Congestive Heart Failure (CHF) & Common Cardiac Interventions – Reference Guide

Overview of CHF and Pathophysiology

Definition: Congestive Heart Failure (CHF) is a syndrome where the heart cannot pump effectively to meet the body's needs, leading to inadequate cardiac output and congestion in the lungs or peripheral tissues (Heart Failure). It often results from structural or functional problems such as coronary artery disease, hypertension, myocardial infarction, valvular disease, or cardiomyopathy (Heart Failure). Over time, the ventricles may weaken and dilate (systolic HF) or stiffen (diastolic HF), impairing blood ejection or filling. The **ejection fraction (EF)** is a key measure: EF ≤ 40% indicates **HFrEF (heart failure with reduced EF)**, while EF ≥ 50% with HF symptoms is **HFpEF (preserved EF)** (Types of Heart Failure | American Heart Association). (HFmrEF, 41–49%, is mid-range) (Types of Heart Failure | American Heart Association).

Left vs. Right HF:

In **left-sided HF**, the left ventricle fails to pump blood forward, causing blood to back up into the lungs (Types of Heart Failure | American Heart Association). Patients develop **pulmonary congestion** – dyspnea on exertion, orthopnea (difficulty breathing lying flat), paroxysmal nocturnal dyspnea (sudden nighttime SOB), crackles on lung auscultation, and sometimes an extra heart sound S3 (ventricular gallop) due to increased volume (Heart Sounds Topic Review | Learn the Heart - Healio). Fatigue and poor organ perfusion (weak pulses, cool extremities) are common.

In **right-sided HF**, the right ventricle fails, leading to systemic venous congestion (Types of Heart Failure | American Heart Association). Signs include **jugular venous distension (JVD)**, peripheral edema (legs, ankles), ascites, weight gain, and hepatomegaly (Heart Failure (1).pdf) (Heart Failure (1).pdf). Patients often report abdominal bloating and loss of appetite from liver/gut congestion. Right HF frequently results from long-standing left HF (Types of Heart Failure | American Heart Association). Often the term "**congestive heart failure**" is used when fluid buildup (edema) is prominent in lungs and tissues (Types of Heart Failure | American Heart Association).

Common Symptoms: Early on, HF may present with exercise intolerance and fatigue. As it progresses, **shortness of breath** (especially when lying flat) and **cough** (often with frothy sputum in acute pulmonary edema) occur due to pulmonary fluid (Left-Sided Heart Failure: Symptoms, Causes and Treatment). **Edema** in dependent areas (legs, sacrum) occurs with right

HF or advanced left HF. Patients may have rapid weight gain from fluid retention, tachycardia, and in severe cases confusion or dizziness from low cardiac output. It's important for nurses to recognize these signs and symptoms promptly, as worsening symptoms may indicate acute decompensation.

Classification: HF severity is often classified by **NYHA functional class I–IV** (I = no symptoms with ordinary activity; IV = symptoms at rest) and by **ACC/AHA stages A–D** (A = at risk with no disease, to D = refractory HF). This guide focuses on Stage C/D (established HF) in the clinical setting.

Key Diagnostic Tools for Heart Failure

Accurate diagnosis and monitoring of CHF involve a combination of **clinical assessment** and diagnostic tests. No single test confirms HF, but multiple investigations build the picture (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP). Key tools include:

- **Physical Exam:** Critical for initial evaluation. Look for elevated JVD, lung crackles, displaced apical impulse (left ventricular enlargement), S3 heart sound, edema, and vital sign abnormalities (e.g. tachycardia, hypotension). These findings strongly suggest HF in the appropriate context (Heart Failure (HF) - Cardiovascular Disorders - Merck Manuals). Blood pressure and heart rhythm (possible atrial fibrillation) should be assessed, as arrhythmias or uncontrolled hypertension can precipitate HF exacerbations.

- **Electrocardiogram (ECG):** An **ECG** is routine to evaluate heart rhythm and evidence of prior infarction or strain. It may show ischemic changes, Q waves from old MI, left ventricular hypertrophy, or arrhythmias that contribute to HF. While not diagnostic of HF by itself, an ECG helps identify underlying causes (e.g. ischemia, atrial fib) and is recommended for all patients with HF (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP).

- **Laboratory Tests:** Blood tests assist in diagnosing and determining HF severity. **B-type Natriuretic Peptide (BNP)** or NT-proBNP levels rise with increased ventricular pressure and volume, and are very useful markers. **Elevated BNP** (e.g. >100 pg/mL) strongly suggests HF is contributing to dyspnea (Heart Failure (1).pdf). Low BNP (<100) makes HF unlikely as the cause of symptoms (Pulmonary edema - Wikipedia). Other labs include **cardiac enzymes (troponin)** to rule out myocardial infarction as a trigger (troponin may be mildly elevated in acute HF due to strain), **electrolytes and renal function** (to assess consequences of HF and guide diuretic therapy) (Heart Failure - Diagnosis | NHLBI, NIH), **LFTs** (liver congestion), and **thyroid function (TSH)** since thyroid disorders can precipitate HF (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP). Arterial Blood Gases (**ABGs**) are checked in acute decompensation to assess oxygenation and acid-base status; acute pulmonary edema may cause hypoxemia and respiratory acidosis.

- **Chest Imaging (Chest X-Ray):** A **chest X-ray (CXR)** is a fast, noninvasive test to look for signs of pulmonary congestion and cardiomegaly. In HF, a CXR may show an **enlarged cardiac silhouette** (cardiomegaly) and pulmonary edema patterns – e.g. hazy bilateral infiltrates, **Kerley B lines** at lung bases indicating interstitial edema, cephalization of pulmonary vessels (upper lobe blood flow), and possibly pleural effusions (Pulmonary edema - Wikipedia). These findings support the diagnosis of cardiogenic pulmonary edema. **Below is a CXR example of acute pulmonary edema**, with the red shaded areas showing extensive fluid in the lungs (Pulmonary edema - Wikipedia) (image). *(Chest X-ray of acute cardiogenic pulmonary edema, demonstrating bilateral diffuse infiltrates consistent with fluid overload.)*

- **Echocardiography:** An **echocardiogram** (cardiac ultrasound) is **essential** in HF workup. Echo can visualize chamber size, wall motion, valve function, estimate pressures, and crucially measure the **ejection fraction** (Ejection fraction: An important heart test - Mayo Clinic). **Transthoracic echo (TTE)** is noninvasive; a probe on the chest uses ultrasound to create moving images of the heart. It can confirm systolic dysfunction (reduced EF) or diastolic dysfunction (impaired filling) and detect valvular disease or wall-motion abnormalities from prior MI. A **transesophageal echo (TEE)**, where a probe in the esophagus images the heart, may be used if more detail is needed (for example, to better visualize valves or clots), but it is more invasive. Echo is often performed on admission for new HF or periodically to assess EF changes (Heart Failure - Diagnosis | NHLBI, NIH). For known HFrEF, EF helps guide therapy (e.g. EF < 35% has implications for ICD placement). Nurses should ensure HF patients get indicated echos and should know their patient's EF.

- **Hemodynamic Monitoring:** In acute or advanced HF, invasive hemodynamic monitoring may be employed. A **right heart catheterization** (pulmonary artery catheter, or Swan-Ganz) measures intracardiac pressures: RA, RV, pulmonary artery pressures, and pulmonary capillary wedge pressure (an indirect left atrial pressure). This can quantify the severity of congestion and guide IV diuretic or vasopressor therapy. Right heart cath is typically done via a venous access (internal jugular or femoral vein) and floats into the right heart and pulmonary artery (Cardiac Catheterization - RNpedia) (Cardiac Catheterization - RNpedia). Less commonly, a **left heart catheterization** (via an artery, e.g. radial or femoral) is done in HF specifically to evaluate for ischemia – it involves coronary angiography to see blockages and can also measure left ventricular end-diastolic pressure and perform ventriculography to calculate EF (Cardiac Catheterization - RNpedia). In practice, left heart cath is performed if **ischemic heart disease** is suspected as the cause of HF or if an acute coronary syndrome is possible. **Cardiac Catheterization** (left or right) is invasive, so nursing care includes pre-procedure consent and teaching, and post-procedure monitoring of the insertion site (e.g. groin or wrist for bleeding), peripheral pulses, and bed rest instructions. Monitor for complications like bleeding, hematoma, or arrhythmias after cath.

- **Advanced Imaging:** Other tests may be utilized in specific cases. A **Cardiac CT scan** or **Cardiac MRI** can assess ventricular function, wall thickness, scar tissue, and even detect infiltrative diseases. **Cardiac MRI** is the gold standard for measuring volumes and EF and can characterize myocardium (e.g. fibrosis in cardiomyopathy). **Nuclear medicine scans** (MUGA scan) can also measure EF. These are typically ordered by cardiology for detailed evaluation and are not first-line in routine HF diagnosis but may be seen in advanced workups (Ejection fraction: An important heart test - Mayo Clinic) (Ejection fraction: An important heart test - Mayo Clinic). Nurses may encounter patients who had these studies done; understanding their purpose (precise EF measurement, tissue characterization) is helpful.

- **Functional/Cardiac Stress Testing:** While not a direct diagnostic of HF, stress tests (exercise or pharmacologic) might be done to evaluate ischemia in patients with HF or to assess exercise capacity. Also, **6-minute walk tests** are used to gauge functional status in chronic HF.

Bottom line for nurses: Know your patient's key diagnostic data – EF %, BNP level, weight trends on CXR, and any cath or echo results that inform their treatment plan. Recognize that persistent high BNP or a dropping EF may signal worsening HF and prompt changes in therapy (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP). Also, ensure routine labs (electrolytes, kidney function) are monitored, especially when patients are on diuretics or other HF meds, as these values guide safe medication management.

