Review Article • บทฟื้นฟูวิชาการ

Intraoperative Hypotension in Non–cardiac Surgery: a Comprehensive Review of Contemporary Evidence and Management Strategies

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Abstract

Background: Intraoperative hypotension (IOH) is a frequent and critical modifiable risk factor in patients undergoing non-cardiac surgery, exhibiting strong correlations with postoperative organ dysfunction and increased mortality. This comprehensive review synthesizes contemporary evidence regarding the definitions, pathophysiological mechanisms, risk factors, and proactive management strategies for IOH.

Methods: A comprehensive literature review was conducted, analyzing pivotal clinical trials, systematic reviews, and international guidelines published up to 2026 (including ESAIC 2025, ACC/AHA 2024, and AAGBI/BIHS 2026) focusing on hemodynamic management in non-cardiac surgical populations.

Results: The definition of IOH remains heterogeneous, although recent 2025 ESAIC guidelines recommend an absolute intraoperative mean arterial pressure (MAP) threshold of 60-65 mmHg to protect organ perfusion. Despite growing interest in individualized blood pressure targets, recent randomized controlled trials (such as PRETREAT and IMPROVE-multi) and meta-analyses demonstrate that strict individualization or higher MAP targets do not significantly improve long-term functional outcomes or reduce postoperative complications compared to standard targets (≥ 60-65 mmHg). In medication management, recent trial data (POISE-3, SPACE, STOP-OR-NOT) support the 2026 AAGBI/BIHS recommendations to continue ACEIs/ARBs on the day of surgery to mitigate postoperative hypertension without increasing severe complications. For intraoperative monitoring and intervention, goal-directed fluid therapy utilizing dynamic indices (SVV/PPV) and the implementation of AI-driven predictive analytics, such as the Hypotension Prediction Index, offer advanced frameworks to proactively detect and manage impending hypotensive episodes before organ injury occurs. Furthermore, the newly emerged concept of "Protective Hemodynamics" via the 5-step C.L.E.A.R. pathway provides a structured approach to multi-organ preservation.

Conclusion: Effective management of IOH requires a paradigm shift from reactive treatment to proactive prevention. Maintaining a target MAP ≥ 60-65 mmHg, optimizing anesthetic techniques, implementing dynamic fluid protocols, and integrating AI predictive software constitute the core pillars to minimize adverse perioperative outcomes in non-cardiac surgery.

Keywords: goal-directed fluid therapy, hypotension prediction index, intraoperative hypotension, mean arterial pressure, noncardiac surgery, protective hemodynamics

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Background

Each year, more than 300 million surgical procedures are performed worldwide[1], with non-cardiac surgery accounting for approximately 85% of these cases. The incidence of severe complications and mortality during the early postoperative weeks remains profoundly high. Postoperative mortality, which stems from subsequent complications, can affect up to 25% of patients undergoing inpatient surgery. To prevent these complications, identifying and mitigating modifiable risk factors is of paramount importance. Among these, intraoperative hypotension (IOH) represents a critical modifiable risk factor.[1]

Although intraoperative hypotension is common during anesthesia, numerous studies have demonstrated its strong association with adverse postoperative outcomes. These include myocardial injury after non-cardiac surgery (MINS), myocardial infarction, cardiogenic shock, acute kidney injury (AKI) which may later progress to chronic kidney disease, perioperative stroke, delirium, postoperative nausea and vomiting, hospital readmission, intensive care unit admission, and increased mortality rates in both cardiac and non-cardiac surgeries.[1-24] The severity and frequency of these complications exhibit an increased odds ratio (OR) correlated with the depth and duration of IOH, as explicitly highlighted in the 2024 APSF (Anesthesia Patient Safety Foundation) guidelines.[3] In a study by Salmasi et al., patients with a cumulative duration of more than 13 min with a mean arterial pressure (MAP) < 65 mmHg (representing 50% of the study cohort) experienced an elevated risk of myocardial injury and AKI.[6] Furthermore, "Triple Low" state (low Bispectral Index, low MAP, and low minimum alveolar concentration) is coupled with increased mortality rates.[14] Consequently, the 2022 European Society of Cardiology guidelines emphasized that preventing and correcting episodes of IOH is a pivotal responsibility.[15]

Definition

Currently, a universal definition for IOH has not yet been established. Furthermore, the medical community has not determined whether IOH should be defined based on absolute arterial blood pressure thresholds or relative thresholds calculated as a percentage decrease from baseline blood pressure. A systematic review identified more than 140 distinct definitions of IOH across 130 studies; these definitions relied on systolic blood pressure (SBP) or MAP, absolute values or relative changes, or a combination of these criteria. Some authors argue that SBP reflects tissue perfusion and perioperative hemodynamic abnormalities better than MAP, as it serves as an early indicator of hemodynamic shifts, cardiac output, stroke volume, organ perfusion, and systolic cardiac function.[1]

