

CHF

Definition

- A condition in which the heart is not able to pump enough blood to meet the body's metabolic needs or does so in the face of elevated filling pressure due to abnormalities of ejection or abnormalities of filling.

Classification

- In patients presenting with new (or possibly unexplained worsening symptoms of heart failure), document systolic function with echocardiography (HFpEF vs HFrEF), and then exclude ischemic underlying etiology (MI or CAD) with angiography (Ischemic Dilated Cardiomyopathy vs Non-ischemic Dilated Cardiomyopathy).
- Classification of heart failure is important not only for more effective communication but for optimization of medical management

Overview of Heart Failure with Reduced Systolic Function

- Ischemic Etiologies: Coronary Artery Disease (major) and congenital coronary anomalies; myocardial bridging
- Non-Ischemic Etiologies: HTN, valvular heart disease, idiopathic, tachycardia mediated, toxins (EtOH, cocaine, meth, anthracyclines, etc), end-stage infiltrative disease (amyloid, hemochromatosis, sarcoid), endocrine (hypo/hyperthyroidism), myocarditis (viral, Chagas, HIV), radiation, peripartum, vitamin deficiencies (thiamine), genetic/familial,
- Etiologies of Exacerbations: poor dietary and/or medication adherence, myocardial ischemia, inadequate drug therapy, HTN, arrhythmia (e.g., atrial fibrillation), valvular heart disease, fluid overload, PE, infection, thyrotoxicosis, physical or emotional stress.

Evaluation of Acute Heart Failure

- Retrieve old ECHO, stress tests, ECGs, and cath reports.
- Take a thorough history (dyspnea, orthopnea, PND, edema, weight gain, abdominal discomfort, nausea, anorexia, baseline weight).
- Physical examination and clinical assessment:
- Is the patient warm or cold (reduced perfusion)? Look for fatigue, altered mental status, Cheyne-Stokes respiration, pallor, cool extremities, narrow pulse pressure, lactic acidosis, hyponatremia, renal failure.

- Is the patient dry or wet (congested)? (See chart below.)
- Left Sided Congestion (crackles, pulmonary edema, left ventricular S3, elevated JVP) +/- Right-sided congestion: elevated JVP, lower extremity edema, hepato-jugular reflex, pulsatile liver, right ventricular S3
- Labs: CBC, chem-10, coagulation studies, troponin, TSH, ECG (e/o ischemia, hypertrophy, pericarditis, new bundle branch block), CXR (vascular prominence, effusion, cardiomegaly).
- Diagnostic utility of B-type natriuretic peptide (BNP):

1. *BNP is released from the heart (mainly the LV) in response to increased wall tension.*
2. *BNP measurement has limited utility after a good clinical assessment. If the diagnosis of HF is clinically apparent, a BNP test is not indicated. Measurement of BNP levels should not be used as a screening test. If the clinical picture is consistent with HF, do not let a low or normal BNP level necessarily deter appropriate diagnosis and therapy.*
3. *In one study, BNP < 100 pg/mL was helpful in ruling out cardiac causes of dyspnea (<50 pg/mL had 96% negative predictive value for CHF) while BNP > 400 pg/mL is helpful for ruling in cardiac causes of dyspnea. For values <100 pg/mL and >400 pg/mL, consider other causes of dyspnea as well as CHF.*
4. *The level of BNP correlates with the severity of HF. However, patients who are stable and well-compensated may run high BNP levels chronically.*
5. *Obese patients have decreased BNP levels due to adipose clearance.*
6. *Higher baseline levels of BNP are seen in older patients and women. BNP is falsely low in obesity, diuretics, ACEi, beta-blockers, and aldosterone antagonist use. BNP is low in constrictive pericarditis*

Acute Management of Heart Failure Exacerbation.

Therapy should be tailored to the hemodynamic profile (Volume status vs Perfusion) as described below:

1. Volume Overload (Wet) with Good Perfusion (Warm) -> Most common hospital presentation of CHF. IV diuretics ± nitrates, afterload reduction (ACEI/ARB, hydralazine, nitrates), ± aldosterone antagonists and beta-blockers when optimized.
2. Volume Overload (Wet) with Poor Perfusion (Cold) -> Likely requires ICU care. IV diuretics, ± afterload reduction, ± inotropes/inodilators. In severe or refractory cases, sometimes PA catheter-guided therapy* is needed, PUF/CVVH, consideration of advanced therapies (LVAD, transplant evaluation).
3. Non-volume Overload (Dry) with Poor Perfusion (Cold) -> Represents 10% of cases, challenging to treat. Often associated with cardio-renal syndrome. Inotropes/inodilators, afterload reduction, and advanced therapies can be considered.
4. Non-volume Overload (Dry) with Good Perfusion (Warm) -> Compensated HF. Usually can be treated as an outpatient. Maintain volume status and prevent disease progression with rx. Chronic management with beta-blockers, ACEI/ARB, aldosterone antagonists, loop diuretics.

