

Intraoperative Cuff–Arterial Pressure Discordance and Postoperative Creatinine Doubling: A Retrospective MOVER Cohort Study

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Abstract

Background

Intraoperative cuff and invasive arterial mean arterial pressure (MAP) may cross clinically used hypotension thresholds in opposite directions. We evaluated whether a directional cuff-arterial discordance phenotype was associated with postoperative creatinine doubling.

Methods

We conducted a retrospective cohort study using the MOVER perioperative electronic health record dataset. Adults with complete core covariates, baseline and postoperative creatinine measurements, and at least five one-to-one paired intraoperative MAP observations were eligible. The primary cohort retained the first chronologically eligible operation per patient. The exposure was arterial MAP < 65 mmHg with paired cuff MAP $\geq$ 75 mmHg in at least 10% of valid pairs. The primary outcome was postoperative creatinine $\geq$ 2 times baseline within 7 days. Multivariable logistic regression adjusted for age, recorded sex, ASA physical status, surgical duration, baseline creatinine, preoperative anemia, general anesthesia, inpatient status, procedure category, and paired-measurement count.

Results

The primary cohort included 3,075 patients; 691 met the exposure definition. Creatinine doubling occurred in 60/691 exposed patients (8.68%) and 132/2,384 unexposed patients (5.54%). The adjusted odds ratio was 1.61 (95% CI, 1.16–2.23; $P = 0.0041$). The association remained significant in the all-operations analysis with patient-clustered standard errors (OR, 1.70; 95% CI, 1.28–2.25), when the cuff threshold was $\geq$ 65 mmHg (OR, 1.42; 95% CI, 1.05–1.92), and when analyses required at least 10 or 20 paired measurements. However, after additional adjustment for the fraction of paired arterial MAP observations below 65 mmHg, the estimate attenuated and was no longer statistically significant (OR, 1.32; 95% CI, 0.90–1.95; $P = 0.1565$). Any creatinine-defined acute kidney injury and in-hospital death were not significantly associated with the primary exposure.

Conclusions

Discordance was associated with creatinine doubling, but the association weakened after adjustment for arterial hypotension burden. Cuff readings above the hypotension threshold may miss lower arterial pressures. Relying on cuff values alone could therefore miss an opportunity for risk-based postoperative kidney assessment. In high-risk surgery, arterial monitoring should be considered when clinically indicated. This study cannot establish that either monitoring or surveillance prevents kidney injury.

Introduction

Postoperative renal injury remains a clinically important complication of major surgery. Intraoperative hypotension is associated with acute kidney injury (AKI), and the risk appears to depend on both the depth and duration of low arterial pressure [2–7]. The usual clinical definitions, however, assume that the measured blood pressure accurately represents the pressure relevant to organ perfusion.

Intraoperative blood pressure is often measured by intermittent oscillometric cuff monitoring, continuous invasive arterial monitoring, or both. These methods do not always agree [1, 12, 13]. A cuff MAP can remain above a specified threshold while a time-matched arterial MAP is below that threshold. This discordance identifies a difference in measured pressure categories; the available data do not show whether clinicians recognized or treated the arterial value differently. Recent work has demonstrated that cuff measurements can misclassify intraoperative blood pressure [1], but the relationship between this measurement-level discordance and postoperative renal outcomes remains uncertain.

We used the MOVER perioperative electronic health record dataset to examine whether a directional pattern of arterial MAP < 65 mmHg accompanied by cuff MAP $\geq$ 75 mmHg was associated with documented postoperative creatinine doubling. We selected creatinine doubling as the primary endpoint because it represents a more clinically consequential laboratory deterioration than a broad, low-threshold AKI definition. Any creatinine-defined AKI and in-hospital death were defined as secondary outcomes before this final rerun.

