Cardiogenic Shock in Perioperative and Intraoperative Settings: A Team Approach

Faisal Masud, MD; *Gaurav Gheewala,* NP; *Martin Giesecke,* MD; *EE Suarez,* MD; *Iqbal Ratnani,* MD

HOUSTON METHODIST DEBAKEY HEART & VASCULAR CENTER, HOUSTON METHODIST HOSPITAL, HOUSTON, TEXAS

ABSTRACT: Cardiogenic shock (CS) is a multifactorial disease process with high morbidity and mortality. When it occurs in a peri- or intraoperative setting, factors such as surgery, anesthesia, and post-surgical physiology can negatively affect patient outcomes. Since patient needs often escalate during CS—from medications to mechanical support to palliative care—this disease demands a multidisciplinary approach that encompasses all aspects of medical delivery. Preliminary studies have indicated that a multidisciplinary team approach to CS results in earlier diagnosis and treatment and improves patient outcomes. Here we discuss various management strategies for CS from an anesthesiology, surgery, and critical care perspective.

INTRODUCTION

Despite advances in therapeutics and diagnostic devices, cardiogenic shock (CS) continues to have high mortality and morbidity rates. Well defined by the American Heart Association (AHA), CS is a state in which ineffective cardiac output (CO) caused by a primary cardiac disorder results in clinical and biochemical manifestations of inadequate tissue perfusion.[1] However, when CS takes place in a peri- and intraoperative environment, factors such as surgery, anesthesia, and post-surgical physiological issues can have a negative impact on patient outcomes. Studies have indicated that 2% to 6% of patients who undergo cardiac surgery develop postcardiotomy shock.[2,3] This may be attributable to low CO (a result, in part, of myocardial hibernation, stunning, or inadequate cardioprotection), systemic vasodilation, or both.[1-5] This review offers perspectives from an anesthesiologist, cardiovascular surgeon, and intensivist who have worked as a team to manage patients with CS.

CRITICAL CARE MANAGEMENT: ROLE OF INTENSIVISTS

Managing CS in the intensive care unit is a complex rendering of an integrated care system in which the intensivist plays a central role in all preoperative care decisions (Figure 1). Here, we focus on some crucial aspects of critical care beyond standard management strategies discussed elsewhere in this issue.

Initial treatment for CS should include fluid resuscitation, vasopressors, inotropes, and other modalities to prevent or treat end-organ hypoperfusion.[6] The prolonged hemodynamic instability during CS requires placement of an arterial catheter to monitor arterial blood pressure and for blood sampling. Patients also receive a central venous catheter insertion for vasoactive agent infusion and to monitor filling pressure and mixed venous oxygenation.

Invasive hemodynamic monitoring with a pulmonary artery (PA) catheter is a reasonable strategy for management of CS and a valuable tool in patients who do not respond as expected, although its routine use cannot be recommended due to lack of strong evidence. The valuable hemodynamic data from a PA catheter–such as pulmonary artery pressures, pulmonary vascular resistance, the impact on the right ventricle, mixed venous oxygenation, and response to therapies–favors its use,[7] and the key information about end-organ perfusion markers such as urine output, capillary refill time, arterial blood gas, and lactic acid cannot be underestimated. In addition, transthoracic echocardiography is playing a vital role in the diagnosis and management of CS because it is noninvasive, quick, and provides valuable information.

Achieving Euvolemia

For any form of shock, euvolemia is mandatory to achieve hemodynamic stability and improve distal capillary perfusion. While CS is defined as a state of low CO, a lack of preload (with low intravascular volume) can be the cause of a low CO state, and fluid resuscitation may be helpful to attain intravascular euvolemia and hence increase CO. Careful fluid resuscitation is vital when there is decreased left ventricular (LV) ejection fraction and pulmonary edema; furthermore, excessive fluid may further increase LV end-diastolic pressure and worsen pulmonary edema.[8] Therefore, vigilant hemodynamic monitoring during this period is critical.

