Case-Based Device Therapy for Heart Failure

Ulrika Birgersdotter-Green
Eric Adler
Editors

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Section I
Management of Cardiogenic Shock

Section Editor: Hao A. Tran

Assessment of the Shock Patient and Hemodynamic Monitoring

Jorge Silva Enciso

Case Vignette

A 50 year-old female with past medical history of breast cancer and chemotherapy, paroxysmal atrial fibrillation and diabetes mellitus, presents with dyspnea on exertion, orthopnea and paroxysmal nocturnal dyspnea. On exam, she is hypotensive (83/61 mmHg), tachycardic (100 beats per min), and tachypneic (20 breaths per min). She has a regular rhythm, systolic ejection murmur at the left apex 3/6, jugular venous distention up to the mandible, sign of hepatojugular reflex, leg edema 2+, and cool distal peripheries. Her blood work is significant for a BUN 33 mmol/dL, creatinine 1.64 mg/dL, total bilirubin 2.42 mg/dL, lactate 2.4 mmol/L. Her NT pro-BNP is 6310 pg/mL, HS-troponin 18 ng/L. Her echocardiogram shows an LV ejection fraction of 12%, end diastolic dimension 6.7 cm, reduced RV function, pulmonary artery systolic pressure of 47 mmHg, moderate-severe mitral regurgitation and severe tricuspid regurgitation. A pulmonary artery catheter was placed showing the following hemodynamics:

Variable	Value	Variable	Value
RA, mmHg	11	SVR, dyn/cm/sec^5	1600
PA, mmHg	55/33/39	PVR, woods units	237
PCWP, mmHg	21	RA:PCWP ratio	0.52
PA Saturation, %	47	PAPi ratio	2.0

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Variable	Value	Variable	Value
AO Saturation, %	99	CPO, watts	0.41
Cardiac Output, L/min	2.3	BP, mmHg	106/67/80
Cardiac Index, L/min/m^2	1.8	HR, bpm	132

The patient is started on Norepinephrine that is promptly escalated to 11 μg/kg/min, Vasopressin 0.04 UI/hr, Dobutamine 3 μg/kg/min and Milrinone 0.25 μg/kg/min.

Definition

Cardiogenic Shock (CS) complicates 5–10% of acute myocardial infarction (AMI) cases with an in-hospital mortality of 41–50% which has been unchanged over 2 decades. Among survivors of AMI, up to 19% of patients will experience a readmission after discharge, with 30% of them developing recurrent heart failure symptoms. Furthermore, 30% of all CS cases present as acute decompensation of chronic systolic heart failure [1]. A higher incidence of CS is seen in elder patients, female gender, patients with diabetes or a prior history of LV dysfunction. Classically, cardiogenic shock has been defined as tissue hypoperfusion and hypoxia due to impaired cardiac function and low cardiac output. It is manifested by abnormal clinical and biomarkers of end organ dysfunction that require either pharmacological or mechanical circulatory support interventions [1]. However, the parameters that define CS differ due to the complexity of its presentation.

Clinical trials defining CS have resolved to count on 3 indicators of cardiac performance: (1) a systolic blood pressure <90 mmHg and use of drugs or devices to maintain BP above 90 mmHg; (2) Cardiac Index of ≤2.2 ml/min/m^2 and a capillary wedge pressure ≥15 mmHg; (3) altered mental status, decreased urine output ≤30 ml/h and lactate ≥2 mmol/L. Clinical features of CS have varied across clinical trials leading to lack of uniformity in defining CS patients which has impact clinical trial outcomes. Recently, The Society of Cardiovascular Angiography and Interventions (SCAI) has developed a classification system as a referendum to differentiate patient subsets and risk stratify their morbidity and mortality risk. This schema allows rapid interpretation and categorization of patients to strategize which therapeutics will benefit each individual (Table 1) [2].

Causes

1. **Acute myocardial infarction (AMI)**. Accounts for 30–80% of the causes of CS, with ST-segment elevation MI being the most common presentation compared to Non-ST elevation MI. ST-segment elevation MI is the leading cause

Table 1 SCAI Cardiogenic shock classification

SCAI shock stage	Physical exam	Biomarkers	Hemodynamics
A	• Normal JVP, clear lung sounds, • Strong distal pulses • Normal mentation	• Normal renal function and lactate	• SBP > 100 mmHg • CI > 2.5 • CVP < 10 • PASAT ≥ 65%
B	• Elevated JVP, rales • Strong distal pulses • Normal mentation	• Minimal renal function impairment • Elevated BNP • Normal Lactate	• SBP < 100 OR MAP < 60 OR > 30 mmHg drop • Pulse ≥ 100 • CI ≥ 2.2 • PASAT ≥ 65%
C	• Ashen, mottled, dusky skin • Volume overload, extensive rales, Killip 3–4, Bipap or mechanical ventilation • Acute AMS	• Lactate > 2 • Creatnine doubling or > 50% drop in GFR, UO < 30 mL/hr • Increased BNP • Increased LFT	• Drugs/Device to maintain BP above Stage B • CI ≤ 2.2, PCWP ≥ 15, RA/WP ≥ 0.8, PAPi < 1.85, CPO ≤ 0.6
D	• Any stage C	• Stage C + Deteriorating	• Any stage C AND requiring multiple pressors, OR addition of MCS to maintain perfusion
E	• Near Pulselessness, cardiac collapse, defibrillator use • Mechanical Ventilation	• Lactate > 5 • pH < 7.2	• No SBP w/o resuscitation • PEA or refractory VT/VF • Hypotension despite maximal support

of death in patients with AMI with an in-hospital mortality close to 36–50% [3]. The clinical presentation of patients with CS are predominantly left ventricular failure (78.5%), severe mitral regurgitation (6.9%), ventricular septal rupture (3.9%), right ventricular failure (2.8%) and cardiac tamponade (1.4%) [4]. Among those who survive to discharge 18.6% have a 30-day risk of readmission (median time of 10 days) with the most common cause being heart failure (39%) followed by new myocardial infarction (15%) and arrhythmias (11%) [5]. Compared to other causes of CS, patients with CS-AMI present with a higher number of cardiovascular co-morbidities including hypertension, diabetes mellitus and smoking. Similarly, compared to other causes of CS, a significant number of CS-AMI patients require mechanical circulatory support, mechanical ventilation and renal replacement therapy at the time of their presentation due to the clinical severity of CS with substantial metabolic disturbances (i.e. higher lactate acidemia, elevated liver function test and renal dysfunction) [6].

