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PROTOCOLS AND GUIDELINES 2025-2026 ON THE MANAGEMENT OF CARDIAC SHOCK: FROM ASSESSMENT TO HEMODYNAMIC SUPPORT

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Abstract: Cardiogenic shock is one of the leading causes of mortality in Brazil, as the main causative condition is acute myocardial infarction (AMI), and its management remains a challenge despite advances in therapeutic options. It is caused by severe impairment of myocardial performance, resulting in decreased cardiac output (CO), hypoperfusion and target organ hypoxia. In this context, clinically, it presents as hypotension refractory to volume replacement, with characteristics of hypoperfusion of target organs that require pharmacological or mechanical intervention. AMI accounts for 81% of cases of cardiogenic shock. This article aims to conduct a literature review to report on the 2025 update of the *American College of Cardiology* (ACC) guidelines, the pharmacological and mechanical therapeutic options cited, and their applicability in practice. The scientific databases used were: *Scientific Electronic Library Online* (Scielo), *National Library of Medicine* (NIH), *PubMed*, whose definition was performed by the Health Sciences Descriptors (DeCS) using keywords such as "Cardiogenic Shock," "*American College of Cardiology*," "Vasoactive Drugs," "Mechanical Circulatory Support Devices," using the Boolean operators AND and OR. Articles used were from 2021 to 2026. The ACC guideline refers to a standardization of institutional care for cardiogenic shock, however, it is a fundamental challenge, in addition to repeated assessment and rapid decision-making. First-line interventions to stabilize patients in cardiogenic shock typically include optimization of volume status and administration of vasoactive and inotropic agents. However, evidence on the selection of medications for circulatory support, complication rates, standardized assessment of medication failure, and treatment goals

in cardiogenic shock is limited. Titration of catecholamines to the lowest dose that maintains adequate organ perfusion is advisable to avoid unwanted adrenergic effects. Mechanical circulatory support devices can provide partial or total circulatory support and may have differential effects on myocardial oxygen consumption, reduction of left ventricular (LV) overload, LV wall stress, or coronary artery perfusion, as well as support for right ventricular (RV) or biventricular dominant shock. Although it is the most widely used temporary mechanical circulatory support device in many centers, few data demonstrate a clinical or hemodynamic benefit of intra-aortic balloon pump in cardiogenic shock. It can be concluded that early application of mechanical circulatory support in stages A-B in the 2025 ACC guideline reduces mortality, but requires more precise studies of the target population and inclusion of other types of cardiogenic shock. Among the most commonly used vasoactive drugs mentioned were norepinephrine, dobutamine, and epinephrine.

INTRODUCTION

Cardiogenic shock is a state of ineffective cardiac output (CO) caused mainly by cardiac dysfunction, resulting in insufficient perfusion of target organs, with a high mortality rate of 40% to 50% (Na et al., 2022). According to the *American Heart Association* (AHA),(2023) the compensatory physiological mechanisms of cardiogenic shock include endogenous sympathetic stimulation, which increases CO by elevating heart rate (HR) and myocardial contractility. In addition to the direct cardiac effects produced by these endogenous compounds, peripheral vasoconstriction contributes to increased systemic vascular resistance (SVR) and mean arterial pressure (MAP).

Although vasopressor therapy plays a key role in maintaining hemodynamic status in the treatment of patients with cardiogenic shock, identifying and treating the underlying etiology that caused cardiogenic shock is essential for the management and conduct (Na et al., 2022) , as presented in the 2025 *American College of Cardiology* (ACC) guideline, according to the Society for Cardiovascular Angiography and Interventions (SCAI) classification of cardiogenic shock severity, early revascularization is associated with improved clinical outcomes in cardiogenic shock secondary to acute coronary syndrome (ACS) (*et al*., 2025) .

Cardiogenic shock can be classified by its dominant involvement, whether left ventricular (LV), right ventricular (RV), or biventricular. Among the etiologies, acute myocardial infarction (AMI) is the most frequent cause of cardiogenic shock, with an incidence of 5% to 15% and mortality greater than 50% (*et* al., 2025) . Meanwhile, heart failure (HF) is estimated at around 8.3% in adults over 65 years *of age*., 2024) .

