



International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

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ARTICLE TITLE	CARDIOGENIC SHOCK: CONTEMPORARY MANAGEMENT, RISK STRATIFICATION AND PHENOTYPING
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DOI	https://doi.org/10.31435/ijitss.3(51).2026.6438
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RECEIVED	30 May 2026
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ACCEPTED	03 September 2026
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PUBLISHED	15 September 2026
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CARDIOGENIC SHOCK: CONTEMPORARY MANAGEMENT, RISK STRATIFICATION AND PHENOTYPING

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ABSTRACT

Background. Cardiogenic shock (CS) is a highly lethal clinical syndrome characterised by inadequate cardiac output and end-organ hypoperfusion. Despite advances in contemporary management, mortality remains unacceptably high. The aetiological spectrum of CS is broadening beyond acute myocardial infarction, and management continues to be challenged by limited and often conflicting evidence.

Aim. To review the current evidence regarding the diagnosis, classification, risk stratification and management of cardiogenic shock, with particular emphasis on clinical phenotyping and the role of mechanical circulatory support.

Material and methods. A narrative literature review was conducted using published studies, randomised trials, observational data, and contemporary international guidelines addressing the diagnosis and management of cardiogenic shock.

Results. Early identification, treatment of the underlying cause and haemodynamic stabilisation with vasoactive agents remain the cornerstones of CS management. While early revascularisation in acute myocardial infarction-related cardiogenic shock has demonstrated mortality benefits, evidence supporting the use of mechanical circulatory support remains largely inconclusive, and the optimal timing, modality and patient selection are uncertain. The heterogeneity of the CS population, together with the lack of universally accepted diagnostic criteria and classification systems, complicates trial design and interpretation and has stimulated interest in phenotype-based approaches.

Conclusions. Cardiogenic shock remains a complex and heterogeneous syndrome associated with high mortality. Improved diagnostic frameworks, refined risk stratification and phenotype-guided management strategies may facilitate more individualised treatment and improve future research and clinical outcomes.

KEYWORDS

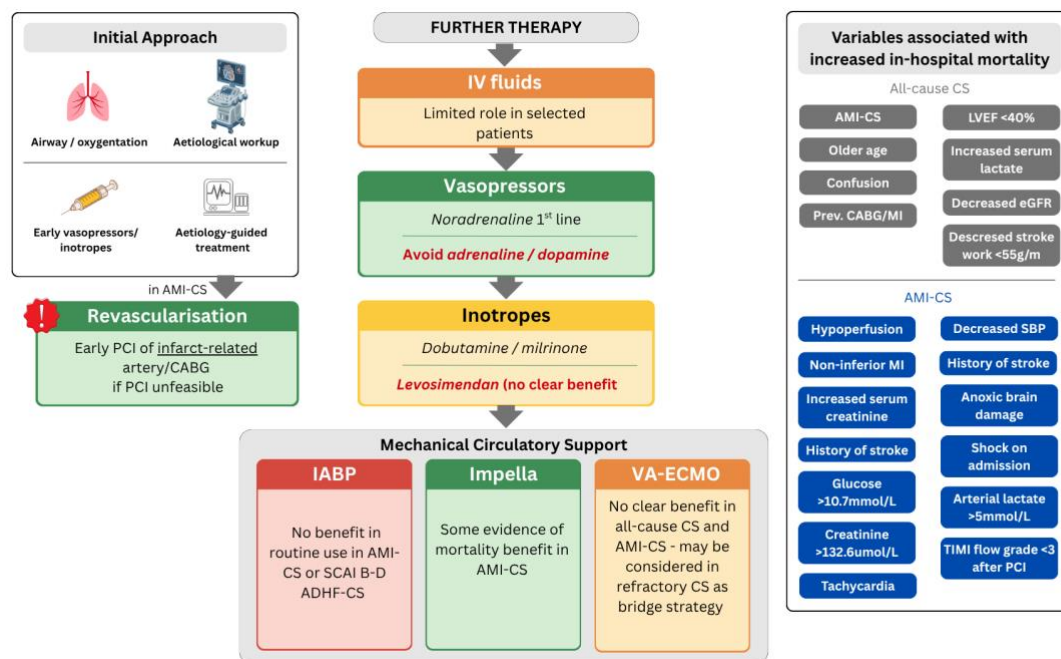
Cardiogenic Shock, Heart Failure, AMI-CS, HF-CS, Extracorporeal Membrane Oxygenation, Mechanical Circulatory Support

CITATION

Rajzer, P., Szczuciński, W., Drelich, K., Polczyk, J., Zając, M., Golińska, Z., Deptuch, W., Moćko, P., Rurkowska, J., & Ogorzałek, F. (2026). Cardiogenic shock: Contemporary management, risk stratification and phenotyping. *International Journal of Innovative Technologies in Social Science*, 3(51). [https://doi.org/10.31435/ijitss.3\(51\).2026.6438](https://doi.org/10.31435/ijitss.3(51).2026.6438)

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Central illustration: Summary of evidence behind cardiogenic shock management strategies and factors associated with increased hospital mortality. AMI-CS - acute myocardial infarction cardiogenic shock; PCI – percutaneous coronary intervention; CABG – coronary artery bypass grafting; IABP – intraaortic balloon pump; ADHF-CS – acute decompensated heart failure cardiogenic shock; LVEF – left ventricular ejection fraction; SBP – systolic blood pressure; VA-ECMO – venoarterial extracorporeal membrane oxygenation.

1. Introduction

Cardiogenic shock (CS) – a state of critical end-organ hypoperfusion due to pump failure – carries an exceptionally high mortality. It complicates approximately 5–10% of acute myocardial infarctions and despite advanced reperfusion and intensive care, short-term mortality remains on the order of 30–50% (Blumer et al., 2024). Given this grim prognosis, cardiogenic shock is a major focus of cardiology guidelines and research.

Progress in CS care has been limited by its marked heterogeneity and inconsistent definitions. Variability in inclusion criteria across studies (e.g. blood pressure thresholds, lactate levels, or requirement for vasoactive support) complicates comparisons and meta-analyses. CS is now recognised as a heterogeneous syndrome arising from multiple aetiologies (Lüsebrink et al., 2024), with recent frameworks such as SCAI staging (Jentzer et al., 2021) highlighting distinct subphenotypes and severity stages. However, the lack of a universally accepted definition continues to hinder the development of clear treatment algorithms.

Randomised trials of mechanical circulatory support (MCS) have yielded inconsistent results, with no clear consensus on their impact on outcomes. While some strategies have shown potential benefit (Møller et al., 2024), others have failed to demonstrate survival advantage (Thiele et al., 2023; Thiele et al., 2013), highlighting significant uncertainty in the role of MCS in CS. Key challenges remain regarding optimal patient selection, timing of device initiation, and the influence of haemodynamic phenotype and shock severity on treatment response.

There is therefore increasing interest in phenotype- and biomarker-guided approaches. Emerging data suggest distinct CS phenotypes (e.g. inflammatory or cardiorenal) may inform tailored management (Zweck et al., 2021). Early recognition, risk stratification (e.g. CardShock or IABP-SHOCK II scores (Harjola et al., 2015; Pöss et al., 2017)), and protocolised care pathways have been proposed to improve outcomes. This review aims to highlight the current evidence behind CS management and outline ongoing research into clinical phenotyping and risk stratification.

1.1 Definition of Cardiogenic SHOCK and Diagnostic Criteria

Cardiogenic shock has been broadly defined as an ineffective cardiac output resulting from primary cardiac dysfunction, with evidence of organ hypoperfusion (Diepen et al., 2017; McDonagh et al., 2021). Unfortunately, variations exist in diagnostic criteria across clinical trials, registries and institutions, leading to challenges in comparing data and establishing clear, universal clinical pathways. Fig. 1 outlines several proposed criteria, each with its strengths and limitations. A recent shift in diagnostic criteria and guidelines reflects the variety of clinical presentations of patients with CS, encompassing those with normotension in addition to the usual, persistently hypotensive patient. Since isolated hypoperfusion has been suggested to carry a higher mortality risk than isolated hypotension, it is crucial to recognise this CS subtype in clinical practice (Jentzer et al., 2021). Some criteria outline objective haemodynamic parameters which, albeit helpful in confirming the diagnosis and ruling out alternative causes of shock, may not be practicable to obtain in some institutions and settings. Some argue that assessment of cardiac output should, however, form an integral part of CS workup and diagnostic criteria, quoting BP as a poor surrogate of cardiac output, the widespread availability of ultrasound to aid non-invasive haemodynamic assessment and the objective of mechanical circulatory support being the restoration of CO (sometimes not associated with restoration of BP) (Ostadal & Belohlavek, 2024).