In general patients should be closely monitored:

- Telemetry monitoring (high risk for ventricular arrhythmia)
- Oxygen via NC or face-mask, goal >92% saturation. If needed, CPAP or BiPAP confers mortality benefit and reduces the need for mechanical ventilation.
- Monitor K, Mg, Cr (remember Cr may be elevated on admission secondary to venous congestion at the glomerulus; if HF-related, Cr should fall with diuresis). Do not hold diuretics for BP.
- Restrict sodium (<2g) and also fluids (<1500cc) if hyponatremic; daily weights, strict I&Os.

Overview and Specifics of Treatment

1. Immediate Considerations: Diuresis, Inotropes, Afterload reduction
2. Guideline Directed Therapy: ACE-i, Beta blockers, Aldosterone antagonists, Hydralazine/Nitrates
3. Devices: ICD, CRT, CRT-D
4. Advanced Therapies: Mechanical circulatory support, Transplant

Diuresis:

Loop diuretics:

- For Veno-dilation and natriuresis. With advanced right heart failure and diuretic resistance, use IV as oral absorption is diminished by bowel edema. Decongestion of hepatic and renal vascular beds will improve organ function. Reduction in LV wall stress and mitral regurgitation will improve cardiac output and BP.
- Initial dosing: 2.5 times normal oral diuretic dose (DOSE trial). If inadequate response (< 100 – 200mL UOP in first 30 mins), double dose and closely monitor UOP
- Consider furosemide/bumex gtt in patients with severe volume overload and/or sub-optimal response to bolus dosing of diuretics.
- Furosemide gtt: Give initial 20-80 mg IV bolus (higher doses for patients with worse renal function; lower doses for diuretic naïve patients). Then start gtt at 5 mg/hr (range 5-20 mg/hr). FENa can be monitored to determine effectiveness of natriuresis (maximum FENa = 20%).
- Bumex gtt: Give initial 1-2 mg bolus. Then start bumex gtt at 0.5 mg/hr (range 0.2-2 mg/hr).
- If inadequate diuresis with loop diuretic alone, try adding a thiazide diuretic such as chlorothiazide IV/PO 125-500 mg or metolazone 2.5-10 mg PO, diuril IV dosed 30 min before the loop diuretic. Metolazone PO should precede IV loop diuretic by at least 2-4 hours. Monitor serum electrolytes (Na, K⁺, and Mg²⁺) closely, replete as necessary.
- Markers of adequate diuresis include resolution of dyspnea, decreased JVP, Cr normalization, return to baseline weight. JVP should be closely monitored to avoid 'bumping patient's creatinine.'
- Transition to PO diuretics and monitor response to PO prior to discharge

Ultrafiltration:

Fluid removal with no effect on serum electrolytes, consideration reserved for in cases of acute decompensated heart failure inadequately responsive to aggressive diuretic regimen. Increased risk of serious adverse events and no difference in weight loss at 96 hours when compared to pharmacologic therapy (CARRESS-HF).

Optimizing Hemodynamics/Inotropes:

Dobutamine:

- Beta-adrenergic agonist (predominantly beta-1), inotrope, mild arterial vasodilator. Generally the inotrope of choice in cardiogenic shock.
- Minimal effects on left sided filling pressures. At higher doses (>10 - 15 mcg/kg/min) can exert significant alpha-adrenergic activity increasing SVR and worsening forward flow. Effectiveness curtailed with concomitant beta blockers.
- Adverse effects: increased myocardial oxygen demand, hypotension, ventricular arrhythmias
- Tolerance can develop with beta receptor down-regulation.
- Normal dose range: 2.0 - 5.0 mcg/kg/min, can be run peripherally
- Titrate to UOP, CO, SvO₂ if a right heart catheter is in place.

