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ORIGINAL ARTICLE

Hemodynamic effects of topical lidocaine on the laryngoscope blade and trachea during endotracheal intubation: a prospective, double-blind, randomized study

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Abstract
Purpose Minimizing hemodynamic changes during the peri-intubation period is a concern for anesthesiologists. We investigated the effect of lidocaine sprayed on the laryngoscope blade and trachea on hemodynamics during direct laryngoscopic intubation.
Methods Seventy-two patients were randomly allocated to one of four groups: 10 % lidocaine was sprayed either on the laryngoscope blade (group L), on the trachea (group V), or on the laryngoscope blade and the trachea (group LV). No lidocaine was used in group C. Anesthesia was induced in all patients with remifentanil (effect site concentration: 4.0 ng/ml) and propofol (effect site concentration 4.0 μg/ml) continuous infusion using a target control infusion (TCI) device. Mean arterial pressure (MAP) and heart rate (HR) were recorded during the peri-intubation period.
Results Changes in MAP and HR over time were markedly different among the four groups ($P < 0.05$). MAP at 1 min post-intubation was significantly lower in groups L, V, and LV than in group C (86.1 ± 12.7, 85.3 ± 12.6, and 83.7 ± 13.1 vs 106.3 ± 22.9 mmHg, $P < 0.01$). Maximum MAP values were lower in groups L and LV than in group C ($P < 0.05$). HRs at 1, 2, and 3 min post-intubation were lower in group LV than in group C (70.4 ± 9.0 vs 84.2 ± 15.3; 64.0 ± 8.1 vs 79.2 ± 15.4; 61.6 ± 8.3 vs 77.2 ± 14.5 beats/min, $P < 0.01$, respectively).
Conclusions Lidocaine sprayed on the laryngoscope blade and/or trachea reduced the hemodynamic response to laryngoscopic intubation during the post-intubation period following anesthetic induction with remifentanil and propofol using a TCI device.

Keywords Hemodynamic response · Intubation · Laryngoscopy · Lidocaine · Trachea

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Introduction

The pressor response to tracheal intubation results in tachycardia, dysrhythmia, and hypertension [1]. The hemodynamic changes associated with laryngoscopic intubation may be harmful in patients with cerebrovascular disease or traumatic brain injury because they can induce cerebral aneurismal re-rupture or secondary brain insult such as increased intracerebral hemorrhage and worsening cerebral edema [2–5]. In addition, increased sympathetic activation may cause myocardial ischemia in patients with pre-existing coronary disease [6]. The hemodynamic response to laryngoscopic intubation is thought to be due to the upward and forward elevation of the laryngoscope [7] as well as tracheal mucosa stimulation by direct contact with the endotracheal tube [8].

Most studies that have focused on preventing tracheal mucosa stimulation have demonstrated that topical lidocaine spray on the trachea effectively reduces the hemodynamic response to laryngoscopy and endotracheal intubation [8–11]. However, in clinical practice, blind orolaryngeal application of lidocaine without direct laryngoscopic guidance may not ensure adequate anesthesia, although one report showed that administering oropharyngeal lidocaine provides good topical anesthesia of the larynx and trachea [12]. The use of direct laryngoscopy itself can affect the

hemodynamic response, particularly during the forward and upward movement of the laryngoscope blade [7]. We hypothesized that topical lidocaine applied to the tip and inner edge of the curved blade of a direct laryngoscope would provide good regional anesthesia on the contacted mucosa of the vallecula.

In this study, we determined whether hemodynamic changes during direct laryngoscopic intubation could be effectively mitigated by applying lidocaine to a direct laryngoscope and the tracheal mucosa.

Materials and methods

This prospective, double blinded, randomized study was approved by the Institutional Review Board at Seoul National University Hospital and informed consent was obtained from all patients. This study is registered at Clinicaltrials.gov (NCT01737437). Seventy-two patients scheduled for elective neurosurgery under general anesthesia at Seoul National University Hospital from August 2012 to November 2012 were enrolled. Patients aged 20–60 years with an American Society of Anesthesiologists (ASA) physical status classification of I or II were recruited. Those with ASA physical status classification III or IV, uncontrolled hypertension, diabetes with autonomic neuropathy, anticipated difficult airway, severe coronary disease, or patients who were receiving medication that affects blood pressure and heart rate were excluded.

