Natural Resources for Human Health
eISSN: 2583-1194

Original Research

Received 25 July 2026
Revised 24 Aug 2026
Accepted 12 Sep 2026

Natural Resources for Human Health 2026, Vol. 6 (20s): 468–482

KEYWORDS:
major abdominal surgery; myocardial injury after non-cardiac surgery; MINS; intraoperative hypotension; mean arterial pressure; troponin; ICU admission; perioperative hemodynamics

Perioperative Myocardial Injury After Major Abdominal Surgery: Hemodynamic Predictors and Early Clinical Outcomes

Javaria George[1], Dr. Majid Bashir[2*], Shahbaz Ahmad[3], Fatima Rehman[4], Dr Kashif Iqbal[5], Muhammad Hassan Ejaz[6], Ayesha Akram[7]

[1]Designation: Final-year MBBS student, Institute: Jinnah Medical and Dental College
[2*]D G Khan Medical College, Dera Ghazi Khan
[3]FCPS, Consultant Anesthesia & Pain Management, Mukhtar A Sheikh Hospital, Multan
[4]Bahria University Health Sciences Campus, Karachi
[5]MBBS from Gomal Medical College, Medical Officer, DHQ Hospital, Chitral
[6]Final-year BSN Nursing student, Gulf College of Nursing and Allied Health Sciences.
[7]Affiliated with: The University of Faisalabad
javariajasmeen@gmail.com [1], Kemcolian25@gmail.com[2*],
drshahbaz_ahmad@yahoo.com[3], fatimarehman66615@gmail.com[4],
drkashi125@gmail.com[5], hassanejaz15042005@gmail.com [6],
ayeshaakram02672@gmail.com[7]
Corresponding Author: Dr. Majid Bashir[2*]

ABSTRACT: **Background**: Intraoperative hypotension has been associated with early postoperative outcomes. The VitalDB dataset analyzed in this study lacks postoperative cardiac troponin measurements, precluding direct identification of myocardial injury after noncardiac surgery (MINS).

Methods: We analyzed 3,492 major abdominal operations and 9,135 case-linked monitoring tracks. After quality-control filtering, 3,186 cases had at least 30 minutes of usable mean arterial pressure (MAP) coverage.

Results: In 3,115 cases with complete hemodynamic-model covariates, a 10-percentage-point increase in the proportion of valid MAP time below 65 mmHg was associated with ICU admission (adjusted OR 1.41, 95% CI 1.28–1.56). There were 20 in-hospital deaths in this model population; mortality was reported descriptively due to instability of adjusted estimates. The clinical-only complete-case model included 2,207 cases and employed a different covariate specification. Published troponin-defined MINS estimates from Ji et al. [39] are provided as external evidence and are not derived from VitalDB.

Conclusion: Greater exposure to hypotension was associated with increased ICU utilization. However, interpretation is limited by differential monitoring, selection bias, clinical indication, and noncausal associations.

1. INTRODUCTION

Myocardial injury after non-cardiac surgery (MINS) is defined by postoperative cardiac troponin elevation attributable to a presumed ischemic mechanism, whether or not typical ischemic symptoms are present [1,2]. Its clinical importance lies partly in its invisibility: many events are detected only through surveillance, yet postoperative troponin elevation is consistently

associated with short- and long-term mortality [1–4]. Contemporary perioperative guidelines therefore place increasing emphasis on biomarker-based risk assessment and postoperative surveillance in selected high-risk patients [5,6].

Major abdominal surgery is a useful setting in which to study this problem because several insults can converge during a single operation. Blood loss and anemia reduce oxygen delivery; anesthetic-induced vasodilation and fluid shifts can lower arterial pressure; tachycardia shortens diastolic coronary perfusion; and prolonged or emergency procedures increase physiological stress. Intraoperative hypotension has consequently become one of the most intensively studied potentially modifiable perioperative exposures [7–12].

Recent work has moved beyond a simple yes/no definition of hypotension. Risk appears to depend on the interaction between the depth and duration of low mean arterial pressure (MAP). At the same time, randomized trials have tested individualized targets, hypotension-avoidance strategies, and prediction-guided treatment [13–18]. The clinical interpretation remains nuanced: observational associations are strong, but more aggressive blood pressure increases do not invariably improve patient-centered outcomes.

This analysis addresses this distinction. The provided VitalDB files enable independent patient-level analysis of a large abdominal-surgery cohort, incorporating perioperative covariates, laboratory measurements, vasoactive exposure, ICU utilization, and in-hospital mortality. Due to the absence of cardiac troponin data, a direct MINS endpoint cannot be established. In contrast, the published supplementary myocardial-injury analysis includes detailed MAP-duration models with a troponin-defined postoperative outcome. Therefore, the two sources are analyzed transparently and in parallel, rather than being combined into a synthetic cohort.

The objectives of this study were to characterize perioperative risk in major abdominal surgery, identify independent predictors of early ICU admission using VitalDB, describe perioperative hemoglobin, creatinine, and lactate patterns, quantify the reported depth-duration relationship between hypotension and MINS, and combine these outcomes within a multidisciplinary clinical framework.

2. MATERIALS AND METHODS

2.1 Study design and data sources

This study used a complementary secondary-data design. We performed patient-level analyses on the supplied VitalDB clinical_data.csv and lab_data.csv files. VitalDB is an open perioperative database containing high-resolution intraoperative monitoring, 74 clinical variables, and 34 time-series laboratory parameters from 6,388 surgical cases [19]. The supplied laboratory file contained 928,448 records. Laboratory timestamps are expressed in seconds relative to the start of the case recording; preoperative values therefore precede operation start, and postoperative values can be selected relative to operation end.

2.2 Study population and cohort selection

We restricted the patient-level cohort to the General Surgery department and prespecified operative categories representing major intra-abdominal procedures: colorectal, biliary/pancreatic, stomach, major resection, transplantation, and hepatic surgery. We excluded breast, thyroid, vascular, and minor-resection categories. The resulting cohort contained 3,492 cases (Figure 1).