Commonly utilized definitions include: an SBP < 80 mmHg, a greater than 20% reduction in SBP from baseline, and a combined definition involving an absolute SBP below 100 mmHg and/or a 30% reduction from baseline. Depending on the applied definition of IOH, the incidence varies extensively, ranging on average from 40% to 90%, with some studies reporting rates

between 5% and 99%.[1] Even if IOH is defined as a MAP < 65 mmHg for 1 min (with some definitions requiring a duration of ≥ 5 min), a criterion widely used in both clinical practice and clinical trials-up to 88% of patients monitored continuously via invasive arterial lines still exhibit hypotension. However, the recent guideline recommends using intraoperative MAP as the standard for assessing and managing IOH.[27] This preference arises because MAP reflects the perfusion pressure for most bodily organs and is less influenced by measurement techniques, location, or artifact interference compared to systolic and diastolic blood pressures. The MAP threshold specified in these guidelines is 60 mmHg.

In a survey conducted by Bækgaard et al., a MAP threshold of either 60 or 65 mmHg was most frequently utilized by clinicians to guide initial diagnosis and vasopressor administration.[28] Approximately one-third of intraoperative hypotensive episodes occur during the interval between anesthesia induction and surgical incision; this condition may be termed post-induction hypotension or pre-incision hypotension.[1] Consequently, managing this specific window represents a critical foundation for IOH prevention. Post-induction hypotension must be distinguished from hypotensive episodes occurring during the surgical procedure itself, as their underlying etiologies differ. While post-induction hypotension is primarily driven by anesthetic management, IOH occurring intraoperatively can be attributed to multifactorial elements related to both anesthesia administration and the surgical intervention itself.

Optimal or individualized blood pressure management

Given that IOH is associated with postoperative complications and mortality, a pivotal remaining question is which arterial blood pressure (ABP) threshold is optimal for each specific patient undergoing surgery under anesthesia. Based on data from large retrospective cohort studies, an absolute MAP threshold of 65 mmHg has been recommended as the minimum intervention trigger.[1] This is because intraoperative MAP values below this limit are linked to organ injury in the non-cardiac surgery population.

However, applying this absolute MAP threshold universally ignores the reality that normal baseline ABP values vary widely among individuals. Many patients, particularly those with chronic arterial hypertension and elderly populations (MAP > 70 mmHg should be maintained to mitigate the risk of major adverse postoperative cardiac events; however, the precise MAP threshold for patients over 80 years old remains undetermined) may require higher MAP levels.[17] Furthermore, the optimal MAP depends on organ-specific perfusion thresholds, surgical progress (e.g., patient repositioning, arterial clamping, hemorrhage), and the type of procedure. The INPRESS trial, the only RCT comparing individualized SBP management against standard care during general anesthesia for major surgery, demonstrated a significant reduction in the risk of postoperative organ dysfunction in the individualized group compared to the standard management group.[31] Sun et al. defined dynamic MAP thresholds

(< 60/65/70 mmHg) to individualize IOH and perioperative hypotension (POH) management, aiming to minimize postoperative organ injury and improve perioperative outcomes.[18]

Another fundamental question concerns which baseline value a patient's intraoperative target MAP or ABP should be modeled after. Pre-induction MAP values should not be utilized as a surrogate for an individual's normal daily MAP; instead, ambulatory or sleeping blood pressure values (true baseline blood pressure) should be adopted. Bergholz et al. utilized pre-operative nighttime MAP thresholds rather than the standard 65 mmHg target in patients undergoing major non-cardiac surgery.[32] However, the IMPROVE-multi-trial demonstrated that utilizing a preoperative nighttime MAP threshold failed to reduce complications within the first 7 postoperative days in high-risk patients undergoing major abdominal surgery, compared to a standard MAP threshold of 65 mmHg or higher.[33]

Moreover, proactive IOH prevention incorporating individualized MAP thresholds (70/80/90 mmHg) tailored to specific patient risk profiles (low/moderate/high) failed to improve functional disability at 6 months post-operatively compared to standard reactive treatment triggered at a MAP of 65 mmHg in the PRETREAT RCT. Notably, this trial was terminated early due to futility.[21] In a meta-analysis, IOH management based on moderate (60-75 mmHg) versus high (> 75 mmHg) MAP thresholds yielded no statistically significant differences in postoperative complications.[35] Similarly, a recent review concluded that a MAP threshold of ≥ 60-65 mmHg remains appropriate for patients undergoing non-cardiac surgery, rather than shifting toward higher or strictly individualized targets.[36]

Pathophysiology

Multiple pathophysiological mechanisms can lead to IOH in surgical patients. IOH may arise from vasodilation (e.g., secondary to anesthetic agents or systemic inflammatory response), intravascular hypovolemia (e.g., due to hemorrhage), low cardiac output (e.g., driven by bradycardia or low stroke volume), elevated intrathoracic pressure (e.g., from mechanical ventilation), sympathetic nervous system impairment, or compromised baroreflex autoregulation. Consequently, the etiologies underlying IOH are highly diverse (Figure 1).[36]

Etiology

Perioperative hypotension (POH) is frequently influenced by factors such as patient health status, anesthesia management, and surgical modality. Elevated risk is notably associated with advanced age, low preoperative blood pressure, and the chronic use of antihypertensive medications. Intraoperative hypotension (IOH) typically correlates with anesthetic depth, vasodilation, and blood loss. Postoperative hypotension, which occurs in approximately 20% of patients, is commonly linked to myocardial ischemia, hypovolemia, sepsis, or other clinical complications.