Patients did not receive any premedication and had fasted for at least 8 h. After entering the operating room, all patients were monitored by standard non-invasive methods, including a three-lead electrocardiogram, pulse oximetry, non-invasive arterial blood pressure, and bispectral index (BIS). The BIS value, mean arterial pressure (MAP), and baseline heart rate (HR) were recorded at that time. After standard monitoring, the patients were induced with remifentanil (effect site concentration: 4.0 ng/ml) and propofol (effect site concentration 4.0 μg/ml) continuous infusion using a target-controlled infusion (TCI) device with preoxygenation of 100 % oxygen via a facial mask. Rocuronium (0.6 mg/kg) was used to facilitate endotracheal intubation. A radial artery catheter was used for direct continuous arterial pressure monitoring after the rocuronium injection.

Patients were randomly divided into four groups ($n = 18$ each) based on whether topical lidocaine was applied to the tracheal mucosa, the laryngoscope, both, or none. Randomization was accomplished using computer-generated random numbers. The assignments were concealed in opaque envelopes and opened immediately before induction of anesthesia by a nurse who was blinded to this study and was in charge of preparing the study drugs. Normal saline (0.9 %) was applied to the trachea and laryngoscope blade in group C. In group L, 10 % lidocaine (Xylocaine spray, AstraZeneca Korea, Seoul, Korea) was applied to the laryngoscope blade and 0.9 % normal saline was applied to the trachea. In group V, 0.9 % normal saline was applied to the laryngoscope blade and 10 % lidocaine was applied to the trachea. In group LV, 10 % lidocaine was applied to both the laryngoscope blade and trachea. Lidocaine (10 %) or normal saline (0.9 %) was applied four times using the same pump-metered spray device. The lidocaine spray device was designed to deliver 10-mg lidocaine in each puff. Two lidocaine spray bottles wrapped in white paper, one of which contained normal saline, were used to blind patients and the intubator.

Patients were placed in the supine position with a 6-cm headrest [13]. Topical lidocaine or normal saline was directly sprayed on the tracheal mucosa under laryngoscopic guidance 2 min after the rocuronium injection (no. 3 Macintosh laryngoscope blade for females, no. 4 for males), if necessary, with external laryngeal manipulation. The laryngoscope was kept in situ without upward and forward movement for 1 min, after which endotracheal intubation was performed in the sniffing position by one anesthesiologist, who was blinded to the study and had performed more than 1,000 laryngoscopic intubations, with minimal lifting force required for a good glottic view. Cormack and Lehane grade was noted by the anesthesiologist [14]. The use of a stylet and external laryngeal manipulation was also noted. Intubation time was defined as the time to endotracheal intubation from a forward and upward movement of the direct laryngoscope blade, not from laryngoscope insertion. After confirming correct endotracheal tube placement by capnography, ventilation was controlled, and end-tidal carbon dioxide was maintained at 30–35 mmHg. Anesthesia was maintained with continuous infusion of remifentanil and propofol at the same concentration as induction until 3 min post-intubation.

An independent investigator recorded the BIS and the hemodynamic parameters such as MAP and HR at baseline, after induction, during upward and forward movement of the direct laryngoscope and 1 min later, during intubation, and every minute for 3 min after endotracheal intubation.

The primary outcome measurements were the MAP and HR. The secondary outcome measurements were the maximum MAP and HR during the peri-intubation period.

A previous study showed that the mean MAP 1 min after intubation is 80 mmHg and its standard deviation is 19 mmHg when propofol (effect site concentration 4.0 μg/ml) and remifentanil (effect site concentration 4.0 ng/ml) continuous infusion using a TCI pump is used to induce anesthesia [1]. Sample size was calculated to have a power of 80 at a 5 % significance level (two-tailed) to detect a MAP difference of 19 mmHg 1 min after intubation between groups C and LV.

