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ORIGINAL ARTICLE

Intraoperative oliguria modifies the association between cardiac power index and postoperative acute kidney injury: a retrospective cohort study

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Abstract

Purpose Postoperative acute kidney injury (AKI) is common and associated with adverse outcomes. Intraoperative hypotension and oliguria are risk factors, yet management remains mean arterial pressure (MAP)-centric. Cardiac power index (CPI) may capture pressure–flow conditions, and its association with AKI may depend on intraoperative urine output.

Methods We analyzed 814 adults undergoing non-cardiac surgery with FloTrac™ monitoring. AKI was defined by the KDIGO creatinine criteria. The main predictors were CPI0.4_AUT, the time-integrated deficit of CPI below 0.40 W/m^2, and intraoperative urine output (mL/kg/h). Multivariable logistic regression models, adjusted for the AKI risk index and other covariates, were fitted with and without a CPI0.4_AUT × urine-output interaction, and average marginal effects were calculated.

Results AKI occurred in 59 patients (7.2%). Without the interaction term, CPI0.4_AUT was not statistically significant at the 0.05 level (adjusted odds ratio [aOR] 1.02, 95% confidence interval [CI] 1.00–1.04, P = 0.10). With the interaction, CPI0.4_AUT was significantly associated with AKI (aOR 1.05, 95% CI 1.02–1.08, P = 0.004), and the CPI0.4_AUT × urine-output term also reached significance (aOR 0.97, 95% CI 0.93–0.99, P = 0.03). Average marginal effects showed that higher CPI0.4_AUT was associated with a higher predicted AKI probability at low urine output (< 0.8 mL/kg/h), but this association was minimal when urine output was ≥ 0.9 mL/kg/h.

Conclusions Low intraoperative CPI with oliguria was associated with a higher predicted probability of AKI, whereas the CPI-AKI association weakened with increased urine output. Integrating CPI and urine output may help characterize hemodynamic management beyond MAP-centric management.

Keywords Acute kidney injury · Cardiac power index · Hemodynamic monitoring · Oliguria · Urine output

Introduction

Postoperative acute kidney injury (AKI) is a major perioperative complication associated with increased mortality and adverse long-term outcomes. Intraoperative hypotension has been linked to an increased risk of AKI [1, 2], presumably via reduced renal perfusion [3]. Consequently, maintaining mean arterial pressure (MAP) above a given threshold has become a common anesthetic goal. However, despite more frequent vasoconstrictor use and shorter durations of hypotension, AKI incidence remains substantial, whereas crystalloid administration has decreased over time [4], suggesting that MAP alone may not adequately reflect renal perfusion.

Renal perfusion depends on both pressure and flow: renal perfusion pressure is approximated by MAP, whereas renal blood flow depends on cardiac output (CO). Even when MAP is maintained, hypovolemia or impaired cardiac function can reduce CO and compromise renal perfusion. Thus, perioperative kidney protection may require joint assessment of MAP and CO.

The cardiac power index (CPI), the product of MAP and CO normalized to body surface area, reflects overall pressure–flow conditions and has prognostic value in critically ill and cardiac populations [5–7]. Oliguria is another

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intraoperative risk factor for AKI [8, 9], underscoring the importance of adequate systemic pressure and flow.

Despite these considerations, data on the association of intraoperative cumulative CPI deficit to low CPI with postoperative AKI in non-cardiac surgery are limited. Furthermore, even with similar cumulative burden of low CPI, AKI risk may differ according to the presence and severity of oliguria. Whether intraoperative urine output modifies the association between low CPI and AKI—specifically, whether the AKI risk associated with low CPI is amplified in the presence of oliguria—has not been rigorously evaluated in the perioperative setting.

Therefore, we investigated the association between intraoperative CPI and postoperative AKI in a large cohort of non-cardiac surgical patients. We focused on the cumulative CPI deficit below a threshold and its relationship with AKI after adjustment for established AKI risk factors, and we examined whether intraoperative urine output modified this association across urine-output levels to inform renal-protective hemodynamic strategies that jointly consider perfusion pressure and blood flow.

Methods

Ethical approval was obtained from the Medical Research Ethics Committee of the Shimane University Faculty of Medicine on January 24, 2024 (approval no. 7485). The committee reviewed and approved the use of an opt-out approach for informed consent; thus, written consent was not obtained from each patient. This manuscript adheres to the applicable STROBE guidelines. A data analysis and statistical plan was created after the data were accessed.

Study design

This retrospective exploratory study included patients who underwent surgery between April 2013 and November 2023 under hemodynamic monitoring with FloTrac™ (the 4th-generation algorithm; Edwards Lifesciences Corporation, Irvine, CA, USA), an arterial pressure–based CO monitor. Patients with serum creatinine levels measured within 1 month before surgery and anesthesia and within 7 days postoperatively and with no missing data for intraoperative blood pressure and CO were included. For patients who underwent more than one eligible surgery during the study period, only the first procedure was included to avoid multiple inclusions of the same individual. No prior power analysis was performed.

