



Evaluation of Predictive Indicators for Post-Intubation Hypotension

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Abstract

Post-intubation hypotension is one of the causes of hemodynamic disorders after tracheal intubation, especially for emergency intubation in the emergency department or intensive care unit, due to the characteristics of these patients with serious conditions and time constraints. Therefore, early and rapid prediction of the risk of post-tracheal intubation hypotension and emphasis on early interventional measures are crucial. Therefore, it is crucial to predict the possibility of hemodynamic instability after emergency tracheal intubation in critically ill patients. According to existing studies, the risk factors for post-intubation hypotension include various factors such as the patient's physiological parameters, drug induction before intubation, and variability in the skills of the healthcare team; however, due to the individual bias effect of some of these indicators and the difficulty of real-time detection, they cannot provide practical value to the clinic. In response to these existing problems, some studies have reported that ultrasonic measurements, a non-invasive, convenient, and dynamically evaluable parameter in real-time, can predict hypotension after tracheal intubation, providing evidence for clinical intervention to improve the prognosis of critical illness.

Keywords

Endotracheal Intubation, Hemodynamic Disturbance, Ultrasonic Measurement, Post-Intubation Hypotension, Prediction Index

Introduction

Endotracheal intubation (ETI) is a critical skill, particularly for clinicians in anesthesiology, emergency medicine, and intensive care. Although ETI is often lifesaving for critically ill patients, it carries several complications, including hypoxemia, hypotension, bradycardia, and cardiopulmonary arrest, which can negatively affect hemodynamic stability, hospital stay length, and survival rates. Research shows that patients who develop hypotension within 1-2 hours' post-intubation—defined as 1) mean arterial pressure < 65 mmHg; 2) systolic blood pressure < 80 mmHg or a 40%

decrease from baseline systolic pressure; or 3) initiation or escalation of vasopressors within 30 minutes—tend to experience longer hospital stays and higher in-hospital mortality, with an incidence ranging from 20% to 45% [1]. Among the patients in the intensive care unit, the incidence of hypotension after intubation ranges between 20-52%; in the emergency department, post-intubation hypotension is significantly associated with higher mortality and prolonged hospitalization [2]. In addition, the post-intubation period is one of the most vulnerable periods for hypotension. Even if of

limited duration, it is associated with serious complications and death, while studies have reported that almost one-third of hypotension during surgery occurs after ETI and is associated with an independent risk of postoperative acute kidney injury [3]. The most common mechanisms include, among others, hypovolemia, sympathetic nervous system depression, decreased vascular tone, depressed myocardial contractility, and inadequate venous return due to intrathoracic pressure [4,5].

Currently, clinical prediction of post-intubation hypotension involves a combination of dynamic vital signs, such as preoperative hypotension, respiratory rate, heart rate, urine output, arterial lactate levels, skin temperature, and neurological symptoms. However, these indicators are subject to significant individual variability, which reduces predictive accuracy. Often, they show minimal changes before post-intubation hypotension develops, limiting their effectiveness for timely prediction. This study aims to identify and evaluate additional predictive indicators of post-intubation hypotension to improve early risk assessment and patient outcomes.

Shock Index

The Shock Index (SI) is the ratio of pulse rate (beats/min) to systolic blood pressure [mmHg], which is usually considered to be a normal value of 0.5-0.7. Due to the clinical advantages of being rapid, simple, noninvasive, and easy to obtain repeatedly, it is commonly used as an effective indicator for assessing the hemodynamic stability of the organism and identifying patients with acute and critical illnesses [6]. In a retrospective study by Heffner et al. involving 465 patients undergoing endotracheal intubation in the emergency department, elevated SI was found to predict cardiovascular instability and hemodynamic deterioration, establishing it as a strong predictor of post-intubation hypotension and poor clinical outcomes [7]. Building on this, Trivedi et al. identified a pre-intubation SI threshold of 0.8 as optimal for identifying high-risk patients in the emergency department, while an SI threshold of 0.9 was found for ICU patients. This discrepancy is likely due to differences in urgency when assessing patient condition in the two settings [8].

