

High intraoperative pulse pressure is a risk factor for postoperative acute kidney injury in a cohort of abdominal surgery patients: An exploratory study

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Abstract

Background. Both intraoperative hypotension and hypertension have been reported to increase the occurrence of acute kidney injury (AKI). However, the impact of the intraoperative pulse pressure (PP) on the latter complications remains relatively unknown.

Objectives. To explore whether high intraoperative PP values are associated with postoperative AKI.

Materials and methods. The data for this study come from a prospective cohort study in which patients who underwent abdominal surgery between October 1, 2018 and July 15, 2019 in university hospital in Katowice, Poland were included in the analysis. Pre- and intraoperative data, including blood pressure measurements, were acquired from medical charts. Several PP thresholds were applied: >50, >55, >60, >65, >70, >75, >80, >85, and >90 mm Hg. Additionally, by analyzing the maximal PP during the procedures, the cutoff point for the occurrence of outcomes was estimated. Postoperative AKI was considered as the outcome of the study. Univariable and multivariable analyses were performed to assess PP relationship with AKI.

Results. Four hundred and ninety-four patients were included in the analysis. The AKI was present in 32 (6.5%) cases. The receiver operating characteristic (ROC) curve analysis estimated a cutoff point of >84 mm Hg of maximal PP to be associated with the outcome. The PP values above 80 mm Hg and onward were successfully included in the multivariable statistical models. A model in which PP > 90 mm Hg (odds ratio (OR) = 4.03; 95% confidence interval (95% CI): [1.53; 10.62]) was included, had the best predicting value in predicting hypoperfusion injury (area under the receiver operating characteristics (AUROC) = 0.88). Apart from PP, intraoperative hypotension, presence of chronic arterial hypertension, chronic kidney disease, and procedure duration were independently associated with AKI.

Conclusions. High intraoperative PP may be associated with the occurrence of postoperative AKI. However, the effect of high PP should be confirmed in other noncardiac populations to prove the generalizability of our results.

Key words: acute kidney injury, hemodynamic monitoring, general surgery, pulse pressure

Background

Hypoperfusion-related organ injury is a fairly frequent perioperative complication.^{1–4} Intraoperative hypotension (IOH) has been linked with postoperative myocardial injury (MI), acute kidney injury (AKI) and stroke.^{1–3} Perioperative Quality Initiative (POQI) consensus statement on intraoperative blood pressure underlines that mean arterial pressure (MAP) below 60–70 mm Hg and systolic blood pressure (SBP) below 100 mm Hg are associated with hypoperfusion-related organ injury and death.⁴ However, hypertensive events during surgery may also worsen the prognosis, as intraoperative episodes of SBP above 160 mm Hg have been correlated with the risk of myocardial injury and infarction.⁴ Lastly, diastolic blood pressure (DBP) below 50 mm Hg is also reported to be harmful.⁵

Although ambulatory pulse pressure (PP) is considered one of the best predictors of cardiovascular risk, it has been poorly investigated in the perioperative period.⁶ The association between high preoperative PP values and the relationship with postoperative complications (mainly myocardial infarction, AKI and stroke) has been explored mostly in cardiosurgical patient populations. The POQI has called for further research on the matter in noncardiac surgery.⁷

Objectives

In an exploratory fashion, we sought to verify whether elevated intraoperative PP values are associated with postoperative AKI in the abdominal surgery population.

Materials and methods

The data used in this study come from a prospective cohort study previously published by our team.⁸ We screened 576 consecutive patients who underwent abdominal surgery between October 1, 2018 and July 15, 2019,

in a University Hospital in Katowice, Poland. Procedures of organ procurement (n = 11), reoperations (n = 24), procedures performed in local anesthesia or monitored anesthesia supervision (n = 33), procedures classified as immediate according to the National Confidential Enquiry into Patient Outcome and Death (NCEPOD) Classification of Intervention⁹ (n = 14), and patients with proven cardiac valve defects (n = 14) were excluded from the study (Fig. 1). Demographic and medical data were recorded, including sex, age, weight, height, and comorbidities and their pharmacological treatment, according to the International Classification of Diseases (ICD-10) criteria.¹⁰ Body mass index (BMI) and Charlson comorbidity index (CCI) were subsequently calculated. Type and duration of anesthesia, as well as type, duration and urgency of surgery were recorded. Perioperative risk was assessed based on an individual patient's risk, according to the American Society of Anesthesiologists (ASA) physical status (PS) classification,¹¹ and procedural risk, according to the European Society of Cardiology and European Society of Anaesthesiology recommendations.¹² Primary arterial hypertension was diagnosed based on medical records.