Therefore, this article aims to analyze the 2025 ACC guidelines on the management and conduct of cardiogenic shock, the use of vasoactive drugs in these patients, and mechanical circulatory support devices.

METHODOLOGY

This article is a narrative literature review using scientific databases such as *Scientific Electronic Library Online* (Scielo), *National Library of Medicine* (NIH), and *PubMed*, which were defined by the Health Sciences Descriptors (DeCS) using keywords such

as "Cardiogenic Shock," "*American College of* Cardiology," "Vasoactive Drugs," and "Mechanical Circulatory Support Devices," using the Boolean operators AND and OR. The inclusion criteria adopted were: articles published between 2021 and 2026; articles included outside this period were those with relevant and important results for development; articles in English and Portuguese; articles with free access; original articles and review articles.

The exclusion criteria were: articles in languages other than English and Portuguese, articles addressing protocols and therapies related to the pathology, paid articles, articles that did not demonstrate relevant data, duplicate articles, theses, and monographs. The articles were selected and filtered after reading the title, abstract, and text of the articles. After applying these filters, 33 articles were found, and 26 articles were selected for this study.

RESULTS AND DISCUSSION

Definition of cardiogenic shock

In this context, cardiogenic shock is considered to be one of the three hemodynamic phenotypes of circulatory shock, observed as a complex syndrome that progresses with hypoperfusion due to primary cardiac injury with decreased CO, neurohormonal activation, compensatory systemic vasoconstriction, and elevated systemic vascular resistance (*et* al., 2025) . These compensatory responses occur at the expense of maladaptive increases in cardiac afterload, myocardial oxygen demand, and filling pressures, resulting in reductions in coronary perfusion pressure (AHA, 2023).

Key updates to the *American College of Cardiology* (ACC) guideline on Cardiogenic Shock

The ACC guideline emphasizes the use of the SUSPECT mnemonic to aid in the diagnosis of cardiogenic shock, as evidenced in Table 1. SUSPECT (Signs/symptoms, urine output, sustained hypotension, perfusion, ECG/Echocardiogram (ECO), congestion, and screening) consists of an introductory assessment of symptoms/signs, consisting of symptoms consistent with signs of shock, hypoperfusion, low CO, and target organ involvement, such as altered level of consciousness, confusion, precordial pain or pressure, cold, clammy extremities, rapid pulse, low pulse pressure (<25% of systolic blood pressure [SBP]), elevated jugular venous pressure, rales, crackles, orthopnea, paroxysmal nocturnal dyspnea, lower limb edema, as well as oliguria or anuria considered as <30mL/h, SBP <90 mmHg, mean arterial pressure (MAP) <65 mmHg for >30 min or decrease >30 mmHg from baseline, as well as metabolic and perfusion changes such as: lactic acid >2 mmol/L, ALT >200 U/L or >3× the upper limit of normal, creatinine ≥2 times the upper limit of normal, pH <7.2, metabolic acidosis, ECG or ECHO evaluation for the presence of acute ischemia, cardiac chamber dilation, and signs of congestion (Sinha *et al.*, 2025) .

Therefore, the guideline emphasizes that the initial suspicion and diagnosis of cardiogenic shock does not necessarily require more invasive tests; however, these are necessary to assess the patient's ventricular function and congestion (*et* al., 2025) . In addition, according to Martelli and Tiberio(2025) , the study was conducted mainly in patients hospitalized for cardiogenic shock secondary to myocardial ischemia with