Medications in Heart Failure Management

Medications are the cornerstone of CHF treatment. Patients with HF typically require multiple drug classes, each targeting a different aspect of the syndrome (Medications Used to Treat Heart Failure | American Heart Association) (Medications Used to Treat Heart Failure | American Heart Association). Proper use of these medications can improve symptoms, **reduce hospitalizations, and prolong survival** in many patients (Heart failure drug treatment: the fantastic four - PMC) (Heart failure drug treatment: the fantastic four - PMC). This section covers common HF medications, their mechanisms, nursing considerations, and key side effects. (Refer to the comparison table at the end of this section for a summary.)

1. Diuretics (Preload Reducers): Diuretics help eliminate excess fluid, reducing blood volume (**preload**) and relieving pulmonary and peripheral congestion. **Loop diuretics** like **furosemide (Lasix)** and **bumetanide** are potent and commonly used for acute and chronic HF to relieve edema and SOB. They act on the renal loop of Henle to promote sodium and water excretion. **Thiazide diuretics** (e.g. **hydrochlorothiazide**) are milder and often used for hypertension but can be added in HF for additional diuresis. **Potassium-sparing diuretics** (e.g. **spironolactone**, **eplerenone**) are weaker diuretics primarily used for their aldosterone-blocking effects (see "aldosterone antagonists" below), but also spare K^+.

- *Indications:* Fluid overload in HF – any patient with signs of volume overload (edema, rales, JVD) will likely be on a diuretic to achieve **euvolemia**. Loop diuretics are first-line for symptomatic relief in HFrEF and HFpEF with congestion (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP). They do **not** by themselves improve mortality, but they **improve symptoms** and exercise tolerance (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP).
- *Mechanism:* In HF, the kidneys often retain fluid due to low perfusion. Diuretics block sodium reabsorption in the nephron, increasing diuresis. Loop diuretics (furosemide) cause strong diuresis even in reduced kidney function, which is helpful in HF. This reduces preload and pulmonary congestion almost immediately (within hours), improving breathing.
- *Nursing Considerations:* **Monitor intake/output and daily weights** to track fluid loss. A daily weight change of >2 lbs can signal fluid shifts. **Check electrolytes**, especially potassium: loop and thiazide diuretics can cause **hypokalemia** and **hyponatremia**. Low K^+ predisposes to arrhythmias (and digoxin toxicity if the patient is on digoxin). Ensure the patient is on potassium supplements or a potassium-sparing agent if needed. **Monitor blood pressure** – excessive diuresis can lead to hypotension and dizziness. Instruct patients to change positions slowly (orthostatic hypotension risk). Give diuretics in the **morning** (and afternoon if BID) to avoid nocturnal diuresis disrupting sleep. For IV diuretics, monitor for rapid urine output and be prepared with a bathroom/bedpan plan. **Renal function** should be watched; overdiuresis can impair kidney perfusion (rising BUN/Cr). Assess lung sounds and edema to evaluate effectiveness.
- *Side Effects:* **Volume depletion** (dehydration, hypotension), **electrolyte imbalances** (↓K, ↓Mg, ↓Na), and dizziness. Loop diuretics can cause **ototoxicity** (hearing damage) at high doses or rapid IV administration. Thiazides can increase calcium and uric acid levels (potential to precipitate gout). Spironolactone can cause **hyperkalemia** (especially if combined with ACE inhibitors or ARBs) and endocrine side effects like breast tenderness or gynecomastia (in men).
- *Signs of Toxicity:* Excess diuresis can lead to prerenal azotemia (↑BUN, dehydration). Watch for confusion, extreme weakness, or arrhythmias due to electrolyte losses. In HF, the goal is gentle daily diuresis to relieve symptoms without causing acute kidney injury or symptomatic hypotension.

2. RAAS Inhibitors (Afterload Reducers): The renin-angiotensin-aldosterone system (RAAS) is often overactive in HF, leading to vasoconstriction and fluid retention. Medications that inhibit RAAS help **reduce afterload** (arterial resistance) and **preload**, and have proven mortality benefits in HFrEF (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP). Key classes: **ACE Inhibitors, ARBs, and ARNIs.**

- **ACE Inhibitors (ACEIs)** – e.g. **enalapril, lisinopril, ramipril**.
    - *Mechanism:* ACEIs block the conversion of angiotensin I to angiotensin II. This leads to **arterial vasodilation** and reduced aldosterone release. The net effect is lower blood pressure and decreased workload on the heart. By widening blood vessels, ACEIs **lower afterload**, making it easier for the heart to eject blood

(Medications Used to Treat Heart Failure | American Heart Association) (Medications Used to Treat Heart Failure | American Heart Association). They also reduce sodium/water retention (via less aldosterone) to some degree, helping with preload.

- *Indications:* **HFrEF (EF <40%)** – ACE inhibitors are first-line therapy in Stage B or C HFrEF (even asymptomatic) to improve survival (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP). They are started as soon as HF with reduced EF is diagnosed, unless contraindicated. Also indicated post-MI with low EF, or in diabetics with cardiovascular risk (Stage A/B) to prevent HF.
- *Nursing Considerations:* Monitor **blood pressure** (risk of hypotension, especially first doses). Educate patients that a persistent **dry cough** can occur (due to bradykinin buildup); if intolerable, they may be switched to an ARB (Medications Used to Treat Heart Failure | American Heart Association). Check **renal function and potassium** – ACEIs can increase K^+ and creatinine. It's normal for creatinine to bump slightly, but >30% rise may indicate renal issues. Ensure the patient avoids potassium-rich salt substitutes. ACEIs are contraindicated in pregnancy and history of angioedema.
- *Side Effects:* **Cough** (common), **hyperkalemia**, **hypotension**, dizziness. **Angioedema** (rapid swelling of face/airway) is a rare but serious allergic reaction – if this occurs, the drug must be stopped and not reintroduced.
- *Toxicity:* Severe hypotension (especially if dehydrated or on diuretics) – hold or reduce dose if SBP <90 and symptomatic. Significant hyperkalemia (>5.5) or renal dysfunction are signs to adjust therapy. Overdose could cause refractory hypotension and is managed supportively.

- **Angiotensin II Receptor Blockers (ARBs)** – e.g. **losartan, valsartan, candesartan**.

  - *Mechanism:* ARBs block the angiotensin II receptor, preventing the effects of angiotensin II (vasoconstriction and aldosterone secretion). Their effects are similar to ACEIs – **vasodilation and reduced afterload** (Medications Used to Treat Heart Failure | American Heart Association). They are essentially an alternative to ACEIs, working downstream in the RAAS.
  - *Indications:* HFrEF in patients who cannot tolerate an ACEI (often due to cough or angioedema). ARBs also improve outcomes in HF and are recommended if ACEIs are not suitable (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP). They can also be used in addition to beta-blockers in Stage B (as in post-MI with low EF).
  - *Nursing Considerations:* Similar to ACEIs: monitor BP, renal function, K^+. ARBs do **not cause cough** (they don't affect bradykinin), so they are a good substitute if ACEI cough is problematic (Medications Used to Treat Heart Failure | American Heart Association). Angioedema is far less common with ARBs but still use cautiously if patient had ACEI angioedema.
  - *Side Effects:* Hypotension, dizziness, hyperkalemia. Rarely angioedema. ARBs are also contraindicated in pregnancy.

    - *Toxicity:* Overdose leads to hypotension; treat supportively. Marked hyperkalemia or renal impairment requires dose adjustment or holding the medication.
- **ARNI (Angiotensin Receptor–Neprilysin Inhibitor)** – **sacubitril/valsartan (Entresto)**.
    - *Mechanism:* This combination of sacubitril (a neprilysin inhibitor) with valsartan (an ARB) enhances neurohormonal modulation. **Sacubitril** blocks neprilysin, an enzyme that breaks down natriuretic peptides; this increases levels of beneficial natriuretic peptides, causing vasodilation and sodium excretion (Medications Used to Treat Heart Failure | American Heart Association). Meanwhile **valsartan** provides ARB effects. Together, ARNI leads to vasodilation, reduced afterload and preload, less remodeling, and diuresis.
    - *Indications:* **Chronic HFrEF (NYHA II–III)** to replace an ACEI or ARB in appropriate patients (Medications Used to Treat Heart Failure | American Heart Association). Guidelines now recommend ARNIs as a **first-line option** in place of ACEI/ARB for symptomatic HFrEF, due to superior outcomes in trials (The four pillars of HFrEF therapy: is it time to treat heart failure ...) (Heart failure drug treatment: the fantastic four - PMC). Patients are often transitioned to Entresto from an ACEI if they remain symptomatic. *Note:* there must be a 36-hour washout when switching from an ACEI to Entresto to avoid angioedema.
    - *Nursing Considerations:* Similar monitoring as ACEI/ARB – blood pressure and labs for renal function and K^+. **Do not combine with an ACEI** (risk of serious angioedema) (Medications Used to Treat Heart Failure | American Heart Association). Educate the patient that this is a combination medication; they should not be on a separate ARB while taking Entresto. Also, because neprilysin inhibitors can raise bradykinin like ACEIs, **angioedema risk** is present – watch for swelling of lips/tongue.
    - *Side Effects:* Hypotension is relatively common; also dizziness, cough (less than ACEI but can occur), hyperkalemia, and renal effects.
    - *Toxicity:* Profound hypotension or angioedema are the main concerns. If angioedema occurs, discontinue immediately and provide appropriate care.

