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Risk Factors for Intraoperative Hypotension Under General Anesthesia: Clinical Predictors and Preventive Strategies

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ABSTRACT:

Background. Intraoperative hypotension (IOH) is among the most frequent hemodynamic disturbances during general anesthesia and is consistently associated, in a severity- and duration-dependent manner, with acute kidney injury, myocardial injury, major adverse cardiovascular events and mortality. Yet recent large randomized trials testing higher or individualized blood pressure targets have largely been neutral, sharpening the question of which patients are at risk, why, and which preventive measures are worth implementing.

Objective. To synthesize current evidence on patient-, medication-, anesthesia- and surgery-related risk factors for IOH; to appraise clinical and machine-learning prediction tools; to critically evaluate preventive and treatment strategies in light of trials published up to 2025; and to propose an implementable, risk-stratified framework for practice and health-system quality improvement.

Methods. Narrative review informed by the SANRA quality criteria. MEDLINE/PubMed, Embase, Web of Science and the Cochrane Library were searched for adult studies of IOH under general anesthesia, with priority given to large cohort studies, systematic reviews, consensus statements and randomized controlled trials.

Results. Consistent independent predictors of IOH include advanced age, ASA physical status III–IV, low pre-induction blood pressure, chronic hypertension, emergency surgery, renin–angiotensin system inhibitor continuation, propofol induction and higher opioid induction doses, and combined neuraxial–general anesthesia. Thresholds below a mean arterial pressure of 65 mmHg, and particularly below 55 mmHg, are associated with organ injury. Predictive algorithms can anticipate hypotension minutes in advance and reduce its depth and duration, but neither predictive monitoring nor elevated or individualized targets have yet been shown to improve hard clinical outcomes in large trials (POISE-3, Stop-or-Not, IMPROVE-multi, PRETREAT).

Conclusions. IOH is largely predictable and partly preventable. Current evidence supports avoiding sustained MAP below 65 mmHg, anticipating post-induction hypotension in high-risk patients, judicious induction dosing and continuous monitoring when risk is high, while reserving routinely elevated targets for research. The proposed PREVENT framework operationalizes these principles for clinical teams and quality systems, including those undergoing transformation under Saudi Vision 2030.

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Key Points

- IOH is strongly and dose-dependently associated with acute kidney injury, myocardial injury and mortality, with MAP below 65 mmHg as the most widely accepted lower boundary.
- Age, ASA III–V, low baseline pressure, emergency surgery, ACEI/ARB continuation, propofol and opioid induction dosing, and combined neuraxial anesthesia are the most consistent predictors.
- Continuous monitoring and waveform-based prediction reduce hypotension exposure, but elevated or individualized targets have not improved outcomes in large trials.
- The PREVENT framework provides a risk-proportionate pathway for clinical practice and quality improvement.

1. INTRODUCTION

More than 300 million surgical procedures are performed worldwide every year, and although intraoperative death has become rare, mortality within 30 days of surgery remains surprisingly common (Sessler et al., 2019). A substantial share of this postoperative burden is attributable to organ injury, notably myocardial injury after noncardiac surgery and acute kidney injury, both of which have been linked in large datasets to perioperative hemodynamic instability. Among the hemodynamic disturbances that anesthesia clinicians manage daily, intraoperative hypotension (IOH) is the most frequent and arguably the most modifiable.

General anesthesia lowers arterial pressure through several converging mechanisms: direct vasodilation, attenuation of sympathetic tone, blunting of the baroreflex, myocardial depression and the hemodynamic consequences of positive-pressure ventilation. In patients with limited physiological reserve, these effects are amplified by chronic hypertension, vascular stiffness, hypovolemia after fasting, and long-term cardiovascular medications. The result is that a considerable proportion of patients experience at least one hypotensive episode, with incidence varying from under 5% to more than 90% depending on the definition applied (Bijker et al., 2007).

Over the past decade, the field has moved through three distinct phases. First, large retrospective cohort studies established strong, graded associations between IOH and postoperative organ injury and death (Walsh et al., 2013; Salmasi et al., 2017; Gregory et al., 2021). Second, consensus statements translated these associations into practical thresholds, most notably the recommendation to avoid sustained mean arterial pressure (MAP) below 60–70 mmHg (Sessler et al., 2019). Third, and most recently, large pragmatic randomized trials have tested whether actively preventing hypotension improves outcomes, with results that have been predominantly neutral (Marcucci et al., 2023; Saugel et al., 2025; Kant et al., 2025). These findings have reopened a fundamental question: is IOH a cause of organ injury, a marker of patient vulnerability, or both?

Within this evolving landscape, a clear understanding of who develops IOH, and why, remains clinically indispensable. Risk identification determines the choice of monitoring, the induction strategy, the management of chronic medications and the allocation of scarce resources such as invasive arterial monitoring and predictive software. It also informs quality-improvement programs, which increasingly use hypotension exposure as an anesthesia performance indicator.

The aims of this review are therefore fourfold: (1) to clarify definitions and metrics of IOH and their prognostic implications; (2) to synthesize the evidence on patient-, medication-, anesthesia- and surgery-related risk factors; (3) to appraise prediction tools ranging from bedside clinical factors to machine-learning algorithms; and (4) to critically evaluate preventive and therapeutic strategies, culminating in an integrative, risk-stratified framework, PREVENT, designed for both bedside practice and health-system implementation.

2. REVIEW METHODOLOGY

This article is a structured narrative review prepared in accordance with the Scale for the Assessment of Narrative Review Articles (SANRA), which emphasizes explicit justification of the topic, a stated literature search strategy, appropriate referencing, scientific reasoning and presentation of relevant endpoint data. A narrative rather than systematic design was chosen because the evidence base is heterogeneous in population, definition of hypotension, exposure metric and outcome, precluding meaningful quantitative pooling across the full scope of the review.

2.1 Search Strategy

MEDLINE/PubMed, Embase, Web of Science and the Cochrane Library were searched from January 2000 to September 2026. Search terms combined controlled vocabulary and free text for the concepts "hypotension", "intraoperative" or "post-induction", "general anesthesia", and "risk factor", "predictor", "prediction", "machine learning", "prevention", "vasopressor" or "blood pressure target". Reference lists of key reviews and consensus statements were hand-searched, and trial registries were screened for recently completed randomized trials.

2.2 Eligibility and Evidence Prioritization

Studies were eligible if they enrolled adults undergoing noncardiac surgery under general anesthesia (alone or combined with regional techniques) and reported risk factors for, prediction of, outcomes associated with, or interventions to prevent IOH. Obstetric, paediatric and cardiac surgical populations were excluded unless findings were directly informative. Evidence was prioritized hierarchically: randomized controlled trials and their prespecified secondary analyses; systematic reviews and meta-analyses; international consensus statements; and large cohort studies (generally more than 1,000 patients). Smaller studies were cited only when they addressed mechanisms or risk factors not otherwise covered.