There is no protocol to guide fluid resuscitation. Since the associated risk may outweigh the benefit, fluid-challenge

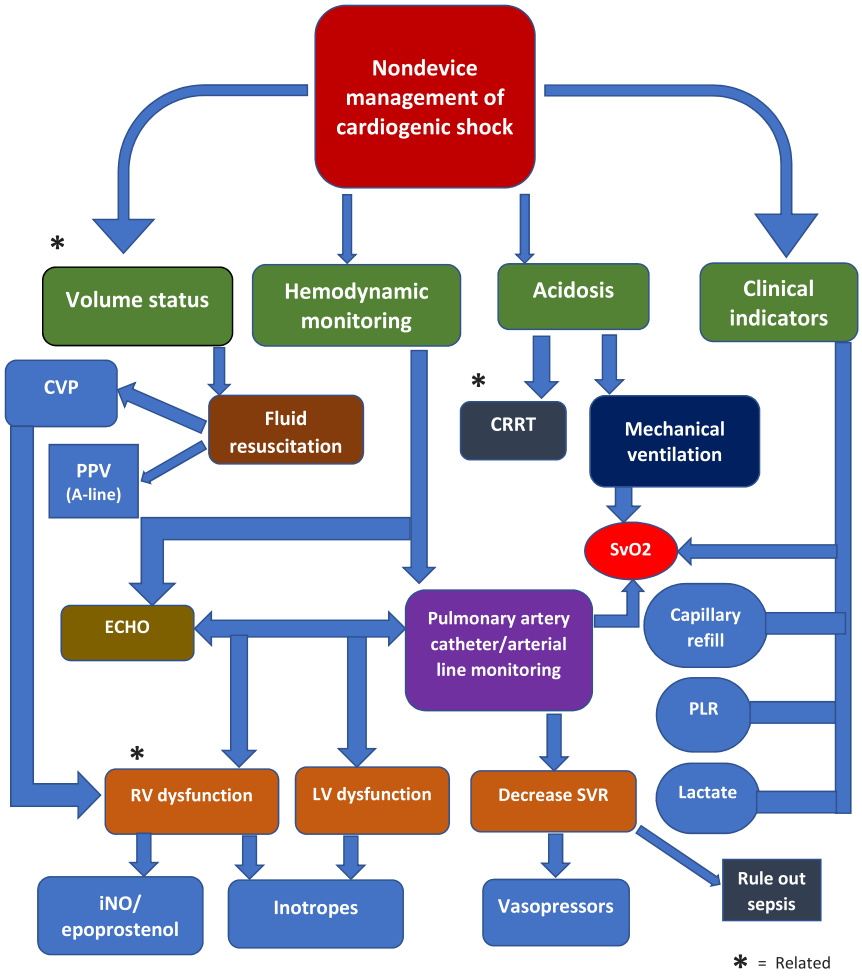

Figure 1.
The mind map: critical care management of cardiogenic shock. CVP: central venous pressure; PPV: pulse pressure variation; CRRT: continuous renal replacement therapy; SvO2: mixed venous oxygen saturation; PLR: passive leg raising; RV: right ventricular; LV: left ventricular; SVR: systemic vascular resistance; iNO: inhaled nitric oxide

therapy is a better option in CS patients.[9] For the fluid challenge, crystalloid solutions are the likely choice because they are readily available and cost effective. Ringers lactate is an isotonic crystalloid fluid that is preferable to normal saline, which may induce hyperchloremic acidosis. There is no advantage of using a colloid solution over a crystalloid solution other than in patients with severe hypoalbuminemia.[9] The fluid should be carefully administered at a rate between 250 mL and 500 mL over 20 to 30 minutes until it induces the desired response, including increased systolic blood pressure, improved urine output, decreased capillary refill time, and improved pulse pressure variation. In the event of worsening pulmonary edema, it should be stopped immediately.[10]

Mechanical Ventilation

There is a misconception that mechanical ventilation (MV) is required only for respiratory failure, but early treatment of hypoxemia associated with pulmonary edema in CS seems to be a successful strategy. Oxygen administration should be started early in patients with acute hypoxemia to prevent pulmonary hypertension. The prevalence of MV in CS is 78% to 88%.[1] It protects the airway, decreases the work of breathing, improves oxygenation and ventilation, and helps to correct acidosis. In addition, MV provides positive end-expiratory pressure, which improves gas exchange by recruiting alveoli and forcing fluid from the alveoli to the circulatory system. Positive end-expiratory pressure from MV reduces LV afterload by lowering transthoracic pulmonary pressures and preload, and it improves oxygen delivery to the myocardium.[1] Finally, it improves right ventricular (RV) function by reducing pulmonary vasoconstriction associated with hypoxemia. The major complication associated with invasive MV is circulatory collapse, especially around intubation, since positive pressure ventilation decreases venous return and can result in hypovolemia. Circulatory insufficiency can be avoided by infusing fluid before intubation to increase preload. It also may help to maintain a low tidal volume and low peak inspiratory pressure after intubation.[8]

