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Intraoperative hemodynamic imbalance quantification: clinical validation of heart rate to mean blood pressure ratio in predicting myocardial injury after noncardiac surgery

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Abstract

Background The effects of isolated heart rate (HR) and mean blood pressure (MBP) on myocardial injury after noncardiac surgery (MINS) have been investigated, but the combined impact of intraoperative HR and MBP remains unclear. This study aimed to assess the influence of the heart rate–mean arterial pressure ratio (HMR) on MINS to optimize hemodynamic management.

Methods This retrospective cohort study included adult patients who underwent general anesthesia and postoperative troponin measurements at Meizhou People's Hospital. The primary exposure was the time-weighted area above the HMR threshold (1.0) (TWAAT-HMR > 1.0), and the primary outcome was MINS within one postoperative day. The diagnostic performance of TWAAT-HMR > 1.0, the time-weighted area under MBP < 60 mmHg, and the time-weighted area above HR > 100 bpm was evaluated using Receiver Operating Characteristic (ROC) analysis. Logistic regression and restricted cubic splines (RCS) were used to assess the association between HMR and MINS. Sensitivity analyses were conducted to confirm the robustness of the findings, and subgroup analyses examined potential interactions with age, sex, and body mass index.

Results Among 699 patients, the incidence of MINS was 9.4%. TWAAT-HMR > 1.0 demonstrated superior predictive accuracy for MINS compared to time-weighted areas under/above MBP and HR (AUC: 0.708 vs. 0.646 and 0.640, respectively). TWAAT-HMR > 1.0 was identified as an independent risk factor for MINS (odds ratio [OR] = 1.71, 95% confidence interval [CI] 1.35–2.17, $p < 0.001$). RCS analysis showed a linear increase in MINS risk with rising HMR (p for non-linearity = 0.507). Sensitivity and subgroup analyses supported the primary findings.

Conclusion Elevated HMR is associated with a higher risk of MINS in adults undergoing general anesthesia. HMR monitoring may serve as a valuable parameter for optimizing perioperative hemodynamic management.

Keywords General anesthesia, Noncardiac surgery, Troponin, Heart rate, Hypotension

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Introduction

Myocardial injury after noncardiac surgery (MINS) is defined as myocardial ischemia-induced damage occurring within 30 days postoperatively and is associated with poor prognosis [1]. Risk factors include advanced age, male sex, hypertension, diabetes, coronary heart disease (CHD), peripheral artery disease, cerebrovascular disease, heart failure, atrial fibrillation, intraoperative hypotension (IOH), and tachycardia [2–5]. MINS is commonly diagnosed using elevated cardiac troponin (cTn) or high-sensitivity cTn, with reported incidence rates ranging from 3 to 16%, depending on diagnostic criteria and study populations [6, 7]. A significant cTn elevation within three days postoperatively increases 30-day mortality, which has been reported between 9.8% and 12.6% [8–12].

The primary mechanisms underlying MINS include coronary artery thrombosis [13–15] and myocardial oxygen supply-demand imbalance [16], with the latter being the predominant factor [17]. Both hypotension and tachycardia contribute to MINS by exacerbating this imbalance [16, 18–21]. A meta-analysis on IOH found that prolonged exposure to low mean blood pressure (MBP) increases the risk of myocardial injury [22]. Similarly, the VISION study reported an association between heart rate (HR) > 100 bpm and MINS, particularly when combined with systolic blood pressure (SBP) < 100 mmHg [23]. However, while IOH and tachycardia frequently co-occur, the VISION study analyzed these parameters separately, potentially limiting the clinical applicability of its risk assessment framework. Given their dynamic interplay, a more integrated approach may better reflect perioperative hemodynamic risks.

Maintaining the intraoperative heart rate to MBP ratio (HMR) ≤ 1 may help balance myocardial oxygen supply and demand. Unlike isolated HR or MBP measurements, HMR integrates both parameters at the same time point. However, research on its association with MINS remains limited. Establishing this relationship could enhance intraoperative hemodynamic management, particularly in settings with restricted monitoring capabilities. A defined HMR threshold may aid in preventing perioperative cardiovascular complications.

This study aims to investigate the association between intraoperative HMR and MINS in patients undergoing general anesthesia using data from the Meizhou People's Hospital cohort. To our knowledge, this is the first study to evaluate HMR—an index integrating IOH and tachycardia—as a predictor of MINS. We hypothesize that an increased time-weighted area above the predefined HMR threshold (1.0) (TWAAT-HMR > 1.0) and prolonged high HMR exposure are independently associated with an elevated risk of MINS.

Methods and materials

Human ethics and consent to participate declarations

This retrospective study was conducted at Meizhou People's Hospital and approved by its Institutional Review Board (IRB) (Ethics No. 2023-C-93). Given the study's retrospective design, the IRB waived the requirement for informed consent. The study was registered in the Chinese Clinical Trial Registry on March 18, 2024 (ChiCTR2400081991) and conducted in accordance with the principles of the Declaration of Helsinki. The report follows the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines [24].

