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Prognostic value of right atrial pressure-corrected cardiac power index in cardiogenic shock

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Abstract

Aim The pulmonary artery catheter (PAC)-derived cardiac power index (CPI) has been found of prognostic value in cardiogenic shock (CS) patients. The original CPI equation included the right atrial pressure (RAP), accounting for heart filling pressure as a determinant of systolic myocardial work, but this term was subsequently omitted. We hypothesized that the original CPI formula (CPI_{RAP}) is superior to current CPI for risk stratification in CS.

Methods and results A single-centre cohort of 80 consecutive Society for Cardiovascular Angiography and Interventions (SCAI) B-D CS patients with available PAC records was included. Overall in-hospital mortality was 21.3%. Results showed CPI_{RAP} to be the strongest haemodynamic predictor of in-hospital death (p_{adj} = 0.038), outperforming CPI [area under the receiver operating characteristic (ROC) curves: 0.726 and 0.673, *P*-for-difference = 0.025]. When the population was stratified according to the identified CPI_{RAP} (0.28 W/m^2) and accepted CPI (0.32 W/m^2) thresholds, the cohort with discordant indexes (low CPI_{RAP} and high CPI) comprised a group of 13 patients featuring a congested phenotype with frequent right ventricle or biventricular involvement. In this group, in-hospital mortality was high (30.8%) similar to those with concordant low CPI and CPI_{RAP}.

Conclusion Incorporating RAP in CPI calculation (CPI_{RAP}) improves the prognostic yield in patients with CS SCAI B-D. A cut-off of 0.28 W/m^2 identifies patients at higher risk of in-hospital mortality. The improved prognostic value of CPI_{RAP} may derive from identification of patients with more intravascular congestion who may experience substantial in-hospital mortality, uncaptured by the commonly used CPI equation.

Introduction

Cardiogenic shock (CS) is a progressively increasing admission diagnosis in cardiac intensive care units (CICU).[1] A significant proportion of CS patients requiring inotropic and/or mechanical circulatory support (MCS) receives invasive haemodynamic monitoring with a pulmonary artery catheter (PAC).[2–4] PAC-derived measures of cardiac hydraulic power and vascular congestion may offer indirect information on

myocardial contractile state and cardiac function and help guide therapeutic interventions. Retrospective data suggest improved outcomes with PAC use in CS patients.[5–7]

Availability of these invasive measures allows calculation of traditional hydraulic indexes. In Guyton's cardiovascular model, both right ventricle (RV) and left ventricle (LV) are viewed as a unified pump where right atrial pressure (RAP) represents the input and mean arterial pressure (MAP) and cardiac output (CO) or cardiac index (CI) represent the

outputs.[8] Cardiac power output (CPO) and the body surface area (BSA)-indexed cardiac power index (CPI) are directly proportional to the product of MAP and CO or CI, respectively, and are indexes of the mechanical power generated by the heart pump.[9] CPO and CPI have been shown to be powerful predictors of outcomes both in heart failure and CS patients.[9–12] An accepted CPO threshold of 0.53 W and CPI threshold of 0.32 W/m^2 identify patients at risk of dismal prognosis in acute heart failure (AHF) and CS scenarios.[10,13,14] In its current, 'usual', and most widely used form, the formula to calculate CPI is

$$CPI = \frac{MAP \times CI}{451}$$

However, in its original description by Tan,[9] CPI was defined as

$$CPI_{RAP} = \frac{(MAP - RAP) \times CI}{451}$$

The physiological and mathematical foundations of this second formula have been recently revisited by Lim.[15] Incorporation of RAP in the calculation eliminates the work done to the heart pump due to filling and distention of the chambers and is represented by the area below the end-diastolic pressure–volume relationship and between the end-diastolic and end-systolic volumes in the pressure–volume loop plane. As such, CPI$_{RAP}$ more accurately reflects the net power generated by active contraction. It is obvious that at high MAP and low RAP conditions (i.e. normal physiology), the contribution of RAP is modest and may be omitted without major difference in the resulting CPI value. However, in scenarios characterized by low MAP and high RAP, common features in CS, CPO, and CPI may be significantly affected by inclusion of RAP in the calculation.

We therefore hypothesized that inclusion of RAP in calculating CPI (CPI$_{RAP}$), to reflect the true systolic power generated by the heart, may provide superior prognostication compared with that provided by CPI in the settings of AHF and CS.

Methods

Study design

We reviewed all available PAC measurements prospectively recorded in our centralized CICU (IRCCS 'San Raffaele Hospital', Milan, Italy) database from June 2020 to September 2021. We selected all consecutive patients meeting the Society for Cardiovascular Angiography and Interventions (SCAI) B to D CS stages according to the SCAI classification.[16] Indication to PAC insertion was left to the treating physician based on clinical profile and concomitant

need of MCS. All PAC assessments were prospectively registered in a dedicated database at the time of haemodynamic assessment. Haemodynamic measures were performed by dedicated personnel (LB, FC, MG, VP, and SS) with expertise in PAC insertion, management, and invasive data interpretation with the aim of obtaining complete haemodynamic profiling.[6] All patients who received PAC also had invasive arterial pressure monitoring. Index PAC assessment was used for the purpose of this analysis. Laboratory tests were performed at the time of PAC insertion. Medical records for clinical, imaging, and laboratory data were reviewed by two of the authors (GB and GG) blinded to the selected study outcome and to the study design.

Phenotypes of right, left, and biventricular failure are presented according to the cut-off of pulmonary capillary wedge pressure (PCWP, 18 mmHg) and RAP (12 mmHg) from the Cardiogenic Shock Working Group (CSWG) registry.[17,18]

The outcome of interest was in-hospital mortality.

Statistical analyses

Categorical variables are reported as proportions, whereas continuous variables are reported as median and interquartile range (IQR). Continuous variables from independent groups were compared by the Kruskal–Wallis rank sum test, and the categorical variables with Fisher's exact test.

A univariable binary logistic regression was obtained to identify variables significantly associated with the outcome of interest. After univariable analysis, a purposeful selection of covariates was performed to test the multivariable association of the variables of interest. Considering the low number of events, multiple models with backward selection with a limited number of covariates (all with univariable $P < 0.010$) were evaluated in order to avoid overfitting.

