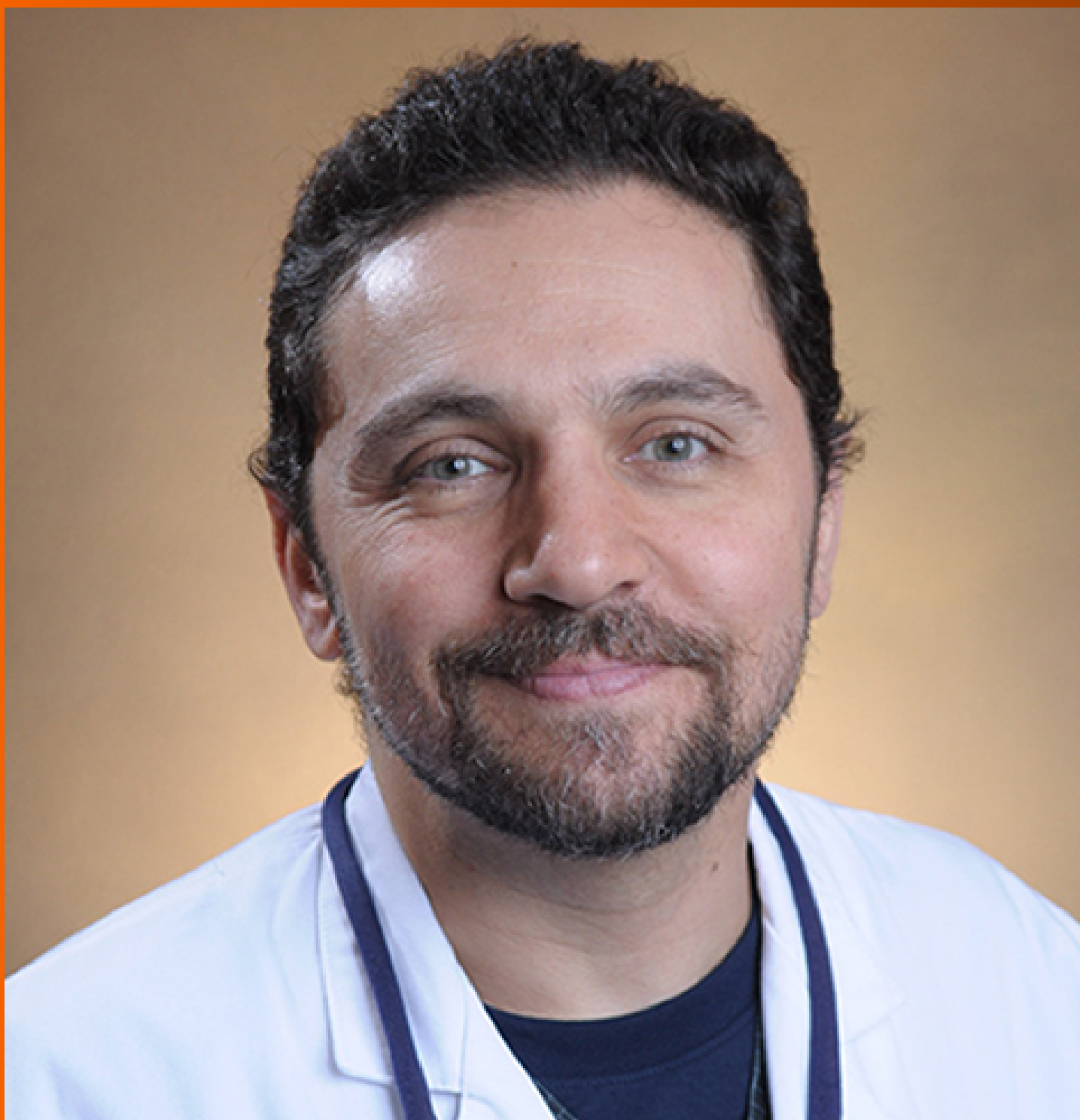


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# Pulmonary artery catheterization in acute myocardial infarction complicated by cardiogenic shock: A review of contemporary literature

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## Abstract

Acute myocardial infarction (AMI) with left ventricular (LV) dysfunction patients, the most common cause of cardiogenic shock (CS), have acutely deteriorating hemodynamic status. The frequent use of vasopressor and inotropic pharmacologic interventions along with mechanical circulatory support (MCS) in these patients necessitates invasive hemodynamic monitoring. After the pivotal Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness trial failed to show a significant improvement in clinical outcomes in shock patients managed with a pulmonary artery catheter (PAC), the use of PAC has become less popular in clinical practice. In this review, we summarize currently available literature to summarize the indications, clinical relevance, and recommendations for use of PAC in the setting of AMI-CS.

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**Core Tip:** The unstable hemodynamic status in acute myocardial infarction-cardiogenic shock patients and frequent use of vasopressor and inotropic medications along with mechanical circulatory support devices, may suggest a role for invasive hemodynamic monitoring with a pulmonary artery catheter (PAC) to help improve outcomes. In this review, we summarize the currently available literature to summarize the indications, clinical relevance, and recommendations for use of PAC in the setting of acute myocardial infarction-cardiogenic shock.

