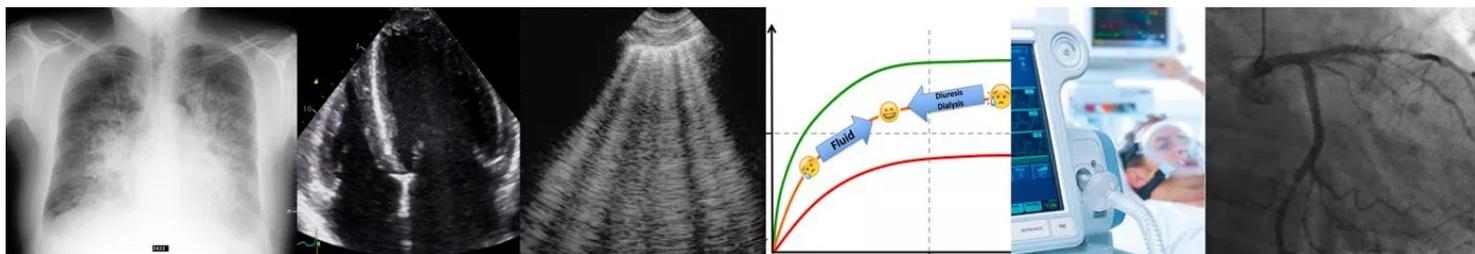




The Internet Book of Critical Care

Cardiogenic shock & severe CHF

November 13, 2016 by [Josh Farkas](#)



(<https://io.wp.com/emcrit.org/wp-content/uploads/2016/11/chfopener.jpg>)

CONTENTS

- [intro: what this chapter is about \(#introduction\)](#)
- [hemodynamic evaluation & risk stratification \(#hemodynamic_evaluation_&_risk_stratification\)](#)
- [causes of heart failure decompensation \(#causes_of_heart_failure_decompensation\)](#)
- [investigations \(#investigations\)](#)
- **treatment**
 - [1\) fix the lungs \(#rx_#1- treat_the_lungs_\(effusion_&_pulm_edema\)\)](#)
 - [2\) optimize the MAP \(#rx_#2- optimize_the_MAP\)](#)
 - [3\) optimize volume status \(#rx_#3- optimize_volume_status\)](#)
 - [4\) consider inotrope for HFrEF \(#rx_#4- consider_inotrope_for_HFrEF\)](#)
 - [5\) treat underlying etiology \(#rx_#5- treat_underlying_etiology\)](#)
 - [6\) mechanical circulatory support \(#rx_#6- temporary_mechanical_circulatory_support\)](#)
 - [7\) things to avoid \(#rx_#7- interventions_to_avoid\)](#)
- [algorithm \(#algorithm\)](#)
- [podcast \(#podcast\)](#)
- [questions & discussion \(#questions_&_discussion\)](#)
- [pitfalls \(#pitfalls\)](#)

introduction

[\(back to contents\) \(#top\)](#)

this chapter is about LV failure

- LV failure spans a *spectrum* of severity which ranges from mild heart failure decompensation to frank cardiogenic shock.
- Cardiogenic shock isn't necessarily a discrete entity, but rather may be conceptualized as the most severe form of heart failure.¹
 - Patients with severe heart failure may go in and out of cardiogenic shock, depending on their management.

this chapter is *not* about:

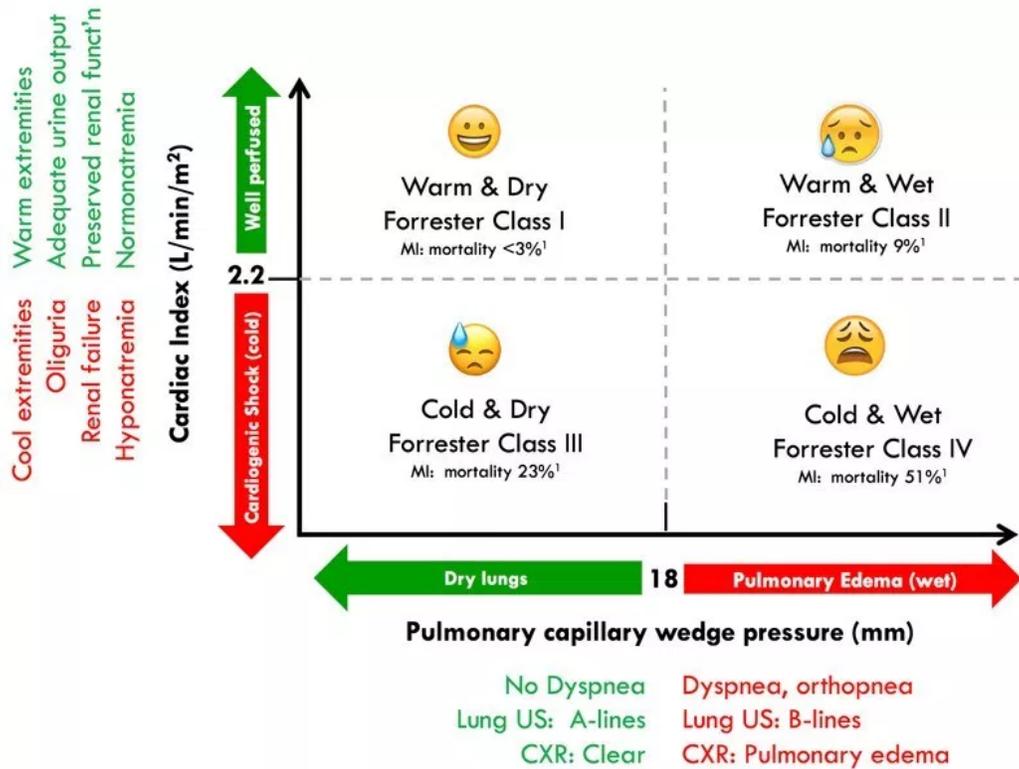
- [SCAPE \(https://emcrit.org/emcrit/scape/\)](https://emcrit.org/emcrit/scape/) (Sympathetic Crashing Acute Pulmonary Edema), a distinct form of rapid-onset heart failure which is associated with hypertension.
 - The basic principles in this chapter will apply to SCAPE. However, the chapter on SCAPE will be more clinically applicable to that scenario.
- Right ventricular failure (cor pulmonale).
- Less common types of heart failure with unique physiology (e.g. acute valvular regurgitation, hypertrophic cardiomyopathy, dynamic LV outflow tract obstruction).