3. Beta-Adrenergic Blockers (Beta-Blockers): Beta-blockers improve HF by slowing the heart rate and reducing myocardial oxygen demand, allowing better filling and decreasing harmful neurohormonal activation. Surprisingly, although they initially can make patients feel worse, long-term they **improve EF, reduce symptoms, and increase survival** in HFrEF (Heart failure drug treatment: the fantastic four - PMC) (Heart failure drug treatment: the fantastic four - PMC).

- *Examples:* **Metoprolol succinate (Toprol XL)**, **Carvedilol (Coreg)**, **Bisoprolol** – these three have strong evidence in HFrEF (Medications Used to Treat Heart Failure | American Heart Association). Carvedilol blocks beta and alpha receptors (providing additional vasodilation), while metoprolol and bisoprolol are selective beta-1 blockers. (Other beta-blockers without proven benefit in HF, like atenolol or propranolol, are generally **avoided** in HFrEF (Medications Used to Treat Heart Failure | American Heart Association).)

- *Mechanism:* By blocking beta-1 adrenergic receptors in the heart, beta-blockers **slow the heart rate and reduce the force of contraction** initially (Medications Used to Treat Heart Failure | American Heart Association). This can improve diastolic filling time and reduce arrhythmia risk. Over time, beta-blockade counteracts the chronic sympathetic overactivation in HF that causes tachycardia and contributes to remodeling. The result is **reverse remodeling** – the left ventricle can actually become less dilated and function better with sustained beta-blocker therapy. Beta-blockers also reduce renin release from kidneys (helping RAAS).
- *Indications:* **HFrEF (EF <40%)**, NYHA class II-III, stable patients. Beta-blockers are recommended for all stable HFrEF patients in Stage B or C, added to ACEI/ARB therapy (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP). It's important the patient be clinically stable (no recent IV inotropes, not in acute decompensation with pulmonary edema) at initiation. They are usually started at *low doses* and slowly titrated up to target doses used in trials. In HFpEF, beta-blockers may be used to control heart rate especially if afib or hypertension, but mortality benefit is clear in HFrEF.
- *Nursing Considerations:* **Start low, go slow.** Monitor heart rate and blood pressure carefully. **Bradycardia** and **hypotension** are common when initiating or up-titrating – if HR <50 or new AV block or SBP <90 with symptoms, notify the provider; dosage adjustments may be needed. Teach patients that they may not feel better right away – in fact, fatigue or slight worsening of symptoms can occur in the first 1-2 weeks. Encourage them to continue as tolerated because benefits occur over months. **Monitor for asthma or COPD exacerbation** (especially with nonselective beta blockers or higher doses, bronchospasm can occur). Carvedilol can cause more hypotension due to alpha blockade; giving it with food and in divided doses can lessen dizziness. **Do not abruptly withdraw** beta-blockers, as rebound tachycardia or ischemia can occur – taper if discontinuing.
- *Side Effects:* **Bradycardia**, **hypotension**, dizziness, fatigue, and depression. May mask signs of hypoglycemia in diabetics (caution patients to monitor blood sugar closely). Sexual dysfunction can occur. In asthma patients, wheezing or SOB might worsen (use beta-1 selective in these patients and monitor).
- *Signs of Toxicity:* Severe bradycardia, hypotension unresponsive to IV fluids, heart block, or acute decompensation (if the dose is too high for patient tolerance). Overdose of beta-blockers can cause shock and is treated with supportive care, possibly glucagon and other medications.

4. Aldosterone Antagonists (MRAs): These are **potassium-sparing diuretics** that block aldosterone's effects. **Spironolactone** and **Eplerenone** fall in this category. While mild diuretics, their main benefit in HF is blunting the cardiac fibrosis and remodeling caused by aldosterone.

- *Mechanism:* They **antagonize aldosterone** at receptors in the distal nephron, causing modest sodium excretion while retaining potassium (Medications Used to Treat Heart Failure | American Heart Association) (Medications Used to Treat Heart Failure | American Heart Association). In HF, aldosterone levels are high (even with ACEI/ARB on board), which leads to sodium retention, potassium loss, and myocardial fibrosis. By

blocking aldosterone, these drugs reduce fibrotic remodeling of the heart and vasculature and help maintain K^+.

- *Indications:* **HFrEF (EF <35%)** with ongoing symptoms (NYHA II-IV) despite ACEI (or ARB/ARNI) + beta-blocker (Medications Used to Treat Heart Failure | American Heart Association). They have a Class I recommendation in guidelines for these patients, unless contraindicated, due to proven mortality reduction. Post-MI patients with low EF or HF symptoms also benefit. Note: In **HFpEF**, some data suggests MRA may help if EF 45-50% with elevated BNP, but benefit is less clear.
- *Nursing Considerations:* Check baseline **kidney function and potassium** before starting – these drugs can cause **hyperkalemia**, especially in patients with CKD or if combined with ACEI/ARB. Generally avoid if Cr >2.5 (or eGFR <30) or K^+ ≥5.0 prior to initiation. Monitor K^+ and Cr within 3 days, 1 week, and monthly in the first few months. Educate patients to avoid high potassium diet or supplements unless directed. Spironolactone can cause endocrine side effects (it has some anti-androgen activity): men may develop **gynecomastia**, erectile dysfunction; women may have menstrual irregularities. Eplerenone is costlier but has fewer hormonal side effects (more selective for aldosterone receptor). Continue to monitor blood pressure (though MRAs only mildly lower BP).
- *Side Effects:* **Hyperkalemia** is the major concern. Others: gynecomastia (spironolactone) (Medications Used to Treat Heart Failure | American Heart Association), breast tenderness, and GI upset. Generally well tolerated aside from those.
- *Toxicity:* Manifested as high potassium – muscle weakness, peaked T-waves on ECG, arrhythmias. If K^+ rises too much, dose reduction or cessation is needed, and treatment of hyperkalemia as appropriate.

5. Vasodilators (Nitrates and Hydralazine): Nitrates (e.g. **nitroglycerin**, **isosorbide dinitrate**) and **Hydralazine** are often used in combination to reduce cardiac workload. Nitrates primarily dilate veins (reducing preload) and also coronary arteries, while hydralazine dilates arteries (reducing afterload) (Medications Used to Treat Heart Failure | American Heart Association). The combination of **hydralazine + isosorbide dinitrate** is particularly beneficial in HFrEF for patients who cannot tolerate ACEI/ARB, and it has added benefit in African American patients with HFrEF (per A-HeFT trial) (Heart failure management guidelines: New recommendations and ...).

- *Mechanism:* **Nitrates** convert to nitric oxide in the vascular endothelium, causing smooth muscle relaxation. This leads to **venous dilation**, which pools blood in peripheral veins and decreases venous return to the heart (lower preload), and also dilates coronary arteries to improve blood flow to myocardium (Medications Used to Treat Heart Failure | American Heart Association). **Hydralazine** acts directly on arterial smooth muscle to prevent contraction, thereby lowering systemic vascular resistance (afterload). By reducing afterload, hydralazine helps increase forward cardiac output from a weak LV. In combo, hydral-nitrates reduce both preload and afterload significantly.
- *Indications:* **HFrEF** patients who remain symptomatic. Particularly indicated in self-identified **Black/African American patients with HFrEF** NYHA III-IV on optimal ACEI/BB therapy (Heart failure management guidelines: New recommendations and ...),

or any patient who cannot take ACEI/ARB due to intolerance or renal issues – hydralazine + isosorbide dinitrate is an alternative regimen to provide mortality benefit. Also used in acute settings: **IV nitroglycerin** is common in acute pulmonary edema for rapid preload reduction and relief of hypertension. Long-acting oral nitrates (isosorbide mononitrate) may be used in HFpEF or for coexisting angina.

- *Nursing Considerations:* **Monitor blood pressure** – these vasodilators can cause **significant hypotension** (especially nitrates). Headache is extremely common with nitrates (due to cerebral vasodilation) – advise patients that headaches often diminish over time; acetaminophen can help. Ensure a "nitrate-free interval" for long-term nitrates (e.g., 10-12 hours overnight) to prevent tolerance. Hydralazine can cause reflex tachycardia; often it's given with a beta-blocker to counteract that when used for hypertension. Monitor for **orthostatic hypotension** and instruct patient to rise slowly. If using topical nitrate patches or paste, follow protocols (wear gloves when handling paste, remove old patches, rotate sites). **Avoid nitrates in patients who have taken phosphodiesterase inhibitors (e.g., sildenafil) in the last 24-48 hours** – risk of serious hypotension.
- *Side Effects:* Nitrates: headache, flushing, dizziness, hypotension. Hydralazine: headache, tachycardia, palpitations, anorexia, and in rare cases a lupus-like syndrome with long-term use (fever, joint pain, rash).
- *Toxicity:* Extreme hypotension (especially if combined with other vasodilators or overdose of nitroglycerin). Nitrate overdose may also cause reflex tachycardia and in severe cases methemoglobinemia (presenting as hypoxia and chocolate-colored blood). Hydralazine toxicity could present as hypotension and tachyarrhythmia.

6. Positive Inotropes: In advanced or acute decompensated HF, medications that **increase myocardial contractility** can be lifesaving or improve symptoms. However, chronic use of most inotropes is associated with worse mortality, so in chronic HF only one oral inotrope (digoxin) is used selectively. IV inotropes (dobutamine, milrinone) are used for acute cardiogenic shock or as palliative bridge in end-stage HF.

