

Association of Cumulative Intraoperative Hypotension Burden with High-Sensitivity Troponin-Defined Myocardial Injury after Major Non-Cardiac Surgery: A Prospective Cohort Study

S. Prasanth¹, Anupam Patel², Priyank Preyas³

¹Assistant Professor, Department of General Medicine, Dhanalakshmi Srinivasan Institute of Medical Sciences and Hospital, Perambalur, Tamil Nadu, India

²Senior Resident, Department of Obstetrics and Gynaecology, Government Medical College, Surat, Gujarat, India

³Junior Resident, Department of Anaesthesiology, Aarupadai Veedu Medical College and Hospital, Vinayaka Missions Research Foundation (VMRF-DU), Puducherry, India

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Corresponding author: Dr. Priyank Preyas

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Abstract

Background: Myocardial injury after non-cardiac surgery (MINS) is an important perioperative cardiovascular complication and is frequently clinically silent. Intraoperative hypotension has been implicated as a potentially modifiable contributor to perioperative myocardial injury.

Objectives: To evaluate the association between intraoperative hypotension burden and postoperative MINS, to assess the relationship between the severity and duration of hypotension and myocardial injury, and to determine the discriminatory ability of intraoperative haemodynamic indices for predicting MINS.

Methods: This prospective observational cohort study included 300 adults undergoing major non-cardiac surgery at a tertiary care teaching hospital. Intraoperative hypotension was primarily defined as mean arterial pressure (MAP) <65 mmHg. Cumulative duration below this threshold, lowest MAP, and time-weighted average (TWA) MAP deficit were recorded. High-sensitivity cardiac troponin T was measured preoperatively and at approximately 24 and 48 hours postoperatively.

Results: MINS occurred in 59/300 participants (19.7%). Patients with MINS were older (64.5 ± 9.2 vs 59.1 ± 10.8 years; $p < 0.001$) and more frequently had coronary artery disease and higher perioperative cardiovascular risk. Any MAP <65 mmHg occurred in 72.9% of patients with MINS compared with 43.2% without MINS ($p < 0.001$). Median cumulative hypotension duration was 15.7 versus 0.0 minutes ($p < 0.001$), and MINS incidence increased from 10.5% with no hypotension to 47.9% with ≥ 20 minutes of MAP <65 mmHg. Each 0.5-mmHg increase in TWA MAP deficit independently predicted MINS (adjusted OR 2.94; 95% CI 1.81–4.79), while each additional 10 minutes below MAP 65 mmHg increased the odds by 63% (adjusted OR 1.63; 95% CI 1.32–2.01). TWA MAP deficit showed an AUC of 0.721.

Conclusion: Greater intraoperative hypotension burden was independently associated with MINS, with a clear exposure–response relationship between hypotension duration and myocardial injury.

Keywords: Intraoperative Hypotension, Myocardial Injury After Non-Cardiac Surgery, High-Sensitivity Troponin, Mean Arterial Pressure, Non-Cardiac Surgery, Perioperative Cardiovascular Risk.

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Introduction

Cardiovascular complications remain an important contributor to morbidity and mortality after major non-cardiac surgery, particularly among older patients and those with underlying cardiovascular disease. In a nationwide analysis of 9,566,277 major non-cardiac surgical hospitalisations, Smilowitz et al. identified perioperative acute myocardial infarction in 84,093 patients (0.88%); affected patients had an in-hospital mortality of

18.0% compared with 1.5% among those without myocardial infarction.[1] The clinical burden of perioperative myocardial damage extends beyond conventionally diagnosed myocardial infarction. Park and Lee described myocardial injury after non-cardiac surgery (MINS) as postoperative cardiac troponin elevation attributable to an ischaemic mechanism after exclusion of non-ischaemic causes and emphasised that most MINS

events occur without typical ischaemic symptoms. [2] They further highlighted myocardial oxygen supply-demand mismatch as the predominant pathophysiological mechanism, implicating haemodynamic disturbances, anaemia, hypoxaemia and tachycardia as potentially relevant perioperative contributors.[2]

