emDocs

The EM Educator Series

The EM Educator Series: Empyema – Cardiogenic Shock – Don't be shock'ed, it's not sepsis

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Case 1:
A 52-year-old male with known heart failure and an ejection fraction of 10% presents with severe shortness of breath and respiratory distress.

Case 2:
A 65-year-old female presents with fatigue, chest pain, and shortness of breath. She is hypotensive and hypoxemia. She has an elevated WBC and lactate, and CXR shows multiple infiltrates. The clinician is treating her for presumed sepsis, but she is worsening with IV fluids.

Questions for Learners:

1. What is a systematic approach to shock?
2. What are the etiologies of cardiogenic shock?
3. What are the ways patients present with cardiogenic shock?
4. What is the utility of labs and imaging?
5. What is the ED-focused management?

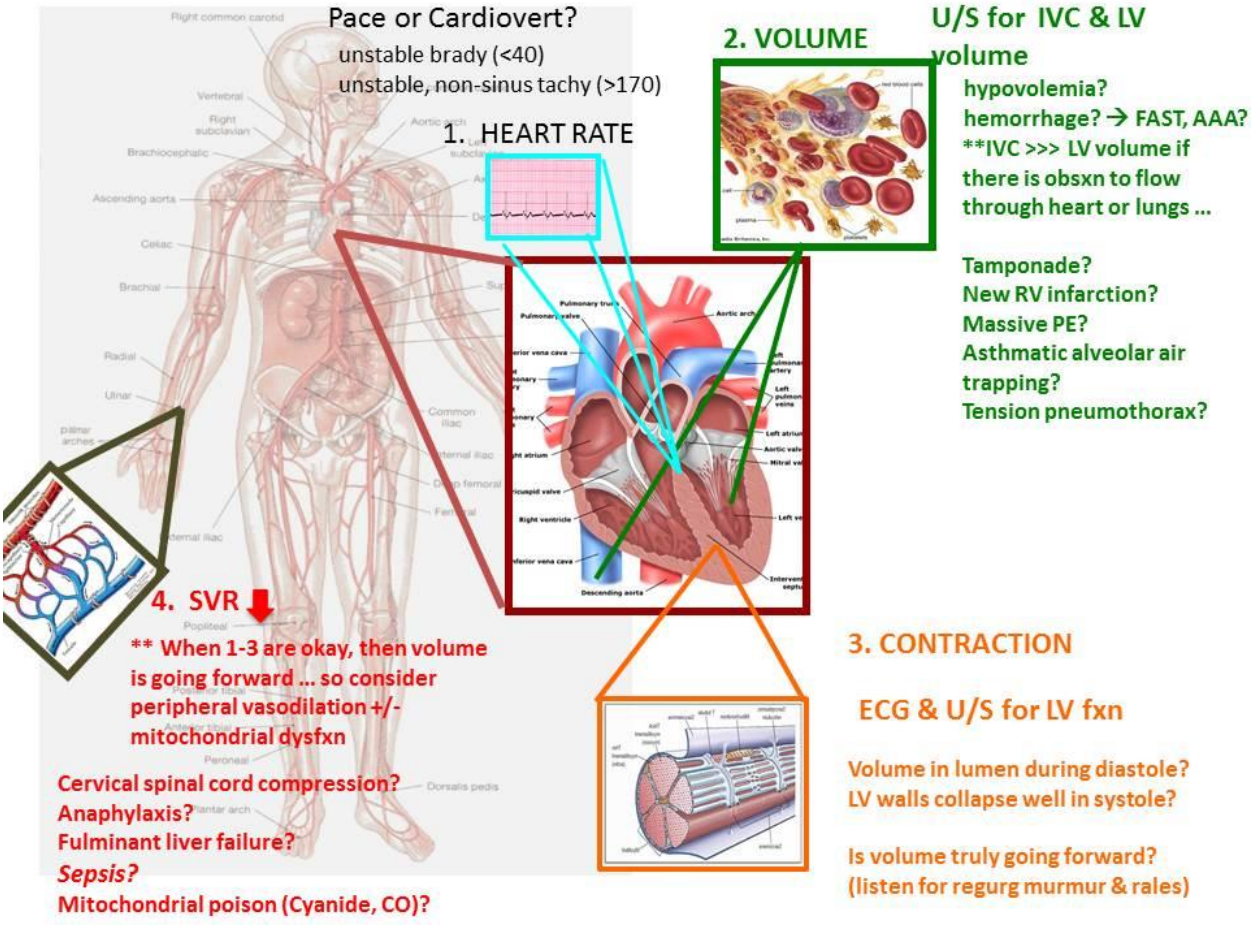
Suggested Resources:

- Articles
  - IBCC – Shock
  - IBCC – CHF
  - emDOCs – Diagnosing Cardiogenic Shock
  - Emergency Medicine Cases
- Journal Articles
  - American Journal of EM – Identifying cardiogenic shock in the emergency department
  - Cardiology Clinics

Answers for Learners:

1. What is a systematic approach to shock?

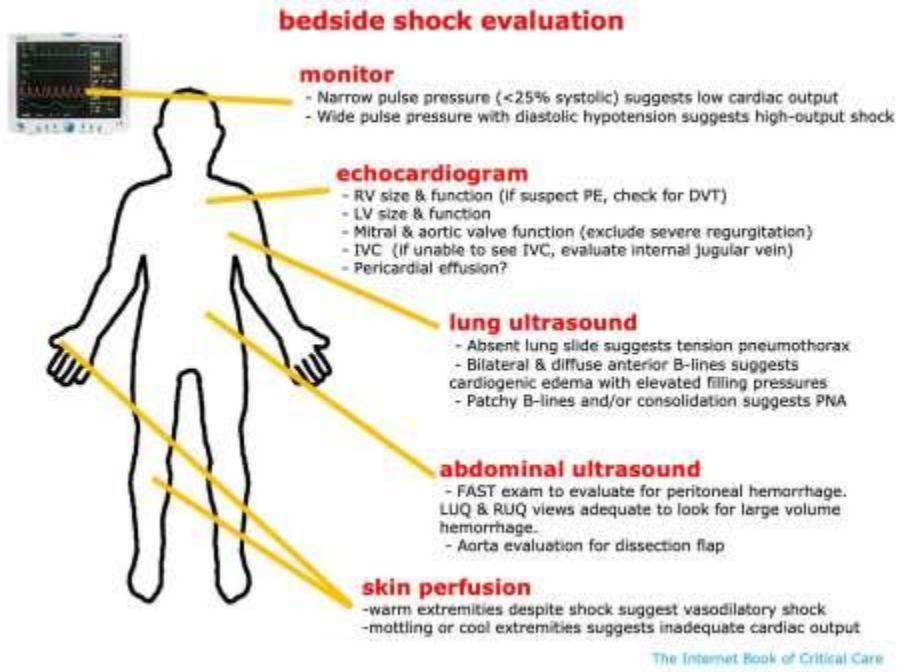
emDOCs primer https://www.emdocs.net/hypotensive-ed-patient-sequential-systematic-approach/



history and data review

- ? Cardiac history (Especially any prior information about cardiac structure/function such as EKG, echo, or even chest CT showing chamber size).
- ? Adrenal disease (Noting: Patients chronically on oral steroid may be assumed to be insufficient).
- ? History of venous thromboembolic disease.
- ? Immunosuppression, ? Invasive devices (e.g. hemodialysis catheters).
- ? Recent procedures or trauma.
- ? Current medications & *changes* in medication list.

examination



labs

- Electrolytes (including Ca/Mg/Phos).
- Complete blood count with differential.
- Coagulation studies.
- CRP (C-reactive protein).
- Lactate.
- If septic shock is suspected:
    - Blood cultures x2.
    - Urinalysis with reflex culture.
    - Sputum culture if clinically indicated.
    - Procalcitonin (if initiating antibiotics).
- Endocrine evaluation:
    - Random cortisol level (if adrenal insufficiency is possible).
    - TSH (if thyroid storm suspected).
- Troponin (if EKG/history suggest acute MI).

