e-ISSN (online): 2745-9497

Journal of Anesthesiology and Clinical Research (JACR)

Journal website: https://hmpublisher.com/index.php/jacr

Hemodynamic Attenuation During Tracheal Intubation: A Randomized Comparative Analysis of Video vs. Direct Laryngoscopy in Adult Elective Surgery

Imam Safi'i[1]*, Arie Zainul Fatoni[1], Taufiq Agus Siswagama[1], Ahmad Feza Fadhlurrahman[1]

[1]Departement of Anesthesiology and Intensive Care, Brawijaya Medicine, Malang, Indonesia

ARTICLE INFO

Keywords:
Hemodynamics
Intratracheal
Intubation
Laryngoscopy
Sympathoadrenal response

***Corresponding author:**
Imam Safi'i

E-mail address:
dr.imams85.tnial@gmail.com

All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/jacr.v7i1.850

A B S T R A C T

Introduction: Laryngoscopy and tracheal intubation inevitably trigger a sympathoadrenal response, manifesting as tachycardia and hypertension. While video laryngoscopy (VL) offers improved glottic visualization compared to direct laryngoscopy (DL), its efficacy in specifically attenuating this hemodynamic stress remains a subject of debate. This study investigates whether VL provides superior hemodynamic stability during the critical post-intubation period by analyzing the rate pressure product (RPP) and temporal hemodynamic interactions. **Methods:** In this prospective, single-blind, randomized controlled trial, 40 adult patients (ASA I-II) undergoing elective surgery were allocated to either Group VL (GlideScope, n=20) or Group DL (Macintosh, n=20). Anesthesia was strictly standardized with Fentanyl 2 mcg/kg, Propofol 2 mg/kg, and Atracurium 0.5 mg/kg. Hemodynamic parameters, including systolic blood pressure (SBP), mean arterial pressure (MAP), and heart rate (HR), were recorded at baseline (T0) and at 1 (T1), 2 (T2), and 5 (T5) minutes post-intubation. The primary analysis utilized a general linear model (Repeated Measures ANOVA) to assess Time-Group interactions, corrected for sphericity. **Results:** Demographics were homogeneous between groups. A significant Time-Group interaction was observed for MAP (p less than 0.001), indicating a blunted pressor response curve in the VL group. Heart Rate at 1-minute post-intubation was significantly lower in Group VL (75.45 plus or minus 11.23 bpm) compared to Group DL (90.15 plus or minus 15.22 bpm; p equals 0.001). Analysis of the rate pressure product revealed that Group DL approached ischemic thresholds, whereas Group VL maintained significantly lower myocardial workload at minutes 1 and 2 (p less than 0.01). **Conclusion:** Video laryngoscopy significantly attenuates the reflex tachycardia and arterial pressure surge associated with tracheal intubation compared to direct laryngoscopy. VL is recommended to minimize cardiovascular stress in susceptible surgical populations.

1. Introduction

Airway management constitutes a foundational pillar of anesthesiology, representing a critical intersection where procedural skill meets physiological preservation.[1] While the primary objective of endotracheal intubation is to secure a patent airway for ventilation and oxygenation, the procedure itself acts as a double-edged sword. The mechanical maneuver required to visualize the glottis and pass the tracheal tube delivers one of the most potent noxious stimuli encountered during the entire perioperative period. This stimulus is not merely a local mechanical event but a trigger for a systemic physiological cascade that challenges the homeostatic reserve of the patient, particularly during the vulnerable phase of anesthesia induction.[2] The physiological mechanism driving this response is rooted in the dense innervation of the upper airway. The manipulation of supraglottic tissues—

specifically the forceful stretching of the epipharynx and the deep pressure exerted on the base of the tongue and vallecula—activates a robust somatovisceral reflex arc. This afferent input is mediated primarily by the glossopharyngeal nerve (CN IX) and the vagus nerve (CN X). These neural signals travel rapidly to the brainstem, specifically stimulating the vasomotor center within the nucleus tractus solitarius. The integration of these impulses precipitates a widespread and immediate sympathoadrenal discharge.[3]

Clinically, this reflex arc manifests as a profound pressor response, characterized by a rapid surge in plasma catecholamine concentrations. The sequelae include acute hypertension, significant tachycardia, and a consequent spike in myocardial oxygen demand. In patients with robust physiological reserves, this transient hyperdynamic state is generally well-tolerated and resolves as the stimulus ceases.[4] However, the demographic landscape of surgical patients is increasingly complex. In individuals with compromised cardiovascular reserve—such as those suffering from coronary artery disease, intracranial pathology, or severe, uncontrolled hypertension—these hemodynamic fluctuations are far from benign.[5] In these high-risk populations, the intubation-induced surge can disrupt the delicate balance between myocardial oxygen supply and demand, potentially precipitating subendocardial ischemia, destabilizing arterial plaques, inducing arrhythmias, or causing cerebral hemorrhage due to spikes in intracranial pressure. Consequently, attenuating this reflex response has become a critical clinical objective in modern anesthetic practice. The magnitude of this hemodynamic disturbance is intimately linked to the mechanical invasiveness of the laryngoscopy technique employed. Traditional direct laryngoscopy (DL), utilizing the ubiquitous Macintosh blade, relies on a line-of-sight geometric principle. To visualize the glottic opening, the anesthesiologist must align the oral, pharyngeal, and laryngeal axes into a single straight line. Achieving this alignment often necessitates a significant anterior lifting force to elevate the mandible and compress the soft tissues of the tongue base. It is this high-force manipulation of the vallecula that maximally stimulates the proprioceptors and nociceptors responsible for driving the sympathoadrenal reflex.[6]

In contrast, the advent of video laryngoscopy (VL) has introduced a paradigm shift in airway management mechanics.[7] By integrating camera optics at the distal tip of the blade, VL allows for an indirect view of the glottis, decoupling visualization from the requirement for linear geometric alignment. This look-around-the-corner capability theoretically reduces the need for forceful tissue compression and cervical spine manipulation. The hypothesis follows that by requiring less lifting force to expose the larynx, VL should significantly reduce the intensity of mechanoreceptor stimulation, thereby dampening the afferent signals to the brainstem and blunting the subsequent pressor response.

