SAN ANTONIO

INTERVENTIONAL FORUM

Cardiogenic Shock Consensus Statement

September 1, 2023

Dear Colleagues,

In 2019, the San Antonio Interventional Forum **(SAIF)** officially endorsed the National Cardiogenic Shock Initiative **(NCSI)** Algorithm for managing acute myocardial infarction complicated by cardiogenic shock **(AMI-CS)**. This endorsement has prompted many local physicians to adopt key principles of cardiogenic shock **(CS)** treatment more broadly. As mechanical circulatory support **(MCS)** capabilities have improved, we have seen a notable change in practice. However, as our understanding of CS evolves, it is important to update and refine community standards for South Texas.

SAIF recognized the potential of standardized care by officially endorsing the NCSI Algorithm before the completion of the study. Although a definitive trial to establish a treatment paradigm and enhance guidelines is yet to be concluded, preliminary observational trials such as Inova 2019, NCSI 2021, and J-PVAD 2022 have shown promising outcomes with survival rates of 82%, 71%, and 81%, respectively. These trials signify substantial progress in the management of cardiogenic shock.

The SAIF Cardiogenic Shock Committee Consensus Statement **(CSCS)** serves as a complementary resource and guide for all hospitals, aligning with the actions of the Regional Resuscitation Committee led by Southwest Texas Regional Advisory Counsel **(STRAC)**. The primary objective is to establish a hierarchy of capable medical facilities that prioritize patient treatment and ensure appropriate levels of care based on the patient's condition and institutional capabilities. The CSCS offered by the SAIF Cardiogenic Shock Committee, approved by the Board, represents our most comprehensive resource for all facilities in South Texas.

These principles include early identification, following an algorithm-based approach, **prioritizing MCS before percutaneous coronary intervention (PCI)**, routine use of Swan-Ganz catheters, and ensuring timely escalation of care. Prompt treatment and early assessment to determine eligibility for advanced therapies or recovery are strongly advocated, especially in hospitals with cath lab capabilities. **Early activation for complete shock evaluation is crucial in these cases.**

To further aid in patient care, we recommend using the Cardiogenic Shock Working Group app provided in the CSCS, the new medical management algorithm, and the updated MCS algorithm. This app defines different phenotypes and stages of cardiogenic shock, facilitating aggressive treatment, particularly in phenotypes II/III and SCAI Stages D/E when recovery or advanced therapies are feasible.

In summary, as knowledge and capabilities progress, it is vital to establish all-inclusive care systems that are in line with suitable plans both within and between facilities. This can only be accomplished through a meticulous standardized approach. SAIF actively encourages cooperation with hospitals, STRAC, and treating physicians to fill care gaps and improve patient outcomes. We warmly invite and encourage your active involvement in this collective ***Call to Action*** to achieve the best possible results for patients in South Texas.

Respectfully,

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Cardiogenic Shock Medical Management Algorithm

Cardiogenic Shock Criteria:
If LVEF < 40% **WITH evidence of end-organ hypoperfusion:** (Lactate ≥ 3, ↓urine output (< 0.5 ml/kg/hr), cool extremities, or AMS)
AND /OR
Hemodynamic Criteria: SBP < 90 mmHg for 20 minutes, HR >100, or Pulse Pressure < 20 mmHg

Transfer to ICU, Insert A-line, **Strongly** Consider PA catheter

Hypoperfusion/ Low CO
"Cold & Dry"
- Thready pulses
- Low pulse pressure
- Cold distal extremities
- Slow capillary refill
- CI < 2
- SvO2 < 50%

Consider Inotropic Agent:
- If SBP > 100 AND eGFR ≥ 30
  - Milrinone 0.25 mcg/kg/min
- If SBP < 100 Order of choice
  1. Dobutamine 3-5 mcg/kg/min
  2. Dopamine 3-5 mcg/kg/min
  3. Norepinephrine 0.02-0.1 mcg/kg/min
  4. Epinephrine 0.02-0.1 mcg/kg/min

Monitor for arrhythmias (AFib/ VTach)

Hypoperfusion with Congestion/ Volume Overloaded with Low CO
"Cold & Wet"
- Cold distal extremities
- ↑ JVD
- Edema (pulm/peripheral)
- RA/CVP > 12
- CI < 2
- SvO2 < 50%

Consider:
- Inotropes (left)
- Diuretics (right)

Congestion/ Volume Overloaded
"Warm & Wet"
- ↑ JVD
- Pulmonary edema/ Crackles
- Dyspnea/Orthopnea
- Peripheral edema
- RA/CVP > 12

Consider Diuretics and Vasodilators:
- If < 10 lb gain above euvolemic weight
  - Diuretic IV pushes
- If > 10 lb gain above euvolemic weight
  - Lasix gtt 10-40 mg/hr
  - Bumex gtt 0.5-1 mg/hr
- If SBP > 100 AND eGFR ≥ 30
  - Milrinone 0.25 mcg/kg/min
  - Nitroglycerin
  - Nitroprusside (PA catheter)

If 2 moderate doses or 1 high dose level exceeded, OR if CI remains < 2, OR if repeat lactate does not downtrend, consider escalation to MCS device and/or **Cardiogenic Shock Team Consult**