2. **Acute Heart Failure (AHF)**. Accounts for 46% of causes of CS based on contemporary data from critical care registries. It is associated with a 31% in-hospital mortality. Patients within this group present with high filling pressures,

low oxygen delivery, higher burden of atrial arrhythmias or ventricular arrhythmias, pulmonary hypertension, chronic kidney disease and severe valvular disease requiring often invasive hemodynamic monitoring, higher use of vasoactive medications and mechanical circulatory support for stabilization (26% of Non-Ischemic Cardiomyopathy compared to 61% of AMI patients) [6]. MAY NEED TO EXPAND THE AHF CAUSES SECTION TO TYPES OF AHF ICM VERSUS NICM

3. **Non-AMI causes**. Other causes of CS are less common and can occur concomitant to the most common causes of CS including valvular heart disease (valvular stenosis or acute insufficiency,) (11%), myocarditis (2%), stress induced cardiomyopathy (2%), post-partum cardiomyopathy, hypertrophic cardiomyopathy and aortic dissection, all which can rapidly deteriorate through direct or indirect impact on the myocardial function (Table 2).

Pathophysiology

Cardiogenic shock precipitates when there is profound depression of the myocardial function resulting in deleterious consequences to end organ perfusion triggering a downward spiral of low cardiac output, reduced blood pressure, ischemia

Table 2 Causes of cardiogenic shock

Acute Myocardial infarction	Heart failure	Valvular-native or prosthetic	Electrical
Mechanical complication • Ventricular septal rupture • Papillary Muscle Rupture • Free Wall Rupture • Cardiac tamponade	• Ischemic Cardiomypathy • Dilated Cardiomyopathy	Stenosis	Atrial arrhythmias
Mitral regurgitation	Myocarditis	Acute regurgitation	Ventricular Tachycardia
Right Ventricular Infarction	Stress induced cardiomyopathy	Valvular Obstruction	Bradycardia
Left Ventricular Dysfunction	Pregnancy associated • Peripartum cardiomyopathy • Coronary Artery Dissection	Leaflet failure	
	Post-Cardiotomy shock	Valve dehiscence	
	Outflow obstruction • Hypertrophic cardiomyopathy		

with the latter enhancing the vicious cycle of perpetual shock. Mechanisms to counterbalance this negative cycle include vasoconstriction and fluid retention with the goal to maintain tissue perfusion and cardiac output. However, in the presence of cardiogenic shock, a cascade of inflammatory markers is released due to poor perfusion. Reactive oxygen species, nitric oxide synthase, peroxy-nitrite and interleukins among other markers will promote vasodilation, reduce catecholamine sensitivity and reduce contractility ultimately affecting myocardial performance [7]. With persistence of inadequate forward flow, the remaining viable myocardium starts to increase its oxygen demand and consumption, compromising further global ventricular function due to ischemia. When left ventricular dysfunction progresses over the course of the shock stage, pulmonary artery pressures and left sided pressures commence to increase leading to interventricular septum displacement to the right ventricular cavity reducing preload to the right ventricle (RV). The acute changes in pressure load deteriorate RV function triggering a rise in venous pressures. This leads to alterations in right ventricular structure causing cavity dilation and displacing the interventricular septum to the left ventricular space, compromising left ventricular diastolic filling and reducing coronary and systemic perfusion causing end organ damage [8].

Similar to CS from left ventricular dysfunction, the pathogenesis of cardiogenic shock due to right ventricular dysfunction (RVD) is associated with poor prognosis. In the presence of acute myocardial infarction, acute RVD presents with ischemia, arrhythmias, cytokine releases (i.e. tumor necrosis factor-α, interleukins) inducing further impact on systolic and diastolic function, poor tolerance to changes in afterload, pulmonary vasoconstriction due to hypoxia and increase risk of microthrombi and emboli. Furthermore, in those patients that require mechanical ventilation, RV function is negatively affected by acute changes in preload an afterload from elevated intra pulmonary pressures, especially when high positive end expiratory pressure ventilation is required [9]. With the abrupt changes in load, RV stroke volume is decreased, RV systolic pressure is reduced prompting reduction in LV end diastolic filling which in turn will contribute to coronary and systemic hypoperfusion. Overtime reduction in RV contractility results in annular and cavity dilation leading to tricuspid regurgitation. The increased regurgitant volume will further exacerbate RV dilation and drive ventricular inter-dependence to affect LV filling begetting a vicious cycle of hypoperfusion. As 20–40% of the RV systolic function is derived from interventricular and LV contraction, once ventricular interdependence develops, it is paramount to maintain and enhance ventricular performance to halt the shock sequence.

