Prognosis and treatment of cardiogenic shock complicating acute myocardial infarction

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INTRODUCTION

Cardiogenic shock (CS) is a clinical condition of inadequate tissue (end-organ) perfusion due to cardiac dysfunction. The definition includes the following hemodynamic parameters: persistent hypotension (systolic blood pressure <80 to 90 mmHg or mean arterial pressure 30 mmHg lower than baseline) with severe reduction in the cardiac index (<1.8 L/min per m^2 without support or <2 to 2.2 L/min per m^2 with support) and adequate or elevated filling pressures [1].

The most common etiology of CS is an acute myocardial infarction (MI; usually ST-elevation MI [STEMI]) with left ventricular failure, but it can also be caused by mechanical complications such as acute mitral regurgitation or rupture of either the ventricular septal or free walls. However, any cause of acute severe left or right ventricular dysfunction may lead to CS.

The prognosis and therapy of CS complicating acute MI will be reviewed here. The pathophysiology, clinical manifestations, and diagnosis of this disorder are discussed separately. (See "Clinical manifestations and diagnosis of cardiogenic shock in acute myocardial infarction".)

EPIDEMIOLOGY

The incidence of CS complicating acute MI has been falling since the mid-1970s. In a report from one United States metropolitan area, the incidence of CS was around 7 percent between 1975 and 1990 and has decreased to between 5.5 to 6.0 percent since then [2]. The decrease in the incidence of shock and the associated improvement in overall mortality in part reflect increased use of coronary reperfusion strategies for MI, including primary percutaneous coronary intervention and fibrinolytic therapy, which by restoring patency to the infarct-related artery, can limit infarct size, reduce the risk of shock, and improve survival [3-6].

OUR APPROACH

For patients determined to have cardiogenic shock (see 'Introduction' above), we take the following sequential steps in management:

- Ventilatory support is initiated in patients who meet criteria. (See 'Ventilatory support' below.)
- Hemodynamic support is initiated, usually with inotropic agents or with norepinephrine in patients who have marked hypotension (ie, systolic blood pressure <80 mmHg). We do not routinely place an intraaortic balloon pump. Benefit may exist in patients with mechanical defects and selected other patients who are rapidly deteriorating. (See 'Hemodynamic support' below.)
- We avoid beta blockers. We give aspirin 325 mg but do not give an oral $P2Y_{12}$ receptor blocker until after diagnostic coronary angiography. (See 'Medical therapy' below.)

VENTILATORY SUPPORT

Ventilatory support may be required for several reasons in patients with CS (see "Overview of initiating invasive mechanical ventilation in adults in the intensive care unit"):

- To protect the airway and maintain oxygen supply in patients with a deterioration in consciousness or cardiac arrest.
- To treat acute respiratory failure, most often due to cardiogenic pulmonary edema. (See "Treatment of acute decompensated heart failure: General considerations".)
- To raise the arterial pH in metabolic acidosis. (See "Bicarbonate therapy in lactic acidosis".)

HEMODYNAMIC SUPPORT

Prompt management of hypotension and hypoperfusion is essential. Both pharmacologic and nonpharmacologic methods of circulatory support are rapidly employed to reverse hypotension, maintain vital organ perfusion, and maintain coronary perfusion pressure [7-10].

Hemodynamic monitoring may be useful in patients with refractory shock despite revascularization. However, reperfusion therapy should not be delayed for insertion of a balloon-tipped pulmonary artery catheter. (See 'Role of pulmonary artery catheter' below.)

Volume status — Careful attention should be given to volume status, although it may be difficult to evaluate in CS patients [11]. Hypovolemia may be present, particularly in the setting of diuretic use or vomiting. An empiric intravenous volume challenge of 250 mL of isotonic saline can be given prior to right heart catheterization in patients with suspected CS when there is no evidence of pulmonary congestion on physical examination or chest radiograph and the patient is not in respiratory distress [12]. Overly vigorous fluid challenges in patients with extensive left ventricular infarction, particularly older adults, will result in pulmonary edema and should be avoided. On the other hand, patients with volume overload and cardiogenic pulmonary edema without hypotension may require therapy with diuretics, morphine, supplemental oxygen, and vasodilators. The management of this complication is discussed separately. (See "Treatment of acute decompensated heart failure: General considerations".)

CS may be secondary to right ventricular shock due to a right ventricular MI. Volume repletion has a role in these patients that have low right-sided filling pressures with hypovolemia. However, excess fluid administration results in a shift of the interventricular septum into the left ventricle, with restriction to left ventricular filling in patients with severe right ventricular dysfunction. (See "Right ventricular myocardial infarction", section on 'Optimization of right ventricular preload'.)

