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Review

Hemodynamic Support in Cardiogenic Shock in the Cardiac Catheterization Laboratory

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Abstract

Cardiogenic shock is a life-threatening, time-sensitive syndrome characterized by clinical and biochemical tissue hypoperfusion caused by circulatory failure secondary to inadequate cardiac output. Inadequate cardiac contractility secondary to acute myocardial infarction appears on the top of the list of the most prevalent etiologies of this syndrome. Despite some advances in its management, this primary cardiac disorder still has an extremely high mortality. In addition to treating the main etiology, immediate hemodynamic support is necessary to reduce the risk of developing multi-organ dysfunction and to preserve cell metabolism, as soon as we suspect it, even when needed in the catheterization laboratory. The cardiac catheterization laboratory has become a pivotal setting for implementing rapid hemodynamic support measures, such as pharmacological interventions and mechanical circulatory support, during critical procedures. Despite inotrope pharmacological treatment, mechanical circulatory support has recently garnered significant interest in this field. The aim of this review is to analyze hemodynamic support in cardiogenic shock in the most common contemporary scenario: the cardiac catheterization laboratory.

Keywords: cardiogenic shock; mechanical circulatory support; acute myocardial infarction

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1. Introduction

Cardiogenic shock (CS) is a clinical syndrome mainly defined by insufficient cardiac output due to a primary cardiac disorder, resulting in tissue hypoperfusion, that can lead to multi-organ failure and death, representing the most severe form of acute heart failure syndromes [1–3].

It represents the final common pathway of different causes. Historically, acute myocardial infarction (AMI) has been the most common cause of CS; however, in the recent decades, there has been a shift in its epidemiology, with non-AMI-related CS now predominating in some centers [4]. Cardiac damage can be either acute—due to a sudden loss of myocardial tissue, as seen in conditions like AMI or myocarditis—or progressive, as seen in patients with chronic decompensated heart failure (HF) that can deteriorate due to significant triggers [1,5].

Despite notable treatment advances that have improved prognosis, mortality remains unacceptably high, ranging from 30 to 60% in contemporary registries and clinical trials in patients who receive an adequate treatment [4,6]. As in every time-dependent process, this prognosis can be reversible if the triggering cause is identified and managed and appropriate measures are taken to achieve adequate cardiovascular support to maintain optimal systemic perfusion [7]. This involves three key steps: (i) immediate stabilization

and transfer to an intensive care unit, (ii) identification and treatment of the underlying cause, and (iii) correction of end-organ hypoperfusion to prevent or reverse organ failure [5]. The identification and treatment of the underlying cause, such as angioplasty in the case of AMI-related CS, must be performed first, alongside stabilization measures, in order to improve survival, without being postponed by non-urgent actions [8–10]. Those steps must be followed by a transfer to the intensive care unit.

Immediate hemodynamic support is crucial for restoring cellular metabolism and preventing the progression of systemic and myocardial ischemia, which can lead to a "shock spiral" that often leads to circulatory collapse and death [11,12]. Hemodynamic support can be achieved using pharmacologic agents or mechanical circulatory support (MCS). Pharmacologic therapy, consisting mainly of inotropic drugs and vasopressors, has a key role, with over 90% of patients receiving at least one vasoactive agent [5,11]. On the other hand, several temporary MCS devices have been developed to enhance hemodynamic status in affected patients [13]. Current guidelines recommend the early use of MCS for patients who do not respond to fluid resuscitation and pharmacologic agents, serving as a bridge to recovery, further evaluation, transplantation, or permanent left ventricular assistance device implantation. However, MCS implantation is associated with several challenges such as the need for specialized multidisciplinary teams and a lack of high-quality evidence regarding its impact on patient outcomes [1].

The goal of this article is to review the management of CS with a focus on hemodynamic support and, in particular, on its most common contemporary setting: the cardiac catheterization laboratory.

2. Cardiogenic Shock

2.1. Evolving Definition of Cardiogenic Shock

Classically, CS has been defined by sustained hypotension (systolic blood pressure < 90 mmHg, or the need for vasoactive medications), accompanied by tissue hypoperfusion due to a low cardiac output (cardiac index < 2.2 L/min/m^2) and congestion (pulmonary capillary wedge pressure, PCWP, $\geq$15 mmHg or pulmonary congestion on imaging) [11,14]. However, recent consensus statements from the Shock Academic Research Consortium (SHARC) and the American Heart Association have therefore expanded the definition of CS as a cardiac disorder that results in both clinical and biochemical evidence of sustained tissue hypoperfusion [15]. The motive of that changing definition has two main reasons:

1. Invasive hemodynamic assessment is not readily available on initial presentation. Therefore, the presence of clinical signs of hypoperfusion (i.e., cold clammy extremities, mental confusion, oliguria, and narrow pulse pressure) and/or biochemical manifestations of hypoperfusion (elevated creatinine in serum, metabolic acidosis, and elevated lactate in serum) in the context of a cardiac disorder is sufficient to suspect CS [5].
2. Hypotension may not be present in all patients with CS. Some patients, especially in the initial stages with preserved compensatory mechanisms, may present without hypotension and are classified as "normotensive CS" [4]. This phenotype, more common in younger patients, is associated with a higher risk for significant hemodynamic deterioration and requires increased clinical vigilance [15].

CS represents a downward spiral of worsening myocardial function and systemic hypoperfusion, particularly when it begins with an ischemic insult, such as AMI, which causes regional myocardial necrosis and results in both systolic and diastolic dysfunction. This leads to a significant reduction in cardiac output and impaired tissue perfusion. In response to diminished perfusion, reflex sympathetic activation occurs, increasing circulating catecholamines and causing systemic vasoconstriction. While initially compensatory,

this vasoconstriction exacerbates afterload and further impairs cardiac function. Simultaneously, fluid retention and an increased plasma volume (preload) contribute to elevated filling pressures, pulmonary congestion, and pulmonary edema. The combination of rising preload and afterload perpetuates a vicious cycle of worsening myocardial ischemia, reduced coronary perfusion pressure, and declining cardiac output. As cardiac output continues to fall, tissue hypoperfusion becomes more severe. Cerebral and splanchnic vasoconstriction may cause mental status changes, intestinal ischemia, and disruption of the intestinal barrier. Splanchnic vasoconstriction, driven by catecholamines, not only leads to ischemic injury to the liver and bowel, but also exacerbates volume overload by mobilizing up to 50% of the total blood volume back into the central circulation. This redistribution further strains the failing heart and worsens systemic congestion [11,16]. In parallel, the inflammatory response triggered by myocardial injury and hypoperfusion further contributes to endothelial dysfunction, capillary leak, and metabolic derangements. Once multi-organ failure ensues, even the restoration of hemodynamic parameters may not be sufficient to reverse the downward course, and the prognosis then becomes poor, often culminating in death [11,12,16].

