

Cardiogenic Shock and Pulmonary Edema

Chapter 316 | Part 8: Critical Care Medicine

KEY CLINICAL POINTS

- 1 Cardiogenic shock (CS) is a low cardiac output state resulting in life-threatening end-organ hypoperfusion and hypoxia.
- 2 SCAI classification stages: A (At risk), B (Beginning), C (Classical), D (Deteriorating or 'doom'), and E (Extremis).
- 3 Norepinephrine is the preferred first-line vasopressor over dopamine or epinephrine based on randomized trials.
- 4 Early revascularization (PCI/CABG) is the only evidence-based treatment strategy for mortality reduction in CS due to MI.
- 5 Culprit-only PCI is preferred in multi-vessel disease to reduce mortality and renal replacement therapy.
- 6 Lactate level >2 mmol/L defines shock; levels >8 mmol/L indicate SCAI stage E.
- 7 Echocardiogram is essential for identifying etiology, mechanical complications (VSR, MR), and RV involvement.
- 8 In isolated RV failure, optimize right atrial pressure to 10–15 mmHg and avoid excess fluids.
- 9 VA-ECMO showed higher complications and no survival benefit compared to control in the ECLS-SHOCK trial.
- 10 Hemoglobin transfusion target is >7.0 g/dL; glucose should be managed to ≤ 180 mg/dL (10.0 mmol/L).

DEFINITION & OVERVIEW

- **Definition (Harrison's 22e):** *Cardiogenic shock (CS) is a low cardiac output state resulting in life-threatening end-organ hypoperfusion and hypoxia.*
- **Clinical Presentation:** Characterized by persistent hypotension, often with an associated lactate >2 mmol/L. Objective parameters like cardiac index or pulmonary capillary wedge pressure can confirm the cause but are not mandatory.
- **SCAI Classification (Figure 316-2):**
 - Stage A: At risk (impending progression)
 - Stage B: Beginning (relative hypotension or tachycardia without hypoperfusion)
 - Stage C: Classical (standard clinical criteria for CS)
 - Stage D: Deteriorating or 'doom' (signals of deterioration)
 - Stage E: Extremis (cardiac arrest, requiring CPR or ECMO)

EPIDEMIOLOGY

- **Incidence:** 5–10% of cases complicating acute MI; incidence has decreased due to early mechanical reperfusion.
 - **Primary Cause:** LV failure accounts for ~80% of CS cases in MI.
 - **Other Causes:** Acute severe MR, VSR, RV failure, free wall rupture, or tamponade. Takotsubo syndrome is a recognized uncommon cause of transient CS.
 - **Risk Factors:** Older age, prior MI, DM, anterior MI location, and multivessel CAD with extensive stenosis.
 - **Timing:** 25% present on admission; 25% develop within 6 hours; 25% develop later on the first day.
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ETIOLOGY & PATHOPHYSIOLOGY

- **Primary Myocardial Failure (Table 316-1):**
 - Acute MI (most frequent)
 - Mechanical complications: VSR, papillary muscle/chordal rupture (severe MR), free wall rupture.
 - Other: Takotsubo syndrome, myocarditis, cardiomyopathy, valvular disease (Aortic/Mitral stenosis or regurgitation).
 - **Pathophysiology Cascade (Figure 316-2):**
 - The "Vicious Cycle": $\downarrow$ Contractility $\rightarrow$ $\downarrow$ Cardiac Output $\rightarrow$ $\downarrow$ BP $\rightarrow$ $\uparrow$ Myocardial Ischemia $\rightarrow$ further $\downarrow$ Contractility.
 - **Systemic Response:** SIRS triggered by cardiac injury leads to inflammatory cytokines and NO production, causing pathologically induced vasodilation and potential cardiotoxicity (peroxynitrite).
 - **Compounding Factors:** Lactic acidosis and hypoxemia reduce the efficacy of catecholamines; high-volume transfusions may trigger inflammation.
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CLINICAL FEATURES

- **General Presentation:** Dyspnea, pallor, apprehension, diaphoresis, altered mental status. Pulse is typically weak and rapid or severely bradycardic.
- **Physical Exam:** Tachypnea, JVD, weak apical pulse, soft S1, S3 gallop, crackles (in LV failure).
- **Electrocardiogram:** Q waves/ST elevation in multiple leads or LBBB; ST elevation in aVR and multi-lead depression suggest severe left main stenosis.
- **Chest Roentgenogram:** Pulmonary vascular congestion/edema (present in most); heart size may be normal (first MI) or enlarged (previous MI).
- **Laboratory Findings:**
 - WBC & CRP: Typically elevated.
 - Renal Function: Progressively impaired; Cystatin C/NGAL do not add prognostic value over creatinine.
 - Hepatic Transaminases: Elevated in ~20% (marker of high mortality).
 - Lactate: >2 mmol/L (shock); >8 mmol/L (SCAI stage E).
 - Glucose: Often elevated; strong independent predictor for mortality.
 - Cardiac Markers: CK, MB, Troponin I/T markedly elevated in acute MI.