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## INTRODUCTION

Cardiogenic shock (CS) is a high-acuity hemodynamically diverse state of end-organ hypoperfusion that is frequently associated with multisystem organ failure. Acute myocardial infarction (AMI) with left ventricular (LV) dysfunction remains the most frequent cause of CS[1,2]. AMI related CS (AMI-CS) continues to be associated with high mortality (30%-40%) even in the contemporary era of early reperfusion, increasing use and availability of MCS devices and multidisciplinary shock teams[3-5]. In contrary to conventional teaching, the hemodynamic profile of CS patients is dynamic across a wide clinical spectrum depending on its stage of development[6]. The acutely deteriorating hemodynamic status in AMI-CS patients and nearly ubiquitous use of vasopressor and inotropic medication along with mechanical circulatory support (MCS) devices, underscore the importance of invasive hemodynamic monitoring to help in providing optimal therapies for these patients.

Although earlier randomized clinical trials (RCTs) including the pivotal Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) trial failed to show a significant improvement in clinical outcomes in shock patients managed with a pulmonary artery catheter (PAC), this data may not be representative of AMI-CS patients as it involved hemodynamically stable patients with heart failure and specifically excluded CS patients[7].

Earlier data from RCTs also failed to show mortality benefit in CS with use of PAC [8-11]. But CS is not a homogeneous disorder and AMI-CS being a distinct entity with markedly different therapeutic/interventional options and management protocols were grossly under-represented (5%-20%) in those studies. Extrapolation of data from these prior studies in the realms of heart failure and critical care and applying it to AMI-CS population may warrant caution and further deliberation[12]. Recent registry-based data allude to improved mortality especially in patients with heart failure and CS with use of PAC[13]. The 2016 European Society of Cardiology guidelines for treatment of heart failure also suggest use of PAC in patients with refractory CS despite pharmacological treatment LOE IIB [C] or being considered for MCS or heart transplantation LOE I [A][14].

Although there are a few earlier reviews on PAC use, they were focused on diagnosis and management of CS patients undergoing MCS[15,16]. However, the use of PAC in AMI-CS subset of patients requires more critical discussion due to multiple recent studies in this arena and addition of intriguing new data regarding its clinical utility. In this review, we intend to explore the indications and recommendations for use of PAC in the setting of AMI-CS and review the recent literature supporting it.



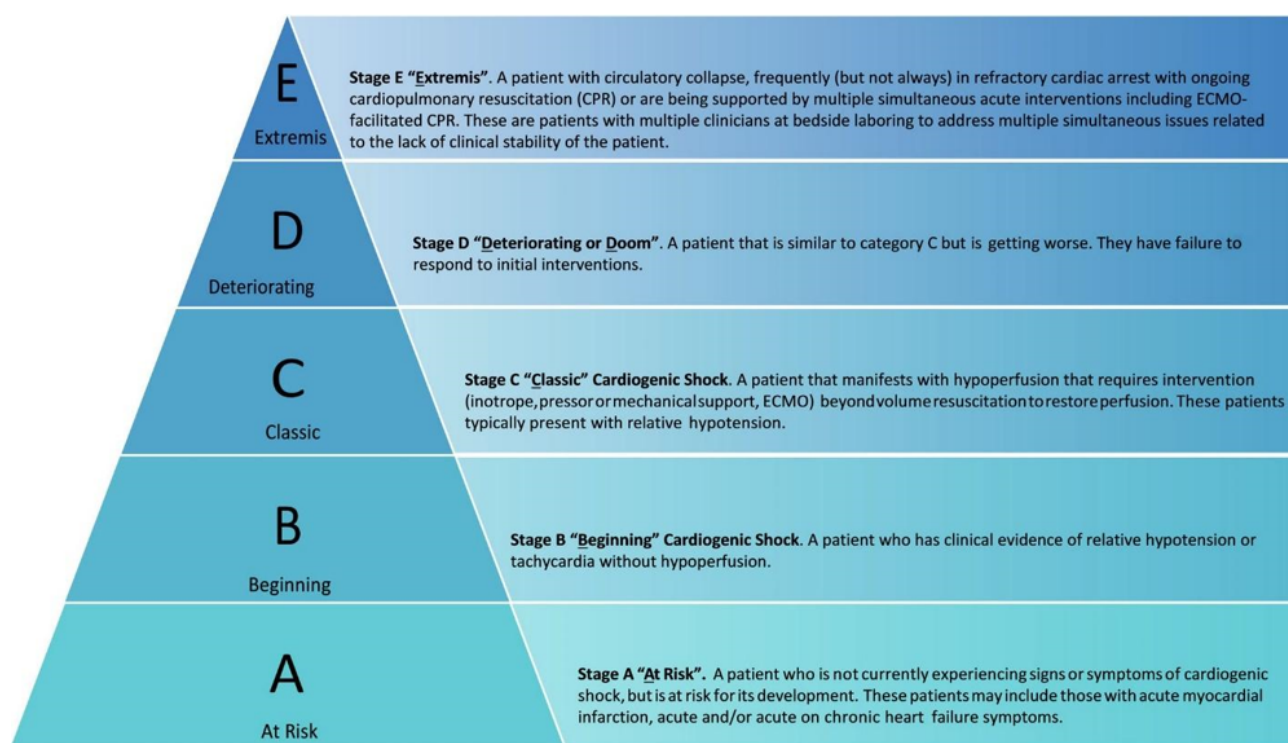
### Epidemiological trends of PAC use in AMI-CS