Despite the sound physiological rationale and the clear anatomical advantages of video laryngoscopy, the global literature regarding its hemodynamic superiority remains surprisingly conflicting. While some investigations report distinct stability and a softer physiological profile with VL, others have found no significant clinical difference compared to traditional direct laryngoscopy. This discordance in the literature may be attributed to methodological heterogeneity across studies. A significant number of previous trials have been limited by variable anesthetic induction protocols, where inconsistent dosing of opioids or varying intervals between induction and intubation introduce pharmacological confounders. These pharmacological tails can mask the true physiological effect of the airway device, making it difficult to isolate the mechanical stimulus of the laryngoscope as the primary variable.[8]

Furthermore, the metrics used to define hemodynamic stability have often been superficial. Many studies rely solely on absolute values of heart rate or systolic blood pressure, which offer only a fragmented view of cardiac workload. Fewer studies have utilized the rate pressure product (RPP)—the product of heart rate and systolic blood pressure—as a primary outcome measure. The RPP serves as a superior surrogate for myocardial oxygen consumption (MVO_2), providing a more holistic assessment of the stress placed on the heart during the critical post-

intubation window. Additionally, there is a paucity of research that conducts detailed, granular analyses of the specific interaction between time and device type, particularly within specific demographic populations, such as in Indonesia, where anatomical variations may exist.[9,10]

This study aims to perform a granular comparative analysis of the hemodynamic profiles between video laryngoscopy and direct laryngoscopy to definitively address these inconsistencies. The novelty of this research lies in its rigorous methodological standardization of the induction sequence, strictly controlling drug dosages and timing to isolate the mechanical stimulus of the laryngoscopy from pharmacological noise. Furthermore, this study employs a sophisticated mixed-model repeated measures ANOVA to evaluate the rate pressure product (RPP) and the specific time-group interaction. This statistical approach allows for the determination of the precise temporal window and trajectory of the hemodynamic interaction, moving beyond simple point-prevalence comparisons. We specifically hypothesize that video laryngoscopy will demonstrate a significantly lower rate pressure product and a blunted pressor response curve compared to direct laryngoscopy, offering protective benefits against the intubation surge in the first two minutes post-intubation.

2. Methods

This was a prospective, single-blind, randomized controlled clinical trial conducted at the Central Operating Theatre of Dr. Saiful Anwar Regional General Hospital, East Java, Indonesia. The study protocol adhered to the principles of the Declaration of Helsinki and was approved by the institutional ethics committee. Written informed consent was obtained from all participants prior to enrollment. The trial was registered in the institutional clinical research registry.

The study population consisted of a defined cohort of adult patients ranging in age from 20 to 55 years, all of whom were scheduled to undergo elective surgical procedures requiring general anesthesia. To ensure a homogeneous baseline of physiological health and minimize anesthetic risk, enrollment was strictly limited to individuals classified as American Society of Anesthesiologists (ASA) physical status I or II. A primary methodological objective was to evaluate hemodynamic responses within the context of anatomically standard airways; consequently, strict inclusion criteria regarding airway geometry were enforced. Eligible participants were required to present with a Mallampati score of I or II and a predicted Cormack-Lehane grade of I or II. Furthermore, specific anatomical measurements were mandated for inclusion, including a thyromental distance exceeding 6 cm and an interincisor gap greater than 3 cm, thereby ensuring adequate mouth opening and mandibular space for laryngoscopy.

To isolate the mechanical stimulus of laryngoscopy as the primary independent variable, rigorous exclusion criteria were implemented to eliminate potential anatomical and physiological confounders. Patients exhibiting characteristics predictive of a difficult airway, such as a Mallampati score of 3 or 4, a history of intraoral pathology, or cervical spine instability, were categorically excluded from the study. Additionally, to prevent the skewing of hemodynamic data by pre-existing pathologies, the researchers excluded individuals with morbid obesity, defined as a Body Mass Index greater than 35 kg/m^2, as well as those with severe cardiovascular comorbidities, including uncontrolled hypertension, arrhythmias, or established ischemic heart disease. Finally, to ensure that the observed sympathoadrenal reflexes were not pharmacologically masked, patients currently prescribed beta-blockers or other rate-limiting medications were omitted from the analysis

The sample size calculation was based on detecting a clinically significant difference in Heart Rate (HR). Based on pilot data, we anticipated a mean difference of 12 beats per minute (bpm) between groups at 1-minute post-intubation, with a standard deviation of 13 bpm. Using a power of 80% and an alpha error of 0.05, a sample size of 18 patients per group was required. To account for potential dropouts, technical failures, or data loss, a total sample size of 40 patients was determined (n=20 per group).

Patients were randomly allocated using a computer-generated sequence into two groups: (1) Group VL: Intubation performed using the GlideScope Video

Laryngoscope with a size 3 or 4 hyperangulated blade; (2) Group DL: Intubation performed using a standard Macintosh laryngoscope with a size 3 or 4 curved blade. Allocation concealment was ensured using sequentially numbered, opaque, sealed envelopes (SNOSE), which were opened by the attending anesthesiologist only upon the patient's entry into the operating theater.