MAP (mean arterial pressure), CO (cardiac output), SVR (systemic vascular resistance), CVP (central venous pressure)

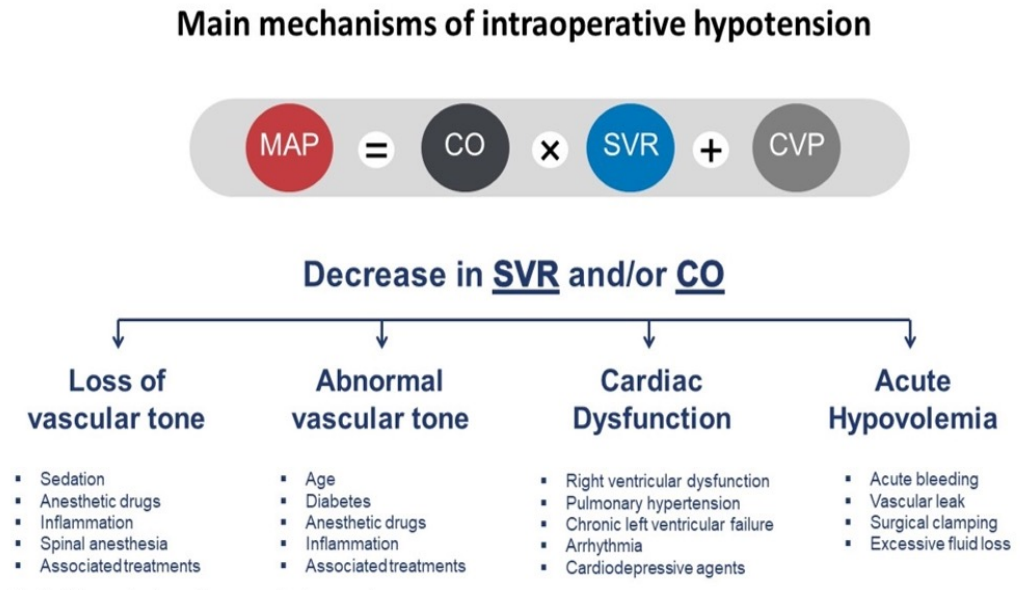


Figure 1 Main mechanisms of intraoperative hypotension (Reproduced with permission from Joosten et al.36)

Risk factors

Numerous risk factors for IOH have been established, including advanced age, elevated ASA physical status classification (III or IV), male sex, low pre-induction SBP and MAP, long-term antihypertensive therapy, multiple systemic comorbidities, the continuation of angiotensin-converting enzyme inhibitors (ACEIs) and angiotensin II receptor blockers (ARBs) on the day of surgery, propofol-based general anesthesia, combined general and regional anesthesia, prolonged operative duration, high-risk surgery, and emergency procedures. Conversely, peripheral nerve plexus blocks are considered a protective factor against IOH compared to general anesthesia. Classically, antihypertensive medications such as ACEIs, ARBs, beta-blockers, and alpha-2 agonists are known to exacerbate the risk of hypotensive episodes during anesthesia induction and maintenance, which has been linked to adverse cardiovascular outcomes and mortality.

However, the POISE-3[37], SPACE[38], and STOP-OR-NOT[39] trials all concluded that there was no significant difference in major complications (e.g., myocardial injury) between patients who withheld versus those who continued ACEIs/ARBs up to the day of surgery. The PREOP-ACEI trial showed that withholding these medications reduced intraoperative and postoperative hypotension but increased the incidence of postoperative hypertension.[40] Similarly, the STOP-OR-NOT trial demonstrated

an increased rate of intraoperative hypotension in the continuation group.[39] These trials formed the cornerstone for the 2026 guidelines, which recommend continuing all antihypertensive therapies, including ACEIs and ARBs, on the day of surgery, as this approach does not alter complication rates while successfully mitigating postoperative hypertension.[41]