After the data from ESCAPE trial was published, there was a notable decrease in PAC use for hemodynamically unstable patients except for AMI-CS. A recently published studies of a nationally representative population of AMI-CS and HF showed up to 75% decrease in PAC use between 2000 and 2014 despite a concomitant increase in patient acuity[13,17]. Significantly higher PAC use was seen in younger patients, patients of white race and those with higher baseline comorbidity, non-cardiac organ failure, and on MCS[17]. Interestingly, PAC was utilized 10 times more frequently in patients with HF and CS as compared to HF patients without CS between 2004 and 2014[13]. Another study involving medicare beneficiaries looked at 457193 hospitalized patients with PACs and showed that the use of PAC decreased by about 2/3<sup>rd</sup>s from 6.28 per 1000 admissions in 1999 to 2.02 per 1000 admissions in 2013 ( $P < 0.001$ ). The study also noted that the decrease use of PAC was more pronounced in patients with respiratory failure [29.9 PACs placed per 1000 admission in 1999 to 2.3 in 2013 (92.3% reduction),  $P < 0.001$  for trend] as compared to PAC use for AMI [20.0 PACs placed per 1000 admissions in 1999 to 5.2 in 2013 (decreased by 74.0%)  $P < 0.001$ ]. Interestingly, the study also noted a nadir in 2009 followed by a subsequent increase in use of PAC for heart failure patients (9.1 PACs placed per 1000 admissions in 1999 to 4.0 in 2009 to 5.8 in 2013) and this was also associated with improved in-hospital mortality, 30-d mortality, and reduced length of stay[18]. A study by Khera *et al*[19] looking at the trends in PAC use among HF patients in the United States from the National Inpatient Sample (NIS) data, 2001 to 2012 showed a decrease in PAC use in CS from 8.2% in 2001 to 6.7% in 2007, but then there was an upward trend up to 14% in 2012 and its use was more common in the larger academic facilities with advanced HF therapies. Similarly, more recent studies using NIS data from 2000-2014, looking at 364001 admissions with AMI-CS showed that PAC was used in 8.1% of patients but there was a 75% decrease during over the study period (13.9% to 5.4%)[17]. While another NIS based study looking at more recent data of 1531878 hospitalized patients with CS (0.3% of total hospital admissions) from January 1, 2004-December 31, 2018, showed a significant increase in the trend for utilization of PAC in CS patients (both AMI-CS and non AMI-CS) reaching up to 17% in 2018 as compared to 10% utilization in the immediate post-ESCAPE trial era ( $P$ -trend  $< 0.001$ )[20].

In the European literature, a study by Sionis *et al*[6] using an observational, prospective, multicenter, European registry showed that CS patients treated in academic centers noted PAC use 82 (37.4%) of the 219 patients over a course of 2 years. Rossello *et al*[21] noted that a PAC was used in 64% of patients with CS from 2005-2009. In Japan, the use of PAC was seen in 16.8% of patients[22]. Overall, the use of PAC is more common in European countries than in United States and other non-European countries. Earlier studies also noted higher use of PAC in patients with higher socioeconomic status and with insurance coverage, large urban hospitals and in patients with MCS which may relate to social disparities in care among this population and paradigm shift in the management of AMI-CS using newer per-cutaneous MCS devices that may require constant hemodynamic data feedback for effective and safe utilization[17].

### Pathophysiology of CS and the role of PAC

Regardless of the etiology, CS is a primary pump failure (increased residual volume and intracardiac pressures in one or both ventricles), which could be from right ventricular, LV or biventricular dysfunction, resulting in hemodynamic compromise and multi-organ failure[23-25]. PAC measures direct and indirect parameters which can be used to differentiate right-sided, left-sided, or biventricular dysfunction. For instance, a high central venous pressure (CVP) to pulmonary capillary wedge pressure (PCWP) ratio indicates right-ventricular (RV) failure[26]. Similarly, low pulmonary artery pulsatility index (PAPi), a more accurate measure of RV function, is associated with high CVP, PCWP, mean PA pressure and low cardiac index (CI). In contrast, these parameters measured with other non-invasive parameters such as echocardiography are not as accurate as with PAC[27]. The Society for Cardiovascular Angiography and Interventions (SCAI) recently proposed a five-stage classification system for CS: A – at risk – at risk of developing symptoms of CS but currently asymptomatic; B – beginning – patient who has relative hypotension but no signs of hypoperfusion; C – classic – patients require inotropic or MCS; D – deteriorating – C getting worse with failure to respond to aforementioned therapies; and E – extremis – circulatory collapse and refractory cardiac arrest (Figure 1)[28]. SCAI classification is used for prognostication purposes as the in-hospital mortality has been shown to rise progressively with each advancing SCAI stage[29,30]. Use of PAC measured para-



**Figure 1 Stages of cardiogenic shock classified by the Society of Cardiovascular Angiography and Intervention.** CPR: Cardiopulmonary resuscitation; ECMO: Extracorporeal membrane oxygenation. Citation: Baran DA, Grines CL, Bailey S, Burkhoff D, Hall SA, Henry TD, Hollenberg SM, Kapur NK, O'Neill W, Ornato JP, Stelling K, Thiele H, van Diepen S, Naidu SS. SCAI clinical expert consensus statement on the classification of cardiogenic shock. *Catheter Cardiovasc Interv.* 2019; 94(1): 29-37. Copyright© The Authors 2021. Published by John Wiley and Sons. The authors have obtained the permission for figure using from the Wiley Periodicals Inc.