Early Recognition of Shock

Clinical features present during the Initial evaluation of the individual with CS include hypotension (systolic blood pressure less than 90 mmHg), diminished pulses, elevated jugular venous pressure, dyspnea, cool peripheries, delated

Table 3 Clinical distinct features of ventricular dysfunction

Features of LV dysfunction	Features of RV dysfunction
Pulmonary rales and/or wheeze	Increase jugular venous pressure
Displaced point of maximal impulse	Tricuspid regurgitation
Mitral or aortic regurgitation	Hepatomegaly
	Hepato-jugular reflex
	Lower extremity edema

capillary refill and altered mental status. However distinct characteristics upon presentation can guide the clinician to elucidate between which ventricle is compromised (see Table 3).

It is important to recognize however that presence of elevated JVP can be seen in both right and left ventricular dysfunction as recent studies show that more than 70 percent of individuals with acute heart failure present with left and right sided concordant hemodynamics (right atrial pressure $\geq$ 12 mmHg equates to a pulmonary capillary wedge pressure $\geq$ 30 mmHg) supporting the notion of JVP as an estimator of pulmonary capillary wedge pressure [10].

- **Electrocardiogram Interpretation**

  In patients with initial presentation of CS-AMI, ECG is essential in the decision process for management of patients suspected of ACS. The ECG should be ordered within 10 min of arrival to the emergency room and If the initial ECG is non-diagnostic, serial ECG should be obtained every 15–30 min. Any ST segment deviation should promptly be determined for acute coronary intervention. Presence of ST segment elevation in 2 or more contiguous leads indicates urgent reperfusion, ST segment depressions, transient ST-elevation ($\geq$0.5 mm [0.05 mV]), or new T wave inversion symmetrical in the precordial leads ($\geq$2 mm [0.2 mV]) are strongly suspicious for acute coronary syndrome (ACS) [11]. Presence of Q waves reflect size and extension of the MI and predicts lower ejection fraction [12]. Ventricular or atrial arrhythmias can also be suggestive for ACS as up to 6% of patients can develop ventricular tachycardia or ventricular fibrillation within an hour of symptom presentation. Most commonly however patients with ACS can present with non-sustained monomorphic in the first 24–48 h after an AMI and usually associated with regional ischemia. Sustained VT is less common but can be seen in ST-elevation AMI associated with larger infarction areas [13].

Risk Assessment

Once clinical identification of CS is established, phenotyping the hemodynamic presentation is essential to guide therapy. The common presenting theme is a low cardiac index with a variable preload, volume and systemic vascular resistance.

A framework has been defined to characterize the hemodynamic status of patients presenting with CS. The classic cold and wet profile is seen in more than 60% of patients with CS-AMI while those with cold and dry profile (isolated hypoperfusion) are seen in close to 30% of patients with CS-MI (Table 4). Moreover, the mortality associated with each profile relies vastly on the presence of hypoperfusion independent on the presence or absence of pulmonary congestion. In the SHOCK trial, hypoperfusion was defined by oliguria <30 ml/hr or cold peripheries which identifies individuals with evidence of end organ dysfunction. The study showed an in-hospital mortality of 70% for those with hypoperfusion without pulmonary congestion compared to 60% with presence of both hypoperfusion and congestion. Those with no hypoperfusion with or without congestion had a 20% mortality [14]. Similarly those patients presenting with the wet and warm profile have a commensurate mortality risk to those in other profiles. This group is characterized by low cardiac index, low-normal systemic vascular resistance and elevated wedge pressure. In those presenting with ST segment elevation AMI, 25% met systemic inflammatory response syndrome (SIRS) criteria defined as presence of two or more of the following: 1. heart rate >90 beats/min; 2. respiratory rate >20 breaths/min; 3. body temperature >38 or <36 °C; 4. leukocyte count >12 or $<4 \times 10^9$/L. For those with SIRS at the time of AMI presentation prognosis is poor with a mortality risk of 31% and a 2–threefold risk for death, shock, heart failure and stroke at 90 days [15].

The basis of profiling patients with CS remotes to the early era of AMI managed by thrombolytic therapy. Originally developed in 1967, the Killip-Kimball classification is based on the bedside clinical assessment of patients presenting with left ventricular dysfunction due to AMI. The classification is divided in 4 categories: class (I) no clinical signs of heart failure; class (II) HF with jugular venous distention, rales and S3 on heart auscultation; class (III) overt pulmonary edema and class (IV) cardiogenic shock and hypoperfusion. The significance of this classification remains relevant today as many studies continue to validate its association with mortality. A recent study examining the temporal trend in outcomes of AMI patients stratified by Killip class showed that this classification remains an independent predictor of mortality with a 3 to fourfold risk of death post-MI specifically in those with Killip class greater than or equal to 2. Patients

Table 4 Hemodynamic profiles in cardiogenic shock

	Volume		
Perfusion		Dry	Wet
	Warm	Increased CI Low SVRi Low-Normal PCWP	Low CI Low-Normal SVRi High PCWP
	Cold	Low CI High SVRi Low-Normal PCPW	Low CI High SVRi High PCWP

CI: Cardiac Index; PCWP: Pulmonary Capillary Wedge Pressure; SVRi: Systemic vascular resistance index

with higher Killip class exhibited more complications including acute kidney injury, new onset atrial fibrillation and ventricular arrhythmias [16].

Risk Scores

Risk prediction in CS is limited due to the heterogeneity of its presentation and causes leading to CS. About one fifth of the causes are not related to AMI however all CS cases share similar variables that can forecast patient outcomes. However, their use in predicting short-term mortality and survival after MCS is helpful. The advantage of risk classifying CS patients is to rapidly determine severity of presentation and facilitate clinical decision making utilizing readily available data obtained with in 24 hr of CS presentation (Table 5).

Biomarkers

Evaluation of myocardial injury severity through biomarker data is paramount as they serve to support the diagnosis of CS, distinguish the hemodynamic profile, determine prognosis. The continuous assessment of the biomarker profile can portend the temporal status of a patient in shock and define treatment effects that may identify responders and non-responders to therapy. The changes in biomarkers overtime can also help predict myocardial recovery.

