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Hemodynamic monitoring in cardiogenic shock

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Abstract

Purpose of Review—Cardiogenic shock remains a major cause of mortality today. With recent advancements in invasive mechanical support strategies, reperfusion practice, and a new classification scheme being proposed for cardiogenic shock, an updated review of the latest hemodynamic monitoring techniques is important.

Recent Findings—Multiple recent studies have emerged supporting the use of pulmonary artery catheters in the cardiogenic shock population. Data likewise continues to emerge on the use of echocardiography and biomarker measurement in the care of these patients.

Summary—The integration of multiple forms of hemodynamic monitoring, spanning non-invasive and invasive modalities, is important in the diagnosis, staging, initial treatment, and subsequent management of the cardiogenic shock patient.

Keywords

Cardiogenic shock; hemodynamic monitoring; echocardiography; pulmonary artery catheterization

INTRODUCTION

Mortality from cardiogenic shock remains elevated, ranging from 40–67% among the most severe of cases [1], despite advances in recent years in invasive mechanical support and reperfusion practice. In effort to improve disease state stratification, recent guidelines endorsed by a multitude of medical societies have been released. These guidelines, proposed by the Society for Cardiovascular Angiography and Intervention (SCAI), outline a new classification scheme for the stages of cardiogenic shock (Table 1) [2**]. Sequential hemodynamic monitoring is essential for the accurate diagnosis, staging, risk stratification, and management of cardiogenic shock. There is not a single monitoring approach for all patients. Various forms of monitoring allow the identification of patients requiring medical therapy and/or invasive mechanical support, and to gauge the response to therapy. This review focuses on publications arising within the last two years as discovered using PubMed and Google Scholar search engines.

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Conflicts of Interest: none

FORMS OF HEMODYNAMIC MONITORING

Forms of hemodynamic monitoring that continue to show promise for the management of cardiogenic shock include peripheral arterial catheterization, pulmonary artery catheterization, biomarker measurement, and serial echocardiography. A recent observational study integrating these various forms of hemodynamic monitoring with a multidisciplinary diagnostic and therapeutic team (SHOCK Team) approach showed improvement in 30-day survival rates from 47% pre-intervention to 58% and 77% in the two subsequent years post-implementation [3]. Use of advanced monitoring techniques should complement, and not replace, targeted clinical examination of other markers of perfusion and cardiac function, such as level of consciousness, respiratory effort, lung sounds, bowel sounds, capillary refill time, urine output, edema, and skin temperature.

Peripheral Arterial Catheterization

Peripheral arterial catheterization is recommended to allow for continuous monitoring of systolic blood pressure (SBP) and mean arterial pressure (MAP) [4**]. This facilitates easy and frequent titration of inotropes and/or vasopressors, which are commonly needed in cardiogenic shock patients with advanced disease. Of additional clinical importance, a recent analysis comparing invasive and non-invasive (auscultatory or oscillometric) approaches in cardiogenic shock patients showed that noninvasive methods tend to overestimate blood pressure during episodes of hypotension [5]. Thus, if arterial pressure values need to be accurately measured, invasive monitoring is recommended.

Blood pressure can vary with the stage of cardiogenic shock, but basic consensus definitions have long included hypotension (variably defined as SBP<90, MAP<60 or >30 mmHg less than baseline), accompanied by tissue hypoperfusion, as core components of the definition of cardiogenic shock [2**,4**]. A recent retrospective 1,002 patient analysis of cardiogenic shock patients found an inverse relationship between the mean MAP in the first 24 hours and hospital mortality, with significantly increased mortality in patients with a mean MAP <65 mmHg [6].

Right Heart & Pulmonary Artery Catheterization

Right heart catheterization with placement of a pulmonary artery catheter (PAC) facilitates invasive measurement of intracardiac pressures, performance of thermodilution cardiac output measures, and measurement of mixed-venous oxygen saturation. These data can be used to calculate cardiac index, cardiac power output/index (CPO/CPI) and pulmonary artery pulsatility index [7]. These measures interpreted together provide a robust assessment of left and right heart systolic performance. The data can be used to make diagnostic and treatment decisions, such as the initiation and discontinuation of appropriate mechanical support devices based on the degree and type of ventricular dysfunction [8,9**]. As an example, CPO ≤0.53 was the strongest predictor of in-hospital mortality in patients with acute myocardial infarction complicated by cardiogenic shock (AMICS), from the SHOCK registry [10]. The use of cardiac power output as a predictor of mortality has been shown to be valid in patients undergoing early mechanical support, and CPO was superior to systolic blood pressure measurements [11]. That finding was not universal, however, and data is