Double-blinding of the intubating anesthesiologist was not possible due to the distinct nature of the devices. To ensure single-blinding of the data collection, a modified protocol was used. The outcome assessor was positioned behind a sterile screen that obscured their view of the airway device being used. Additionally, all hemodynamic data were extracted post-hoc from the digital memory of the anesthesia monitor by a researcher who was blinded to the group allocation.

Standard monitoring (Non-Invasive Blood Pressure [NIBP], Electrocardiogram [ECG], Pulse Oximetry [SpO_2]) was established upon arrival. Pre-oxygenation was performed for 3 minutes with 100% oxygen. Induction of anesthesia was strictly standardized to minimize pharmacological variability: (i) Fentanyl: 2 mcg/kg IV; (ii) Propofol: 2 mg/kg IV; (iii) Atracurium: 0.5 mg/kg IV. Laryngoscopy was initiated exactly 3 minutes post-muscle relaxant administration to ensure peak effect of both the neuromuscular blocker and the opioid. All procedures were performed by senior anesthesiologists experienced in both techniques to mitigate learning curve bias. Intubation time was limited to a maximum of 45 seconds. Patients requiring more than one intubation attempt were excluded from the analysis to prevent the confounding effect of repeated stimulation.

Hemodynamic variables—Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), and Heart Rate (HR)—were recorded at four precise time points: (i) T0: Pre-induction baseline (after 5 minutes of rest); (ii) T1: 1 minute post-intubation; (iii) T2: 2 minutes post-intubation; (iv) T5: 5 minutes post-intubation. Secondary outcome (derived), the rate pressure product (RPP), a correlate of myocardial oxygen consumption, was calculated using the formula: RPP = Systolic Blood Pressure × Heart Rate.

Data were analyzed using SPSS version 26.0. Normality was assessed using the Shapiro-Wilk test. Continuous variables with normal distribution were compared using the Independent T-Test. Categorical variables were analyzed using the Chi-Square test. To assess the interaction between the device used and the time intervals, a general linear model (repeated measures ANOVA) was employed. The assumption of Sphericity was tested using Mauchly's Test. If Sphericity was violated (p less than 0.05), the degrees of freedom were corrected using the Greenhouse-Geisser estimate. Effect sizes were reported as partial eta squared for ANOVA and Cohen's d for t-tests to quantify the magnitude of the difference. A p-value less than 0.05 was considered statistically significant.

3. Results

Table 1 delineates the demographic and baseline physiological characteristics of the 40 adult subjects enrolled in the trial, stratified by airway management technique. The randomization methodology successfully yielded two well-balanced cohorts with no statistically significant inter-group differences. Demographic analysis revealed homogeneity in mean age (36.50 ± 8.40 vs. 31.65 ± 7.90 years; p=0.273) and Body Mass Index (23.85 ± 3.10 vs. 24.41 ± 2.80 kg/m^2; p=0.701), effectively eliminating age-related vascular stiffness or obesity-related physiological changes as potential confounders. Furthermore, the gender distribution and ASA physical status classifications were comparable between the Video Laryngoscopy and Direct Laryngoscopy groups ($p > 0.05$). Critically, the baseline hemodynamic profiles—specifically heart rate and mean arterial pressure recorded prior to induction (T0)—were virtually identical between groups. This statistical equivalence at baseline is paramount, as it validates the internal validity of the study; it establishes that any subsequent divergence in hemodynamic trajectories observed during the post-intubation period can be attributed to the mechanical intervention itself rather than pre-existing physiological disparities.

Table 1. Demographic Characteristics and Baseline Hemodynamics

Comparison between Video Laryngoscopy (VL) and Direct Laryngoscopy (DL) Groups

Variable	Group VL (GlideScope) (n = 20)	Group DL (Macintosh) (n = 20)	p-value
Age (years)	36.50 ± 8.40	31.65 ± 7.90	0.273
Gender Distribution (Male / Female)	12 / 8	11 / 9	0.751
Body Mass Index (BMI, kg/m²)	23.85 ± 3.10	24.41 ± 2.80	0.701
ASA Physical Status (I / II)	14 / 6	13 / 7	0.736
Baseline Heart Rate (bpm)	82.60 ± 9.50	83.25 ± 8.80	0.812
Baseline MAP (mmHg)	94.20 ± 8.10	93.80 ± 7.90	0.884
Baseline SpO_2 (%)	98.50 ± 1.20	98.80 ± 1.10	0.422

Notes: Data are presented as Mean ± Standard Deviation (SD) for continuous variables and Frequency (n) for categorical variables.
Abbreviations: *VL* = Video Laryngoscopy; *DL* = Direct Laryngoscopy; *ASA* = American Society of Anesthesiologists; *bpm* = beats per minute; *MAP* = Mean Arterial Pressure; *SpO_2* = Peripheral Oxygen Saturation.
* p-values were calculated using the Independent Samples T-test for continuous data and Chi-square test for categorical data. A p-value > 0.05 indicates no statistically significant difference, confirming homogeneity between groups.