The statistical analysis was performed using SPSS ver. 20.0 (SPSS Inc., Chicago, IL, USA). Parametric and non-parametric data were compared using a cross-table with a chi-square test and analysis of variance (ANOVA), respectively. The hemodynamic data were compared using repeated-measures ANOVA for time by treatment effect, followed by ANOVA with a Bonferroni test to compare hemodynamic data at each time point. A P value <0.05 was considered significant for all tests.

Results

A total of 72 patients were enrolled in this study. No significant differences were found among the four groups with respect to demographic and intubation data (Table 1). We did not use a stylet for endotracheal intubation in any patient. External laryngeal manipulation was used in two, six, four, and two patients in groups C, L, V, and LV, respectively.

Changes in MAP over time among the four groups were markedly different (Table 2; Fig. 1, $P < 0.05$). Baseline MAP was 101.0 ± 10.7, 102.2 ± 14.9, 99.5 ± 15.2, and 99.1 ± 15.4 mmHg in groups C, L, V, and LV, respectively. MAP decreased in all groups from the baseline value immediately after anesthetic induction with remifentanil and propofol. No significant differences in MAP were observed before intubation among the four groups except MBP at 1-min post-laryngoscopy between group C and group L (91.6 ± 15.9 vs 80.5 ± 16.4 mmHg, $P < 0.05$). The MAP in group C was significantly higher than in groups L, V, and LV at 1 min post-intubation (106.3 ± 22.9 vs 86.1 ± 12.7, 85.3 ± 12.6, and 83.7 ± 13.1 mmHg, $P < 0.01$), and higher than in group L and LV at 2 min post-intubation (95.1 ± 18.4 vs 77.5 ± 10.7 and 80.0 ± 12.4 mmHg, $P < 0.01$), and higher than in group LV at 3 min post-intubation (88.1 ± 17.7 vs 75.2 ± 13.4 mmHg, $P < 0.05$).

Changes in HR over time among the four groups were also different (Table 2; Fig. 2, $P < 0.05$). Baseline HR was 75.8 ± 16.8, 78.8 ± 13.9, 78.2 ± 15.6, and 68.9 ± 12.5 beats/min in groups C, L, V, and LV, respectively. No significant differences in HR were observed prior to intubation among the four groups. HR was markedly lower in group LV than in group C at 1 min post-intubation (70.4 ± 9.0 vs 84.2 ± 15.3 beats/min, $P < 0.01$), lower than in groups C and V at 2 min post-intubation (64.0 ± 8.1 vs 79.2 ± 15.4 beats/min, $P < 0.01$; vs 64.0 ± 8.1 vs 76.4 ± 12.6 beats/min, $P < 0.05$), and lower than in groups C, L, and LV at 3 min post-intubation (61.6 ± 8.3 vs 77.2 ± 14.5 beats/min, $P < 0.01$; 61.6 ± 8.3 vs 72.7 ± 11.8 and 72.4 ± 11.3 beats/min, $P < 0.05$).

Maximum MAP during the peri-intubation period was significantly higher in group C than in groups LV and L (108.7 ± 22.1 vs 89.9 ± 14.0 and 91.3 ± 14.6, $P < 0.05$), but no significant difference was observed between groups L or V and LV (Table 3). Maximum HR during the peri-intubation period was higher in group C than in group LV (88.1 ± 13.2 vs 73.9 ± 9.1 beats/min, $P < 0.01$). The number of patients with increased MBP >30 % of baseline value was higher in group C compared with the other groups ($P < 0.05$). The number of patients with decreased MBP >30 % of the baseline value was comparable among the four groups.

Baseline BIS values were 93.2 ± 4.2, 91.7 ± 7.0, 95.6 ± 2.5, and 94.0 ± 5.0 in groups C, L, V, and LV, respectively. After anesthetic induction, the BIS value significantly decreased to 55.6 ± 10.7 for group C, 52.1 ± 11.1 for group L, 51.6 ± 7.9 for group V, 51.4 ± 12.2 for group LV ($P < 0.01$). After that time, the BIS value was maintained in the range of 30–70 in all groups. Changes in the BIS value over time among the four groups were not different.