conflicting in a separate cohort from the Cardiogenic Shock Working Group registry, with a higher proportion of cardiogenic shock due to decompensated heart failure, where they found that CPO was not significantly predictive of mortality. They did, however, identify biventricular and right ventricular congestion as significant mortality predictors, and suggest further study of CPO in its application to different cohorts of cardiogenic shock patients [12]. Accordingly, CPO, though useful, may not replace systolic and mean arterial pressure-based classifications in assessing risk.

The hemodynamic presentation of cardiogenic shock can be variable, based on the degree of compensatory increases in systemic vasomotor tone, the underlying etiology, and any concomitant systemic inflammatory response syndrome. The data from the PAC can identify such mixed forms of shock and stratify a patient's position in the classic framework of "wet vs. dry" and "warm vs. cold," which guide the approach for vasopressors and volume management (Table 2). Unmasking mixed shock (patients in cardiogenic shock who have a prominent vasoplegic component) and those with normotensive cardiogenic shock (patients with hypoperfusion due to poor forward flow but with compensatory elevated vascular tone) are two important diagnoses facilitated by PAC placement [4**]. Furthermore, the use of serial hemodynamic measurements gauges response to therapy and can identify the need for escalation of support.

Routine use of pulmonary artery catheters in critical care medicine has been widely debated over the past few decades, secondary to neutral meta-analysis data on mortality and survival benefit for critically ill patients [13]. Additionally, the ESCAPE trial evaluated the effect of PAC for patients with severe symptomatic heart failure and yielded overall neutral results, with no clinical survival benefit and no excess mortality, despite a predictable increase in PAC-related adverse events [14]. Broad application of this trial to all cardiogenic shock patients, however, should be cautioned. The trial was designed to avoid inclusion of unstable patients who might require urgent PA catherization to guide management, including patients with previous inotrope requirement during the hospitalization or with severe acute kidney injuries. Despite the previous neutral data described, multiple societies and experts in their most recent statements continue to recommend PAC use in cardiogenic shock patients, particularly among complicated patient presentations [2**,4**,9**]. Additionally, recent international survey data has identified the use of a PAC in cardiogenic shock patients by a majority of practicing physicians [15]. This continues to be an area of evolving research.

A growing body of evidence in recent years has supported PAC use in cardiogenic shock patients. A recent multicenter retrospective review from the Cardiogenic Shock Working Group analyzed outcomes in 1,414 cardiogenic shock patients, stratified by SCAI stage and by the degree of PAC use (complete data, incomplete data, or no PAC data) prior to initiation of mechanical support [16*]. They found significant differences in mortality between PAC-use groups in the overall cohort as well as each SCAI stage. The patients with complete PAC assessment had the lowest in-hospital mortality across all SCAI stages. The findings were consistent with other published reports, including a single center retrospective study published in 2017 that found lower short and long-term mortality associated with PAC use, although only for the subset of cardiogenic shock patients without acute coronary syndrome [17]. This is in addition to other multi-center registry data published in 2019 showing an

association between PAC use and lower mortality and in-hospital cardiac arrest rates [18*]. Similar findings of improved mortality have been seen specifically for AMICS patients who underwent Impella™ placement, with and without PAC monitoring [19]. While these studies are not without limitations, they represent the strongest observational evidence in support of ongoing PAC use in the diagnosis and management of cardiogenic shock.

