

Cardiogenic Shock Systems of Care Best Practices. Part 1: Prehospital and Emergency Department Evaluation and Management

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Abstract

This paper explores best practices for the prehospital and emergency department evaluation and management of cardiogenic shock, a critical condition marked by high mortality rates, within a contemporary cardiogenic shock system of care. It highlights the significance of early identification and immediate intervention to prevent irreversible cardiac and non-cardiac organ damage. Emphasizing multidisciplinary team-based care and data-driven insights, the paper details optimal strategies for managing cardiogenic shock from initial contact, noting challenges in prehospital identification and treatment due to patient and disease heterogeneity and the variability in response capabilities of prehospital teams.

Keywords

Cardiogenic shock, emergency medicine, prehospital medicine, system of care

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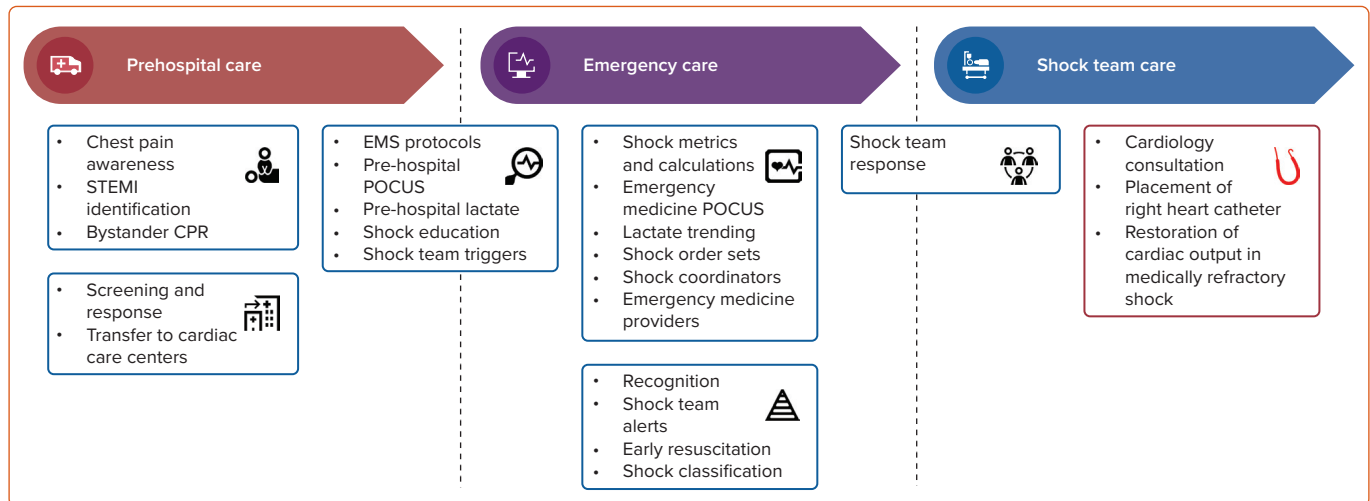
Cardiogenic shock (CS) represents a spectrum of hemodynamic derangements in which cardiac output is insufficient to provide adequate tissue perfusion, leading to a life-threatening condition that can often progress to multiorgan failure and death. Despite significant advances in percutaneous coronary interventions (PCI) and mechanical circulatory support (MCS), mortality rates for CS patients have remained largely unchanged over past decades.^{1–3} The time-sensitive nature of CS underscores the critical importance of early identification and prompt, effective interventions to restore tissue perfusion and prevent irreversible cardiac and extracardiac damage.

Effective management of CS demands a coordinated, multidisciplinary effort across clinical spaces and institutions. Over the past decade, global centers have made substantial progress by assembling interdisciplinary CS systems of care, challenging traditional practices, and applying data-driven insights to drive improvements in care. These evolving care models have highlighted the importance of early recognition and structured treatment strategies and serial clinical

reassessments. Given the volumes-to-outcome relationship in the management of CS, professional organizations have advocated for standardization and regionalization of care for CS patients.^{4–6}

The integration of disease-specific, multidisciplinary team-based care, starting from the initial point of first medical contact, has been a cornerstone of modern medicine, extending from trauma and cerebrovascular emergencies to advanced coronary artery disease, pulmonary embolism, and now CS.^{7,8} CS systems of care leverage the expertise of specialists from diverse fields to manage CS patients promptly and expertly across the entire spectrum of care (*Figure 1*). Prehospital and emergency department (ED) teams play a pivotal role in the management of CS, because timely diagnosis and initial stabilization significantly affect patient survival. This paper describes the contemporary practices for prehospital and ED management of CS, outlining optimal management strategies based on current evidence. Accompanying papers will describe all phases of the contemporary CS systems of care, from symptom onset through recovery.^{9,10}

Figure 1: Emergency Care Pathway for Cardiogenic Shock Survival



EMS = emergency medical services; POCUS = point-of-care ultrasound; STEMI = ST-elevation MI.