Vasopressors and inotropes — Based on the lower rate of arrhythmias and the trend toward lower mortality presented below, our experts suggest starting with norepinephrine. We attempt to minimize the number of agents and their dose.

In some patients, measurement of hemodynamic parameters such as cardiac output and arterial pressure (as well as the calculation of systemic vascular resistance) with an arterial line and a pulmonary artery catheter may guide the choice of vasopressors and inotropes. However, there is no evidence that titrating medications based on hemodynamics improves outcomes. (See "Pulmonary artery catheterization: Indications, contraindications, and complications in

adults", section on 'Severe cardiogenic shock' and "Pulmonary artery catheterization: Interpretation of hemodynamic values and waveforms in adults".)

Sympathomimetic inotropic and vasopressor agents (table 1) are the mainstay of hemodynamic support [12]. However, there is very little evidence to guide the use of one drug over another or a particular combination of drugs:

- Norepinephrine is a potent vasopressor with some positive inotropic properties that may be used for rapid initial circulatory support for CS. The minimum required dose should be used. (See "Use of vasopressors and inotropes", section on 'Norepinephrine'.)

- Dopamine is a vasopressor; its effect varies based upon the dose range administered. At low doses, it has primarily positive inotropic effects but at higher doses it stimulates alpha adrenergic receptors, resulting in vasoconstriction and increased systemic vascular resistance. This may produce an undesirable elevation in pulmonary capillary wedge pressure (PCWP). The minimum required dose should be used. (See "Use of vasopressors and inotropes", section on 'Dopamine'.)

While dopamine has historically been chosen before norepinephrine, some evidence suggests that outcomes may be better with norepinephrine as the initial agent. In a trial of 1679 patients with circulatory shock due to varying etiologies (eg, septic, hypovolemic, and CS) who were randomly assigned to initial therapy with either dopamine or norepinephrine, there was a trend toward a higher rate of death at 28 days with dopamine, and there were significantly more arrhythmias, predominantly atrial fibrillation. There was no difference in the treatment effect based on shock type, including in the subset of 280 patients with CS [13].

We generally begin therapy with a vasopressor (usually norepinephrine) in patients with severe hypotension (ie, systolic blood pressure <80 mmHg). The administration of dobutamine, an inotropic agent (table 1), is limited to less sick patients with a low cardiac index, high PCWP, and borderline low blood pressure but without severe hypotension. Nonhypotensive patients in a low output state with high PCWP can also be treated with dobutamine plus a vasodilator (intravenous nitroglycerin or nitroprusside). This combination will further reduce both afterload and preload. (See "Use of vasopressors and inotropes", section on 'Dobutamine'.)

Intraaortic balloon pump — The available evidence does not support the routine use of an intraaortic balloon pump (IABP) in most patients with acute MI complicated by CS in whom primary percutaneous coronary intervention (PCI) is attempted or performed or in whom fibrinolytic therapy is administered. However, benefit may exist in patients with mechanical defects (such as mitral regurgitation or a ventricular septal defect) and selected other patients who are rapidly deteriorating. (See "Intraaortic balloon pump counterpulsation" and "Acute

myocardial infarction: Mechanical complications", section on 'Rupture of the interventricular septum' and "Acute myocardial infarction: Mechanical complications", section on 'Papillary muscle rupture'.)

The best evidence against the routine use of IABP for patients with MI comes from the IABP-SHOCK II trial in which 600 patients with CS complicating acute MI (ST-elevation and non-ST elevation MI) were randomly assigned to the device or no device [14]. All patients were expected to undergo early revascularization (predominantly with PCI) and to receive best available medical care. At 30 days, there was no difference in the rate of all-cause mortality (39.7 versus 41.3 percent, respectively; relative risk 0.96, 95% CI 0.79-1.17). There were no significant differences in secondary end points such as length of stay in the intensive care unit, renal function, or the rates of major bleeding, peripheral ischemic complications, sepsis, or stroke. There was no difference in mortality at long-term follow-up of 12 months and 6.2 years (52 versus 51 and 66.3 versus 67.0 percent, respectively) [15,16]. A 2015 meta-analysis of seven studies (n = 790), including IABP-SHOCK, came to similar conclusions [17].

One weakness of the study was crossover of patients in the control group to IABP for reasons other than the development of a mechanical complication (26 of 30 insertions were thought to be protocol violations). However, a per-protocol adjusted analysis that excluded patients who crossed over came to the same conclusions. It is possible that rapidly deteriorating patients may not have been enrolled and if enrolled, crossed over; the study cohort may represent those who stabilized on vasopressor/inotropic support. Thus, the trial results may not apply to severe shock with rapid deterioration. Further data and longer follow-up are needed to better understand subsets that may benefit from IABP.