Continuous Renal Replacement Therapy

Between 13% and 28% of patients with CS develop acute kidney injury, and nearly 20% require renal replacement therapy.[1] For these hemodynamically compromised patients, continuous renal replacement therapy (CRRT) is a much better option than intermittent hemodialysis, which can cause a drastic fluid imbalance.[11] The initial goal of CRRT is to correct metabolic acidosis and remove toxins from the body. An optimal atmosphere for the RV is necessary, especially postoperatively in individuals with a tenuous RV reserve. In certain situations, early utilization of CRRT might be a favorable strategy to achieve a normal metabolic milieu and an optimal preload (central venous pressure between 10-12 cm H_2O).

Inotropes and Vasopressors

The decision of whether to start an inotrope and/or vasopressor as first-line therapy in an intensive care unit should be guided by a quick assessment of the overall cardiovascular system and the primary driving hemodynamic factor. This may necessitate a quick bedside transthoracic echocardiogram, a noninvasive hemodynamic monitoring tool such as the Cheetah NICOM (Cheetah Medical) and ClearSight system (Edwards Lifesciences), or invasive hemodynamic assessment with a device such as the Edwards Vigileo (Edwards Lifesciences). Details of the various medications that can be used are mentioned elsewhere in this issue ("Management of Cardiogenic Shock in a Cardiac Intensive Care Unit" by Kim, Sunkara, and Varnado). A newer vasopressor agent, an angiotensin-II stimulant, has been approved by the US Food and Drug Administration for increasing blood pressure in adult patients with septic and distributive shock. It is used primarily on patients in the vasodilatory shock state who have not responded to conventional vasopressors. Because angiotensin II has a higher risk of thromboembolism, it should be used with caution.[12]

INTRAOPERATIVE MANAGEMENT: ROLE OF CARDIAC ANESTHESIOLOGISTS

There are multiple possible etiologies for CS in the perioperative arena. The most common is acute myocardial infarction with LV dysfunction,[1,13] which can occur in at-risk patients in the cardiovascular operating room, especially during noncardiac surgeries. Intraoperative CS poses unique challenges, particularly in noncardiac surgery, due to delays in recognition and timely revascularization in the setting of ischemia. On the other hand, with the patient already in the operating room, placement of mechanical support therapy may occur more quickly.

In the operating room, anesthesiologists are the central gatekeepers of optimal outcomes for high-risk individuals; they must be keenly aware of variation in the types of shock and etiologies of decreased CO, such as ventricular dysfunction, LV outflow tract obstruction, acute valvular pathology, arrhythmias, and obstructive shock. Obstructive shock can include a pulmonary thromboembolism (ie, amniotic fluid embolism in a parturient patient), air embolism, pericardial tamponade, tension pneumothorax or hemothorax, and abdominal compartment syndrome. Patients with a hypertrophic septal wall (eg, physiological asymmetric septal hypertrophy, hypertrophic obstructive cardiomyopathy) can develop a dynamic obstruction of their LV outflow tract due to hypovolemia, tachycardia, vasodilation, and/or high sympathetic tone.[10] In these situations, the solution may sometimes be to retract inotropes rather than increase them. Acute valvular pathology can include florid mitral insufficiency from a ruptured chordae or acute aortic insufficiency from an aortic dissection that includes the aortic annulus.

Recent reviews do an excellent job of describing the pathophysiology, diagnosis, and treatment of CS.[1,4,6,14-16] Much has been written with regard to CS in the setting of STEMI and some in the setting of acute on chronic heart failure, but there is very limited understanding of the pathobiology, systems, and protocols to manage intraoperative CS.[16] Although CS can occur unexpectedly and acutely in the operating room, the cardiac anesthesiologist typically plays a key role in close monitoring of these patients and could be prepared for these situations. A thorough preoperative assessment is key to develop an appropriate plan for each patient.

