Cardiogenic Shock

3

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3.1 Introduction

Cardiogenic shock is a serious complication of acute myocardial infarction and is an important cause of hospital death. Cardiogenic shock is a condition in which your heart suddenly can't pump enough blood to meet your body's needs. The condition is most often caused by a severe heart attack. Cardiogenic shock is rare, but it's often fatal if not treated immediately. If treated immediately, about half the people who develop the condition survive. The incidence of cardiogenic shock is about 5% in patients with acute myocardial infarction (AMI) and three times more ST-segment elevation myocardial infarction (STEMI) than in non-STEMI [1]. Recent advances in early treatment, technological advancement, and pharmacologic treatment have improved the prognosis of patients and improved long-term survival and quality of life. Therefore, the mortality rate due to cardiogenic shock is also decreasing, and the prognosis of the high-risk patients is better than the previous one [2].

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Table 3.1 The definition of CS consists of hemodynamic instability of various parameters

I.	Persistent hypotension: systolic blood pressure <90 mmHg or mean arterial pressure 30 mmHg lower than baseline
II.	Severe reduction in cardiac index: <1.8 L/min/m^2 without support or <2.0–2.2 L/min/m^2 with support
III.	Adequate or elevated filling pressure: left ventricular end-diastolic pressure >18 mmHg or right ventricular end-diastolic pressure >10–15 mmHg.

3.2 Definition of Cardiogenic Shock

Cardiogenic shock is a life-threatening medical condition resulting from an inadequate circulation of blood due to primary failure of the ventricles of the heart to function effectively (Table 3.1). Signs of tissue hypoperfusion include low urine production, cool extremities, and altered mental of consciousness.

3.3 Pathophysiology

The most common cause of cardiogenic shock is pump failure due to extensive myocardial infarction (MI) with damage to the heart muscle and subsequent depression of myocardial contractility. Additional causes of cardiogenic shock are listed in Table 3.2 [3]. Other mechanical

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Table 3.2 Causes of cardiogenic shock

Acute myocardial infarction
Pump failure
Large infarction
Smaller infarction with preexisting left ventricular dysfunction
Infarction extension
Reinfarction
Infarction expansion
Mechanical complications
Acute mitral regurgitation caused by papillary muscle rupture
Ventricular septal defect
Free-wall rupture
Pericardial tamponade
Right ventricular infarction
Other conditions
End-stage cardiomyopathy
Myocarditis
Myocardial contusion
Prolonged cardiopulmonary bypass
Septic shock with severe myocardial depression
Left ventricular outflow tract obstruction
Aortic stenosis
Hypertrophic obstructive cardiomyopathy
Obstruction to left ventricular filing
Mitral stenosis
Left atrial myxoma
Acute mitral regurgitation (chordal rupture)
Acute aortic insufficiency

complications following myocardial injury after MI are acute mitral regurgitation resulting from papillary muscle rupture, ventricular septal defect, and free-wall rupture. Mechanical complication must be strongly suspected in patients with cardiogenic shock complicating non-anterior MI, especially complications of a first MI.

Sepsis, hemorrhage, and bowel ischemia also cause cardiogenic shock, which severely reduces the myocardial contractility. These causes require proper treatment through suspicion or recognition of the cause as well as support of the myocardial function.

Acute myocarditis, takotsubo cardiomyopathy, hypertrophic cardiomyopathy, and myocardial contusion may lead to cardiogenic shock in the absence of significant coronary artery disease. Acute valvular regurgitation of left ventricular (LV) output caused by endocarditis or chordal rupture may also cause cardiogenic shock. Acute aortic insufficiency due to aortic dissection, cardiac tamponade, or massive pulmonary embolism can present as cardiogenic shock without associated pulmonary edema.

Cardiogenic shock is a clinical syndrome characterized by systemic hypotension and hypoperfusion secondary to insufficient cardiac output. LV pump failure is a major cause of cardiogenic shock, but right ventricular (RV) failure and macro/microcirculation system are also responsible for cardiogenic shock. Recent research has suggested that the peripheral vasculature, neurohormonal, and cytokine systems play a role in the pathogenesis and persistence of cardiogenic shock [4–10].

