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Advanced Markers for Hemodynamic Monitoring in Cardiogenic Shock and End-Stage Heart Failure: A Mini Review

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Abstract

Right heart catheterization (RHC) provides critical hemodynamic insights by measuring atrial, ventricular, and pulmonary artery pressures, as well as cardiac output (CO). Although the use of RHC has decreased, its application has been linked to improved outcomes. Advanced hemodynamic markers such as cardiac power output (CPO), aortic pulsatility index (API), pulmonary artery pulsatility index (PAPi), right atrial pressure to pulmonary capillary wedge pressure ratio (RAP/PCWP) and right ventricular stroke work index (RVSWI) have been introduced to enhance risk stratification in cardiogenic shock (CS) and end-stage heart failure (HF) patients. CPO has emerged as a potent prognostic tool, with values below 0.6 Watts significantly associated with mortality. Similarly, API and PAPi have demonstrated strong predictive power for adverse outcomes, including death and the need for advanced HF therapies. RAP/PCWP ratio is shown to be a valuable a prognostic tool for RV dysfunction, mortality, and adverse outcomes. Despite mixed evidence on the prognostic utility of RVSWI, its physiologic relevance in assessing right ventricular function remains important. A novel clinical observation, involving patients with an RAP numerically greater than pulmonary artery saturation, was associated with a 71% 30-day mortality rate, underscoring the potential prognostic value of this finding. This review aims to summarize key advanced hemodynamic markers and their role in improving risk stratification and guiding treatment in CS and end-stage HF. The integration of these markers into clinical practice holds the potential to enhance personalized care and improve outcomes for patients with CS and advanced HF.

Keywords Cardiac Power Output · Pulmonary Artery Pulsatility Index · Aortic Pulsatility Index · Cardiogenic Shock · Right Ventricular Stroke Work Index · Right Atrial Pressure to Pulmonary Capillary Wedge Pressure Ratio

Abbreviations

API	Aortic pulsatility index
BP	Blood pressure
CI	Cardiac index
CO	Cardiac output
CPO	Cardiac power output
CS	Cardiogenic shock
HF	Heart failure
HTx	Heart transplant
iFick	Indirect Fick
LV	Left ventricle
LVAD	Left ventricular assist device
PA	Pulmonary artery
PAPi	Pulmonary artery pulsatility index
PCWP	Pulmonary capillary wedge pressure
RAP	Right atrial pressure
RHC	Right heart catheterization
ROC	Receiver operating characteristic
RV	Right ventricle
RVSWI	Right ventricle stroke work index
SVI	Stroke volume index

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Introduction

Right heart catheterization (RHC) offers direct insights into hemodynamic status by measurement of atrial, ventricular, pulmonary artery (PA) pressures, and cardiac output (CO)

[1, 2]. Following the Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) trial a significant decrease in RHC utilization was observed [3]. Notably, a 75% decrease was reported in the use of PA catheters in patients with acute myocardial infarction complicated by cardiogenic shock (CS) [4]. However, recently RHC in CS has been associated with improved outcomes and increased use of downstream advanced heart failure (HF) therapies [5–9]. Findings from RHC that have been associated with mortality include systolic PA pressure, systolic and diastolic blood pressure (BP), as well as disproportionate increase in right to left ventricular (LV) filling pressures [10, 11]. Advanced hemodynamics markers have been introduced for hemodynamic monitoring in CS, including cardiac power output (CPO), aortic pulsatility index (API), pulmonary artery pulsatility index (PAPi), right atrial pressure to pulmonary capillary wedge pressure ratio (RAP/PCWP) and right ventricle stroke work index (RVSWI). This review aims to (1) summarize these advanced hemodynamic markers; (2) evaluate their association with outcomes in patients with CS and end-stage HF; (3) present an observation from clinical practice in patients undergoing RHC with the unusual constellation of findings of a right atrial pressure (RAP) numerically higher than PA saturation.