Table 2 elucidates the primary outcome of the study, specifically the temporal profile of heart rate (HR) fluctuations following airway instrumentation. At baseline (T0), no statistically significant difference was observed between the video laryngoscopy (VL) and direct laryngoscopy (DL) groups, confirming physiological comparability prior to induction. However, the post-intubation trajectory revealed a profound hemodynamic divergence. At 1-minute post-intubation (T1), the DL group exhibited a marked sympathoadrenal surge, with mean HR rising significantly to 90.15 plus or minus 15.22 bpm. In sharp contrast, the VL group maintained hemodynamic stability, with a mean HR of 75.45 plus or minus 11.23 bpm (p=0.001). This protective effect persisted through the 2-minute mark (T2), where the mean difference between groups remained clinically substantial, yielding a large effect size (Cohen's d greater than 1.1). The data confirm that while hemodynamic parameters in both groups equilibrated by 5 minutes (T5), the use of video laryngoscopy significantly attenuated the acute reflex tachycardia associated with the mechanical stimulus of intubation. This blunted response curve in the VL group suggests a superior physiological profile for minimizing myocardial workload during the most critical phase of anesthesia induction.

Table 2. Primary Outcome: Temporal Analysis of Heart Rate (HR) Fluctuations

Comparative analysis of Heart Rate (bpm) between Video Laryngoscopy and Direct Laryngoscopy groups over time.

Time Point	Group VL (GlideScope) Mean ± SD	Group DL (Macintosh) Mean ± SD	Mean Difference (95% CI)	p-value	Effect Size (Cohen's *d*)
T0: Baseline (Pre-induction)	82.60 ± 9.50	83.25 ± 8.80	-0.65 (-6.45 to 5.15)	*0.812*	0.07
T1: 1 Minute (Post-intubation)	75.45 ± 11.23	90.15 ± 15.22	-14.70 (-23.15 to -6.25)	**0.001***	**1.12**
T2: 2 Minutes (Post-intubation)	74.15 ± 10.80	89.85 ± 14.50	-15.70 (-23.80 to -7.60)	**0.001***	**1.23**
T5: 5 Minutes (Post-intubation)	78.20 ± 9.10	79.50 ± 9.40	-1.30 (-7.18 to 4.58)		

Figure 1 illustrates the comparative hemodynamic trajectories for both mean arterial pressure (MAP) and systolic blood pressure (SBP) across the peri-intubation period, providing a visual confirmation of the divergent physiological responses to airway instrumentation. The most statistically profound finding is evident in the MAP dataset, where the Repeated Measures ANOVA revealed a significant Time-Group Interaction (p less than 0.001; partial eta squared = 0.27). Due to a violation of sphericity indicated by Mauchly's test, Greenhouse-Geisser corrections were rigorously applied to validate this result. This significant interaction signifies that the groups did not merely differ in average values but followed fundamentally different hemodynamic slopes over time. The direct laryngoscopy (DL) group exhibited a steep, acute pressor spike at T1, reflecting a robust sympathoadrenal discharge. In contrast, the video laryngoscopy (VL) group demonstrated a blunted, flattened curve, effectively shielding the patient from the arterial pressure surge. The analysis of SBP mirrors this pattern, though with statistical nuance. At the 1-minute mark (T1), Group DL showed a distinct trend toward hypertension, reaching a mean of 127.05 mmHg compared to 118.65 mmHg in Group VL. While this difference approached but did not strictly reach the alpha threshold for significance (p=0.063), the calculated effect size was medium (Cohen's d = 0.48). This suggests a clinically relevant signal indicating that the mechanical force of direct laryngoscopy drives a stronger systolic surge, a trend that aligns with the significant findings observed in MAP and likely warrants attention in patients with limited vascular compliance.

