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Cardiogenic Shock in the Canadian Landscape: Key Concepts for the Practicing Clinician

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Introduction

Cardiogenic shock (CS) is generally defined as a state of end-organ hypoperfusion secondary to an inability of the heart to deliver sufficient oxygenated blood to the tissues.[1] Although CS is often initiated by an event that specifically affects the cardiovascular system, without prompt intervention, it can lead to a cascade of insults on other organ systems that result in additional morbidity and mortality. Despite advances in temporary mechanical circulatory support (MCS) technology over the past 2 decades, studies have consistently reported a 30% to 50% mortality rate for patients with CS at 6 to 12 months, though this rate may exceed 70% depending on the severity of the shock and individual patient factors.[2] This review will provide an overview of key concepts in CS including current definitions, hemodynamic assessment, shock state classifications, and prognostication.

Etiology of Cardiogenic Shock

In contemporary cardiac intensive care units, several conditions can lead to CS. Acute myocardial infarction (AMI), complicated by CS (AMICS), accounts for approximately 30% of shock cases, while the incidence of CS complicating AMI has been reported to be between 7% to 10%.[3] Non-ischemic cardiomyopathies are responsible for 28% of cases, ischemia without AMI comprises 18%, and various other causes (valve dysfunction, arrhythmia, among others) are responsible for 17%. The remaining 7% of cases are reported as unknown or missing.[4]

Definitions of Cardiogenic Shock

CS is a clinical diagnosis, however, criteria have been proposed to standardize its definition.

The landmark SHOCK trial defined CS based on 3 variables:

1. Hypotension (a systolic blood pressure of <90 mm Hg for at least 30 minutes or the need for supportive measures to maintain a systolic blood pressure of >90 mm Hg
2. Evidence of end-organ hypoperfusion (cool extremities or a urine output of <30 mL per hour)
3. Impaired cardiac hemodynamics, defined as a cardiac index (CI) of less than 2.2 litres per minute per square metre of body-surface area, and a pulmonary artery wedge pressure (PAWP) of at least 15 mm Hg.[3]

Although subsequent studies have introduced slight variations to these criteria, the overall concepts have remained consistent. Definitions of hypoperfusion have been broadened to include an elevated arterial lactate level (>2 mmol/L), acute kidney injury (creatinine ≥2 times the upper limit of normal), acute hepatic injury (ALT >3 times the upper limit of normal), cool or mottled extremities, or altered mental status not explained by an alternate cause. Similarly, hemodynamic criteria have been broadened to include a systemic vascular resistance index (SVRI) >2200 dynes/(cm·sec-5).[2]

Key Hemodynamic Indices in Cardiogenic Shock

The altered hemodynamics in CS are often best assessed by right heart catheterization. This procedure can provide critical information in the initial assessment of CS and in monitoring the response to therapy. In addition to the indices

Hemodynamic Index	Formula	Normal Value
CPO	$\frac{(Cardiac\ output)(Mean\ arterial\ pressure)}{451}$	>0.6 Watts[5]
PAPi	$\frac{(PASP - PADP)}{RAP}$	>1.85[33]
RVSWi	$(mPAP - RAP)(SVi)(0.0136)$	8-12 g/m/beat/m^{2}[,34]
RAP:PAWP	$\frac{RAP}{PAWP}$	0.43-0.75[35]

Table 1. Key hemodynamic indices in shock evaluation; *courtesy of Ayaaz K. Sachedina, MD, FRCPC and Jordan D. Gibson, MD, FRCPC*

Abbreviations: CPO: cardiac power output, **mPAP:** mean pulmonary artery pressure, **PADP:** pulmonary artery diastolic pressure, **PAWP:** pulmonary artery wedge pressure, **PASP:** pulmonary artery systolic pressure, **PAPi:** pulmonary artery pulsatility index, **RAP:** right atrial pressure, **RVSWi:** right ventricular stroke work index, **SVi:** stroke volume index

mentioned above for defining CS, other important parameters include cardiac power output (CPO), pulmonary artery pulsatility index (PAPi), and right atrial to pulmonary artery wedge pressure ratio (RA:PAWP) **(Table 1)**.