Cardiac Power Output

CPO is the rate of hydraulic energy inputted into the systemic vasculature measured at the aortic root. Calculated as the product of mean arterial pressure and CO, divided by 451, CPO at rest typically measures around 1 Watt in healthy individuals, with a significant increase to approximately 6 Watts during exercise (Table 1) [12–14]. CPO is a strong predictor of mortality in chronic HF with peak exercise CPO minus resting CPO reflecting reserve in the system. Those with CPO of < 1Watt had a rate of mortality or heart transplant (HTx) at 12 months greater than 90% [12]. In patients in CS, most or all of the reserve is recruited, therefore resting values obtained at the bedside reflect peak cardiac power and the limitations of the underlying myocardium [10, 12, 13, 15]. In the SHOCK (Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock) trial registry, multivariate analysis revealed that CPO and LV work were the only independent hemodynamic correlates of in-hospital mortality, with CPO being a stronger correlate than LV work. A value of 0.53 Watts was found to be optimal with a positive and negative predictive valve of 58% and 71% respectively for predicting in-hospital mortality [10]. This finding has been replicated in several subsequent studies with various optimal CPO cut-offs proposed ranging from 0.53 to 0.6 Watts [10, 16, 17]. In a cohort of 204 consecutive patients admitted for CS, CPO of < 0.6 Watts at 24 h held an odds ratio of 10.2 for mortality in multi-variable analysis [16]. Importantly, the original CPO calculation included RAP with subsequent iterations excluding this value. In the ESCAPE dataset, CPO excluding RAP was not correlated with outcomes while inclusion of RAP resulted in a significant correlation [18]. CPO is a powerful prognostic marker in CS and has made its way into clinical use as a primary indicator for escalation of hemodynamic support.

Aortic Pulsatility Index

API is a novel marker integrating both left ventricular output and left-sided filling pressure by combining arterial pulse pressure—correlated with stroke volume under fixed systemic compliance—with pulmonary capillary wedge pressure (PCWP), which in the absence of pulmonary venous

Table 1 Summary of novel hemodynamic markers, including calculation formulas, proposed dichotomous thresholds, and association with adverse outcomes in cardiogenic shock (CS) and chronic heart failure (HF). *PAPi: Pulmonary artery pulsatility index; RVSWI: Right ventricular stroke work index; API: Aortic pulsatility index; CPO: Cardiac power output; PAPP: Pulmonary artery pulse pressure; RAP: Right atrial pressure; SVI: Stroke volume index; mPAP: Mean pulmonary artery pressure; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; PCWP: Pulmonary capillary wedge pressure; MAP: Mean arterial pressure; CO: Cardiac output; Ao: Aorta; PA: Pulmonary artery; RA: Right atrium; RV: Right ventricle; LV: Left ventricle; HTx: Heart transplant*

Parameter	How to Determine	Proposed Dichotomous Threshold	Association with Adverse Outcomes
CPO	MAP × CO/451	CS: < 0.6 Chronic HF < 1.0	Lower CPO associated with increased mortality [10, 12, 16, 17]
API	(SBP-DBP)/PCWP	CS: ≥ 1.45 Chronic HF: ≥ 2.9	Higher API associated with freedom from advanced therapies or death [24–26]
PAPi	PAPP/RAP	CS: < 1.0 Chronic HF: < 2.56	Lower PAPi associated with mortality, RV mechanical support and need for advanced therapies [16, 28, 29]
RAP/PCWP	RAP/PCWP	CS: > 0.63 Chronic HF: > 0.63	Higher RAP/PCWP associated with mortality and adverse outcomes [11, 16, 31–33, 35–39]
RSVSWI	SVI × (mPAP-RAP)	N/A	Inconsistently associated with outcomes [23, 29, 37, 40–43]

disease reflects left atrial pressures and thus indicates pulmonary congestion (Table 1) [19–22]. Calculated as systolic BP minus diastolic BP divided by PCWP, API is a LV measurement analogous to the PAPi [23]. In their study of 224 patients, Belkin et al. found that, in univariable analysis, a lower baseline API was significantly associated with progression to advanced therapies or death within 30 days compared to those receiving continued medical management [24]. Similarly, in multivariate analysis, a higher API was strongly associated with freedom from advanced therapies or death after adjusting for baseline characteristics and RHC measurements. Receiver operating characteristic (ROC) analysis identified an optimal cutoff of 1.45, with Kaplan–Meier analysis showing that an API ≥ 1.45 was associated with a higher rate of freedom from the primary outcome (79%) compared to an API < 1.45 (48%) [24]. In a post-hoc analysis of the ESCAPE trial by Belkin et. al., among other predictors including CI, CPO and PAPi, API was the most effective predictor of the composite endpoint of death, HTx, or left ventricular assist device (LVAD) at 6 months. In ROC analysis, API demonstrated the best predictive performance with a cutoff of 2.9, compared to CI, CPO, and PAPI, showing higher sensitivity (76.2%). Kaplan–Meier analyses revealed that an API ≥ 2.9 was associated with a greater likelihood of freedom from the primary outcome compared to an API < 2.9 (83.5% vs 58.4%, $P = 0.001$) [25]. In a single-center study including 453 patients with advanced HF who underwent RHC, API was found to be a significant predictor of freedom from the composite outcome of death, LVAD implantation, total artificial heart implantation or HTx and all-cause mortality in both univariate and multivariate analyses [26]. Recently, API has been identified as a hemodynamic measurement to guide weaning from percutaneous LV mechanical support (Impella® CP) [27]. Through an integration of pressure and volume measurement, API offers a comprehensive assessment of a patient's clinical status in both chronic HF and CS through an easy to calculate single measure. Further insight into the utility of API as a tool to escalate, and de-escalate, mechanical circulatory support will be telling about its broad clinical applicability in the coming years.

