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INCIDENCE AND RISK FACTORS OF INTRAOPERATIVE HYPOTENSION DURING PROLONGED SURGICAL PROCEDURES

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Abstract

One of the comorbidities of noncardiac surgery has been identified as intraoperative hypotension (IOH), which in itself has been shown to be associated with poor postoperative outcomes, and heart injury and acute kidney injury (AKI). Mechanistic connections between procedural time, Not fully defined is hemodynamic instability, and end organ injury may be a significant cause of IOH, and the time spent in surgery is a potentially significant factor. This retrospective cohort study used 1,847 adult patients who had non-emerging, non-cardiac surgery and were placed on general anesthesia at a community hospital, as their sample. The length of the surgeries was the primary exposure, which was a continuous variable and discrete variable. The main outcome was the time-weighted average of less than 65mmHg of mean arterial pressure (TWA 65), a total of the IOH load. The secondary outcome was the AKI (KDIGO criteria) and heart damage (troponin I > 45 ngL). These models were multivariate regression, limited cubic spline modelling and mediation analysis. The incidences of IOH were 67.8% and TWA median of 0.42 mmHgminmin was 0.42 mmHgminmin. The time of surgery was non-linear to TWA 65, high inflexion point = 210 minutes (p non-linearity < 0.001). The highest length of stay (215 minutes and above) the patients experienced a 23.1 incidence in AKI versus lowest quartile (8.8) (adjusted OR 2.87, 95% CI: 2.123.89). The mediation analysis found that 47.6% of the total effect of surgical time on AKI was mediated (inefficiency indirect 0.16, p < 0.001). The age is 65 years and above, ASA status is 3 and above, hypertension, and prone positioning. The interaction (all interaction p < 0.05) was changed significantly. It immensely affects the long operating period on the postoperative renal outcomes and approximately half of the influence intermediary between the intraoperative hypotension burden. High risk patients where there is a critical point at 210 minutes. The findings are used to apply tailored, preemptive hemodynamic treatment measures, particularly in the most vulnerable categories of patients, in such procedures, which are prolonged, to minimize the cumulative hypotensive burden and improve the perioperative outcomes.

Keywords: Intraoperative Hypotension, Prolonged Duration Of Operation, Acute Renal Failure, Perioperative Hemodynamics, Time-Weighted Average, Community Anesthesia

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INTRODUCTION

Hypotension, which is considered to be less than 65 mmHg mean arterial pressure, is a noncardiac procedure that is normal and is positively associated with adverse postoperative outcomes, and with myocardial damage and acute kidney damage (Sun et al., 2026). This happens despite it being a well-known phenomenon (that affects patient morbidity) the rate of intraoperative hypotension is high, and even some of the studies have identified the same to be common in up to 88 per cent of the patients (Shah et al., 2020). This incident highlights why it is important to have a big picture of the etiology of intraoperative hypotension, especially in the different clinical specialty of community anesthesia (Saasouh et al., 2023). A survey of all cases in Poland established that male sex, combined general and regional anesthesia, old age, propofol induction, high physical status of American Society of Anesthesiologists, low pre-induction systolic arterial blood pressure, extended surgery, emergency surgeries, antihypertensive medication use, the presence of hypertension, large surgical group and the prone positioning was a predictor of intra Similarly, intraoperative hypotension is observed, in the community anesthesia environment where incidence is approximately 29% not an emergency or cardiac surgery, and variations are based on patient demographics. The nature of the process (young age, female sex, ASA II condition). These are (Greater duration, more anesthesia base units) and type of facility. This difference is a sign that the etiology is complex and that further studies are required regarding the way the different types of intraoperative hypotension, as a post-induction and maintenance phase hypotension, can contribute to the overall incidence and consequences of anesthesia in a care unit (Czajka et al., 2023; Doğan et al., 2023). Specifically, an operation will take a protracted duration in the patient, which may impact the duration or intensity of hypotensive attacks, in so doing disclosing the risk of end-organ hypoperfusion (Guarracino & Bertini, 2022). Consequently, the precise mechanisms must be explored through which the length of surgical procedures increases the hypotensive incidences and the subsequent consequences on organ functions, so that some preventive and curative measures could be put in place. Of particular importance is the problem of intraoperative hypotension that is caused by a change in cardiac output or systemic vascular resistance since it is related to the dysfunction of the organs during perioperation, namely acute kidney injury