Prehospital Identification and Management of the Patient with Cardiogenic Shock

Despite years of research and technological advances, the mortality of CS patients remains exceptionally high. Identifying these patients at all entry points of the healthcare system is of the utmost importance, including in the prehospital and ED environments. Of all patients who present to the ED in shock, up to 15% have CS, with many of these patients presenting via ambulance, cared for first by emergency medical services (EMS) clinicians.^{9–11} EMS providers may also be involved in the transport of patients with CS from more rural facilities to tertiary or quaternary cardiac centers. As such, identifying areas of opportunity for the triage and treatment of CS patients in the prehospital setting is critical.

Although the heterogeneity and acuity of CS patients do not lend themselves well to randomized clinical trials, there are data suggesting that prehospital interventions for patients with a similar disease process, namely acute heart failure (AHF), improve mortality. Miró et al. performed a retrospective analysis of the EAHFE registry looking at prehospital interventions to treat AHF that included supplemental oxygen, non-invasive ventilation, intravenous diuretic administration, or nitroglycerin.¹² In that study, patients in AHF (with and without CS) managed with low-intensity prehospital treatment (i.e. receiving no intervention or only one intervention of the four therapies) had significantly higher odds of 7-day mortality than patients who received high-intensity treatment (two or more therapies initiated) from prehospital teams.¹² Another retrospective analysis of patients who received pharmacotherapy by EMS clinicians versus those who did not receive any therapy suggested that the OR of survival was higher (2.51; 95% CI [1.37–4.55]) when therapies were initiated in the prehospital environment.¹³ Extrapolation of existing data for the prehospital treatment of trauma or ST-segment elevation myocardial infarction (STEMI), in which first medical contact to intervention time is key, would also suggest that prompt identification, triage, and management of CS patients by EMS clinicians could improve outcomes.^{14,15}

Challenges of Identification and Management of Cardiogenic Shock in the Prehospital Setting

There are notable challenges to the diagnosis and management of CS patients during the prehospital phase of care.¹⁶ CS itself is a heterogeneous condition with varied presentations and symptomatology, with historic challenges in diagnosis only heightened by the resource-limited

prehospital environment. Diagnostic tools in the field have traditionally been limited, and many of the data points used by inpatient clinicians to determine the etiology of shock are not available to the prehospital provider. Classical physical examination findings of AHF or CS, such as a loud murmur, rales on pulmonary examination, increased jugular venous pressure, or physical examination markers of global hypoperfusion, perform notoriously poorly in the identification of heart failure under the best of circumstances, and are even more difficult to discern in the loud, and at times chaotic, prehospital environment.¹⁷ Studies specific to the prehospital environment have found that it is very difficult to differentiate between AHF (with or without CS) and other causes of dyspnea, especially in the setting of incomplete patient history and limited clinical diagnostics.^{16,17}