Natural Resources for Human Health
eISSN: 2583-1194

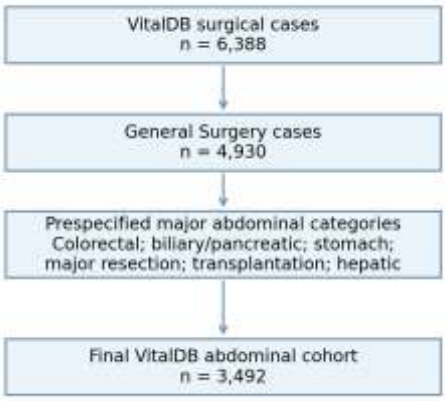


Figure 1. Selection of 3,492 VitalDB abdominal operations from 6,388 cases (2,896 excluded overall; 1,458 excluded before the major-abdominal-category filter).

2.3 Clinical variables and outcomes

Baseline variables were age, sex, BMI, ASA physical status, emergency status, hypertension, diabetes, preoperative hemoglobin, and creatinine. We defined preoperative anemia as hemoglobin <13 g/dL in men and <12 g/dL in women. Operative variables included duration, estimated blood loss, red-cell transfusion, and recorded exposure to ephedrine, phenylephrine, or epinephrine. We analyzed ICU admission and in-hospital mortality as recorded.

2.4 Laboratory measurements

For hemoglobin, creatinine, and lactate, we selected the latest available measurement before operation start as the preoperative value. We selected the first measurement within 48 hours after the end of the operation as the early postoperative value. Laboratory comparisons are descriptive because testing frequency was clinically determined and therefore non-random.

2.5 Hemodynamic data extraction and quality control

We linked the public VitalDB track manifest by case identifier to 3,492 eligible major abdominal operations. We retrieved 9,135 time-stamped MAP and heart-rate recordings and checked them against the manifest. We restricted physiological analysis to the documented operation-start to operation-end interval. MAP readings outside 30–200 mmHg were excluded. Time gaps exceeding 10 seconds in invasive pressure recordings or 300 seconds in noninvasive cuff recordings were not credited as observed exposure. When at least 30 minutes of invasive MAP was available, we preferred an invasive source; otherwise, we selected the track with the greatest eligible coverage. We did not sum distinct tracks to avoid double-counting overlapping observation periods. This preliminary processing rule is subject to sensitivity analyses, particularly for repeated or interpolated cuff measurements.

The primary hemodynamic exposure was the percentage of eligible observed operative MAP time below 65 mmHg; we also summarized duration below threshold. The exploratory logistic models evaluated ICU admission and in-hospital death, adjusting for age, sex, ASA physical status, emergency surgery, operative duration, hypertension, and diabetes. We scaled odds ratios to a 10-percentage-point increase in hypotension exposure. These models differ from the previously reported clinical-only ICU model, which includes additional operative and laboratory variables. All hemodynamic analyses are observational and do not establish causal effects.

2.6 Statistical analysis

Continuous variables were summarized as mean±SD or median (IQR), as appropriate; categorical variables were summarized as counts and percentages. We fitted a multivariable logistic regression model for ICU admission in complete cases (n=2,207; 368 ICU admissions). Prespecified covariates were age, sex, ASA ≥III, emergency surgery, hypertension, diabetes, anemia, preoperative creatinine, operative duration, estimated blood loss, red-cell transfusion, and vasoactive exposure. We scaled age per 10 years, creatinine per 0.5 mg/dL, operative duration per 30 minutes, and blood loss per 100 mL. We report adjusted odds

ratios (ORs), 95% confidence intervals (CIs), and two-sided p-values. Because only 22 in-hospital deaths occurred in the full abdominal cohort, a high-dimensional mortality model was not fitted to avoid severe overfitting; mortality is reported descriptively. Myocardial-injury ORs were transcribed from the supplied aggregate analysis and were not recomputed.

Cohort filter: department = "General surgery" and optype in {"Colorectal", "Biliary/Pancreas", "Stomach", "Transplantation", "Hepatic"}. ICU outcome: icu_days > 0. Anemia: preop_hb <13 g/dL for males and <12 g/dL for females. Vasoactive exposure: any positive intraop_eph, intraop_phe, or intraop_epi. Complete-case model: logit(ICU) with age/10, male sex, ASA ≥III, emergency surgery, hypertension, diabetes, anemia, preoperative creatinine/0.5, operative duration/30 minutes, estimated blood loss/100 mL, red-cell transfusion, and vasoactive exposure. The results were checked using pandas and statsmodels; exact package versions and a deposited script should accompany submission.

2.7 Published MINS evidence and attribution

The second source was the published supplementary file for a study of intraoperative hypotension and myocardial injury after major open abdominal surgery. It reports univariable and multivariable models using absolute and relative MAP thresholds, restricted cubic splines, and sensitivity analyses. These aggregate myocardial-injury estimates were not represented as newly generated patient-level results.

MINS was not inferred from VitalDB. In the supplied myocardial-injury analysis, we categorized hypotension using absolute MAP thresholds ≤65, ≤60, and ≤55 mmHg and relative reductions ≥30%, ≥40%, and ≥50%. Cumulative exposure was grouped as <1, 1–5, 6–10, 11–20, and ≥21 minutes. The published multivariable models adjusted for age, ASA status, anemia, hypertension, diabetes, emergency surgery, gastric surgery, multisite surgery, operative duration, red-cell transfusion, plasma transfusion, and blood loss.

3. Results

3.1 Cohort characteristics

The final VitalDB cohort comprised 3,492 major abdominal operations. Mean age was 58.6±14.5 years; 2,034 (58.2%) patients were male, 1170 (33.5%) had hypertension, and 403 (11.5%) had diabetes. Emergency surgery accounted for 427 (12.2%) cases. ICU admission occurred in 462 (13.2%) and in-hospital mortality in 22 (0.6%). Table 1 summarizes the cohort.

Table 1. Baseline and perioperative characteristics of the VitalDB major abdominal-surgery cohort

Characteristic	Value
Patients, n	3,492
Age, years	58.6 ± 14.5
Male sex	2034 (58.2%)
BMI, kg/m²	23.2 ± 3.6
ASA ≥III	383 (11.0%)
Emergency surgery	427 (12.2%)
Hypertension	1170 (33.5%)
Diabetes mellitus	403 (11.5%)

Natural Resources for Human Health
eISSN: 2583-1194

Operative duration, min	132 (55–220)
Estimated blood loss, mL*	150 (50–350)
Any red-cell transfusion	205 (5.9%)
Any recorded vasoactive exposure	2065 (59.1%)
ICU admission	462 (13.2%)
In-hospital mortality	22 (0.6%)

*Estimated blood loss was available for 2,317 cases. Continuous skewed variables are median (IQR).