The SBP and DBP were measured on a nondominant arm using an automated noninvasive oscillometric BP monitoring device (Dräger Infinity Gamma XL; Dräger, Lübeck, Germany) with a cuff of appropriate size, depending on a patient's arm circumference, and recorded in 5-minute intervals during anesthesia, from the first preinduction measurement until the last measurement during recovery from anesthesia in the operating theater. The MAP values were automatically calculated. Pulse pressure was calculated as the difference between SBP and DBP. The need for norepinephrine (NE) use and its doses, together with intraoperative fluid balance, were analyzed.

Taking into consideration other studies on clinical consequences of abnormal PP, and the fact that PP revolves usually around values of 40 mm Hg, we distinguished following absolute PP thresholds: >50, >55, >60, >65, >70, >75, >80, >85, and >90 mm Hg.^{5,13–15} Additionally, by analyzing

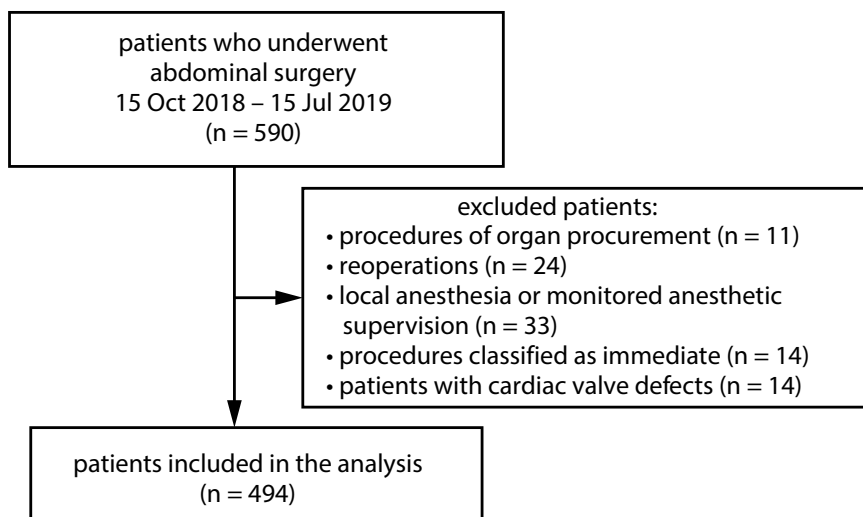


Fig. 1. Flow diagram for the patient selection process

the maximal PP during the procedure, the best cutoff point associated with the occurrence of AKI was estimated. We used maximal PP (presented as the median of all maximal PPs recorded among all the patients) and not an average or a median, due to a number of reasons. Firstly, the average value is much more confounded by extreme values of the distribution of numbers. Secondly, choosing maximal PP over average or median PP is better suited for finding a cutoff point for PP that is associated with postoperative AKI. In this study, we explored the role of high PP values; therefore, we naturally sought higher values and tried to find thresholds that would be easily identifiable by a clinician. In contrast, the average or the median value reflects rather a global trend in values and does not bring any specific information.

Moreover, we analyzed the occurrence of high systolic (defined as SBP > 160 mm Hg¹⁶), low diastolic (defined as DBP < 50 mm Hg¹⁷) and low mean arterial pressure (defined as MAP < 60 mm Hg¹⁸). We excluded pre-induction measurements in order to assess only those BP values that occurred during anesthesia.

In the postoperative period, the incidents of AKI were recorded and defined as a serum creatinine increase ≥ 0.3 mg/dL within 48 h or an increase in serum creatinine by ≥ 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days.¹⁹ This outcome was considered as the endpoint. In addition, incidents of AKI were classified as stages based on Kidney Disease Improving Global Outcomes (KDIGO) guidelines.²⁰

Strengthening the Reporting of OBServational studies in Epidemiology (STROBE) statement was applied for appropriate reporting.²¹

Statistical analysis was performed using MedCalc statistical software v. 18.1 (MedCalc Software Ltd., Ostend, Belgium). Continuous variables were expressed as median

and interquartile range (IQR). Qualitative variables were expressed as absolute values and/or percentages. Between-group differences for quantitative variables were assessed using the Mann–Whitney U test. Their distribution was verified with the Shapiro–Wilk test. The χ^2 tests were applied for qualitative variables. The correlation was assessed using Spearman's rank correlation coefficient. The receiver operating characteristic (ROC) curve analysis was implemented to assess the relationship between AKI and maximal PP. In order to control the potential confounding factors, we used multivariable logistic regression with all variables that achieved p-value < 0.1 in the univariable analysis. The Hosmer–Lemeshow test was performed to assess the goodness-of-fit of multivariable logistic regressions. If applicable, odds ratios (ORs) and area under the receiver operating characteristics (AUROC) with 95% confidence intervals (95% CIs) were calculated. All tests were two-tailed. A value of p < 0.05 was considered statistically significant.

