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Chapter 7

# Cardiogenic Shock

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Additional information is available at the end of the chapter

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**Abstract**

Cardiogenic shock is the second most common cause of circulatory shock, occurs secondary to myocardial infarction, which accounts for 80% of the cases, and remains one of the leading causes of death in patients with acute myocardial infarction. Cardiogenic shock carries a high morbidity and mortality despite recent advances in medical and mechanical therapies. Cardiogenic shock also occurs in non-acute coronary syndrome conditions, such as Takotsubo cardiomyopathy, fulminant myocarditis, end stage heart failure, and others. In this chapter, we provide a brief review on the pathophysiology, diagnosis, and acute management of cardiogenic shock patients. We will focus more on the management of acute coronary syndrome related cardiogenic shock, given that it is the most common etiology.

**Keywords:** cardiogenic shock, acute coronary syndrome, hemodynamic support, mechanical circulatory support devices, vasopressors, inotropes

## 1. Definition

Circulatory shock is defined as the failure to meet the body's cellular oxygen demands. It typically occurs when the systolic blood pressure falls below 90 mmHg or the mean arterial blood pressure falls below 65 mmHg for 30 min. In circulatory shock there are signs of tissue hypoperfusion such as altered mental status, decreased urine output (<0.5 ml/kg/h), cold and clammy skin, and elevated serum lactic acid level (>1.5 mmol/l) [1].

Cardiogenic shock (CS) is the shock that results from cardiac causes and can be defined as a circulatory failure in addition to severely reduced cardiac index (<1.8 L/min/$m^2$ without support or <2.0–2.2 L/min/m with support) in the presence of adequate filling pressures (left ventricular end diastolic pressure (LVEDP) > 18 mmHg or right ventricular end diastolic pressure >10–15 mmHg) [2].

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To differentiate CS from other types of shock, the following general hemodynamic measures can be used with the help of echocardiography or pulmonary artery catheterization (**Table 1**).

| | Cardiogenic | Distributive (e.g. septic shock) | Hypovolemic | Obstructive | |
|---|---|---|---|---|---|
| | | | | PE | Tamponade |
| PCWP/LVEDP | ↑ | Unchanged or ↓ | ↓ | Usually unchanged | ↑ |
| SVR | ↑ or unchanged | ↓ | ↑ | ↑ | ↑ |
| CI/CO | ↓ | ↑ But might be ↓ | ↓ | ↓ | ↓ |

PCWP, pulmonary capillary wedge pressure, LVEDP, left ventricular end diastolic pressure; SVR, systematic vascular resistance; CI, cardiac index; CO, cardiac output.

**Table 1.** General hemodynamic measures to differentiate between cardiogenic shock and other types of shock.

## 2. Epidemiology

CS is the second most common type of circulatory shock representing 16% of patients presenting with shock [3]. CS complicates up to 8.6% of patients with ST segment elevation myocardial infarction (STEMI) and about 2.5% of patients with non-ST segment elevation myocardial infarction (NSTEMI), and remains one of the leading causes of death in patients presenting with acute myocardial infarction (AMI) [4]. Despite the advancement in the medical and technological management, CS carries a poor prognosis with high morbidity and mortality (40–60% of patients with CS will die within 6 months) [5–7].

AMI is the most common cause of CS, and patients with AMI older than 75 years tend to present more frequently with CS than patients younger than 75 [2–4, 8].

## 3. Etiology and pathophysiology

Acute coronary syndrome (ACS) leading to ischemia and left ventricular (or right ventricular) failure is the leading cause of CS and represents around 80% of CS cases (8% of those are caused by mechanical complications of AMI such as ventricular septal rupture, free wall rupture, papillary muscle rupture and acute mitral regurgitations) [7].

The pathophysiology of ischemia leading to CS is illustrated as a vicious cycle in **Figure 1**. AMI may lead to severe left ventricular (LV) dysfunction and pump failure. The hypotension that accompanies CS leads to the release of inflammatory cytokines and catecholamines leading to increased contractility, which in turn leads to increased myocardial oxygen demand that

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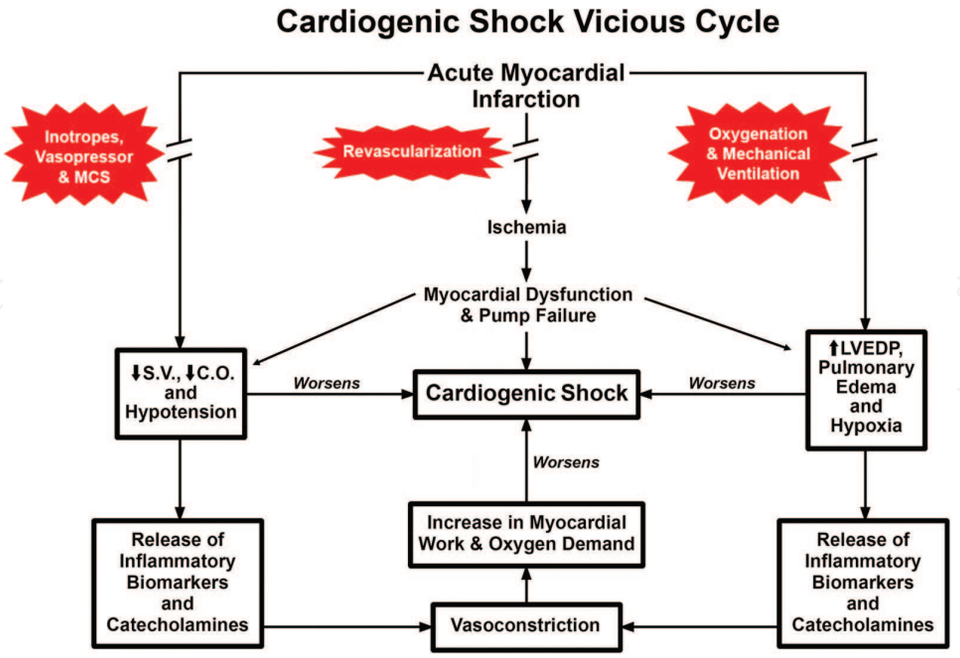



**Figure 1.** The vicious cycle of cardiogenic shock. SV, stroke volume; CO, cardiac output; LVEDP, left ventricular end diastolic pressure; MCS, mechanical circulatory support.

causes worsening of the ischemia and shock state. The increase in catecholamines also causes peripheral vasoconstriction that in turn leads to an increase in the afterload, worsening the ischemia and the shock state [2].