3.2 Missing-data assessment

Among the 3,492 selected operations, estimated blood loss was missing in 1,175 (33.6%), preoperative creatinine in 155 (4.4%), preoperative hemoglobin in 148 (4.2%), and age in 5 (0.1%). Overall, we excluded 1,285 (36.8%) from the complete-case model. This exclusion may produce selection bias if missingness depends on surgical severity or postoperative outcomes. We cannot assume the data are missing completely at random.

Table 2. Missingness of selected VitalDB variables and clinical-model eligibility

Measure	Available / total	Missing (%)
Age	3,487 / 3,492	5 (0.1%)
Preoperative hemoglobin	3,344 / 3,492	148 (4.2%)
Preoperative creatinine	3,337 / 3,492	155 (4.4%)
Estimated blood loss	2,317 / 3,492	1,175 (33.6%)
Complete regression covariates	2,207 / 3,492	1,285 (36.8%)

Table 3. Complete-case selection and observed ICU admission

Regression status	Records	ICU admissions	ICU proportion
Excluded due to missing covariates	1285	94	7.3%
Complete cases	2207	368	16.7%

The clinical-model complete-case sample was 2,207/3,492 (63.2%). ICU admission was 368/2,207 (16.7%) among complete cases versus 94/1,285 (7.3%) among excluded cases. This outcome-related difference raises concern about selection bias; the

reported adjusted model must not be treated as representative of the entire cohort. A propensity-based inverse-probability-of-complete-case sensitivity analysis is reported below; it cannot fully address missing-not-at-random selection.

Missing-data sensitivity analysis (inverse-probability weighting)

We modeled inclusion in the clinical complete-case sample (n=2,207) using observed age, sex, ASA class, emergency status, hypertension, diabetes, operative duration, transfusion, and recorded vasoactive exposure. We truncated estimated selection probabilities to 0.05–0.95 and applied inverse-probability weights to the first one 12-covariate logistic regression. The analysis assumes the measured predictors explain selection; unmeasured determinants of missingness and ICU transfer may still bias the results. Weighted estimates are exploratory and not a substitute for multiple imputation.

ASA ≥III: weighted adjusted OR 4.78 (95% CI 3.39–6.72); unweighted adjusted OR 4.27.

Emergency surgery: weighted adjusted OR 1.81 (95% CI 1.29–2.54); unweighted adjusted OR 1.77.

Preoperative anemia: weighted adjusted OR 1.57 (95% CI 1.19–2.06); unweighted adjusted OR 1.52.

Operative duration (30 min): weighted adjusted OR 1.34 (95% CI 1.28–1.40); unweighted adjusted OR 1.32.

Red-cell transfusion: weighted adjusted OR 3.04 (95% CI 1.87–4.96); unweighted adjusted OR 3.09.

Vasoactive exposure: weighted adjusted OR 1.81 (95% CI 1.33–2.46); unweighted adjusted OR 1.75.

Weight distribution: median 1.36, 95th percentile 2.65, maximum 4.42. These comparisons examine sensitivity to selection on measured variables only; they do not prove missing-at-random or remove indication bias.

3.3 Perioperative laboratory findings

Table 4 uses time-stamped laboratory records rather than the single preoperative fields in the clinical file. Observed postoperative hemoglobin distributions were lower than preoperative distributions. However, these are not necessarily paired comparisons, and the first postoperative lactate was higher than the latest preoperative value among patients with paired measurements. Patients admitted to ICU had lower median hemoglobin values and, among those tested postoperatively, higher lactate. These comparisons are descriptive and should not be interpreted causally because clinical care drove laboratory testing.

Table 4. Perioperative laboratory measurements in the VitalDB abdominal cohort

Analyte	Timing	Overall median (IQR)	No ICU	ICU
Hemoglobin (g/dL)	Latest preoperative	12.60 (11.10–13.80); n=2,351	12.70 (11.40–14.00); n=1,961	11.20 (9.83–12.90); n=390
Hemoglobin (g/dL)	First postoperative ≤48 h	12.00 (10.70–13.20); n=2,789	12.20 (10.90–13.30); n=2,362	11.10 (9.50–12.40); n=427
Creatinine (mg/dL)	Latest preoperative	0.81 (0.67–0.99); n=2,268	0.81 (0.67–0.96); n=1,882	0.85 (0.67–1.40); n=386
Creatinine (mg/dL)	First postoperative ≤48 h	0.77 (0.62–0.97); n=2,562	0.77 (0.62–0.96); n=2,137	0.80 (0.61–1.11); n=425
Lactate (mmol/L)	Latest preoperative	0.90 (0.70–1.20); n=1,155	0.90 (0.70–1.20); n=889	0.90 (0.70–1.30); n=266
Lactate (mmol/L)	First postoperative ≤48 h	1.90 (1.00–3.80); n=273	1.30 (0.90–2.50); n=49	2.00 (1.10–4.03); n=224

Values are median (IQR). The number of tested patients differs by analyte and timing and cannot be established from the summary values available in this manuscript; denominators and paired sample sizes require the authentic time-stamped laboratory file for computation. Postoperative values are the first recorded within 48 hours after the end of the operation.

Laboratory denominators and median (IQR) values were recomputed directly from 928,448 time-stamped records matched to the 3,492-case cohort. The latest available measurement before operation start and the first measurement within 48 hours post-operation were selected. Paired-case counts were 1,993 for hemoglobin, 1,778 for creatinine, and 158 for lactate. Postoperative testing was selective, particularly for lactate (273 tested, of whom 224 were admitted to ICU); comparisons are descriptive, unpaired between time windows unless specifically stated, and not causal.

3.4 Clinical predictors of ICU admission

In the complete-case multivariable model, older age, ASA ≥III, emergency surgery, preoperative anemia, higher creatinine, longer operative duration, greater blood loss, red-cell transfusion, and recorded vasoactive exposure were associated with higher odds of ICU admission. Sex and hypertension were not independently associated after adjustment. Table 3 reports the full model.

