Intraoperative Hypotension in Non-Cardiac Surgery: Pathophysiology, Monitoring and Individualized Management

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Abstract. *Intraoperative hypotension (IOH) is one of the most frequent haemodynamic disturbances in anaesthetic practice and, depending on its depth and duration, has been associated with acute kidney injury, myocardial injury, stroke and mortality after non-cardiac surgery. Nevertheless, a single pressure threshold does not carry the same risk for every patient, and the causal contribution of hypotension to organ injury remains incompletely defined. The aim of this narrative review was to synthesise current evidence on the definition, mechanisms, monitoring and management of IOH and to translate it into a practical, cause-directed approach. Observational cohorts, randomised controlled trials, systematic reviews and international consensus statements, published predominantly between 2005 and 2025, were examined. The evidence indicates that the risk of organ injury rises progressively when mean arterial pressure (MAP) falls below approximately 65 mmHg or more than 20–30 % below baseline, and that cumulative exposure (depth × duration) is more informative than any single reading. An individualised strategy targeting systolic pressure within 10 % of baseline reduced postoperative organ dysfunction in high-risk patients in one randomised trial, whereas a hypotension-avoidance strategy applied to a broad population did not improve vascular outcomes. Effective management combines accurate measurement, rapid identification of the dominant mechanism (vasodilation, hypovolaemia, reduced contractility, arrhythmia or obstruction) and targeted therapy with fluids, vasopressors or inotropes, followed by reassessment. Minimising the depth and duration of hypotension, rather than chasing a universal number, should be the guiding principle of perioperative blood-pressure management.*

Keywords: *intraoperative hypotension; mean arterial pressure; anaesthesia; haemodynamic monitoring; vasopressors; organ perfusion; perioperative safety*

1. Introduction

A substantial proportion of patients undergoing non-cardiac surgery experience at least one episode of arterial hypotension during anaesthesia, after induction, after neuraxial block, during haemorrhage or in response to surgical manipulation and positioning. Although most episodes are brief, their cumulative burden can be considerable, particularly in elderly patients and in those with cardiovascular or renal disease.

Interest in intraoperative hypotension (IOH) has grown since large observational studies demonstrated a graded association between low intraoperative mean arterial pressure (MAP) and postoperative acute kidney injury (AKI), myocardial injury after non-cardiac surgery (MINS) and mortality (Monk et al., 2005; Walsh et al., 2013; Salmasi et al., 2017).

A systematic review subsequently confirmed that the risk of organ injury increases with both the depth and the duration of hypotension (Wesselink et al., 2018). These findings have shaped international consensus statements that recommend avoiding prolonged MAP values below 60–65 mmHg in adults undergoing non-cardiac surgery (Sessler et al., 2019; Saugel et al., 2024).

However, association does not establish causation. Patients who become hypotensive are often older, sicker and exposed to more extensive surgery, and the mechanism that produces hypotension, such as sepsis, bleeding or heart failure, may itself be harmful. Moreover, there is no universally accepted definition of IOH. Consequently, clinicians face two practical questions: what blood-pressure level is safe for an individual patient, and how should hypotension be treated once it occurs?

The objectives of this review were therefore (i) to summarise current definitions of IOH and the evidence linking it with postoperative organ injury; (ii) to describe the principal pathophysiological mechanisms; (iii) to discuss the strengths and limitations of blood-pressure monitoring methods; (iv) to evaluate the evidence for individualised blood-pressure targets; and (v) to propose a practical, cause-directed algorithm for prevention and treatment of IOH.

2. Materials and Methods

This article was prepared as a narrative literature review. Relevant publications were identified in PubMed/MEDLINE, Scopus and Google Scholar using combinations of the terms "intraoperative hypotension", "mean arterial pressure", "non-cardiac surgery", "acute kidney injury", "myocardial injury", "vasopressor", "individualized blood pressure" and "haemodynamic monitoring".