Intraoperative hypotension is particularly important because it is common, objectively measurable and potentially modifiable. Walsh et al., analysing 33,330 non-cardiac surgical procedures, demonstrated a graded relationship between the duration of MAP <55 mmHg and myocardial injury; compared with patients without such hypotension, adjusted odds ratios for myocardial injury were 1.30, 1.47, 1.79 and 1.82 for exposures lasting 1–5, 6–10, 11–20 and >20 minutes, respectively.[3] Sessler et al., in 9,765 participants from the POISE-2 cohort, similarly found that each additional 10 minutes of clinically important intraoperative hypotension was associated with an 8% increase in the relative effect on myocardial infarction or 30-day mortality.[4] The clinical relevance of preserving arterial pressure is further supported by Futier et al., who reported postoperative organ dysfunction in 38.1% of high-risk patients managed with an individualised blood-pressure strategy compared with 51.7% receiving standard management (relative risk 0.73; 95% CI 0.56–0.94).[5] Collectively, these observations support evaluation of both the depth and cumulative duration of intraoperative hypotension in relation to postoperative myocardial injury. Against this background, the present study aimed to evaluate the association between intraoperative hypotension burden and postoperative myocardial injury, as assessed by high-sensitivity cardiac troponin, among patients undergoing major non-cardiac surgery. It further aimed to determine the relationship of the duration and severity of intraoperative hypotension with myocardial injury, to identify clinically relevant haemodynamic thresholds associated with increased risk, and to assess whether intraoperative hypotension independently predicted postoperative myocardial injury after adjustment for relevant perioperative cardiovascular and surgical risk factors.

Materials and Methods

This was a single-centre, hospital-based, prospective observational cohort study conducted in the Department of Anaesthesiology, in collaboration with the Department of Cardiology, at a tertiary care teaching hospital over a period of two years, from January 2025 to December 2026. The study was commenced after obtaining approval from the Institutional Human Ethics Committee (IHEC). All eligible participants were provided with a Participant Information Sheet (PIS) explaining the purpose, procedures, potential risks

and benefits of the study, and written informed consent was obtained before enrolment. Adult patients aged ≥ 18 years who underwent elective major non-cardiac surgery under general or neuraxial anaesthesia, required at least overnight postoperative hospitalisation, and for whom intraoperative haemodynamic monitoring and perioperative high-sensitivity cardiac troponin measurements could be obtained were included. Patients undergoing cardiac or major vascular surgery involving cardiopulmonary bypass, those undergoing emergency surgery, patients with acute coronary syndrome or acute myocardial infarction before surgery, those with haemodynamic instability requiring preoperative vasopressor or inotropic support, patients with sepsis or other acute conditions known to cause non-ischaemic myocardial injury at the time of enrolment were excluded from the study.

The sample size was calculated based on the study by Hallqvist et al., who reported postoperative myocardial damage, defined using high-sensitivity cardiac troponin T, in 30% of patients undergoing major elective non-cardiac surgery.[6] Assuming an expected proportion of 30%, a 95% confidence level, and an absolute precision of 5.5%, the minimum required sample size was calculated using the formula $n = Z^2 \times p \times (1-p) / d^2$, where $Z=1.96$, $p=0.30$, and $d=0.055$. Considering 10% nonresponse rate, the calculated sample size was rounded off to 300 participants; enrolled using nonprobability sampling technique – purposive sampling/complete enumeration. Data were collected prospectively for all enrolled participants using a predesigned structured case-record form. Before surgery, detailed demographic and anthropometric characteristics, including age, sex, body mass index and smoking status, were recorded together with relevant cardiovascular and systemic comorbidities. Current cardiovascular medications were documented. Preoperative functional status, American Society of Anesthesiologists physical status, Revised Cardiac Risk Index, type and cardiovascular risk category of the planned surgical procedure, and relevant baseline investigations were recorded. Laboratory evaluation included haemoglobin, serum creatinine and estimated glomerular filtration rate, and a 12-lead electrocardiogram was reviewed were clinically indicated or available as part of routine perioperative assessment. A baseline hs-cTnT/hs-cTnI was obtained before surgery to facilitate differentiation of pre-existing chronic troponin elevation from acute perioperative myocardial injury. Baseline arterial blood pressure was recorded before administration of anaesthetic or vasoactive drugs, with the patient at rest, and the average of available stable pre-induction measurements was considered the reference blood pressure. Intraoperatively, systolic blood pressure,

diastolic blood pressure, mean arterial pressure (MAP), heart rate and peripheral oxygen saturation were prospectively recorded from the anaesthesia monitoring system throughout the period from induction of anaesthesia until completion of anaesthesia. Blood pressure was measured using non-invasive oscillometric monitoring at intervals not exceeding 5 minutes or continuously using an invasive arterial catheter when placement of an arterial line was clinically indicated; the method of monitoring was documented for each participant. Intraoperative hypotension was primarily evaluated using an absolute MAP threshold of <65 mmHg. For each patient, the occurrence of hypotension, lowest recorded MAP, number of hypotensive episodes, cumulative duration below MAP 65 mmHg, and total area under the MAP 65-mmHg threshold were determined. Hypotension burden was additionally expressed as the time-weighted average MAP deficit below 65 mmHg, calculated by dividing the area under the 65-mmHg threshold by the total duration of intraoperative blood-pressure monitoring, thereby incorporating both the depth and duration of hypotension.[7] Other intraoperative variables recorded included the type and duration of anaesthesia, duration and type of surgery, estimated blood loss, intravenous crystalloid and colloid administration, blood and blood-component transfusion, urine output, episodes of hypoxaemia or significant arrhythmia, and the administration of vasopressors or inotropes, including the agent used, route of administration and cumulative dose or infusion requirement. Postoperatively, participants were clinically assessed and hs-cTn was measured at approximately 24 and 48 hours after surgery using the same laboratory assay as that used for the baseline measurement. Additional troponin measurements, electrocardiography, echocardiography or other cardiac investigations were performed when clinically indicated. Postoperative myocardial injury was identified from serial hs-cTn measurements using the assay-specific 99th-percentile upper reference limit, with the pattern and preoperative value used to distinguish acute from chronic myocardial injury. Myocardial injury after non-cardiac surgery (MINS) was adjudicated when an acute postoperative troponin elevation was considered attributable to a presumed ischaemic mechanism, irrespective of the presence of symptoms, after exclusion of an overt non-ischaemic cause such as sepsis, pulmonary embolism, acute stroke or acute decompensated heart failure.[8]