hemodynamic evaluation & risk stratification

[\(back to contents\) \(#top\)](#)

bedside hemodynamic assessment: try to determine the following

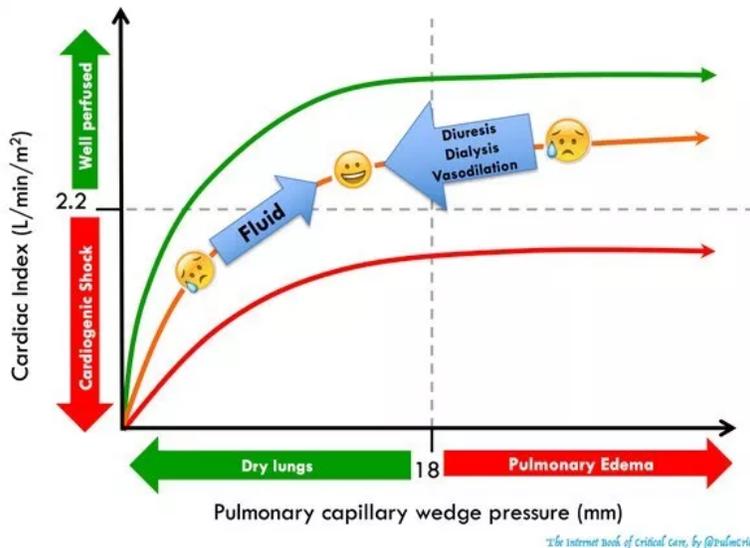
- **Cardiac index (systemic perfusion)**
 - Adequate cardiac index is suggested by warm extremities, normal capillary refill, preserved renal function, good urine output, and adequate mentation.
 - Inadequate cardiac index may be suggested by cool extremities, poor capillary refill, acute kidney injury, oliguria, poor mentation, and elevated transaminases ("shock liver").
 - Normal mentation doesn't prove that perfusion is adequate. Some patients in occult cardiogenic shock may have normal mentation despite malperfusion of other organs (e.g. shock liver and acute kidney injury).
- **Pulmonary capillary wedge pressure (pulmonary congestion)**
 - High wedge pressure is suggested by *pulmonary edema* (dyspnea, rales on lung auscultation, edema on chest X-ray, and B-lines on lung ultrasound).
 - Low wedge pressure is suggested by *dry lungs* (no dyspnea, clear lungs on auscultation and chest X-ray, A-lines on lung ultrasound).
 - The best test to determine wedge pressure is *lung ultrasonography*. Bilateral diffuse B-lines imply elevated wedge pressure, whereas bilateral A-lines suggest a low or normal wedge pressure. Ultrasonography is more sensitive than chest X-ray or exam to detect mild cardiogenic pulmonary edema.
- **Total body volume status (systemic congestion)**
 - Note that it's possible for patients to have an elevated pulmonary capillary wedge pressure *without* total body volume overload (e.g. euvoolemia plus an acutely deteriorating left ventricle).
 - Clinical history can be very useful here: is there a history of volume loss (e.g. gastroenteritis, over-diuresis) or volume gain (e.g. diuretic nonadherence, iatrogenic fluid administration)? Weight gain or loss?
 - Echocardiographic assessment of the inferior vena cava and jugular veins may allow estimation of the central venous pressure.
 - Peripheral pitting edema suggests systemic congestion.



