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Marwan F. Jumean
The University of Texas Health Science Center-Houston, marwan.f.jumean@uth.tmc.edu

Sriram Nathan
The University of Texas Health Science Center at Houston, sriram.nathan@uth.tmc.edu

Igor D. Gregoric
The University of Texas Health Science Center at Houston, igor.d.gregoric@uth.tmc.edu

Biswajit Kar
The University of Texas Health Science Center at Houston, biswajit.kar@uth.tmc.edu

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Houston SHOCK: A Practical Scoring System Incorporating Cardiogenic Shock Dynamic Changes

Marwan Jumean, MD; Sriram Nathan, MD; Igor D. Gregoric, MD; Biswajit Kar, MD
Department of Advanced Cardiopulmonary Therapies and Transplantation, The University of Texas Health Science Center at Houston, Houston TX, USA

Email: Biswajit.Kar@uth.tmc.edu

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Abstract

Cardiogenic shock has an unacceptably high mortality rate and additional tools are needed to improve outcomes. The Society of Cardiovascular Angiography and Interventions (SCAI) shock severity classification has provided a unified definition of shock severity that has proven to be reproducible and predictive of survival. However, cardiogenic shock assessment goes beyond standardizing its severity, and a uniform and practical approach to comprehensive assessment that may guide therapy in a dynamic state is currently lacking.

Since cardiogenic shock is a rapidly evolving pathophysiological catastrophe, we propose a new assessment tool – the Houston SHOCK Score – which incorporates dynamic changes. The acronym SHOCK can be used to emphasize five key aspects of patients in cardiogenic shock: **S**everity, **H**emodynamics, **O**nset, **C**auses, and **K**inetics. We believe this tool provides physicians with vital information that will facilitate appropriate care by incorporating dynamic changes in the patient's profile.

Keywords: cardiogenic shock, mechanical circulatory support device, SCAI shock

Background

Cardiogenic shock (CS) is a devastating clinical condition with an overall mortality rate ranging from 25-50%.[1-4] Mortality remains high despite advances in medical management,[2] the adoption of early revascularization and emergent reperfusion strategies after acute myocardial infarction (AMI),[1] and the advent and widespread utilization of percutaneous mechanical circulatory support (pMCS) device therapy.[5] In fact, in an analysis of 56,497 patients in the CATH-PCI registry, the mortality rates from AMI-CS rose from 27.6% in 2005-2006 to 30.6% in 2011-2013 ($P < .0001$).[5] In addition, the burden on healthcare systems is significant. CS has an unacceptably high 30-day readmission rate of 18.6% amongst survivors of AMI-CS.[6]

The Society for Cardiovascular Angiography and Interventions (SCAI) expert consensus statement on the classification of CS offers a standardized taxonomy for providers.[7] It supports the early identification and triage of patients presenting with CS in a simple, readily applicable, and intuitive manner. The classification is a step closer to the standardization of a CS definition. Based on this new ***severity*** classification, CS is classified into five categories, from at-risk

Table 1. Profiles of shock based on invasive hemodynamic assessment.

Cardiogenic	**Classic** (cold and wet): reduced CI, high SVRI, elevated PCWP **Mixed** (warm and wet): reduced CI, reduced SVRI, elevated PCWP **Euvolemic** (cold and dry): reduced CI, elevated SVRI, normal PCWP
Hypovolemic	Characterized by reduced intravascular volume; typically with reduced CI, elevated SVRI, and reduced PCWP
Distributive	Characterized by severe peripheral vasodilatation; typically with increased CI, reduced SVRI, and reduced PCWP
Obstructive	**Tamponade**: reduced CI, elevated SVRI, and elevated PCWP **Pulmonary**: reduced CI, elevated SVRI, and reduced PCWP
Right Ventricular	Reduced CI, elevated SVRI, reduced PCWP, elevated RAP, elevated RA:PCWP ratio, reduced pulmonary artery pulsatility, reduced PAPi, reduced RVSWI

Abbreviations: CI, cardiac index; PAPi, pulmonary artery pulse pressure index; PCWP, pulmonary capillary wedge pressure; RAP, right atrial pressure; RVSWI, right ventricular stroke work index; SVRI, systemic vascular resistance index

(stage A) to extreme CS (Stage E); however, CS assessment goes beyond standardizing its severity. A comprehensive, uniform approach is currently lacking, especially one that considers changes in patients' clinical or hemodynamic status, response to therapy, and trajectory. Comprehensive classification of CS is difficult due to a myriad of reasons that we will review below.