Methods

Study design and data source

We conducted a retrospective cohort study using MOVER, a publicly accessible, deidentified perioperative electronic health record dataset containing surgical case information, intraoperative flowsheet measurements, laboratory results, and discharge disposition [15, 16]. The first chronologically eligible operation per patient was the unit of analysis in the primary cohort. All eligible operations were retained in a sensitivity analysis. The exposure definition, outcome definition, and adjustment set were fixed before the revised analysis. Reporting was structured in accordance with the STROBE principles for observational studies [11].

Eligibility and cohort construction

We included adults aged 18 years or older with valid operating-room entry and exit times, recorded age, sex and ASA physical status, a preoperative hemoglobin value, a baseline creatinine measured within 30 days before surgery, and at least one postoperative creatinine within 7 days after operating-room exit. Baseline creatinine was the latest eligible preoperative value, and postoperative creatinine was the maximum value observed during the 7-day window. Operations entered the monitoring cohort when at

least five valid one-to-one paired arterial and cuff MAP observations were available. Transplant operations were excluded because postoperative creatinine trajectories after transplantation are influenced by graft-specific and immunologic factors. For patients with more than one eligible nontransplant operation, only the first chronologically eligible operation was retained in the primary cohort.

Blood pressure pairing and exposure definition

Arterial MAP was identified from the intraoperative flowsheet labels 'Arterial Line MAP (ART)' and 'MAP-ART A-line'. Cuff MAP was identified from 'NIBP - MAP'. Values outside 30–200 mmHg were treated as invalid. After averaging duplicate values within each device and minute, all eligible ART–cuff candidate pairs within ± 2 minutes were ranked by absolute time difference. The closest eligible pair was retained, and both its arterial and cuff minute were then removed from further matching. Thus, each arterial and cuff minute contributed to at most one pair. This procedure avoids inflating the number of paired observations through many-to-many matching and preserves pair-level timestamps for audit.

The primary exposure was a directional cuff-arterial hypotension-discordance phenotype: arterial MAP < 65 mmHg with paired cuff MAP ≥ 75 mmHg in at least 10% of valid pairs. MAP < 65 mmHg is a widely used intraoperative hypotension threshold [2–7], and the 10-mmHg separation was used to reduce classification of small measurement fluctuations as clinically meaningful discordance. The 10% paired-reading threshold was an operational exposure-burden criterion. Sensitivity analyses used cuff MAP ≥ 65 mmHg, cuff MAP > 75 mmHg to match the strict boundary used in the recent misclassification study [1], and a requirement for at least two qualifying discordant pairs. These proportions describe paired readings and do not represent duration-weighted hypotension burden.

Outcomes

The primary outcome was documented postoperative creatinine doubling, defined as a maximum measured postoperative creatinine ≥ 2 times baseline within 7 days of operating-room exit. This is a creatinine-based endpoint corresponding to the creatinine component of the KDIGO stage 2 threshold; it was not intended to capture all KDIGO stage 2–3 AKI because urine output, kidney-replacement therapy, and other criteria were not evaluated. The first secondary outcome was creatinine-defined postoperative AKI, defined as a maximum postoperative creatinine ≥ 1.5 times baseline within 7 days or an increase of at least 0.3 mg/dL within the first 48 postoperative hours. These thresholds follow the creatinine components of KDIGO criteria [10], and the 7-day postoperative window follows the ADQI–POQI consensus framework for postoperative AKI [9]. The second secondary outcome was in-hospital death, identified from discharge disposition. These definitions were applied to the same operation-level cohort.

Covariates and statistical analysis

The prespecified adjustment set included age, recorded sex, ASA physical status category, logarithm of surgical duration, logarithm of baseline creatinine, preoperative anemia, general anesthesia, inpatient status, broad procedure category, and logarithm of the number of paired MAP observations. Sex was recorded in MOVER as male or female and modeled as a binary covariate. Anemia was defined as hemoglobin < 13.0 g/dL in men or < 12.0 g/dL in women. Procedure categories were derived from the primary procedure name using fixed text rules. The ICU administration flag was excluded because its timing and clinical meaning could not be validated. Logistic regression estimated odds ratios (ORs) and 95% CIs with sandwich robust standard errors. Two-sided $P < 0.05$ was considered statistically significant.