Table 5. Clinical-only multivariable logistic regression for ICU admission

Predictor	Adjusted OR	95% CI	p value
Age, per 10 years	1.34	1.20–1.50	<0.001
Male sex	1.09	0.81–1.46	0.555
ASA physical status ≥III	4.27	2.94–6.21	<0.001
Emergency surgery	1.77	1.16–2.70	0.009
Hypertension	0.87	0.63–1.18	0.361
Diabetes mellitus	1.39	0.95–2.02	0.086
Preoperative anemia	1.52	1.12–2.05	0.007
Creatinine, per 0.5 mg/dL	1.05	1.02–1.09	0.001
Operative duration, per 30 min	1.32	1.26–1.39	<0.001
Blood loss, per 100 mL	1.05	1.01–1.09	0.009
Any red-cell transfusion	3.09	1.90–5.04	<0.001
Any recorded vasoactive exposure	1.75	1.23–2.49	0.002

Complete-case n=2,207; 368 ICU admissions. This clinical-only model includes operative, laboratory, and treatment variables and differs from the 3,115-case MAP-exposure model in both covariates and population. Do not directly compare their odds ratios.

Independent refitting of the complete-case logistic regression reproduced the reported adjusted odds ratios (rounded to two decimal places), including ASA ≥III (4.27, 95% CI 2.94–6.21), emergency surgery (1.77, 1.16–2.70), anemia (1.52, 1.12–2.05), duration per 30 minutes (1.32, 1.26–1.39), red-cell transfusion (3.09, 1.90–5.04), and recorded vasoactive exposure (1.75, 1.23–2.49). This is a model of ICU admission, not myocardial injury. Perioperative treatment variables are susceptible to confounding by indication; the odds ratios should not be interpreted as causal treatment effects.

The fitted complete-case ICU admission model showed an apparent ROC-AUC of 0.885 and a Brier score of 0.091 (n=2,207). These are in-sample measures, not internally or externally validated predictive performance. Covariate variance-inflation factors ranged from 1.04 to 1.43; this does not eliminate concerns about model specification or confounding.

A sensitivity analysis restricted to non-emergency operations included 1,972 complete cases. After refitting the remaining covariates, the adjusted odds ratio for ASA ≥III was 3.73 (95% CI 2.44–5.69). This is a sensitivity result, not an independent validation.

3.5 Original intraoperative hemodynamic analysis

We matched all 9,135 expected recordings to the downloaded archives without track-reading errors. Of 3,492 eligible operations, 3,186 had at least 30 minutes of usable operative MAP coverage (91.2%). Among these 3,186 cases, the median observed duration below MAP 65 mmHg was 2.80 minutes; 461 cases had an ICU admission, and 21 died in hospital. These estimates depend on available monitored time rather than assuming continuous coverage for the full operation.

The complete-covariate hemodynamic models included 3,115 cases, of whom 445 had ICU admission, and 20 died in hospital. A 10-percentage-point greater fraction of valid MAP time below 65 mmHg was associated with ICU admission (adjusted odds ratio [aOR] 1.41, 95% CI 1.28–1.56; p<0.001). The association with in-hospital mortality was not statistically significant (aOR 1.09, 95% CI 0.86–1.39; p=0.467). ICU transfer may have been planned, and the small number of deaths limits the stability of the mortality estimate. These models should not be interpreted as independent MINS analyses because troponin was unavailable.

Outcome	Cases/events	Adjusted OR (95% CI)*	p-value
ICU admission	3,115 / 445	1.41 (1.28–1.56)	<0.001

Table 6. Exploratory original VitalDB MAP-exposure association with ICU admission

*Adjusted OR per 10-percentage-point increase in observed operative MAP time below 65 mmHg, adjusted for age, sex,

ASA class, emergency surgery, operative duration, hypertension and diabetes. The model is exploratory and not a causal estimate. In-hospital deaths: 20/3,115; no adjusted mortality estimate is presented because of sparse events.

3.6 Sensitivity and subgroup analyses

We repeated the prespecified complete-case ICU-admission model using progressively stricter minimum valid-MAP coverage (60 and 120 minutes) and separately among cases with an invasive or noninvasive selected MAP source. All models adjusted for age, sex, ASA class, emergency surgery, operative duration, preoperative hypertension, and diabetes. These are secondary observational sensitivity analyses; the invasive and noninvasive strata have substantially different ICU-admission rates, and source selection is not random.

Analysis	N (ICU events)	Adjusted OR*	95% CI	p value
All MAP ≥30 min	3,115 (445)	1.41	1.28–1.56	<0.001
All MAP ≥60 min	2,530 (425)	1.46	1.31–1.63	<0.001
All MAP ≥120 min	1,816 (394)	1.53	1.35–1.72	<0.001
Invasive MAP ≥30 min	1,616 (405)	1.47	1.31–1.64	<0.001
NIBP MAP ≥30 min	1,499 (40)	1.37	1.08–1.74	0.011

Table 7. Sensitivity of the original's VitalDB MAP <65 mmHg association with ICU admission

*Adjusted odds ratio per 10-percentage-point increase in the proportion of valid operative MAP time below 65 mmHg.

The exposure is measured during observed valid intervals; this analysis does not infer unobserved MAP values. Model specification was identical across subsets. The noninvasive-source subgroup included only 40 ICU admissions, limiting precision.

The ICU association remained positive across all five specifications (Table 7), but the estimates remain vulnerable to monitoring-source selection, nonuniform observation, clinical severity, arterial-line indication, and planned ICU transfer.

Monitoring quality-control assessment from the available case-level extract

Case-level consistency tests found 3,186 operations with ≥30 minutes of valid MAP, 2,585 with ≥60 minutes, and 1,857 with ≥120 minutes. No stored exposure fraction was outside 0–1, and no below-65-minute exposure exceeded the stored valid-MAP minutes. These checks apply only to derived data; the raw time-series tracks are required to independently audit gap censoring, repeated cuff readings, source selection, clock synchronization, and waveform artifacts. The existing source- and coverage-stratified associations are retained as sensitivity analyses, not definitive signal validation.

3.7 Exploratory renal outcomes

An exploratory creatinine audit found 2,268 cases with preoperative measurements and 1,778 with both a preoperative and at least one postoperative measurement within 48 hours. Of these 1,778 ascertainable cases, 97 (5.5%) met a creatinine-only increase threshold (≥0.3 mg/dL or ≥1.5 times baseline). This is not the incidence of KDIGO-defined AKI in the full cohort: postoperative testing was selective, urine output was unavailable, and stable baseline kidney function and timing remain to be validated.