(1) Mortality numbers from Forrester 1976 PMID 790191. Mortality is probably lower today. *The internet book of critical care, by @drumcrit*

<https://i2.wp.com/emcrit.org/wp-content/uploads/2016/11/forres1.jpg> **Forrester classifications**

- Based on the pulmonary capillary wedge pressure and the cardiac index, patients may be categorized as shown above. These categorizations have direct implications for prognosis and treatment.²
- First, imagine overlaying cardiac output curves over this classification system (shown below).
 - Green curve: normal cardiac output function
 - Orange curve: moderate heart failure
 - Red curve: severe heart failure
- Patients who are warm/wet may often be managed with volume removal and/or vasodilation to reduce their afterload (vasodilation shifts fluid out of the lungs without affecting the total body volume).
- Patients who are cold/dry may often be managed by fluid administration:

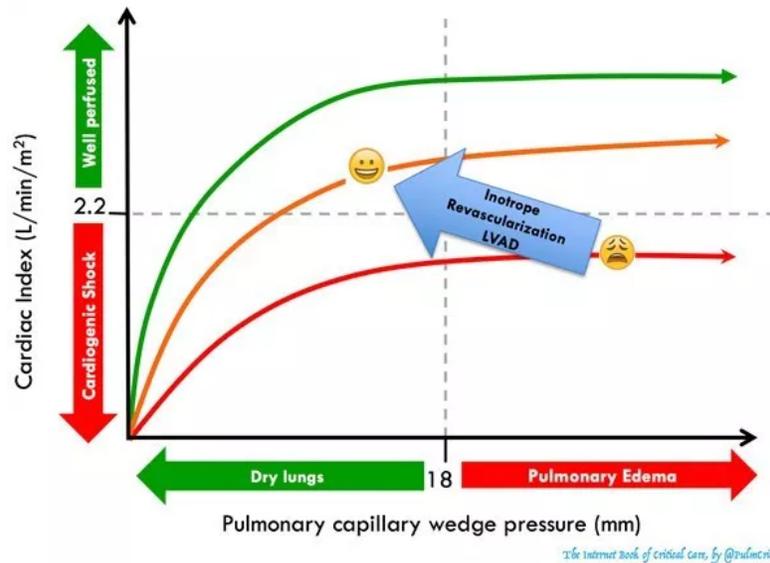


The internet book of critical care, by @drumcrit

<https://i2.wp.com/emcrit.org/wp-content/uploads/2016/11/forrest2.jpg>

classic presentation of cardiogenic shock: patients who are cold & wet

- Cardiogenic shock may be roughly conceptualized as requiring two components:
 - (1) Systemic hypoperfusion due to low cardiac output (cold).
 - (2) Filling pressures are elevated (wet).
- Patients in cardiogenic shock *cannot be fixed* with volume administration or removal.
 - Giving volume will worsen their pulmonary congestion (making them wetter).
 - Removing volume will worsen their systemic hypoperfusion (making them colder).
- Management of cardiogenic shock usually requires interventions to improve *cardiac function* (e.g., inotropic medications, revascularization, or a mechanical support device).
- Cardiogenic shock patients may look deceptively OK, but they are indeed critically ill.
 - Early recognition facilitates appropriate ICU management.
 - The patient with unrecognized cardiogenic shock will generally *fail* to respond to non-intensive therapy, running in circles (typically the patient is initially diuresed, then develops worsening renal failure, then is given fluid back, then develops pulmonary edema, then transferred to ICU).



(<https://i.wp.com/emcrit.org/wp-content/uploads/2016/11/imageci.jpg>)



EMUSS

@emuss_uk

Cardiogenic shock, what to expect #POCUS, poor LV, Raised JVP, distended IVC, B lines & Pleural Effusion #FOAMus #rcemlearning @TrEmatus @echo_urg @Echoscopie @echotainment @girardphilippe1 @EmergencyEcho @pocusireland @POCUSEcho @echocardiac @EcosemesA @GTEcografia @hopeful9613



103 12:47 PM - Feb 21, 2018

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vasodilated cardiogenic shock

- To make things confusing, cardiogenic shock may trigger a systemic inflammatory response with elevated cytokine levels and *reduced systemic vascular resistance*. This may occur later in the course of cardiogenic shock, possibly due to ischemic tissue damage. This condition will mimic septic shock.³
- To add further to the confusion, some patients with *septic* shock will develop a sepsis-induced cardiomyopathy. So, advanced-stage septic shock and advanced-stage cardiogenic shock can look clinically quite similar (e.g., shock, vasodilation, reduced systolic heart failure, systemic inflammation).
 - This may represent a final common pathway of the dying patient.

HFpEF vs. HFrEF

- Heart failure patients may be classified as heart failure with reduced ejection fraction (<40%, HFrEF, a.k.a. "systolic failure") vs. heart failure with preserved ejection fraction (HFpEF, a.k.a. "diastolic dysfunction").
- Differentiating HFpEF vs. HFrEF can be done with bedside echocardiography.
 - HFrEF: reduced ejection fraction
 - HFpEF: preserved ejection fraction. Presence of heart failure is suggested by dilated left atrium, left ventricular hypertrophy, and pulmonary congestion (B-lines on lung ultrasonography).⁴
- Treatment of these disorders is generally similar, with a few differences:
 - HFpEF patients shouldn't be treated with inotropes.
 - HFpEF patients may be more preload-dependent, thus at higher risk for hypotension following diuresis.

causes of heart failure decompensation

[\(back to contents\) \(#top\)](#)

volume alteration

- Acute volume overload (e.g. diuretic nonadherence, dietary indiscretion)
- Acute hypovolemia (e.g. over-diuresis, reduced oral intake, gastroenteritis)

acute reduction in LV ejection fraction

- Acute MI
- Takotsubo cardiomyopathy, post-cardiac arrest stunning
- Tachyarrhythmia
- Peripartum cardiomyopathy
- Myocarditis (e.g. viral, SLE, giant-cell)

arrhythmia

- Bradyarrhythmia
- Tachyarrhythmia (most often new-onset atrial fibrillation)

other

- Thyroid disease
- Medications
 - Toxicity (e.g. excess beta-blocker, digoxin toxicity)
 - Medication nonadherence
- Uncontrolled hypertension