CPO is a measure that considers both cardiac output and the ability of the heart to generate systemic flow and blood pressure. A CPO cutoff of less than 0.6 W has been shown to have the best sensitivity and specificity for predicting worsening heart failure in patients admitted with CS.[5]

In addition to assessing left ventricular (LV) function and filling pressures, right heart catheterization is a powerful tool for assessing right ventricular (RV) function. The PAPi is used to assess right heart function, with lower values suggesting right heart dysfunction.[6] Multiple studies have shown an increase in adverse outcomes, including all-cause mortality, in a variety of patient populations with a low PAPi.[6–8] Although there is no universally agreed upon PAPi value to identify "high-risk" individuals, a recent study found that hospitalized patients in the lowest 3 quartiles undergoing right heart catheterization had increased mortality compared with those in the highest PAPi quartile, suggesting that PAPi may play a role in modelling risk across a range of cardiac presentations. Other helpful RV hemodynamic indices include the RA:PAWP and the right ventricular stroke work index (RVSWi) **(Table 1)**. An elevated RA:PAWP value is also associated with right heart dysfunction. While an elevated RA:PAWP appears to be associated with an increase in mortality, the RVSWi may be less predictive of outcomes than the PAPi and RA:PAWP.[6]

Finally, while not widely used in clinical practice for prognosticating shock, evidence suggests that microvascular perfusion parameters may be associated with adverse outcomes in CS.[9,10] This topic is the subject of much ongoing research and holds promise for improving prognostication and serving as a potential future therapeutic target.

Appropriate hemodynamic assessment and identification of impaired cardiac function with univentricular or biventricular involvement is imperative for determining the appropriate treatment strategies. Selecting a specific medical therapy and MCS strategy that is directed to the area and degree of cardiac impairment facilitates more effective and supportive intervention.

Classification of Cardiogenic Shock

A key concept in the management and ongoing research of CS is its inherent heterogeneity, both in severity and underlying etiology. The most widely used classification in contemporary practice and research is The Society for Cardiovascular Angiography and Interventions (SCAI) classification of shock. This scheme **(Figure 1)** was first proposed in 2019 in an effort to improve upon the Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) definition.[11] The SCAI shock classification is an ordinal scale that grades shock severity from A (at risk of CS) through to E

(in extremis). Numerous validation studies have shown that the SCAI classification consistently predicts increasing mortality from grades B to E.[12–14]

While the SCAI classification has been helpful for standardizing and stratifying shock severity, providing a more consistent description of patient populations in both clinical practice and research, limitations have been described.[15] These limitations include the lack of discrimination of shock severity from stages C to D, the lack of discrimination between LV, RV, and biventricular failure, and the absence of therapeutic guidance based on clinical presentation. An updated SCAI classification was recently proposed that incorporated additional modifiers into the SCAI shock classification to address these limitations.[16] In this scheme, it was suggested that SCAI stage C be further stratified as follows: hypoperfusion with normal blood pressure, hypoperfusion with hypotension or 1 vasopressor, or hypoperfusion with hypotension and >1 vasopressor. Within each strata, it was further suggested to add a modifier to identify LV failure, RV failure, or biventricular failure. For defining SCAI stages D and E, specific cutoffs are also suggested for lactate levels and for the number of vasopressors to add granularity, thereby defining these as discrete states on a continuum of CS severity.[15]

The etiology of CS contributes another layer of heterogeneity to this patient population. The Shock Academic Research Consortium (SHARC) recently proposed a framework for classifying the underlying causes of CS, which will improve discrimination of CS phenotypes in future research studies **(Figure 1)**. They suggested classifying shock into several types: acute myocardial infarction-related CS (AMI-CS) with or without ST-segment elevation; heart failure-related CS (HF-CS), which can be further classified as de novo or acute-on-chronic; secondary CS (from another, non-myocardial cause); and post-cardiotomy CS (in the setting of cardiac surgery), which can be further classified by surgery type. In the future, machine learning approaches may also play a role in further stratifying phenotypes of CS.[17]

Prognosis and Outcomes of CS States

The SCAI shock classification has been shown to be a helpful prognosticator of mortality during the initial assessments and reassessments of patients with CS. Early validation studies of the SCAI shock classification reported in hospital or 30-day mortality rates of 33.9% for SCAI B, 10.7-53.9% for SCAI C, 24-66.9% for SCAI D, and 42.0-77.4% for SCAI E, respectively.[12,18–20] In recent years, there has been a focus on the utility of conducting serial assessments of the SCAI shock classification. This approach recognizes the dynamic trajectory of patients with CS, however, most early validation studies assigned a shock class at the time of admission. In a recent study,