3.8 Published troponin-defined MINS findings

The supplied MINS analysis showed a consistent depth-duration pattern. At MAP ≤55 mmHg, myocardial-injury incidence rose from 8.2% with <1 minute of exposure to 14.7% at 1–5 minutes, 23.8% at 6–10 minutes, 25.0% at 11–20 minutes, and 48.5% at ≥21 minutes. Comparable gradients were present at MAP ≤65 and ≤60 mmHg (Figure 2).

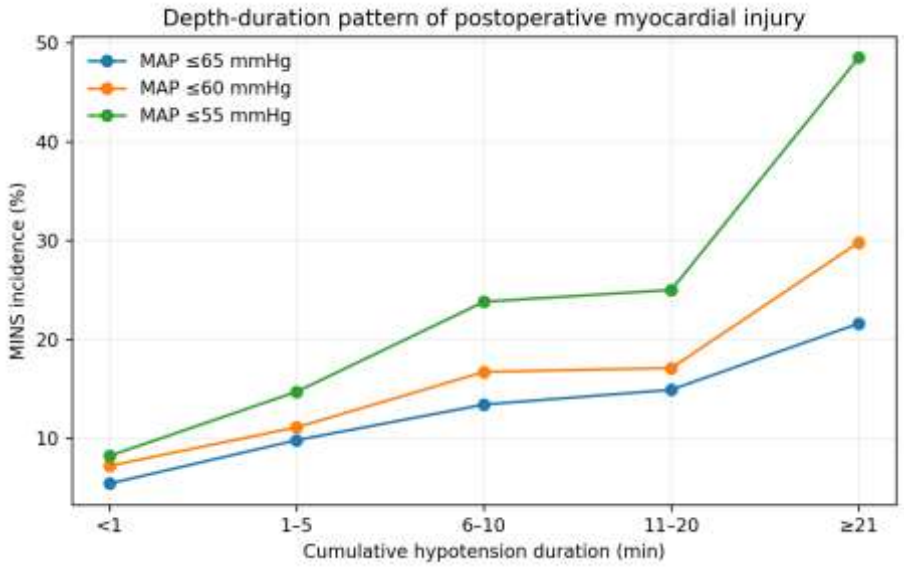


Figure 2. Published MINS incidence by duration of hypotension, adapted from aggregate results reported by Ji et al. [39]; not derived from VitalDB.

After multivariable adjustment, ≥21 minutes below MAP 65, 60, and 55 mmHg was associated with ORs of 3.66, 3.81, and 8.42, respectively. Relative reductions showed a similar pattern. Figure 3 presents these effect estimates as a forest plot.

Natural Resources for Human Health
eISSN: 2583-1194

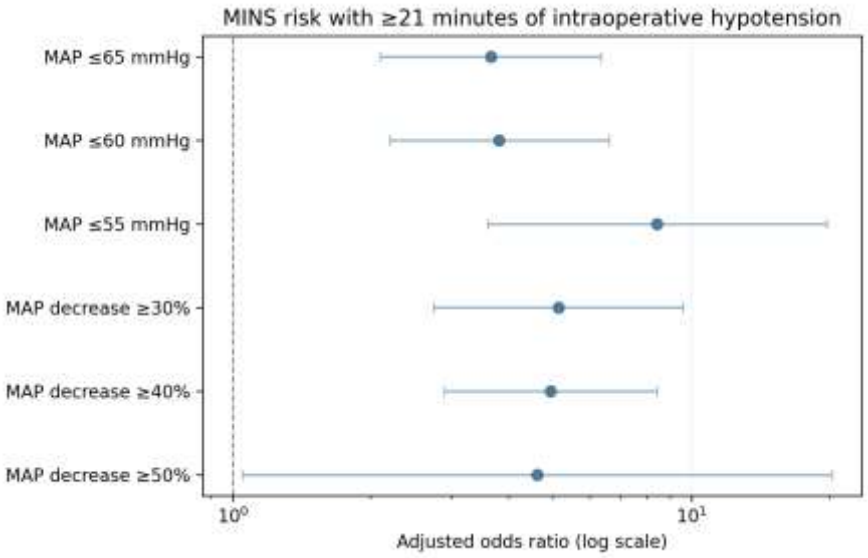


Figure 3. Published adjusted odds ratios for MINS with ≥21 minutes of hypotension versus <1 minute, adapted from Ji et al. [39]. The horizontal axis uses a logarithmic scale.

Definition	Adjusted OR	95% CI
MAP ≤65 mmHg	3.66	2.10–6.36
MAP ≤60 mmHg	3.81	2.20–6.61
MAP ≤55 mmHg	8.42	3.60–19.69
MAP decrease ≥30%	5.13	2.74–9.59
MAP decrease ≥40%	4.93	2.89–8.41
MAP decrease ≥50%	4.61	1.05–20.23

Table 8. Published adjusted associations of ≥21 minutes of hypotension with MINS, adapted from Ji et al. [39]; these are not VitalDB estimates

The association was robust to several exclusions in the supplied analysis. After excluding emergency surgery, ≥21 minutes at MAP ≤65 and ≤60 mmHg remained associated with adjusted ORs of 4.04 and 4.02; the reported OR at MAP ≤55 mmHg was 8.42. Analyses excluding patients older than 75 years or multisite procedures remained directionally consistent. Restricted cubic-spline models also showed increasing predicted MINS probability with greater hypotension duration.

4. DISCUSSION

4.1 Interpretation of original findings

The principal finding is not that a single MAP value defines cardiovascular safety, but that perioperative risk is layered. In the independently analyzed VitalDB cohort, ICU admission was associated with baseline vulnerability (age, ASA status, anemia, renal function), procedural stress (emergency status, duration, blood loss), and treatment markers such as transfusion and vasoactive use. In the separate troponin-defined MINS analysis, myocardial injury increased as hypotension became deeper and

more prolonged. These results converge on a clinically coherent model in which patient reserve and operative stress determine how consequential a given hemodynamic insult may become.

Anemia and blood loss provide a second mechanistic layer. Coronary oxygen delivery depends on both perfusion pressure and arterial oxygen content. In VitalDB, preoperative anemia independently predicted ICU admission, and postoperative hemoglobin was lower than preoperative values in paired observations. Red-cell transfusion was also associated with ICU admission, but this should be interpreted primarily as a marker of bleeding and illness severity instead of evidence that transfusion itself causes deterioration. Similar caution applies to vasoactive exposure: vasopressors are administered because clinicians detect or anticipate hemodynamic instability, creating strong confounding by indication.