Definitions of cardiogenic shock – key criteria comparison					
Definition	 Hypotension	 Hypoperfusion	 Congestion	 Haemodynamics	 Normotensive shock included?
SARC (2023)	✓ SBP <90 mmHg for ≥30 min or need for vasopressors/inotropes to maintain SBP >90 mmHg	✓ Altered mental status • Cold, clammy extremities • Urine output <30 mL/h • Lactate >2 mmol/L • Creatinine ≥2× ULN / urine output <0.5 mL/kg/h • Acute liver injury (ALT >3× ULN)	✓ Clinical or imaging evidence of elevated filling pressures (e.g. pulmonary congestion)	✓ Haemodynamic definition (optional): • CI ≤2.2 L/(min·m ²) • SVR >2200 dynes/(cm·sec ⁻⁴)	✓ Yes SBP ≥90 mmHg without the need for vasopressors, inotropes or MCS + evidence of hypoperfusion (alternative causes or ↑ hypoperfusion markers excluded)
KAMIR-NIH (2018)	✓ SBP <90 mmHg for >30 min or need for supportive management to maintain SBP >90 mmHg	✓ At least 1 of the following: • Cool extremities • Decreased urine output • Increased lactic acid level • Altered mental status	✓ Clinical signs of pulmonary congestion	✗ Not included	✗ No
ESC (2021)	✗ Not required (no SBP threshold)	✓ • Cold, sweaty extremities • Oliguria • Altered mental state • Dizziness • Narrow pulse pressure • Raised lactate • Raised creatinine • Narrowed pulse pressure	✗ Not required	✗ Not required	✓ Yes No SBP threshold (normotensive shock included)
EHS-PCI (2012)	✓ SBP <90 mmHg for >30 min or need for supportive intervention to maintain SBP >90 mmHg	✓ Evidence of end-organ damage (altered mental status / oliguria / raised lactate / increased creatinine / metabolic acidosis)	✗ Not required	✓ Increased filling pressures (e.g. PCWP >15 mmHg)	✗ No
IABP-SHOCK II (2012)	✓ SBP <90 mmHg for ≥30 min or need for supportive measures to maintain SBP >90 mmHg	✓ Evidence of hypoperfusion: • Altered mental status • Cold, clammy extremities • Urine output <30 mL/h • Lactate >2 mmol/L	✗ Not required	✗ Not required	✗ No
SHOCK trial (1999)	✓ SBP <90 mmHg for ≥30 min or need for supportive measures to maintain SBP >90 mmHg	✓ Evidence of end-organ hypoperfusion: • Cool extremities • Urine output <30 mL/h • Heart rate ≥60 bpm	✗ Not required (not part of definition)	✓ Required • CI ≤2.2 L/min/m ² • PCWP >15 mmHg	✗ No

SBP – systolic blood pressure; CI – cardiac index; PCWP – pulmonary capillary wedge pressure; SVR – systemic vascular resistance;
ULN – upper limit of normal; MCS – mechanical circulatory support

Fig. 1. Key diagnostic criteria for cardiogenic shock from clinical trials and guidelines (Bauer et al., 2012; Lee et al., 2018; McDonagh et al., 2021; Thiele et al., 2012; Waksman et al., 2023). CS – cardiogenic shock; SBP – systolic blood pressure; MCS – mechanical circulatory support; ALT – alanine aminotransferase; CI – cardiac index; SVR – systemic vascular resistance; PCWP – pulmonary capillary wedge pressure.

1.2 Classification of CS

The high degree of heterogeneity of patients with cardiogenic shock calls for a classification system both for clinical and research applications. CS can be categorized based on its primary aetiology, severity, and hemodynamic profile. Each subtype necessitates personalized management and presents its own unique risk profile. Other pathophysiological factors like the type of ventricular failure (LV, RV, BiV), acuity of CS onset and dynamic features of the presentation should also be taken into consideration (Jain et al., 2021; Yerasi et al., 2022).

The range of CS aetiologies is broad, ranging from ischaemia, decompensated or de novo heart failure and post-cardiotomy CS to non-myocardial aetiologies like valvular disease, pericardial pathology or arrhythmias. CS classification has been outlined in Fig. 2.

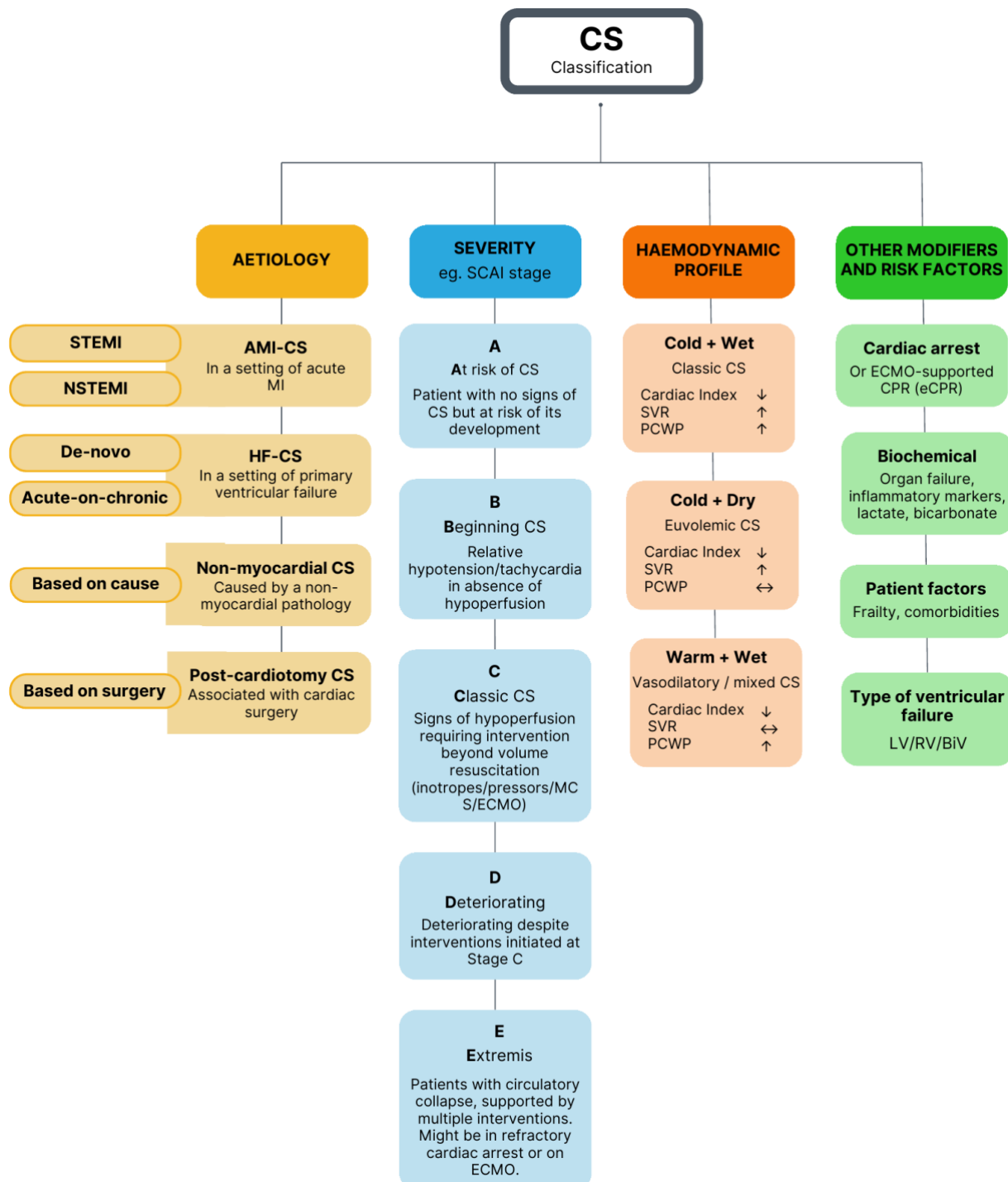


Fig. 2. Classification of cardiogenic shock based on aetiology, severity and haemodynamic profile (Baran et al., 2019; Lüsebrink et al., 2024; Waksman et al., 2023). CS – cardiogenic shock; STEMI – ST-elevation myocardial infarction; NSTEMI – non-ST-elevation myocardial infarction; AMI-CS – acute myocardial infarction cardiogenic shock; HF-CS – heart failure cardiogenic shock; MCS – mechanical circulatory support; ECMO – extracorporeal membrane oxygenation; SVR – systemic vascular resistance; PCWP – pulmonary capillary wedge pressure; CPR – cardiopulmonary resuscitation; LV – left ventricle; RV – right ventricle; BiV – biventricular.