Symptoms/Signs:	Altered mental status, confusion, chest pain or pressure, cold and clammy extremities, rapid pulse, low pulse pressure (<25% of SBP), elevated jugular venous pressure, rales, crackles, orthopnea, paroxysmal nocturnal dyspnea, lower limb edema.
Ur*ine output*:	Oliguria or anuria, <30 mL/h (<0.5 mL/[kg·h])
Sust*ained hypotension*:	SBP <90 mmHg, MAP <65 mmHg for >30 min or decrease >30 mmHg from baseline, or need for pharmacological or mechanical support to maintain SBP >90 mmHg.
Perfusion:	Assess markers of poor target organ perfusion, including lactic acid >2 mmol/L, ALT >200 U/L or >3× the upper limit of normal, creatinine ≥2× the upper limit of normal, pH <7.2, metabolic acidosis with no other known cause.
ECG/Echocardiogram:	Assess acute ischemia, including ECG and ultrasonographic evidence of STEMI (regional wall motion abnormalities); evidence of LV or RV dilation and systolic dysfunction; valve pathology.
Congestion:	Presence or absence of congestion based on physical signs and hemodynamics; clarification of ventricular involvement (LV vs. RV vs. BiV).
Screening:	Activation of the appropriate triage/shock team or possible transfer to a higher level of care.

ALT = alanine aminotransferase; BiV = biventricular; ECG = electrocardiogram; LV = left ventricle; MAP = mean arterial pressure; RV = right ventricle; SBP = systolic blood pressure; STEMI = ST-segment elevation myocardial infarction.

Table 1. SUSPECT: A mnemonic to aid in confirming the diagnosis of cardiogenic shock.

Source: ACC, 2025.

ST elevation and depression verified on ECG and cardiac abnormalities such as elevated natriuretic peptides, pulmonary congestion, or systemic congestion. important factors considered in the guideline were the inclusion of hypotension and hypoperfusion in conjunction with studies conducted on patients with congestion and reduced cardiac output with a pulmonary artery catheter; therefore, medical history and physical examination remain extremely important in the initial assessment of the patient.

In this context, although the guideline reports that the best *database* was used for the study, there are still other etiologies that would be necessary to complement it. In addition, the study's evaluation dynamics are organized and close to the actual evolution of patients, but it would not be applicable to low- and medium-complexity hospitals. Additionally, according to da Silva *et al*.,(2026)conduzida nas bases de dados PubMed, SciELO, ScienceDirect, LILACS e Consensus.app, utilizando descritores controlados e combinações booleanas. Após aplicação dos critérios de inclusão e exclusão, foram selecionados 40 artigos. Os resultados foram organizados em quatro eixos temáticos: estratégias de revascularização e suporte hemodinâmico precoce; utilização de suportes circulatórios mecânicos; inovações tecnológicas e modelos preditivos; e diretrizes e consensos clínicos recentes. As evidências indicam que a revascularização

