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REVIEW

Peri-intubation Cardiovascular Collapse During Emergency Airway Management

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ABSTRACT

Emergency airway management is a lifesaving procedure but can be associated with significant risks, including hypoxia, hypotension, cardiac arrest, and death. Peri-intubation hypotension, reported in ≥ 40% of cases, is strongly associated with increased morbidity and mortality. While clinical guidelines emphasize the importance of preoxygenation and hemodynamic optimization prior to intubation, the latter remains poorly defined, with limited available data to guide evidence-based strategies to mitigate cardiovascular collapse during rapid sequence intubation. This review synthesizes current knowledge on the epidemiology, risk factors, and pathophysiology of peri-intubation hemodynamic deterioration. We review targeted strategies for hemodynamic optimization of physiologic parameters before intubation. These include volume expansion with fluid resuscitation, vasopressor utilization, selection of pharmacologic agents, invasive hemodynamic monitoring, and advanced preoxygenation techniques. In selected high-risk patients, we also discuss the potential role of extracorporeal membrane oxygenation as an adjunctive or rescue therapy. Our goal is to provide airway specialists with a comprehensive framework for mitigating cardiovascular collapse during emergent airway management and to stimulate further research into this high-risk and understudied domain.

Keywords: Emergency airway management; Peri-intubation hypotension; Rapid sequence intubation; Post-intubation adverse events

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Key Summary Points

Peri-intubation hypotension affects ≥40% of critically ill patients undergoing emergency airway management and is associated with increased morbidity and mortality.

While rapid sequence intubation is commonly performed in unstable patients, current guidelines offer limited practical guidance for mitigating hemodynamic collapse in this high-risk population.

This review summarizes the epidemiology, risk factors, and pathophysiologic determinants underlying peri-intubation cardiovascular deterioration, highlighting the need for a proactive and individualized management.

Key evidence-based strategies discussed include pre-intubation risk stratification, fluid resuscitation when indicated, push-dose and continuous vasopressor use, pharmacologic agent selection and dosing, preoxygenation methods, the role of invasive hemodynamic monitoring, and use of rescue strategies such as extracorporeal membrane oxygenation.

Standardizing definitions of peri-intubation hypotension should be a research priority, along with defining optimal vasopressor use, evaluating the role of invasive arterial monitoring, and investigating the effectiveness of bundled airway management strategies.

INTRODUCTION

Emergency airway management in critically ill patients carries a high risk of cardiovascular collapse, particularly in those with underlying physiologic derangements such as hypotension, shock, and severe hypoxemia [1, 2]. Despite improvements in first-pass success rates during rapid sequence intubation over the past decade, the incidence of adverse events, such as hypoxia, hypotension, cardiac arrest, and death, remains high [3–12]. To mitigate these risks, clinical guidelines emphasize the importance of hemodynamic optimization prior to intubation, yet specific strategies remain poorly defined [13, 14]. In the absence of standardized protocols and robust evidence, there is a critical need to develop and validate evidence-based approaches to guide peri-intubation hemodynamic management and enhance safety in this high-risk population [15].

Ethical Approval

This article is based on previously conducted studies and does not contain any new studies with human participants or animals performed by any of the authors.

EPIDEMIOLOGY

Peri-intubation cardiovascular collapse during rapid sequence intubation is a common, yet serious, complication with reported rates exceeding 40% [1, 3, 16]. Peri-intubation hypotension itself has no standardized definition, with related terms such as "cardiovascular collapse" and "hemodynamic instability" used inconsistently across studies, as noted in this review. These events are associated with an increased risk of multi-organ failure, cardiac arrest, and death [1, 9, 17–24]. Even brief or modest reductions in mean arterial pressure during intubation may contribute to increased risk for kidney dysfunction, myocardial injury, and mortality, underscoring the importance of prevention, early recognition, and intervention before the onset of severe tissue and organ hypoxia [20, 25].

RISK FACTORS

Patient-Related Risk Factors

Physiologically Difficult Airway Features

Physiologic derangements during critical illness significantly increase the risk of cardiovascular collapse and other adverse events during or after intubation by reducing physiologic reserve, impairing compensatory mechanisms, and amplifying hemodynamic stress [15]. These factors are further compounded and magnified during intubation by the effects of anesthetic induction agents and the transition to positive-pressure ventilation, which can precipitate abrupt cardiovascular decompensation. Previously described causes of physiologically difficult airway management include pre-existing hypotension, shock, hypoxemia, right ventricular dysfunction, intracranial hypertension, severe metabolic acidosis, obesity, and pregnancy, among others [2, 15, 26].

Anatomically Difficult Airway Features

Anatomically difficult airways are also associated with an increased risk of complications during emergency airway management, largely due to the increased number of intubation attempts, which is strongly correlated with adverse events [11, 13, 27]. Early recognition of features predicting difficult laryngoscopy, bag-valve-mask ventilation, extraglottic device placement, and cricothyrotomy are essential for optimizing first-pass success and minimizing complications. Although this review focuses on physiologic optimization, awareness of anatomically difficult airway features (e.g., limited mouth opening, reduced neck mobility, large tongue, facial trauma, airway obstruction, cervical spine immobilization, lack of jaw protrusion, and obesity) remains equally critical to ensure safe and effective airway management [28]. Structured pre-intubation assessments such as the LEMON assessment, MACOCHA score, and other predictive tools can help anticipate anatomic challenges and guide appropriate planning, including the use of adjuncts or early escalation to alternative airway strategies [29].

Procedure-Related Risk Factors

The selection and dosing of induction agents, especially those with vasodilatory or cardiodepressive properties, may influence hemodynamic stability during airway management [15]. Device choice also plays a role, as video laryngoscopy has been shown to improve first-pass success and reduce the number of attempts and associated complications [30]. Operator experience is another key factor, as novice providers are more likely to require multiple attempts, prolonging apnea and further exacerbating hemodynamic stress [3, 4, 7, 31]. Finally, effective team dynamics, including clear role assignment, closed-loop communication, and coordinated preparation, are essential to minimize delays, reduce errors, and ensure efficient execution during emergent airway management [15, 32]. These risk factors are summarized in Table 1.

Modifiable Risk Factors

While numerous studies have identified patient-related and procedure-related risk factors for peri-intubation hypotension, many are non-modifiable in the acute setting [16, 18, 20, 23, 24, 33–35]. However, some modifiable variables, such as pre-intubation systolic blood pressure, have been associated with increased risk [1, 20, 33, 34, 36]. Identifying and addressing modifiable risk factors is likely critical to improving outcomes, though high-quality studies validating strategies to reduce peri-intubation hypotension are lacking.