- **Digoxin:**
    - *Mechanism:* Cardiac glycoside that inhibits the Na^+/K^+ ATPase pump in cardiac cells, leading to increased intracellular calcium and stronger contractions. It also slows AV node conduction, providing rate control in atrial fibrillation. Result: **increased contractility (positive inotropy)** and **slowed heart rate** (especially in afib).
    - *Indications:* Was once a mainstay, now used in **HFrEF (EF <40%) with persistent symptoms** despite optimal beta blocker, ACEI, diuretics. It can improve symptoms and exercise tolerance and reduce hospitalizations, but it **does not improve survival**. Often used if the patient has **atrial fibrillation with rapid rate** and HF (helps control rate and support output). In Stage D HF, low-dose digoxin may be continued for symptom relief.
    - *Nursing Considerations:* **Monitor heart rate and rhythm** – dig slows AV conduction, risk of bradycardia or AV block. Always check **apical pulse for 1 full**

minute before administration; withhold if pulse <60 bpm and notify provider (as per facility protocol). **Monitor potassium level** closely: hypokalemia (from diuretics) greatly increases risk of digoxin toxicity. Also monitor kidney function – digoxin is renally excreted; dose may need adjustment in renal impairment. Educate patient on recognizing toxicity signs (see below) and to keep potassium intake consistent.

- *Side Effects:* GI upset (nausea, vomiting, anorexia) can occur even at therapeutic levels. Fatigue, weakness, and mental status changes may be noted. It can precipitate arrhythmias (because it increases automaticity while slowing AV conduction).
- *Toxicity:* **Digoxin toxicity** is a critical concern. Signs include *GI symptoms* (anorexia, nausea, vomiting), *visual disturbances* (blurred vision, yellow-green halos around lights), confusion, and *cardiac arrhythmias* (extra beats, AV block, atrial tachyarrhythmias, ventricular arrhythmias). Digoxin has a narrow therapeutic range (~0.5–2.0 ng/mL). Toxicity is more likely with hypokalemia, hypomagnesemia, or reduced renal clearance. If toxicity is suspected, obtain a digoxin level and EKG (look for AV block, bradycardia, PVCs, or bidirectional VT). Antidote **Digoxin Immune Fab (Digibind)** may be used for severe toxicity. Nurses must be vigilant in monitoring for these signs, especially in elderly patients.

- **IV Inotropes (Dobutamine, Milrinone):**
  - *Mechanism:* **Dobutamine** is a beta-1 agonist (with some beta-2), causing increased contractility and mild vasodilation. **Milrinone** is a phosphodiesterase-3 inhibitor that increases cAMP in heart cells (positive inotrope) and in vascular smooth muscle (vasodilator); it's sometimes called an inodilator. Both increase cardiac output in acute settings.
  - *Indications:* **Acute decompensated HF with low output** (cardiogenic shock or end-organ hypoperfusion) – they are given IV in ICU to stabilize patients. Also used as a temporary bridge to decision or transplant, or as palliative therapy in stage D HF (continuous home infusions) when no other options remain. Not used long-term routinely due to mortality risk.
  - *Nursing Considerations:* These are IV drip medications, typically in ICU or advanced HF unit. They require continuous **hemodynamic monitoring**: watch blood pressure (risk of hypotension, especially milrinone due to vasodilation) and cardiac rhythm (risk of arrhythmias). **Dobutamine** can provoke tachycardia or arrhythmias – continuous telemetry is required. **Milrinone** can cause significant vasodilation; monitor for hypotension and also check liver/kidney as it's excreted renally. Ensure IV access is secure (central line preferred for vesicants). For home therapy patients, educate on pump use and to report any palpitations or dizziness immediately.
  - *Side Effects:* Arrhythmias (atrial or ventricular), hypotension, headache (especially milrinone due to vasodilation), tremors (dobutamine).

- *Toxicity:* Over-infusion can lead to severe arrhythmias or profound hypotension. For dobutamine, watch for chest pain or excessive tachycardia as it increases myocardial oxygen demand (can provoke ischemia). These infusions are titrated carefully to avoid such issues.

7. Other Medications and Considerations:

- **SGLT2 Inhibitors:** Originally diabetes meds, **dapagliflozin** and **empagliflozin** are now approved for HFrEF (even without diabetes) because they modestly improve outcomes. They cause the kidneys to excrete glucose and sodium (diuretic effect, weight loss) (Medications Used to Treat Heart Failure | American Heart Association) (Medications Used to Treat Heart Failure | American Heart Association). Indicated in HFrEF NYHA II-IV to reduce hospitalization and CV death. Nursing notes: watch for genital yeast infections, monitor renal function (don't use if GFR too low), and patient should report any ketoacidosis symptoms if diabetic. These are usually added by cardiology HF specialists.

- **Ivabradine:** Slows sinus node (reduces HR) without blood pressure effect, used in HFrEF with resting HR ≥70 on max beta-blocker. It lowers hospitalization risk. Nurses should monitor heart rate (can cause bradycardia or luminous phenomena in vision).

- **Anticoagulation:** Not for HF per se, but if patient is in **atrial fibrillation** (common in HF) or has a low EF with intracardiac thrombus, they may be on warfarin or a NOAC to prevent clots. Ensure INR monitoring for warfarin and patient education on bleeding precautions, as needed.

- **Vasopressin Antagonists (e.g. Tolvaptan):** Occasionally used short-term for severe hyponatremia in HF to free water excrete. Not routine; monitor sodium closely if used.

- **Analgesics/Sedatives:** Morphine used to be given in acute pulmonary edema for anxiety and vasodilation; now it's used sparingly due to respiratory depression risk, but you might see low-dose morphine in acute settings to help relieve distress. Also, anxiety can worsen dyspnea, so a calm environment or low-dose anxiolytic may be helpful in acute settings (with caution).

Medication Summary Table: Below is a summary of major medication classes in CHF:*Table: Summary of common HF medications, their actions, and nursing considerations.*

This medication guide highlights the most common therapies. Other drugs like SGLT2 inhibitors and ivabradine are emerging treatments included in updated guidelines pmc.ncbi.nlm.nih.gov.

Medication Class	Mechanism & Benefits	Key Nursing Considerations
Loop Diuretics (Furosemide, etc)	Inhibit $Na^+/K^+/Cl^-$ reabsorption in loop of Henle → strong diuresis. Reduce preload & relieve congestion aafp.org.	Monitor I&O, daily weight; watch K^+ (risk hypoK) and BP. Can cause dehydration, hypotension, kidney injury. Give in AM. Assess lung sounds & edema for effect.
Thiazide Diuretics (HCTZ)	Inhibit NaCl reabsorption in distal tubule → moderate diuresis. Lower BP, mild edema relief.	Monitor K^+, Na^+ (can drop both). Often added if loop diuresis inadequate. Less effective if GFR low.
ACE Inhibitors (Lisinopril, etc)	Block Angiotensin II formation → vasodilation (↓afterload) and ↓aldosterone (less fluid retention). Slow HF progression, improve survival aafp.org.	Check BP & renal labs; **cough** common heart.org. Watch K^+ (risk hyperK). Contraindicated in pregnancy. Monitor for angioedema (rare).
ARBs (Losartan, Valsartan)	Block Ang II receptors → similar vasodilatory & anti-aldosterone effects as ACEI. Improve outcomes in HFrEF aafp.org.	Use if ACEI not tolerated (no cough) heart.org. Monitor BP, K^+, renal function. Avoid in pregnancy. Angioedema possible but less likely.
ARNI (Entresto: sacubitril/valsartan)	Neprilysin inhibitor + ARB → ↑natriuretic peptides (vasodilation, diuresis) + RAAS blockade heart.org. Further reduces mortality in HFrEF vs ACEI.	Ensure 36h washout from ACEI before start heart.org. Monitor BP (often causes low BP), K^+, renal. Educate about angioedema risk & to avoid ACEI concurrently.

Beta-Blockers (Metoprolol, Carvedilol)	Block β1 (±β2) receptors → slow HR, ↓BP, allow ventricle to fill better, anti-arrhythmic. Over time, improve EF and survival pmc.ncbi.nlm.nih.gov.	Start low dose in stable HF, titrate up slowly aafp.org. Check HR (hold if <50) and BP. Can initially cause fatigue or worse symptoms – reassure patient. Contraindicated in acute decomp HF, asthma caution (use selective).
Aldosterone Antagonists (Spironolactone)	Block aldosterone receptor → mild diuresis, K^+ retention, reduced fibrosis. Lowers mortality in HFrEF NYHA II–IV heart.org.	Monitor **K^+ closely** (risk of hyperkalemia) heart.org, especially with ACEI/ARB. Check renal function; avoid if Cr >2.5. Spironolactone: watch for gynecomastia in males.
Hydralazine + Nitrate (Bidil or separate)	Hydralazine: arterial dilator (↓afterload). Nitrate: venodilator (↓preload) heart.org. Together improve symptoms & survival in HFrEF (esp. Black patients) sciencedirect.com.	Monitor BP (risk of hypotension). Headache common (nitrates). Hydralazine can cause reflex tachycardia – often given with β-blocker. Ensure nitrate-free interval to prevent tolerance. Educate to avoid ED meds with nitrates.
Digoxin (cardiac glycoside)	Inhibits Na^+/K^+ ATPase → ↑Ca^{2+} in heart cells (stronger contraction); slows AV node (↓HR). Improves symptoms, exercise tolerance (not survival).	Check apical pulse, hold if <60. Monitor digoxin level (0.5–2 ng/mL) and **K^+** (hypoK increases toxicity risk). Watch for toxicity: GI upset, vision changes, arrhythmias – report immediately.
IV Inotropes (Dobutamine, Milrinone)	β1 agonist (dobutamine) or PDE-3 inhibitor (milrinone) → ↑contractility; milrinone also vasodilates (↓afterload). Used in acute HF or cardiogenic shock.	ICU monitoring – continuous BP, EKG. Titrate per orders. Risk of arrhythmias – have defib/pacing nearby. Assess perfusion (urine output, mentation) to gauge effect. For milrinone, monitor BP and renal function. Typically short-term use.

