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Intraoperative Hypotension — From Blood Pressure Thresholds to Individualized Hemodynamic Management: Current State of Knowledge

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Abstract

Introduction and Purpose. Intraoperative hypotension (IOH) is common during non-cardiac surgery and has repeatedly been associated with postoperative organ injury. Its clinical significance, however, is not always straightforward. Definitions of IOH vary between studies, arterial pressure alone does not identify the underlying hemodynamic disturbance, and the optimal blood pressure for an individual patient remains uncertain. This review examines current evidence on the clinical consequences, risk assessment, monitoring, and management of IOH, with particular attention to the limitations of fixed blood pressure thresholds and the role of individualized hemodynamic management.

Materials and Methods. A structured literature search of PubMed/MEDLINE was performed, with the final search completed on 16 August 2026. The review focused primarily on studies published between January 2015 and August 2026, while relevant earlier studies were also considered. Randomized and observational studies, systematic reviews, meta-analyses, and current perioperative guidelines and consensus statements were considered.

Results. Observational studies generally show stronger associations between IOH and postoperative organ injury when hypotension is deeper or lasts longer, particularly for acute kidney injury and myocardial injury. These findings do not establish that hypotension itself is the sole cause of postoperative complications. Pragmatic MAP thresholds remain useful in clinical practice, but an optimal target for all patients has not been identified. Current consensus recommendations favour treatment based on the presumed hemodynamic cause rather than a uniform response to a low MAP value. Continuous and predictive monitoring can reduce exposure to hypotension, although better blood pressure control has not consistently resulted in better patient-centred outcomes. Postoperative hypotension remains less well characterized.

Conclusions. The clinical significance of IOH cannot be determined from a single blood pressure value alone. Early recognition and cause-directed treatment remain central to management, while the optimal degree of blood pressure individualization is uncertain. Further research should clarify which hypotensive episodes represent modifiable postoperative risk and which patients are most likely to benefit from individualized monitoring and treatment.

Keywords: intraoperative hypotension; mean arterial pressure; perioperative hemodynamics; blood pressure monitoring; hemodynamic management; non-cardiac surgery

1. Introduction

Intraoperative hypotension (IOH) is a frequent problem during anesthesia and has been linked to several postoperative complications, most notably acute kidney injury and myocardial injury [1,2]. Although blood pressure is monitored routinely throughout surgery, there is still no single definition of IOH that is used consistently in clinical studies [1]. The optimal blood pressure target is also uncertain [2,3]. In practice, recognizing a low blood pressure is usually straightforward, while deciding when it becomes clinically important and how aggressively it should be treated is more difficult.

This problem is reflected in the way IOH has been defined in the literature. A review of 318 studies involving adults undergoing non-cardiac surgery showed considerable variation between the definitions used [1]. Most studies relied on an absolute blood pressure threshold, some used a decrease from the patient's baseline value, and nearly half did not include the duration of hypotension in their definition [1]. This makes direct comparison between studies difficult and contributes to the wide variation in the reported incidence of IOH. Mean arterial pressure (MAP) is now commonly used to guide intraoperative blood pressure management, with values around 60–65 mmHg serving as pragmatic lower reference points in current perioperative recommendations rather than as universal physiological thresholds [2,3].

The clinical importance of IOH depends on more than the measured pressure alone. The depth and duration of hypotension, the patient's susceptibility, and the underlying hemodynamic disturbance all influence its interpretation [2,3]. Similar reductions in MAP may result from different mechanisms and may therefore require different treatment [2,3]. Correcting the pressure value without considering its cause may be insufficient.

Much of the evidence linking IOH with postoperative complications comes from large observational studies. Deeper and longer hypotension has repeatedly been associated with a higher risk of organ injury, particularly acute kidney injury and myocardial injury after non-cardiac surgery [2,4]. These associations are clinically important, but they do not establish that hypotension is the sole or direct cause of postoperative injury [2]. Patient comorbidity, cardiovascular reserve, blood loss, and surgical complexity may contribute both to the development of IOH and to postoperative outcomes.

Randomized trials provide a less consistent picture. Avoiding severe or prolonged hypotension remains an important part of intraoperative care, but strategies targeting higher or individualized blood pressures have not consistently improved postoperative outcomes [2,5].

This discrepancy leaves several questions unresolved, including how hypotension severity and duration should be interpreted, which patients are most susceptible to harm, and whether management based on the underlying hemodynamic disturbance can improve outcomes beyond treatment guided mainly by arterial pressure.