Because SI is influenced by many factors such as the patient's body temperature, age, measurement position, and other variables, it is unstable. In recent years, with the deepening of research, other related indices have been gradually derived on the basis of SI; among them, Liu et al. proposed a modified shock index (MSI), which replaces the ratio of the original heart rate and systolic blood pressure (SBP) with the ratio of the heart rate and mean arterial pressure (MAP) to calculate the shock index [9]. The blood pressure (SBP) ratio was replaced by the ratio of heart rate to mean arterial pressure (MAP) to calculate the shock index, with a reference range of approximately 0.34-1.70 beats/(min-mmHg) (1 mmHg = 0.133 kPa); its positive predictive value is greater than that of SI [10]. Because this value covers the changes in heart rate, systolic blood pressure, diastolic blood pressure, and other indicators, it integrates cardiovascular circulation and breaks through the limitation of relying solely on heart rate and systolic blood pressure to judge the condition. The increase or decrease of MSI reflects the hypodynamic and hyperdynamic states of the body's blood circulation, respectively, and is related to patient mortality, with the risk of death increasing as MSI increases. This makes MSI a better predictor than SI in both emergency department and intensive care unit patients. For patients in the emergency department and intensive care unit, it is a better predictor of disease severity and post-discharge morbidity and mortality than SI, but there is a lack of clinical studies on whether it can predict the risk of post-tracheal intubation hypotension [11].

In our clinical work, since blood pressure and heart rate are affected by age, we found that the accuracy of applying SI to determine whether a patient is in shock and the cutoff value for the degree of shock varies among different age groups. Therefore, after excluding the effect of age on blood pressure and heart rate, we introduced in this study the age shock index, i.e., SI multiplied by age. Initially used to predict mortality in elderly trauma patients [12,13], AgeSI has since been linked to mortality in non-trauma patients as well, with evidence suggesting it outperforms both SI and MSI in predicting poor outcomes. In patients with acute myocardial infarction, AgeSI has also been associated with mortality, outperforming both SI and MSI in this context. However, evidence for AgeSI as a predictor of

post-intubation hypotension remains limited [14]. A study by Lee et al. identified pre-intubation SI, AgeSI, and MSI as predictors of post-intubation hypotension, with AgeSI showing a significant correlation with post-intubation hypotension in patients with stable hemodynamics prior to intubation. After adjusting for confounders, $MSI > 1.3$ and $SI > 0.7$ were found to be statistically significant predictors of post-intubation hypotension incidence, with AgeSI emerging as the most effective predictor among these parameters [10].

Hypotension Prediction Score (HYPS)

Studies have shown that patients who develop hypotension after tracheal intubation have increased morbidity and mortality. To enable early identification of critically ill patients at risk for hypotension following intubation, a new multivariable risk scoring system—the Hypotension Prediction Score (HYPS)—was developed [15]. This score includes 11 variables: severity of illness (APACHE II score), age, presence of sepsis, intubation due to respiratory failure, intubation during cardiac arrest, intubation with mean arterial pressure < 65 mmHg, diuretic use within the previous 24 hours, systolic blood pressure below 130 mmHg, catecholamine or phenylephrine use within 1 hour prior to intubation, and use of etomidate during intubation. Each of these factors has been shown to independently correlate with post-intubation hypotension. Where systolic blood pressure, once below 130 mmHg, increases the risk of post-intubation hypotension, even if diastolic blood pressure is twice the normal value. Interestingly, when using mean arterial pressure rather than systolic pressure, we found that being below the threshold of 95 mmHg was associated with post-intubation hypotension. Thus, perhaps achieving a higher perfusion pressure by mean arterial pressure or systolic pressure in critically ill patients may prevent post-intubation hypotension and related adverse outcomes [16].