- Hypophosphatemia

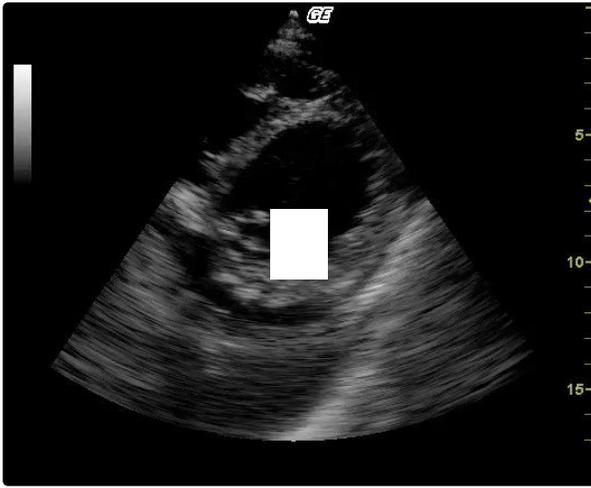


Augusto Hernandez M

@augustoraulhm

Previously healthy 33yo female with refractory cardiogenic shock in her 2 week post partum at ICU: PPCM

@EchoResPract #POCUS @grupomedicopty



72 5:01 PM - Aug 17, 2017

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investigations

[\(back to contents\) \(#top\)](#)

cardiac imaging

- EKG
- Echo

Assessing Left Ventricular Function



labs

- CBC, Electrolytes including Ca/Mg/Phos (if hypocalcemia suspected check iCa)
- Troponin
- Lactate level
- Liver function tests (marked transaminase elevation suggests shock liver with poor cardiac output)
- TSH if thyroid disease suspected
- Digoxin level for patients on digoxin
- Brain natriuretic peptide (BNP) levels are *unhelpful* (cardiopulmonary ultrasonography is a superior test).



Lauren Westafer

@LWestafer

But [#hasslebias](#) ..

(Get over it & do better test)

BNP: + LR 2, – LR 0.2

Lung US: + LR 22, – LR 0.03 [foamcast.org/2015/12/08/epi...](#)

[#FOAMed](#)) [twitter.com/shivanimody/st...](#)

Shivani Mody @ShivaniMody

Just take a minute and do the lung US to help differentiate between CHF vs COPD @nobleultrasound #EEM17AllNYC

26 8:45 AM - Mar 29, 2017

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Swan-Ganz catheter?

- Hemodynamic assessment can generally be made non-invasively as described above. Furthermore, high-quality echocardiographic images with doppler can provide substantial hemodynamic information (e.g. cardiac output based on the velocity-time integral).⁵
- Reasons for avoiding a Swan-Ganz catheter include:⁶

- 1) Swan-Ganz catheterization is an invasive procedure which carries risk of pneumothorax, line infection, arrhythmia, pulmonary artery perforation, and heart block. These risks aren't merely academic; I've seen all of these complications.
- 2) Swan-Ganz catheterization will always reveal abnormal numbers, but it's unknown what we should do with this data.⁷ Specifically, there is no defined goal for cardiac output or systemic vascular resistance. A cardiac index which may be adequate for one patient will leave another patient in cardiogenic shock.
- 3) Swan-Ganz catheterization tends to encourage fluid management based on static filling pressures. However, these pressures (even the hallowed pulmonary capillary wedge pressure) do not predict fluid-responsiveness.⁸ Titrating fluid administration or diuresis against the wedge pressure is thus an inferior strategy compared to empirically trialing fluids and carefully monitoring the patient's clinical response.
- 4) Numerous studies have failed to show benefit from Swan-Ganz catheterization both in critically ill patients overall and also specifically in heart failure patients.^{9 10 11 12} The ESCAPE trial, a multicenter RCT in heart failure, showed that Swan-Ganz catheterization increased adverse events without offering benefit.¹³
- 5) Over time, there has been steady improvement in echocardiography. Meanwhile, physicians and nurses are becoming *less* skilled at insertion and troubleshooting of Swan-Ganz catheters. Altogether, this means that the added value of Swan-Ganz catheter beyond echocardiography is perpetually declining. Given that the Swan-Ganz catheter had dubious value in its heyday (the 1990s), it's even *less* beneficial currently.
- Routine use of Swan-Ganz catheterization is not recommended by AHA guidelines, even in cardiogenic shock.³ Reasons to consider Swan-Ganz catheterization may include:
 - Unresponsiveness to initial therapy.
 - Documentation of hemodynamics to determine candidacy for cardiac transplantation or ventricular assist device.

rx #1- treat the lungs (effusion & pulm edema)

[\(back to contents\) \(#top\)](#)

BiPAP (noninvasive ventilation)

