**Anestezjologia • Ratownictwo • Nauka • Praktyka / Anaesthesiology • Rescue Medicine • Science • Practice**

**ARTYKUŁ POGLĄDOWY / REVIEW PAPER**
Otrzymano/Submitted: 10.04.2026 • Zaakceptowano/Accepted: 03.06.2026


# *Post-induction hypotension – risk factors and preventive strategies. A literature review*

**Aleksandra Polańska[1], Mikołaj Polewka[1], Filip Przybył[1], Karol Piotrowski[1], Małgorzata Jerczak[1], Tomasz Pluszyński[1], Natalia Padula[1], Iga Piórkowska[1], Kacper Paczosa[1]**
[1] Medical University of Silesia in Katowice, Faculty of Medical Sciences in Katowice

## Abstract

Post-induction hypotension (PIH) is a significant decrease in arterial blood pressure occurring shortly after anesthetic administration, typically before surgery begins. As one of the most frequent forms of perioperative instability, PIH is associated with increased risk of organ injury and higher mortality. This review examines the multifactorial pathophysiology, including intravascular volume, cardiac performance, and autonomic regulation. Modern risk stratification goes beyond clinical history, incorporating point-of-care ultrasonography, dynamic hemodynamic assessment, and non-invasive autonomic monitoring. Identifying high-risk patients enables a shift from reactive management to a proactive, physiology-based strategy. Individualized fluid therapy, precise drug titration, and preemptive vasopressor use are key to enhancing patient safety during anesthetic induction.
*Anestezjologia i Ratownictwo 2026; 20: 140-147. doi:10.53139/AIR.20262015*

*Keywords: post-induction hypotension, general anesthesia, risk factors, prevention, point-of-care ultrasound*

## Introduction

Post-induction hypotension (PIH) represents one of the earliest and most frequent forms of hemodynamic instability during general anesthesia, typically occurring within 10-20 minutes after anesthetic administration [1-4]. Nearly one-third of intraoperative hypotensive episodes arise during the interval between induction and surgical incision [5-8]. Despite its high prevalence, there is no universally accepted definition of PIH. Most commonly, it is defined as a mean arterial pressure (MAP) below 65 mmHg or a 20-30% reduction in MAP or systolic blood pressure (SBP) from baseline values [2,4,6,9-11].

Reported incidence varies widely depending on patient characteristics and surgical context, ranging from 17-18% in general surgical populations [12] to as high as 70-87% among patients receiving renin-angiotensin-aldosterone system (RAAS) inhibitors [13]. Even brief MAP reductions below 60-70 mmHg have been associated with organ hypoperfusion [14,15] and increased risk of acute kidney injury, myocardial injury, and perioperative mortality [8,15-20].

This review aims to summarize current evidence on the pathophysiology of PIH, identify patients at increased risk, and discuss available strategies for its prevention and management.

## Materials and methods

This narrative review was based on a structured search of PubMed and Embase using the terms ("post-induction hypotension" AND "risk factors") OR ("post-induction hypotension" AND "prevention"). Articles published between January 2020 and January 2026 were screened. Studies addressing the pathophysiology, risk factors, prediction, prevention, or management of post-induction hypotension in adult patients undergoing general anesthesia were included. After removal of duplicates and exclusion of irrelevant

studies or unavailable full texts, 39 articles were included. The selected literature comprised randomized trials, observational studies, and review articles, and the evidence was synthesized narratively.

## Pathophysiology of PIH

PIH arises from the interaction between anesthetic-induced cardiovascular depression and limited patient-specific hemodynamic reserve. At its core lies a disruption of autonomic nervous system (ANS) regulation, which, under physiological conditions, maintains vascular tone, myocardial contractility, and venous return [21].

