ACUTE HEART FAILURE

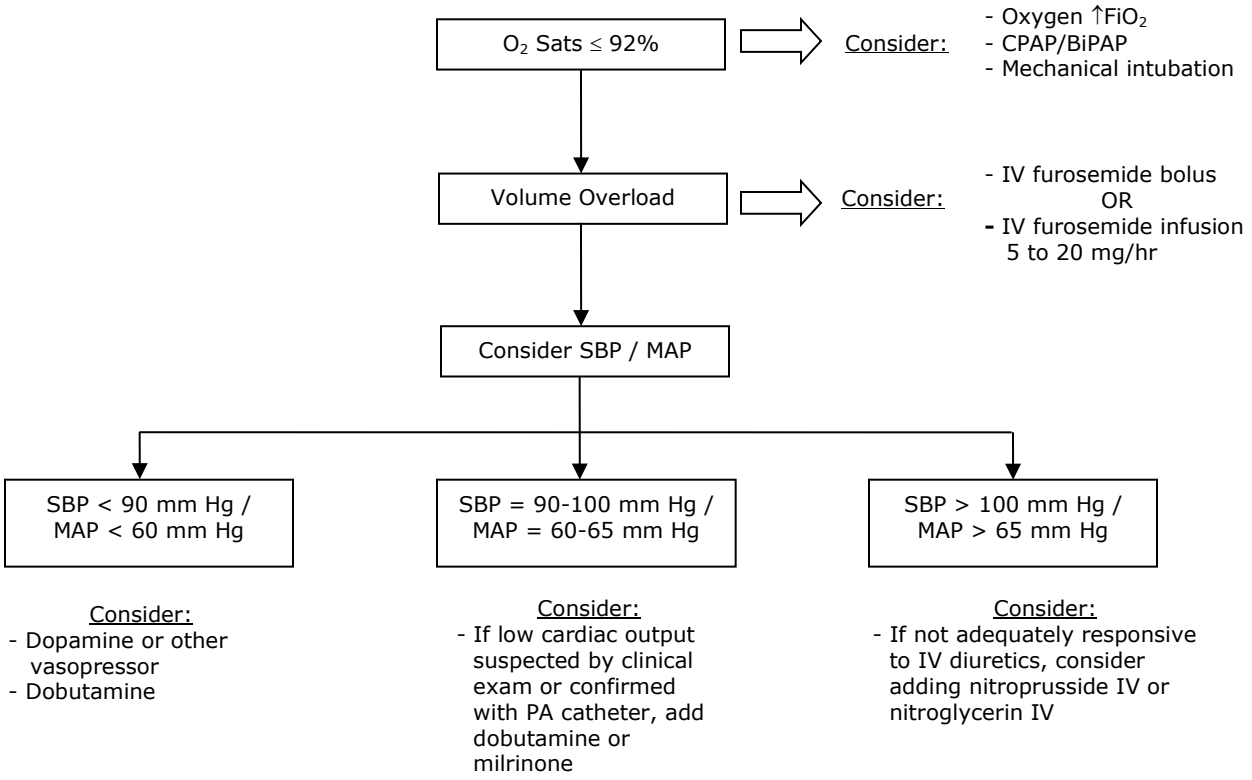
- Refers to new or worsening signs and symptoms of heart failure (HF) that are usually caused by volume overload and/or hypoperfusion
- A relatively equal percentage of patients with acutely decompensated HF have impaired versus preserved LV systolic function
- Precipitating cause(s) for acute HF should be sought for appropriate treatment and to prevent future acute events
- The majority of patients do present with features of congestion/fluid overload on clinical assessment
- Acute HF requiring hospitalization may be a marker of deterioration and worsening prognosis

KEY POINTS

- Oral chronic HF therapy should generally be continued **in the absence of** hemodynamic instability or contraindications
- Patients admitted with significant worsening of renal function should be considered for a reduction in, or temporary discontinuation of ACE inhibitors, ARBs, and/or mineralocorticoid receptor antagonists until renal function improves
- Continuation of beta-blocker upon admission for acute HF is safe, unless the patient is symptomatic from hypotension or bradycardia
- Routine use of vasodilators and positive inotropes have not been shown to improve survival in hemodynamically stable patients
- Ultrafiltration may be of benefit in relieving congestion particularly in diuretic-resistant patients, but a recent study suggests it may be no more effective than pharmacologic therapy in most patients
- Vasopressin receptor antagonists (e.g. tolvaptan) can rapidly and effectively reduce body weight and restore serum sodium in hyponatremic patients with congestion but their use has not been associated with mortality benefits
- Monitor fluid intake and output, vital signs, body weight, clinical signs and symptoms of systemic perfusion and congestion, and daily serum electrolytes and creatinine