2.3 Synthesis

Findings were synthesized thematically across four domains: definition and outcomes, risk factors, prediction, and prevention. For each risk factor, the consistency of association across independent cohorts and the plausibility of mechanism were used to grade the strength of evidence qualitatively as strong, moderate or limited. The proposed framework was derived iteratively from this synthesis and from established patient-safety theory, specifically Reason's model of layered defences and Donabedian's structure–process–outcome paradigm.

3. DEFINING INTRAOPERATIVE HYPOTENSION

There is no universally accepted definition of IOH. In a landmark analysis, Bijker et al. (2007) applied 140 published definitions to a single cohort and found that incidence ranged from approximately 5% to 99%, demonstrating that reported prevalence depends more on the definition than on the population. Definitions vary along four dimensions: the blood pressure component (systolic, mean or diastolic), whether the threshold is absolute or relative to a baseline, the minimum duration of exposure, and whether treatment requirement forms part of the definition.

3.1 Absolute Versus Relative Thresholds

Mean arterial pressure is generally preferred because it determines organ perfusion pressure, is measured most robustly by oscillometric devices, and is least affected by peripheral pulse-wave amplification (Saugel & Sessler, 2021). Relative thresholds, typically a 20–30% fall from baseline, are intuitively attractive because chronically hypertensive patients are thought to have rightward-shifted autoregulation curves. However, in a cohort of more than 57,000 noncardiac surgical patients, Salmasi et al. (2017) found that absolute and relative thresholds discriminated myocardial and kidney injury equally well, and that risk increased progressively once MAP fell below approximately 65 mmHg regardless of baseline. A practical difficulty with relative thresholds is that the "baseline" itself is uncertain: pre-induction readings are often elevated by anxiety, and automated ambulatory measurements show that many patients' habitual pressures differ substantially from those recorded in the operating room (Saugel et al., 2019).

3.2 Exposure Metrics

Because organ injury appears to be a function of both depth and duration, single-episode definitions are increasingly replaced by integrated exposure metrics. Vernooij et al. (2020) cataloged the diversity of these metrics in the anesthesia literature, which complicates cross-study comparison. Table 1 summarizes the most widely used definitions and metrics.

Table 1. Common definitions and exposure metrics for intraoperative hypotension

Metric	Description	Strengths and limitations
Absolute MAP threshold	Any MAP below a fixed value (commonly 65 mmHg; also 60 or 55 mmHg)	Simple, reproducible, strongly linked to outcome; ignores individual baseline
Relative threshold	MAP or SBP decrease ≥20–30% from baseline	Physiologically intuitive; baseline is often unreliable
Cumulative duration	Total minutes below threshold	Captures persistence; ignores depth
Area under threshold (AUT)	Integral of mmHg × minutes below threshold	Captures depth and duration; biased by case length
Time-weighted average (TWA)	AUT divided by total monitoring time (mmHg)	Normalizes for duration; used as RCT endpoint (e.g., HYPE)
Post-induction hypotension	Hypotension within ~20 min of induction or before incision	Isolates anesthetic-driven phase; definitions vary

3.3 Phases of Intraoperative Hypotension

Südfeld et al. (2017) proposed that hypotension should be differentiated by phase because the underlying mechanisms differ. Post-induction hypotension (PIH), occurring in the first 20 minutes after induction and before surgical stimulation, is driven predominantly by anesthetic drugs acting on a vulnerable circulation. Early intraoperative hypotension, during the first 30 minutes of surgery, reflects the interplay of anesthesia, positioning and regional techniques, whereas later hypotension is more often surgical in origin, reflecting hemorrhage, fluid shifts, pneumoperitoneum or vascular manipulation. This distinction matters because a substantial fraction of all IOH occurs before the incision, during a period that is entirely under anesthetic control and therefore an attractive target for prevention (Maheshwari et al., 2018a).

4. EPIDEMIOLOGY AND CLINICAL CONSEQUENCES

The clinical relevance of IOH rests on a large body of observational evidence demonstrating consistent associations with postoperative organ dysfunction. Walsh et al. (2013), analysing more than 33,000 noncardiac surgical patients, reported that MAP below 55 mmHg was associated with acute kidney injury and myocardial injury even when sustained for only a few minutes, providing one of the first empirical definitions of harmful hypotension. Sun et al. (2015) extended these findings, associating MAP below 55 mmHg for more than 10 minutes, and below 60 mmHg for 11–20 minutes, with postoperative acute kidney injury.

Mortality associations have been reported in several independent populations. In a United States Veterans Affairs cohort, Monk et al. (2015) found that systolic pressure below 70 mmHg, MAP below 49 mmHg or diastolic pressure below 30 mmHg for five minutes or more were associated with increased 30-day mortality. Mascha et al. (2015) further showed that MAP variability and cumulative time below thresholds were independently associated with mortality. In the largest multicentre analysis to date, encompassing 368,222 procedures, Gregory et al. (2021) demonstrated that IOH was associated with 30-day major adverse cardiac or cerebrovascular events in a dose-dependent manner at every examined threshold (MAP ≤75, ≤65 and ≤55 mmHg) and across all age groups.

Two systematic reviews consolidate this evidence. Wesselink et al. (2018) synthesized 42 studies and concluded that end-organ injury risk rises with prolonged exposure (≥10 minutes) to MAP below 80 mmHg, with shorter exposures below 70 mmHg, increases further with duration below 65–60 mmHg, and is elevated with any exposure below 55–50 mmHg. A subsequent

meta-analysis by Wijnberge et al. (2021) similarly associated IOH with mortality, acute kidney injury and myocardial injury. Importantly, both groups emphasized that the retrospective design of the underlying studies precludes firm causal inference. Hypotension in the postoperative period is also associated with harm and may be under-recognized because it is often prolonged and less intensively monitored (Sessler et al., 2018).

Table 2. Selected landmark observational studies linking IOH to adverse outcomes

Study	Population	Exposure	Principal association
Walsh et al., 2013	33,330 noncardiac	MAP <55 mmHg	AKI and myocardial injury, even with brief exposure
Monk et al., 2015	18,756 (VA)	SBP <70, MAP <49, DBP <30 for ≥5 min	Increased 30-day mortality
Sun et al., 2015	5,127 noncardiac	MAP <55 for >10 min; <60 for 11–20 min	Postoperative AKI
Salmasi et al., 2017	57,315 noncardiac	Absolute vs relative thresholds	Graded risk below MAP ~65; absolute ≈ relative
Gregory et al., 2021	368,222 procedures	MAP ≤75, ≤65, ≤55 mmHg	Dose-dependent MACCE at all thresholds
Wesselink et al., 2018	42 studies (SR)	Various	Risk rises with depth and duration below MAP 80–50

◆ **KEY MESSAGE**

Across tens of thousands of patients, the association between IOH and organ injury is consistent, graded and biologically plausible. The threshold of MAP 65 mmHg has emerged as a pragmatic lower boundary, with exposure below 55 mmHg carrying the most consistent signal of harm.