UNOS Heart Committee Inotrope/Pressor Dose Ranges

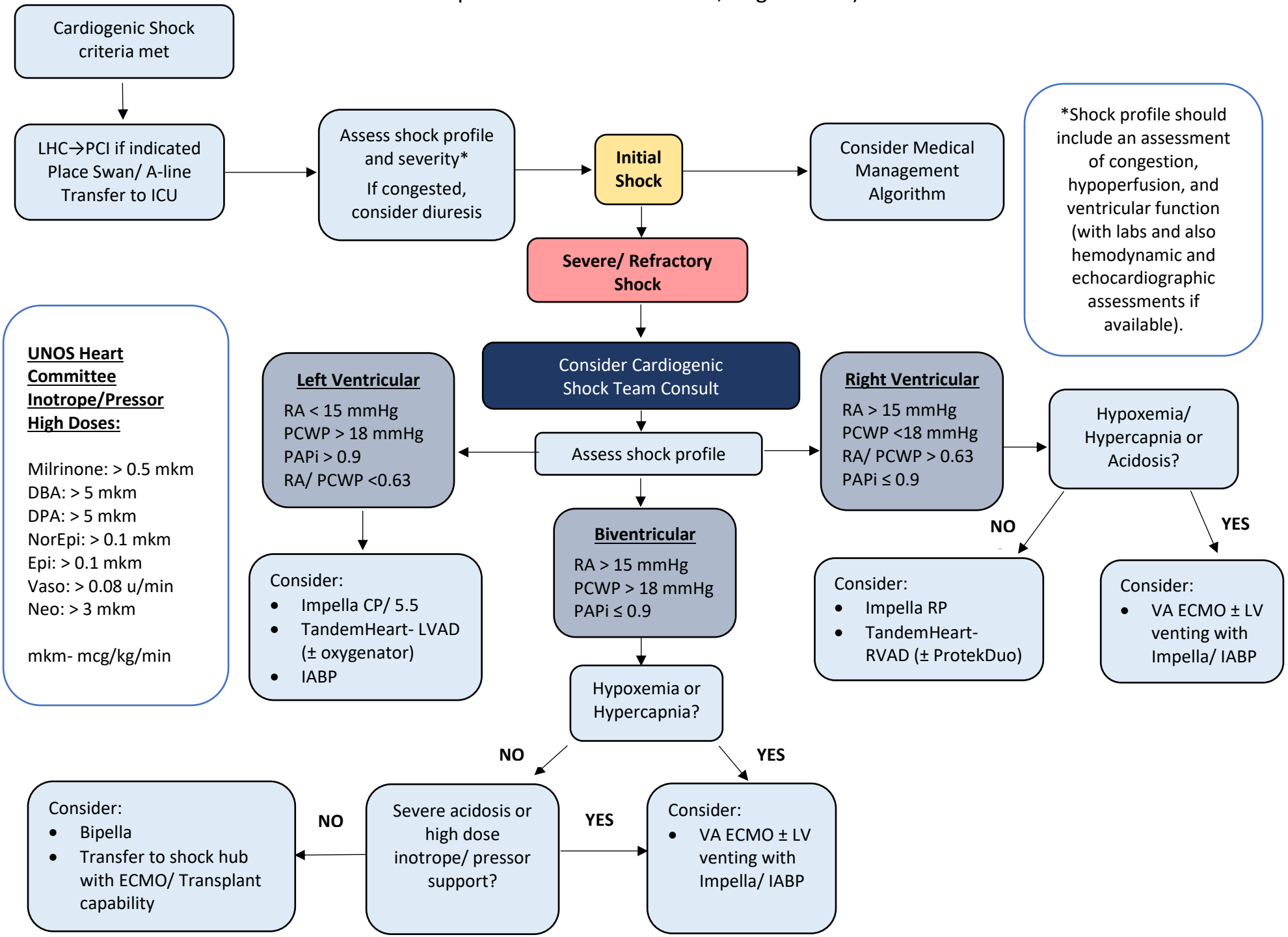
Low Doses:
Milrinone: 0.01-0.35 mkm
DBA: 0.01-3 mkm
DPA: 0.01-2 mkm
NorEpi: 0.01-0.04 mkm
Epi: 0.01-0.05 mkm
Vaso: 0.01—0.04 u/min
Neo: 0.01-1 mkm

Moderate Doses:
Milrinone: 0.375-0.5 mkm
DBA: 3-5 mkm
DPA: 2.1-5 mkm
NorEpi: 0.05-0.1 mkm
Epi: 0.06-0.09 mkm
Vaso: 0.05—0.08 u/min
Neo: 1.1-3 mkm

High Doses:
Milrinone: > 0.5 mkm
DBA: > 5 mkm
DPA: > 5 mkm
NorEpi: > 0.1 mkm
Epi: > 0.1 mkm
Vaso: > 0.08 u/min
Neo: > 3 mkm

Mechanical Circulatory Support Escalation Algorithm

Adapted from AHA Circulation, August 2022)



Cardiogenic Shock Consensus Statement

SAIF Cardiogenic Shock

CS is a severe medical condition that poses a significant and life-threatening risk. Despite advancements in medical technology and knowledge, the mortality rate for CS has remained unacceptably high, exceeding 50% for over two decades. Although there is increasing evidence supporting the early identification of CS, hemodynamic monitoring, and the escalation of mechanical circulatory support, there is still a wide range of treatment strategies and outcomes.