ACUTE HEART FAILURE

Figure 1. Algorithm for Treatment of Acute Heart Failure

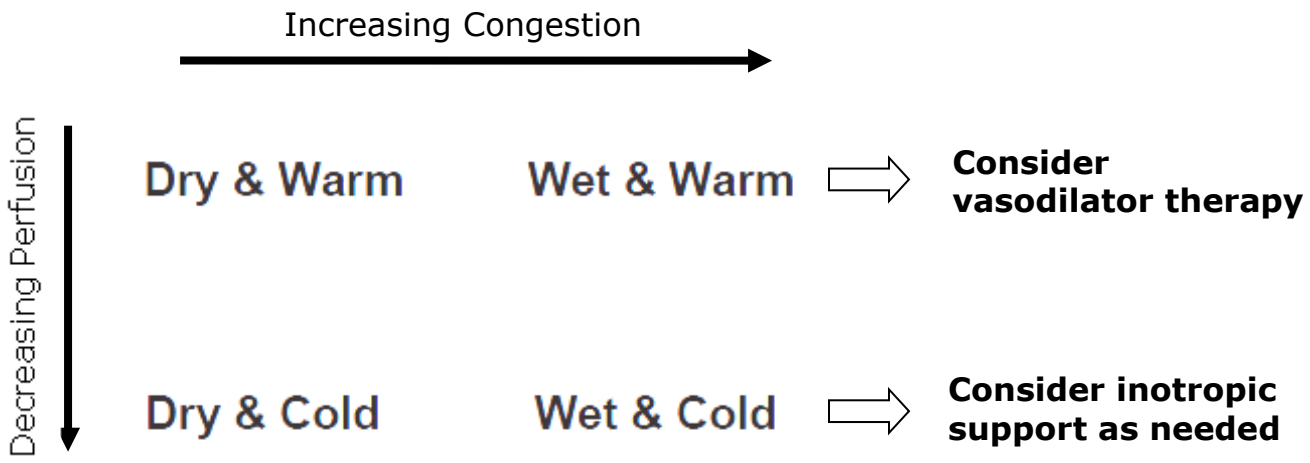


ACUTE HEART FAILURE

COMMON FACTORS THAT PRECIPITATE ACUTE DECOMPENSATED HEART FAILURE

- Nonadherence with medication regimen
- Nonadherence with diet (e.g. sodium intake and/or fluid restriction)
- Acute myocardial ischemia
- Uncorrected high blood pressure
- New onset and/or deterioration in atrial fibrillation and other arrhythmias
- Recent addition of negative inotropic drugs (e.g. verapamil, diltiazem, nifedipine, beta-blockers)
- Pulmonary embolus
- Initiation of drugs that increase salt retention (e.g. steroids, thiazolidinediones, NSAIDs)
- Excessive alcohol or illicit drug use (e.g. cocaine, amphetamines)
- Endocrine abnormalities (e.g. diabetes mellitus, hyperthyroidism, hypothyroidism)
- Concurrent infections (e.g. pneumonia, viral illnesses)
- Additional acute cardiovascular disorders (e.g. endocarditis, myopericarditis, aortic dissection)

Figure 2. Classification of Patients Presenting with Acute Decompensated Heart Failure



The clinical goals of pharmacologic therapy include:

- Relieve congestive symptoms or optimize volume status
- Treat symptoms of low cardiac output

PHARMACOLOGIC THERAPY

To relieve congestion:

- Mild volume overload – IV furosemide boluses
- Moderate to severe volume overload
  - Increase IV furosemide bolus dose/frequency
  - Consider furosemide continuous infusion if the nursing unit permits as per the UHN Restricted Nursing IV Drug List
  - Add metolazone/hydrochlorothiazide to furosemide

- Add IV vasodilators (nitroprusside or nitroglycerin)
- Ultrafiltration may be considered for persistent congestion despite optimized diuretic therapy

To increase cardiac output:

- If SBP < 90 mmHg or MAP < 60 mmHg, consider vasopressors and/or dobutamine
- If SBP = 90-100 mmHg or MAP = 60-65 mmHg, consider dobutamine or milrinone
- If SBP > 100 mmHg or MAP > 65 mmHg, consider IV vasodilators (nitroprusside or nitroglycerin)

- ACE inhibitors should not be initiated in the acute setting (e.g. first 8-12 hours) unless elevated BP is present – they should be initiated after the acute event (e.g. > 24 hours)
- Initiation of beta-blocker therapy is recommended after optimization of volume status and successful discontinuation of intravenous diuretics, vasodilators, and inotropic agents. Beta-blocker therapy should be initiated at a low dose and only in stable patients.