Table 5 Risk scores utilized in cardiogenic shock

This table 5 Risk ScoreRisk score/trial	Components
Shock trial	Clinical Score: Age, shock on admission, end-organ hypoperfusion, anoxic brain injury, systolic blood pressure, prior CABG, noninferior MI, and creatinine ≥ 1.9 mg/dL. Hemodynamic Score: LV stroke work, LVEF < 28%. The limitations of this score is based on the treatments offered at the time period (1993–1999), and not with contemporary therapeutic resources existent to treat shock [38]
CardShock trial	ACS etiology, age, previous MI, prior CABG, confusion at presentation, low LVEF, lactate levels, eGFR. The risk tool was validated in 384 patients from the IABP-SHOCK II trial and showed an AUC 0.85 for mortality prediction [39]
IABP-SHOCK II score	Age > 73 years (1 point); 2) history of stroke (2 points); Glucose > 191 mg/dL (1 point); Creatinine > 1.5 mg /dL (1 point); lactate > 5 mmol/L (2 points); TIMI flow < 3 after PCI (2 points). Risk categories based on the points where low 0–2 points, intermediate 3–4 points and high 5–9 points with mortality rates of 23.8%, 49.2% and 76.6% respectively. The AUC for short-term mortality in AMI-CS was 0.73. When validated with patients included in CardShock, IABP-SHOCK II score showed a similar AUC 0.73 [40]

Within 12 h of Ý metabolic panel, blood count, arterial blood gas and lactate should be obtained. Electrolyte evaluation, liver and renal function parameters are important elements of end organ perfusion Cardiac enzymes should be obtained serially and trend every 6 h. Frequent monitoring of cardiac markers can reveal the degree of injury the myocardium has sustained since the initial event. The following are some biomarkers that have demonstrated prognostic value in patients with cardiogenic shock:

- **N-terminal pro-B-type natriuretic peptide (NT-proBNP)**. NT-proBNP should be obtained as it can help prognosticate outcomes in cardiogenic shock patients. In a sub study from the IABP shock trial, NT-proBNP values were higher among non survivors compared to survivors specially in those with impaired renal function, signaling a degree of advanced shock stage and end organ dysfunction [17]. It is important to note that high natriuretic peptide levels do not necessarily correlate with elevated filling pressures however in those admitted to ICU with shock, NT-proBNP remain an independent predictor of ICU mortality with a 15-fold risk of death compared to those with levels <1200 pg/mL [18].
- **Lactate**. As a marker of tissue hypoperfusion, it has been associated with a high 30-day mortality. In patients presenting with ACS, admission lactate is predictor of in-hospital mortality when added to other indicators of shock including, systolic blood pressure, LV ejection fraction and peripheral hypoperfusion [19]. Similar to patients presenting with ACS, those with admitted to the ICU with acute decompensated heart failure (ADHF) can be risk stratified by determining the lactate on admission. In a study of 754 consecutive patients with CS-ADHF, the admission lactate had a greater power to predict in-hospital mortality with a twofold risk, especially in those with levels greater than 3.2 mmol/L [20]. Others have also shown that even in the absence of shock, patients with heart failure related to AMI, there is a 28% thirty day mortality when lactate is greater than 2.5 mmol/L [21]. It is recommended that lactate measurements should be obtain every 1–4 h and that repeated assessments can inform about persistence of shock. Absence of lactate clearance from blood is associated with a poor prognosis, as studies have shown that a clearance of less than 10% in 12 h from admission identifies a high-risk subset of patients for death [22]. Additionally, determining the level of bicarbonate at admission has been associated with a high mortality risk at short and long term follow up. In a study of 165 ischemic patients admitted with cardiogenic shock, those with in the lowest tertile of bicarbonate levels had a 15.5 (IQR 12.8–16.6) were associated with a twofold risk for 1 year mortality [23].
- **Troponin**. Cardiac troponin beyond its diagnostic power for detecting AMI, has been determined to be a successful tool in predicting mortality. The degree of troponin elevation can determine outcomes in patients presenting with CS-AMI. In the Global Registry of Acute Coronary Events, the maximum 24-h troponin (either I or T) presenting with non-ST segment elevation MI (NSTEMI) was analyzed in 16,318 patients. For each ten-fold increase in the baseline value, there was a significant linear trend for worse outcomes including ventricular arrhythmias, cardiogenic shock, new onset heart failure and death. The degree of troponin elevation was found to be a strong predictor for early and late mortality [24]. Furthermore, in patients that continue to have elevated circulating

troponin levels over the first 30 days following a hospitalization, it suggests ongoing myocardial injury associated with chronic remodeling and risk for all-cause mortality [25].

Echocardiography

Echocardiography in the acute setting can be beneficial in differentiating the causes of cardiogenic shock. A focused echocardiogram should be done in the initial evaluation of CS patients as it provides vital information about LV and RV contraction, intravascular fluid status, presence of pericardial effusion and tamponade. In those presenting with AMI, detecting mechanical complications is of sum importance to dictate the opportune therapies for stabilization. In other cases of CS, it help assess left ventricular function, right ventricular function and acute valvular heart disease. In the SHOCK trial, mechanical complications accounted for 12% of the causes of CS with severe valvular heart disease being the most common one (predominantly moderate mitral regurgitation), followed by ventricular septal rupture and tamponade. Moreover, in CS patients presenting with moderate MR, there is a 6 to sevenfold risk of 30-day mortality [4, 26]. However, in recent years the mortality related to mechanical complications in ST segment elevation MI (STEMI) patients have decreased to almost 25%, with free wall rupture representing now the most common complication, requiring pericardiocentesis due to cardiac tamponade with hemodynamic compromise [27].