Because there is no consensus statement on preoperative evaluation of patients at risk for CS, it seems appropriate to evaluate all available data in the preoperative setting.[17] A pertinent physical exam is important to provide the anesthesiologist with probable sites for vascular access. While an electrocardiogram can provide a baseline, it is important to note the presence of conduction blocks to weigh the risk of floating a PA catheter when needed without fluoroscopy guidance. Radiological evidence (chest x-ray, computed tomography scan) of pericardial and pleural effusions and pulmonary status are key factors that could drive intra- and postoperative decisions regarding cardiorespiratory maintenance. Reviewing ongoing medical therapy (such as infusions) is equally important, as is assessing certain oral medications that might need to be discontinued. In the preoperative setting, an indwelling PA catheter may provide important information regarding RV function and continuous cardiac index measurements.[18]

There is no consensus on the best anesthetic agents or techniques to use in patients with or at high risk of CS. Thus, anesthetic management is based on expert opinion and knowledge of concomitant pathophysiology. There are several reasons why it is prudent to reduce doses of induction medications and to wait for a longer period to gauge the effect. First, the cardiovascular effects of an induction agent can be exaggerated in patients with CS because their volume of distribution is decreased.[15] Second, these patients will likely have low CO, so it will take longer for an injected dose of medication to reach the brain and cause a loss of consciousness. To some degree, all induction agents, including etomidate and ketamine, may cause a loss of centrally mediated sympathetic tone; thus, vasodilation and hypotension must be expected and appropriately treated to avoid end-organ hypoperfusion. Vasoconstrictors such as vasopressin

and norepinephrine are effective in this application. In fact, vasopressin is effective in increasing systemic vascular resistance without significantly altering pulmonary vascular resistance,[19] and norepinephrine use is associated with a lower incidence of arrhythmias.[1] Also, it is well accepted that preoperative inotropic and vasopressor support should be continued intraoperatively. However, recent studies are shedding light on the worsened outcomes associated with the use of beta mimetics and phosphodiesterase inhibitor inotropes in patients with chronic heart failure, and further studies will be required to determine if this also applies to patients with CS.[20-22]

Maintenance of anesthesia is typically achieved with low-dose inhalational agents and judicious administration of relatively short-acting opioids.[15] There is inadequate data to determine which mode of ventilation is best in patients with CS.[1] Maintaining adequate arterial oxygen pressure may require relatively high concentrations of inspired oxygen. Still, one must consider the risk of hyperoxia in patients in the intensive care unit in general and in some subsets of patients with CS.[23-26]

The complexities of intraoperative CS make it challenging for anesthesiologists to care for these patients. Thus, systems of care should be developed to include the anesthesiologist in the decision-making process for surgical candidacy and risk and to encourage a comprehensive and team-based approach to the treatment of CS. While the current consensus for CS teams focuses on the medical team caring for shock outside of the operating room, similar efforts must be made to focus on intraoperative CS.

ROLE OF CARDIAC SURGEONS: BRIDGING THE CONTINUUM OF PERIOPERATIVE CARE

Surgeons play a key role in maintaining continuity of care and have unique perspectives in complex decision making for surgical candidates. On the CS team, they are central to device selection and implementation of temporary mechanical circulatory support devices (tMCS) such as extracorporeal membrane oxygenation (ECMO), Impella (Abiomed), and the intra-aortic balloon pump (IABP). The details of tMCS are elucidated in "Acute Mechanical Circulatory Support for Cardiogenic Shock" by Telukuntla and Estep in this issue, and we address general comments specific to perioperative care here.

While there is increasing recognition of the negative impact of prolonged pressor and inotropic agents in patients in CS and a focus on early utilization of mechanical support, there is no consensus on device selection. Selecting the most appropriate type of device is complex and needs to integrate patient-specific criteria and a concerted long-term plan. For example, it has been recognized that patients classified as INTERMACS 1 who undergo operative durable support have significantly worse outcomes.[27] Support strategies in these settings have favored less invasive and less traumatic tMCS such as the axillary 5.0 Impella or an IABP that can be left in place for preoperative patient optimization.

Furthermore, although escalating tMCS from less robust and invasive to higher support (such as from IABP to ECMO) may minimize the risk of vascular and other complications, it contradicts the strategy of upfront maximal support with a gradual wean to break the cycle of CS. The use of tMCS at our institution has varied depending on the severity of CS. In addition, the Impella 5.0 requires a surgical cut down, typically at the axillary artery, making it a difficult strategy in an urgent situation where there is no time to mobilize a surgical team and transfer a patient to the operating room. The Impella 5.0 system can be used (1) for patients with smoldering shock or exacerbations of heart failure, (2) preemptively in patients with a low ejection fraction undergoing high-risk cardiac surgery, and (3) increasingly in patients needing combined ECMO/Impella for cardiac unloading. Although outcomes for CS had remained relatively unchanged for more than a decade after several years of progress,[28] consortiums are once again describing improved outcomes after following shock protocols that include early robust temporary support.[29]