Postoperative myocardial infarction was differentiated from isolated myocardial injury in accordance with the Fourth Universal Definition of Myocardial Infarction, which required acute myocardial injury together with clinical, electrocardiographic, imaging or angiographic

evidence of myocardial ischaemia. Postoperative variables potentially influencing myocardial injury, including hypotension, hypoxaemia, significant anaemia, major bleeding or transfusion, tachyarrhythmia, sepsis and renal dysfunction, were also documented.

Statistical Analysis: Statistical analysis was performed using IBM SPSS Statistics for Windows, version 28.0 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality using the Shapiro–Wilk test and inspection of histograms and Q–Q plots. Normally distributed continuous variables were expressed as mean±standard deviation and compared using the independent-samples Student's t-test, whereas non-normally distributed variables were presented as median with interquartile range and compared using the Mann–Whitney U test. Categorical variables were expressed as frequencies and percentages and compared using the chi-square test or Fisher's exact test, as appropriate. The association between intraoperative hypotension burden and MINS was evaluated using multivariable binary logistic regression, and adjusted odds ratios with 95% confidence intervals were reported. Separate regression models were constructed for time-weighted average MAP deficit and cumulative duration of MAP <65 mmHg because of collinearity between these haemodynamic exposure measures. Model discrimination was assessed using the C-statistic. Receiver operating characteristic curve analysis was performed to evaluate the ability of intraoperative hypotension parameters to discriminate patients with and without MINS, and the area under the curve with 95% confidence intervals was calculated. Optimal cut-off values were identified using the Youden index, with corresponding sensitivity and specificity reported. A two-tailed p value <0.05 was considered statistically significant.

Results

Among the 300 participants, myocardial injury after non-cardiac surgery occurred in 59 (19.7%). Patients who developed MINS were older than those without MINS (64.5±9.2 vs 59.1±10.8 years; $p<0.001$) and more frequently had coronary artery disease (25.4% vs 12.0%; $p=0.009$), an abnormal preoperative electrocardiogram (35.6% vs 13.7%; $p<0.001$), ASA physical status III/IV (71.2% vs 54.8%; $p=0.022$), and an RCRI score ≥ 2 (23.7% vs 10.0%; $p=0.005$). In contrast, the distribution of surgical specialty, anaesthetic technique, operative duration, estimated blood loss, fluid administration, transfusion and vasopressor use was broadly comparable between groups. Intraoperative hypotension was substantially more frequent and prolonged among patients who developed MINS: any MAP <65 mmHg occurred in 72.9% compared

with 43.2% of those without MINS ($p < 0.001$), while the cumulative duration below MAP 65 mmHg was 15.7 (0.0–28.2) minutes versus 0.0 (0.0–11.1) minutes, respectively ($p < 0.001$). The lowest intraoperative MAP was also lower in the MINS group (63.9 ± 5.4 vs 66.0 ± 5.8 mmHg; $p = 0.012$), and the time-weighted average MAP deficit below 65 mmHg was higher [0.24 (0.00–0.51) vs 0.00 (0.00–0.13) mmHg; $p < 0.001$]. A clear exposure-response relationship was observed, with the incidence of MINS increasing from 10.5% among patients with no exposure below MAP 65 mmHg to 15.6%, 24.1% and 47.9% among those exposed for 1–9, 10–19 and ≥ 20 minutes, respectively ($p < 0.001$).

Acute postoperative myocardial injury occurred in 65 (21.7%) participants, of whom 59 (19.7%) were adjudicated as having myocardial injury after non-cardiac surgery (MINS). Although preoperative hs-cTnT concentrations were comparable between groups, patients with MINS had markedly higher hs-cTnT concentrations at 24 hours [20.7 (16.2–27.4) vs 6.8 (4.8–9.4) ng/L] and 48 hours [15.4 (12.3–22.5) vs 6.4 (4.9–9.0) ng/L] (both $p < 0.001$).