Study population

Inclusion criteria: Patients were eligible if they were aged ≥ 40 years, underwent noncardiac surgery under general anesthesia (GA), and had recorded postoperative cardiac troponin (cTn) measurements, as well as intraoperative HR, SBP, and diastolic blood pressure (DBP). Exclusion criteria: Patients were excluded if they lacked postoperative cTn measurements, intraoperative HR, SBP, or DBP records, baseline characteristics, or had severe infections, oxygenation disorders, or major blood loss.

Confounding variables

Patient characteristics included age, gender, body mass index (BMI), and nicotine dependence. Preoperative comorbidities comprised hypertension, atrial fibrillation, CHD, congestive heart failure (CHF), chronic obstructive pulmonary disease (COPD), stroke history, and peripheral vascular disease (PVD). Medication history encompassed rate-controlling drugs (β-blockers, diltiazem, verapamil), statins, heparin, and aspirin. Preoperative laboratory parameters included estimated glomerular filtration rate (eGFR), hemoglobin (Hb), and glucose levels. Surgical factors considered were the American Society of Anesthesiologists (ASA) classification, urgency of surgery, surgical type, duration of GA, intraoperative blood loss, and red blood cell (RBC) transfusion. Comorbidities were identified using ICD-10 codes.

Exposures

The primary exposure was the intraoperative time-weighted area above the predefined HMR threshold (1.0) (TWAAT-HMR > 1.0). HMR index = HR (beats/min) / MBP (mmHg). The TWAAT-HMR = (depth HMR below HMR threshold 1.0 × time spent below HMR threshold 1.0 in minutes) / length of anesthesia in minutes (Supplementary Figure S1). TWAAT-HMR > 1.0 accounts for both dynamic HMR changes and temporal variations, minimizing the influence of individual time points. The time-weighted averaging reduces transient extreme values' impact on the overall mean, thereby more accurately

Table 1 Baseline characteristics of patients grouped by MINS within postoperative 1 day

	MINS within postoperative 1 day		*p*
Variables	**No, *N* = 633**	**Yes, *N* = 66**	
TWAAT-HMR > 1.0 quartiles	0.02[0.00, 0.07]	0.07[0.03, 0.20]	< 0.001
Age (years)	64[55, 73]	67[61, 76]	0.012
Gender (woman)	252(39.8)	19(28.8)	0.080
BMI ≥ 24 kg/m^2	189(29.9)	10(15.2)	0.012
Past medical history			
Hypertension	126(19.9)	17(25.8)	0.262
Atrial fibrillation	2(0.3)	1(1.5)	0.258
COPD	44(7.0)	11(16.7)	0.005
Coronary heart disease	22(3.5)	3(4.5)	0.723
Peripheral Vascular Disease	26(4.1)	4(6.1)	0.516
History of stroke	29(4.6)	5(7.6)	0.359
Congestive heart failure	6(0.9)	3(4.5)	0.045
Diabetes	63(10.0)	6(9.1)	0.823
Nicotine dependence	83(13.1)	12(18.2)	0.253
Preoperative medications ≤ 7 days before surgery			
Rate-controlling medications	24(3.8)	0(0.0)	0.155
Aspirin	14(2.2)	1(1.5)	> 0.999
Statins	43(6.8)	7(10.6)	0.310
Preoperative heparin ≤ 24 h before surgery	116(18.3)	16(24.2)	0.242
Preoperative eGFR mL/min/1.73 m^2			< 0.001
≤ 44	27(4.3)	7(10.6)	
45–59	59(9.3)	15(22.7)	
≥ 60	547(86.4)	44(66.7)	
Preoperative hemoglobin (g/L)	131[114, 144]	129[115, 140]	0.652
Preoperative glucose (mmol/L)	5.26[4.57, 7.22]	5.33[4.62, 6.44]	0.933
Preoperative triglyceride (mmol/L)	1.18[0.84, 1.56]	1.19[0.82, 1.65]	0.588
ASA physical status			0.121
I-II	41(6.5)	1(1.5)	
III	419(66.2)	41(62.1)	
IV	173(27.3)	24(36.4)	
Emergency	97(15.3)	20(30.3)	0.002
Surgery type			0.132
Low-risk surgery	26(4.1)	6(9.1)	
Thoracic surgery	432(68.2)	46(69.7)	
Non-thoracic major surgery	175(27.6)	14(21.2)	
Intraoperative blood bleeding (ml)	50[20, 84]	50[20, 100]	0.089
Intraoperative RBC infusion	16 (2.5)	6 (9.1)	0.001

Categorical data are presented as n (%), and for continuous variables, data are presented as median [quartile]. MINS: Myocardial Injury after Noncardiac Surgery; HMR: heart rate to mean blood pressure Ratio; TWAAT-HMR: time-weighted area above the HMR threshold; BMI: Body Mass Index; COPD: Chronic Obstructive Pulmonary Disease; Rate-controlling medications: verapamil, diltiazem and β-blockers; eGFR: Estimated Glomerular Filtration Rate; ASA: American Society of Anesthesiologists; RBC: red blood cell; eGFR was calculated using the CKD-EPI formula

representing the overall HMR level. MBP and HR were measured using non-invasive and/or invasive methods at 5-minute intervals intraoperatively.