HYPS scores ranged from -1.5 to 29 (median 7.5, interquartile range 4 to 12.5), and when scores were below the low-risk threshold of 1.5, they had a positive predictive value of 11.9% and a negative predictive value of 88.1%. When the score was above the high-risk threshold of 19, it had a positive predictive value of

71.9% and a negative predictive value of 28.1%. When the score was 9.5, it had high specificity and sensitivity, and its predictive ability was optimal [15]. Similarly, research has demonstrated a positive correlation between the risk of post-intubation hypotension in emergency departments and the HYPS score. Higher scores were associated with an increased likelihood of post-intubation hypotension, with the high-risk group exhibiting a notably high incidence rate of 33% following ETI [17]. Furthermore, this study revealed that the HYPS scoring system demonstrated superior predictive value for post-intubation hypotension compared to the SI. This enhanced predictive capability enables clinicians to identify high-risk patients for hypotension more effectively in emergency settings, facilitating the implementation of appropriate interventional measures.

Heart Rate Variability (HRV)

Heart rate variability (HRV) is the difference or variation in the cardiac beat cycle over consecutive heartbeats, in other words, the ratio of low-frequency power to high-frequency power of the heart's beat rate, with a normal value of 1.5-2. It is a valuable indicator of the modulation of the cardiovascular system, mainly through neurohumoral factors, and thus a possible predictor of sudden cardiac death and arrhythmic events. It is a valuable indicator for predicting sudden cardiac death and arrhythmic events. By measuring HRV in individuals, we can obtain information about autonomic nervous system activity, stress response, etc. It is a known tool for predicting hypotension and common bradycardia. Although studies on the correlation between HRV and post-intubation hypotension are limited in China, international research has shown that a low HRV ratio is significantly associated with hypotension, while a high HRV ratio is strongly linked to bradycardia. In other words, HRV can predict the development of hypotension and bradycardia in patients undergoing elective surgery with spinal anesthesia [18]. This conclusion has been further supported by additional research, suggesting that preoperative HRV analysis can help identify patients at risk of post-induction hypotension and serves as a promising predictive tool for post-intubation hypotension [19].

Perfusion Index and Perfusion Variability Index

The perfusion index (PI) is a parameter of oximetry that indicates the ratio of pulsatile to static non-pulsatile blood flow at the site being monitored (e.g., fingers, toes, earlobes), with a normal range of 4-5. PI reflects the perfusion status of the monitored site, with low values indicating potential hypotensive shock, intravascular volume depletion, or poor peripheral circulation. A study [20] on hypotension incidence in general anesthesia patients found that PI correlates with systolic blood pressure and is a better predictor of hypotension than mean arterial pressure. Additionally, the study suggested that PI has a high negative predictive value for hypotension before tracheal intubation under propofol anesthesia, supporting its use alongside other circulatory monitoring parameters to assess the coupling between microcirculation and macrocirculation.

Furthermore, numerous studies have confirmed that PI reflects tissue hypoperfusion and is significantly associated with patient mortality rates [21], justifying its use as a peripheral tissue perfusion index to predict outcomes in patients with pregnancy-induced hypertension (post-intubation hypotension).

The pulse perfusion variation index (PVI) is a non-invasive, continuous dynamic respiratory variation index that reflects changes in pulse perfusion (PI) during the respiratory cycle [22]. This indirectly indicates the relationship between intrathoracic pressure and cardiac preload, enabling dynamic assessment of volume responsiveness, particularly in mechanically ventilated patients. Generally, when PVI exceeds 15%, it suggests low volume responsiveness, and a PVI greater than 47% may predict hypotension, though its sensitivity (15%) and specificity (58%) are relatively low [23]. A prospective observational study on propofol-induced hypotension found no significant association between baseline PVI > 15% and PI < 1.05 with the occurrence of hypotension [24]. Therefore, while PVI and PI cannot fully predict hypotension, they remain clinically useful for identifying high-risk patients with low intravascular volume.