2. Management Strategies in Cardiogenic SHOCK

2.1 Initial Investigations and Stabilisation

Initial management of cardiogenic shock (CS) focuses on rapid haemodynamic stabilisation and identification of the underlying aetiology. Early ECG, arterial blood gas analysis, and bedside echocardiography are central to diagnosis, risk stratification, and treatment selection, with point-of-care echocardiography playing a key role in defining shock phenotype, ventricular function, filling pressures, and mechanical complications. In patients requiring invasive mechanical ventilation, lung-protective ventilation strategies using lower tidal volumes are associated with a reduced incidence of right ventricular failure (Price et al., 2010).

Fluid management remains challenging and should be individualised according to the patient's haemodynamic profile. While a limited fluid challenge may be appropriate in selected patients without evidence of congestion, excessive volume administration can worsen pulmonary oedema and impair cardiac performance. Invasive haemodynamic monitoring and dynamic assessments of fluid responsiveness may help guide therapy where available (Srinivasan & Kundu, 2024). This is particularly relevant in right ventricular-predominant shock, where excessive preload augmentation may be detrimental, especially in the presence of elevated right atrial pressures (Berisha et al., 1990; Ferrario et al., 1994; SINIORAKIS et al., 1994). Consequently, contemporary management emphasises early phenotypic assessment and haemodynamically guided fluid administration to optimise organ perfusion while avoiding volume overload.

2.2 Revascularisation in AMI-CS

The 1999 SHOCK trial (detailed descriptions of trials discussed can be found in Supplementary Table 1) emphasised the need for early revascularisation in AMI-CS (Hochman et al., 1999). The trial randomised 302 patients to either receive emergency revascularisation (coronary artery bypass graft (CABG) or percutaneous coronary intervention (PCI)) or undergo initial medical stabilisation. Although the primary endpoint (30-day, all-cause mortality) was not met, mortality at 6 months was significantly lower in patients receiving early revascularisation. This benefit was seen during 1-year, 3-year and 6-year follow-up (HR of death for revascularisation vs medical stabilisation 0.74; 95% CI 0.57-0.97; $p = 0.03$) (Hochman et al., 2006; Hochman et al., 1999). Data from a Swiss AMIS registry including 23696 patients also supports this survival benefit (Jeger et al., 2008). Survival rates were similar in patients in the SHOCK trial who underwent PCI or CABG (White et al., 2005).

Multivessel disease is prevalent in patients with AMI related cardiogenic shock, representing around 80% of patients (Peters et al., 2023; Tamis-Holland et al., 2022; Thiele et al., 2017). CULPRIT-SHOCK was a landmark, 2017 trial which tackled the clinical dilemma of whether to pursue multivessel PCI in patients with cardiogenic shock, or whether infarct-related vessel revascularisation favours survival in such patients. Indeed, it showed the superiority of culprit-vessel PCI over multivessel PCI in terms of mortality, translating into revised international guidelines (Thiele et al., 2017). As such, the European Society of Cardiology (ESC) recommends immediate PCI of the infarct-related artery in AMI-CS, and CABG if PCI is unfeasible or unsuccessful (Class I, Level B) (Byrne et al., 2023). US-based guidelines echo this approach, recommending emergency, culprit-vessel revascularisation via PCI or CABG irrespective of time from symptoms onset (Level 1, Class B-R) (Rao et al., 2025). PCI of the noninfarct-related artery is ill-advised (Class 3, Level B-R). Finally, the ESC make a Class IIa, Level C recommendation to consider fibrinolysis in STEMI patients developing CS, if primary PCI is unavailable within 120 minutes of diagnosis, once mechanical complications of MI have been ruled out.

2.3 Vasoactive Pharmacotherapy in CS Management

Vasoactive agents remain central to the management of cardiogenic shock (CS), with most patients requiring a combination of vasopressors and inotropes to restore perfusion and maintain adequate cardiac output (Berg et al., 2019; Heidenreich et al., 2022; McDonagh et al., 2021). Drug selection should be guided by the underlying haemodynamic profile, with the primary goal of achieving adequate organ perfusion while minimising adverse effects.

Noradrenaline is recommended as the first-line vasopressor in CS. It effectively increases mean arterial pressure with a relatively favourable safety profile compared with other agents (Ibanez et al., 2018; Levy et al., 2015; McDonagh et al., 2021; Werdan et al., 2021). Evidence from randomised and observational studies has consistently shown worse outcomes with adrenaline, including higher rates of refractory shock and metabolic complications (Bougouin et al., 2022; Léopold et al., 2018; Levy et al., 2018; Tarvasmäki et al., 2016). Dopamine has also been associated with increased arrhythmias and mortality and is no longer recommended in this setting (De Backer et al., 2010).

Inotropes are particularly useful in persistent hypoperfusion despite adequate mean arterial pressure (MAP), although they increase myocardial oxygen demand due to tachycardia (Laghlam et al., 2024). While a MAP >65 mmHg is commonly targeted, evidence for benefits of higher MAPs is mixed (Ameloot et al., 2020; Du et al., 2016; Levy et al., 2015; Parlow et al., 2021; Perez et al., 2014). Dobutamine and milrinone are also commonly used agents, with the DOREMI trial demonstrating no significant difference in clinical outcomes between these agents in patients with cardiogenic shock (both AMI-CS and all-aetiology CS) (Jung et al., 2021; Mathew et al., 2021). CAPITAL-DOREMI 2 is currently investigating inotropes (dobutamine or milrinone) versus placebo in SCAI stage C/D shock (Parlow et al., 2023). In practice, dobutamine is often preferred due to its shorter half-life and ease of titration, whereas milrinone may be considered in patients receiving beta-blockers or with pulmonary hypertension.

Levosimendan, a calcium sensitiser with inodilatory properties, has been proposed as an alternative to catecholamine-based inotropes, particularly due to its ability to enhance myocardial contractility without substantially increasing myocardial oxygen demand. This pharmacological profile makes it theoretically attractive in patients with ischaemic cardiomyopathy or those receiving beta-blockers, in whom adrenergic responsiveness may be attenuated. Additionally, its vasodilatory effects may reduce afterload and improve cardiac output, although at the expense of hypotension. Despite these theoretical advantages, clinical trial data have been inconsistent. Earlier studies, such as the SURVIVE trial, did not demonstrate a mortality benefit compared with dobutamine, although subgroup analyses suggested a possible benefit in patients receiving beta-blockers (Mebazaa et al., 2009; Mebazaa et al., 2007). Subsequent meta-analyses have reported mixed results, often limited by heterogeneity and inclusion of patients without severe cardiogenic shock (Fernando et al., 2022; Schumann et al., 2018; Uhlig et al., 2020). Combination therapy with noradrenaline showed similar outcomes between levosimendan and dobutamine (Tarvasmäki et al., 2016). LevoHeartShock, an ongoing trial, is assessing early levosimendan vs placebo in CS patients treated already treated with standard therapy (NCT04020263). The primary composite endpoint is 30-day, all-cause mortality and/or ECMO implantation and/or dialysis. The study is anticipated to end in January 2028. Levosimendan's inodilator properties combined with limited increases in myocardial oxygen demand also make it an attractive alternative to dobutamine in venoarterial ECMO (VA-ECMO), where LV unloading may be needed to counteract the increased afterload and prevent pulmonary oedema (Cholley et al., 2019). Evidence for its use in this setting is limited and of inadequate quality to inform clinical practice (Distelmaier et al., 2016; Vally et al., 2019). The recent LEVOECMO trial evaluated levosimendan in patients with cardiogenic shock requiring VA-ECMO support, finding no significant difference in successful weaning from ECMO or in clinically relevant outcomes compared with placebo (Combes et al., 2026).

