

ORIGINAL ARTICLE

Video Versus Direct Laryngoscopy and The Hemodynamic Response and Myocardial Workload During Tracheal Intubation Under Standardized Anesthetic Induction: A Randomized Trial

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ABSTRACT

Background. Tracheal intubation triggers a sympathoadrenal reflex that raises heart rate and blood pressure. Whether video laryngoscopy (VL) reduces this response is debated.

Objective. To compare GlideScope VL with Macintosh direct laryngoscopy (DL) for the hemodynamic response and rate pressure product (RPP) under standardized induction.

Methods. In this single-blind randomized trial, 40 adults (ASA I–II) received VL (n = 20) or DL (n = 20). Induction was fentanyl 2 mcg/kg, propofol 2 mg/kg, atracurium 0.5 mg/kg, and 1.5% sevoflurane, with laryngoscopy 3 minutes after the relaxant. Heart rate (HR), systolic (SBP) and mean arterial pressure (MAP) were recorded at baseline (T0) and 1 (T1), 2 (T2), and 5 (T5) minutes after intubation.

Results. HR was lower with VL at T1 (75.45 ± 11.23 vs. 90.15 ± 15.22 bpm, $p = 0.001$) and T2 (74.15 ± 10.80 vs. 89.85 ± 14.50 bpm, $p < 0.001$). MAP showed a Time $\times$ Group interaction ($p < 0.001$) and was lower with VL at T1 (96.30 ± 7.80 vs. 108.40 ± 9.60 mmHg, $p < 0.001$). SBP at T1 did not differ significantly ($p = 0.052$). RPP was lower with VL at T1 ($8,952 \pm 1,480$ vs. $11,453 \pm 2,210$) and T2 ($8,649 \pm 1,390$ vs. $11,213 \pm 2,150$ bpm-mmHg; both $p < 0.001$). First-pass success was 100% vs. 90% ($p = 0.487$).

Conclusion. In young ASA I–II patients, VL was associated with lower HR, MAP, and RPP 1–2 minutes after intubation than DL; SBP and first-pass success did not differ significantly.

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1. Introduction

Tracheal intubation is the standard way to secure the airway during general anesthesia, but exposing the glottis and passing the tube are among the strongest noxious stimuli of the perioperative period.^{1–3} The stimulus starts a somatosensory and neurohumoral reflex, and it arrives while the cardiovascular depression of induction is at its peak.⁴ Afferent fibers from the tongue base, vallecula, epiglottis, and vocal cords run in the glossopharyngeal nerve and the internal branch of the superior laryngeal nerve (a branch of the vagus). They reach the nucleus tractus solitarius and the rostral ventrolateral medulla and lead to preganglionic sympathetic outflow.^{5–8}

The efferent limb releases norepinephrine and epinephrine, which raise vascular resistance and, through beta-1 receptors, heart rate and contractility.^{9–11} Healthy patients usually tolerate this brief response. In patients with coronary artery disease, intracranial pathology, or vascular aneurysms it may be harmful.¹² A higher heart rate and systolic pressure increase myocardial oxygen demand and shorten diastole, when most coronary perfusion occurs.^{13,14} The rate pressure product (RPP), systolic pressure multiplied by heart rate, is a clinical surrogate for myocardial oxygen consumption, and values above about

12,000 bpm-mmHg have been used as a marker of increased ischemic risk.³

The size of the response is thought to depend on the force that the laryngoscope puts on the tongue base.⁵ Direct laryngoscopy (DL) with a Macintosh blade needs line-of-sight alignment of the oral, pharyngeal, and laryngeal axes and a considerable lifting force, often cited as 35 to 45 N.^{6,7} Video laryngoscopy (VL) with a hyperangulated blade, such as the GlideScope, gives an indirect view around the tongue base and is expected to need less force (about 5 to 15 N) and to cause less afferent stimulation.^{9–11} Clinical trials disagree. Some report blunted hemodynamic responses with VL, and others find no difference.¹² Differences in depth of induction, opioid dose, and the interval between relaxant and laryngoscopy may explain part of the inconsistency.¹³

In routine practice, hemodynamic instability in the first minutes of induction is often accepted as unavoidable.^{8,9} Yet short episodes of hypertension or hypotension after induction have been associated with postoperative myocardial injury, acute kidney injury, and longer recovery.^{10,11} Many surgical patients in Indonesia and the region have undiagnosed or poorly controlled cardiovascular risk factors.^{6,7} Procedural success alone, such as placement of the tube at the first attempt, does not describe the physiological cost of airway instrumentation.^{12–14}

This randomized trial tested whether GlideScope VL reduces the hemodynamic response and RPP after tracheal intubation, compared with Macintosh DL, when induction is standardized with weight-based fentanyl, propofol, and atracurium and a fixed 3-minute interval before laryngoscopy.^{1,2} We hypothesized that VL would reduce the rise in heart rate and mean arterial pressure and keep the RPP lower during the first two minutes after intubation.^{3,4}

2. Methods

2.1. Study design, setting, and ethics

This was a prospective, parallel-group, single-blind, randomized controlled trial, reported according to the CONSORT 2010 statement.¹⁵ It took place in the Central Operating Theatres of Dr. Saiful Anwar Regional General Hospital, Faculty of Medicine, Universitas Brawijaya, Malang, East Java, Indonesia, from January to December 2025. The protocol was approved by the CMHC Research Center Ethics Committee, Indonesia (Reference number 2024/0912) and followed the Declaration of Helsinki and the ICH Good Clinical Practice guidelines.^{15,16} During the preoperative anesthetic evaluation, every eligible patient received a verbal and written explanation of the study and gave written informed consent.¹⁷