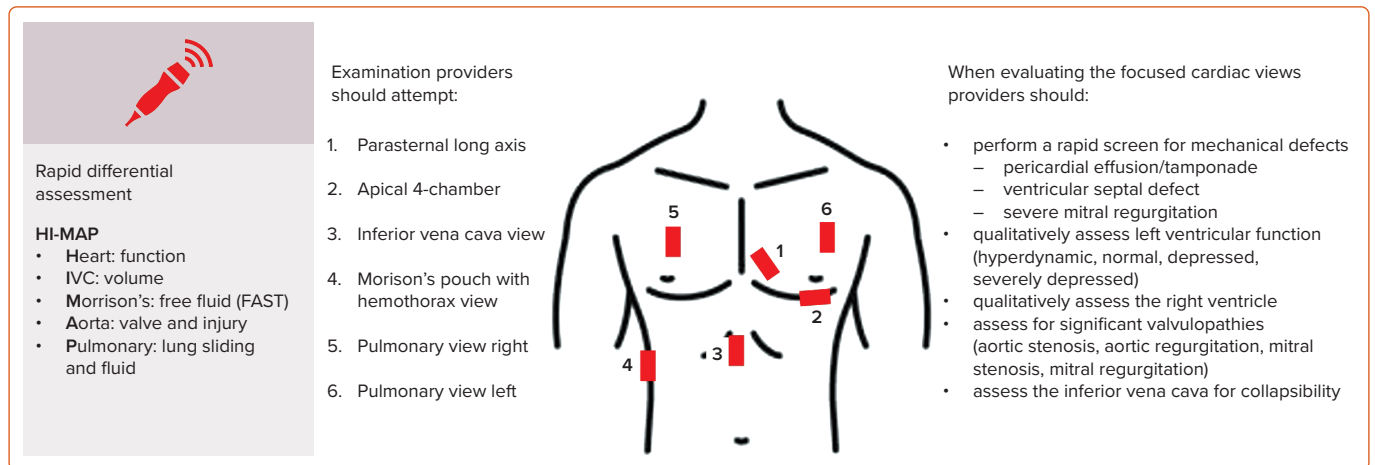
Another challenge is the significant variability in response, capability, and guidelines of prehospital teams treating patients who experience heart failure and are at risk of progression to CS. Many prehospital units may not be equipped to manage patients with CS, with one study finding that basic life support units may be dispatched to a disproportionately high number of patients who are ultimately diagnosed with AHF or CS.¹⁴ Even among advanced life support units, there is notable variability in clinical guidelines, as well as medications and equipment (e.g. non-invasive ventilation) stocked on responding units. Prehospital teams may also be hampered in their ability to initiate therapies for CS patients due to logistics concerns; for example, in very urban settings, transport times can be so short that initiation of therapies may be limited or deprioritized.

Finally, the education of prehospital clinicians may contribute to difficulty with early recognition. Traditionally, CS has been taught to EMS clinicians (and many others) as a binary condition where the diagnosis hinges on the presence of hypotension.¹⁸ Such a view of CS anchored in blood pressure rather than perfusion may fail to identify patients who are at very high risk of progression along their clinical course to fulminant shock or patients with evidence of end-organ hypoperfusion despite a “normal” blood pressure.

Improving Early Recognition of Cardiogenic Shock in the Prehospital Environment

The diagnosis of CS in the prehospital setting is challenging compared with diagnoses of trauma, stroke, or STEMI patients. However, the

Figure 2: Initial Point-of-care Ultrasound Assessment for Cardiogenic Shock: Prehospital and Emergency Department



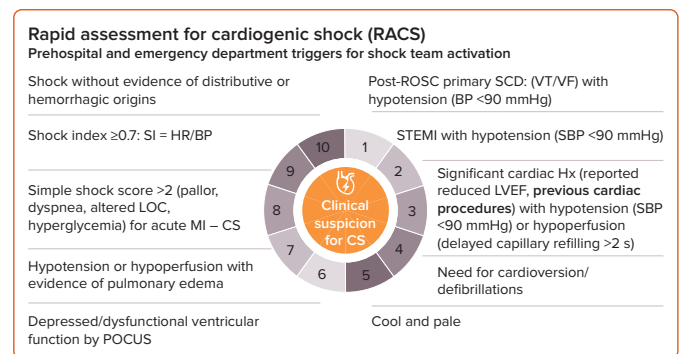
recently published Society for Cardiovascular Angiography and Interventions (SCAI) shock classification system provides a unique opportunity to improve the recognition of CS in the prehospital environment.¹⁹ Its tiered system of shock severity, supported by perturbations in the physical examination, biochemical markers, and hemodynamics, allows for more flexibility in the diagnosis of CS that could be adopted by EMS clinicians.

Despite the challenges with physical examination in the prehospital setting, there is good evidence to suggest that EMS clinicians can accurately diagnose CS by physical examination. In a relatively small single-center study of 40 patients in Poland who experienced acute coronary syndrome and shock, four examination findings were able to discriminate patients with CS exceptionally well: fainting or impaired consciousness, pale skin, dyspnea, and hyperglycemia (blood glucose >11.1 mmol/l or >200 mg/dl).²⁰ Described as the “4S Score” (4 Simple Shock Systems Scale), a value of two or more demonstrated excellent discrimination of CS in the setting of acute coronary syndrome and could be easily performed in the prehospital environment.²⁰

Previously relegated to the hospital setting, many prehospital emergency services and critical care transport medicine teams now have access to point-of-care laboratory testing for biochemical markers that may suggest CS. Specific tests that are incorporated into determining the SCAI shock stages, such as lactate, B-type natriuretic peptide, serum creatinine, and pH, are available to prehospital clinicians, although cost and logistics may be limiting.^{21,22}

Finally, although more invasive approaches to measuring hemodynamics, such as pulmonary artery catheter (PAC) insertion, are not feasible in the prehospital environment, continuous blood pressure monitoring by arterial catheter can safely be initiated by prehospital and critical care transport medicine crews in systems with advanced capabilities.²³ Portable ultrasound machines may be used to non-invasively evaluate hemodynamics such as cardiac output (or, even more simply, gross assessment of left and right ventricular function and inferior vena cava screening for volume assessment) in the prehospital setting (Figure 2), but would require advanced training and education for EMS personnel, and thus may be limited to specific teams and locales.²⁴ Figure 3 shows key triggers that prehospital providers can use to recognize CS in the prehospital environment.