Sensitivity analyses included all eligible operations with patient-level cluster-robust standard errors; alternative cuff thresholds of ≥ 65 and > 75 mmHg; a requirement for at least two qualifying discordant pairs; restriction to operations with at least 10 or at least 20 paired measurements; and additional adjustment for the fraction of paired observations with arterial MAP < 65 mmHg. The last model was considered potentially overadjusted because arterial hypotension is a component of the exposure phenotype.

As a post hoc descriptive analysis, we identified patients whose every available paired cuff MAP was > 65 mmHg, then counted those with at least one paired arterial MAP < 65 mmHg and their postoperative creatinine-doubling events. This analysis was limited to paired readings in the dual-monitored cohort; no inferential comparison was specified.

Results

Cohort construction

The initial eligible clinical and laboratory cohort contained 19,514 operations with valid operating-room times, complete core covariates, preoperative hemoglobin, and baseline and postoperative creatinine measurements. Of these, 5,407 had linked intraoperative monitoring and case data, and 4,188 had at least five valid unique paired MAP observations. Exclusion of 292 transplant operations left 3,896 eligible nontransplant operations from 3,075 patients. The primary cohort retained the first chronologically eligible operation per patient and therefore included 3,075 operations; 821 subsequent eligible operations were excluded from the primary analysis. The primary cohort contained 192 creatinine-doubling events, 659 any-AKI events, and 133 in-hospital deaths. Cohort construction is shown in Fig. 1.

The 5,407 operations with linked monitoring data differed from the 14,107 laboratory-complete operations without linked monitoring data: the linked group was older (mean age, 56.7 vs 53.0 years), had higher baseline creatinine (1.70 vs 1.39 mg/dL), longer operations (274.6 vs 165.8 minutes), and a higher crude any-AKI frequency (26.4% vs 13.8%). This comparison was used to characterize monitoring selection, rather than to impute an exposure for cases without paired blood pressure data.

Figure 1 Cohort construction and primary analysis. The primary exposure required arterial mean arterial pressure (MAP) < 65 mmHg with paired cuff MAP ≥ 75 mmHg in at least 10% of valid pairs.

Baseline characteristics

Baseline and operative characteristics of the primary cohort are presented in Table 1.

Table 1
Baseline and operative characteristics of the primary cohort

Characteristic	Directional discordance (n = 691)	No directional discordance (n = 2384)
Age, years	59.6 (18.5)	57.7 (17.9)
Male sex	446 (64.5%)	1495 (62.7%)
ASA physical status 1	2 (0.3%)	21 (0.9%)
ASA physical status 2	46 (6.7%)	180 (7.6%)
ASA physical status 3	334 (48.3%)	1196 (50.2%)
ASA physical status 4	259 (37.5%)	874 (36.7%)
ASA physical status 5	50 (7.2%)	113 (4.7%)
Baseline creatinine, mg/dL	0.90 (0.70–1.30)	0.80 (0.60–1.10)
Preoperative anemia	491 (71.1%)	1660 (69.6%)
Surgical duration, min	246.0 (169.0-352.5)	260.0 (183.0-375.0)
General anesthesia	663 (95.9%)	2301 (96.5%)
Inpatient class	688 (99.6%)	2372 (99.5%)
Paired MAP measurements	12 (8–20)	13 (8–22)
Cardiac procedure	39 (5.6%)	154 (6.5%)
Neurosurgery/Spine procedure	205 (29.7%)	771 (32.3%)
Vascular procedure	54 (7.8%)	163 (6.8%)
Thoracic procedure	15 (2.2%)	76 (3.2%)
Abdominal/General procedure	152 (22.0%)	472 (19.8%)
Orthopedic/Trauma procedure	98 (14.2%)	371 (15.6%)
Urologic procedure	5 (0.7%)	18 (0.8%)
Head/Neck procedure	18 (2.6%)	66 (2.8%)
Gynecologic/Obstetric procedure	3 (0.4%)	12 (0.5%)
Other procedure	102 (14.8%)	281 (11.8%)

Values are mean (standard deviation), median (interquartile range), or number (percentage), as appropriate. MAP, mean arterial pressure.