Notably, the 2024 ACC/AHA guidelines recommend a tailored approach regarding medication withholding. Withholding these agents for high-risk surgeries may be beneficial to minimize hypotension, whereas continuation remains reasonable in patients with heart failure and chronic kidney disease.[42] This individualized management was also highlighted; they noted that withholding medications should be considered in cases involving prolonged surgery with high anticipated blood loss, and decisions should also depend on institutional protocols, preoperative NT-proBNP levels, and the anesthetic modality.[45] Continuing beta-blocker therapy is recommended for patients on a stable dose based on specific clinical scenarios. Conversely, initiating beta-blocker therapy on the day of surgery for patients without a clear clinical indication is strongly discouraged due to an increased risk of postoperative mortality.[46] Furthermore, the 2024 KDIGO guidelines recommend withholding these medications in vulnerable patients but emphasize the critical need for early postoperative resumption, particularly in patients with heart failure and proteinuric kidney disease. This approach may represent the safest equilibrium between renal protection and hemodynamic stability.[48]

Prevention

Preoperative risk assessment and stratification

Identification of high-risk patients: Patients categorized as high-risk include elderly populations, individuals with an elevated ASA physical status classification, chronic hypertension, heart failure, long-standing diabetes mellitus (which induces autonomic neuropathy), or those undergoing emergency surgery.

Cardiovascular medication management: Detailed in Risk factors.

Optimizing anesthetic techniques during anesthesia induction

Early co-administration of vasopressors: Rather than waiting for hypotensive episodes to occur, current clinical trends favor the early co-administration of low-dose vasopressors immediately upon induction, particularly with propofol-based induction (e.g., combining low-dose phenylephrine or norepinephrine) for major or high-risk surgeries to stabilize vascular tone. Trocheris-Fumery et al. utilized low-concentration diluted norepinephrine (0.016 mg/mL) administered via continuous intravenous infusion at a rate of 0.48 mg/h or 8 mcg/min.[50] Meanwhile, Thomsen et al. utilized a higher concentration of norepinephrine (3 mg/50 mL of 0.9% NaCl, equating to 0.06 mg/mL) with a variable infusion rate ranging from 0.03 mcg/kg/minute to 0.2 mcg/kg/min.[30]

Selection of hemodynamically safe induction agents: Etomidate or ketamine should

be utilized as alternatives to propofol in hemodynamically unstable patients or those with heart failure.

Dose titration in spinal anesthesia: In cesarean sections or lower-body surgeries, reducing the local anesthetic dose and combining it with opioids minimizes sympathetic blockade, thereby mitigating vasodilation-induced hypotension.

Optimizing anesthetic depth monitoring during maintenance of anesthesia

The 2025 ESAIC guidelines recommend the utilization of neuromonitoring such as electroencephalography, near-infrared spectroscopy (NIRS), to monitor and optimize anesthetic depth, titrate anesthetic dosing, and minimize adverse events. Notably, NIRS is specifically indicated for patients at high risk for cerebral hypoperfusion.[27]

Goal-directed fluid therapy (GDFT)

Avoidance of fluid overload: Aggressive fluid loading as a prophylactic measure against hypotension is no longer recommended, as it causes tissue edema and respiratory failure.

Utilization of dynamic indices: The 2025 ESAIC guidelines recommend using dynamic parameters, such as stroke volume variation (SVV) or pulse pressure variation (PPV), to accurately assess whether a patient is genuinely fluid-responsive, while discouraging the use of central venous pressure (CVP) and the passive leg raising (PLR) test.[27]

Fluid challenge: The 2025 ESAIC guidelines recommend that fluid boluses should only be administered when clinical evidence of hypovolemia or tissue hypoperfusion is present, integrated with continuous stroke volume (SV) or cardiac output (CO) monitoring to evaluate fluid responsiveness.[27]

For elective major abdominal surgeries, the 2025 ESAIC guidelines recommend appropriate fluid administration and note that a positive fluid balance of up to 1-2 L at the end of surgery is tolerable. This is because intraoperative fluid balance in these procedures depends on multifactorial elements, including occult blood loss and the prevention of AKI.[27]

Echocardiography

The 2025 ESAIC guidelines recommend the utilization of echocardiography (transthoracic echocardiography or transesophageal echocardiography) if patients exhibit intraoperative or postoperative hemodynamic instability, particularly when there is a lack of response to initial therapy or when the underlying mechanism of the event remains unclear.[27]

Continuous prophylactic vasopressor infusion

Low-dose continuous intravenous infusion: Commencing a continuous infusion of norepinephrine or phenylephrine immediately following anesthesia induction or regional blockade helps maintain stable systemic vascular resistance.

Phenylephrine: This remains the first-line choice in obstetric anesthesia (cesarean section) because it minimizes fetal acidosis and

reduces maternal postoperative nausea and vomiting.