2.4 Escalation to Mechanical Circulatory Support

Mechanical circulatory support (MCS) is frequently employed in cardiogenic shock (CS), although high-quality evidence guiding patient selection, timing, and device choice remains limited. Its primary role is to stabilise haemodynamics, reduce ventricular workload, and improve end-organ perfusion, typically as a bridge to recovery, decision, or advanced therapies (Behnam N. Tehrani et al., 2022).

Current practice increasingly favours early consideration of MCS in patients with persistent hypoperfusion despite optimal pharmacological therapy. Observational data suggest that early device implantation—particularly prior to revascularisation in AMI-CS—may improve outcomes, especially when integrated into protocolised care pathways with invasive haemodynamic monitoring. However, these findings are not consistently supported by randomised data (Basir et al., 2019; Basir et al., 2018; Basir et al., 2017; Taleb et al., 2019; Tehrani et al., 2019).

2.5 Intra-aortic Balloon Pump vs Standard Care

Due to uncertainty in MCS patient selection, several trials have assessed the efficacy of various support strategies. In IABP-SHOCK II, IABP use in AMI-CS showed no mortality benefit at 30 days, nor differences in haemodynamic or renal outcomes, or long-term quality of life at 12-month follow-up (Thiele et al., 2012). Rates of reinfarction and recurrent revascularisation at 12 months were comparable in patients on IABP and those without (Thiele et al., 2013). There seems to be no benefit of IABP in infarct size reduction in AMI-CS (Patel et al., 2011). Accordingly, European and US guidelines no longer recommend routine IABP in AMI-CS unless mechanical complications are present (Byrne et al., 2023; Diepen et al., 2017), and real-world data shows a 50% decline in use (Cohen et al., 2025).

Since patients with HF-CS have a different underlying haemodynamic pathophysiology, characterised by a ventricular afterload mismatch with little preload reserve, even modest augmentation of afterload can translate into increased cardiac output, making IABP mechanistically viable in early SCAI stages of HF-CS. However, the Altshock-2 trial in SCAI B–D CS secondary to acute decompensated HF found no difference in 60-day survival or successful bridge to heart replacement therapy, leading to early termination for futility (Nuccia Morici et al., 2025). IABP might thus not be appropriate as an early intervention in this patient population, likely having a similar efficacy to less invasive options like inotropes.

2.6 PVADS vs Standard Care

Several trials have investigated the Impella (Abiomed; Danvers, Massachusetts) and other percutaneously implanted ventricular assist devices (pVADs), both as standalone support and for LV unloading in VA-ECMO. A meta-analysis of three RCTs comparing pVADs (TandemHeart (LivaNova; London, UK), Impella 2.5) with IABP showed higher CI, MAP, and lower PCWP with pVADs, but also more bleeding and no mortality benefit (Cheng et al., 2009). The DanGer-SHOCK trial was the first large MCS trial to show a lower 180 day mortality in patients on Impella compared to standard care {at the expense of a higher incidence of adverse events and renal replacement therapy} (Møller et al., 2024). These results are likely to influence the choice of MCS in the near future, with microaxial-flow pumps preferred over IABP.

2.7 Venoarterial Extracorporeal Membrane Oxygenation (VA-ECMO)

ECMO, unlike other MCS modalities, provides both circulatory and respiratory support and is sometimes used in Stage E CS or cardiac arrest, with some data suggesting reduced 30-day mortality in this setting (Ouweneel et al., 2016) (although patient selection for ECMO in cardiac arrest is still debated (Yannopoulos et al., 2017)). It is easier to implant than the TandemHeart or Impella 5.5, and can be used as part of pre-hospital emergency care teams (Levy et al., 2015). Since retrograde flow from femoral arterial cannulation increases LV afterload, adjunctive LV unloading is sometimes required (Combes et al., 2020; Schrage et al., 2018).

Several trials investigated the use of VA-ECMO in CS patients. The ECMO-CS trial showed no benefit of early VA-ECMO implantation in patients in severe or rapidly deteriorating, any-cause CS in terms of the 30-day composite outcome (death from any cause, resuscitated circulatory arrest, and implementation of another MCS device) (Ostadal et al., 2023). Interpretation of ECMO-CS data is, however, limited by high crossover (39% escalated to ECMO in control arm) and heterogeneous inclusion criteria, potentially diluting benefit in subgroups more likely to respond. EURO-SHOCK, halted early due to COVID-19 showed non-significant mortality reduction with VA-ECMO in AMI-CS patients, but increased rate of bleeding and vascular complications (Banning et al., 2023). Published concurrently, the ECLS-SHOCK trial yielded findings consistent with those of EURO-SHOCK, finding no difference in 30-day mortality between VA-ECMO and standard therapy in AMI-CS (Thiele et al., 2023).

2.8 Impella vs IABP

Comparing the change in cardiac index in 26 AMI-CS patients on either IABP or Impella LP2.5, im et al. (Seyfarth et al., 2008) found a significantly greater increase in the Impella group 30 minutes post implantation. This did not translate into reduced mortality however, with rates of 46% in both intervention groups. The 2017 IMPRESS trial enrolling patients with AMI-CS yielded results consistent with the above, with no difference in mortality between Impella and IABP groups (Ouweneel et al., 2017). It should be noted that in both trials, the time of MCS initiation (pre- or post-PCI) was at the discretion of the operator – this differs from current recommendations regarding early MCS in this patient population. There is also some evidence that Impella reduces pre- and post-procedural major adverse events compared with IABP in cases of high-risk PCI, although these studies did not enrol patients with cardiogenic shock specifically (Dangas et al., 2014; KOVACIC et al., 2015; O'Neill et al., 2012).

2.9 Impella vs IABP for LV Unloading in Patients on VA-ECMO

A recent systematic review and meta-analysis reviewed results from 7 observational studies comparing Impella and IABP for LV unloading in VA-ECMO (Gandhi et al., 2023). Among 698 patients included, no difference in short-term mortality, transition to durable LVAD/cardiac transplantation, initiation of RRT, stroke or limb ischaemia were seen. Major bleeding was more common in patients unloaded with Impella vs IABP (57.2% vs 39.7%; RR 1.66, 95% CI 1.12-2.44), and so was the incidence of haemolysis (31% vs 7%; RR 4.61, 95% CI 1.24-17.17).

Yeo et al. in their observational study based on data from the Nationwide Readmissions Database compared LV unloading strategies (IABP and Impella) with ECMO alone in 15 980 patients on VA-ECMO (Yeo et al., 2024). In-hospital mortality was lower in patients on ECMO and IABP compared to ECMO alone, whereas ECMO with Impella and ECMO alone had similar in-hospital mortality. ECMO with Impella had a higher incidence of bleeding events and AKI requiring haemodialysis. Compared to ECMO+IABP, ECMO+Impella was associated with a higher incidence of, higher in-hospital and 40-day mortality.

In another retrospective review of 283 patients on either ECMO alone or ECMO unloaded with Impella or IABP, Char et al. found an increased 180-day survival in patients unloaded with IABP compared with ECMO alone, whereas no difference in survival was observed in the ECMO+Impella group (Char et al., 2023). A higher incidence of bleeding events was noted in the ECMO+Impella group compared with other groups.

3. Staging and Risk Stratification

Over the past decades, multiple risk scores have been proposed for stratification of risk in cardiogenic shock patients, ranging from general Intensive Care Unit ICU scores for unselected patients admitted with shock (APACHE, SOFA) (Kellner et al., 2013) to those designed for patients in whom cardiac dysfunction is the primary contributor to shock. The vast majority of those scores have been modelled from trials and registries investigating AMI-CS, and many lack generalisability in non-AMI related cardiogenic shock populations. Here, we discuss several prominent risk stratification tools in the general CS population, in AMI-CS patients and in those already on MCS therapy. We also discuss clinical phenotyping strategies, biomarker-based risk stratification and broad patient variables that convey prognostic value.

3.1 Society for Cardiovascular Angiography and Intervention (SCAI) Staging

Several approaches have been suggested for severity staging of CS. The one most used was developed by the Society for Cardiovascular Angiography and Intervention (SCAI), who proposed five levels of CS severity (outlined in Figure 2).