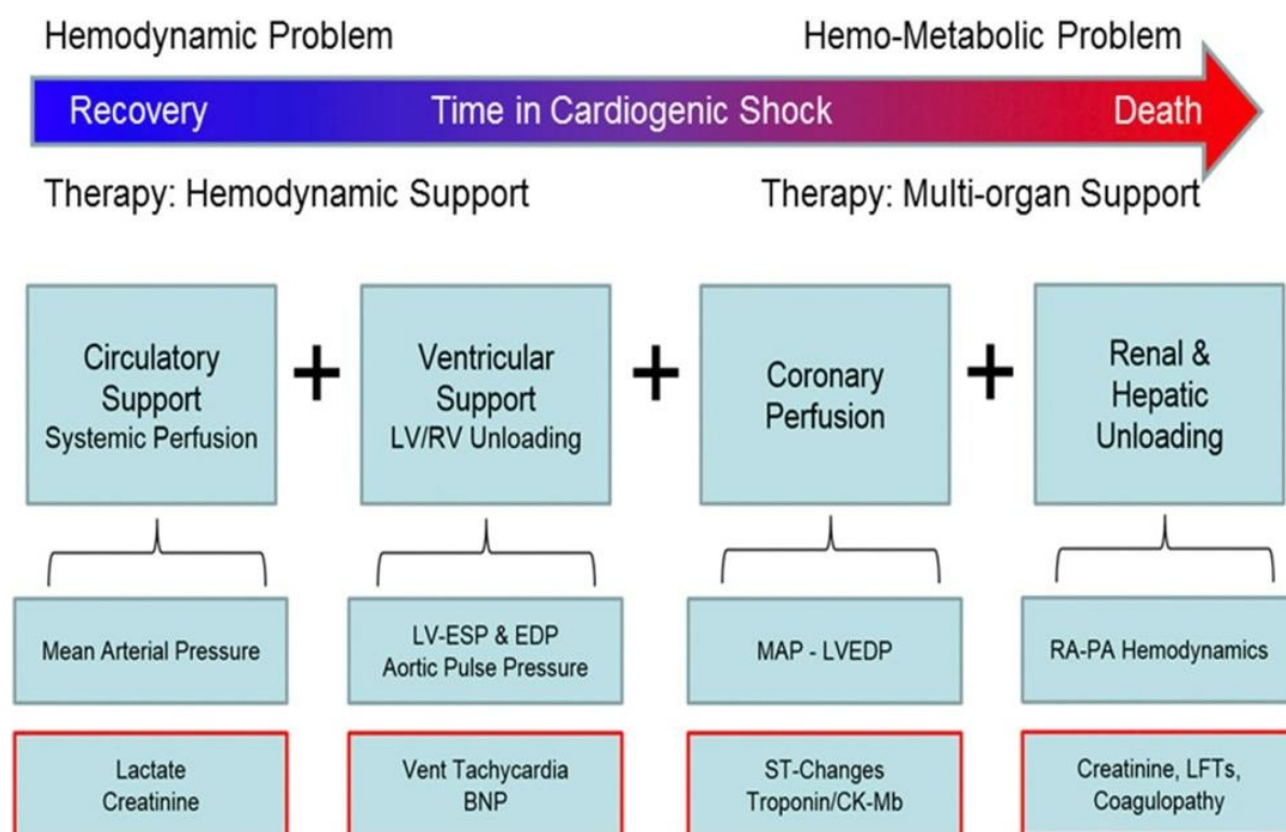
meters and prognostication through SCAI classification can facilitate clinical decision making in deciding the therapy and its clinical utility[31].

Untreated or sub-optimally treated CS results in a state of persistent tissue hypoperfusion with accumulation of lactic acid metabolites which transitions the early potentially reversible hemodynamic insult of CS to a more complex 'hemo-metabolic' cascade with refractory CS (Figure 2)[32]. All aspects of hemodynamic support including adequate circulatory support, optimal LV unloading, restoring myocardial perfusion and achieving significant decongestion must be fulfilled in a timely manner to effectively treat and reverse the hemodynamic compromise of CS[32,33].

Adequate circulatory support is defined by an increase in mean arterial pressure (MAP) and enhanced microvascular blood flow resulting in adequate organ perfusion. Ventricular unloading, which is defined as a reduction in myocardial work and wall stress, is best achieved by reducing native ventricular pressure and volume[32,33]. Myocardial perfusion, increased epicardial and microvascular coronary blood flow, often improves with adequate circulatory and ventricular support. Decongestion refers to a reduction in total body volume resulting in decreased venous filling pressures [32]. The importance of aforementioned aspects of CS is crucial to highlight, since selective therapies such as inotropes and vasopressors although may increase MAP, but they do not improve microvascular organ perfusion[32]. In addition, inotropes and vasopressors disproportionately increase LV afterload, myocardial work/wall stress, and myocardial ischemia eventually culminating refractory CS and increased in-hospital mortality[32].

### **Clinical utility of hemodynamic parameters from the PAC**

The use of hemodynamic parameters obtained through the PAC are essential indicators for decision-making during the selection, initiation, titration and weaning of pharmacological as well as MCS support in AMI-CS patients. Emerging new evidence suggests that the use of PAC among patients with CS is associated with lower mortality and lower in-hospital cardiac arrest possibly by improved patient selection and better utilization of hemodynamic data to guide management[13]. Early acquisition of hemodynamic data like cardiac output (CO), cardiac filling pressures and systemic vascular resistances (SVR) and pulmonary vascular resistances (PVR) would



**Figure 2 Hemo-metabolic cascade of acute myocardial infarction with cardiogenic shock.** BNP: B-type natriuretic peptide; CK: Creatinine kinase; ESP: End-systolic pressure; LFT: Liver function tests; LV: Left ventricular; LVEDP: Left ventricular end-diastolic pressure; MAP: Mean arterial pressure; PA: Pulmonary artery; RA: Right atrium; RV: Right ventricular. Citation: Esposito ML, Kapur NK. Acute mechanical circulatory support for cardiogenic shock: the "door to support" time. *F1000Res*. 2017 May 22; 6: 737. Copyright© The Authors 2021. Published by Taylor and Francis Group. The authors have obtained the permission for figure using from the Taylor and Francis Group.