CS also occurs in the absence of coronary artery disease; those etiologies represent around 20% of CS cases. The non-ACS-related CS patients tend to do slightly better than those with ACS [7]. Those conditions may include hypertrophic cardiomyopathy, end stage heart failure, acute fulminant myocarditis, severe valvular stenosis, and acute valvular regurgitation secondary to trauma or infection. CS complicates about 10% of patients presenting with Takotsubo cardiomyopathy and carries a poorer prognosis than the rest of Takotsubo cardiomyopathy population [1, 9, 10].

CS could also occur secondary to right ventricular (RV) dysfunction and failure secondary to RV ischemia, acute pulmonary embolism, pulmonary hypertension (PH) and others [2, 11, 12].

The right ventricle is affected in nearly 50% of inferior STEMI patients, however, RV infarction leading to CS occurs in approximately 5% of CS cases caused by AMI; despite that, it carries high mortality similar to that of LV failure. RV failure leads to decreased transpulmonary delivery of LV preload and intraventricular dependence, which in turn may lead to decreased LV filling. The RV end diastolic pressure in CS secondary to RV failure is usually very high, exceeding 20 mmHg [2, 11–13].

**Figure 2** summarizes the most common causes of CS.

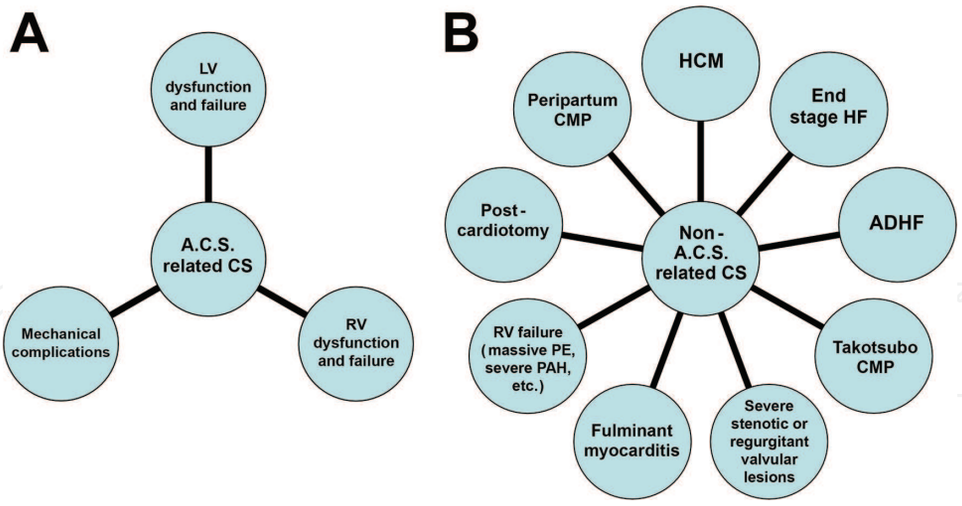



**Figure 2.** The most common causes of cardiogenic shock. (A) ACS represents 80% of CS cases, (B) non-ACS etiologies, which represent 20% of CS causes. ACS, acute coronary syndrome; LV, left ventricle; RV, right ventricle; HCM, hypertrophic cardiomyopathy; ADHF, acute decompensated heart failure; CMP, cardiomyopathy; PE, pulmonary embolism; PAH, pulmonary arterial hypertension.

## 4. Diagnosis and clinical presentation

The diagnosis of CS requires a high index of suspicion due to its high morbidity and mortality. It should be noted that up to 70% of patients with CS will develop shock later during their hospital stay [4].

Most patients with CS are critically ill and might complain of chest pain and/or dyspnea. There are physical exam findings that are more specific to CS than other types of shock, such as elevated jugular venous pressure (JVP), S3 gallop and the presence of pulmonary rales. In fact, the presence of elevated JVP > 8 $cmH_2O$ and rales more than one-third of the lung bases predicted CS with very high sensitivity and specificity [14]. The risk factors that are associated with a higher risk of CS in ACS patients are female gender, diabetes mellitus, anterior wall MI, prior history of MI and older age [14, 15].

Other signs and symptoms of CS are generally those of tissue hypoperfusion, such as the presence of hypotension (SBP < 90 mmHg or MAP < 65 mmHg) in addition to tachycardia, altered mentation, decreased urine output and cold and clammy skin.

Electrocardiogram (ECG) and chest X-ray (CXR) should be obtained in all patients presenting with shock. CXR in CS may show pulmonary edema, pleural effusion, pulmonary vascular congestion or enlarged cardiac silhouette. Cardiac troponin is also mandatory for all patients with suspicion of shock from cardiac causes at the time of presentation and then repeated within 3–6 h [16].

ECG can help diagnose acute STEMI, Q waves or any active cardiac ischemia; although in a routine general practice only about 50% of patients with suspected NSTEMI will have ECG changes that are diagnostic of myocardial infarction at the time of presentation [17].

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The presentation ECG carries prognostic information, as well, and can identify high-risk patients. In an analysis from the SHOCK trial [17], which included CS patients caused by AMI, a higher baseline heart rate was associated with a higher one-year mortality. Also, in CS patients secondary to inferior MI who received medical management, a longer QRS duration and a higher sum of ST segment depression in all leads were associated with a higher one-year mortality [17].

Echocardiography is of utmost importance in the evaluation of shock patients especially when the etiology of shock is not well established. It is noninvasive and readily available at bedside. It helps identify severe valvular regurgitant or stenotic lesions, evaluate for ventricular or septal rupture post-AMI and check for cardiac tamponade.

Two-dimensional echocardiography allows for the identification of LV ejection fraction, assessment of segmental wall motion abnormalities and RV function. Doppler echocardiography allows for the assessment of early mitral filling velocity (E) and the mitral annulus tissue velocity (e') which greatly helps the clinician identifying elevated LV filling pressures with excellent sensitivity and specificity. E:e' > 15 correlates with LVEDP > 14 mmHg and E/e' < 8 correlates with normal LVEDP [18, 19].