In response to the pressing situation at hand, SAIF is adopting a proactive approach to highlight the significance of standardized care. A key aspect of this approach involves accurate diagnosis of various CS phenotypes and stages to determine the level of urgency in decision-making and intervention. Additionally, precise, and timely diagnosis aids in identifying the underlying cause and guiding potential escalation strategies.

SAIF advocates for the use of the Society for Cardiovascular Angiography and Interventions Stages of Cardiogenic Shock (SCAI SHOCK Stages) and algorithms based on the hemodynamics obtained from a Swan-Ganz catheter to guide treatment decisions. To support this approach, SAIF endorses the tools provided in this document and encourages the implementation of a comprehensive, facility-based program that can effectively meet the needs of critically ill patients.

As an organization, we firmly believe that improving outcomes in CS requires a collaborative effort between SAIF, individual hospitals, advanced capability centers, and STRAC. By coordinating shock care and establishing a community standard, we can form a strong partnership that will improve patient outcomes and save lives.

Acknowledging NCSI

In 2019, SAIF took the proactive step of endorsing the NCSI before its completion. We highly appreciate the significant contributions that NCSI has made in the field of AMI-CS, both in terms of advancing research and enhancing patient care. Although NCSI was not a prospective, randomly controlled trial, it achieved remarkable outcomes in 406 patients, with a survival rate of 71% upon discharge and over 90% native heart recovery **(Table 1)**.

Table 1: NCSI Outcomes

Stage	Discharge	30-Days	1-Year
C & D (N=295)	79%	77%	62%
E (N=111)	54%	49%	31%
Overall (N=406)	**71%**	**68%**	**53%**

188 (46%) patients had cardiac arrest

SAIF believes that the most optimal results in CS can be obtained by implementing a treatment strategy based on algorithms, multidisciplinary management, and coordinated care. However, we also acknowledge the importance of tailoring these plans to suit the specific capabilities and experiences of each facility and team. Therefore, it is of utmost importance to implement an accessible medical management and open-label MCS algorithm that can serve as a widely accepted standard for South Texas.

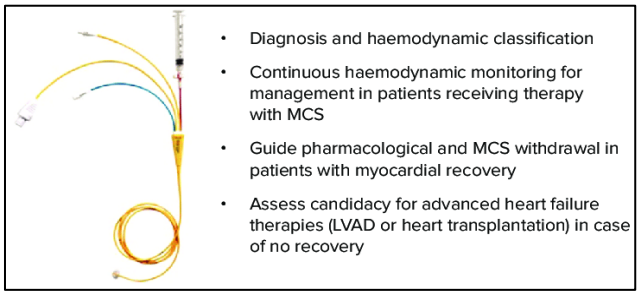
Vasopressors and the Swan-Ganz Catheter

Vasopressors and inotropes are commonly used as the initial treatment for CS with the aim of restoring hemodynamic stability. While these medications can be effective, it is essential to consider their potential drawbacks. These drugs increase afterload and oxygen demand, which may lead to end-organ hypoperfusion, lactic acidosis, and refractory shock.

To optimize the use of these medications, effective dosing and frequent reassessment is necessary. Weight-based dosing has been introduced to address some of these concerns, allowing for individualized dosing. It is important to note that the appropriate dosing levels for cardiac patients may not always be well-understood or applied in clinical settings. Hence, emphasizing extensive education and awareness within the ICU regarding the low, moderate, and high dose classifications by the United Network of Organ Sharing **(UNOS) (Exhibit 1)** and ensuring proper dosing is imperative. This will facilitate the safe and effective use of vasopressors and inotropes in the management of CS.

A pervasive challenge in managing CS is finding a balance between non-invasive hemodynamic monitoring and invasive tools like the Swan-Ganz catheter. Early trials using Swan-Ganz catheters to guide treatment in acute myocardial infarction patients showed harm. However, the ESCAPE trial demonstrated benefits for a specific cohort of patients with severe symptomatic decompensated heart failure. Consequently, high-level tertiary and quaternary centers witnessed a rise in the regular utilization of Swan-Ganz catheters as clinicians deemed invasive monitoring necessary for critically ill patients. Conversely, the usage of such catheters diminished in community hospitals during the same period.

Figure 1: Edwards Swan-Ganz Catheter



Recommendations for invasive hemodynamic monitoring in patients with cardiogenic shock from the Society for Cardiovascular Angiography and Interventions/Heart Failure Society of America.44

The SCAI/Heart Failure Society of America 2017 expert consensus document proposed practical uses of invasive hemodynamics, including continuous monitoring for patients receiving MCS, guiding pharmacological and MCS withdrawal in patients with myocardial recovery, and assessing candidacy for escalation and advanced heart failure therapies **(Figure 1)**. The Swan-Ganz catheter, which had declined in usage, is now being rediscovered as a valuable tool for various steps in cardiogenic shock management, from diagnosis to weaning from MCS.

Etiology and Cardiogenic Shock Phenotypes

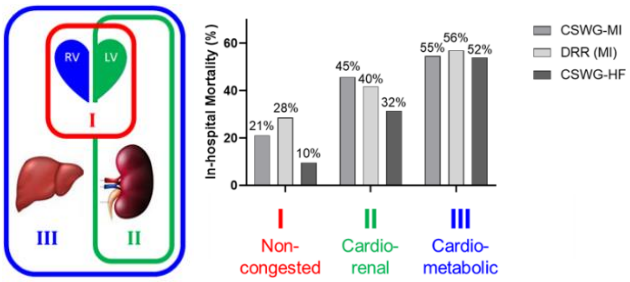
CS occurs more frequently in cases of ST-segment elevation myocardial infarction **(STEMI)** compared to non-ST-segment elevation myocardial infarction **(NSTEMI)**. The overall management of STEMI has improved significantly due to heightened community awareness, the establishment of clinical networks, and increased emphasis on primary PCI. Additionally, early identification of shock within 90 minutes of the door-to-balloon window has likely contributed to higher survival rates in STEMI-CS patients compared to the NSTEMI-CS population when there is less urgency to activate the Cath Lab.