Postoperative hypotension (44.1% vs 7.9%), significant anaemia (30.5% vs 8.3%), myocardial infarction (15.3% vs 0%), and requirement for ICU admission or continued ICU care (42.4% vs 17.8%) were also more frequent among patients with MINS, and their median hospital stay was longer [8.0 (6.0–9.0) vs 6.0 (5.0–7.0) days; $p < 0.001$].

On multivariable analysis, increasing age and greater intraoperative hypotension burden remained independently associated with MINS; each 0.5-mmHg increase in time-weighted average MAP deficit below 65 mmHg was associated with an adjusted odds ratio of 2.94 (95% CI 1.81–4.79; $p < 0.001$), while each additional 10 minutes below MAP 65 mmHg was associated with an adjusted odds ratio of 1.63 (95% CI 1.32–2.01; $p < 0.001$). The corresponding models demonstrated good discrimination, with C-statistics of 0.792 and 0.779, respectively. On ROC analysis, time-weighted MAP deficit showed an AUC of 0.721 (95% CI 0.646–0.795), with an optimal cut-off > 0.23 mmHg, whereas cumulative duration below MAP 65 mmHg yielded an AUC of 0.711 (95% CI 0.633–0.786) with an optimal cut-off > 6.4 minutes.

Table 1: Baseline clinical characteristics according to MINS status

		Overall (N=300)	No MINS (n=241)	MINS (n=59)	p value
Age (years)		60.1 $\pm$ 10.7	59.1 $\pm$ 10.8	64.5 $\pm$ 9.2	<0.001
Male sex		161 (53.7)	125 (51.9)	36 (61.0)	0.207
Body mass index (kg/m ²)		25.8 $\pm$ 3.9	25.8 $\pm$ 3.9	25.8 $\pm$ 4.1	0.976
Current smoking		58 (19.3)	47 (19.5)	11 (18.6)	0.881
Hypertension		145 (48.3)	115 (47.7)	30 (50.8)	0.666
Diabetes mellitus		108 (36.0)	82 (34.0)	26 (44.1)	0.150
Dyslipidaemia		77 (25.7)	60 (24.9)	17 (28.8)	0.537
Coronary artery disease		44 (14.7)	29 (12.0)	15 (25.4)	0.009
Previous myocardial infarction		19 (6.3)	12 (5.0)	7 (11.9)	0.070
Prior PCI/CABG		25 (8.3)	16 (6.6)	9 (15.3)	0.061
Heart failure		17 (5.7)	14 (5.8)	3 (5.1)	1.000
Cerebrovascular disease		10 (3.3)	5 (2.1)	5 (8.5)	0.028
Peripheral arterial disease		9 (3.0)	8 (3.3)	1 (1.7)	1.000
Chronic kidney disease		33 (11.0)	27 (11.2)	6 (10.2)	0.820
Cardiovascular medications	Beta-blocker	58 (19.3)	41 (17.0)	17 (28.8)	0.040
	Renin-angiotensin system inhibitor	105 (35.0)	83 (34.4)	22 (37.3)	0.681
	Calcium-channel blocker	66 (22.0)	52 (21.6)	14 (23.7)	0.721
	Diuretic	42 (14.0)	30 (12.4)	12 (20.3)	0.117
	Statin	91 (30.3)	69 (28.6)	22 (37.3)	0.195
	Antiplatelet agent	46 (15.3)	31 (12.9)	15 (25.4)	0.016
	Anticoagulant	12 (4.0)	10 (4.1)	2 (3.4)	1.000
Reduced preoperative functional capacity		41 (13.7)	30 (12.4)	11 (18.6)	0.214
ASA physical status III/IV		174 (58.0)	132 (54.8)	42 (71.2)	0.022
RCRI	RCRI 0	135 (45.0)	117 (48.5)	18 (30.5)	0.005
	RCRI 1	127 (42.3)	100 (41.5)	27 (45.8)	
	RCRI ≥ 2	38 (12.7)	24 (10.0)	14 (23.7)	
Haemoglobin (g/dL)		12.4 $\pm$ 1.4	12.4 $\pm$ 1.5	12.4 $\pm$ 1.4	0.928
Serum creatinine (mg/dL)		0.86 (0.73–1.08)	0.87 (0.75–1.10)	0.84 (0.70–1.04)	0.708

eGFR (mL/min/1.73 m ²)	56.7 ± 13.6	57.3 ± 13.8	54.3 ± 12.7	0.110
Baseline hs-cTnT (ng/L)	6.2 (4.9–8.4)	6.4 (4.9–8.8)	6.2 (4.9–8.1)	0.758
Abnormal preoperative ECG	54 (18.0)	33 (13.7)	21 (35.6)	<0.001
Reference pre-induction MAP (mmHg)	97.8 ± 9.9	97.5 ± 9.7	99.2 ± 10.6	0.285

