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Review Article

Cardiogenic shock: contemporary concepts, hemodynamic targets, and escalation to mechanical circulatory support

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Abstract

Cardiogenic shock (CS) is a syndrome of sustained tissue hypoperfusion caused by primary cardiac pump failure and remains associated with high short-term mortality despite improvements in reperfusion, intensive care, and acute mechanical circulatory support (aMCS). Modern defini-tions emphasize not only hypotension but also clinical and biochemical evidence of inadequate cellular oxygen utilization, frequently driven by macro–microcirculatory uncoupling. This review summarizes contemporary definitions and staging, core pathophysiology, bedside diagnosis and monitoring, pragmatic early hemodynamic targets, evidence-based management of AMI-related CS, and structured escalation from vasoactive drugs to aMCS. A practical focus is placed on the first 24 hours, when achieving adequate flow and tissue perfusion appears prognostically decisive.

Introduction

Cardiogenic shock is a critical state of systemic hypoperfusion due to cardiac pump failure, often complicated by multiorgan dysfunction. Mortality remains substantial, particularly in advanced shock and when multiorgan failure develops.[1] While AMI has historically accounted for most cases, contemporary cohorts include a growing proportion of non-ischemic etiologies (decompensated chronic heart failure, myocarditis, Takotsubo, arrhythmias, and post-cardiotomy shock). Regardless of cause, outcomes depend on rapid recognition, early etiology-directed therapy, and timely escalation when initial resuscitation fails.

Development

Definition and staging

Operational definitions integrate: (i) hypotension or the need for vasopressors/inotropes/aMCS to maintain perfusion; (ii) signs of end-organ hypoperfusion (altered mentation, mottling/cool extremities, oliguria, rising creatinine/transaminases); and (iii) objective evidence of low cardiac output and/or elevated filling pressures.[1, 2] Importantly, arterial pressure alone is insufficient: tissue hypoperfusion may persist despite apparently acceptable MAP, reflecting loss of hemodynamic coherence.[1]

Staging frameworks improve communication and facilitate escalation. The SCAI classification (A–E) ranges from “at risk” (A) to “extremis” (E) and is associated with prognosis. In a pooled cohort with serial hemodynamics, admission staging clustered predominantly in stages C–E, reflecting the ICU population.[2]

Etiology and phenotypes

CS is a syndrome with heterogeneous phenotypes. The dominant hemodynamic pattern may be LV failure (pulmonary congestion, elevated wedge pressure), RV failure (systemic venous congestion, low PAPi), or biventricular failure; a subset evolves a mixed phenotype with vasoplegia and systemic inflammation.[1] Etiologic workup should be parallelized with resuscitation. In practice, a focused bedside echocardiogram at presentation is indispensable (LV/RV function, regional wall motion abnormalities, mechanical complications, severe valvular lesions, pericardial effusion/tamponade).

Pathophysiology: from pump failure to microcirculatory dysfunction

The initial insult reduces stroke volume and cardiac output, activating sympathetic vasoconstriction and neurohormonal pathways. As shock progresses, inflammation, endothelial dysfunction, and microvascular flow heterogeneity impair diffusion and tissue oxygen extraction. A key contemporary concept is the loss of “hemodynamic coherence”: normalization of macrocirculatory variables (e.g., MAP) may not

translate into improved cellular oxygen delivery or utilization.[1] Clinically, persistent hyperlactatemia or elevated venous–arterial CO_2 gaps despite corrected blood pressure suggest ongoing tissue hypoperfusion and should prompt reassessment of flow, phenotype, and escalation strategy.

Initial evaluation and monitoring

Parallel diagnostics and treatment: Early steps include: airway/ventilation as needed; rapid ECG and cardiac biomarkers; lactate and blood gas evaluation; bedside echocardiography; and early coronary angiography when AMI is suspected.[1]

Invasive hemodynamics: When phenotype or response to therapy is unclear, a pulmonary artery catheter can guide decision-making. Beyond absolute pressures and CI, derived indices can provide actionable insight:

Cardiac power output/index (CPO/CPI): a composite of pressure and flow approximating cardiac hydraulic work; lower values are associated with worse outcomes.[2]

PAPi: assists identification of RV failure and need for RV-targeted support.

Tissue perfusion surrogates: $ScvO_2$, venous–arterial CO_2 gap (ΔPCO_2), and $\Delta PCO_2/C(a– v)O_2$ ratio.[2]

Early (first 24 h) hemodynamic targets

In a post hoc analysis of two prospective cohorts with serial assessments at admission, 6, 12, and 24 hours, mean macrocirculatory and tissue perfusion variables during the first 24 hours were associated with 30-day mortality, and clinically relevant thresholds were proposed.[2] While these are not definitive "goal-directed" mandates, they offer pragmatic resuscitation goals: maintain adequate perfusion pressure while ensuring sufficient flow and improving tissue perfusion markers.