Recent studies indicate that non-ischemic causes contribute to over 50% of CS cases, reflecting the increasing prevalence of heart failure. Although in-hospital mortality may be lower for HF-CS, many patients require short/long-term ventricular assist device or transplant during their hospital stay, in contrast to those with native heart survival. The Cardiogenic Shock Working Group **(CSWG)** has recently identified an association between de novo heart failure **(DNHF)** CS and increased in-hospital death, cardiac arrest, and faster progression to maximum severity stage according to SCAI criteria.

Cardiogenic Shock Consensus Statement

CS exhibits a diverse range of complexities, treatment options, and associated outcomes. **Early diagnosis and decision-making within the crucial initial hours can profoundly impact the clinical trajectory of many patients.** The CSWG has identified three distinct phenotypes: phenotype I (non-congested), phenotype II (cardio-renal), and phenotype III (cardiometabolic). These phenotypes are associated with increasing mortality rates and show a correlation with SCAI Stages **(Figure 2)**.

Figure 2: Cardiogenic Shock Phenotypes and Mortality



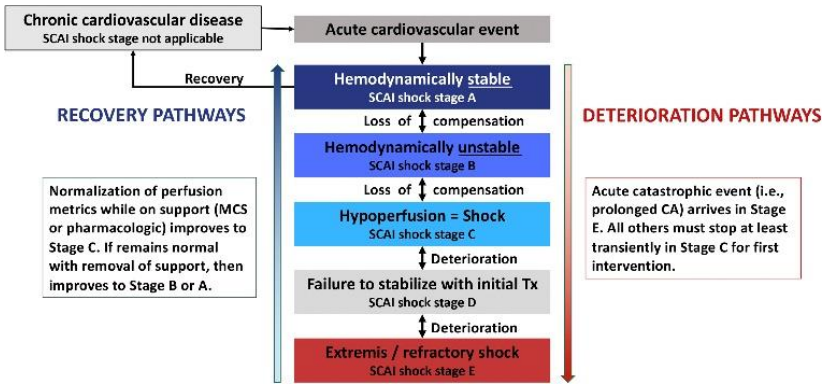
Hemodynamically, **phenotype I** is characterized by relatively stable cardiovascular parameters, lower heart rate, and filling pressures compared to the other phenotypes. These characteristics indicate a non-congested patient with a greater likelihood of recovery. On the other hand, **phenotype II** patients were typically older and had more co-existing conditions. They displayed a lower heart rate, elevated cardiac filling pressures, and a deterioration in kidney function, indicating a cardio-renal phenotype. Patients with cardiometabolic shock, referred to as **phenotype III**, exhibit several characteristic features. These include elevated levels of lactate and alanine aminotransferase, increased heart rate, and elevated right atrial pressure. Additionally, they present with low blood pressure, decreased cardiac power output, and cardiac index. These indicators suggest multiorgan involvement and are often accompanied by transaminase elevation and lactic acidosis in patients with CS.

Overall, these findings highlight the significance of accurately identifying the etiology and distinguishing between different phenotypes of CS. This precise identification can assist in determining the most suitable management strategy, the necessity for advanced heart failure consultation, and potential escalation.

SCAI Shock Stages

The classification of SCAI shock stages has gained significant popularity since its introduction in 2019, with researchers utilizing it in various clinical settings to guide treatment decisions and assess risk. As patients progress through different shock stages, their care trajectories evolve accordingly, posing challenges when transferred between healthcare facilities in terms of predicting optimal therapy timing. SAIF recommends incorporating the **Updated SCAI Shock Classification System with (A) Modifier for Cardiac Arrest (Exhibit 2)** as a fundamental aspect of patient management within a comprehensive hub and spoke model for cardiogenic shock care.

Figure 3: Recovery and Deterioration Pathways



The recent 2022 update on SCAI Shock has provided important clarifications and additions. Refinements have been made to the definition of cardiac arrest, and revisions to the SCAI SHOCK pyramid**.** Updated SCAI Stages with Descriptors **(Exhibit 3)** has been introduced to capture frequently observed variables. These updates emphasize the need to differentiate "high-risk" patients with severe shock

and compromised hemodynamics from those categorized as "high-risk" due to nonmodifiable mortality risk factors as they move through the deterioration and recovery pathways **(Figure 3)**.

Nonmodifiable mortality risk factors are factors that increase the risk of mortality in patients experiencing CS but cannot be changed or modified. These factors include pre-existing comorbidities (such as chronic kidney disease or advanced heart failure), frailty, certain genetic or inherited conditions, advanced age, and cardiac arrest. Decision-making becomes challenging when dealing with patients of advanced age and those who have experienced cardiac arrest.

