Acute Heart Failure: FAQs

The chart below addresses common clinical questions about pharmacotherapy of acute heart failure. See our toolbox, Improving Heart Failure Care, for strategies and resources for preventing heart failure readmissions. Screen for nonadherence and look for opportunities for patient education.20

**Abbreviations:** ACEI = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; eGFR = estimated glomerular filtration rate; LVAD = left ventricular assist device; MI = myocardial infarction; PCWP = pulmonary capillary wedge pressure; SBP = systolic blood pressure

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| What are the main presentations of acute heart failure?                            | • One way of thinking about acute heart failure is to classify the patient as “wet” or “dry” (presence or absence of congestion) and “warm” or “cold” (adequate perfusion or poor perfusion).2,3 Physical finding such as orthopnea, jugular venous distension, rales, S3 gallop, hepatomegaly, hepatojugular reflux, ascites, or edema (pulmonary, bilateral peripheral) suggest congestion.2,4 Indicators of poor perfusion include narrow pulse pressure, cool extremities, oliguria, dizziness, confusion, and hypotension.2,4  
  • Warm and dry = no congestion and adequate perfusion2  
  • Warm and wet = congestion with adequate perfusion.2 Common presentation.3 Patients usually have normal or elevated SBP.4  
  • Cold and dry = no congestion with poor perfusion2 (rare3)  
  • Cold and wet = congestion with poor perfusion2  
  Note that:  
  • Hypoperfusion is not the same as hypotension, but hypoperfused patients are often hypotensive.4  
  • Over 90% of patients are “wet.”3  
  • The “cold” presentation has a higher risk of mortality.3 |
| How is each presentation of acute heart failure generally treated, initially?        | **Warm and wet:**  
  • Diuretic.2,4 Consider adjuvant vasodilator.2,4  
  **Warm and dry** (compensated):4  
  • Adjust oral meds.4  
  **Dry and cold** (hypoperfused, hypovolemic):4  
  • Consider cautious fluid challenge (250 to 500 mL normal saline over 20 to 30 min), then inotrope if still hypoperfused.4,8  
  **Wet and cold:**  
  • SBP <90 mmHg: inotrope.4 Consider vasopressor (e.g., norepinephrine) if refractory.4 Add diuretic once perfusion is adequate.4  
  • SBP ≥90 mmHg: vasodilator and diuretic. Consider inotrope if refractory.4 |
### Clinical question  | Pertinent information
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How are diuretics used in acute heart failure? | **Volume overload ("wet" presentation): intravenous** loop diuretic.<sup>1,2</sup>
- If patient is on a loop diuretic at home, start with an intravenous dose the same or greater (1 to 2.5 x) than the total home oral dose as intermittent boluses every eight to 12 hours, or as a continuous infusion.<sup>1,2,20</sup> When converting, consider these furosemide equivalents:
  - Bumetanide 0.5 to 1 mg orally = furosemide 40 mg orally<sup>22</sup>
  - Torsemide 10 to 20 mg orally = furosemide 40 mg orally<sup>22</sup>
- Example dosing (if patient is not on a loop diuretic at home):<sup>1</sup>
  - CrCl ≥60 mL/min/1.73 m<sup>2</sup>, furosemide 20 to 40 mg two or three times daily, or 5 to 20 mg/hour infusion.<sup>1</sup>
  - CrCl <60 mL/min/1.73 m<sup>2</sup>, furosemide 20 to 80 mg two or three times daily, or 5 to 20 mg/hour infusion.<sup>1</sup>
  - Give a bolus dose before starting continuous infusion.<sup>22</sup>
- If diuresis is brisk but transient, increase the loop diuretic frequency to three or four times daily.<sup>20</sup>
- If response is inadequate (e.g., <500 mL in four to six hours), double the loop diuretic dose.<sup>22</sup> Repeat if needed to achieve adequate urine output, until maximum dose is reached.<sup>22</sup> Consider switch to continuous infusion after first 24 hours if poor response persists, and/or add a thiazide or acetazolamide.<sup>2,20,22</sup> Consider ultrafiltration.<sup>4</sup>
  - Consider a max total daily IV furosemide dose of 400 to 500 mg,<sup>20</sup> or bumetanide 10 to 15 mg.<sup>22</sup>
  - Metolazone 2.5 to 20 mg once daily.<sup>13</sup>
  - Acetazolamide 250 mg once or twice daily.<sup>13,20</sup>
  - Hydrochlorothiazide 25 to 100 mg once or twice daily.<sup>13</sup>
  - Not necessary to time thiazide so that it is given prior to loop.<sup>12</sup>
- When switching to oral diuretic:
  - consider that oral furosemide bioavailability is only about 50%.<sup>1</sup>
  - optimize guideline-directed medical therapy.<sup>20</sup> For a quick review, see our chart, Heart Failure Treatment at a Glance.<sup>1</sup>