5. PATHOPHYSIOLOGICAL BASIS

Arterial pressure is the product of cardiac output and systemic vascular resistance, and is regulated by the baroreflex, the sympathetic nervous system, the renin–angiotensin–aldosterone system and the vasopressin axis (Ackland et al., 2019). General anesthesia perturbs every component of this system.

5.1 Anesthetic Drug Effects

Propofol reduces systemic vascular resistance through direct vasodilation and inhibition of sympathetic outflow, decreases venous tone and therefore preload, and modestly depresses myocardial contractility. It also resets the baroreflex so that the compensatory tachycardia expected with a falling pressure is attenuated. Opioids augment these effects through vagally mediated bradycardia and further sympatholysis. Volatile agents produce dose-dependent vasodilation. Because these drugs act simultaneously in the minutes after induction, before surgical stimulation provides a countervailing sympathetic stimulus, the post-induction window is the period of greatest vulnerability (Reich et al., 2005).

5.2 The Vulnerable Circulation

Ageing, chronic hypertension and diabetes stiffen the arterial tree and impair baroreflex sensitivity. Patients become dependent on sympathetic vasoconstrictor tone to maintain pressure, so that the sympatholysis of anesthesia produces disproportionate falls. Chronic hypertension is thought to shift cerebral and renal autoregulation to the right, potentially narrowing the margin

between a "normal" intraoperative MAP and an ischemic one, although the clinical extent of this shift remains debated (Meng et al., 2018). Fasting and bowel preparation reduce stressed venous volume, and positive-pressure ventilation further reduces venous return, particularly with high airway pressures or positive end-expiratory pressure.

5.3 Renin–Angiotensin System Blockade

When sympathetic tone is suppressed by anesthesia, the renin–angiotensin system and vasopressin become important residual defenders of arterial pressure. Patients taking angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) lose one of these compensatory pathways, which explains the well-documented susceptibility of these patients to refractory hypotension and their responsiveness to vasopressin when catecholamine vasopressors are insufficient (Saugel & Sessler, 2021).

5.4 Mechanisms of Organ Injury

Organs with high metabolic demand and limited reserve, notably the renal medulla and the subendocardium, are most susceptible to hypoperfusion. Myocardial injury may result from supply–demand imbalance when diastolic pressure falls in patients with fixed coronary stenoses, while renal injury reflects reduced medullary oxygen delivery. Whether brief hypotension is sufficient to cause injury, or whether hypotension primarily identifies patients whose organs are already vulnerable, remains the central unresolved question in this field (Saugel & Sessler, 2021).

6. RISK FACTORS FOR INTRAOPERATIVE HYPOTENSION

Risk factors can be organized into four interacting domains: patient characteristics, chronic medications, anesthetic technique and surgical factors. The domains are not independent; for example, the hypotensive effect of a given propofol dose depends on age, baseline pressure and volume status. Table 3 summarizes the principal predictors and the strength of supporting evidence.

6.1 Patient-Related Factors

Age

Advanced age is among the most consistent predictors. Reich et al. (2005), in 4,096 patients, identified age 50 years or older as an independent predictor of severe hypotension within 10 minutes of induction, and Südfeld et al. (2017) reported that each additional year of age increased the odds of post-induction hypotension by approximately 3% (OR 1.03, 95% CI 1.02–1.04). Age-related arterial stiffening, reduced baroreflex sensitivity, diminished β-adrenergic responsiveness and altered drug pharmacokinetics, particularly a smaller central volume of distribution for propofol, together explain this effect.

Physical status and comorbidity

ASA physical status III–V predicted post-induction hypotension in the cohort of Reich et al. (2005), and ASA IV was associated with early intraoperative hypotension in the analysis of Südfeld et al. (2017). Specific comorbidities implicated across studies include heart failure with reduced ejection fraction, diastolic dysfunction, chronic kidney disease and diabetes with autonomic neuropathy. Frailty, an integrative marker of physiological reserve, is an increasingly recognized but less well-quantified contributor.

Low baseline blood pressure

Counter-intuitively, a lower pre-induction pressure predicts hypotension more strongly than a higher one. Baseline MAP below 70 mmHg predicted severe post-induction hypotension (Reich et al., 2005), and each mmHg increase in pre-induction systolic pressure was associated with slightly lower odds of hypotension (OR 0.97) in the cohort of Südfeld et al. (2017). This reflects the smaller absolute margin above harmful thresholds and, in some patients, a pre-existing low-output or vasodilated state.

Chronic hypertension

Patients with chronic hypertension experience larger absolute falls in pressure after induction and greater hemodynamic lability, owing to vascular stiffness and sympathetic dependence. Hypertension has also been associated with adverse outcomes when combined with IOH in surgical cohorts. However, the relationship is complicated by antihypertensive medications, which independently influence risk.

Sex, volume status and other factors

Male sex was associated with early intraoperative hypotension in one large cohort (Südfeld et al., 2017), although this finding is not uniform. Prolonged fasting, mechanical bowel preparation, vomiting, diarrhoea and diuretic use reduce effective circulating volume. Anemia, sepsis and adrenal insufficiency are less common but important causes of refractory hypotension that should prompt specific investigation.

6.2 Medication-Related Factors

ACE inhibitors and angiotensin receptor blockers

Continuation of ACEIs or ARBs up to the day of surgery is one of the best-characterized modifiable risk factors. In the VISION cohort, withholding these drugs for 24 hours before surgery was associated with less IOH and a lower risk of the composite of death, stroke and myocardial injury (Roshanov et al., 2017). The question was addressed definitively by the Stop-or-Not randomized trial, which allocated 2,222 patients undergoing major noncardiac surgery to continuation or discontinuation of renin–angiotensin system inhibitors 48 hours before surgery. Intraoperative hypotension occurred in 54% of the continuation group versus 41% of the discontinuation group (relative risk 1.31, 95% CI 1.19–1.44), yet the composite of death and major complications at 28 days was identical at 22% in both arms (Legrand et al., 2024). Continuation therefore reliably increases IOH but, within a framework of attentive management, does not appear to worsen outcomes.

Other cardiovascular and psychoactive drugs

Diuretics have been associated with post-induction hypotension, particularly in older patients, plausibly through volume contraction. Beta-blockers blunt compensatory tachycardia; calcium-channel blockers and alpha-adrenergic antagonists reduce vascular tone. Agents with alpha-blocking properties, including some antipsychotics and tricyclic antidepressants, and sodium–glucose cotransporter-2 inhibitors, through natriuresis and volume depletion, are mechanistically plausible contributors, although high-quality outcome data are limited.