radiologic studies

- EKG is occasionally helpful (e.g., may reveal occlusive MI, or RV strain).
- CXR (e.g., may reveal pneumonia, or cardiogenic edema implying LV failure).
- CT may be considered depending on the clinical scenario:
    - CTA to evaluate for pulmonary embolism.
    - CT abdomen/pelvis to evaluate for septic focus.

differential & categorization

Findings on ultrasonography and physical examination may be integrated as shown below. This tends to work best in previously-healthy patients with a single mechanism of shock. Patients with multiple chronic problems or multifactorial shock may defy categorization.

bedside approach to undifferentiated shock/hypotension

	IVC or Jugular vein size	Lung POCUS	Significant pericardial effusion	RV dilation	LVEF	Mitral or aortic regurg?	Cardiac Output
Distributive shock - Septic shock* - Anaphylaxis* - Adrenal crisis*, Thyroid storm - Post-cardiac arrest SIRS - Pancreatitis, Hepatic failure - Neurogenic (trauma, spinal anesth) - Vasodilatory medications	↓↓ or normal	Normal (or focal abnormality from pneumonia)	-	-	nl / ↑	-	**High output:** - Warm extremities. - Low diastolic Bp. - Wide pulse pressure. - May be febrile. - May look toxic. - Capillary refill is variable.
Hypovolemic shock - Vomiting, diarrhea, overdiuresis - Hemorrhage (GI, peritoneal, RP) **Abdominal compartm. syndrome**	↓↓↓	Normal	-	-	nl / ↑	-	**Low output** - Cool extremities. - Diastolic Bp may be normal. - Narrow pulse pressure. - Delayed capillary refill.
RV failure - PE, - Decompensated chronic PH - RV myocardial infarction	↑↑	Normal	-	+	nl / ↑	-	
Tamponade	↑	Normal	+	-	nl / ↑	-	
Tension pneumothorax	↑	No slide on affected hemithorax	-	-	nl / ↑	-	
AutoPEEP / high airway pressure - Asthma > COPD > ARDS - Exacerbated by hypovolemia - Dx based on history, vent waves	nl/↑	Normal	-	+/-	nl / ↑	-	
LV failure - MI - Myocarditis, postpartum CM - Takotsubo cardiomyopathy - Beta-blocker overdose	nl/↑	B-lines everywhere	-	+/-	↓↓	+/-	
Valve dysfunction - Endocarditis - Post-MI papillary muscle rupture - Prosthetic valve dysfunction **LV outflow obstruction (LVOTO)** - Elderly with chronic HTN - HOCM or Takotsubo CM	nl/↑	B-lines everywhere	-	-	nl / ↑	+	

POCUS can generally diagnose RV failure, tamponade, LV failure, or valve dysfunction. Unfortunately, hypovolemic and distributive shock often look relatively unremarkable on POCUS (both may present with a collapsed IVC). Thus, additional clues about the cardiac output will often be needed to sort out these etiologies. Unfortunately, in the later stages of shock, these distinctions may tend to blend together. For example, low cardiac output may eventually lead to tissue damage that causes inflammation, causing vasodilation. Alternatively, prolonged septic shock often leads to a stress cardiomyopathy with a fall in cardiac output. Thus, archetypal distinctions are most accurate in sorting out patients who present relatively *early* in their disease course.

*Anaphylaxis can kill within minutes, if suspected then start epinephrine *without* delay.

Adrenal crisis: If suspected, immediately start empiric steroid. Dexamethasone 6 mg may be useful since this doesn't interfere with performing an ACTH stimulation test later on.

Septic shock is the most common cause of shock in critically ill adults. If a reasonably through evaluation fails to reveal any obvious cause of the shock (e.g., cardiopulmonary POCUS is unremarkable and the history is nonspecific), septic shock is likely. When in doubt, treat empirically for sepsis.