Figure 3: Rapid Assessment for Cardiogenic Shock: Prehospital and Emergency Department Triggers for Shock Team Activation



BP = blood pressure; CS = cardiogenic shock; HR = heart rate; Hx = history; LOC = level of consciousness; LVEF = left ventricular ejection fraction; POCUS = point-of-care ultrasound; ROSC = return of spontaneous circulation; SBP = systolic blood pressure; SCD = sudden cardiac death; SI = shock index; STEMI = ST-elevation MI; VT = ventricular tachycardia.

Prehospital Management of Cardiogenic Shock

Prehospital management of CS patients should focus primarily on resuscitation, stabilization, and optimization of oxygenation, ventilation, and hemodynamics. Although capabilities of different prehospital systems will vary significantly, the overarching principles of prehospital care for the possible CS patient should consistently focus on these principles with an emphasis on simplicity when possible.

Initial support of oxygenation and ventilation should include non-invasive positive pressure ventilation (NIPPV). Not only does NIPPV decrease the work of breathing, improve oxygenation, and support ventilation, but it is also hemodynamically advantageous in the setting of CS. Increased positive pressure in the thorax may simultaneously decrease preload on the right heart and decrease relative afterload on the left heart. Optimizing mean arterial pressure with a goal of 65 mmHg is reasonable. Norepinephrine is the preferred initial vasopressor in this setting, and intravenous fluid boluses should generally be avoided or used with exceptional caution depending on the clinical history.^{23–27} To improve inotropy, starting an inotrope may be warranted.^{28,29} Many prehospital organizations do not carry dobutamine or milrinone; therefore, the addition of low-dose epinephrine (a mixed inotrope and vasopressor) may be a reasonable addition to support hemodynamics.^{30–32}

Diuretic therapy has been a staple of heart failure therapy in the prehospital environment. In the early stages of CS (i.e. SCAI stages A or B), there may be utility in the administration of intravenous diuretics to optimize volume status, decrease congestion, improve oxygenation, and optimize hemodynamics.^{12,13} However, if the patient's hemodynamics deteriorate and pressor support is required, withholding diuretic therapy in the prehospital setting may be appropriate. Ideally, more complex decision-making regarding vasoactive medication selection and dosing would occur in real time through direct communication with an immediately available supervising physician (online medical control), allowing active, case-specific guidance during transport rather than relying solely on prewritten standing orders.

Optimizing Appropriate Prehospital Triage of the Cardiogenic Shock Patient

As with any time-sensitive condition, optimal outcomes rely on getting the right patient to the right facility at the right time. Although the US has adopted standardized systems of care for conditions such as trauma and stroke, networked care for patients with CS is sorely lacking.³³ For the past decade, national societies such as the American Heart Association and leaders within the field have argued for a standardized national model for regionalization of care for CS patients, although this has not yet come to fruition.^{5,6,34}

One facet of a regionalized hub-and-spoke model is the prehospital triage of patients to appropriate facilities, as is seen with the trauma, stroke, and STEMI center designations. For patients with CS, this could mean initial triage to a center with the ability to perform PCI and implant temporary MCS devices (*Figure 4A*). Ideally, these facilities would additionally offer a dedicated cardiac critical care unit with a variety of cardiac services, including durable MCS and cardiac transplantation. Taking inspiration from the STEMI model of prehospital notification and cath lab activation, hospitals could be pre-notified by EMS crews for patients with suspected CS.³⁵ It is important to emphasize that such shock systems should complement and supplement, but not replace, currently successful triage and transport systems for STEMI.

Although regionalized models of care are not standardized across the US, some states have made progress towards regionalized models of care. The state of Washington has used a formal triage system for prehospital cardiac patients beyond the standard STEMI process, including those with cardiac arrest and those with a concern for cardiogenic pulmonary edema. In this system, EMS clinicians may bypass closer facilities in favor of a "Level 1 Cardiac Hospital" with more appropriate resources when able.³⁶ Other states have enacted similar systems, but significant regional variability remains in how patients with CS are triaged and transported between facilities, and how much increased distance and time delay is acceptable to prioritize one center over another. However, it is clear that any effort to regionalize and standardize the treatment of patients with suspected or confirmed CS will require close collaboration with EMS clinicians and cardiac and critical care specialists, because prehospital providers represent the first link in the chain of survival for many CS patients.