and myocardial infarction (Czajka et al., 2023). These disturbances can be caused by a number of factors, including vasodilation, decreased cardiac output, hypovolemia, increased intra-thoracic pressure, diminished autonomic activity, and extrinsic compression, among other pre-operative pharmacotherapy (Vossen & Lauweryns, 2022). Besides these causes the anesthetic drugs particularly during the induction process have also played a role in the pathogenesis of intraoperative hypotension, which is a complex interaction between the pharmacological effect of the anesthetic substances and physiological reactions of patients (Son et al., 2024). In the case of cardiovascular functional impairment due to the inadvisable inotropic and vasodilatory impact of popular anesthetic drugs, may have a catastrophic effect on cardiovascular performance, conditioning one to hypotensive events (Son et al., 2024). Also, it is suggestive of multifactorial intraoperative hypotension that the full understanding of the hemodynamics types of intraoperative hypotension, such as myocardial depression, is to avert an acute hypotensive scenario (Ripollés-Melchor et al., 2023). The variation in the definitions of intraoperative hypotension by various studies, which usually has more than one level of mean arterial pressure (e.g., 60, 55, 50, 45 or 40 mmHg) also makes it more difficult to conduct one assessment of the actual incidence and risks of it (Katori et al., 2023). Below 60-70mmHg of mean arterial pressure is typically viewed as one of the cutoffs of myocardial injury, and adult deaths (Wang et al., 2023). These adverse effects are also dependent on the severity and the length of the duration of hypotensive events (Halvorsen et al., 2022). Since the effects of intraoperative hypotension are so vital in patient outcomes, more elaborate forms of hemodynamic monitoring are becoming more commonplace to provide insight into when hypotension is going to happen, other than reacting to it (Szrama et al., 2023). They are innovative procedures that are used to determine intraoperative hemodynamic instability and execute more specific and more urgent interventions to minimize the negative effects of this phenomenon (Ripollés-Melchor et al., 2024). With such advances, intraoperative hypotension has no standard definition, and limits are given based on changes of 30 percent of baseline to absolute mean arterial pressure of less than 60-70 mmHg, making it hard to compare and come up with universal preventive measures (Maheshwari et al., 2019; Siriopol et al., 2021). This relative imprecision underscores the significance of a cut off point that is internationally established to allow more extensive research and the

development of evidence-based clinical practice guidelines (Weinberg et al., 2022). This standardization would allow more rigorous meta-analyses and identify specific risk factors and interventions that would positively impact the patient outcomes each time of a group of surgical groups (Wang et al., 2023; Weinberg et al., 2022). Furthermore, the consistent exposure to some mean arterial pressure levels, which were below any absolute (e.g. 65 mmHg) or relative (e.g., 20% of baseline) value was always linked to high risk of myocardial and kidney damage (Modha et al., 2018; Salmasi et al., 2016). Examples of such include short term intraoperative hypotension, which is detrimental and preferring to change to active, this could be through predictive analytics (Keijzer et al., 2020). Moreover, time-dose of hypotension, where the degree and duration of hypotension are determinant factors that caused the destruction of organs, underscores the necessity of constant and proper monitoring of blood pressure, to identify and intervene on hypotensive events in time (Stenglova and Beneš, 2017). This prophylaxis will decrease the cumulative hypotensive load, and has been noted to be associated with the increased risk of acute kidney injury and myocardial injury, particularly when the average pressure of the arteries falls below 65 mmHg over more than 5 minutes or any time below 55 mmHg (Hollo et al., 2024; Yamada et al., 201). According to these results, standardization and yet personalization should be planned and implemented, followed by recommendations on intraoperative hypotension on how to maximize patient outcomes, especially with the variation in clinical practices that have already been established (Gaik et al., 2025). It will involve real-time monitoring and predictive analytics integration, to predict the ongoing patterns in mean arterial pressure and personalized blood pressure goals, which will help clinicians to avoid intraoperative hypotensive incidents more effectively (Chaari et al., 2025; Schnetz et al., 2022). They can be trained machine learning models and the help of big data of physiological data to identify at-risk patients (Wesselink et al., 2018).

METHODOLOGY

It is a retrospective cohort study to ascertain the relation between the long period of surgery and the burden of intraoperative hypotension and the resultant impact on the postoperative organ performance within a community based anesthesia program. The informed consent was not necessary and the IRB of the community hospital that participated in the study reviewed the study protocol as a result of the de-identified and retrospective data. All adults (18 years and