The aim of this review is to evaluate current evidence on IOH in adults undergoing non-cardiac surgery, focusing on its definition, severity and duration, relationship with postoperative organ injury, and current approaches to monitoring and treatment. Particular attention is given to blood pressure targets and to the difference between the strong associations reported in observational studies and the less consistent results of randomized trials evaluating more intensive or individualized blood pressure management.

2. Materials and Methods

A structured literature search was conducted in PubMed/MEDLINE, with the final search completed on 16 August 2026. The review focused primarily on publications from January 2015 to August 2026. Earlier studies were considered when they provided relevant evidence on the definition of intraoperative hypotension, the development of blood pressure thresholds, or other concepts important to perioperative blood pressure management.

The search covered terms related to intraoperative hypotension, perioperative hypotension, mean arterial pressure, blood pressure thresholds, postoperative outcomes, risk factors, hemodynamic monitoring, hypotension prediction, vasopressor therapy, and individualized blood pressure management. The main focus was on adults undergoing non-cardiac surgery.

Randomized controlled trials, observational studies, systematic reviews, and meta-analyses were considered, together with current clinical practice guidelines and perioperative consensus statements relevant to the definition, monitoring, or management of IOH.

Studies limited to cardiac, obstetric, or paediatric populations were outside the main scope of the review because of important differences in patient characteristics and perioperative hemodynamic management.

3. Pathophysiology and Hemodynamic Consequences

Intraoperative hypotension can result from several hemodynamic disturbances, and more than one mechanism is often present at the same time. Arterial pressure depends mainly on cardiac output and systemic vascular resistance, both of which may change rapidly during anesthesia.

Induction of general anesthesia commonly reduces sympathetic tone and causes vasodilation, while some anesthetic agents can also decrease myocardial contractility. Venous return and cardiac output may consequently fall, which partly explains why hypotension is particularly common shortly after induction [6].

The cause of hypotension may change during the course of an operation. Vasodilation can predominate initially, while blood loss or other changes in circulating volume may become more important later. Cardiac factors also need to be considered. Impaired myocardial function, changes in ventricular preload or afterload, bradycardia, and tachyarrhythmias can all reduce cardiac output and contribute to a fall in arterial pressure. In practice, these mechanisms frequently overlap. Two patients with the same mean arterial pressure (MAP) may therefore have different hemodynamic disturbances and may not require the same approach to treatment [2,3,6].

The relationship between arterial pressure and organ blood flow is more complex than the blood pressure value itself suggests. Autoregulation can maintain relatively stable blood flow across a range of perfusion pressures, but below an organ's lower autoregulatory limit blood flow becomes increasingly pressure-dependent. This limit is not fixed and may differ between organs and patients. How reliably clinically relevant autoregulatory thresholds can be identified during surgery remains uncertain [2,6].

MAP should therefore be regarded as an important hemodynamic variable rather than a direct measure of tissue perfusion. Organ perfusion depends on the pressure gradient across the vascular bed, so elevated venous or tissue pressure can reduce effective perfusion even when MAP appears acceptable [2]. Systemic arterial pressure may also fail to reflect changes at the microcirculatory level. Restoration of MAP to an acceptable value does not necessarily confirm normalization of regional blood flow or oxygen delivery. Conversely, a brief decrease in MAP does not inevitably result in tissue injury [2,6].

The depth and duration of hypotension are important when considering its potential consequences. A brief episode of moderate hypotension is not equivalent to a prolonged or profound reduction in arterial pressure. Observational studies generally show greater postoperative risk with increasing hypotension exposure, particularly for acute kidney injury and myocardial injury [2,4]. These associations support limiting the overall burden of hypotension but do not establish that hypotension is the sole cause of postoperative injury [2].

The clinical course of IOH can be viewed as a continuum rather than as a sequence defined by a single pressure threshold. An initial decrease in arterial pressure may be tolerated and resolve without detectable consequences. As hypotension becomes deeper or persists, maintaining adequate organ perfusion and oxygen delivery may become more difficult, particularly in patients with limited physiological reserve. Reduced perfusion may then contribute to cellular dysfunction and clinically apparent organ injury. The point at which this occurs cannot currently be defined by a single MAP value applicable to every patient [2,6].

Current perioperative recommendations use pragmatic lower MAP thresholds, generally around 60–65 mmHg, rather than a universal physiological boundary [2,3]. These values are population-based clinical reference points rather than optimal pressures for every patient. Interpretation of IOH therefore requires consideration of arterial pressure together with its depth and duration, the likely hemodynamic mechanism, and the clinical context [2,3].