2.2. Severity Classification

To standardize and unify the language surrounding CS, and to help with its prognosis, the Society of Cardiovascular Angiography and Interventions (SCAI) proposed a CS classification scheme in 2019 [17]. The SCAI-CS staging system categorizes patients with CS into five stages based on severity (Table 1):

- Stage A ("at risk"): stable patients with acute cardiac conditions that put them at risk of developing CS.
- Stage B ("beginning" or pre-shock): characterized by hemodynamic instability (relative hypotension or tachycardia) without obvious signs of organ hypoperfusion such as physical signs, elevated serum creatinine, metabolic acidosis, or elevated serum lactate.
- Stage C ("classic"): classic definition of CS, with obvious signs of hypoperfusion (i.e., cold extremities, elevation of serum lactate, elevation of creatine, or low urine output requiring intervention (inotropic support).
- Stage D ("deterioration"): deteriorating hemodynamic conditions despite initial treatments or supportive interventions. Advanced interventions such as mechanical circulatory support need to be considered to stabilize the patient.
- Stage E ("extremis"): extreme shock even with circulatory support or/and cardiac arrest with ongoing cardiopulmonary resuscitation (CPR).

Table 1. Severity classification of cardiogenic shock.

	Physical Examination	**Laboratory Tests**	**Hemodynamics**
SCAI A (at risk) Patient who is not currently experiencing signs or symptoms of CS.	Normal JVP Lung sound Warm and well-perfused	Normal exams	Normotensive (SBP $\geq$ 100 mmHg or normal for patient) Hemodynamic: CI $\geq$ 2.5 L/min CVP < 10 mmHg PCWP $\leq$ 15 mmHg PA saturation $\geq$ 65%
SCAI B (beginning) Relative hypotension or tachycardia without hypoperfusion.	Elevated JVP Rales in lung fields Warm and well-perfused	Normal lactate Minimal renal function impairment Elevated BNP	SBP < 90 mmHg OR MAP < 60 mmHg OR > 30 mmHg drop from baseline Heart rate $\geq$ 100 bpm Hemodynamic: CI $\geq$ 2.2 L/min PA saturation $\geq$ 65%

Table 1. *Cont.*

	Physical Examination	Laboratory Tests	Hemodynamics
SCAI C (classic) Hypoperfusion that requires intervention beyond volume resuscitation.	May include any of the following: Looks unwell, panicked, ashen, mottled, or dusky Cold, clammy Volume overload Extensive rales Killip–Kimball classification 3 or 4 Mechanical ventilatory support Cold, clammy Acute alteration in mental status Urine output < 30 mL/h	May include any of: Lactate $\geq$ 2 mmL/L Creatinine doubling OR > 50% drop in GFR Increased LFTs Elevated BNP	May include any of the following: >30 mmHg SPB drop from baseline AND drugs/device used to maintain BP above target CI < 2.2 L/min PCWP > 15 mmHg RAP/PCWP $\geq$ 0.8 PAPi < 1.85 CPO $\leq$ 0.6 W
SCAI D (deteriorating) Similar to category C but getting worse.	Any of stage C	Any of stage C AND Deteriorating	Any of stage C AND requiring multiple vasopressors OR addition of MCS devices to maintain perfusion
SCAI E (extremis) Cardiac arrest with ongoing CPR and/or ECMO, being supported by multiple interventions.	Near pulselessness Cardiac collapse Mechanical ventilation Defibrillator used	"Trying to die" CPR (A-modifier) PH $\leq$ 7.2 Lactate $\geq$ 5 mmoL/L	No SBP without resuscitation PEA or refractory VT/VF Hypotension despite maximal support

Legend: BiPAP, bilevel positive airway pressure; BNP, brain natriuretic peptide; BP, blood pressure; CI, cardiac index; CPO, cardiac power output; CPR, cardiopulmonary resuscitation; CS, cardiogenic shock; CVP, central venous pressure; ECMO, extracorporeal membrane oxygenation; GFR, glomerular filtration rate; JVP, jugular venous pressure; LFT, liver function test; MAP, mean arterial pressure; MCS, mechanical circulatory support; PA, pulmonary artery; PAPi, pulmonary artery pulsatility index, PCWP, pulmonary capillary wedge pressure; PEA, pulseless electrical activity; RAP, right atrial pressure; SBP, systolic blood pressure; VF, ventricular fibrillation; VT, ventricular tachyarrhythmia.

The revised SCAI Shock Classification highlights the spectrum of CS severity and mortality risk that exists within each SCAI shock stage [18]. Observational studies have shown a correlation between higher SCAI stages and increased mortality [13,18,19]. The mortality risk observed in patients with pre-shock (SCAI Shock stage B) and mild or normotensive CS (SCAI Shock Stage C) is not trivial, emphasizing the importance of recognizing CS early during the disease process [4].

Some lessons learned from the SCAI shock validation studies are the following:

- The distinction between SCAI shock stage B and SCAI shock stage C is critical and requires the integration of multiple clinical and laboratory exams. Patients with hypoperfusion without hypotension are at a higher risk of adverse outcomes than those patients with hypotension and normal perfusion [20]. However, this distinction may be difficult, especially in cases of mixed shock phenotypes.
- Shock classification based on required therapeutic interventions: Patients needing vasoactive drugs or MCS for hypoperfusion or hemodynamic compromise are classified as stage C. If additional vasoactive drugs or MCS devices are required, the patient progresses to stage D. If perfusion cannot be restored with multiple therapies or extremely high doses of vasoactive drugs are necessary, the patient is classified as stage E [21].
- Cardiac arrest (CA) modifier clarification: CA represents an "A modifier" in the SCAI classification. CA events are heterogeneous and currently, there is no specific CPR duration that qualifies a patient for the A modifier, which should refer to those at risk of an anoxic brain injury, defined as a Glasgow Coma Scale (GCS) score of less than 9 or the absence of a motor response to voice [22]. However, its heterogeneity and the following characteristics may be considered when making a decision: type of patient,

place of the cardiac arrest, time to return of spontaneous circulation, duration of the cardiac arrest, rhythm of the CA, etiology, and time to defibrillation. It is difficult to establish just one severity parameter in such a complex situation.