Primary and secondary outcomes

Creatinine doubling occurred in 60 of 691 exposed patients (8.68%) and 132 of 2,384 unexposed patients (5.54%), an absolute risk difference of 3.14 percentage points. The crude OR was 1.62, and the adjusted OR was 1.61 (95% CI, 1.16–2.23; $P = 0.0041$). Any AKI occurred in 169/691 exposed patients and 490/2,384 unexposed patients; the adjusted association was not statistically significant (OR, 1.10; 95% CI, 0.89–1.37; $P = 0.3775$). In-hospital death occurred in 39/691 exposed patients and 94/2,384 unexposed patients and was not significantly associated with the exposure (OR, 1.22; 95% CI, 0.81–1.84; $P = 0.3365$).

In the post hoc paired-reading description, 1,637 patients had cuff MAP > 65 mmHg in every available paired observation; 91 (5.56%) had postoperative creatinine doubling. Of these patients, 871 (53.2%) had at least one paired arterial MAP < 65 mmHg, and 56 of 871 (6.43%) had creatinine doubling. Among the remaining 766 patients without a paired arterial MAP < 65 mmHg, 35 (4.57%) had creatinine doubling. These are descriptive counts, not an estimate of missed hypotension among patients monitored only by cuff.

Associations with postoperative outcomes and sensitivity analyses are summarized in Table 2.

Table 2
Associations between cuff–arterial hypotension discordance and postoperative outcomes

Analysis	N	Crude OR	Adjusted OR	95% CI	P value
Primary: creatinine doubling	3075	1.62	1.61	1.16–2.23	0.0041
Secondary: any AKI	3075	1.25	1.10	0.89–1.37	0.3775
Secondary: in-hospital death	3075	1.46	1.22	0.81–1.84	0.3365
All eligible operations, clustered	3896	1.68	1.70	1.28–2.25	0.0003
Cuff MAP $\geq$ 65 mmHg	3075	1.41	1.42	1.05–1.92	0.0214
Cuff MAP > 75 mmHg	3075	1.69	1.65	1.18–2.29	0.0031
$\geq$ 2 discordant pairs	3075	1.73	1.73	1.21–2.48	0.0027
$\geq$ 10 paired measurements	2043	1.66	1.66	1.11–2.48	0.0130
$\geq$ 20 paired measurements	881	1.63	1.84	1.03–3.31	0.0408
Adjusted for arterial MAP < 65 burden	3075	1.62	1.32	0.90–1.95	0.1565

The primary cohort retained one operation per patient. All models adjusted for age, recorded sex, ASA physical status, surgical duration, baseline creatinine, preoperative anemia, general anesthesia, inpatient status, procedure category, and paired-measurement count. The all-operations analysis used patient-level cluster-robust standard errors. AKI, acute kidney injury; MAP, mean arterial pressure; OR, odds ratio.

Sensitivity analyses

The association with creatinine doubling remained significant in the all-operations analysis (OR, 1.70; 95% CI, 1.28–2.25), with cuff thresholds of ≥ 65 mmHg (OR, 1.42; 95% CI, 1.05–1.92) and > 75 mmHg (OR, 1.65; 95% CI, 1.18–2.29), when at least two discordant pairs were required (OR, 1.73; 95% CI, 1.21–2.48), and after restriction to at least 10 or at least 20 paired measurements. Additional adjustment for total arterial MAP < 65 burden attenuated the association (OR, 1.32; 95% CI, 0.90–1.95; $P = 0.1565$).