Priority was given to large observational cohorts that quantified the relationship between intraoperative blood pressure and postoperative outcomes, randomised controlled trials of blood-pressure management strategies, systematic reviews and meta-analyses, and consensus statements of the Perioperative Quality Initiative (POQI). Articles published predominantly between 2005 and 2025 in English were considered; studies limited to cardiac surgery, obstetric anaesthesia or paediatric populations were not the focus of this review.

Findings were synthesised thematically (definition, pathophysiology, monitoring, targets and treatment). Because the review was not designed as a systematic review or meta-analysis, no PRISMA selection flow, risk-of-bias scoring or pooled statistical analysis is presented, and the conclusions should be interpreted in this context.

3. Results

3.1. Definition, incidence and association with outcomes

IOH is most frequently defined either by an absolute threshold, typically MAP < 65 mmHg or systolic blood pressure (SBP) < 90 mmHg, or by a relative decrease of 20–30 % from the patient's preoperative baseline. Depending on the definition chosen, reported incidence varies from less than 10 % to more than 90 % of anaesthetics, which illustrates how strongly the threshold determines the apparent burden of the problem.

In a retrospective cohort of more than 33 000 non-cardiac surgical patients, Walsh et al. (2013) found that the risk of AKI and myocardial injury increased when MAP remained below 55 mmHg, and that even short exposures of a few minutes were associated with adverse outcomes. Salmasi et al. (2017) analysed more than 57 000 patients and showed that MAP below 65 mmHg was associated with AKI and myocardial injury, and that relative thresholds did not predict outcomes better than absolute values. The key studies are summarised in Table 1.

Table 1. Selected studies linking intraoperative blood pressure with postoperative outcomes after non-cardiac surgery.

Study	Design and population	Exposure / intervention	Principal finding
Monk et al., 2005	Prospective cohort, 1 064 adults	Intraoperative systolic hypotension, anaesthetic depth	Cumulative systolic hypotension independently associated with 1-year mortality
Walsh et al., 2013	Retrospective cohort, 33 330 patients	Minutes with MAP below 55–75 mmHg	Risk of AKI and myocardial injury rose when MAP < 55 mmHg, even for short periods
Salmasi et al., 2017	Retrospective cohort, 57 315 patients	Absolute (MAP < 65 mmHg) vs relative thresholds	MAP < 65 mmHg associated with AKI and myocardial injury; relative thresholds not superior
Futier et al., 2017 (INPRESS)	RCT, 298 high-risk patients, major abdominal surgery	SBP within 10 % of baseline vs standard care	Individualised management reduced postoperative organ dysfunction (38.1 % vs 51.7 %)
Wesselink et al., 2018	Systematic review, 42 studies	Various definitions of IOH	Risk of organ injury rises when MAP < 80 mmHg for > 10 min and increases further with lower and longer exposure (e.g. MAP < 65 mmHg for > 5 min)
Wijnberge et al., 2020 (HYPE)	RCT, 68 patients	Machine-learning early-warning system	Median time-weighted average of hypotension reduced; clinical outcomes not powered
Marcucci et al., 2023 (POISE-3)	RCT, 7 490 patients	Hypotension-avoidance vs hypertension-avoidance antihypertensive strategy	No significant difference in composite vascular outcomes

These data consistently indicate a dose–response relationship: the lower the pressure and the longer it persists, the greater the risk. For this reason, metrics such as area under the threshold or time-weighted average MAP below 65 mmHg are increasingly used in research, as they integrate both depth and duration (Wesselink et al., 2018).

3.2. Pathophysiological mechanisms

Arterial pressure is the product of cardiac output and systemic vascular resistance; hypotension therefore reflects vasodilation, reduced preload, impaired contractility, inappropriate heart rate or rhythm, or obstruction to flow. Intravenous and inhalational anaesthetics reduce sympathetic tone, cause arterial and venous dilatation and, at higher concentrations, depress myocardial contractility. Venodilation reduces venous return, a mechanism amplified in patients who are hypovolaemic after prolonged fasting, bowel preparation or diuretic therapy.