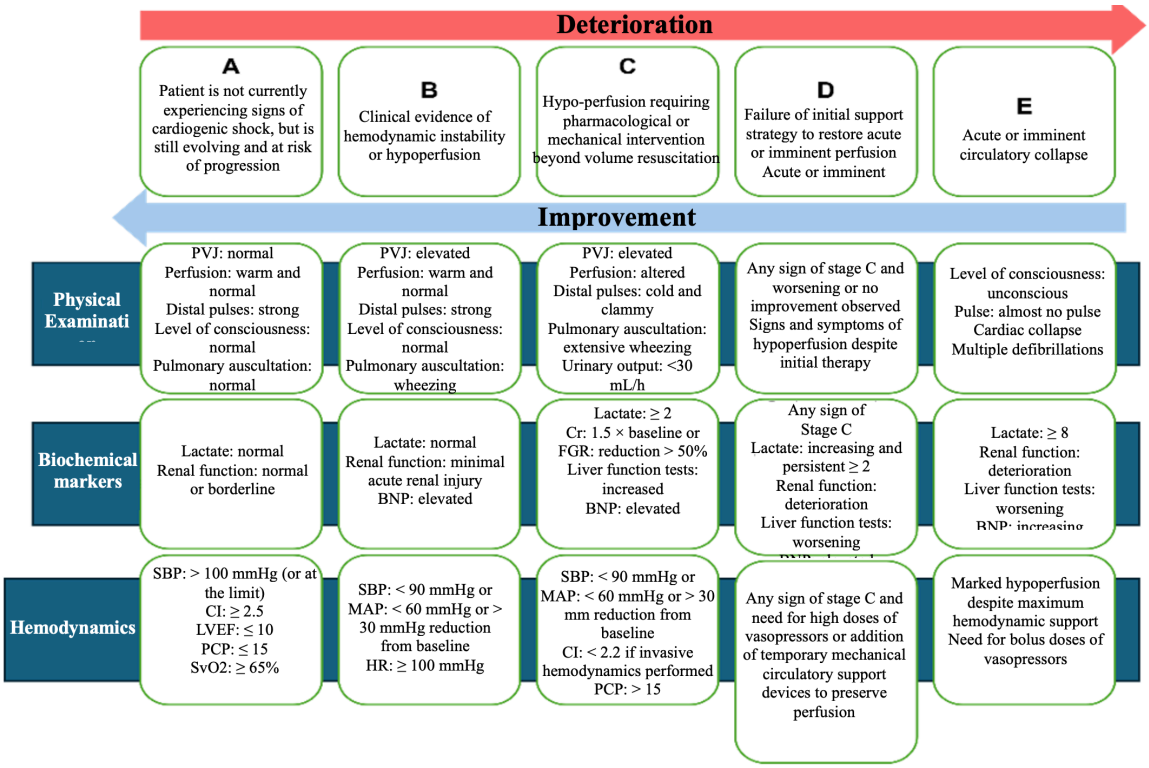
precoce e o uso racional de dispositivos de suporte circulatório, aliados à atuação de equipes multidisciplinares (Shock Teams , early coronary revascularization is still the main therapeutic approach for patients who have developed cardiogenic shock due to acute coronary syndrome (ACS), as found in the CULPRIT-SHOCK study, while the use of a pulmonary artery catheter, with monitoring of cardiac index, pulmonary capillary pressure, and central venous saturation remain important factors for preventing complications in these patients, as well as the adjustment of vasoactive drugs such as inotropes and vasopressors.

According to Sinha *et al.* (2025), the initial assessment of the affected patient should be performed within 30 minutes, with ECG and ECHO evaluation, with screening being the last step and guiding the next course of action. From 60 to 90 minutes, initial shock management should be chosen, and if a complex level of care is essential, the patient should be transferred within the hospital or to the catheterization laboratory, where approaches such as percutaneous coronary intervention, coronary angiography, femoral angiography, right heart catheterization, or temporary mechanical circulation support will be performed. Echo findings that should be considered were: decreased systolic function of the right ventricle (RV) or left ventricle (LV), cardiac tamponade, or acute valvular heart disease.

In addition, within 6 to 24 hours, priority should be given to stabilizing the patient, therapy for pulmonary congestion, metabolic and hemodynamic disorders, personalized interventions such as coronary artery bypass grafting, heart transplantation, or palliative treatment (Souza *et al.*, 2025) .

The ACC guideline also emphasizes the use of the SCAI severity classification criteria, as shown in Figure 2, translated from the original guideline, which ranges from A to E in terms of severity. Three main parameters are used: physical examination, jugular venous pressure measurement, perfusion, distal pulses, altered level of consciousness, pulmonary auscultation, urinary output, signs of hypoperfusion, and evaluation of biochemical markers such as lactate, creatinine, glomerular filtration rate, liver function, brain natriuretic peptide (BNP), acidosis (pH), and invasive hemodynamic assessment through mean arterial pressure (MAP), central venous pressure (CVP), pulmonary capillary pressure, pulmonary artery oxygen saturation (SvO2), and al., 2025) .