Induction agents acutely suppress sympathetic activity and blunt baroreflex-mediated compensatory responses. Propofol produces dose-dependent hypotension through combined systemic vasodilation and negative inotropy, with effects persisting up to 10 minutes after administration [22]. Direct vascular smooth muscle relaxation increases venous capacitance and reduces preload, while myocardial depression further lowers cardiac output [8,23]. In addition, propofol impairs baroreflex sensitivity, limiting the capacity to counteract reductions in systemic vascular resistance [9,14]. Thiopental exerts similar hemodynamic effects and additionally inhibits medullary vasomotor centers, further contributing to hypotension [24]. Opioids, such as fentanyl and remifentanil, reduce sympathetic outflow via μ-opioid receptor activation, promoting bradycardia and amplifying blood pressure reductions [2,25].

These pharmacologic effects are amplified in the presence of relative hypovolemia. Occult volume depletion - resulting from prolonged fasting, preoperative bowel preparation, or nasogastric drainage - frequently predisposes patients to marked blood pressure reductions after induction [3,20,26,27]. Positive pressure ventilation, particularly when combined with PEEP (Positive End-Expiratory Pressure), can further impair venous return, thereby exacerbating preload reduction [14,15,23].

Finally, impaired cardiovascular reserve limits compensatory capacity. Elderly patients and those with hypertension or diabetes often exhibit autonomic dysfunction and reduced baroreflex responsiveness [11,28]. Structural cardiac abnormalities - including regional wall motion abnormalities (RWMA), elevated left ventricular filling pressures (E/e'), and reduced end-diastolic volume - limit the Frank-Starling reserve, thereby diminishing tolerance to pharmacologic vasodilation [1,22,29]. Chronic beta-blocker or angiotensin receptor blocker (ARB) therapy alters autonomic tone and attenuates catecholamine responsiveness, rendering patients particularly vulnerable to peri-induction hemodynamic instability [10,17,30].

## Risk factors for PIH

PIH is influenced by several categories of risk factors, including demographic and clinical characteristics, baseline hemodynamic and metabolic parameters, intravascular volume status, and pharmacologic or procedural variables. These factors rarely occur in isolation and result in a common outcome: reduced capacity to compensate for the drop in sympathetic tone and systemic vascular resistance during induction. The goal of identifying them is practical - to enable risk stratification and anticipation of hypotension before induction rather than its treatment after onset.

### 1. Demographic and patient-related characteristics

Older age (>50-60 years) is the most consistent predictor of PIH [24], and risk rises steadily - in one cohort the adjusted odds of PIH were ~2.8-fold higher at ≥30 years and ~3.5-fold higher at ≥60 years versus 18-29 years [24]**.** Age, however, mainly reflects reduced physiological reserve rather than being a risk factor in itself, which is why frailty predicts PIH even after adjustment for age, American Society of Anesthesiologists (ASA) class and comorbidities [31]. Frailty can be assessed in clinical practice using a brief validated tool, such as the 5-item FRAIL scale (fatigue, resistance [ability to climb stairs], ambulation, illnesses, and loss of weight), scored from 0 to 5. A positive frailty score should place the patient in a high-risk category regardless of chronological age, as it more accurately reflects reduced physiological reserve than age alone.

Body mass index (BMI) shows a U-shaped relationship with PIH: both low and high BMI are associated with greater risk, and a moderate BMI appears most protective [32]. A low BMI is the more clinically useful warning sign, as it marks limited physiological reserve - consistent with reports linking lower body mass to PIH [33]. In practice, a low BMI deserves attention and overweight should not be taken as reassurance.

Overall clinical status, commonly summarized by ASA physical status ≥ II, is associated with PIH mainly because it reflects accumulated comorbidities and

reduced physiological reserve; in a prospective cohort, ASA ≥ II was an independent predictor of post-induction hypotension [24]. However, the label itself is less important than the functional impairment behind it, and identifying specific organ reserve is more useful than relying on the score alone.

The comorbidities most strongly associated with PIH are those that impair cardiac and renal reserve. Markers of myocardial dysfunction appear particularly relevant: preoperative RWMA were associated with a markedly increased risk of PIH (OR ≈ 6.6), as was an elevated E/e' ratio indicating diastolic dysfunction [22]. Reduced renal function has also been linked to lower post-induction blood pressure in a large prospective cohort [29]. Clinically, this translates into treating a history of ischemic heart disease, heart failure, left ventricular dysfunction, or chronic kidney disease as an a priori high-risk state. Early recognition therefore allows hemodynamic protection to be initiated from the moment of induction.