Receiver operating characteristic (ROC) curves were obtained to assess and compare the discriminatory power of CPI$_{RAP}$ and CPI. The DeLong test was used to compare the ROC curves. The 'closest topleft' point method was used to identify the optimal CPI$_{RAP}$ cut-off for predicting the outcome of interest, and patients were accordingly stratified.

Survival during hospital stay was also evaluated according to the Kaplan–Meier method, and survival among groups stratified by the identified CPI$_{RAP}$ cut-off was compared with the Breslow test. A $P < 0.05$ was considered statistically significant. All analyses were performed with RStudio (Version 1.3.1093, RStudio, PBC).

Results

In the study time span, a total of 177 patients meeting SCAI B through D criteria were admitted to our CICU. Of these, 80 patients had one available baseline PAC haemodynamic

assessment and were included in this study. Median age was 65 (56, 72) years, and 63 (78.8%) patients were males. On admission, 76 (95.0%) were on SCAI stage B or C. Median BSA was 1.93 (1.79, 2.09) m^2. CS aetiology was acute coronary syndrome (ACS) in 27 (33.8%) and acute decompensated heart failure (ADHF) in 53 (66.2%). Serum lactate on admission was 2.75 (1.50, 6.18) mmol/L. Left ventricular ejection fraction (LVEF) was 20 (15, 27)%. On haemodynamic assessment, heart rate was 95 (85, 111) b.p.m., MAP was 81 (74, 88) mmHg, CI was 2.23 (1.79, 2.93) L/min/m^2, CPI was 0.40 (0.32, 0.53) W/m^2, and CPI$_{RAP}$ was 0.31 (0.23, 0.40) W/m^2. During ICU stay, intra-aortic balloon pump was implanted in 45 (56.2%) patients, Impella pump was implanted in 14 (17.5%) patients, and veno-arterial extracorporeal membrane oxygenation (ECMO) was run in 3 (3.8%) patients. Median ICU stay was 12 (6, 19) days, and in-hospital stay was 16 (11, 31) days. A total of 6 (7.4%) patients underwent LVAD implant, and 1 (1.2%) underwent heart transplant. In-hospital death occurred in 17 (21.3%) patients.

Study cohort baseline clinical and haemodynamic variables, according to in-hospital death outcome, are summarized in *Table 1*. Non-survivors were older, had worse right ventricle systolic function on echocardiography, more often received mechanical ventilation, and, at PAC assessment, presented with lower CPI and CPI$_{RAP}$, lower stroke volume, and higher degree of pulmonary circulation overload.

Univariate and multivariate analyses for in-hospital death

Univariate analysis identified several variables associated with in-hospital death, including age, SCAI stage D CS, systolic pulmonary artery pressure (sPAP), CPI, and CPI$_{RAP}$. In a multivariable model including age and CPI$_{RAP}$, both age [odds ratio (OR) = 1.07; 95% confidence interval (95%CI): 1.02–1.15; p_{adj} = 0.018] and CPI$_{RAP}$ (OR = 0.54; 95%CI: 0.27–0.92 for 0.1 W/m^2 increase; p_{adj} = 0.043) were significantly associated with the outcome of in-hospital death (*Table 2*). CPI$_{RAP}$ remained significantly associated with in-hospital death also when adjusted for sPAP (Model 2), SCAI stage on admission (Model 3), CI (Model 4), and stroke volume index (Model 5). Summary statistics of these models are available in the Supporting Information. Unadjusted estimated in-hospital death probability based on CPI$_{RAP}$ and CPI values is presented in *Figure 1*.

Based on ROC analysis, CPI$_{RAP}$ outperformed both CPI and CI for the in-hospital death outcome (*Figure 2*). The area under the curve (AUC) was 0.725 (95%CI: 0.589–0.862) for CPI$_{RAP}$, 0.673 (95%CI: 0.529–0.817) for CPI, and 0.626 (95%CI: 0.481–0.771) for CI. Based on the DeLong test, the AUC for CPI$_{RAP}$ was significantly higher than that of the other two indexes (CPI$_{RAP}$ vs. CPI: P = 0.025; CPI$_{RAP}$ vs. CI: P = 0.047). The closest topleft point method identified a

CPI$_{RAP}$ value of 0.28 W/m^2 as the optimal threshold value for the in-hospital death outcome, with a specificity of 0.76 and a sensitivity of 0.59.

Based on a survival analysis with Kaplan–Meier estimates (*Figure 3*), patients with a CPI$_{RAP} \leq$ 0.28 W/m^2 demonstrated higher 30 day mortality: 40 (SE 9.8)% vs. 24.6 (SE 10.4)%, P = 0.023.

Reclassification analyses

We explored the impact of population reclassification by the identified CPI$_{RAP}$ threshold of 0.28 W/m^2 as compared to the accepted CPI threshold of 0.32 W/m^2. A total of 22 (27.5%) patients demonstrated concordantly 'low' CPI and CPI$_{RAP}$, 45 (56.3%) patients demonstrated concordantly 'high' CPI and CPI$_{RAP}$, and 13 (16.2%) patients demonstrated discordantly 'high' CPI and 'low' CPI$_{RAP}$. Therefore, among those with 'high' CPI, 22.4% was reclassified as 'low' CPI$_{RAP}$.

We then assessed clinical and haemodynamic characteristics of the three groups defined according to CPI and CPI$_{RAP}$ agreement (*Table 3*). Notably, the discordant group showed a greater proportion of reduced tricuspid annular plane systolic excursion (TAPSE) on admission as compared to concordantly high and concordantly low groups (61.5 vs. 25.0 vs. 16.7%; P = 0.048), and of note, no difference in tricuspid regurgitation severity was found between strata. The discordant group also demonstrated higher serum creatinine values (P = 0.041) and CSWG-defined biventricular congestion (69.2 vs. 28.9 vs. 45.5%; P = 0.033). They also exhibited an intermediate PAC haemodynamic profile between those in the concordantly high and low groups in terms of CI, MAP, systemic and pulmonary resistances, and LV and RV stroke work. Nevertheless, the discordant group presented features of greater pulmonary overload including the highest PCWP [24 (21, 27) vs. 18 (13, 23) vs. 18 (13, 25) mmHg; P = 0.016] and highest mean pulmonary artery pressure [mPAP; 40 (32, 45) vs. 29 (22, 38) vs. 29 (24, 40) mmHg; P = 0.048] and a nominally lower pulmonary artery pulsatility index (PAPi).