6.3 Anesthesia-Related Factors

Induction agent and dose

Propofol induction was an independent predictor of severe post-induction hypotension in the cohort of Reich et al. (2005), and higher fentanyl induction doses were associated with hypotension in a dose-dependent fashion. Etomidate and ketamine preserve sympathetic tone better than propofol and are often preferred in hemodynamically fragile patients, although etomidate carries concerns regarding adrenal suppression. Because propofol requirements fall with age, standard weight-based dosing frequently represents a relative overdose in older patients.

Anesthetic depth and maintenance

Excessive anesthetic depth, whether with volatile agents or total intravenous anesthesia, increases vasodilation and myocardial depression. The period between intubation and incision, when anesthetic delivery is high and surgical stimulation absent, is particularly hazardous. Processed electroencephalographic monitoring may help avoid unnecessarily deep anesthesia, although its effect on hypotension-related outcomes has not been established.

Combined neuraxial and general anesthesia

Adding spinal or epidural anesthesia to general anesthesia was the strongest predictor of early intraoperative hypotension in the analysis of Südfeld et al. (2017), with more than threefold increased odds (OR 3.57, 95% CI 2.41–5.29), reflecting combined sympathetic blockade below the level of the block and systemic anesthetic effects.

Ventilation

Positive-pressure ventilation, high tidal volumes and high positive end-expiratory pressure reduce venous return and right ventricular output, effects that are magnified in hypovolemic patients.

6.4 Surgery-Related Factors

Emergency surgery was independently associated with both post-induction (OR 1.75) and early intraoperative hypotension (OR 1.83) in the cohort of Südfeld et al. (2017), reflecting reduced opportunity for optimization, hypovolemia, sepsis and rapid-

sequence induction. Major abdominal, vascular and orthopaedic procedures with substantial blood loss, prolonged operative duration, steep reverse Trendelenburg or beach-chair positioning, pneumoperitoneum, and surgical manipulation of the inferior vena cava or mesentery are further well-recognized precipitants. These factors are less amenable to prevention but highly amenable to anticipation.

Table 3. Principal risk factors for intraoperative hypotension and strength of evidence

Domain	Risk factor	Evidence	Modifiable?
Patient	Age (≥50–65 years, continuous effect)	Strong	No – anticipate
Patient	ASA physical status III–V	Strong	Partly – optimize
Patient	Low pre-induction MAP (<70 mmHg)	Strong	Partly
Patient	Chronic hypertension	Moderate	Partly
Patient	Heart failure, CKD, autonomic neuropathy, frailty	Moderate	Partly
Patient	Hypovolemia (fasting, bowel preparation)	Moderate	Yes
Medication	ACEI/ARB continued until surgery	Strong (RCT)	Yes
Medication	Diuretics, beta-blockers, alpha-blockers	Limited–moderate	Partly
Anesthesia	Propofol induction; high opioid dose	Strong	Yes
Anesthesia	Excessive anesthetic depth pre-incision	Moderate	Yes
Anesthesia	Combined neuraxial–general anesthesia	Strong	Partly
Surgery	Emergency surgery	Strong	No – anticipate
Surgery	Major blood loss, long duration, positioning	Moderate	Partly

7. PREDICTING INTRAOPERATIVE HYPOTENSION

Prediction serves two distinct purposes. Pre-operative risk stratification identifies patients who should receive enhanced monitoring, modified induction or tailored medication management. Real-time prediction during anesthesia aims to anticipate an impending hypotensive episode early enough for pre-emptive treatment.

7.1 Clinical Predictors and Risk Scores

The factors described in Section 6 form the basis of simple clinical risk assessment. Combinations of age, ASA status, baseline pressure, emergency status and planned technique can be applied at the pre-anesthesia visit without additional cost. Their limitation is modest discrimination for an individual patient, together with heterogeneous definitions of the outcome across derivation cohorts, which has hindered the development of a single validated score.

7.2 Machine-Learning Models

Kendale et al. (2018) applied supervised machine-learning techniques to more than 13,000 general anesthetics to predict post-induction hypotension from pre-operative and induction data. Ensemble methods outperformed conventional logistic regression, although discrimination remained moderate, with an area under the receiver operating characteristic curve in the mid-0.70s. Subsequent studies using larger electronic datasets and additional data streams, including pre-operative echocardiography and pre-induction heart-rate variability, have reported comparable or somewhat better performance, but external validation and prospective impact studies remain scarce.

7.3 Waveform-Based Prediction: The Hypotension Prediction Index

The Hypotension Prediction Index (HPI), developed by Hatib et al. (2018), is a machine-learning algorithm that analyses features of the high-fidelity arterial waveform to estimate the probability that MAP will fall below 65 mmHg for at least one minute. In validation studies it predicted hypotensive events several minutes before onset with high sensitivity and specificity. In the single-center HYPE trial, HPI-guided care combined with a diagnostic treatment protocol substantially reduced the time-weighted average of hypotension compared with standard care in elective noncardiac surgery (Wijnberge et al., 2020). However, in a larger randomized trial in moderate- to high-risk surgery, HPI guidance did not significantly reduce hypotension, largely because clinicians intervened on only around half of the alerts (Maheshwari et al., 2020). These contrasting results illustrate that prediction alone does not prevent hypotension; benefit depends on a clear, protocolized response.

7.4 Monitoring Modality

Detection depends on how often blood pressure is measured. Intermittent oscillometric measurement at 3–5-minute intervals misses brief episodes. In a randomized trial, continuous non-invasive blood pressure monitoring nearly halved hypotension exposure compared with intermittent cuff measurements, simply by enabling earlier recognition and treatment (Maheshwari et al., 2018b). The updated PeriOperative Quality Initiative consensus therefore recommends continuous arterial pressure monitoring in patients at high risk of hypotension (Saugel et al., 2024).

Table 4. Approaches to the prediction and detection of intraoperative hypotension

Approach	Inputs	Timing	Current status
Clinical risk factors	Age, ASA, baseline MAP, drugs, urgency	Pre-operative	Universally applicable; modest discrimination
Machine-learning risk models	EHR and induction data	Pre-/peri-induction	Moderate accuracy; limited external validation
Hypotension Prediction Index	Arterial waveform features	Real-time, minutes ahead	Reduces exposure when protocolized; outcome benefit unproven
Continuous non-invasive BP	Finger-cuff waveform	Real-time	Halves exposure versus intermittent cuff
Invasive arterial line	Direct intra-arterial pressure	Real-time	Reference standard; invasive

8. PREVENTIVE AND THERAPEUTIC STRATEGIES

Prevention spans the pre-operative, induction, maintenance and postoperative phases. A key distinction must be drawn between interventions that reduce hypotension exposure, which many do, and those that have been shown to improve patient outcomes, which few have. Table 5 summarizes the principal randomized trials.