Independent of other risk factors, the ability to withstand the insult of cardiogenic shock decreases with age. However, making a binary decision based on age alone is difficult due to the uncertainty surrounding its consideration. Defining the clinical significance of cardiac arrest is also complex. Cardiac arrest situations can vary, with some cases having no impact on clinical trajectory, while others may result in anoxic brain injury. At present, cardiac arrest should refer to patients with potential anoxic brain injury, indicated by a Glasgow Coma Scale score of less than 9 or the absence of motor response to voice.

Nonmodifiable risk factors play an important role in determining the clinical trajectory of patients with cardiogenic shock, regardless of the SCAI stages. Identifying these factors helps determine the prognosis and optimize palliative care for many of these patients.

Patient Management

Patients who have experienced STEMI complicated by CS and those who arrive at the hospital in SCAI Shock Stages D and E are commonly transferred directly to the Cath Lab with minimal delay. It is crucial to identify and treat early-stage shock **inpatients** with the same urgency regardless of their etiology. This involves **rapid mobilization of the Cath Lab** for patients in SCAI Shock Stage C and higher, defining coronary anatomy in cases of both AMI-CS and HF-CS, and use of the tools in the CSCS to improve outcomes.

According to the findings of the SCAI-CSWG, approximately 90% of patients in SCAI Stage B of cardiogenic shock progress to more advanced stages, underscoring the significance of early intervention. Interestingly, the time from baseline to reach the maximum stage is longer in Stage C compared to Stage B, highlighting the importance of initiating treatment promptly. For patients in the beginning stages of shock, it is recommended to place a Swan-Ganz Catheter, initiate a transfer to the Intensive Care Unit, and manage their care using the prescribed Medical Management Algorithm, unless a MCS strategy is indicated and available.

When patients arrive in the Cath Lab, it is imperative that they have a comprehensive shock evaluation gathering the clinical data in **(Figure 4)**. Ideally, a hemodynamic assessment via complete heart catheterization and labs should be drawn. The CSWG app **(Figure 5)** has calculators for SCAI Shock Stages, hemodynamics, and shock phenotypes.

Figure 4: Clinical Data for Comprehensive Shock Evaluation

Shock Stage Calculator	Hemodynamics Calculator	Shock Phenotype Calculator
Systolic Blood Pressure	Height	Age
Mean Arterial Blood Pressure	Weight	Serum Creatinine (mg/dL)
Serum lactate (mmol/L)	SaO-2 (%)	Serum Bicarbonate (mmol/L)
Serum alanine aminotransferase (ALT)	SvO2 (%)	Serum Alanine Transaminase (U/L)
Blood pH	Hemoglobin (g/dL)	Serum Lactate (mmol/L)
Number of vasopressors/inotropes	Heart Rate (beats/min)	Platelet Count (K/uL)
Number of mechanical circulatory devices	Age	White Cell Count (K/uL)
Out of hospital Cardiac Arrest	Systolic Blood Pressure (mm Hg)	
	Diastolic Blood Pressure (mm Hg)	
	Right Atrial Pressure (mm Hg)	
	Pulmonary Artery Systolic Pressure (mm Hg)	
	Pulmonary Artery Diastolic Pressure (mm Hg)	
	Pulmonary Capillary Wedge Pressure / Left Ventricular End Diastolic Pressure (mm Hg)	

In the context of CS, the preferred method of revascularization is PCI. Insights from NCSI suggest that the timing of MCS before PCI may provide benefit. However, the question of complete revascularization is complex and goes beyond the scope of this document. The limitations of complete revascularization likely involve multiple factors, including anatomical complexity, operator expertise, and fatigue.

Figure 5: CSWG App

When a patient's anatomy does not allow for PCI, the decision regarding urgent CABG becomes more nuanced, and it is recommended to involve the Heart Team. Full circulatory MCS and a Heart Failure Specialist is highly recommended in such cases where urgent surgical revascularization is warranted. One area that requires further exploration is the use of full circulatory support, LV unloading, and optimization for surgery in patients with TIMI II/III flow.

An innovative approach is the use of the Stafford-Prasad Shock Board **(Exhibit 5)** to monitor laboratory results, pressor requirements, and hemodynamics. Regular reassessments help minimize the chances of missing an opportunity for timely intervention and allow for potential reversal of a deteriorating condition. The Stafford-Prasad Shock Board not only serves as a visual aid but also encourages all healthcare team members to actively advocate for the patient. To streamline the care process, corresponding order sets have been created to align with the Shock Board, with updates scheduled at four-hour intervals during the first 24 hours.

Decisions regarding escalation and de-escalation should be made by a multidisciplinary team as a patient progresses down the recovery or deterioration pathway. Heart teams should have a low threshold for seeking consultation from a system and/or regional shock teams at specialized centers, depending on their experience and comfort level in treating CS patients.

Key factors to consider when determining the need for escalation include increasing lactate levels, the use of 2 moderate or 1 high-dose inotrope, a cardiac index below 2, a cardiac power output of less than 0.8 on pressors, non-modifiable risk factors and goals of treatment. In non-AMI shock patients, involving a heart failure specialist at the onset of care can change the longitudinal care trajectory with an early screening for advanced therapies.

Though there may not be a universal consensus on the specific incidence rates, studies suggest that approximately 20-40% of patients with CS experience right heart failure. Patients who develop right ventricular or bi-ventricular failure often fall into phenotype III, characterized by significant venous congestion. Similar to HF-CS, the SAIF organization strongly recommends early consultation with a heart failure specialist and the implementation of aggressive therapy when necessary.