In-hospital mortality was significantly different across groups and measured at 30.8 vs. 11.1 vs. 36.4% (P = 0.026) for discordant, concordantly high, and concordantly low cohorts, respectively (*Figure 4*).

Discussion

The main findings of this study may be summarized as follows (*Figure 5*):

- CPI calculation with inclusion of RAP (CPI$_{RAP}$) was the strongest haemodynamic variable associated with in-hospital mortality, proving superior to usual CPI.
- The CPI$_{RAP}$ 0.28 W/m^2 threshold best identifies patients at greater risk of in-hospital death.

Table 1 Baseline characteristics and invasive haemodynamic according to in-hospital death outcome

	Overall (*n* = 80)	In-hospital survival (*n* = 63)	In-hospital death (*n* = 17)	*P*-value
Clinical features				
Males	63 (78.8%)	51 (81.0%)	12 (70.6%)	0.340
Age	67 (57, 74)	66.00 (55, 73)	74 (68, 79)	**0.002**
Aetiology				0.152
ACS	24 (38.1%)	3 (17.6%)	27 (33.8%)	
ADHF	39 (61.9%)	14 (82.4%)	53 (66.2%)	
Serum creatinine (mg/dL)	1.56 (1.12, 2.26)	1.52 (1.12, 2.20)	1.80 (1.17, 2.49)	0.393
Total bilirubin (mg/dL)	1.00 (0.61, 1.64)	1.00 (0.51, 1.61)	1.30 (0.80, 2.00)	0.192
Direct bilirubin (mg/dL)	0.57 (0.30, 0.90)	0.48 (0.28, 0.81)	0.80 (0.51, 1.12)	**0.022**
Known coronary artery disease				1.000
Single-vessel	18 (23.4%)	14 (23.3%)	4 (23.5%)	
Two-vessel	10 (13.0%)	8 (13.3%)	2 (11.8%)	
Three-vessel	13 (16.9%)	10 (16.7%)	3 (17.6%)	
Pre-existing HFrEF	35 (46.1%)	26 (43.3%)	9 (56.2%)	0.701
Admission SpO$_2$ (%)	98.5 (96.8, 100.0)	98.0 (97.0, 100.0)	99.0 (96.0, 100.0)	0.484
Admission lactate (mmol/L)	2.75 (1.50, 6.18)	2.40 (1.43, 6.18)	4.03 (2.10, 6.00)	0.201
Admission LVEF (%)	20 (15, 27)	20 (15, 26)	20 (15, 33)	0.618
Admission RV dysfunction (echo)				**0.024**
TAPSE <18	18 (25.4%)	16 (28.1%)	2 (14.3%)	
TAPSE 14–10	16 (22.5%)	11 (19.3%)	5 (35.7%)	
TAPSE <10	21 (29.6%)	14 (24.6%)	7 (50.0%)	
Tricuspid regurgitation				0.061
None/trace	5 (6.2%)	5 (7.9%)	0 (0.0%)	
Mild	41 (51.2%)	36 (57.1%)	5 (29.4%)	
Moderate	23 (28.8%)	15 (23.8%)	8 (47.1%)	
Severe	11 (13.8%)	7 (11.1%)	4 (23.5%)	
Intensive care management				
Any inotrope	63 (85.1%)	48 (81.4%)	15 (100.0%)	0.106
IABP support	45 (56.2%)	34 (54.0%)	11 (64.7%)	0.583
Impella support	14 (17.5%)	12 (19.0%)	2 (11.8%)	0.722
VA-ECMO support	3 (3.8%)	3 (4.8%)	0 (0.0%)	1.000
Mechanical ventilation	43 (59.7%)	30 (52.6%)	13 (86.7%)	**0.019**
Haemodynamic profile				
CSWG isolated LV congestion	16 (20.0%)	11 (17.5%)	5 (29.4%)	0.312
CSWG isolated RV congestion	6 (7.5%)	6 (9.5%)	0 (0.0%)	0.333
CSWG biventricular congestion	32 (40.0%)	24 (38.1%)	8 (47.1%)	0.581
CSWG RV or biventricular congestion	38 (47.5%)	30 (47.6%)	8 (47.1%)	1.000
CSWG euvolemic	26 (32.5%)	16 (35.6%)	1 (7.7%)	0.098
SCAI CS stages				0.056
B	38 (47.5%)	31 (49.2%)	7 (41.2%)	
C	38 (47.5%)	31 (49.2%)	7 (41.2%)	
D	4 (5.0%)	1 (1.6%)	3 (17.6%)	
Invasive haemodynamics				
CPI (W/m^2)	0.40 (0.32, 0.53)	0.43 (0.36, 0.55)	0.34 (0.26, 0.40)	**0.030**
CPI$_{RAP}$ (W/m^2)	0.31 (0.23, 0.40)	0.32 (0.25, 0.44)	0.23 (0.17, 0.32)	**0.005**
CI (L/min/m^2)	2.23 (1.79, 2.93)	2.30 (1.85, 3.05)	2.00 (1.50, 2.50)	0.112
HR (b.p.m.)	95 (85, 111)	94 (87, 110)	98 (80, 114)	0.724
MAP (mmHg)	81 (74, 88)	81 (75, 89)	77 (74, 82)	0.193
SV (mL)	47 (35, 63)	51 (36, 65)	37 (30, 48)	**0.027**
SVi (mL/m^2)	24 (19, 32)	26 (19, 35)	23 (17, 25)	0.110
PCWP (mmHg)	19 (14, 25)	18 (13, 24)	21 (19, 25)	0.143
RAP (mmHg)	11 (8, 15)	11 (8, 15)	11 (8, 17)	0.671
mPAP (mmHg)	30 (24, 40)	29 (24, 39)	39 (32, 42)	**0.033**
sPAP (mmHg)	42 (33, 59)	40 (33, 55)	58 (47, 69)	**0.013**
dPAP (mmHg)	22 (19, 29)	21 (18, 28)	28 (23, 29)	0.053
SVR (WU)	15.60 (10.78, 22.50)	14.90 (10.75, 21.50)	19.40 (14.30, 24.10)	0.146
PVR (WU)	2.54 (1.48, 3.81)	2.07 (1.46, 3.62)	3.81 (2.54, 6.17)	**0.008**
PAPi	1.74 (1.32, 2.85)	1.60 (1.17, 2.62)	2.45 (1.87, 3.58)	0.080
LVSWi (cJ/m^2)	20.01 (15.58, 28.29)	21.90 (16.41, 28.68)	17.25 (12.68, 19.43)	**0.007**
RVSWi (cJ/m^2)	7.12 (4.76, 8.84)	7.02 (4.58, 9.13)	7.96 (5.74, 8.21)	0.939