Pulmonary Artery Pulsatility Index

PAPi is calculated as PA systolic pressure minus PA diastolic pressure divided by RAP, and serves as an indicator of right heart function based on two key physiological factors: PA pulse pressure, which provides an indirect measure of right ventricle's (RV) contractile performance in relation to afterload; and RAP, a measure of the RV's ability to protect the venous system from the PA pressure (Table 1). By assessing these parameters, PAPi offers insights into the RV's ability to generate adequate pressure against resistance and the extent of RV failure. PAPi has been identified in numerous studies as a predictive marker of outcomes in patients with CS and end-stage HF. Korabathina et al. in a study including patients with acute inferior myocardial infarction and suspected RV dysfunction showed that PAPi had the highest sensitivity (88.9%) and specificity (98.3%) for predicting in-hospital mortality and/or requirement of a percutaneous RV support device. ROC analysis revealed that a PAPi of ≤ 0.9 achieved 100% sensitivity and 98.3% specificity (C-statistic: 0.998) in predicting these outcomes [28]. Tehrani et al. conducted a study with 204 patients with CS and showed that PAPi < 1.0 was an independent predictor of 30-day mortality and was subsequently incorporated into the proposed treatment algorithm for managing CS [16]. In a post-hoc analysis of the ESCAPE trial including 190 patients Kochav et al. highlighted that PAPi was associated with clinical, echocardiographic and hemodynamic signs of RV failure and was an independent predictor of the primary endpoint of death or hospitalization at 6 months. ROC analysis yielded a cutoff value for PAPi of 3.65 (sensitivity 83%, specificity 31%, positive predictive value 71%) [23]. In a study in 416 patients with end-stage HF by Bayram et al., PAPi ≤ 2.56 was associated with a higher adverse cardiac event risk compared to PAPi > 2.56 and could predict the composite outcome of LVAD implantation, HTx or cardiac mortality with 56.7% sensitivity and 51.3% specificity at 1 year [29]. Zern et al. categorized PAPi values of 8285 patients into quartiles (Q1: 0.3–2.2, Q2: 2.21–3.3, Q3: 3.4–5.5, and Q4: 5.6–30.0) and no cutoff was specified in this study. Multivariate analysis revealed that patients in the lowest PAPi quartile had a 60% greater risk of death compared to those in the highest quartile, as well as an increased risk of major adverse cardiac events and HF hospitalizations [30]. PAPi is an important marker for assessing RV function in a variety of clinical settings including CS, decompensated HF, chronic HF and pre-LVAD with strong prognostication of morbidity and mortality. It predicts adverse outcomes such as mortality and the need for advanced therapies, enabling early identification of high-risk patients and guiding timely interventions, which is essential for improving clinical outcomes.

Right Atrial Pressure to Pulmonary Capillary Wedge Pressure Ratio

This advanced hemodynamic marker, calculated as a ratio of RAP/PCWP, reflects the balance between right and left-sided filling pressures, giving insight into RV function (Table 1). Under normal conditions, RAP/PCWP ratio is 0.5 and this "concordant" relationship is present in 75% of patients allowing for an estimate of PCWP based on jugular venous pressure with the other 25% being "discordant" [31,