PATHOPHYSIOLOGY

Multiple converging physiologic mechanisms contribute to the risk of hemodynamic instability during airway management in critically ill patients [25, 33]. The administration of induction agents can result in vasoplegia, sympatholysis, or myocardial depression, leading to decreased systemic vascular resistance and impaired cardiac contractility [37]. The initiation of positive-pressure ventilation eliminates the negative intrathoracic pressure generated by spontaneous breathing, which normally augments venous return [2]. This transition increases intrathoracic and right atrial pressures, reduces the pressure gradient driving venous return, and leads to decreased preload,

Table 1 Risk factors for hemodynamic instability during emergency airway management

	Risk factors	Examples
Patient-related	Physiologically difficult airway features*	Pre-existing hypotension, hypoxemia, shock, right ventricular dysfunction, severe metabolic acidosis, etc.
	Anatomically difficult airway features	Limited mouth opening, reduced neck mobility, large tongue, facial trauma, airway obstruction, cervical spine immobilization, lack of jaw protrusion, obesity
Procedure-related	Pharmacologic agents*	Use of induction agents with vasodilatory or myocardial depressant effects
	Intubation device*	Direct laryngoscopy associated with increased complications as compared to video laryngoscopy
	Operator experience*	Less experienced providers associated with increased number of attempts and adverse events
	Team dynamics*	Poor communication, unclear role assignments, and uncoordinated preparation increase risk

*Modifiable factors; targeted strategies may help mitigate associated risks

stroke volume, and cardiac output. These effects are particularly detrimental in patients with hypovolemia or vasodilation, where low mean systemic filling pressure further compromises venous return. Rising intrathoracic pressure also increases right ventricular afterload, which, in the setting of existing right ventricular dysfunction or elevated pulmonary vascular resistance, may precipitate acute right ventricular failure and impair left ventricular filling. During the apneic phase, an abrupt loss of compensatory hyperventilation can worsen acidosis, while hypoxemia and hypercapnia may rapidly ensue, further impairing myocardial performance [2]. The cumulative burden of these stressors can lead to sudden hypotension, cardiac arrest, and death if not anticipated and proactively managed. These interrelated mechanisms are visually summarized in Fig. 1.

RISK STRATIFICATION FOR PERI-INTUBATION HYPOTENSION

Although several risk factors are known to predispose patients to immediate hypotension during emergent endotracheal intubation, the absence of widely adopted prediction tools often results in failure to recognize at-risk patients, leading to abrupt cardiovascular collapse and suboptimal clinical responses [3, 20].

One proposed tool to aid in risk stratification is the HYPS (hypotension prediction score), which was derived and validated in a cohort of critically ill patients [20, 38]. This score incorporates clinical variables such as pre-intubation systolic blood pressure, vasopressor use, age, and severity of illness to estimate the likelihood of post-intubation hypotension, with values ≥ 2 indicating increased risk [20]. While this score shows promise in identifying at-risk patients, its use in emergent settings may be limited by the need to calculate multiple variables in a time-sensitive environment.

Alternatively, a rapid and pragmatic approach using the shock index (calculated as heart rate divided by systolic blood pressure) and the modified shock index (calculated as heart rate divided by mean arterial pressure) may help predict post-intubation hypotension and mortality. Shock index values ≥ 0.7–0.8 have been associated with increased risk, whereas evidence for the modified shock index is less clear, though values ≥ 0.9 have been linked to worse

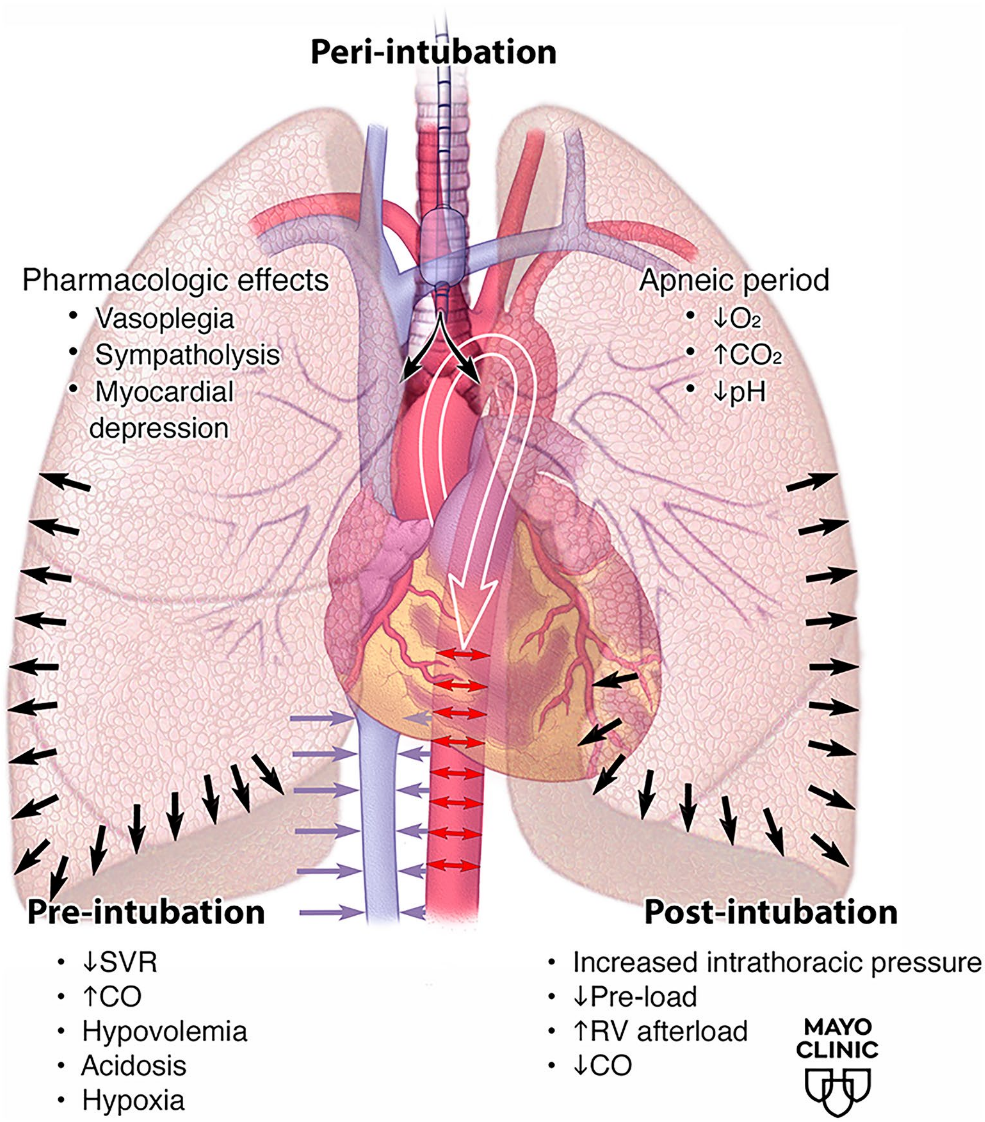

Fig. 1 Pathophysiology of peri-intubation cardiovascular collapse during rapid sequence intubation. *CO* cardiac output, *RV* right ventricle, *SVR* systemic vascular resistance. Used with permission of Mayo Foundation for Medical Education and Research, all rights reserved

outcomes [2, 9, 23, 24, 26, 39–43]. When at-risk patients are identified, providers should consider additional resuscitative measures prior to intubation, if time permits, although evidence supporting pre-intubation interventions to improve cardiovascular stability remains limited [1, 3].