- Acute BiPAP: Patients in respiratory distress due to heart failure generally respond nicely to BiPAP. This is strongly supported by evidence in heart failure:
 - BiPAP has been shown to reduce intubation and mortality.
 - BiPAP reduces cardiac preload and afterload (physiologic effects similar to an ACE inhibitor).
 - It's not merely enough to place the patient on BiPAP – for maximal benefit the pressures should be up-titrated as tolerated (figure below). The most important parameter is the expiratory pressure, which should be ramped up rapidly if possible. More on noninvasive ventilation use [here \(https://emcrit.org/pulmcrit/bipap-hfnc/\)](https://emcrit.org/pulmcrit/bipap-hfnc/).
- Chronic nocturnal BiPAP: Patients with heart failure plus sleep apnea or obesity hypoventilation may greatly benefit from ongoing nocturnal CPAP or BiPAP, respectively. Although CPAP/BiPAP is technically indicated for sleep-disordered breathing, it can also help a lot with the heart failure component (e.g. promote pulmonary decongestion overnight).

BiPAP titration in CHF

- **Example**
 - Start: iPAP 10 cm / ePAP 5 cm
 - Increase: iPAP 15 cm / ePAP 10 cm
 - Increase: iPAP 18 cm / ePAP 14 cm
- **Basic principles**
 - Mean airway pressure provides goodness
 - Ramp up the ePAP aggressively to achieve high mean airway pressure
 - Could also use CPAP alone (titrate from 5 cm up to 15 cm).

BiPAP titration in everything else

- **Example**
 - Start: iPAP 10 cm / ePAP 5 cm
 - Increase: iPAP 15 cm / ePAP 5 cm
 - Increase: iPAP 18 cm / ePAP 5 cm
- **Basic principles**
 - Driving pressure supports the work of breathing (equal to iPAP-ePAP).
 - Ramp up iPAP to increase the driving pressure.

The Internet Book of Critical Care, by @pulmcrit

<https://io.wp.com/emcrit.org/wp-content/uploads/2016/11/bipaptrr.jpg> **intubation**

- Often needed for frank cardiogenic shock (especially patients with delirium due to brain hypoperfusion).
- Advantages:
 - Provides full support for the work of breathing, which may allow shunting of blood away from the diaphragm and towards vital organs.

- Stabilizes patients for procedures that require lying flat (e.g. cardiac catheterization)
- Disadvantage: intubation in cardiogenic shock carries risks of hypotension/arrest, so be careful.
- When in doubt about the need for intubation: initiate BiPAP without delay, optimize other factors as rapidly as possible (e.g. Rx #2-5). Continually re-evaluate and intubate if necessary. Even if the patient does eventually require intubation, it's often safer to resuscitate them before intubation.

drainage of large effusions

- If the patient isn't in respiratory distress, then effusions should be managed with diuresis and optimization of heart failure. However, it can take large effusions a long time to resorb. If the patient has large effusion(s) and this is causing significant respiratory distress or hypoxemia, then therapeutic drainage may be beneficial.¹⁴

inhaled pulmonary vasodilator

- Inhaled epoprostanol or nitric oxide may be considered for an intubated patient with biventricular failure or severe hypoxemia. Physiological benefits include:
 - (1) Reduction in right ventricular afterload may improve cardiac output among patients with right ventricular failure.
 - (2) Inhaled pulmonary vasodilators will improve perfusion:ventilation matching and thereby improve the oxygen saturation.
- There is a risk that improved RV function will dump more blood into the left ventricle, thereby increasing the pulmonary capillary wedge pressure and exacerbating cardiogenic pulmonary edema. However, in the intubated patient this generally isn't a major problem.

rx #2- optimize the MAP

[\(back to contents\) \(#top\)](#)

For a patient with decompensated heart failure, the blood pressure needs to be high enough to perfuse the organs. However, if the pressure is too high, this will increase the workload on the heart (excessive afterload). Often an ideal blood pressure will be in the low-normal range (e.g. MAP ~65 mm).

hypertension (or high-normal Bp) should be managed with afterload reduction

- Afterload reduction is highly beneficial if the patient has enough blood pressure to tolerate it. Afterload reduction may improve cardiac output, decongest the lungs, and reduce the myocardial workload. It's a win-win-win.
- In the acute phase, a high-dose **nitroglycerine** infusion is the safest vasodilator.
 - High doses (up to 200-250 mcg/min) may be needed to achieve arterial vasodilation, titrated against the patient's blood pressure.
- Once the patient has stabilized a bit, this may be transitioned to an oral agent:
 - An ACE-inhibitor or ARB is good at afterload reduction. However, this increases the risk of renal failure, especially in a tenuous patient who is being actively diuresed.
 - The combination of **hydralazine** plus **isosorbide dinitrate** has similar physiologic effects compared to an ACE-inhibitor without the nephrotoxicity. The usual starting dose is isosorbide dinitrate 20 mg PO q6hr and hydralazine 37.5 mg PO q6hr. If blood pressure remains high/normal, this may be up-titrated to target twice the initial dose.¹⁵

hypotension may be managed with an inopressor (e.g., epinephrine or norepinephrine)

