General Anesthesia

Tracheal lidocaine attenuates the cardiovascular response to endotracheal intubation

[L'administration trachéale de lidocaïne diminue la réponse cardio-vasculaire à l'intubation endotrachéale]

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Purpose: In order to examine the efficacy of tracheal lidocaine (TL) for attenuation of the cardiovascular responses to endotracheal intubation (EI), we compared the cardiovascular responses to TL alone and EI with TL, with those to EI without TL.

Methods: Seventy-five patients (ASA I-II) were studied. Anesthesia was induced with fentanyl $2 \mu\text{g}\cdot\text{kg}^{-1}$ iv, thiamylal $5 \text{ mg}\cdot\text{kg}^{-1}$ iv and sevoflurane 1.0% in oxygen. Vecuronium $0.12 \text{ mg}\cdot\text{kg}^{-1}$ was used to facilitate EI. In Group A ($n=25$), three minutes after induction, EI was performed. In Group B ($n=25$), three minutes after induction, the patients received TL (4% lidocaine, 4 mL). This was followed by immediate EI. In Group C ($n=25$), EI was performed two minutes after TL. Heart rate, arterial blood pressure and rate-pressure product (RPP) were measured from one minute before induction until five minutes after EI.

Results: The changes of RPP caused by TL alone in Group C (TL; $+34.6 \pm 29.0\%$, mean $\pm$ SD) were significantly ($P < 0.01$) less than those caused by EI without TL in Group A ($+77.3 \pm 42.6\%$). EI after TL in Group C did not cause significant changes in RPP ($+5.4 \pm 15.2\%$). There were no significant differences between Groups A and B ($+58.3 \pm 36.6\%$).

Conclusion: We conclude that the cardiovascular responses to TL alone are half as great as those to EI without TL, and that TL is effective for attenuation of the cardiovascular responses to EI. EI should be performed more than two minutes after TL.

Objectif : Évaluer l'efficacité de l'administration trachéale de lidocaïne (LT) utilisée pour atténuer les réactions cardio-vasculaires à l'intubation endotrachéale (IE) en comparant les réponses cardio-vasculaires à la LT seule et à l'IE avec LT, avec les réponses à l'IE sans LT.

Méthode : L'étude a porté sur 75 patients d'état physique ASA I-II. L'anesthésie a été induite avec $2 \mu\text{g}\cdot\text{kg}^{-1}$ de fentanyl iv, $5 \text{ mg}\cdot\text{kg}^{-1}$ de thiamylal iv et du sévoflurane à 1,0 % dans de l'oxygène. On a utilisé $0,12 \text{ mg}\cdot\text{kg}^{-1}$ de vécuronium pour faciliter l'IE. Dans le groupe A ($n = 25$), on a réalisé l'IE trois minutes après l'induction. Dans le groupe B ($n = 25$), les patients ont reçu la LT (4 mL à 4 %) trois minutes après l'induction. Ce qui a été immédiatement suivi de l'IE. Dans le groupe C ($n = 25$), l'IE a été faite deux minutes après la LT. La fréquence cardiaque, la tension artérielle et le produit de la tension systolique par la fréquence des contractions cardiaques (PTF) ont été mesurés à partir d'une minute avant l'induction jusqu'à cinq minutes après l'IE.

Résultats : Les changements de PTF causés par la LT seule dans le groupe C (LT; $+34,6 \pm 29,0 \%$, moyenne $\pm$ écart type) ont été significativement ($P < 0,01$) moindres que ceux qui ont été causés par l'IE sans LT dans le groupe A ($+77,3 \pm 42,6 \%$). L'IE qui a suivi la LT dans le groupe C n'a pas modifié significativement le PTF ($+5,4 \pm 15,2 \%$). Les différences n'étaient pas significatives entre les groupes A et B ($+58,3 \pm 36,6 \%$).

Conclusion : Les réponses cardio-vasculaires à la LT seule sont moitié moins importantes que les réponses à l'IE sans LT et la LT est efficace pour diminuer les réponses cardio-vasculaires à l'IE. L'IE doit toutefois être faite plus de deux minutes après l'injection de LT.