Patients receiving renal replacement therapy; undergoing nephrectomy, including partial nephrectomy; receiving intraoperative furosemide or mannitol; requiring circulatory assist devices such as extracorporeal membrane oxygenation; classified as the American Society of Anesthesiologists physical status (ASA-PS) 5 or 6; undergoing cardiac surgery with an artificial heart–lung machine or other cardiac procedures (pulsatile coronary artery bypass, percutaneous aortic valve replacement, or percutaneous mitral valvuloplasty); organ transplant recipients; or aged < 18 years were excluded. Outcomes were defined according to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria, with AKI defined as an increase in serum creatinine of 0.3 mg/dL within 48 h or a > 1.5-fold increase from baseline. Urine volume criteria were not used to define AKI. The reference creatinine value was the most recent measurement obtained within 1 month preoperatively.

Data collection and processing

Relevant data were collected from the medical and anesthesia records of the study participants, including blood pressure, CO, surgery and anesthesia-related information, and preoperative and postoperative serum creatinine levels. Details of the procedures for calculating urine output, noradrenaline equivalents, blood loss, total fluid volume and CPI are presented in Online Resource 1.

A complete case analysis was performed to address missing data in the covariates. Separately, for intraoperative 1-min hemodynamic time-series data, non-physiological artifacts (CO < 0.5 or > 9 L/min; MAP < 10 or > 150 mmHg) were identified and replaced with linear interpolation between adjacent valid measurements before calculating CPI and related indices.

Statistical analysis

We used a staged, hypothesis-driven analysis. We initially hypothesized that lower intraoperative CPI would be associated with postoperative AKI. Low urine output is an established AKI risk factor and is physiologically influenced by CPI; therefore, we refined this hypothesis and posited that the CPI–AKI association would be modified by intraoperative urine output. We evaluated a CPI × urine output interaction term in multivariable models.

CPI was evaluated using the area under the threshold (CPI_AUT), defined as the time-integrated deficit of CPI below a fixed threshold. For each minute, when CPI fell below the threshold, the difference between the threshold and the observed CPI was calculated and then integrated over the intraoperative period. Thus, a larger CPI_AUT indicates longer and/or deeper periods during which CPI remains below the threshold. CPI thresholds between 0.32 and 0.46 W/m^2 were examined; the main analysis used 0.40 W/m^2, the rounded midpoint (0.39 W/m^2). The range was selected based on CPI values corresponding to commonly

used MAP and cardiac index targets, as well as reported values in healthy adults, allowing for lower pressures and flows during anesthesia [10]. Using a similar approach, we derived MAP65_AUT (area under MAP < 65 mmHg) and CI2.8_AUT (area under cardiac index < 2.8 L/min/m^2). SVV13_AAT was defined as the area above stroke volume variation ≥ 13%.

Baseline characteristics were summarized by AKI status using medians (interquartile ranges) for continuous variables and counts (percentages) for categorical variables. Between-group differences were assessed with the Mann–Whitney U test for continuous variables and chi-squared or Fisher's exact tests for categorical variables.

For the primary analysis, the outcome was postoperative AKI (KDIGO creatinine criteria). Multivariable logistic regression models were fitted to estimate odds ratios (ORs) and 95% confidence intervals (CIs). The primary variables of interest were CPI_AUT at a threshold of 0.40 W/m^2 (CPI0.4_AUT) and intraoperative urine output (mL/kg/h), both treated as continuous variables. Covariates were the AKI risk index [11] (a five-class score based on age, sex, abdominal surgery, emergency surgery, diabetes, active congestive heart failure, ascites, hypertension, and preoperative renal dysfunction), preoperative estimated glomerular filtration rate (eGFR; Japanese Society of Nephrology equation), hemoglobin, albumin, the use of potentially nephrotoxic medications (diuretics; angiotensin-converting-enzyme inhibitors [ACEIs]; angiotensin receptor blockers [ARBs]; angiotensin receptor–neprilysin inhibitors [ARNIs]; and non-steroidal anti-inflammatory drugs [NSAIDs]), body mass index (BMI), duration of surgery, blood loss per kilogram of body weight, total intraoperative fluid administration per kilogram of body weight per hour, and intraoperative noradrenaline-equivalent dose. eGFR values > 200 mL/min/1.73 m^2 were truncated at 200. ORs for continuous variables were interpreted per 10 mL/min/1.73 m^2 increase in preoperative eGFR, per 30-min increase in operation time, and per 0.01 μg/kg/min increase in noradrenaline-equivalent dose.

We specified two multivariable logistic models: a primary model that included a CPI0.4_AUT × urine-output interaction term, and an otherwise identical model without the interaction term for comparison. Variance inflation factors (VIFs) were calculated to assess multicollinearity (VIF > 10 considered problematic), and a likelihood-ratio test compared the models with and without the interaction term.

Average marginal effects (AMEs) were calculated using the marginal effects package, estimating the change in predicted AKI probability associated with a 1-unit increase in urine output across levels of CPI0.4_AUT and, conversely, with a 1-unit increase in CPI0.4_AUT across urine-output levels. Estimates were averaged across patients while holding other covariates at observed values. Positive AMEs indicate a higher model-predicted probability of postoperative AKI, whereas negative AMEs indicate a lower model-predicted probability.

To examine robustness, we performed exploratory subgroup and sensitivity analyses, including models additionally adjusted for measures of cardiac function and inflammation, models using alternative specifications of the AKI risk index and alternative intraoperative hemodynamic metrics, models incorporating additional perioperative covariates; analyses using different CPI thresholds; and a categorical analysis of four hemodynamic groups defined by CPI0.4_AUT and CI2.8_AUT. Full model specifications and detailed results are provided in Online Resources 3–7. All regression analyses were complete-case analyses with respect to covariates. Statistical analyses were performed using R version 4.4.1 (R Foundation for Statistical Computing, Vienna, Austria) with logistic regression (glm, family = binomial); 95% CIs were obtained using profile likelihood, and two-sided $p < 0.05$ was considered statistically significant.