Echocardiography

Since echocardiographic assessment can occur immediately and is non-invasive, it should be performed urgently in the assessment of a possible cardiogenic shock patient [4**]. A basic assessment of left and right ventricular contractility can help support or refute the diagnosis of cardiogenic shock, as well as identify emergent life-threatening etiologies of shock such as cardiac tamponade. More detailed examination can assess right and left heart geometry, quantify diastolic dysfunction, reveal regional wall motion abnormalities (suggestive of coronary artery disease), acute or chronic valvular abnormalities, outflow obstruction, as well as mechanical complications such as septal or ventricular free wall rupture, papillary muscle rupture, or chordae tendineae rupture [20*,21]. Echocardiography can also be used to noninvasively estimate cardiac output, pulmonary artery systolic pressure and via calculation, systemic vascular resistance [22]. A recent large 5,453 patient retrospective database study showed that multiple echocardiographic parameters (including low stroke volume index and high E/e' ratio) correlated with SCAI stages and mortality, particularly among patients with less severe stages of shock [23*]. A recent randomized controlled trial evaluated the effect of serial miniature transesophageal echocardiography (TEE) on time to resolution of hemodynamic instability in ICU patients with undifferentiated circulatory shock [24*]. There was no difference in the resolution of hemodynamic instability at their primary endpoint of 6 days, but there was an improvement with TEE when the data was analyzed for the first 72 hours. This suggests a role for TEE but needs to be studied in the cardiogenic shock population exclusively and in the larger numbers to verify. Finally, a small retrospective study found that the ratio of corrected left ventricular ejection time to pulmonary artery wedge pressure independently predicted successful weaning from veno-arterial extracorporeal support [25]. These studies together provide important evidence supporting the ongoing use of echocardiography in the hemodynamic assessment of patients with cardiogenic shock.

Blood Chemistries

While no particular biomarker is diagnostic of cardiogenic shock, serial monitoring of several laboratory markers can support the diagnosis and monitor the progress of treatment. Although nonspecific, monitoring basic chemistries such as liver function tests, renal function tests, and lactate is recommended for an assessment of end organ perfusion [26]. A recent large sub-study of patients with AMICS identified arterial lactate measured at 8 hours (using a cutoff of 3.1) as the best predictor of mortality, superior to baseline measurement and calculation of lactate clearance [27*]. Another recent secondary analysis of cardiogenic shock patients found that lactate measurements at 6, 12, and 24 hours were predictors of 30-day mortality, and additionally that relative change in lactate in the first 24 hours predicts survival [28]. In a separate cohort of AMICS patients who underwent percutaneous coronary angiography and Impella™ placement, combining lactate levels with hemodynamic data

(lactate >4 or <4 and CPO >0.6 or <0.6) at 12–24 hours post-procedure was the best predictor of survival [11]. Troponin and brain natriuretic peptide are useful indicators for acute coronary syndrome and acute heart failure [2**], respectively, but their role specifically for serial hemodynamic monitoring of cardiogenic shock is less well-established. Many additional novel biomarkers continue to be studied, but none of these have entered the mainstream of cardiogenic shock management at the present time [29].

Other Noninvasive and Minimally-Invasive Modalities

As the use of PAC fell out of favor in the early 2000's, multiple additional noninvasive modalities were proposed as alternatives to PAC's to measure cardiac output [30]. These modalities include the chest bioreactance techniques, minimally-invasive pulse-contour analyses, and transpulmonary dilution.

The chest bioreactance technique is used by the Starling™ Non-Invasive Cardiac Output Monitor (NICOM) device (Baxter Medical, Chicago). The NICOM device was previously evaluated for use in a variety of intensive care unit settings and was shown to have acceptable accuracy, precision, and responsiveness for cardiac output measurements in comparison to PAC thermodilution [31,32]. A more recent study, however, specifically evaluated use of NICOM for cardiogenic shock patients and showed poor correlation compared to both Fick and PAC thermodilution [33]. Potentially, the poor correlation of NICOM may be related to the thoracic fluid overload and low flow state seen in cardiogenic shock patients affecting such impedance-based measurements. To our knowledge, the use of NICOM for hemodynamic monitoring in cardiogenic shock has not been endorsed in any societal guidelines.

Pulse-contour analysis devices have similarly been proposed as non-invasive or minimally-invasive cardiac output monitors but not been validated specifically for the cardiogenic shock population and have not been endorsed in any societal guidelines or studied in recent literature. Transthoracic dilution measures, using a central venous and arterial catheter, calculate cardiac output, global end-diastolic volume, and extravascular lung water. In a recent trial for AMICS, investigators compared the use of PiCCO™ to a control group utilizing central venous pressure, heart rate, and blood pressure monitoring alone [34]. The study demonstrated favorable outcomes in APACHE and SOFA scores, length of stay, and cardiac indices in the days following initiation. To our knowledge, use of this device for cardiogenic shock management has not been endorsed in any societal guidelines. These minimally invasive devices can all be used to estimate cardiac output but do not give the additional information provided by a PAC.