Key Points/Best Practices

- EMS should have regular training on the prehospital presentation and identification of CS states.
- EMS should have a pathway for communication and pre-alerting of CS patients based on prehospital triggers.
- EMS leaders should work with local shock centers to develop triage protocols for CS patients.

- Shock centers should provide routine feedback to EMS providers on shock patients.

Emergency Department Care for the Patient with Cardiogenic Shock

Prompt recognition and management is key to interrupting the natural evolution of CS, and preventing the development of irreversible multiorgan dysfunction is necessary, as with other life-threatening pathologies.^{37–40} International consensus guidelines from the American Heart Association and the European Society of Cardiology agree that there is clear benefit to the early diagnosis and management of CS.^{39,40} It is natural, then, for the ED to play a key role in the triage and care of the patient with CS (*Figure 4*).

Available data suggest that prompt identification and guideline-based treatment of CS demonstrate improved outcomes, leading to a shorter hospital length of stay and decreased mortality.^{12,41,42} In addition, early initiation of pharmacological management, such as with diuretics, shows a reduction in mortality. Finally, in-hospital initiatives to reduce door-to-support time for patients in CS using invasive hemodynamic management, including the initiation of MCS, have shown promising reductions in admission mortality.⁴³

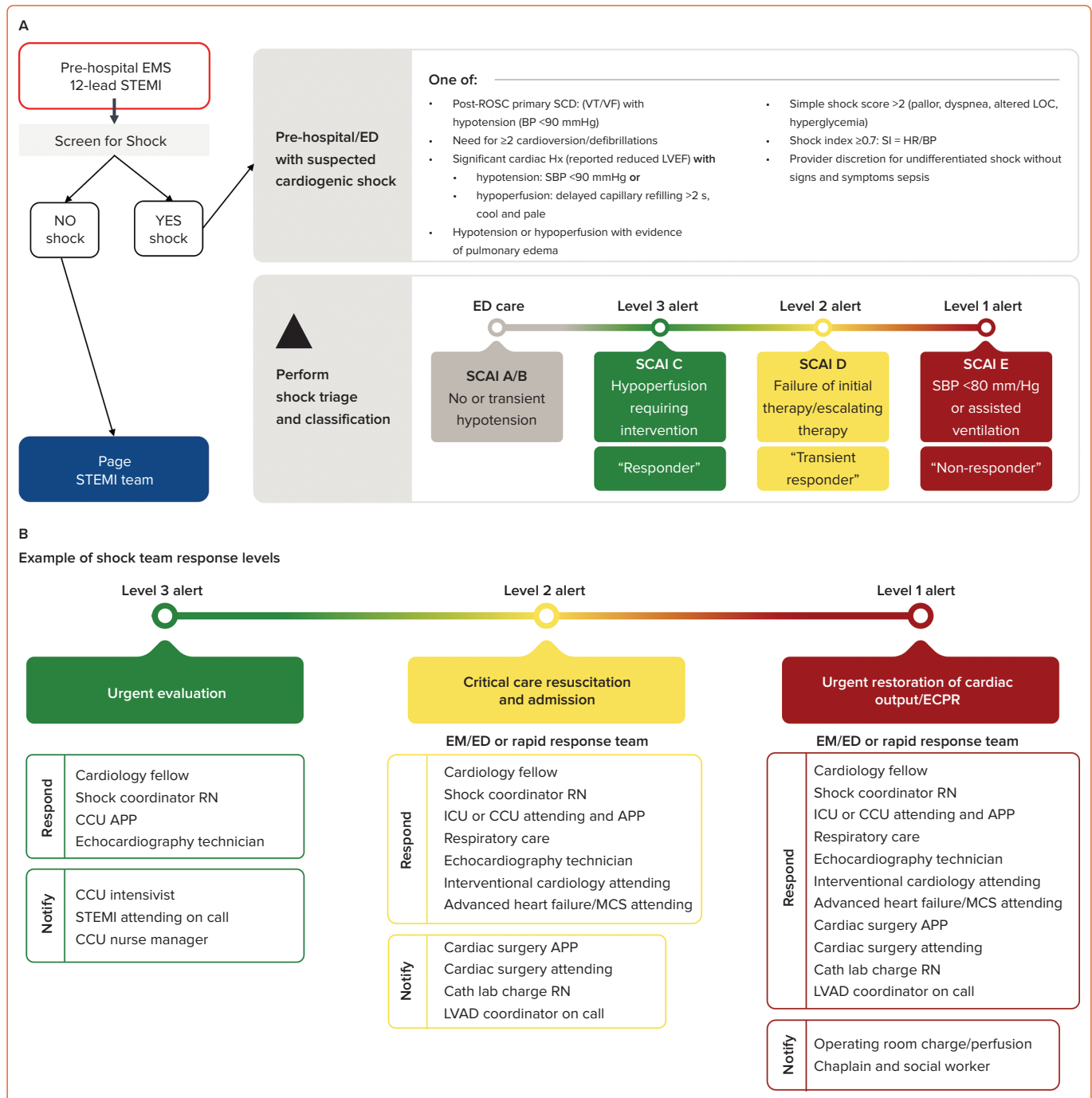
There are notable challenges to the diagnosis and management of CS in the ED. Emergency physicians are expected to accurately diagnose and treat a wide spectrum of illnesses across multiple organ systems and medical specialties. Patients with CS arrive to the ED with varied phenotypic presentations that overlap with other shock states, such as sepsis.⁴⁴ Furthermore, in resource-limited settings or critical access EDs without access to the full spectrum of diagnostic resources and specialist physicians, differentiating between CS and other conditions can be very challenging.^{19,20,45} In addition, previous out-of-date definitions and education about CS may contribute to a delay in diagnosis. Traditionally, CS has been taught as a binary condition in which the diagnosis hinges on the presence of hypotension, defined somewhat arbitrarily as a systolic blood pressure <90 mmHg or mean arterial pressure <65 mmHg.^{46,47} This approach fails to identify patients who are at very high risk of progression along their clinical course to frank shock but present with what is otherwise a "normal" blood pressure, despite clear evidence of end-organ hypoperfusion.^{48–50} Expedient but simple physical examination and laboratory testing, such as bedside volume assessment and point-of-care lactic acid measurements, can help recognize these patients.