Positive-pressure ventilation further impedes venous return. Neuraxial anaesthesia produces sympathectomy proportional to the height of the block, and high blocks may also cause bradycardia through blockade of cardio-accelerator fibres. Surgical factors, including haemorrhage, compression of the inferior vena cava, pneumoperitoneum, reverse Trendelenburg or prone positioning and manipulation of the heart or great vessels, can cause sudden falls in pressure.

Chronic antihypertensive therapy modifies the haemodynamic response to anaesthesia. Angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers attenuate the renin–angiotensin response to vasodilation and are associated with post-induction hypotension that may respond poorly to conventional vasopressors. In practice, several mechanisms often coexist, so the cause must be identified before treatment is chosen.

3.3. Monitoring and measurement limitations

Non-invasive oscillometric cuff measurement, usually performed every 3–5 minutes, is the standard for most patients. Its intermittent nature means that rapid changes after induction may be missed, and accuracy depends on correct cuff size and position; oscillometric devices also tend to overestimate low pressures.

Invasive arterial monitoring provides continuous beat-to-beat pressure and allows arterial blood sampling and analysis of the pressure waveform. It is indicated in patients

with significant cardiovascular disease, anticipated rapid blood loss, use of vasoactive infusions or major surgery. However, the transducer must be zeroed and levelled at the phlebostatic axis, and over-damped or under-damped waveforms can produce misleading values. Continuous non-invasive finger-cuff technologies and machine-learning algorithms that predict impending hypotension are emerging tools; in the HYPE trial, an early-warning system reduced the time-weighted average of hypotension, although the study was not powered for clinical outcomes (Wijnberge et al., 2020).

3.4. Individualised blood-pressure targets

MAP of 65 mmHg is commonly used as a practical lower threshold, but it is not a universal target. Autoregulation of cerebral and renal blood flow is shifted to higher pressures in patients with chronic hypertension, and the preoperative baseline varies widely between individuals. POQI consensus statements therefore recommend that MAP below 60–65 mmHg should be avoided and that higher targets may be considered in patients with chronic hypertension or elevated risk (Sessler et al., 2019; Saugel et al., 2024).

The INPRESS trial randomised 298 high-risk patients undergoing major surgery to an individualised strategy, maintaining SBP within 10 % of the resting preoperative value with a norepinephrine infusion, or to standard care in which ephedrine boluses were given only if SBP fell below 80 mmHg or 40 % below baseline. The composite of systemic inflammatory response syndrome and organ dysfunction within seven days occurred in 38.1 % of the individualised group compared with 51.7 % of the standard group (Futier et al., 2017). In contrast, the POISE-3 trial, which compared a hypotension-avoidance strategy (withholding antihypertensive drugs and targeting MAP ≥ 80 mmHg) with continuation of antihypertensives, found no significant difference in the composite of vascular death and major vascular complications (Marcucci et al., 2023). Together, these trials suggest that benefit is most likely in high-risk patients and when hypotension is actively prevented during surgery.

3.5. Cause-directed treatment, fluids and vasopressors

When hypotension is detected, the first step is to confirm that the measurement is genuine. Next, heart rate and rhythm, bleeding, anaesthetic depth, ventilator settings and surgical events are reviewed. Hypovolaemia and blood loss are treated with fluids or blood products according to clinical indications and, where available, dynamic indices of fluid responsiveness such as pulse pressure variation or stroke-volume variation. Vasodilation, which is the predominant mechanism after induction, responds to vasopressors such as phenylephrine or norepinephrine; reduction of anaesthetic depth should also be considered.