Results

Among 1,086 anesthesia cases monitored with FloTrac™, 272 were excluded owing to renal replacement therapy, age < 18 years, cardiac surgery, intraoperative furosemide or mannitol use, nephrectomy, missing data, or duplicate cases. The remaining 814 patients were included in the final analysis (Fig. 1). No patients had ASA-PS 5 or 6. Postoperative AKI developed in 59 patients (7.2%). In the subgroup of 445 patients with left ventricular ejection fraction data, 41 (9.2%) developed AKI.

The median age was 70 years, and the most common surgical categories were urologic, hepatobiliary, pancreatic, and lower gastrointestinal procedures (Table 1). As shown in Table 1, several preoperative characteristics differed between groups. Intraoperatively, patients who developed AKI experienced greater blood loss and lower urine

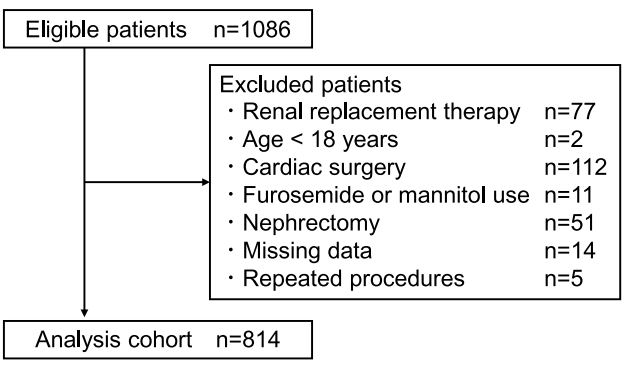

Fig. 1 Flow chart illustrating study inclusion and exclusion criteria. Abbreviations: n, number

Table 1 Patient background and type of surgery by AKI status

Characteristic	Overall N = 814	no AKI n = 755	AKI n = 59	P-value*
Female sex	281 (35%)	271 (36%)	10 (17%)	0.003
Age (years)	70 [64–77]	70 [63–77]	74 [68–79]	0.02
BMI (kg/m^2)	22.5 [20.1–24.8]	22.3 [20.0–24.7]	23.9 [21.0–26.3]	0.007
Type of surgery				0.6
Urologic surgery	185 (23%)	164 (22%)	21 (36%)	
Hepatobiliary surgery	118 (14%)	106 (14%)	12 (20%)	
Pancreatic surgery	101 (12%)	95 (13%)	6 (10%)	
Lower gastrointestinal surgery	87 (11%)	79 (10%)	8 (14%)	
Gynecologic surgery	67 (8.2%)	65 (8.6%)	2 (3.4%)	
Other surgeries	256 (31%)	246 (33%)	10 (17%)	
Emergency surgery	111 (14%)	101 (13%)	10 (17%)	0.4
ASA physical status				0.15
Class I	18 (2.2%)	18 (2.4%)	0 (0%)	
Class II	557 (68%)	521 (69%)	36 (61%)	
Class III	221 (27%)	201 (27%)	20 (34%)	
Class IV	18 (2.2%)	15 (2.0%)	3 (5.1%)	
AKI risk index				< 0.001
Class I	224 (28%)	216 (29%)	8 (14%)	
Class II	257 (32%)	246 (33%)	11 (19%)	
Class III	239 (29%)	214 (28%)	25 (42%)	
Class IV	81 (10%)	68 (9.0%)	13 (22%)	
Class V	13 (1.6%)	11 (1.5%)	2 (3.4%)	
Hypertension	451 (55%)	409 (54%)	42 (71%)	0.01
Diabetes	207 (25%)	188 (25%)	19 (32%)	0.2
History of chronic heart failure	62 (7.6%)	55 (7.3%)	7 (12%)	0.2
Preoperative chronic use of diuretics / ACEIs / ARBs / ARNIs / NSAIDs	334 (41%)	297 (39%)	37 (63%)	< 0.001
Preoperative creatinine (mg/dL)	0.73 [0.62–0.89]	0.73 [0.61–0.89]	0.79 [0.65–1.02]	0.01
Preoperative eGFR (mL/min/1.73m^2)	75 [62–87]	75 [62–87]	71 [57–85]	0.06
Preoperative hemoglobin (g/dL)	13 [11–14]	13 [11–14]	12 [10–14]	0.2
Preoperative albumin (g/dL)	4.0 [3.5–4.3]	4.0 [3.5–4.3]	3.9 [3.5–4.2]	0.07
Operation time (min)	330 [205–440]	322 [200–432]	393 [282–540]	0.002
Total blood loss (mL/kg)	1.9 [0.0–5.4]	1.7 [0.0–5.1]	4.7 [1.3–9.5]	< 0.001
Any operative transfusion	101 (12%)	88 (12%)	13 (22%)	0.02
Total intraoperative fluid volume (mL/kg/h)	8.9 [6.9–12.6]	8.9 [6.9–12.6]	8.8 [5.9–11.7]	0.13
Noradrenaline equivalent (μg/kg/min)	0 [0–0.0045]	0 [0–0.0043]	0 [0–0.020]	0.04
Urine output (mL/kg/h)	1.6 [0.9–2.8]	1.7 [1.0–2.9]	0.8 [0.4–1.4]	< 0.001
Area under the threshold of 0.4 for cardiac power index (W·min/m^2)	7.1 [1.6–16.3]	6.6 [1.5–15.4]	12.1 [5.3–26.5]	< 0.001
Area under the threshold of 65 for mean arterial pressure (mmHg·min)	188 [45–687]	178 [43–630]	490 [119–1394]	< 0.001
Area under the threshold of 2.8 for cardiac index (L/m^2)	78 [18–173]	76 [17–171]	112 [60–215]	0.006
Area above the threshold of 13% for stroke volume variation (%·min)	144 [9.9–664]	145 [10–646]	125 [6.4–945]	0.6