4. Epidemiology and Clinical Course

The reported incidence of intraoperative hypotension varies considerably between studies and depends on the definition used, the population examined, and the type and duration of surgery [1]. Blood pressure thresholds are particularly important. A definition based on MAP <65 mmHg will identify more episodes than one using a lower absolute threshold, while relative decreases from baseline identify a partly different group of patients [1]. Reported incidence should therefore be interpreted in relation to the definition used rather than as a fixed estimate of how often IOH occurs.

In a prospective cohort of 720 adults undergoing elective non-cardiac surgery, IOH occurred in 33.1% of patients when defined as MAP <65 mmHg for at least one minute. A relative decrease of more than 30% from baseline was observed in 39.9% [7]. These results illustrate how substantially the estimated frequency can change with the definition applied. Differences in patient characteristics, anesthetic technique, surgical setting, and blood pressure measurement may add further variation between studies.

The course of IOH is also variable. Many episodes are brief and occur around induction of anesthesia, whereas others develop later or persist despite initial treatment [6]. Observational data indicate that postoperative risk is related not only to whether hypotension occurs but also to the accumulated exposure [2,4,8]. The kidney and myocardium have been studied most extensively in this context [2,4,8].

In a retrospective analysis of 33,330 non-cardiac surgical procedures, exposure to MAP below 55 mmHg was associated with both acute kidney injury and myocardial injury, with stronger associations as the duration of hypotension increased [8]. Similar exposure-dependent relationships have been reported in other observational cohorts using different MAP thresholds [2,4]. These findings make the lowest recorded pressure alone a poor description of hypotension exposure and support considering both its depth and duration.

Acute kidney injury is among the outcomes most consistently associated with IOH [2,4,9]. Associations have also been reported for myocardial injury and myocardial infarction, while findings for mortality, stroke, delirium, and other postoperative complications are less uniform [9]. A systematic review and meta-analysis of 72 studies reported associations between IOH and several major postoperative outcomes, including 30-day mortality, acute kidney injury, myocardial injury, myocardial infarction, stroke, and postoperative delirium [9]. Most of the included studies, however, were observational, and the certainty of evidence was low or very low for several outcomes [9]. The reported associations should therefore not be interpreted as proof that IOH directly caused these complications.

This limitation is clinically relevant because trials comparing different intraoperative blood pressure targets have not reproduced the observational findings with the same consistency [5]. Epidemiological data support limiting substantial hypotension exposure, but they do not identify a single treatment target that has been shown to improve outcomes in all patients [2,5]. The implications of this discrepancy for blood pressure management are discussed later in this review.

Table 1. Postoperative outcomes associated with intraoperative hypotension: findings from observational studies and blood pressure-target trials

Outcome	Observational findings	Findings from BP-target trials	Overall interpretation
Acute kidney injury	Repeated association with increasing depth and duration of IOH	Higher or individualized targets have not consistently reduced AKI	Association is well documented, but benefit from routinely higher

			targets remains unproven
Myocardial injury	Exposure-dependent association reported in large cohorts	Higher targets have not reduced myocardial injury consistently	Association is well described; causal and therapeutic implications remain uncertain
Mortality	Higher risk reported with greater hypotension exposure	No consistent mortality benefit from higher targets	Association is influenced by patient and perioperative risk; causal contribution remains uncertain
Postoperative delirium	Association reported, but results are heterogeneous	Recent randomized evidence suggests possible benefit from higher targets	Potential treatment signal, but current evidence remains insufficient for a firm conclusion

Abbreviations: AKI, acute kidney injury; BP, blood pressure; IOH, intraoperative hypotension.

Source: Authors' own elaboration based on current evidence [4,7,8].

5. Diagnosis and Risk Stratification

Recognition of intraoperative hypotension depends partly on how blood pressure is measured and how often measurements are obtained. Intermittent oscillometric monitoring is sufficient for many patients undergoing non-cardiac surgery and remains widely used in routine practice. Its limitation is straightforward: blood pressure between consecutive measurements is unknown, so short hypotensive episodes may be missed or detected with delay. Continuous arterial pressure monitoring provides a more complete record and allows rapid changes to be recognized earlier. Current consensus recommendations suggest considering continuous arterial pressure monitoring because it can reduce the severity and duration of IOH compared with intermittent measurements [2]. Randomized evidence also suggests that continuous non-invasive blood pressure monitoring can reduce intraoperative hypotension compared with intermittent cuff measurements, although the available trials are relatively small [11]. Whether better detection and control of hypotension also improve postoperative outcomes remains uncertain [2].