- Age might be included as a major risk for adverse outcomes that modifies risk across the SCAI shock stages [22].
- Maintaining the simplicity and flexibility of the original scale is key.
- The SCAI shock stage should be reassessed at intervals, the timing of which will depend on the initial severity: the improvement of the SCAI SHOCK stage by even one category is a powerful favorable prognostic indicator, and conversely, a maintaining or declining SCAI SHOCK is a potent negative marker [23].

However, the SCAI classification has certain disadvantages, with the most significant being that it does not consider the etiology of the CS. Its design was focused on patients presenting acutely, but acute and acute-on-chronic processes can differ in important ways as there exist physiological adaptations in chronic patients. As will be explained later, patients with decompensated HF may exhibit a different hemodynamic profile due to chronic adaptations. These compensatory mechanisms may result in a lower SCAI SHOCK stage or a deceptively stable clinical picture, despite high-risk hemodynamics [21,24].

Another limitation of the SCAI classification is that it has not undergone widespread validation in critical areas such as prehospital settings, emergency care, or among patients with chronic mechanical support or following cardiac surgery. Additionally, the absence of well-defined timelines for reevaluating the SCAI score during different phases of care could affect the reliability of mortality risk assessments [25]. Finally, disparities in how institutions interpret the system, especially regarding the criteria for initiating MCS, highlight the pressing need for greater standardization.

2.3. Cause and Pathophysiological Features

As addressed before, AMI still represents the most common cause of CS. However, over the past few decades, the epidemiological landscape of CS has shifted, with HF now surpassing AMI as the primary cause in some centers [14] Figure 1.

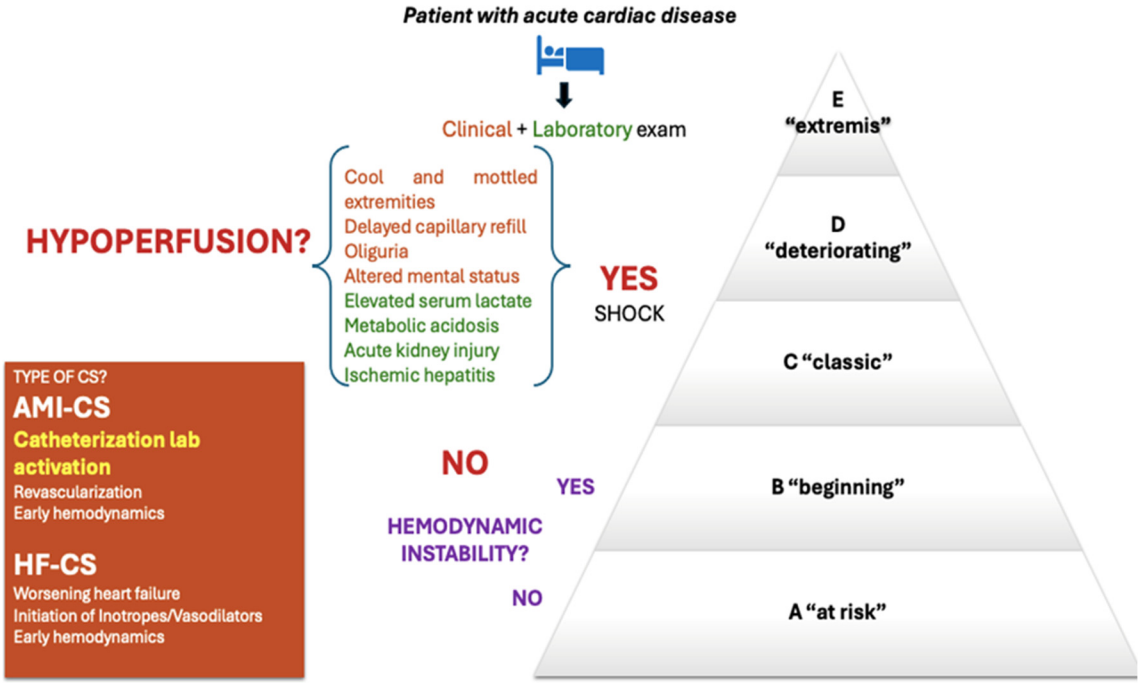


Figure 1. Cardiogenic shock: definition, severity, and etiology.

2.3.1. Acute Myocardial Infarction (AMI) Related to CS

As the population ages and frailty become more common, the incidence of acute coronary syndrome (ACS) is increasing, especially in older patients [26]. In this population, there is also a higher prevalence of complex coronary artery disease (CAD), including more cases of multi-vessel disease, chronic total occlusions, and calcified vessels [27]. Up to 10% of patients presenting with AMI are expected to have significant hemodynamic disturbances. In individuals with existing CAD, even minor ischemic damage can lead to CS, potentially compromising up to 40% of the myocardial mass [27]. Severe left ventricular dysfunction (LVEF < 35%) is the most common presentation of CS in the setting of AMI, most frequently occurring after an anterior AMI [28]. CS is a dynamic condition that may occur prior to or following reperfusion, even if those patients who successfully got through acute intervention may later develop CS [17].

The therapeutic focus of AMI-related CS is on immediate revascularization. The SHOCK trial showed that early revascularization (via percutaneous coronary intervention, PCI, or coronary artery bypass grafting) significantly improved survival rates in patients with cardiogenic shock compared to medical therapy alone, with a lower 30-day mortality rate, also long-term outcomes in survival and recovery [8]. After the results of the Culprit Lesion Only PCI versus Multi-vessel PCI in Cardiogenic Shock (CULPRIT-SHOCK) trial, revascularization of only the culprit vessel is recommended in CS patients with multi-vessel CAD [29].

2.3.2. Heart-Failure (HF) Cardiogenic Shock

This type accounts for 50% of cases and is becoming increasingly common [30]. In fact, a recent analysis from the United States showed that the incidence of AMI-related CS had decreased from 39.2% to 28.5% in the last decade [31]. Consequently, multidisciplinary teams, including cardiologists and critical care specialists, should be aware of this epidemiological shift. The use of advanced hemodynamic monitoring, such as right heart catheterization and the integration of percutaneous MCS devices, has become increasingly important. Moreover, additional intracoronary techniques performed in the cardiac catheterization laboratory, such as intracoronary imaging or functional assessment, may play a more prominent role in evaluating non-AMI causes of CS.

Multiple etiologies are associated with HF-CS, including myocarditis, takotsubo syndrome, isolated right ventricular failure, cardiomyopathies, severe valvular heart disease, post-cardiac surgery CS, and post-resuscitation shock [1]. The pathophysiology of HF-CS varies depending on whether it is de novo or an acute-on-chronic HF-CS [27].