Classification Complications

First, patients with CS present with a wide spectrum of presentations. The inability to accurately recognize the different ***hemodynamic phenotypes*** of CS (Table 1) in a timely fashion is a major contributing factor to poor outcomes. The current definitions of CS used in clinical trials[7-9] require a systolic blood pressure (SBP) of less than 90 mmHg despite pressor support with evidence of end-organ hypoperfusion and/or hemodynamic parameters of reduced cardiac index (CI). However, septic and hypovolemic shock also manifest with hypotension and end-organ hypoperfusion (Table 2), and assessment of hemodynamics is oftentimes not readily available. Furthermore, mixed forms of shock frequently exist simultaneously.[9] In addition to including absolute blood pressure parameters in the definition of CS, the degree of hypotension relative to the patient's preexisting blood pressure should be considered in any attempt to redefine the spectrum of CS. In fact, 5.2% of patients in the SHOCK trial registry were in CS but had an SBP > 90 mmHg, and 7.1% had no evidence of organ hypoperfusion with an SBP < 90 mmHg; yet, both groups of patients have increased mortality.[8]

Second, the ***onset of CS*** and its impact on outcomes has not been fully studied. Traditionally, *acute* onset refers to CS cases that develop within less than 24 hours of the onset of symptoms and are typically seen in acute ischemic events or electrical storms. A *subacute* onset, defined as symptom onset of fewer than 7 days, is seen most often in mechanical complications of myocardial infarction and acute myocarditis. Finally, *chronic* onset is defined as symptom onset of greater than 7 days but is typically seen in patients with known stage D heart failure.

Third, CS is the final culmination of ***multiple disease states***. The heterogeneity of CS is akin to heart failure with preserved ejection fraction (HFpEF) heterogeneity in clinical trials. An impact on long-term mortality has not been seen in most HFpEF clinical trials due to the different underlying pathophysiological mechanisms of diseases that lead to HFpEF. Outcomes of post-cardiotomy shock vary dramatically from those of acute myocarditis. A patient suffering AMI-related CS behaves differently than a stage D heart failure patient presenting with CS. The hemodynamic profiles of these two subsets of patient populations are different, with the latter having higher filling pressures, pulmonary artery pressures, and a different metabolic

Table 2. Parameters of end-organ hypoperfusion.

Clinical Parameters	Impaired mental status, UOP < 40cc/hour, progressive pulmonary edema
Hemodynamic Parameters	High doses of pressors to maintain adequate BP is defined as: Norepinephrine dose of > 0.2mcg/kg/min Epinephrine dose of > 0.2mcg/kg/min Dopamine > 5mcg/kg/m Two vasoactive agents
Biochemical Parameters	Creatinine > 0.4mg/dL X baseline, elevated AST/ALT > 4X upper limit, lactate > 4.0 units

Abbreviations: AST/ALT, aspartate aminotransferase/alanine aminotransferase; BP, blood pressure; UOP, urinary output

profile.[10] As such, one would expect the hemodynamic goals of management to differ based on the inciting event.

Lastly, there are aspects of CS that are not addressed in the current literature, especially responses to initial therapy. Many questions arise when considering the approach to CS management. For example, it is not clear if full pMCS support followed by de-escalation in CS would be superior to a strategy of tailored escalation of pMCS support, or whether an approach of maximum pMCS support be utilized routinely in stages C to E of CS. It is unclear if dynamic changes in the patient's hemodynamic status and response to therapy play a role and whether the "***kinetics"*** of either approach has an impact on mortality.

Mortality in CS is not solely related to the initial myocardial insult and the acute drop in CI. In fact, in a meta-analysis of randomized controlled trials of pMCS, Thiele et. al demonstrated the improvement in hemodynamic profiles of patients who were supported with pMCS when compared to those supported with an intra-aortic balloon pump (IABP).[11] Reductions in pulmonary capillary wedge pressure and improvements in CI and mean arterial pressure were observed; however, an improvement in 30-day mortality was not found.[11] Thus, it remains to be seen if this lack of effect on mortality was due to the limited power of the study or the true absence of an effect. Indeed, the complex pathophysiological way in which the body responds to the initial insult that leads to progressive cardiac dysfunction varies from one patient to another. The variations in the activation of systemic inflammatory response syndrome (SIRS), the extent of metabolic derangements, the impact of pMCS on SIRS activation, and the complex interplay between the different pMCS types and the CS patient (eg, the balance between the salubrious hemodynamic effects and the complications associated with these large bore devices) need to be further studied as they play a major role in the outcomes of these patients. As such, the differentiation of CS patients based on the use of a pMCS device is inadequate. pMCS support should be tailored to different hemodynamics of shock presentation and active changes in the patient's profile. A CS patient who **stabilizes** on an IABP may need to be classified differently than a patient who requires veno-arterial extracorporeal membrane oxygenation for hemodynamic support due to **refractory** CS. Treatment during or following stabilization should be tailored to the inciting cause of CS and the likelihood of reversibility of its cause.