Two earlier observational studies support the findings in IABP-SHOCK II [18-20].

For those patients with CS undergoing PCI in whom an IABP is chosen, the optimal timing of placement is unknown, as the observational evidence is conflicting. In IABP-SHOCK II, there was no difference in outcomes between those who received an IABP before or after PCI. In a study of 48 patients with MI and CS, a significantly lower in-hospital mortality rate at 30 days (19 versus 69 percent) was found in those who received the IABP before as opposed to after PCI [21]. However, an earlier analysis from the SHOCK trial registry suggested that the mortality was similar whether IABP was placed before or after PCI [22].

For patients whose hemodynamic parameters and clinical status are rapidly deteriorating while on vasopressor and inotropic support after revascularization, and who are candidates for bridge to transplantation or durable ventricular assist device, IABP is not as effective for hemodynamic support as other temporary mechanical circulatory support devices.

The role of IABP in MI patients treated with fibrinolytic therapy who will be transferred for possible revascularization is not well established, as there is little evidence that can be used to guide the formation of recommendations. (See "Diagnosis and management of failed fibrinolysis or threatened reocclusion in acute ST-elevation myocardial infarction", section on 'Primary failure'.)

Other mechanical devices — Though outcome evidence is lacking, temporary mechanical circulatory support (MCS) devices are increasingly being used in patients with CS. The selection of devices is usually dependent on center-specific experience and whether the patient requires univentricular or biventricular support. (See "Short-term mechanical circulatory assist devices", section on 'Non-IABP percutaneous circulatory devices'.)

These include:

- Left ventricular and biventricular assist devices. In the setting of CS, these surgically implanted devices are usually placed as a bridge to recovery in patients who had rapid reperfusion but with persistent hypoperfusion, or as a bridge to transplantation or a durable left ventricular assist device (LVAD) in eligible patients in whom ventricular function is not expected to recover.

- Percutaneous left atrial-to-femoral arterial ventricular assist device. This device (TandemHeart) is placed via the femoral vein and across the interatrial septum to provide temporary circulatory support while performing high-risk PCI or awaiting ventricular recovery.

- Percutaneous cardiopulmonary bypass support with use of an extracorporeal membrane oxygenator (ECMO) may be utilized when oxygenation is severely impaired or when rapid/bedside biventricular support is necessary for urgent stabilization [23,24].

  In critically ill patients with multi-organ failure secondary to CS, a strategy of using ECMO to stabilize the patient and allow organ function recovery, followed by a delayed coronary artery bypass graft surgery, could be considered. This strategy might be particularly useful in patients who present late (more than six hours after initiation of symptoms) and in patients with mechanical complications of MI [25]. (See "Extracorporeal life support in adults in the intensive care unit: Overview".)

- Percutaneous transvalvular LVAD. This device (Impella 2.5 or Impella CP) is placed via the femoral artery, retrograde across the aortic valve into the left ventricle. It has a microaxial pump that decompresses the left ventricle and delivers a maximum flow of 2.5 to 4.0 L/min into the ascending aorta. In a multicenter observational registry of 120 patients with

CS after acute MI who received the Impella 2.5 device following initial IABP support, 30-day mortality was 64.2 percent [26]. Without a comparison group, we do not know how this would compare with no device or with other devices.

In a 2019 retrospective analysis, the use of Impella was not associated with lower 30-day mortality compared with matched patients from the IABP-SHOCK trial treated with an IABP or medical therapy [27]. In April 2016, the US Food and Drug Administration approved the Impella device for use in acute MI-related CS based on circulatory support effect, not based on improved clinical outcomes.

- The Impella RP System (Abiomed, United States) is a right ventricular assist device that provides peripherally-placed circulatory support in patients with refractory right ventricular shock. The pump is inserted via the femoral vein, into the right atrium, and through to the pulmonary artery, and can provide flow of up to 5 L/min [28].

- Several small randomized trials have compared percutaneous temporary LVAD support with IABP support in patients with CS after an acute MI [29-31]. Two meta-analyses have evaluated mortality using data from these small trials:

  - In a 2017 meta-analysis of 148 patients randomly assigned to either TandemHeart or Impella VAD or an IABP, there was no difference in 30-day mortality (risk ratio [RR] 1.01, 95% CI 0.70-1.44, p = 0.98) for percutaneous LVAD support compared with IABP control [32].

  - In a second 2017 meta-analysis that evaluated 95 patients randomly assigned to Impella or IABP, there was no difference in mortality at either 30 days (RR 0.99, 95% CI 0.62-1.58) or at six months (RR 1.15, 95% CI 0.74-1.48) between the Impella and the IABP cohort [33].