HF-CS patients typically exhibit a slightly different profile compared to AMI-related CS. Notably, patients with chronic HF often experience reduced functional capacity and persistent pulmonary congestion due to their chronically low cardiac output. In contrast to AMI-CS, patients with acute-on-chronic HF tend to show more pronounced hemodynamic abnormalities on invasive assessment yet often maintain preserved markers of end-organ function and lack overt hypotension [30].

The clinical management of non-AMI-related CS slightly differs, emphasizing the optimization of medical therapy, such as the use of inotropes and vasopressors to support cardiac function, while addressing precipitating factors like arrhythmias or electrolyte imbalances. Early recognition remains essential for all patients with CS regardless of the underlying cause [32].

3. Therapeutic Approach to Cardiogenic Shock

3.1. Vasoactive Drugs in CS

In the context of CS, vasoactive drugs (VDs) are essential to stabilize the patient while simultaneously addressing the underlying cause, especially during the initial stages. These agents, when used judiciously, help to increase cardiac output, raise blood pressure, and improve tissue perfusion. They, however, present risks and limitations, due to potential adverse hemodynamic effects, including an increase in myocardial oxygen consumption. The balance between efficacy and potential harm must always be carefully weighed. The selection of a specific VD should be guided by the patient's hemodynamic profile, the underlying etiology of the shock, and the potential adverse effects associated with each drug [1]. VD are typically classified, depending on their mechanism of action, into inotropes, vasopressors, and inodilators (Table 2).

Table 2. Vasoactive drugs in the management cardiogenic shock.

Drug	Mechanism of Action	Dose	Advantages	Disadvantages
Dobutamine	β1 and β2 agonist, mild α1 effects at higher doses.	2–20 mcg/kg/min	Potent inotrope improving cardiac output. Mild vasodilation at low doses.	Increases myocardial oxygen consumption. Risk of arrhythmias and hypotension at high doses.
Norepinephrine	Predominantly α1 agonist with mild β1 inotropic effects.	0.05–1.0 mcg/kg/min	Potent vasoconstriction, increases SVR, and maintains coronary perfusion pressure without excessive HR increase.	Limited chronotropic effect. May cause ischemia. Increases afterload.
Epinephrine	Non-selective α1, β1, and β2 agonist.	0.01–0.5 mcg/kg/min	Potent inotropic and chronotropic effects. Useful in refractory shock.	Can induce stress cardiomyopathy. Increases lactate production.
Dopamine	α1, β1, and dopaminergic agonist.	2–20 mcg/kg/min	Useful for hypotension with concurrent low CO.	Dose-dependent effects. Higher risk of arrhythmias. No proven renal protection.
Levosimendan	Calcium sensitizer and ATP-sensitive K + channel opener.	0.05–0.2 mcg/kg/min	Increases contractility without increasing myocardial oxygen demand.	Limited data on long-term survival benefit. Hypotension risk due to vasodilation.
Milrinone	Phosphodiesterase-3 inhibitor, increases cAMP and intracellular calcium.	0.375–0.75 mcg/kg/min	Inotropic effects even in patients on beta-blockers.	Long half-life requires cautious use in renal failure. Hypotension risk due to vasodilation.
Vasopressin	V1 receptor agonist and V2.	0.03 units/min	Efficacy in acidosis and refractory hypotension.	Risk of ischemia and peripheral vasoconstriction. Risk of hyponatremia.
Phenylephrine	Pure α1 agonist.	0.5–10 mcg/min	Increases SVR without direct impact on heart rate.	Reflex bradycardia. Not suitable for patients with low-output states.

Legend. SVR: Systemic Vascular Resistance. cAMP: Cyclic Adenosine Monophosphate. CO: Cardiac Output. HR: Heart Rate.

(a) Dobutamine is considered a first-line inotropic drug in CS. It is a synthetic amine with strong β1 and β2 receptor agonist properties (3:1 ratio), making it a potent inotrope with weak chronotropic effects. This allows for increased myocardial contractility with moderate effects on heart rate. Its dosing is critical when managing cardiovascular conditions [12]. At low doses, dobutamine induces mild vasodilation through β2 activation, while at high doses, it can cause vasoconstriction via α1 receptor stimulation, potentially causing an imbalance between oxygen supply and demand, especially in ischemic conditions [33]. However, dobutamine has not shown a survival benefit in clinical trials, and moreover, concerns have arisen regarding a potential detrimental effect [34].

(b) Norepinephrine is also considered a first-line vasopressor in CS. It predominantly stimulates α1-adrenergic receptors, causing potent vasoconstriction and, to a lesser extent, β1 receptors provide a mild inotropic effect. This makes norepinephrine ideal for increasing systemic vascular resistance and maintaining coronary perfusion pressure without significantly raising heart rate, thus minimizing the risk of increasing myocardial oxygen demand. Its short half-life allows for precise titration. Studies, such as the Sepsis Occurrence in Acutely Ill Patients-II (SOAP-II) trial, have shown favorable outcomes in patients with CS, with a lower incidence of arrhythmias (24.1% vs. 12.4%, $p < 0.001$) and a trend towards reduced 28-day mortality in the cardiogenic shock subgroup (HR 0.86, 95% CI: 0.72–1.02, $p = 0.07$) compared to dopamine [35].

(c) Epinephrine is reserved for refractory shock or mixed shock due to its powerful inotropic, chronotropic, and vasoconstrictor effects. It is an endogenous catecholamine that stimulates α1, β1, and β2 receptors. Epinephrine carries significant risks, including increased lactate production and a higher likelihood of myocardial ischemia and arrhythmias. Clinical trials, such as OptimaCC (Epinephrine Versus Norepinephrine for Cardiogenic Shock After Acute Myocardial Infarction), have reported an increased incidence of refractory CS and higher mortality with epinephrine compared to norepinephrine (HR 1.75, 95% CI: 1.06–2.88, $p = 0.03$), cautioning against its use in certain populations [36].

(d) Dopamine was once a standard therapy for CS due to its mixed α1, β1, and dopaminergic receptor effects. Nowadays, it has fallen out of favor due to higher rates of arrhythmias and increased mortality, particularly at the higher doses needed for vasopressor effects. Low doses were believed to offer renal protection through dopaminergic receptor activation; nonetheless this theory has been debunked, and evidence now shows no benefit in renal outcomes contributing to its reduced use [37].