Abbreviations: AKI, acute kidney injury; BMI, body mass index; ASA, American Society of Anesthesiologists; ACEIs, angiotensin-converting-enzyme inhibitors; ARBs, angiotensin receptor blockers; ARNIs, angiotensin receptor-neprilysin inhibitors; NSAIDs, non-steroidal anti-inflammatory drugs; eGFR, estimated glomerular filtration rate

Data are presented as absolute numbers (percentage) or median (25th, 75th percentile). No patients had an ASA physical status of 5 or 6

* Comparisons of patient background and surgical characteristics between the AKI and non-AKI groups were performed using the Mann–Whitney U test for continuous variables and the chi-square test for categorical variables. P-values indicate the statistical significance of differences between the groups

output, and exhibited higher CPI0.4_AUT, MAP65_AUT and CI2.8_AUT values, consistent with the hypothesized hemodynamic risk profile. Findings in the EF subgroup paralleled those of the overall cohort (Online Resource 2).

In the multivariable logistic regression analysis, the model without an interaction term (Model 2) showed that higher AKI risk index class, preoperative chronic use of potentially nephrotoxic medications, and greater intraoperative blood loss remained significantly associated with AKI, whereas BMI, duration of surgery, total intraoperative fluid volume, and noradrenaline-equivalent dose were not (Table 2). In this model, higher urine output was independently associated with lower odds of postoperative AKI (adjusted odds ratio [aOR] 0.56, 95% CI 0.38–0.77, P = 0.001), whereas CPI0.4_AUT was not statistically significant when averaged over all urine output levels (aOR 1.02, 95% CI 1.00–1.04, P = 0.10).

When the CPI0.4_AUT × urine output interaction was added (Model 1), the aOR for CPI0.4_AUT at a urine output of 0 mL/kg/h was 1.05 (95% CI 1.02–1.08, P = 0.004), indicating that larger CPI deficits were significantly associated with higher odds of postoperative AKI. The urine output term was not statistically significant in this interaction model (aOR 0.75, 95% CI 0.49–1.03, P = 0.14), whereas the interaction term was significantly less than 1 (aOR 0.97, 95% CI 0.93–0.99, P = 0.03), indicating that the CPI0.4_AUT–AKI association varied across urine output levels. A likelihood ratio test favored Model 1 over Model 2 ($\chi^2(1) = 5.6$, $P = 0.02$). VIFs for all predictors, including the interaction term, were < 4, suggesting no problematic multicollinearity.

Table 2 Logistic regression analysis for AKI using CPI0.4_AUT: comparison of models with and without the CPI0.4_AUT–urine output interaction

			Model 1: with interaction		Model 2: without interaction	
Variable	Unadjusted OR (95% CI)	P-value*	Adjusted OR (95% CI)	P-value*	Adjusted OR (95% CI)	P-value*
AKI risk index	1.76 (1.36–2.28)	< 0.001	1.35 (0.98–1.86)	0.06	1.38 (1.01–1.90)	0.04
Preoperative eGFR (per 10 mL/min/1.73m^2)	0.87 (0.77–0.99)	0.04	0.97 (0.84–1.10)	0.64	0.98 (0.85–1.11)	0.72
Preoperative hemoglobin (g/dL)	0.91 (0.80–1.03)	0.14	0.89 (0.75–1.06)	0.18	0.89 (0.75–1.06)	0.18
Preoperative albumin (g/dL)	0.79 (0.54–1.18)	0.23	0.91 (0.51–1.65)	0.75	0.89 (0.50–1.60)	0.69
Preoperative chronic use of diuretics / ACEIs / ARBs / ARNIs / NSAIDs	2.59 (1.51–4.55)	0.001	1.94 (1.06–3.64)	0.03	2.00 (1.10–3.72)	0.03
BMI (kg/m^2)	1.00 (0.97–1.01)	0.96	0.99 (0.93–1.01)	0.66	0.99 (0.94–1.01)	0.66
Operation time (per 30 min)	1.06 (1.02–1.11)	0.004	1.02 (0.96–1.08)	0.49	1.02 (0.96–1.08)	0.48
Total intraoperative fluid volume (mL/kg/h)	0.95 (0.90–1.00)	0.09	0.97 (0.90–1.04)	0.46	0.98 (0.90–1.04)	0.55
Noradrenaline equivalent (per 0.01 μg/kg/min)	1.02 (0.97–1.05)	0.40	1.02 (0.97–1.07)	0.29	1.03 (0.97–1.07)	0.26
Total blood loss (mL/kg)	1.03(1.01–1.05)	0.004	1.04 (1.01–1.07)	0.005	1.04 (1.01–1.07)	0.01
Urine output (mL/kg/h)	0.48 (0.33–0.66)	< 0.001	0.75(0.49–1.03)	0.14	0.56 (0.38–0.77)	0.001
Area under the threshold of 0.4 for cardiac power index (CPI0.4_AUT, W·min/m^2)	1.03 (1.01–1.05)	< 0.001	1.05(1.02–1.08)	0.004	1.02 (1.00–1.04)	0.10
CPI0.4_AUT × urine output (interaction term)	-	-	0.97 (0.93–0.99)	0.03	-	-