32]. An elevated RAP/PCWP ratio, representing half of the "discordant" cohort, suggests significant RV dysfunction and is more specific if RAP itself is elevated above normal. Increased RAP/PCWP has been identified as a predictive marker of outcomes in patients with acute myocardial infarction, CS, end-stage HF, and as a predictor of RV failure after LVAD implantation with 0.63 and 0.86 being the most common ratios found to be prognostic [33, 34]. In the setting of acute myocardial infarction, Lopez-Sendon et al. identified that a ratio of RA /PCWP > 0.86 was associated with pathological evidence of RV infarction at necropsy [34]. Several subsequent studies have demonstrated the utility of the RA/PCWP as an index of biventricular congestion and the relative contribution of LV or RV failure [11, 16, 31, 32, 35–37]. In a study of patients with CS from the Critical Care Cardiology Trials Network, RAP/PCWP was significantly associated with in-hospital mortality in both univariate and multivariate analysis [35]. This observation is supported by the finding of a RAP/PCWP > 0.63 at 24 h being statistically associated with 30-day mortality in a cohort of 204 patients with CS [16]. However, in a hemodynamic analysis of patients included in the SHOCK trial and registry, RAP/PCWP was not associated with mortality at 30 days or at discharge [37]. In a post-hoc analysis of the ESCAPE trial database, RAP/PCWP was independently associated with hospitalization or death [32]. Similarly, Grodin et al. in a retrospective analysis containing adult advanced HF patients showed that the association of RAP/PCWP with mortality persisted after multivariable adjustments [11]. Moreover, RAP/PCWP > 0.63 was found to be an independent predictor of RV failure after LVAD implantation [33]. Prognostic significance of RAP/PCWP has also been associated with mortality after LVAD implantation and HTX [31, 33, 38, 39]. In conclusion, RAP/PCWP ratio is a valuable hemodynamic marker that provides insights into biventricular congestion and prognosis with particular insight into RV function and reserve. This advanced hemodynamic marker can help identify patients at high risk of RV failure, guide therapeutic strategies to optimize unloading, and inform decision-making for LVAD implantation.

Right Ventricular Stroke Work Index

Calculated as the product of stroke volume index (SVI) and the difference between mean PA pressure and RAP normalized to body surface area, RVSWI reflects the RV's contractile performance in a given preload/afterload state (Table 1). A low mean PA pressure with an elevated RAP in the setting of moderate or poor SVI suggests decoupling of the RV-PA and inability to recruit further RV contractile function. The results of studies investigating the relationship between RVSWI and outcomes differ according to their populations. Jain et al. in a study including 1414 with CS from the Cardiogenic Shock Working Group (CSWG) showed that RVSWI was among the variables associated with in-hospital mortality in a univariate analysis, with non-survivors having a significantly lower RVSWI than survivors (4.83 g-m/m^2 vs 5.56 g-m/m^2, $P = 0.002$). However, RVSWI was not included as an independent predictor in the multivariate model. Further analyses in the same study revealed that patients with HF related CS, had a significantly higher RVSWI, compared to patients with acute myocardial infarction. Paradoxically, RVSWI was associated with mortality only in patients with HF related CS [40]. Lala et al. in a hemodynamic analysis of patients included in the SHOCK trial and registry demonstrated that RVSWI was not a significant predictor of mortality at 30 days or at discharge [37]. In patients with HF with reduced ejection fraction, lower RVSWI values have been associated with increased HF hospitalization, while in patients with HF with preserved ejection fraction higher RVSWI values were independently associated with renal function [41, 42]. However, in two studies in patients with end-stage HF conducted by Cesini et al. and Kochav et al. RVSWI was not associated with primary outcomes including LVAD implantation, urgent HTx, hospitalization at 6 months and death [23, 43]. In a study with similar population including patients with end-stage HF by Bayram et al., RVSWI was not associated with adverse cardiac events in both univariate and multivariate analysis [29]. Despite RVSWI having clear physiologic relevance as a marker of RV contractile performance, the mixed results regarding its association with outcomes across various populations, particularly in CS, raise concerns about its reliability as a predictive metric in clinical practice and limit its utility in guiding clinical decision-making.

Right Atrial Pressure Higher than Pulmonary Artery Saturation: An Ominous Finding at Right Heart Catheterization

In clinical practice, we observed patients with the unusual constellation of findings of a RAP numerically higher than PA saturation. In order to determine the frequency and the prognostic utility of this finding we conducted a retrospective study, involving 4621 patients who underwent RHC from November 2016 to January 2023 at a single academic medical center. RHC results, including direct pressure measurements and recorded waveforms, were analyzed, with CO and CI calculated using both the indirect Fick (iFick) and thermodilution methods. After excluding 104 patients without PA saturation data, 14 patients were identified with RAP greater than PA saturation. These cases were manually reviewed by a heart failure cardiologist, focusing on end-expiratory values.