The SCAI shock stages are a more optimal way to understand the CS patient along their clinical trajectory.¹⁹ This description of CS on a spectrum of severity may facilitate a paradigm shift in conceptualizing the disease, highlighting that the majority of CS patients actually present in stage A or stage B without overt hypotension.⁵¹ Notably, these patients in the earliest stages of CS are often under-recognized and have a surprisingly high mortality.^{50,52} Additional evidence suggests that stage B CS patients are at very high risk of clinical deterioration, with 41–90% of these patients progressing to stage C or more severe stages of shock.^{53,54}

Improving Emergency Department Identification of Cardiogenic Shock

Perhaps one of the most helpful aspects of the updates to the SCAI shock classification system is that it addresses three domains that encapsulate each of the stages of shock: physical examination, biochemical markers, and hemodynamics. Many of these parameters may facilitate early identification of CS in the ED when advanced diagnostics are not immediately available or even in more resource-limited settings.⁵⁵

Figure 4: Cardiogenic Shock Triage, Alert Activation, and Response Framework



A: Cardiogenic shock triage and notifications: emergency medicine shock triage. B: Cardiogenic shock alert and responses. APP = advanced practice provider; BP = blood pressure; CCU = cardiac care unit; CS = cardiogenic shock; ECPR = extracorporeal cardiopulmonary resuscitation; ED = emergency department; EMS = emergency medical services; HR = heart rate; Hx = history; ICU = intensive care unit; LOC = level of consciousness; LVAD = left ventricular assist device; LVEF = left ventricular ejection fraction; MCS = mechanical circulatory support; POCUS = point-of-care ultrasound; RN = registered nurse; ROSC = return of spontaneous circulation; SBP = systolic blood pressure; SCAI = Society for Cardiovascular Angiography and Interventions; SCD = sudden cardiac death; SI = shock index; STEMI = ST-elevation MI; VT = ventricular tachycardia.