In general, myocardial dysfunction is severe enough to cause cardiogenic shock. In the case of cardiogenic shock, myocardial contractility disturbance causes a decrease in the afterload, lowering the blood pressure, resulting in systemic hypoperfusion. The mean depression of LV ejection fraction (EF) is moderate to severe (30%), with a wide range of EF and LV sizes recorded [11]. Metabolic disorders occur in the areas of the remote myocardium and in the infarct region [12]. Hypoperfusion causes release of catecholamines, which increase contractility and peripheral blood flow, but catecholamines also increase myocardial oxygen demand and cause proarrhythmic and myocardiotoxic effects. Cardiogenic shock is not the only result of severe depression of LV function due to extensive myocardial ischemia or injury. Depressed myocardial contractility is accompanied by inadequate systemic vasoconstriction as a result from a systemic inflammatory response to extensive myocardial injury in cardiogenic shock.

RV failure can contribute to cardiogenic shock, but the ratio of predominant cardiogenic shock due to mainly RV failure is only 5% [13]. However, cardiogenic shock due to isolated RV failure is associated with a higher risk of death, as with LV failure. RV failure reduces cardiac output and ventricular interdependence, eventually decreasing LV filling. Treatment of RV failure with cardiogenic shock is focused on ensuring adequate right-heart filling pressure to maintain cardiac output and adequate LV preload.

Decreased cardiac output due to MI and progressive myocardial ischemia cause the release of catecholamines, which constrict peripheral arterial vessels to maintain the perfusion of important organs. Activation of the neurohormone cascade promotes salt and moisture retention. This can improve perfusion, but worsens pulmonary edema.

The reflex mechanism of increased systemic vascular resistance (SVR) is not generally effective, as evidenced by the variable SVR, with average SVR during cardiogenic shock in the normal range despite vasopressor therapy [14].

Excess nitric oxide (NO) can also contribute to systemic inflammatory response syndrome. MI is associated with increased expression of inducible NO synthase, which leads to excess NO, which inhibits vasoconstriction, myocardial function, and catecholamine action [9, 10].

3.4 Treatment and Management

3.4.1 Initial Approach and Diagnosis

Cardiogenic shock is defined as hypotension (SBP <90 mmHg) despite adequate filling status with signs of hypoperfusion. A patient in cardiogenic shock should undergo immediate comprehensive assessment. Chest X-ray, electrocardiogram (ECG), and echocardiography are required immediately in all patients with suspected cardiogenic shock. Chest X-ray can be a useful test for the diagnosis of cardiogenic shock. Pulmonary venous congestion, pleural effusion, interstitial or alveolar edema, and cardiomegaly are the most specific findings for cardiogenic shock, although in up to 20% of patients with cardiogenic shock chest X-ray is nearly normal [15]. ECG is rarely normal in cardiogenic shock. It is also helpful in identifying underlying cardiac disease and potential precipitants [16]. Immediate echocardiography is mandatory only in patients with hemodynamic instability in cardiogenic shock and in patients suspected of acute life-threatening structural or functional cardiac abnormalities. The following laboratory assessments should be performed at admission on the blood of all patients with cardiogenic shock: cardiac troponin, natriuretic peptides (BNP), blood urea nitrogen (BUN), creatinine, electrolytes (sodium, potassium), liver function tests, thyroid-stimulating hormone (TSH), serum glucose complete blood count, and D-dimer.

A plasma BNP level should be measured in all patients with acute dyspnea and suspected cardiogenic shock to help in the differentiation of cardiogenic shock from noncardiac causes of acute dyspnea. BNP have high sensitivity, and normal levels in patients with suspected acute heart failure make the diagnosis unlikely [17–21]. The level of BNP is an important predictor of cardiovascular events (reinfarction, cardiogenic shock, sustained ventricular tachycardia, ventricular fibrillation, angina, symptoms of left ventricular dysfunction) in patients with acute coronary syndrome and provides better predictive power than the troponin level [22].

Measurement of cardiac troponin is useful for detection of acute coronary syndrome (ACS) as the underlying cause of cardiogenic shock. However, elevated concentrations of circulating cardiac troponins are detected in the vast majority of patients with cardiogenic shock, often without obvious myocardial ischemia or an acute coronary event, suggesting ongoing myocyte injury or necrosis in these patients [23]. The patients of cardiogenic shock have been found to have close association with increased level of serum cardiac troponin-I. The troponin ratio was independently associated with the development of cardiogenic shock [24, 25].