Once hypotension is detected, crossing a predefined MAP threshold is only the starting point of the assessment. The blood pressure trend and the setting in which the decrease occurs are also relevant. An isolated fall after induction differs from recurrent or persistent hypotension during a prolonged operation or ongoing blood loss. Absolute MAP thresholds remain useful in everyday practice, but they neither identify the cause of hypotension nor provide a direct measure of organ perfusion [2,3]. Additional assessment becomes particularly important when hypotension persists, recurs, or cannot be readily explained by standard monitoring.

Risk assessment begins before induction. Older age is among the factors most consistently associated with IOH, while hypertension, diabetes, treatment with renin–angiotensin system inhibitors, higher ASA status, and emergency surgery have also been associated with IOH in pooled analyses [10]. The available evidence includes heterogeneous surgical populations and definitions, which limits direct comparison between studies [10]. No single preoperative characteristic reliably predicts which patient will develop hypotension. In practice, several patient- and procedure-related factors need to be considered together.

Preoperative assessment should consider not only the likelihood of IOH, but also its potential consequences. The latter may be particularly important in patients with limited cardiovascular or renal reserve and in those undergoing high-risk surgery [2,3]. A similar reduction in arterial pressure may therefore have different clinical importance in different patients. These

considerations can influence how closely blood pressure is monitored and whether additional hemodynamic information is needed during the procedure.

Advanced hemodynamic monitoring is not required in every patient. In selected high-risk situations, stroke volume (SV) or cardiac output (CO) measurements can provide information that is not available from arterial pressure alone, especially when the hemodynamic disturbance is unclear. Current ESAIC guidance suggests considering SV or CO monitoring in patients with a high baseline risk of complications and in those undergoing high-risk non-cardiac surgery [3]. Routine maximization of SV or CO is not recommended [3]. Instead, these variables can be used to better characterize the hemodynamic state and assess the response to interventions when standard monitoring provides insufficient information.

Predictive monitoring has been developed to identify patients at risk of an imminent hypotensive episode. The Hypotension Prediction Index (HPI) uses features derived from the arterial pressure waveform to estimate the likelihood of impending hypotension. Randomized studies have shown that HPI-guided strategies can reduce hypotension exposure in some surgical populations [12], although this effect has not been consistent across all trials. More recent pooled evidence suggests an overall reduction in several measures of hypotension exposure, including its duration and time-weighted burden [13]. Evidence that improved blood pressure control translates into fewer major postoperative complications remains inconclusive [13]. HPI may therefore help clinicians recognize and respond to impending hypotension earlier, but it should not yet be regarded as a strategy proven to prevent postoperative organ injury.

Table 2. Factors relevant to risk assessment and hemodynamic monitoring in intraoperative hypotension

Domain	**Factors to consider**	**Clinical relevance**	**Monitoring consideration**
Patient-related factors	Older age, higher ASA status, hypertension,	Associated with increased susceptibility to IOH in pooled analyses	Consider as part of the overall preoperative risk assessment

	diabetes, ACEI/ARB use		
Procedural context	Emergency surgery; procedures with expected major hemodynamic changes	Emergency surgery is associated with IOH; major hemodynamic changes may require closer surveillance	Consider more frequent or continuous BP monitoring when rapid changes are expected
BP pattern	Persistent, recurrent, or rapidly changing hypotension	Intermittent measurements may incompletely characterize BP changes	Continuous BP monitoring provides a more complete record
Hemodynamic uncertainty	Hypotension not adequately explained by standard monitoring	Arterial pressure alone may not identify the underlying hemodynamic disturbance	Consider SV or CO monitoring in selected high-risk patients or procedures

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; ASA, American Society of Anesthesiologists; BP, blood pressure; CO, cardiac output; IOH, intraoperative hypotension; SV, stroke volume

Source: Authors' own elaboration based on current evidence and perioperative consensus recommendations [2,3,9]

6. Management and Follow-up

Management of intraoperative hypotension should aim to correct clinically relevant reductions in arterial pressure without treating every decrease in MAP in the same way. Current recommendations support maintaining intraoperative MAP at approximately 60 mmHg or higher, particularly in patients at increased perioperative risk [2,3]. This is a practical lower reference point rather than an optimal target for every patient [2,3].

Treatment should reflect the likely cause of hypotension [2,3]. Vasodilation, hypovolaemia, changes in heart rate, and impaired cardiac output can produce similar reductions in MAP but require different responses. Vasopressors are appropriate when reduced vascular tone predominates, whereas fluid administration is more likely to be useful when intravascular volume depletion is present [2,3]. More than one mechanism may be involved, particularly during major surgery. The response to the initial intervention should therefore be reassessed before treatment is escalated.