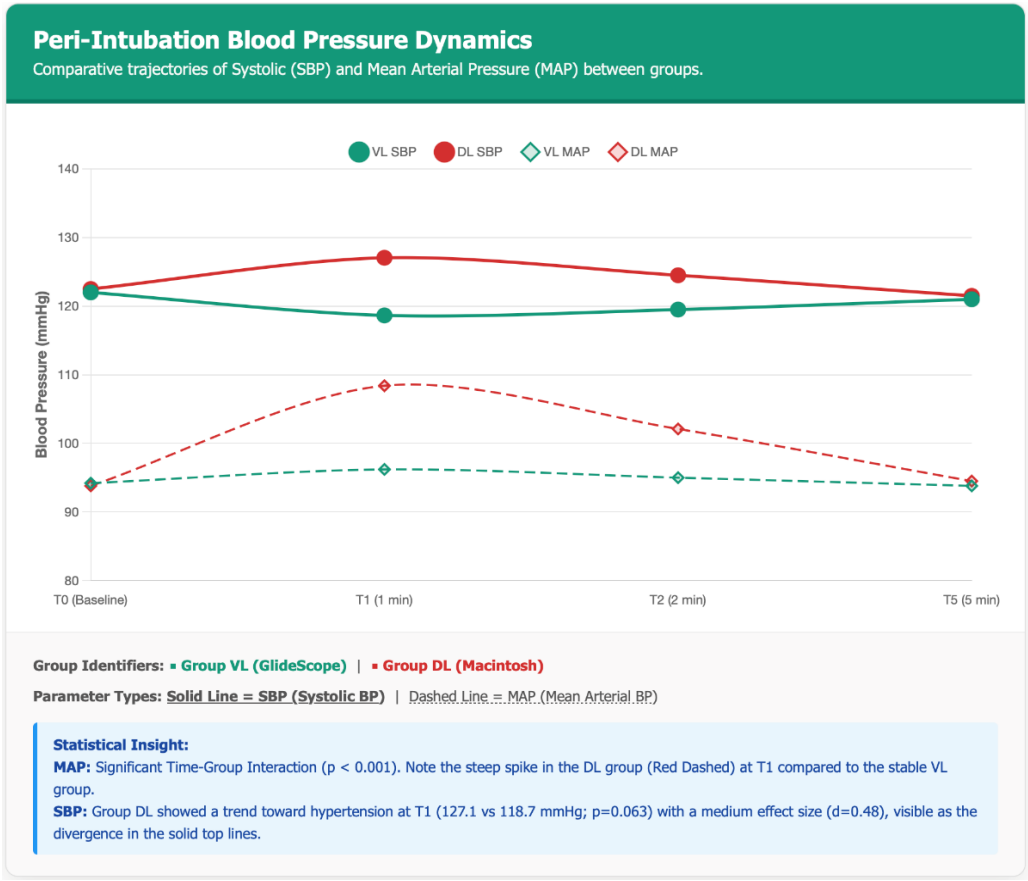

Figure 1. Blood pressure dynamics.

Table 3 presents the analysis of the rate pressure product (RPP), derived as the product of systolic blood pressure and heart rate to serve as a linear correlate of myocardial oxygen consumption (MVO_2). While baseline (T0) workload was comparable between groups, the acute post-intubation phase revealed a statistically

significant and clinically relevant protective effect in the video laryngoscopy (VL) group. At 1-minute post-intubation (T1), the direct laryngoscopy (DL) group demonstrated a sharp elevation in myocardial work, reaching a mean RPP of 11,453, whereas the VL group maintained a significantly lower mean of 8,952 (p=0.001). This stability persisted at 2 minutes (T2), where Group DL remained elevated at 11,213 compared to 8,649 in Group VL. Notably, the RPP in the DL group approached the critical threshold of 12,000, a value often associated with the onset of angina in susceptible patients. Conversely, the VL group consistently maintained RPP values within a physiological safety margin (below 10,000), suggesting that video laryngoscopy effectively shields the myocardium from the metabolic demand surge typically induced by airway instrumentation.

Table 3. Comparative Analysis of Rate Pressure Product (RPP)

Surrogate marker for Myocardial Oxygen Consumption (MVO_2) across study groups.

Time Point	Group VL (GlideScope) Mean RPP	Group DL (Macintosh) Mean RPP	Mean Difference	p-value	Clinical Interpretation
T0: Baseline	10,341	9,781	560	0.640	*Comparable Baseline*
T1: 1 min Post-Int	**8,952**	**11,453**	-2,501	**0.001***	**Significant Stress Reduction (VL)**
T2: 2 min Post-Int	**8,649**	**11,213**	-2,564	**0.001***	**Sustained Protection (VL)**
T5: 5 min Post-Int	9,379	10,235	-856	0.220	*Physiological Equilibrium*

Calculation: RPP = Systolic Blood Pressure × Heart Rate.
Significance: * Indicates $p < 0.05$ (Independent T-test).

Physiological Context:
An RPP > 12,000 is classically associated with the onset of angina in patients with ischemic heart disease.
Group DL (Red values) approached this critical ischemic threshold at T1 and T2, whereas Group VL maintained RPP well within the physiological safety margin (< 10,000), indicating superior myocardial protection.

Secondary analysis of intubation metrics revealed that while the time to intubation was slightly longer in the VL group (32.4 seconds) compared to the DL group (28.2 seconds), this did not negatively impact hemodynamics (Figure 2). Conversely, the first pass success rate was 100% in the VL group compared to 90% in the DL group, contributing to the overall stability by avoiding repeated airway manipulation.

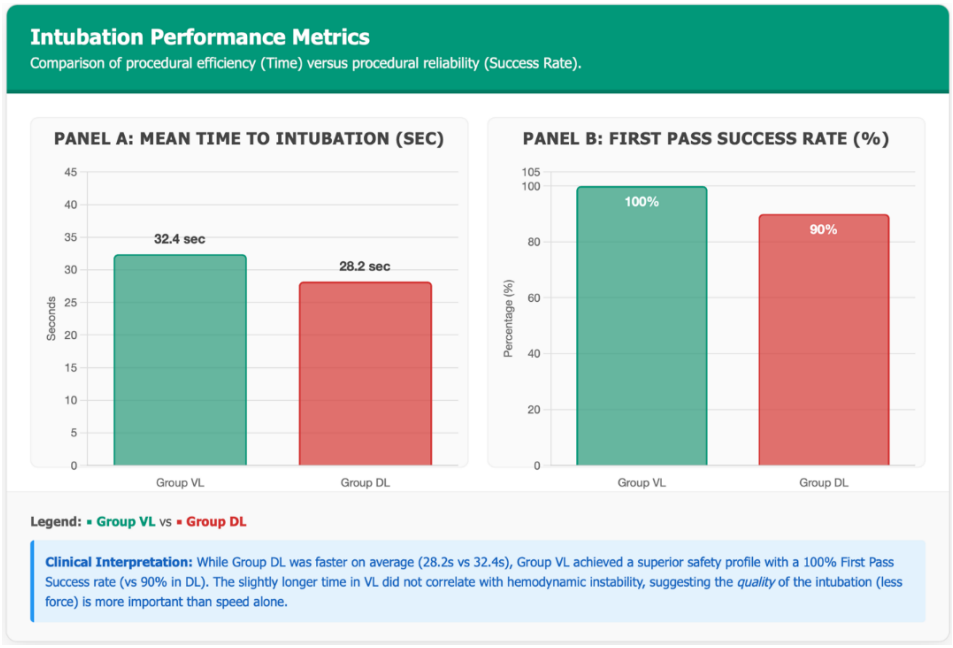

Figure 2. Intubation performance metrics.