(e) Levosimendan has emerged as a promising alternative inotropic agent in CS. It enhances myocardial contractile proteins' sensitivity to calcium, improving contractility without increasing myocardial oxygen consumption. For this, levosimendan does not rely on increasing intracellular calcium or on the cyclic adenosine monophosphate (cAMP) mechanisms that are thought to underlie catecholamines' and PDE inhibitors' (PDEis) adverse effects. Levosimendan also presents vasodilatory effects, through ATP-sensitive potassium channels, reducing afterload and improving coronary perfusion. In fact, levosimendan has the fastest onset of action among cardiac inotropes, which may provide additional benefits in an acute scenario. Clinical trials, such as the RUSSLAN trial (Randomized study on Safety and effectivenesS of Levosimendan in patients with left ventricular failure due to an Acute myocardial iNfarct), demonstrated a significant reduction in mortality at 14 days compared to placebo in patients with acute heart failure (11.7% vs. 19.6%; hazard ratio 0.56, 95% CI 0.33–0.95; $p = 0.031$) [38]. The Levosimendan in Acute heart Failure following myocardial infarction (LEAF) trial showed a reduction in major adverse cardiac events (MACEs)

at 31 days compared to dobutamine (13.1% vs. 23.3%, $p = 0.049$) [39]. It is important to take into account that levosimendan may not be the ideal drug in the scenario of acute CS in the catheterization lab due to its slower onset of action (30–60 min). Some authors support its role as a preconditioning agent before high-risk angioplasty procedures, similar to its use prior to coronary bypass surgeries in patients with a depressed LVEF [40]. However, randomized clinical trials are needed to support further recommendations.

(f) Milrinone may be used in CS. It is a PDEi that shares inotropic and vasodilatory effects with dobutamine but differs in its mechanism of action. Milrinone prevents cAMP breakdown by inhibiting PDE3 receptors, leading to increased intracellular calcium and enhanced myocardial contractility. It thus offers a treatment advantage in heart failure patients on beta-blockers, as, unlike catecholamines, it does not depend on adrenergic receptor stimulation. Its vasodilatory effects reduce afterload. Its longer half-life warrants careful monitoring, especially in patients with renal dysfunction. The DOREMI (Dobutamine Compared with Milrinone) trial compared milrinone to dobutamine in patients with cardiogenic shock and found no significant difference in the primary composite outcome (in-hospital death from any cause, resuscitated cardiac arrest, receipt of a cardiac transplant or mechanical circulatory support, non-fatal myocardial infarction, transient ischemic attack or stroke, or initiation of renal replacement therapy.) (HR 0.90; 95% CI, 0.69–1.19; $p = 0.47$) [41].

(g) Vasopressin is often used as a second-line vasopressor in vasodilatory shock states, such as septic shock, but it can also be useful in CS, particularly in cases of refractory hypotension or acidosis. It is an endogenous hormone, which acts on V1 receptors inducing vasoconstriction and on V2 receptors promoting water retention. Vasopressin's effects are independent of adrenergic receptors, making it a valuable option in catecholamine-resistant shock. A study by Nguyen et al. showed that vasopressin reduced norepinephrine dose requirements in catecholamine-resistant shock (median norepinephrine equivalent dose of 0.14 mcg/kg/min vs. 0.23 mcg/kg/min, $p = 0.04$) [42]. Moreover, vasopressin has been associated with systemic vasoconstriction with relatively fewer effects on the pulmonary circulation, producing a desirable decrease in the pulmonary vascular resistance/systemic vascular resistance ratio [43].

(h) Phenylephrine is generally avoided in cardiogenic shock, due to its potential to induce reflex bradycardia and its lack of inotropic effects. It is a pure α1 agonist, which from a pathophysiological standpoint, may be beneficial during transcatheter aortic valve replacement in patients with aortic stenosis, as it may increase blood pressure without exerting positive inotropic effects [44].

3.2. Device Therapy in CS

The treatment of CS with MCS devices has evolved significantly, particularly after the introduction of VA-ECMO (Getinge, Wayne, NJ, USA) and the Impella (Abiomed, Danvers, MA, USA)®. These technologies offer crucial hemodynamic support but come with distinct challenges and risks, requiring a careful balance between their benefits and potential complications, such as hemolysis, thromboembolism, and increased afterload. The selection of MCS devices must be personalized, taking into account each patient's specific hemodynamic status and long-term prognosis (Table 3). Meticulous post-implantation care, as well as careful management, by multidisciplinary and experienced teams, particularly during patient transport or interventions such as those in catheterization lab setting, are essential to minimize complications. Continued refinement in protocols will improve patient outcomes in this complex clinical setting.

Table 3. Mechanical circulatory support devices in cardiogenic shock.

Device	Mechanism of Action	Advantages	Complications	Estimated Cost (€-euros)
IABP (Intra-Aortic Balloon Pump)	A balloon inflates in the descending aorta during diastole to improve coronary perfusion and deflates in systole to reduce afterload.	Improves coronary perfusion. Relatively low cost. Widely available.	Limited hemodynamic benefits. Risk of peripheral ischemia. Less effective in severe ventricular dysfunction.	800–1500 €
Impella 2.5 (Abiomed, Danvers, MA, USA)®	Axial flow pump that provides up to 2.5 L/min blood flow, inserted percutaneously via femoral artery.	Easy percutaneous insertion. Provides adequate support for moderate left ventricular dysfunction.	Bleeding. Hemolysis. Risk of vascular injury during insertion.	18,000–23,000 €
Impella CP (Abiomed, Danvers, MA, USA)®	Axial flow pump delivering 3.5–4.3 L/min flow, used for more severe cases with additional monitoring features (Smart Assist).	Increased flow capacity. Enhanced hemodynamic monitoring with Smart Assist system. Suited for high-risk interventions.	Similar to Impella 2.5® but with additional risks due to higher flow, such as device migration and hemolysis.	20,000–25,000 €
Impella RP(Abiomed, Danvers, MA, USA)®	Provides 4 L/min of blood flow, specifically designed for right ventricular support.	Specialized for right ventricular failure. Effective unloading of the right ventricle.	Specific for RV use. Risk of vascular injury, bleeding, and hemolysis.	22,000–25,000 €
VA-ECMO (Getinge, Wayne, NJ, USA)	Cardiopulmonary support device that oxygenates blood outside the body and returns it to arterial circulation.	Simultaneous circulatory and respiratory support. Useful in severe cardiogenic shock.	Increased ventricular afterload. Risk of renal failure. Vascular complications.	45,000–75,000 € per case, including monitoring and consumables costs.
Temporary Ventricular Overdrive Pacing (VOP)	Increases heart rate to suppress arrhythmias, effective in stabilizing electrical storms.	Quick stabilization of arrhythmias. Useful as a bridge to permanent ICD implantation.	Requires constant monitoring. Possible discomfort from pacing leads.	600–1300 €

Legend. MCS: Mechanical Circulatory Support. IABP: Intra-Aortic Balloon Pump. VA-ECMO: Veno-Arterial Extracorporeal Membrane Oxygenation. VOP: Ventricular Overdrive Pacing. RV: Right Ventricular.