Table 2: Surgical, anaesthetic and intraoperative characteristics

		Overall	No MINS	MINS	p value
Surgical specialty	Abdominal	114 (38.0)	92 (38.2)	22 (37.3)	0.058
	Orthopaedic	82 (27.3)	70 (29.0)	12 (20.3)	
	Urologic	48 (16.0)	41 (17.0)	7 (11.9)	
	Gynaecologic	32 (10.7)	22 (9.1)	10 (16.9)	
	Thoracic	14 (4.7)	11 (4.6)	3 (5.1)	
	Other	10 (3.3)	5 (2.1)	5 (8.5)	
High-risk surgical category		86 (28.7)	64 (26.6)	22 (37.3)	0.102
Anaesthetic technique	General	242 (80.7)	194 (80.5)	48 (81.4)	0.504
	Neuraxial	33 (11.0)	25 (10.4)	8 (13.6)	
	Combined	25 (8.3)	22 (9.1)	3 (5.1)	
Surgical duration (min)		191.2 ± 73.4	194.9 ± 74.3	175.9 ± 68.3	0.062
Anaesthesia duration (min)		226.9 ± 73.9	230.6 ± 75.0	211.6 ± 67.9	0.062
Estimated blood loss (mL)		203.3 (119.5–335.7)	202.5 (119.4–334.7)	224.7 (125.4–362.1)	0.393
Crystalloid administered (mL)		2163 ± 495	2156 ± 503	2189 ± 466	0.640
Urine output (mL/kg/h)		0.73 ± 0.22	0.72 ± 0.23	0.76 ± 0.19	0.182
Invasive arterial monitoring		97 (32.3)	77 (32.0)	20 (33.9)	0.774
Colloid administration		27 (9.0)	23 (9.5)	4 (6.8)	0.506
Blood-component transfusion		25 (8.3)	20 (8.3)	5 (8.5)	1.000
Intraoperative hypoxaemia		22 (7.3)	19 (7.9)	3 (5.1)	0.586
Significant intraoperative arrhythmia		19 (6.3)	16 (6.6)	3 (5.1)	1.000
Any vasopressor/inotrope		170 (56.7)	132 (54.8)	38 (64.4)	0.181
Phenylephrine bolus use		93 (31.0)	70 (29.0)	23 (39.0)	0.139
Norepinephrine infusion use		76 (25.3)	61 (25.3)	15 (25.4)	0.986
Phenylephrine cumulative dose among recipients (µg)		213 (143–328)	213 (137–325)	242 (154–332)	0.897
Norepinephrine maximum infusion rate among recipients (µg/kg/min)		0.057 (0.041–0.078)	0.057 (0.044–0.078)	0.048 (0.040–0.084)	0.695

Table 3: Intraoperative hypotension exposure and myocardial injury

		Overall	No MINS	MINS	p value
Lowest intraoperative MAP (mmHg)		65.6 ± 5.8	66.0 ± 5.8	63.9 ± 5.4	0.012
Number of MAP <65-mmHg episodes		0.0 (0.0–2.0)	0.0 (0.0–2.0)	2.0 (0.0–3.5)	<0.001
Cumulative MAP	<65 duration (min)	0.0 (0.0–14.2)	0.0 (0.0–11.1)	15.7 (0.0–28.2)	<0.001
	<60 duration (min)	0.0 (0.0–4.8)	0.0 (0.0–0.5)	4.2 (0.0–20.9)	
	<55 duration (min)	0.0 (0.0–0.0)	0.0 (0.0–0.0)	0.0 (0.0–10.4)	
Area under MAP 65 threshold (mmHg.min)		0.0 (0.0–48.1)	0.0 (0.0–28.0)	62.6 (0.0–124.2)	<0.001
Time-weighted average MAP deficit <65 (mmHg)		0.00 (0.00–0.21)	0.00 (0.00–0.13)	0.24 (0.00–0.51)	<0.001
Any MAP <65 mmHg		147 (49.0)	104 (43.2)	43 (72.9)	<0.001
MAP reduction >20% from baseline		277 (92.3)	221 (91.7)	56 (94.9)	0.586
Marked SBP reduction >50% from baseline		12 (4.0)	8 (3.3)	4 (6.8)	0.261
MINS incidence by cumulative MAP <65 duration	0 min	16/153 (10.5%)	—	—	<0.001
	1–9 min	7/45 (15.6%)	—	—	
	10–19 min	13/54 (24.1%)	—	—	
	≥20 min	23/48 (47.9%)	—	—	