Outcome

The primary outcome was MINS within the first postoperative day. To reduce confounding from other postoperative factors, only patients with cTn measurements within 24 h postoperatively were included. If multiple measurements were available, the peak cTn value was used. MINS was diagnosed when postoperative cTn levels exceeded the 99th percentile upper reference limit without evidence of nonischemic causes [2, 25].

Statistical analysis

Patients were stratified into four groups based on TWAAT-HMR > 1.0 quartiles. Continuous variables, assessed using the Shapiro-Wilk test, were non-normally distributed and presented as medians [quartiles]. Group differences for continuous variables were evaluated using the Wilcoxon or Kruskal-Wallis rank sum tests. Categorical variables were expressed as frequencies (percentages) and compared using Pearson's Chi-squared test or Fisher's exact test. Receiver operating characteristic (ROC) curves were constructed to compare the diagnostic performance of various indicators (HMR, MBP, HR, and SBP×HR) and their extreme values in predicting early MINS. Logistic regression analysis was conducted to determine the odds ratio (OR) for the association between TWAAT-HMR > 1.0, the highest HMR, and early MINS. Model 1: unadjusted model; Model 2: age, gender, BMI ≥ 24 kg/m^2; Model 3: included age, gender, BMI ≥ 24 kg/m^2, COPD, CHF, preoperative eGFR, emergency, intraoperative blood loss, intraoperative RBC infusion (variables with $p < 0.010$ in Table 1). A three-node restricted cubic spline (RCS) model was applied to multivariable logistic regression to explore potential non-linear relationships between exposures and early MINS. Sensitivity analyses were performed by excluding patients with (1) MBP <55 mmHg and HR <55 bpm or (2) MBP >120 mmHg and HR >120 bpm. Post-hoc subgroup analyses and interaction tests were conducted for age, gender, and BMI.

Data analysis was performed using R (4.2.3) and Zstats v0.90 (www.medsta.cn/software). A two-tailed P-value < 0.05 was considered statistically significant.

Results

Patient selection

A total of 2,303 patients who underwent GA with postoperative cTn measurement were identified at Meizhou People's Hospital. Of these, 699 patients met the inclusion criteria and were included in the analysis (Fig. 1).

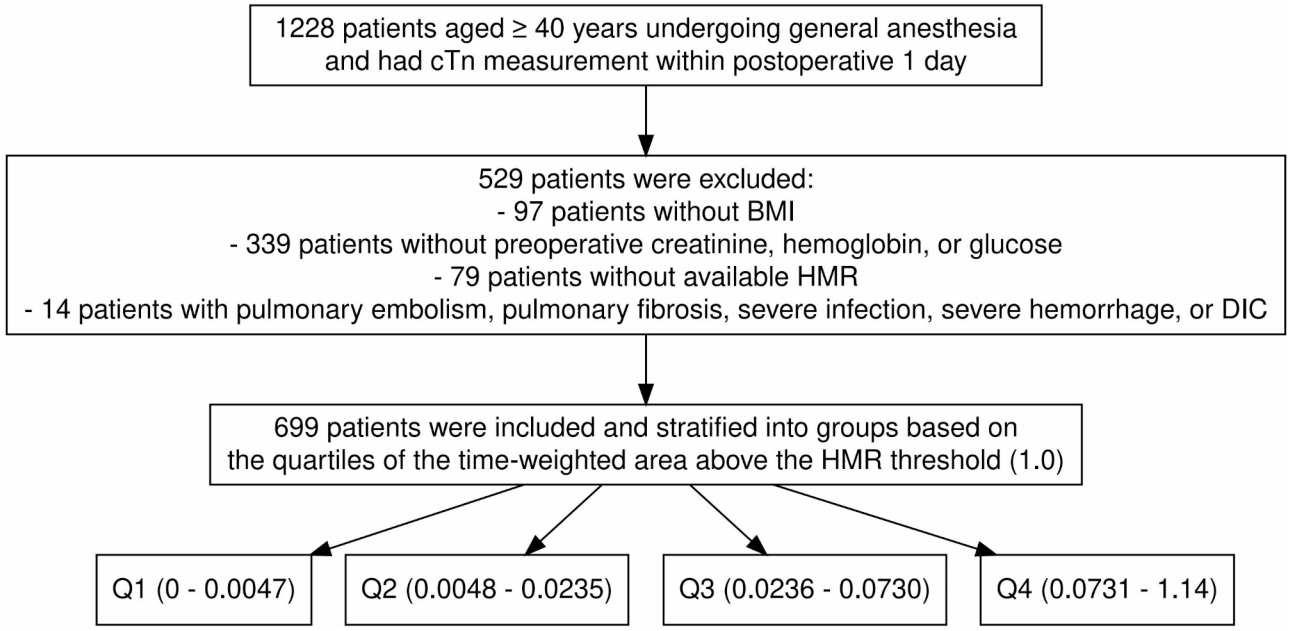

Fig. 1 Flowchart of participant inclusion and exclusion. cTn: cardiac troponin; BMI: body mass index; HMR: heart rate to mean arterial pressure ratio; DIC: disseminated intravascular coagulation

Patients were stratified into quartiles based on intraoperative TWAAT-HMR > 1.0.