Both multivariable models were adjusted for the following covariates: AKI risk index, preoperative eGFR, preoperative hemoglobin, preoperative albumin, preoperative chronic use of diuretics / ACEIs / ARBs / ARNIs / NSAIDs, BMI, operation time, total intraoperative fluid volume, noradrenaline equivalent, total blood loss, urine output and CPI0.4_AUT. Model 1 (with interaction) additionally included the product term CPI0.4_AUT × urine output

*P-values were derived from the Wald test; the 95% CI was calculated using the profile likelihood method. Odds ratios for continuous variables represent the change in odds per 10 mL/min/1.73m^2 increase in eGFR, per 30-min increase in operation time, per 1 mL/kg increase in blood loss, per 1 mL/kg/h increase in urine output and total intraoperative fluid volume, and per 0.01 μg/kg/min increase in noradrenaline-equivalent dose

Abbreviations: OR, odds ratio; CI, confidence interval; AUT, area under the threshold; CPI, cardiac power index; AKI, acute kidney injury; BMI, body mass index; ACEIs, angiotensin-converting-enzyme inhibitors; ARBs, angiotensin receptor blockers; ARNIs, angiotensin receptor-neprilysin inhibitors; NSAIDs, non-steroidal anti-inflammatory drugs; eGFR, estimated glomerular filtration rate

To facilitate interpretation of the interaction, we examined AMEs for CPI0.4_AUT and urine output (Figs. 2 and 3). The AME of a 1 mL/kg/h increase in urine output at each level of CPI0.4_AUT was generally negative, indicating that higher urine output was associated with a lower model-predicted probability of AKI. When CPI0.4_AUT was near zero, this reduction was small and not significant. As CPI0.4_AUT increased, the magnitude of the negative AMEs increased, suggesting that a 1 mL/kg/h higher urine output was associated with a larger decrease in the model-predicted probability of AKI at higher cumulative CPI deficits. When CPI0.4_AUT was moderately to markedly elevated, a 1 mL/kg/h higher urine output was estimated to be associated with an approximately 1–2 percentage-point lower predicted AKI probability.

Conversely, the AME of a 1 W·min/m^2 increase in CPI0.4_AUT was positive at urine outputs approximately < 0.8 mL/kg/h, indicating that larger CPI deficits were associated with significantly higher model-predicted probability of AKI in this low-urine-output range. As urine output increased, these AMEs approached zero; at urine outputs roughly ≥ 0.9 mL/kg/h, the AMEs were close to zero with 95% CIs crossing zero, indicating that the increase in predicted AKI probability associated with a 1-unit increase in CPI0.4_AUT was no longer statistically significant.

Subgroup and supplementary analyses were broadly consistent with the primary results. Across subgroup analyses, including the EF subgroup, the subgroup with preoperative CRP and COPD data, and analyses replacing the composite AKI risk index with its individual components plus heart failure and high-risk surgery indicators, the direction and significance of the aOR for CPI0.4_AUT and the CPI0.4_AUT × urine output interaction were consistent with the primary model. However, in the EF subgroup, the interaction term was negative but only borderline significant (Online Resource 3).

When CPI0.4_AUT was replaced by MAP65_AUT or CI2.8_AUT, associations with AKI were less consistent. In models without interaction terms, higher urine output remained inversely associated with AKI. In models with interaction terms, the CI2.8_AUT × urine output interaction was significantly negative, whereas no clear interaction was observed between CPI0.4_AUT and preoperative MAP (Online Resource 4). In models adding individual covariates (total vasopressor bolus dose, SVV13_AAT, preoperative MAP, or intraoperative transfusion), aOR estimates for CPI0.4_AUT and the interaction term were