A	At risk for CS without signs or symptoms	**AMI-CS**	Shock in the setting of ACS (may be NSTEMI or STEMI).
B	Hypotension and/or tachycardia without hypoperfusion		
C	Hypoperfusion requiring pharmacologic or mechanical support	**HF-CS**	Shock secondary to right, left or biventricular failure in the absence of ACS. May be de novo or acute on chronic.
D	Clinical condition worsening despite efforts to support	**Secondary CS**	Due to another non-myocardial cardiac cause (arrhythmic or valve disease).
E	Patient in extremis and may have ongoing CPR	**Post-cardiotomy CS**	Following cardiac surgery.

Figure 1. Classification of cardiogenic shock. a) SCAI classification of CS severity b) SHARC classification of CS phenotypes; *courtesy of Ayaaz K. Sachedina, MD, FRCPC and Jordan D. Gibson, MD, FRCPC*

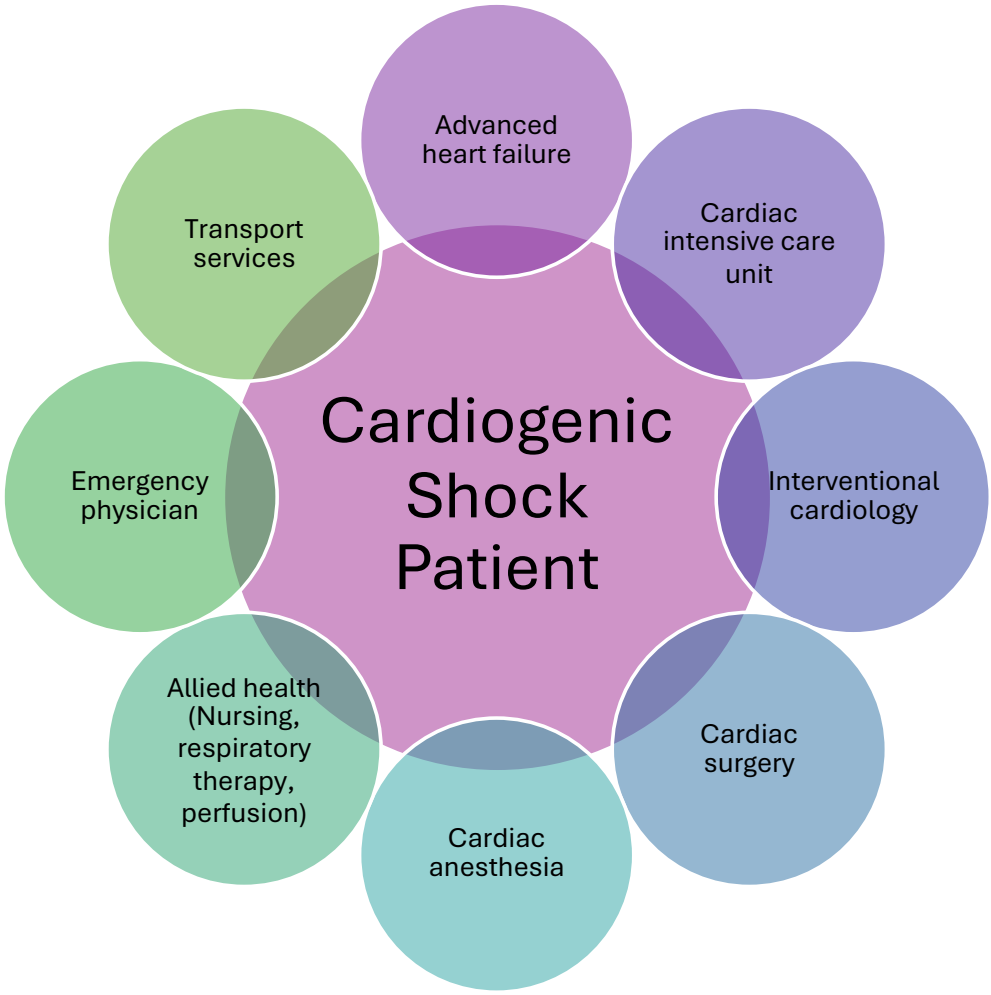

Figure 2. Multidisciplinary team required for management of a patient with cardiogenic shock; *courtesy of Ayaaz K. Sachedina, MD, FRCPC and Jordan D. Gibson, MD, FRCPC*