In patients with cardiogenic shock complicating ACS, an immediate coronary angiography is recommended with an intent to perform coronary revascularization. Invasive monitoring with an arterial line should also be considered.

3.4.2 General Support Measures

Prehospital emergency medical service should be considered for transfer to a specialized cardiac care center if cardiogenic shock is suspected. Emergency department care is a temporizing measure during the preparation for revascularization in the cardiac catheterization laboratory or surgical intervention for mechanical failure.

Antithrombotic therapy with aspirin and heparin should be given as routinely recommended for MI. Clopidogrel may be deferred until after emergency angiography, because on the basis of angiographic findings coronary artery bypass grafting (CABG) may be performed immediately. Clopidogrel is indicated in all patients who undergo percutaneous coronary intervention (PCI), and on the basis of extrapolation of data from MI patients who were not in shock it should also be useful in patients with shock as well. Negative inotropes and vasodilators (including nitroglycerin) should be avoided. Arterial oxygenation and near-normal pH should be maintained to minimize ischemia. Intensive insulin therapy improves survival in hyperglycemic critically ill patients and is recommended for use in complicated MI. There should be a low threshold to institute mechanical ventilation via mask or endotracheal tube. Positive end-expiratory pressure decreases preload and afterload. Mechanical ventilation also reduces work of breathing (Fig. 3.1).

3.4.3 Hemodynamic Management

Fluid is given in RV infarct with hypotension. Because some patients with cardiogenic shock develop hypotension without pulmonary edema, an appropriate amount of fluid can be administered. If there is no improvement in perfusion with fluid challenge, or there is hypoperfusion with pulmonary edema, vasopressors or inotropes are considered.

Pulmonary artery (PA) catheterization is frequently performed to confirm the diagnosis of cardiogenic shock, to ensure adequate filling pressure, and to guide changes in therapy. Individualized PA catheter use is recommended for MI patients with severe hypotension [26]. However, many centers have chosen to manage cardiogenic shock without PA catheterization. Clinical evaluation with echocardiography is a reasonable alternative. Both PA systolic pressure and wedge pressure can be accurately estimated with Doppler echocardiography, and in particular the finding of a short mitral deceleration time

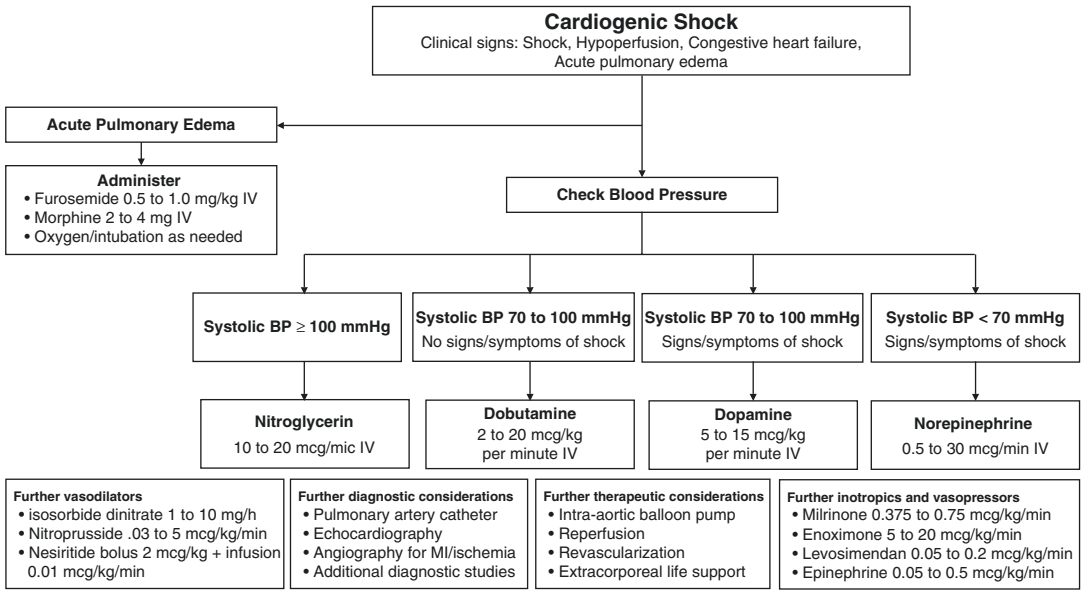

Fig. 3.1 Emergency management of complicated ST-elevation myocardial infarction. The emergency management of patients with cardiogenic shock, acute pulmonary edema, or both is outlined. *SBP* systolic blood pressure, *IV* intravenous, *BP* blood pressure, *MI* myocardial infarction. *Furosemide less than 0.5 mg/kg for new-onset acute pulmonary edema without hypovolemia; 1 mg/kg for acute or chronic volume overload, and renal insufficiency. Combinations of medications, e.g., dobutamine and dopamine, may be used

(≤140 ms) is highly predictive of pulmonary capillary wedge pressure ≥20 mm Hg in cardiogenic shock [27].