MCS devices were used in 3.1 percent of the cases reported in the National Cardiovascular Data Registry. In these cases, MCS was initiated before revascularization 27.7 percent of the time, during the revascularization procedure 49.9 percent, and 22.4 percent post-procedure [34]. Appropriately powered randomized trials are needed to assess whether use of MCS improves clinical outcomes, and to define the optimal strategy for MCS use in CS. The utilization of these devices is discussed in detail elsewhere. (See "Short-term mechanical circulatory assist devices".)

Role of pulmonary artery catheter — Pulmonary artery catheters may be used to guide management of patients with shock and support treatment decisions [35]. However, their use in CS patients, with or without mechanical circulatory support, has not been proven to save lives. The use of pulmonary artery catheters has not been systematically evaluated in randomized

clinical trials. Experts have widely differing views on their utility. Pulmonary artery catheter use may assist in device selection and weaning of patients in CS managed with mechanical circulatory support devices [36].

MEDICAL THERAPY

For patients with CS associated with acute myocardial infarction (MI), we use the following approach to drug treatment:

- Beta blockers should be avoided despite the fact that in the broad population of patients with acute MI, beta blockers are given early. In patients with CS or "pre-shock," in which the cardiac output is diminished but hypotension has not yet developed, drugs that have negative inotropic activity will likely worsen the clinical situation. Often, tachycardia is a reflection of response to low stroke volume in order to maintain cardiac output. (See "Overview of the acute management of ST-elevation myocardial infarction" and "Overview of the acute management of non-ST-elevation acute coronary syndromes", section on 'Initial medical therapy'.)

- In patients with CS in whom revascularization is planned, we give aspirin 325 mg. We do not give an oral $P2Y_{12}$ receptor blocker until after diagnostic coronary angiography in the event that coronary artery bypass surgery needs to be performed. (See "Acute ST-elevation myocardial infarction: Antiplatelet therapy", section on 'Summary and recommendations' and "Acute non-ST-elevation acute coronary syndromes: Early antiplatelet therapy", section on 'Summary and recommendations'.)

REPERFUSION/REVASCULARIZATION

Early, successful revascularization improves outcomes compared with medical therapy. Some patients will require only percutaneous coronary intervention (PCI) of the infarct-related artery, others may require immediate coronary artery bypass surgery (CABG), and some may require both. This may involve emergent percutaneous recanalization of the infarct-related artery in the catheterization laboratory followed by emergent/urgent or staged revascularization with CABG.

Our approach — Our recommendations for the use of reperfusion therapy in patients with MI complicated by CS are similar to those for most patients with MI and differ principally in the level of evidence. For many patients, we proceed with immediate PCI of culprit lesions rather than CABG due to the much shorter time needed to establish reperfusion. (See "Acute ST-elevation myocardial infarction: Selecting a reperfusion strategy", section on 'Fibrinolysis'.)

Our approach to reperfusion is as follows:

- Immediate CABG is the reperfusion treatment of choice for patients with associated mechanical complications.

- In patients with ST-elevation MI (STEMI), revascularization with either PCI or CABG is preferred to fibrinolytic therapy if it can be performed in a timely manner (within 120 minutes from initial hospital presentation for PCI) [37]. (See "Acute ST-elevation myocardial infarction: Selecting a reperfusion strategy", section on 'Summary and recommendations'.)

- For MI (either STEMI or non-ST-elevation MI [NSTEMI]) patients with one- or two-vessel coronary disease and technically suitable lesions, immediate PCI of the culprit vessel is preferred to CABG [38]. (See 'Percutaneous coronary intervention' below.)

  Immediate reperfusion with PCI is also preferred to CABG for amenable severe stenosis or total occlusion of the left main coronary artery, particularly if it is due to thrombotic occlusion. (See 'Left main coronary artery disease' below.)

  Patients with advanced three-vessel disease, with or without left main coronary artery disease, who have no identifiable single culprit lesion should be considered for immediate CABG if available.

- Patients admitted to hospitals without facilities for revascularization who do not have contraindications to invasive care should be immediately transferred to a tertiary care center with such facilities [12]. If there is an anticipated long delay in transport for cardiac catheterization **and** the risks of fibrinolysis are low **and** the duration of MI symptoms is less than three hours, we recommend rapid initiation of fibrinolytic therapy (<30 minutes) prior to transfer.

- Patients who survive initial therapy that does not include emergency PCI or CABG should be referred for immediate coronary angiography and potential revascularization [12]. This recommendation is based upon the high prevalence of a low left ventricular ejection fraction and three-vessel or left main coronary artery disease [39], settings in which revascularization is associated with a long-term reduction in mortality.