Fluid administration requires particular care. A low MAP does not necessarily indicate hypovolaemia, and fluid responsiveness alone is not an indication for fluid loading [3]. ESAIC guidance supports fluid administration when clinical or metabolic findings suggest hypovolaemia or tissue hypoperfusion rather than solely on the basis of a positive fluid responsiveness test [3]. This distinction matters in vasodilatory hypotension and impaired cardiac function, where repeated fluid administration may increase intravascular volume without addressing the main hemodynamic problem.

Vasopressors remain central when reduced vascular tone is the main cause of hypotension, but no single agent has established superiority across non-cardiac surgical populations [2,3]. Phenylephrine and norepinephrine differ in their hemodynamic effects, which may influence drug choice in individual patients. Evidence for differences in postoperative outcomes is less convincing. In the multicentre VEGA-1 feasibility trial, norepinephrine and phenylephrine were not associated with significant differences in major postoperative outcomes, although the study was not powered to establish clinical superiority [14]. Vasopressor choice should therefore be guided mainly by the hemodynamic disturbance rather than an assumed outcome advantage of one agent over another.

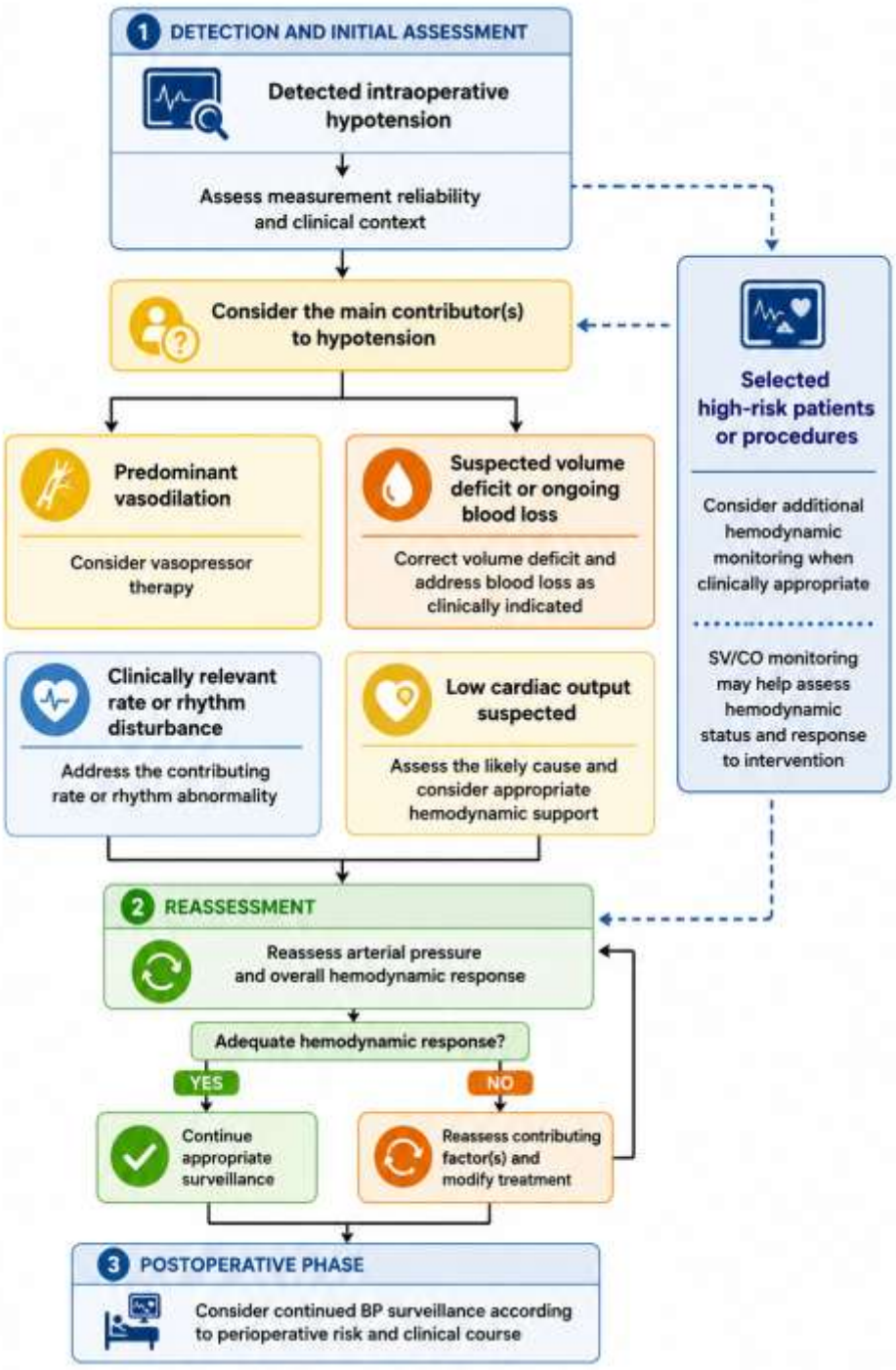
Higher blood pressure targets may be appropriate in selected patients, but there is currently no validated method for defining an optimal MAP for an individual patient [2,3,5]. Management