Pulmonary artery (PA) catheterization—Swan-Ganz catheter—is an excellent tool for confirming the diagnosis and guiding the medical and mechanical management. In CS, there is an increase in the right atrial (RA) pressure, RV systolic and diastolic pressures and pulmonary capillary wedge pressure (PCWP), and a decrease in the cardiac output and index (**Figure 3**). SVR can also be calculated using the PA catheter and is frequently elevated in CS patients. Currently, the main indication for PA catheter use is to establish the diagnosis of CS when the

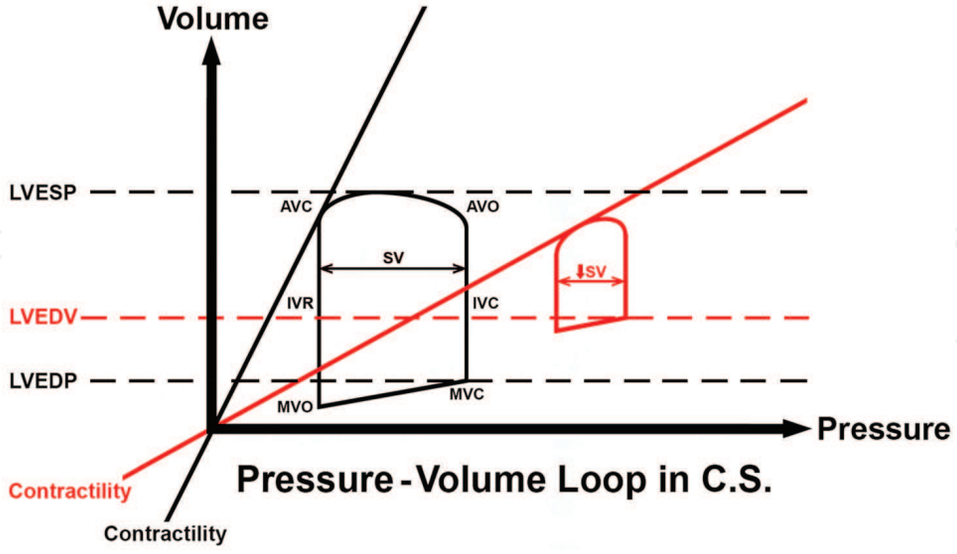



**Figure 3.** The pressure volume loop in cardiogenic shock. The left loop is that of a normal individual while the right one is the CS loop. In CS, there is an increase in LVEDP and LVDEV; there is a decrease in contractility and SV. LVEDP, left ventricular end diastolic pressure; LVESP, left ventricular end systolic pressure; LVEDV, left ventricular end diastolic volume; SV, stroke volume; AVO, aortic valve opens; AVC, aortic valve closes; MVO, mitral valve opens; MVC, mitral valve closes; IVC, isovolumetric contraction; IVR, isovolumetric relaxation.

clinical picture is not clear, or when hemodynamic stabilization is not achieved despite escalating doses of vasopressors and inotropes. PA catheter is also recommended when mechanical circulatory support devices are considered. It should be noted that the routine use of PA catheter is discouraged in patients with a confirmed diagnosis and those who stabilize rather quickly [20].

## 5. Treatment

Since the most common etiology behind CS is ACS, the mainstay of therapy is coronary revascularization to relieve the vicious cycle of ischemia-shock state. Treatment also involves general supportive measures, pharmacotherapy, vasopressors, inotropes and mechanical circulatory support (MCS) in the setting of refractory shock (**Figure 4**).

### 5.1. General measures and pharmacotherapy used in acute coronary syndrome

All patients with suspected AMI—STEMI or NSTEMI—should receive a loading dose of aspirin (162–325 mg) as a chew non-enteric coated capsule and a maintenance dose of aspirin should be continued indefinitely after that. A high dose statin (atorvastatin 80 mg) is also indicated in all patients presenting with AMI without contraindications and should be continued indefinitely. Treatment with high dose statins for ACS patients reduced the risk of death, recurrent myocardial infarction, stroke and the need for coronary revascularization. Oxygen therapy is indicated for all patients with hypoxemia ($O_2$ saturation < 90%) [16, 21].

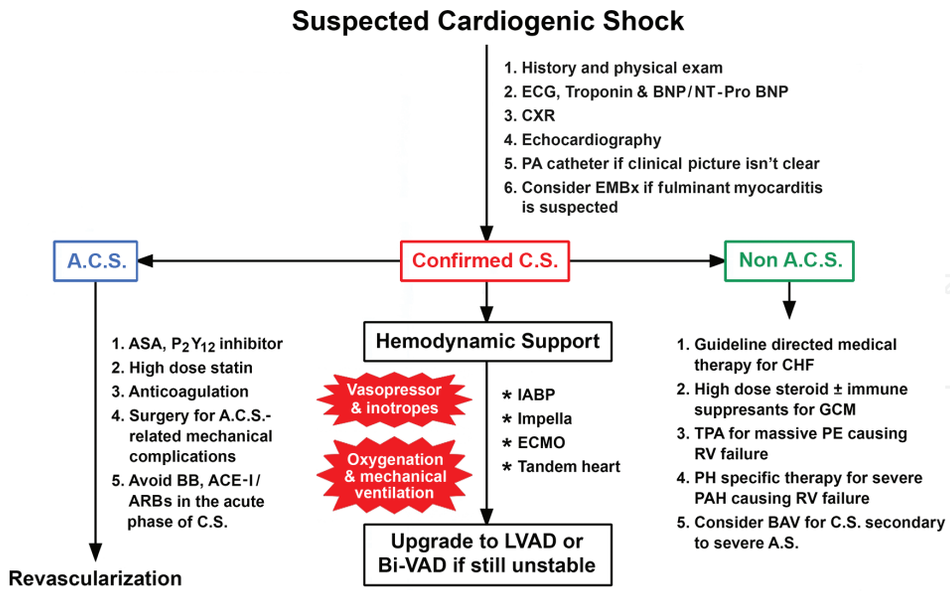



**Figure 4.** Cardiogenic shock treatment flow chart. CXR , chest X-ray; PA, pulmonary artery; EMBx, endomyocardial biopsy; A.C.S, acute coronary syndrome; BB, beta blockers; ACE-I, angiotensin converting enzyme inhibitors; ARB, angiotensin receptor blockers; LVAD, left ventricular assist device; Bi-VAD, biventricular assist device; CHF, congestive heart failure; GCM, giant cell myocarditis; TPA , tissue plasminogen activator; PE, pulmonary embolism; PH, pulmonary hypertension; PAH, pulmonary artery hypertension; BAV, balloon aortic valvuloplasty.