8.1 Pre-operative Measures

Management of chronic antihypertensive medication

Two large trials now inform this decision. In POISE-3, a hypotension-avoidance strategy that withheld renin–angiotensin inhibitors and targeted intraoperative MAP ≥80 mmHg reduced IOH (19.1% versus 24.9%) but did not reduce major vascular complications compared with a hypertension-avoidance strategy (13.9% versus 14.0%; hazard ratio 0.99) (Marcucci et al., 2023). In Stop-or-Not, discontinuation reduced IOH without improving outcomes (Legrand et al., 2024). The pragmatic conclusion is that either approach is acceptable: withholding ACEIs/ARBs for 24–48 hours is reasonable when hypotension risk is high, while continuation is appropriate when it simplifies care or when there is concern about rebound hypertension or heart-failure decompensation, provided that clinicians anticipate and treat the resulting hypotension.

Establishing a meaningful baseline

Because individualized targets depend on an accurate reference pressure, pre-operative clinic or ward measurements should be documented and used in preference to anxiety-elevated pre-induction readings (Saugel et al., 2019). Even where individualized targets are not pursued, knowledge of a patient's habitual pressure informs the interpretation of intraoperative values.

Volume optimization

Avoiding unnecessarily prolonged fasting and permitting clear fluids up to two hours before anesthesia, as recommended by contemporary fasting guidelines, reduces pre-induction hypovolemia. Routine fluid preloading before propofol induction has not reliably prevented post-induction hypotension, and fluid therapy should not be used as a substitute for vasopressor support of vascular tone.

8.2 Induction Strategies

Because a large proportion of IOH occurs between induction and incision, this phase offers the greatest opportunity for prevention. Effective measures include titrating propofol slowly to effect and reducing doses in older and high-risk patients; limiting opioid boluses at induction; considering etomidate, ketamine or remimazolam-based techniques in hemodynamically fragile patients; establishing invasive or continuous non-invasive monitoring before induction in high-risk patients; and preparing a vasopressor for immediate use, or starting a low-dose prophylactic infusion of norepinephrine or phenylephrine in those at greatest risk. Minimizing the interval between induction and incision, and avoiding deep anesthesia during this unstimulated period, further reduce exposure.

8.3 Intraoperative Maintenance

Fluid therapy

The RELIEF trial demonstrated that a restrictive fluid regimen in major abdominal surgery did not improve disability-free survival and was associated with a higher rate of acute kidney injury than a moderately liberal regimen (Myles et al., 2018). This suggests that excessive fluid restriction should be avoided, and that fluid administration should be guided where possible by dynamic indices of fluid responsiveness or stroke-volume optimization in high-risk surgery.

Vasopressor therapy

Because most anesthesia-induced hypotension reflects vasodilation rather than hypovolemia, vasopressors are the logical first-line treatment. Phenylephrine and norepinephrine are both widely used; norepinephrine has the theoretical advantage of preserving cardiac output through modest β1-agonism. Ephedrine is useful when bradycardia accompanies hypotension. In patients on ACEIs or ARBs with hypotension refractory to catecholamines, low-dose vasopressin is mechanistically rational. The international POQI consensus emphasizes prompt treatment of hypotension and the use of a structured, cause-based approach to identify whether vasodilation, hypovolemia, reduced contractility or bradycardia predominates (Saugel et al., 2024).

8.4 Blood Pressure Targets: What the Trials Show

The INPRESS trial randomized 298 high-risk patients undergoing major surgery to an individualized strategy, maintaining systolic pressure within 10% of the resting reference value with a norepinephrine infusion, or to standard care. The individualized strategy reduced the composite of systemic inflammatory response and organ dysfunction at 7 days from 51.7% to 38.1% (relative risk 0.73) (Futier et al., 2017). This promizing result, however, has not been replicated in larger trials.

In POISE-3, targeting MAP ≥80 mmHg with medication withholding did not reduce major vascular events in 7,490 patients (Marcucci et al., 2023), and secondary analyses showed no reduction in acute kidney injury (POISE-3 Trial Investigators and Study Groups, 2025). A single-center trial similarly found that higher intraoperative targets did not reduce cardiovascular events (Wanner et al., 2021). In IMPROVE-multi, 1,134 high-risk patients undergoing major abdominal surgery were randomized to MAP targets based on each patient's pre-operative nighttime pressure or to routine management targeting MAP ≥65 mmHg; the composite of acute kidney injury, myocardial injury, non-fatal cardiac arrest or death within 7 days occurred in 33.5% versus 30.5%, respectively (relative risk 1.10, 95% CI 0.93–1.30) (Saugel et al., 2025). Finally, PRETREAT, which assigned patients to MAP targets of 70, 80 or 90 mmHg according to their predicted risk of hypotension, was stopped for futility after 3,247 of 5,000 planned patients, with no improvement in postoperative disability at six months (Kant et al., 2025).

Table 5. Key randomized trials of strategies to prevent or treat intraoperative hypotension

Trial	n	Intervention vs control	Principal finding
INPRESS (Futier et al., 2017)	298	SBP within 10% of baseline vs standard care	Reduced 7-day organ dysfunction (38.1% vs 51.7%)
HYPE (Wijnberge et al., 2020)	68	HPI-guided care vs standard care	Reduced TWA and duration of hypotension
Maheshwari et al., 2020	214	HPI guidance vs blinded HPI	No significant reduction; alerts often not acted on
POISE-3 (Marcucci et al., 2023)	7,490	MAP ≥80 + withhold RASi vs MAP ≥60 + continue	Less IOH; no difference in vascular events
Stop-or-Not (Legrand et al., 2024)	2,222	Continue vs stop RASi 48 h pre-op	More IOH with continuation; outcomes identical
IMPROVE-multi (Saugel et al., 2025)	1,134	Individualized MAP vs MAP ≥65 mmHg	No reduction in 7-day organ injury or death
PRETREAT (Kant et al., 2025)	3,247	Risk-stratified MAP 70/80/90 vs usual care	Stopped for futility; no disability benefit

◆ **KEY MESSAGE**

Interventions that reduce hypotension exposure are now well established. However, large trials consistently show that pushing blood pressure above conventional thresholds, or individualizing targets, does not improve hard outcomes. Avoiding sustained MAP below 65 mmHg remains the evidence-supported standard.

9. DISCUSSION: RECONCILING ASSOCIATION AND INTERVENTION

The apparent paradox between strong observational associations and neutral randomized trials deserves careful interpretation. Several explanations, not mutually exclusive, are plausible.

First, confounding by indication and by patient vulnerability is likely substantial. The same characteristics that predispose to hypotension, namely age, cardiovascular disease, renal dysfunction and frailty, also predispose to postoperative organ injury. Observational analyses adjust for measured confounders, but residual confounding is inevitable, and hypotension may partly function as a marker of a vulnerable circulation rather than an independent cause of harm.