should also be reassessed once arterial pressure has improved, as restoration of MAP does not necessarily mean that the underlying hemodynamic disturbance has been corrected [2,3]. In selected high-risk situations, stroke volume or cardiac output monitoring may provide useful additional information, although routine maximization of these variables is not recommended [3].

Hemodynamic surveillance may still be necessary after surgery. Postoperative hypotension can go undetected because blood pressure is usually measured less frequently outside the operating room, allowing some episodes to persist before recognition [2,15]. Postoperative hypotension has also been associated with myocardial injury and other adverse outcomes after non-cardiac surgery [15,16]. However, clinically relevant postoperative thresholds and the optimal monitoring strategy remain less clearly established than for the intraoperative period [2]. Closer observation may be appropriate after substantial intraoperative instability, ongoing blood loss, or persistent circulatory abnormalities.

The need for further postoperative assessment depends on baseline risk, the extent of intraoperative instability, and the subsequent clinical course. A brief episode that resolves promptly in an otherwise stable patient is different from prolonged or recurrent hypotension during major surgery. Persistent hemodynamic abnormalities or signs of organ dysfunction require further evaluation according to the clinical context.

Figure 1. Cause-directed assessment and management of intraoperative hypotension



The framework summarizes principles derived from current perioperative hemodynamic recommendations. Several mechanisms may contribute to hypotension simultaneously, and management should be adapted to the clinical context and response to treatment. The figure represents a conceptual framework and should not be interpreted as a validated treatment algorithm.

Abbreviations: BP, blood pressure; CO, cardiac output; IOH, intraoperative hypotension; SV, stroke volume.

Source: Authors' own elaboration based on current POQI and ESAIC recommendations [2,3].

7. Current Challenges and Future Perspectives

A major limitation of the literature on intraoperative hypotension is the gap between observational and interventional evidence. Large cohort studies consistently associate deeper and longer hypotension with postoperative organ injury, but trials aimed at reducing hypotension or maintaining higher arterial pressures have not shown equally consistent clinical benefits [2,4,5,8,9]. IOH may contribute to postoperative injury, but it may also reflect severe illness, surgical complexity, blood loss, or cardiovascular dysfunction. Observational studies cannot fully separate these effects, making it difficult to determine how much of the associated risk can actually be reduced by preventing or treating hypotension [2,9].

Randomized evidence on individualized blood pressure management is also mixed. In the INPRESS trial, individualized systolic blood pressure management based on the patient's preoperative resting pressure reduced a composite measure of postoperative organ dysfunction compared with standard management in high-risk patients undergoing major surgery [17]. However, this finding has not been reproduced consistently across subsequent trials, and differences in patient populations, blood pressure targets, and intervention strategies complicate direct comparison between studies [5,17,18].

The IMPROVE-multi randomized trial illustrates this uncertainty. In this study, 1,142 high-risk patients undergoing major abdominal surgery were randomized to individualized or routine perioperative blood pressure management. Individualized management based on preoperative nighttime MAP did not reduce the composite of acute kidney injury, acute myocardial injury, nonfatal cardiac arrest, or death within 7 days compared with routine management targeting MAP ≥65 mmHg [18]. This result does not support this particular method of setting an individualized blood pressure target, but it does not rule out other approaches to individualized hemodynamic management.

MAP also has limitations as a treatment target because it does not identify the mechanism responsible for hypotension. Current recommendations therefore favour treatment according to the presumed hemodynamic cause rather than the pressure value alone [2,3]. Whether

additional hemodynamic variables can provide more useful treatment targets than MAP remains uncertain.

The same uncertainty applies to newer monitoring and treatment technologies. Continuous blood pressure monitoring, predictive systems, and automated vasopressor delivery can improve blood pressure control and reduce hypotension exposure [2,11–13,19,20]. Closed-loop vasopressor systems, for example, increase the time spent within predefined pressure targets and reduce time in hypotension compared with manual titration [19,20]. However, the available randomized trials remain relatively small, and it is uncertain whether better hemodynamic control translates into fewer clinically important postoperative complications [19,20]. Better control of a physiological variable should not be assumed to provide clinical benefit. Future trials should therefore focus on organ injury, major complications, and mortality rather than mainly on blood pressure metrics. They should also account for baseline risk, surgical setting, and the mechanism responsible for hypotension.