**2. Baseline hemodynamic and metabolic parameters**

Pre-induction hemodynamic status often provides more clinically relevant information than baseline medical history. A low baseline SBP and MAP combined with an elevated resting heart rate are strong predictors of PIH [10,12] - with tachycardia frequently reflecting compensated hypovolemia that becomes unmasked after induction. Conversely, a higher baseline blood pressure appears protective [1].

Importantly, baseline blood pressure should be interpreted relative to the patient's usual values rather than population norms. In this context, the combination of low MAP and elevated heart rate may indicate limited compensatory reserve, warranting cautious induction dosing and careful assessment of intravascular volume status.

Laboratory markers provide additional and potentially modifiable information. Elevated total cholesterol (≥5.2 mmol/L) was independently associated with increased PIH risk (~1.58-fold; 50.6% vs 27.3% incidence in high- versus normal-cholesterol patients) [5], reflecting endothelial dysfunction and reduced vascular responsiveness to vasodilation. Similarly, anemia and hypoalbuminemia indicate reduced physiological reserve and chronic disease burden [12] and should be considered during preoperative optimization whenever possible.

**3. Intravascular volume status**

Of the factors that can actually be changed, preoperative hypovolemia is the most important - to the point that PIH has been described as "a fluid relationship" [14]. The volume deficit is often hidden: prolonged fasting, aggressive bowel preparation before abdominal surgery, or trauma-related losses reduce preload and only become clinically apparent once anesthetic-induced vasodilation occurs [7,18,20,26]. A related and easily missed contributor in older patients is postprandial hypotension, which reflects poor autonomic handling of blood pooling in the gut and may flag patients prone to exaggerated peri-induction falls [21]. Because volume can be assessed and corrected beforehand, this is the most actionable factor of all. The practical detail is covered in the prediction and prevention sections.

**4. Chronic medications**

Chronic pharmacotherapy also plays a significant role: long-term use of angiotensin-converting enzyme inhibitors (ACEI), ARBs, and beta-blockers attenuates compensatory cardiovascular responses, limiting the ability to restore blood pressure via increased cardiac output [10,13,22]. For risk assessment, chronic RAAS or beta-blockade is best read as pre-blunted compensation that warrants extra caution, with particular weight given to long-acting agents; whether to withhold them is addressed under prevention.

**5. Induction phase**

The induction technique is itself a determinant of PIH, because unlike age, comorbidities, or baseline physiology, the choice and dose of induction agents are modifiable factors introduced by the anesthesiologist. Propofol and thiopental carry the highest hypotensive profile, and the effect is dose-dependent and mediated predominantly by a reduction in systemic vascular resistance rather than by myocardial depression [8]; so higher bolus doses are associated with greater risk. Hypnotics with a more stable hemodynamic profile - remimazolam, etomidate, and ketamine/esketamine - are associated with a substantially lower incidence of PIH, an advantage most pronounced precisely in the patients already identified as vulnerable, namely the elderly and those with hypertension or cardiac disease [11,13,34]. The practical inference for risk stratification is that the same patient may move between risk categories according to the agent and dose selected, so the

planned induction should be regarded as part of the risk profile rather than as a separate matter.

Co-administered drugs modulate this risk further. High opioid doses deepen the sympatholytic, vasodilatory response, and neuromuscular blocking agents such as rocuronium may contribute additively to cardiovascular depression [8]. Recognizing these agents as risk-modifying rather than hemodynamically neutral matters because their combined effect can be anticipated before induction in a patient already flagged as high-risk; the corresponding strategies for their use are addressed in the section on therapeutic management.

**6. Procedural factors**

Procedural factors such as emergency surgery, highly invasive procedures, the lithotomy position, and prolonged induction increase the risk of PIH [1,3,30]. Higher risk is also seen in oncology patients [4] and those undergoing structural cardiac interventions such as transcatheter aortic valve implantation (TAVI) [35]. These factors are largely non-modifiable, so their main value is in guiding preparation and monitoring rather than intervention.