Overall, individual physical examination findings are notoriously poor at predicting low cardiac output states and CS in and of themselves. Various reviews of the literature have shown no single examination performs well in identifying CS.⁵⁶ Despite this, it is certainly reasonable to conduct a thorough physical examination of any patient with suspected CS. Some classic physical examination findings, such as jugular venous distension, pulmonary rales or crackles, delayed capillary refill, peripheral edema, and cold extremities, may provide some clues as to the presence of early CS or perhaps help differentiate it from other forms of shock.^{57,58} The 4S Score, described above, may aid

ED providers in identifying CS patients in the setting of acute coronary syndrome.⁵⁹

Laboratory tests commonly available in the ED can help identify the presence of CS. These tests correlate with the different SCAI shock stages and include lactate, B-type natriuretic peptide, serum creatinine, a chemistry panel (with specific attention to serum glucose, sodium, and creatinine), complete blood count, liver function tests, and arterial/venous blood gas analysis.^{19,60} No single laboratory test is particularly sensitive or specific for the diagnosis of CS, but in the appropriate context the tests

can provide critical information about CS severity and other organ system dysfunction. Not all EDs offer point-of-care testing, and results may also be delayed and less useful for immediate decision-making.

Although more invasive approaches to measuring hemodynamics, such as a PAC, may not be feasible in the ED, less invasive means may raise suspicion of CS. In addition to non-invasive blood pressure measurements, initiation of minimally invasive continuous blood pressure monitoring by way of an arterial catheter can safely be initiated by emergency medicine physicians.

Point-of-care ultrasound (POCUS) is ubiquitous in the contemporary ED setting and has found significant utility in differentiating critically ill patients (*Figure 2*). It can be used to non-invasively evaluate hemodynamics, cardiac function, and pulmonary congestion.^{61,62} Simple, qualitative estimation of cardiac function may be helpful in identifying CS and has demonstrated reasonable accuracy by emergency medicine providers.⁶³ A more quantitative approach may be more accurate and is also feasible with POCUS.⁶⁴ Measurement of left ventricular outflow tract velocity time interval using pulse wave Doppler can be used to calculate stroke volume when the left ventricular outflow tract diameter is also measured. When multiplied by the heart rate, this value can be used to estimate cardiac output.^{65,66} Furthermore, various parameters of right heart function can be evaluated by POCUS, including right ventricular chamber size, tricuspid annular plane systolic excursion, and peak systolic tricuspid velocity (s).^{67,68} Although all these echocardiographic measurements have limitations, there is value in incorporating them as objective measures of cardiac function. Overall, POCUS may be an effective, non-invasive bedside modality to quickly identify CS patients in the ED.

Central venous oxygen saturation ($S_{cv}O_2$) may also be helpful in identifying CS. Although a true mixed venous oxygenation saturation (obtained from pulmonary arterial blood using a PAC) is typically used in the intensive care unit setting and provides more accurate estimates of cardiac output, $S_{cv}O_2$ measured from a central line provides a reasonable surrogate.⁶⁹ A normal $S_{cv}O_2$ value in ED and intensive care unit populations is generally considered $>70\%$. Values $<60\%$ are associated with CS states and decreased tissue perfusion.²⁸ One ED investigation demonstrated a mean $S_{cv}O_2$ in “mildly decompensated” heart failure of 51% and in “severely decompensated” heart failure of 32% .²⁹ Therefore, although decreased $S_{cv}O_2$ is not necessarily specific to CS, it may support the diagnosis of CS in the appropriate clinical context.

Finally, invasive hemodynamic monitoring with PACs may be of use for the early diagnosis of CS, guidance of initial interventions, and prompt recognition of the need to escalate support.^{31,70–72} Both the European Society of Cardiology and SCAI recommend PAC placement in their guidelines for the management of CS.^{40,73} In the past, PAC placement was often considered a procedure relegated to the cardiac cath lab or the cardiac intensive care unit. However, with the development of ED critical care areas and specialized ED resuscitation units, placing a PAC in the ED may be reasonable in some centers when prompt transfer to the cardiac intensive care unit or cath lab is not feasible. *Figure 4* shows how ED providers can use many different tools to recognize CS within the ED and activate the shock team based on the severity of shock present.

Enhancing Management of Cardiogenic Shock in the Emergency Department

Once CS is identified in the ED, rapid resuscitation, stabilization, and

coordination of care is imperative. The initial steps of ED management are crucial in halting the progression of CS and the development of multiorgan failure.

First, oxygenation and ventilation must be supported. The vast majority of patients in CS exhibit evidence of acute hypoxic respiratory insufficiency or failure requiring respiratory support.^{74,75} There is strong evidence that NIPPV decreases mortality and morbidity in the setting of heart failure with associated respiratory insufficiency.⁷⁶ In addition to supporting oxygenation, improving ventilation, and decreasing work of breathing, NIPPV demonstrates hemodynamic benefits as well. Increasing intrathoracic pressure causes decreased preload and afterload for the right and left ventricle, respectively. In addition, it may reduce right ventricular afterload by counteracting hypoxic pulmonary vasoconstriction.^{77,78} Should NIPPV fail, especially in the setting of worsening mental status and decreased respiratory drive, escalation to endotracheal intubation and invasive mechanical ventilation is indicated.