- For patients with ongoing evidence of systemic hypoperfusion despite revascularization and inotropic therapy (refractory CS) who may be candidates for transplantation or durable left ventricular assist device therapy (LVAD), we typically employ initiation of temporary mechanical circulatory support. Device selection is driven by the degree of hypoperfusion and whether the patient has univentricular or biventricular involvement

(see 'Other mechanical devices' above). Device choice and device optimization may be driven by data from continuous right heart catheterization monitoring in this cohort of patients. (See 'Role of pulmonary artery catheter' above.)

Fibrinolysis — Although reperfusion can be established with fibrinolysis in patients with STEMI, we prefer direct revascularization with either PCI or CABG. Fibrinolysis is recommended if PCI is not possible or if it is significantly delayed [40,41]. (See "Acute ST-elevation myocardial infarction: Selecting a reperfusion strategy" and "Acute ST-elevation myocardial infarction: The use of fibrinolytic therapy".)

Based upon the apparent benefit in those who did not receive immediate revascularization and the absence of adverse effects in those who underwent invasive procedures, fibrinolytic therapy should be given to patients who present to a facility without primary PCI and who cannot be transferred for primary PCI capability in a timely manner, particularly those who present within three hours of symptoms. However, all patients should be transferred to a PCI-capable facility as soon as possible.

Percutaneous coronary intervention — In appropriate patients, PCI is preferred to no reperfusion or to fibrinolysis for patients with CS complicating acute STEMI and NSTEMI [42-47] (see 'Our approach' above). Immediate PCI should be performed on the culprit lesion(s) of the infarct-related artery. For most patients with CS and acute MI, we do not perform nonculprit lesion PCI. (See "Acute coronary syndromes: Approach to nonculprit lesions".)

Nonrandomized [45] and randomized trials [46] have shown mortality benefit to early revascularization with PCI. Patients with CS on admission have a higher in-hospital mortality than the majority of patients who develop shock after hospitalization [48,49]. However, in the SHOCK trial and registry, patients with shock on admission derived the same in-hospital mortality benefit from emergency revascularization (60 versus 82 percent) as those who developed shock later (46 versus 62 percent) [48].

A 2017 meta-analysis of 10 cohort studies that included over 6000 patients with CS showed a higher mortality with multivessel PCI than with culprit-lesion-only PCI (37.5 versus 28.8 percent; $p = 0.001$) [50]. The CULPRIT-SHOCK trial, published after the meta-analysis, randomly assigned 706 patients with both STEMI and NSTEMI with CS to PCI of the culprit lesion only, with the option of stage revascularization of nonculprit lesions or immediate multivessel PCI [51]. Staged revascularization was performed in 17.7 percent of the culprit-lesion-only group. The following findings were noted:

- At 30 days, the primary end point (a composite of death or renal failure leading to renal-replacement therapy) occurred less often in the culprit-lesion-only group (45.9 versus 55.4

percent; relative risk [RR] 0.83, 95% CI 0.71-0.96).

- At 30 days, the secondary end point (all-cause death) occurred less often in the culprit-lesion-only group (RR 0.84, 95% CI 0.72-0.98).

- At one year, death occurred in 50.0 and 56.9 percent of the two groups, respectively (RR 0.88, 95% CI 0.76-1.01) [52].

  The rate of a composite of death or recurrent infarction was 50.9 and 58.4 percent, respectively (RR 0.87, 95% CI 0.76-1.00). Repeat revascularization occurred more often with culprit-only PCI (32.3 versus 9.4 percent; RR 3.44, 95% CI 2.39-4.95) as did rehospitalization for heart failure (5.2 versus 1.2 percent; RR 4.46, 95% CI 1.53-13.04).

Left main coronary artery disease — Patients with CS who have an occluded or severely stenosed left main coronary artery present complex clinical challenges. (See "Left main coronary artery disease".)

- Immediate CABG is the reperfusion treatment of choice if there are associated mechanical complications. (See "Acute myocardial infarction: Mechanical complications".)

- In patients without mechanical complications, and when the left main coronary artery is the culprit lesion on angiography (eg, thrombotic occlusion, high-grade lesion), we proceed with immediate PCI in a manner analogous to primary PCI in STEMI. The rationale is to provide reperfusion therapy as rapidly as possible. Registry data show that PCI of the left main coronary artery in shock is feasible, is used more often than CABG, and is performed much earlier. Although adjustment for covariates, including considering PCI and CABG as time-varying covariates, suggests better outcomes for CABG, the data are confounded by selection of lower-risk patients for CABG who on average survived for several days [53].

- Immediate CABG should be considered for patients with left main coronary artery stenosis associated with severe three-vessel disease, particularly if a high SYNTAX score is present. (See 'Coronary artery bypass graft surgery' below and "Revascularization in patients with stable coronary artery disease: Coronary artery bypass graft surgery versus percutaneous coronary intervention", section on 'Outcomes based on lesion severity'.)