Postoperative hypotension remains less well studied than IOH. Observational evidence associates postoperative hypotension with myocardial injury and other adverse outcomes [15,16], but clinically relevant pressure thresholds are not well established, and it remains unclear which patients benefit from more intensive monitoring or earlier treatment [2]. The appropriate duration and intensity of postoperative blood pressure surveillance are also uncertain, particularly after major surgery or substantial intraoperative hemodynamic instability [2]. Strategies developed for the operating room cannot automatically be assumed to apply after surgery.

A single MAP value is unlikely to define an optimal treatment target for all patients [2,3,5]. Future studies should determine whether clinically relevant thresholds can be identified for particular patients or organs, which episodes of hypotension represent modifiable risk, and which patients benefit from earlier or more intensive intervention. Another important question is whether treatment guided by the underlying hemodynamic disturbance improves outcomes compared with management based mainly on arterial pressure thresholds.

8. Conclusions

Intraoperative hypotension is common during non-cardiac surgery and is consistently associated with postoperative organ injury, particularly when reductions in arterial pressure are deeper or prolonged. These associations are strongest for acute kidney injury and myocardial

injury, although observational evidence alone cannot establish how much of this risk is directly caused by hypotension.

A single MAP threshold is unlikely to represent an optimal treatment target for every patient. Current evidence supports avoiding substantial hypotension while considering the likely hemodynamic cause, the duration and severity of the episode, and the patient's baseline risk. More intensive or predictive monitoring can reduce hypotension exposure, but improved blood pressure control has not consistently resulted in better postoperative outcomes.

The main challenge is therefore not simply to maintain a predefined arterial pressure, but to identify which hypotensive episodes are clinically important and potentially modifiable. Current evidence supports a cause-directed approach to treatment, while the clinical benefit of more individualized hemodynamic strategies remains to be established.

Declaration of the use of generative AI and AI-assisted technologies in the writing process

During the preparation of this manuscript, the author used ChatGPT (OpenAI) to assist with language refinement, improvement of clarity and readability, and manuscript structuring and formatting. After using this tool, the author critically reviewed and edited the content as needed and takes full responsibility for the final content of the manuscript.

References

1. Weinberg L, Li SY, Louis M, Karp J, Poci N, Carp BS, et al. Reported definitions of intraoperative hypotension in adults undergoing non-cardiac surgery under general anaesthesia: a review. *BMC Anesthesiol.* 2022;22:69. doi:10.1186/s12871-022-01605-9.

2. Saugel B, Fletcher N, Gan TJ, Grocott MPW, Myles PS, Sessler DI; PeriOperative Quality Initiative XI (POQI XI) Workgroup Members. PeriOperative Quality Initiative (POQI) international consensus statement on perioperative arterial pressure management. *Br J Anaesth.* 2024;133(2):264–276. doi:10.1016/j.bja.2024.04.046.

3. Saugel B, Buhre W, Chew MS, Cholley B, Coburn M, Cohen B, et al. Intra-operative haemodynamic monitoring and management of adults having noncardiac surgery: a statement from the European Society of Anaesthesiology and Intensive Care. *Eur J Anaesthesiol.* 2025;42(6):543–556. doi:10.1097/EJA.0000000000002174.

4. Salmasi V, Maheshwari K, Yang D, Mascha EJ, Singh A, Sessler DI, Kurz A. Relationship between intraoperative hypotension, defined by either reduction from baseline or absolute thresholds, and acute kidney and myocardial injury after noncardiac surgery: a retrospective cohort analysis. *Anesthesiology.* 2017;126(1):47–65. doi:10.1097/ALN.0000000000001432.

5. Chiou K, Vikram S, Rowshan K, Shah S, Modha D, Engelman E, et al. Higher versus routine intraoperative blood pressure targets in noncardiac surgery: a systematic review and meta-analysis with trial sequential analysis of randomized trials. *Anesthesiology.* 2026. doi:10.1097/ALN.0000000000006190.

6. Kouz K, Hoppe P, Briesenick L, Saugel B. Intraoperative hypotension: pathophysiology, clinical relevance, and therapeutic approaches. *Indian J Anaesth.* 2020;64(2):90–96. doi:10.4103/ija.IJA_939_19.

7. Terwindt LE, Tol JTM, van der Ven WH, Kurucz VC, Noteboom SH, Breel JS, et al. The influence of anesthetic drug strategy on the incidence of post-induction hypotension in elective, non-cardiac surgery: a prospective observational cohort study. *J Clin Anesth.* 2024;99:111671. doi:10.1016/j.jclinane.2024.111671.