The Internet Book of Critical Care, emcrit.org/IBCC/shock

2. What are the etiologies of cardiogenic shock?

Most studies of CS focus on patients with CS secondary to myocardial infarctions (MIs) involving the left ventricle. Although MIs are the primary cause of CS (~70%), any cause of ventricular dysfunction and reduced CO or cardiac index (CO/body surface area) as a potential cause must be considered.6 This includes, but is not limited to, nonischemic causes of right heart failure, myocarditis, takotsubo cardiomyopathy, hypertrophic cardiomyopathy, or valvular heart disease. To make things more challenging, CS is a continuum rather than a static state, ranging from worsening heart failure to refractory shock with irreversible end organ damage. CS becomes even more variable with the occurrence of secondary insults such as arrhythmias or progressive ischemia and acidosis.3 It should be noted that in 2/3 of cases, CS is not present on admission but later develops within 48 hours of hospitalization as the patient progresses down the continuum of shock. It is important to frequently reevaluate patients' vital signs, symptoms, physical exam, and bedside echo.

CAUSES OF CARDIOGENIC SHOCK
Acute MI: • 79% LVF • 7% acute MR • 4% VSD • 3% isolated RVF • 2% tamponade or cardiac rupture • 7% other
Other: • Left ventricular outflow tract or filling obstruction • Severe myocardial depression secondary to septic shock • Myocardial contusion • Myocarditis • Cardiomyopathy • Acute chordal rupture with MR • Acute aortic insufficiency • Prolonged cardiopulmonary bypass

Think of the causes of cardiogenic shock in 4 categories (keeping in mind that #1 and #2 require emergent mechanical repair):

1. Acute coronary syndromes
2. Mechanical (ie. severe aortic stenosis, endocarditis, ruptured valve, free wall rupture)
3. Myocarditis
4. Progressive non-ischemic chronic heart failure

3. What are the ways patients present with cardiogenic shock?

Classically, patients with CS present with complaints of dyspnea, chest pain, fatigue, and/or ankle swelling. Physical exam may reveal signs of congestion including peripheral edema, jugular venous distension (JVD), crackles/rales on auscultation, and signs of hypoperfusion such as cool, poorly perfused extremities (Table 2). In a small retrospective review of 30 patients in undifferentiated shock, those with CS were more likely to have JVD (80% compared to 0% and 20%), cold skin (57.1% compared to 14.3% and. 28.5%), and pulmonary rales (75% vs 16.7% and 8.3%) compared to patients with distributive and hypovolemic shock, respectively. In another prospective study with 68 patients, residents used specific clinical exam findings to differentiate categories of shock. CS was categorized by SBP less than 90, signs of low output (cold hands, poor capillary refill, and weak pulse), elevated jugular venous pressure (JVP)> 7 cmH2O, S3 gallop, and crackles to 1/3 of the lungs. Of 68 patients, 11 met criteria for CS. In patients with echocardiographic evidence of low cardiac output, elevated JVP predicted CS with an accuracy of 80%, which was unchanged when adding the presence of crackles.

Physical exam findings associated with cardiogenic shock
Congestion: • JVD • Rales • Peripheral edema/ascites • Hepatosplenomegaly • Orthopnea • Abdominal jugular reflex
Low perfusion: • Cold extremities • Hypotension • Tachycardia • Tachypnea • Narrowed pulse pressure
Other: • Ventricular gallop (S3) • Displaced PMI

Although JVP is a useful proxy for elevated wedge pressures, it may be difficult to assess due to body habitus and positioning of the patient (head of the bed should be elevated 45 degrees which can be difficult in patients with severe orthopnea). JVP is measured by calculating the highest pulsation point in cm above the sternal angle and then add 5 (as the right atrium is 5 cm below the sternal angle), which correlates to distension in cmH20 (Figure 2). Elevated values are often considered greater than 6-8 cmH20. Of note, elevated JVP is associated with increased risk of mortality, with a relative risk (RR) of 1.52.