Results

A total number of patients included in the analysis was 494, out of which 239 (46%) were male. The median age of participants was 65 years (IQR 46–68). Older age, higher ASA-PS class and higher CCI were found to be significant preoperative risk factors for the occurrence of AKI. Detailed preoperative population characteristics are presented in Table 1, whereas intraoperative population characteristics are presented in Table 2. The primary outcome (AKI) was diagnosed in 32 (6.7%) patients. According to KDIGO criteria, 24 patients (75%) suffered from stage 1 AKI, 5 patients (15.6%) from stage 2 AKI and 3 patients (9.4%) from stage 3 AKI.¹⁹ Pre-induction PP was not associated with the outcome (Table 1).

Table 1. Preoperative population characteristics

Variable	AKI (–) (n = 462)	AKI (+) (n = 32)	p-value	df/test-value
Age [years]	61 (44–68)	67 (62–73)	0.0002	U-value = 5010
Male (n)	213 (46.1)	17 (53.1)	0.4738	df = 1
BMI [kg/m ²]	25.6 (22.5–29.0)	27.4 (22.6–30.7)	0.2318	U-value = 6426
Arterial hypertension	197 (42.6)	26 (81.2)	<0.0001	df = 1
Chronic kidney disease (n)	8 (1.7)	5 (15.6)	<0.0001	df = 1
Pre-induction SBP [mm Hg]	140 (125–155)	142.5 (132.5–155)	0.1985	U-value = 6308
Pre-induction MAP [mm Hg]	101.7 (92–110)	101.5 (95–113)	0.5473	U-value = 6297
Pre-induction PP [mm Hg]	56 (48–66)	59 (50–75)	0.2595	U-value = 6439
ASA-PS I/II	279 (60.4)	10 (31.2)	0.0012	df = 1
ASA-PS III/IV/V	183 (39.6)	22 (68.7)	0.0012	df = 1
CCI [pts]	3 (1–5)	5 (3–7)	0.0001	U-value = 4434
Premedication	278 (60.2)	22 (62.5)	0.7949	df = 1

AKI – acute kidney injury; PP – pulse pressure; ASA-PS – American Society of Anesthesiologists physical class; BMI – body mass index; SBP – systolic blood pressure; MAP – mean arterial pressure; CCI – Charlson comorbidity index; df – degrees of freedom. Age, BMI, pre-induction SBP, pre-induction MAP, pre-induction PP, and CCI were analyzed using Mann–Whitney test, whereas sex, arterial hypertension, chronic kidney disease, ASA-PS, and premedication were tested with χ^2 test.

Table 2. Intraoperative population characteristics

Variable	AKI (–) (n = 462)	AKI (+) (n = 32)	p-value	df/test-value
General + epidural anesthesia (n)	23 (6.5)	9 (28.1)	<0.0001	df = 1
Procedure risk I (n)*	43 (9.3)	1 (3.1)	0.2356	df = 1
Procedure risk II (n)*	308 (66.7)	17 (53.1)	0.1188	df = 1
Procedure risk III (n)*	111 (24.0)	14 (43.7)	0.0132	df = 1
Oncological procedure (n)	216 (46.8)	22 (68.7)	0.0161	df = 1
Catecholamine use (n)	194 (42)	26 (81.2)	<0.0001	df = 1
Catecholamine dose [μ g/kg/min]	0.06 (0.042–0.091)	0.073 (0.061–0.108)	0.1166	U-value = 1636
Procedure duration [min]	220.0 (120.0–330.0)	392.5 (255.0–557.0)	<0.0001	U-value = 3297
Fluid dose [mL/kg/h]	6.79 (5.16–8.80)	6.64 (4.71–8.59)	0.5337	U-value = 6906
Mean arterial pressure [mm Hg]	83.33 (78.33–88.33)	85.17 (78.33–89.67)	0.5765	U-value = 6956
Minimal pulse pressure during anesthesia [mm Hg]	30 (25–35)	25 (20–32)	0.0419	U-value = 5816
Median pulse pressure during anesthesia [mm Hg]	45.0 (40–51.2)	50 (45–60)	0.0028	U-value = 5058
Maximal pulse pressure during anesthesia [mm Hg]	65 (56–75)	82.5 (66–93.5)	<0.0001	U-value = 4018
MAP < 60 mm Hg during anesthesia (n)	106 (22.9)	14 (43.7)	0.0080	df = 1
SBP > 160 mm Hg during anesthesia (n)	98 (21.2)	10 (31.2)	0.1844	df = 1
DBP < 50 mm Hg during anesthesia (n)	133 (28.8)	15 (46.9)	0.0309	df = 1