ACS, acute coronary syndrome; ADHF, acute decompensated heart failure; CI, cardiac index; CPI, cardiac power index; CSWG, Cardiogenic Shock Working Group; CPI, cardiac power index; dPAP, diastolic pulmonary artery pressure; HFrEF, heart failure with reduced ejection fraction; HR, heart rate; IABP, intra-aortic balloon pump; LV, left ventricle; LVEF, left ventricular ejection fraction; LVSW, left ventricular stroke work index; MAP, mean arterial pressure; mPAP, mean pulmonary artery pressure; PAPi, pulmonary artery pulsatility index; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistances; RAP, right atrial pressure; RV, right ventricle; RVSW, right ventricular stroke work index; SCAI, Society for Cardiovascular Angiography and Interventions; sPAP, systolic pulmonary artery pressure; SpO$_2$, peripheral oxygen saturation; SV, stroke volume; SVi, stroke volume index; SVR, systemic vascular resistances; TAPSE, tricuspid annular plane systolic excursion; VA-ECMO, veno-arterial extracorporeal membrane oxygenation. Categorial variables are expressed as count and proportions, and continuous variable as medians (25th–75th quartiles).
Values in bold are significant P-values <0.05

**Table 2** Logistic regression analysis (in-hospital death outcome)

Variable	OR (95%CI)	P-value	OR (95%CI)	P_{adj}-value
	Univariate logistic regression		Multivariate logistic regression	
Aetiology (ADHF)	2.87 (0.83–13.4)	0.125	—	—
Age (years)	1.09 (1.03–1.17)	**0.006**	1.07 (1.02–1.15)	**0.018**
SCAI (SCAI C)	1.00 (0.31–3.25)	1.000	—	—
SCAI (SCAI D)	13.29 (1.46–292.38)	**0.035**	—	—
Serum creatinine (mg/dL)	0.98 (0.17–1.04)	0.712	—	—
Serum lactate (mmol/L)	1.01 (0.99–1.12)	0.576	—	—
LVEF (%)	0.99 (0.94–1.04)	0.881	—	—
CI (L/min/m^2)	0.54 (0.24–1.02)	0.092	—	—
MAP (mmHg)	0.96 (0.91–1.01)	0.162	—	—
sPAP (mmHg)	1.04 (1.01–1.08)	**0.014**	—	—
SVi (mL/m^2)	0.94 (0.87–1.00)	0.065	—	—
CPI (W/m^2)[a]	0.62 (0.38–0.92)	**0.034**	—	—
CPI_{RAP} (W/m^2)[a]	0.48 (0.24–0.81)	**0.015**	0.54 (0.27–0.92)	**0.043**
SVR (WU)	1.04 (0.97–1.11)	0.228	—	—

ADHF, acute decompensated heart failure; CI, cardiac index; CPI, cardiac power index; LVEF, left ventricular ejection fraction; MAP, mean arterial pressure; OR, odds ratio; SCAI, Society for Cardiovascular Angiography and Interventions; sPAP, systolic pulmonary arterial pressure; SVi, stroke volume index; SVR, systemic vascular resistances; 95%CI, 95% confidence interval.
[a]Coefficients are presented for a 0.1 W/m^2 increase in the CPI and CPI_{RAP} indexes.
Values in bold are significant P-values <0.05

Figure 1 Unadjusted estimated in-hospital mortality probability by CPI_{RAP} (left panel) and CPI (right panel) with 95% confidence intervals. The dashed line (left panel) identifies the CPI_{RAP} 0.28 W/m^2 threshold identified as the best cut point at receiver operating characteristic curves (see text for details). The dashed line (right panel) identifies the accepted CPI threshold at 0.32 W/m^2. CPI, cardiac power index.
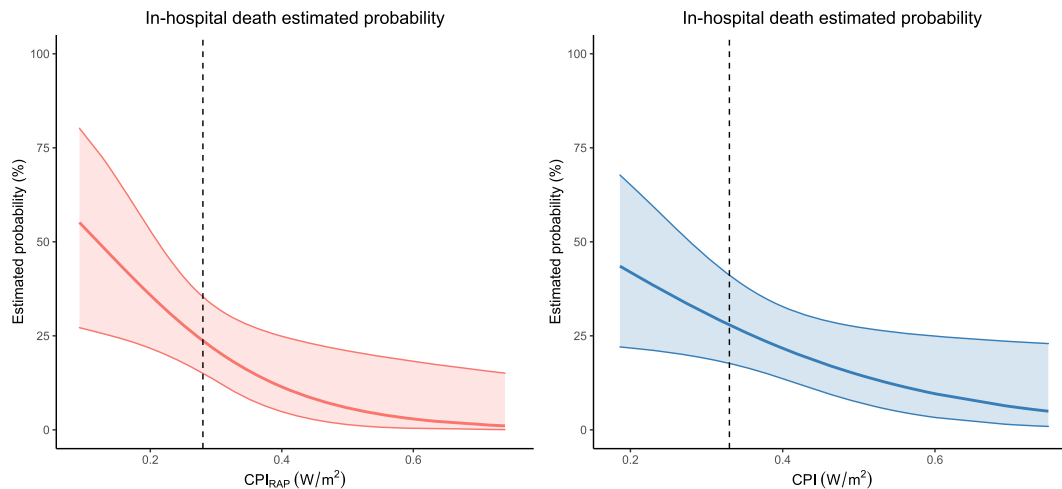
- Those patients with 'high' CPI reclassified as 'low' CPI_{RAP} (≤0.28 W/m^2) experienced substantial in-hospital mortality.
- Patients with 'high' CPI reclassified as 'low' CPI_{RAP} comprised a phenotype of congestion with frequent RV or biventricular involvement.