DIFFERENTIAL DIAGNOSIS

- **Pulmonary Edema Differentiation:**
 - Cardiogenic: High PCW (confirmed by PAC or Echocardiogram).
 - Non-cardiogenic: Sepsis, neurogenic injury, or fluid overload without LV failure.
 - **Diagnostic Clues:**
 - Echocardiography: Identifies systolic/diastolic dysfunction and valvular lesions.
 - ECG: ST elevation/Q waves indicate acute MI (requires immediate reperfusion).
 - BNP: Substantially elevated levels support heart failure as the cause of dyspnea.
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INVESTIGATIONS & DIAGNOSIS

1. **Initial Assessment:** Focused history, physical exam, ECG, Chest X-ray, ABG, Lactate, and blood labs.
 2. **Echocardiogram (Prompt):**
 - Identify etiology (MI, mechanical complications like VSR/MR, or tamponade).
 - Assess LV/RV function and pulmonary infiltration (Table 316-2).
 3. **Hemodynamic Monitoring (PAC):**
 - Recommended for management uncertainty or severe CS unresponsive to initial therapy.
 - Used to confirm: RV involvement, L-to-R shunting, pulmonary artery pressures, transpulmonary gradient, and SVR.
 - Differentiation of non-cardiogenic edema (e.g., identifying low PCW in sepsis).
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MANAGEMENT & TREATMENT

1. **Initial Stabilization:**
 - Ventilation: Required in ~80% for hypoxemia and work of breathing.
 - Glucose Control: Target ≤ 180 mg/dL (10.0 mmol/L).
 - Hemoglobin: Target >7.0 g/dL.
 - Medication Adjustment: Discontinue negative inotropes; treat bradyarrhythmias with pacing.
2. **Reperfusion & Revascularization:**
 - Strategy: Rapid revascularization of the infarct-related artery is the only evidence-based mortality reduction strategy.
 - Choice: PCI (preferred) or CABG if anatomy is not amenable to PCI.
 - Multi-vessel Disease: Culprit-only PCI preferred over immediate multi-vessel PCI (reduces mortality and RRT need).
3. **Vasopressors & Inotropes:**
 - First-line Vasopressor: Norepinephrine (preferred over dopamine/epinephrine due to fewer arrhythmias and lower lactate).
 - Dobutamine: Used for inotropy; note that its vasodilating activity may preclude use if vasoconstriction is needed.
4. **Mechanical Circulatory Support (MCS):**
 - IABP: Standard initial support; used in patients with early revascularization (especially within 30 min).
 - Evidence: VA-ECMO showed higher complications and no survival benefit compared to control in the ECLS-SHOCK trial.

- Selection Logic (Flowchart 1):
- If Not Stabilized → Short-term percutaneous MCS.
- If Severe Neurologic Deficit → Consider long-term surgical MCS.
- If Preservation of LV Function → Bridge to Recovery; else, Bridge to Decision/Destination Therapy.

5. Right Ventricular (RV) Failure Specifics:

- Target: Optimize right atrial pressure to 10–15 mmHg.
- Fluid Management: Avoid excess fluids.

PROGNOSIS & COMPLICATIONS

• Prognostic Markers:

- Lactate: >8 mmol/L indicates high-risk (SCAI Stage E).
- Glucose: Elevated at admission is a strong independent predictor of mortality.
- Hepatic Transaminases: Elevation in ~20% of patients correlates with higher mortality.
- Time to Reperfusion: Outcome benefit correlates strongly with the time between symptom onset and reperfusion.

SPECIAL CONSIDERATIONS

• Right Ventricular Failure:

- Identification: RV involvement, pulmonary artery pressure, and SVR changes (Table 316-3).
- Management: Target RA pressure 10–15 mmHg; avoid excess fluids.

• Mechanical Complications:

- Identify via Echocardiogram: VSR, papillary muscle/chordal rupture, free wall rupture.

KEY PEARLS & CLINICAL TRAPS

• Norepinephrine is the first-line choice for vasopressor therapy in CS.

- Culprit-only PCI is the standard for multi-vessel disease to reduce mortality and RRT.
- Lactate >8 mmol/L identifies patients in 'extremis' (Stage E).
- Echocardiogram is the primary tool to differentiate mechanical complications from simple myocardial failure.
- SCAI Stage D represents a state of "doom" or impending collapse.

FLOWCHARTS

Emergency Management of CS (Flowchart 1)

1. **Initial Assessment:** Identify cause (LV dysfunction, RV dysfunction, or Mechanical complication).
2. **Revascularization Path:**
 - Emergency PCI of culprit lesion → (If not amenable) → Emergency CABG.
3. **Hemodynamic Support Path:**
 - If signs of low blood pressure → Fluid challenge → Invasive blood pressure monitoring → IABP.
4. **Stabilization Check:**
 - Is patient stabilized?

- Yes → Proceed to standard care/less intensive MCS.
- No → Short-term percutaneous MCS in selected/refractory cases.

5. MCS Selection Path (based on specific criteria):

- Assessment of "Preservation of LV function" and "Severe neurologic deficit":
- If severe neurologic deficit → Long-term surgical MCS.
- If viable myocardium → Bridge to Recovery.
- If no recovery expected → Destination Therapy.