PRE-INTUBATION HEMODYNAMIC OPTIMIZATION STRATEGIES DURING EMERGENCY AIRWAY MANAGEMENT

Preoxygenation

Hypoxemia is common during emergent tracheal intubation, reflects impaired physiologic reserve, and is associated with an increased risk of cardiac arrest and death [7, 34, 44]. Preoxygenation is therefore a foundational component of rapid sequence intubation, aimed at extending the duration of safe apnea by maximizing oxygen reserves and delaying critical desaturation.

Traditional preoxygenation techniques include spontaneous ventilation with 100% fractional inspired oxygen (FiO_2) for ≥ 3 min or eight vital capacity breaths using oxygen delivery systems such as non-rebreather masks at flush flow rate, high-flow nasal cannula, or bag-valve-mask [14, 26, 45]. However, these may be inadequate in critically ill patients with reduced functional residual capacity, high shunt physiology, or impaired respiratory mechanics [15].

Emerging evidence supports noninvasive positive-pressure ventilation as a more effective preoxygenation strategy, with randomized controlled studies demonstrating improved oxygenation and reduced hypoxemia, and concordant expert consensus providing additional support [15, 45, 46]. In parallel, the long-standing practice of maintaining an apneic period between induction and laryngoscopy to reduce aspiration risk has been recently challenged; multicenter randomized trials demonstrate that continued ventilation with noninvasive or bag-mask techniques during this period significantly decreases peri-intubation hypoxemia without increasing aspiration events, with expert consensus further endorsing this approach in appropriate patients [13, 44, 46, 47].

While noninvasive ventilation may theoretically precipitate hypotension by increasing intrathoracic pressure and impairing venous return, all patients undergoing emergency intubation are ultimately exposed to positive-pressure ventilation and its hemodynamic consequences. Initiating this exposure before induction and paralysis may unmask hemodynamic vulnerability in a controlled, sequential manner, allowing instability to be corrected before the additive effects of sedative agents occur. This approach may offer an opportunity to reduce compounded hemodynamic collapse in the highest-risk patients, although it has not been rigorously studied and requires further investigation.

In select cases, such as agitated, delirious, or uncooperative patients, medication-assisted preoxygenation using a delayed sequence intubation approach may be necessary. This involves administering a sedative-hypnotic to facilitate preoxygenation before neuromuscular paralysis and proceeding with intubation [13, 15, 48, 49].

Finally, it remains unknown whether the use of inhaled pulmonary vasodilators (e.g., nitric oxide, aerosolized prostacyclin) for hemodynamic optimization prior to emergent airway management may provide benefit in specific patient populations by attenuating expected increases in right ventricular afterload, such as those with severe pulmonary hypertension, right ventricular dysfunction, or profound hypoxemia [2, 50–52].

Fluid Resuscitation

Volume resuscitation is a critical step in mitigating the risk of peri-intubation hemodynamic collapse. However, empiric administration of fixed-volume fluid boluses has not demonstrated benefit across all critically ill populations. Two randomized trials evaluating the routine use of a 500-mL fluid bolus prior to induction found no significant reduction in the incidence of cardiovascular collapse [53, 54]. In PREPARE I, subgroup analysis raised the hypothesis that patients preoxygenated with noninvasive ventilation may be at increased risk of cardiovascular collapse and could potentially benefit from fluid bolus administration. However, the subsequent PREPARE II trial, designed to test this hypothesis, did not demonstrate a benefit of empiric fluid bolus administration among patients receiving noninvasive ventilation [53, 54]. These findings may reflect variability in fluid responsiveness, as some patients are not intravascularly depleted whereas others may require substantially larger volumes [54]. As such, fluid resuscitation likely should be guided by individualized clinical assessment. While routine pre-intubation fluid administration may not be warranted for all critically ill patients, it remains an essential component of care for those with hypovolemia [15]. When clinically feasible, ensuring adequate volume resuscitation prior to induction remains a priority, with fluid type, volume, and rate tailored to the physiologic profile.

Vasopressors

Push-Dose Vasopressors

Bolus dose or "push-dose" vasopressors have emerged as a practical strategy to treat peri-intubation hypotension during emergent airway management [25]. Peripherally administered agents, such as phenylephrine (100 µg/mL in a 10-mL syringe), epinephrine (10 µg/mL in a 5-mL syringe), or ephedrine (5 mg/mL in a 10-mL syringe) are commonly utilized for their rapid onset, ease of bedside administration, and ability to effectively increase blood pressure [25, 55, 56]. These agents exert immediate hemodynamic effects and are often employed as a bridge to initiation of vasopressor infusions or aggressive volume resuscitation when time constraints preclude early definitive therapy [25, 57].

Despite these hemodynamic benefits, evidence-based studies supporting the use of push-dose vasopressors remain sparse. One retrospective study found no significant difference in cardiovascular outcomes between patients receiving push-dose phenylephrine compared to those receiving continuous infusion norepinephrine, suggesting that vasopressor selection may be influenced more by clinical context and availability than by differences in efficacy [57]. Another multicenter, propensity-matched cohort study in patients with septic shock found that administering a phenylephrine push prior to norepinephrine initiation was associated with a higher rate of early hemodynamic stability, but not sustained stability at 12 h, indicating that while push-dose vasopressors may offer transient benefit, patients often experience sustained hypotension necessitating continuous vasopressor support [58].

Push-dose vasopressor use is not without potential risk. Phenylephrine is a selective alpha-1 adrenergic agonist that induces peripheral vasoconstriction, leading to increased systemic vascular resistance and blood pressure. However, the resulting rise in afterload may also reduce cardiac output, particularly in patients with compromised ventricular function [25]. Epinephrine is a non-selective alpha- and beta-adrenergic agonist that also increases blood pressure through peripheral vasoconstriction but additionally enhances cardiac output via beta-adrenergic stimulation. However, in a retrospective study compared to push-dose phenylephrine, epinephrine produced a greater increase in systolic blood pressure but was associated with a significantly higher rate of dosing errors [56].

Multiple reports have raised concern about the potential of medication errors, dose miscalculations, and adverse events when push-dose vasopressors are administered without standardized preparation or oversight [55–57]. These risks are further compounded when clinicians are required to dilute, label, and select appropriate doses at the bedside [56]. To mitigate risks, the use of pre-mixed syringes, pharmacist collaboration, and monitoring protocols when administering push-dose vasopressors is recommended [13].

Continuous Vasopressor Infusions

Although push-dose vasopressors transiently increase blood pressure, their short duration often results in recurrent hypotension, with most patients ultimately requiring a continuous vasopressor infusion [25, 57]. Starting a vasopressor infusion prior to induction may help blunt the hemodynamic stress associated with sedative agents and the transition to positive-pressure ventilation. While previously considered high-risk, peripheral vasopressor administration is now regarded as safe for short durations when properly monitored, allowing early initiation without central access [59–62].

The evidence regarding vasopressor use to prevent peri-intubation hypotension remains inconclusive. In a multicenter, propensity-matched secondary analysis of two clinical trials, prophylactic vasopressor administration was not associated with a reduced incidence of peri-intubation hypotension [63]. Similarly, a recent scoping review found no definitive evidence that vasopressors reduce post-intubation hypotension [64].