Conclusion

The Cardiogenic Shock Consensus Statement underscores the significance of integrated systems, collaboration, and algorithms to improve outcomes for patients with CS. Early identification and prompt treatment of initial-stage shock, coupled with rapid mobilization of the Cath Lab and utilization of tools like the Stafford-Prasad Shock Board, play a crucial role. Early involvement of heart failure specialists and implementation of coordinated care systems optimize resources and ensure timely interventions. This collaborative approach, coupled with standardized care, saves lives, enhances patient outcomes, and upholds the highest level. We strongly urge physicians to actively engage, lead within their respective institutions, form teams to identify, treat, and evaluate quality through an intra and inter-hospital plan that prioritizes patient well-being to achieve the best outcomes for CS patients in South Texas.

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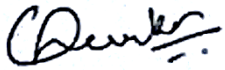

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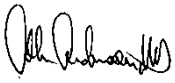

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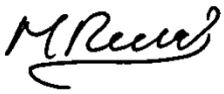

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Exhibit 1: UNOS High, Moderate, and Low Pressor/Inotrope Definitions

	Clinical Indication	Dose (mcg/kg/min)			Dose			Major Side Effects
		Low	Moderate	High	Low	Moderate	High	
Inotropes								
Epinephrine	Shock (cardiogenic, vasodilatory) Cardiac arrest Bronchospasm/anaphylaxis Symptomatic bradycardia or heart block unresponsive to atropine or pacing	0.01 – 0.05	0.06 – 0.09	>0.1				Ventricular arrhythmias Severe hypertension resulting in cerebrovascular hemorrhage Cardiac ischemia Sudden cardiac death
Milrinone	Low CO (decompensated HF, after cardiotomy)	0.01 – 0.35	0.375 – 0.5	>0.5				Ventricular arrhythmias Hypotension Cardiac ischemia Torsade des pointes
Dobutamine	Low CO (decompensated HF, cardiogenic shock, sepsis-induced myocardial dysfunction) Symptomatic bradycardia unresponsive to atropine or pacing	0.01 – 3	3 – 5	>5				Tachycardia Increased ventricular response rate in patients with atrial fibrillation Ventricular arrhythmias Cardiac ischemia Hypertension (especially nonselective β-blocker patients) Hypotension
Dopamine	Shock (cardiogenic, vasodilatory) HF Symptomatic bradycardia unresponsive to atropine or pacing	0.01 – 2	2.1 – 5	>5				Severe hypertension (especially in patients taking nonselective β-blockers) Ventricular arrhythmias Cardiac ischemia Tissue ischemia/gangrene (high doses or due to tissue extravasation)
Vasopressors								
Norepinephrine	Shock (vasodilatory, cardiogenic)	0.01 – 0.04	0.05 – 0.1	>0.1	<5	5 – < 12	≥12	Arrhythmias Bradycardia Peripheral (digital) ischemia Hypertension (especially nonselective β-blocker patients)
Vasopressin	Shock (vasodilatory, cardiogenic) Cardiac arrest	0.01 – 0.04	0.05 – 0.08	>0.08				Arrhythmias Hypertension Decreased CO (at doses >0.4 U/min) Cardiac ischemia Severe peripheral vasoconstriction causing ischemia (especially skin) Splanchnic vasoconstriction
Phenylephrine	Hypotension (vagally mediated, medication-induced) Increase MAP with AS and hypotension Decrease LVOT gradient in HCM	0.01 – 1	1.1 – 3	>3				Reflex bradycardia Hypertension (especially with nonselective β-blockers) Severe peripheral and visceral vasoconstriction Tissue necrosis with extravasation