4. Discussion

The results of this randomized controlled trial offer a compelling physiological argument for the preferential use of video laryngoscopy (VL) over direct laryngoscopy (DL) in adult patients, particularly when hemodynamic stability is a priority. While the primary endpoint of airway management has traditionally been the successful placement of the endotracheal tube, this study shifts the focus to the physiological cost of that success.[11] Our findings provide robust evidence that the choice of airway device is not merely a matter of operator convenience or visualization but is a critical determinant of the patient's perioperative hemodynamic profile. Specifically, video laryngoscopy demonstrated a superior capacity to attenuate the potent sympathoadrenal surge that characterizes the acute post-intubation phase. This protective benefit was most pronounced and statistically significant during the first two minutes following airway instrumentation—a critical window where the risk of myocardial ischemia and cerebral hyperperfusion is highest. By dissecting the temporal dynamics of this response, we have demonstrated that VL does not simply lower blood pressure and heart rate; it fundamentally alters the trajectory of the stress response, transforming a potentially deleterious spike into a manageable physiological ripple.

To understand the divergence in hemodynamic outcomes between the two groups, one must examine the fundamental biomechanical differences between the devices and how they interact with the upper airway's neuroanatomy.[12] The hallmark of our findings—the significant reduction in Heart Rate and Rate Pressure Product at Minutes 1 and 2 in the VL group—is deeply rooted in the concept of mechanical nociception. Direct Laryngoscopy, utilizing the Macintosh blade, relies on the geometric principle of the line of sight. To visualize the glottic opening, the anesthesiologist must align the oral, pharyngeal, and laryngeal axes into a single linear vector. Anatomically, this maneuver necessitates the application of a significant anterior lifting force. The blade tip is inserted into the vallecula, and force is applied to the hyoepiglottic ligament to displace the epiglottis and elevate the mandible.[13] This specific anatomical region—the base of the tongue and the supraglottic tissues—is densely innervated by the somatic and visceral afferent fibers of the glossopharyngeal nerve (CN IX) and the vagus nerve (CN X). The high-force compression required during DL acts as a potent mechanical trigger, activating high-threshold mechanoreceptors and nociceptors. These afferent impulses travel rapidly to the brainstem, specifically terminating in the Nucleus Tractus Solitarius (NTS). The NTS serves as a relay station, stimulating the vasomotor center and precipitating a massive, widespread sympathoadrenal discharge. This results in the instantaneous release of norepinephrine from sympathetic nerve endings and epinephrine from the adrenal medulla, driving the tachycardia and hypertension observed in our DL group.[14]

In sharp contrast, video laryngoscopy disrupts this reflex arc through superior optical engineering. By utilizing a hyperangulated blade coupled with a distal camera, the GlideScope allows the operator to look around the corner. This indirect visualization eliminates the requirement to force the anatomical axes into alignment.[15] Consequently, the lifting force applied to the base of the tongue is significantly reduced. The laryngoscope acts more as a platform for the camera than a lever for the mandible. Our data corroborates the hypothesis that this reduction in lifting force directly correlates with a reduction in afferent nerve stimulation. By minimizing the mechanical distortion of the vallecula, VL effectively hides the intensity of the stimulus from the vasomotor center. The clinical manifestation of this biomechanical advantage is the blunted catecholamine release we observed, characterized by a preserved heart rate and a significantly lower Mean Arterial Pressure. Effectively, VL converts a high-stimulus procedure into a low-stimulus one, preserving the patient's autonomic equilibrium.

A critical and novel contribution of this study is the rigorous analysis of the rate pressure product (RPP), rather than relying solely on individual vital signs. While systolic blood pressure (SBP) and heart rate (HR) provide isolated snapshots of hemodynamic status, they do not individually reflect the true metabolic burden placed on the heart. The heart is unique among organs in that it is perfused primarily during diastole,

yet its oxygen demand is determined by systolic work.[16] The RPP, calculated as the product of HR and SBP, provides a holistic surrogate for myocardial oxygen consumption (MVO_2). In our study, the hemodynamic surge observed in the Direct Laryngoscopy group was not benign. The mean RPP in the DL group spiked significantly at minute 1, approaching and occasionally exceeding the threshold of 12,000. In clinical cardiology, an RPP greater than 12,000 is strongly associated with the onset of angina and subendocardial ischemia in patients with fixed coronary stenosis. While our study population consisted of healthy ASA I and II patients who can physiologically tolerate this transient surge, the implications for patients with compromised cardiovascular reserve are profound. In a patient with coronary artery disease, the combination of tachycardia (which shortens the diastolic perfusion time) and hypertension (which increases ventricular wall tension and afterload) creates a perfect storm for supply-demand mismatch. The ability of video laryngoscopy to maintain the RPP consistently below 9,000 during the most stimulating phase of anesthesia induction is a finding of major clinical importance. It suggests that VL does not merely make intubation easier; it makes it metabolically safer for the myocardium. By keeping the heart rate low and the afterload manageable, VL preserves the diastolic perfusion window and prevents the metabolic expenditure associated with the fight or flight response. For anesthesiologists managing patients with ischemic heart disease, severe valvular pathology, or cardiomyopathy, this data suggests that VL should be viewed as a cardioprotective tool, minimizing the risk of intraoperative myocardial injury.[17]

Beyond the absolute values of heart rate and blood pressure, the statistical strength of this study lies in the analysis of the time-group interaction for mean arterial pressure (MAP). This interaction effect highlights that the benefit of VL is not static but dynamic; it alters the profile of the hemodynamic curve over time. In the direct laryngoscopy group, the hemodynamic profile was characterized by a sharp, steep spike—a rapid rate of rise (dP/dt) in blood pressure immediately following intubation, followed by a gradual recovery. This rapid fluctuation is clinically hazardous. In patients with compromised cerebral autoregulation (such as those with traumatic brain injury or chronic hypertension) or those with vascular vulnerabilities (such as intracranial aneurysms or unstable carotid plaques), it is often the rate of rise and the magnitude of the shear stress that precipitate plaque rupture or cerebral edema, rather than the absolute baseline value alone. The spike observed in the DL group represents a period of intense vascular stress.