The increasing incidence of CS, coupled with persistently high rates of CS-related mortality, has led to a reappraisal of invasive haemodynamic monitoring with PAC.[4,19] Several variables provide an outline of cardiovascular physiology and may improve clinical management of CS patients. The CPO and CPI, measures of ventricular pump power, emerged as important prognostic markers in CS.[9–11] In its original description by Tan et al., a normal resting CPO of 1 W was derived, assuming a normal cardiovascular 'physiology' (arterial blood pressure 120/80 mmHg; RAP 3 mmHg; CO 5 L/min). Several subsequent publications omitted the RAP term from the equation for calculations of both CPO and CPI. Although the contribution of RAP may be marginal in euvolemic, stable, ambulatory patients, this omission may introduce an important error at lower MAP and higher RAP, a common haemodynamic profile in CS patients. Notably, venous congestion is common in CS and may be present in up to 58% of patients[18]: when both lower MAP and higher RAP values are present, the discrepancy between calculations is greatest. From a clinical perspective,

Figure 2 Receiver operating characteristic curves for CPI$_{RAP}$, CPI, and CI for the endpoint of interest of in-hospital mortality. AUC, area under the curve; CPI, cardiac power index.
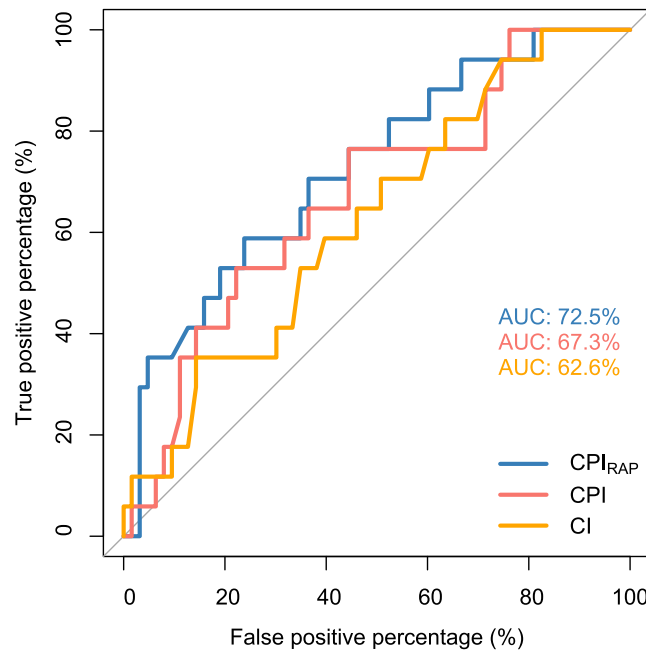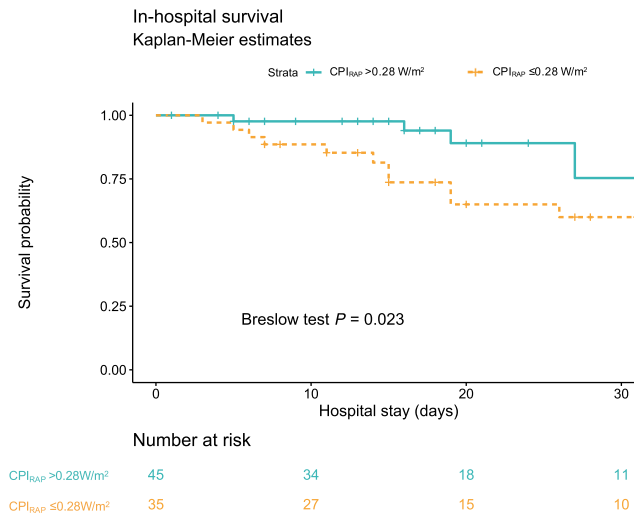

Figure 3 Kaplan–Meier curves showing in-hospital survival stratified according to the CPI$_{RAP}$ 0.28 W/m^2 threshold. Follow-up days are truncated at Day 30. CPI, cardiac power index.

CPI calculation with the currently widespread formula systematically overestimates heart hydraulic power. The pathophysiological reason underlying this concept is that by omitting RAP from the calculation, the diastolic chamber filling power (which represents work done to the heart) is included in the calculation, thus overestimating true cardiac systolic power (Supporting Information).

Our study demonstrates the independent prognostic relevance of CPI$_{RAP}$ (i.e. a CPI calculated with the inclusion of the RAP value). The CPI$_{RAP}$ index better discriminated those patients who would experience in-hospital death than CPI or CI. Based on the identified cut-off of 0.28 W/m^2, we explored the clinical and haemodynamic characteristics of the groups classified on the agreement between CPI and CPI$_{RAP}$. Patients

Table 3 Baseline characteristics and invasive haemodynamic according to CPI and CPI$_{RAP}$ agreement groups