Ultrasound Measurement

Inferior Vena Cava Collapse Index (IVC-CI):

Post-intubation hypotension is primarily associated with insufficient blood volume and decreased vascular tone, both of which reflect changes in hemodynamics. Bedside ultrasound, a fast, non-invasive, multifunctional, and repeatable imaging method, is widely applicable across various clinical settings, from emergency departments to intensive care units. The inferior vena cava (IVC) is the largest venous trunk in the body, collecting venous blood from the abdomen, pelvis, and lower extremities and flowing into the right atrium. Its internal diameter is measured by ultrasound to assess the right atrium, central venous pressure, and cardiovascular volume; the internal diameter of the inferior vena cava during expiration is 18.8 ± 3 mm, and during inspiration is 21.3 ± 4 mm. Hemodynamics exploration of the internal diameter of the inferior vena cava allows rapid assessment of intravascular and extravascular volume status and volume response [2]. The underlying mechanism of IVC-based assessments is that the IVC diameter varies with respiratory cycles. During spontaneous inhalation, the negative pressure in the thoracic cavity reduces right atrial pressure, creating a pressure gradient that drives increased venous return through the IVC, leading to IVC collapse. This variation can be quantified using the IVC collapse index (IVC-CI), calculated as follows: $\text{IVC-CI} = (\text{dIVCmax} - \text{dIVCmin}) / \text{dIVCmax} \times 100\%$, where dIVCmax represents the maximum IVC diameter at end-inspiration, and dIVCmin is the minimum diameter at end-expiration.

Dipti et al.'s meta-analysis demonstrated a correlation between dIVCmax and hypovolemic states [25]. When IVC-CI is less than 20%, intravascular hypovolemia can effectively be excluded. Conversely, when dIVCmax is less than 2.1 cm and IVC-CI exceeds 50%, the likelihood of hypotension increases [26]; however, this does not exclude the possibility of intravascular volume overload. Research also identified IVC-CI as a significant independent predictor of hypotension following induction [27]. Therefore, IVC-CI provides critical insight into volume status, aiding in the prediction of post-intubation hypotension. A study on elderly patients undergoing spinal anesthesia found that IVC-guided fluid management significantly reduced the incidence of hypotension by monitoring IVC-CI before and after anesthesia. Notably, IVC-CI showed

superior predictive accuracy for hypotension compared to dIVCmax [28]. Furthermore, Zhang et al. demonstrated that in hypertensive patients, dIVCmax exhibited superior specificity and sensitivity in predicting post-intubation hypotension compared to the IVC-CI, which showed no significant predictive performance. Specifically, at an optimal threshold of 1.24 cm, dIVCmax achieved a specificity of 94.4%, surpassing that of IVC-CI [29]. These findings suggest that dIVCmax may serve as a more effective predictor of post-intubation hypotension in hypertensive patients. Additionally, pre-intubation MAP has been identified as a crucial factor in predicting post-intubation hypotension. These results align with previous domestic and international studies, which consistently indicate that lower pre-intubation MAP or systolic blood pressure represents a significant risk factor for post-intubation hypotension.

Subclavian Vein Collapse Index (SCV-CI):

The subclavian vein (SCV) is a continuation of the axillary veins of both upper limbs and is generally mildly upwardly bowed, about 3-4 cm long, with an internal diameter of about 1-2 cm. It begins at the outer edge of the first rib and then joins the internal jugular vein a few centimeters anteriorly under the clavicle to form the cephalic vein, which continues to the superior vena cava to enter the heart. It is usually unaffected by intra-abdominal hypertension or positive end-expiratory pressure. The minimum diameter (dSCVmin) and the maximum diameter (dSCVmax) of the subclavian vein are assessed in M-mode with a moderate sweep speed during natural and deep inspiration, and the subclavian vein collapse index is calculated using the formula $(dSCVmax - dSCVmin) / dSCVmax \times 100$ [30].

Studies suggest that SCV-CI can predict fluid responsiveness in hypovolemic shock patients with spontaneous breathing and has a positive correlation with the IVC-CI, indicating that SCV-CI may sometimes substitute for IVC-CI in assessing fluid responsiveness in spontaneously breathing patients [31]. Furthermore, multiple studies have demonstrated that SCV-CI can predict fluid responsiveness in mechanically ventilated patients. Among 120 patients undergoing general anesthesia, an SCV-CI threshold of 36% (with 90%

sensitivity and 87% specificity) was identified for predicting post-induction hypotension, similar to the IVC-CI threshold of 37% (with 94% sensitivity and 84% specificity). This suggests that both SCV-CI and IVC-CI are effective predictors of post-intubation hypotension [32,33].