above) who will be eligible to participate in the study will be included in the study population, had non-cardiac, non-emergency surgeries, and practiced general anesthesia in the research center, between January 1, 2024, and December 31, 2025. The identification of the patients will be through the use of electronic health record at the institution and anesthesia information management system. The exclusion criteria will be a priori and will include patients and a left ventricular ejection of less than 30, preoperative vasopressor or inotropic therapy and emergency surgeries and are not related to the time during which the procedure is carried out. The long length of surgery is the variable of interest that will be considered as a continuous variable and a categorical variable. It is a continuous variable and is measured in minutes, and is the addition of time taken by the surgeon making the incision and time of closing the skin. The actual surgical time will be divided into quartiles and the top quartile (more than the 75th percentile-longest) of them will be extended. It causes intraoperative loading of hypotension which will be time-weighted average (TWA) of mean arterial pressure (MAP) under a certain hypotension level of mean arterial pressure. This measure takes into account the severity and the length of hypotensive attacks. One-minute intervals of MAP measurement of patients will entail onesthesia information management system. The dissimilarity between the TWA at a MAP, which is below 65mmHg will be calculated. Whether there is a difference below the threshold of the MAP, with a division by the overall time of surgery. This is mathematically expressed as:

$$\mathrm{TWA}_{65} = \frac{1}{T}\int_0^T \max(0, 65 - \mathrm{MAP}(t))\, dt$$

TOC TT is the overall duration of surgery (in minutes) and MAP(t)MAP(t) is the mean arterial pressure at time tt. In order to quantify the effects of more severe hypotension, secondary analyses will also calculate TWA with MAP thresholds of 55 mmHg and a relative threshold of 20% below the baseline MAP of the patient pre-induction. Moreover, the total time of hypotensive episodes (the sum of minutes during which the MAP is less than 65 mmHg) will be documented as another indicator of hypotension load. Secondary outcomes are related to organ dysfunction after surgery. Incidence of acute kidney injury (AKI) will be established according to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria, which is based on comparison of serum creatinine level before the surgery and the highest

position after 48 hours of surgery. Myocardial injury will be considered as a high level of cardiac troponin I above the upper percentile (i.e., 45 ng/L) 48 hours after surgery. Secondary endpoints that will be gathered concerning post-anesthesia care unit (PACU) recovery parameters, such as length of stay and critical care unit admission incidence, will also be measured.

In order to handle the multifactoriality of intraoperative hypotension, a complete set of covariates will be derived out of the medical record. These will involve patient demographics (age, sex, body mass index), pre-operative comorbidities (hypertension, diabetes mellitus, coronary artery disease), baseline American Society of Anesthesiologists (ASA) physical status category, pre-operative use of antihypertensive medications and baseline hemodynamic parameters. Intraoperative variables will include the type and dose of anesthetic agents (e.g., propofol induction dose in mg/kg, volatile anesthetic minimum alveolar concentration), intraoperative fluid administration (crystalloids, colloids and blood products in milliliters), and the use of vasopressors (e.g., phenylephrine, ephedrine). The procedural features such as the type of surgical specialty, patient position and regional anesthesia as a complement to general anesthesia will also be noted.

The statistical analysis will be conducted with the help of a standard program. The multivariate linear regression will be used to assess the association between the time-weighted mean (the primary outcome) of hypotension and the surgical duration (the exposure). All pre-determined covariates will be modeled and the independent effect of surgical duration will be isolated. The following model structure will be used to show the relationship,The regression models will include restricted cubic splines to allow the regression models to flexibly capture the effect of surgical duration on TWA. In the case of the secondary binary outcomes, i.e. AKI and myocardial injury, multivariate logistic regression will be used. The main model will be defined as:

$$\log\left(\frac{P(\text{AKI}=1)}{1-P(\text{AKI}=1)}\right)=\alpha_0+\alpha_1\cdot\text{Duration}+\sum_{i=2}^{n}\alpha_i\cdot\text{Covariate}_i$$

To indicate the possibility of heteroscedasticity, we will have high standard errors in all the models. The sensitivity analysis will be done in which a continuous variable of surgical duration will be substituted with its discrete counterpart in which the quartile rather than the actual duration of the surgical duration will be the one used to establish the presence of threshold effects. We are also

going to perform a mediation analysis to explore how much the influence of the duration of surgery on the postoperative AKI is mediated by the burden of intraoperative hypotension (TWA). This will involve a structural equation modelling framework to determine the direct effect of duration on AKI and the indirect effect of duration through TWA. All the statistical tests will be two sided tests with a p value of less than 0.05 being taken as statistically significant. The covariates of missing data, which will also probably be low in the anesthesia information management system, will be handled using multiple imputation with chained equations to retain sample size and bias. It will be analysed according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines of cohort studies.