Discussion

Principal findings

In this retrospective cohort of 3,075 patients with audited time-matched intraoperative blood pressure measurements, the directional cuff-arterial hypotension-discordance phenotype was associated with higher odds of documented postoperative creatinine doubling. The exposed group had an absolute creatinine-doubling risk 3.14 percentage points higher than the unexposed group, and adjusted odds were 61% higher. The association remained significant across alternative cuff thresholds, stricter exposure-frequency requirements, minimum paired-measurement counts, and an analysis retaining all eligible operations. It attenuated and was no longer statistically significant after additional adjustment for the fraction of paired arterial MAP observations below 65 mmHg (OR, 1.32; 95% CI, 0.90–1.95; $P = 0.1565$). The broader creatinine-defined AKI endpoint and in-hospital death were not significantly associated with the primary exposure.

Clinical interpretation

The exposure identifies paired observations in which the invasive arterial MAP was below 65 mmHg while the cuff MAP was at or above 75 mmHg. The association was attenuated after adjustment for arterial hypotension burden; therefore, the result should be interpreted as an association of the discordance phenotype and accompanying arterial hypotension, rather than evidence that device disagreement independently causes kidney injury.

For patients already monitored with both devices, a higher cuff MAP should not by itself dismiss a lower arterial MAP. When marked discordance occurs, clinicians should verify cuff placement and limb, transducer leveling and zeroing, and arterial waveform quality. If the arterial signal remains technically reliable, the lower arterial pressure warrants particular attention and clinical assessment rather than reassurance from the cuff value [1, 12, 13]. Given the higher observed rate of creatinine doubling, postoperative kidney function also merits risk-based assessment, including serum creatinine when clinically indicated [9]. The data do not show whether clinicians recognized or acted on discordance, and this study did not test whether any particular response or surveillance strategy improves outcomes.

The paired data show that a cuff MAP ≥ 75 mmHg can coincide with an arterial MAP < 65 mmHg among dual-monitored patients. In high-risk major surgery managed with cuff monitoring alone, an apparently

adequate cuff value could therefore fail to reveal lower intra-arterial pressure [1, 13]. Our selected dual-monitored cohort cannot quantify how often this occurs in cuff-only patients or whether adding an arterial catheter prevents AKI. The findings do not support placing an arterial catheter in every patient. Invasive monitoring may be considered for major procedures or marked hemodynamic variability when supported by patient- and procedure-specific clinical indications [5, 13]; this remains individualized clinical judgment rather than a benefit demonstrated here.

Relationship to previous work

Prior perioperative studies have linked intraoperative hypotension to postoperative AKI and other organ injury [2–9], while randomized work suggests that individualized pressure management can reduce postoperative organ dysfunction in selected high-risk patients [8]. A recent study quantified blood-pressure misclassification caused by noninvasive oscillometric measurements [1]. A comparative study reported that cuff inflation can alter intra-arterial blood pressure values [12], and a perioperative review summarized measurement limitations and blood-pressure target considerations [13]. Lehman and colleagues analyzed 27,022 paired ICU measurements and evaluated AKI in 1,633 patients; at $\text{MAP} \leq 60$ mmHg, AKI prevalence did not differ significantly by measurement technique [14]. The present study differs in setting, exposure definition, and primary outcome. Its attenuation after adjustment for arterial hypotension burden indicates that the result should be interpreted as an association of the discordance phenotype and its accompanying arterial hypotension, rather than an independent causal effect of device disagreement.

Strengths and limitations

Strengths include audited time-matched measurements, one-to-one pairing that prevents reuse of minute-level readings, one chronologically selected operation per patient in the primary cohort, explicit adjustment for recorded sex, a clinically consequential primary endpoint, and concordant results across several exposure and data-density sensitivity analyses.

