Hemodynamics of Cardiogenic Shock

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KEYWORDS

• Cardiogenic shock • Hypoperfusion • Hemodynamic • Pressure-volume loops
• Right heart catheterization

KEY POINTS

- Treatment of cardiogenic shock remains a clinical challenge.
- Greater understanding of the pathophysiology of cardiogenic shock from different causes and of the available treatment strategies is leading to new treatment concepts.
- If the left ventricular dysfunction is based on ischemia or infarction, changes in myocardial perfusion occurring at different stages of the process can play pivotal roles.
- It is important that clinicians appreciate and understand the physiologic meaning of these measurements and take them into account when treating patients with cardiogenic shock.

INTRODUCTION

Cardiogenic shock (CS) represents an advanced state of morbidity along the pathophysiologic pathway of end-organ hypoperfusion caused by reduced cardiac output (CO) and blood pressure (Table 1). Acute coronary syndromes (ACSs) remain the most common cause of CS, with an estimated 100 to 120,000 patients in the United States and Europe subsequently having CS after ACS each year.[1] The spectrum of hypoperfusion states caused by low CO ranges from pre-CS to refractory CS and can be characterized by an array of hemodynamic parameters. This review provides the foundation for a hemodynamic understanding of CS including the use of hemodynamic monitoring for diagnosis and treatment, the cardiac and vascular determinants of CS, and a hemodynamic approach to risk stratification and management of CS.

DEFINITIONS

The spectrum of CS can be divided into pre-CS, CS, and refractory CS—whereby each state is characterized by increasing levels of tissue hypoperfusion and poorer response to treatment but have in common an underlying reduction in CO. Although several different parameters have been used to define CS, the most widely used definitions focus on hemodynamic parameters based on blood pressure and cardiac index (CI).[2] Abnormalities of central venous pressure, pulmonary capillary wedge pressure (PCWP), and systemic vascular resistance (SVR) are typically involved but not always included in CS definitions owing to variability in measurement, and serum lactate is often included to provide objective evidence of end-organ hypoperfusion. For each of these parameters, it is well recognized that there is a continuum ranging from the completely normal condition to a state of refractory CS. Current management strategies rely on this continuum, in particular by drawing attention to patients who are on the verge of significant end-organ dysfunction development in whom early intervention can be particularly effective.

In this regard, the state of pre-CS, also referred to as *nonhypotensive cardiogenic shock*, has been

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Table 1
Definitions of pre–cardiogenic shock, cardiogenic shock, and refractory cardiogenic shock according to clinical and hemodynamic criteria and response to therapy

	Pre-CS (Nonhypotensive)	CS	Refractory CS
Clinical criteria	Signs of peripheral hypoperfusion: Oliguria (urine output <30 mL/h) Cold extremities Altered mental status Increased serum lactate	Signs of peripheral hypoperfusion	Signs of peripheral hypoperfusion
Hemodynamic criteria	SBP ≥90 mm Hg without circulatory support[3]	SBP <90 for >30 min or the need for pharmacologic or intra-aortic balloon pump support to maintain a systolic blood pressure >90 mm Hg or mean arterial pressure 30 mm Hg lower than baseline. Cardiac index <2.2 L/min/m^2. Elevated filling pressures of the left, right, or both ventricles	Same as CS
Response to treatment			Ongoing evidence of tissue hypoperfusion despite administration of adequate doses of 2 vasoactive medications and treatment of the underlying etiology.[9]

discussed and defined as clinical evidence of peripheral hypoperfusion with systolic blood pressure (SBP) more than 90 mm Hg without vasopressor circulatory support. Compared with patients with CS, patients with pre-CS had similar CI, left ventricular ejection fraction (LVEF), and PCWP but higher SVR (1753 ± 675 vs 1389 ± 689 dyn/cm/sec^{-5}, $P = .07$).[3] Notably, patients with pre-CS are often difficult to identify because of subtle signs of hypoperfusion; however, proper diagnosis can be important because of high rates of in-hospital mortality (as high as 43%).[3]

CS has been defined clinically as (1) SBP less than 90 mm Hg for greater than 30 minutes or use of vasopressors to achieve those levels; (2) evidence of pulmonary edema or elevated left ventricle (LV) filling pressures (LV end diastolic pressure or PCWP); (3) evidence of organ hypoperfusion including at least one of the following: (a) change in mental status; (b) cold, clammy skin; (c) oliguria; (d) increased serum lactate.[4,5] Finally, refractory-CS can be defined as CS unresponsive to medical or mechanical support.

The use of invasive hemodynamic measurements is important for definitive diagnosis and for characterizing the extent and site of the cardiac pathologic condition through the measurement of right-sided filling pressures, pulmonary pressures, wedge pressures, and CO.

ETIOLOGY

A multitude of processes can lead to CS. CS can occur acutely in a patient without prior cardiac history or progressively in a patient with long-standing chronic heart failure. The most prevalent etiology of CS remains ACS (including ST-segment elevation myocardial infarction [MI] and non–ST-segment elevation acute coronary system), which accounts for nearly 80% of cases. Despite advances in treatment and revascularization, CS remains the most lethal complication of MI, with mortality rates ranging from 38% to 65% in different cohorts.[6–8] CS in ACS results most commonly from myocardial dysfunction caused by ischemia or infarct but can also be caused by mechanical