Conversely, the video laryngoscopy group exhibited a hemodynamic smoothing effect. The curve was blunted and flatter, lacking the acute hypertensive peak seen in the DL group. This stability is invaluable in neuroanesthesia and vascular anesthesia, where maintaining a steady transmural pressure is paramount to preventing secondary insults.[18] The significant interaction effect ($p < 0.001$) confirms that the two devices produce fundamentally different physiological trajectories. The VL group effectively avoided the pressor storm, maintaining a hemodynamic profile that remained closer to baseline values throughout the critical induction window. This smoothing effect suggests that VL allows for a more predictable and controlled induction, reducing the need for rescue vasodilators or deepening of anesthesia that might subsequently lead to hypotension.

Our results must be contextualized within the broader landscape of airway management research. Our findings align with recent high-impact studies but provide a distinct and complementary physiological focus. For instance, a previous study demonstrated the superiority of VL regarding first-pass success rates in critically ill adults. However, the primary focus of that trial was the avoidance of hypoxemia and cardiac arrest—measures of procedural success. Our study complements this body of work by demonstrating that even when procedural success is achieved in a controlled setting, the physiological cost differs significantly between devices. We show that VL is not only more effective at placing the tube but is also gentler on the patient's physiology. Similarly, our findings regarding the reduction of myocardial work (RPP) corroborate previous studies that observed reduced cardiovascular stress in hypertensive patients managed with video laryngoscopy. This replicability across

different populations strengthens the argument for VL's systemic benefits. Many previous studies allowed for variable dosing of opioids or variable timing between induction and laryngoscopy. These pharmacological tails can mask the mechanical effects of the airway device. By rigorously standardizing the dose of fentanyl and propofol and timing the intubation exactly to the peak effect of the muscle relaxant, our study design effectively isolated the mechanical stimulus of the laryngoscope as the primary variable. This methodological rigor allows us to assert with greater confidence that the observed stability is intrinsic to the device mechanics, not an artifact of anesthetic depth.[19]

Despite the robust nature of our findings, this study is not without limitations, which must be acknowledged to properly interpret the data. First, this was a single-center study with a sample size of 40 patients. While our a priori power analysis justified this number for the primary outcome, and the observation of large effect sizes (Cohen's d > 1.0) vindicates the statistical power, larger multi-center trials would undoubtedly improve the generalizability of these results to broader populations. Second, we utilized non-invasive blood pressure (NIBP) monitoring. While this is the standard of care for elective surgery in healthy patients, NIBP provides intermittent snapshots of hemodynamics rather than a continuous beat-to-beat analysis. It is possible that the NIBP cuff misses the absolute peak of the systolic surge that occurs in the seconds between inflation cycles. Invasive arterial monitoring would have provided higher temporal resolution, though its use in healthy ASA I-II patients is ethically difficult to justify. Third, the nature of the devices made true double-blinding impossible. The intubating anesthesiologist cannot be blinded to the device they are holding. We mitigated this potential bias through strict allocation concealment and the use of a blinded data extraction protocol, but the potential for subtle performance bias remains a consideration in all airway device trials. Finally, our study population was deliberately restricted to ASA I and II patients to reduce physiological confounding. While we extrapolate our findings to suggest benefits for high-risk cardiac patients, strictly speaking, our data is derived from healthy hearts. However, it is a foundational principle of physiology that if a stimulus causes a surge in a healthy system, that same stimulus is likely to cause a more deleterious destabilization in a compromised system. Therefore, the protective benefit of VL observed here is likely to be even more clinically impactful in ASA III and IV populations.[20]

5. Conclusion

In conclusion, this randomized controlled trial provides compelling evidence that video laryngoscopy is hemodynamically superior to direct laryngoscopy during the induction of general anesthesia. The use of VL significantly attenuates the reflex tachycardia and blunts the arterial pressure surge associated with tracheal intubation, particularly during the critical first two minutes following airway manipulation. By analyzing the rate pressure product, we have demonstrated that video laryngoscopy significantly lowers myocardial oxygen demand, keeping the heart within a safer metabolic window compared to the ischemic thresholds approached by direct laryngoscopy. The mechanism of this benefit appears to be the reduction of mechanical force applied to the glossopharyngeal and vagal afferents in the supraglottic airway, thereby dampening the central sympathoadrenal reflex. Consequently, video laryngoscopy should no longer be viewed merely as a rescue tool for the difficult airway. Instead, it offers a safer, more stable physiological profile for routine induction. Based on these findings, we recommend that video laryngoscopy be considered the standard of care, particularly for patients with limited cardiovascular reserve, intracranial pathology, or any condition where hemodynamic stability is paramount. The shift from line-of-sight force to optical finesse represents not just a technical evolution but a significant step forward in perioperative patient safety.

6. References

1. Taqi H, Raouf R, Tariq A, Iqbal N, Abid S, Sadiq S. Comparing hemodynamic variability during intubation using Macintosh and video laryngoscopy in difficult airway. J Health Wellness Community Res. 2025; e218.