AKI – acute kidney injury; df – degrees of freedom; MAP – mean arterial pressure; SBP – systolic blood pressure; DBP – diastolic blood pressure; * according to European Society of Cardiology and European Society of Anaesthesiology recommendations.¹¹ General + epidural anesthesia, procedure risks, oncological procedures, catecholamine use, MAP < 60 mm Hg, SBP > 160 mm Hg, and DBP < 50 mm Hg were tested with χ^2 test, whereas catecholamine dose, procedure duration, fluid dose, MAP, and minimal, maximal and median pulse pressure during anesthesia were tested using Mann–Whitney test.

Table 3. Correlation between pulse pressure and systolic blood pressure, diastolic blood pressure and mean arterial pressure

Variable	Median pulse pressure AKI (–) (n = 462)	Median pulse pressure AKI (+) (n = 32)
Median systolic blood pressure	R = 0.649; p < 0.01	R = 0.604; p < 0.01
Median diastolic blood pressure	R = –0.214; p < 0.01	R = –0.623; p < 0.01
Pre-induction pulse pressure	R = 0.446; p < 0.01	R = 0.447; p < 0.01

AKI – acute kidney injury. The values are Spearman's rank correlation coefficients.

In patients who developed AKI, PP more negatively correlated with DBP than in patients without AKI (Table 3).

Maximal PP registered over the course of the procedure was associated with the outcome (AUROC = 0.728; p < 0.001), with a cutoff point >84 mm Hg (Fig. 2).

In univariable analyses, all PP thresholds, except for >50 mm Hg, were statistically significant predictors of AKI (Fig. 3). In multivariable logistic regressions, PP values >80, >84, >85, and >90 mm Hg were included in the final statistical models. It was discovered that PP > 90 mm Hg predicted AKI with the highest accuracy, even after the adjustment for various confounding factors, including intraoperative hypertension (Table 4). Low DBP (<50 mm Hg) and high SBP (>160 mm Hg) were not significant in the multivariable models.

Discussion

The main finding of our exploratory study is that increasing intraoperative values of PP were associated with the occurrence of postoperative AKI. This association persisted after adjusting for confounding factors (most importantly: high SBP and low DBP). We found a cutoff point of >84 mm Hg of maximal PP to be associated with AKI. In regards to the predetermined thresholds, PP above 80 mm Hg and onward was linked to AKI. Pulse pressure above 90 mm Hg, out of all PP thresholds applied, appeared to be the best predictor of postoperative AKI.

To our knowledge, this is the first study investigating the role of intraoperative PP in abdominal surgery in such a complex manner. It is known that increased ambulatory

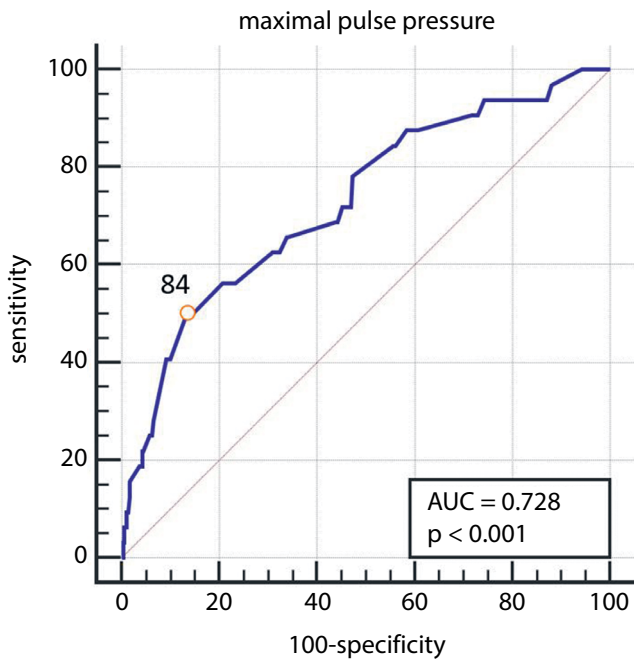


Fig. 2. The receiver operating characteristic (ROC) curve analysis of maximal pulse pressure (PP) values registered over the course of procedure

AUC – area under the curve.