Table 1 Demographic and intubation data

	Group C ($n = 18$)	Group L ($n = 18$)	Group V ($n = 18$)	Group LV ($n = 18$)
Age (years)	40.1 ± 13.5	46.7 ± 12.7	42.6 ± 11.4	43.3 ± 11.4
Gender (M/F)	10/8	7/11	7/11	7/11
Height (cm)	164.4 ± 9.3	158.7 ± 10.0	163.9 ± 9.5	164.9 ± 6.1
Weight (kg)	66.5 ± 13.9	60.5 ± 12.4	63.9 ± 12.1	64.1 ± 9.1
BMI (kg/m^2)	24.4 ± 3.9	24.0 ± 3.8	23.7 ± 3.6	23.5 ± 3.5
Intubation time[a] (s)	9.1 ± 2.3	10.4 ± 3.2	9.5 ± 2.5	9.2 ± 2.5
Cormack and Lehane grade (1:2:3)	14:4:0	14:3:1	15:2:1	14:3:1
Cricoid compression (yes/no)	2:16	6:12	4:14	2:16

Data are presented as mean ± SD or number

Group C, control; Group L, 10 % lidocaine spray is applied to the laryngoscope blade; Group V, 10 % lidocaine spray is applied to the trachea; Group LV, 10 % lidocaine spray is applied to the laryngoscope blade and trachea

[a] The time required for direct laryngoscope insertion is excluded because the tip of direct laryngoscope is already placed at the vallecula

Table 2 Changes of bispectral index and hemodynamic parameters during peri-intubation period

	Group	Baseline	After anesthetic induction	During laryngoscopy for IT spray	1 min after laryngoscopy	During intubation	1 min after intubation	2 min after intubation	3 min after intubation
MAP (mmHg)	Group C	101.0 ± 10.7	77.9 ± 12.5*	76.8 ± 13.6*	91.6 ± 15.9	96.2 ± 19.7	106.3 ± 22.9*	95.1 ± 18.4	88.1 ± 17.7
	Group L	102.2 ± 14.9	71.1 ± 11.5*	70.6 ± 11.2*	80.5 ± 16.4‡	81.4 ± 15.2	86.1 ± 12.7†, §	77.5 ± 10.7§	77.0 ± 12.5
	Group V	99.5 ± 15.2	73.8 ± 13.8*	74.1 ± 9.2*	93.5 ± 13.0*	90.0 ± 11.6*	85.3 ± 12.6§	83.3 ± 10.9	80.5 ± 10.6†
	Group LV	99.1 ± 15.4	70.5 ± 13.0*	73.0 ± 14.0*	86.7 ± 14.5†	86.6 ± 14.2†	83.7 ± 13.1§	80.0 ± 12.4§	75.2 ± 13.4*, ‡
HR (beats/min)	Group C	75.8 ± 16.8	63.9 ± 11.5*	65.8 ± 9.5*	69.4 ± 13.1†	75.7 ± 14.3	84.2 ± 15.3*	79.2 ± 15.4†	77.2 ± 14.5
	Group L	78.8 ± 13.9	68.0 ± 10.7*	67.0 ± 11.4*	68.4 ± 12.4*	68.6 ± 13.5*	79.5 ± 12.4*	72.9 ± 11.4	72.7 ± 11.8
	Group V	78.2 ± 15.6	66.9 ± 12.7*	65.9 ± 12.7*	76.9 ± 12.8*	76.6 ± 13.7	80.6 ± 12.5*	76.4 ± 12.6	72.4 ± 11.3
	Group LV	68.9 ± 12.5	58.6 ± 8.6*	58.7 ± 8.4*	71.3 ± 10.7*	70.1 ± 10.8*	70.4 ± 9.0*,§	64.0 ± 8.1§, $	61.6 ± 8.3*, §, $, ¶

Data are presented as mean ± SD

Group C, control; Group L, 10 % lidocaine spray is applied to the laryngoscope blade; Group V, 10 % lidocaine spray is applied to the trachea; Group LV, 10 % lidocaine spray is applied to the laryngoscope blade and trachea