How are vasodilators used in acute heart failure? | Vasodilators decrease preload and afterload. Thus, they may indirectly increase cardiac output.<sup>4</sup>
- Vasodilators reduce dyspnea, but do not reduce mortality or rehospitalization.<sup>1</sup>
- Vasodilators are first-line agents (with a diuretic) for the "warm and wet," and "cold and wet" presentation with SBP ≥90 mmHg.<sup>4</sup>
- Vasodilators should not be used if SBP <90 mmHg or if the patient has symptomatic hypotension.<sup>4</sup> They should be used with caution in patients with aortic or mitral stenosis.<sup>4</sup> Canadian guidelines recommend use only if SBP >100 mmHg.<sup>1</sup>
- Hypotension is associated with poor outcomes in heart failure, so vasodilators should be dosed conservatively.<sup>4</sup>
- **Vasodilator choices** include nitroglycerin and nitroprusside (nesiritide no longer marketed as of February 2018).<sup>1</sup> There
### Clinical question
Vasodilator use in acute heart failure, continued

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<td>is less evidence for nitroprusside than nitroglycerin in heart failure.¹</td>
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<td>• <strong>Nitroglycerin</strong>: primarily a venodilator (preload reducer).²,⁶ May increase cardiac output. Consider for patients with MI and pulmonary edema (due to preload reduction).⁶ May cause headache and tolerance.⁶</td>
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<td>• <strong>Nitroprusside</strong>: decreases preload and afterload. Consider for patients with hypertension or severe mitral regurgitation. Adverse effects include hypotension, reflex tachycardia, coronary steal, cyanide toxicity, rebound in heart failure if tapered too quickly.⁶</td>
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<td>• <strong>Nesiritide (no longer marketed)</strong>: was considered for patients with PCWP &gt; 18 mmHg after diuretics and nitroglycerin.⁶</td>
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### How are vasopressors used in acute heart failure?

| Vasopressors are used for cardiogenic shock.⁷ See our chart, *Vasopressors for Shock*, for more information. |
| Vasopressors are also used for “wet and cold” presentations refractory to first-line therapy.⁴ |
| Norepinephrine is preferred for hypotensive patients.⁴,⁵ It increases blood pressure and also provides inotropic support.⁵ |