In cases of cardiogenic shock secondary to acute heart failure (CS-AHF), distinct echocardiographic markers have been found to provide additional information to stratify patients at risk of worsening shock and poor prognosis. Studies have shown that a reduced ejection fraction, high wall motion score index, elevated E/e' ratio>13 m/s, moderate to severe mitral regurgitation, presence of LV outflow obstruction, elevated pulmonary systolic pressure and right ventricular involvement are associated with increase in hospital mortality [28]. Early recognition of these high-risk individuals can rapidly triage which patients need to escalate their hemodynamic support with either intravenous inotropic drugs and/or mechanical circulatory support (MCS). Furthermore, once hemodynamic stabilization occurs, daily echocardiograms at the bedside can determine myocardial recovery or persistent systolic dysfunction, myocardial complications post-AMI and short term MCS device adjustment.

Hemodynamic Monitoring

Urgent assessment of signs of hypoperfusion in all patients with CS is recommended by obtaining continuous blood pressure monitoring through an arterial line, telemetry for heart rate and arrhythmia evaluation, continuous pulse

oximetry for oxygen saturation, temperature and urine output. Additionally, pulse pressure should be closely monitored with a goal SBP ≥ 90 mmHg and MAP 60–65 mmHg. Central venous catheter insertion should also be obtained to administer vasopressors or inotropes, monitor CVP and mixed central venous oxygen saturation.

The use of invasive hemodynamic through a pulmonary artery catheter (PAC) is critical for establishing the diagnosis of cardiogenic shock. Determining the cardiac index and filling pressures ascertains the category and severity of shock and risks stratify patients. It can also provide information about the fluid status, adequate oxygen delivery as determined by the mixed venous oxygen saturation (SVO2) and pulmonary vascular resistance. The PAC can also distinguish cardiogenic vs. mixed shock as the latter can be seen in 20% of CS cases.

Although PAC utilization in CS has decreased over the past decade, studies have shown that its use is associated with corrections in reclassification of CS, improved outcomes and increased survival. The goal of hemodynamic monitoring is directed towards improving tissue perfusion through stabilization or enhancing parameters that will make a significant impact on outcomes. It should not only focus on improving cardiac function but also reducing filling pressures. A sub-analysis from the CardSHock study investigating the use of PAC in a real-world setting showed that those managed by PAC received more often inotropes, vasopressors, mechanical ventilation, renal replacement therapy and mechanical assist devices. The cardiac index, cardiac power output index and stroke volume index where the highest predictors for 30-day mortality allowing for reclassification of CS patients [29]. This is partly due to better decision strategies to guide therapy based on the hemodynamic data obtained [30].

The PAC can assist in choosing which vasopressor or inotropic drug to initiate and titrate, select which patient will benefit from acute MCS insertion for isolated LV, isolated RV or biventricular support and guide weaning of pharmacological or mechanical support. This is of importance as response to any intervention is dependent on volume status, intrinsic RV function, systemic and vascular resistances, and presence of valvulopathy.

A multitude of hemodynamic parameters can be obtained by PAC measurement which the clinician can integrate into their decision making:

	Mean	Range
Right Atrium, mmHg	4	−1 to 8
Right Ventricle Systolic, mmHg	24	15 to 28
Right Ventricle End Diastolic, mmHg	4	0 to 8
Pulmonary Artery Systolic, mmHg	24	15 to 28
Pulmonary Artery Diastolic, mmHg	10	5 to 16
Pulmonary Artery Mean, mmHg	16	10–22
Pulmonary Capillary Wedge, mmHg	9	6 to 15

	Mean	Range
Cardiac Output, mL/min	6	4 to 8
Cardiac Index, mL/min/m^2	3.4	2.8 to 4.2
Systemic Vascular Resistance	14.4 (1150)	11.3 to 17.5 (900 to 1400)
Pulmonary Vascular Resistance	2.5 (200)	1.9 to 3.1 (150 to 250)
Transpulmonary Gradient	<12 mmHg	PAP mean—PCWP mean
Diastolic Pulmonary Gradient	<7 mmHg	PAP diastolic—PCPWP mean

The PAC can also assess if there is RV involvement in CS. Right ventricular dysfunction (RVD) can be defined by readily available hemodynamic parameters obtained by PAC which include:

1. Right atrial pressure (RAP) >10 mmHg
2. Right atrial to pulmonary capillary wedge ratio >0.63
3. Pulmonary artery pulsatility index (PAPi) <2. This parameter represents the ratio of PA pulse pressure to RAP calculated as: pulmonary artery systolic pressure—pulmonary artery diastolic pressure/right atrial pressure
4. Right ventricular stroke work index <450 g-m/m^2, determined by mean PA pressure—mean RAP x stroke volume index

Recognizing markers of RVD is important as 23–24% of CS-AMI present with RVD (CVP > 10 mmHg), while 15% present with severe RVD (CVP > 15 mmHg). Even more, biventricular failure (represented by elevated CVP > 15 mmHg and PCWP > 15 mmHg) is the most common hemodynamic profile occurring in 38% of patients which is associated with poor prognosis and not uncommonly requiring biventricular mechanical support [31].

Other important hemodynamic parameters that have proven to be significant prognosticators in CS are the cardiac power output (CPO) and cardiac power index (CPI) is derived from obtaining the cardiac output and mean arterial pressure. The CPO is calculated as CO x MAP/451. A CPO <0.6 W/m^2 which been associated with increased 30 day in-hospital mortality in patients with CS at 24 h after CS diagnosis and implementing supportive therapies [32, 33].