LARYNGOSCOPY and endotracheal intubation provoke cardiovascular responses that include hypertension, tachycardia and dysrhythmias. These hemodynamic changes may be associated with myocardial ischemia, myocardial failure and cerebral hemorrhage in critically ill

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Accepted for publication February 6, 2001.

Revision accepted March 28, 2001.

patients.^{1,2} A number of drugs, including calcium channel blocker,³⁻⁵ beta-adrenergic blockers,^{6,7} *iv* lidocaine⁸ and narcotics⁶ have been used in an attempt to prevent the cardiovascular responses to endotracheal intubation. Intratracheal administration of lidocaine (tracheal lidocaine) is also widely used for the attenuation of those cardiovascular responses. This method may avoid unexpected or excessive hypotension that can be associated with antihypertensive drug use. The efficacy of tracheal lidocaine for attenuation of the cardiovascular responses to tracheal intubation has not been fully established and some authors have questioned its efficacy.⁸⁻¹⁰ It is unknown whether the magnitude of the cardiovascular responses to tracheal lidocaine alone is less than that of endotracheal intubation without topical anesthesia. If the magnitude of the cardiovascular responses to tracheal lidocaine alone is as great as that produced by endotracheal intubation, it would have limited usefulness as an attenuating drug.

In this study, in order to elucidate the effectiveness of tracheal lidocaine in blunting the cardiovascular responses, we compared the cardiovascular responses to endotracheal lidocaine and tracheal intubation with tracheal lidocaine in ASA class I or II patients without cardiovascular disease.

Methods

After obtaining institutional approval and informed consent, 75 patients without cardiovascular disease (ASA physical status I-II) undergoing elective surgery under general anesthesia were studied. Patients were premedicated with diazepam 5 mg or 10 mg and ranitidine 150 mg *po* was given 90 min before induction of anesthesia. On arrival in the operating room an *iv* infusion of acetate Ringer solution was started. Routine monitoring, including arterial blood pressure (AP), an electrocardiogram (ECG) and oxygen saturation (SpO₂) were introduced. The AP was measured automatically and registered with an automated non-invasive AP monitor (Jentow®, Nippon Colin, Komaki, Japan). Heart rate (HR) was monitored by ECG. Anesthesia was induced with fentanyl 2 µg·kg⁻¹ *iv*, thi-amylal 5 mg·kg⁻¹ *iv* and sevoflurane 1.0% (inspired) in oxygen. Vecuronium 0.12 mg·kg⁻¹ *iv* was used to facilitate endotracheal intubation. In the control group (Group A), three minutes after induction, direct laryngoscopy with a standard Macintosh laryngoscope blade was performed and endotracheal intubation was undertaken. In Group B, three minutes after induction, the trachea was sprayed with 4% lidocaine 4 mL under direct vision with a standard Macintosh laryngoscope through a laryngotracheal anesthesia set (LTA®, Abott Ireland, Rep. of Ireland). This was followed by imme-

TABLE I Demographic data

	Group A (n=25)	Group B (n=25)	Group C (n=25)
Age (yr)	49.3 ± 16.8	49.1 ± 16.8	49.3 ± 15.5
Sex (female/male)	13/12	12/13	13/12
Height (cm)	159.2 ± 6.9	159.9 ± 9.4	158.6 ± 7.4
Weight (kg)	61.7 ± 10.8	57.3 ± 11.8	58.6 ± 12.1

Data are mean ± standard deviation.

diate endotracheal intubation. In Group C, three minutes after induction, the trachea was sprayed with 4% lidocaine 4 mL. The laryngoscope was removed and, two minutes later, laryngoscopic intubation was performed. During anesthesia, ventilation was assisted or controlled and end-tidal carbon dioxide was maintained at 35–40 mmHg. All intubations and tracheal lidocaine administrations were accomplished within 30 sec.

Baseline values of mean AP (MAP) (mmHg), systolic AP (SAP) (mmHg), diastolic AP (DAP) (mmHg) and HR (beats·min⁻¹) were measured one minute before induction of anesthesia.

In Groups A and B, subsequent measurements were taken immediately before endotracheal intubation, one minute after endotracheal intubation, thereafter every one minute until five minutes after endotracheal intubation. In Group C, subsequent measurements were taken immediately before tracheal lidocaine, one minute after tracheal lidocaine, two minutes after tracheal lidocaine (immediately before endotracheal intubation), one minute after endotracheal intubation and thereafter every one minute until five minutes after endotracheal intubation. The rate-pressure product (RPP) was calculated by multiplying SAP by HR.