The retrospective design limits causal inference. Patients receiving both arterial and cuff monitoring were selected by clinical judgment and may differ from patients monitored with a cuff alone. Requiring postoperative creatinine creates outcome-ascertainment and selection bias because testing is more likely in patients who are clinically unwell or have a renal indication. The exposure is a proportion of paired readings rather than a duration-weighted measure. Matching used a deterministic closest-pair procedure within a two-minute window rather than a global optimization algorithm, and paired values were not necessarily simultaneous. Arterial waveform damping, leveling errors, cuff placement, limb selection, and clinical responses to discordant values could not be fully assessed. Baseline and postoperative creatinine values were independently reconstructed from source laboratory collection timestamps and matched the derived values for all 4,206 operations in the raw pairing audit; however, creatinine sampling may miss transient injury, and postoperative elevation can have causes unrelated to hypotension. Although repeated operations were removed from the primary cohort and retained with clustered inference in sensitivity analysis, external validation is required.

Conclusion

Among adults undergoing surgery with time-matched invasive and cuff blood pressure measurements, arterial MAP < 65 mmHg with cuff MAP $\geq$ 75 mmHg in at least 10% of paired observations was associated with higher odds of documented postoperative creatinine doubling. The association attenuated and was no longer statistically significant after additional adjustment for arterial hypotension burden. In this dual-monitored cohort, a cuff MAP above the hypotension threshold could coincide with arterial hypotension. For similar patients monitored only by cuff, or when a concurrent arterial value is disregarded, such hypotension might go unrecognized, potentially missing an opportunity to identify patients who warrant closer postoperative kidney assessment. For high-risk major operations, clinicians should consider invasive arterial monitoring when patient- and procedure-specific indications support it. This study cannot quantify the frequency of missed hypotension in cuff-only patients or show that monitoring or surveillance changes prevent kidney injury.

Abbreviations

AKI

acute kidney injury

ART

arterial-line measurement

ASA

American Society of Anesthesiologists

CI

confidence interval

KDIGO

Kidney Disease:Improving Global Outcomes

MAP

mean arterial pressure

MOVER

Medical Informatics Operating Room Vitals and Events Repository

NIBP

noninvasive blood pressure

OR

odds ratio

STROBE

Strengthening the Reporting of Observational Studies in Epidemiology.

Declarations

Ethics approval and consent to participate

This secondary analysis used publicly available, deidentified data from the MOVER repository under the repository's data-use terms. No patients were contacted or recruited. Because the analysis used nonidentifiable secondary data, additional ethics approval and individual informed consent were not required.

Consent for publication

All authors have read and approved the final manuscript and consent to its publication. The manuscript contains no individual-level data requiring consent for publication.

Clinical trial number: not applicable.

Competing interests

The authors declare that they have no competing interests.

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Author Contribution

Zitong Liu: Conceptualization, methodology, data curation, formal analysis, visualization, and drafting of the original manuscript. Ziqiao Zhuang: Methodology, interpretation of the results, literature verification, and critical revision of the manuscript. Shuo Wang: Clinical interpretation of the findings, discussion of the relevant literature, and revision of the manuscript. Xiaoyun Xu, Erna Wu, and Fengyuan Ma: Clinical interpretation of the findings and revision of the manuscript. Sihao Chen, Zhaorong Xiao, and Fengzhe Che: Review of the study results and revision of the manuscript. Weiwei Liu: Study supervision, interpretation of the results, critical revision of the manuscript, and final approval. All authors read and approved the final manuscript.

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Data Availability

The datasets used and/or analyzed during the current study are available from the University of California, Irvine Medical Informatics Operating Room Vitals and Events Repository (MOVER) at <https://doi.org/10.24432/C5VS5G> and <https://mover-download.ics.uci.edu/> under the repository's data-use terms. Patient-level data cannot be redistributed by the authors.