Microcirculation

The 2025 ESAIC guidelines recommend measuring blood lactate levels and, when feasible, central venous oxygen saturation ($ScvO_2$) if tissue hypoperfusion or hypoxia is clinically suspected.[27]

Application of AI-driven predictive analytics

Hypotension Prediction Index (HPI): This represents a major technological advancement developed by Hatib et al.[51] The AI-driven algorithm analyzes invasive (or continuous non-invasive) arterial waveform morphology to provide a warning of impending hypotensive risks 5-15 min prior to onset. When the HPI reading is elevated, clinicians can proactively intervene to address the underlying etiology (whether driven by vasodilation, hypovolemia, or myocardial dysfunction) before the MAP drops below 65 mmHg. However, a recent meta-analysis noted that the implementation of HPI software during abdominal surgery did not improve definitive patient outcomes.[52]

Impact of intermittent pneumatic peristaltic compression

Studies have demonstrated that the application of intermittent pneumatic peristaltic compression devices on the lower extremities reduces the incidence of hypotensive episodes and decreases the total volume of intravenous fluid required.[1]

Management

Due to the robust correlation between IOH and adverse postoperative outcomes, immediate management of IOH is imperative. The choice of therapeutic interventions remains a subject of ongoing debate, as it remains unclear which specific treatment strategy significantly alters clinical outcomes. Common therapeutic approaches rely on vasoactive agents, particularly vasopressors and fluid resuscitation. However, a definitive consensus has yet to be reached regarding which specific vasopressor should be preferred to increase vascular tone and arterial blood pressure intraoperatively. Large-scale randomized controlled trials are urgently warranted to determine the most effective management strategy for IOH.[1]

Nevertheless, a novel concept termed "Protective Hemodynamics" has recently emerged to minimize the risks associated with hypotension management. This hemodynamic preservation approach incorporates a 5-step strategy designed to optimize organ protection, known by the acronym C.L.E.A.R. (C = customize targets, L = limit catecholamines, E = enhance flow focusing on microcirculation and regional blood flow, A = adjust fluid balance guided by echocardiography with optimal cardiac output and microvascular blood flow, R = resolve underlying conditions)[14,53]

For patients without advanced monitoring, IOH management can be guided by clinical algorithms from NYSORA (Table 1).[54] Notably, the 2025 ESAIC guidelines recommend that

Table 1 NYSORA management algorithm for IOH[54]

NYSORA management algorithm for IOH (www.nysora.com	
Hypotension (MAP < 65 mmHg): Rapid correction of perioperative hypotension is essential to safeguard cerebral, spinal, and organ perfusion ↓	
Avoid excessive depth of anesthesia ↓	
Inform the surgeon and ventilate with 100% O_2 ↓	
Cycle BP & scan monitor for HR, rhythm, ST changes, SaO_2, $etCO_2$, PAP ↓	
Manage fluids (crystalloid solution) ↓	
Administer vasopressors and positive inotropic agents Ephedrine: 5-10 mg bolus Phenylephrine: 50-100 mcg bolus/continuous infusion 10-110 mcg/min Vasopressin: 1-4 units bolus dose/continuous infusion at 0.01-0.04 units/min Norepinephrine: 4-8 mcg bolus/continuous infusion at 1-20 mcg/min Epinephrine: 8-16 mcg bolus dose Terlipressin: 1mg ↓	
Trendelenburg position ↓	
Pulse absent • Perform advanced life support	**Pulse present** • Assess tracheal deviation, JVD, subcutaneous crepitus, bilateral air entry • ↑ CVP/ PCWP: obstructive or cardiogenic • ↓ CVP/ PCWWP: hypovolemic or distributive • Peripheral perfusion • Look at the surgical field for blood loss, IVC compression

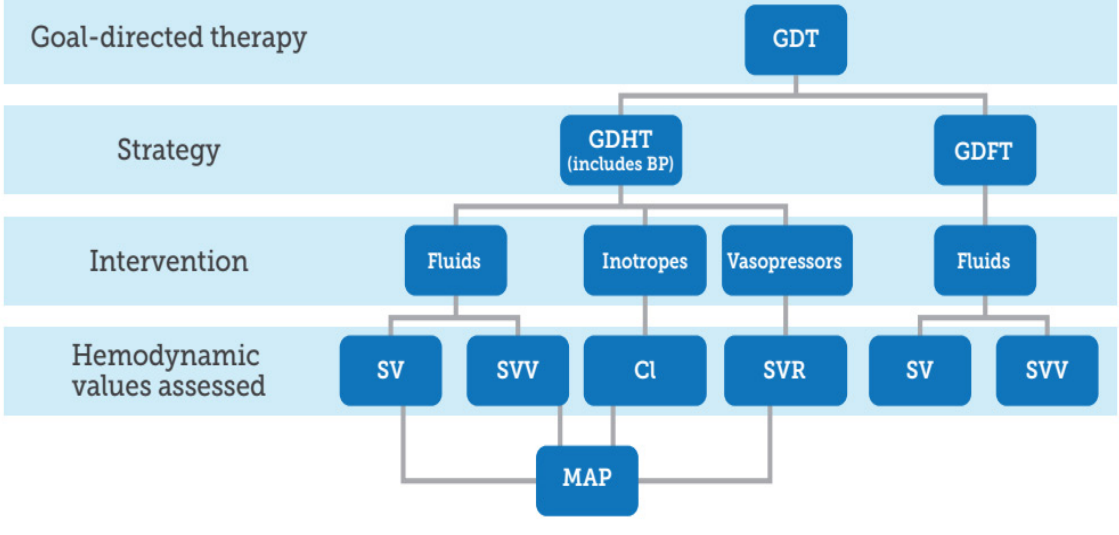
intraoperative blood pressure must be measured at least every 3 to 5 min when using non-invasive blood pressure (NIBP) cuffs. If invasive ABP monitoring is indicated, placing a radial artery catheter under local anesthesia prior to anesthesia induction is preferred to minimize complications. Furthermore, these guidelines emphasize that arterial blood pressure (whether invasive or non-invasive) must not be utilized as a surrogate for CO.[27]