Dopamine:

- Low dose acts through dopamine receptors increasing renal blood flow and natriuresis (1 - 3 µg/kg/min). Intermediate doses act through beta-adrenergic receptors to increase HR and contractility (2 - 10 µg/kg/min). High doses stimulate alpha-adrenergic receptors increasing afterload, this may be detrimental in HF (>10 µg/kg/min).
- Use only to stabilize hypotensive patients.
- Can run peripherally for <24 hours as long as a viable PIV has been established

Milrinone:

- Phosphodiesterase-3 inhibitor, inodilator
- Hemodynamic profile: Reduces right- and left-sided filling pressures, reduces pulmonary and systemic vascular resistance, direct positive inotropic effect. Longer half-life than dobutamine, more effective in beta-blocked patients and suspected dobutamine unresponsiveness due to beta receptor down-regulation.
- Adverse effects: Toxicity profile similar to dobutamine, although slightly less tachycardia and myocardial oxygen demand.
- Normal dose range: 0.375 - 0.75 mcg/kg/min; starting dose in renal dysfunction is 0.25 mcg/kg/min.
- Do not give a bolus prior to starting infusion even though doing so is suggested in the package insert. Bolus dosing is associated with more hypotension and is rarely indicated.

Digoxin:

- Na-K-ATPase inhibition improves contractility, anti-adrenergic effect also beneficial. The only safe PO inotrope. Symptomatic improvement and decrease in hospitalizations, but no mortality benefit in HF (DIG trial).
- Normal dose range: 0.125-0.25 mg daily, loading dose not indicated in HF.
- Optimal serum concentrations: 0.5-0.8 ng/mL in men, 0.5-0.9 ng/mL in women
- Adverse effects: confusion, visual disturbances, arrhythmias (conduction disease, pAT)

Nitrates:

- Preferential venodilator useful in acute setting to reduce pulmonary edema and other congestive symptoms. Reduces LV filling pressure and SVR.
- Sublingual, nitropaste, or IV nitroglycerin (start at 10-20 mcg/min and titrate as permitted by BP).
- When stable, convert to PO nitrate (e.g. start with isosorbide dinitrate 10 mg PO TID).
- Avoid with concurrent PDE-5 inhibitor, NO reductase inhibitor, severe aortic stenosis, cyanide toxicity.
- Adverse effects: headache (common), hypotension, tachyphylaxis

Guideline-Directed Therapy

ACE inhibitors:

- For afterload reduction and neuro-hormonal effects. Have a proven mortality benefit (CONSENSUS trial), prevent re-hospitalization, stop adverse LV remodeling in chronic HF (SOLVD trial). This effect is independent of the anti-hypertensive effect.
- If ACE inhibitor causes cough, try ARB (angiotensin II receptor blocker). Always attempt re-challenge with ACE inhibitor in future as ACE confers improved mortality benefit over ARB.
- Dosing: Captopril 3.125 – 6.25 mg PO q8hr and increase dose by dose as BP permits.
- Monitor K⁺, and Cr (up to 30% increase in Cr is acceptable).
- Once stable, switch to equivalent dose of once-daily ACE inhibitor.
- Target dosing: Lisinopril 40mg PO Qday.

Beta-blockers:

- For hemodynamic benefits, beneficial LV remodeling, decreasing frequency of PVCs. Multiple trials showing mortality benefit, reduced hospitalizations, symptom mitigation (MERIT-HF, PRECISE, CIBIS-I & II, etc.).
- Metoprolol ER, carvedilol, and bisoprolol have symptom and mortality benefit and slow disease progression for NYHA class II-IV chronic HF.
- Do not initiate in acute setting (OPTIMIZE-HF trial). Once patient is adequately diuresed, initiate beta blocker with appropriate up-titration on outpatient basis.
- Do not withdraw in exacerbation unless patient is hypotensive or in cardiogenic shock.
- Initial dosing: Start with metoprolol ER 12.5-25 mg PO daily or carvedilol 3.125 mg PO BID and titrate up as permitted by HR and BP.

- Target dosing: Metoprolol ER 200 mg daily or carvedilol 25 mg BID (50 mg BID if > 85 kg). COMET trial showed greater benefit with carvedilol vs. short-acting metoprolol. Also recent work showed that HR may be more important than final dose.

Aldosterone Antagonists:

- Survival benefit derived from potassium sparing action lowering risk of hypokalemia-associated arrhythmia as well as blocking of mineralocorticoid activity.
- Indicated for the following populations:
 - NYHA class II HF + LVEF \leq 30% (EMPHASIS-HF trial)
 - NYHA class III-IV HF + LVEF < 35% (RALES trial)
 - recent MI + LVEF \leq 40% + symptomatic HF or DM (EPHESUS trial)
- Contraindications: Cr > 2.5 mg/dL in men, Cr > 2.0 mg/dL in women, K⁺ > 5 meq/L.
- Initial dosing: Spironolactone 25 mg daily (every other day dosing for eGFR < 50 mL/min), titrate up to 50 mg daily if K⁺ < 5 meq/L at 4 weeks.
- Target dosing: Spironolactone 25-50 mg daily
- Can consider changing to eplerenone (much more expensive) if undesired endocrine side effects (e.g. gynecomastia) with spironolactone.
- Monitor K⁺.