(a) Intra-aortic balloon pumps (IABPs) are frequently used in CS, in situations such as mechanical complications of AMI and decompensated acute HF. The device consists of a latex or silicone-based balloon, using helium as an inert gas for inflation controlled by an external console. The IABP is inserted into the descending aorta, usually via the femoral artery. IABPs work by inflating during diastole to increase coronary blood flow and deflating during systole to reduce afterload, thus theoretically improving cardiac output. While they are widely available and affordable, as well as helpful in stabilizing hemodynamics, their efficacy is limited and it carries risks such as limb ischemia, infection, hemolysis, balloon rupture, and gas loss, thus translating into inconsistent survival benefits in CS. The IABP-SHOCK II (Intra-aortic Balloon Pump in Cardiogenic Shock) trial showed no significant reduction in mortality at 30 days (39.7% in the IABP group vs. 41.3% in the control group; RR 0.96, 95% CI: 0.79–1.17, $p = 0.69$) [45,46], leading to the 2023 European Society of Cardiology Acute Coronary Syndrome guidelines recommendation against its routine use [47]. Although IABPs have been associated with higher costs than conservative treatment, their lower upfront costs make them a more economical choice in less severe cases or when resource availability is constrained [48].

(b) Micro-axial flow pumps (Impella Abiomed, Danvers, MA, USA®) are frequently used in CS to improve cardiac output. The device is an intravascular MCS pump that is inserted into the left ventricle percutaneously or surgically. It operates by drawing blood directly from the left ventricle and pumping it into the ascending aorta. It reduces both systolic and diastolic pressure within the left ventricle, decreases afterload and myocardial oxygen consumption, and enhances systemic perfusion, promoting myocardial recovery. The different models, including Impella 2.5®, CP®, 5.0® and the latest model 5.5®, offer varying levels of flow support, delivering between 2.5 and 5.5 L/min, depending on the required circulatory assistance. Additionally, Impella RP® is designed to support right ventricular function, providing up to 4.0 L/min in cases of right ventricular failure. Although the Impella is less invasive than VA-ECMO, its ability to provide blood flow is limited, making it more suitable for patients with moderate heart failure or less severe shock. In the DanGer Shock (Danish–German Cardiogenic Shock) trial, the use of Impella CP® in patients with AMI complicated by CS, combined with standard care, significantly reduced 180-day mortality compared to standard care alone (45.8% vs. 58.5%; HR 0.74, 95% CI: 0.55–0.99, $p = 0.04$). However, there was an increase in serious complications such as hemolysis, limb ischemia, and the need for renal replacement therapy [49]. The last released model, Impella 5.5®, has shown additional survival benefits in observational studies [50]. Impella use has been associated with a lower cost compared to ECMO, and it is additionally associated with long-term cost savings by reducing the need for long-term LVAD or a heart transplant [51].

(c) Veno-arterial extracorporeal membrane oxygenation (VA-ECMO, (Getinge, Wayne, NJ, USA)) is a life-saving technique used in critical care for patients with severe CS. It works by temporarily taking over the functions of the heart and lungs. Blood is drained from the body through a large vein, passed through an oxygenator, and then pumped back into the arterial system, ensuring that oxygenated blood reaches vital organs. Despite its life-saving potential, VA-ECMO is not without risks, including complications, such as hemolysis, thromboembolism, and increased ventricular afterload caused by retrograde flow, which can potentially worsen heart failure. Recent studies have raised questions about the efficacy of VA-ECMO in improving clinical outcomes. The 2023 ECMO-CS trial found that immediate implementation of VA-ECMO did not significantly improve clinical outcomes compared to early conservative therapy in patients with CS. The composite primary endpoint (death, resuscitated cardiac arrest, or use of another mechanical support device) occurred in 63.8% of patients in the VA-ECMO group versus 71.2% in the early conservative group (HR 0.72; 95% CI 0.46–1.12; $p = 0.21$). Additionally, 30 days all-cause mortality remains high in both groups (50.0% in the VA-ECMO group versus 47.5% in the conservative group, $p = 0.21$) [52]. Similarly, the ECLS-SHOCK trial explored VA-ECMO in patients with myocardial infarction-related CS. It found no significant mortality benefit at 30 days, with 47.8% mortality in the ECMO group and 49.0% in the control group (RR, 0.98; 95% CI, 0.80–1.19; $p = 0.81$). In fact, patients receiving VA-ECMO experienced significantly more complications, including a higher risk of bleeding (OR 2,4 95% CI1.55–3.84) and peripheral ischemia (OR 3.53; 95% CI1.70–7.34) [9]. Moreover, a recent metanalysis of pooled data from four randomized trials confirmed that VA-ECMO did not reduce 30-day mortality in patients with infarct-related CS. The study reported similar mortality rates between the VA-ECMO and control groups (OR, 0.93; 95% CI, 0.66–1.29) but with higher related complications (bleeding or vascular complications). These findings suggest that while VA-ECMO can be a vital tool in managing cardiogenic shock, it should be used cautiously and reserved for carefully selected

patients, as current evidence does not demonstrate a clear survival benefit in this population. Of note, VA-ECMO should be the preferred MCS in cases of biventricular severe dysfunction, profound CS with end-organ hypoperfusion unsuitable to be managed by an Impella alone, and refractory CA, or in cases of severe respiratory compromise (such as acute respiratory distress complicating CS). Its cost-effectiveness is generally lower than other MCS devices due to higher costs and longer hospital stays, making it more justifiable in cases of severe multi-organ failure or cardiac arrest, or as a bridge to cardiac transplantation [53].

(d) Temporary ventricular overdrive pacing: although it cannot be defined as an MCS, this non-pharmacological strategy could be effective in stabilizing patients with CS secondary to a refractory electrical storm (ES) after an AMI when antiarrhythmic medications fail. It works by increasing the heart rate, which helps suppressing recurrent ventricular arrhythmias [54].