According to Kapur et al.,(2022) during the initial assessment and physical examination, findings such as lethargy, confusion, altered mental status, cold and clammy extremities, prolonged capillary refill time (>2 s), and reduced urine output (<30 mL/h or <0.5 mL/kg/h), even in the absence of hypotension, should also raise suspicion of acute coronary syndrome within the parameters discussed previously. Also according to Jentzer *et al.*,(2025) respiratory status should be assessed for additional signs of congestion or volume overload such as tachypnea, orthopnea, decreased arterial oxygen saturation, as well as possible symptoms of early progression such as nausea, vomiting, abdominal pain, early satiety, and decreased appetite, reflecting evidence of gastrointestinal ischemia due to inadequate cardiac output, although these signs are nonspecific and should be considered with the patient's main clinical presentation.



BNP: brain natriuretic peptide; CI: cardiac index; Cr: creatinine clearance; CVP: central venous pressure; GFR: glomerular filtration rate; HR: heart rate; JVP: jugular venous pressure; ALT ¼ liver function test; MAP: mean arterial pressure; PPC: pulmonary capillary pressure; SBP: systolic blood pressure; SCAI: Society for Cardiovascular Angiography and Interventions;

Table 2: Figure translated from the SCAI group criteria for severity classification in cardiogenic shock from the 2025 ACC guideline

Source: ACC, 2025 and Kapur *et al.*, (2022)

According to Dil *et al.*,(2025) the criteria for SCAI Stage A described above remain ambiguous, as the studies did not specifically define extensive myocardial infarction (EMI), neglected the impact of infarct location, and did not include microvascular dysfunction and systemic inflammation in the progression of the disease in the study design. In this context, patients with broad pathophysiology and opposing risks ranging from minimal to critical are included, in addition to the lack of specific biomarkers to identify patients at real risk of shock progression. Although it is important to reassess the patient's condition, such as continuous monitoring of lactate, hemodynamics, and biochemical markers, in countries such as Brazil, this does not occur in practice, as there are still places where adequate prevention cannot be performed in patients with significant family history and phenotype. in addition to multiple issues within the hospital and emergency room environment, such as transportation, intra-hospital transfer documentation and bureaucratic issues, as well as the lack of advanced medications for ventilatory and hemodynamic management for these patients, and the absence of blood gas analysis devices in the units described.

Furthermore, in the context of invasive hemodynamics, despite the use of SvO2 and PCP as parameters, there are no rando-

Category	Agent(s)	Mechanism of Action/Receptor Binding	Dosing	Hemodynamic Effects			
				SVR	BP	CO	HR
Inopressor	Norepinephrine	α1 (+++), β1 (++), β2 (+)	0.05-1 µg/kg/min	↑↑	↑↑	↑	↑
	Epinephrine	β1 (+++), α1 (++), β2 (++)	0.01-0.5 µg/kg/min	↑↑	↑↑	↑↑	↑↑
	Dopamine	D1 (+++), β1 (++), α1 (+)	Low: 2-5 µg/kg/min Intermediate: 5-10 µg/kg/min High: 10-20 µg/kg/min	↑↑	↑↑	↑	↑↑
Inodilator	Dobutamine	β1 (+++), β2 (++)	2-10 µg/kg/min	↓↔	↓↔	↑↑	↑
	Milrinone	PDE-3 inhibitor	0.125-0.5 µg/kg/min	↓↓	↓↓	↑↑	↔↑
Vasopressor	Phenylephrine	α1 (+++)	0.1-10 µg/kg/min	↑	↑↑	↔↓	↔↓
	Vasopressin	Vasopressin receptor	0.01-0.04 U/min	↑↑	↑↑	↔↓	↔↓
Vasodilator	Nitroprusside	NO production	0.3-10 µg/kg/min	↓	↓	↑↔	↑↔
	Nitroglycerin	Converts to NO	25-200 µg/min	↓	↓	↑↔	↑↔
Chronotrope	Isoproterenol	β1 (+++), β2 (+++)	2-20 µg/min	↓	↔	↑	↑↑
	Dopamine	See above					
Inotrope	Levosimendan*	Binds to troponin C, making it more sensitive to calcium thereby improving interaction between troponin C and I	0.05-0.2 µg/kg/min	↓	↓	↑	↔

*Not approved by the Food and Drug Administration (FDA) for clinical use in the United States. ↑ increased effects; ↓ decreased effects; ↔ neutral effects; +++ strong binding; ++ moderate binding; + weak binding; α1 receptor a-1; ß1 receptor b-1; ß2 receptor b-2; BP: blood pressure; CO: cardiac output; D1 ¼ D1 receptor; HR: heart rate; NO: nitric oxide; PDE-3: phosphodiesterase 3; SVR: systemic vascular resistance.