## Methods for predicting PIH

Contemporary anesthesiology increasingly aims to objectify the assessment of hemodynamic risk through advanced bedside diagnostics, dynamic testing, and artificial intelligence-based tools. Central to PIH prediction is a structured evaluation of three key domains: intravascular volume status, cardiovascular functional reserve and ANS balance prior to the administration of induction agents.

**1. Ultrasonographic assessment of intravascular status and vessels**

The initial step in risk stratification involves the assessment of preload and venous capacitance. Point-of-care ultrasonography (POCUS) plays a pivotal role in this context. One of the most widely used screening tools is inferior vena cava (IVC) ultrasonography, with the inferior vena cava collapsibility index (IVCCI), calculated as IVCCI = (maximum diameter during expiration - minimum diameter during inspiration)/ maximum diameter during expiration [27]. High IVCCI values (>50-70%) indicate hypovolemia, whereas values below 20% suggest normo- or hypervolemia. Golhar M et al. reported that, in spontaneously breathing patients, IVCCI was a strong predictor of PIH, with an ROC-derived optimal cut-off of ≥38% and a sensitivity of 95.5% [27].

When IVC assessment is limited or inconclusive, alternative venous measurements such as superior vena cava collapsibility/distensibility (SVCDI) or subclavian vein collapsibility index (SCVCI) may be employed, as they retain predictive accuracy even in patients with arrhythmias such as atrial fibrillation [7,36]. In elderly and non-ventilated patients, corrected carotid flow time (FTc) provides a reliable, noninvasive marker of circulating blood volume, with values below approximately 330-350 ms strongly identifying patients with reduced effective preload [18,20].

**2. Advanced assessment of cardiac function and dynamic parameters**

While venous and preload parameters identify patients with reduced circulating volume, they do not capture the myocardium's capacity to compensate for anesthetic-induced vasodilation. Therefore, evaluating intrinsic cardiac performance is essential for a comprehensive PIH risk assessment. Parameters such as cardiac output (CO), cardiac power output (CPO), and stroke volume index (SVI) provide more robust information regarding hypotension susceptibility than static variables alone [8,14,37]. Transthoracic echocardiography (TTE) can detect subclinical myocardial dysfunction, and additional echocardiographic predictors, such as RWMA and an elevated E/e' ratio (an index estimating left ventricular filling pressure), indicate diastolic dysfunction and impaired ventricular filling reserve [1,22,29].

Dynamic testing with the passive leg raise (PLR) using TTE allows measurement of ΔVTI - the change in velocity-time integral in the LVOT (left ventricular outflow tract). Aissaoui Y et al. demonstrated that an increase of ≥18% identifies patients at high risk of PIH, with a sensitivity of 88% [15]. By combining preload assessment with dynamic cardiac responses, clinicians can better determine which patients will benefit from pre-induction fluid therapy.

**3. Analysis of ANS function**

Beyond intravascular volume and cardiac performance, the integrity of autonomic regulation plays a critical role in maintaining blood pressure stability during anesthetic induction. Patients with impaired baroreflex sensitivity or sympathetic predominance

are particularly prone to exaggerated vasoplegia due to limited compensatory capacity [9].

Several noninvasive methods allow quantification of autonomic function and its impact on hemodynamic stability. Heart rate variability (HRV) analysis and deceleration capacity (DC) are commonly used to measure autonomic balance. DC derived from a 5-minute preoperative electrocardiogram may provide the highest predictive value, helping identify patients at elevated risk of peri-induction hypotension [9].

Preoperative pupillary light reflex (pPLR) assessment provides additional insight into autonomic responsiveness. Among pupillometric parameters, maximal constriction velocity (MCV) appears most predictive for PIH, with lower values indicating impaired autonomic regulation. In the study by Shao L et al., an MCV threshold of 0.316 s/mm strongly correlated with PIH occurrence [38]. Complementary to these measures, the pleth variability index (PVi) derived from pulse oximetry provides further information on intravascular responsiveness, allowing a multifaceted preoperative assessment [12].