Since PAC is an invasive procedure, its insertion should be guided with caution as complications can occur in 5% of the cases including: insertion site hematoma, arterial puncture, pulmonary artery hemorrhage, pulmonary artery puncture, arrhythmias catheter related blood stream infections and endocarditis.

Hemodynamic Risk Profiling

The SCAI stages serves as a robust indicator for profiling CS patients based on their initial presentation (Table 1). With each incremental stage there is a 1.53 to 6.8-fold increase in-hospital mortality risk [34]. Among those with ongoing hypoperfusion and deterioration based on presence of hemodynamic indicators of

biventricular failure (high RAP:PCPW ratio, low CPO, low PAPi), requiring multiple vasopressors for ongoing support, are at highest risk for becoming refractory to therapy and at greatest need for MCS. The in-hospital mortality for those in refractory shock can range from 40 to 67% [35]. Thus, early recognition and rapid progression of the severity of CS is critical for survival and improved outcomes.

Hemodynamic Goal Directed Therapy

Initial evaluation of invasive hemodynamics during the acute phase of shock can serve to identify and institute adequate support measures for stabilization. The initial measurements of cardiac index, pulmonary capillary wedge pressure, pulmonary artery oxygen saturation, pulmonary artery pulsatility index can assist clinicians in determining which therapies provide the maximum benefit. Studies have shown that when interventions are started on early hours of CS, survival outcomes improve. In patients with CS-AMI requiring MCS in the first 12–24 h of presentation, a CPO >0.6 W and lactate <4 mg/dL show a 95% in-hospital survival to discharge compared to those with a CPO < 0.6 W and lactate >4 mg/dL who have a predicted 30% survival. Additionally, once MCS is initiated more than 50% of patients reduce the number of inotropes, improve cardiac performance measures, oxygenation, lactate and achieve a lower heart rate. Establishing shock protocols emphasizes standard practices that can promptly identify patients in need of early MCS.

Even though macro-circulatory changes can be seen with prompt fluid resuscitation, micro-circulatory dysfunction can persist signaling poor perfusion pressure. Correction of flow alterations occurring at tissue level is critical as impaired endothelial vasoreactivity, reduced blood cell rheology, platelet aggregation and micro-thrombosis can accelerate organ failure and make all efforts of MCS futile. Optimization of oxygen transport based $ScvO_2$, lactate, veno-arterial difference in CO_2 and sublingual microcirculatory flow by administration of fluids, red blood cell transfusions, and inotropes is in parallel important to MCS initiation [36].

Establishing Weaning Versus Dependence

One of the overarching goals of every shock patient should be to achieve myocardial recovery and survival to discharge. Daily assessments are required to evaluate underlying cardiac function, hemodynamic changes, biomarker trend and vasopressor requirements. The later has been proven to be a marker of poor prognosis when the number of vasopressors or inotropes escalates rapidly. Indeed, patients who required more than 2 inotropes have a 65% 30-day mortality risk compared to those with one or none vasopressors. By assessing hemodynamic trends, the clinician can rapidly identify if escalation or de-escalation of support is warranted. Several observational studies and inherent institutional protocols have been established to dictate when a patient can be weaned off support. These include:

1. Cardiac index ≥ 2.2 L/min/m^2
2. Cardiac power output > 0.6 W
3. PCWP ≤ 18 mmHg
4. PAPi ≥ 1.5
5. MAP ≥ 65 mmHg
6. CVP ≤ 15 mmHg
7. Heart Rate < 120 bpm
8. LVEF $\geq 25\%$
9. TAPSE > 14 mm

If such recovery parameters are not met then consideration for increasing hemodynamic support should be considered with either a short-term MCS (impella, intra-aortic balloon pump, VA-ECMO). If such weaning trials are occurring while on MCS then evaluation for advanced therapies are to be sought including durable left ventricular assist device or heart transplantation.

Timing of Percutaneous Mechanical Circulatory Support

The initial management strategies to stabilize CS includes IV fluids, inotropes and vasopressors, however about 8% of patients evolve into progressive or refractory shock with an expected mortality of ~70%. Moreover, mortality increases rapidly with the number of vasoactive drugs use with only 35% survival when 2 or more inotropes are used and are associated with increase myocardial oxygen consumption, increase afterload and vasoconstriction that may impair microcirculation [37]. In these stages aggressive interventions are needed to stop the accelerated pace of shock. Short-term MCS inserted either percutaneously or surgically can be used as a bridge to myocardial recovery, bridge to decision when neurological function is unclear or multi-organ failure may preclude a decision for advanced heart failure therapies including LVAD or heart transplant; or as bridge to another durable device. The advantage of short-term MCS is to allow hemodynamic optimization and potential reversal of end-organ dysfunction before moving forward with other therapies or palliative care.

It is important then to recognize the initial insult leading to CS and understand the underlying myocardial reserve to withstand circulatory collapse. The primary objective of managing CS patients is to achieve coronary perfusion via revascularization when needed, achieve circulatory support to preserve a viable mean blood pressure and unload the left and/or right ventricle to reduce the deleterious effects of increase afterload and oxygen demand.

The 2015 SCAI statement on the use of percutaneous MCS recommends implementing early placement of approved MCS devices in those who failed to stabilize with initial support. Prompt ventricular unloading enhances myocardial performance and reduces mechanical power expenditure by: (1) lowering PCWC; (2) minimizing myocardial wall stress and ventricular work; (3) reducing myocardial oxygen demand; (4) augmenting coronary perfusion. Studies have shown that early MCS implementation with the impella device is associated with better

survival specially in those when MCS is implemented less than 75 min from shock onset. In a study of 287 patients presenting with CS-AMI who underwent percutaneous coronary intervention with a mean LVEF of 25%, only 44 survive to discharge. Time to MCS was associated with improved survival before PCI or requiring inotropes and vasopressors [37].