Table 3: Vasoactive agents used in cardiogenic shock

Source: ACC, 2025.

mized studies with results in a defined population using pulmonary artery catheters in the context of ACS. The results of the ESCAPE study (*Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness*) were conducted in patients with severe heart failure (*et al.*, 2025) .

The guideline also addresses pharmacological treatment, with a primary focus on treating congestion with thiazide diuretics, intravenous loop diuretics, and renal replacement therapy if indicated, due to microcirculatory ischemia, optimizing cardiac output, and increasing perfusion of vital organs, while hypoperfusion can be treated with intravenous administration of vasoactive drugs, such as inotropics, chronotropics, inopressors, inodilators, vasodilators, and vasopressors, as shown in Table 3.

Regarding mechanical circulatory support devices, the guideline is based on the severity classification of circulatory shock, whether it is of AMI or HF origin, as well as the assessment of the presence of arrhythmias, hypoxia, contraindications to the use of certain devices, and the use of intravenous antiplatelet agents (Sinha *et al.*, 2025) .

If the patient's profile is SCAI B and in the initial period, regardless of the type of cardiogenic shock (LV, RV, or biventricular involvement), guidelines for the use of vasoactive agents may be considered. The use of these devices remains uncertain, while with SCAI C classification, treatment differs depending on the involvement. If LV involvement is dominant, the use of intra-aortic balloon therapy or the Impella CP device - low-profile transcatheter (ICP) or Impella 5.5 (I5.5) (SmartAssis - short-term microaxial heart pump) should be considered. If it is dominant for the RV or biventricular, Pro-Tek Duo/CentriMag (PTD/CM) (double-lumen device introduced through

the right internal jugular vein) or Impella RP Flex (IRP) or I5.5 should be considered if cardiogenic shock consists of ACS I. If it is dominant for the LV, ICP or I5.5 or temporary transapical or transseptal LV assist device; if it is dominant for the right side, opt for extracorporeal venoarterial oxygenation or LV ventilation or I5.5 or PTD/CM or IRP if it is SCAI E. There are two options: extracorporeal venoarterial oxygenation or LV ventilation (Sinha *et al.*, 2025) .

Pharmacological treatment and the use of vasoactive drugs in cardiogenic shock

According to the study by Greenwood *et al.*,(2025) early administration of inotropes within 8 hours after classification using SCAI criteria resulted in a lower need for vasopressors, higher peak lactate levels, and reduced mortality, despite similar disease severity scores and baseline cardiac function among the patients analyzed. Cardiac calcitropics, such as catecholamines and phosphodiesterase 3 inhibitors, increase intracellular calcium concentration to increase contractility and are considered to have a direct action, as opposed to secondary effects on chronotropy and vascular tone (Sinha *et al.*, 2025) .

Although inotropics are beneficial in the treatment of cardiogenic shock, in Takotsubo syndrome, if the etiology is due to LV outflow tract obstruction (LVOTO), catecholaminergic inotropics are contraindicated, and the preferred treatment would be fluid therapy and short-acting beta-blockers (*et al.*, 2026)2026 .

According to Fibiger *et al.*,(2025)sendo a principal causa de morte em pacientes com infarto agudo do miocárdio (IAM dobutamine is a synthetic catecholaminergic inotropic agent that increases cardiac contractility through its stimulatory effect on myocardial beta-1 receptors. It also affects the peripheral vasculature due to its combined action on vascular alpha-1 and beta-2 receptors. In clinical practice, low doses of dobutamine (<5 μg/kg/min) for patients with acute heart failure lead to increased cardiac output through increased inotropy and reduce afterload by exerting a vasodilator effect on the peripheral arterial vasculature, with rapid action in emergencies, but it can also have adverse effects such as increased HR and arrhythmias (Guerra *et al.,*2024).