- Hypotension requires treatment to defend coronary and end-organ perfusion.
- **Norepinephrine** is widely recommended as a front-line agent for cardiogenic shock. Norepinephrine will improve the blood pressure, but there is a risk that excessive afterload could drop the cardiac output.
- **Epinephrine** may be a reasonable choice for a patient with reduced ejection fraction, hypotension, and poor cardiac output. At low doses (e.g. 0-5 mcg/min) epinephrine acts predominantly as an *inotroph*. However, unlike dobutamine, epinephrine doesn't cause vasodilation.¹⁶ The net effect of low-dose epinephrine is often an improvement in blood pressure and cardiac output, without affecting systemic vascular resistance much.
- Dopamine should be avoided, given evidence of harm compared to norepinephrine in the SOAP-II trial.¹⁷

rx #3- optimize volume status

[\(back to contents\) \(#top\)](#)

fluid administration

- Consider giving a fluid challenge if the following conditions are met:
 - (1) There is insufficient end-organ perfusion (e.g. acute kidney injury).
 - (2) No evidence of pulmonary congestion (e.g. no B-lines on lung ultrasonography).
 - (3) Overall assessment suggests true hypovolemia (e.g. no systemic congestion).
- Fluid should be given in boluses of 500-1000 ml fluid challenges, with careful determination of the effect on the patient. If fluid isn't causing clinical improvement, don't give more.
- Be careful – static hemodynamic parameters (e.g. CVP, pulmonary capillary wedge pressure) do not predict fluid-responsiveness and should not be used as the primary determinant of fluid administration.⁸

fluid removal

- Consider diuresis if the following conditions are met:
 - (1) There is significant pulmonary and/or systemic congestion.
 - (2) Overall assessment suggests total body fluid overload.
- For patients who aren't responding adequately to furosemide, consider adding a thiazide diuretic (e.g. metolazone 5 mg daily or indapamide 5 mg daily). This may enhance sodium excretion, with improved clearance of extravascular edema fluid.^{18 19} Patients with severe systemic congestion may have poor PO intake, so they may require IV diuretics only (e.g. IV furosemide plus IV chlorothiazide).
- Patients with substantially elevated central venous pressure can experience an *improvement* in renal function with diuresis, because decreasing venous congestion will increase blood flow through the kidney. The driving pressure through the kidneys is equal to the MAP minus the CVP, so lowering the CVP may increase renal perfusion:

$$\text{Renal Perfusion Pressure} = (\text{MAP} - \text{CVP})$$

rx #4- consider inotrope for HFrEF

[\(back to contents\) \(#top\)](#)

avoid catecholamine inotropes when possible

- Inotropes will cause a short-term improvement in hemodynamics. Unfortunately, available evidence indicates that inotrope use associates with worse outcomes.²⁰ Available prospective RCT data is scanty, but it likewise suggests that inotropes may be harmful.²¹
- Inotropes should be used only if necessary, for the following indications:²²
 - **Hypoperfusion with low-normal blood pressure** (e.g. acute kidney injury with poor urine output despite #1-3 above).
 - **Refractory cardiogenic pulmonary edema:** Front-line therapies for cardiogenic pulmonary edema include #1-3 above: BiPAP, nitroglycerine (if blood pressure is adequate), and diuresis (if there is evidence of volume overload). Some patients will fail to respond to these treatments, especially hypotensive patients in whom nitroglycerine or diuresis is contraindicated. In such patients inotropes may be used with a goal of reducing the pulmonary capillary wedge pressure and decongesting the lungs.

dobutamine vs milrinone?

- Overall both agents are generally similar. Both may cause hypotension (milrinone somewhat more than dobutamine) so they shouldn't be used in profoundly hypotensive patients (generally start with blood pressure control first, see Rx step #2 above).
- Dobutamine has a shorter half-life, making it is more readily titratable. This may be preferable for immediate stabilization of an acutely ill patient (e.g. a patient with marked pulmonary edema, on the verge of requiring intubation).
- Milrinone may be favored in heart failure, because it provides more effective vasodilation and might avoid toxicity from overstimulation of beta-receptors. Unfortunately, milrinone is cleared by the kidneys, so dose titration in renal failure can be tricky. Even with normal renal function the half-life of milrinone is long (2.3 hours), making rapid titration impossible.
- There aren't prospective RCTs comparing the two agents, so ultimately selection is somewhat subjective.

digoxin

- Digoxin is the only positive inotropic agent whose use doesn't correlate with increased mortality. It's not a particularly powerful inotrope, but it might be the safest (with close monitoring of digoxin levels).
- Digoxin can be considered for patients with long-standing atrial fibrillation and systolic heart failure.

- Patients with new-onset atrial fibrillation might benefit from cardioversion to sinus rhythm instead.
- Digoxin generally isn't used as a front-line agent for heart failure, but can be considered when the patient is failing to respond to other therapies.
- With intravenous loading, improvement may occur over several hours.

rx #5- treat underlying etiology

[\(back to contents\) \(#top\)](#)

arrhythmia treatment

- If shock is caused by new-onset tachyarrhythmia (e.g. atrial fibrillation), then reversion to sinus rhythm may be beneficial. However, if the heart rate isn't very high then be careful – slowing down the heart rate may actually [aggravate matters](https://emcrit.org/squirt/af/).

cardiogenic shock due to MI

- Treat with medical therapies for type-I MI (e.g. aspirin, P2Y12 inhibitor, anticoagulation).
- Revascularization is essential. This is beneficial even at delayed timepoints.²³
 - Thrombolysis works poorly in cardiogenic shock – PCI or CABG is generally necessary.