	Overall ($n = 80$)	Concordantly high ($n = 45$)	Discordant ($n = 13$)	Concordantly low ($n = 22$)	*P*-value
Clinical features					
Males	63 (78.8%)	34 (75.6%)	12 (92.3%)	17 (77.3%)	0.486
Age	67 (57, 74)	65 (56, 72)	68 (55, 78)	73 (67.3, 76.8)	0.064
Aetiology					0.999
ACS	27 (33.8%)	15 (33.3%)	4 (30.8%)	8 (36.4%)	
ADHF	53 (66.2%)	30 (66.7%)	9 (69.2%)	14 (63.6%)	
Serum creatinine (mg/dL)	1.56 (1.12, 2.26)	1.46 (1.02, 1.86)	2.50 (1.70, 3.09)	1.70 (1.17, 2.50)	**0.041**
Total bilirubin (mg/dL)	1.00 (0.61, 1.64)	1.00 (0.56, 1.72)	1.16 (0.80, 1.42)	1.08 (0.62, 1.99)	0.859
Direct bilirubin (mg/dL)	0.57 (0.30, 0.90)	0.57 (0.27, 0.90)	0.70 (0.37, 1.00)	0.49 (0.30, 0.86)	0.649
Known coronary artery disease					0.414
Single vessel	18 (23.4%)	11 (25.6%)	3 (23.1%)	4 (19.0%)	
Two vessels	10 (13.0%)	5 (11.6%)	3 (23.1%)	2 (9.5%)	
Three vessels	13 (16.9%)	5 (11.6%)	1 (7.7%)	7 (33.3%)	
Pre-existing HFrEF	35 (46.1%)	19 (44.2%)	5 (38.5%)	11 (55.0%)	0.749
Admission SpO$_2$ (%)	98.5 (96.8, 100.0)	99.0 (97.3, 100.0)	97.0 (96.0, 100.0)	98.0 (94.0, 100.0)	0.425
Admission lactate (mmol/L)	2.75 (1.50, 6.18)	1.80 (1.26, 5.40)	4.03 (1.96, 6.18)	5.00 (2.10, 9.00)	0.064
Admission LVEF (%)	20 (15, 27)	20 (15, 31)	19 (15, 25)	20 (15, 27)	0.666
Admission RV dysfunction (echo)					**0.048**
TAPSE <18	18 (25.4%)	8 (20.0%)	3 (23.1%)	7 (38.9%)	
TAPSE 14–10	16 (22.5%)	9 (22.5%)	2 (15.4%)	5 (27.8%)	
TAPSE <10	21 (29.6%)	10 (25.0%)	8 (61.5%)	3 (16.7%)	
Tricuspid regurgitation					0.825
None/trace	5 (6.2%)	4 (8.9%)	0 (0.0%)	1 (4.5%)	
Mild	41 (51.2%)	25 (55.6%)	6 (46.2%)	10 (45.5%)	
Moderate	23 (28.8%)	10 (22.2%)	5 (38.5%)	8 (36.4%)	
Severe	11 (13.8%)	6 (13.3%)	2 (15.4%)	3 (13.6%)	
Intensive care management					
Any inotrope	63 (85.1%)	35 (83.3%)	11 (84.6%)	17 (89.5%)	0.906
IABP support	45 (56.2%)	24 (53.3%)	9 (69.2%)	12 (54.5%)	0.641
Impella support	14 (17.5%)	6 (13.3%)	2 (15.4%)	6 (27.3%)	0.362
VA-ECMO support	3 (3.8%)	2 (4.4%)	1 (7.7%)	0 (0.0%)	0.562
Mechanical ventilation	43 (59.7%)	21 (51.2%)	9 (69.2%)	13 (72.2%)	0.274
Haemodynamic profile					
CSWG isolated LV congestion	16 (20.0%)	11 (24.4%)	3 (23.1%)	2 (9.1%)	0.338
CSWG isolated RV congestion	6 (7.5%)	5 (11.1%)	0 (0.0%)	1 (4.5%)	0.602
CSWG biventricular congestion	32 (40.0%)	13 (28.9%)	9 (69.2%)	10 (45.5%)	**0.033**
CSWG RV or biventricular congestion	38 (47.5%)	18 (40.0%)	9 (69.2%)	11 (50.0%)	0.178
CSWG euvolemic	26 (32.5%)	16 (35.6%)	1 (7.7%)	9 (40.9%)	0.098
SCAI CS stages					0.291
B	38 (47.5%)	24 (53.3%)	4 (30.8%)	10 (45.5%)	
C	38 (47.5%)	20 (44.4%)	7 (53.8%)	11 (50.0%)	
D	4 (5.0%)	1 (2.2%)	2 (15.4%)	1 (4.5%)	
Invasive haemodynamics					
CPI (W/m^2)	0.40 (0.32, 0.53)	0.52 (0.44, 0.60)	0.37 (0.36, 0.39)	0.26 (0.24, 0.30)	<**0.001**
CPI$_{RAP}$ (W/m^2)	0.31 (0.23, 0.40)	0.39 (0.34, 0.49)	0.26 (0.25, 0.28)	0.20 (0.17, 0.22)	<**0.001**
CI (L/min/m^2)	2.23 (1.79, 2.93)	2.73 (2.26, 3.54)	2.06 (2.00, 2.33)	1.50 (1.33, 1.74)	<**0.001**
HR (b.p.m.)	95 (85, 111)	98 (90, 113)	93 (86, 110)	92 (73, 103)	0.221
MAP (mmHg)	81 (74, 88)	84 (76, 92)	81 (76, 85)	77 (73, 81)	**0.047**
SV (mL)	47 (35, 63)	59 (44, 67)	41 (39, 50)	31 (26, 37)	<**0.001**
SVi (mL/m^2)	24 (19, 32)	31 (23, 37)	22 (18, 25)	18 (15, 22)	<**0.001**
PCWP (mmHg)	19 (14, 25)	18 (13, 23)	24 (20, 25)	18 (13, 25)	0.016
RAP (mmHg)	11 (8, 15)	11 (7, 14)	12 (11, 17)	12 (7, 17)	0.182
mPAP (mmHg)	30 (24, 40)	29 (22, 38)	40 (32, 45)	29 (24, 40)	**0.048**
sPAP (mmHg)	42 (33, 59)	41 (31, 54)	51 (40, 66)	44 (33, 59)	0.109
dPAP (mmHg)	22 (19, 29)	21 (16, 28)	27 (24, 32)	22 (19, 30)	0.064
SVR (WU)	15.60 (10.78, 22.50)	13.30 (9.50, 18.60)	16.50 (12.60, 19.30)	23.45 (18.05, 27.65)	<**0.001**
PVR (WU)	2.54 (1.48, 3.81)	1.81 (1.43, 2.71)	3.05 (2.07, 3.81)	3.74 (3.16, 6.15)	<**0.001**
PAPi	1.74 (1.32, 2.85)	2.00 (1.40, 2.80)	1.53 (1.08, 3.25)	1.80 (1.18, 2.56)	0.622
LVSWi (cJ/m^2)	20.01 (15.58, 28.29)	27.46 (21.54, 31.64)	17.25 (14.13, 18.48)	13.52 (10.92, 16.93)	<**0.001**
RVSWi (cJ/m^2)	7.12 (4.76, 8.84)	8.20 (6.30, 9.45)	6.83 (5.39, 8.37)	4.29 (2.91, 6.27)	<**0.001**

