume or raises filling pressure. Baroreceptors and the kidney sense low effective volume and activate the sympathetic system, the renin–angiotensin–aldosterone system (RAAS) and vasopressin. Short term this holds BP and output; sustained for months, the same response becomes the disease.

Angiotensin II and aldosterone drive myocyte hypertrophy, apoptosis and fibrosis; norepinephrine is toxic to myocytes, provokes arrhythmias and downregulates β_1 receptors; vasopressin causes hyponatremia. Wall stretch releases the counterweight, ANP and BNP (natriuresis, vasodilation, RAAS inhibition), which neprilysin degrades: the reason for sacubitril. ARNi, ACE inhibitors, ARBs, β-blockers and MRAs lower mortality by blocking these pathways, and SGLT2 inhibitors by natriuretic and metabolic effects; diuretics, digoxin and inotropes relieve symptoms without lowering mortality.

In HFrEF the LV dilates and becomes spherical. By Laplace's law, wall stress = pressure × radius ÷ (2 × wall thickness), so a bigger, thinner ventricle has higher wall stress and oxygen demand; the stretched mitral annulus adds functional MR. In HFpEF, hypertension, age, obesity, diabetes and inflammation make a thick, stiff ventricle: a small volume gain or BP surge raises LVEDP sharply (flash pulmonary edema), and AF or tachycardia, which cost atrial kick and filling time, are poorly tolerated.

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