Hydralazine + Nitrates:

- Hydralazine is a preferential arterial vasodilator.
- Indicated for black patients with NYHA class III-IV HF and LVEF < 40% on maximal medical therapy.
- Initial dosing: Hydralazine 25 mg TID + isosorbide dinitrate 20 mg TID or combination isosorbide dinitrate-hydralazine 37.5 mg-20 mg TID. Titrate up every 2-4 weeks as permitted by BP.
- Target dosing: Hydralazine 75 mg TID + isosorbide dinitrate 40 mg TID.
- Can consider isosorbide mononitrate (30-120 mg daily) as alternative to TID-dosed isosorbide dinitrate.

ICD and CRT

Indicated only after medical optimization and removal of applicable vices (no active drug use) – please see applicable section.

Implantable cardiac defibrillator (ICD): Primary prevention of sudden cardiac death in populations at increased risk for life-threatening ventricular arrhythmias. Mortality benefit for NYHA class II-III HF + LVEF \leq 35% despite optimal medical management for at least 3 months (SCD-HeFT trial).

Cardiac resynchronization therapy (CRT): HF frequently leads to intraventricular conduction delay and ventricular dyssynchrony. Resynchronization (biventricular pacing) improves pump function with mortality benefit, symptom improvement, and decreased hospitalizations for NYHA class II-III HF + LVEF \leq 35% + LBBB + QRS \geq 150 ms despite optimal medical management (MIRACLE, CARE-HF, COMPANION, MADIT-CRT trials).

Most patients who meet criteria for ICD also meet criteria for CRT and vice versa. Combination devices (CRT-D) confer superior mortality benefit when compared to ICD or CRT alone (REVERSE, MADIT-CRT, COMPANION trials).

Advanced Therapies

Mechanical circulatory support

1. Intra-aortic balloon pump (IABP): Temporary treatment for HF refractory to medical management, systolic unloading and improved coronary perfusion, particularly useful in patients with ischemia or mitral regurgitation, placed in cath lab (see corresponding section).
2. Left ventricular assist device (LVAD): Can be used as a bridge to recovery, a bridge to transplant, or “destination” therapy for outpatient use. Placed in OR.

Cardiac transplantation: Consider early involvement of transplant/advanced HF team in patients with new, rapidly progressive, severe HF, or advanced HF refractory to treatment.

Overview and Management of Heart Failure with Preserved Systolic Function

Etiologies

- Etiologies include: ischemic heart disease (most common), hypertensive heart disease, diabetic cardiomyopathy, high output cardiac failure (e.g., severe anemia, AV fistulae, thyrotoxicosis), restrictive cardiomyopathy (radiation injury, infiltrative disease [e.g., amyloidosis], metabolic storage disease [e.g., Fabry’s disease], endocardial fibrosis), and hypertrophic cardiomyopathy, valvular heart disease (e.g., AS, MR). Exclude HF due to constrictive pericarditis or due to isolated right heart failure (pulmonary HTN, cor pulmonale)
- Etiologies of exacerbation: Anything that increases LV wall stiffness will predispose to exacerbation of diastolic HF. Common precipitants include ischemia, tachyarrhythmia, HTN.

Evaluations

- CBC (anemia), chem-10 (CKD), LFTs (cirrhosis), TFTs (thyrotoxicosis), lipid panel, A1c, urine microalbumin, SPEP/UPEP as indicated
- Diastolic dysfunction can be assessed non-invasively by echo (LVH, impaired filling, valve disease, atrial enlargement, rule out constrictive pericarditis) or invasively by cardiac catheterization.
- If echo is unrevealing, consider cardiac catheterization to evaluate for occult arterial disease and, occasionally, endomyocardial biopsy.
- On exam, look for HTN, S4, signs of LVH.

Treatment

- Similar to treatment for systolic HF as outlined above. Address underlying etiology (ischemia, HTN, infiltrative process, etc)
- Evidence is weak for guiding treatment with commonly used drugs for systolic failure; many studies show marginal or no benefit, nevertheless optimization is important.
- Treat congestion (diuretics, ultrafiltration/dialysis, salt restriction)

Discharge Planning

- All HF patients should receive the pneumococcal and influenza vaccines.
- Advance directive education, palliative care involvement (depending on severity and co-morbidities), goals of care discussions for patients with recurrent hospitalizations.
- Early outpatient follow-up and/or telephone nurse follow-up after discharge help reduce readmissions.
- Heart failure education, including dietary modification (fluid and salt restriction), scale and instruction regarding daily weights, diuretic self-titration teaching, avoidance of cardio-toxic recreational drugs, explain rationale of all medications.

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