Since the SCV is unaffected by intra-abdominal pressure or end-expiratory positive pressure, some studies have explored using SCV diameter, measured by ultrasound, as a predictor for post-intubation hypotension. For example, Kent's study indicated that a dSCVmax of less than 8.0 cm and an SCV-CI exceeding 38.24% before spinal anesthesia, or a dSCVmax below 6.5 cm and an SCV-CI above 37.68% post-anesthesia, can predict hypotension following spinal anesthesia [34]. However, real-time measurements post-anesthesia are more clinically valuable. Compared to SCV-CI, dSCVmax has lower accuracy for predicting post-spinal hypotension, possibly due to individual variations in SCV diameter. This conclusion is supported by additional studies [35], which suggest that SCV-CI is more sensitive than SCV diameter measurement. Therefore, while SCV-CI and dSCVmax both show potential as predictors for post-intubation hypotension, further research is required to determine which measurement offers greater clinical value.

Internal Jugular Vein Area (IJV-A):

Ultrasonography of the internal jugular vein (IJV) can be used to estimate CVP, intraventricular blood volume, and fluid responsiveness. In patients in shock, the passive elevated lower extremity test has been shown to be helpful in assessing fluid volume deficit or fluid responsiveness [36]. Similar to the elevated lower extremity test, changes in the size of the internal jugular vein (IJV) from a supine position to a head-down position may reflect fluid volume status. IJV assessment is much simpler compared to ultrasound measurement of the IVC. The maximum area of the IJV (IJV-Area) is measured primarily by ultrasound in the supine and head-down position of the patient. The minimum value of IJV-A in the supine position is about 0.09 cm², the maximum value is about 3.12 cm², and the mean value is about 1.29 cm² (standard deviation 0.68); the minimum value of IJV-A in the head-low-feet-high position is 0.15 cm², the maximum value is about 4.88

cm², and the mean value is about 1.85 cm² (standard deviation 0.78) [37].

Studies have shown that the area of the internal jugular vein measured by ultrasound in the head-down position is larger in the hypotensive group compared to the non-hypotensive group. For example, a study by Okamura et al. [37] found that in the head-down position, the area of the internal jugular vein in the hypotensive group (standard deviation) was 2.02 (0.86) cm² compared to 1.72 (0.68) cm² in the non-hypotensive group. This suggests the presence of a relationship. The study also used multiple logistic regression analysis to consider variables affecting internal jugular vein area, such as age, calcium channel antagonists, ACEIs/ARBs, and basal mean blood pressure. The results showed that the internal jugular vein area measured by ultrasound in the head-down position prior to induction of tracheal intubation with general anesthesia was an independent predictor of hypotension. However, it should be noted that the sensitivity as well as the specificity of this index in this study was not high, and a small number of patients had a lower IJV-A in the head-down position than in the supine position. The mechanism for this may be the contraction of the IJV due to sympathetic activation, which reduces the IJV area [38]. Therefore, we should not rely on this index alone for prediction in clinical applications but should combine it with other indexes for comprehensive judgment.

In addition, for special populations such as pregnant women, the jugular vein area cannot completely predict hypotension. This is due to the increase in systemic blood volume and widening of the jugular vein area in these patients due to hormones. A recent foreign study on hypotension after spinal anesthesia in pregnant women also suggests that the parameters of IJV-A and IJV-D during spontaneous and deep respiration cannot be used as reliable predictors of postoperative hypotension after spinal anesthesia during cesarean delivery [39]. This may indicate that the occurrence of hypotension during the induction of anesthesia may depend on factors other than intravascular volume. However, since this study was a single-center study, this conclusion needs to be verified by more studies in the future.