Determinants of outcomes — The clinical response to primary PCI is highly variable. While some patients improve rapidly, others show no immediate hemodynamic improvement, and a few transiently deteriorate after reperfusion is established, particularly if there is late reperfusion. This is also true for late reperfusion with fibrinolysis. Data from the

nonrandomized SHOCK trial registry suggest that the in-hospital mortality after PCI is related to the degree of reperfusion achieved in the infarct-related artery [54]. Among 276 patients undergoing PCI, the mortality for Thrombolysis in Myocardial Infarction (TIMI) grade 3 (normal), grade 2, or grade 0/1 flow was 33, 50, and 86 percent, respectively. A similar relationship to TIMI flow grade was noted in a report from the ALKK primary PCI registry in Germany (37, 66, and 78 percent in-hospital mortality, respectively) [55]. (See "Diagnosis and management of failed fibrinolysis or threatened reocclusion in acute ST-elevation myocardial infarction", section on 'Primary failure'.)

The time from symptom onset to PCI may be another determinant of outcome. In the ALKK report, the in-hospital mortality was 44 percent in patients receiving primary and rescue PCI, as well as urgent CABG, within three hours from symptom onset; 45 percent from three to six hours; 54 percent from 6 to 12 hours; and 58 percent from 12 to 24 hours [55]. However, the benefits of primary PCI were seen in both early and late presenters in the SHOCK trial [46]. As a result, late presenters should not be denied emergency revascularization based upon timing alone. (See 'Percutaneous coronary intervention' above.)

Coronary artery bypass graft surgery — The majority of patients with CS after MI have significant left main coronary artery or three-vessel disease (16 and 53 percent, respectively, in the SHOCK trial registry) [39]. In such patients, the ability to achieve complete revascularization makes CABG a potentially critical therapeutic strategy. A surgical approach also permits the correction of concomitant severe mitral regurgitation, which is often present. Pooled data on 370 patients in 22 studies revealed an in-hospital mortality rate of 36 percent when CABG was performed during the hospitalization for acute MI with CS [43]. (See "Coronary artery bypass graft surgery in patients with acute ST-elevation myocardial infarction", section on 'Cardiogenic shock'.)

The relatively low mortality rate in these nonrandomized series may reflect a true benefit or selection bias in which patients at lowest risk are selected for CABG. However, similar findings (41 percent overall mortality) were noted in the patients who underwent emergency CABG in the SHOCK trial within six hours of randomization [46]. Despite this benefit, CABG is underutilized in the community setting. In a review from the National Registry of Myocardial Infarction of 25,311 patients with CS seen from 1995 to 2004 in the United States, the overall rate of immediate CABG was stable at about 3 percent [56].

The relative efficacy of PCI and CABG was evaluated in the 128 patients with predominant left ventricular failure who underwent emergency revascularization in the SHOCK trial [57]. Not surprisingly, the 47 patients (37 percent) who underwent CABG were significantly more likely to have diabetes and three-vessel or left main coronary artery disease; 85 percent of these

patients received two or more grafts, and 52 percent received three or more grafts. Despite the more extensive disease in the CABG group, overall survival was similar to PCI at 30 days (57 versus 56 percent with PCI) and one year (47 versus 52 percent). The similar outcomes, despite the worse disease in patients undergoing CABG, may reflect in part the higher rate of complete revascularization (87 versus 23 percent with PCI).

Long-term outcome — In addition to the short-term benefits, follow-up reports from the SHOCK trial demonstrated that the benefit of revascularization persists for many years. At one year, early revascularization was associated with a lower mortality rate (eg, 53 versus 66 percent) [47]. In addition, the patients who were assigned to emergency revascularization were significantly more likely to remain stable after discharge (71 versus 44 percent at one year) and less likely to worsen or die after 30 days (15 versus 34 percent) [58].

The mortality benefit persisted at six years (67 versus 80 percent with initial medical stabilization) [59]. The previously observed significant interaction between age and treatment effect was no longer evident on long-term follow-up.

Evidence of long-term benefit was also suggested in a subset analysis from the Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries (GUSTO-I) trial of over 39,000 patients who were alive at 30 days after an acute MI, 1306 of whom had been in CS [60]. Revascularization within 30 days was performed in 44 percent of the patients with CS; the one-year mortality in these patients was significantly lower than those who were not revascularized (8 versus 15 percent, odds ratio 0.6 after adjusting for differences in baseline characteristics). Not surprisingly, the survivors of CS had higher one-year mortality than those without shock (12 versus 3 percent).