Cohort characteristics

Baseline characteristics are summarized in Table 2. The median age of the cohort was 64 years, with women comprising 38.8% of the population. The most prevalent comorbidities were hypertension (20.5%), diabetes (9.9%), and COPD (7.9%). Regarding surgical procedures, 27.0% of patients underwent thoracic surgery, while 68.4% underwent non-thoracic major surgeries. The median TWAAT-HMR > 1.0 was 0.02 [0.00, 0.07]. The overall incidence of early MINS was 9.4%. As shown in Table 1, patients with early MINS tended to have higher TWAAT-HMR > 1.0 values and were generally older with a higher prevalence of comorbidities. Conversely, patients without early MINS had a higher proportion of females, better preoperative eGFR, and a greater likelihood of undergoing elective surgery (Table 1).

ROC curves

TWAAT-HMR > 1.0 demonstrated the highest discriminatory ability for early MINS compared to the time-weighted area under/above MBP < 60 mmHg, HR > 100 bpm, and SBP×HR > 12,000 (AUC: 0.708, 0.646, 0.640, and 0.552, respectively; Supplementary Table S2). The TWAAT-HMR > 1.0 had a sensitivity of 72.7% and a specificity of 60.8%. Additionally, the highest intraoperative HMR exhibited a superior AUC compared to the lowest MBP, highest HR, and highest SBP×HR (Supplementary Table S3). The optimal HMR cutoff value was determined as 1.3 using the Youden index. To improve clinical applicability and feasibility, an HMR threshold of 1.0 was adopted for analysis (Supplementary Table S3). The duration of HMR > 1.0 also had a higher AUC than the duration of SBP > 160 mmHg or < 90 mmHg, HR > 99 bpm, and SBP×HR > 12,000 (Supplementary Table S3).

Outcome

Multivariable logistic regression demonstrated that TWAAT-HMR > 1.0, the highest intraoperative HMR, and the duration of HMR > 1.0 were independently associated with an increased risk of MINS (OR 1.71; 95% CI 1.35–2.17; $p < 0.001$; Table 3; Supplementary Table S3). A significant difference in MINS incidence was observed between the Q3/Q4 and Q1 groups (p for trend < 0.001). Multivariable-adjusted RCS analysis indicated a linear association between TWAAT-HMR > 1.0 and MINS risk (p for non-linearity = 0.507; Fig. 2). Similar linear relationships were observed for the highest HMR and the duration of HMR > 1.0 (p for non-linearity = 0.417, 0.382, respectively; Supplementary Figure S4).

Sensitivity analysis

To assess result stability, a sensitivity analysis was performed by excluding patients with extreme MBP and HR values (MBP > 120 mmHg and HR > 120 bpm, or MBP < 55 mmHg and HR < 55 bpm). The analysis of the remaining cohort confirmed that a higher TWAAT-HMR > 1.0 remained independently associated with an increased risk of MINS (OR 1.47; 95% CI 1.13–1.93;

Table 2 Baseline characteristics of patients stratified by the time-weighted the area above the curve HMR threshold of 1.0