retrospectively assessing shock severity every 4 hours over the first day of a cardiac intensive care unit admission in a population with and without shock, improved the prognostication accuracy.[21] It has also been shown that a single re-evaluation of the SCAI classification at 24 hours, whether it is improving or worsening, along with the maximum SCAI class assigned over a patient's course, offers additional prognostic information.[22–24]

The long-term outcomes amongst survivors of CS are highly variable. While some patients recover with minimal support, a recent report using data from the United States National Inpatient Sample of 332120 patients identified with CS, 5710 (1.7%) went on to receive an LV assist device, or cardiac transplant during their index admission.[25] An awareness and understanding of these possible trajectories is essential in managing patients with CS. The early involvement of advanced heart failure specialists in the care of patients with CS can help identify those who are not showing signs of independent recovery and optimize their candidacy for long-term advanced therapies or transplant.

The Role of Shock Teams

The first 24 hours following a patient's admission for CS are critical for their outcome. Early activation of multidisciplinary specialists through shock team models has been shown to improve outcomes in CS **(Figure 2)**.[26,27] Leveraging the expertise of a diverse group of specialists facilitates the optimization and standardization of clinical practices, which historically have varied

amongst different centres depending on the volume of CS that they treat.[28]

Involving clinicians with different areas of expertise early in the management of a patient, and facilitating their transfer to a specialized cardiac centre, can improve outcomes. Several studies have independently shown improvements in patient survival with the involvement of a CS team.[26,28] The involvement of CS teams can also facilitate the earlier deployment of advanced MCS and increase the use of pulmonary artery catheters. This approach helps to better identify the etiology of a patient's shock state and guide subsequent treatment. Given the heterogeneity of CS, it is important to recognize that a "one-size-fits-all" approach to it does not exist and that future research should strive to consider shock phenotypes when assessing responses to specific therapies.[29,30] While many advanced MCS options are available to support patients with CS, there is no evidence to date that suggests the superiority of one device over another, or over medical management, in all-comers with CS. However, when treatment strategies are guided by the patient's etiology and degree of hemodynamic impairment, there is a greater potential for more effective and directed therapy. Earlier involvement of a CS team during the patient's management course can help with these decisions.

Unique Features of Cardiogenic Shock Management in Canada

In Canada, the delivery of CS care has unique features, particularly when comparing it to care in the United States. Firstly, the geographic and per capita density of centres capable of offering advanced therapies and transplants is relatively low.[31,32] In Canada, there are 9 centres that offer the full spectrum of care for CS patients, including cardiac transplant.[31] Thus, access to advanced cardiac centres with full MCS options and heart transplant services can be challenging due to the limited number of centres offering these services and the large catchment areas for each centre.[31]

In Canada, there is also significant variability of mechanical support options available by centre. A recent survey of all Canadian centres with cardiac catheterization and revascularization capabilities (N=46) reported that intra-aortic balloon pumps (IABP) were available at all centres, however, percutaneous LV assist devices were available at only 39.1%, and extracorporeal membrane oxygenation (ECMO) was available at 65.2%. In a forthcoming report comparing shock management and outcomes between Canada and the United States, the use of pulmonary artery catheters and Impellas was significantly higher in the United States compared to Canada. The adjusted mortality for patients presenting with CS was also reported to be lower in the United States compared to Canada (29.4% vs 37.1%, $p = 0.0004$)[36]. Exploring and addressing the reasons for these differences will be important for future research.

Conclusion

Despite advancements in Cardiology over recent decades, mortality rates for CS remain high in Canada and globally. CS is a heterogeneous condition, and its management is further complicated by the unique and diverse treatment settings within the Canadian landscape of cardiovascular care. Moving forward, hospital centres will require ongoing efforts to define pathways to ensure prompt initiation and ongoing discussion of care for patients with CS. In addition, further evidence will be required to define the best therapeutic options for specific phenotypes of patients presenting with this heterogeneous condition.

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