help not only to define the nature of CS (univentricular or biventricular) but also to evaluate the patient's response to various advanced therapies[32]. For instance, the use of PAC in such patients would be indispensable in assessing response to therapies, guiding management and optimize device settings especially when escalating or de-escalating MCS and is supported by the current heart failure guidelines (Table 1)[14, 16]. In patients with CS, continuous hemodynamic feedback from a PAC can guide management by helping to optimize volume status, titrate vasoactive medications in a more targeted fashion as well as detect any complications such as pump thrombosis which usually manifests as recurrence of CS with sudden elevation of PA and PCWPs [33,34]. A more recent reanalysis of the ESCAPE trial data published in 2016 showed that advanced heart failure patients with a PAC who achieved a post-treatment goal of PCWP + right atrial pressure (RAP) < 30 mmHg was associated with a 6-mo mortality rate of 8.7%, as compared to 45.3% ( $P$ -value < 0.0001) in patients who have failed to achieve that target[35]. A recently released scientific statement from the American Heart Association does endorse use of PAC in difficult clinical scenarios such as when treating patients with cardiorenal syndrome as the real-time hemodynamic data obtained through PAC will help to identify and treat subclinical congestion and avoid over diuresis and intravascular underfilling and thereby improving the hemodynamics and subsequent end organ perfusion to the heart and kidneys[36] (Table 2).

Several algorithms have been proposed to help manage and potentially improve outcomes in patients with AMI-CS and early acquisition of hemodynamic data using a PAC and prompt action remain a central theme across all the various protocols. According to the National Cardiogenic Shock Initiative, in order to achieve four aspects of hemodynamic support equation, it is important to maintain thrombolysis in myocardial infarction - 3 flow,  $CPO > 0.6$  ( $CPO = MAP \times CO / 451$ ) and  $PAPi > 0.90$  [ $PAPi = (\text{systolic pulmonary artery pressure} - \text{diastolic pulmonary artery pressure}) / CVP$ ][37-39]. Four different management pathways could be evaluated from CPO and PAPi. Therefore, the hemodynamics obtained from the use of a PAC are crucial in determining further management.



**Table 1 Studies evaluating outcomes with use of pulmonary artery catheter in patients with cardiogenic shock**

| Author (year)                           | Study type                  | Region/sites                | Time period            | n       | Study population               | Outcomes                            | Conclusion  |
|---|-----------------------------|-----------------------------|------------------------|---------|--------------------------------|-------------------------------------|---|
| Sotomi <i>et al</i> [22] (2014)         | Prospective observational   | Japan-multicenter           | 2007-2011              | 1004    | ADHF                           | All-cause mortality                 | Decreased all-cause mortality in PAC cohort on inotropic support or lower SBP |
| Sionis <i>et al</i> [6] (2020)          | Prospective observational   | Europe-multicenter          | 2010-2012              | 219     | CS, hypotension or severe LCOS | 30-d mortality                      | No mortality difference. CI, CPI, and SVI-predictors of 30-d mortality        |
| Rossello <i>et al</i> [21] (2017)       | Prospective observational   | Spain-single center         | 2005-2009              | 179     | CS                             | Short- and long-term mortality      | Lower long-term and short-term mortality                                      |
| Hernandez <i>et al</i> [13] (2019)      | Retrospective observational | United States-multicenter   | 2004-2014              | 9431944 | ADHF and CS                    | Mortality                           | Lower mortality   |
| Doshi <i>et al</i> [54] (2018)          | Retrospective observational | United States-multicenter   | 2005-2014              | 842369  | CS                             | In-hospital mortality               | Lower mortality   |
| Cohen <i>et al</i> [55] (1)(2005)       | Retrospective observational | International-multicenter   | –                      | 26437   | ACS                            | 30-d mortality                      | Higher mortality  |
| Gore <i>et al</i> [56](1987)            | Retrospective observational | United States-multicenter   | 1975, 1978, 1981, 1984 | 3263    | AMI                            | In-hospital and long-term mortality | No mortality difference   |
| Vallabhajosyula <i>et al</i> [17](2020) | Retrospective observational | United States-multicenter   | 2000-2014              | 364001  | AMI-CS                         | In-hospital mortality               | No mortality difference   |
| Zorzi <i>et al</i> [52] (2019)          | Retrospective observational | Switzerland-single center   | 2008-2011              | 91      | CS                             | Mortality                           | Increase in PAC in first 24 h   |
| Garan <i>et al</i> [34] (2020)          | Retrospective observational | United States-multicenter   | 2016-2019              | 1414    | CS                             | In-hospital mortality               | Lower mortality   |
| Cooper <i>et al</i> [57] (2015)(3)      | Retrospective observational | United States-single center | 2002-2008              | 217     | AMI                            | CS diagnosis                        | Echocardiography-based criteria can be used to accurately diagnose CS         |

ACS: Acute coronary syndrome; ADHF: Acute decompensated heart failure; AMI: Acute myocardial infarction; CI: Cardiac index; CS: Cardiogenic shock; CPI: Cardiac power index; HF: Heart failure; LCOS: Low cardiac output syndrome; PAC: Pulmonary artery catheterization; SBP: Systolic blood pressure; SVI: Stroke volume index.