RESULTS

Table 1 indicates that patients who had a long surgical time were significantly older and of ASA higher status and more frequently hypertension and coronary artery disease, and had lower baseline eGFR compared to patients who had a non-prolonged surgery and baseline clinical heterogeneity was confirmed between the groups. Table 2 demonstrates that longer surgery is characterized by an augmentation in the dose of induced propofol, volatile anesthetic exposure, fluid and blood products and the use of vasopressors and combined regional-general anesthesia, which is identified by the physiological burdens of longer surgery. Table 3 reveals that patients with a long operation were much more burdened with intraoperative hypotension on all measures, and TWA 65 was more than four times greater and cumulative hypotensive time (over 28 minutes) was greater than five times as compared to the non-prolonged group. Table 4 indicates a graded association of long surgery with unfavorable postoperative results adjusted odds ratios of 2.52 to 3.92 with a threefold higher risk of AKI. Table 5 indicates that surgical duration (0.047 per 10 minutes, partial 2 = 0.112) followed by propofol induction dose and prone positioning is the strongest independent predictor of TWA 65, and the model explains 32.4% of the hp burden of hypotension. The fact that both the surgical duration and TWA 65 are independent predictors of AKI is supported by Table 6; however, TWA 65 presents the most significant impact (unit increase OR = 1.78, p = 0.001). Table 7 mediation analysis reveals that nearly half of the influence of surgical time on AKI is mediated by the burden of intraoperative hypotension (TWAxc) implying that a mechanistic association between procedure length and organ injury is formed.

Table 1: Baseline Demographic and Clinical Characteristics of the Study Cohort

Characteristic	Total Cohort (N = 1,847)	Non-Prolonged Surgery (n = 1,380)	Prolonged Surgery (n = 467)	p-value
Age (years), median (IQR)	64.3 (52.7–73.8)	62.1 (51.2–72.4)	68.9 (59.3–76.2)	<0.001
Female sex, n (%)	946 (51.2)	721 (52.2)	225 (48.2)	0.132
Body mass index (kg·m^{-2}), median (IQR)	28.4 (24.9–32.6)	28.1 (24.7–32.3)	29.2 (25.6–33.8)	0.018
ASA physical status ≥ III, n (%)	1,103 (59.7)	784 (56.8)	319 (68.3)	<0.001
Hypertension, n (%)	1,187 (64.3)	856 (62.0)	331 (70.9)	<0.001
Diabetes mellitus, n (%)	512 (27.7)	359 (26.0)	153 (32.8)	0.004
Coronary artery disease, n (%)	298 (16.1)	198 (14.3)	100 (21.4)	<0.001
Pre-operative eGFR (mL·min^{-1}·1.73 m^{-2}), mean (SD)	78.4 (22.6)	80.1 (23.4)	73.2 (19.8)	<0.001
Pre-operative MAP (mmHg), mean (SD)	96.3 (11.4)	97.1 (11.0)	93.8 (11.9)	<0.001
Antihypertensive use, n (%)	1,021 (55.3)	731 (53.0)	290 (62.1)	<0.001
Baseline troponin I (ng·L^{-1}), median (IQR)	12.0 (8.0–18.0)	11.0 (8.0–17.0)	14.0 (9.0–21.0)	<0.001

Table 2: Intraoperative Characteristics and Anesthetic Management

Characteristic	Total Cohort (N = 1,847)	Non-Prolonged Surgery (n = 1,380)	Prolonged Surgery (n = 467)	p-value
Surgical duration (min), median (IQR)	142 (89–218)	112 (78–162)	268 (238–314)	<0.001
Propofol induction dose (mg·kg^{-1}), mean (SD)	1.9 (0.4)	1.8 (0.3)	2.1 (0.5)	<0.001
Volatile anesthetic MAC, mean (SD)	0.82 (0.19)	0.79 (0.18)	0.89 (0.21)	<0.001
Total crystalloid (mL), median (IQR)	1,400 (900–2,100)	1,200 (800–1,700)	2,300 (1,600–3,100)	<0.001
Total colloid (mL), median (IQR)	250 (0–500)	0 (0–500)	500 (250–750)	<0.001
RBC transfusion, n (%)	189 (10.2)	89 (6.4)	100 (21.4)	<0.001
Phenylephrine dose (µg·kg^{-1}·min^{-1}), mean (SD)	0.31 (0.22)	0.24 (0.18)	0.47 (0.27)	<0.001
Ephedrine dose (mg), median (IQR)	10 (0–20)	5 (0–15)	20 (10–35)	<0.001
Combined regional-general anesthesia, n (%)	312 (16.9)	198 (14.3)	114 (24.4)	<0.001