INVASIVE HEMODYNAMIC MONITORING

Routine use of invasive hemodynamic monitoring with a pulmonary artery (PA) catheter is not recommended in normotensive patients with acute HF and congestion with symptomatic response to diuretics and vasodilators.

PA catheter is, however, useful in the following situations:

- Presumed cardiogenic shock requiring escalating vasopressor therapy and consideration of mechanical circulatory support
- Severe clinical decompensation in which therapy is limited by uncertain contributions of elevated filling pressures, hypoperfusion, and vascular tone
- Apparent dependence on intravenous inotropic infusions after initial clinical improvement
- Persistent severe symptoms despite adjustment of recommended therapies
- To guide therapy in the presence of severe diffuse pulmonary disease

ACUTE HEART FAILURE

INOTROPES

Drug	Dosage	$t_{1/2}$	Receptor Affinity				Effects				Adverse Effects
			α_1	β_1	β_2	DA	CO	HR	SVR	PVR	
Dobutamine	<5 mcg/kg/min	2-3 min	+	++++	++	0	↑	↑	↓	↔	↑/↓ BP, headache, tachyarrhythmias, nausea, fever, hypersensitivity, cardiac ischemia
	5 to 20 mcg/kg/min						↑	↑	↔	↔	
Milrinone	Loading dose (optional): 25 to 50 mcg/kg bolus over 10 min 0.125 to 0.75 mcg/kg/min consider using lower doses in renal impairment	2.5 h	N/A (phosphodiesterase inhibitor)				↑	↑	↓	↓	tachyarrhythmias, ↓BP, cardiac ischemia
Dopamine	<3 mcg/kg/min	2-20 min	++++	++++	++	++	↑/↔	↔	↓	↔	tachyarrhythmias, headache, nausea, tissue necrosis, ↑BP, cardiac ischemia
	3 to 10 mcg/kg/min						↑	↑	↔	↔	
	10 to 20 mcg/kg/min						↑	↑	↑	↔	

DA = dopamine receptors

VASODILATORS

Drug	Dosage	$t_{1/2}$	Mechanism of Action	Clinical Response	Hemodynamic Effects			Adverse Effects
					CO	SVR	PCWP	
Nitroprusside	0.25 to 3 mcg/kg/min	2 min	Acts on vascular smooth muscle and increases synthesis of nitric oxide	Mixed arterial-venous vasodilation	↑	↓	↓	Hypotension, rebound phenomenon after abrupt withdrawal, cyanide and thiocyanate toxicity
Nitroglycerin	5 to 200 mcg/min	1-4 min	Activates guanylate cyclase to ↑ cGMP in vascular smooth muscle, by serving as nitric oxide donor	- Venodilation - Mild arterial vasodilation - Coronary vasodilation with beneficial effects on myocardial oxygen demand and supply	↑/↔	↓/↔	↓	Hypotension, tolerance, headache

ACUTE HEART FAILURE

DIURETIC DOSING FOR THE TREATMENT OF ACUTE HEART FAILURE

Based on The Diuretic Optimization Strategies Evaluation (DOSE) trial:

- There is no difference between furosemide continuous infusion and intermittent bolus dosing in either symptoms or renal function
- There was a trend towards greater symptom improvement with high compared with low dose diuretics without a significant difference in renal function
- In summary, there is no advantage in the routine use of continuous diuretic infusions and a higher dose of diuretics could be considered, with close observation of renal function and electrolytes

Creatinine clearance*	Patient	Initial IV dose**	Maintenance dose
≥60 mL/min/1.73m^2	New-onset HF or no maintenance diuretic therapy	Furosemide 20-40 mg 2-3 times daily	Lowest diuretic dose that allows for diuresis is the ideal dose
	Established HF or chronic oral diuretic therapy	Furosemide bolus same as oral dose 1:1***	
<60 mL/min/1.73m^2	New-onset HF or no maintenance diuretic therapy	Furosemide 20-80 mg 2-3 times daily	
	Established HF or chronic oral diuretic therapy	Furosemide bolus same as oral dose 1:1***	

*Creatinine clearance is calculated from the Cockroft-Gault or Modified Diet in Renal Disease formula
**Intravenous continuous furosemide at doses of 5 to 20mg/h is also an option.
***Example: if patient was on 40 mg orally, initial IV dose would be 40 mg IV

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Prepared by: Yvonne Kwan BScPhm, ACPR
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Reviewed by: Dr. Michael McDonald

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The Pharmacotherapy Handbook is intended to be used as a tool to aid in the appropriate prescribing and administration of cardiovascular formulary agents.

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