Carotid Artery (Corrected Flow Time and Peak Velocity Variation):

Hypotension after tracheal intubation induced by anesthesia is relatively common in clinical practice. Based on the undoubtedly favorable premise of noninvasive, easily accessible measurements, carotid artery corrected blood flow time (FTc) and carotid artery blood flow peak velocity ($\Delta V_{\text{peak-CA}}$) were suggested as a simple and easy method for assessing patients' volume status and predicting volume responsiveness during voluntary respiratory states. The reasons for this are as follows: first, the carotid arteries are large and shallow, making Doppler flow acquisition easy. Second, in the emergency department as well as in the intensive care unit, blood flow is preferentially distributed from the brachial artery to the carotid artery in the event of unexpected hemodynamic instability, when it may be the only palpable artery. Ultrasound was used to obtain the common carotid artery flow spectrum and then to calculate the FTc and the $\Delta V_{\text{peak-CA}}$, in which the FTc was calculated using a simplified formula: $\text{FTc} = \text{FT} + [1.29 \times (\text{HR} - 60)]$. The maximum and minimum values of the peak velocity during one respiratory cycle were measured automatically and recorded. $\Delta V_{\text{peak-CA}}$ was calculated as follows: $100 \times (\text{maximum peak velocity} - \text{minimum peak velocity}) / [(\text{maximum peak velocity} + \text{minimum peak velocity}) / 2]$, and the average value of the three consecutive measurements was recorded [40].

FTc is a complex static index, and it has been reported that the use of FTc for in vivo fluid optimization during surgery not only reduces the rate of complications, improves patient recovery, and shortens the postoperative hospital stay [41], but also predicts the volume response in mechanically ventilated patients and shows no significant difference between it and the change in noninvasive pulse pressure. A study by Maitra et al. [42] demonstrated for the first time that carotid artery FTc and ΔV_{peak} were able to predict possible hypotension in patients undergoing surgery after induction of tracheal intubation under general anesthesia. The findings of this study were further supplemented by Wang's study. That is, the area under the working characteristic curve of subjects with carotid FTc was 0.87, with an optimal cutoff value of 379.1 ms, a sensitivity of 72.2%, and a specificity of 93.7%,

whereas the area under the ROC curve of subjects with ΔV_{peak} was 0.67, with an optimal cutoff value of 7.5%, a sensitivity of 55.6%, and a specificity of 75.0%, demonstrating the value of both in predicting hypotension [43]. Multivariate logistic regression analysis revealed that carotid FTc was an independent predictor of hypotension after tracheal intubation in the elderly. In elderly patients, each 1-millisecond decrease in carotid FTc increased the risk of post-tracheal intubation hypotension by 8%, but the results of this study do not apply to elderly patients undergoing induction with isoprotorenol, which may increase the incidence of hypotension. However, in some of the studies, FTc was not a predictor of volume response, possibly because patients with hemodynamic disorders that affect FTc to predict volume response were not excluded or because vasoconstriction induced by some medications, such as norepinephrine, may result in a lower FTc. At the same time, a low FTc is not always associated with a low left ventricular preload, and it may even represent a state of humoral overload, which implies that a fluid challenge based on FTc alone may further worsen the hemodynamic situation [41]. Therefore, in this context, there may be no single parameter that can guide fluid therapy in all cases, and FTc may be very useful when used in combination with other clinical information such as inferior vena cava measurements.

To date, there have been many studies aimed at determining the cutoff value of ΔV_{peak} for predicting volume responsiveness as well as for distinguishing volume resuscitation responders from nonresponders. Thus, carotid ΔV_{peak} has been shown to be an accurate method of assessing preload and a promising variable for predicting volume responsiveness in patients undergoing mechanical ventilation procedures or in patients with different types of shock. Notably, a study by Song et al. [44] confirmed that carotid ΔV_{peak} was the most appropriate method for predicting fluid responsiveness in mechanically ventilated coronary revascularization patients with a cutoff value of 11%. Furthermore, these findings indicate that measuring corrected flow time (FTc) and peak velocity variation ($\Delta V_{\text{peak}}\text{-CA}$) in the carotid artery can effectively predict volume responsiveness in mechanically ventilated patients. These parameters show promise as predictive indicators for potential post-intubation hypotension

risk.