CONCLUSION

A significant new amount of literature has emerged over the past several years surrounding diagnosis and treatment of cardiogenic shock. Multimodal hemodynamic monitoring remains paramount to the diagnosis, staging, and implementation of treatment. In particular, new data highlights the re-emergence of the pulmonary artery catheter as an important tool for this patient population, though the overall level of evidence remains limited. Further prospective analyses or trials linking the pulmonary artery catheter with management

decisions and patient-centered outcomes in the cardiogenic shock population would be helpful as next steps.

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KEY POINTS

- Serial hemodynamic monitoring is paramount to the diagnosis, initial treatment, and subsequent management of cardiogenic shock.
- All patients with cardiogenic shock should have an arterial line placed for blood pressure monitoring and titration of vasoactive infusions.
- Invasive monitoring with a pulmonary artery catheter should be considered, particularly in patients with diagnostic uncertainty or in those patients who fail to respond to initial therapy.
- Echocardiography should be used early in the diagnosis of cardiogenic shock and serial assessments may aid management.

Table 1.

Hemodynamic Characteristics of the SCAI Classification of Cardiogenic Shock*

Stage		Physical Examination	Biochemical markers	Hemodynamics
A.	**A**t Risk	Normal CVP, no rales, warm, good peripheral pulses, normal mentation	Normal labs, lactate and renal function	SBP >100 or normal for pt. If done: CI ≥2.5, CVP <10, S_vO_2 >65%
B.	**B**eginning CS	↑CVP, rales, good peripheral pulses, normal mentation	Minimal renal functional impairment, elevated BNP	SBP <90 or MAP <60 or >30 decrease, HR ≥100. If done: CI ≥2.2, S_vO_2 ≥65%
C.	**C**lassic CS	May include any: Looks unwell, panicked, ashen, volume overload, rales, Killip class 3–4, cold/clammy, altered MS, decreased UO	May include any: Lactate>2, doubling creatinine or 50% drop in GFR, Increased LFTs, elevated BNP	May include any: SBP <90, MAP <60 or >30 decrease, and drugs/device used to maintain BP above target, CI <2.2, P_{pao} >15, CVP/P_{pao} ≥0.8, PAPI <1.85, CPO ≤0.6
D.	**D**eteriorating/ Doom	Any of Stage C	Any of Stage C and deteriorating	Any of Stage C and require multiple pressors, mechanical circulatory support to maintain flow
E.	**E**xtremis	Near pulselessness, cardiac collapse, mechanical ventilation, defibrillator used	"Trying to die" pH ≤7.2, lactate ≥5	No SBP without resuscitation, pulseless electrical activity, refractory VT/VF, hypotension despite maximal support

*Abbreviations: Brain natriuretic peptide: BNP, cardiac index: CI, cardiac power output: CPO, cardiogenic shock: CS, central venous pressure: CVP, glomerular filtration rate: GFR, liver function tests: LFTs, mean arterial pressure: MAP, mental status: MS, mixed venous O_2 saturation: S_VO_2, pulmonary arterial occlusion pressure: P_{pao}, pulmonary artery pulsatility index: PAPI, patient: pt, systolic blood pressure: SBP, urine output: UO, ventricular tachycardia/fibrillation: VT/VF. Modified from reference 2. Baran et al. Catheter Cardiovasc Interv 2019;94:29-37.

Table 2.

Variable Hemodynamic Phenotypes of Cardiogenic Shock*

	“Dry”	**“Wet”**
“Warm”	Low SVR with Normal or Decreased P_{pao} = Vasodilatory (not Cardiogenic) Shock	Low SVR with Elevated P_{pao} = Mixed Shock (Cardiogenic Shock with vasoplegia)
“Cold”	Elevated SVR with Normal or Decreased P_{pao} = Euvolemic Cardiogenic Shock	Elevated SVR with Elevated P_{pao} = Classic Cardiogenic Shock

*Abbreviations: Pulmonary artery occlusion pressure: P_{pao}, systemic vascular resistance: SVR