Variables	Overall *N* = 699	Q1, *N* = 175	Q2, *N* = 174	Q3, *N* = 175	Q4, *N* = 175	*p*
TWAAT-HMR > 1.0 range		0–0.0047	0.0048–0.0235	0.0236–0.0730	0.0731–1.14	
TWAAT-HMR > 1.0 quartiles	0.02[0.00, 0.07]	0.00[0.00, 0.00]	0.01[0.01, 0.02]	0.04[0.03, 0.06]	0.13[0.10, 0.21]	
Age (years)	64 [55, 73]	62 [55, 72]	64 [56, 72]	64 [56, 72]	65 [55, 76]	0.435
Gender (woman)	271(38.8)	79(45.1)	69(39.7)	61(34.9)	62(35.4)	0.172
BMI ≥ 24 kg/m^2	199(28.5)	60(34.3)	49(28.2)	40(22.9)	50(28.6)	0.131
Past medical history (%)						
Hypertension	143(20.5)	44(25.1)	31(17.8)	34(19.4)	34(19.4)	0.343
Atrial fibrillation	3(0.4)	0(0.0)	0(0.0)	0(0.0)	3(1.7)	0.062
COPD	55(7.9)	8(4.6)	11(6.3)	19(10.9)	17(9.7)	0.103
Coronary heart disease	25(3.6)	10(5.7)	4(2.3)	5(2.9)	6(3.4)	0.332
Peripheral Vascular Disease	30(4.3)	7(4.0)	5(2.9)	9(5.1)	9(5.1)	0.681
History of stroke	34(4.9)	13(7.4)	4(2.3)	7(4.0)	10(5.7)	0.138
Congestive heart failure	9(1.3)	2(1.1)	1(0.6)	1(0.6)	5(2.9)	0.334
Diabetes	69(9.9)	19(10.9)	13(7.5)	21(12.0)	16(9.1)	0.510
Nicotine dependence	95(13.6)	18(10.3)	30(17.2)	27(15.4)	20(11.4)	0.187
Preoperative medications ≤ 7 days before surgery						
Rate-controlling medications	24(3.4)	7(4.0)	10(5.7)	0(0.0)	7(4.0)	0.025
Aspirin	15(2.1)	7(4.0)	3(1.7)	1(0.6)	4(2.3)	0.173
Statins	50(7.2)	20(11.4)	10(5.7)	6(3.4)	14(8.0)	0.027
Preoperative heparin ≤ 24 h before surgery	132(18.9)	31(17.7)	29(16.7)	27(15.4)	45(25.7)	0.060
Preoperative eGFR mL/min/1.73 m^2						0.282
≤ 44	34(4.9)	10(5.7)	6(3.4)	8(4.6)	10(5.7)	
45–59	74(10.6)	16(9.1)	12(6.9)	21(12.0)	25(14.3)	
≥ 60	591(84.5)	149(85.1)	156(89.7)	146(83.4)	140(80.0)	
Preoperative hemoglobin (g/L)	129[115, 141]	131[117, 143]	130[117, 139]	130[119, 139]	122[106, 143]	0.123
Preoperative glucose (mmol/L)	5.32[4.62, 6.50]	5.30[4.67, 6.20]	5.23[4.62, 6.20]	5.17[4.52, 6.24]	5.92[4.86, 7.63]	< 0.001
Preoperative triglyceride (mmol/L)	1.19[0.82, 1.63]	1.17[0.81, 1.57]	1.22[0.90, 1.67]	1.19[0.84, 1.72]	1.16[0.78, 1.58]	0.300
ASA physical status						< 0.001
I-II	42(6.0)	19(10.9)	7(4.0)	10(5.7)	6(3.4)	
III	460(65.8)	113(64.6)	134(77.0)	120(68.6)	93(53.1)	
IV	197(28.2)	43(24.6)	33(19.0)	45(25.7)	76(43.4)	
Emergency	117(16.7)	20(11.4)	16(9.2)	27(15.4)	54(30.9)	< 0.001
Surgery type						< 0.001
Low-risk surgery	32(4.6)	7(4.0)	5(2.9)	8(4.6)	12(6.9)	
Non-thoracic major surgery	478(68.4)	148(84.6)	114(65.5)	105(60.0)	111(63.4)	
Thoracic surgery	189(27.0)	20(11.4)	55(31.6)	62(35.4)	52(29.7)	
Intraoperative RBC infusion	22 (3.2)	1 (0.6)	5 (2.9)	4 (2.3)	12 (6.9)	0.007
Intraoperative blood bleeding (ml)	50[20, 100]	30[20, 50]	50[20, 100]	50[20, 100]	50[20, 100]	0.030
MINS within postoperative 1 day	66(9.4)	4(2.3)	12(6.9)	18(10.3)	32(18.3)	< 0.001

Categorical data are presented as n (%), and for continuous variables, data are presented as median [quartile]. HMR: heart rate to mean blood pressure Ratio; TWAAT-HMR: time-weighted area above the HMR threshold; BMI: Body Mass Index; COPD: Chronic Obstructive Pulmonary Disease; Rate-controlling medications: verapamil, diltiazem and β-blockers; eGFR: Estimated Glomerular Filtration Rate; ASA: American Society of Anesthesiologists; RBC: red blood cell; eGFR was calculated using the CKD-EPI formula; MINS: Myocardial Injury after Noncardiac Surgery.

$p = 0.004$; Supplementary Table S5). As TWAAT-HMR > 1.0 increased, MINS risk significantly rose.

Post-hoc subgroup analysis

Subgroup analyses were largely consistent with the primary findings (Fig. 3). However, in patients with BMI > 24 kg/m², TWAAT-HMR > 1.0 was not significantly associated with early MINS (OR 1.05; 95% CI 0.96–1.14; $p = 0.336$). No significant interaction effects were observed between subgroups.

Discussion

Our study is the first to establish the synergistic effect of concurrent HR and MBP on early MINS risk. We demonstrate that a higher TWAAT-HMR > 1.0 is independently and linearly associated with an increased risk of early MINS. By explicitly quantifying the HMR threshold, our findings offer a novel perspective for intraoperative hemodynamic management. To our knowledge, research on the association between HMR and MINS remains limited.

Table 3 Results of logistic regression for MINS within postoperative 1 day and the time-weighted the area above the HMR threshold (1.0)

	Crude Model 1		Adjusted Model 2		Adjusted Model 3	
Variables	**OR (95%CI)**	***p***	**OR (95%CI)**	***p***	**OR (95%CI)**	***p***
Continuous TWAAT-HMR > 1.0 per 0.1	1.89(1.52–2.35)	< 0.001	1.88(1.50–2.34)	< 0.001	1.71(1.35–2.17)	< 0.001
The TWAAT-HMR threshold (1.0) range						
Q1(0–0.0047)	Reference	/	Reference	/	Reference	/
Q2(0.0048–0.0235)	3.17(1.01–10.02)	0.050	3.03(0.95–9.65)	0.060	2.92(0.91–9.44)	0.073
Q3(0.0236–0.0730)	4.90(1.62–14.80)	0.005	4.35(1.43–13.21)	0.010	3.76(1.22–11.65)	0.022
Q4(0.0731–1.14)	9.57(3.30–27.69)	< 0.001	8.83(3.03–25.70)	< 0.001	6.08(2.03–18.26)	0.001
p for trend	< 0.001		< 0.001			< 0.001

Crude Model 1: unadjusted.