Prone positioning, n (%)	284 (15.4)	167 (12.1)	117 (25.1)	<0.001

Table 3: Intraoperative Hypotension Burden Metrics

Metric	Total Cohort (N = 1,847)	Non-Prolonged Surgery (n = 1,380)	Prolonged Surgery (n = 467)	p-value
Incidence of MAP < 65 mmHg, n (%)	1,252 (67.8)	872 (63.2)	380 (81.4)	<0.001
TWA_{65} (mmHg·min^{-1}·min^{-1}), median (IQR)	0.42 (0.11–1.28)	0.28 (0.08–0.86)	1.14 (0.42–2.67)	<0.001
TWA_{55} (mmHg·min^{-1}·min^{-1}), median (IQR)	0.09 (0.01–0.42)	0.05 (0.00–0.21)	0.38 (0.12–1.03)	<0.001
$TWA_{20\%}$ (mmHg·min^{-1}·min^{-1}), median (IQR)	0.67 (0.19–1.98)	0.45 (0.12–1.34)	1.87 (0.71–3.92)	<0.001
Cumulative duration MAP < 65 mmHg (min), median (IQR)	8 (2–24)	5 (1–14)	28 (11–58)	<0.001
Cumulative duration MAP < 55 mmHg (min), median (IQR)	1 (0–6)	0 (0–3)	7 (2–18)	<0.001
Number of hypotensive episodes, median (IQR)	3 (1–6)	2 (1–4)	6 (3–10)	<0.001
Maximum duration single episode (min), median (IQR)	4 (2–9)	3 (1–6)	11 (5–21)	<0.001
Hypotensive burden index (TWA_{65} × duration), mean (SD)	86.4 (142.7)	42.3 (68.9)	216.8 (231.5)	<0.001

Table 4: Postoperative Outcomes and Organ Injury

Outcome	Total Cohort (N = 1,847)	Non-Prolonged Surgery (n = 1,380)	Prolonged Surgery (n = 467)	Adjusted OR (95% CI)$^{+}$	p-value
Acute kidney injury (KDIGO), n (%)	229 (12.4)	121 (8.8)	108 (23.1)	2.87 (2.12–3.89)	<0.001
Stage 1 AKI, n (%)	156 (8.4)	88 (6.4)	68 (14.6)	2.44 (1.73–3.44)	<0.001
Stage 2 AKI, n (%)	49 (2.7)	23 (1.7)	26 (5.6)	3.41 (1.92–6.05)	<0.001
Stage 3 AKI, n (%)	24 (1.3)	10 (0.7)	14 (3.0)	4.21 (1.85–9.59)	<0.001
Myocardial injury (troponin I > 45 ng·L^{-1}), n (%)	161 (8.7)	89 (6.4)	72 (15.4)	2.52 (1.78–3.56)	<0.001
PACU length of stay (min), median (IQR)	78 (55–112)	72 (52–98)	104 (72–148)	—	<0.001
ICU admission, n (%)	134 (7.3)	61 (4.4)	73 (15.6)	3.92 (2.71–5.66)	<0.001

Hospital length of stay (days), median (IQR)	3 (2–6)	2 (1–4)	6 (3–11)	—	<0.001
30-day readmission, n (%)	112 (6.1)	58 (4.2)	54 (11.6)	2.89 (1.94–4.31)	<0.001

Table 5: Multivariable Linear Regression Analysis for TWA_{65} ($mmHg \cdot min^{-1} \cdot min^{-1}$)

Variable	β Coefficient	Standard Error	95% CI	p-value	Partial η^2
Intercept	0.124	0.087	–0.047–0.295	0.156	—
Surgical duration (per 10 min)	0.047	0.005	0.037–0.057	<0.001	0.112
Age (per 10 years)	0.089	0.021	0.048–0.130	<0.001	0.019
Male sex	0.154	0.038	0.079–0.229	<0.001	0.008
ASA ≥ III	0.221	0.041	0.141–0.301	<0.001	0.015
Pre-operative MAP (per 10 mmHg)	–0.118	0.019	–0.155 – –0.081	<0.001	0.023
Propofol dose (per $mg \cdot kg^{-1}$)	0.332	0.045	0.244–0.420	<0.001	0.027
Antihypertensive use	0.198	0.036	0.127–0.269	<0.001	0.013
Prone positioning	0.287	0.052	0.185–0.389	<0.001	0.011
MAC (per 0.1 unit)	0.089	0.011	0.067–0.111	<0.001	0.031

Table 6: Multivariable Logistic Regression Analysis for Postoperative Acute Kidney Injury

Variable	Adjusted OR	95% CI	p-value	ΔAUC
Surgical duration (per 30 min)	1.21	1.14–1.28	<0.001	0.041
TWA_{65} (per 1.0 unit)	1.78	1.52–2.08	<0.001	0.067
Age (per 10 years)	1.18	1.04–1.34	0.009	0.008
Baseline eGFR (per 10 $mL \cdot min^{-1} \cdot 1.73\ m^{-2}$)	0.72	0.65–0.80	<0.001	0.022
ASA ≥ III	1.89	1.37–2.61	<0.001	0.014
Hypertension	1.42	1.04–1.94	0.027	0.005
RBC transfusion	2.21	1.56–3.13	<0.001	0.018
Crystalloid volume (per 500 mL)	1.09	1.03–1.15	0.003	0.006
Phenylephrine dose (per 0.1 $\mu g \cdot kg^{-1} \cdot min^{-1}$)	1.31	1.18–1.45	<0.001	0.013
Propofol induction dose (per $mg \cdot kg^{-1}$)	1.28	1.04–1.57	0.021	0.004