Table 4: Serial troponin measurements and postoperative clinical outcomes

	Overall	No MINS	MINS	p value
Preoperative hs-cTnT (ng/L)	6.2 (4.9–8.4)	6.4 (4.9–8.8)	6.2 (4.9–8.1)	0.758
24-hour hs-cTnT (ng/L)	8.0 (5.2–12.5)	6.8 (4.8–9.4)	20.7 (16.2–27.4)	<0.001
48-hour hs-cTnT (ng/L)	7.2 (5.1–11.4)	6.4 (4.9–9.0)	15.4 (12.3–22.5)	<0.001
Peak postoperative hs-cTnT (ng/L)	8.5 (5.8–14.0)	7.0 (5.4–9.9)	22.4 (16.6–30.4)	<0.001
Postoperative hypotension	45 (15.0)	19 (7.9)	26 (44.1)	<0.001
Postoperative hypoxaemia	25 (8.3)	15 (6.2)	10 (16.9)	0.015
Significant postoperative anaemia	38 (12.7)	20 (8.3)	18 (30.5)	<0.001
Major bleeding	17 (5.7)	8 (3.3)	9 (15.3)	0.002
Tachyarrhythmia	16 (5.3)	8 (3.3)	8 (13.6)	0.005
Sepsis	6 (2.0)	6 (2.5)	0 (0.0)	0.602
Postoperative renal dysfunction	27 (9.0)	18 (7.5)	9 (15.3)	0.061
Postoperative myocardial infarction	9 (3.0)	0 (0.0)	9 (15.3)	<0.001
Ischaemic symptoms	12 (4.0)	0 (0.0)	12 (20.3)	<0.001
Ischaemic ECG changes	14 (4.7)	0 (0.0)	14 (23.7)	<0.001
ICU admission/continued ICU care	68 (22.7)	43 (17.8)	25 (42.4)	<0.001
Hospital length of stay (days)	6.0 (5.0–8.0)	6.0 (5.0–7.0)	8.0 (6.0–9.0)	<0.001
Acute postoperative myocardial injury occurred in 65 (21.7%) participants; 59 (19.7%) were adjudicated as MINS and 6 (2.0%) as non-ischaemic injury.				

Table 5: Multivariable logistic regression for predictors of MINS

Predictor	Adjusted OR	95% CI	p value
Model A: time-weighted hypotension burden			
Age, per 10 years	1.53	1.11–2.10	0.009
Coronary artery disease	1.78	0.72–4.40	0.209
RCRI ≥ 2	2.10	0.81–5.47	0.129
High-risk surgery	1.45	0.71–2.98	0.312
TWA deficit, per 0.5 mmHg	2.94	1.81–4.79	<0.001
Model B: cumulative duration below MAP 65 mmHg			
Age, per 10 years	1.52	1.12–2.07	0.008
Coronary artery disease	1.79	0.73–4.40	0.203
RCRI ≥ 2	1.99	0.76–5.19	0.159
High-risk surgery	1.46	0.72–2.97	0.290
MAP <65 duration, per 10 min	1.63	1.32–2.01	<0.001
Model A C-statistic = 0.792; Model B C-statistic = 0.779. Separate models were fitted for time-weighted deficit and cumulative hypotension duration because these measures capture overlapping haemodynamic exposure and were not entered simultaneously.			

Table 6: Discrimination of intraoperative hypotension measures for MINS

	AUC	95% CI	Optimal cut-off	Sensitivity (%)	Specificity (%)
Cumulative duration MAP <65 mmHg (min)	0.711	0.633–0.786	>6.4	71.2	68.0
Time-weighted average MAP deficit <65 mmHg (mmHg)	0.721	0.646–0.795	>0.23	52.5	85.1
Lowest intraoperative MAP (mmHg)	0.626	0.550–0.703	<64.8	55.9	72.6

Discussion

The present study demonstrated that myocardial injury after non-cardiac surgery was a relatively frequent perioperative cardiovascular complication, occurring in 59 of 300 participants (19.7%). The American Heart Association scientific statement by Ruetzler et al. reported that MINS occurs in approximately 20% of adults undergoing major inpatient non-cardiac surgery.[8] Similarly, the VISION investigators, in a prospective international cohort of 21,842 patients, identified

MINS in 3,904 patients (17.9%).[9] Puelacher et al., using systematic perioperative hs-cTnT surveillance in 2,546 non-cardiac surgical procedures, detected perioperative myocardial injury after 397 procedures (16%). [10]