IT intratracheal

* $P < 0.01$ compared with baseline within groups

† $P < 0.05$ compared with baseline within groups

‡ $P < 0.05$ compared with Group C

§ $P < 0.01$ compared with Group C

$ $P < 0.05$ compared with Group V

¶ $P < 0.05$ compared with Group L

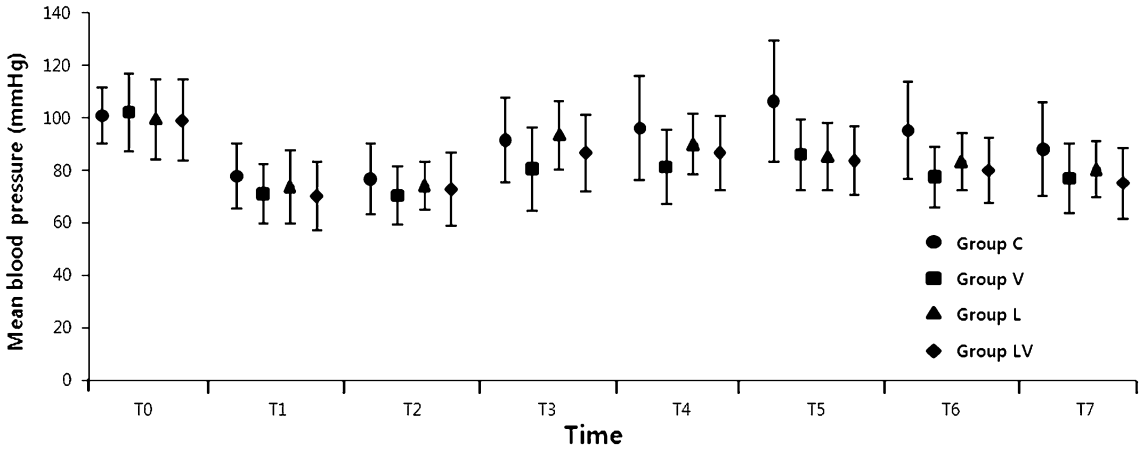

Fig. 1 This graph shows changes in mean arterial pressure (mean and standard deviation) over time. *T0* baseline, *T1* after anesthetic induction, *T2* during laryngoscopy for intratracheal spray, *T3* 1 min after laryngoscopy, *T4* during intubation, *T5* 1 min after intubation, *T6* 2 min after intubation, *T7* 3 min after intubation

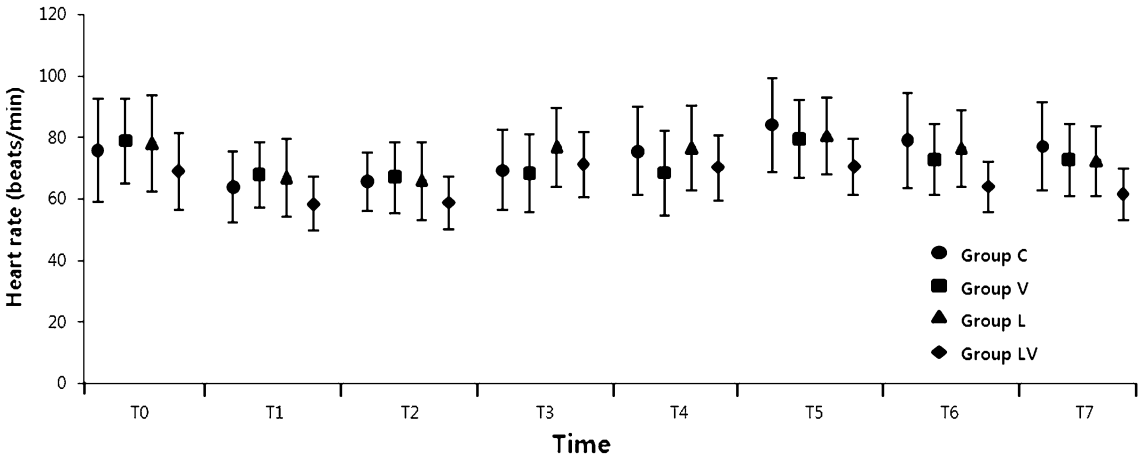

Fig. 2 This graph shows changes in heart rate (mean and standard deviation) over time. *T0* baseline, *T1* after anesthetic induction, *T2* during laryngoscopy for intratracheal spray, *T3* 1 min after laryngoscopy, *T4* during intubation, *T5* 1 min after intubation, *T6* 2 min after intubation, *T7* 3 min after intubation

No patient demonstrated laryngeal spasm, coughing, movement, or arrhythmia while under anesthesia.