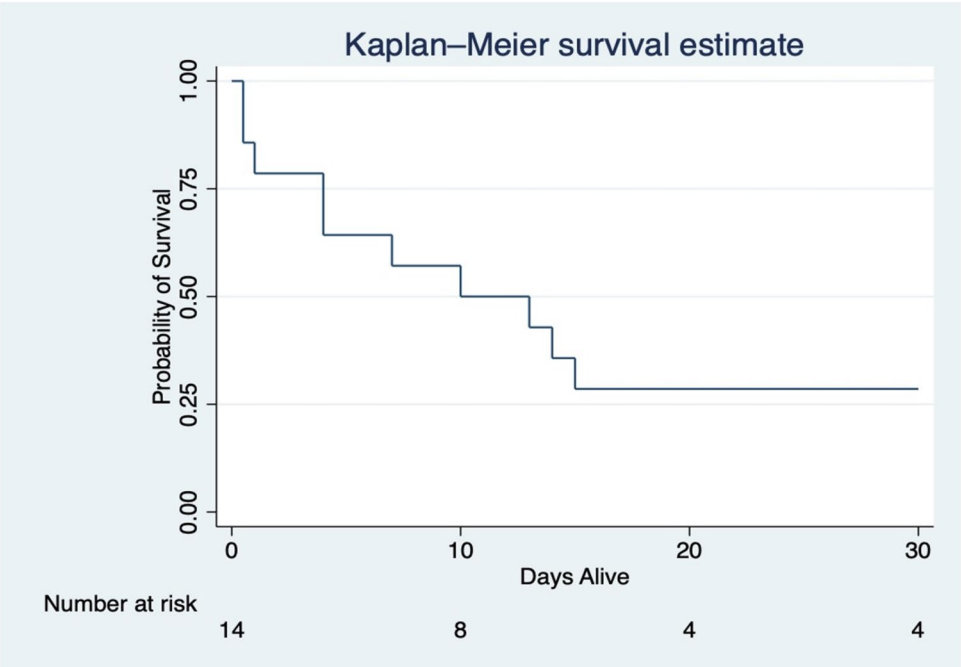

Fig. 1 Kaplan Meier curve for 30-day survival analysis patients with right atrial pressure (RAP) greater than pulmonary artery (PA) saturation

Overall, 14 patients (0.3%) exhibited a RAP numerically higher than PA saturation. The final cohort was predominantly White ($n = 11$, 78.7%), male ($n = 9$, 64.3%), with a mean age of 49.7 ± 14.7 years. The most common cause of shock etiology was nonischemic cardiomyopathy ($n = 6$,42.9%), followed by acute coronary syndrome ($n = 4$, 28.6%). Median [interquartile range] length of stay was 16.5 [6,21] days during index admission with 7 (50%) patients requiring temporary mechanical circulatory support. RAP was 29.1 ± 4.2 mmHg and PA saturation 22.1 ± 4.1%. CO via iFick was 2.6 ± 1.0 L/min and via thermodilution 2.4 ± 0.7 L/min, with a CI of 1.3 ± 0.5 (range: 0.7–2.3) L/min/m2 and 1.2 ± 0.3 (range: 0.7–1.6) L/min/m2 respectively. PA pressures included a systolic pressure of 58.0 ± 11.8 mmHg, and diastolic pressure of 39.4 ± 8.5 mmHg, with a pulmonary capillary wedge pressure of 37.6 ± 10.9 mmHg. Systemic BP was 106.1 ± 29.3 mmHg for systolic and 60.9 ± 20.0 mmHg for diastolic, with an average heart rate of 112.2 ± 30.5 bpm. PAPi was calculated at 0.56 [0.37, 0.82], CPO at 0.38 [0.31, 0.60], and API at 1.19 [0.71,1.75]). Ten out of the 14 patients (71%) died during the first 30 days after their RHC with a median time to death of 11.5 days (Fig. 1). Of the four patients who survived, one was bridged to recovery with venoarterial extracorporeal membrane oxygenation, two with dual inotropes and one received a durable LVAD.

The finding of a RAP numerically higher than the PA saturation is very rare (0.3%) but when found is an ominous finding and portends a very high 30-day mortality rate. While further studies are needed, recognizing the prognostic utility of this finding as part of a broader hemodynamic assessment may provide an additional tool to help guide early decisions regarding the potential need for escalation of circulatory support, ultimately aiding in improved risk stratification and management.