Aditya Bharadwaj
@adityadoc1

#cardiogenicshock left main PCI via #RadialFirst #impella out on day2, d/c home day4 @SVRaoMD @rwyeh @rajivxgulati @Radial_ICG @DrSethdb



10 3:10 PM - Sep 17, 2017

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anemia?

- Although heart failure patients are often anemic, this usually isn't the cause of their decompensation. As a general rule, treatment of the dyspneic patient with blood transfusion in the expectation that this will improve pulmonary status is disappointing.
- Patients should be transfused to standard transfusion targets: >7 mg/dL (>70 g/L) or, in a patient with evidence of active myocardial ischemia, >8 mg/dL (>80 g/L).

rx #6- temporary mechanical circulatory support

[\(back to contents\) \(#top\)](#)

Indicated for end-organ dysfunction refractory to #1-5 above. Perhaps the most important end-organ to support is the kidneys. If the patient develops severe renal failure, this aggravates matters greatly. Depending on the context, mechanical support may play a variety of different roles:

- Bridge to recovery.
- Bridge to surgically-implanted ventricular assist device (VAD).
- Bridge to cardiac transplant.
- Bridge to re-assessment, ideally following resolution of multi-organ failure ("bridge to bridge").

Options include aortic balloon pumps, percutaneous centrifugal pumps, and full veno-arterial ECMO. Controversy remains regarding the ideal timing and use of various devices. Expert consultation is required. One problem with most of these devices is that they constrain the patient to bed with very limited mobility.²⁹

factors involved in determining mechanical support device

- Is support needed for the left ventricle, the right ventricle, or both?
- Ability to tolerate anticoagulation? (This is required for both temporary & durable devices.)
-

intra-aortic balloon pumps (IABP)

- Most popular devices overall.
- Most thoroughly investigated. Unfortunately, RCTs consistently fail to show improvement in patient-centered outcomes.^{24 25}
- May augment cardiac output by 0.3-0.5 liters/minute. (31374209)
- Contraindications: Severe peripheral artery disease, moderate-to-severe aortic regurgitation, aortic disease.

impella

- Evidentiary basis?
 - LV-Impella failed to show any difference when compared to an intra-aortic balloon pump in one small RCT.²⁶
 - Recent retrospective registry study compared matched patients treated with impella vs. IABP: patients treated with impella had some improvement in renal function, more bleeding, more peripheral vascular complications, and no difference in mortality (30586755).
- Contraindications: LV thrombus, mechanical aortic valve, severe aortic stenosis, moderate-to-severe aortic regurgitation, severe peripheral arterial disease, inability to anti-coagulate (31374209).
- There is even less evidence regarding most temporary mechanical devices (e.g. RV-impella, TandemHeart, RV-TandemHeart, Thoratec, Aortix, Reitan pump).²⁷

VA-ECMO

- Seems to show the most promise, as a rapidly deployable strategy capable of supporting the sickest patients (patients with respiratory failure and biventricular heart failure).²⁸ Unfortunately, this isn't yet widely available.



josh farkas
@PulmCrit

if you had to pick *one* mechanical support device to have at your hospital for use in cardiogenic shock, what would it be?

- 15% intra-aortic balloon pump
- 12% impella (single, LV only)
- 1% tandem heart
- 72% VA ECMO

486 votes • Final results

15 7:22 AM - Oct 11, 2018

[23 people are talking about this](#)

rx #7- interventions to avoid

[\(back to contents\) \(#top\)](#)

- Nephrotoxic medications (e.g. NSAIDs, ACEi/ARB).
- Don't try to suppress a sinus tachycardia. This is often a *compensatory* mechanism that may be keeping the patient alive.
- Avoid using diltiazem for rate control in AF patients with decompensated heart failure and reduced ejection fraction (the negative inotropic effects may be problematic). More on the unstable AF patient [here \(https://emcrit.org/squirt/af/\)](https://emcrit.org/squirt/af/).

- Don't treat mild, stable hyponatremia with an infusion of 3% saline or salt tablets. Patients with heart failure commonly have mild hyponatremia. This will generally tend to resolve with treatment of the underlying heart failure (e.g. diuresis with furosemide).
- Fluid and sodium restriction haven't shown benefit in RCTs.^{30 31} Hospital food often isn't great, so the most humane thing is probably to provide a regular diet. Follow fluid balance and use diuretics if needed.

be very careful with beta blockers in decompensated heart failure

- Beta-blockers are fantastic for *chronic, compensated* heart failure, but potentially dangerous in decompensated heart failure (negative inotropy may further impair cardiac function).
- Beta-blockers shouldn't be *started* in the context of decompensated heart failure.
- It is controversial whether beta-blockers should be *continued* among patients who were previously taking them.
 - Beta-blockers should be held in patients with cardiogenic shock.
 - For patients who aren't in shock, beta-blockers may be continued (perhaps at a reduced dose initially).
- Please note that a beta-blocker is the *opposite* to giving an inotrope. So any *enthusiasm* for using dobutamine in heart failure should translate into an equal and opposite *aversion* towards beta-blockers.