PP is strongly associated with cardiovascular events, not only in the general population but also in cardiac surgery setting, irrespective of the presence of chronic arterial hypertension.^{6,22,23} Pulse pressure stands as a proxy for general vascular health and reflects cardiovascular risk better than isolated measurements of either SBP or DBP.²⁴ Generally, a value of PP is determined by stroke volume, left ventricle contractility and arterial compliance. Interestingly, pre-induction PP values (a reflection of baseline PP) alone were not significantly related to the outcome. In studies by Abbott

et al. and Mitrev et al., it was found that increasing values of ambulatory and pre-induction PP were significantly related to the increased occurrence of postoperative MI and AKI.^{5,25} It must be remembered, however, that those studies were performed among cardiac surgery patients with pre-existing cardiac morbidities, and the effect of preoperative PP might have been more significant than in the noncardiac setting. The fact that in our cohort pre-induction PP was not associated with AKI, gave us more space to explore the impact of intraoperative values. Nevertheless, intraoperative PP positively correlated with the pre-induction values. The negative correlation between PP and DBP was especially interesting, since it was 2 times stronger in patients with the compromised outcome. Lowered DBP is known to decrease coronary perfusion and could be associated with the development of hypoperfusion-induced organ injury.^{16,26,27} However, after taking into account low DBP (<50 mm Hg) in multivariate analyses, PP thresholds remained significant and low DBP was not included in the models.

We discovered that patients who experienced AKI exhibited higher values of PP, and the ORs varied, depending on the threshold applied. Contrary to our hypothesis, Ahuja et al., in a large cohort of 23,000 patients, found that PP below 35 mm Hg was linked to postoperative MI and AKI.¹⁷ Indeed, in our cohort, the AKI group experienced lower minimal PP compared to the non-AKI group (median 25 mm Hg compared to 30 mm Hg). Low PP is thought to predict cardiovascular events in patients with impaired cardiac function: decreased contractility of left ventricle causes SBP to achieve lower values and negatively impact the value of PP. It must be remembered that Ahuja et al. explored only the lowest values of PP and called for further research regarding high intraoperative PP.¹⁷

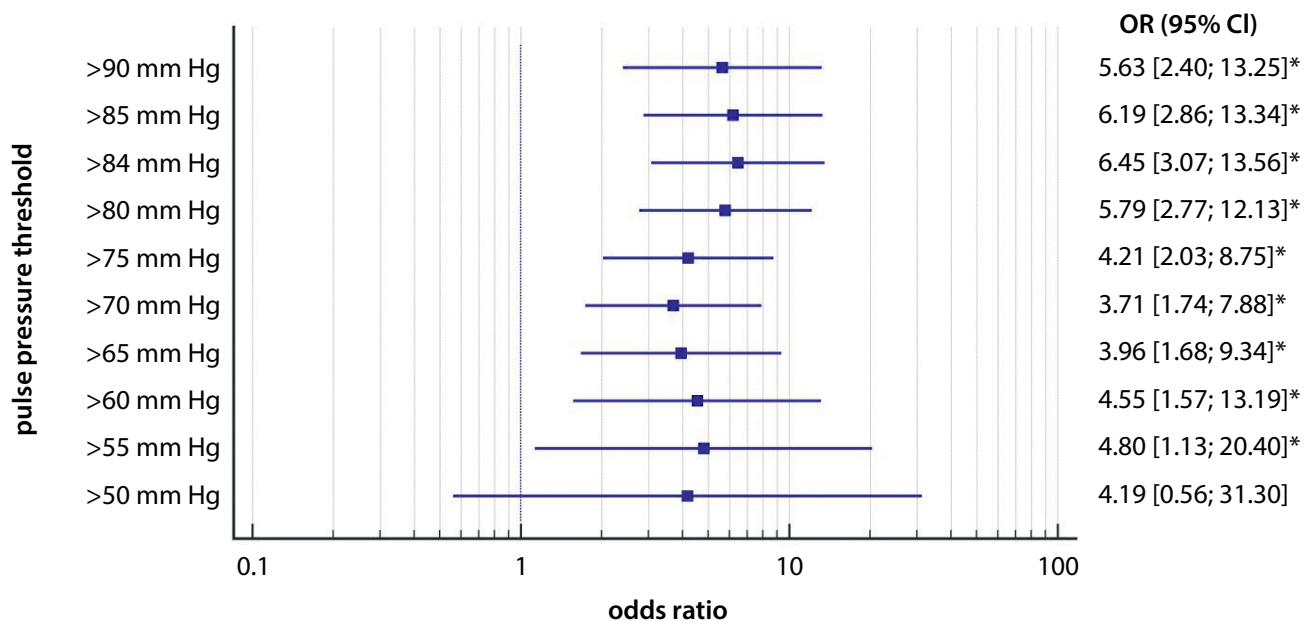


Fig. 3. Pulse pressure thresholds and their relationship with acute kidney injury. The box represents odds ratio (OR) whereas the whiskers represent confidence intervals (CIs). The asterisk represents statistically significant values