**4. Machine learning models and nomograms**

Given the multifactorial and nonlinear nature of PIH risk, traditional statistical approaches are increasingly supplemented by machine learning techniques. Algorithms such as Random Forest and ensemble decision tree models integrate multiple variables - including age, ASA classification, induction drug doses, and baseline MAP - to estimate hypotension risk in real time [12,16,17].

These models facilitate construction of individualized risk nomograms, enabling proactive optimization of anesthetic management, such as tailored drug titration and early preventive interventions [1,2,16,35]. By combining advanced ultrasonography, cardiac assessment, autonomic evaluation, and machine learning, clinicians can achieve a comprehensive, individualized prediction of PIH, improving patient safety during induction.

## Prevention strategies and therapeutic management

Prevention of PIH has evolved from protocol-driven management toward individualized, physiology-guided strategies aimed at mitigating perioperative morbidity and mortality [9,13,22]. Rather than reacting to hypotension after it occurs, contemporary approaches focus on correcting modifiable vulnerabilities across four interrelated domains: preload optimization, myocardial protection, vascular tone preservation, and continuous risk surveillance.

**1. Pre-induction hemodynamic optimization**

optimization of intravascular volume prior to anesthetic administration constitutes the first modifiable target. Identifying fluid-responsive patients allows correction of relative hypovolemia before exposure to vasodilatory or negative inotropic agents while avoiding unnecessary fluid loading in patients unlikely to benefit. This is particularly important in elderly patients and those with impaired cardiac reserve, for whom excessive fluid administration may worsen cardiovascular status.

Several bedside parameters may help identify patients who are most likely to respond to pre-induction fluid therapy. Li M et al. established an FTc cut-off value of <340.7 ms and demonstrated that fluid therapy guided by this threshold significantly reduces the incidence of PIH by restoring effective preload before induction [20]. Similar reductions in hypotension - approaching 50% - have been observed when fluid administration is targeted to patients with increased IVCCI and a positive ΔVTI-PLR response, without increasing the risk of fluid overload [15,26,36]. Importantly, Aissaoui et al. found that ΔVTI-PLR had greater predictive value for PIH than IVCCI alone [15]. Li X et al. further demonstrated that, in patients with atrial fibrillation, a preoperative IVCCI >34.1% may serve as a useful noninvasive predictor of PIH [36].

In patients with limited cardiac reserve, where liberal fluid administration may be deleterious, intermittent pneumatic leg compression offers a non-pharmacologic alternative for augmenting venous return, improving early post-induction MAP stability, and reducing ephedrine requirements [39].

**2. Pharmacologic modulation of induction**

Pharmacologic strategy represents the second preventive axis. Since hemodynamic instability during induction is mainly caused by a sudden drop in systemic vascular resistance [8], the aim is to limit drug-induced circulatory effects through appropriate agent selection, careful titration, and controlled administration. Titration to clinical effect remains fundamental [14], and target-controlled infusion (TCI) enhances stability

relative to manual bolus dosing by achieving therapeutic concentrations more gradually, reducing the incidence of hypotensive episodes (13% vs 34%) [37].

In patients at increased risk - including the elderly, oncology patients, and those treated with ACEI or ARBs - propofol is not an obligatory induction agent, and substitution with a more hemodynamically stable hypnotic is a key preventive step [4,11,13,25,34]. Remimazolam is the best-supported alternative: in elderly hypertensive patients it substantially reduced the incidence of PIH compared with propofol (46.5% vs 73.2%) [11], produced significantly less PIH in patients on renin-angiotensin system blockade [13], and in cardiac surgery it reduced the area under the baseline-MAP curve during the first 10 minutes after induction by approximately 23% while lowering vasopressor requirements (60% vs 88% of patients) [34]. Its rapid metabolism by tissue esterases and reversibility with flumazenil make it particularly suitable for elderly or frail patients and for those with hepatic or renal dysfunction. Etomidate, which exerts minimal effect on myocardial contractility, is often preferred when cardiovascular reserve is limited. Compared with etomidate, propofol produces significantly more hypotension at induction, particularly in hypovolemic patients, and etomidate is specifically recommended as an alternative when the pre-induction MAP is below 70 mmHg [4]. However, its transient adrenocortical suppression supports reserving it for selected high-risk cases rather than routine use. Adjunctive agents, including lidocaine and low-dose esketamine (0.2 mg/kg), may provide additional attenuation of induction-related blood pressure decreases [6,19].