Several randomized controlled trials, including the ongoing PREVENTION (NCT05014581) and FLUVA (NCT05318066) studies, are specifically evaluating whether early initiation of norepinephrine reduces the risk of peri-intubation cardiovascular collapse. Importantly, factors such as infusion dead space, delivery rate, and device type may influence the onset and magnitude of hemodynamic effect. While definitive conclusions await these trial results, expert consensus and clinical guidelines currently endorse anticipatory vasopressor use in patients with or at risk for hypotension [15, 26].

Incorporating vasopressors into a broader peri-intubation bundle, including pre-induction risk assessment, individualized fluid resuscitation, use of push-dose vasopressors and vasopressor infusions, may offer a practical strategy to reduce the risk of peri-intubation cardiovascular collapse [65]. Further research is needed to define optimal agents, timing, dosing strategies, hemodynamic targets, and comparative effectiveness in high-risk intubations.

Premedication for Rapid Sequence Intubation

Fentanyl may be indicated in select patient populations as a pretreatment/premedication for rapid sequence intubation to blunt the sympathetic response to laryngoscopy, such as in patients with significantly elevated intracranial pressure [2, 66, 67]. However, it should not be used in routine clinical practice for all emergent intubations, as its administration has been associated with an increased risk of post-intubation hypotension in retrospective and randomized controlled studies [4, 68–70]. Careful consideration of patient-specific factors is essential before use.

Medications for Induction and Neuromuscular Blockade

Full-Dose Induction and Neuromuscular Blockade

Medication selection during rapid sequence intubation may further influence the risk of post-intubation hypotension depending on their pharmacologic effects [1, 17, 20, 37]. Etomidate and ketamine are generally preferred over propofol and midazolam in patients at risk for cardiovascular compromise due to their dose-dependent vasodilatory and cardiodepressive effects [1, 6, 14, 15, 17, 18, 37, 71]. Etomidate is considered “hemodynamically neutral” with minimal cardiovascular depression. However, it has been associated with adrenocortical suppression through transient 11β-hydroxylase inhibition as well as possible harm in some studies, though results are heterogeneous and may depend on specific outcome timing, leaving clinical relevance uncertain [37, 72–75]. In contrast, ketamine provides cardiovascular stimulation through sympathomimetic effects, though this benefit may be attenuated in catecholamine-depleted states, potentially resulting in hypotension from its negative inotropic effects. Admixtures of ketamine and propofol (e.g., ketofol) have shown comparable hemodynamic stability to reduced-dose etomidate in limited evidence, including a case series, a meta-analysis, and a randomized trial, though the optimal ratio is neither well defined nor approved [15, 76–78]. Randomized controlled trials have not shown significant differences in major outcomes between ketamine and etomidate during rapid sequence intubation [18, 72, 79].

For neuromuscular blockade, succinylcholine and rocuronium are the preferred agents for rapid sequence intubation [13, 15]. Succinylcholine offers a rapid onset and short duration of action but is associated with the risk of hyperkalemia in patients with burns, neuromuscular disease, or prolonged immobility. Rocuronium avoids these complications and is generally preferred in such scenarios, though its longer duration of action may limit the ability to perform early neurologic assessments. Some retrospective cohort studies have found that patients receiving neuromuscular blockade were less likely to develop post-intubation hypotension, but this association requires further investigation [16, 24].

Reduced-Dose Induction Strategy

In significantly unstable patients, dose reduction of induction agents has been variably employed as a strategy to mitigate cardiovascular collapse [17, 18, 24]. For instance, etomidate may be administered at doses ≤0.2 mg/kg and ketamine at ≤0.75–1.25 mg/kg, depending on the clinical context [80, 81]. This approach aims to balance the need for sedation while minimizing vasodilation and myocardial depression. Despite this approach being utilized in clinical practice, the evidence supporting reduced-dose induction is largely retrospective and subject to selection bias. Comparative studies have yielded mixed results, and no standardized definition exists to delineate what constitutes a safe "reduced dose" across different induction agents [18, 24, 80–82]. Furthermore, this strategy is not without potential drawbacks, including an increased risk of awareness under neuromuscular blockade and a false sense of reassurance that may delay or detract from appropriate hemodynamic optimization prior to intubation [80, 81]. While dose reduction may represent a reasonable strategy in unstable critically ill patients, prospective randomized controlled trials are needed to confirm its safety and efficacy, and to determine the optimal dosing thresholds for commonly used induction agents. Accordingly, available data support prioritizing resuscitative interventions, such as intravenous fluids and vasopressors, to stabilize the patient prior to induction, rather than relying solely on dose adjustment or induction agent selection to prevent peri-intubation hypotension [80, 82].

Intubation Without Sedation or Neuromuscular Blockade

In select critically ill patients, the administration of any induction agent may pose an unacceptably high risk of cardiovascular collapse. In these cases, awake intubation prioritizing use of topical anesthetics, without the use of induction agents or paralytics, may offer the safest alternative by preserving spontaneous ventilation and hemodynamic tone. This technique requires patient cooperation and meticulous topicalization but may be lifesaving when induction would otherwise precipitate profound hypotension or cardiac arrest [83].

Ideal candidates are those at high risk of decompensation who can tolerate local anesthetic techniques, although selection is largely guided by clinical judgment. In contrast, patients with severe agitation, altered mental status, or heavy secretions may not be suitable candidates because of difficulty achieving effective topicalization and visualization [83]. Success is also influenced by operator experience, equipment, and team preparation. Comprehensive descriptions of topicalization techniques (e.g., atomization, nebulization, equipment) have been published elsewhere [83]. When performed appropriately, awake fiberoptic intubation can provide a hemodynamically stable alternative, though its technical demands and patient selection criteria limit its broader applicability.

Invasive Blood Pressure Monitoring

Accurate and continuous blood pressure monitoring may be useful during emergency airway management, particularly in critically ill patients at risk for peri-intubation hypotension or cardiovascular collapse. Noninvasive blood pressure measurements can be delayed, intermittent, and unreliable in critically ill patients, especially during periods of hemodynamic instability [59, 84–87]. In contrast, invasive arterial blood pressure monitoring provides real-time, beat-to-beat measurements that enable early detection of hemodynamic deterioration and more precise titration of vasoactive agents [59].

The value of continuous invasive blood pressure monitoring was further supported by a randomized trial showing that continuous intra-arterial blood pressure monitoring, compared to noninvasive blood pressure monitoring during induction of anesthesia, significantly reduced the extent and duration of hypotension [88]. These findings reinforce the importance of integrating invasive arterial monitoring into a peri-intubation hemodynamic strategy, when feasible, especially in high-risk patients as defined by the HYPS, pre-intubation shock index, modified shock index, or other clinical parameters.

ROLE OF RESCUE AND ADJUNCTIVE THERAPIES

In select critically ill patients at high risk for peri-intubation cardiovascular collapse, conventional airway management strategies may be unsafe or inadequate. For patients with profound physiologic instability, such as profound shock or severe pulmonary hypertension with right ventricular failure, adjunctive therapies may offer the only viable path to a safe and controlled intubation. While these approaches are resource-intensive, they can be lifesaving when incorporated early and thoughtfully into a physiology-driven airway plan.