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Figures

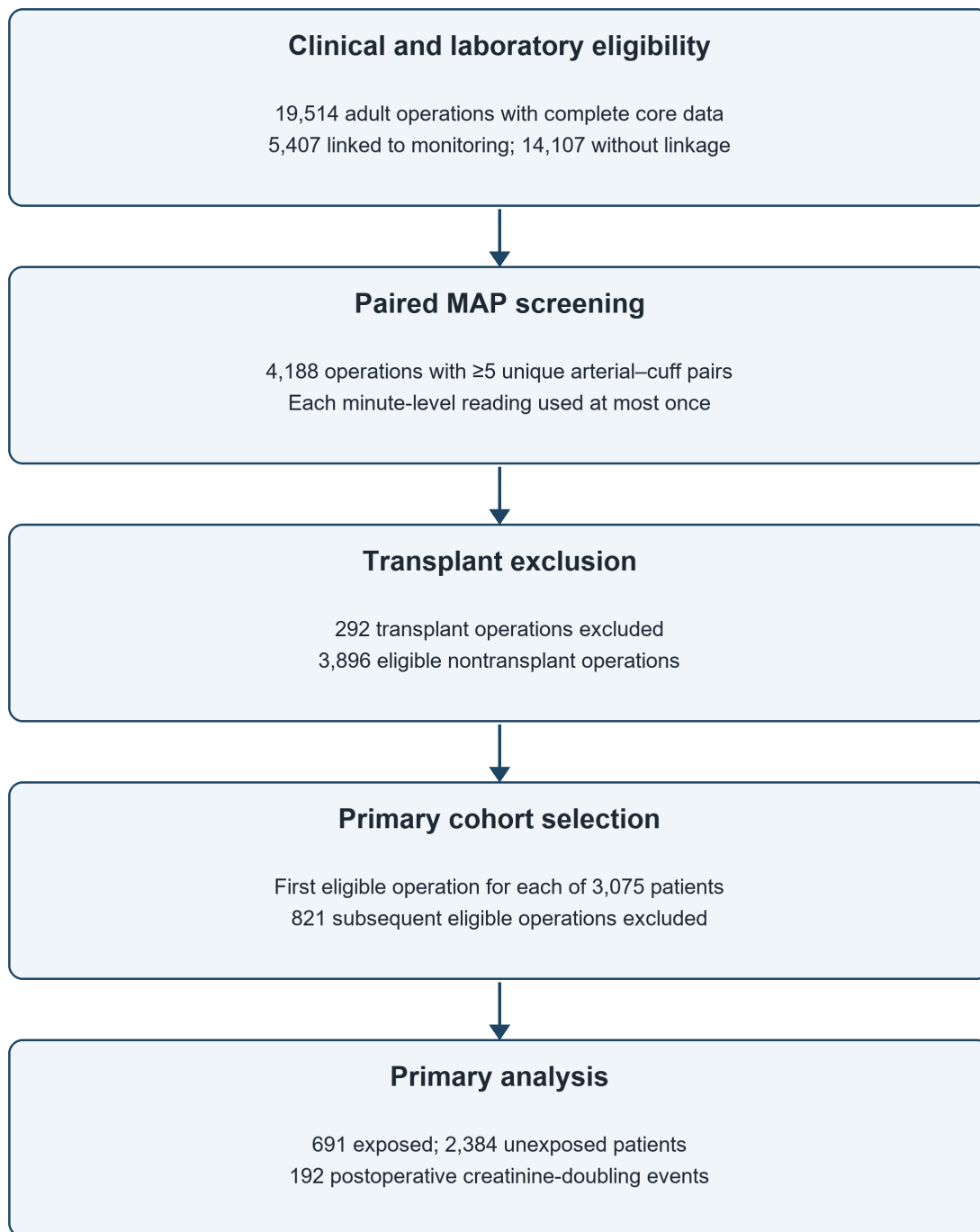


Figure 1

Cohort construction and primary analysis. The primary exposure required arterial mean arterial pressure (MAP) < 65 mmHg with paired cuff MAP ≥ 75 mmHg in at least 10% of valid pairs.