Adjusted Model 2: adjusted for age, gender, BMI ≥ 24 kg/m^2

Adjusted Model 3: adjusted for age, gender, BMI ≥ 24 kg/m^2, COPD, CHF, preoperative eGFR, emergency, intraoperative blood loss, intraoperative RBC infusion.

Abbreviations: MINS: Myocardial Injury after Noncardiac Surgery; OR: Odds ratio; CI: confidence interval; HMR: heart rate to mean blood pressure ratio; TWAAT-HMR: time-weighted area above the HMR threshold; BMI: Body Mass Index; COPD: Chronic Obstructive Pulmonary Disease; CHF: congestive heart failure; eGFR: Estimated Glomerular Filtration Rate; RBC: red blood cell.

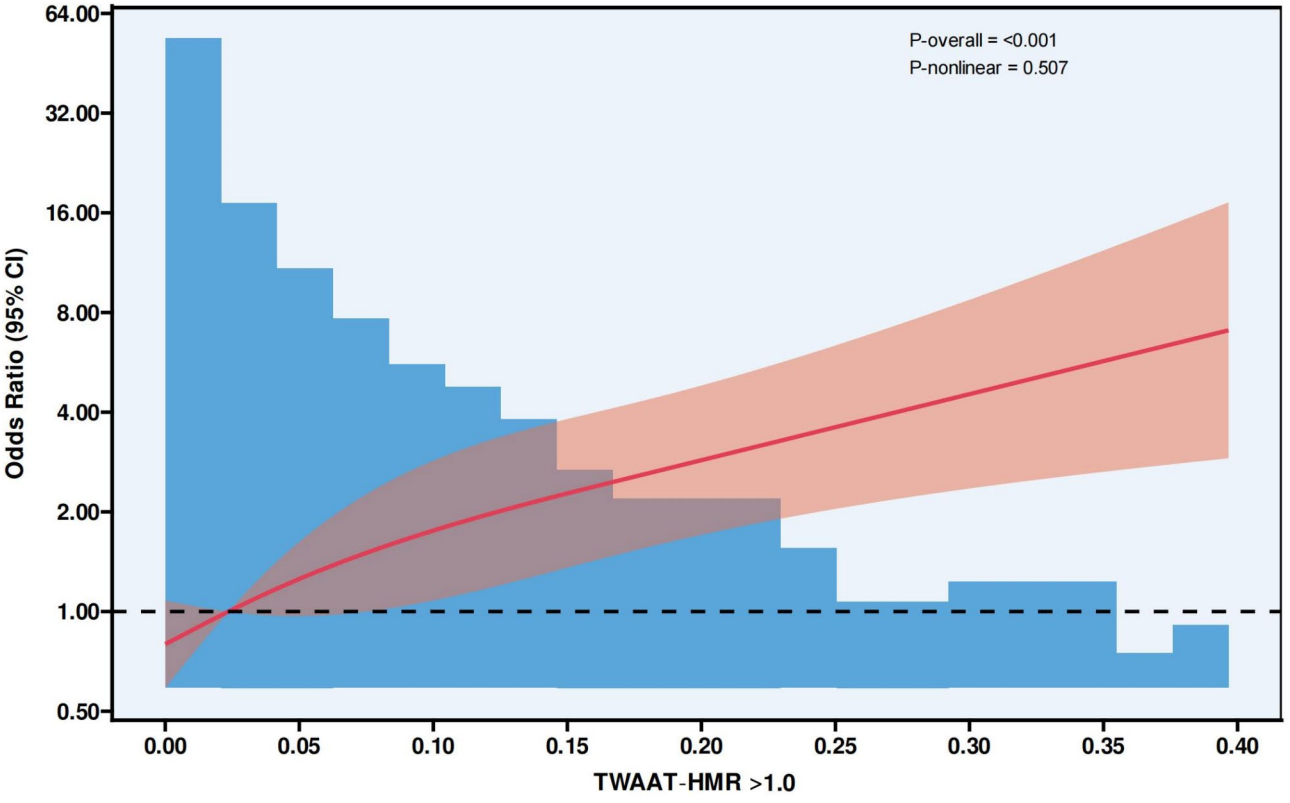

Fig. 2 RCS of the time-weighted area above HMR threshold (1.0) and early MINS. The odds ratios (OR) for MINS are adjusted for age, gender, BMI ≥ 24 kg/m2, COPD, CHF, preoperative eGFR, emergency, intraoperative blood loss, intraoperative RBC infusion. The red line represents OR. The light-red area represents a 95% confidence interval. RCS: restricted cubic spline; MINS: Myocardial Injury after Noncardiac surgery; HMR: heart rate to mean arterial pressure ratio; BMI: Body Mass Index; COPD: Chronic Obstructive Pulmonary Disease; CHF: Congestive Heart Failure; eGFR: estimated Glomerular Filtration Rate; RBC: Red Blood Cell