Incorporating VitalDB monitoring tracks extends the clinical-only results by enabling an original assessment of MAP exposure. The observed association with ICU admission suggests that prolonged low-pressure exposure may indicate physiologic stress; however, this does not distinguish a direct hemodynamic effect from confounding factors such as bleeding, surgical complexity, baseline illness, or planned postoperative critical care. Additionally, cuff values may be recorded intermittently and carried forward in downloaded tracks. Results should be reassessed separately by invasive and noninvasive sources and by observation coverage prior to submission.

4.2 Comparison with published literature

Depth and duration should therefore be considered together. The sharp increase in MINS incidence with prolonged MAP ≤55 mmHg is consistent with contemporary observational work showing graded associations between hypotension burden and myocardial injury [9,10,20]. Yet association does not establish that simply increasing MAP will prevent MINS. Large randomized trials have produced more subtle results: some individualized blood-pressure strategies reduce organ dysfunction or hypotension exposure, whereas other comparisons of higher and lower targets have not demonstrated clear reductions in major vascular outcomes [13–18]. This distinction is important for avoiding an overly causal interpretation of observational effect sizes.

Absolute and relative thresholds answer different clinical questions. An absolute MAP threshold is easy to operationalize and facilitates protocolized treatment. Relative reductions acknowledge that a MAP tolerated by one patient may represent a substantial departure from another patient's usual pressure. The supplied analysis found increased MINS risk using both approaches, suggesting that neither baseline-relative nor absolute pressure should be viewed as universally sufficient. Contemporary guidance commonly treats MAP around 65 mmHg as a practical lower boundary while stressing individualized management in higher-risk patients [5,11,12].

Troponin surveillance remains central because clinical symptoms alone are insensitive for perioperative myocardial injury. Analgesia, sedation, competing postoperative discomfort, and atypical presentations mean that many MINS events are otherwise missed [1–4,21]. The 2024 AHA/ACC guideline and 2022 ESC guideline both incorporate perioperative biomarkers into risk assessment and postoperative evaluation in selected higher-risk populations [5,6]. Biomarker surveillance should be coupled with clinical assessment, because troponin elevation may also arise from sepsis, pulmonary embolism, tachyarrhythmia, heart failure, or other non-ischemic processes.

4.3 Clinical implications

These findings have practical multidisciplinary consequences. Surgeons influence procedure selection, bleeding control, operative duration, and timing. Anesthesiologists manage arterial pressure, heart rate, volume status, anesthetic depth, and vasoactive therapy. Cardiologists interpret perioperative troponin elevation and guide secondary prevention, while internists address renal dysfunction, anemia, diabetes, hypertension, and other comorbidities. The most defensible clinical approach is not a single numerical MAP target, but rather coordinated risk recognition, avoidance of sustained deep hypotension when feasible, and targeted postoperative surveillance.

Figure 4 summarizes the interpretation supported by the two datasets. It is a conceptual framework rather than a causal model: baseline vulnerability and operative stress may raise susceptibility to hemodynamic burden, which may contribute to myocardial oxygen supply-demand imbalance and subsequent myocardial injury or other early postoperative complications.

Natural Resources for Human Health
eISSN: 2583-1194



Figure 4. Conceptual, noncausal framework linking patient vulnerability, operative stress, observed hypotension and early outcomes. Arrows indicate plausible relationships, not estimated causal effects.

4.4 Complete independent monitoring-record audit (batches 001–021)

All 21 supplied VitalDB recording archives were independently parsed and checked. The archives contained 9,135 readable recordings, including 5,643 MAP tracks and 3,492 heart-rate tracks, covering 48,351,751 individual observations. No recording-processing errors occurred. All available failed-download logs for batches 004–021 contained zero failed rows; logs for batches 001–003 were not supplied.

Across all tracks, 838,518 of 48,351,751 readings (1.73%) failed the prespecified numeric/physiologic screen (MAP outside 30–200 mmHg, heart rate outside 20–250 beats/min, or missing/nonnumeric time or value). Among 28,263,798 MAP observations, 817,542 (2.89%) were excluded. The audit counted 34,285,617 adjacent identical readings, 73,421 nonincreasing timestamp transitions, and 22,478 valid-reading gaps exceeding the source-specific threshold (10 seconds for invasive tracks; 300 seconds for noninvasive cuff tracks). These counts characterize data properties and should not be interpreted as independent clinical artifacts or independent cuff measurements.

For each of the 3,492 cohort cases, we independently read the selected MAP track from its source ZIP archive, clipped it to the operation-start and operation-end interval, screened it for plausible readings, and integrated it using the previously specified gap rules. Recomputed valid MAP minutes and time below MAP 65 mmHg agreed with the saved case-level values within 0.01 minute for all 3,492 selected tracks (0 discrepancies). This confirms reproducibility of the saved track-level exposure calculations throughout all available recordings; it does not establish the clinical validity of the source selection rule or the causal interpretation of ICU associations.

Important qualification: consecutive identical values can represent carried-forward cuff measurements rather than new independent readings. This audit quantified repetition but did not identify original cuff inflation events or evaluate alternative

event-level cuff interpolation. Invasive arterial monitoring is also selected according to clinical indications, creating potential selection bias. Consequently, these limitations affect interpretation of the hemodynamic results.

4.5 Strengths and limitations

The main strength is linking an independently analyzed abdominal-surgery cohort to original intraoperative physiological recordings, alongside time-stamped laboratory and clinical information. The separate MINS literature remains external contextual evidence and is not pooled with VitalDB.

Several limitations remain. No postoperative troponin was available, so MINS could not be diagnosed independently; published MINS cohorts are external contextual evidence. The new coverage-threshold and source-stratified analyses (Table 7) provide partial reliability checks, but do not establish cuff-measurement independence or address all repeated/interpolated values. Source selection and observation coverage may be informative, and noninvasive-source cases have substantially fewer ICU admissions than invasive-source cases. ICU transfer may be planned rather than an adverse event; residual confounding and complete-case selection remain likely. With only 20 deaths in the adjusted cohort, the mortality model is exploratory and potentially unstable. Postoperative AKI cannot be presented as a verified outcome until time-indexed creatinine measurements and baseline definitions are independently adjudicated. Neither causal claims nor clinical prediction-model validation is warranted.