Central Extracorporeal Membrane Oxygenation

Venoarterial ECMO produces the most robust hemodynamic support and oxygenation. It requires no assistance from the patient's own cardiac or pulmonary systems, and peripheral ECMO may quickly be placed at the bedside in severely unstable patients. As a central circuit, ECMO has the unique advantage of serving as stable support for post-cardiotomy shock and can be easily converted from the cannula insertions for cardiopulmonary bypass. The most common central cannulation strategies use either bicaval or right atrial venous cannulation and direct aortic arterial return. These cannulas are clamped and quickly connected to an ECMO circuit with rapid reinstitution of flow. Once on ECMO, the heparin used for cardiopulmonary bypass may be completely reversed with protamine for completion of hemostasis. The cannula may be further secured by the surgeon with the chest generally left open for re-exploration within 24 to 48 hours, at which point the chest is washed out and the patient may be assessed for weaning off support.

In situations where there is slim possibility of recovery and a need to bridge to a more durable device or cardiac replacement, unique configurations such as an LV apical cannulation or pulmonary arterial cannulation can potentially be transitioned to an extracorporeal pulsatile or continuous flow device. When

such a strategy is used, the circuit is typically planned for chest closure. This bridge to cardiac replacement is mostly necessary in the context of biventricular support or in situations where a tMCS device (eg, 5.0 Impella) is unavailable or use of such a device is contraindicated.

Data from the Extracorporeal Life Support Organization continue to show high mortality in patients requiring ECMO support, with venoarterial ECMO mortality in the 57% range[30]; however, there is no head-to-head comparison of ECMO versus medical management in acute severe CS, where much higher mortality outcomes might be anticipated. Despite the associated risks, ECMO use has been increasing around the nation and world.[30] There was a temporary reduction in Medicare reimbursement for ECMO in 2019, but the decision was reversed starting in 2020.[31] Most of the literature related to ECMO does not delineate between central and peripheral cannulation.

ECMO does have some drawbacks, including no inherent LV unloading, and its use can lead to higher intracardiac pressures and increased myocardial strain. Moreover, for those with severe heart failure, the increased afterload may result in severe pulmonary edema, causing damage to another organ system and further increasing the risk of mortality.[32] As such, the combination of ECMO with another support modality, such as ECMO/IABP and ECMO/Impella, has been gaining prominence as an unloading strategy. The need for such unloading seems to be controversial in patients with a central ECMO, but there is increasing recognition of such a possibility in situations of poor cardiac function. These unloading modalities can also serve as a potential de-escalation strategy for those patients recovering function and approaching candidacy for ECMO decannulation. A direct LV cannula can be placed for a central ECMO circuit if a tMCS device is not used.

TIME TO PAUSE

There will be times when patients may not adequately respond to medical and/or surgical interventions; this is when a palliative care specialist may come into play. Much has been studied about palliative care in patients with heart failure, and the AHA as well as international societies have recommended that palliative care be part of the multidisciplinary team taking care of these patients. This should apply to patients in CS as well. There may be scenarios in which a patient requires acute interventions in the operative and/or perioperative state, and time may not allow for family and team members to review all possible options—for example, MCS and ECMO, both of which may offer temporary improvement. For this reason, it is imperative that the multidisciplinary team discuss goals of care and exit strategies with the family at the earliest possible state, preferably before any surgical treatments take place. Palliative care specialists can be consulted and are a good resource to help with family dynamics and goals of care. Early engagement of palliative care provides more effective communication and increased family satisfaction if and when hospice and other comfort measures are needed.

Considering its high mortality and morbidity rates and overall impact on patients and families, CS in the perioperative setting requires a multidisciplinary team approach. Regular and frequent communication among the health care team members and with the patients' families about prognosis and goals facilitates a better outcome for all involved.

KEY POINTS

- Among patients who have cardiac surgery, 2% to 6% develop cardiogenic shock (CS).
- CS can occur rather unexpectedly and acutely in the operating room and needs a multidisciplinary approach for timely planning.
- There is no current consensus on strategies for managing preoperative CS, and efforts must focus on building consensus in this regard.
- Palliative care should be included in the multidisciplinary care team to manage patients in CS.

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Dr. Masud is on the advisory board of La Jolla Pharmaceuticals Company and Portolla Pharmaceuticals. Dr. Suarez is an advisor for Medtronic and Abbot and on the speaker's bureau for Abiomed.

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