2.2. Participants

We included adults aged 20 to 55 years with ASA physical status I or II who were scheduled for elective surgery under general endotracheal anesthesia with mechanical ventilation.^{18,19} To limit anatomical variation, patients also needed a modified Mallampati class I or II, an anticipated Cormack-Lehane grade I or II, a thyromental distance above 6.0 cm, an inter-incisor gap above 3.0 cm, and normal cervical spine mobility.²⁰ Exclusion criteria were an anticipated difficult airway (Mallampati class III or IV, retrognathia, limited neck extension), body mass index (BMI) above 35.0 kg/m², known coronary artery disease, uncontrolled hypertension at baseline (systolic BP > 140 mmHg or diastolic BP > 90 mmHg), resting dysrhythmia, long-term beta-blocker or calcium channel blocker therapy, severe gastroesophageal reflux disease, and a history of adverse anesthetic reactions.²¹

2.3. Sample size

The sample size was based on the difference in heart rate (HR) between groups 1 minute after intubation.²² We assumed a difference of 12 bpm and a standard deviation (SD) of 13 bpm, taken from preliminary institutional pilot observations and published variance data.²³ With a two-sided α of 0.05 and power of 80%, 19 patients per arm were required ($n = 2 \times (1.96 + 0.84)^2 \times 13^2 / 12^2 = 18.4$, rounded up).²⁴ The submitted text gave 18 per arm. Enrollment was set at 20 per group to allow for dropouts, protocol deviations, and measurement artifacts.²⁵

2.4. Randomization and blinding

Participants were allocated 1:1 to Group VL (GlideScope video laryngoscope with a hyperangulated blade, size 3 or 4) or Group DL (Macintosh direct laryngoscope with a curved blade, size 3 or 4).²⁶ A computer-generated random permuted block sequence (block size 4) was used. An independent clinical research coordinator who took no part in perioperative care prepared sequentially numbered, opaque, sealed envelopes (SNOSE). The attending anesthesia team opened the assigned envelope in the operating

theatre, after baseline monitoring was in place and just before induction.^{15,16} Blinding of the operator was impossible. The outcome assessor who acquired the physiological data sat behind an opaque barrier that hid the operator's hands and the airway equipment.^{17,18} Hemodynamic data were stored in the memory of a multiparameter monitor (Mindray BeneVision N17, Shenzhen, China) and exported afterward by an independent investigator who was unaware of group assignment.¹⁹

2.5. Anesthesia and intubation

On arrival in the operating theatre, patients lay in the sniffing position. Continuous five-lead electrocardiography (ECG), non-invasive blood pressure (NIBP) with an appropriately sized arm cuff, and pulse oximetry (SpO₂) were started.^{20,21} An 18-gauge cannula was placed in an upper-extremity vein, and Ringer's lactate ran at 5 mL/kg/h. Patients rested undisturbed for 5 minutes before baseline values were recorded.²² Pre-oxygenation with 100% oxygen at 8 L/min through a tight-fitting face mask lasted exactly 3 minutes.²³

Induction was uniform in both groups.²⁴ Fentanyl 2.0 mcg/kg was given intravenously. Sixty seconds later, propofol 2.0 mg/kg was given over 30 seconds until the eyelash reflex was lost and the patient no longer answered verbally.²⁵ Atracurium 0.5 mg/kg followed immediately.²⁶ Gentle manual mask ventilation with 100% oxygen and 1.5% sevoflurane then continued for exactly 3 minutes.¹⁶ All intubations were done by senior anesthesiologists, each with more than 200 intubations with both devices.¹⁵ Tube placement was confirmed by capnography (EtCO₂ > 30 mmHg for 5 consecutive breaths) and chest auscultation.¹⁷ No surgical draping, incision, or other stimulation was allowed during the 5-minute study window.¹⁸

Before enrollment, all attending anesthesiologists and research assistants completed a protocol calibration on airway manikins to harmonize blade insertion depth, lifting technique, and data logging.^{18–20} Before each case the GlideScope was checked for camera focus, the anti-fogging heater, and blade integrity.²¹ In Group DL, stainless steel Macintosh blades (size 3 for women, size 4 for men) were used with a fiberoptic light source of at least 2,500 lux.²²

2.6. Outcomes and measurements

The primary outcome was HR 1 minute after intubation. Secondary outcomes were systolic (SBP), diastolic (DBP), and mean arterial pressure (MAP), the rate pressure product (RPP = SBP × HR), time to intubation (TTI, from passage of the blade beyond the incisors to capnography-confirmed tube placement), first-pass success, number of attempts, and Cormack-Lehane grade.^{19–21} Values were logged at baseline (T0, after 5 minutes of rest before induction) and at 1 (T1), 2 (T2), and 5 minutes (T5) after intubation.¹⁹ Software logged ECG, plethysmography, and oscillometric pressure every 5 seconds, and the NIBP cuff was set to cycle at T0, T1, T2, and T5 from a stopwatch started at tube cuff inflation.^{19–24} Manual overrides were not allowed during the observation period.^{25,26} No measurement was planned between the end of induction and laryngoscopy.