Pharmacological treatment, such as inotropic and vasopressor agents, should be used in the lowest possible doses. Higher vasopressor doses are associated with poorer survival [28]. This indicates both severe hemodynamic disturbances and direct toxic effects. Use of inotropic and vasopressor agents is always required to maintain coronary and systemic perfusion until the IABP is placed or until the shock is resolved. There are very little studies on comparisons of vasopressors. The American College of Cardiology/American Heart Association guidelines recommend norepinephrine for more severe hypotension due to its high potency [26]. Although norepinephrine has inotropic properties, dobutamine is often necessary in this condition. Use of dopamine in this setting can be associated with excess risk [29].

Levosimendan may also be used in combination with an inotropic agent or vasopressor. Levosimendan infusion in severe cardiogenic shock complicating AMI in addition to dobutamine and norepinephrine improved survival and cardiovascular hemodynamics without leading to hypotension [30, 31]. Milrinone can also be another alternative to nonischemic patients [32, 33].

3.4.4 Mechanical Support (Fig. 3.2)

Intra-aortic balloon pump (IABP) counterpulsation has long been the mainstay of mechanical therapy for cardiogenic shock. Use of an IABP improves coronary and peripheral perfusion via diastolic balloon inflation and augments LV performance via systolic balloon deflation with an acute decrease in afterload. Accurate timing of inflation and deflation provides optimal support. Not every patient has a hemodynamic response to IABP; response predicts better outcome [34]. IABP support should be instituted as quickly as possible, even before any transfer for revascularization if a skilled operator is available and insertion can be performed quickly.

The use of IABP counterpulsation can be useful for patients with cardiogenic shock after STEMI who do not quickly stabilize with pharmacological therapy [35].

In the large National Registry of Myocardial Infarction, IABP use was independently associated with survival at centers with higher rates of IABP use, whether PCI, fibrinolytic therapy, or no reperfusion had been used [36]. Complications associated with IABP are less common in the modern era; in the largest series, the overall and major complication rates were 7.2% and 2.8%, respectively [37] (Fig. 3.3).