In patients with intraoperative hemodynamic monitoring, the 2025 ESAIC guidelines recommend advanced hemodynamic monitoring for surgical patients with severe

cardiopulmonary disease (e.g., severe heart failure, severe pulmonary hypertension) and those undergoing high-risk surgeries (e.g., liver transplantation).[27] In these scenarios, IOH management can be directed by synthesizing and analyzing hemodynamic parameters based on the availability of the monitoring device coupled with clinical data. Accordingly, fluid administration, vasopressors, and inotropic therapies are delivered via goal-directed therapy (GDT) (Table 2 and Figures 2 and 3).

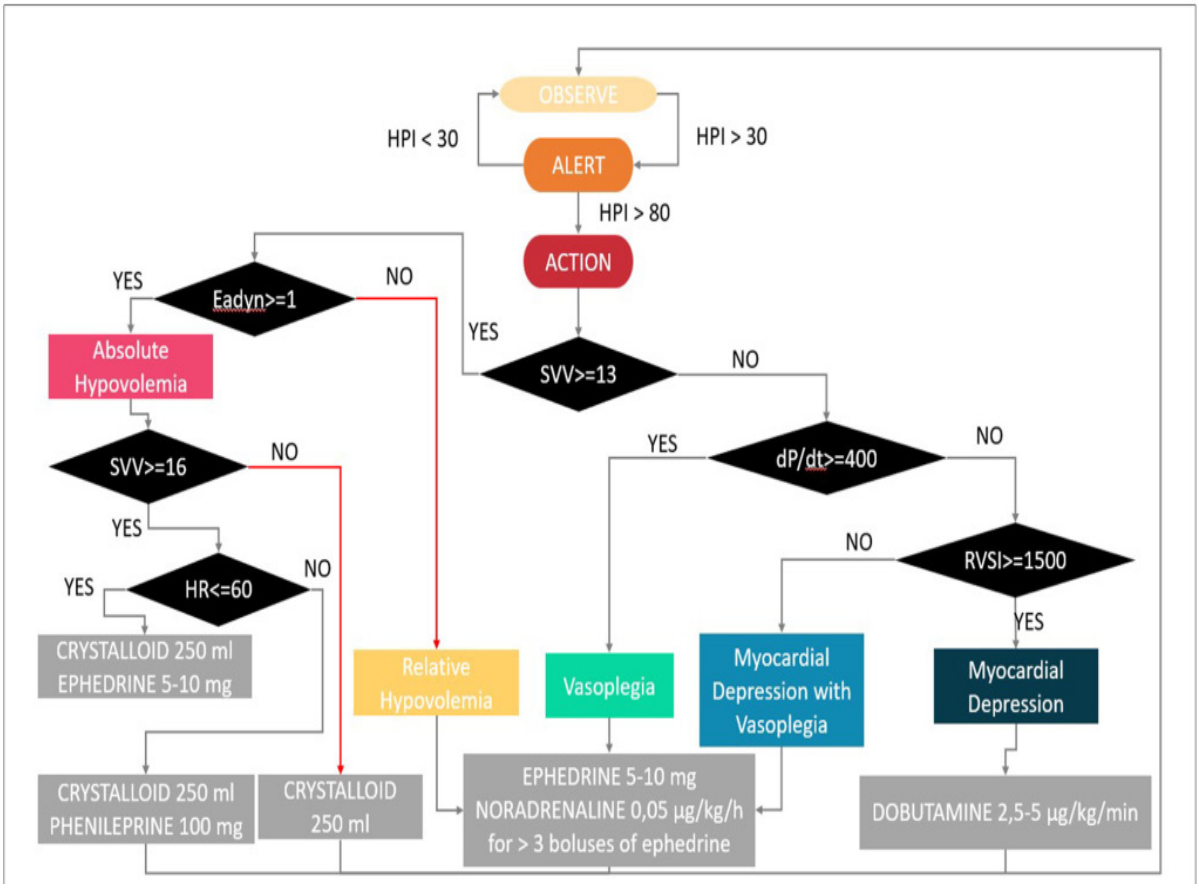
Table 2 Five cardiovascular pathophysiological phenotypes[55]