Pharmacologic support

Vasoactive therapy is a bridge to definitive treatment and/or aMCS. In general, norepinephrine is preferred as first-line vasopressor to maintain MAP with less arrhythmogenicity than dopamine in shock.[1,3] Inotropes are selected by phenotype and blood pressure:

a. Dobutamine is often used to augment contractility and forward flow, frequently combined with norepinephrine if vasodilation occurs.

b. Milrinone may be useful in RV dysfunction and pulmonary hypertension but can worsen hypotension and accumulates in renal failure; in a randomized trial comparing milrinone vs dobutamine in CS, no significant difference in primary outcomes was observed.[4]

c. Epinephrine is generally reserved for refractory shock due to tachyarrhythmias and metabolic effects, and should not be used when a safer escalation plan is available.

Etiology-directed therapy: AMI complicated by cardiogenic shock

In AMI-CS, early revascularization improves survival.[5] Systems of care should minimize delays to reperfusion, given the steep relationship between time-to-treatment and mortality in shock complicating STEMI.[6] For multivessel disease, an important nuance is strategy: in CULPRIT-SHOCK, immediate multivessel PCI increased early harm compared with culprit-lesion-only PCI, with no long-term survival advantage.[7] Thus, a culprit-only approach is generally preferred in the acute shock phase, with staged PCI considered once stabilized.

Escalation to acute mechanical circulatory support aMCS should be considered when perfusion targets are not achieved despite optimized preload, ventilation/oxygenation, rhythm control, and vasoactive/inotropic therapy, especially in SCAI stages C–E.[1] A structured pathway can reduce therapeutic inertia and prevent excessive catecholamine escalation without a definitive plan.Evidence comparing Impella vs VA-ECMO remains limited by the absence of randomized trials. A propensity score-matched/adjusted meta-analysis reported lower short-term mortality and fewer bleeding events requiring transfusion with Impella compared with VA-ECMO, but the certainty of evidence was low and residual confounding is likely.[8] Consequently, device choice should be individualized, guided by phenotype, contraindications, and institutional expertise, ideally within a multidisciplinary shock team.

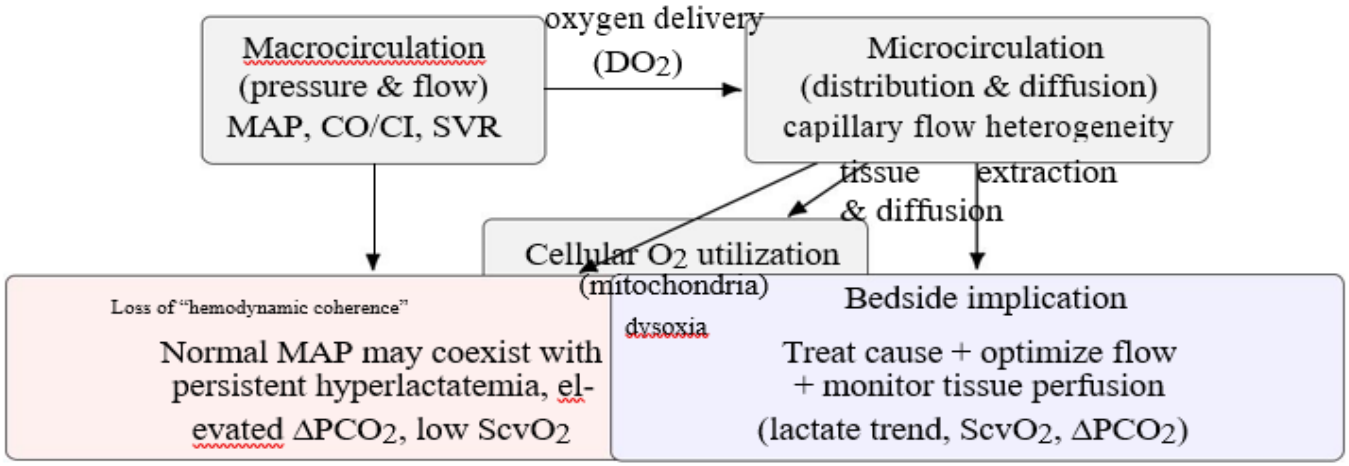

Figure 1 Macro–microcirculatory dissociation in cardiogenic shock (schematic). Restoring blood pressure alone may not normalize tissue perfusion when microvascular flow is heterogeneous or cellular oxygen utilization is impaired.

Table 1 Pragmatic bedside criteria suggesting cardiogenic shock (operational).