Mechanical Circulatory Support

Extracorporeal life support can serve as an adjunctive or rescue therapy to achieve the primary endpoint of oxygenation and perfusion for patients in whom oxygen delivery and perfusion cannot be stabilized through conventional means. Candidacy for extracorporeal membrane oxygenation (ECMO) should be determined according to predefined institutional protocols as part of the pre-intubation planning process for high-risk patients. This is especially relevant for patients already requiring high-dose vasoactive or respiratory support prior to intubation.

In extremely rare situations, the risk of peri-intubation cardiac arrest may be so high, such as in patients with critical airway obstruction or severe pulmonary hypertension with right ventricular dysfunction, that the preferred strategy may be to avoid intubation altogether and proceed directly to ECMO before airway management is attempted [89, 90]. If intubation remains a reasonable option, the ECMO team may consider staying on standby for select high-risk cases, as this has been associated with improved outcomes compared to emergent, unplanned ECMO initiation [51, 91]. In such cases, preemptive placement of 5-French femoral arterial and venous sheath catheters prior to intubation may be considered in consultation with the ECMO team to enable rapid cannulation in the event of peri-intubation hemodynamic collapse or cardiac arrest [91]. However, this approach remains largely theoretical in this setting and should be regarded as a potential focus for future investigation.

The effectiveness of ECMO in this setting relies on early recognition, coordinated multidisciplinary planning, and timely preparation. In appropriately selected patients, ECMO provides cardiopulmonary support while facilitating recovery or serving as a bridge to definitive interventions [92]. A clinical algorithm outlining the approach to airway management in patients at intermediate or high-risk of peri-intubation hypotension is summarized in Fig. 2.

FUTURE DIRECTIONS

The proactive use of push-dose vasopressors represents a particularly promising area for investigation. Future studies should aim to develop a systematic, protocolized approach to their administration by identifying the most effective agents, dosing strategies, indications, and optimal timing. Comparative effectiveness research is needed to assess whether a

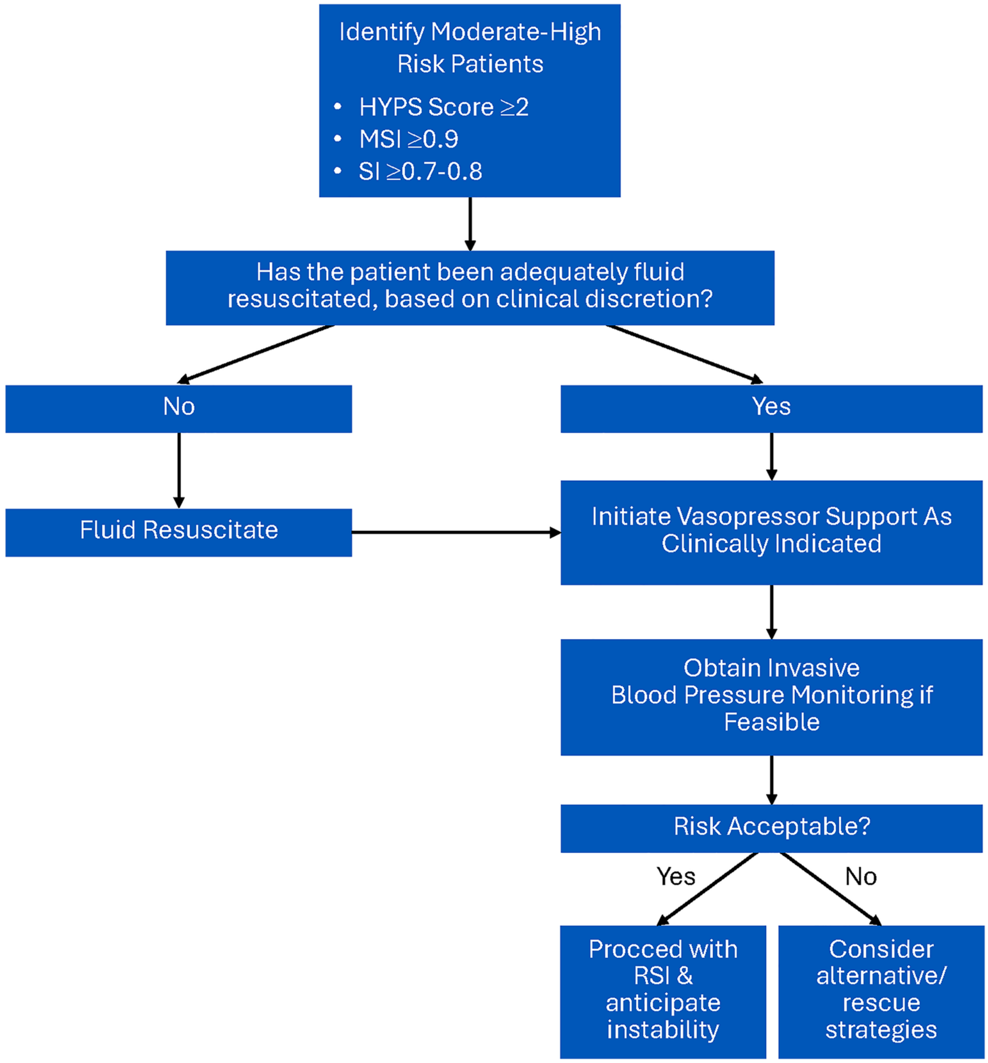

Fig. 2 Proposed clinical algorithm for hemodynamic optimization of moderate-high risk patients during rapid sequence intubation. *HYPS* hypotension prediction score, *MSI* modified shock index, *RSI* rapid sequence intubation, *SI* shock index

proactive, structured use of push-dose vasopressors improves outcomes compared to reactive administration.

Similarly, the preemptive initiation of vasopressor infusions prior to induction, particularly in patients with borderline or declining blood pressure, warrants additional focused investigation. Although increasingly used in practice, the ideal timing, choice of agent, and optimal indications for initiating continuous vasopressors in this setting remain undefined.

The role of invasive arterial blood pressure monitoring during emergent intubation remains largely undefined. Studies are needed to evaluate its use in high-risk patients and to clarify whether it is associated with improved recognition and management of hemodynamic instability. Targeting specific hemodynamic thresholds prior to induction (e.g., systolic blood pressure ≥ 130 mmHg), either alone or as part of a bundled resuscitation strategy, merits prospective validation.

Finally, integrating physiological risk stratification and optimization into structured airway safety checklists and clinical pathways may facilitate early recognition of high-risk patients, streamline intervention, and enhance team-based preparedness [93]. It is likely that no single intervention alone will confer maximal benefit; rather, a bundle of coordinated, evidence-based interventions may ultimately yield the greatest improvement in outcomes as has been the case in other areas such as central venous access, sepsis care, respiratory failure, and surgery [93–98].

CONCLUSION

Emergency airway management in critically ill patients carries a substantial risk of cardiovascular collapse, particularly in those with concomitant physiologic compromise. Despite growing awareness of this risk, approaches to hemodynamic optimization remain highly variable and are guided by limited evidence. Identifying high-risk patients, tailoring airway management strategies to their vulnerabilities, and implementing supportive measures such as fluid resuscitation, vasopressor use, invasive blood pressure monitoring, and adjunctive and rescue therapies are paramount for optimizing outcomes.