Beta blockers (BB), angiotensin converting enzyme inhibitors (ACE-I) or angiotensin receptor blockers (ARB) should be avoided in patients at risk for CS [16, 21].

In patients with STEMI, a loading dose of a P2Y12 inhibitor should be administered as early as possible or at the time of primary coronary intervention (PCI) (clopidogrel 600 mg, ticagrelor 180 mg or prasugrel 60 mg). Patients with NSTEMI who are undergoing early revascularization should also receive a loading dose of a P2Y12 inhibitor as soon as possible. It should be noted that prasugrel is contraindicated in patients with prior history of stroke or transient ischemic attack (TIA) [16, 21–23].

All patients with STEMI undergoing PCI should receive anticoagulation unless they have contra-indications. Unfractionated heparin (UFH) can be used with or without glycoprotein (GP) IIb/IIIA inhibitors. The recommended dose of UFH is 50–70 units/kg as IV bolus if used with GP IIb/IIIa inhibitors to achieve a therapeutic activated clotting time (ACT) of 200–250 s, or 70–100 u/kg as a bolus if used without GP IIb/IIIa inhibitors to achieve therapeutic ACT of (250–300 s). Bivalirudin can be used in STEMI patients as well, and is preferred as a monotherapy over the combination of UFH-GP IIb/IIIa inhibitor in patients at high risk for bleeding [21].

In patients with NSTEMI the anticoagulation regimen differs slightly from patients with STEMI, UFH can be used with a loading dose of 60 u/kg (maximum dose of 4000 units) followed by infusion of 12 u/kg/h with (maximum dose of 1000 u/h) adjusted to keep therapeutic activated partial thromboplastin time (PTT) during the period of treatment. Enoxaparin is another option for anticoagulation at a dose of 1 mg/kg every 12 h. Most NSTEMI patients presenting with CS will undergo early revascularization, which makes bivalirudin another good option for anticoagulation as bivalirudin is only indicated in NSTEMI patients who undergo early invasive strategy [16].

Most clinicians prefer to use UFH in the setting of CS complicating an NSTEMI given that most of these patients will undergo early invasive strategy, and UFH has the advantage to turn on and off, or even reverse rather easily.

GP IIb/IIIa inhibitors might be considered for NSTEMI patients undergoing early invasive strategy and are treated with dual antiplatelet therapy (DAPT) [16].

### 5.2. Revascularization

Early revascularization is the cornerstone of treatment in AMI patients presenting with CS. The randomized SHOCK trial proved a statistically significant mortality benefit at 6 months in AMI patients complicated by CS treated with emergency revascularization as opposed to medical stabilization [5]. The non-randomized SHOCK registry also showed the same mortality benefit of early revascularization in patients older than 75 [24].

The goal in STEMI patients is first medical contact (FMC) to device time of less than 90 min, and revascularization can still be done even up to 12 h after ischemic symptoms onset. But, in patients with CS complicating a STEMI, revascularization should be performed regardless of the time of symptoms onset. It is also reasonable to intervene on non-infarct arteries in STEMI patients complicated by CS at the time of PCI [21].

Early revascularization within 2 h of presentation should be done in all NSTEMI patients with CS, as well as those with high-risk features (such as refractory angina, electrical instability, signs of heart failure or worsening mitral regurgitation, as well as sustained ventricular tachycardia or fibrillation) [16].

PCI is not the only option for revascularization; coronary artery bypass grafting (CABG) should be considered especially if successful PCI is not feasible, there are mechanical complications such as ventricular septal or papillary muscle rupture, and in those with left main disease or three vessels, CAD. Emergent CABG can be done within 2–4 h in capable facilities [16, 25].

Thirty-six percent of patients undergoing revascularization in the SHOCK trial underwent CABG; those patients were more likely to be diabetic and have left main or three vessels CAD. The survival rate at 30 days and at 1 year was similar between those who underwent PCI or CABG in the SHOCK trial [26].

Compared to patients without CS undergoing CABG, those with CS were more likely to have had suffered AMI within 24 h prior to CABG, were more likely to have left main disease, have lower ejection fraction and were more likely to have intra-aortic balloon pump (IABP) used preoperatively [25].

It should be noted that patients with CS undergoing CABG have worse morbidity and mortality and longer intensive care unit (ICU) stay than those without CS. And even though older age was associated with higher morbidity and mortality, around 70% of patients with CS above the age of 75 survived this major surgery making CABG suitable for carefully selected elderly CS patients [25].

### 5.3. Fibrinolysis

If PCI cannot be performed within 120 min of FMC in STEMI patients, fibrinolytics can be used in those without contraindications and even up to 12 h after symptoms onset, and up to 24 h in those with large areas of ischemia, hemodynamic instability, or have clinical or ECG signs of continuous ischemia. **Table 2** summarizes the absolute contraindications to fibrinolysis [21].

| | |
|---|---|
| Any prior intracranial hemorrhage | Any active bleeding or bleeding diathesis (not including menses) |
| Known malignant intracranial neoplasm | Suspected aortic dissection |
| Known cerebral structural vascular lesion | Ischemic stroke within the past 3 months (except for those with ischemic stroke in the past 4.5 h) |
| Severe uncontrolled refractory hypertension | Any significant closed head or facial trauma in the past 3 months |
| Intracranial or intraspinal surgery in the past 2 months | If streptokinase is used, prior treatment within the previous 6 months (streptokinase is antigenic) |

**Table 2.** Absolute contraindications to fibrinolytics [21].

Patients with RV infarction secondary to proximal right coronary artery (RCA) occlusion with extensive clot burden might be resistant to fibrinolytic therapy; there is also a higher rate of re-occlusion after thrombolysis of the RCA [13, 27, 28].

Patients with CS secondary to STEMI who are treated with fibrinolytics should be transferred immediately to a PCI-capable facility after receiving fibrinolysis.

In patients with NSTEMI, fibrinolytics are contraindicated; those patients should be stabilized and transferred immediately to a PCI-capable facility for coronary angiography and revascularization [16].