Table 4. Multivariate logistic regression models in predicting the occurrence of acute kidney injury (AKI)

Variable*	Model			
	PP > 80 mm Hg (1/0)	PP > 84 mm Hg (1/0)	PP > 85 mm Hg (1/0)	PP > 90 mm Hg (1/0)
Pulse pressure	OR = 2.61 95% CI: [1.13; 6.05] β = 0.96 p = 0.0245	OR = 3.13 95% CI: [1.35–7.22] β = 1.14 p = 0.0011	OR = 3.17 95% CI: [1.33–7.54] β = 1.15 p = 0.0090	OR = 4.03 95% CI: [1.53–10.62] β = 1.39 p = 0.0048
Chronic arterial hypertension (1/0)	OR = 3.13 95% CI: [1.17–8.35] β = 1.14 p = 0.0226	OR = 3.07 95% CI: [1.29–7.06] β = 1.12 p = 0.0079	OR = 3.20 95% CI: [1.21–8.46] β = 1.16 p = 0.0192	OR = 4.20 95% CI: [1.56–11.35] β = 1.43 p = 0.0046
Procedure duration (per 1 min)	OR = 1.006 95% CI: [1.003–1.008] β = 0.0062 p < 0.0001	OR = 1.006 95% CI: [1.003–1.009] β = 0.0063 p < 0.0001	OR = 1.007 95% CI: [1.003–1.009] β = 0.0066 p < 0.0001	OR = 1.007 95% CI: [1.003–1.009] β = 0.0066 p < 0.0001
Chronic kidney disease (1/0)	OR = 6.72 95% CI: [1.71–26.45] β = 1.90 p < 0.0001	OR = 6.59 95% CI: [1.66–26.17] β = 1.88 p < 0.0001	OR = 5.93 95% CI: [1.47–23.84] β = 1.78 p < 0.0001	OR = 6.14 95% CI: [1.54–24.53] β = 1.81 p < 0.0001
Intraoperative hypotension (MAP < 60 mm Hg) (1/0)	not included	not included	not included	OR = 2.54 95% CI: [1.07–6.04] β = 0.93 p = 0.0102
AUROC	0.861; (0.828–0.891); p < 0.0001	0.866; (0.83–0.89); p < 0.0001	0.862; (0.83–0.89); p < 0.0001	0.880; (0.85–0.91); p < 0.0001
Hosmer–Lemeshow test	χ^2 = 3.49; p = 0.8995	χ^2 = 7.71; p = 0.4621	χ^2 = 4.68; p = 0.7887	χ^2 = 3.87; p = 0.8768

PP – pulse pressure; MAP – mean arterial pressure; AUROC – area under the receiver operating characteristics; OR – odds ratio; 95% CI – 95% confidence interval; β – coefficient; p – p-value. * Variables that failed to be significant in the multivariable models were as follows: PP > 50 mm Hg, PP > 55 mm Hg, PP > 60 mm Hg, PP > 65 mm Hg, PP > 70 mm Hg, PP > 75 mm Hg, age, American Society of Anesthesiologists (ASA) III/IV/V, Charlson comorbidity index (CCI), adjunction of regional anesthesia, procedure risk (III), oncological procedure, catecholamine use, systolic blood pressure (SBP) >160 mm Hg, diastolic blood pressure (DBP) <50 mm Hg, SBP (per 1 mm Hg).

High PP could influence systemic circulation in numerous ways. First, kidneys have a high resting blood flow. With the increase of PP, perfusion of this organ becomes more pulsatile and it is thought to damage endothelium and smooth muscle and induce shear stress, which can cause plaque to rupture and form thrombosis.^{28–30} Additionally, high PP can decrease flow-mediated vasodilation.³¹ What is also worth mentioning is that increased PP causes aortic lumen to decrease, which results in ventricular-aortic decoupling, characterized by cardiac output that is too great to be accommodated by aortic lumen (leading to the impaired cardiac output with preserved systolic function).^{5,32}