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Declarations

Conflict of Interest. Samuel I. Garcia has nothing to disclose. Nathan J. Smischney has nothing to disclose. Benjamin J. Sandefur has nothing to disclose. Jacopo D'Andria Ursoleo has nothing to disclose. Diana J. Kelm has nothing to disclose. Patrick M. Wieruszewski is a consultant for Wolters Kluwer/UpToDate and serves on the Editorial Advisory board for *Pulmonary Therapy*.

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REFERENCES

1. Russotto V, Tassistro E, Myatra SN, et al. Peri-intubation cardiovascular collapse in patients who are critically ill: insights from the INTUBE study. Am J Respir Crit Care Med. 2022;206(4):449–58.
2. Mosier JM, Joshi R, Hypes C, Pacheco G, Valenzuela T, Sakles JC. The physiologically difficult airway. West J Emerg Med. 2015;16(7):1109–17.
3. Russotto V, Myatra SN, Laffey JG, et al. Intubation practices and adverse peri-intubation events in critically ill patients from 29 countries. JAMA. 2021;325(12):1164–72.

4. Griesdale DE, Bosma TL, Kurth T, Isac G, Chittock DR. Complications of endotracheal intubation in the critically ill. Intensive Care Med. 2008;34(10):1835–42.

5. Benedetto WJ, Hess DR, Gettings E, et al. Urgent tracheal intubation in general hospital units: an observational study. J Clin Anesth. 2007;19(1):20–4.

6. Jaber S, Amraoui J, Lefrant JY, et al. Clinical practice and risk factors for immediate complications of endotracheal intubation in the intensive care unit: a prospective, multiple-center study. Crit Care Med. 2006;34(9):2355–61.

7. McKown AC, Casey JD, Russell DW, et al. Risk factors for and prediction of hypoxemia during tracheal intubation of critically ill adults. Ann Am Thorac Soc. 2018;15(11):1320–7.

8. Simpson GD, Ross MJ, McKeown DW, Ray DC. Tracheal intubation in the critically ill: a multi-centre national study of practice and complications. Br J Anaesth. 2012;108(5):792–9.

9. Heffner AC, Swords DS, Neale MN, Jones AE. Incidence and factors associated with cardiac arrest complicating emergency airway management. Resuscitation. 2013;84(11):1500–4.

10. Smischney NJ, Seisa MO, Heise KJ, et al. Practice of intubation of the critically ill at Mayo Clinic. J Intensive Care Med. 2019;34(3):204–11.

11. Brown CA 3rd, Bair AE, Pallin DJ, Walls RM. Techniques, success, and adverse events of emergency department adult intubations. Ann Emerg Med. 2015;65(4):363-370.e361.

12. Hypes C, Sakles J, Joshi R, et al. Failure to achieve first attempt success at intubation using video laryngoscopy is associated with increased complications. Intern Emerg Med. 2017;12(8):1235–43.

13. Acquisto NM, Mosier JM, Bittner EA, et al. Society of Critical Care Medicine clinical practice guidelines for rapid sequence intubation in the critically ill adult patient. Crit Care Med. 2023;51(10):1411–30.

14. Higgs A, McGrath BA, Goddard C, et al. Guidelines for the management of tracheal intubation in critically ill adults. Br J Anaesth. 2018;120(2):323–52.

15. Karamchandani K, Nasa P, Jarzebowski M, et al. Tracheal intubation in critically ill adults with a physiologically difficult airway. An international Delphi study. Intensive Care Med. 2024;50(10):1563–79.

16. Green RS, Edwards J, Sabri E, Fergusson D. Evaluation of the incidence, risk factors, and impact on patient outcomes of postintubation hemodynamic instability. CJEM. 2012;14(2):74–82.

17. Marler J, Howland R, Kimmons LA, Mohrien K, Vandigo JE, Jones GM. Safety of propofol when used for rapid sequence intubation in septic patients: a multicenter cohort study. Hosp Pharm. 2022;57(2):287–93.

18. Kim JM, Shin TG, Hwang SY, et al. Sedative dose and patient variable impacts on postintubation hypotension in emergency airway management. Am J Emerg Med. 2019;37(7):1248–53.

19. Jones AE, Yiannibas V, Johnson C, Kline JA. Emergency department hypotension predicts sudden unexpected in-hospital mortality: a prospective cohort study. Chest. 2006;130(4):941–6.

20. Smischney NJ, Kashyap R, Khanna AK, et al. Risk factors for and prediction of post-intubation hypotension in critically ill adults: a multicenter prospective cohort study. PLoS ONE. 2020;15(8):e0233852.

21. Schwartz DE, Matthay MA, Cohen NH. Death and other complications of emergency airway management in critically ill adults. A prospective investigation of 297 tracheal intubations. Anesthesiology. 1995;82(2):367–76.

22. Cook TM, Woodall N, Harper J, Benger J, Fourth National Audit Project. Major complications of airway management in the UK: results of the Fourth National Audit Project of the Royal College of Anaesthetists and the Difficult Airway Society. Part 2: intensive care and emergency departments. BJA Br J Anaesth. 2011;106(5):632–42.

23. Smischney NJ, Seisa MO, Heise KJ, et al. Predictors of hemodynamic derangement during intubation in the critically ill: a nested case-control study of hemodynamic management-Part II. J Crit Care. 2018;44:179–84.

24. Heffner AC, Swords DS, Nussbaum ML, Kline JA, Jones AE. Predictors of the complication of postintubation hypotension during emergency airway management. J Crit Care. 2012;27(6):587–93.

25. Panchal AR, Satyanarayan A, Bahadir JD, Hays D, Mosier J. Efficacy of bolus-dose phenylephrine for peri-intubation hypotension. J Emerg Med. 2015;49(4):488–94.

26. Kornas RL, Owyang CG, Sakles JC, Foley LJ, Mosier JM. Evaluation and management of the physiologically difficult airway: consensus recommendations from Society for Airway Management. Anesth Analg. 2021;132(2):395–405.

27. Sakles JC, Chiu S, Mosier J, Walker C, Stolz U. The importance of first pass success when performing orotracheal intubation in the emergency department. Acad Emerg Med. 2013;20(1):71–8.

28. Detsky ME, Jivraj N, Adhikari NK, et al. Will this patient be difficult to intubate? The rational clinical examination systematic review. JAMA. 2019;321(5):493–503.

29. De Jong A, Molinari N, Terzi N, et al. Early identification of patients at risk for difficult intubation in the intensive care unit: development and validation of the MACOCHA score in a multicenter cohort study. Am J Respir Crit Care Med. 2013;187(8):832–9.

30. Prekker ME, Driver BE, Trent SA, et al. Video versus direct laryngoscopy for tracheal intubation of critically ill adults. N Engl J Med. 2023;389(5):418–29.

31. Garcia SI, Sandefur BJ, Campbell RL, et al. First-attempt intubation success among emergency medicine trainees by laryngoscopic device and training year: a national emergency airway registry study. Ann Emerg Med. 2023;81(6):649–57.

32. Garcia SI, Finch AS, Ridgeway JL, et al. Understanding team dynamics and culture of safety using video reflexive ethnography during real-time emergent intubation. Ann Am Thorac Soc. 2024;21(7):1065–73.