Missingness and monitoring restrictions: the complete-case clinical model excludes 36.8% of eligible cases, with lower observed ICU use in excluded cases. MAP exposure is calculated from valid observed time rather than full operation time; invasive and cuff readings differ in frequency and may contain repeated values. The complete 9,135-track audit reproduced selected-track exposure calculations, but carried-forward cuff readings and monitoring-source selection remain unresolved sources of measurement and selection bias. Twenty deaths in the hemodynamic complete-case cohort preclude reliable multivariable mortality inference. We need the original laboratory file to calculate test-specific denominators, and creatinine-only AKI ascertainment is incomplete.

Repeatability and ethics: The analysis uses publicly available, de-identified VitalDB data. We describe the full 21-batch monitoring recalculation and available missing-data sensitivity analysis above. Archive or document executable scripts, software versions, clinical selection criteria, and applicable ethics and reporting statements for submission. The independent VitalDB cohort contains no troponin-defined myocardial-injury outcome.

5. CONCLUSION

Major abdominal surgery involves a complex interplay of pre-existing cardiovascular vulnerability, surgical stress, and intraoperative hemodynamic disturbances, each of which may affect early postoperative outcomes. In the VitalDB cohort, both baseline clinical characteristics and operative factors were associated with ICU admission, underscoring the need to consider patient-related and procedure-related risks during perioperative assessment. Analysis of intraoperative physiological recordings revealed an exploratory association between a greater proportion of operative time with mean arterial pressure (MAP) below 65 mmHg and an increased likelihood of ICU admission. However, the association with in-hospital mortality was inconclusive, suggesting that the observed hemodynamic relationship may not consistently apply to different clinical outcomes.

Interpretation of these findings requires caution, as several methodological and clinical factors may influence the observed associations. These factors include differences between arterial and noninvasive blood pressure measurements, intermittent cuff sampling, missing physiological recording intervals, institutional ICU admission practices, and residual confounding. Although coverage-restricted and MAP-source-stratified sensitivity analyses showed directionally consistent associations with ICU admission, substantial selection bias and confounding by indication may persist. Specifically, patients with greater preoperative vulnerability or more complex surgical courses may be more likely to experience hypotension and require postoperative intensive care, complicating efforts to establish an independent causal relationship.

Externally published findings regarding troponin-defined myocardial injury after noncardiac surgery (MINS) provide complementary clinical context and emphasize the importance of perioperative cardiovascular monitoring. However, these findings differ from the present VitalDB analysis, in which postoperative troponin-defined myocardial injury was not independently established as an outcome. Therefore, the observed relationships between intraoperative hypotension and ICU

admission should not be interpreted as direct evidence of myocardial injury or as confirmation that hypotension causes adverse cardiac events.

Future research should prioritize external validation of the physiological exposure algorithm, standardized selection of blood pressure sources, improved management of missing monitoring intervals, and prospective measurement of postoperative cardiac biomarkers. Larger multicenter studies that include serial troponin measurements, comprehensive cardiovascular risk profiles, and standardized postoperative outcome definitions are needed to determine whether the duration and burden of intraoperative hypotension independently predict myocardial injury and other clinically significant complications. Overall, the current findings indicate that intraoperative hemodynamic instability may inform postoperative risk assessment following major abdominal surgery. However, its independent prognostic value, clinical applicability, and potential role in guiding preventive interventions require further investigation before drawing definitive conclusions.

REFERENCES

[1] Ruetzler K, Smilowitz NR, Berger JS, et al. Diagnosis and management of patients with myocardial injury after noncardiac surgery: a scientific statement from the American Heart Association. Circulation. 2021;144:e287-e305. doi:10.1161/CIR.0000000000001024.

[2] Devereaux PJ, Szczeklik W. Myocardial injury after non-cardiac surgery: diagnosis and management. Eur Heart J. 2020;41:3083-3091.

[3] Devereaux PJ, Biccard BM, Sigamani A, et al. Association of postoperative high-sensitivity troponin levels with myocardial injury and 30-day mortality among patients undergoing noncardiac surgery. JAMA. 2017;317:1642-1651.

[4] Botto F, Alonso-Coello P, Chan MTV, et al. Myocardial injury after noncardiac surgery: a large international prospective cohort study establishing diagnostic criteria, characteristics, predictors, and 30-day outcomes. Anesthesiology. 2014;120:564-578.

[5] Thompson A, Fleischmann KE, Smilowitz NR, et al. 2024 AHA/ACC multisociety guideline for perioperative cardiovascular management for noncardiac surgery. Circulation. 2024;150:e351-e442. doi:10.1161/CIR.0000000000001285.

[6] Halvorsen S, Mehilli J, Cassese S, et al. 2022 ESC Guidelines on cardiovascular assessment and management of patients undergoing non-cardiac surgery. Eur Heart J. 2022;43:3826-3924.

[7] Walsh M, Devereaux PJ, Garg AX, et al. Relationship between intraoperative mean arterial pressure and clinical outcomes after noncardiac surgery. Anesthesiology. 2013;119:507-515.

[8] Salmasi V, Maheshwari K, Yang D, et al. Relationship between intraoperative hypotension, defined by either reduction from baseline or absolute thresholds, and acute kidney and myocardial injury after noncardiac surgery. Anesthesiology. 2017;126:47-65.

[9] Wesselink EM, Wagemakers SH, van Waes JAR, Wanderer JP, van Klei WA, Kappen TH. Associations between intraoperative hypotension, duration of surgery and postoperative myocardial injury after noncardiac surgery. Br J Anaesth. 2022;129:487-496. doi:10.1016/j.bja.2022.06.034.

[10] Wesselink EM, Kappen TH, Torn HM, Slooter AJC, van Klei WA. Intraoperative hypotension and the risk of postoperative adverse outcomes: a systematic review. Br J Anaesth. 2018;121:706-721.

[11] Sessler DI, Bloomstone JA, Aronson S, et al. Perioperative Quality Initiative consensus statement on intraoperative blood pressure, risk and outcomes for elective surgery. Br J Anaesth. 2019;122:563-574.

[12] Saugel B, Sessler DI. Perioperative blood pressure management. Anesthesiology. 2021;134:250-261.