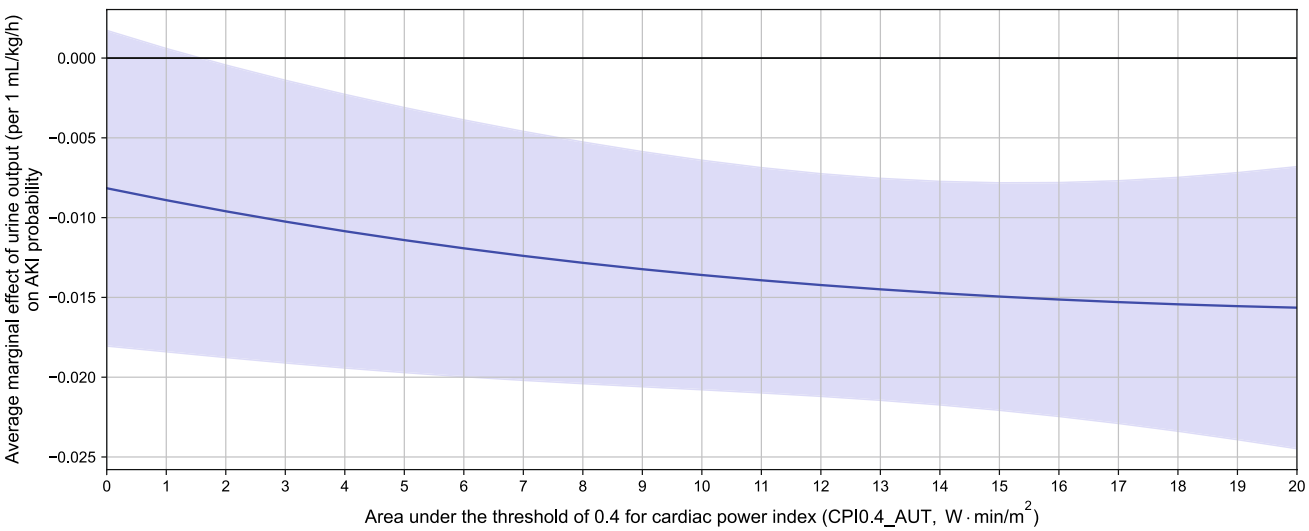

Fig. 2 Average marginal effect of increased urine output on model-predicted AKI probability across levels of CPI0.4_AUT The horizontal axis shows CPI0.4_AUT, calculated by summing over time the difference between the threshold value of 0.40 W/m^2 and the observed CPI whenever CPI is below this threshold (units: W·min/m^2). The vertical axis shows the average marginal effect (AME) on the predicted probability of postoperative AKI associated with increasing urine output by 1 mL/kg/h. Positive AME values indicate that a 1 mL/kg/h higher urine output is associated with a higher model-predicted probability of postoperative AKI, whereas negative AME values indicate an association with a lower model-predicted probability of postoperative AKI. The solid line represents the estimated AME, and the shaded area represents its 95% confidence interval. As shown in the figure, AME values are generally below zero across the observed range of CPI0.4_AUT, indicating that a 1 mL/kg/h higher urine output is consistently associated with a change in the model-predicted probability of AKI toward lower values. Moreover, as CPI0.4_AUT increases (i.e., as CPI remains below the threshold for longer periods and/or by greater deficits), the AMEs shift further in the negative direction, suggesting that the estimated reduction in predicted AKI probability associated with a 1 mL/kg/h increase in urine output becomes larger when the cumulative deficit in CPI is greater. **Abbreviations:** CPI0.4_AUT, area under the threshold of 0.4 W/m^2 for cardiac power index (W·min/m^2); AME, average marginal effect; CPI, cardiac power index; AKI, acute kidney injury