Stages A-B are characterised by the absence of hypoperfusion, with patients with risk factors for CS categorised as Stage A and those with hypotension or tachycardia being at Stage B (now increasingly considered “preshock” (Jentzer et al., 2021)). Stages C-E, in contrast, are defined by hypoperfusion, with higher stages requiring increasingly intensified interventions in the form of vasoactive compounds, MCS or ECMO. The SCAI system does not, however, differentiate between the normotensive, hypoperfused CS clinical phenotype, which tends to occur in early-stage C shock when increased systemic vascular resistance is able to compensate, the isolated hypotensive phenotype and the hypotensive, hypoperfused phenotype. Jentzer showed an in-hospital mortality of 17.2% for the first group, 9.3% for the second and 33.8% for the

hypotensive and hypoperfused group (Jentzer et al., 2021). SCAI staging also fails to account for the type of ventricular failure (RV, LV vs biventricular failure). Previous analyses have shown significantly poorer outcomes in biventricular failure compared with LVF alone (Hamon et al., 2008; Kuchibhotla et al., 2017; Mehta et al., 2001), whereas studies investigating outcomes of cardiogenic shock with predominant RV failure compared to shock with LV failure yielded conflicting results (Brodie et al., 2007; Jacobs et al., 2003; Josiassen et al., 2021). A post-hoc analysis of the landmark SHOCK trial found that patients with RV-predominant shock (5.3%) were younger, had a smaller prevalence of multivessel disease and prior AMI, and had a shorter median time between the index MI and the diagnosis of shock than patients with LV-predominant shock (94.7%). No significant difference in in-hospital mortality was found (53.1% in RV group versus 60.8% in LV group, $p = 0.296$). Despite this, the SCAI staging system has been validated in multiple studies, with increasing mortality from stages B to E, regardless of aetiology (Baran et al., 2020; Hanson et al., 2020; Jentzer et al., 2019; Pareek et al., 2021; Schrage et al., 2020; Thayer et al., 2020).

3.2 Cardshock Study

This was a multicentre, prospective, observational study enrolling patients with both AMI (81%; 68% ST-elevation myocardial infarction and 8% mechanical complications) and non-AMI aetiologies (mostly severe chronic heart failure and valvular causes) of cardiogenic shock (Harjola et al., 2015). A risk prediction model for in-hospital mortality was proposed, based on variables independently associated with all-cause death in multivariable logistic regression analysis. Apart from variables that are more obviously associated with worse prognosis, like markers of hypoperfusion or older age, AMI-CS was also found to be an independent predictor of mortality, despite the high rates of revascularisation and a clinical presentation similar to patients with non-AMI cardiogenic shock. Older age, confusion, previous coronary artery bypass graft (CABG)/MI, LV ejection fraction (LVEF) $<40\%$, serum lactate and eGFR were all associated with increased hospital mortality. The model showed good discrimination, with an area under the curve (AUC) of 0.85 (95% CI 0.79–0.90; $P < 0.001$) for in-hospital mortality. The score was validated using the cohort from the IABP-SHOCK II trial (Thiele et al., 2012). The score has good clinical utility, is practicable to calculate and considers the increased risk of AMI-CS.

3.3 Risk Stratification in AMI-CS

Data from the SHOCK trial and registry formed a basis for an early risk prediction model. The randomised trial investigated the value of early revascularisation in AMI-CS to reduce mortality (Hochman et al., 1999). Based on data from 294 patients from the trial and 923 patients from the registry, Sleeper et al. developed a Stage 1 severity scoring system based on clinical variables, and a Stage 2 severity scoring system that included haemodynamic parameters from patients with pulmonary artery (PA) catheters inserted (Sleeper et al., 2010). Overall, the in-hospital 30-day mortality among included patients with AMI-CS was 57%. The stage 1 multivariate modelling identified 8 risk factors – age (OR 1.27 per 5y increase), shock on admission (OR 1.92), end-organ hypoperfusion (OR 4.16), anoxic brain injury (OR 21.52), systolic blood pressure (SBP) (OR 1.09 per 5mmHg decrease), prior CABG (OR 2.00), non-inferior MI (OR 1.39) and creatinine $\geq 1.9\text{mg/dL}$ (OR 1.68) (c-statistic 0.74). Supplementary Table 2a shows the scoring for the identified risk factors. Mortality increases with rising severity scores, reaching 88% in the upper deciles. The stage 2 multivariate analysis was based on data from 872 patients who underwent haemodynamic monitoring. Inpatient, 30-day mortality in this group was 50%. The model consists of five variables – age (OR 1.23 per 5y increase), end-organ hypoperfusion (OR 3.92), anoxic brain injury (OR 12.1), stroke work (OR 0.84 per 5 units) and LVEF $<28\%$ (OR 2.20) (c-statistic 0.75). The scoring outline can be appreciated in Supplementary Table 2b.

A subsequent risk stratification model was developed from the IABP-SHOCK II trial patient cohort (Pöss et al., 2017). The model was developed based on data from 480 patients from the randomised population. They identified the following six variables that served as independent predictors of 30-day mortality, and these were used as score parameters – age >73 (HR 1.54), history of stroke (HR 2.09), glucose $>10.6\text{mmol/L}$ at admission (HR 1.48), creatinine $>132.6\mu\text{mol/L}$ at admission (HR 1.57), arterial lactate $>5\text{mmol/L}$ at admission (HR 1.98), Thrombolysis After Myocardial Infarction (TIMI) flow grade <3 after PCI (HR 2.73). Supplementary Table 3 illustrates the scoring system proposed. The model was internally validated on 98 registry patients (c-statistic 0.79), and externally validated on 137 patients from the CardShock trial cohort (c-statistic 0.73), with both showing good discrimination.

Prior studies have also established individual variables that were predictors of poor outcome in AMI-CS. Fig. 3 shows patient variables frequently cited in AMI-CS risk prediction scores (Kalra et al., 2021).

Despite differences in derivation cohorts, most risk scores consistently identify age, lactate, renal dysfunction and markers of end-organ injury as dominant predictors of mortality. Notably, many variables incorporated into contemporary phenotyping models overlap substantially with those used in traditional risk scores, raising the possibility that phenotyping may represent a more sophisticated reformulation of established prognostic factors rather than a fundamentally new concept.

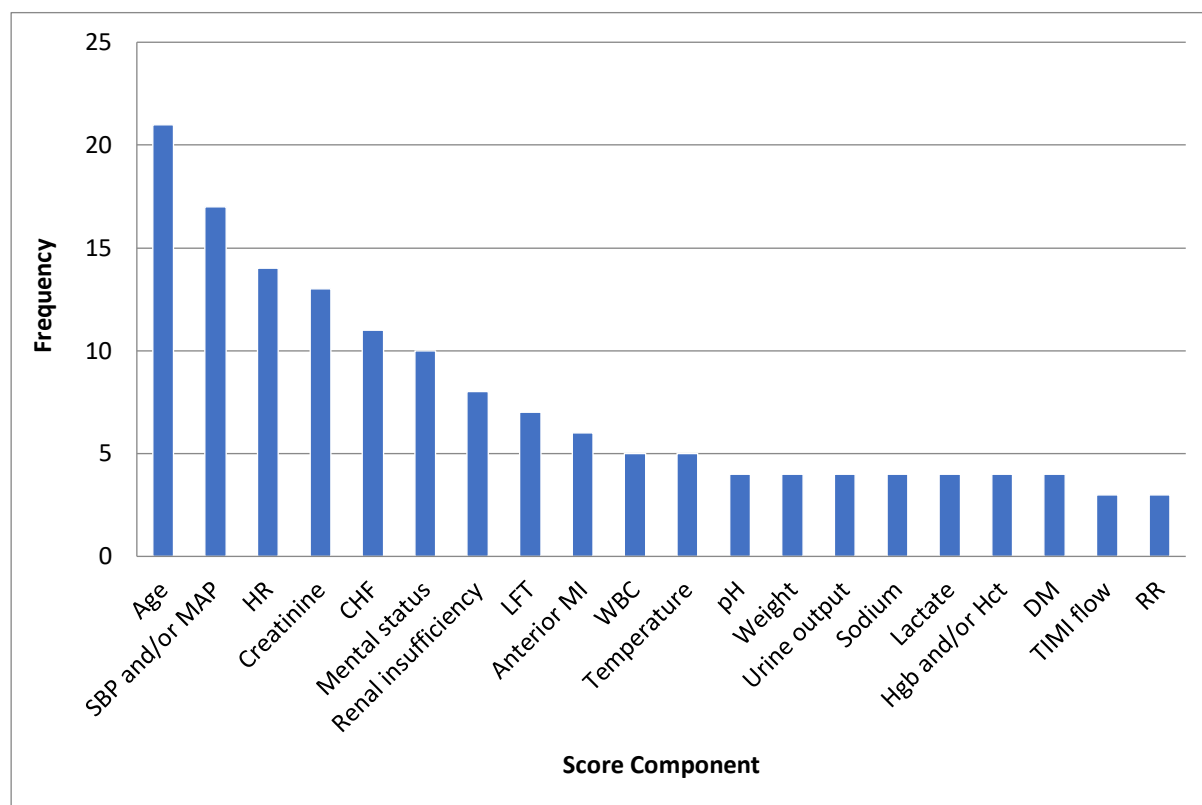


Fig. 3. Variables frequently cited in AMI-CS risk prediction scores. Adapted from Kalra et al. (Kalra et al., 2021). SBP – systolic blood pressure; MAP – mean arterial pressure; HR – heart rate; CHF – congestive heart failure; LFT – liver function tests; WBC – white blood cells; Hgb – haemoglobin; Hct – haematocrit; DM – diabetes mellitus; TIMI – thrombolysis in myocardial infarction; RR – respiratory rate.