Always refer to the latest HF guidelines for comprehensive recommendations, and use clinical judgment for individual patient needs.

Common Cardiac Interventions in HF Patients

Many patients on a cardiac unit, particularly those with CHF, have underlying coronary artery or valvular disease. They may undergo **invasive interventions or surgeries** to treat the root cause of their heart failure or related cardiac issues. Understanding these procedures and the nursing care before/after is crucial for safe patient management. Below we cover **Coronary Artery Bypass Grafting (CABG)**, **Heart Valve Repair/Replacement**, and **Percutaneous Coronary Intervention (PCI)** – common interventions you may encounter.

Coronary Artery Bypass Grafting (CABG):
CABG is a major **open-heart surgery** to treat severe coronary artery disease. In a CABG, a surgeon takes blood vessel grafts from elsewhere (commonly the **internal mammary artery** from the chest, and/or the **saphenous vein** from the leg) and creates new routes to bypass blocked coronary arteries (Coronary Artery Bypass Graft - StatPearls - NCBI Bookshelf). This restores blood flow to ischemic heart muscle, relieving angina and potentially improving heart function (Coronary Artery Bypass Graft - StatPearls - NCBI Bookshelf) (Coronary Artery Bypass Graft - StatPearls - NCBI Bookshelf). In the illustration below, you can see a heart with three bypass grafts: a left internal thoracic (mammary) artery graft to the LAD, and two vein grafts (one on the right coronary, one on a branch of the circumflex) (Coronary artery bypass surgery - Wikipedia) (image). *(Image: Heart with bypass grafts – vessels from chest wall and leg attached to bypass blocked arteries, re-routing blood flow (Coronary artery bypass surgery - Wikipedia).)*

- **Purpose:** CABG is typically done for multi-vessel coronary disease or left main disease when stents are insufficient or not feasible. It is indicated in patients with significant blockages causing symptoms or impairing heart function, especially if anatomy is complex. For HF patients, if ischemic cardiomyopathy (HF due to CAD) is present, CABG can improve EF and symptoms by revascularizing viable myocardium (Coronary Artery Bypass Graft - StatPearls - NCBI Bookshelf). It **improves survival in certain high-risk coronary disease** (like left main or 3-vessel disease with low EF) compared to medical therapy or PCI (Coronary artery bypass surgery - Wikipedia) (Coronary artery bypass surgery - Wikipedia).

- **Pre-Op Nursing Role:** Focus on **patient education and optimization.** Ensure the patient understands the procedure (use teach-back for concepts like “bypass” meaning new pathway around blockages (Coronary Artery Bypass Grafts | Heart and Vascular Center | DHMC and Clinics)). Typical pre-op orders include NPO after midnight, obtaining baseline labs (CBC, BMP, coagulation studies, type & cross for blood), and possibly special bathing (chlorhexidine wash to reduce infection risk). **Assess and mark peripheral pulses** (for post-op comparison, especially if a vein will be harvested from the leg). Discuss what to expect: an incision in the chest (and possibly leg incisions), placement on cardiopulmonary bypass machine, the likelihood of waking up intubated in ICU, chest tubes, and pacing wires. Address anxiety – this is a big surgery; provide emotional support and involve family in teaching if appropriate. Ensure consent is signed and all pre-op checklist items (like removing jewelry, dentures, etc.) are done. Administer

pre-op medications as ordered (sometimes a benzodiazepine for relaxation, antibiotics for prophylaxis).

- **Post-Op Nursing Care:** CABG patients go to CVICU immediately post-op. As a floor nurse, you may receive them after 24-48 hours once stable, or you may care for them in a step-down. Key points:

    - **Airway/Breathing:** They will be extubated usually within 6-12 hours. Encourage **pulmonary hygiene**: coughing, deep breathing, incentive spirometry – very important to prevent atelectasis and pneumonia. Manage pain so they can cough (splint the chest incision with a pillow).

    - **Circulation:** Monitor vital signs and heart rhythm continuously. Post-CABG arrhythmias are common (especially **atrial fibrillation** in ~30% of patients); be ready to administer beta-blockers or amiodarone if ordered (Caring for a patient after coronary artery bypass graft surgery - LWW). Check perfusion: skin color, temp, peripheral pulses.

    - **Hemodynamics:** Watch blood pressure closely – hypotension may indicate bleeding, and hypertension can stress the new grafts. Often parameters will be set (e.g., keep SBP 90-120). They will have one or more **chest tubes** draining fluid from around the heart; **monitor chest tube output** hourly initially. Report excessive drainage (e.g., >150 mL/hr) as it could indicate hemorrhage, and be alert to sudden cessation of output which could signify a tamponade (fluid accumulation compressing the heart). Maintain patency of chest tubes (no kinks, ensure dependent drainage). If pacing wires are present (epicardial wires), know how to handle them (keep capped and secured; if heart block or bradycardia occurs, they may be attached to a temporary pacemaker).

    - **Fluid balance:** They often have significant fluid shifts post-op. Monitor I&O, daily weights, and adjust IV fluids per protocol. Edema is common due to bypass pump; diuretics may be started on day 1-2 post-op to reach dry weight.

    - **Incision care:** They will have a **sternotomy incision** (down the middle of chest) and likely a leg incision (if vein harvested). Keep incisions clean and dry; follow surgeon's orders for dressing changes (some use occlusive dressings for 48h, others leave open to air if dry). Watch for signs of infection (redness, warmth, drainage). **Sternal precautions** are typically in place: no lifting >5-10 lbs, avoid using arms to push/pull (to prevent dehiscence of sternum). Instruct to hug a pillow when coughing or moving.

    - **Pain management:** They will have significant pain from the sternotomy and possibly the leg. Regularly assess pain (expect they'll be on IV or PO analgesics). Adequate pain control is crucial for them to participate in breathing

exercises and mobilization.

- **Mobility:** Early mobilization is important – usually sitting up on day 1, and ambulating by day 2 if stable. Work with physical therapy. Prevent DVT with compression devices until ambulating well.

- **Other:** Monitor labs – expect an inflammatory response (WBC may be elevated, low-grade fever common first 1-2 days). Manage blood sugars even in non-diabetics (often on insulin drip or sliding scale, as tight glucose control improves outcomes post-op). If the patient received blood transfusions or a long pump run, watch kidney function (creatinine) and cognitive status (some experience "pump head," cognitive changes that usually improve). Provide **emotional support** – patients can feel vulnerable with new equipment (chest tubes, etc.) and may have mood swings or even post-CABG depression. Engage family support, and consider pastoral care or counselor if needed.

- **Complications to watch for: Bleeding** (e.g., from chest tubes, internal; signs: dropping BP, increased HR, output in drains), **cardiac tamponade** (beck's triad: JVD, muffled heart sounds, hypotension; also sudden stop of chest tube output and pulsus paradoxus), **arrhythmias** (atrial fib, heart blocks, ventricular arrhythmias), **infections** (sternal wound infection or pneumonia – fever >101°F and purulent sternal drainage could indicate mediastinitis, a serious complication), **pericardial friction rub** (post-pericardiotomy syndrome). Also monitor for **renal failure** and **stroke** (neuro checks each shift – stroke risk exists from aortic plaque emboli during surgery).

- **Outcomes:** With successful CABG, patients often have relief of angina and improved exercise capacity. If HF was due to ischemia, and viable myocardium is revascularized, EF can improve over months. Long term, patients need risk factor management (no smoking, healthy diet, cardiac rehab exercise program) to keep grafts patent. Grafts can reocclude over years, especially vein grafts, so aspirin and statin therapy are standard post CABG to improve graft longevity.

Valve Repair or Replacement (Valve Surgery):
Heart failure can be caused or exacerbated by **valvular heart disease** (e.g., aortic stenosis, mitral regurgitation). Surgical or percutaneous intervention on valves can dramatically improve HF symptoms when the valve lesion is corrected.

- **Common Valve Issues:** Severe **aortic stenosis (AS)** causes pressure overload on LV (can lead to hypertrophy and HF) – replacing a tight aortic valve often yields big improvements in symptoms. Severe **mitral regurgitation (MR)** causes volume overload and pulmonary congestion – repair or replacement can alleviate HF. Other valves (aortic regurg, mitral stenosis, tricuspid or pulmonary valve disease) can also cause or worsen

HF and may need intervention.

- **Approaches:** Valves can be fixed via **open-heart surgery** (traditional valve replacement or repair with a surgical approach using cardiopulmonary bypass) or via **transcatheter techniques** (like TAVR – Transcatheter Aortic Valve Replacement, done via catheter without open surgery for aortic valves, or MitraClip for certain mitral regurg cases).

- **Nursing Considerations Pre-Op:** Similar to CABG if it's open surgery: patient education, consent, pre-op testing (including often a cardiac cath prior to valve surgery to check coronary arteries). If it's a transcatheter procedure (TAVR), still need consent and NPO; often done in cath lab or hybrid OR. Check for specific instructions (for TAVR, maybe different anticoagulation considerations). Ensure dental clearance for valve surgery because of endocarditis risk (often patients need dental exam pre valve replacement).