Limitations



The abovementioned findings should be analyzed with caution due to possible confounding factors. First, the vast majority of patients had their BP measured with the oscillometric method. Due to an imperfect algorithmic method of distinguishing SBP and DBP, such patients have a higher risk of discrepancy between the registered and real BP. The discrepancy, especially in SBP, is more often expressed in patients with stiffer arteries and higher PPs.³³ In a study by Kayrak et al., oscillometric measurements led to the underestimation of PP in a group of patients with isolated

systolic hypertension (but not in subjects with mixed hypertension).³⁴ Secondly, the true association between high intraoperative PP and AKI is, to a certain extent, determined by the preoperative PP values. Despite the fact that pre-induction PP was not significantly related to the outcome in our analysis, it is possible that intraoperative PP is only a reflection of the overall cardiovascular condition and does not impair organ perfusion in a short-term period (such as the duration of surgical procedure). Thirdly, pre-induction BP value was defined as baseline MAP. It is possible that such measurement does not represent the true baseline, as it could be influenced by stress or premedication. Additionally, the BP measurements were recorded in 5-minute intervals, and therefore, a risk of underrecognition of PP changes exists. Finally, our analysis was restricted to a limited population of abdominal patients, which reduces the generalizability of our results into all noncardiac surgery settings.

Conclusions

High intraoperative PP may be associated with AKI in patients undergoing abdominal surgery. However, the effect of high PP should be confirmed in other noncardiac populations to prove the generalizability of our results.