33. Lin CC, Chen KF, Shih CP, Seak CJ, Hsu KH. The prognostic factors of hypotension after rapid sequence intubation. Am J Emerg Med. 2008;26(8):845–51.

34. Elmer J, Brown F, Martin-Gill C, Guyette FX. Prevalence and predictors of post-intubation hypotension in prehospital trauma care. Prehosp Emerg Care. 2020;24(4):461–9.

35. Perbet S, De Jong A, Delmas J, et al. Incidence of and risk factors for severe cardiovascular collapse after endotracheal intubation in the ICU: a multicenter observational study. Crit Care. 2015;19(1):257.

36. Halliday SJ, Casey JD, Rice TW, et al. Risk factors for cardiovascular collapse during tracheal intubation of critically iII adults. Ann Am Thorac Soc. 2020;17(8):1021–4.

37. Foster M, Self M, Gelber A, et al. Ketamine is not associated with more post-intubation hypotension than etomidate in patients undergoing endotracheal intubation. Am J Emerg Med. 2022;61:131–6.

38. Smischney NJ, Surani SR, Montgomery A, et al. Hypotension prediction score for endotracheal intubation in critically ill patients: a post hoc analysis of the HEMAIR study. J Intensive Care Med. 2022;37(11):1467–79.

39. Althunayyan SM. Shock index as a predictor of post-intubation hypotension and cardiac arrest; a review of the current evidence. Bull Emerg Trauma. 2019;7(1):21–7.

40. Trivedi S, Demirci O, Arteaga G, Kashyap R, Smischney NJ. Evaluation of preintubation shock index and modified shock index as predictors of postintubation hypotension and other short-term outcomes. J Crit Care. 2015;30(4):861.e861-867.

41. Cannon CM, Braxton CC, Kling-Smith M, Mahnken JD, Carlton E, Moncure M. Utility of the shock index in predicting mortality in traumatically injured patients. J Trauma. 2009;67(6):1426–30.

42. Smischney NJ, Seisa MO, Schroeder DR. Association of shock indices with peri-intubation hypotension and other outcomes: a sub-study of the KEEP PACE trial. J Intensive Care Med. 2024;39(9):866–74.

43. Lee K, Jang JS, Kim J, Suh YJ. Age shock index, shock index, and modified shock index for predicting postintubation hypotension in the emergency department. Am J Emerg Med. 2020;38(5):911–5.

44. Casey JD, Janz DR, Russell DW, et al. Bag-mask ventilation during tracheal intubation of critically ill adults. N Engl J Med. 2019;380(9):811–21.

45. Baillard C, Fosse JP, Sebbane M, et al. Noninvasive ventilation improves preoxygenation before intubation of hypoxic patients. Am J Respir Crit Care Med. 2006;174(2):171–7.

46. Gibbs KW, Semler MW, Driver BE, et al. Noninvasive ventilation for preoxygenation during emergency intubation. N Engl J Med. 2024;390(23):2165–77.

47. Frat JP, Ricard JD, Quenot JP, et al. Non-invasive ventilation versus high-flow nasal cannula oxygen therapy with apnoeic oxygenation for preoxygenation before intubation of patients with acute hypoxaemic respiratory failure: a randomised, multicentre, open-label trial. Lancet Respir Med. 2019;7(4):303–12.

48. Weingart SD, Trueger NS, Wong N, Scofi J, Singh N, Rudolph SS. Delayed sequence intubation: a prospective observational study. Ann Emerg Med. 2015;65(4):349–55.

49. Waack J, Shepherd M, Andrew E, Bernard S, Smith K. Delayed sequence intubation by intensive care flight paramedics in Victoria, Australia. Prehosp Emerg Care. 2018;22(5):588–94.

50. Shekar K, Varkey S, Cornmell G, et al. Feasibility of non-invasive nitric oxide gas inhalation to prevent endotracheal intubation in patients with acute hypoxemic respiratory failure: a single-centre experience. Nitric Oxide. 2021;116:35–7.

51. Rajagopal S, Ruetzler K, Ghadimi K, et al. Evaluation and management of pulmonary hypertension in noncardiac surgery: a scientific statement from the American Heart Association. Circulation. 2023;147(17):1317–43.

52. Thompson A, Fleischmann KE, Smilowitz NR, et al. 2024 AHA/ACC/ACS/ASNC/HRS/SCA/SCCT/SCMR/SVM guideline for perioperative cardiovascular management for noncardiac surgery: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. J Am Coll Cardiol. 2024;84(19):1869–969.

53. Russell DW, Casey JD, Gibbs KW, et al. Effect of fluid bolus administration on cardiovascular collapse among critically ill patients undergoing tracheal intubation: a randomized clinical trial. JAMA. 2022;328(3):270–9.

54. Janz DR, Casey JD, Semler MW, et al. Effect of a fluid bolus on cardiovascular collapse among critically ill adults undergoing tracheal intubation (PrePARE): a randomised controlled trial. Lancet Respir Med. 2019;7(12):1039–47.

55. Rotando A, Picard L, Delibert S, Chase K, Jones CMC, Acquisto NM. Push dose pressors: experience in critically ill patients outside of the operating room. Am J Emerg Med. 2019;37(3):494–8.

56. Nam E, Fitter S, Moussavi K. Comparison of push-dose phenylephrine and epinephrine in the emergency department. Am J Emerg Med. 2022;52:43–9.

57. Schmitt CJ, Mattson AE, Brown CS, et al. The incidence of cardiovascular instability in patients receiving various vasopressor strategies for peri-intubation hypotension. Am J Emerg Med. 2023;65:104–8.

58. Hawn JM, Bauer SR, Yerke J, et al. Effect of phenylephrine push before continuous infusion norepinephrine in patients with septic shock. Chest. 2021;159(5):1875–83.

59. Evans L, Rhodes A, Alhazzani W, et al. Surviving Sepsis Campaign: international guidelines for management of sepsis and septic shock 2021. Crit Care Med. 2021;49(11):e1063–143.

60. Cardenas-Garcia J, Schaub KF, Belchikov YG, Narasimhan M, Koenig SJ, Mayo PH. Safety of peripheral intravenous administration of vasoactive medication. J Hosp Med. 2015;10(9):581–5.

61. Tian DH, Smyth C, Keijzers G, et al. Safety of peripheral administration of vasopressor medications: a systematic review. Emerg Med Australas. 2020;32(2):220–7.

62. Loubani OM, Green RS. A systematic review of extravasation and local tissue injury from administration of vasopressors through peripheral intravenous catheters and central venous catheters. J Crit Care. 2015;30(3):653.e659-617.

63. Fuchita M, Pattee J, Russell DW, et al. Prophylactic administration of vasopressors prior to emergency intubation in critically ill patients: a secondary analysis of two multicenter clinical trials. Crit Care Explor. 2023;5(7):e0946.

64. Marks PLG, Domm JM, Miller L, Yao Z, Gould J, Loubani O. The use of vasopressors to reduce post-intubation hypotension in critically ill adult patients undergoing emergent endotracheal intubation: a scoping review. CJEM. 2024;26(11):804–13.