Conclusions

In this review we attempted to underscore the importance of advanced hemodynamic markers in the management of CS and end-stage HF, with CPO, API, PAPi, RAP/PCWP and RVSWI each contributing unique insights into patient prognosis (Fig. 2). Despite the recent reported decrease in the use of PA catheters, the integration of these markers into clinical practice has the potential to improve risk stratification and guide therapeutic decision-making, ultimately leading to better outcomes for patients with CS and end-stage HF. The identification of novel hemodynamic patterns, such as RAP greater than PA saturation, may offer the opportunity to improve phenotyping of patients with CS and allow for increased personalization of CS care. However, variability in the predictive thresholds and the need for further validation of these markers in diverse patient populations highlight the ongoing need for research in this area.

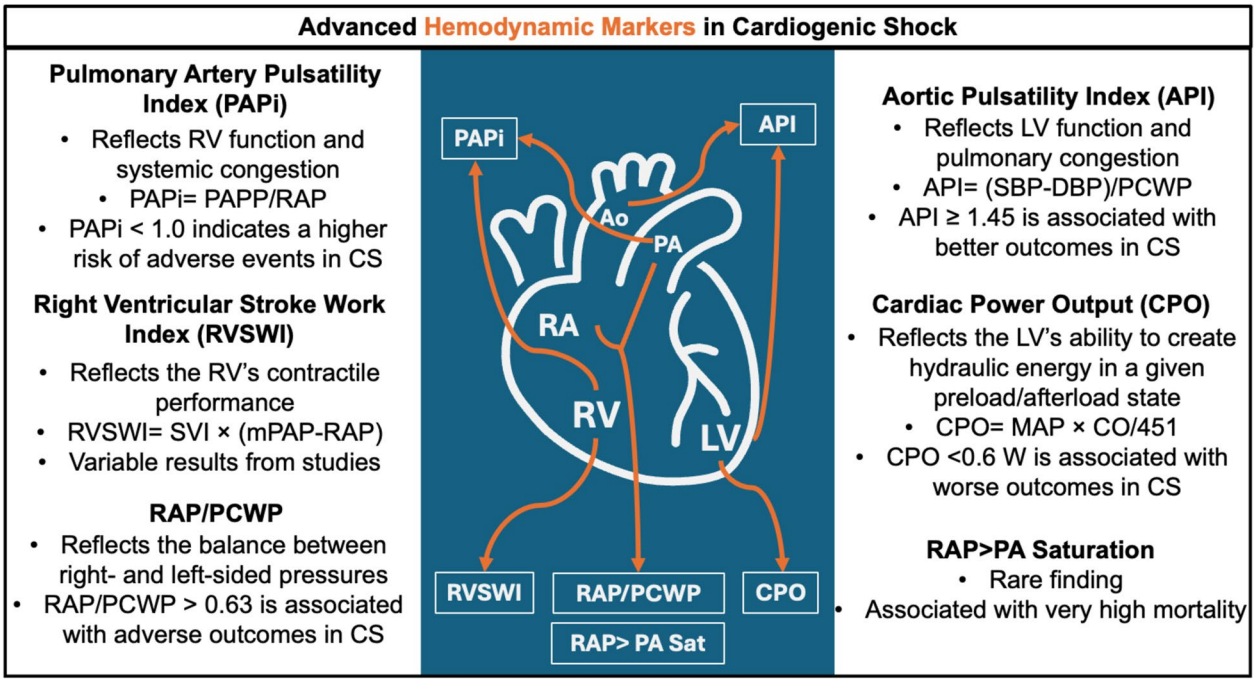

Fig. 2 Summary of the advanced hemodynamic markers in cardiogenic shock. *PAPi: Pulmonary artery pulsatility index; RVSWI: Right ventricular stroke work index; API: Aortic pulsatility index; CPO: Cardiac power output; PAPP: Pulmonary artery pulse pressure; RAP: Right atrial pressure; SVI: Stroke volume index; mPAP: Mean pulmonary artery pressure; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; PCWP: Pulmonary capillary wedge pressure; MAP: Mean arterial pressure; CO: Cardiac output; Ao: Aorta; PA: Pulmonary artery; RA: Right atrium; RV: Right ventricle; LV: Left ventricle*

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Data Availability No datasets were generated or analysed during the current study.

Declarations

Competing Interests Sotiria Liori serves as an associate editor of this journal.

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