Other co-administered drugs also influence hemodynamic stability during induction. Opioids should be used in the lowest effective dose, as large boluses can worsen hemodynamic depression; in this context, remifentanil has been associated with less PIH than fentanyl in some cohorts [25]. Neuromuscular blocking agents such as rocuronium may further contribute to cardiovascular depression during induction and should therefore be used cautiously in already high-risk patients [8].

Overall, substituting remimazolam or etomidate in selected patients reduces both the incidence of PIH and the need for vasopressor support, but agent selection is an adjunct to - not a replacement for - careful dose titration, an individualized induction plan, and readiness to administer a vasopressor.

**3. Early vasopressor strategy**

Mounting evidence suggests that anesthetic-induced vasoplegia constitutes the predominant mechanism underlying PIH [8]. Consequently, proactive vasopressor administration has shifted from rescue therapy to a preventive strategy. Preemptive bolus dosing of phenylephrine, ephedrine, or norepinephrine before or during induction effectively reduces the incidence and severity of hypotension, particularly in patients with chronic hypertension and multiple comorbidities [2,8,23]. In patients with RWMA, prophylactic norepinephrine is especially valuable for maintaining coronary perfusion pressure and protecting the myocardium [29]. At the same time, excessive doses of opioids should be avoided, as opioid-induced vasodilation may exacerbate hemodynamic instability [4,35].

**4. Integrated monitoring and predictive systems**

The final component of modern prevention lies in dynamic monitoring and predictive analytics. Continuous noninvasive blood pressure monitoring systems (e.g., ClearSight) enable real-time detection of rapid hemodynamic deterioration [2,8].

Integration of machine learning models with electronic medical records facilitates early automatic identification of high-risk patients and supports timely preventive interventions [12,16,17]. By combining bedside diagnostics (POCUS, TTE) with predictive algorithms, clinicians can transition from reactive management to anticipatory hemodynamic control, thereby enhancing induction safety and reducing perioperative complications [1,15,22].

## Conclusions

PIH is a common and clinically significant complication of general anesthesia, resulting from the interaction between anesthetic-induced cardiovascular depression and limited patient-specific hemodynamic reserve. Even brief peri-induction hypotension is associated with adverse postoperative outcomes, highlighting the need for early identification and prevention.

Risk assessment should extend beyond demographic factors and comorbidities to include careful evaluation of baseline hemodynamic status. This begins with a thorough preoperative history to determine the patient's usual blood pressure values. In patients with chronic hypertension, post-induction values that appear normotensive in absolute terms may represent

a significant relative decrease and potentially lead to organ hypoperfusion if the patient is physiologically adapted to higher pressures. Further assessment of intravascular volume, myocardial function, and autonomic reserve enhances the identification of patients at increased risk.

Effective prevention requires correction of modifiable factors before induction, careful titration of anesthetic agents, and timely administration of vasopressors when indicated. Transitioning from reactive treatment to proactive, physiology-based hemodynamic management is essential for optimizing perioperative safety.

Conflict of interest
None

Correspondence address
Aleksandra Polańska
ul. Fabryczna 13A/20
40-611 Katowice
(+48) 692 216 516
polanska.aleksandra@o2.pl