Exhibit 2: Updated SCAI Stages with (A) Modifier for CA Arrest

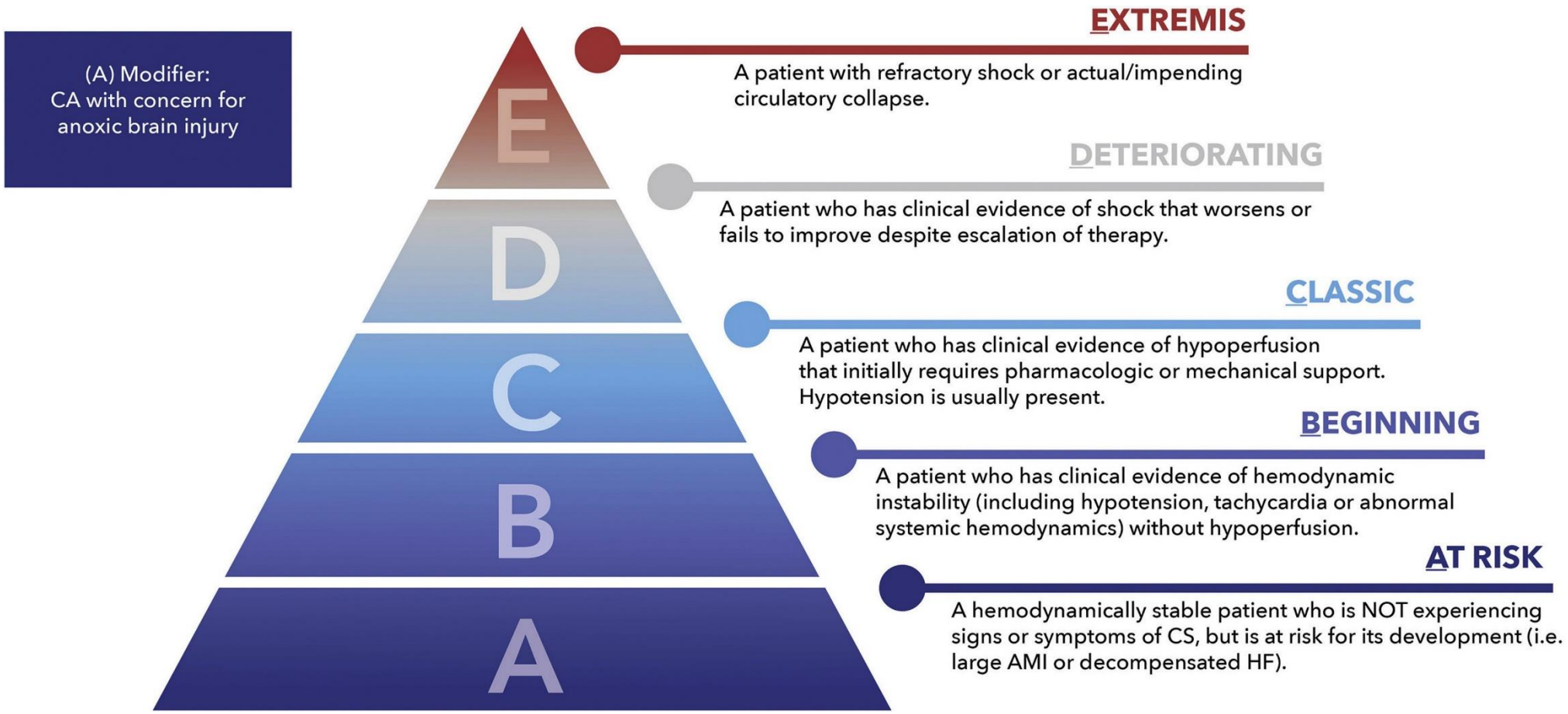


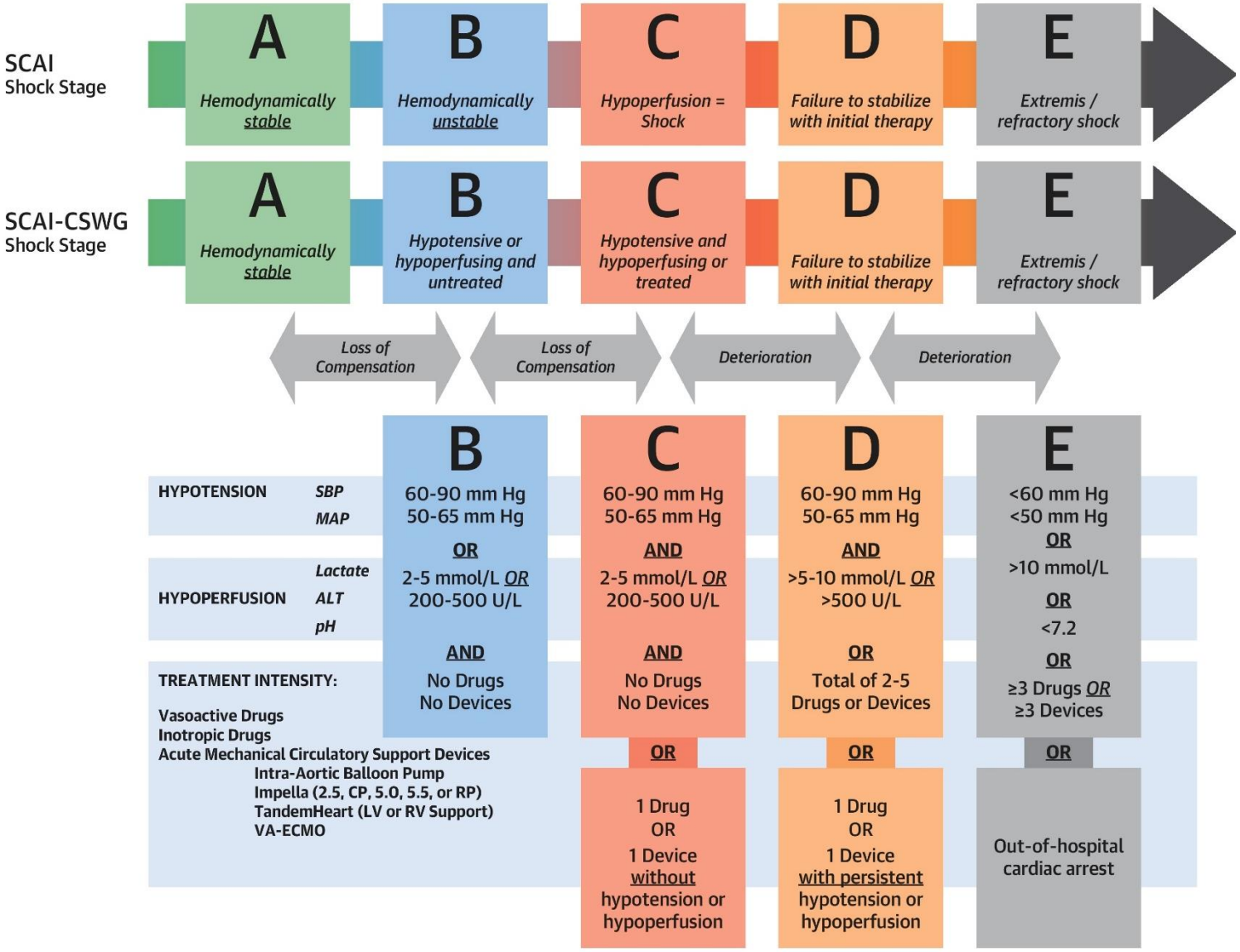
Exhibit 3: Updated SCAI Stages with Descriptors