Safety limits were set in advance.^{23,24} Severe hypertension was SBP above 160 mmHg or MAP above 115 mmHg for more than two consecutive cycles, and severe tachycardia was HR above 100 bpm.^{25,26} Persistent hypertension or tachycardia beyond the study window was treated with esmolol 0.5 mg/kg or fentanyl 1 mcg/kg, and hypotension (MAP < 65 mmHg) with ephedrine 5 mg and a crystalloid

bolus.^{15,16} Rescue treatment, airway mucosal bleeding, dental injury, and sore throat at 24 hours were recorded on case report forms.^{17,18}

2.7. Statistical analysis

Analyses used IBM SPSS Statistics, version 26.0 (IBM Corp., Armonk, NY, USA). Normality was checked with the Shapiro-Wilk test.²² Continuous variables are given as mean $\pm$ SD and were compared with the independent-samples t-test (Welch's correction when Levene's test showed unequal variances).²³ Categorical variables are given as n (%) and were compared with Fisher's exact test, or Pearson's chi-square test when expected counts were adequate.²⁴ Changes over time were analyzed with repeated-measures analysis of variance (ANOVA), with Time as the within-subject factor and Group as the between-subject factor. Sphericity was tested with Mauchly's test, and the Greenhouse-Geisser correction was applied when it was violated.^{25,26} Effect sizes are Cohen's d and partial eta

Table 1. Baseline characteristics of the study patients (n = 40)

Characteristic	Group VL (GlideScope, n = 20)	Group DL (Macintosh, n = 20)
Age, years	36.50 $\pm$ 8.40	31.65 $\pm$ 7.90
Male / female, n (%)	12 (60.0) / 8 (40.0)	11 (55.0) / 9 (45.0)
Body mass index, kg/m ²	23.85 $\pm$ 3.10	24.41 $\pm$ 2.80
ASA physical status I / II, n (%)	14 (70.0) / 6 (30.0)	13 (65.0) / 7 (35.0)
Heart rate, bpm	82.60 $\pm$ 9.50	83.25 $\pm$ 8.80
Systolic blood pressure, mmHg	122.05 $\pm$ 10.40	122.60 $\pm$ 9.80
Diastolic blood pressure, mmHg	80.25 $\pm$ 7.30	79.40 $\pm$ 6.90
Mean arterial pressure, mmHg	94.20 $\pm$ 8.10	93.80 $\pm$ 7.90
SpO ₂ , %	98.50 $\pm$ 1.20	98.80 $\pm$ 1.10

Notes: Values are mean $\pm$ SD unless stated. Hemodynamic values were recorded after 5 minutes of rest before induction. No significance tests were applied to baseline characteristics (CONSORT 2010). ASA, American Society of Anesthesiologists; SpO₂, peripheral oxygen saturation.

squared (η^2).¹⁶ Exploratory multivariable linear regression (4 predictors, n = 40) was used to examine factors related to MAP at T1.¹⁵ No significance tests were applied to baseline characteristics. A two-sided p < 0.05 was considered significant, with no adjustment for multiple comparisons.¹⁷

3. Results

3.1. Participants and baseline characteristics

Between January and December 2025, 48 adults were assessed for eligibility and 8 were excluded: 5 did not meet the inclusion criteria (2 predicted difficult airway, 2 BMI > 35.0 kg/m², and 1 on long-term rate-limiting antihypertensive therapy) and 3 declined. Forty patients were randomized to Group VL (n = 20) or Group DL (n = 20), and all completed the protocol, with no loss to follow-up and no protocol violation (Figure 1).