Discussion

Our data show that topical application of lidocaine to the laryngoscope blade, trachea, or both sites attenuated the cardiovascular response to laryngoscopic intubation during the peri-intubation period.

Endotracheal intubation using direct laryngoscopy requires elevation of the epiglottis and exposure of the glottis opening, which is obtained by a forward and upward movement of the laryngoscope blade. Such laryngoscopic manipulation can result in circulatory responses such as hypertension and tachycardia. Shribman et al. [7] reported that laryngoscopy alone generates the same pressor and sympathoadrenal responses as laryngoscopy followed by intubation. We expected that topical lidocaine applied to the laryngoscope would provide regional anesthesia of the contacted mucosa of the vallecula, thereby attenuating the hemodynamic effects of laryngoscopic intubation. In our study, the laryngoscope blade was sprayed with lidocaine and was kept in the vallecula without upward and forward movement for 1 min. This procedure effectively attenuated the hemodynamic response during the early post-intubation period by obtunding sensory input from mechanical stimulation generated by the forward and upward movement of the laryngoscope. Spraying the laryngoscope blade with lidocaine is an easy, simple, and effective adjuvant method to reduce hemodynamic changes during the peri-intubation period. A disadvantage of this method is that it takes time

Table 3 Maximum and minimum values of hemodynamic parameters during peri-intubation period

	Group C ($n = 18$)	Group L ($n = 18$)	Group V ($n = 18$)	Group LV ($n = 18$)
MAP (mmHg)				
Pre-induction	101.0 ± 10.7	102.2 ± 14.9	99.5 ± 15.2	99.1 ± 15.4
Maximum	108.7 ± 22.1	91.3 ± 14.6*	96.2 ± 12.1	89.9 ± 14.0*
Minimum	73.8 ± 12.7	67.7 ± 10.0	69.5 ± 9.8	67.0 ± 11.3
Increased MAP >30 % of pre-induction value (*n*)	5	0*	0*	0*
Decreased MAP >30 % of pre-induction value (*n*)	6	11	8	9
HR (beats/min)				
Pre-induction	75.8 ± 16.8	78.8 ± 13.9	78.2 ± 15.6	68.9 ± 12.5
Maximum	88.1 ± 13.2	80.6 ± 12.5	83.2 ± 12.0	73.9 ± 9.1†
Minimum	61.9 ± 9.6	64.8 ± 11.1	64.3 ± 13.1	57.4 ± 8.8
Increased HR >30 % of pre-induction value (*n*)	4	2	2	3
Decreased HR >30 % of pre-induction value (*n*)	2	4	2	2

Data are presented as mean ± SD

Group C, control; Group L, 10 % lidocaine spray is applied to the laryngoscope blade; Group V, 10 % lidocaine spray is applied to the trachea; Group LV, 10 % lidocaine spray is applied to the laryngoscope blade and trachea

MAP mean arterial pressure, *HR* heart rate

* $P < 0.05$ compared with Group C

† $P < 0.01$ compared with Group C

before the beneficial effect is exerted on the pressor response to laryngoscopic elevation. Moreover, because manual ventilation is not allowed for 1 min (contact time between the topical lidocaine sprayed on the laryngoscope blade and the mucosa of the vallecula), this method may be not suitable for some clinical situations, especially in morbidly obese patients, those with severe chronic obstructive pulmonary disease, parturient females with a low functional residual capacity and decreased oxygen reserves, and patients at risk of aspiration.