The INOVA Heart and Vascular Institute algorithm adopts a similar ‘combat’ approach to managing CS and primarily relies on 5 key areas of focus which include rapid identification of shock, early right heart catheterization, expedited initiation and early escalation of percutaneous MCS as appropriate, minimization of vasopressor and inotrope use, and, meaningful patient recovery and survival[40,41]. In addition to the routinely measured hemodynamic parameters using a PAC, the INOVA pathway emphasizes on measurement of CPO ( $< 0.6$ ), right atrial (RA): PCWP ( $> 0.63$ ) and PAPI ( $< 1.5$ ) as well as other metrics such as serum lactate ( $> 2$  mmol/L) and tricuspid annular plane systolic excursion ( $< 14$  mm) to help diagnose the presence of RV failure and need for RV mechanical support in CS as well as guide management including initiating or escalating/de-escalating percutaneous MCS[40].

In another prospective study by Garan *et al*[42] comparing outcomes of veno-arterial extracorporeal membrane oxygenation and a percutaneous ventricular assist device an institutional CS algorithm was used to guide selection of MCS. Of the 51 patients, 31 (76.4%) underwent invasive hemodynamic assessment with a PAC before the first device initiation and both groups had very similar hemodynamic parameters as measured by the PAC including, RA pressure, PCWP, CPO and CI[42]. The Utah Cardiac Recovery Shock Team also emphasizes the importance of multidisciplinary shock team approach and early use of PAC to guide MCS selection and improve in-hospital mortality (61.0% for shock team *vs* 47.9% for control;  $P = 0.041$ ) and 30-d all-cause mortality [hazard ratio (HR): 0.61, 95% cumulative incidence (CI): 0.41-0.93] in refractory AMI-CS[41]. The University of Ottawa Heart Institute adopted a multidisciplinary code shock team approach to CS and demonstrated improved long-term survival[43]. In their study as well, hemodynamic monitoring with a PAC was done in 62% of patients (66% for treatment *vs* 50% for control,  $P = 0.13$ ) for a median duration of 4 d (IQR, 2-6)[43].

**Table 2 Current guidelines on pulmonary artery catheterization in cardiogenic shock**

| Guideline                                | Recommendation   |
|--|--|
| 2011 ACCF/AHA CABG[51]                   | Invasive hemodynamic monitoring with PAC is required before induction of anesthesia in patients with CS undergoing CABG (Class I; level of evidence C)   |
| 2013 ACCF/AHA HF [52]                    | Invasive hemodynamic monitoring should be performed in patients with respiratory distress or impaired perfusion – when intracardiac filling pressures could not be determined from clinical assessment (Class I; level of evidence C)<br><br>Invasive hemodynamic monitoring is also recommended for patients with persistent acute HF symptoms despite empiric HF therapy adjusts and with one of following: (1) Systemic or pulmonary vascular resistance; or fluid status or perfusion is uncertain; (2) Low systolic blood pressure despite initial therapy; (3) Worsening renal function; (4) Candidate for pressor support; and (5) Candidate for MCS or heart transplant (Class IIa; level of evidence C) |
| The 2013 ISHLT MCS [53]                  | Patients undergoing procedure MCS device placement should have insertion of large-bore intra-venous line, arterial line, and pulmonary catheter for monitoring and intra-venous access (Class I; level of evidence B)  |
| 2016 ESC HF[11]                          | Routine invasive hemodynamic evaluation is not indicated for diagnosis of HF – PAC could be used in hemodynamically unstable patients with unknown mechanism of deterioration<br><br>PAC could be used for acute HF who have refractory symptoms despite pharmacological treatment (Class IIb; level of evidence C)<br><br>PAC along with right heart catheterization is recommended for evaluation of patients for MCS or heart transplantation (Class I; level of evidence C)  |
| 2017 SCAI/HFSA Invasive Hemodynamics[54] | Continuous hemodynamic monitoring is required for patients receiving MCS<br><br>Continuous hemodynamic monitoring is used for withdrawal of MCS and pharmacologic support  |

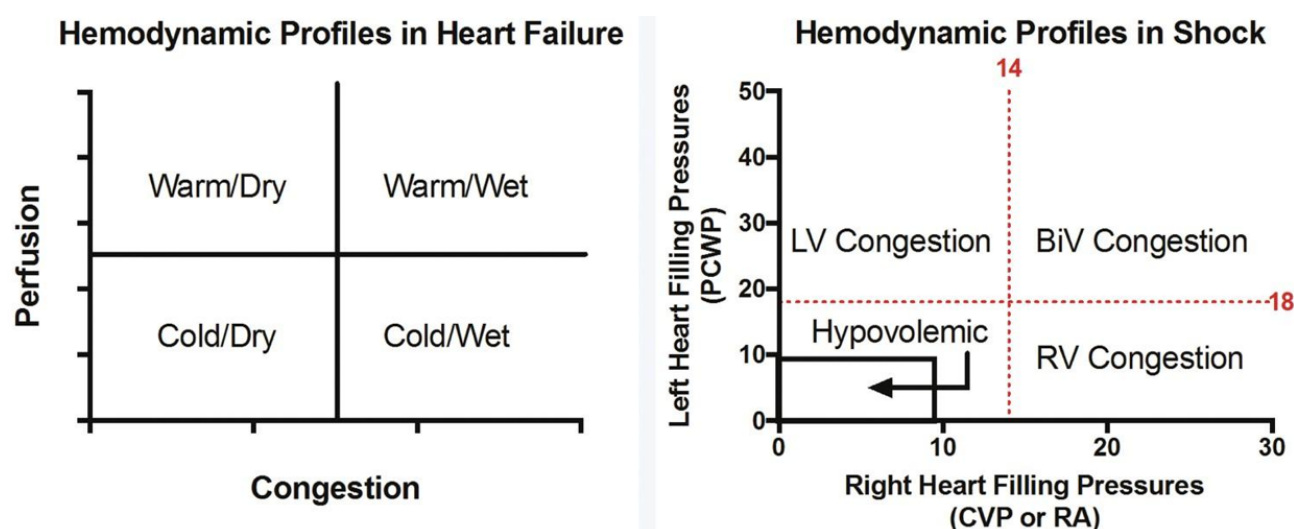
ACCF: American College of Cardiology Foundation; AHA: American Heart Association; CABG: Coronary artery bypass grafting; CS: Cardiogenic shock; ESC: European Society of Cardiology; HF: Heart failure; HFSA: Heart Failure Society of America; ISHLT: International Society of Heart and Lung Transplantation; MCS: Mechanical circulatory support; PAC: Pulmonary artery catheter; SCAI: Society of Cardiovascular Angiography and Intervention.