- **Post-Op Care:** If open surgery, the immediate post-op care is very similar to CABG (sternotomy incision, possible bypass graft at same time if needed, etc.). Monitor for the same complications. Additionally, for valve patients:

    - For **mechanical valve replacements**, patients will require lifelong anticoagulation (usually warfarin). Nurses must monitor INR and teach patients about anticoagulation safety (avoid high-risk activities for bleeding, consistent diet if on warfarin, regular blood checks).
    - **Bioprosthetic valves** (tissue valves) usually don't require long-term warfarin (often just aspirin), but they may degenerate in ~10-15 years.
    - Watch for signs of prosthetic valve dysfunction or thrombosis: new murmurs, HF symptoms, or **embolic events** (stroke symptoms could indicate a clot on the valve).
    - **Infective endocarditis prophylaxis:** After valve surgery, patients will need antibiotic prophylaxis before certain dental or surgical procedures – ensure they are educated on this.
    - If it's a **transcatheter valve (TAVR)**, recovery is quicker: typically just a catheter insertion site (femoral artery) to monitor. Ensure good groin site care (like after PCI: observe for hematoma, keep leg straight for prescribed time if applicable). TAVR patients still go to ICU initially; nurse monitors for conduction issues (new heart block can occur requiring a pacemaker sometimes). Usually less invasive but still needs careful observation.
- **Outcomes:** Successful valve interventions often lead to marked improvement in HF symptoms. For example, a patient with severe aortic stenosis and heart failure can see increased EF and stamina after a new valve relieves the outflow obstruction. Mitral regurg patients often have less pulmonary edema once the valve is fixed. The overall HF prognosis improves if the valvular problem is resolved. However, if myocardial damage is long-standing, HF may persist, but usually at a better baseline.

Percutaneous Coronary Intervention (PCI):
Also known as **angioplasty with stenting**, PCI is a non-surgical procedure to open blocked coronary arteries. Many CHF patients have ischemic heart disease, and you will encounter patients who have had PCI for heart attacks or angina. Additionally, acute decompensation of HF can be triggered by myocardial infarction, leading to emergent PCI.

- **Procedure Overview:** PCI is done in the cardiac catheterization lab. A cardiologist accesses the arterial system (commonly radial artery in wrist or femoral artery in groin) and threads a catheter to the coronary arteries. They identify blockages on angiography. Then a balloon is inflated at the blockage site to dilate the artery (**angioplasty**), and usually a **stent** (mesh wire tube) is deployed to keep the artery open (Coronary stent - Wikipedia) (Coronary stent - Wikipedia). This restores blood flow to the heart muscle. For an acute MI, this aborts the infarction if done quickly, saving heart muscle. The diagram below illustrates stent placement: (A) catheter across lesion, (B) balloon inflating and expanding stent to compress plaque, (C) opened artery with stent in place (Coronary stent - Wikipedia).

- **Indications:**

    - **Acute coronary syndrome (ACS)** – PCI is the frontline treatment for ST-elevation MI (primary PCI within 90 minutes of arrival is the goal). It's also done for unstable angina or NSTEMI if significant lesions are found.
    - **Stable angina** – if symptoms aren't controlled by meds or stress testing shows high risk, PCI can be done to improve blood flow.
    - In context of HF, if a patient's HF is ischemic, PCI might be used to open arteries to improve EF (less invasive than CABG if suitable anatomy). Or if a patient with known HF develops chest pain and an MI, they'll undergo PCI. So you'll see HF patients post-PCI in your unit.
- **Nursing Role – Pre-PCI:** If a patient is scheduled for an elective PCI, ensure they understand the procedure. Check for **allergy to contrast dye or shellfish** (premedicate if needed). Ensure **renal function** is acceptable (contrast can worsen kidney function; hydration pre and post can help). Patient will be NPO except clear liquids (depending on policy) and should have IV access. If emergent (MI code), you may be involved in the rapid prep: quick consent (if possible), shaving the groin, etc. For **STEMI** patients, time is critical – preparation happens rapidly. Ensure any **anticoagulants or antiplatelet** usage is known to the team (they often get additional antiplatelet like P2Y12 inhibitors loading dose pre/during PCI). Educate that they may feel flushing or chest discomfort when the dye is injected, which is normal but should be reported if severe.

- **Post-PCI Nursing Care:** After PCI, patients may return with a vascular closure device in the artery or a pressure dressing. Key care points:

    - **Monitor the access site** (groin or wrist) for bleeding or hematoma. Check under the patient as well, as blood can pool. If femoral, keep the leg straight for the

prescribed time (often 4-6 hours if a manual closure or shorter if a closure device). If radial, the patient will have a tight wrist band (e.g., TR band) – monitor that site and gradual release per protocol.
    - **Frequent vital signs** per protocol (e.g., q15min x2h, etc.) and **neurovascular checks** of the extremity (distal pulses, cap refill, temperature, sensation) (Cardiac Catheterization | Johns Hopkins Medicine). Diminished pulses or cool, pale limb could indicate arterial obstruction or severe hematoma compressing vessels.
    - **Bedrest**: For femoral access, typically bedrest for a few hours; no heavy lifting or straining. For radial, the patient can sit up sooner, but avoid using that wrist and lifting with that arm for about 24-48 hours.
    - **Heart monitoring**: The patient should be on telemetry – watch for reperfusion arrhythmias (sometimes after an artery is opened, transient arrhythmias like VT can occur). Also, an MI patient could have arrhythmias from the infarct area.
    - **Chest pain**: The patient should have *no chest pain* after a successful PCI to an infarct-related artery. If chest pain recurs, treat it as an emergency – could indicate acute stent thrombosis or closure. Immediately notify the provider if new chest pain or ST changes on monitor.
    - **Medication**: Ensure the patient receives the prescribed **dual antiplatelet therapy (DAPT)** – typically aspirin indefinitely and a P2Y12 inhibitor (like clopidogrel, ticagrelor) for 6-12 months at least (Coronary stent - Wikipedia). These are crucial to prevent stent clots. Educate patient on medication adherence – stopping those prematurely can be life-threatening (stent thrombosis). Also, they might be on high-intensity statin therapy post-PCI (to stabilize plaques). Check that they have these orders and understand them.
    - **Hydration**: Encourage fluids if not contraindicated to help flush out contrast and protect kidneys. Monitor urine output.
    - **Back pain**: If femoral approach, sometimes lying flat can cause back discomfort. Ensure comfort, and that could be normal vs. severe back pain which could hint at retroperitoneal bleed (if blood leaking internally – watch for hypotension and flank bruising).
    - Provide patient education on risk factor modification and symptoms to report (for instance, if they had a stent placed, advise them to seek care for any recurrence of chest pain).
- **Outcomes:** PCI can abort a heart attack, improving survival and preserving heart muscle (so HF doesn't worsen). In chronic HF, opening an artery can improve EF if a significant viable myocardium was hibernating due to low blood flow. However, not all HF patients qualify for PCI – only if discrete blockages are present. After PCI, patients often experience relief of angina and can exercise more, which helps conditioning in HF. It's not a cure for HF, but part of the management of underlying CAD.

AHA Guideline-Based Heart Failure Management

The American Heart Association (AHA) and American College of Cardiology (ACC) publish guidelines that outline evidence-based management of heart failure. These guidelines emphasize a **multidisciplinary, systematic approach** to HF care to improve patient outcomes. Key elements include staging of HF, lifestyle modifications, medication therapy (often called **GDMT – Guideline-Directed Medical Therapy**), device therapies, and target outcomes to monitor. Here we summarize some AHA guideline-based standards and priorities (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP) (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP):

- **Risk Factor Management (Stage A):** For patients at risk of HF (e.g., hypertension, diabetes, CAD without symptoms), **preventive measures** are priority. Control hypertension and lipids aggressively (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP). Encourage regular exercise, weight management, and smoking cessation. These measures can prevent progression to symptomatic HF. Nurses in clinics should reinforce adherence to these lifestyle changes and medications for comorbidities.

- **Early Treatment of Structural Heart Disease (Stage B):** This stage is asymptomatic HF (e.g., low EF after an MI but no symptoms). Guidelines recommend starting **ACE inhibitors (or ARBs)** and **beta-blockers** in all patients with EF ≤40% even if asymptomatic (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP). This can prevent development of symptoms and reduce mortality. For example, a post-MI patient with EF 30% should be on an ACEI and beta-blocker (assuming no contraindications) to prevent remodeling (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP). Nurses should ensure these meds are initiated before discharge in post-MI or cardiomyopathy patients (core measures). Also in Stage B, if a patient has severe valvular disease, **surgical correction** should be considered before symptoms worsen (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP).

- **Comprehensive Treatment of Symptomatic HF (Stage C):** Stage C is established, symptomatic HF (most CHF patients on the unit). The **management priorities** per guidelines include:

    - **Combination of medications** proven to improve outcomes in HFrEF: specifically, the **"Four Pillars"** of HFrEF therapy are:
        1. **RAAS inhibition:** ACEI or ARB, or preferably ARNI (The four pillars of HFrEF therapy: is it time to treat heart failure ...).
        2. **Beta-blocker** (one of the proven ones) (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP).

3. **Aldosterone antagonist** (MRA) for appropriate patients (EF ≤35%, class II-IV) (Medications Used to Treat Heart Failure | American Heart Association).
4. **SGLT2 inhibitor** (dapagliflozin/empagliflozin) – recent addition with Class A recommendation for HFrEF (The four pillars of HFrEF therapy: is it time to treat heart failure ...).
   These, along with diuretics for symptom control, form the core of GDMT in HFrEF (Heart failure drug treatment: the fantastic four - PMC) (Heart failure drug treatment: the fantastic four - PMC). Every HFrEF patient should be evaluated for tolerance of these meds. For HFpEF, the medication impact on mortality is less clear, but treating blood pressure (ACEI/ARB, BB), managing fluid with diuretics, and addressing comorbidities is advised.