Five cardiovascular pathophysiological phenotypes analyzed via hemodynamic monitoring of intraoperative hypotension	
Pathophysiological phenotypes	**Corresponding hemodynamic parameters**
Relative hypovolemia	SVV > 13% (stroke volume variation) + Eadyn < 1 (dynamic arterial elastance)
Absolute hypovolemia	SVV > 13% + Eadyn > 1
Vasoplegia	SVV < 13% + dP/dt > 400 mmHg/s (peak arterial pressure derivative)
Myocardial depression	SVV < 13% + dP/dt < 400 mmHg/s + SVRI > 1,500 dyn.s/cm^5/m^2 (systemic vascular resistance index)
Myocardial depression with vasoplegia	SVV < 13% + dP/dt < 400 mmHg/s + SVRI < 1,500 dyn.s/cm^5/m^2



GDHT (goal-directed hemodynamic therapy), GDFT (goal-directed fluid therapy), SVV (stroke volume variation), CI (cardiac index), SVR (systemic vascular resistance)

Figure 2 General principles of GDT involving fluid resuscitation, vasopressors, and inotropic agents (Reproduced with permission from Yerdon et al.[2])



HPI (hypotension prediction index), Eadyn (dynamic arterial elastance), dP/dt (peak arterial pressure derivative), RVSI (systemic vascular resistance index), SVV (stroke volume variation)

Figure 3 Hemodynamic management based on HPI, SVV%, Eadyn, RVSI, and dP/dt via the Acumen IQ Sensor, HemoSphere platform, and hemodynamic monitoring (Reproduced with permission from Valbuena-Bueno et al.[55])

In patients monitored via hemodynamic monitors, the AI-driven HPI software, utilized through the invasive Acumen IQ Sensor (or the continuous non-invasive Acumen IQ Cuff) paired with the HemoSphere platform, integrates and analyzes dynamic parameters including MAP, HPI, SVV%, Eadyn, dP/dt, CI, and SVRI. Based on these algorithmic calculations, specific diagnostic and therapeutic pathways are directed.[55,56] Figure 3 and figure 4 illustrate a representative example of a management algorithm integrating HPI and hemodynamic monitoring.

Currently, both invasive and non-invasive versions of the HPI software (introduced in 2018 and granted US FDA clearance in June 2021) demonstrate exceptionally high diagnostic sensitivity and specificity (approximately 90%). Despite certain limitations, it has been widely implemented worldwide.[4,11,13,51,57,61,62,64-67] Research indicates a strong correlation between HPI values and MAP; specifically, an HPI reading

of 85% corresponds to a patient MAP of approximately 72-76 mmHg.[4,14,24] Mulder et al. outlined a trial comparing MAP-guided versus HPI-guided strategies for IOH prevention in patients undergoing elective moderate-to-major non-cardiac surgery.[60] Francis et al. designed a trial evaluating clinical outcomes in brain tumor surgeries complicated by IOH with and without the utilization of HPI tools.[63] Furthermore, HPI has been applied to guide norepinephrine weaning in patients experiencing post-cardiac surgery vasoplegic shock (which affects 9-44% of cases).[5] Managing IOH via AI-driven predictive analytics also contributes to reducing cognitive burden, stress, and burnout syndrome among anesthesia practitioners.[7]

During major liver resection, minimizing intraoperative blood loss is critical, as hemorrhage and blood transfusions are strongly associated with worsened surgical prognoses. Consequently, a GDFT strategy is employed, utilizing dynamic SVV% targets (titrated to 13-15% or up to 20% during the hepatic dissection, transection, and suturing phases) (Figure 5).[68-70]

Conclusion

IOH remains one of the most critical and actionable modifiable risk factors directly tied to adverse postoperative outcomes, including MINS, AKI, and increased short- and long-term