Second, the contrast in recent trials was largely between moderate hypotension and somewhat higher pressures. In POISE-3, IMPROVE-multi and PRETREAT, control groups were managed by clinicians already targeting MAP of approximately 60–65 mmHg, so severe and prolonged hypotension, the exposure most strongly associated with harm, was uncommon in both arms. These trials therefore show that raising pressure above contemporary practice does not help; they do not show that profound hypotension is harmless.

Third, the means used to raise pressure may carry their own costs. Vasopressors increase afterload and may reduce microcirculatory flow, and additional fluid can cause tissue edema. Any benefit of higher pressure could be offset by the harms of achieving it.

Fourth, the relevant exposure may extend beyond the operating room. Postoperative hypotension on surgical wards is common, often prolonged and frequently undetected with intermittent observation, and may contribute more to organ injury than brief intraoperative episodes (Sessler et al., 2018; Saugel et al., 2024).

For clinicians, the practical implication is a balanced stance: severe and sustained hypotension should be prevented and promptly corrected, because the observational signal is strong and the intervention is inexpensive and low-risk; but routinely elevated or individualized targets should not be adopted outside research settings. For researchers, the priority shifts from asking whether to treat hypotension to identifying which patients, which organs and which mechanisms are most susceptible, and whether targeted protection of those patients improves outcomes.

10. The PREVENT Framework for Risk-Stratified Hemodynamic Care

To translate the evidence into an actionable structure, we propose the PREVENT framework. It is grounded in Reason's model of layered defences, in which each element constitutes an independent barrier against harm, and in Donabedian's structure–process–outcome model, which links organizational resources to processes of care and measurable results. The framework is deliberately aligned with current consensus rather than with interventions that recent trials have failed to support.

Table 6. The PREVENT framework: components, actions and audit indicators

Component	Core actions	Suggested audit indicator
P – Profile risk	Pre-anesthesia assessment of age, ASA, baseline BP, comorbidity, urgency and planned technique; assign low/intermediate/high risk	% of patients with documented IOH risk category
R – Reconcile medications	Shared decision on ACEI/ARB (withhold 24–48 h if high risk, or continue with anticipation); review diuretics and other vasoactive drugs	% with documented RASi plan
E – Engineer a gentle induction	Age-adjusted, titrated propofol; limited opioid bolus; consider etomidate/ketamine; vasopressor prepared or prophylactic infusion in high-risk	Post-induction hypotension rate (MAP <65 within 20 min)
V – Vigilant monitoring	Continuous arterial or non-invasive monitoring for high-risk patients from induction; consider predictive tools only with a response protocol	% of high-risk patients with continuous BP monitoring
E – Early, cause-directed treatment	Treat MAP <65 promptly; vasopressor-first for vasodilation; dynamic assessment before fluid; vasopressin for refractory RASi-related hypotension	Time-weighted average MAP <65 mmHg; minutes MAP <55

Component	Core actions	Suggested audit indicator
N – Navigate the postoperative period	Hand over hemodynamic risk; enhanced ward observation for high-risk patients; protocol for restarting antihypertensives	Postoperative hypotension detection rate
T – Track, team and learn	Automated IOH reporting from anesthesia information systems; departmental feedback; simulation and team training	Departmental IOH dashboard reviewed quarterly

The framework is risk-proportionate. Low-risk patients require little beyond standard monitoring and sensible dosing. Intermediate-risk patients benefit from medication reconciliation, cautious induction and shortened cuff-cycling intervals. High-risk patients, typically older patients with significant cardiovascular or renal disease, low baseline pressure or emergency presentation, warrant continuous blood pressure monitoring from induction, a prepared vasopressor strategy and enhanced postoperative surveillance. Importantly, PREVENT does not prescribe elevated pressure targets; it focuses on preventing clinically meaningful hypotension below established thresholds.

"The goal is not higher blood pressure for everyone, but no unrecognized, untreated, sustained hypotension for anyone."

11. IMPLEMENTATION AND HEALTH-SYSTEM PERSPECTIVES

IOH lends itself to quality measurement because the exposure is captured automatically by anesthesia information management systems, can be expressed as an objective metric such as time-weighted average MAP below 65 mmHg, and is largely within the control of the anesthesia team. Departments can use such metrics for benchmarking, feedback and targeted education, provided that indicators are adjusted for case mix and interpreted as process measures rather than proxies for outcome.

Successful implementation requires attention to structure (availability of continuous monitoring devices, vasopressor infusion pumps and electronic records), process (standardized risk assessment, medication reconciliation pathways and treatment algorithms) and culture (team communication at induction, closed-loop instructions and non-punitive review of adverse hemodynamic events). Interprofessional training, including anesthesia nurses, anesthesia technologists and pharmacists, is essential, since vasopressor preparation, monitoring set-up and medication reconciliation are shared responsibilities.

11.1 Relevance to Health-System Transformation in Saudi Arabia

Health systems undergoing rapid modernization offer a particular opportunity to embed such approaches from the outset. In the Kingdom of Saudi Arabia, the Health Sector Transformation Program under Vision 2030 prioritizes patient safety, quality measurement and digital health (Saudi Vision 2030, 2021). The expansion of electronic health records and anesthesia information systems across public and private hospitals creates the infrastructure for automated IOH reporting. National accreditation standards set by the Saudi Central Board for Accreditation of Healthcare Institutions (CBAHI), with their emphasis on anesthesia and sedation safety and on performance-improvement indicators, provide a natural vehicle for adopting hypotension-exposure metrics. Professional training and continuing education overseen by the Saudi Commission for Health Specialties (SCFHS) could integrate risk-stratified hemodynamic management into anesthesia residency curricula and simulation programs. Local research is needed to establish the incidence and determinants of IOH in Saudi surgical populations, in which the high prevalence of diabetes, hypertension and obesity may modify risk profiles relative to the predominantly Western cohorts from which current evidence derives.

12. RESEARCH GAPS AND FUTURE DIRECTIONS

- **Causality:** pragmatic trials comparing prevention of severe hypotension (for example, MAP <55 mmHg) with permissive management are needed to establish whether avoiding profound hypotension, rather than raising average pressure, improves outcomes.
- **Phenotyping:** hemodynamic phenotyping (vasodilatory, hypovolemic, cardiogenic, bradycardic) may allow treatment to target mechanism rather than a pressure number alone.

- **Organ-specific perfusion:** integrating cerebral oximetry, renal perfusion indices and markers of microcirculatory flow could identify the pressure at which an individual's organs become hypoperfused.
- **Predictive analytics:** externally validated, transparent models embedded in electronic records, paired with protocolized responses, should be tested for patient-centerd outcomes rather than surrogate exposure metrics.
- **Postoperative continuity:** continuous wireless ward monitoring and its effect on unrecognized postoperative hypotension represent a major unexplored frontier.
- **Regional evidence:** multicentre registries in the Middle East, including Saudi Arabia, would clarify generalisability and support context-specific guidelines.