## References

1. Xu Y, Tao L, Luo Y, et al. The role of echocardiographic volume parameters in predicting post-induction hypotension in geriatric patients under general anesthesia: a retrospective study. BMC Geriatr 2026;26:165.
2. Terwindt LE, Tol JTM, van der Ven WH, 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.
3. Grillot N, Gonzalez V, Deransy R, Rouhani A, Cintrat G, Rooze P et al. Post-induction hypotension during rapid sequence intubation in the operating room: A post hoc analysis of the randomized controlled REMICRUSH trial.Anaesth Crit Care Pain Med 2025;44: 101502.
4. Mohammad Shehata I, Elhassan A, Alejandro Munoz D, et al. Intraoperative Hypotension Increased Risk in the Oncological Patient. Anesth Pain Med 2021;11:e112830.
5. Yang Y, Gong C, Jiang H, et al. Association between elevated serum total cholesterol and increased risk of post-induction hypotension in elderly patients undergoing non-cardiac surgery: a retrospective cohort study.Lipids Health Dis 2025;24:338.
6. Elsherbiny M, Hasanin A, Kasem S, et al. Comparison of different ratios of propofol-ketamine admixture in rapid-sequence induction of anesthesia for emergency laparotomy: a randomized controlled trial. BMC Anesthesiol 2023;23:329.
7. Wang B, Hui K, Xiong J, et al. Effect of subclavian vein diameter combined with perioperative fluid therapy on preventing post-induction hypotension in patients with ASA status I or II. BMC Anesthesiol 2024;24:138.
8. Saugel B, Bebert EJ, Briesenick L, et al. Mechanisms contributing to hypotension after anesthetic induction with sufentanil, propofol, and rocuronium: a prospective observational study. J Clin Monit Comput 2022;36:341-7.
9. Qi H, Shi M, Wu T, et al. Short-term deceleration capacity of heart rate predicts post-induction hypotension in patients with low ASA status: a prospective observational study. BMC Anesthesiol. 2025;25:395.
10. Oh CS, Park JY, Kim SH. Comparison of effects of telmisartan versus valsartan on post-induction hypotension during noncardiac surgery: a prospective observational study. Korean J Anesthesiol. 2024;77: 335-44.
11. Zhou C, Wang X, Zhou M, et al. Comparison of Remimazolam and Propofol on Post-Induction Hypotension in Elderly Hypertensive Patients: A Randomized Controlled Trial. Drug Des Devel Ther 2025;19:3425-35.
12. Chen M, Zhang D. Machine learning-based prediction of post-induction hypotension: identifying risk factors and enhancing anesthesia management. BMC Med Inform Decis Mak 2025;25:96.
13. Song SW, Kim S, Park JH, et al. Post-induction hypotension with remimazolam versus propofol in patients routinely administered angiotensin axis blockades: a randomized control trial. BMC Anesthesiol 2023;23: 19.
14. Wong GTC, Irwin MG. Post-induction hypotension: a fluid relationship? Anaesthesia 2021;76:15-18.
15. Aissaoui Y, Jozwiak M, Bahi M, et al. Prediction of post-induction hypotension by point-of-care echocardiography: A prospective observational study. Anaesth Crit Care Pain Med 2022;41:101090.
16. Li XF, Huang YZ, Tang JY, et al. Development of a random forest model for hypotension prediction after anesthesia induction for cardiac surgery. World J Clin Cases 2021;9:8729-39.
17. Katsin M, Glebov M, Berkenstadt H, et al. Developing a machine learning-based prediction model for postinduction hypotension. J Clin Monit Comput 2025;39:889-99.
18. Huang S, Liao Z, Chen A, et al. Effect of carotid corrected flow time combined with perioperative fluid therapy on preventing hypotension after general anesthesia induction in elderly patients: A prospective cohort study. Int J Surg 2024;110:799-809.