### 5.4. Vasopressors and inotropes

There is no optimal vasopressor or inotrope in the setting of CS, but catecholamines are the most frequently used vasopressors, with norepinephrine and dopamine being the most widely used. Catecholamines exhibit their effects through the stimulation of A1, B1, B2, and dopaminergic receptors (D1 and D2) [9, 29].

Norepinephrine is a potent A1 agonist; it induces an increase in systolic blood pressure (SBP), diastolic blood pressure (DBP), and the pulse pressure. Norepinephrine has minimal effect on myocardial contractility and HR [29].

Dopamine produces a multitude of effects at different doses: at lower doses (<3 ug/kg/min), it works primarily on the D1 receptors and causes coronary and renal vasodilatation; at intermediate doses (3–10 ug/kg/min), dopamine stimulates the B receptors and causes an increase in inotropy and HR; and at higher doses (10–20 ug/kg/min), dopamine works primarily on A1 receptors and causes vasoconstriction. The renal vasodilatory effect—so-called renal dose—of low dose dopamine remains controversial, and glomerular filtration rate (GFR) does not change with use of those renal doses of dopamine [30, 31].

Epinephrine has high affinity towards A1, B1 and B2 receptors, with B effects more pronounced at lower doses and Alpha effects at higher doses. Prolonged use of epinephrine is associated with direct cardiac toxicity through damage to the arterial wall that results in myocardial necrosis and stimulation of myocyte apoptosis [29, 32].

Vasopressin or "antidiuretic hormone" is a non-adrenergic vasopressor; it stimulates the V1 and V2 receptors. The stimulation of the V1 receptors causes vasoconstriction while the stimulation of the V2 receptors enhances water reabsorption in the renal collecting ducts. It augments the pressor effect of norepinephrine and has no effect on cardiac output (CO). Vasopressin's pressor effect is relatively preserved during the acidotic state that develops in most shock patients [29, 33].

Dobutamine is a B1 and B2 agonist; it primarily induces an inotropic effect, exhibits a modest increase in HR and causes peripheral vasodilatation through the stimulation of B2 receptors. Dobutamine induces an increase in the cardiac output and a reduction in the LVEDP. Pharmacologic tolerance to dobutamine usually develops after 72 h of use. Dobutamine could induce arrhythmias, myocardial ischemia and tachycardia, especially at higher doses (>15 ug/kg/min), but these effects are reversed rather rapidly due to the short half-life of the drug (2.3 min).

The prolonged use of dobutamine (7–52 days) is associated with much higher 6-month mortality [29, 30, 34–36].

Milrinone is a noncatecholamine inotrope and peripheral vasodilator, has lusitropic effect and has less effect on HR than dobutamine. Milrinone works through the inhibition of phosphodiesterase enzymes (PDE), which in turn, leads to an increase in intracellular cyclic adenosine monophosphate (cAMP), which leads to an increase in the rate of entry and removal of calcium from the cardiac myocytes thus increasing myocardial contractility. Milrinone has been mainly used in the treatment of advanced severe heart failure patients, and—to date—there have been head-to-head trials comparing dobutamine to milrinone. Milrinone should be avoided in advanced kidney disease patients as it is cleared renally [30, 37, 38].

Levosimendan is a calcium-sensitizing agent that enhances myocardial inotropy and lusitropy and causes peripheral vasodilation, and it is not yet approved for use in the USA. Levosimendan is associated with similar mortality rates as compared to dobutamine but it tends to cause more peripheral vasodilation and hypotension than dobutamine [30, 39, 40].

Norepinephrine is preferred over dopamine as dopamine has been associated with a higher incidence of arrhythmias and a higher rate of death at 28 days in the CS patient subgroup [3].

In CS secondary to RV infarction, IV fluids are always the first line, but the excessive administration of IV fluids beyond an RA pressure of 15 mmHg could result in the deterioration of LV performance, and the use of dobutamine in this scenario can be particularly helpful in improving myocardial performance. Despite the severe hemodynamic compromise, arrhythmias, and increased in-hospital mortality, many patients with severe RV infarction recover within 3–10 days and typically, global RV function recovers within 3–12 months [13, 29, 41].

Vasopressors and inotropes are essential in stabilizing CS patients but caution should always be taken with their use. The use of these agents causes an increase in the myocardial oxygen demand and can induce arrhythmias, and thus their use should always be individualized and guided by hemodynamic monitoring. The long-term use of inotropes is strongly discouraged, and should only be considered as a bridge to heart transplantation or ventricular assist devices (VAD) or as a palliative therapy in advanced heart failure patients [20, 29].

It is recommended to combine two small doses of vasopressors and inotropes than the use of a maximal dose of a single agent to avoid dose-related adverse events, also, the addition of vasopressin can help with "catecholamine sparing" [29]. The use of epinephrine in CS patients is associated with higher 90-day mortality independent of a prior cardiac arrest, and, thus, its use is discouraged unless it is a last resort medication [42].

Our experience with these vasoactive agents in CS has been to initiate norepinephrine followed by an inotrope and then a stepwise approach in the addition of further vasopressors and/or inotropes in the setting of refractory shock. A concomitant shock etiology, such as septic shock, should always be investigated as the choice of these agents might differ.

References [20, 29] provide further information about inotropes and their mechanism of action.

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## 6. Mechanical circulatory support devices

In certain patients with CS, hemodynamic stabilization might not be achieved despite aggressive pharmacotherapy and revascularization, as a result, percutaneous mechanical circulatory support (MCS) devices might be considered for temporary stabilization [43]. The optimal MCS device offers rapid hemodynamic stabilization along with a low complication rate. To date, no trial has shown mortality benefit with the use of these devices in CS patients.

### 6.1. Intra-aortic balloon pump counterpulsation

IABP counterpulsation is the most common form of percutaneous LV support. The original idea of counterpulsation started in the 1960s as an external counterpulsation device stimulating the hemidiaphragm around the distal thoracic aorta with each diastole. IABP is implanted percutaneously through either of the femoral arteries using a double lumen catheter that is 7.5–8 Fr and is placed in the thoracic aorta with its tip distal to the left subclavian artery take off, and its proximal portion above the renal vessels (**Figure 5**) [43, 44].