TABLES & FIGURES

Table 316-1: Etiologies of Cardiogenic Shock

- **Myocardial Infarction:** LV failure, VSR, papillary muscle/chordal rupture (severe MR), free wall rupture.
- **Other MI Complications:** Post-cardiac arrest, postcardiotomy, arrhythmias, myocarditis.
- **Structural/Other:** Takotsubo syndrome, HCM with outflow obstruction, Aortic dissection, Valvular disease (Aortic/Mitral stenosis or regurgitation).
- **RV Failure Specifics:** Acute MI, cor pulmonale, pericardial tamponade, severe acidosis/hypoxemia.

Table 316-2: Utility of Echocardiogram

- **Ventricular Function:** Identifies predominantly left, right, or biventricular involvement.
- **Hemodynamics:** Evaluates IVC diameter/collapse, estimated pulmonary artery systolic pressure, and estimated left atrial pressure.
- **Pulmonary:** Detects pleural effusion, lung edema, pneumothorax, and pulmonary infiltration.

Table 316-3: Hemodynamic Patterns

- **Normal Values:** RA <6; RVS <25; RVD 0–12; PAS <25; PAD 0–12; PCW 6–12; CI ≥ 2.5; SVR (800–1600).
- **Pulmonary Edema:** ↑ PCW, ↑ SVR, ↓ CI.
- **Cardiac Tamponade:** ↑ RA, ↑ PCW, ↑ SVR, ↓ CI.
- **Ventricular Septal Rupture:** ↑ RA, ↑ PCW, ↑ SVR, ↑ PBF (Pulmonary Blood Flow), ↓ SBF (Systemic Blood Flow).
- **Septic Shock:** ↓ RA, ↓ PCW, ↓ SVR.

REFERENCE TABLES

TABLE 316-1

Etiologies of Cardiogenic Shock a and Cardiogenic Pulmonary Edema Etiologies of Cardiogenic Shock or Pulmonary Edema...

- Etiologies of Cardiogenic Shock or Pulmonary Edema
- Acute myocardial infarction/ischemia
- Left ventricular failure
- Ventricular septal rupture
- Papillary muscle/chordal rupture–severe mitral regurgitation
- Ventricular free wall rupture

- Other conditions complicating large myocardial infarctions
- Excess negative inotropic or vasodilator medications
- Post–cardiac arrest
- Postcardiotomy
- Refractory sustained supraventricular or ventricular tachyarrhythmias
- Refractory sustained bradyarrhythmias
- Acute fulminant myocarditis
- End-stage cardiomyopathy
- Takotsubo syndrome/apical ballooning syndrome
- Hypertrophic cardiomyopathy with severe outflow obstruction
- Aortic dissection with aortic insufficiency or tamponade
- Severe valvular heart disease
- Critical aortic or mitral stenosis
- Acute severe aortic regurgitation or mitral regurgitation
- Toxic/metabolic
- β Blocker or calcium channel antagonist overdose
- Pheochromocytoma
- Scorpion venom
- Hypertensive crisis
- Post–cardiac arrest stunning
- Myocardial depression in setting of septic shock or systemic inflammatory response syndrome
- Myocardial contusion
- Other Etiologies of Cardiogenic Shock^b
- Right ventricular failure due to:
 - Acute myocardial infarction
 - Acute or decompensated chronic cor pulmonale
 - Pericardial tamponade
 - Toxic/metabolic
 - Severe acidosis, severe hypoxemia

TABLE 316-2

Utility of the Echocardiogram in Cardiogenic Shock or Pulmonary Edema

CLINICAL QUESTION	INFORMATION
Ventricular function	Predominantly left, right, or biventricular involvement
Hemodynamics	Volume assessment by inferior vena cava diameter and inspiratory collapse Estimated pulmonary artery systolic pressure Estimated left atrial pressure

CLINICAL QUESTION	INFORMATION
Pulmonary	Pleural effusion Lung edema Pneumothorax Pulmonary infiltration

TABLE 316-3

**Hemodynamic Patterns a Normal values MI without pulmonary edema b Pulmonary edema
Cardiogenic shock**

	RA, mmHg	RVS, mmHg	RVD, mmHg	PAS, mmHg	PAD, mmHg	PCW, mmHg
Normal values	<6	<25	0–12	<25	0–12	<6–12
	—	—	—	—	—	~13 (5–18)
Pulmonary edema	↔↑	↔↑	↔↑	↑	↑	↑
	↔↑ ↑	↔↑ ↓↔↑d	↔↑ ↑	↔↑ ↓↔↑d	↑ ↔↓↑d	↑ ↓↔↑d
Cardiac tamponade	↑	↔↑	↑	↔↑	↔↑	↔↑
	↔↑	↑	↔↑	↑	↑	↑
Ventricular septal rupture	↑	↔↑	↑	↔↑	↔↑	↔↑
	↓	↔↓	↔↓	↓	↓	↓
Septic shock	↓	↔↓	↔↓	↓	↓	↓