3.4 Risk Stratification in Patients on MCS

Severity scores for patients on MCS are crucial both for prognostication and to help physicians select patients suitable for MCS therapy. Several studies investigated factors associated with worse outcomes in patients on veno-arterial extracorporeal membrane oxygenation (VA-ECMO). The ENCOURAGE score, derived from a French multicenter registry of 138 patients with AMI-CS treated with VA-ECMO, offers a context-specific risk stratification tool, using seven pre-ECMO parameters – age >60 (OR 2.63), female sex (OR 4.35), body mass index (BMI) >25kg/m² (OR 3.10), Glasgow Coma Scale (GCS) <6 (OR 3.09), creatinine >150μmol/L (OR 2.60), elevated serum lactate (2-8mmol/L – OR 4.71; >8mmol/L – OR 8.71) and prothrombin activity <50% (OR 2.80). It demonstrated strong discriminative ability (AUC 0.85) for in-hospital mortality. While promising in its targeted population, its use is limited by a lack of external validation and restriction to AMI-CS cases.

The SAVE score (Survival After Veno-Arterial ECMO) was another model developed to predict in-hospital survival in patients with cardiogenic shock treated with VA-ECMO, irrespective of the underlying aetiology (Schmidt et al., 2015). Derived from the ELSO registry, it included 3846 adult patients, making it one of the largest cohorts used for risk stratification in this context. The score incorporates 12 pre-ECMO variables, including age, CS aetiology (e.g., myocarditis, AMI), organ failure, weight, duration of intubation, and prior cardiac arrest (detailed in Supplementary Table 4). It stratifies patients into five risk groups, with

predicted survival ranging from <20% in the lowest category to >70% in the highest. The SAVE score demonstrated good discriminative ability (AUC 0.68) and has been externally validated on a cohort of 161 patients (AUC 0.9), offering a practical tool to support decision-making regarding ECMO initiation, prognostication, and resource allocation.

3.5 Clinical Phenotyping

Clinical phenotyping has emerged as a pivotal approach in the management of cardiogenic shock (CS), offering a nuanced understanding of patient heterogeneity and informing tailored therapeutic strategies. Recent studies have identified distinct CS phenotypes characterized by varying clinical and biochemical profiles, which correlate with differential outcomes.

In one study, Zweck and colleagues sought to determine whether clinical phenotypes identified in the acute phase of cardiogenic shock could predict distinct clinical profiles and inpatient mortality (Zweck et al., 2021). Using clustering analysis in 1959 patients with AMI-CS and acute-on-chronic CS, three phenotypes were identified: phenotype I (low-risk, preserved organ function), phenotype II (comorbidities, renal dysfunction, anaemia and congestion), and phenotype III (high-risk, with hepatic dysfunction, lactic acidosis, inflammation and right-sided congestion). These phenotypes were associated with progressively increasing mortality, a relationship that persisted across SCAI stages and in patients with non-AMI CS. Findings were validated in an independent cohort of 796 patients, with consistent phenotype characteristics and mortality gradients. Notably, outcomes following mechanical circulatory support varied by phenotype, suggesting potential differences in treatment response. The study is, however, limited by its retrospective design, reliance on observational data, and incomplete adjustment for confounders. Furthermore, overlap with established risk markers raises uncertainty as to whether phenotyping provides incremental prognostic value beyond existing models or applying clinical judgment to independent variables (Mebazaa & Soussi, 2023).

Soussi et al. conducted a secondary analysis of the FROG-ICU cohort to investigate heterogeneity among cardiogenic shock (CS) survivors at ICU discharge and its prognostic implications (Soussi et al., 2024). Among 173 patients included in the study—all meeting SCAI shock stages C to E—CS was of ischaemic origin in 38% of cases. Using latent class analysis on 15 routine clinical and laboratory variables, the authors identified two distinct phenotypes at ICU discharge. Phenotype B, comprising 38% of the cohort, was characterized by greater systemic inflammation (e.g. elevated procalcitonin and IL-6), ongoing kidney injury, endothelial dysfunction (e.g. bio-adrenomedullin), and myocardial fibrosis (e.g. galectin-3). This group also had more frequent non-ischaemic CS aetiology and right ventricular dysfunction on admission. Patients in Phenotype B had significantly higher one-year mortality (adjusted HR 2.83, 95% CI 1.21–6.60, $p = 0.016$) and worse physical quality of life at 3 months. These findings emphasize the prognostic relevance of late-stage phenotyping in CS, highlighting a potential role for biomarker-guided, individualised care pathways. Other variables like the degree of ventriculo-arterial uncoupling (Guinot et al., 2022) or microcirculatory impairment (Schemmelmann et al., 2024) have also been proposed as subphenotypes useful clinically to guide patient care. The syndromic nature of cardiogenic shock and the lack of significant improvements in outcomes despite advancements in management options should prompt further enquiry into clinical phenotyping using machine learning models, molecular/omics-based approaches and existing biomarkers and physiologic measurements (Soussi et al., 2025).

4. Summary

Cardiogenic shock remains a highly lethal clinical syndrome characterised by profound haemodynamic compromise and progressive multi-organ dysfunction. Despite significant advances in understanding its pathophysiology, risk stratification, and management, outcomes remain poor, particularly among patients presenting with advanced shock or established end-organ injury. This review highlights the complex and heterogeneous nature of CS, encompassing diverse aetiologies as well as a broad spectrum of clinical presentations. Importantly, normotensive hypoperfused states are increasingly recognised as high-risk phenotypes that may be underappreciated in routine clinical practice.

Efforts to standardise the diagnosis and classification of CS, including the SCAI staging system and SHARC criteria, represent important advances in improving clinical communication, facilitating research, and guiding management. However, existing classification systems do not fully capture important clinical and

haemodynamic nuances, including isolated right or left ventricular failure, dynamic changes in perfusion status, and the impact of patient-specific comorbidities.

Current pharmacological management remains guided largely by physiological rationale, clinical experience, and limited trial evidence. The superiority of noradrenaline over adrenaline and dopamine is increasingly supported by its more favourable haemodynamic profile and lower incidence of adverse effects. However, evidence supporting one inotrope over another remains limited, with major trials demonstrating no clear superiority of milrinone, dobutamine, or levosimendan. Furthermore, many of these studies excluded patients with the most severe forms of shock, limiting the generalisability of their findings. Although levosimendan may offer theoretical advantages in patients receiving beta-blockers and as an adjunct to VA-ECMO for reducing left ventricular afterload, robust outcome data are lacking.

Mechanical circulatory support remains a promising but unsettled frontier. While observational studies support early MCS within protocolised care pathways incorporating invasive haemodynamic monitoring, major RCTs including IABP-SHOCK II, ECLS-SHOCK, and ECMO-CS have failed to demonstrate consistent mortality benefits. In VA-ECMO studies, heterogeneous patient populations, delayed initiation, crossover between treatment arms, and high complication rates may have obscured benefits in selected patients. These findings suggest that MCS is unlikely to provide universal benefit but may improve outcomes when applied early in carefully selected patients and combined with strategies to minimise complications, including left ventricular unloading. The recent DanGer-SHOCK trial suggests a survival benefit with Impella in AMI-CS, offset by increased bleeding and renal complications, highlighting the need for more refined patient selection and device-specific treatment algorithms. The optimal unloading strategy during VA-ECMO also remains uncertain, with emerging data suggesting that IABP may offer advantages over Impella in some clinical settings.