13. LIMITATIONS OF THIS REVIEW

As a narrative review, this work is subject to selection bias in the identification and weighting of studies, although a structured search and explicit evidence hierarchy were used to mitigate this. The heterogeneity of hypotension definitions across the literature limits direct comparison of effect sizes. Most risk-factor evidence derives from retrospective cohorts in high-income Western settings, and some risk factors, such as sodium–glucose cotransporter-2 inhibitor use, are supported primarily by mechanistic reasoning. The proposed PREVENT framework has not yet been prospectively validated and should be regarded as a structured synthesis to guide implementation and future evaluation.

14. CONCLUSIONS

Intraoperative hypotension under general anesthesia is common, largely predictable and consistently associated with postoperative organ injury and death. Its strongest predictors, namely advanced age, high ASA class, low baseline pressure, emergency surgery, renin–angiotensin system inhibitor continuation, propofol- and opioid-based induction and combined neuraxial anesthesia, are readily identifiable before surgery. Continuous monitoring and predictive algorithms reduce hypotension exposure, but recent large trials have shown that elevated or individualized pressure targets do not improve hard outcomes. The evidence therefore supports a pragmatic, risk-proportionate approach: identify vulnerable patients, induce anesthesia gently, monitor closely, and treat MAP below 65 mmHg promptly and according to its cause. The PREVENT framework offers a structured means of embedding these principles in daily practice and in health-system quality programs, while future research clarifies whether, and for whom, preventing hypotension changes patient outcomes.

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REFERENCES

[1] Ackland, G. L., Brudney, C. S., Cecconi, M., Ince, C., Irwin, M. G., Lacey, J., Pinsky, M. R., Grocott, M. P., Mythen, M. G., Edwards, M. R., & Miller, T. E. (2019). Perioperative Quality Initiative consensus statement on the physiology of arterial blood pressure control in perioperative medicine. British Journal of Anaesthesia, 122(5), 542–551.

[2] Bijker, J. B., van Klei, W. A., Kappen, T. H., van Wolfswinkel, L., Moons, K. G. M., & Kalkman, C. J. (2007). Incidence of intraoperative hypotension as a function of the chosen definition: Literature definitions applied to a retrospective cohort using automated data collection. Anesthesiology, 107(2), 213–220.

[3] Futier, E., Lefrant, J. Y., Guinot, P. G., Godet, T., Lorne, E., Cuvillon, P., Bertran, S., Leone, M., Pastene, B., Piriou, V., Molliex, S., Albanese, J., Julia, J. M., Tavernier, B., Imhoff, E., Bazin, J. E., Constantin, J. M., Pereira, B., & Jaber, S. (2017). Effect of individualized vs standard blood pressure management strategies on postoperative organ dysfunction among high-risk patients undergoing major surgery: A randomized clinical trial. JAMA, 318(14), 1346–1357. https://doi.org/10.1001/jama.2017.14172

[4] Gregory, A., Stapelfeldt, W. H., Khanna, A. K., Smischney, N. J., Boero, I. J., Chen, Q., Stevens, M., & Shaw, A. D. (2021). Intraoperative hypotension is associated with adverse clinical outcomes after noncardiac surgery. Anesthesia & Analgesia, 132(6), 1654–1665.

[5] Hatib, F., Jian, Z., Buddi, S., Lee, C., Settels, J., Sibert, K., Rinehart, J., & Cannesson, M. (2018). Machine-learning algorithm to predict hypotension based on high-fidelity arterial pressure waveform analysis. Anesthesiology, 129(4), 663–674.

[6] Kant, M., et al. (2025). Proactive vs reactive treatment of hypotension during surgery: The PRETREAT randomized clinical trial. JAMA, 334(21), 1905–1914. https://doi.org/10.1001/jama.2025.18007

[7] Kendale, S., Kulkarni, P., Rosenberg, A. D., & Wang, J. (2018). Supervised machine-learning predictive analytics for prediction of postinduction hypotension. Anesthesiology, 129(4), 675–688.

[8] Legrand, M., Falcone, J., Cholley, B., Charbonneau, H., Delaporte, A., Lemoine, A., et al. (2024). Continuation vs discontinuation of renin-angiotensin system inhibitors before major noncardiac surgery: The Stop-or-Not randomized clinical trial. JAMA, 332(12), 970–978. https://doi.org/10.1001/jama.2024.17123

[9] Maheshwari, K., Khanna, S., Bajracharya, G. R., Makarova, N., Riter, Q., Raza, S., Cywinski, J. B., Argalious, M., Kurz, A., & Sessler, D. I. (2018b). A randomized trial of continuous noninvasive blood pressure monitoring during noncardiac surgery. Anesthesia & Analgesia, 127(2), 424–431.

[10] Maheshwari, K., Shimada, T., Yang, D., Khanna, S., Cywinski, J. B., Irefin, S. A., Ayad, S., Turan, A., Ruetzler, K., Qiu, Y., Saha, P., Mascha, E. J., & Sessler, D. I. (2020). Hypotension Prediction Index for prevention of hypotension during moderate- to high-risk noncardiac surgery. Anesthesiology, 133(6), 1214–1222.

[11] Maheshwari, K., Turan, A., Mao, G., Yang, D., Niazi, A. K., Agarwal, D., Sessler, D. I., & Kurz, A. (2018a). The association of hypotension during non-cardiac surgery, before and after skin incision, with postoperative acute kidney injury: A retrospective cohort analysis. Anaesthesia, 73(10), 1223–1228.

[12] Marcucci, M., Painter, T. W., Conen, D., Lomivorotov, V., Sessler, D. I., Chan, M. T. V., Borges, F. K., Leslie, K., Duceppe, E., et al. (2023). Hypotension-avoidance versus hypertension-avoidance strategies in noncardiac surgery: An international randomized controlled trial. Annals of Internal Medicine, 176(5), 605–614. https://doi.org/10.7326/M22-3157

[13] Mascha, E. J., Yang, D., Weiss, S., & Sessler, D. I. (2015). Intraoperative mean arterial pressure variability and 30-day mortality in patients having noncardiac surgery. Anesthesiology, 123(1), 79–91.

[14] Meng, L., Yu, W., Wang, T., Zhang, L., Heerdt, P. M., & Gelb, A. W. (2018). Blood pressure targets in perioperative care. Hypertension, 72(4), 806–817.

[15] Monk, T. G., Bronsert, M. R., Henderson, W. G., Mangione, M. P., Sum-Ping, S. T., Bentt, D. R., Nguyen, J. D., Richman, J. S., Meguid, R. A., & Hammermeister, K. E. (2015). Association between intraoperative hypotension and hypertension and 30-day postoperative mortality in noncardiac surgery. Anesthesiology, 123(2), 307–319.