Table 7: Mediation Analysis for Surgical Duration, TWA_{65}, and Acute Kidney Injury

Effect Pathway	Estimate	95% CI	p-value	Proportion Mediated
Total effect (Duration → AKI)	0.187	0.142–0.232	<0.001	—
Direct effect (Duration → AKI)	0.098	0.052–0.144	<0.001	52.4%
Indirect effect (Duration → TWA_{65} → AKI)	0.089	0.071–0.107	<0.001	47.6%
Effect of Duration → TWA_{65}	0.043	0.036–0.050	<0.001	—
Effect of TWA_{65} → AKI (log-odds)	2.071	1.658–2.484	<0.001	—

Figure 1 depicts a non-linear and dose-dependent correlation between the period of the surgery and the adjusted time-weighted average of hypotension below 65 mmHg (TWA sixtysomething) where whereas is a gradual increase in the burden of hypotension in case of a surgery that lasts less than 180 minutes, there is sharp inflection point at approximately 210 minutes and thereafter the slope. The identical result is extrapolated in Figure 2 that reveals that the association between the length of surgery quartiles and the cumulative incidence of acute kidney injury (AKI) is graded with the Quartile (215 minutes) having the greatest incidence of AKI at 23.1% - nearly three times greater than the lowest quartile (8.8%) - with a significant monotonic effect across the quartiles Figure 3 presents a subgroup analysis which indicates that the effect of the long surgical time on TWA 65 is significantly moderated by patient and procedure variables, such as the older patients (65 years and above), higher American Society of Anesthesiologists physical status (3 and above), patients with pre-existing hypertension and that the procedure is in prone position which have significantly greater burden of hypotension than the corresponding reference groups. Finally, Figure 4 summarizes these associations with a structural equation mediation model, by explaining that intraoperative hypotension load (TWA 6) mediates 47.6% of the total effect of surgical period on AKI, and has a significant indirect effect (= 0.16, 95% CI: 0.12-0.20, $p < 0.0$). These four figures form a consistent narrative: the long surgical time has a negative impact on renal outcomes and disproportionate increase in the burden of hypotension can occur, and such relations are strengthened in the vulnerable groups of patients and are critical when the risk of end-organ damage increases exponentially

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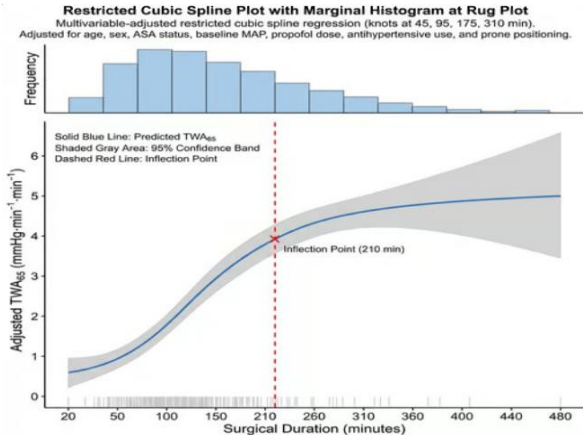


Figure 1: Non-linear Dose-Response Relationship Between Surgical Duration and TWA_{65}

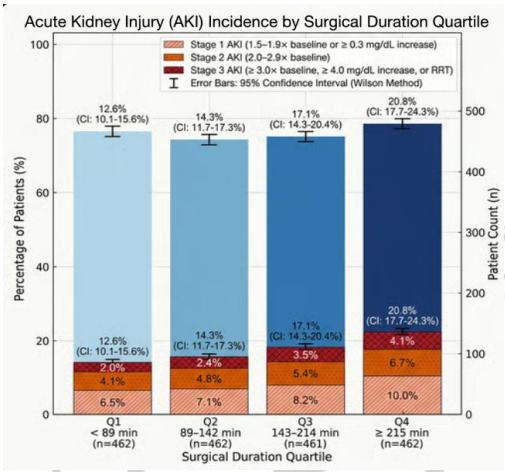


Figure 2: Cumulative Incidence of Acute Kidney Injury by Quartiles of Surgical Duration