IABP is a form of internal counterpulsation and acts as an assisting circulatory support device that inflates during diastole and deflates during systole. Its main mechanism is by diastolic augmentation during inflation that contributes to the coronary, cerebral, and systemic circulation. The presystolic deflation lowers the impedance to systolic ejection and subsequently lowers the myocardial work and oxygen demand. IABP usually causes between 0.5 and 1.0

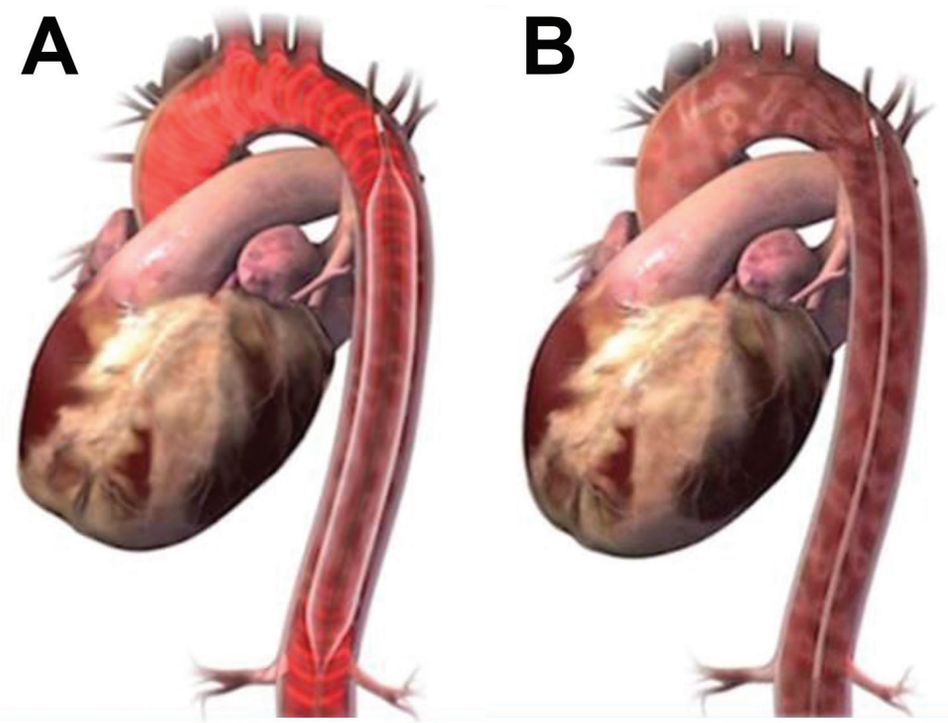



**Figure 5.** Intra-aortic balloon pump. The left panel shows the balloon inflation during diastole and the right panel shows the balloon deflation during systole. Reproduced with permission from Getinge.

L/min increase in the CO. IABP induces around 10% drop in SBP indicating proper systolic unloading, causes an increase in DBP which in turn improves the coronary perfusion and leads to a net increase in the mean arterial pressure (MAP). There is also an increase in the LV ejection fraction with IABP and a decrease in the LV end diastolic volume and pressure [44–47].

Despite all the hemodynamic advantages with IABP, studies have failed to show any mortality benefit with its use. The SHOCK II trial, which compared IABP vs. medical stabilization, showed no difference in mortality along with other variables such as time to hemodynamic stabilization, length of ICU stay, the dose and duration of catecholamines, and changes in renal function [6, 48].

Currently the main indication for IABP counterpulsation is CS refractory to pharmacotherapy; IABP is currently a class IIa indication for the treatment of CS complicating a STEMI in the American Heart Association/American College of Cardiology guidelines (AHA/ACC), while its routine use in CS is discouraged by the European Society of Cardiology [21, 49].

Other indications where IABP can help stabilize the patient include refractory heart failure, papillary muscle rupture or acute mitral regurgitation, ventricular septal rupture, refractory unstable angina, high-risk PCI or the inability to wean from cardiopulmonary bypass [44, 49, 50].

The absolute contraindications to IABP are significant aortic regurgitation and aortic dissection. Other relative exclusion criteria include: significant peripheral arterial disease (PAD) that precludes placement, severe coagulopathy, active infection, and cancer with metastasis [44].

The complication rate with IABP is rather rare with thrombocytopenia and fever being the most common (about 50% and 40% of patients, respectively). Other major complications include: major limb ischemia (0.9% of patients); severe access site bleeding (0.8%); amputation (0.1%); balloon leak (1%); and IABP-related mortality (0.05%). The main risk factors associated with IABP complications are female gender, PAD, small body surface area (BSA) (BSA < 1.65 $m^2$), and advanced age (>75 years) [51, 52].

Due to the lack of data, the use of anticoagulation with IABP is variable among different centers. Most centers, like ours, use anticoagulation, but some will not, especially with 1:1 pumping [43].

### 6.2. Impella devices

The Impella device is a nonpulsatile, axial flow device that is implanted inside the LV percutaneously, commonly through the femoral artery for the 2.5 Impella or with surgical cutdown, commonly through the axillary artery for the 5.0 Impella. The Impella acts as a pump that propels blood from the LV into the ascending aorta (**Figure 6**) [43].

The Impella device has three versions; 2.5 Impella, which is a 12 Fr system that provides a maximal flow of 2.5 L/min, the 5.0 Impella which is a 21 Fr system and provides a maximal flow of 5 L/min, and the CP Impella, which is a 14 Fr system that provides between 3 and 4 L/min of flow [43, 53].