In patients with chronic kidney disease (CKD) who underwent stress testing with magnetic resonance imaging (MRI) under dobutamine administration, adverse events such as life-threatening arrhythmias, AMI, or death were reported in up to 1.0% of patients (*et* al., 2023)the best non-invasive approach to assess CAD in these patients remains unclear. We sought to investigate the accuracy, safety, and prognosis of patients with severe CKD undergoing dobutamine stress cardiac magnetic resonance imaging (CMR . Among other effects associated with dobutamine, there are others that are due to sympathetic hyperstimulation, such as chest discomfort, palpitations, tremors, headache, dyspnea, hypertension, nausea, vomiting, and eosinophilic myocarditis.

According to Mahadevappa *et al.*,(2024) myoclonus is a neurological side effect that can be caused by dobutamine infusion, specifically in patients with CKD. In addition, through another study conducted with a patient with heart failure, CKD, and psychiatric disease, it was proven through the neurology department that the myoclonus was caused by a side effect of the me-

dication caused by the administration of dobutamine. The symptoms ceased shortly after the suspension of dobutamine, and the patient was discharged with a regimen of milrinone and amiodarone (Sunnaa *et al.*, 2024) . However, the patient did not survive after another cardiogenic shock. It can be seen that there are still no studies or data in the literature to support patients who present this type of adverse effect and associated pathologies.

While chronotropic agents increase CO predominantly by increasing HR, and inopressors such as dopamine, norepinephrine, and epinephrine increase CO and SVR, the administration of norepinephrine, a more potent vasoconstrictor than dopamine, is suggested in cases of marked hypotension with systolic blood pressure < 70 mmHg. In 2010, the *Sepsis Occurrence in Acutely Ill Patients* (SOAP II) study included a randomized clinical trial comparing the use of dopamine and norepinephrine in patients with shock. The study concluded that the 28-day mortality rate was higher among patients with prespecified cardiogenic shock (n = 280) treated with dopamine than those treated with norepinephrine, considered to be approximately 110 fewer deaths per 1000 patients (Hosoya *et al.*, 2025) .

The AHA and ACC guidelines recommend norepinephrine as the first choice for maintaining a MAP > 65 mmHg in patients with AMI in cardiogenic shock. However, they did not report survival with the use of norepinephrine for cardiogenic shock in clinical settings, noting that there was a lower incidence of refractory shock but comparable mortality(AHA, 2019) .

However, according to a study conducted by the AHA in 2025, Patel *et al.*,(2025) epinephrine, dopamine norepinephrine was common to all combinations, the three main ones being norepinephrine-epinephrine, norepinephrine-vasopressin, and norepinephrine-dobutamine. Therefore, this suggests that physicians generally use norepinephrine as a first-line agent, with variability in the selection of the secondary vasoactive agent (inopressor, vasopressor, inodilator) determined by the physiology of the shock manifested. To this end, we observed greater use of norepinephrine-dobutamine in patients with HF with cardiogenic shock, a phenotype that may more commonly present with depressed CD, but with preserved vascular tone, compared to the greater use of norepinephrine-epinephrine in patients with AMI with cardiogenic shock or de novo HF with cardiogenic shock, both subtypes in which acute target organ damage occurs.

Complementing the above, according to Bougouin *et al.*,(2022) in a study of 766 patients in 5 hospitals, among patients with post-resuscitation shock after out-of-hospital cardiac arrest, the use of epinephrine was associated with higher all-cause mortality and specific cardiovascular mortality, compared to norepinephrine infusion.

Regarding dopamine, Na *et al.*(2022) described in their study that the use of norepinephrine as a first-line vasopressor was not associated with reduced hospital mortality or early recovery of hemodynamic parameters, but may reduce the use of additional vasopressors compared to the use of dopamine in patients with cardiogenic shock.