Excessive crystalloid contributes to oedema and pulmonary complications, whereas inadequate replacement causes hypoperfusion. Vasopressor choice depends on the mechanism, heart rate and cardiac output: phenylephrine may cause reflex bradycardia and reduce cardiac output, ephedrine provides mixed effects useful in bradycardia, and norepinephrine provides vasoconstriction with modest inotropy and is suitable for continuous infusion. Impaired contractility may require an inotrope, while bradycardia, arrhythmia, high neuraxial block and anaphylaxis require specific treatment. Proposed elements of a stepwise approach are presented in Table 2.

Table 2. Practical cause-directed approach to intraoperative hypotension.

Step	Action	Key considerations
1. Before surgery	Record baseline blood pressure; assess cardiovascular risk and medications	Decide on individual MAP target; consider holding ACE inhibitors/ARBs on the day of surgery according to local policy
2. Induction	Close monitoring (1–2 min cuff cycles or arterial line in high-risk patients); titrate induction agents	Prepare vasopressor in advance; avoid excessive anaesthetic depth
3. Verify	Confirm the reading: cuff size and position, transducer zero and level, waveform quality	Treat the patient, not an artefact
4. Identify the cause	Review heart rate/rhythm, bleeding, depth of anaesthesia, ventilation, surgical events	Consider vasodilation, hypovolaemia, low contractility, arrhythmia, obstruction, anaphylaxis
5. Treat	Fluids or blood products for hypovolaemia; vasopressor for vasodilation; inotrope for low contractility; specific therapy for arrhythmia	Use dynamic indices of fluid responsiveness where available
6. Reassess and document	Re-evaluate pressure, perfusion and response; record depth and duration of hypotension	Continue surveillance into the post-anaesthesia care unit and ward

4. Discussion

This review highlights three principles that should guide the management of IOH. First, hypotension should be understood as a continuous exposure rather than a binary event. The consistent dose–response relationship between the depth and duration of low pressure and postoperative organ injury suggests that brief, mild reductions are of limited importance, whereas sustained MAP below 65 mmHg, and particularly below 55 mmHg, deserves prompt correction (Walsh et al., 2013; Salmasi et al., 2017; Wesselink et al., 2018).

Second, the optimal target depends on the patient. The INPRESS trial demonstrated that an individualised target based on the patient's own baseline reduced organ dysfunction in high-risk patients (Futier et al., 2017), whereas a strategy applied broadly in POISE-3 did not improve vascular outcomes (Marcucci et al., 2023). These results are not contradictory: they differ in population, intervention and timing, and they emphasise that the benefit of tighter pressure control is concentrated in vulnerable patients and depends on how it is achieved.

Third, treatment must be directed at the mechanism. A blood-pressure value alone does not reveal whether the problem is vasodilation, hypovolaemia, poor contractility or obstruction, and the same MAP may reflect very different cardiac output and organ blood flow. Clinical assessment, waveform analysis and, where available, cardiac output monitoring or point-of-care ultrasound allow a rational choice of therapy.

Institutional protocols that define baseline measurement, risk-adapted monitoring and pre-prepared vasopressors can reduce variability in care; post-induction hypotension is a particularly attractive target because it is common and predictable.

This review has limitations. It is a narrative synthesis rather than a systematic review, and the selection of studies may be subject to bias. Most of the outcome data are observational and cannot exclude residual confounding, and the randomised evidence is limited to a small number of trials with heterogeneous populations and interventions. Future research should include adequately powered trials of individualised pressure targets, evaluation of predictive monitoring technologies on patient-centred outcomes, and studies that integrate blood pressure with measures of flow and tissue perfusion.

5. Conclusion

Management of intraoperative hypotension is not simply about increasing a blood-pressure number. The central tasks are to identify the cause of impaired perfusion, to limit the depth and duration of hypotension and to individualise pressure targets according to the patient's baseline and risk profile. A MAP of 65 mmHg is a reasonable lower limit

for most adults, but higher targets may be justified in patients with chronic hypertension or high cardiovascular and renal risk. Accurate monitoring, cause-directed use of fluids, vasopressors and inotropes, and standardised institutional protocols can improve perioperative safety in non-cardiac surgery.

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