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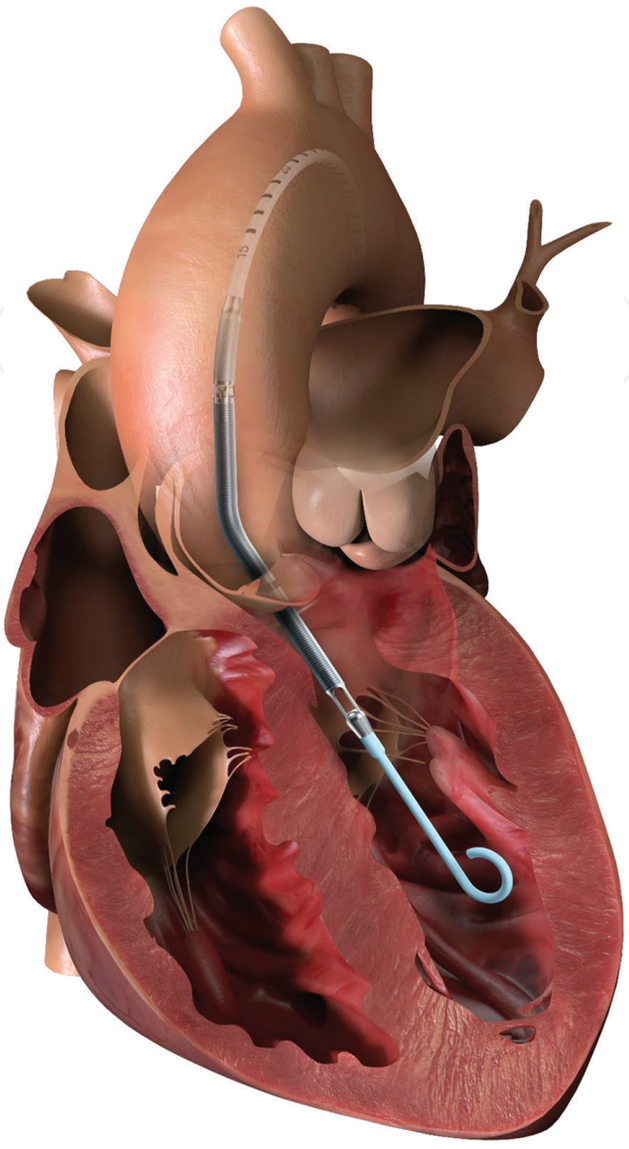


**Figure 6.** The Impella device with the pump inside the left ventricle and the outer catheter inside the aorta. Reproduced with permission from Abiomed.

The Impella unloads the LV, reduces the left ventricular end diastolic volume (LVEDV) and the LV wall tension and improves the systemic and coronary perfusion through an increase in the mean arterial pressure. The Impella device requires an adequate RV function (or an RV assist device) to maintain adequate LV preload, and unlike the IABP, the Impella devices can work properly through transient arrhythmias.

The main indications of the Impella devices are similar to those of the IABP counterpulsation with slight differences, for example, the Impella may worsen right-left shunting in patients with ventricular septal defect (VSD).

The main contraindications to Impella are mechanical aortic valve and LV thrombus. Other relative exclusion criteria are severe aortic regurgitation and severe PAD. The most common complications are those of vascular nature such as access site bleeding, retroperitoneal

hematoma, limb ischemia and vascular injury. Hemolysis is also common with the Impella device due to the mechanical shear stress of the device on the red blood cells. In addition, anticoagulation is generally required during treatment with Impella [43, 53].

Compared to the IABP, Impella does provide greater hemodynamic support but it has not been shown to change the mortality [54]. In the largest most recent randomized controlled trial (the IMPRESS trial) comparing Impella to IABP in CS complicating AMI; 48 patients with severe CS complicating STEMI were randomized to the Impella device (24 patients) and to IABP (24 patients), the mortality at 30 days and at 6 months was similar between the two groups (50% in both groups at 6 months). Of note: those were extremely ill patients with 92% of the entire group having cardiac arrest prior to randomization, and half the mortality at 6 months was attributed to brain damage in both groups [55].

And although not commonly done, the successful use of Impella in combination with IABP has been reported [56].

A brief comparison between the Impella and the IABP is summarized in **Table 3**.

### 6.3. Other percutaneous mechanical circulatory support devices

The IABP and the Impella are not the only circulatory support devices used in CS, there are other—less commonly used—devices such as the Tandemheart, the extracorporeal membrane oxygenation (ECMO) and others.

The TandemHeart is left atrial to aorta support device that is inserted percutaneously and requires a transseptal puncture to access the left atrium. It bypasses the LV and pumps blood—extracorporeally—from the left atrium into the iliofemoral arterial system (**Figure 7**) [43, 57].

| | **Impella** | **IABP** |
|---|---|---|
| ECG | Unrelated to systole or diastole | Inflates with diastole and deflates with systole |
| CO | Up to 5 L/min of CO | Modest increase in CO (0.5–1 L increase CO) |
| LVEDV | Reduces LVEDV and LVEDP | Reduces LVEDV and LVEDP |
| Catheter size | Between 12 and 21 Fr | 7.5–8 Fr |
| Rhythm | Does not require a stable rhythm (although asystole and VF are poorly tolerated) | Requires a stable rhythm |
| Absolute contraindications | Mechanical AV, LV thrombus | Severe AR, aortic dissection |
| Complications | Similar complication profile of vascular injury and access site bleeding, with these complications being slightly higher with the Impella | |
| Mortality | No difference in mortality between both devices in CS patients complicating AMI | |

CO, cardiac output; LVEDV, left ventricular end diastolic volume; LV, left ventricle; AV, aortic valve; AR, aortic regurgitation.