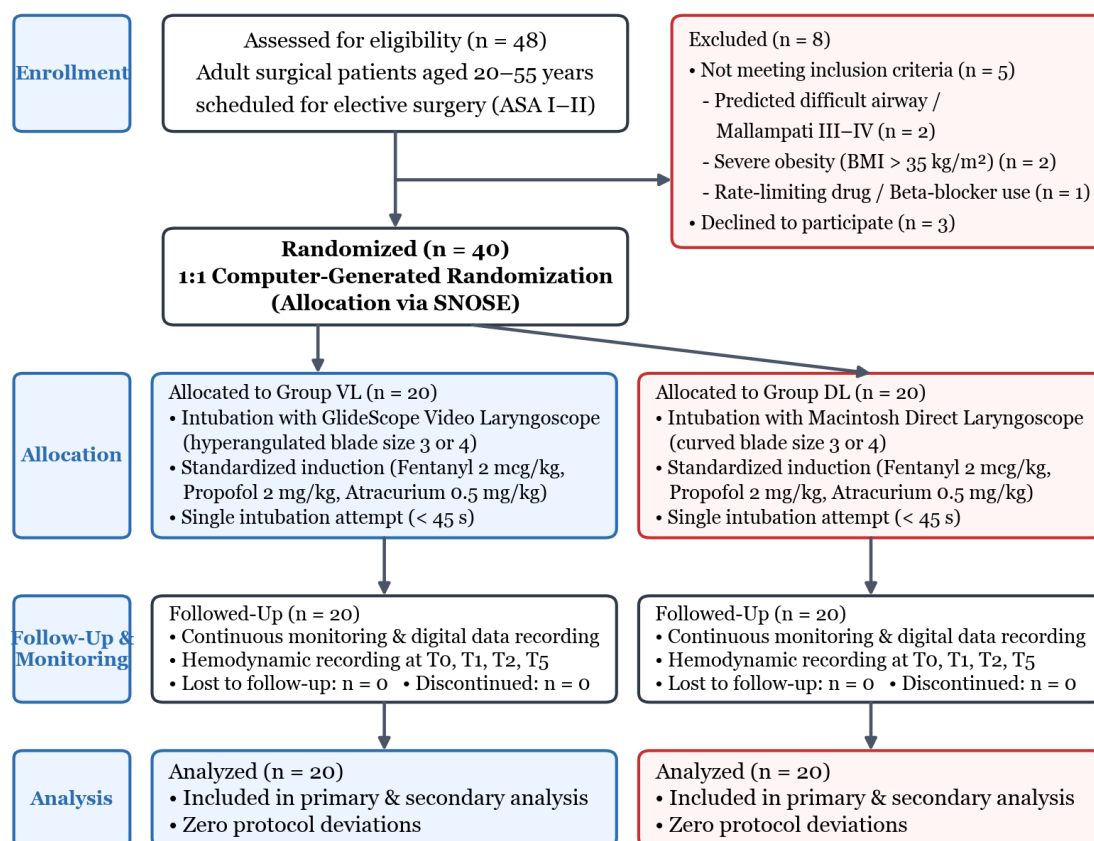


Figure 1. CONSORT 2010 flow diagram of participant screening, randomization, allocation, follow-up, and analysis. VL, video laryngoscopy; DL, direct laryngoscopy; SNOSE, sequentially numbered, opaque, sealed envelopes.

Baseline characteristics are given in Table 1. Mean age was 36.50 ± 8.40 years in Group VL and 31.65 ± 7.90 years in Group DL (standardized difference 0.59). Group VL included 12 men (60.0%) and 8 women (40.0%), and Group DL 11 men (55.0%) and 9 women (45.0%). ASA physical status I/II was 14/6 in Group VL and 13/7 in Group DL. Baseline HR, SBP, DBP, MAP, and SpO_2 were similar.

3.2. Heart rate

Heart rate differed after intubation (Table 2, Figure 2A). Baseline HR was 82.60 ± 9.50 bpm in Group VL and 83.25 ± 8.80

± 8.80 bpm in Group DL. At T1, HR was 75.45 ± 11.23 bpm with VL and 90.15 ± 15.22 bpm with DL (mean difference -14.70 bpm, 95% CI -23.15 to -6.25 , $p = 0.001$, Cohen's $d = 1.12$). At T2 it was 74.15 ± 10.80 and 89.85 ± 14.50 bpm (mean difference -15.70 bpm, 95% CI -23.80 to -7.60 , $p < 0.001$, $d = 1.23$). By T5 the groups were similar (78.20 ± 9.10 vs. 79.50 ± 9.40 bpm, $p = 0.658$). From baseline to T1, mean HR rose by 6.90 bpm (8.3%) in Group DL and fell by 7.15 bpm (8.7%) in Group VL.

Table 2. Heart rate (bpm) before and after intubation

Time point	Group VL	Group DL	Mean difference (95% CI)	p	Cohen's d
T0, baseline	82.60 ± 9.50	83.25 ± 8.80	-0.65 (-6.45 to 5.15)	0.812	0.07
T1, 1 min	75.45 ± 11.23	90.15 ± 15.22	-14.70 (-23.15 to -6.25)	0.001	1.12
T2, 2 min	74.15 ± 10.80	89.85 ± 14.50	-15.70 (-23.80 to -7.60)	< 0.001	1.23
T5, 5 min	78.20 ± 9.10	79.50 ± 9.40	-1.30 (-7.18 to 4.58)	0.658	0.14

Notes: Values are mean $\pm$ SD. Independent-samples t-test (Welch's correction where appropriate). T0, before induction; T1, T2, and T5, 1, 2, and 5 minutes after intubation. VL, video laryngoscopy; DL, direct laryngoscopy; CI, confidence interval.

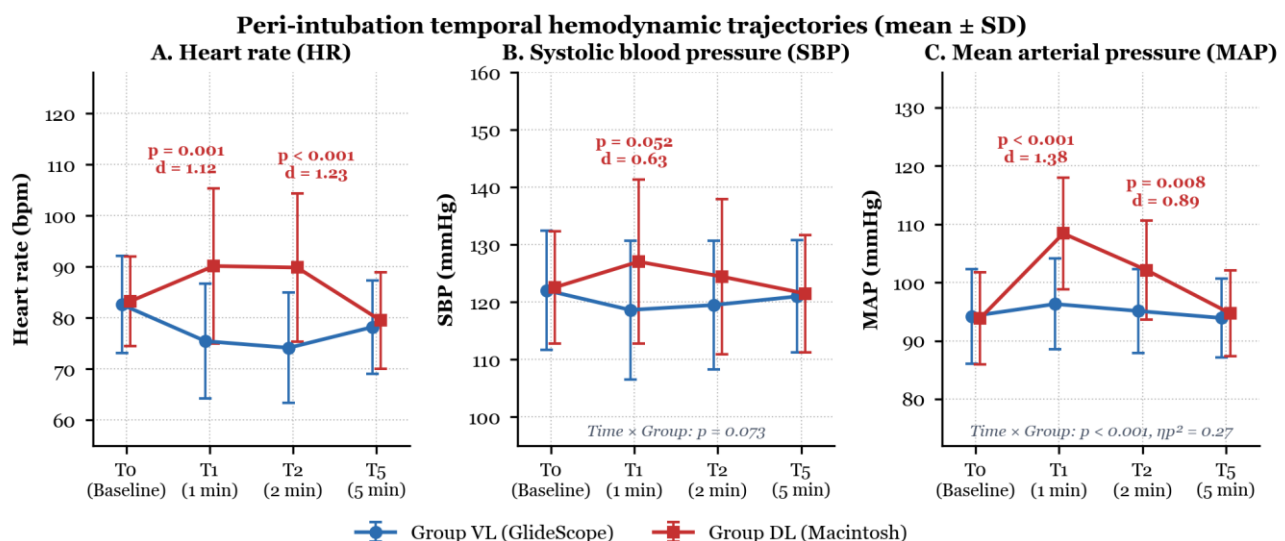


Figure 2. Heart rate (A), systolic blood pressure (B), and mean arterial pressure (C) at baseline (T0) and 1 (T1), 2 (T2), and 5 minutes (T5) after intubation (mean $\pm$ SD; $n = 20$ per group). p values are for between-group comparisons at that time point; d is Cohen's d. The Time $\times$ Group interaction is from repeated-measures ANOVA.