3.3. Optimal Timing

Given its complexity, providing a universal recommendation on whether to use hemodynamic support during high-risk PCI is challenging. On the one hand, early MCS has been associated with improve coronary and systemic perfusion, reducing the myocardial workload and creating a safer environment in which to perform percutaneous reperfusion. On the other hand, early PCI remains the gold standard for managing AMI-associated CS. Of particular concern is the acute total occlusion of the unprotected left main coronary artery (ATOLMCA), which, although accounting for less than 2% of all AMI cases, is a catastrophic condition with extremely high mortality rates. A recently published study has shown that, despite the progressive use of vasoactive drugs and MCS over time, patients with ATOLMCA often present with cardiac arrest and continue to have a poor prognosis [55]. In such critical scenarios, an MCS-first strategy may provide systemic stabilization, enhancing the safety of subsequent interventions. Ultimately, the choice between MCS-first or reperfusion-first strategies should be based on the patient's stability, the availability of resources and the multidisciplinary team approach.

4. Hemodynamic Support During High-Risk Percutaneous Coronary Interventions

4.1. Definition of High-Risk Percutaneous Coronary Intervention (PCI)

High-risk PCI is defined as a coronary intervention procedure with a significant increase in the risk of intra or periprocedural complications due to the patient's clinical profile, anatomical characteristics, or a combination of both [56,57] (Figure 2). In contrast, complex angioplasty specifically refers to cases where the coronary anatomy poses a significant technical challenge for the interventional cardiologist. These characteristics may include severe coronary calcifications, a high thrombus burden, marked arterial tortuosity, or the presence of chronic total occlusions. Successfully managing these conditions often requires the use of advanced techniques (e.g., plaque modification techniques such as rotablation or intravascular lithotripsy) and intracoronary imaging techniques such as optimal coherence tomography (OCT) or intravascular ultrasound (IVUS) [57,58]. In high-risk PCI, all anatomical and clinical factors involved should be carefully evaluated. This approach allows for the design of an optimal strategy that ensures the best outcomes and patient safety. CAD anatomical characteristics represent the most variable and technically challenging factor. These include multi-vessel disease, unprotected left main CAD, chronic total occlusions, extensive lesions (>60 mm), and severe calcifications [59]. Comorbidities such as chronic kidney disease, advanced age, or peripheral vascular disease significantly impact both short and long-term prognosis. However, the patient's hemodynamic status, particularly

in cases of CS, plays a crucial role in determining both prognosis and therapeutic approach, as timely intervention becomes critical [27].

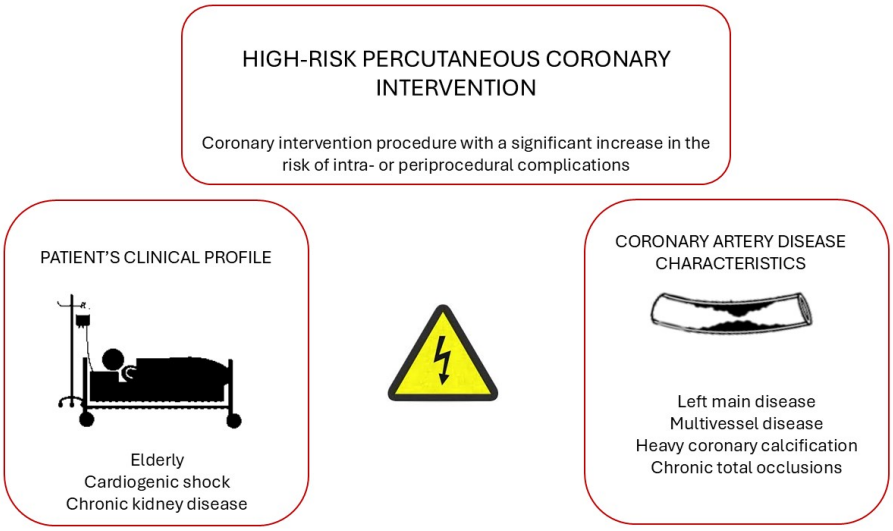


Figure 2. Definition and identification of high-risk percutaneous coronary intervention.

4.2. MCS During High-Risk PCI

Temporary MCS plays a pivotal role in the catheterization laboratory as contributes to the stabilization of cardiovascular function before and during the revascularization procedure. Currently, several MCS devices are available offering safe and effective treatment options for this high-risk population. We will focus on the use of the IABP and Impella® devices, which are the most commonly employed and available in this scenario.

(a) IABP

As addressed before, an IABP is an MCS device aimed at increasing cardiac output by providing indirect physiological assistance, reducing myocardial oxygen demand and improving coronary and cerebral perfusion. The catheter balloon tip is radiopaque, facilitating its location in the descending thoracic aorta, approximately 3 cm distal to the left subclavian artery. The most frequent size is 7.5 Fr. The femoral artery is the preferred approach for retrograde access in the descending aorta. It is the access point of choice in patients without peripheral vascular disease, and less frequently, the axillary artery or a transthoracic approach is used. Severe aortic regurgitation, aortic dissection, and severe peripheral and aortic arteriosclerosis are the main contraindications, among others. As a temporary mechanical assist device, its use is generally recommended for a duration under 7 days [60].

IABP use during high-risk PCI has been associated with improved outcomes in the balloon pump-assisted coronary intervention study (BCIS-1) trial. Elective IABP use during PCI was associated with a 34% relative reduction in all-cause mortality compared with unsupported PCI [61]. Moreover, pre-operative IABP use in high-risk patients reduces short- and middle-term mortality rates, along with a notable decrease in rates of severe respiratory disorders [62].

(b) Microaxial flow pump (Impella, Abiomed, Danvers, MA, USA®)

This percutaneous MCS device provides continuous, non-pulsatile, laminar flow through a microaxial pump. It effectively unloads the left ventricle, reducing end-diastolic pressure and myocardial oxygen demand, which improves coronary perfusion and optimizes cardiac output [53]. The cannula's distal end, which serves as the inlet, should be positioned 3.5 cm from the aortic valve plane, while the outlet end should be placed in the

ascending aorta. Three catheters are available for percutaneous insertion (the Impella 2.5 [12 Fr, maximum flow 2.5 L/min], CP [14 Fr, 3.5 L/min] and RP [for right ventricular, RV unloading into the pulmonary artery, 23 Fr, 4.0 L/min]). In high-risk PCI, the Impella CP is the most widely used. Table 3 provides more information regarding specific characteristics of different types of Impella devices. The most common complications related to Impella support are hemorrhagic events secondary to hemolysis, peripheral vascular complications and stroke. Specially, the Impella had a higher incidence of bleeding complications when compared to IABP has been described [63–66].

