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Maya Guglin

*Indiana University, mguglin@iu.edu*

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**Recommended Citation**

Guglin, Maya (2022) "Hemodynamic Goals in Shock Management: Is There One Target for All?," *Journal of Shock and Hemodynamics*: Vol. 1(2) :E2022126 [https://doi.org/10.57905/josh/e2022126](https://doi.org/10.57905/josh/e2022126)

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2022 Symposium Presentation

Hemodynamic Goals in Shock Management: Is There One Target for All?

Maya Guglin, MD, PhD
Indiana University

Email: mguglin@iu.edu

Received November 14, 2022
Published December 1, 2022

Abstract

The current guidelines for managing cardiogenic shock lack specificity and clarification. The main criterion for cardiogenic shock is low cardiac output, and the most important goal is to achieve adequate output from a shock state. Because of the complex nature of cardiogenic shock, a “one-size-fits-all” outline may not be the best solution. Historically, hemodynamic goals in cardiogenic shock are copied from septic shock. Because septic shock and cardiogenic shock are different hemodynamic entities, the goals should be different.

Keywords: cardiogenic shock, hemodynamics, management

Background

In a statement from the American Heart Association on critical care unit monitoring, there is only one paragraph that outlines the hemodynamic goals to manage cardiogenic shock. It states:

The optimal [mean arterial pressure] MAP likely differs from patient to patient, and the risks of hypoperfusion with lower MAPs must be balanced (and individualized) with the potentially deleterious impact of vasoactive agents on myocardial oxygen demand, ischemia, and arrhythmia associated with higher MAP targets.¹

While certainly appropriate, the guidelines lack direct and specific goals for managing cardiogenic shock. Any recommendations come from studies of septic shock. In contrast, guidelines on septic shock are clear and specific.

The guidelines from the Surviving Sepsis Campaign² state similar goals:

- Central venous pressure (CVP) of 8-12 mmHg
- Mean arterial pressure (MAP) greater than 65 mmHg
- Mixed venous saturation (SvO₂) greater than 65%
- Urine output greater than 0.5 mL kg h⁻¹

Septic and cardiogenic shock studies in the context of guideline refinement will be reviewed.

Studies Related to Septic Shock

The Surviving Sepsis Campaign referenced a randomized trial comparing goal-directed therapy to standard therapy.³ The in-hospital mortality for goal-directed therapy was 30.5% versus 46.5% with standard therapy.

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A post hoc data analysis of a multicenter trial investigated the association of MAP and vasopressor load in septic shock patients. Similar mortality rates were seen when patients were grouped into quartiles based on MAP (from 70-100 mm Hg). When the quartiles were based on vasopressor load and dose, there was a stepwise increase in mortality with each increasing quartile.

In a retrospective study evaluating arterial blood pressure during sepsis and outcome, the best results were seen in patients with a MAP between 60 and 65 mmHg. The time spent below these values correlated with increased mortality risk, with an odds ratio of 2.96.

**Septic Shock versus Cardiogenic Shock**

Septic shock and cardiogenic shock are hemodynamically different. They share some common features, such as end-organ hypoperfusion, tissue hypoperfusion, and cardiac index but differ in cardiac output, wedge pressure, CVP, etc. Because they are entirely different entities, the hemodynamic goals for septic shock should not be applied to the cardiogenic shock setting. This is especially important since not all cardiogenic shock cases are created equal.

Cardiogenic shock can be caused by a pulmonary embolism and acute right ventricular failure with an underfilled ventricle that creates low cardiac output. Cardiogenic shock can result from acute myocardial infarction with left ventricular failure, high wedge pressure, and normal right atrial pressure. Depending on ideology, there are differences in how patients go into cardiogenic shock. Hypertension, hypoperfusion, decreased cardiac output, and possible congestion are all commonly seen after the immediate impact of arterial occlusion in acute myocardial infarction-related shock. The same can also be seen in cardiogenic shock caused by heart failure; however, the process is gradual rather than acute. To curate more specific priorities and hemodynamic goals for managing cardiogenic shock, the differences between cardiogenic shock and septic shock, and even the different etiologies of cardiogenic shock, need to be further explored through prospective studies.

There are different mortality profiles depending on the type of congestion. Right ventricular congestion, left ventricular congestion, and bi-ventricular congestion exist, and all are seen in patients with cardiogenic shock. Right ventricular and bi-ventricular congestion carry higher mortality risks than left ventricular congestion. In the setting of acute myocardial infarction, left ventricular congestion carries a higher risk of mortality than heart failure-related shock.

We need to design and conduct randomized trials in patients with cardiogenic shock to define appropriate hemodynamic goals for each type of shock of cardiogenic origin. For any type of shock, the specific goals should provide guidance to achieve normal cardiac output, adequate perfusion of end organs, and an euvolemic state.

**Conclusion**

The main criterion for cardiogenic shock is low cardiac output, and the most important goal is to achieve adequate cardiac output from a shock state. There may not be a “one size fits all” solution because of the variety of cardiogenic shock types; however, the current guidelines for goal-directed management need further clarification and specificity. For any type of cardiogenic shock, we need to achieve normal cardiac output, adequate perfusion of end organs, and an euvolemic state. Prospective studies comparing and investigating different sets of goals are needed.

**References**