These findings support the observed incidence in the present cohort and emphasise that myocardial injury constitutes a considerably larger perioperative burden than clinically apparent myocardial infarction alone. Patients who developed MINS in the present study were

significantly older than those without MINS (64.5 ± 9.2 vs 59.1 ± 10.8 years), and had a greater prevalence of coronary artery disease (25.4% vs 12.0%), abnormal preoperative electrocardiographic findings (35.6% vs 13.7%), ASA physical status III/IV (71.2% vs 54.8%), and an RCRI score ≥ 2 (23.7% vs 10.0%). These observations indicate that underlying cardiovascular substrate and overall systemic disease burden influence vulnerability to perioperative myocardial injury. Age and established cardiovascular disease are sufficiently important predictors that the 2022 ESC guideline recommends perioperative hs-cTn measurement before and at 24 and 48 hours after intermediate- or high-risk surgery in patients aged ≥ 65 years or those with established cardiovascular disease or cardiovascular risk factors.[7] Duceppe et al. similarly incorporated age ≥ 65 years, significant cardiovascular disease and RCRI ≥ 1 into the Canadian Cardiovascular Society criteria for postoperative troponin surveillance.[11]

The principal finding of the present study was the strong association between intraoperative hypotension burden and MINS. Exposure to any MAP < 65 mmHg occurred in 72.9% of patients with MINS compared with 43.2% of patients without MINS, while cumulative exposure below 65 mmHg was substantially longer [15.7 (0.0–28.2) vs 0.0 (0.0–11.1) minutes]. Salmasi et al. similarly demonstrated that MAP values below an absolute threshold of approximately 65 mmHg were progressively associated with both myocardial and renal injury, and that the odds of organ injury increased as the duration of hypotension increased.[12] Importantly, they found that relative reductions from baseline did not provide stronger discrimination than absolute MAP thresholds, supporting the use of MAP < 65 mmHg as the primary haemodynamic exposure in the present study. A particularly important observation was the graded exposure–response relationship between duration of hypotension and MINS. MINS occurred in only 10.5% of patients with no exposure below MAP 65 mmHg but increased to 15.6% with 1–9 minutes, 24.1% with 10–19 minutes, and 47.9% with ≥ 20 minutes of exposure ($p < 0.001$). This pattern is consistent with the concept that hypotension-associated organ injury is determined by both depth and duration rather than by an isolated low blood-pressure measurement. The 2024 PeriOperative Quality Initiative consensus concluded that MAP values below approximately 60–70 mmHg are associated with myocardial injury, acute kidney injury, myocardial infarction and mortality, with injury increasing according to hypotension severity and duration.[13] The 2024 AHA/ACC perioperative guideline similarly identifies a harm threshold around MAP < 65 mmHg or SBP < 90 mmHg sustained for

approximately 15 minutes and recommends maintaining intraoperative MAP at ≥ 60 –65 mmHg to reduce myocardial injury.[14] The present findings also demonstrate why quantification of hypotension burden is more informative than recording the lowest MAP alone. Although the lowest MAP was significantly lower in patients with MINS (63.9 ± 5.4 vs 66.0 ± 5.8 mmHg), the difference was relatively modest. In contrast, time-weighted average (TWA) MAP deficit below 65 mmHg differed markedly between groups [0.24 (0.00–0.51) vs 0.00 (0.00–0.13) mmHg]. Hallqvist et al. previously observed postoperative hs-cTnT elevation in 90 of 300 patients (30%) undergoing major elective non-cardiac surgery and reported that an intraoperative SBP reduction $> 50\%$ from baseline lasting > 5 minutes independently predicted myocardial damage (OR 4.4; 95% CI 1.8–11.1).[6] The present results extend this concept by demonstrating the usefulness of a continuous burden measure incorporating both magnitude and duration of pressure reduction.

Serial troponin measurements further supported an acute perioperative pattern of injury. Baseline hs-cTnT values were virtually identical between MINS and non-MINS groups, whereas concentrations diverged substantially postoperatively, reaching 20.7 (16.2–27.4) versus 6.8 (4.8–9.4) ng/L at 24 hours and 15.4 (12.3–22.5) versus 6.4 (4.9–9.0) ng/L at 48 hours. The VISION investigators demonstrated the prognostic significance of such postoperative hs-cTnT elevations: among 21,842 patients, peak postoperative hs-cTnT concentrations of 20 to < 65 ng/L were associated with 3.0% 30-day mortality, compared with 0.5% when hs-cTnT was < 20 ng/L.[15] Furthermore, 93.1% of the 3,904 patients with MINS in VISION had no ischaemic symptoms, demonstrating why systematic biomarker surveillance is necessary. The distinction between myocardial injury and overt myocardial infarction was similarly apparent in the present study. Although 65 participants (21.7%) developed acute postoperative myocardial injury and 59 (19.7%) fulfilled criteria for MINS, only nine patients, corresponding to 15.3% of the MINS group, fulfilled criteria for postoperative myocardial infarction. Thus, most MINS events would not have been represented by conventional myocardial infarction endpoints. Devereaux et al. previously reported that 58.2% of patients with MINS did not fulfil the universal definition of myocardial infarction, while only 15.8% experienced an ischaemic symptom.[9] Puelacher et al. likewise found typical chest pain in only 6% and any ischaemic symptoms in 18% of patients with perioperative myocardial injury.[10] Postoperative physiological disturbances were also concentrated among patients with MINS. Postoperative hypotension occurred in 44.1%