Data are expressed as mean ± standard deviation (SD). One-way ANOVA for repeated measurement followed by Student's *t* test was used for the intra-group comparisons. The pre-induction values, the maximum and the minimum values of the hemodynamic variables during intubation and the changes in the hemodynamic variables caused by endotracheal intubation and tracheal lidocaine were analyzed with one-way factorial ANOVA with Scheffe's *F* post hoc test. *P* < 0.05 was considered as statistically significant.

Results

The patients' demographic data are shown in Table I. None were taking cardioactive or antihypertensive medication.

HR, MAP and RPP increased significantly (*P* < 0.01) following endotracheal intubation in Groups A and B in comparison with the pre-induction values

FIGURE 1 The hemodynamic changes during intubation period in Groups A and B. Data are mean $\pm$ standard deviation. $\square$ =Group A; $\bullet$ =Group B. HR=heart rate (beats $\cdot$ min $^{-1}$); MAP=mean arterial pressure (mmHg); RPP=rate-pressure product; EI=endotracheal intubation. * P < 0.05; ** P < 0.01 compared with pre-induction values.

FIGURE 2 The hemodynamic changes during intubation period in Group C. Data are mean $\pm$ standard deviation. HR=heart rate (beats $\cdot$ min $^{-1}$); MAP=mean arterial pressure (mmHg); RPP=rate-pressure product; TL=tracheal lidocaine; EI=endotracheal intubation. * P < 0.05; ** P < 0.01 compared with pre-induction values.

TABLE II Maximum and minimum values in hemodynamic variables during intubation period

	Group A	Group B	Group C
HR (beats·min ⁻¹)			
Pre-induction	72 ± 12	72 ± 13	73 ± 13
Maximum	88 ± 14	83 ± 11	77 ± 16*
Minimum	66 ± 12	63 ± 10	63 ± 13
MAP (mmHg)			
Pre-induction	93 ± 19	97 ± 16	93 ± 16
Maximum	117 ± 18	115 ± 19	100 ± 16**#
Minimum	77 ± 13	75 ± 15	73 ± 11
SAP (mmHg)			
Pre-induction	132 ± 21	135 ± 19	129 ± 21
Maximum	157 ± 21	151 ± 19	136 ± 21**
Minimum	109 ± 17	105 ± 16	102 ± 14
DAP (mmHg)			
Pre-induction	74 ± 14	77 ± 14	73 ± 16
Maximum	93 ± 14	91 ± 17	80 ± 15**
Minimum	60 ± 11	59 ± 14	56 ± 11
RPP			
Pre-induction	9576 ± 2410	9800 ± 2723	9294 ± 2101
Maximum	13799 ± 3369	12480 ± 3238	10279 ± 2871**
Minimum	7346 ± 1774	6781 ± 1869	6517 ± 1731

Data are mean ± standard deviation. HR=heart rate; MAP=mean arterial pressure; SAP=systolic arterial pressure; DAP=diastolic arterial pressure; RPP=rate-pressure product. **P* < 0.05; ***P* < 0.01 compared with Group A; #*P* < 0.05 compared with Group B.

TABLE III Changes (%) in hemodynamic variables from pre-EI (or pre-TL) values 1 min after EI (or TL)

	Group A EI	Group B TL + EI	Group C TL	EI
HR	+28.8 ± 21.2	+20.7 ± 15.4	+11.7 ± 12.3**	+0.6 ± 9.1**
MAP	+39.6 ± 18.8	+37.0 ± 20.0	+18.9 ± 19.3**##	+3.1 ± 10.9**
RPP	+77.3 ± 42.6	+58.3 ± 36.6	+34.6 ± 29.0**	+5.4 ± 15.2**

Data are mean ± standard deviation. TL=tracheal lidocaine; EI=endotracheal intubation; HR=heart rate; MAP=mean arterial pressure; RPP=rate-pressure product. ***P* < 0.01 compared with Group A (EI); ##*P* < 0.01 compared with Group B (TL + EI).