Although observational and registry data suggest that early initiation of MCS favors good outcomes, appropriate patient selection including patient age, comorbidities, hemodynamic and laboratory values institutional experience and device related complications are key elements that have to be taken into account when consider MCS.

Shock Team Approach

Our current understanding of CS has evolved over the past decade with attention being focused towards preservation of end organ perfusion while minimizing adverse events when patients are supported on conventional therapy. The key to improve outcomes in CS is to stablish a pattern of early recognition markers of CS to allocate appropriate therapies. The success of door-to balloon time in STEMI has been in large part due to training of emergency personnel to detect clinical, ECG, and laboratory criteria of acute ischemia due to coronary occlusion. A similar approach should be boarded for early triage of patients and avoid delaying evaluation and management of CS patients. Cardiac shock centers have demonstrated improved outcomes when care pathways are established and followed based on current best practices standards. When a standardized approach is use survival from CS can improve dramatically. In a study of 204 patients, from the INOVA group from a task force to develop a management protocol for CS patients. The algorithm approach focused on 5 objectives:

1. Rapid identification of the CS state
2. Early invasive hemodynamic implementation
3. Minimize use of vasopressors and inotropes
4. Early MCS implant for the left and/or right ventricle
5. Assess and achieve myocardial recovery

The authors noted that after implementing the shock team approach the survival increased from 47% for CS-AMI and CS_ADHF to 58 and 77%. The most common cause of death was multiorgan failure in 80% of the patients. Those who required MCS for every 1-h delay in escalation to MCS was associated with a 10% increase risk of death. Overall, the complexity of CS etiologies requires a multi-disciplinary team approach with the clinical skills, hemodynamic expertise and technical skills for percutaneous MCS insertion and management. In tertiary shock care centers, the team is mostly conformed of interventional cardiologist, advanced heart failure specialist, nephrologist, critical care specialist, cardiac surgeon, palliative care, neurologist, pharmacist. A proposed algorithm based on current scientific statement for CS management (Fig. 1).

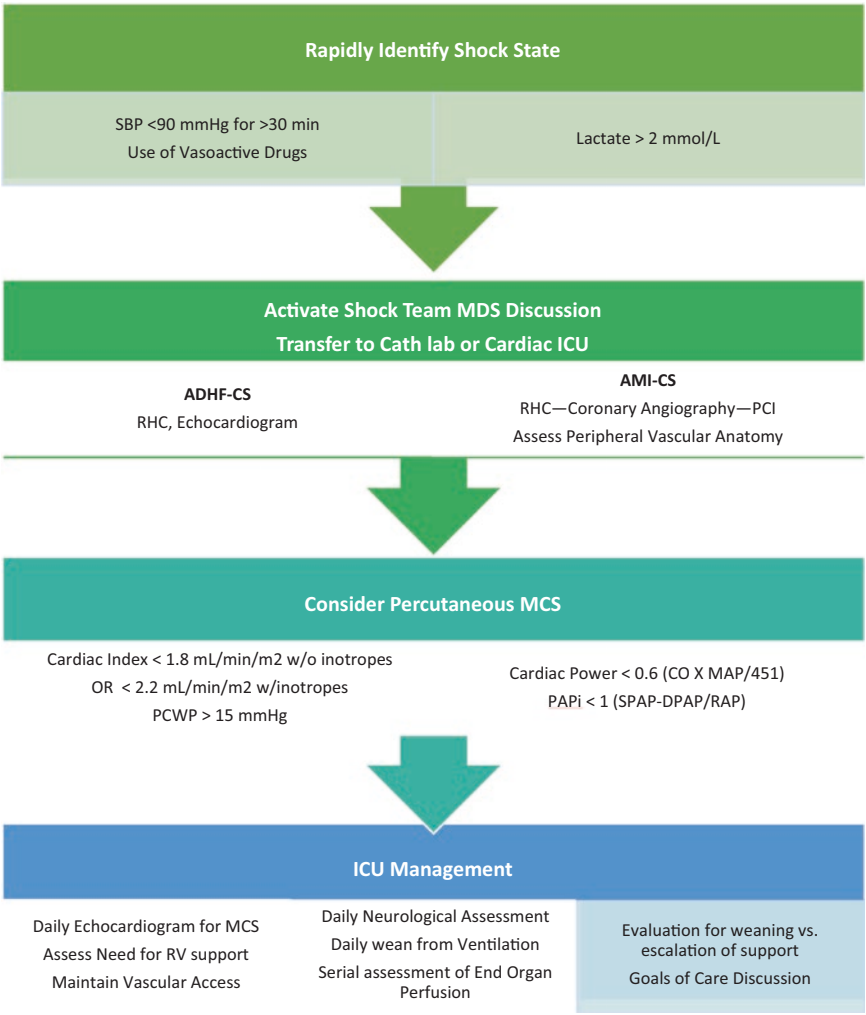

Fig. 1 Cardiogenic shock management algorithm

Key Points

1. Identify Type and severity of Cardiogenic Shock: ACS vs non-ACS
2. Use hemodynamic data to guide clinical decision making
3. Use Vasoactive Drugs to maintain MAP > 65 mmHg
4. Trend hemodynamic and biomarker data (CPO, PAPi, lactate, CO2, creatinine)
5. Expedite Early Ventricular Unloading with MCS and Select type
6. Enhance Coronary perfusion

7. Preserve Renal and Hepatic Function
8. Maintain Vascular access
9. Achieve recovery and survival
10. Refractory Shock = Escalation to MCS