[13] Futier E, Lefrant JY, Guinot PG, et al. Effect of individualized vs standard blood pressure management strategies on postoperative organ dysfunction among high-risk patients undergoing major surgery. JAMA. 2017;318:1346-1357.

[14] Marcucci M, Painter TW, Conen D, et al. Hypotension-avoidance versus hypertension-avoidance strategies in noncardiac surgery: an international randomized controlled trial. Ann Intern Med. 2023;176:605-614.

[15] Yoshikawa Y, Maeda M, Kunigo T, et al. Effect of using hypotension prediction index versus conventional goal-directed hemodynamic management to reduce intraoperative hypotension in non-cardiac surgery: a randomized controlled trial. J Clin Anesth. 2024;93:111348. doi:10.1016/j.jclinane.2023.111348.

[16] Saugel B, Meidert AS, Brunkhorst FM, et al. Individualized Perioperative Blood Pressure Management in Patients Undergoing Major Abdominal Surgery: The IMPROVE-multi Randomized Clinical Trial. JAMA. 2025;334(21):1893-1904. doi:10.1001/jama.2025.17235.
[17] Kant M, van Klei WA, Hollmann MW, et al. Proactive vs Reactive Treatment of Hypotension During Surgery: The PRETREAT Randomized Clinical Trial. JAMA. 2025;334(21):1905-1914. doi:10.1001/jama.2025.18007.
[18] Li K, Hu Z, Li W, Shah K, Sessler D. Tight perioperative blood pressure management to reduce complications: a randomized feasibility trial. BMJ Open. 2023;13(11):e071328. doi:10.1136/bmjopen-2022-071328.
[19] Lee HC, Park Y, Yoon SB, et al. VitalDB, a high-fidelity multi-parameter vital signs database in surgical patients. Sci Data. 2022;9:279. doi:10.1038/s41597-022-01411-5.
[20] Cai J, Tang M, Wu H, et al. Association of intraoperative hypotension and severe postoperative complications during non-cardiac surgery in adult patients: a systematic review and meta-analysis. Heliyon. 2023;9(5):e15997. doi:10.1016/j.heliyon.2023.e15997.
[21] Chew MS, Saugel B, Lurati-Buse G. Perioperative troponin surveillance in major noncardiac surgery: a narrative review. Br J Anaesth. 2023;130:21-28. doi:10.1016/j.bja.2022.08.041.
[22] Thygesen K, Alpert JS, Jaffe AS, et al. Fourth universal definition of myocardial infarction (2018). Eur Heart J. 2019;40:237-269.
[23] Sessler DI, Meyhoff CS, Zimmerman NM, et al. Period-dependent associations between hypotension during and for four days after noncardiac surgery and a composite of myocardial infarction and death. Anesthesiology. 2018;128:317-327.
[24] Liem VGB, Hoeks SE, Mol KHJM, et al. Postoperative hypotension after noncardiac surgery and the association with myocardial injury. Anesthesiology. 2020;133(3):510–522. doi:10.1097/ALN.0000000000003368.
[25] van Waes JAR, van Klei WA, Wijeysundera DN, et al. Association between intraoperative hypotension and myocardial injury after vascular surgery. Anesthesiology. 2016;124:35-44. doi:10.1097/ALN.0000000000000922.
[26] VISION Study Investigators. Association between postoperative troponin levels and 30-day mortality among patients undergoing noncardiac surgery. JAMA. 2012;307:2295-2304.
[27] Devereaux PJ, Duceppe E, Guyatt G, et al. Dabigatran in patients with myocardial injury after non-cardiac surgery (MANAGE): an international, randomized, placebo-controlled trial. Lancet. 2018;391:2325-2334.
[28] Smilowitz NR, Redel-Traub G, Hausvater A, et al. Myocardial injury after noncardiac surgery: a systematic review and meta-analysis. Cardiol Rev. 2019;27:267-273.
[29] Puelacher C, Lurati Buse G, Seeberger D, et al. Perioperative myocardial injury after noncardiac surgery: incidence, mortality, and characterization. Circulation. 2018;137:1221-1232.
[30] Abbott TEF, Pearse RM, Archbold RA, et al. A prospective international multicentre cohort study of intraoperative heart rate and systolic blood pressure and myocardial injury after noncardiac surgery. Br J Anaesth. 2018;120:935-945.
[31] Mascha EJ, Yang D, Weiss S, Sessler DI. Intraoperative mean arterial pressure variability and 30-day mortality in patients having noncardiac surgery. Anesthesiology. 2015;123:79-91.
[32] Maheshwari K, Turan A, Mao G, et al. The association of hypotension during non-cardiac surgery, before and after skin incision, with postoperative acute kidney injury. Anaesthesia. 2018;73:1223-1228.
[33] Monk TG, Bronsert MR, Henderson WG, et al. Association between intraoperative hypotension and hypertension and 30-day postoperative mortality in noncardiac surgery. Anesthesiology. 2015;123:307-319.
[34] Sun LY, Wijeysundera DN, Tait GA, Beattie WS. Association of intraoperative hypotension with acute kidney injury after elective noncardiac surgery. Anesthesiology. 2015;123:515-523.
[35] Khanna AK, Maheshwari K, Mao G, et al. Association between mean arterial pressure and acute kidney injury and a composite of myocardial injury and mortality in postoperative critically ill patients. Crit Care Med. 2019;47:910-917.
[36] Bello C, Rössler J, Shehata P, Smilowitz NR, Ruetzler K. Perioperative strategies to reduce risk of myocardial injury after non-cardiac surgery (MINS): a narrative review. J Clin Anesth. 2023;87:111106. doi:10.1016/j.jclinane.2023.111106.
[37] Spahn DR, Muñoz M, Klein AA, Levy JH, Zacharowski K. Patient blood management: effectiveness and future potential. Anesthesiology. 2020;133:212-222.
[38] Muñoz M, Acheson AG, Auerbach M, et al. International consensus statement on the peri-operative management of anemia and iron deficiency. Anaesthesia. 2017;72:233-247.
[39] Ji H, Xia R, Tong X, Lv S, Wang W, Cheng M, et al. Association between intraoperative hypotension and myocardial injury after major open abdominal surgery. Front Med (Lausanne). 2026;13:1806976. doi:10.3389/fmed.2026.1806976.