19. Zhang CQ, Liu DQ, Chen BY, et al. Esketamine attenuates hemodynamic oscillations during anesthesia induction in elderly gastrointestinal surgical patients: a randomized trial. BMC Anesthesiol 2025;25:506.
20. Li M, Li F, Yu J, et al. The impact of pre-rehydration guided by carotid corrected flow time on hypotension prevention following general anesthesia induction in patients undergoing gastrointestinal surgery: a prospective randomized controlled trial. Front Med (Lausanne) 2024;11:1416574.
21. Li T, Zang H, Che L, Cui Q, Xu L. Correlation between postprandial hypotension and post-induction hypotension in the elderly: a protocol for a prospective cohort study. BMJ Open 2025;15:e095466.
22. Tarao K, Daimon M, Son K, et al. Risk factors including preoperative echocardiographic parameters for post-induction hypotension in general anesthesia. J Cardiol 2021;78:230-6.
23. B M, Botta B, Balasubramanian K, S S, S K. A Comparative Study of Preloading With Ringer's Lactate and Intravenous Ephedrine for the Prevention of Hypotension Due to Propofol During Induction of General Anesthesia. Cureus 2025;17:e89097.
24. Hulualem Nega M, Ahmed SA, Tawuye HY, Mustofa SY. Incidence and factors associated with post-induction hypotension among adult surgical patients: Prospective follow-up study. Int J Surg Open 2022;49: 00565.
25. Zhang J, Zhao C, Sun H, et al. Remifentanil reduces post-induction hypotension compared to fentanyl in thoracoscopic esophagectomy: a retrospective cohort study. Front Pharmacol 2025;16:1660228.
26. Dana E, Arzola C, Khan JS. Prevention of hypotension after induction of general anesthesia using point-of-care ultrasound to guide fluid management: a randomized controlled trial. Can J Anaesth 2024;71:1219-28.
27. Golhar M, Manisha, Yadav T, Johar S. Evaluation of Inferior Vena Cava Collapsibility Index as a Predictor of Hypotension Following General Anaesthesia with Thiopentone Induction: An Observational Study. J Clin Diagn Res 2023;17:UC17-UC21.
28. Zhang Y, Wang X, Sang X, et al. Effect of Fluid Therapy in Early Morning on the Incidence of Post-Induction Hypotension During Non-Cardiac Surgery After Noon: A Single-Center Retrospective Study. Drug Des Devel Ther 2024;18:1339-47.
29. Son K, Tarao K, Daimon M, et al. Preoperative echocardiography and anesthetic drugs as predictors of post-induction hypotension during general anesthesia: a prospective observational study. Sci Rep 2024;14:25717.
30. Tamsett Z, Douglas N, King C, et al. Does the choice of induction agent in rapid sequence intubation in the emergency department influence the incidence of post-induction hypotension? Emerg Med Australas 2024;36:340-7.
31. Yu J, Che L, Wang R, et al. Association of preoperative frailty and post-induction hypotension in elderly patients undergoing major non-cardiac surgery: a prospective observational cohort study. Clin Interv Aging 2025; 20:1283-92.
32. Wang Y, Wang J, Zhang Y, et al. Body mass index and risk of post-induction hypotension in older adults undergoing non-cardiac surgery: a retrospective cohort study. BMC Geriatr 2025;25:939.
33. Czajka S, Putowski Z, Krzych ŁJ. Post-induction hypotension and intraoperative hypotension as potential separate risk factors for the adverse outcome: a cohort study. J Anesth 2023;37:442-50.
34. Ju JW, Lee DJ, Chung J, et al. Effect of remimazolam versus propofol on hypotension after anesthetic induction in patients undergoing coronary artery bypass grafting: A randomized controlled trial. J Clin Anesth 2024;98:111580.
35. Noto K, Uchida S, Kinoshita H, et al. Predictive model for post-induction hypotension in patients undergoing transcatheter aortic valve implantation: a retrospective observational study. JA Clin Rep 2024;10:33.
36. Li X, Yang G, Hao S, et al. Ultrasonography evaluation of the inferior vena cava collapsibility index and the superior vena cava distensibility index for predicting hypotension during general anesthesia in atrial fibrillation patients: a prospective observational study. BMC Anesthesiol 2025;25:440.
37. Yildirim SA, Dogan L, Sarikaya ZT, et al. Hypotension after Anesthesia Induction: Target-Controlled Infusion Versus Manual Anesthesia Induction of Propofol. J Clin Med 2023;12:5280.
38. Shao L, Zhou Y, Yue Z, et al. Pupil maximum constriction velocity predicts post-induction hypotension in patients with lower ASA status: a prospective observational study. BMC Anesthesiol 2022;22:274.
39. Park JY, Yu J, Kim CS, et al. Effect of pneumatic leg compression on post-induction hypotension in elderly patients undergoing robot-assisted laparoscopic prostatectomy: a double-blind randomised controlled trial. Anaesthesia 2023;78:730-8.