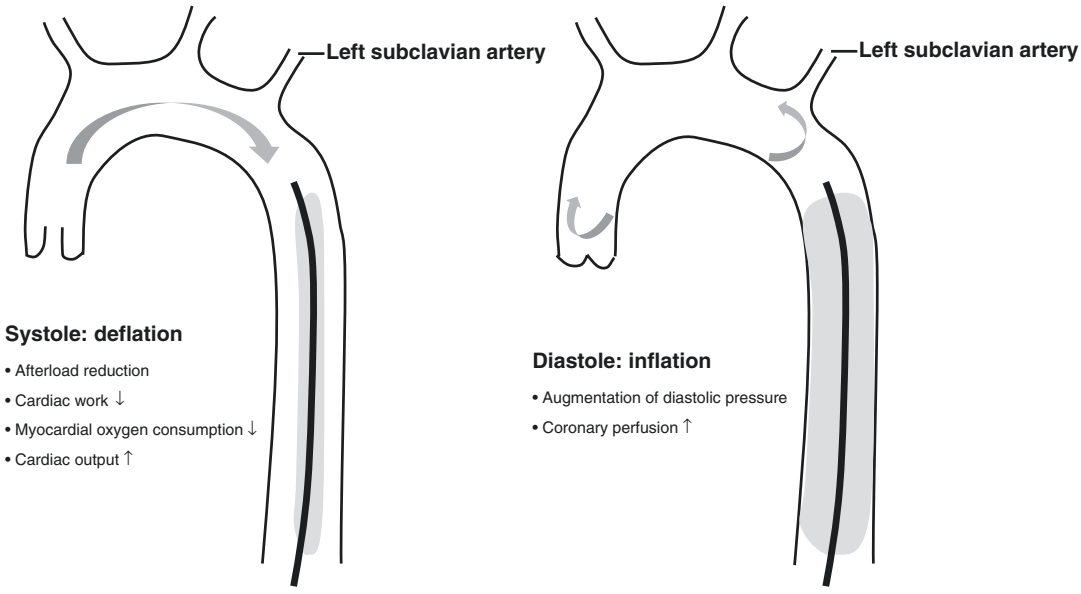

Fig. 3.2 Intra-aortic counterpulsation balloon pump

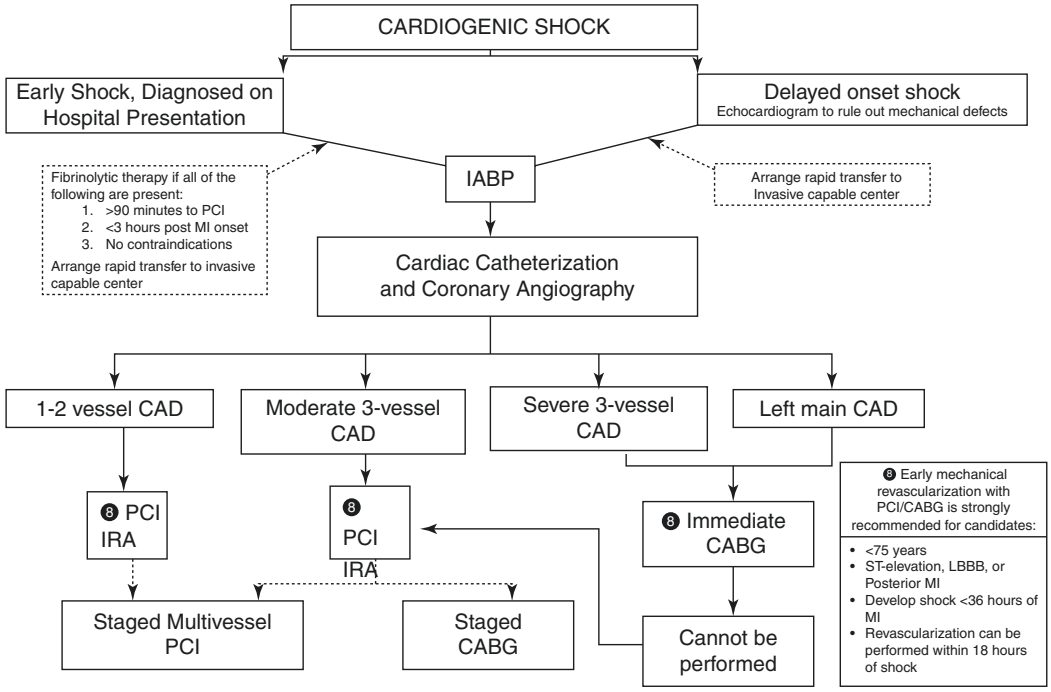

Fig. 3.3 Recommendations for initial reperfusion therapy when cardiogenic shock complicates acute MI. Early mechanical revascularization with PCI or CABG is strongly recommended for suitable candidates 75 years of age and for selected elderly patients (see text). Eighty-five percent of shock cases are diagnosed after initial therapy for acute MI, but most patients develop shock within 24 h. Intra-aortic balloon pump (IABP) is recommended when shock is not quickly reversed with pharmacological therapy, as a stabilizing measure for patients who are candidates for further invasive care. Dashed lines indicate that the procedure should be performed in patients with specific indications only. Recommendations for staged CABG and multivessel PCI are discussed in the text, as are definitions of moderate and severe three-vessel CAD. LBBB indicates left bundle-branch block (*Circulation* 2003;107:2998–3002)

3.4.5 Reperfusion

The survival benefit of early revascularization in cardiogenic shock, reported in several observational studies, was shown convincingly in the randomized SHOCK trial, which found a 13% absolute increase in 1-year survival in patients assigned to early revascularization [11, 38].

Emergency revascularization with either PCI or CABG is recommended in suitable patients with cardiogenic shock due to pump failure after STEMI irrespective of the time delay from MI onset [35]. In the absence of contraindications, fibrinolytic therapy should be administered to patients with STEMI and cardiogenic shock who are unsuitable candidates for either PCI or CABG [31]. Thrombolytic therapy is less effective but is indicated when PCI is impossible or if a delay has occurred in transport for PCI and when MI and cardiogenic shock onset were within 3 h.