**Table 3.** A brief comparison between intra-aortic balloon pump (IABP) and Impella.

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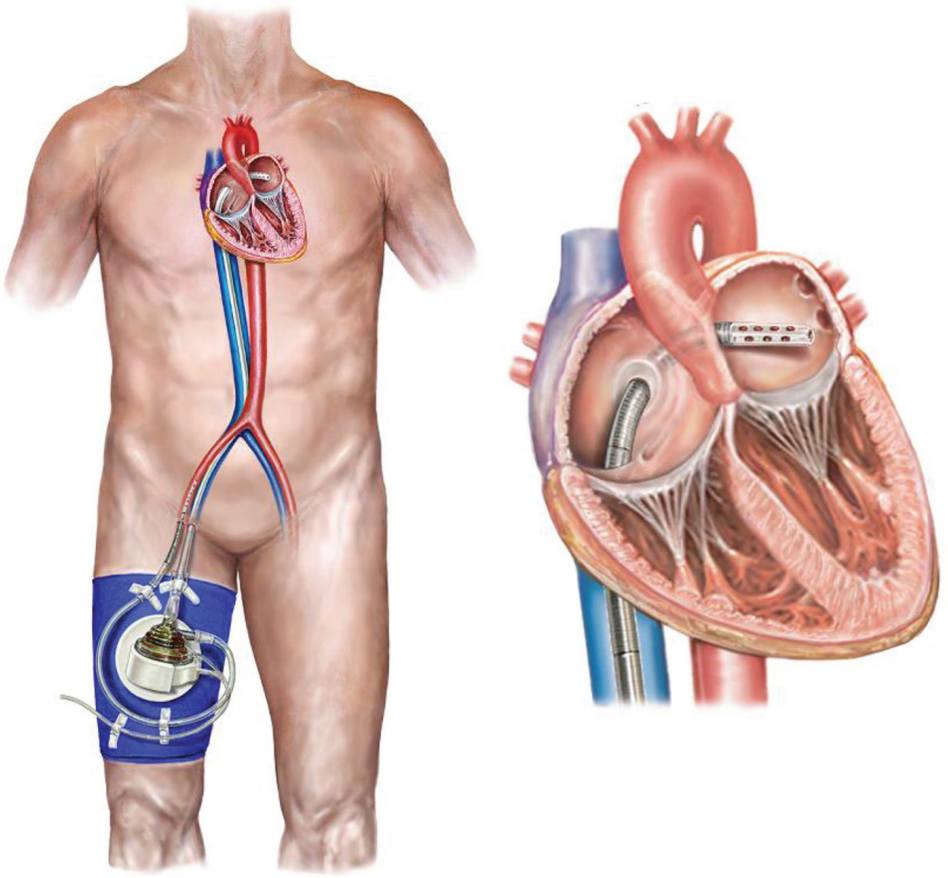


**Figure 7.** The TandemHeart. The left panel shows the entire system: there is a venous catheter and an arterial catheter, and the pump is situated extracorporeally. The right panel shows the transseptal puncture and how the venous catheter bypasses the left ventricle. Reproduced with permission from Tandemlife.

The TandemHeart device has two separate catheters, a 21 Fr venous catheter that goes transseptally and aspirates the LA blood and an arterial perfusion outflow cannula between 15 and 19 Fr. The TandemHeart pump can provide flow rates up to 4.5 L/min of assisted cardiac output [8, 43].

The TandemHeart has been studied in severe refractory CS patients not responding to vasopressors/inotropes in combination with IABP. The TandemHeart significantly improved the hemodynamics in this extremely ill population, along with PCWP, lactic acid levels and creatinine levels. This device can also be used as a bridge to a more definitive therapy such as left ventricular assist device (LVAD) or heart transplantation [28].

ECMO can provide a full pulmonary and/or cardiac support for those with failing hearts and/or lungs. The ECMO device can be either venoarterial (V-A ECMO) or venovenous (V-V ECMO); the V-A ECMO is ideal for those with CS and poor oxygenation while the V-V ECMO provides oxygenation only when the cardiac hemodynamics are stable. The venous catheter size is usually 20 Fr and the arterial catheter size is 17 Fr. ECMO can provide even more than 6 L/min of CO depending on catheter size and unlike other MCS devices, a trained perfusionist is required to manage the ECMO [43].

IABP, Impella, TandemHeart and ECMO can all be used in the setting of CS with slight differences in indications. They offer hemodynamic support, and it is recommended that one of these devices be inserted rapidly in CS if hemodynamic stability cannot be achieved with fluid resuscitation and/or pharmacotherapy. The experience with these devices in CS patients has been to start with an IABP along with vasopressors/inotropes, and if hemodynamic stability cannot be achieved, one may consider upgrading to one of the more powerful percutaneous MCS devices. Although these devices are FDA approved for the use of up to 6 h, they have been used successfully for days in patients with prolonged shock [43].

Our center's experience is to insert an IABP or an Impella—depending on operator's experience—rapidly in CS patients secondary to AMI prior to attempted revascularization. We recommend—as it is endorsed by the 2015 SCAI/ACC/HFSA/STS consensus document for the use of MCS devices—that one of these devices inserted rapidly if hemodynamic stability cannot be achieved rapidly with pharmacotherapy.

Other devices are being used such as the right ventricular assist devices (RVAD), which is used for the failing RV, and others. For further read on these devices and other MCS devices, refer to the 2015 SCAI/ACC/HFSA/STS expert consensus statement on the use of percutaneous MCS [43].

## 7. Treatment considerations in non-ACS related CS

The mechanical complications of AMI such as acute MR, papillary muscle rupture, ventricular septal rupture and LV free wall rupture are catastrophic, and carry very high mortality and are surgical emergencies. IABP helps stabilize these patients, especially acute MR patients, and the other MCS devices can be used in these situations as well.

RV failure resulting in CS also carries high mortality; ECMO or RVAD might be especially helpful in this situation. In CS secondary to massive pulmonary embolism, fibrinolysis (or mechanical thrombectomy) might be helpful, and in RV failure secondary to severe pulmonary arterial hypertension, the use of pulmonary hypertension (PH) specific therapy might provide improvement in the PA pressures and RV function.

The treatment considerations in acute decompensated heart failure (ADHF) and end stage cardiomyopathy are those of the heart failure guidelines [20], and the above-mentioned MCS devices can be used interchangeably.

In most patients with myocarditis, the course is usually self-limiting and presents with acute heart failure; on the other hand, fulminant myocarditis will present with acute severe heart failure and even CS. Close to 90% of patients with fulminant myocarditis will have full recovery with minimal long-term sequelae if recognized early. The treatment of CS secondary to fulminant myocarditis includes hemodynamic support with pharmacotherapy or MCS devices, along with high dose steroids with or without immunosuppressants if giant cell myocarditis is diagnosed [58].

## 8. Summary and conclusion

Cardiogenic shock still carries high morbidity and mortality and remains the leading cause of death in acute myocardial infarction patients. Early recognition and treatment is the key to improving survival, and early revascularization in CS secondary to myocardial infarction remains the cornerstone of therapy in these patients. The early use of vasopressors/inotropes is recommended in this population, and the early use of the mechanical circulatory support devices is encouraged if hemodynamic stability cannot be achieved rapidly with pharmacotherapy.

One should keep in mind the mechanical complications of myocardial infarction and the grave prognosis if not recognized early.

There is a multitude of etiologies for non-ACS related cardiogenic shock; those should be treated similarly with vasopressors/inotropes, and MCS devices, keeping in mind guidelines directed medical therapy for those with congestive heart failure.

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