Recommendation

Given that CS outcomes are highly dependent on the ***severity*** at the time of presentation *AND* the above-mentioned elements including the dynamic changes in the patient's profile, which we refer to as "Kinetics," we propose incorporating the following five elements to provide a more comprehensive assessment of the CS patient. The five variables, encapsulated by the acronym SHOCK are: (1) the **S**everity of CS; (2) the **H**emodynamic profile of the patient, (3) the **O**nset of CS symptoms, (4) the **C**ause of CS, and (5) the **K**inetics of CS (Figure 1). This approach offers healthcare providers and researchers a uniform language, a standard platform, and clinically relevant parameters that include dynamic changes and response to therapy that allow for a more accurate portrayal of the clinical picture of a CS patient. This proposed assessment will support the accurate identification of CS patients with similar pathophysiology and severity while addressing most of the aforementioned shortcomings of the current CS definitions. We believe that this comprehensive assessment will fulfill a vital need to be able to compare the effects of different therapies and aid the design of meaningful trials in specific subsets of patients with CS taking into consideration the hemodynamic profiles of patients, the kinetics of patients, and the likelihood of reversibility of causes.

The following is a description of the five parameters of the SHOCK Scoring System.

Table 3. Houston Shock Score.

Variable		Points
Severity	SCAI A-C SCAI D, E	0 1
Hemodynamics	Classic Mixed & Euvolemic	0 1
Onset	Acute Chronic	0 1
Cause	Acute Treatable Other Causes	0 1
Kinetics	Stabilized Refractory/Worsening	0 2

Severity

As stated earlier, the SCAI expert consensus statement on the classification of CS simplifies the severity into one of five stages, mirroring the Interagency Registry for Mechanically Assisted Circulatory Support profiling of advanced heart failure patients.[7] In a recent single-center retrospective study of 10,004 patients admitted to an intensive care unit, the unadjusted hospital mortality rose steadily as the severity of CS increased.[12] Mortality in stage A was 3.0%, stage B 7.1%, stage C 12.4%, stage D 40.4%, and stage E 67% ($P < .001$); each higher SCAI shock stage was associated with increased hospital mortality with an adjusted odds ratio of 1.53 to 6.8 (all $P < .001$).[12] Thus, we suggest the first assessment should include ascertaining the severity of CS via the SCAI system. The Houston SHOCK Score assigns a value of 0 to patients classified as stages A-C, and those individuals classified as stages D or E are assigned a value of 1 (Table 3).

Hemodynamics

While the classic form of CS is frequently seen, the hemodynamic profile is varied and can be classified in one of three main presentations:[9]

1. The classic cold and wet, the most frequently encountered form, is often seen in AMI-CS
2. Mixed CS (warm and wet), and
3. Euvolemic CS (cold and dry) — often seen in chronic heart failure patients who present in a decompensated state

Since CS management is dependent on the predominant phenotype, the second step in our assessment model is to account for such variability in presentation. The SHOCK Score encourages users to provide a more detailed classification of shock after invasive and non-invasive hemodynamic assessments are attained. Patients would be delineated by one of the following types of shock: cardiogenic, hypovolemic, distributive, obstructive, or right ventricular (Table 1). The Houston SHOCK Score assigns a value of 0 to patients classified as classic; those individuals classified as mixed or euvolemic are assigned a value of 1.

Onset

As mentioned earlier, an AMI-CS patient behaves differently than a stage D heart failure patient presenting with CS. Such differentiation is critical when looking at therapeutic modalities and mortality outcomes. CS onset after AMI occurred within 24 hours in 74% of patients in the SHOCK trial registry.[13] However, patients in stage D heart failure often have acute episodes of decompensation. Further, the hemodynamic profiles of these two subsets of patients are different with the latter having higher filling pressures, pulmonary artery pressures, and a different metabolic profile.[10] The Houston SHOCK Score assigns a value of 0 to patients with acute onset, and those classified as chronic are assigned a value of 1.