The ratio between the RA pressure and PCWP (RA:PCWP ratio) could help us gain insight into the possibility of RV failure in AMI and prognosis in patients with CS[32]. The RA:PCWP ratio can be used analogous to the classic  $2 \times 2$  table in HF patients to classify patients as hypovolemic, LV-, RV-, or BiV dominant congestion (Figure 3)[44, 45]. Prior studies have successfully demonstrated PAPI as a simple and reliable hemodynamic measure to predict in-hospital mortality after acute inferior wall MI with high sensitivity and specificity as well as predict RV failure after left ventricular assist devices implantation[46,47].

A recent review on AMI-CS emphasizes the need for systems of care with early recognition and transportation of AMI-CS patients to level I dedicated cardiac shock care centers along with use of pre-PCI implantation of MCS devices with “door-to-support” time  $\leq 90$  min and consistent use of PAC for accurate hemodynamic monitoring to help improve survival and outcomes in these patients[48]. It is important to recognize that PAC does not have any intrinsic therapeutic effect and by itself would not improve outcomes but rather facilitates decisions that could translate to favorable outcomes by prompt and appropriate action guided by the real-time monitoring of hemodynamic data. For instance, escalation of device therapy from a primarily LV support to biventricular device support with Bipella (right and left sided Impella) may be warranted if hemodynamic monitoring with PAC suggests biventricular failure with CPO  $< 0.6$  and PAPI  $< 0.9$  to help reverse the progression of AMI-CS[25].

### ***Differentiating AMI-CS from CS in chronic congestive heart failure***

Although CS is often referred to as one homogenous entity the CS phenotype in AMI patients may be very distinct from that in end stage heart failure patients and such early distinction could have significant prognostic and therapeutic implications. There are also considerable differences between CS from AMI *vs* heart failure – chronicity in heart failure along with neurohumoral dysregulation (especially shock) and changes stemming from heart failure therapy[49]. CS from AMI has low filling, lower pulmonary artery pressures, higher oxygen delivery ( $DO_2$ ), lower oxygen-hemoglobin affinity ( $P50$ ), and more severe metabolic acidosis in comparison with CS from end-stage heart failure (ESHF)[49]. Further, there is higher inpatient mortality in patients with acute HF related *vs* acute on chronic HF related CS even with similar hemodynamic characteristics such as MAP, CO, cardiac power index (CPI)[50].



**Figure 3 Congestive profiles in cardiogenic shock.** Clinical assessment of hemodynamic conditions in decompensated heart failure is traditionally categorized into four groups based on systemic perfusion and congestive status using a two-by-two table. Cardiogenic shock is categorized as having LV-, RV-, or BiV-dominant congestion or hypovolemia. Treatment approaches may be tailored to each of these four categories. BiV: Biventricular; CVP: Central venous pressure; LV: Left ventricular; PCWP: Pulmonary capillary wedge pressure; RA: Right atrial; RV: Right ventricular. Citation: Esposito ML, Kapur NK. Acute mechanical circulatory support for cardiogenic shock: the "door to support" time. *F1000Res*. 2017 May 22; 6: 737. Copyright© The Authors 2021. Published by Taylor and Francis Group. The authors have obtained the permission for figure using from the Taylor and Francis Group.

A recent single-center study by Lim *et al*[49] looking at patients with CS due to AMI ( $n = 26$ ) and ESHF ( $n = 42$ ) who underwent MCS (extracorporeal life support, Impella or temporary ventricular assist devices) suggested that the ESHF-CS patients had higher filling and pulmonary artery pressures but lower oxygen delivery, greater anaerobic metabolism with less severe metabolic acidosis as compared to the AMI-CS patients.

### Clinical outcomes in CS patients with PAC and hemodynamic monitoring

More recent data from the CS literature have shown potential short- and long-term mortality implications of invasive hemodynamic data. In the CardShock study, which used an observational, prospective, multicenter, European registry, the CI, CPI and stroke volume index were the strongest 30-d mortality predictors in addition to the previously validated CardShock risk score (Table 1)[6]. Similarly, an earlier study looking at 541 patients with CS who were enrolled in the Should we emergently revascularize Occluded Coronaries for CS (SHOCK) trial registry suggested that CP [odd ratio (OR): 0.60, 95%CI: 0.44-0.83,  $P < 0.002$ ;  $n = 181$ ] and CPI (OR: 0.65, 95%CI: 0.48-0.87,  $P < 0.004$ ;  $n = 178$ ) are the strongest independent hemodynamic correlate of in-hospital mortality in patients with CS[37], but this was not shown to be predictive in a more recent study involving a large multi-center registry[34]. Data from this large multicenter registry study representing real-world patients with CS in the contemporary acute MCS era suggested that decreased MAP along with an increased RAP significantly associated with higher mortality but PCWP, CPO and CI did not appear to impact mortality consistently[34].