(Figure 1). In Group C, there were no significant differences in HR, MAP and RPP one minute after tracheal lidocaine and endotracheal intubation in comparison with the pre-induction values (Figure 2). The pre-induction values and the minimum values of HR, AP and RPP during the intubation period were comparable within the groups. The maximum values of HR, AP and RPP during the intubation period in Group C were significantly less than those in Group A while there were no significant differences between Groups A and B (Table II). The changes in hemodynamic variables from the pre-intubation values (or the pre-tracheal lidocaine values) one minute after intubation (or tracheal lidocaine) in all three groups are shown in Table III. Endotracheal intu-

bation in Groups A and B, and tracheal lidocaine in Group C caused a significant increase in the measured hemodynamic variables in comparison with both the pre-intubation values and the pre-tracheal lidocaine values. However, the changes of hemodynamic variables caused by tracheal lidocaine alone in Group C were significantly less than those caused by endotracheal intubation without tracheal lidocaine in Group A while there were no significant differences between Groups A and B. Endotracheal intubation two minutes after tracheal lidocaine in Group C did not cause significant changes in hemodynamic variables.

Discussion

Several studies have examined the efficacy of tracheal lidocaine for attenuation of the cardiovascular responses to endotracheal intubation. The results of these studies have been contradictory. Denlinger *et al.*¹¹ showed that a simple tracheal spray with lidocaine attenuated the hypertensive responses to endotracheal intubation when compared with saline tracheal spray. Others showed that the application of topical anesthesia to the upper airway and trachea failed to prevent the pressor responses to endotracheal intubation.⁸⁻¹⁰ In the study of Denlinger *et al.*,¹¹ endotracheal intubation was performed more than two minutes after a tracheal spray with local anesthetics while endotracheal intubation was performed less than one minute after topical anesthesia in the studies that indicated the ineffectiveness of tracheal lidocaine.⁸⁻¹⁰ Our study suggests that differing intervals between tracheal lidocaine and endotracheal intubation probably caused the inconsistent conclusions reported in other investigations. Our results show that endotracheal intubation performed two minutes after tracheal lidocaine attenuates the cardiovascular responses to endotracheal intubation.

In the present study, the time point of endotracheal intubation in Group C was two to three minutes later than in Groups A and B. Since sevoflurane 1% (inspired) was started at the induction of anesthesia and continued until five minutes after endotracheal intubation, it is possible that the patients in Group C were more deeply anesthetized when their trachea was intubated, than those in Groups A and B. A previous study reported that laryngoscopic intubation under 2% end-tidal sevoflurane concentration increased SAP by 36 mmHg and HR by 36 (beats·min⁻¹) in ASA class I or II gynecological patients.¹² Therefore, it is not likely that the attenuation of the cardiovascular responses to endotracheal intubation in Group C was due to deep sevoflurane anesthesia. Most of the previous studies designed to examine the effectiveness of tracheal lidocaine place emphasis on the

cardiovascular changes after endotracheal intubation. Therefore, in these studies, the cardiovascular responses to endotracheal intubation following a tracheal spray with local anesthetic were compared with those following the topical application of saline or *iv* application of lidocaine.^{8,9,11} The present study emphasizes the cardiovascular responses to tracheal lidocaine as well as those to endotracheal intubation with or without tracheal lidocaine. Although our study demonstrates that tracheal lidocaine alone provokes a significant increase in HR and blood pressure, the magnitude of these cardiovascular responses to tracheal lidocaine was half that of tracheal intubation without topical anesthesia. Tracheal lidocaine blocked the cardiovascular responses to endotracheal intubation. In addition, the present study also shows that endotracheal intubation with tracheal lidocaine does not produce an increased risk of hypotension during induction of anesthesia. This study suggests that tracheal lidocaine can reduce the dose of narcotics required to block the cardiovascular responses to endotracheal intubation, and may be a useful strategy when combined with other drugs to decrease the risk of hypotension or delayed emergence.

We conclude that tracheal lidocaine is an effective method for attenuating the cardiovascular responses to endotracheal intubation without producing an increased risk of hypotension. Endotracheal intubation should be performed more than two minutes after tracheal lidocaine in order to attenuate the cardiovascular responses to endotracheal intubation.

Acknowledgement

We thank Professor Harry G.G. Kingston, Department of Anesthesiology, Oregon Health Science University, School of Medicine, for his helpful comments.

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