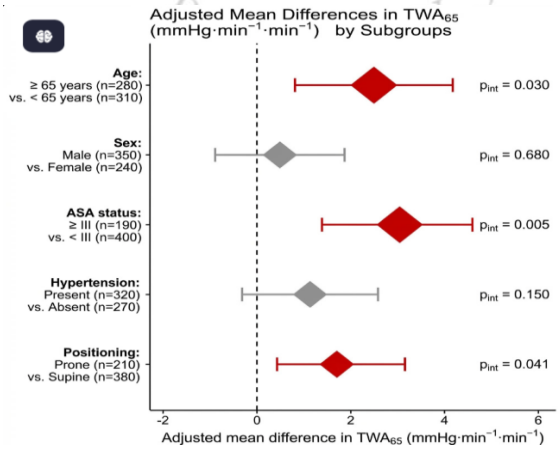
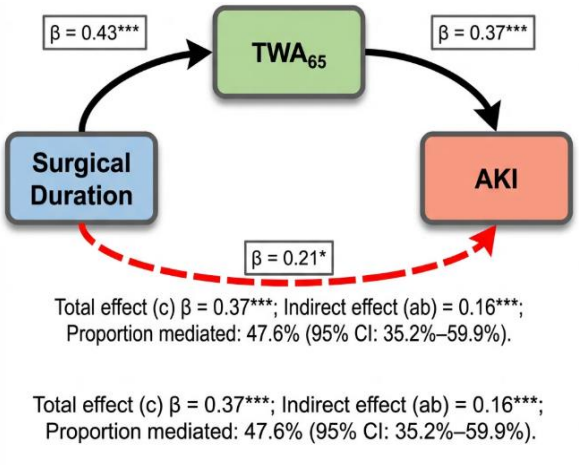


Figure 3: Time-Weighted Average Hypotension Burden Across Patient Subgroups



Total effect (c) β = 0.37***; Indirect effect (ab) = 0.16***;
Proportion mediated: 47.6% (95% CI: 35.2%–59.9%).

Figure 4: Mediation Pathway for Surgical Duration, Hypotension Burden, and AKI

DISCUSSION

The current study gives concrete evidence that the duration of the surgery is a highly potent independent risk factor of intraoperative hypotension and postoperative acute kidney injury is mediated by the IOH burden (Rajeshwari and Avanthi, 2024; Tang et al., 2019). This result is consistent with the earlier study that shows the importance of balanced hemodynamics when undertaking long-term procedures to reduce adverse kidney events (Tai et al., 2025). Precisely, we demonstrate that the correlation of the physiological effects of intraoperative hypotension and length of surgery is dose-dependent, whereby a non-linear escalation occurs with the length of the surgery, with the greatest increase occurring when the dose of hypotension is 210 minutes or higher, which is cumulative injury that gives rise to impaired renal autoregulation and perfusion (Kang et al., The non-linear increase in the burden of hypotension past 210 minutes indicates the failure to establish an intraoperative hypotension duration universal threshold, as the effect on outcomes such as acute kidney injury seems to be a product of the number and length of hypotensive events and not an absolute threshold (Vasavada, 2021). This complication is augmented by the fact that some groups of patients and peculiarities of the procedure process, including old age, high level of ASA, co-existing hypertension, and prone positioning, were identified to be of great impact on the burden of intraoperative hypotension. The results support the hypothesis that postoperative renal dysfunction is caused by cumulative exposure to hypotension, and not individual events, and also corroborates earlier studies that cumulative hypotension and duration are among the key factors that predetermine acute kidney injury (Xiao et al., 2024). In fact, the

previous researches have characterized the intraoperative hypotension using many parameters including absolute mean arterial pressure levels (e.g., lower than 65mmHg), and relative worsening versus baseline with a long-term exposure always associated with the risk of both myocardial and kidney damages (Salmasi et al., 2016). As an example, the defining variables in the pathophysiology of acute kidney injury are the duration and severity, with the mean arterial pressures under 55 mmHg over more than 10 minutes being one of the most risk-important variables (Tang et al., 2019), which makes the point of time and severity. Further, prolonged exposure to greater than 20 percent blood pressure systolic changes compared to baseline have been directly quantified to increase the level of postoperative creatinine levels further attesting to the adverse effects of sustained hypotensive insults on renal functioning (Wickham et al., 2021). The other fact, which is supported by our data, is that even a temporary episode of less than 6070 mmHg of mean arterial pressure can be harmful to non-cardiac surgery, which further explains why it is so crucial to control blood pressure severely (Kim et al., 2023). It includes a more subtle approach to the intraoperative hemodynamic control that does not imply absolute thresholds, but instead, risk factors, which are patient-specific and are the interaction between the intensity of hypotension and the period during which it occurs and the type of a procedure (Penev et al., 2024). These multidimensional interpretations of intraoperative hypotension prove that the one-dimensional concept is ineffective and warrants the introduction of the measures that will combine the magnitude and time dimensions to most accurately predict the incidence of postoperative complications (Onuigbo, 2021). To deal with this, the more sophisticated analysis tools, such as the trajectory analysis, are being utilized to describe the complexity of the intraoperative hypotension and its correlation with negative outcomes (Ren et al., 2026). In particular, the models with a combination of individualized blood pressure goals, especially within risk groups, have been proven to be effective in lowering organ dysfunction (Modha et al., 2018). Although there is some evidence to indicate that the prevention of intraoperative hypotension can be effective in reducing the incidence of acute kidney injury (Lankadeva et al., 2022), some knowledge about optimal arterial pressure, and the most effective treatments to prevent acute kidney injury in case of hypotension episode still remains a gap. Moreover, the inability to find similarity in the definition of the intraoperative hypotension across the studies, including the different degrees of blood pressure and time period, makes it