The Nursing Students Competence Instrument shock team protocols used cardiac power output[37], and PAPi[39,51] as hemodynamic criteria for MCS patient selection, assessing response to therapy and for escalation/de-escalation of MCS. In this study, CPO ( $> 0.6$  or  $< 0.6$  W) and lactate ( $> 4$  or  $< 4$  mg/dL) at 12-24 h was shown to have the best prognostic value in predicting survival as patients with persistently higher lactate levels ( $> 4$  mmol/L) and low CPO ( $< 0.6$  W) at 12-24 h while on Impella support will have a higher mortality (50%) and such patients should be evaluated for escalation of MCS[38].

Another retrospective single center study looking at 91 consecutive patients with CS due to primary LV failure, who had PAC within the first 24 h showed that a reduced compliance of the pulmonary artery (CPA), worsened right ventricular dysfunction and was independently associated with increased mortality in patients with CS and increased from 4.5% in the quartile of patients with highest CPA to 43.5% in the lowest CPA quartile[52].

Literature has shown beneficial, non-significant, and deleterious effects of PAC in CS patients (Table 1). In a study by Hernandez *et al*[13] utilizing the NIS database, patients with CS and PAC use had lower mortality (35.1% *vs* 39.2%, OR: 0.91;  $P < 0.001$ ) and lower in-hospital cardiac arrest (14.9% *vs* 18.3%, OR: 0.77;  $P < 0.001$ ) which persisted even after propensity score matching. The Acute Decompensated Heart Failure Syndromes registry which was an prospective, multicenter observational study in which 813 patients (16.8%) were managed with PACs, of which 502 patients (PAC group) were propensity core-matched with 502 controls (control group) showed that PAC guided management in advanced HF patients with CS requiring inotropes (HR: 0.22; 95%CI: 0.08-0.57;  $P = 0.002$ ) and are hypotensive (systolic blood pressure  $\leq 100$  mmHg; HR: 0.09; 95%CI: 0.01-0.70;  $P = 0.021$ ) had an in-hospital mortality benefit compared to those managed without PAC derived hemodynamic data[22]. Another recent study from the Cardiogenic Shock Working Group looking at 1414 patients with CS showed that use of complete PAC-derived hemodynamic data prior to MCS initiation in 1190 (84%) patients with advanced CS stages was associated with improved survival from CS ( $P < 0.001$ ). Patients with no PAC assessment had worse in-hospital mortality as compared to patients who were assessed with PAC (OR: 1.57; 95%CI: 1.06-2.33)[34]. Another recent study involving 15259 AMI-CS patients treated rapidly with an Impella for MCS along with use of invasive hemodynamic monitoring with a PAC as the first strategy had significantly better survival rates (63%) as compared to the controls (49%) ( $P < 0.001$ )[53].

Interestingly, a single center study with 129 patients admitted with CS and followed for 5 years showed that the use of PAC in patients with CS was associated with lower short-term (HR: 0.55, 95%CI: 0.35-0.86,  $P = 0.008$ ) and long-term mortality rates (HR: 0.63, 95%CI: 0.41-0.97,  $P = 0.035$ ) even after adjustment for age, gender and the presence of shock upon admission but this benefit was only significant in those patients without acute coronary syndrome (ACS)[21]. This merits future studies on outcomes of PAC in ACS *vs* non-ACS patients.

In contrast, CardShock study was an observational, prospective, multicenter, European registry study in which more than one-third of patients were managed with a PAC. The findings from this study suggest that use of PAC was associated with a more aggressive treatment strategy but did not increase the 30-d mortality[6]. Similarly, a retrospective single center study looking at 91 consecutive patients with CS due to primary LV failure, who had PAC within the first 24 h showed with increased mortality in patients with CS[52]. The discrepancy in the outcomes of mortality with PAC invites future multi-center and international trials as deciding factors to assess the efficacy of PAC in comparison with PAC in AMI-CS sub-set of population.

### Limitations

This review is based on the results of currently available observational, single/multi-center, and national cohorts. However, the contribution of confounding factors in these studies is unknown. For instance, use of PAC could be significantly higher in critically ill patients thus confounding the results of in-hospital, 30-d mortality and other relevant clinical outcomes. Therefore, the role of PAC in AMI-CS patients may need to be further explored through well-designed future RCTs.

### Future directions

As PAC by itself has no intrinsic therapeutic benefit, future studies focused on testing the workflows and appropriate interventions that would allow prompt acquisition and action on hemodynamic information from the PAC including the timing, selection, management, and weaning of temporary MCS. There is also an ongoing clinical trial looking at whether PAC guided LV mechanical unloading after PCI for acute anterior wall MI will attenuate post-infarct scar and cardiac remodeling. The data from this study may further define the clinical utility of PAC in guiding the need for mechanical LV unloading to help improve clinical outcomes in the setting of AMI-CS.

## CONCLUSION

In conclusion, PAC has shown to be useful in monitoring treatment parameters, tailoring treatments, and predict prognosis in AMI-CS patients. Several hemodynamic parameters acquired using PAC are critical to not only defining the etiology of AMI-CS (univentricular or Bi-ventricular) but also vital to the selection, initiation, titration of both pharmacological and MCS devices in these patients that may help better



outcomes. Early identification of CS with a targeted shock to device time of < 90 min along with dedicated multidisciplinary shock teams and designated shock centers will be critical to favorably affecting mortality outcomes in this extremely sick patient population. However, the contradicting benefits of in-hospital and 30-d mortality in AMI-CS requires further understanding of the processes and treatment strategies using larger RCTs.

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