3.3. Blood pressure

MAP and SBP over time are shown in Table 3 and Figure 2B and 2C. For MAP, repeated-measures ANOVA showed effects of Time ($F = 21.84$, $p < 0.001$) and Group ($F = 9.85$, $p = 0.003$) and a Time $\times$ Group interaction ($F(2.41, 91.58) = 14.12$, $p < 0.001$, $\eta^2 = 0.27$). Sphericity was violated (Mauchly's $W = 0.62$, $p = 0.002$), so the Greenhouse-Geisser correction ($\epsilon = 0.803$) was used. In Group DL, MAP rose by 15.57% from baseline to 108.40 ± 9.60 mmHg at T1; in

Group VL it rose by 2.23% to 96.30 ± 7.80 mmHg. The difference at T1 was -12.10 mmHg (95% CI -17.65 to -6.55 , $p < 0.001$, $d = 1.38$). At T2 it was -7.00 mmHg (95% CI -12.02 to -1.98 , $p = 0.008$, $d = 0.89$), and at T5 the groups were similar ($p = 0.724$).

SBP at T1 was 118.65 ± 12.10 mmHg with VL and 127.05 ± 14.30 mmHg with DL (mean difference -8.40 mmHg, 95% CI -16.88 to 0.08 , $p = 0.052$, $d = 0.63$). This difference was not significant, nor was the Time $\times$ Group interaction for SBP ($F(2.68, 101.8) = 2.45$, $p = 0.073$, $\eta^2 = 0.06$).

Table 3. Systolic and mean arterial blood pressure (mmHg) before and after intubation

Variable and time point	Group VL	Group DL	Mean difference (95% CI)	p
Systolic blood pressure				
T0, baseline	122.05 ± 10.40	122.60 ± 9.80	-0.55 (-6.98 to 5.88)	0.865
T1, 1 min	118.65 ± 12.10	127.05 ± 14.30	-8.40 (-16.88 to 0.08)	0.052
T2, 2 min	119.50 ± 11.20	124.45 ± 13.50	-4.95 (-12.82 to 2.92)	0.218
T5, 5 min	121.00 ± 9.80	121.50 ± 10.20	-0.50 (-6.85 to 5.85)	0.876
Mean arterial pressure				
T0, baseline	94.20 ± 8.10	93.80 ± 7.90	0.40 (-4.71 to 5.51)	0.884
T1, 1 min	96.30 ± 7.80	108.40 ± 9.60	-12.10 (-17.65 to -6.55)	< 0.001
T2, 2 min	95.10 ± 7.20	102.10 ± 8.50	-7.00 (-12.02 to -1.98)	0.008
T5, 5 min	93.90 ± 6.80	94.70 ± 7.40	-0.80 (-5.32 to 3.72)	0.724

Notes: Values are mean ± SD. Repeated-measures ANOVA: systolic pressure, Time × Group $F(2,68, 101.8) = 2.45$, $p = 0.073$, $\eta^2 = 0.06$; mean arterial pressure, Time × Group $F(2,41, 91.58) = 14.12$, $p < 0.001$, $\eta^2 = 0.27$ (Mauchly's $W = 0.62$, $p = 0.002$; Greenhouse-Geisser $\epsilon = 0.803$). The systolic pressure difference at T1 was recomputed from the printed means and SDs (see Change Log). VL, video laryngoscopy; DL, direct laryngoscopy.

3.4. Rate pressure product

RPP over time is given in Table 4 and Figure 3. At baseline, RPP was $10,341 \pm 1,520$ bpm·mmHg in Group VL and $9,781 \pm 1,410$ bpm·mmHg in Group DL ($p = 0.235$). At T1, RPP was $8,952 \pm 1,480$ bpm·mmHg with VL and $11,453 \pm 2,210$ bpm·mmHg with DL (mean difference $-2,501$ bpm·mmHg,

95% CI $-3,695$ to $-1,307$, $p < 0.001$). At T2 it was $8,649 \pm 1,390$ and $11,213 \pm 2,150$ bpm·mmHg (mean difference $-2,564$, 95% CI $-3,712$ to $-1,416$, $p < 0.001$). The difference at T5 ($9,379 \pm 1,280$ vs. $10,235 \pm 1,510$ bpm·mmHg; mean difference -856 , 95% CI $-1,745$ to 33) was not significant ($p = 0.061$). Mean RPP in Group DL did not reach $12,000$ bpm·mmHg at any time point.

Table 4. Rate pressure product (bpm·mmHg) before and after intubation

Time point	Group VL	Group DL	Mean difference (95% CI)	p
T0, baseline	10,341 ± 1,520	9,781 ± 1,410	560 (-375 to 1,495)	0.235
T1, 1 min	8,952 ± 1,480	11,453 ± 2,210	-2,501 (-3,695 to -1,307)	< 0.001
T2, 2 min	8,649 ± 1,390	11,213 ± 2,150	-2,564 (-3,712 to -1,416)	< 0.001
T5, 5 min	9,379 ± 1,280	10,235 ± 1,510	-856 (-1,745 to 33)	0.061

Notes: Values are mean ± SD. RPP = systolic blood pressure × heart rate. Independent-samples t-test. p values at T0 and T5 were recomputed from the printed statistics (see Change Log). [Author query: baseline, T2, and T5 RPP values do not follow from the printed mean SBP and HR; please verify.] VL, video laryngoscopy; DL, direct laryngoscopy.