### How are inotropes used in acute heart failure?

| Inotropes are options for patients with severely reduced cardiac output (i.e., SBP < 90 mmHg and/or end organ hypoperfusion; dry and cold or wet and cold).⁴ |
| If the patient is hypotensive due to hypovolemia (dry and cold), this should be corrected before an inotrope is used.⁴ |
| If the patient is wet and cold and SBP < 90 mmHg, an inotrope is first-line.⁴ |
| If the patient is wet and cold and SBP ≥ 90 mmHg, consider an inotrope if the patient is refractory to a diuretic and vasodilator.⁴ |
| Inotropes may increase mortality.¹,⁴ They can cause arrhythmias and myocardial ischemia, and require blood pressure and continuous electrocardiographic monitoring.⁴ Guidelines recommend that inotropes should be reserved for hemodynamically unstable patients (e.g., hypoperfusion, symptomatic hypotension) because they have not been shown to improve outcomes beyond their immediate hemodynamic effects, yet pose risks.¹,⁴ |
| **Inotrope options include** dopamine, dobutamine, and milrinone.¹,² Dobutamine is usually preferred.⁴,⁶ Choice depends on the patient’s hemodynamics and clinical scenario.⁵ Consider the following choices: |
| Recent beta-blocker use (and not responding to initial dobutamine titration): milrinone (action not affected by beta-blockers).⁵,⁶,⁸,¹⁴ |
| Hypotension: dobutamine (if not in shock) or dopamine (to increase blood pressure).⁴,⁵,⁸ |
| Pulmonary hypertension: milrinone (reduces pulmonary vascular resistance).⁵ |
| Cardiorenal syndrome: dopamine or dobutamine (easier to titrate than milrinone [long half-life so delay to steady-state; renally cleared]).⁵ |
| Ischemic heart disease: dobutamine (minimal effect on heart rate vs dopamine; less arrhythmogenic than dopamine; increases cardiac output at least as much as dopamine with lower oxygen consumption; milrinone may increase mortality).⁵,⁶,⁸,⁹ |
| Tolerance to dobutamine: milrinone (tolerance to dobutamine begins within 48 to 72 hours).¹⁴,¹⁵ |

*Continued…*
### Clinical question | Pertinent information
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Inotrope use in acute heart failure, continued | • Use the lowest dose and shortest duration needed to meet the therapeutic goal.5  
• In select end-stage heart failure patients, dobutamine, milrinone, or dopamine may be used to palliate symptoms and improve functional class, as a bridge to transplant or LVAD, or with LVAD.11 Does not improve survival.11

Can you start or continue a beta-blocker in a patient with acute heart failure? | • Continue if patient is hemodynamically stable and there are no contraindications.2  
• In patients who present with symptomatic hypotension or bradycardia, or hypoperfusion, consider reducing the beta-blocker dose or holding.1,4 Unless the patient is in shock, it is preferable to reduce the dose rather than hold the beta-blocker.1,4 Holding beta-blockers in acute heart failure is associated with increased mortality.4  
• In patients with the “warm wet” presentation who are diuretic-refractory, reduce the beta-blocker dose by 50%. Hold the beta-blocker if congestion remains unresponsive, or if inotropes are needed.20  
• In the event that dobutamine is needed, consider that metoprolol may enhance dobutamine’s effect on cardiac output (perhaps due to beta-1 upregulation), while carvedilol may attenuate it.10  
• If the beta-blocker is held, try to restart it before discharge.1  
• Wait until the patient is hemodynamically stable and euclidean to initiate beta-blocker therapy.4,19 Start with a low dose, and be particularly cautious in patients who required an inotrope.2 Arrange for close follow-up post-discharge.19 Attempt dosage increase every two weeks to target (or maximally tolerated) dose.23  
• In patients with heart failure with preserved ejection fraction (HFP EF), beta-blockers can be used to treat hypertension or atrial fibrillation.2