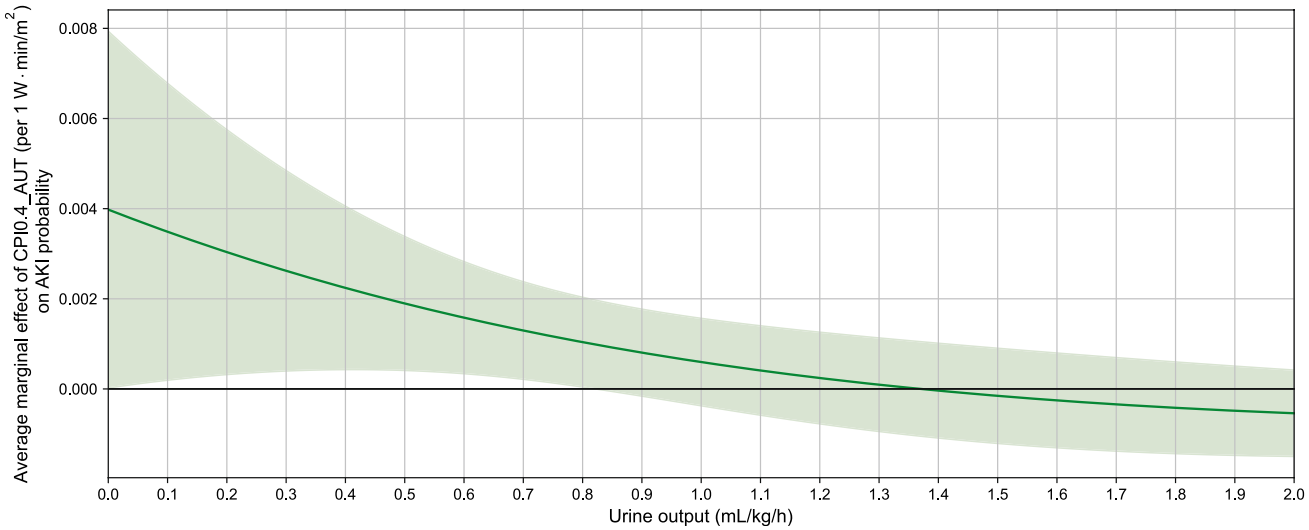

Fig. 3 Average marginal effect of increases in CPI0.4_AUT on model-predicted AKI probability across levels of urine output The horizontal axis shows urine output (mL/kg/h), and the vertical axis shows the average marginal effect (AME) on the predicted probability of postoperative AKI associated with a 1-unit increase in CPI0.4_AUT (1 W·min/m^2). CPI0.4_AUT was calculated by summing over time the difference between the threshold value of 0.40 W/m^2 and the observed CPI whenever CPI is below this threshold. Positive AME values indicate that a 1-unit higher CPI0.4_AUT is associated with a higher model-predicted probability of postoperative AKI, whereas negative AME values indicate an association with a lower model-predicted probability of postoperative AKI. The solid line represents the estimated AME, and the shaded area represents its 95% confidence interval. At low urine output levels (in this cohort, approximately < 0.8 mL/kg/h), AME values are greater than zero, indicating that larger CPI0.4_AUT is associated with a higher model-predicted probability of AKI. As urine output increases, AME values move toward zero, and at urine outputs of approximately ≥ 0.9 mL/kg/h, the AME estimates are close to zero and their 95% confidence intervals cross zero, indicating that the increase in the model-predicted probability of AKI associated with a 1-unit increase in CPI0.4_AUT is no longer statistically significant. At higher urine output levels, the point estimates of the AME become slightly negative, although the 95% confidence intervals still include zero, suggesting that when urine output is adequately maintained, the CPI0.4_AUT-associated increase in the model-predicted probability of AKI may be smaller, but it is not completely reversed. **Abbreviations:** AKI, acute kidney injury; CPI0.4_AUT, area under the threshold of 0.4 W/m^2 for cardiac power index (W·min/m^2); AME, average marginal effect; CPI, cardiac power index

similar to those in the main analysis, and their statistical significance was unchanged (Online Resource 5).

In sensitivity analyses using CPI thresholds from 0.32 to 0.46 W/m^2, interaction models consistently yielded the same pattern of a positive CPI_AUT association and a negative CPI_AUT × urine output interaction, whereas models without interaction continued to show a weak association with CPI_AUT and a constant inverse association with urine output (Online Resource 6). Finally, when CPI0.4_AUT and CI2.8_AUT were dichotomized at their medians to form four hemodynamic groups, AKI incidence was higher in the groups with high CPI0.4_AUT and lowest in the low-CPI0.4_AUT/high-CI2.8_AUT group; only the high-CPI0.4_AUT/high-CI2.8_AUT group showed a statistically significant increase in unadjusted AKI odds compared with the reference, whereas the high-CPI0.4_AUT/low-CI2.8_AUT group showed a similar trend that did not reach significance (Online Resource 7).

Discussion

This study yielded two main findings. First, among non-cardiac surgical patients monitored intraoperatively with FloTrac™, a larger cumulative CPI deficit below 0.4 W/m^2 (higher CPI0.4_AUT) was associated with higher odds of postoperative AKI. Second, the CPI0.4_AUT–AKI association differed across intraoperative urine-output levels, consistent with a statistically significant CPI0.4_AUT × urine-output interaction. In average marginal-effects analyses, higher CPI0.4_AUT was accompanied by a larger absolute decrease in model-predicted AKI probability per 1 mL/kg/h higher urine output: when CPI0.4_AUT was moderately to markedly elevated, a 1 mL/kg/h higher urine output was estimated to be associated with an approximately 1–2 percentage-point lower predicted AKI probability (e.g., from 10 to 8%–9%). By contrast, at lower urine outputs (approximately < 0.8 mL/

kg/h), higher CPI0.4_AUT was associated with a more pronounced increase in the model-predicted probability of AKI, whereas at urine outputs of roughly ≥ 0.9 mL/kg/h, the marginal effect of CPI0.4_AUT was small and statistically non-significant. These findings suggest that the combination of low CPI and oliguria is associated with particularly high postoperative AKI probability, whereas the CPI0.4_AUT-associated increase in model-predicted AKI probability was less pronounced at higher urine output levels.

In multivariable models including CPI0.4_AUT and intraoperative urine output as primary predictors, the model without the interaction term (Model 2) showed that higher urine output was independently associated with lower odds of postoperative AKI, whereas the main term of CPI0.4_AUT did not reach conventional statistical significance (P = 0.10). By contrast, in the interaction model, the estimated CPI0.4_AUT–AKI association at a urine output of 0 mL/kg/h was clearly significant, and the interaction term was significantly less than 1, indicating that the CPI0.4_AUT–AKI association depended on urine output. A likelihood-ratio test favored the interaction model, supporting explicit modeling of the interdependence between CPI0.4_AUT and urine output.

This interaction has important implications for AKI pathophysiology. CPI integrates MAP and CO (or cardiac index) and may therefore reflect global and organ perfusion more comprehensively than MAP alone. In our cohort, patients who developed AKI had higher MAP65_AUT values, indicating a greater cumulative burden of intraoperative hypotension, yet when MAP65_AUT was used as the primary hemodynamic metric, its association with AKI and its interaction with urine output were less consistent than for CPI0.4_AUT. Likewise, in models using CI2.8_AUT, the interaction with urine output was significantly negative, but the CI2.8_AUT main term was significant only in the interaction model, not in the model without interaction. Although odds-ratio magnitudes cannot be directly compared across CPI0.4_AUT, MAP65_AUT, and CI2.8_AUT because of differing scales, CPI0.4_AUT showed a more consistent association with AKI across model specifications and subgroups. This pattern suggests that CPI, as the product of pressure and flow, may capture the risk of renal hypoperfusion and organ ischemia more sensitively than either pressure or flow alone. In a "pre-renal" state characterized by oliguria, even modest, cumulative reductions in cardiac pump performance may act as the final trigger that pushes the kidneys beyond their tolerance threshold.