Case Conclusion

After unsuccessful improvement in the patient's hemodynamic, clinical and perfusion status, a decision is made to start mechanical circulatory support with notable improvement in atrial and ventricular filling pressures, cardiac index and lactate. Weeks after maintaining stabilization with MCS the patient underwent successful heart transplantation without complications:

	Inotrope	24 hours post-MCS
RA, mmHg	11	8
PA, mmHg	55/33/39	44/20/29
PCWP, mmHg	21	17
PA Saturation, %	47	58
AO Saturation, %	99	100
Cardiac Output, L/min	2.3/1.8	2.9/2.2
Cardiac Index, L/min/m^2	1.8	2.2
SVR, dyn/cm/sec^5	1600	2041
PVR, Woods unit	237	248
RA:PCWP ratio	0.52	0.47
PAPi ratio	2	3
CPO, watts	0.41	0.51
BP, mmHg	106/67/80	96/44/79
HR, bpm	132	88
Lactate mmol/L	2.4	1.6

Conclusion

Cardiogenic shock is complex syndrome that requires a multidisciplinary approach to improve outcomes. The current SCAI classification can allow for proper differentiation of CS subsets and determine the hemodynamic profile. The advantage of utilizing PAC hemodynamic guided therapy can confirm eh presence and severity of CS where the cold and wet is the most frequent CS phenotype.

The use of vasopressors and inotrope for initial stabilization of CS patients is beneficial, however the longer duration on these vasoactive drugs is counterbalanced by their negative side effects. Trending arterial lactate is helpful in prognosticating and identifying refractory CS. The early recognition of high-risk CS patients will allow for prompt implementation of MCS to improve cardiac while avoiding the cardiotoxic effect of vasopressors. Similarly, those patients that fail to achieve myocardial recovery should be considered for long term durable MCS.

Future Direction

The Shock team approach has been popularized in tertiary centers and has quickly been adopted by many hospital systems. The early mobilization of a multidisciplinary team to address medical and surgical needs of the patient may prove to be cost-effective and timely. Early recognition of cardiogenic shock as well has been the center of discussion with artificial intelligence embedded in electronic medical record systems. These ubiquitous systems actively collect continuous variables to alert practitioners by the use of best practice advisories.

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Temporary Mechanical Circulatory Support

Daniel Walters and Ryan Reeves

Clinical Vignette 1

A 55 year-old man with a past medical history notable for HIV infection and AIDS, tobacco abuse, coronary artery disease, and a prior percutaneous coronary intervention (PCI) to an unknown vessel presented with acute chest pain and anterior ST-segment elevations. He was hypotensive with a blood pressure of 85/52, tachycardic with a heart rate of 112 in sinus rhythm, and demonstrated crackles on pulmonary auscultation. Emergent angiography demonstrated left anterior descending artery stent thrombosis. Successful angioplasty and stenting were performed, however he remained persistently hypotensive and required norepinephrine for blood pressure support. Subsequently, a 50 mL IABP was placed from the right femoral artery. He was brought to the cardiac care unit, where over the next 48 h his condition improved. The IABP was weaned and removed on hospital day three with manual pressure for hemostasis, and discharged to home on hospital day five (Tables 1, 2, 3).

Introduction: IABP

The IABP was the first widely available non-pharmacologic modality that could alter cardiovascular hemodynamics and for decades was the standard therapeutic device for percutaneous MCS [1]. It continues to be the most widely used system

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Table 1 SCAI/ACC/HFSA/STS consensus statement summary

Suggested indications for percutaneous mechanical circulatory support
• Complications of acute myocardial infarction
• Severe heart failure in the setting of non-ischemic cardiomyopathy
• Acute cardiac allograft failure
• Post-transplant right ventricular failure
• Patients slow to wean from cardiopulmonary bypass following heart surgery
• Refractory arrhythmias
• Prophylactic use for high risk percutaneous coronary intervention*
• High-risk or complex ablation of ventricular tachycardia
• High-risk percutaneous valve interventions

*HR-PCI encompass those age 70, ongoing ischemic and LV systolic dysfunction EF < 40%, previous CABG, acute coronary syndromes complicated by unstable hemodynamics (wedge pressure ≥ 15 mmHg, mean pulmonary arterial pressure ≥ 50 mmHg), post-AMI angina, Killip class III-IV and CS

with approximately 50,000 per year being implanted for cardiogenic shock alone [2]. Indications for use include the following: acute or chronic cardiogenic shock, decompensated congestive heart failure refractory to medical therapy, acute myocardial infarction (AMI), critical left main or three vessel coronary artery disease, adjunctive support for high risk/complex PCI, and refractory arrhythmia [3, 4]. Introduced through the peripheral vasculature, the IABP is advanced over a guidewire to the proximal descending thoracic aorta, just distal to the great vessels. The hemodynamic effects of counterpulsation include: increased diastolic pressure and coronary perfusion, decreased afterload, increased stroke volume, and decreased stroke work and myocardial consumption, which lead to an improvement in cardiac output (0.5–1.5 L/min) and metabolic clearance of lactate [3, 5–7]. The hemodynamic benefits are dependent on balloon position, presence of cardiac arrhythmias and tachycardia, timing of balloon inflation, and systemic vascular resistance. Systemic anticoagulation may reduce device-associated thrombosis, and is recommended. If ongoing bleeding precludes anticoagulation, a systole to balloon inflation ratio of 1:1 is recommended to reduce stasis and the potential for thrombosis.

IABP and Acute Myocardial Infarction

Initial reports demonstrated the benefits of the IABP in AMI complicated by cardiogenic shock, with a significant reduction of in-hospital mortality, however, patients receiving IABP were younger, more often received inotropic support, and were more aggressively treated with coronary angioplasty and bypass surgery [8–10]. This early experience, although derived from a sub-analysis of