Stroke Volume Variation:

Studies have shown [45] that transthoracic echocardiographic parameters, such as stroke volume variation (SVV), inferior vena cava variability, and pulse pressure variation, are significantly correlated with changes in blood volume. Pulse-continuous cardiac output monitoring is considered the gold standard for assessing volume responsiveness and volume status; however, it is complex, invasive, time-consuming, costly, and associated with various complications, leading to low patient acceptance. In contrast, transthoracic echocardiography accurately reflects stroke volume (SV) by determining the time-integral of the blood flow rate in the left ventricular outflow tract and clinically reflects stroke volume variability (SVV) by calculating the ratio of the difference between the highest and the lowest stroke volume values to the mean stroke volume value, with a normal value of between 0 and 10%. In clinical practice, SVV is used to assess volume responsiveness, predict the effect of fluid therapy, and monitor the effects of mechanical ventilation. The high SVV values relative to the actual values during the circulatory compensation phase, especially during the autonomic respiratory state, and the correlation with hypotension suggest that SVV has a good predictive value in predicting hypotension. This suggests that SVV has good predictive value for hypotension. Furthermore, SVV is inversely related to blood volume; as SVV increases, volume becomes more insufficient, and MAP decreases. Therefore, measuring SVV before anesthesia induction can provide clinicians with valuable information regarding the patient's preoperative volume status, enabling timely volume resuscitation and the use of vasoactive drugs to prevent hypotension. Another study [46] found that maintaining SVV within the 5%-7% range reduces the incidence of tourniquet-related hypotension in elderly patients undergoing total knee arthroplasty and helps decrease the risk of postoperative myocardial injury. Therefore, echocardiographic measurement of SVV can serve as a useful reference for predicting post-intubation hypotension.

Conclusion

This article focuses on the current indicators that can

predict post-intubation hypotension, including shock index, heart rate variability, per-beat variability, and the use of ultrasound, which, as a noninvasive, convenient, and versatile technology, can be used not only to measure the function of individual organs but also to assess the intravascular and extravascular volume response. This is particularly suitable for the urgent assessment of whether tracheal intubation carries a risk of developing hypotension. Although carotid corrected flow time and carotid peak flow rate variability can effectively predict the risk of hypotension after endotracheal intubation, their calculations are complex and time-consuming compared to those of the inferior vena cava collapse index, jugular vein area, and subclavian vein collapse index, and they are not suitable for the evaluation of emergency preoperative endotracheal intubation. The simplicity and stability of the inferior vena cava collapse index and jugular vein area do not exclude the possibility that the operation and visualization of ultrasound may be affected in clinical application. In this case, it is recommended that they be used in combination with other techniques, such as the perfusion index, heart rate variability, and shock index, etc., and that the above parameters be used for the dynamic assessment of hemodynamics. This will provide real-time evidence for post-intubation hypotension to improve the clinical prognosis of patients with critical illnesses and improve clinical outcomes.

There are limitations in this paper, and the current study on the mechanism of post-tracheal intubation hypotension involves a variety of physiological, pathological, and pharmacological factors, such as the patient's physiological parameters (age, body mass index, lactate level, and albumin level), inducing agents (sedative-hypnotic agents, neuromuscular blocking agents) used during tracheal intubation, different sequences of target-controlled drug administration, and comorbidities (renal failure, respiratory failure), all of which are hypotension-independent risk factors [15]. This paper did not include these physiological mechanisms and the use of inducers within the scope of the study, focusing instead on the early prediction of the risk of possible hypotension after tracheal intubation by noninvasive means. For clinical application, it is recommended that the above early warning indicators

and their involvement in physiological, pathological, pharmacological, and other mechanisms be integrated and analyzed, allowing for future reduction in the incidence of post-intubation hypotension and offering the possibility of precise treatment after the occurrence of post-intubation hypotension.

Conflict of Interest

The authors have read and approved the final version of the manuscript. The authors have no conflicts of interest to declare.

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