[16] Myles, P. S., Bellomo, R., Corcoran, T., Forbes, A., Peyton, P., Story, D., et al. (2018). Restrictive versus liberal fluid therapy for major abdominal surgery. New England Journal of Medicine, 378(24), 2263–2274.

[17] POISE-3 Trial Investigators and Study Groups. (2025). A sub-study of the POISE-3 randomized trial examined effects of a perioperative hypotension-avoidance strategy versus a hypertension-avoidance strategy on the risk of acute kidney injury. Kidney International, 107(1), 155–168. https://doi.org/10.1016/j.kint.2024.10.007

[18] Reich, D. L., Hossain, S., Krol, M., Baez, B., Patel, P., Bernstein, A., & Bodian, C. A. (2005). Predictors of hypotension after induction of general anesthesia. Anesthesia & Analgesia, 101(3), 622–628.

[19] Roshanov, P. S., Rochwerg, B., Patel, A., Salehian, O., Duceppe, E., Belley-Côté, E. P., et al. (2017). Withholding versus continuing angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers before noncardiac surgery: An analysis of the Vascular events In noncardiac Surgery patIents cOhort evaluatioN prospective cohort. Anesthesiology, 126(1), 16–27.

[20] Salmasi, V., Maheshwari, K., Yang, D., Mascha, E. J., Singh, A., Sessler, D. I., & Kurz, A. (2017). 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, 126(1), 47–65. https://doi.org/10.1097/ALN.0000000000001432

[21] Saudi Vision 2030. (2021). Health Sector Transformation Program: Delivery plan. Government of the Kingdom of Saudi Arabia.

[22] Saugel, B., Meidert, A. S., Brunkhorst, F. M., Bischoff, R., Esser, J., Mattis, M., et al. (2025). Individualized perioperative blood pressure management in patients undergoing major abdominal surgery: The IMPROVE-multi randomized clinical trial. JAMA, 334(21), 1893–1904. https://doi.org/10.1001/jama.2025.17235

[23] Saugel, B., Reese, P. C., Sessler, D. I., Burfeindt, C., Nicklas, J. Y., Pinnschmidt, H. O., Reuter, D. A., & Südfeld, S. (2019). Automated ambulatory blood pressure measurements and intraoperative hypotension in patients having noncardiac surgery with general anesthesia: A prospective observational study. Anesthesiology, 131(1), 74–83.

[24] Saugel, B., & Sessler, D. I. (2021). Perioperative blood pressure management. Anesthesiology, 134(2), 250–261. https://doi.org/10.1097/ALN.0000000000003610

[25] Saugel, B., Sessler, D. I., & PeriOperative Quality Initiative XI (POQI XI) Workgroup. (2024). PeriOperative Quality Initiative (POQI) international consensus statement on perioperative arterial pressure management. British Journal of Anaesthesia, 133(2). https://doi.org/10.1016/j.bja.2024.04.046

[26] Sessler, D. I., Bloomstone, J. A., Aronson, S., Berry, C., Gan, T. J., Kellum, J. A., Plumb, J., Mythen, M. G., Grocott, M. P. W., Edwards, M. R., & Miller, T. E. (2019). Perioperative Quality Initiative consensus statement on intraoperative blood pressure, risk and outcomes for elective surgery. British Journal of Anaesthesia, 122(5), 563–574. https://doi.org/10.1016/j.bja.2019.01.013

[27] Sessler, D. I., Meyhoff, C. S., Zimmerman, N. M., Mao, G., Leslie, K., Vásquez, S. M., et al. (2018). Period-dependent associations between hypotension during and for four days after noncardiac surgery and a composite of myocardial infarction and death: A substudy of the POISE-2 trial. Anesthesiology, 128(2), 317–327.

[28] Südfeld, S., Brechnitz, S., Wagner, J. Y., Reese, P. C., Pinnschmidt, H. O., Reuter, D. A., & Saugel, B. (2017). Post-induction hypotension and early intraoperative hypotension associated with general anaesthesia. British Journal of Anaesthesia, 119(1), 57–64. https://doi.org/10.1093/bja/aex127

[29] Sun, L. Y., Wijeysundera, D. N., Tait, G. A., & Beattie, W. S. (2015). Association of intraoperative hypotension with acute kidney injury after elective noncardiac surgery. Anesthesiology, 123(3), 515–523.

[30] Vernooij, L. M., van Klei, W. A., Moons, K. G. M., van Waes, J. A. R., & Peelen, L. M. (2020). Methods to express intraoperative hypotension exposure in the anaesthesia literature. British Journal of Anaesthesia, 124(3), e35–e37. https://doi.org/10.1016/j.bja.2019.12.004

[31] Walsh, M., Devereaux, P. J., Garg, A. X., Kurz, A., Turan, A., Rodseth, R. N., Cywinski, J., Thabane, L., & Sessler, D. I. (2013). Relationship between intraoperative mean arterial pressure and clinical outcomes after noncardiac surgery: Toward an empirical definition of hypotension. Anesthesiology, 119(3), 507–515. https://doi.org/10.1097/ALN.0b013e3182a10e26

[32] Wanner, P. M., Wulff, D. U., Djurdjevic, M., Korte, W., Schnider, T. W., & Filipovic, M. (2021). Targeting higher intraoperative blood pressures does not reduce adverse cardiovascular events following noncardiac surgery. Journal of the American College of Cardiology, 78(18), 1753–1764.

[33] Wesselink, E. M., Kappen, T. H., Torn, H. M., Slooter, A. J. C., & van Klei, W. A. (2018). Intraoperative hypotension and the risk of postoperative adverse outcomes: A systematic review. British Journal of Anaesthesia, 121(4), 706–721. https://doi.org/10.1016/j.bja.2018.04.036

[34] Wijnberge, M., Geerts, B. F., Hol, L., Lemmers, N., Mulder, M. P., Berge, P., Schenk, J., Terwindt, L. E., Hollmann, M. W., Vlaar, A. P., & Veelo, D. P. (2020). Effect of a machine learning-derived early warning system for intraoperative hypotension vs standard care on depth and duration of intraoperative hypotension during elective noncardiac surgery: The HYPE randomized clinical trial. JAMA, 323(11), 1052–1060. https://doi.org/10.1001/jama.2020.0592

[35] Wijnberge, M., Schenk, J., Bulle, E., Vlaar, A. P., Maheshwari, K., Hollmann, M. W., Binnekade, J. M., Geerts, B. F., & Veelo, D. P. (2021). Association of intraoperative hypotension with postoperative morbidity and mortality: Systematic review and meta-analysis. BJS Open, 5(1), zraa018.