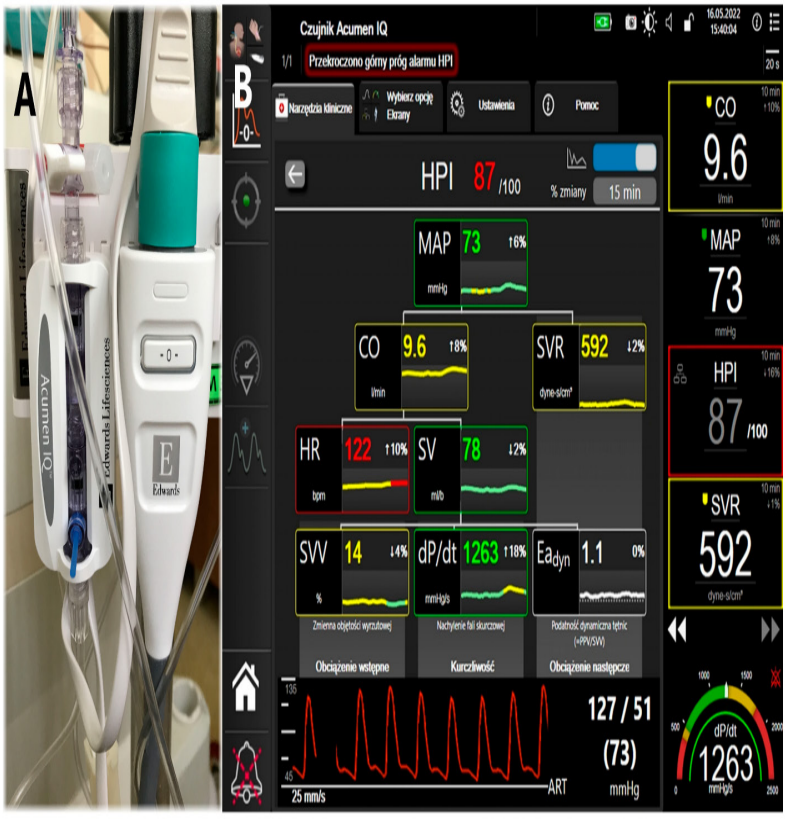


Figure 4 Acumen IQ Sensor, HemoSphere platform, and hemodynamic monitoring equipment (Reproduced with permission from Cylwik et al.[56])

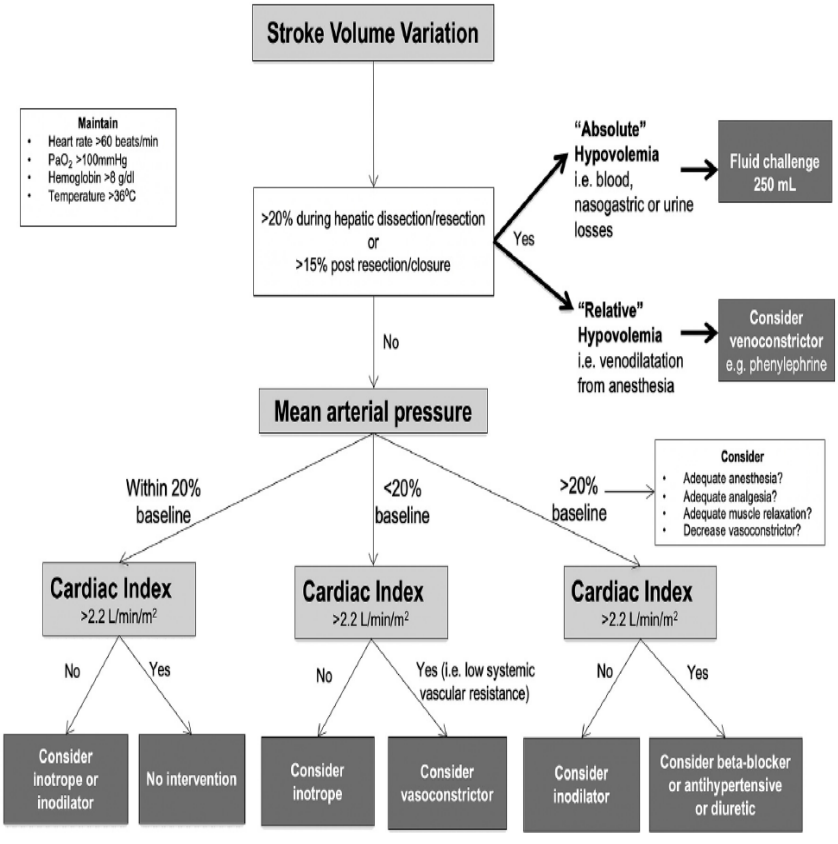


Figure 5 Intraoperative blood loss mitigation strategy: Dynamic stroke volume variation (SVV) adjustments (13-15% to 20%) utilizing the FloTrac sensor on the EV1000 monitor during hepatic dissection, transection, and suturing phases to guide GDFT (Reproduced with permission from Weinberg et al.[70])

mortality in non-cardiac surgery. Contemporary consensus-spearheaded by the 2025 ESAIC guidelines, converges on maintaining an absolute intraoperative MAP threshold of ≥ 60-65 mmHg as the standard for adequate organ perfusion. Strictly individualized or aggressively higher blood pressure targets do not definitively yield superior clinical outcomes over a standard protective target, and that continuing chronic ACEI/ARB therapies on the day of surgery represents a safe equilibrium to mitigate postoperative hypertension. To effectively mitigate the burden of IOH, clinical practice must undergo a decisive paradigm shift from traditional, reactive treatments to proactive, and multi-modal prevention strategies. The integration of the novel "Protective Hemodynamics" framework, and the advent of artificial intelligence and machine learning, empowers clinicians to forecast and preemptively correct impending hypotensive episodes. Ultimately, a tailored combination of these advanced technological tools, rigorous guideline adherence, and a deep understanding of patient-specific pathophysiology constitute the essential pillars to enhance perioperative safety and improve the prognosis of patients undergoing non-cardiac surgery.

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