Also imperative are the maintenance of organ perfusion and the treatment of hypotension. Although the optimal blood pressure in a broad cohort of CS patients has not been clearly identified, an initial mean arterial pressure goal of 65 mmHg is reasonable.⁷⁹ Maintaining mean arterial pressure is paramount to ensuring that vital organs are adequately perfused. Fluid resuscitation is rarely effective and may be harmful, especially in the setting of vascular congestion secondary to poor cardiac output. Available data and guidelines suggest norepinephrine is an appropriate first-line agent.^{25–27}

Although inotropes have not been shown to improve outcomes in the setting of CS, they may have utility in temporarily increasing cardiac output, especially in the setting of hypoperfusion and vascular congestion.^{30,80,81} Available evidence does not clearly support the superiority of a particular inotrope.³² Therefore, either low-dose epinephrine or dobutamine would be a reasonable choice to provide inotropy. Dobutamine (with or without norepinephrine) may confer the advantage of fewer adverse effects of the two, as some evidence demonstrates fewer arrhythmias, less myocardial oxygen consumption, and lower lactate production compared with epinephrine.⁸² Although milrinone overall seems to be generally equivalent in outcomes to dobutamine, its pharmacokinetics may make it suboptimal for initiation in the ED in the setting of CS because its time of onset is longer and its serum concentrations can build up in the setting of kidney injury, which is common in CS patients.⁸³ Negative inotropes, such as diltiazem, should be avoided in the setting of CS. Tachycardia, to include AF with rapid ventricular response and atrial flutter, is often compensatory, and lowering the heart rate with negative inotropic agents without appropriate support of cardiac output may precipitate fulminant shock.

In the setting of heart failure-related CS with clinically significant organ congestion despite an adequate blood pressure (SCAI stage A and B), diuretics may be considered in conjunction with vasopressor or inotropic support, because elevated left ventricular filling pressures are associated with poor outcomes in this patient population.⁸⁴ Loop diuretics, in conjunction with either thiazides or acetazolamide, may be used to provide vascular decongestion, and can be initiated in the ED.^{42,85}

Another important part of caring for the CS patient in the ED is the identification and management of the underlying etiology of the shock state. Perhaps the most valuable interventions in CS are those targeted at

acute, reversible disease processes. Specifically, this means identifying acute coronary syndrome, acute valvulopathy, or acute dysrhythmias.^{86–88} These underlying pathologies may be amenable to percutaneous, surgical, or pharmacological interventions that are often time-sensitive.

To this end, ED clinicians should consult the appropriate cardiac specialty teams as soon as possible. A cornerstone of management for patients with acute MI-associated CS is early revascularization therapy.⁸⁹ In addition to revascularization, interventionalists or other specially-trained physicians can initiate temporary MCS, including percutaneous axial flow devices, extracorporeal membrane oxygenation, and other critical supportive interventions recommended by the American Heart Association and European Society of Cardiology.^{41,42,90} Timely activation of these consultant services and CS teams is important because there is benefit to earlier intervention and shorter door-to-support times.^{18,91–93} In a traditional model, it is often only STEMI patients who go directly from the ED to the cath lab for diagnosis and intervention, and all other patients would be triaged first to an intensive care unit. However, in a modern shock team model, many CS patients go directly from the ED to the cath lab for definitive diagnosis of their condition and consideration for treatment strategies, including the early application of MCS. Much of this can be achieved with the activation of a shock team while the patient is still in the ED.

Key Points/Best Practices

- Emergency medicine providers should receive regular training on clinical practice for patients with CS.
- Guidelines should exist for the emergency medicine provider to initiate a CS response pathway that activates additional resources in an expedited manner.
- Emergency medicine providers should receive regular, organized feedback on outcomes and interventions of CS patients.
- Emergency medicine providers should be part of a multidisciplinary shock committee.

Conclusion

The establishment of CS systems of care as a contemporary care model for managing critically ill CS patients marks a significant advance in the treatment of this challenging and time-sensitive disease process. The prehospital and ED phases play a pivotal role in the management of CS, because timely diagnosis and initial stabilization significantly affect patient survival and serve as the entry point into CS systems of care.

This paper focuses on the critical aspects of identifying and managing CS in the prehospital and ED settings within the context of a contemporary model of CS systems of care. Additional phases of the contemporary CS systems of care are described in the accompanying papers.^{91,92}

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