Can you start or continue an ACE inhibitor or ARB in a patient with acute heart failure? | • Continue if patient is hemodynamically stable and there are no contraindications.2  
• In patients who present with significant deterioration of renal function, consider reducing the ACEI or ARB dose or holding until renal function improves.2  
• Hold or reduce dose in the case of symptomatic hypotension, hypoperfusion, or hyperkalemia.4  
• If the ACEI or ARB is held, try to restart it before discharge.1  
• Unless the patient has elevated SBP, wait until after patient has stabilized (e.g., >24 hours after event, hemodynamically stable, euclidean renal function stable) to initiate ACEI or ARB therapy.1,4,19 Arrange for close follow-up post-discharge.19 Check electrolytes and renal function within a week.20 Attempt dosage increase every two weeks to target (or maximally tolerated) dose.23  
• In patients with heart failure with preserved ejection fraction (HFP EF), it is reasonable to titrate to goal blood pressure (SBP <130 mmHg) in the hospital.20,24 ARBs may reduce readmission.24
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| Can you start sacubitril/valsartan (*Entresto*) in the hospital?                  | - When the decision is made to start sacubitril/valsartan, check patient accessibility/affordability and start any prior authorization process before discharge. If applicable, switch the patient from an ACEI to valsartan to ease transition.  
- Sacubitril/valsartan (*Entresto*) can be started in patients with heart failure with reduced ejection fraction (HFrEF) once the patient has stabilized (e.g., for the past six hours: SBP ≥ 100 mmHg, no symptomatic hypotension, no increase in IV diuretic, no IV vasodilators or nitrates; no inotropes for 24 hours), assuming they meet usual criteria for initiation (e.g., ARB and ACEI discontinued, 36-hour washout from last ACEI dose, taking appropriate guideline-directed therapy, etc).  
- Sacubitril/valsartan can be started at a dose of 24/26 mg twice daily (SBP ≥ 100 to 120 mmHg), or 49/51 mg twice daily (SBP ≥ 120 mmHg).  
- In appropriate patients, starting sacubitril/valsartan is not riskier than starting an ACEI in patients hospitalized for heart failure in regard to renal function, hyperkalemia, hypotension, and angioedema [Evidence level A-1].  
- Arrange for close follow-up post-discharge. Check electrolytes and renal function within a week. Attempt dosage increase every one to two weeks to target (or maximally tolerated) dose. |
| Can you start or continue spironolactone or eplerenone in a patient with acute heart failure? | - Continue if patient is hemodynamically stable and there are no contraindications.  
- In patients who present with significant deterioration of renal function, consider reducing the aldosterone antagonist dose or holding until renal function improves.  
- Hold or reduce dose in the case of symptomatic hypotension, hypoperfusion, or hyperkalemia.  
- If the aldosterone antagonist is held, try to restart it before discharge.  
- There is limited data on starting or intensifying aldosterone antagonists in acute heart failure, but it may be safe to do so if eGFR >30 mL/min/1.73 m² and SBP >90 mmHg [Evidence level B-1]. Patient should be euvoletic, and electrolytes should be stable. Consider for patients with diuretic resistance.  
- Arrange for close follow-up post-discharge. Check electrolytes and renal function within a week. Attempt dosage increase every two weeks to target (or maximally tolerated) dose. Consider need to reduce or discontinue any potassium supplementation, especially if diuretic doses are reduced as symptoms improve.  
- In patients with heart failure with preserved ejection fraction (HFpEF), it is reasonable to titrate to goal blood pressure (SBP <130 mmHg) in the hospital. Aldosterone antagonists may reduce readmission. |
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| Can you start or continue ivabradine (*Corlanor* [U.S.], *Lancora* [Canada]) in a patient with acute heart failure? | - There is only limited data on ivabradine in the acute setting. It has been started in patients with heart rate ≥80 beats per minute, after weaning from vasopressors. (Patients presenting in cardiogenic shock [SBP ≤85 mmHg] were excluded from this study.) [Evidence level B-1].¹⁷  
- Not for patients with heart failure with *preserved ejection fraction* (*HFpEF*).²³ |

*Users of this resource are cautioned to use their own professional judgment and consult any other necessary or appropriate sources prior to making clinical judgments based on the content of this document. Our editors have researched the information with input from experts, government agencies, and national organizations. Information and internet links in this article were current as of the date of publication.*
**Levels of Evidence**

In accordance with our goal of providing Evidence-Based information, we are citing the LEVEL OF EVIDENCE for the clinical recommendations we publish.

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| A     | Good-quality patient-oriented evidence.* | 1. High-quality RCT  
2. SR/Meta-analysis of RCTs with consistent findings  
3. All-or-none study |
| B     | Inconsistent or limited-quality patient-oriented evidence.* | 1. Lower-quality RCT  
2. SR/Meta-analysis with low-quality clinical trials or of studies with inconsistent findings  
3. Cohort study  
4. Case control study |
| C     | Consensus; usual practice; expert opinion; disease-oriented evidence (e.g., physiologic or surrogate endpoints); case series for studies of diagnosis, treatment, prevention, or screening |

*Outcomes that matter to patients (e.g., morbidity, mortality, symptom improvement, quality of life).


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**References**


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