Labs may show a metabolic acidosis (as lactate increases due to peripheral ischemia), renal hypoperfusion with resulting acute kidney injury, and possible evidence of cardiac ischemia with elevated troponin and EKG changes. In the CardShock study, a multicenter, prospective, observational study of 219 CS patients, lactate levels were significantly associated with increased mortality (adjusted odds ratio of 1.4). It is important to note that lactate elevation is not specific to sepsis and can be seen in any hypoperfused state such as CS.

On the other hand, these physical exam findings and hemodynamic parameters do not always hold true. In a study using the SHOCK Trial registry, 5.2% of CS patients did not have overt hypotension although did have signs of peripheral hypoperfusion and low CI. This is likely due to an adaptive catecholamine release in early CS, which increases systemic vascular resistance (SVR) and transiently maintains blood pressure, though generally with a narrow pulse pressure. Even patients with clinically significant pulmonary edema on imaging can present with wheezing or even clear lung sounds rather than rales. In one study, pulmonary congestion was only seen in approximately 2/3 cases of CS secondary to MI.21 Furthermore, even with decreased LV contractility, CS patients may not have a severe reduction in LVEF. In fact, the mean EF in a cohort of CS patients is about 30%, which is reduced but higher than expected.

Though the exam is not perfect, a detailed physical exam looking for signs of congestion and peripheral hypoperfusion along with a careful review of vital signs and labs may be the first hint your patient has cardiogenic shock.

4. What is the utility of labs and imaging?

cardiac imaging:

- EKG.
- Echo.

labs:

- CBC, Electrolytes including Ca/Mg/Phos (if hypocalcemia suspected check ionized calcium).
- 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).

5. What is the ED-focused management?

1. Optimize **oxygenation** with **NIPPV**

Maintaining adequate tissue oxygenation is critical in patients with heart failure and cardiogenic shock, which is usually ideally achieved with NIPPV. It has the added benefit of decreasing preload and afterload. (see **Part 1 for oxygenation strategies in heart failure**)

Clinical pearl: avoid endotracheal intubation whenever possible in the patient in cardiogenic shock as removal of respiratory drive may lead to cardiovascular collapse

Clinical Pitfall: overshooting positive pressure ventilation in the patient with RV failure; positive pressure ventilation can potentially increase RV afterload and therefore should be used with caution in patients with cardiogenic shock resulting from acute RV failure

2. Optimize **blood pressure** with **vasopressors** (eg. norepinephrine) to maintain cardiac/end-organ perfusion targeting a MAP of 65-80
3. Optimize **contractility** with **ionotropes** (eg. dobutamine, milrinone)

1.Optimizing blood pressure with norepinephrine +/- vasopressin: target a MAP of 65-80. This is required to augment end-organ/coronary perfusion. The preferred first line agent is norepinephrine. Vasopressin may be added as a second line agent. While epinephrine and norepinephrine both have been shown to improve MAP and cardiac indices, norepinephrine has a lower incidence of refractory shock compared to epinephrine.

2.Optimizing contractility with dobutamine or milrinone: while dobutamine is a Beta 1 and 2 agonist and milrinone is a phosphodiesterase 3 inhibitor, both agents are inotropes and vasodilators. A recent RCT showed no significant difference in in-hospital survival and major cardiac outcomes with dobutamine versus milrinone in patients in cardiogenic shock.

Our experts recommend starting with dobutamine as it is a shorter acting drug and can be titrated more easily compared to milrinone. However, for patients taking long-acting beta-blockers, milrinone may be the better first option as it works on a different receptor.

> ***Clinical pitfall:*** *giving an ionotrope before initiating a vasopressor may decrease BP further as they are vasodilators, which may lead to cardiovascular collapse; our experts suggest initiating norepinephrine prior to giving an ionotrope in heart failure patients with cardiogenic shock*

4. Optimize **volume status** (crystalloid or diuretics)

Based on clinical and PoCUS assessment of intravascular volume, patients may require gentle and cautious crystalloid administration or diuresis with ongoing assessment of volume status.

> ***Practical pearl:*** *it is imperative to consult cardiology/CV surgery early in the resuscitation of patients with cardiogenic shock as there may be a need for emergent mechanical interventions*