The severity and duration of IOH are critical factors in MINS development [26]. One study found that the association between low SBP and MINS was amplified when HR increased and attenuated when HR decreased during surgery [23]. However, the role of HR in MINS remains controversial. An intraoperative HR ≥ 100 bpm for at least 30 min has been associated with myocardial ischemia [27], whereas another study found no significant association between the intraoperative area above an HR threshold of 80–100 bpm and MINS [28]. Notably, previous studies have primarily investigated IOH and tachycardia separately, without considering their concurrent

Variables	n (%)	OR (95% CI)	P	P for interaction
All patients	699 (100.00)	1.71 (1.35 ~ 2.17)	<0.001	
Age (years)				0.846
< 65	360 (51.50)	1.62 (1.16 ~ 2.28)	0.005	
≥ 65	339 (48.50)	2.01 (1.37 ~ 2.93)	<0.001	
Gender				0.533
Woman	271 (38.77)	2.16 (1.32 ~ 3.53)	0.002	
Man	428 (61.23)	1.64 (1.24 ~ 2.16)	<0.001	
BMI (kg/m^2)				0.164
< 24	500 (71.53)	1.66 (1.30 ~ 2.14)	<0.001	
≥ 24	199 (28.47)	2.57 (1.14 ~ 5.79)	0.023	
Hypertension				0.317
Yes	143 (20.46)	1.45 (0.88 ~ 2.40)	0.143	
No	556 (79.54)	1.79 (1.36 ~ 2.35)	<0.001	

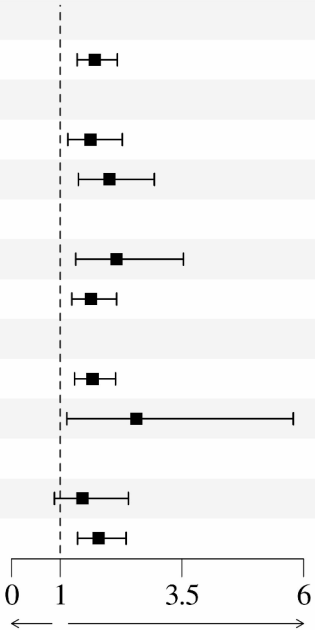

Fig. 3 Post-hoc subgroup analysis. OR: odds ratios; CI: confidence interval; BMI: body mass index

effects. Given that HR's impact on MINS may depend on concurrent MBP status, our study is the first to explicitly quantify the combined impact of HR and MBP through the HMR index, which may better reflect real-world intraoperative hemodynamic interactions. A mismatch between MBP and HR may negatively affect myocardial oxygen supply-demand balance through a synergistic interaction. Prolonged periods of high HMR, indicating sustained imbalances between myocardial oxygen supply and demand, suggest that the duration of elevated HMR plays a crucial role in MINS development. Both the magnitude and duration of HMR elevation contribute to MINS risk, highlighting the importance of intraoperative HMR monitoring. Accordingly, we propose the TWAAT-HMR threshold of 1.0, which quantifies the cumulative burden of HMR elevation in terms of both magnitude and duration. Furthermore, our sensitivity and subgroup analyses were consistent with the primary findings, reinforcing the robustness of our results. However, the subgroup analysis results should be interpreted with caution due to the limited sample size.

The rationale for proposing HMR as a risk indicator for MINS is based on the myocardial oxygen supply-demand balance theory. First, regarding oxygen supply, coronary perfusion pressure is dependent on mean arterial pressure (MAP). A MAP < 65 mmHg reduces coronary perfusion, exacerbating the supply-demand mismatc [16, 26]. IOH is associated with an increased risk of myocardial injury, potentially due to reduced coronary perfusion pressure and ischemia/reperfusion injury. However, a definitive causal relationship remains unproven, as highlighted by a recent meta-analysis of randomized controlled trials (RCTs) [29]. Second, regarding oxygen demand, HR elevation exponentially increases myocardial oxygen consumption by heightening cardiac workload and shortening diastolic filling time, the latter of which may restrict subendocardial perfusion [30]. Tachycardia in the presence of fixed coronary stenosis induces subendocardial necrosis, mirroring perioperative myocardial injury patterns [31]. An elevated HMR—whether driven by tachycardia, hypotension, or both—creates a scenario of stress-induced ischemia, aligning with the pathophysiology of type 2 myocardial infarction, which results from a supply-demand mismatch rather than plaque rupture [25]. The independent association between elevated HMR and early MINS underscores this mechanistic framework and provides novel insights for optimizing intraoperative hemodynamic management.