ACS, acute coronary syndrome; ADHF, acute decompensated heart failure; CI, cardiac index; CPI, cardiac power index; CSWG, Cardiogenic Shock Working Group; dPAP, diastolic pulmonary artery pressure; HFrEF, heart failure with reduced ejection fraction; HR, heart rate; IABP, intra-aortic balloon pump; LV, left ventricle; LVEF, left ventricular ejection fraction; LVSW, left ventricular stroke work index; MAP, mean arterial pressure; mPAP, mean pulmonary artery pressure; PAPi, pulmonary artery pulsatility index; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistances; RAP, right atrial pressure; RV, right ventricle; RVSW, right ventricular stroke work index; SCAI, Society for Cardiovascular Angiography and Interventions; sPAP, systolic pulmonary artery pressure; SpO$_2$, peripheral oxygen saturation; SV, stroke volume; SVi, stroke volume index; SVR, systemic vascular resistances; TAPSE, tricuspid annular plane systolic excursion; VA-ECMO, veno-arterial extracorporeal membrane oxygenation. Categorial variables are expressed as count and proportions, and continuous variables as medians (25th–75th quartiles).
Values in bold are significant P-values <0.05

In-hospital mortality according to CPI calculations agreement

BEST CARDIAC POWER INDEX (CPI) CALCULATION?

$$CPI = \frac{MAP}{451} \times CI$$

$$CPI_{RAP} = \frac{MAP - RAP}{451} \times CI$$

N= 80
cardiogenic shock patients

All with index PAC
hemodynamic assessment

CPI$_{RAP}$ strong *hemodynamic predictor* of in-hospital death
(p_{adj}=0.043)

CPI$_{RAP}$≤0.28 W/m^2 improved outcome *discrimination* vs CPI
(p=0.025)

CPI$_{RAP}$ *reclassified a subset* of patients at high in-hospital mortality risk
(30.8%)
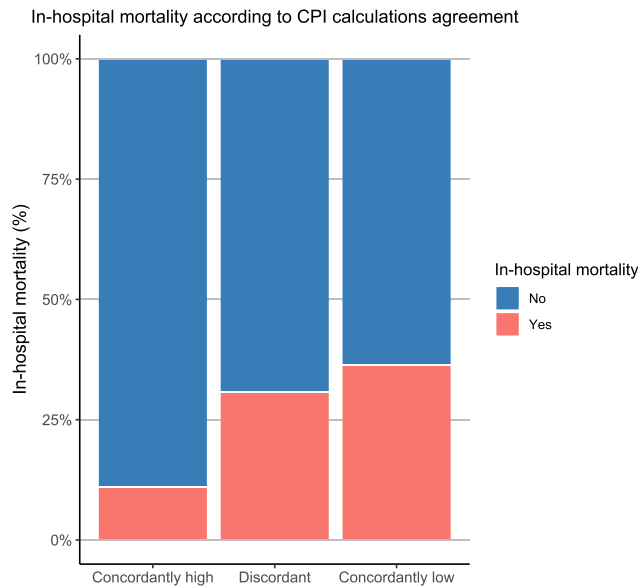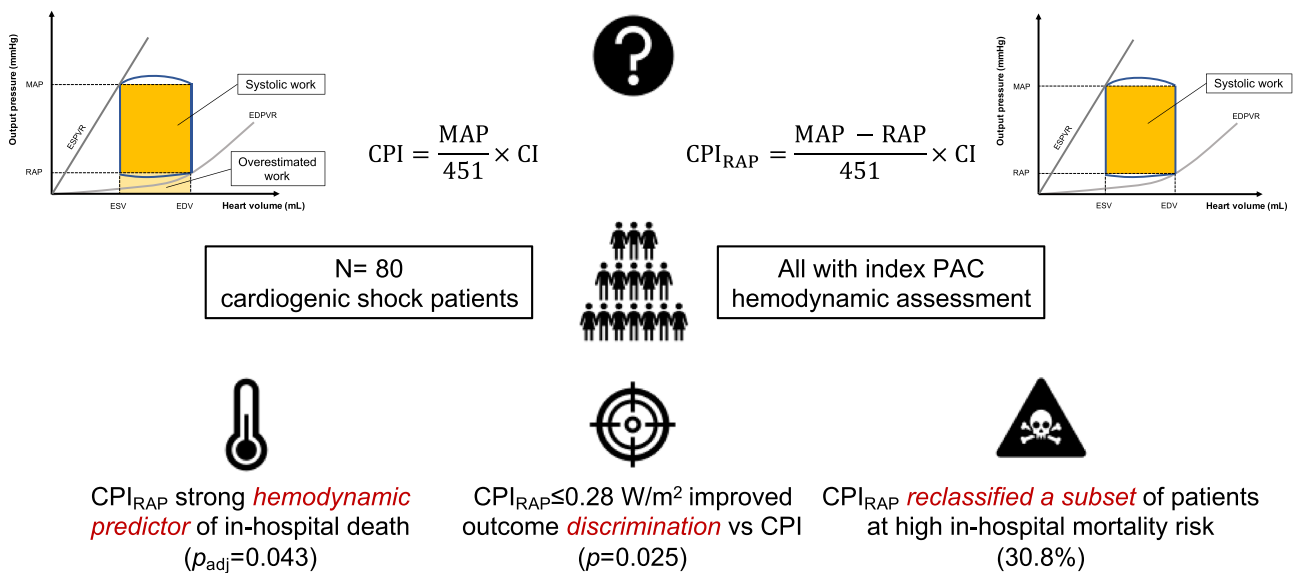
with concordantly low CPI and CPI$_{RAP}$ had the worst indexes of (bi)ventricular power, lowest MAP, and highest systemic and pulmonary vascular resistances, suggesting that they may represent an extreme end of the CS spectrum. On the opposite side, patients with concordantly high CPI and CPI$_{RAP}$ demonstrated the best parameters of (bi)ventricular function. Patients with discordant calculations (CPI > 0.32 W/m^2 and