Risk stratification tools such as the CardShock and IABP-SHOCK II scores provide useful prognostic information, particularly in AMI-CS, although their performance is less robust in non-ischaemic and mixed-aetiology populations. Similarly, emerging phenotype-based approaches have demonstrated promising prognostic value but require prospective validation before widespread implementation.

Several important evidence gaps remain. High-quality randomised trials are needed to better define the role, timing, and patient selection criteria for MCS across different CS phenotypes and SCAI stages, particularly in non-AMI populations. Further research should also clarify the utility of invasive haemodynamic monitoring and evaluate the integration of biomarker-guided and phenotype-based strategies into clinical decision-making. In addition, long-term outcomes beyond in-hospital mortality—including quality of life, neurocognitive function, and rehospitalisation—remain insufficiently studied despite their importance to patients and healthcare systems.

In conclusion, although meaningful progress has been made in the diagnosis and management of cardiogenic shock, treatment remains largely empirical and improvements in survival have been modest. A more personalised, data-driven approach incorporating early phenotyping, invasive haemodynamic assessment, and dynamic risk stratification is likely to improve patient selection and optimise the use of available therapies. Ultimately, improving outcomes in cardiogenic shock may depend less on developing new interventions than on identifying the right intervention, for the right patient, at the right time. Addressing the substantial remaining evidence gaps through collaborative research networks, improved trial design, and standardised care pathways will be essential to achieving this goal.

5. Supplementary Material

Supplementary table 1: Summary of key trials in cardiogenic shock management.

Trial (Year)	Patients (N)	Design	Groups	Primary endpoint	Secondary and safety Endpoints	Results (Key Findings, Statistics)
SHOCK (1999)	n=600 (AMI with CS, LV failure)	RCT, open-label, multicenter	Emergency revascularization (PCI or CABG) vs initial medical therapy	30-day all-cause mortality	Secondary: 6-month mortality	30-day mortality: 39.7% (IABP) vs 41.3% (control) (RR 0.96; 95%CI 0.79–1.17; P=0.69). 6-mo mortality: 50.3% vs 63.1% (P=0.027). No differences in safety endpoints (bleeding, stroke).
CULPRIT-SHOCK (2017)	n=706 (AMI-CS, multivessel CAD)	RCT, open-label, multicenter	Culprit-only PCI ($\pm$ staged PCI) vs immediate multivessel PCI	Death or severe renal failure (RRT) at 30 days	Secondary: death, RRT, MI, stroke	Primary composite: 45.9% (culprit-only) vs 55.4% (multivessel) (RR 0.83; 95%CI 0.71–0.96; P=0.01). Death at 30d: 43.3% vs 51.6% (RR 0.84; 95%CI 0.72–0.98; P=0.03). Non-culprit PCI significantly increased risk. RRT use trended lower (14.5% vs 20.6%, P=0.07). No difference in shock resolution.
OPTIMA-CC (2018)	n=57 (post-AMI CS after PCI)	RCT, double-blind, multicenter	Epinephrine vs norepinephrine infusion	Cardiac index evolution	Primary safety: refractory shock; other: metabolic, arrhythmias	No difference in CI (P=0.43). Refractory shock was higher with epinephrine (37% vs 7%; P=0.008), leading to early trial termination. Epinephrine $\uparrow$ HR and lactate; trial halted due to harm.
CardShock Vasopressor Substudy (2016)	n=219 (CardShock registry: CS of all causes)	Prospective registry (observational)	Analysis by vasopressor type (adrenaline vs none)	90-day all-cause mortality	Secondary/safety: organ injury scores	Adrenaline use (21%) was independently associated with higher 90-day mortality (unadjusted OR 5.2; 95%CI 1.88–14.7; P=0.002; adjusted OR 3.0; 95%CI 1.3–7.2; P=0.013). Adrenaline-treated patients had more organ injury (lactate, acidosis) and dose requirements. Other inotropes (levosimendan, etc.) had no adverse association.

Trial (Year)	Patients (N)	Design	Groups	Primary endpoint	Secondary and safety Endpoints	Results (Key Findings, Statistics)
SOAP-II (2010)	n=1679 (all shock etiologies)	RCT, double-blind, multicenter	Dopamine vs norepinephrine	28-day all-cause mortality	Secondary: arrhythmias, organ-support days	28-day mortality: 52.5% (dopamine) vs 48.5% (norepi); OR 1.17 (95%CI 0.97–1.42; P=0.10). No significant difference. Dopamine caused more arrhythmias: 24.1% vs 12.4% (P<0.001). Norepinephrine had a trend to lower arrhythmias and ICU stay. Multivariate: dopamine was independent predictor of arrhythmias.
DOREMI (2021)	n=192 (CS; 65 with AMI-CS)	RCT, double-blind	Milrinone vs dobutamine	Composite (death, arrest, transplant/MCS, MI, stroke, RRT in-hospital)	Secondary: individual components, death, arrest, MCS, RRT	Primary composite: 49% (milrinone) vs 54% (dobu) (RR 0.90; 95%CI 0.69–1.19; P=0.47). In-hospital death: 37% vs 43% (RR 0.85; 95%CI 0.60–1.21). No significant differences in any endpoint (resuscitated arrest, MCS, RRT).
CAPITAL-DOREMI II (ongoing)	n=346 planned (CS, SCAI C–D)	RCT, double-blind, placebo-controlled	Milrinone vs dobutamine vs placebo	Likely 30-day mortality or hemodynamic composite	Not specified (presumably safety, organ support)	Ongoing trial (NCT05267886) comparing milrinone, dobutamine, and placebo in CS. Expected completion December 2026. No results yet.
SURVIVE (2007)	n=1327 (acute decompensated HF needing inotropes)	RCT, double-blind, multicenter	Levosimendan vs dobutamine	All-cause mortality at 180 days	Secondary: 31-day mortality, days alive/out of hospital, NYHA class, natriuretic peptide levels, etc.	180-day mortality: 26% (levo) vs 28% (dobu); HR 0.91 (95%CI 0.74–1.13; P=0.40). No significant diff. Levosimendan lowered BNP more (P<0.001 at 5d). Safety: more atrial fibrillation with levo (18% vs 10%; P=0.001). Overall no survival benefit.
SURVIVE Substudy – β -blockers (2009)	n=669 (subset of SURVIVE on β -blocker therapy)	Post-hoc subgroup analysis	Levosimendan vs dobutamine	Mortality at days 5 and 14	N/A	In β -blocker patients, levosimendan reduced early mortality: Day 5 death 1.5% vs 5.1% (HR 0.29; 95%CI 0.11–0.78; P=0.01). Day 14: 7.0% vs 10.3% (HR 0.67; 95%CI 0.45–0.99; P=0.045). Suggests Levo may benefit HF pts on β -blockers.

Trial (Year)	Patients (N)	Design	Groups	Primary endpoint	Secondary and safety Endpoints	Results (Key Findings, Statistics)
IABP-SHOCK II (2012)	n=600 (AMI-CS planned revascularization)	RCT, open-label, multicenter	IABP + usual care vs usual care alone	30-day all-cause mortality	Secondary: hemodynamic stabilization, ICU stay, catecholamine dose, complications	30-day mortality: 39.7% (IABP) vs 41.3% (control); RR 0.96 (95%CI 0.79–1.17; P=0.69). No difference in secondary endpoints or in bleeding, stroke, limb ischemia (e.g. bleeding: 3.3% vs 4.4%, P=0.51).
Altshock-2 (2025)	n=101 (HF-CS, SCAI B–D)	RCT, open-label, multicenter (Italy)	Early IABP + standard care vs standard care	Survival or bridge to heart replacement at 60d	Secondary/safety: MCS escalation, complications	60-day composite (survival or bridge): 81% (IABP) vs 75% (control); HR 0.72; P=0.45. No statistical benefit; trial stopped for futility. Escalation to other MCS similar. Complications (bleeding, limb ischemia) comparable between groups. Largest HF-CS IABP RCT to date showed no advantage of routine early IABP.
DanGer-SHOCK (2024)	n=355 (STEMI-CS)	RCT, open-label, multicenter (DK/DE/UK)	Impella CP microaxial pump + SOC vs SOC alone	180-day all-cause mortality	Safety: severe bleeding, limb ischemia, device failure	180-day mortality: 45.8% (Impella) vs 58.5% (control); HR 0.74 (95%CI 0.55–0.99); P=0.04 (NNT≈8). Safety composite: 24.0% vs 6.2% (RR 4.74; 95%CI 2.36–9.55) (more bleeding/limb events). Benefit greatest in more severe hypotension.
Seyfarth et al., 2008	n=25 (AMI-CS)	Prospective, 2-center, randomized trial	Impella LP2.5 vs IABP	Change of cardiac index (CI) from baseline to 30 min after implantation.	Lactic acidosis, hemolysis, and mortality after 30 days	CI after 30 min of support was significantly increased in patients with the Impella LP2.5 compared with patients with IABP (Impella: $\Delta CI = 0.49 \pm 0.46$ l/min/m ² ; IABP: $\Delta CI = 0.11 \pm 0.31$ l/min/m ² ; p = 0.02). 30-day mortality was 46% in both groups
IMPRESS (2013)	n=48 (STEMI + severe CS, ventilated)	RCT, open-label (single-center)	Impella CP vs IABP	30-day (reported 6-month) mortality	Secondary: stroke, MACCE	6-month mortality: 50% (Impella) vs 50% (IABP) (P=0.92). No difference in any endpoint. Small trial (n=48) underpowered; concluded similar outcomes with either support.