While HMR offers a novel approach to quantifying the supply-demand balance, its clinical application must consider broader hemodynamic goals, including vital organ perfusion. Although our study did not define a specific MBP range, appropriate MBP and HR targets can be inferred from existing research. MINS has been associated with an SBP > 160 mmHg and an absolute MBP ≤ 65 mmHg (or a relative decrease of ~ 30% from baseline) [23, 26]. Our findings indicate that groups with an HMR ≤ 1.3 had a median MBP above 62 mmHg and a median HR below 93 bpm. An intraoperative HR > 100 bpm has been linked to MINS, whereas maintaining an HR < 55 bpm for prolonged periods may reduce the risk [23]. A plausible hemodynamic target during GA may be SBP < 160

mmHg, MBP ≥ 65 mmHg (or a relative decrease of ≤ 30% from baseline), and HR ≤ 90 bpm, ensuring adequate perfusion of the heart, kidneys, liver, and brain. More stringent HR control may be required for patients with coronary artery disease, obstructive hypertrophic cardiomyopathy, and aortic valve stenosis. However, strict HR control is not recommended for heart failure patients who rely on a higher HR to maintain cardiac output. Nevertheless, these targets are based on previous studies and should not be rigidly applied. Hemodynamic management strategies based on HMR should focus on protective hemodynamic strategies rather than fixed BP and HR thresholds [32]. The protective hemodynamic strategy emphasizes individualized BP targets, minimizing catecholamine load, optimizing fluid balance, and addressing underlying conditions, all of which align with the HMR concept proposed in this study [32]. By quantifying the relationship between HR and MBP, HMR provides a new perspective for MINS risk stratification, while a flexible, individualized approach to hemodynamic management ensures optimal organ perfusion during surgery. Integrating HMR-based quantitative assessment with personalized protective hemodynamic strategies may improve MINS management and optimize perioperative outcomes. This individualized approach not only aligns with long-term protective strategies but also reduces the risk of over-reliance on single hemodynamic parameters in traditional management.

The clinical value of HMR lies in its ability to integrate myocardial oxygen supply and demand, overcoming the limitations of traditional isolated parameters (HR and MBP) in detecting early imbalances and refining risk stratification. Rational HMR management may help prevent MINS through optimized fluid and blood infusion management, anesthesia depth adjustment, and appropriate use of vasoactive agents. However, individualized patient conditions must be considered rather than relying solely on a single index. HMR serves as a practical and accessible indicator, particularly beneficial for primary healthcare facilities with limited cardiac monitoring capabilities. Future RCTs are essential to validate causality, define population-specific HMR thresholds, and assess protocolized HMR-guided interventions, translating this theoretical framework into clinical practice.

Limitations

This study's retrospective design precludes causal inference. While an independent association between high TWAAT-HMR and an increased risk of MINS was observed, potential residual confounding factors may contribute to this relationship [33]. To minimize external influences, we included only individuals with cTn measurements on the first postoperative day. Additionally, the commonly used anesthesia system's 5-minute data storage interval may fail to capture transient hemodynamic fluctuations. This study did not validate the clinical utility of HMR in intraoperative hemodynamic management during coronary artery bypass grafting (CABG). Given the unclear relationship between HR parameters [34] and post-CABG recovery quality, along with HMR's potential benefits, well-designed RCTs are necessary to establish a causal relationship between HMR and myocardial injury (including cardiac and noncardiac surgeries) and to develop a feasible protocol for HMR-guided hemodynamic management.

Conclusion

Elevated intraoperative HMR is independently associated with an increased risk of early MINS. HMR serves as an effective and practical tool for risk stratification in MINS. Future high-quality RCTs are needed to confirm causality, establish context-specific HMR thresholds, and evaluate HMR-guided strategies to optimize postoperative outcomes.

Abbreviations

MINS	Myocardial Injury after Noncardiac Surgery
CHD	Coronary Heart Disease
IOH	Intraoperative Hypotension
cTn	Cardiac Troponin
HR	Heart Rate
SBP	Systolic Blood Pressure
MBP	Mean Blood Pressure
HMR	HR to MBP Ratio
TWAAT-HMR threshold	Time-Weighted Area Above the HMR Threshold
GA	General Anesthesia
IRB	Institutional Review Board
BMI	Body Mass Index
Hb	Hemoglobin
eGFR	Estimated Glomerular Filtration Rate
ASA	American Society of Anesthesiologists
ROC	Receiver Operating Characteristic
OR	Odds Ratio
RCS	Restricted Cubic Spline
COPD	Chronic Obstructive Pulmonary Disease
PVD	Peripheral Vascular Disease
CHF	Congestive Heart Failure
RCT	Randomized Controlled Trial
CABG	Coronary Artery Bypass Grafting

Supplementary Information

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Supplementary Material 1

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Author contributions

Yuanjun Zhou: study design, data collection and examination, data analysis, and manuscript drafting, manuscript revision; Weiming Chen and Fei Liang: data examination and analysis, manuscript drafting, manuscript revision; Liping Zhong: data examination and data analysis; Yilin Liao: data examination and analysis and the supervision of the study process, manuscript revision; Yuting Zhong: study design, data collection, and examination, data analysis,

manuscript drafting, manuscript revision and supervision of the study process. All authors revised the manuscript and approved the submission.

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Data availability
The corresponding author can grant data access to this study upon request.

Declarations

Ethics approval and consent to participate
This retrospective study was conducted at Meizhou People's Hospital and approved by its Institutional Review Board (IRB) (Ethics No. 2023-C-93). Given the study's retrospective design, the IRB waived the requirement for informed consent. The study was registered in the Chinese Clinical Trial Registry on March 18, 2024 (ChiCTR2400081991) and conducted in accordance with the principles of the Declaration of Helsinki. The report follows the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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