- **Hydralazine + Nitrate** combination is recommended in African American patients with HFrEF who remain symptomatic (Class I recommendation in addition to ACEI/BB) (Heart failure management guidelines: New recommendations and ...), or for any patient who can't take ACEI/ARB.
- **Diuretics** are recommended as needed for fluid control – use enough to relieve edema/ congestion in Stage C HF (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP). There's no mortality benefit, but they are crucial for symptom management.
- **Device Therapy:** If EF remains low (≤35%) on meds, **ICD (Implantable Cardioverter-Defibrillator)** is recommended in many patients to prevent sudden cardiac death (Heart failure management guidelines: New recommendations and ...) (particularly ischemic cardiomyopathy or non-ischemic with class II-III symptoms). Also, if a patient has left bundle branch block with wide QRS (>150ms) and EF ≤35%, **CRT (Cardiac Resynchronization Therapy)** biventricular pacemaker can improve coordination of contraction and increase EF (Heart failure management guidelines: New recommendations and ...). Nurses should ensure eligible patients get referred for these therapies – you might see patients post-ICD implant for monitoring.
- **Education and Self-Care:** AHA guidelines stress patient education in Stage C: diet (2g sodium, fluid restriction ~2L if hyponatremic), daily weights, medication adherence, and symptom monitoring (Heart Failure (1).pdf) (Heart Failure (1).pdf). Nurses provide counseling on low-sodium diet, fluid limit if prescribed, and what to do if weight increases (typically, call provider if >2-3 lbs in 24h or >5 lbs in a week). Ensure discharge instructions include these points (a core measure for HF).
- **Exercise Training:** Cardiac rehabilitation or regular exercise as tolerated is recommended to improve functional status (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP). Even in HF, supervised exercise can reduce symptoms and hospitalizations.
- **Comorbidities:** Manage sleep apnea (consider sleep study/CPAP), anemia (sometimes IV iron is given if ferritin low), diabetes (SGLT2i help both DM and

HF), depression (common in HF), etc. This holistic management improves outcomes.
    - **Follow-Up:** Close follow-up after hospitalization (within 7 days or at least 2 weeks) is recommended to reduce readmissions. Many places have HF clinics or nurse-led phone follow-ups.
- **Advanced Therapies for Refractory HF (Stage D):** If a patient has Stage D (symptoms at rest despite max therapy, frequent hospitalizations), guidelines advise referral to advanced HF specialists for evaluation of **heart transplant** or **mechanical circulatory support (LVAD)** (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP). Also, consideration of **palliative care** and goals of care discussions is important at this stage (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP). As bedside nurses, if you have a patient who frequently is in and out of ICU with HF, encourage them to discuss advanced options with their cardiologist. You might care for patients with LVADs – that's specialized, but remember they require anticoagulation and have unique assessments (often no palpable pulse due to continuous flow device).

    - For Stage D in hospital, sometimes IV inotropes at home or hospice for symptom relief might be an option if no definitive therapies are possible. Nursing includes teaching family how to manage IV infusions and recognizing end-of-life symptoms if hospice.
- **Target Outcomes:** AHA guidelines highlight goals like: improve **NYHA functional class**, increase exercise capacity, prevent hospital admissions, and prolong survival. In practice, we use proxies: maintain **optimal weight** (no big swings from fluid), keep **BP controlled** (e.g., target <130/80 in HF to reduce afterload unless symptomatic hypotension), achieve **HR ~60-70** in sinus (with beta-blocker, or <AF 80 if in afib), and titrate meds to **target doses** from trials if possible (Four Pillars, Fast? Rapid Sequencing of HF Drugs Faces an Uphill ...). We track lab markers too: a **decrease in BNP** over time can indicate improvement; stable or rising BNP might prompt therapy adjustment (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP). An improvement in EF on echo (say from 25% to 35%) after months of therapy is a positive sign (some patients may even have HF **"recovered EF"** if EF normalizes, but they must stay on meds).

    - Patient-centered targets are also key: e.g., "able to walk to mailbox without SOB" or "able to sleep through night without orthopnea." Document these functional goals as they matter greatly to quality of life.
    - **Preventive care** is part of HF standards too: ensure patients get annual **flu shots and pneumococcal vaccine** (respiratory infections can worsen HF).
- **Quality Measures:** Hospitals track HF metrics: ACEI/ARB/ARNI at discharge for EF<40% (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP), documentation of education, 7-day follow-up, etc.

In essence, AHA/ACC guidelines provide a roadmap: treat contributing conditions, employ proven therapies in a stepwise way, and educate/monitor patients to meet specific targets. Always stay updated because HF management evolves (e.g., new meds or indications get added – like SGLT2 inhibitors recently). By following guidelines, you'll be prioritizing interventions that have the strongest evidence for helping your patients (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP).

Practical Nursing Considerations for CHF Care

Managing heart failure goes beyond medications and procedures – **nursing care and patient teaching are pivotal** in preventing exacerbations and maintaining quality of life. Below are practical tips and knowledge areas for bedside nurses on a cardiac unit:

Monitoring Fluid Balance: Careful monitoring of fluid status is a daily nursing task for HF patients. **Daily weights** are one of the most sensitive indicators of fluid gain or loss – same time each morning, after voiding, using the same scale. A change of **1 kg (2.2 lb)** roughly equals 1 liter of fluid gained or lost. Instruct patients to track their weight at home daily and to notify the clinic for significant gains (commonly, 2–3 lbs in one day or 5 lbs in a week triggers a call) (Heart failure - Symptoms and causes - Mayo Clinic). On the unit, compare today's weight to yesterday's and admission baseline – upward trends may necessitate diuretics or evaluation of dietary adherence. **Intake & Output (I&O)** should be measured: ensure all IV fluids, oral intake, urine output, and other losses (diarrhea, drains) are recorded. For patients on fluid restriction (e.g., 2L/day), help them space fluids throughout the day (provide a fluid allowance plan, like a pitcher with their allotment or smaller cups). Monitor for signs of dehydration too, especially if on aggressive diuresis (dry mucous membranes, hypotension). In acute decomp HF, a Foley catheter may be placed for accurate output measurement and diuresis tracking. **Edema assessments** each shift (check pretibial edema, sacral in bedbound, etc.) can gauge if fluid is being mobilized. Also listen to lung sounds for improvement or worsening crackles. By maintaining euvolemia, we reduce cardiac workload and symptoms.

Vital Signs and Oxygenation: Frequent **vital signs** monitoring is needed. Watch for patterns: **tachycardia** could indicate compensation for low output or a response to hypoxia/pain; new **atrial fibrillation with RVR** may severely worsen HF (report new irregular rhythm). **Blood pressure** trends are important – many HF meds lower BP, but we want to maintain perfusion. If SBP is consistently <90 and patient is dizzy, notify provider – medication doses might need adjusting. Conversely, if hypertension is not controlled, it can strain the heart. **Orthostatic BP** checks may be ordered if patient is on diuretics or vasodilators; assist patient slowly from lying to sitting to standing, and see if there's a significant drop or symptoms. **Pulse oximetry**: HF patients with pulmonary edema may need supplemental O_2. Keep O_2 saturation per order (often >92%). Administer oxygen if sat is low or patient is dyspneic – common delivery is nasal cannula 2-4 L/min, or high-flow/BiPAP in acute pulmonary edema. Nurses should titrate oxygen as needed and monitor for improvement in breathlessness. Positioning is a simple but effective intervention: **High Fowler's position** (elevating head of bed) can ease breathing in pulmonary edema by decreasing venous return and improving lung expansion (Heart Failure (1).pdf). Many

patients will self-adjust to sleeping propped up on pillows (ask how many pillows they use at home or if they sleep in a recliner – a gauge of orthopnea severity). Cluster care to allow rest periods, as these patients fatigue easily. If the patient has an order for continuous telemetric monitoring, always respond promptly to alarms – VTach or extreme brady can be lethal.

Medication Management & Monitoring: Nursing's role is critical in ensuring HF meds are given safely. Always check parameters – e.g., apical pulse before beta-blockers or digoxin (hold if too low, as specified), BP before ACEI/ARNI (hold or discuss if SBP <90 and symptomatic). **Assess for side effects/toxicity** each day: ask if any cough (ACEI), dizziness/lightheadedness, or visual disturbances (digoxin). Monitor lab results daily: for example, if patient is on IV furosemide, look at that morning's BMP for K^+ and Cr. Replace potassium per protocol if needed. If patient is on warfarin due to low EF with clot or afib, check INR results – coordinate dose changes with pharmacy and teach patient about maintaining stable vitamin K intake. **Trough levels**: If the patient is on digoxin, a level might be drawn – be aware of when and hold dose if necessary for accurate level draw. For new start Entresto, ensure no ACEI in past 36 hrs (to avoid angioedema). A lot of patient safety rests on nursing double-checks, especially since these patients are often on many medications. Encourage patients to voice any new symptoms after meds – e.g., "I felt dizzy after my lisinopril" – and communicate these to the team. Also, reconcile their home meds with current regimen; sometimes a home medication (like NSAIDs) may worsen HF – ensure patients know to avoid NSAIDs which cause sodium retention and can counteract diuretics. Educate about each medication purpose in lay terms ("this pill helps your heart pump easier by relaxing your blood vessels") to improve adherence (Medications Used to Treat Heart Failure | American Heart Association).