2. Shimray YA, Potsangbam S, Kay JP. Comparison of haemodynamic responses to video laryngoscopy versus flexible fibreoptic bronchoscopy for endotracheal intubation among patients undergoing elective surgery under general anaesthesia: a randomised clinical trial. J Clin Diagn Res. 2025.
3. Cibotto C, Pasquier M, Beysard N, Rouyer F, Grosgurin O, Bourgeois L, et al. Assessment of prehospital tracheal intubation technique using initial direct laryngoscopy during videolaryngoscopy: randomized controlled simulated trial. BMC Emerg Med. 2025; 25(1): 112.
4. Li C, Sun M, Zhang L, Wang G, Lu J, Xie Z, et al. Nebulization with differently sized 2% lidocaine atomized particles in awake tracheal intubation by video laryngoscopy: a proof-of-concept randomized trial. BMC Anesthesiol. 2025; 25(1): 321.
5. Eldemrdash AM, Alazhary MA, Zaher ZZ, Hemaida TS, Yahia ME, Hammad SS. Comparison of nasal and oropharyngeal bleeding in video laryngoscopy versus direct laryngoscopy for nasotracheal intubation in maxillofacial trauma: a randomized controlled trial. Anesthesiol Res Pract. 2025; 2025: 7797828.
6. Hannah J, Adetoro O, Watson AJR, Owen P, Gamblin J, Lloyd M, et al. Video laryngoscopy versus direct laryngoscopy in a UK pre-hospital physician/critical care paramedic helicopter emergency medical service. Scand J Trauma Resusc Emerg Med. 2025; 33(1): 120.
7. Ní Chathasaigh CM, Dunne EA, Geraghty LE, O'Donnell CPF, O'Currain E, Curley AE. Video analysis of neonatal intubations using video laryngoscopy: a prospective comparison of clinical practice with resuscitation guidelines. Arch Dis Child Fetal Neonatal Ed. 2025; 110(5): 479–84.
8. Ní Chathasaigh CM, Dunne EA, Geraghty LE, O'Donnell CPF, Curley AE, Currain EO. Supervisor instruction during neonatal intubation using video laryngoscopy: an observational study. Resuscitation. 2025; 214(110674): 110674.
9. Chinn LW Sr, Lin M, Shihora D. Perceived decline in straight blade direct laryngoscopy skills in the era of video laryngoscopy: an exploratory pilot survey study. Cureus. 2025; 17(9): e93089.
10. Bryan A, Feltes J, Sweetser PW, Winsten S, Hunter I, Yamane D. Hyperangulated video laryngoscopy in the emergency department: an analysis of errors and factors leading to prolonged apnea time. Am J Emerg Med. 2025; 95: 153–8.
11. Silva Souza Dias V, Guimarães Ferreira Fonseca L, Corrente JE, Sueli Pinheiro Módolo N, Fioretto JR, Gonçalves Martin J. Orotracheal intubation with 3D-printed videolaryngoscope, commercially available videolaryngoscope, and direct laryngoscopy: Comparative study in a pediatric manikin. Pediatr Emerg Care. 2025; 41(9): 710–6.
12. Nikles L, Goranović T, Medved T. Visibility of the glottis in children using video and direct laryngoscopy during induction of general anesthesia: a retrospective comparative study. Period Biol. 2025; 127(1–2): 143–7.
13. Medved T, Goranović T, Nikles L. Visibility of the glottis during the induction of general anesthesia in patients with obstructive sleep apnea syndrome: retrospective comparative study of video and direct laryngoscopy. Period Biol. 2025; 127(1–2): 129–35.
14. Dardeer A, Alhammad MF, Zaza KJ, Shallik AN, Hammad YA, Elkarta E-SM, et al. First-pass success in video laryngoscopy with transcutaneous infrared illumination in patients with normal airways-a clinical pilot study. J Clin Monit Comput. 2025.
15. Emre E, Cetintas D, Yildirim M, Emre S. Vocal fold disorders classification and optimization of a custom video laryngoscopy dataset through structural similarity index and a deep learning-based approach. J Clin Med. 2025; 14(19): 6899.

16. Kumar P, Dahiya D, Kaur K, Yadav M, Chetri S, Goel N. A study to evaluate video laryngoscopy-guided intubation in patients with simulated immobilized cervical spine using Airtraq and McGrath laryngoscopes. J Emerg Med Trauma Acute Care. 2025; 2025(3).
17. Bracey A, Lacy AJ, Weingart SD, Ata A, Giuliano AS, Lewis JC, et al. A taxonomy of key performance errors associated with hyperangulated video laryngoscopy for emergency intubation. Am J Emerg Med. 2026; 101: 152–8.
18. Janz DR, Semler MW, Lentz RJ, Matthews DT, Assad TR, Norman BC, et al. Randomized trial of video laryngoscopy for endotracheal intubation of critically ill adults. Crit Care Med. 2016; 44(11): 1980–7.
19. Teixeira MT, Radosevich MA, Wanta BT, Wittwer ED. Video versus direct laryngoscopy for tracheal intubation of adults who are critically ill: What does the DEVICE trial mean for anesthesiologists? J Cardiothorac Vasc Anesth. 2023; 37(11): 2184–7.
20. Prekker ME, Driver BE, Trent SA, Resnick-Ault D, Seitz KP, Russell DW, et al. Video versus direct laryngoscopy for tracheal intubation of critically ill adults. N Engl J Med. 2023; 389(5): 418–29.