The structure of the statistical models is also relevant. In the interaction model, the aOR of 1.05 for CPI0.4_AUT represents the change in AKI odds per 1 W·min/m^2 increase in CPI0.4_AUT at a urine output of 0 mL/kg/h and may appear modest. However, postoperative AKI is strongly linked to long-term mortality and other serious outcomes [12–15], so this magnitude of association is unlikely to be clinically trivial. Moreover, the average marginal-effects analysis showed that, particularly when CPI0.4_AUT was moderately to markedly elevated, increases in urine output were associated with an approximately 1–2 percentage-point lower absolute predicted AKI probability, highlighting low CPI plus low urine output as a clinically relevant "hot spot." In contrast, the simpler model without the interaction term yielded a smaller aOR for CPI0.4_AUT, effectively averaging the CPI0.4_AUT–AKI association across patients with a wide variation in urine outputs and potentially masking the sharp risk increase in patients with both large CPI deficits and low urine output. The clinical value of our findings therefore lies less in a single odds ratio than in showing that the strength of the CPI deficit–AKI association varies across intraoperative urine output levels.

Across all subgroup and sensitivity analyses (including alternative covariate sets, CPI thresholds, and hemodynamic definitions), the CPI–urine output interaction remained directionally consistent, supporting the robustness of the main findings. These findings suggest that the main pattern is unlikely to be explained solely by a specific model specification or arbitrary CPI threshold.

Exploratory categorical analysis showed a compatible pattern with the main analysis, reinforcing CPI's potential value as an integrated pressure–flow indicator.

Our findings are consistent with, and extend, previous reports linking intraoperative hypotension and oliguria to postoperative AKI [16, 17]. Prior work has often evaluated AKI risk within a blood pressure–centric framework, with clinical targets such as maintaining a MAP ≥ 65 mmHg. In addition, perioperative hemodynamic goal-directed therapy that titrates fluids and inotropes to achieve adequate CO and systemic oxygen delivery has been shown to lower the risk of postoperative AKI [18]. In this context, our findings add that an integrated pressure–flow index—CPI—shows a specific interaction with urine output that enables more nuanced risk stratification than MAP alone. In particular, patients who maintain MAP ≥ 65 mmHg but have reduced CPI and persistent oliguria may represent a high-risk group that is underestimated when only blood pressure is considered.

This study has some limitations. First, because of its retrospective design, causal inferences cannot be drawn and residual confounding by unmeasured variables cannot be excluded. Although we adjusted for multiple variables, unmeasured factors (including surgical complexity, clinician judgement, perioperative inflammation, microcirculatory changes, and variability in fluid and vasopressor management) may have influenced the results. Second, this was a single-center study in a Japanese population that excluded cardiac surgery and patients receiving perioperative diuretics, which may limit generalizability to other settings and

populations. Third, urine output was summarized as mean mL/kg/h during the operation, preventing assessment of temporal relationships between oliguria and CPI; therefore, the interaction should be interpreted as a statistical interaction rather than a causal interplay. Fourth, CPI was derived from FloTrac™ data and may be susceptible to measurement error, particularly during arrhythmias or abrupt changes in vascular tone. Fifth, only 59 AKI events occurred, raising the possibility of overfitting in multivariable models. Ultimately, the overall statistical approach was exploratory, with multiple subgroups, sensitivity analyses, and CPI thresholds examined, increasing the risk of type I error. Although the CPI–urine output interaction was consistent across thresholds, the findings should be regarded as hypothesis-generating rather than directly prescriptive for clinical protocols.

Despite these limitations, our results have important implications for practice and research. Even when MAP is maintained at ≥ 65 mmHg, patients with depressed CPI, markedly elevated CPI0.4_AUT, and oliguria (e.g., urine output < 0.5–0.9 mL/kg/h) should be considered at substantially higher risk of postoperative AKI than suggested by blood pressure alone. In such situations, a strategy that goes beyond escalating vasopressors—re-evaluating overall hemodynamics, optimizing volume status and, where appropriate, inotropic support to improve CO and CPI, together with explicit targeting of oliguria correction—may represent a testable hypothesis for AKI prevention. From a practical standpoint, CPI is simply the product of MAP and CO or index, variables that are already monitored in many operating rooms, including when using FloTrac™; thus, CPI can be derived from routinely available hemodynamic data without any additional invasive monitoring. Our findings should thus not be interpreted as requiring CPI monitoring per se, but rather as supporting intraoperative management that interprets pressure and flow jointly—whether via CPI or by considering MAP and CO together—while using urine output as an additional safety signal for kidney perfusion. Because this study is observational, we cannot determine whether specific interventions aimed at increasing urine output (such as diuretics) reduce AKI risk. Nonetheless, the model-based estimate that a 1 mL/kg/h increase in urine output in the presence of substantial CPI deficits may reduce absolute model-predicted probability of AKI by about 1–2 percentage points suggests that the potential population-level impact in major surgery could be clinically meaningful.

In conclusion, in this study, we showed that the association between low intraoperative CPI and postoperative AKI varied with intraoperative urine output. The combination of low CPI and oliguria was associated with particularly high model-predicted AKI probability, whereas the increase in predicted AKI probability associated with higher CPI0.4_AUT was weaker at higher urine output levels. These findings support moving beyond MAP-centric management toward perioperative strategies that monitor and interpret both CPI and urine output. External validation and prospective interventional studies reporting the use of protocols that incorporate both variables as management targets are needed to confirm these observational findings and assess their effectiveness for AKI prevention.

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Data availability The datasets generated and/or analyzed during the current study are not publicly available due to institutional restrictions and the presence of sensitive patient information, but are available from the corresponding author on reasonable request.

Declarations

Conflict of interest The authors declare that there are no conflicts of interest.

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