PROGNOSIS

The short-term prognosis of CS is directly related to the severity of the hemodynamic disorder. Patients most commonly succumb to multiorgan dysfunction due to ongoing organ hypoperfusion [7]. In-hospital mortality is over 50 percent [61,62]. In 2019, the Society for Cardiovascular Angiography and Intervention proposed a classification scheme for patients admitted to a cardiac intensive care unit with CS [63]. In a subsequent single-center study, patients were retrospectively categorized into one of five stages (of severity) of CS based on the presence or absence of hypotension/tachycardia, hypoperfusion, clinical deterioration, or refractory shock [64]. Each higher CS stage was significantly associated with increased hospital mortality. Although this classification system needs additional validation, it has the potential to predict the risk of short-term death.

Long-term survival in patients with myocardial infarction (MI) complicated by CS is improved with timely revascularization in the acute setting, and functional status and quality of life in most survivors are excellent [58,59]. However, in the Intraventricular Aortic Balloon Pump Cardiogenic Shock (IABP-SHOCK) trial (see 'Intraaortic balloon pump' above), mortality was approximately 67 percent at nearly six years [16].

The following have been identified as risk factors for short- and/or long-term mortality in CS:

- Increasing age; clinical signs of severe hypoperfusion such as oliguria, cold or clammy extremities, or biochemical evidence such as increasing lactate [65,66]; elevated creatinine; and neurological involvement such as history of stroke [14,16,67] and anoxic damage [22].
- Abnormal hemodynamic parameters such as reduced mean arterial pressure (MAP) despite supportive therapies, reduced cardiac output (CO), cardiac index, and cardiac power index (MAP x CO/451 x body surface area in m^2) [68].
- Possible non-ST-elevation MI as opposed to ST-elevation MI (STEMI) [69].
- Mortality varies significantly with the location of the culprit lesion and is higher in patients with a left main coronary artery or saphenous vein graft lesion than in those with circumflex, left anterior descending, or right coronary artery lesions (79 and 70 percent versus 37 to 42 percent). Right coronary culprit lesions were associated with the best prognosis [70]. Multivessel disease or prior coronary artery bypass graft surgery are also risk factors for mortality [14,22].
- Echocardiographic predictors of outcome are reduced left ventricular ejection fraction and worsening severity of mitral regurgitation [70,71].
- The time from symptom onset to reperfusion is an important determinant of mortality in patients with STEMI who undergo primary percutaneous coronary intervention [72,73]. (See "Primary percutaneous coronary intervention in acute ST elevation myocardial infarction: Determinants of outcome".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Non-ST-elevation acute coronary syndromes (non-ST-elevation myocardial infarction)" and "Society guideline links: ST-elevation myocardial infarction (STEMI)" and "Society guideline links: Percutaneous coronary intervention".)

SUMMARY AND RECOMMENDATIONS

- For patients with cardiogenic shock (CS) and acute myocardial infarction (MI), we withhold beta blockers until hemodynamics have stabilized. If possible, we give aspirin 325 mg but do not give a $P2Y_{12}$ receptor blocker. (See 'Medical therapy' above.)

- For patients with CS and severe hypotension (systolic blood pressure <80 mmHg), we recommend vasopressors for initial management (**Grade 1B**). Although there is no definitive evidence of the superiority of one vasopressor over another, we suggest beginning with norepinephrine rather than dopamine (**Grade 2B**). (See 'Vasopressors and inotropes' above.).

- For patients in whom mechanical complications (eg, acute mitral regurgitation or rupture of the ventricular septum) are not present and for whom revascularization is planned, we recommend not routinely placing an intraaortic balloon pump (**Grade 1B**). (See 'Intraaortic balloon pump' above.)

- All patients with CS complicating MI should undergo an attempt at reperfusion. (See 'Reperfusion/revascularization' above.)

- Our recommendations for the use of reperfusion therapy in patients with MI complicated by CS are similar to those for most patients with MI (see "Acute ST-elevation myocardial infarction: Selecting a reperfusion strategy", section on 'Summary and recommendations'):

  - For patients with ST-elevation myocardial infarction (MI), we recommend revascularization as opposed to fibrinolytic therapy (**Grade 1A**). This recommendation requires that diagnostic coronary angiography be performed within 120 minutes of initial hospital presentation.

    For those patients who cannot undergo timely coronary angiography, we recommend fibrinolytic therapy rather than no immediate reperfusion (**Grade 1B**).

  - For patients with one- or two-vessel disease who do not have mechanical complications, we recommend percutaneous coronary intervention (PCI) of the infarct-related artery as opposed to coronary artery bypass graft surgery (CABG) (**Grade 1B**).