compared with 7.9% of patients without MINS, while significant postoperative anaemia occurred in 30.5% versus 8.3%. These findings are biologically consistent with myocardial oxygen supply–demand imbalance, in which reduced coronary perfusion pressure and diminished oxygen-carrying capacity may act synergistically in susceptible myocardium. In 1,710 patients aged ≥ 60 years undergoing intermediate- or high-risk non-cardiac surgery, Liem et al. found that postoperative MAP < 65 mmHg lasting more than four hours was associated with myocardial injury (adjusted OR 2.98; 95% CI 1.78–4.98).[16] The 2024 AHA/ACC guideline also recognises postoperative anaemia as a contributor to myocardial ischaemia and type 2 myocardial infarction through impaired tissue oxygen delivery.[14, 17] The multivariable findings strengthen the relationship between cumulative haemodynamic exposure and myocardial injury. After adjustment, each 0.5-mmHg increase in TWA MAP deficit below 65 mmHg was associated with nearly a threefold increase in the odds of MINS (adjusted OR 2.94; 95% CI 1.81–4.79), while every additional 10 minutes below MAP 65 mmHg increased the odds by 63% (adjusted OR 1.63; 95% CI 1.32–2.01). Age also remained independently associated with MINS, whereas coronary artery disease, RCRI ≥ 2 and high-risk surgery did not retain statistical significance after adjustment. This suggests that dynamic intraoperative haemodynamic exposure provided information beyond conventional preoperative risk markers in this cohort. The absence of major between-group differences in anaesthetic technique, surgical duration, estimated blood loss, transfusion and overall vasopressor use further supports hypotension burden itself as the principal intraoperative discriminator.[18]

Finally, ROC analysis demonstrated moderate discriminatory performance for cumulative hypotension measures. TWA MAP deficit yielded an AUC of 0.721 (95% CI 0.646–0.795), with a threshold > 0.23 mmHg providing 52.5% sensitivity and 85.1% specificity, whereas cumulative MAP < 65 -mmHg duration produced an AUC of 0.711 (95% CI 0.633–0.786), with an optimal threshold > 6.4 minutes yielding 71.2% sensitivity and 68.0% specificity. The higher specificity of TWA deficit indicates that a measure integrating depth and duration may identify a smaller subgroup with more substantial haemodynamic exposure, whereas cumulative duration may be more sensitive for screening. The corresponding multivariable C-statistics of 0.792 and 0.779 further suggest that combining hypotension burden with baseline clinical variables offers better discrimination than any single blood-pressure parameter alone, consistent with recommendations to evaluate perioperative cardiovascular risk as an interaction

between patient vulnerability and perioperative physiological stress.

The present study had several limitations. First, it was conducted at a single tertiary care teaching hospital, which may limit the generalisability of the findings to institutions with different patient populations, surgical case-mix, perioperative protocols, and haemodynamic management practices. Second, as an observational study, it could demonstrate associations between intraoperative hypotension burden and myocardial injury after non-cardiac surgery but could not establish a causal relationship, and residual confounding from unmeasured perioperative factors may have persisted despite multivariable adjustment. Third, although serial high-sensitivity cardiac troponin measurements were obtained preoperatively and at approximately 24 and 48 hours postoperatively, myocardial injury occurring outside this surveillance window could have been missed. Fifth, the use of an absolute MAP threshold of < 65 mmHg, although clinically relevant, may not fully account for individual differences in baseline blood pressure, chronic hypertension, or autoregulatory reserve.

Conclusion

The present study demonstrated that myocardial injury after non-cardiac surgery was a frequent postoperative cardiovascular complication, occurring in nearly one-fifth of patients undergoing major non-cardiac surgery. Greater intraoperative hypotension burden, particularly longer cumulative exposure below a mean arterial pressure of 65 mmHg and a higher time-weighted MAP deficit, was independently associated with MINS even after adjustment for relevant preoperative cardiovascular risk factors. A clear exposure–response relationship was observed, with progressively higher rates of MINS as the duration of hypotension increased. Patients with MINS also showed greater postoperative troponin elevation, a higher frequency of postoperative hypotension, anaemia, myocardial infarction and ICU requirement, and a longer hospital stay. These findings support the importance of careful intraoperative blood-pressure surveillance and minimisation of both the depth and duration of hypotension in patients undergoing major non-cardiac surgery, while also highlighting the value of perioperative high-sensitivity cardiac troponin surveillance for detecting otherwise clinically silent myocardial injury.

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