CPI$_{RAP}$ ≤ 0.28 W/m^2) had an intermediate profile in terms of (bi)ventricular function parameters coupled with a profile of pulmonary overload and biventricular congestion. On baseline evaluation, these patients demonstrated more echocardiographic RV dysfunction and worse kidney function. Taken together, these findings suggest that the prognostic refinement obtained with CPI$_{RAP}$ may be ascribed to identification

of patients with biventricular dysfunction and congestion, RV involvement, and possibly splanchnic damage.

Indeed, overt, mainly LV, CS may well be captured by the usual CPI calculation, as lower MAP and CI outweigh the RAP term in the equation, especially if congestion is not extreme (euvolemic CS), and this may be the case of the group with concordantly low CPI and CPI_{RAP} indexes. On the opposite side, CPI_{RAP} may better capture patients with a congested phenotype and predominantly RV or biventricular involvement. These patients represent a high-risk group, as demonstrated by similar in-hospital mortality compared with patients with concordantly low CPI and CPI_{RAP} and by almost three-fold greater mortality compare with patients with concordantly high CPI and CPI_{RAP} (30.8 vs. 36.4 vs. 11.1%). Of note, several recent studies indeed identified venous and biventricular congestion as powerful indicators of in-hospital mortality in CS patients, confirming the high mortality found in this cohort.[17,18,20] Finally, in our population, CS aetiology was mixed, and the majority (66.2%) was due to ADHF, in line with contemporary CICU epidemiology[21]; notably, ADHF patients may frequently exhibit RV dysfunction, a condition that may further prompt PAC insertion.[4] In addition, ADHF aetiology may increase the impact of RAP inclusion in the CPI_{RAP} calculation. These findings confirm and extend, in a contemporary cohort, the prognostic role of RAP-corrected CPO observed in a recent sub-analysis of the Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) trial. However, in that short report, only ADHF patients were considered, and no echocardiographic, metabolic, and haemodynamic variables are presented.[22]

In conclusion, inclusion of RAP in CPI calculation (CPI_{RAP}) better captures the haemodynamic profile of patient SCAI stage B through D CS and refines identification of patients at risk of dismal outcome. Although this hypothesis-generating report does not undermine previous observations on the prognostic role of CPI in its currently widespread calculation, it is a call to further utilize and validate in larger prospective cohorts its original description (CPI_{RAP}), which may better reflect the true systolic power of cardiac pump, especially as CPO and CPI are increasingly incorporated in CS management protocols.[11,23]

Limitations

The major limitation of this study is the small sample size. Nevertheless, the study results seem consistent and are sustained by a sound pathophysiological rationale. Moreover, we selected the strongest study outcome (in-hospital death) that is not susceptible to adjudication bias. These findings, relevant for better characterization of invasive profiles of patients with AHF and CS, remain to be confirmed in larger studies.

Second, the study outcome was retrospectively assessed. However, clinical, haemodynamic, and laboratory data were prospectively recorded in a centralized dedicated database at the time of PAC assessment as per institutional protocol. In addition, clinical data were retrieved by authors blinded to the study design and outcome. Third, index PAC assessment was obtained for each patient at the earliest possible time point after minimum haemodynamic stabilization. In supported patients, PAC assessment does not reflect native heart CPI but rather native power under inotropic stimulation or the sum of native and MCS power. This limitation was also observed in the first validation study of the CPO, in which 95% of patients were on sympathomimetic amines and 27% on intra-aortic balloon pump (IABP), and also reflects real-world practice.[10,24] This drive an inherent bias from the different haemodynamics with vs. without inotropes/MCS in the same patient. Nevertheless, we compared two different formulas with the same variables affected by inotropes/MCS, the effect of inotropes/MCS is the same in the two calculations, and the additive discriminatory power of the CPI_{RAP} results from the RAP term.

Conclusion

Incorporating RAP in CPI calculation (CPI_{RAP}) improves the prognostic yield in patients with CS SCAI B–D. A cut-off of 0.28 W/m^2 identifies patients at higher risk of in-hospital mortality. The improved prognostic value of CPI_{RAP} may derive from identification of patients with more intravascular congestion who may experience substantial in-hospital mortality, uncaptured by traditional CPI equation.

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Conflict of interest

None declared.

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Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Table S1. Logistic regression analysis (in-hospital death outcome).
Table S2. Logistic regression analysis (in-hospital death outcome).
Table S3. Logistic regression analysis (in-hospital death outcome).
Table S4. Logistic regression analysis (in-hospital death outcome).
Figure S1. Pressure volume loop (PVL) for an idealized cardiac cycle. The systolic cardiac work of each cycle is approximated by the rectangle area within a PVL (dark-yellow area). The diastolic work is approximated by the rectangle area between the lower boundary of the PVL and the x-axis (light-yellow area). MAP x CI equals the sum of both the dark- and light-yellow rectangle multiplied for heart rate. In the Guyton model, preload is represented by RAP for the whole heart, thereby the true height of the systolic work rectangle is MAP - RAP. Hence (MAP − RAP) x CI equals the dark-yellow rectangle only, multiplied for heart rate, and represent actual systolic power. It is evident, that CPI, as compared to CPI$_{RAP}$, systematically overestimates true cardiac power by an amount proportional the light-yellow area and, therefore, to RAP. Legend: CPI – cardiac power index; EDPVR – end-diastolic pressure-volume relationship; EDV – end-diastolic volume; ESPVR – end-systolic pressure-volume relationship; ESV – end-systolic volume; MAP – mean arterial pressure; RAP – right atrial pressure.

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