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References

- 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: A retrospective cohort analysis. *Anesthesiology*. 2017;126(1):47–65. doi:10.1097/ALN.0000000000001432
- Bijker JB, Persoon S, Peelen LM, et al. Intraoperative hypotension and perioperative ischemic stroke after general surgery: A nested case-control study. *Anesthesiology*. 2012;116(3):658–664. doi:10.1097/ALN.0b013e3182472320
- 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(4):706–721. doi:10.1016/j.bja.2018.04.036
- McEvoy MD, Gupta R, Koepke EJ, et al. Perioperative quality initiative consensus statement on postoperative blood pressure, risk and outcomes for elective surgery. *Br J Anaesth*. 2019;122:575–86. doi:10.1016/j.bja.2019.01.019
- Abbott TEF, Pearse RM, Archbold RA, et al. Association between preoperative pulse pressure and perioperative myocardial injury: An international observational cohort study of patients undergoing non-cardiac surgery. *Br J Anaesth*. 2017;119(1):78–86. doi:10.1093/bja/aex165
- Cheng S, Xanthakis V, Sullivan LM, Vasan RS. Blood pressure tracking over the adult life course patterns and correlates in the Framingham heart study. 2012;60(6):1393–1399. doi:10.1161/HYPERTENSION.AHA.112.201780
- 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(5):563–574. doi:10.1016/j.bja.2019.01.013
- Czajka S, Putowski Z, Krzych ŁJ. Intraoperative hypotension and its organ-related consequences in hypertensive subjects undergoing abdominal surgery: A cohort study. *Blood Press*. 2021;30(6):1–11. doi:10.1080/08037051.2021.1947777
- National Confidential Enquiry Into Patient Outcome and Death. <https://www.ncepod.org.uk/classification.html>. Accessed January 11, 2021.
- World Health Organization. ICD-10: International statistical classification of diseases and related health problems. 10th revision, 2004. Spanish version, 1st edition published by PAHO as: <https://apps.who.int/iris/handle/10665/42980>.
- Doyle DJ, Goyal A, Bansal P, Garmon EH. *American Society of Anesthesiologists Classification (ASA Class)*. Treasure Island, USA: StatPearls Publishing; 2020. PMID:28722969.
- Kristensen SD, Knuuti J, Saraste A, et al. 2014 ESC/ESA guidelines on non-cardiac surgery: Cardiovascular assessment and management. *Eur Heart J*. 2014;35(35):2383–2431. doi:10.1093/eurheartj/ehu282
- Homan TD, Bordes S, Cichowski E. *Physiology, Pulse Pressure*. Treasure Island, USA: StatPearls Publishing; 2021. PMID:29494015.
- Fontes ML, Aronson S, Mathew JP, et al. Pulse pressure and risk of adverse outcome in coronary bypass surgery. *Anesth Analg*. 2008;107(4):1122–1129. doi:10.1213/ane.0b013e31816ba404
- Blacher J, Evans A, Arveiler D, et al. Residual cardiovascular risk in treated hypertension and hyperlipidaemia: The PRIME study. *J Hum Hypertens*. 2010;24(1):19–26. doi:10.1038/jhh.2009.34
- 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: Results of the VISION study. *Anesth Analg*. 2018;126(6):1936–1945. doi:10.1213/ANE.0000000000002560
- Ahuja S, Mascha EJ, Yang D, et al. Associations of intraoperative radial arterial systolic, diastolic, mean, and pulse pressures with myocardial and acute kidney injury after noncardiac surgery: A retrospective cohort analysis. *Anesthesiology*. 2020;132(2):291–306. doi:10.1097/ALN.0000000000003048
- Sun LY, Wijeyesundera DN, Tait GA, Beattie WS. Association of intraoperative hypotension with acute kidney injury after elective noncardiac surgery. *Anesthesiology*. 2015;123(3):515–523. doi:10.1097/ALN.0000000000000765
- Kellum JA, Lameire N; KDIGO AKI Guideline Work Group. Diagnosis, evaluation, and management of acute kidney injury: A KDIGO summary (Part 1). *Crit Care*. 2013;17(1):204. doi:10.1186/cc11454
- Kellum JA, Lameire N, Aspelin P, et al. Kidney disease: Improving global outcomes (KDIGO) acute kidney injury work group. KDIGO clinical practice guideline for acute kidney injury. *Kidney Int Suppl*. 2012;2(1):1–138. <https://kdigo.org/wp-content/uploads/2016/10/KDIGO-2012-AKI-Guideline-English.pdf>
- von Elm E, Altman DG, Egger M, et al.; STROBE Initiative. The Strengthening of Reporting of Observational Studies in Epidemiology (STROBE) Statement: Guidelines for reporting observational studies. *Int J Surg*. 2014;12(12):1495–1499. doi:10.1016/j.ijsu.2014.07.013
- Mancusi C, Losi MA, Izzo R, et al. Higher pulse pressure and risk for cardiovascular events in patients with essential hypertension: The Campania Salute Network. *Eur J Prev Cardiol*. 2018;25(3):235–243. doi:10.1177/2047487317747498
- Howell SJ, Sear YM, Yeates D, Goldacre M, Sear JW, Foex P. Hypertension, admission blood pressure and perioperative cardiovascular risk. *Anaesthesia*. 1996;51(11):1000–1004. doi:10.1111/j.1365-2044.1996.tb14990.x
- Haider AW, Larson MG, Franklin SS, Levy D; Framingham Heart Study. Systolic blood pressure, diastolic blood pressure, and pulse pressure as predictors of risk for congestive heart failure in the Framingham Heart Study. *Ann Intern Med*. 2003;138(1):10–16. doi:10.7326/0003-4819-138-1-200301070-00006
- Mitrev L, Speich KG, Ng S, et al. Elevated pulse pressure in anesthetized subjects before cardiopulmonary bypass is associated strongly with postoperative acute kidney injury stage. *J Cardiothorac Vasc Anesth*. 2019;33(6):1620–1626. doi:10.1053/j.jvca.2019.01.019
- Assmann G, Cullen P, Evers T, Petzinna D, Schulte H. Importance of arterial pulse pressure as a predictor of coronary heart disease risk in PROCAM. *Eur Heart J*. 2005;26(20):2120–2126. doi:10.1093/eurheartj/ehi467
- Sato R, Luthe SK, Nasu M. Blood pressure and acute kidney injury. *Crit Care*. 2017;21(1):28. doi:10.1186/s13054-017-1611-7
- O'Rourke MF, Safar ME. Relationship between aortic stiffening and microvascular disease in brain and kidney: Cause and logic of therapy. *Hypertension*. 2005;46(1):200–204. doi:10.1161/01.HYP.0000168052.00426.65
- Lyon RT, Runyon-Hass A, Davis HR, Glagov S, Zarins CK. Protection from atherosclerotic lesion formation by reduction of artery wall motion. *J Vasc Surg*. 1987;5(1):59–67. PMID:3795393.
- Traub O, Berk BC. Laminar shear stress: Mechanisms by which endothelial cells transduce an atheroprotective force. *Arterioscler Thromb Vasc Biol*. 1998;18(5):677–685. doi:10.1161/01.atv.18.5.677
- Ceravolo R, Maio R, Pujia A, et al. Pulse pressure and endothelial dysfunction in never-treated hypertensive patients. *J Am Coll Cardiol*. 2003;41(10):1753–1758. doi:10.1016/s0735-1097(03)00295-x
- Mitchell GF, Lacourcière Y, Ouellet JP, et al. Determinants of elevated pulse pressure in middle-aged and older subjects with uncomplicated systolic hypertension: The role of proximal aortic diameter and the aortic pressure-flow relationship. *Circulation*. 2003;108(13):1592–1598. doi:10.1161/01.CIR.0000093435.04334.1F
- Stergiou G, Lourida P, Tzamouranis D, Baibas NM. Unreliable oscillometric blood pressure measurement: Prevalence, repeatability and characteristics of the phenomenon. *J Hum Hypertens*. 2009;23(12):794–800. doi:10.1038/jhh.2009.20
- Kayrak M, Ulgen MS, Yazici M, et al. A comparison of blood pressure and pulse pressure values obtained by oscillometric and central measurements in hypertensive patients. *Blood Press*. 2010;19(2):98–103. doi:10.3109/08037050903516318