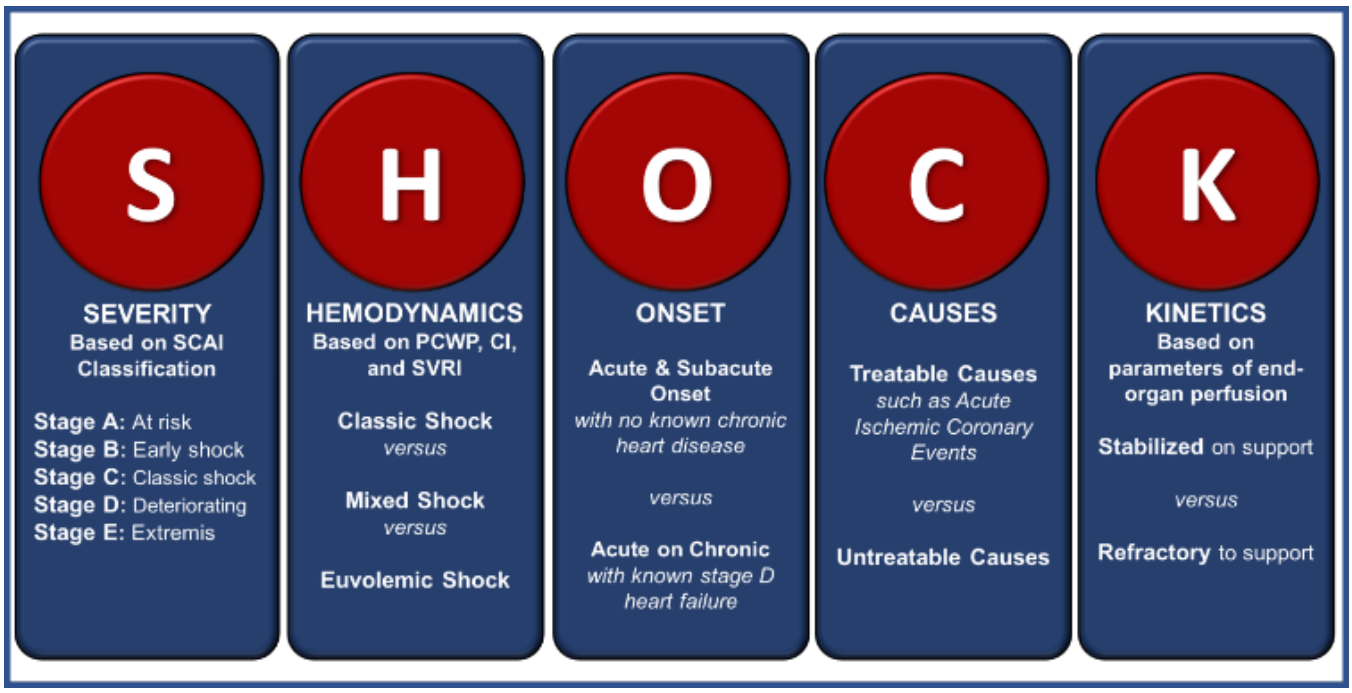
Cause

The only form of CS to have a proven therapy that can impact mortality is AMI-CS. One may argue that the diagnosis of AMI-CS is more readily available and, as such, tailored therapies have existed for decades. Discerning the etiology of CS upon presentation, however, is difficult, and trying to ascertain a diagnosis as quickly as possible can pave the wave to initiating early therapies that can potentially impact mortality. A broad assessment of CS must be made based on the cause. We propose the following etiologies: ischemic due to AMI, ischemic due to a mechanical complication, non-ischemic (such as acute myocarditis, acute on chronic stage D heart failure), right ventricular failure, and electrical storms. The Houston SHOCK Score assigns a value of 0 to patients classified as acute reversible and treatable; all other causes are assigned a value of 1.

Kinetics

Perhaps the least understood aspect of CS management is the kinetics (dynamic changes in the patient's profile) and predicting the outcome of support on pMCS devices. While some consider pMCS support devices a therapeutic option in the armamentarium utilized in CS management, it is crucial to emphasize that in most instances adequate pMCS support is intended to stabilize (*not to treat)* a worsening CS patient. In addition, pMCS devices help determine response to therapy

Figure 1. The Houston SHOCK Score incorporates the dynamic changes seen in this rapidly evolving hemodynamic catastrophe. The acronym SHOCK emphasizes five key aspects of patients in cardiogenic shock - Severity, Hemodynamics, Onset, Causes, and Kinetics - and allows healthcare workers to capture and score the dynamic changes encountered in cardiogenic shock.