Rate pressure product (RPP) dynamics and myocardial workload margins

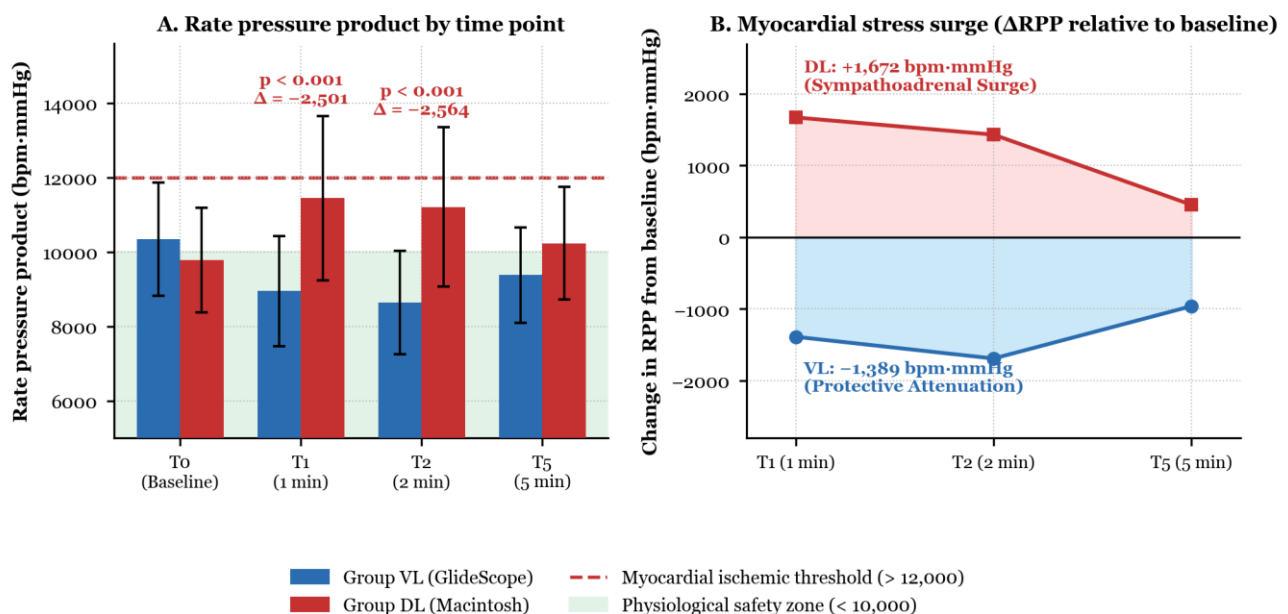


Figure 3. Rate pressure product (RPP) over time. (A) RPP by time point (mean ± SD) with the thresholds of 10,000 and 12,000 bpm·mmHg used in this paper; (B) change in RPP from baseline.

3.5. Airway view, procedure, and regression

Procedural results are in Table 5 and Figure 4. Time to intubation was longer with VL (32.4 ± 4.8 vs. 28.2 ± 3.9 seconds; mean difference 4.2 seconds, 95% CI 1.4 to 7.0, $p = 0.004$). All 20 patients in Group VL had a Cormack-Lehane

grade I view, compared with 15 (75.0%) in Group DL; the other 5 had grade II (Fisher's exact test, $p = 0.047$). First-pass success was 100.0% (20 of 20) with VL and 90.0% (18 of 20) with DL (Fisher's exact test, $p = 0.487$); two Group DL patients needed a second attempt. No patient failed intubation.

Table 5. Procedural outcomes

Outcome	Group VL (n = 20)	Group DL (n = 20)	Effect (95% CI)	p
Time to intubation, seconds	32.4 ± 4.8	28.2 ± 3.9	MD +4.2 (1.4 to 7.0)	0.004
First-pass success, n (%)	20 (100.0)	18 (90.0)	Fisher's exact test	0.487
Attempts, mean ± SD	1.00 ± 0.00	1.10 ± 0.31		
Cormack-Lehane grade I / II, n (%)	20 (100.0) / 0 (0.0)	15 (75.0) / 5 (25.0)	Fisher's exact test	0.047

Notes: Values are mean ± SD unless stated. Time to intubation was compared with the independent-samples *t*-test. The submitted *t* test for the number of attempts was removed because one group had zero variance. MD, mean difference; VL, video laryngoscopy; DL, direct laryngoscopy.