8. Walsh M, Devereaux PJ, Garg AX, Kurz A, Turan A, Rodseth RN, et al. Relationship between intraoperative mean arterial pressure and clinical outcomes after noncardiac surgery: toward an empirical definition of hypotension. *Anesthesiology.* 2013;119(3):507–515. doi:10.1097/ALN.0b013e3182a10e26.

9. Cai J, Tang M, Wu H, Yuan J, Liang H, Wu X, et al. Association of intraoperative hypotension and severe postoperative complications during non-cardiac surgery in adult patients: a systematic review and meta-analysis. *Heliyon.* 2023;9(5):e15997. doi:10.1016/j.heliyon.2023.e15997.

10. Daum N, Bill D, Thiele M, Felber J, von Wedel D, Spies C, et al. Preoperative patient risk factors for intraoperative hypotension: a systematic review and meta-analysis. *Front Cardiovasc Med.* 2025;12:1709004. doi:10.3389/fcvm.2025.1709004.

11. Juri T, Suehiro K, Kimura A, Mukai A, Tanaka K, Yamada T, et al. Impact of continuous non-invasive blood pressure monitoring on hemodynamic fluctuation during general

anesthesia: a randomized controlled study. *J Clin Monit Comput.* 2018;32(6):1005–1013. doi:10.1007/s10877-018-0125-4.

12. Wijnberge M, Geerts BF, Hol L, Lemmers N, Mulder MP, Berge P, et al. 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.* 2020;323(11):1052–1060. doi:10.1001/jama.2020.0592.

13. Piscopo R, Giustiniano E, Meco M, Nisi F, Dalla Corte F, Piccioni F, Cecconi M. Does Hypotension Prediction Index limit the occurrence of postoperative complications associated with intraoperative hypotension? A systematic review and model-averaged Bayesian meta-analysis of statistically sound studies. *J Anesth.* 2026. doi:10.1007/s00540-026-03741-9.

14. Legrand M, Kothari R, Fong N, Palaniappa N, Boldt D, Chen LL, et al.; VEGA-1 Trial Investigators. Norepinephrine versus phenylephrine for treating hypotension during general anaesthesia in adult patients undergoing major noncardiac surgery: a multicentre, open-label, cluster-randomised, crossover, feasibility, and pilot trial. *Br J Anaesth.* 2023;130(5):519–527. doi:10.1016/j.bja.2023.02.004.

15. Liem VGB, Hoeks SE, Mol KHJM, Potters JW, Grüne F, Stolker RJ, van Lier F. Postoperative hypotension after noncardiac surgery and the association with myocardial injury. *Anesthesiology.* 2020;133(3):510–522. doi:10.1097/ALN.0000000000003368.

16. Khanna AK, Shaw AD, Stapelfeldt WH, Boero IJ, Chen Q, Stevens M, et al. Postoperative hypotension and adverse clinical outcomes in patients without intraoperative hypotension, after noncardiac surgery. *Anesth Analg.* 2021;132(5):1410–1420. doi:10.1213/ANE.0000000000005374.

17. Futier E, Lefrant JY, Guinot PG, Godet T, Lorne E, Cuvillon P, et al.; INPRESS Study Group. 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.* 2017;318(14):1346–1357. doi:10.1001/jama.2017.14172.

18. Saugel B, Meidert AS, Brunkhorst FM, Bischoff R, Esser J, Mattis M, et al.; IMPROVE-multi Trial Group. Individualized perioperative blood pressure management in patients undergoing major abdominal surgery: the IMPROVE-multi randomized clinical trial. *JAMA.* 2025;334(21):1893–1904. doi:10.1001/jama.2025.17235.

19. Chiou K, Patrick P, Srivastava I, Engelman E, Rinehart J, Joosten A, Coeckelenbergh S. Closed-loop versus manual vasopressor titration for blood pressure control in surgical and critically ill patients: a systematic review and meta-analysis of randomised trials. *J Clin Anesth.* 2026;112:112230. doi:10.1016/j.jclinane.2026.112230.

20. Alvarado-Sánchez JI, Abril-Calderón DM, Sánchez-Quimbayo I, Oñoro-Patiño L, Vega-Useche C, Reyes-Gonzales JS, Carreño-Hernandez FL. Closed-loop vasopressor systems for hemodynamic control in perioperative and critical care settings: a systematic review and meta-analysis. *J Clin Monit Comput.* 2026. doi:10.1007/s10877-026-01463-7.