65. Nabzdyk CGS, Read MD, Wieruszewski PM, Seelhammer TG. Proactive approach to peri-induction hemodynamic management in critically ill patients. J Cardiothorac Vasc Anesth. 2021;35(12):3489–92.

66. Bucher J, Koyfman A. Intubation of the neurologically injured patient. J Emerg Med. 2015;49(6):920–7.

67. Pouraghaei M, Moharamzadeh P, Soleimanpour H, et al. Comparison between the effects of alfentanil, fentanyl and sufentanil on hemodynamic indices during rapid sequence intubation in the emergency department. Anesth Pain Med. 2014;4(1):e14618.

68. Ferguson I, Buttfield A, Burns B, et al. Fentanyl versus placebo with ketamine and rocuronium for patients undergoing rapid sequence intubation in the emergency department: the FAKT study-a randomized clinical trial. Acad Emerg Med. 2022;29(6):719–28.

69. Takahashi J, Goto T, Okamoto H, et al. Association of fentanyl use in rapid sequence intubation with post-intubation hypotension. Am J Emerg Med. 2018;36(11):2044–9.

70. Bakhsh A, Bakhribah A, Alshehri R, et al. Association between fentanyl use and post-intubation mean arterial pressure during rapid sequence intubation: prospective observational study. West J Emerg Med. 2025;26(1):10–9.

71. Freeman J, Alkhouri H, Knipp R, Fogg T, Gillett M. Mapping haemodynamic changes with rapid sequence induction agents in the emergency department. Emerg Med Australas. 2022;34(2):237–43.

72. Jabre P, Combes X, Lapostolle F, et al. Etomidate versus ketamine for rapid sequence intubation in acutely ill patients: a multicentre randomised controlled trial. Lancet. 2009;374(9686):293–300.

73. Koroki T, Kotani Y, Yaguchi T, et al. Ketamine versus etomidate as an induction agent for tracheal intubation in critically ill adults: a Bayesian meta-analysis. Crit Care. 2024;28(1):48.

74. Bruder EA, Ball IM, Ridi S, Pickett W, Hohl C. Single induction dose of etomidate versus other induction agents for endotracheal intubation in critically ill patients. Cochrane Database Syst Rev. 2015;1(1):CD010225.

75. Kotani Y, Piersanti G, Maiucci G, et al. Etomidate as an induction agent for endotracheal intubation in critically ill patients: a meta-analysis of randomized trials. J Crit Care. 2023;77:154317.

76. de GalloMoraes A, Racedo Africano CJ, Hoskote SS, et al. Ketamine and propofol combination ("ketofol") for endotracheal intubations in critically ill patients: a case series. Am J Case Rep. 2015;16:81–6.

77. Smischney NJ, Seisa MO, Morrow AS, et al. Effect of ketamine/propofol admixture on peri-induction hemodynamics: a systematic review and meta-analysis. Anesthesiol Res Pract. 2020;2020:9637412.

78. Smischney NJ, Nicholson WT, Brown DR, et al. Ketamine/propofol admixture vs etomidate for intubation in the critically ill: KEEP PACE randomized clinical trial. J Trauma Acute Care Surg. 2019;87(4):883–91.

79. Matchett G, Gasanova I, Riccio CA, et al. Etomidate versus ketamine for emergency endotracheal intubation: a randomized clinical trial. Intensive Care Med. 2022;48(1):78–91.

80. Driver BE, Trent SA, Prekker ME, Reardon RF, Brown CA 3rd. Sedative dose for rapid sequence intubation and postintubation hypotension: is there an association? Ann Emerg Med. 2023;82(4):417–24.

81. Mattson AE, Brown CS, Sandefur BJ, Cole K, Haefke B, Cabrera D. Postintubation hypotension following rapid sequence intubation with full- vs reduced-dose induction agent. Am J Health Syst Pharm. 2025;82(3):e148–56.

82. April MD, Arana A, Schauer SG, et al. Ketamine versus etomidate and peri-intubation hypotension: a national emergency airway registry study. Acad Emerg Med. 2020;27(11):1106–15.

83. Sandefur BJ, Driver BE, Long B. Managing awake intubation. Ann Emerg Med. 2025;85(1):21–30.

84. Araghi A, Bander JJ, Guzman JA. Arterial blood pressure monitoring in overweight critically ill patients: invasive or noninvasive? Crit Care. 2006;10(2):R64.

85. Bur A, Hirschl MM, Herkner H, et al. Accuracy of oscillometric blood pressure measurement according to the relation between cuff size and upper-arm circumference in critically ill patients. Crit Care Med. 2000;28(2):371–6.

86. Kaur B, Kaur S, Yaddanapudi LN, Singh NV. Comparison between invasive and noninvasive blood pressure measurements in critically ill patients receiving inotropes. Blood Press Monit. 2019;24(1):24–9.

87. Riley LE, Chen GJ, Latham HE. Comparison of noninvasive blood pressure monitoring with invasive arterial pressure monitoring in medical ICU patients with septic shock. Blood Press Monit. 2017;22(4):202–7.

88. Kouz K, Wegge M, Flick M, et al. Continuous intra-arterial versus intermittent oscillometric arterial pressure monitoring and hypotension during induction of anaesthesia: the AWAKE randomised trial. Br J Anaesth. 2022;129(4):478–86.

89. Malpas G, Hung O, Gilchrist A, et al. The use of extracorporeal membrane oxygenation in the anticipated difficult airway: a case report and systematic review. Can J Anaesth. 2018;65(6):685–97.

90. Lorusso R, Whitman G, Milojevic M, et al. 2020 EACTS/ELSO/STS/AATS expert consensus on post-cardiotomy extracorporeal life support in adult patients. J Thorac Cardiovasc Surg. 2021;161(4):1287–331.

91. Liu C, Li X, Li J, et al. Standby extracorporeal membrane oxygenation: a better strategy for high-risk percutaneous coronary intervention. Front Med (Lausanne). 2024;11:1404479.

92. Wieruszewski PM, Ortoleva JP, Cormican DS, Seelhammer TG. Extracorporeal membrane oxygenation in acute respiratory failure. Pulm Ther. 2023;9(1):109–26.

93. Jaber S, Jung B, Corne P, et al. An intervention to decrease complications related to endotracheal intubation in the intensive care unit: a

prospective, multiple-center study. Intensive Care Med. 2010;36(2):248–55.

94. Stiell IG, Spaite DW, Field B, et al. Advanced life support for out-of-hospital respiratory distress. N Engl J Med. 2007;356(21):2156–64.

95. Ferrer R, Artigas A, Levy MM, et al. Improvement in process of care and outcome after a multicenter severe sepsis educational program in Spain. JAMA. 2008;299(19):2294–303.

96. Micek ST, Roubinian N, Heuring T, et al. Before-after study of a standardized hospital order set for the management of septic shock. Crit Care Med. 2006;34(11):2707–13.

97. Haynes AB, Weiser TG, Berry WR, et al. A surgical safety checklist to reduce morbidity and mortality in a global population. N Engl J Med. 2009;360(5):491–9.

98. Pronovost P, Needham D, Berenholtz S, et al. An intervention to decrease catheter-related bloodstream infections in the ICU. N Engl J Med. 2006;355(26):2725–32.