As in MI without shock, earlier revascularization is better in cardiogenic shock. Presentation 0–6 h after symptom onset was associated with the lowest mortality among cardiogenic shock patients undergoing primary PCI, in which door-to-angiography times were <90 min in approximately three-fourths of patients [39].

3.4.6 Revascularization Approach: Surgery or PCI (Fig. 3.2)

Revascularization in the SHOCK trial could be percutaneous or surgical. Thirty-seven percent of

patients assigned to the early revascularization strategy underwent CABG at a median of 2.7 h after randomization [40]. Despite a higher prevalence of triple-vessel or left main disease and diabetes mellitus in patients who underwent CABG compared with PCI, survival and quality of life were similar [40, 41].

3.4.7 Total Circulatory Support: LV Assist Devices and Extracorporeal Life Support

Temporary mechanical circulatory support with LV assist devices is theoretically appealing to interrupt the vicious spiral of ischemia, hypotension, and myocardial dysfunction, allowing for recovery of stunned and hibernating myocardium and reversal of neurohormonal derangements. Device-related complications and irreversible organ failure remain major limitations.

Compared with IABP, LV assist devices may provide superior hemodynamic support and serve as more effective bridges to recovery or transplantation, though experience with their use in this setting is limited [42, 43].

Alternative LV assist devices for circulatory support may be considered in patients with refractory cardiogenic shock [35] (Fig. 3.4).

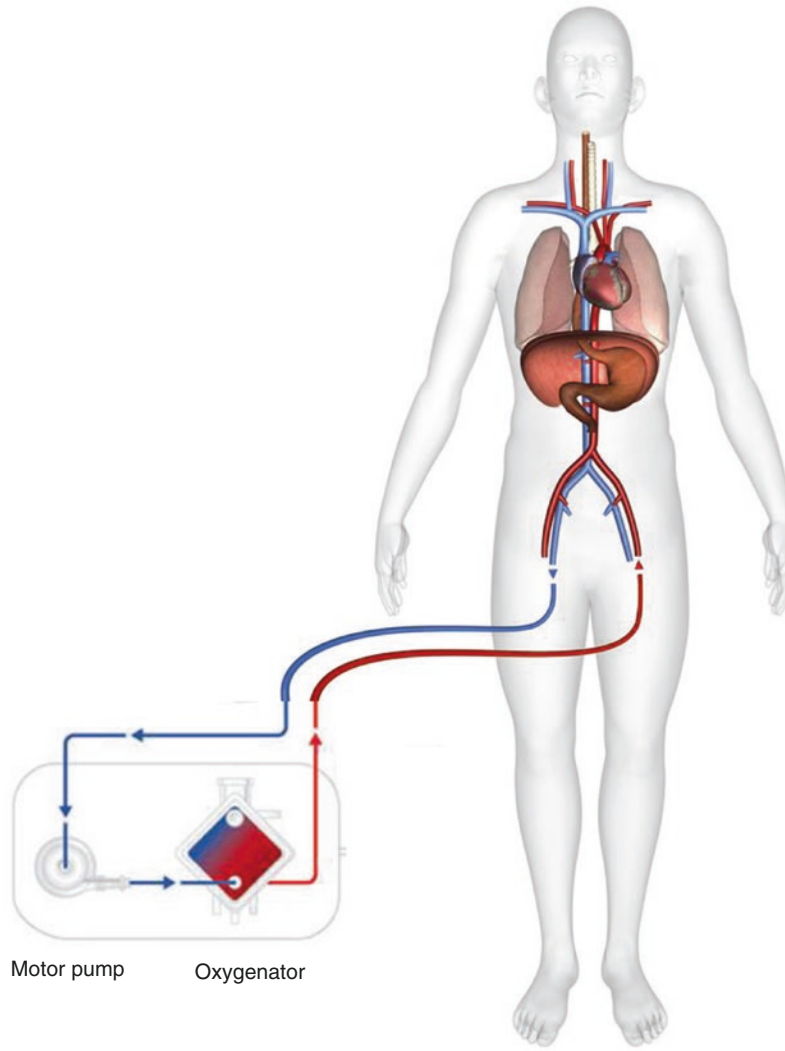

Fig. 3.4 LV assist device and extracorporeal life support

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