algorithm

[\(back to contents\) \(#top\)](#)

severe heart failure & cardiogenic shock

Evaluation

- EKG & echocardiography
- CBC, Lytes including Ca/Mg/Phos
- Troponin, Lactate, Liver function tests if shock is suspected
- TSH and/or digoxin level depending on context



Rx #1 – Treat the lungs

- Consider BiPAP (vs intubation) in cardiogenic pulmonary edema
- Large effusion(s) may be drained if causing acute distress
- Consider inhaled epoprostenol for intubated patient with right ventricular failure or pulmonary hypertension

Rx #2 – Optimize the MAP

- HTN/normotension → Afterload reduction (nitroglycerine infusion or hydralazine 37.5 mg & isosorbide dinitrate 20 mg q6hr)
- Hypotension (severe or w/ organ dysfunction) → Norepinephrine (epinephrine is another option in HFrEF with hypoperfusion)

Rx #3 – Optimize the volume

- Fluid challenge if: hypoperfusion, no pulmonary congestion (no B-lines on ultrasound), assessment suggests total body hypovolemia
- Diuresis if: significant systemic/pulmonary congestion, assessment suggests total body volume overload

Rx #4 – Consider inotrope (usually dobutamine/milrinone) for HFrEF if:

- (a) Normotensive patient with organ hypoperfusion
- (b) Refractory cardiogenic pulmonary edema in hypotensive patient
- Note: *Digoxin* may be considered as a weak inotropic agent in patients with chronic AF, HFrEF, and refractory heart failure.

Rx #5 – Treat underlying etiology

- New-onset tachyarrhythmia causing heart failure: cardioversion, antiarrhythmics
- Ischemic cardiomyopathy: Revascularization, treatment for acute MI if present

Rx #6 – Mechanical circulatory support

- Consider for persistent organ failure – device of choice is patient/institution specific.

Rx #7 – Things to avoid

- Nephrotoxins (e.g. NSAIDs, ACE-inhibitors, angiotensin receptor blockers)
- Initiation of beta-blocker in decompensated heart failure
- Any beta-blocker or calcium-channel blocker (e.g. diltiazem) in a patient with cardiogenic shock

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[\(back to contents\) \(#top\)](#)



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[\(back to contents\) \(#top\)](#)

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- Failure to identify a patient who is cold and wet (Forrester class IV). These patients may not look terrible, but they have cardiogenic shock and generally require ICU admission.
- Treatment plan that focuses on a single intervention (e.g. diuresis), without optimizing other aspects of the patient (e.g. afterload reduction).
- Delayed management of respiratory distress (e.g. with BiPAP, effusion drainage, or intubation).
- Application of an outpatient-style management (e.g. beta-blocker and ACEi/ARB initiation) in a critically ill patient with cardiogenic shock.

Going further:

- **Overview**
 - [Cardiogenic Shock \(https://emcrit.org/emcrit/cardiogenic-shock/\)](https://emcrit.org/emcrit/cardiogenic-shock/) (Scott Weingart, 2009)
 - [Cardiogenic Shock \(http://rebelem.com/cardiogenic-shock/\)](http://rebelem.com/cardiogenic-shock/) (Anand Swaminathan, Rebel EM)
- **Shocky AF patient**
 - [Shocky patient in AF w/ RVR \(https://emcrit.org/squirt/af/\)](https://emcrit.org/squirt/af/) – more complete IBCC chapter to follow eventually (PulmCrit)
- **Swan-Ganz catheters**
 - [Pulmonary artery catheters \(https://lifeinthefastlane.com/cc/pulmonary-artery-catheters/\)](https://lifeinthefastlane.com/cc/pulmonary-artery-catheters/) (Aaron Sparshott, LITFL) & [Literature summaries \(https://lifeinthefastlane.com/cc/pulmonary-artery-catheter-literature-summaries/\)](https://lifeinthefastlane.com/cc/pulmonary-artery-catheter-literature-summaries/) (Chris Nickson)
 - [Why we fail at hemodynamics: the flaw of averages \(https://emcrit.org/pulmcrit/hemodynamics-swan-curse/\)](https://emcrit.org/pulmcrit/hemodynamics-swan-curse/) (PulmCrit)
 - [Alice in Intensiveland \(http://www.gjcu.sgul.ac.uk/teaching/resources/left-field/files/alice_CHEST1995.pdf\)](http://www.gjcu.sgul.ac.uk/teaching/resources/left-field/files/alice_CHEST1995.pdf) (Bartlett, CHEST)
- **BNP is unhelpful**
 - [BNP \(https://lifeinthefastlane.com/cc/brain-natriuretic-peptide-bnp/\)](https://lifeinthefastlane.com/cc/brain-natriuretic-peptide-bnp/) (Chris Nickson, LITFL)
 - [The Case of Dubious Squire \(https://emcrit.org/emnerd/the-case-of-dubious-squire/\)](https://emcrit.org/emnerd/the-case-of-dubious-squire/) (Rory Spiegel, EMNerd)
 - [BNP in the Emergency Department: Does it change management? \(http://www.emdocs.net/bnp-level-in-the-emergency-department-does-it-change-management/\)](http://www.emdocs.net/bnp-level-in-the-emergency-department-does-it-change-management/) Michael Lamberta and Andrew Chertoff, emDocs

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