Inodilators such as milrinone and dobutamine increase CO and reduce afterload through systemic vasodilation. Vasodilators decrease LV preload and/or afterload by reducing systemic vascular resistance, poten-

tially increasing cardiac output (Sinha *et al.*, 2025) .

Mechanical circulatory support devices

Among the main devices used in cardiogenic shock, the intra-aortic balloon pump consists of a counterpulsation mechanism with diastolic inflation and rapid systolic deflation. It has a uniform construction and varies in length (22.0-27.5 cm), in inflated diameter (15-18 mm), and in balloon volume (30-50 mL). The balloon is inflated and deflated rapidly by injecting and removing a gas, typically helium or carbon dioxide, with helium being the preferred agent. Therefore, the ideal balloon size requires that no more than 90% of the aortic diameter be obstructed when the balloon is inflated (Gillespie *et al.*, 2024) .

The intra-aortic balloon decreases afterload, increases diastolic pressure at the aortic root, increases coronary blood flow, decreasing SBP by 10%, end-diastolic aortic pressure by up to 30%, and increases LV ejection fraction (LVEF) and CD by 0.5 and 1 L/min or 30% (Elias *et al.*, 2025) .

Another device mentioned above is Impella, which is an axial flow pump in a *pigtail* catheter that is positioned through the aortic valve to relieve LV overload by providing non-pulsatile blood flow to the ascending aorta. It is used in high-risk percutaneous coronary interventions and in cardiogenic shock surgeries (Chera *et al.*, 2018).

The *IMPRESS in Severe Shock* study (Impella *Versus IABP Reduces Mortality in STEMI Patients Treated With Primary PCI in Severe Cardiogenic Shock*) was the first randomized pilot study to evaluate and compare the efficacy and safety of PCI and intra-aortic balloon in patients with AMI. However, this study did not demonstrate a survival benefit in the 48 patients included. According to a 2019 AHA study by Schrage *et al.*,(2019) routine treatment with Impella was not associated with lower 30-day all-cause mortality compared to a matched cohort from the IABP-SHOCK II study.

In addition, Impella is a device with variations of percutaneous heart pumps that can be implanted in the LV or RV and can provide up to 5.0 L of cardiac output. Recently, the I5.5 was introduced to further increase CO. Therefore, the two most relevant clinical scenarios in which Impella is used are high-risk percutaneous coronary intervention (PCI) and cardiogenic shock. Impella is a temporary ventricular support device intended for short-term use and indicated for the treatment of ongoing cardiogenic shock occurring immediately (<48 h) after AMI or open heart surgery, thus increasing CO and decreasing the end-diastolic pressure of the affected ventricle. The goal of Impella Cardiac Support Therapy is to increase cardiac output and reduce the end-diastolic pressure of the affected ventricle *et* al., 2022) .

The PTD/CM is a catheter with an extracorporeal pump, and its use consists of the implantation of a LVAD (left ventricular assist device) and cardiogenic shock due to severe pulmonary hypertension, usually associated with the CentriMag ventricular assist system. This provides temporary support for the RV, LV, or biventricular, providing temporary support with low rates of device-related complications (both cardiac and systemic. Interrupting the vicious cycle and restoring the hemodynamic stability is a fundamental treatment of CS. Acute coronary syndrome (ACS .

CONCLUSION

Early application of mechanical circulatory support in stages A-B reduces mortality but requires accurate identification of the target population. Only a multidisciplinary approach incorporating dynamic risk stratification and precision methods can minimize the inappropriate use of invasive therapies.

It is concluded that current guidelines and expert statements recommend the use of vasoactive agents to normalize systemic hemodynamics, but evidence on how quickly these drugs should be administered or what titration target they should achieve is scarce. The AHA/ACC guideline still emphasizes the use of norepinephrine as first-line therapy in cardiogenic shock, but treatment remains costly, and studies in patients with different etiologies are still scarce. There is still a need for new, less expensive devices and drug therapy that is less costly for the public sector. In the current context, these patients are still treated late, and mortality rates remain high.

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