Domain	Typical findings (any combination increases likelihood)
Hemodynamics	SBP < 90 mmHg or MAP < 65–70 mmHg or need for vasopressors/inotropes/aMCS to maintain perfusion; narrow pulse pressure; rising filling pressures.
Hypoperfusion	Lactate > 2 mmol/L, oliguria < 0.5 mL/kg/h, mottling/cool extremities, altered consciousness, worsening renal/hepatic indices.
Cardiac disorder / low output	AMI/acute HF/post-cardiotomy; ECG/echo abnormalities; cardiac index often < 2.2 L/min/m^2; low cardiac power output/index.

Table 2 Early (first 24 h) hemodynamic and tissue perfusion targets in cardiogenic shock (pragmatic).

Variable	Threshold associated with worse outcomes
Mean systolic arterial pressure	< 95 mmHg
Mean arterial pressure	< 70 mmHg
Mean cardiac output	< 3.5 L/min
Mean cardiac index	≤ 1.8 L/min/m^2
Mean cardiac power index	< 0.27 W/m^2
Mean $ScvO_2$	< 70%
Mean ΔPCO_2 ($PvCO_2$–$PaCO_2$)	≥ 9 mmHg
Mean $\Delta PCO_2/C(a–v)O_2$ ratio	≥ 1.5 mmHg/mL

Table 3 Common vasoactive agents in cardiogenic shock (bedside summary).

Agent	Primary effect	Practical notes
Norepinephrine	↑ SVR, modest ↑ inotropy	First-line vasopressor; titrate to perfusion targets; monitor arrhythmias/ischemia.
Dobutamine	↑ inotropy, variable ↓ SVR	Useful for low output with adequate MAP; may require concurrent vasopressor.
Milrinone	↑ inotropy/lusitropy, ↓ PVR/SVR	Consider in RV failure/pulmonary HTN; avoid in severe hypotension/renal dysfunction.
Vasopressin	↑ SVR (V1)	Adjunct to reduce norepinephrine dose; minimal direct chronotropy.
Epinephrine	↑ inotropy and SVR	Reserve for refractory shock; higher arrhythmia risk and lactate rise.

Table 4 Acute mechanical circulatory support options in cardiogenic shock (simplified)

Device	Principal physiology	Key considerations
IABP	Modest afterload reduction; coronary perfusion augmentation	Limited CO augmentation; may be used selectively (e.g., ischemia, mechanical complications) as bridge in experienced centers.
Impella (LV unloading)	Direct LV unloading and forward flow	Vascular access, hemolysis; requires adequate RV function/volume; device choice by required flow (CP/5.0/5.5).
VA-ECMO	High-flow circulatory support + oxygenation	Increases LV afterload; may need LV unloading; bleeding, limb ischemia, stroke; useful for profound hypoxemia/biventricular failure.

Discussion

Three themes increasingly shape contemporary CS care. Staging and phenotype drive decisions. CS is not monolithic. SCAI stage, LV/RV dominance, congestion vs hypoperfusion, and vasoplegic/inflammatory components influence drug selection, ventilatory strategy, and device choice.[1] Resuscitation should target perfusion, not only pressure. The first 24 hours appear prognostically decisive. Proposed thresholds for MAP, CI, and CPI should be interpreted alongside tissue perfusion endpoints such as lactate trajectories, $ScvO_2$, and CO_2-derived indices.[2] Persistent abnormalities should prompt reassessment rather than mere escalation of catecholamines. Timely aMCS can prevent catecholamine toxicity and delayed support. Excessive vasoactive requirements reflect inadequate cardiac reserve and contribute to arrhythmia, ischemia, and metabolic stress. Structured pathways that define "failure" of pharmacologic therapy, trigger shock-team consultation, and promote phenotype-guided device selection may improve outcomes, although high-quality randomized evidence is still needed.

Conclusion

Cardiogenic shock is best approached as a syndrome of sustained tissue hypoperfusion due to cardiac pump failure with frequent macro–microcirculatory dissociation. Early diagnosis, etiology-directed therapy (particularly immediate reperfusion in AMI-CS), and resuscitation guided by both macrocirculatory and tissue-perfusion indices are central. Pragmatic targets during the first 24 hours include maintaining MAP near or above 70 mmHg while ensuring adequate forward flow (CI $\gtrsim$1.8 L/min/m^2 and CPI $\gtrsim$0.27 W/m^2) and improving tissue perfusion markers (lactate decline, $ScvO_2$ $\geq$70%, and controlled CO_2 gaps).[2] When targets are not achieved, early escalation to aMCS within a multidisciplinary shock-team model is appropriate, with device selection tailored to phenotype and oxygenation needs.

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Conflicts of interest

Author declares that there is no conflict of interest.

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