Microaxial flow pump MCS use has been studied in high-risk PCI patients in the PROTECT II trial (Prospective, Multi-center, Randomized Controlled Trial of the IMPELLA RECOVER LP 2.5 System Versus Intra-Aortic Balloon Pump in Patients Undergoing Non-Emergent High-Risk PCI). This prospective, multicenter, randomized clinical trial enrolled 452 patients with high-risk PCI, defined by triple-vessel disease or unprotected left main CAD with severely depressed systolic function. Patients were randomized to either Impella 2.5® or IABP. The trial was prematurely stopped due to futility as there were no clinical differences at 30 days, whereas a trend toward better outcomes and better hemodynamic profile was observed at 90 days for the Impella-supported patients [67,68] Similar results were observed in a retrospective study in which a protected high-risk PCI with the Impella 2.5 device strategy was compared to an unprotected approach in patients with multi-vessel CAD and severely reduced LVEF. MCS supported high-risk PCI show similar in- hospital and long-term outcomes and post- procedural adverse events compared to unprotected PCI [69]. The observational PROTECT III trial demonstrated improved revascularization rates, fewer bleeding events, and better cardiovascular outcomes at 90 days in patients undergoing Impella-assisted high-risk PCI, compared to those in the PROTECT II trial population [70]. Currently, a large RCT of Impella-assisted high-risk PCI versus unassisted high-risk PCI, the PROTECT IV trial (Impella-Supported PCI in High-Risk Patients With Complex Coronary Artery Disease and Reduced Left Ventricular Function) is underway [71].

Current clinical practice guidelines recommend elective insertion of an MCS as an adjunct to PCI in high-risk patients with a class 2B recommendation [72]. However, 2023 European guidelines do not routinely recommend the use of MCS based on observational data until further clinical trial evidence is available [47]. On the other hand, management of high-risk PCI with IABP is more cost-effective than the routine use of percutaneous LVADs. The use of IABP as initial therapy in high-risk PCI and cardiogenic shock patients may result in savings of up to $2.5 billion annually in incremental costs to the hospital system [73].

To date, no randomized clinical studies have compared ventricular mechanical assist devices with standard therapies. The CHIP-BCIS3 trial is a prospective, multicenter open-label randomized study designed to access whether elective percutaneous left ventricular unloading is superior to standard care in patients undergoing high-risk, non-emergent PCI. Eligible patients must have severe left ventricular dysfunction and extensive CAD. Exclusions include cardiogenic shock and acute ST-segment elevation AMI. The primary outcome is a composite of death, stroke, myocardial infarction, and other cardiovascular events, analyzed by the win ratio. Trial results are expected in 2025 [74]. Until then, observational studies have shown that prophylactic MCS with an Impella CP or ECMO prevents hemodynamic instability in high-risk PCI patients [75]. However, the optimal timing for initiating MCS remains uncertain, with slightly better outcomes observed in patients receiving MCS before PCI [76,77]. Recently, an algorithm was proposed to identify patients who may benefit most from mechanical circulatory support (MCS) during high-risk percutaneous

coronary interventions (PCI). This algorithm incorporates clinical presentation variables, comorbidities, and anatomical factors. However, external validation is still needed [78].

4.3. MCS in Valvular Heart Disease Percutaneous Procedures

An area that remains unexplored is the use of MCS devices during percutaneous valvular procedures. According to data from the US National Registry, which includes approximately 60,000 patients undergoing transfemoral percutaneous aortic valve replacement (TAVI), only 2.8% of these patients received MCS emergently or electively [79]. Among those, an IABP was the most commonly used device, accounting for 87% of cases, followed by a microaxial flow pump (Impella®) and extracorporeal membrane oxygenation (ECMO), each representing 6% of cases [80].

The use of an IABP during a TAVI has yielded mixed results in observational studies. Some data demonstrate its safety during the procedure, along with the feasibility of early device removal, while other studies report an association with increased adverse outcomes and higher hospitalization costs in this subgroup.

Historically, severe aortic stenosis was considered a contraindication to MCS using microaxial flow pump devices due to potential mechanical interference with the calcified valve leaflets. However, observational data has shown promising results in this scenario, especially after the development of a new generation of devices with higher ventricular support [55,81]. Given current evidence, prophylactic or periprocedural Impella implantation is technically possible and may help stabilize the patient's status. Nonetheless, randomized clinical trials assessing the use of IABPs and Impella devices in this context are still needed.

5. Future Directions

Randomized clinical trials are essential in advancing the management of CS, as they provide robust evidence to guide interventions and improve outcomes. Of note, novel pharmacological agents such as istaroxime (NCT04325035) and intravenous potent antiplatelet therapies (NCT03551964) in AMI-related CS are being studied in randomized clinical trials. Moreover, developing a "cardiogenic shock code" protocol will minimize inequity in access to MCS and improve survival of CS patients [7]. Finally, the formation of multidisciplinary teams including cardiologists, intensivists, and emergency physicians will ensure a comprehensive and coordinated approach, ultimately improving the prognosis of this population [4].

6. Conclusions

Cardiogenic shock remains a high-mortality, time-sensitive condition, with acute myocardial infarction being the leading cause. Early identification, revascularization, and tailored hemodynamic support, including vasoactive drugs and mechanical circulatory support (MCS) devices are crucial for management despite associated risks and costs. Emerging MCS applications, such as in valvular disease, hold promises for improving outcomes, highlighting the need for ongoing research and patient-specific strategies.

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Abbreviations

AMI	Acute myocardial infarction
BiPAP	Bilevel positive airway pressure
BNP	Brain natriuretic peptide
BP	Blood pressure
CAD	Coronary artery disease
CI	Cardiac index
CPO	Cardiac power output
CPR	Cardiopulmonary resuscitation
CS	Cardiogenic shock
CVP	Central venous pressure
ECMO	Extracorporeal membrane oxygenation
GCS	Glasgow coma scale
GFR	Glomerular filtration rate
HF	Heart failure
IABP	Intra-aortic balloon pump
IVUS	Intravascular ultrasound
JVP	Jugular venous pressure
LFT	Liver function test
LVAD	Left ventricular assist device
LVEF	Left ventricular ejection fraction
MAP	Mean arterial pressure
MACEs	Major adverse cardiac events
MCS	Mechanical circulatory support
OCT	Optimal coherence tomography
PA	Pulmonary artery
PAPi	Pulmonary artery pulsatility index
PCI	Percutaneous coronary intervention
PCWP	Pulmonary capillary wedge pressure
PEA	Pulseless electrical activity
PDEi	Phosphodiesterase inhibitor
RAP	Right atrial pressure
SBP	Systolic blood pressure
SCAI	Society of Cardiovascular Angiography and Interventions
VF	Ventricular fibrillation
VD	Vasoactive drugs
VT	Ventricular tachyarrhythmia
cAMP	Cyclic adenosine monophosphate

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