Trial (Year)	Patients (N)	Design	Groups	Primary endpoint	Secondary and safety Endpoints	Results (Key Findings, Statistics)
Yeo et al., 2024	n=15,980	Observational (database analysis)	ECMO only vs ECMO + IABP vs ECMO + Impella	In-hospital mortality (adjusted)	40-day mortality, bleeding, AKI	ECMO+IABP had lower in-hospital mortality vs ECMO alone (aOR 0.83; P=0.02). ECMO+Impella did not reduce mortality (aOR 1.09; P=0.695). Compared to ECMO+IABP, ECMO+Impella had higher 40-day mortality (HR 1.25; P<0.001) and more complications (bleeding aOR 1.21; AKI requiring HD aOR 1.42).
Char et al., 2023	n=283 (VA-ECMO for primary CS)	Retrospective single-center	ECMO only vs ECMO + IABP vs ECMO + Impella	180-day survival	Bleeding, hemodynamics	ECMO+IABP group (n=68) had improved 180-day survival vs ECMO alone (log-rank P=0.005). ECMO+Impella (n=72) showed no survival benefit vs ECMO alone (P=0.66). Bleeding: 52.8% (Impella) vs 37.1% (IABP) vs 17.7% (ECMO only) (P<0.001). Pre-ECMO IABP was an independent protective factor (HR 0.45).
ECMO-CS (2022)	n=117 (rapidly deteriorating or severe CS)	RCT, open-label, multicenter (CZ)	Immediate VA-ECMO vs no immediate VA-ECMO (support allowed if needed)	Composite: death, resuscitated arrest, or new MCS at 30d	Secondary: 30- and 90-day mortality	Primary event (30d composite): 63.8% (early ECMO) vs 71.2% (conservative); HR 0.72 (95%CI 0.46–1.12); P=0.21. Not significant. 30-day mortality ~50% in both. 39% of conservative group eventually received ECMO.
EURO-SHOCK (2023)	n=35 (persistent CS post-PCI in AMI)	RCT, open-label (multinational)	Early VA-ECMO vs continued standard therapy	30-day all-cause mortality	1-year mortality, rehospitalization	Stopped early (COVID); 30-day mortality: 43.8% (ECMO) vs 61.1% (control); HR 0.56 (95%CI 0.21–1.45); P=0.22. 1-year mortality: 51.8% vs 81.5% (HR 0.52; P=0.14). More vascular (21.4% vs 0%) and bleeding (35.7% vs 5.6%) complications with ECMO. Underpowered; trend favoured ECMO but not conclusive.

Trial (Year)	Patients (N)	Design	Groups	Primary endpoint	Secondary and safety Endpoints	Results (Key Findings, Statistics)
ECLS-SHOCK (2023)	n=417 (AMI-CS, high lactate, SCAI C–E)	RCT, open-label (DE/SI)	Early VA-ECMO + SOC vs SOC alone (crossover discouraged; Impella allowed)	30-day all-cause mortality	30-day RRT, neurologic outcome, bleeding, limb events	30-day mortality: 47.8% (ECLS) vs 49.0% (control); RR 0.98 (95%CI 0.80–1.19); P=0.81. No mortality benefit. ECLS had higher moderate-severe bleeding (23.4% vs 9.6%; RR 2.44; 95%CI 1.50–3.95) and more vascular complications (11.0% vs 3.8%; RR 2.86). 30-day RRT trended lower (8.1% vs 13.9%).

Supplementary Table 2a: Stage 1 (clinical) cardiogenic shock scoring system without invasive haemodynamics (Sleeper et al., 2010).

Variable	Points	Variable	Points
Anoxic brain damage	30	Hypoperfusion	14
Shock on admission	6	Prior CABG	7
Noninferior MI	3	Creatinine ≥ 1.9 mg/dL	5
Age, y		Systolic BP, mm Hg	
≤45	0	≤55	12
46–50	2	56–60	11
51–55	5	61–65	10
56–60	7	66–70	9
61–65	10	71–75	8
66–70	12	76–80	7
71–75	15	81–85	6
76–80	17	86–90	5
81–85	20	91–95	4
86–90	22	96–100	3
>90	25	101–105	2
		105–110	1
		>110	0

Supplementary Table 2b: Stage 2 (haemodynamic) scoring system with invasive hemodynamics (Sleeper et al., 2010).

Variable	Points	Variable	Points
Anoxic brain damage	25	Hypoperfusion	14
LVEF <28%	8	Stroke work, g/m	
Age, y		≤5	18
≤45	0	6–15	15
46–51	2	16–25	12
51–55	4	26–35	9
56–60	6	36–45	6
61–65	8	46–55	3
66–70	10	>55	0
71–75	12		
76–80	14		
81–85	16		
86–90	18		
>90	20		

Supplementary Table 3: AMI-CS severity scoring system based on IABP-SHOCK II patient cohort (Pöss et al., 2017).

Variable	Points
Age >73	1
History of stroke	2
Glucose >10.7mmol/L	1
Creatinine >132.6μmol/L	1
Arterial lactate >5mmol/L	2
TIMI flow grade <3 after PCI	2
Risk Categories	Score
Low	1-2
Intermediate	3-4
High	5-9

Supplementary Table 4: Component variables of the SAVE score in patients on VA-ECMO (Schmidt et al., 2015).

Variable		Points
CS aetiology		
Myocarditis		3
Refractory VT/VF		2
Post-heart/lung transplant		3
Congenital heart disease		-3
Other		0
Age (years)		
18-38		7
39-52		4
53-62		3
≥63		0
Weight (kg)		
≤65		1
66-89		2
≥90		0
Acute pre-ECMO organ failure		
Liver failure		-3
Central nervous system (CNS) dysfunction		-3
Renal failure		-3
Chronic renal failure		-6
Duration of intubation prior to ECMO (hours)		
≤10		0
11-29		-2
≥30		-4
Peak inspiratory pressure ≤20cmH ₂ O		3
Cardiac arrest prior to ECMO		-2
Diastolic blood pressure ≥40mmHg prior to ECMO		3
Pulse pressure ≤20mmHg prior to ECMO		-2
HCO ₃ ≤15mmol/L prior to ECMO		-3
SAVE score constant		-6
TOTAL score		-35 to 17
Risk Class	Score	% survival
I	>5	75
II	1 to 5	58
III	-4 to 0	42
IV	-9 to -5	30
V	≤-10	18

Author contributions: Conceptualisation of the study, P.R. and Z.G.; literature search, P.R., W.S., J.P. and W.D.; drafting the manuscript, P.R., W.S., K.D., J.P., M.Z., W.D., P.M., J.R. and F.O.; critical revision of the manuscript, P.R., K.D. and J.R.; editing/formatting of the final manuscript, W.S., K.D., M.Z. and P.M.; creation of tables, W.S. and Z.G.; creation of in-text figures/visualisations, P.R., M.Z., W.D. and J.R.; conceptual input and guidance, Z.G., W.D., P.M. and F.O.; project coordination, P.R., J.P. and F.O.; supervision, P.R. All authors have read and agreed to the published version of the manuscript.

Conflicts of interest: The authors declare there are no conflicts of interest

Institutional Review Board Statement: Not applicable

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