  - For patients with three-vessel or left main coronary artery disease who do not have mechanical complications (eg, acute mitral regurgitation or rupture of either the ventricular septal or free walls), the decision to perform immediate PCI or emergency CABG, with or without PCI of the infarct-related artery, should be decided based on the

availability of rapid CABG, and factors such as the likelihood of successful revascularization with PCI, the extent of disease, and the skill level/experience of the operators. Shock teams, which include interventional cardiologists and cardiac surgeons, should be involved in decision-making.

- Patients with mechanical complications should undergo immediate CABG and an attempt at repairing the mechanical defect.
- For patients with non-ST-elevation MI, we recommend that revascularization be performed as soon as possible as opposed to either fibrinolytic therapy or no reperfusion (**Grade 1B**).

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GRAPHICS

Vasopressors and inotropes in treatment of acute hypotensive states and shock: Adult dose and selected characteristics

Agent	United States trade name	Initial dose	Usual maintenance dose range	Range of maximum doses used in refractory shock
Vasopressors (alpha-1 adrenergic)				
Norepinephrine (noradrenaline)	Levophed	5 to 15 mcg/minute (0.05 to 0.15 mcg/kg/minute) Cardiogenic shock: 0.05 mcg/kg/minute	2 to 80 mcg/minute (0.025 to 1 mcg/kg/minute) Cardiogenic shock: 0.05 to 0.4 mcg/kg/minute	80 to 250 mcg/minute (1 to 3.3 mcg/kg/minute)
Epinephrine (adrenaline)	Adrenalin	1 to 15 mcg/minute (0.01 to 0.2 mcg/kg/minute)	1 to 40 mcg/minute (0.01 to 0.5 mcg/kg/minute)	40 to 160 mcg/minute (0.5 to 2 mcg/kg/minute)

Phenylephrine	Neo-Synephrine, Vazculep	40 to 160 mcg/minute until stabilized (alternatively, 0.5 to 2 mcg/kg/minute)	20 to 400 mcg/minute (0.25 to 5 mcg/kg/minute)	80 to 730 mcg/minute (1.1 to 9.1 mcg/kg/minute)	

Dopamine	Inotropin	2 to 5 mcg/kg/minute	2 to 20 mcg/kg/minute	20 mcg/kg/minute	

Antidiuretic hormone				
Vasopressin (arginine-vasopressin)	Pitressin, Vasostrict	0.03 units/minute	0.01 to 0.04 units/minute (not titrated)	Doses >0.04 units/minute can cause cardiac ischemia and should be reserved for salvage therapy
Inotrope (beta$_1$ adrenergic)				
Dobutamine	Dobutrex	Usual: 2 to 5 mcg/kg/minute (range: 0.5 to 5 mcg/kg/minute; lower doses for less	2 to 10 mcg/kg/minute	20 mcg/kg/minute

		severe cardiac decompensation)		
Inotrope (nonadrenergic, PDE_3 inhibitor)				
Milrinone	Primacor	0.125 to 0.25 mcg/kg/minute	0.125 to 0.75 mcg/kg/minute	0.75 mcg/kg/minute

- All doses shown are for intravenous (IV) administration in adult patients. The initial doses shown in this table may differ from those recommended in immediate post-cardiac arrest management (ie, advanced cardiac life support). For details, refer to the UpToDate topic review of post-cardiac arrest management in adults, section on hemodynamic considerations.
- Vasopressors can cause life-threatening hypotension and hypertension, dysrhythmias, and myocardial ischemia. They should be administered by use of an infusion pump adjusted by clinicians trained and experienced in dose titration of intravenous vasopressors using continuous noninvasive electronic monitoring of blood pressure, heart rate, rhythm, and function. Hypovolemia should be corrected prior to the institution of vasopressor therapy. Reduce infusion rate gradually; avoid sudden discontinuation.
- Vasopressors can cause severe local tissue ischemia; central line administration is preferred. When a patient does not have a central venous catheter, vasopressors can be temporarily administered in a low concentration through an appropriately positioned peripheral venous catheter (ie, in a large vein) for less than 24 hours. The examples of concentrations shown in this table are useful for peripheral (short-term) or central line administration. Closely monitor catheter site throughout infusion to avoid extravasation injury. In event of extravasation, prompt local infiltration of an antidote (eg, phentolamine) may be useful for limiting tissue ischemia. Stop infusion and refer to extravasation management protocol.
- Vasopressor infusions are high-risk medications requiring caution to prevent a medication error and patient harm. To reduce the risk of making a medication error, we suggest that centers have available protocols that include steps on how to prepare and administer vasopressor infusions using a limited number of standardized concentrations. Examples of concentrations and other detail are based on recommendations used at experienced centers; protocols can vary by institution.

D5W: 5% dextrose water; MAP: mean arterial pressure; NS: 0.9% saline.

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