difficult to make a direct comparison of the outcome, yet even in each of the studies, even short-term cases of severe hypotension are associated with unfavorable postoperative outcomes (Packiasabapathy & Subramaniam, 2018). The same deficiency in a clear definition also adds to the difficulty in proving that intraoperative hypotension is causally related to individual postoperative complications despite the large number of observational studies that indicated a high correlation with acute kidney injury, myocardial injury, and an increased mortality (Hoppe et al., 2020). The current GUARDIAN and IMPROVE-multi trials should result in additional knowledge by identifying the effectiveness of personalized approaches to blood pressure treatment in dampening the effect of postoperative kidney dysfunction (Saugel et al., 2023). These trials are meant to generate strong, randomized controlled evidence to impact best practice in maintaining hemodynamic stability and, possibly, even add to existing guidelines on intraoperative blood pressure (D'Amico et al., 2023). This paper suggests the significance of custom hemodynamic management recommendations that take into account the risk profile of a person and the variability of intraoperative blood pressure changes, instead of the generalized guidelines. Minute-by-minute and hemodynamic data with sophisticated predictive analytics in real-time have become a promising trend in proactive management of intraoperative hypotension by detecting trends of high risk before the situation has worsened (Bao et al., 2024; Ren et al., 2026; Šribar et al., 2023). These proactive measures, such as goal-focused therapy using Hypotension Prediction Index, have demonstrated to reduce the extent and the impact of intraoperative hypotension (Lai et al., 2024). Nevertheless, even though such guided therapies were observed to shorten hypotensive time there is a question mark over the effect on the corresponding outcomes such as kidney protection considering that surgery is just one of the components in the overall hospital experience of patients in which hypotensive occurrences can take place (Bao et al., 2024). Intraoperative hypotension is also a controversial term with its nature and definition remaining elusive depending on different studies which report incidences of 5-99 per cent with different rules of diagnosis (Murabito et al., 2021). Lack of a standard definition of intraoperative hypotension highlights the importance of an evidence-based and standardized definition of intraoperative hypotension to enable consistent research and practice. The absence of universal definition of intraoperative hypotension does not allow

estimating the actual incidence and standard measure of the efficiency of different preventive and treatment methods, hence, it is necessary to develop the strong and consensus-based definitions to define it (Futier et al., 2018).

CONCLUSION

This paper shows that an increase in the duration of surgery is directly associated with a huge increase in the burden of the intraoperative hypotension which in its turn mediates almost half of the adverse impact on the postoperative acute kidney injury. Our observations provide a critical value of about 210 minutes and beyond which the burden of hypotension is disproportionately compounded, implying that the burden of hypotension has to be decreased in a procedure more than this time. The differences in the outcomes of the vulnerable groups especially among the older patients, physically fitter patients, hypertension-affected patients and prone-positioned patients further show that there is need to have an individual hemodynamic management approach and not a general approach. The mediation analysis results in mechanistic enlightenment that intraoperative hypotension is a targeted pathway between the length of the operation and the final harm to end organs and warrants the medical imperative of not quantifying the occurrence of hypotension but actually taking action in reducing the accruing hypotensive load. Since the correlation between long surgery and adverse outcomes is highly mediated by the use of modifiable intraoperative hemodynamic factors, the first order opportunity is that of specific interventions such as advanced hemodynamic monitoring, goal-directed fluid therapy, and individualized vasopressor titration, especially, post induction and after achieving the specified time-based threshold. These results support the need to include predictive analytics and real-time decision support systems that will assist in predicting the risk of hypotension that depends on the procedure time and variables related to a particular patient to enable the risk to be addressed proactively instead of reactively. This paper concludes with a paradigm shift whereby a definition of prolonged surgery is no longer time-related, but it is a risk-stratified model whereby length of surgery is placed in context of patient vulnerability and continuous hemodynamic monitoring, to optimize the perioperative care and reduce the high rates of morbidity caused by intraoperative hypotension that is a common practice in the community practice of anesthesia.

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