Stage	Description	Physical examination/bedside findings		Biochemical markers		Hemodynamics	
		Typically includes	May include	Typically includes	May include	Typically includes	May include
A At risk	A patient who is **not currently experiencing signs or symptoms of CS, but is at risk for its development**. These patients may include those with large acute myocardial infarction or prior infarction and/or acute or acute-on-chronic heart failure symptoms.	**Normal JVP** **Warm and well-perfused** • Strong distal pulses • Normal mentation	Clear lung sounds	**Normal lactate**	Normal labs • Normal (or at baseline) renal function	**Normotensive** (SBP ≥100 mmHg or at baseline)	If invasive hemodynamics are assessed: • Cardiac Index ≥2.5 L/min/m^2 (if acute) • CVP ≤10 mmHg • PCWP ≤15 mmHg • PA saturation ≥65%
B Beginning CS	A patient who has **clinical evidence of hemodynamic instability** (including relative hypotension or tachycardia) **without hypoperfusion**.	**Elevated JVP** **Warm and well-perfused** • Strong distal pulses • Normal mentation	Rales in lung fields	**Normal lactate**	Minimal acute renal function impairment Elevated BNP	**Hypotension** • SBP <90 mmHg • MAP <60 mmHg • > 30 mmHg drop from baseline **Tachycardia** • Heart rate ≥100 bpm If invasive hemodynamics assessed **(strongly recommended)** • Cardiac index <2.2 L/min/m^2 • PCWP >15 mmHg	
C Classic CS	A patient who manifests with **hypoperfusion and who requires one intervention (pharmacological or mechanical) beyond volume resuscitation**. These patients typically present with relative hypotension (but hypotension is not required).	**Volume overload**	Looks unwell Acute alteration in mental status Feeling of impending doom Cold and clammy Extensive rales Ashen, mottled, dusky, or cool extremities Delayed capillary refill Urine Output <30 mL/h	**Lactate ≥2 mmol/L**	Creatinine increase to 1.5 x baseline (or 0.3 mg/dL) or > 50% drop in GFR Increased LFTs Elevated BNP		
D Deteriorating	A patient who is similar to category C but is getting worse. **Failure of initial support strategy to restore perfusion** as evidenced by worsening hemodynamics or rising lactate.	**Any of stage C and worsening (or not improving) signs/ symptoms of hypoperfusion despite the initial therapy.**		**Any of stage C and lactate rising and persistently >2 mmol/L**	Deteriorating renal function Worsening LFTs Rising BNP	**Any of stage C and requiring escalating doses or increasing numbers of pressors or addition of a mechanical circulatory support device to maintain perfusion**	
E Extremis	**Actual or impending circulatory collapse**	**Typically unconscious**	Near pulselessness Cardiac collapse Multiple defibrillations	**Lactate ≥8 mmol/L**[a]	CPR (A-modifier) Severe acidosis • pH < 7.2 • Base deficit >10 mEq/L	**Profound hypotension despite maximal hemodynamic support**	Need for bolus doses of vasopressors

BNP, B-type natriuretic peptide; CPR, cardiopulmonary resuscitation; CVP, central venous pressure; GFR, glomerular filtration rate; JVP, jugular venous pressure; LFT, liver function tests; MAP, mean arterial pressure; PA, pulmonary artery; PCWP, pulmonary capillary wedge pressure; SVP, systolic ventricular pressure.

[a] Stage E prospectively is a patient with cardiovascular collapse or ongoing CPR.

Exhibit 4: Clinical Variable and Parameters to Define Society for Cardiovascular and Interventions Stages



Kapur NK, et al. J Am Coll Cardiol. 2022;80(3):185-198.

Exhibit 5: Stafford-Prasad Shock Board

Wt: Kg BSA:				12HR EVALUATE			24HR EVALUATE
First 24hrs Q4	Time:	Time:	Time:	Time:	Time:	Time:	Time:
Date:	0 hrs	4 hrs	8 hrs	12 hrs	16 hrs	20 hrs	24 hrs
SvO2							
SaO2							
Hgb							
CO/CI							
Map							
Cvp							
PaS/PaD							
SVR							
SV							
CPO							
PAPI							
Lactate							
Cr							
Impella P-level							
Flow: l/min							
Dopa / Dobut							
Epi / Mil							
Levo / Vaso							
Isuprel / Neo							
Admission ICU & Daily							
PfHgb							
Ast/Alt							
T-Bili							

Hemodynamic Calculations

Map = SBP + DBP + DBP / 3
CO = SV x HR
CI = CO/BSA
SVR = MAP - CVP (80)/ CO
PVR = mPAP- PCWP (80)/CO
CPO = MAP x CO / 451
PAPI = sPAP - dPAP / CVP
FICK = 3.3 x Dry Weight Kg / Hgb (Sao2 – Svo2) 1.34 (x)10 = CO

Notes:

CPO/PAPI Calculator

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