Procedural efficiency, first-pass reliability, and hemodynamic decoupling

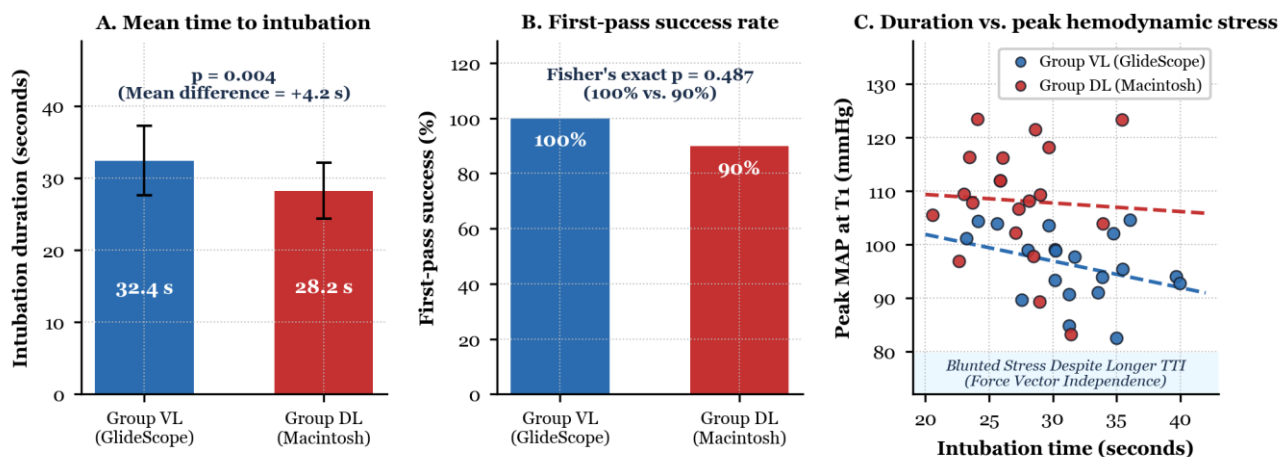


Figure 4. (A) Time to intubation (mean ± SD). (B) First-pass success. (C) Individual MAP at T1 against intubation time, with the fitted line for each group. One Group VL point (about 31.3 seconds) was partly hidden in the original figure and its position was estimated.

In the multivariable linear regression for MAP at T1 (Table 6), device was the strongest predictor (adjusted $\beta = -12.35$ mmHg for VL vs. DL, 95% CI -17.52 to -7.18 , $p < 0.001$; $R^2 = 0.465$, adjusted $R^2 = 0.404$, $F(4, 35) = 7.61$, $p < 0.001$). Baseline MAP was also associated with MAP at T1 ($\beta =$

$+0.38$ per mmHg, 95% CI $+0.06$ to $+0.70$, $p = 0.021$), whereas age ($p = 0.468$) and BMI ($p = 0.576$) were not. Figure 4C shows MAP at T1 against intubation time for each patient.

Table 6. Multivariable linear regression for mean arterial pressure at T1

Predictor	β	SE	95% CI	p
Device (VL vs. DL)	-12.35	2.54	-17.52 to -7.18	< 0.001
Baseline MAP, per mmHg	+0.38	0.16	+0.06 to +0.70	0.021
Age, per year	+0.12	0.16	-0.21 to +0.45	0.468
BMI, per kg/m ²	-0.25	0.44	-1.15 to +0.65	0.576

Notes: Exploratory model, $n = 40$: $R^2 = 0.465$, adjusted $R^2 = 0.404$, $F(4, 35) = 7.61$, $p < 0.001$; Durbin-Watson 1.94; variance inflation factors < 1.3. β , regression coefficient (mmHg); SE, standard error; MAP, mean arterial pressure; BMI, body mass index; VL, video laryngoscopy; DL, direct laryngoscopy.

4. Discussion

4.1. Principal findings

In this randomized trial of 40 low-risk adults with a standardized induction, GlideScope VL was associated with a lower heart rate, MAP, and RPP 1 and 2 minutes after intubation than Macintosh DL.^{27,28} The differences were large for HR ($d > 1.1$), MAP ($d = 1.38$ at T1), and RPP, and they had disappeared by 5 minutes. SBP and first-pass success did not differ significantly. The groups differed because heart rate rose in DL and fell in VL, and the mean rise in DL was modest (6.9 bpm, to 90.15 bpm). Because no measurement was taken between induction and laryngoscopy, the T0–T1 change combines the effects of the induction drugs and of the device.^{29,30} The fall in VL heart rate below baseline fits an induction effect that the device did not offset.

4.2. Possible mechanisms

A biomechanical explanation is plausible but was not tested here (Figure 5).³¹ DL needs line-of-sight alignment and a lifting force on the vallecula and hyoepiglottic ligament, reported at 35 to 45 N.^{32–34} Afferents from this area travel in cranial nerves IX and X to the nucleus tractus solitarius and the rostral ventrolateral medulla, where they drive sympathoadrenal discharge.^{35–38} VL gives an indirect view and is reported to need less force, about 5 to 15 N, which would reduce afferent traffic.^{39–42} Our results fit this hypothesis.^{43–45} We did not, however, measure blade force, neural activity, catecholamines, or vascular resistance, and the cellular and receptor-level signaling that would follow, as well as any effect on cerebral autoregulation, are inferences from the literature, not findings of this trial.

Integrated Neuro-Biomechanical Reflex Arc & Hemodynamic Attenuation Architecture

Comparative Pathophysiological Cascades Between Direct Macintosh and Hyperangulated Video Laryngoscopy