The effect of intratracheal lidocaine spray on the hemodynamic changes related to endotracheal intubation has been controversial. Some studies have reported that a simple tracheal spray with lidocaine effectively attenuates the hypertensive response to endotracheal intubation [8–11, 15]. Other studies have shown that intratracheal lidocaine administration was not beneficial [16–18]. In this study, intratracheal lidocaine spray attenuated the hemodynamic response to endotracheal intubation, confirming that stimulation of the tracheal mucosa with an endotracheal tube contributes to hemodynamic alterations. Our results also showed that there were no significant differences in hemodynamic parameters depending on lidocaine spray on the laryngoscope blade or intratracheal lidocaine spray, suggesting that the tracheal mucosa stimulation contributes to hemodynamic alterations as much as laryngoscopic elevation. Although topical anesthesia on the trachea is an effective method to reduce hemodynamic changes related to endotracheal intubation, the method cannot be used in patients with a difficult airway in whom intratracheal lidocaine spray under laryngoscopic guidance is impossible.

The timing of endotracheal intubation after topical anesthesia of the trachea is important to reduce hemodynamic changes related to intubation. Previous studies have shown that endotracheal intubation should be performed at least 2 min after applying tracheal lidocaine to attenuate the cardiovascular response [8, 11]. In other words, contact time of at least 2 min between the topical anesthetic and the tracheal mucosa is required to provide adequate penetration of lidocaine into the tracheal mucosa for maximum effect. In our study, tracheal intubation was performed 1 min after intratracheal lidocaine spray. We expected that lidocaine applied to the tracheal mucosa would begin to produce topical anesthesia in about 1 min and the extent of the hypertensive response to endotracheal intubation due to such a short contact time can be partially compensated for by a sufficient anesthetic depth. Indeed, our results showed that although the contact time was 1 min, the cardiovascular response to endotracheal intubation was effectively reduced.

The dosage of induction agents can also affect the hemodynamic response to tracheal intubation. The effect-site remifentanil concentrations of 4.0 ng/ml used in the current study may be close to the effect-site concentration of remifentanil that reduces the sympathetic response to endotracheal intubation in 50 % of cases (EC_{50}). A previous report using muscle relaxants demonstrated that an effect-site remifentanil concentration of 4.0 ng/ml in combination with an effect-site propofol concentration of

4.0 μg/ml markedly attenuates the hemodynamic response to endotracheal intubation compared to using propofol without remifentanil [19]. Another report showed that a bolus injection of 1 μg/kg remifentanil, which corresponds to a blood concentration of 4.0 ng/ml, effectively reduces the pressor response to laryngoscopy and tracheal intubation during rapid sequence induction with co-administration of 5 mg/kg thiopental [20]. In cases that do not use muscle relaxants, the EC_{50} of remifentanil for blunting the cardiovascular response to tracheal intubation is 4.4–5.4 ng/ml, although the effect-site propofol concentration varied (3.0–5.4 μg/ml) [21–24]. In this study, remifentanil continuous infusion itself blunted the hemodynamic response to endotracheal intubation. Moreover, spraying the trachea and laryngoscope blade with lidocaine in combination with remifentanil continuous infusion more significantly attenuated the hemodynamic response to endotracheal intubation than did remifentanil continuous infusion alone. Thirty percent of patients in the control group and about 50 % of patients in the three other groups temporarily became hypotensive (a decrease in MAP >30 % of pre-induction value) during the peri-intubation period. Therefore, on application of our study protocol to attenuate the hemodynamic response to laryngoscopy and endotracheal intubation, caution should be used in patients at risk of tissue ischemia.

This study had some limitations. Various laryngoscope-lifting forces can result in different hemodynamic responses. Different induction agents, dosages, and techniques can have markedly different effects on cardiovascular responses to laryngoscopy and endotracheal intubation. Finally, the mean age of subjects in the current study was about 40 years, but their baseline MAP was more than 90 mmHg. We have observed that the initial MBP can be different from a patient's true MBP due to increased anxiety in the operating room; the baseline values of MAP and HR are more precise when patients are allowed to relax for 10–15 min after entering the operating room.

In conclusion, applying topical lidocaine to the trachea and/or the laryngoscope blade effectively attenuated the pressor response to endotracheal intubation during the post-intubation period in patients who were induced with remifentanil and propofol continuous infusion using a TCI device. However, since spraying the laryngoscope blade with lidocaine may not be applicable in some clinical situations, we recommend the use of intratracheal lidocaine spray.

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