Abbreviations: CI, cardiac index; PCWP, pulmonary capillary wedge pressure; SVRI, systemic vascular resistance index

and improve the overall clinical status and the patient's trajectory. While the optimal approach is not fully known, the ability to stabilize a patient by pMCS support represents a different group of patients than those that are unable to be stabilized. By describing the kinetics of CS, i.e. dynamic changes and responsiveness to therapy, patients can be classified as *stabilized* CS vs. *refractory shock* despite support. Assessment of the response to therapy is based on improvement in parameters of perfusion listed in Table 2. This will allow for quicker triage of worsening patients to dedicated shock centers. The Houston SHOCK Score assigns a value of 0 to patients classified as stabilized; those individuals classified as worsening/refractory are assigned a value of 2.

Applicability in Clinical Practice

In addition to offering a standardized approach to comprehensively assess patients in shock, we believe the SHOCK Score can serve important purposes that are yet to be validated. We believe dichotomizing each of the five variables of the Houston SHOCK Score into a score (Table 3) is practical. Healthcare providers can quickly triage the "sicker and refractory" patients to dedicated shock centers and offer time-sensitive therapies. In addition, admission score can be validated prospectively for outcomes including mortality. We believe the higher the score, the higher the acuity of the patient resulting in worse inpatient and long-term outcomes. Our initial scores are arbitrarily dichotomized to 0 and 1 (except for kinetics where 0 and 2 are used, as those in refractory CS should be given a higher weighted score in our opinion); however, we believe that ongoing validation research can offer a more accurate weighted score for some variables. Lastly, similar to SCAI Severity Classification of shock, this scoring system is dynamic and can be applied and utilized in a dynamic manner incorporating new data and response to therapy as available. As the kinetic response is a key component of this score, we believe capturing the score on admission (for triage purposes), and at 72 hours of admission may serve to be of prognostic significance that further validation is necessary. Retrospective data analysis is currently underway at our institution to validate this score.

Clinical Examples

A 68-year-old male presented with two hours of chest pain and was found to have large anterior STEMI, for which he underwent percutaneous coronary intervention (PCI) of the proximal left anterior descending coronary artery. The left circumflex and right coronary arteries were chronically occluded. His blood pressure was 75/44 mmHg on arrival for which an IABP was placed following the PCI. In the ensuing hours, the patient had a low blood pressure of 80/50 mmHg,

cool extremities, S3 gallop, reduced urine output, and elevated lactic acid requiring the addition of high doses of epinephrine and norepinephrine. This patient receives a Houston Shock Score of 4 ($S_1H_0O_1C_0K_2$). Such a high score would allow for a more urgent referral to a shock center and the allocation of the shock center's resources to be ready for support escalation at the time of arrival, such as a percutaneous MCS device implantation.

A 23-year-old female with familial cardiomyopathy on milrinone therapy and known reduced left ventricular systolic function presented for the fourth time in 3 months with 1 week of 4 pillow orthopnea and dyspnea on minimal exertion. On presentation, the patient had an SBP of 88 mmHg, S3 gallop on examination with 3+ pedal edema, and ascites. She underwent a right heart catheterization that revealed a right atrial pressure of 19 mmHg, pulmonary artery pressure of 49/35 mmHg, pulmonary capillary wedge pressure (PCWP) of 32 mmHg, and a CI of 1.7 L/m/m^2. An Impella 5.5 was placed, and her repeat hemodynamics at 24 hours showed a right atrial pressure of 10 mmHg, PCWP of 19 mmHg, and CI of 2.2 L/m/m^2. This patient receives a Houston Shock Score of 2 ($S_0H_0O_1C_1K_0$). While both patients are classified as "classic" CS per SCAI definition, the first patient has a higher Houston Shock Score as he needs more urgent attention, triage to a shock center, and escalation of therapy.

Conclusion

In summary, CS is a disease state with a heterogeneous pathophysiology, and its management starts with a comprehensive assessment that incorporates not only its ***severity***, but also its ***hemodynamic*** profile, ***onset***, ***cause***, and ***kinetic*** response to therapy. The SHOCK Score offers a more comprehensive and standardized taxonomy that can help move this field forward.

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