Diet and Nutrition Education: Low-sodium diet is a cornerstone of HF self-management (Heart Failure (1).pdf). Typical restriction is ~2 grams of sodium per day. Teach patients which foods are high in salt – e.g., processed foods, canned soups, deli meats, fast food, pickles, etc. Work with dietary to provide low-salt options and show patients how meals can still be tasty with herbs instead of salt. If fluid restriction is ordered (often 1.5–2 L/day in advanced HF or hyponatremia), help the patient plan intake: small cups, ice chips for thirst, sugar-free hard candies to moisten mouth, and spacing drinks. Daily weights as mentioned integrate with diet – show how eating salty foods leads to weight bump from water retention. If patient has hyperlipidemia or diabetes, coordinate diabetic or heart-healthy diet teaching – these comorbid conditions worsen HF if uncontrolled. Malnutrition is a risk in severe HF (due to gut edema, poor appetite); involve a nutritionist for high-calorie low-sodium choices if needed.

Promoting Activity and Rest: HF patients should balance **activity with rest**. Encourage any **exercise** as tolerated – even hallway walks – because it improves conditioning. Plan nursing tasks around patient's energy levels: e.g., perform a bed bath after a rest period, not immediately following ambulation. **Cluster care** so they aren't constantly disturbed (promote undisturbed sleep at night as much as possible, since sleep improves cardiac function and compliance with regimen). However, avoid completely bed-bound status unless required – immobility leads to deconditioning and risk of clots. Even sitting up for meals helps. Many HF patients have some degree of anemia and muscle wasting, contributing to fatigue – physical

therapy consult can teach energy conservation techniques and simple strengthening exercises. Check oxygen needs with activity (some patients use O_2 only with exertion). For those in cardiac rehab programs, encourage participation as it's proven to enhance functional capacity.

Preventing and Responding to Acute Decompensation: Nurses often are first to notice if a patient is slipping into acute decompensated HF. Watch for increasing oxygen requirements, new inspiratory crackles or wheezes, new S3 gallop, tachycardia, or a big overnight weight jump. Also subjective reports: "I feel more short of breath today" or "I had to use an extra pillow last night" are red flags. If you suspect fluid overload, double-check diuretic orders – sometimes a dose increase or an extra dose (per sliding scale protocol or PRN) may be indicated (Heart Failure (1).pdf). Communicate concerns early to the provider; early intervention with IV diuresis can prevent ICU transfer. If a patient does develop acute pulmonary edema (extreme SOB, low O_2 sat, frothy sputum), be prepared to position them upright, apply high-flow O_2 or assist with CPAP/BiPAP, possibly administer IV diuretics and vasodilators stat per orders, and call rapid response if severe. Knowing the hospital's HF emergency protocols (like an acute pulmonary edema standing order set) empowers you to act quickly. Often, **BiPAP** can stave off intubation by reducing pulmonary edema (by increasing intrathoracic pressure, it decreases venous return and improves oxygenation). Stay calm and coach patient through breathing if they're anxious – anxiety worsens breathing, so a reassuring nurse at the bedside is vital.

Fall Prevention and Orthostatic Hypotension: Many HF patients, especially elderly ones, are at high risk of falls due to weakness, diuretics causing urgency, and medications causing dizziness (Heart Failure (1).pdf). Implement fall precautions: assist with ambulation, especially at night to the bathroom (diuretics may have them up frequently to urinate). Have a **bedside commode** if appropriate instead of rushing to bathroom. Educate patient to **rise slowly** from bed or chair – sit at edge of bed for a minute before standing. If orthostatic, consider a wheelchair for longer distances until they're steadier. Keep call light in reach and encourage them to call for help. Ensure adequate lighting. Review meds – are they on any sedatives or additional BP meds that could be minimized? After vasodilator or diuretic administration, anticipate a drop in pressure – maybe wait 30 minutes before you take them on a walk. Involve physical therapy if gait is unsteady; a walker might be needed temporarily. Document and communicate any near-fall or dizziness episodes so the team can adjust the care plan. Preventing falls is paramount for safety – a fall with injury could be catastrophic in an anticoagulated HF patient.

Psychosocial and Emotional Support: Living with CHF is stressful – patients often face anxiety about breathlessness or even fear of death, and depression is prevalent. Provide **emotional support** and encouragement. Be a good listener; allow them to express frustrations or fears. Involve family or caregivers in teaching so they feel supported at home. Consider a referral to a **heart failure support group** or have educational materials available – knowing they're not alone helps. If a patient is dealing with end-stage disease, facilitate discussions on advanced directives and wishes (in collaboration with care team). **Palliative care consults** can be helpful not only for end-of-life planning but also for symptom control (e.g., refractory dyspnea management, coping strategies). Social workers can assist with resources, whether it's financial

(medications can be expensive) or arranging home health nursing for follow-up. Spiritual care (chaplain) may provide solace if appropriate.

Interdisciplinary Care: CHF management truly requires a team (Heart Failure (1).pdf). Nurses coordinate with:

- **Cardiologists/HF specialists:** for medical management and advanced therapies.
- **Dietitians:** for tailoring the low-sodium diet and fluid plans (Heart Failure (1).pdf).
- **Pharmacists:** they often educate on meds, ensure optimal dosing, and help with med rec and adherence packaging.
- **Physical and Occupational Therapists:** to improve exercise tolerance and adapt daily activities to energy levels.
- **Case Management:** to arrange heart failure clinic follow-ups, cardiac rehab, or home health monitoring (some programs use telehealth scales and nurse calls).
- **Home Health Nursing:** can check on patient status, draw labs like BNP, reinforce education at home after discharge.
- **Family and Caregivers:** they should be seen as part of the team – educate them on how to support the patient (like helping with weighing, diet, noticing symptom changes). By coordinating this **interprofessional approach**, nurses help ensure continuity of care and support for the patient beyond the hospital (Heart Failure (1).pdf).

Patient Education – The Cornerstone: Every interaction is an opportunity to teach or reinforce. Key points every HF patient should understand (ensure to document that you taught and patient can repeat back):

- **Medication adherence:** Take all meds as prescribed even if feeling well (Heart Failure (1).pdf). Explain each drug's purpose simply, and set up a system (pill box, alarm reminders). Emphasize not skipping diuretics due to inconvenience – instead plan them (e.g., take furosemide in morning so you're not up all night).
- **Symptom monitoring:** Teach them the signs of worsening HF: rapid weight gain, increase in edema, more SOB especially lying flat, needing more pillows, reduced exercise tolerance, frequent dry hacking cough, or needing to stop more often when walking. Also symptoms like confusion or dizziness might mean low output or low BP. They should report these early to their provider; early tweaks in diuretics or meds can prevent a crisis (Heart failure - Symptoms and causes - Mayo Clinic) (Heart failure - Symptoms and causes - Mayo Clinic).
- **Daily weights:** as discussed, how to do it accurately and when to call for changes.
- **Diet and fluids:** Review their sodium restriction and tips to manage thirst on fluid limits if any. Provide written dietary guidelines or involve dietitian for a session. If diabetic, coordinate that diet. Moderate alcohol intake only (if alcohol caused cardiomyopathy, absolutely abstain). If overweight, discuss gradual weight loss to reduce strain on heart.
- **Activity:** Encourage staying active within limits. For some, a formal **cardiac rehab** program is appropriate (especially if HF is post-MI or they have insurance that covers it). For others, walking 15-20 minutes a day or as tolerated. Teach energy conservation: sit

while cooking or showering if needed, spread out chores, etc. Make sure they know to avoid exertion in extreme temperatures, and to rest if symptomatic.

- **Follow-up appointments:** They should see their cardiologist or HF clinic frequently (often 7-10 days post discharge, then every few weeks until stable). Emphasize keeping these appointments and doing labs like INR if on warfarin or kidney function if on ACEI/diuretics. Many readmissions happen from lack of follow-up.
- **When to seek emergency care:** Chest pain unrelieved by nitro (if they have ischemic disease), severe SOB at rest unrelieved by meds, fainting, or confusion – don't wait, call 911.
- **Self-care confidence:** Some patients may feel overwhelmed, so break it down and use teach-back. Provide **written instructions** (many hospitals have an HF booklet or AHA printouts). The AHA's motto for HF patients is "**Know Your Numbers**" – weight, BP, HR, symptoms. Encourage a diary or log.

Finally, celebrate small victories with the patient – like when they lose weight and edema improves, or when they can walk further. Positive reinforcement boosts their confidence in managing HF. Remember, engaged and educated patients are less likely to be readmitted and more likely to have a better quality of life (Heart failure - Symptoms and causes - Mayo Clinic) (Heart failure - Symptoms and causes - Mayo Clinic).

References: This guide incorporates evidence-based information from the American Heart Association and other trusted clinical sources. Key guidelines and findings were drawn from the 2022 AHA/ACC Heart Failure Management Guidelines (emphasizing multi-drug therapy and patient-centric care) (The four pillars of HFrEF therapy: is it time to treat heart failure ...) (Heart failure drug treatment: the fantastic four - PMC), as well as summaries from the ACCF/AHA Heart Failure Guidelines update (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP) (ACCF and AHA Release Guidelines on the Management of Heart Failure | AAFP). Medication mechanisms and considerations are supported by the AHA Heart Failure Medication compendium (Medications Used to Treat Heart Failure | American Heart Association) (Medications Used to Treat Heart Failure | American Heart Association). Diagnostic and intervention details reference standard cardiology texts and clinical resources, including NIH and NHLBI publications on heart failure diagnosis (Heart Failure - Diagnosis | NHLBI, NIH) (Heart Failure - Diagnosis | NHLBI, NIH) and reputable medical manuals. The importance of daily weight monitoring, diet, and patient education is reinforced by AHA patient education materials (Heart Failure (1).pdf) (Heart Failure (1).pdf) and expert consensus. By adhering to these guidelines and best practices, nurses can provide high-quality, lifesaving care to patients with CHF and related cardiac conditions.