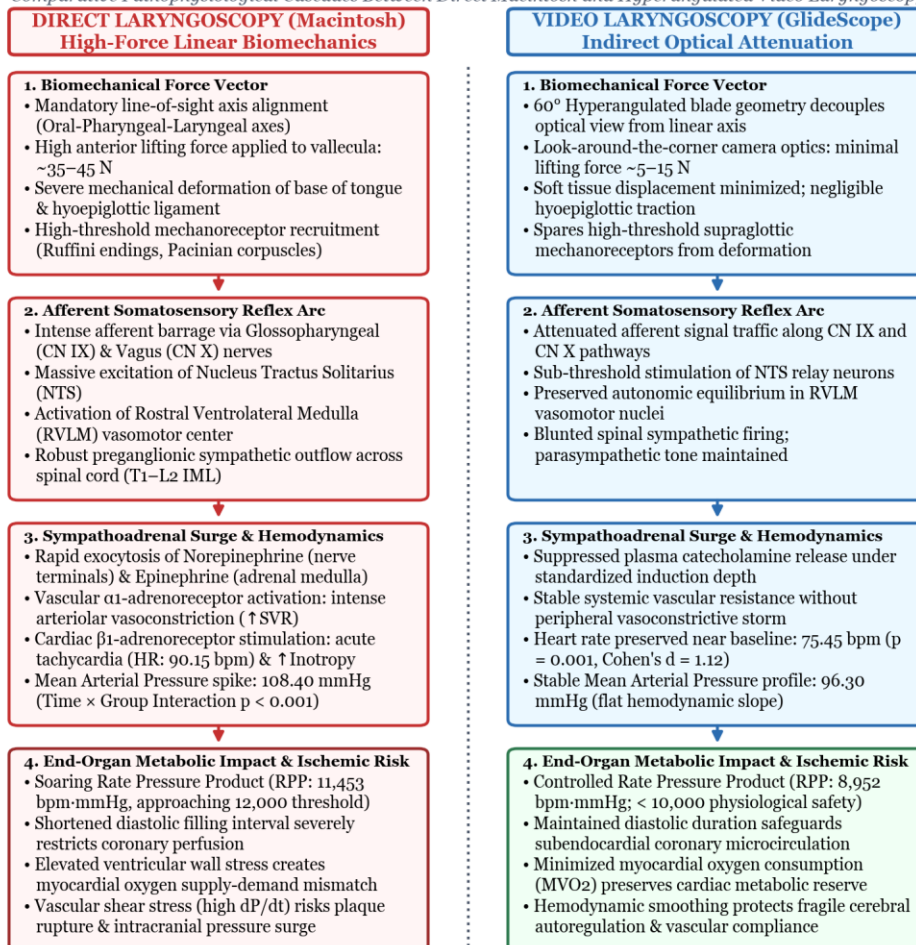


Figure 5. Hypothesized reflex arc and mechanism by which laryngoscope type may influence the hemodynamic response. The pathway is taken from the literature; neural activity, catecholamines, vascular resistance, and blade force were not measured in this trial. HR, MAP, and RPP are group means at T1.

The RPP is a surrogate for myocardial oxygen consumption, and left ventricular perfusion is mainly diastolic, so a lower rate and systolic load may improve the supply–demand balance in patients with fixed coronary stenosis.^{3,4,14,16} Our patients were ASA I–II, normotensive, and free of known coronary disease, and no ischemic outcome was recorded. Cardioprotection and benefit for patients with intracranial or vascular disease therefore remain hypotheses.^{15,17,18}

4.3. Comparison with other studies and implications

Large trials in critically ill adults report that VL improves first-attempt success, but these studies did not examine cardiovascular stress in elective patients.^{18,19} Earlier small studies of the hemodynamic response in elective surgery gave mixed results.^{8,20} Standardizing the depth of induction and the interval before laryngoscopy, as we did, may reduce the variation that obscures a device effect.^{9,21}

Time to intubation was 4.2 seconds longer with VL, and the pressor response did not appear to depend on intubation time within the range observed (about 20 to 40 seconds).^{22–24} VL gave a better view (grade I in all patients) and a numerically higher first-pass rate, but the first-pass difference was not significant and the study was not powered for it.²⁵ The findings suggest that VL may reduce the hemodynamic response to intubation in low-risk patients; they do not by themselves justify routine first-line use, particularly given the cost and slower tube passage of VL.^{26–28}

Trials in patients with cardiovascular or intracranial risk are needed before a change in practice is advised.²⁹

4.4. Strengths and limitations

Strengths are the randomized design, a uniform induction with a fixed interval, experienced operators, automated data capture, and a blinded assessor and data exporter.^{30,31} The limitations are as follows. First, the trial was single-center with 40 patients and one measurement per time point; it was powered for heart rate only.³² Second, there was no post-induction, pre-laryngoscopy measurement, and sevoflurane was given by mask. Third, blood pressure was measured intermittently by oscillometry, which may miss the peak response; beat-to-beat monitoring would give a continuous record.³³ Fourth, the operator could not be blinded, and the RPP thresholds used are not validated for this setting. Fifth, only ASA I–II patients with normal airways were studied.³⁴ Sixth, the regression was exploratory.

5. Conclusion

In this randomized trial of young ASA I–II patients, GlideScope video laryngoscopy was associated with a lower heart rate, mean arterial pressure, and rate pressure product 1 and 2 minutes after intubation than Macintosh direct laryngoscopy under a standardized induction. Systolic pressure and first-pass success did not differ significantly, and cardioprotection was not measured. Larger

multicenter trials that include a post-induction measurement and high-risk patients are needed before video laryngoscopy is recommended for hemodynamic protection.

6. Declarations

Ethical approval and consent to participate. The protocol was approved by the Institutional Health Research Ethics Committee, CMHC Research Center, Indonesia (Reference Number2024/0912). Written informed consent was obtained from all participants. The study followed the Declaration of Helsinki and ICH Good Clinical Practice guidelines.

Consent for publication. Not applicable; no individually identifiable data are presented.

Availability of data and materials. The de-identified datasets generated and analyzed during this study are available from the corresponding author on reasonable request.

Artificial intelligence (AI) use statement. The authors declare that no generative artificial intelligence tool was used in the preparation of this manuscript. The authors reviewed and edited all content and take full responsibility for its integrity and accuracy.

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7. References

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