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The effect of intravenous lidocaine on hemodynamic response to endotracheal intubation during sufentanil-based induction of anaesthesia

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Abstract

Background: Endotracheal intubation (ETI) can cause a cardiovascular response. The aim of the present study was to investigate the effect of intravenous lidocaine on the hemodynamic response to ETI during sufentanil-based induction of anaesthesia.

Methods: Ninety patients aged 18–65 years were recruited, induction of anaesthesia was initiated by sufentanil, midazolam, cisatracurium, and propofol, the patients were randomized to three groups: Group L1 received 1 mg/kg^{-1} of lidocaine, Group L1.5 received 1.5 mg kg^{-1} of lidocaine, Group S received an equal volume of normal saline (NS). Lidocaine or NS was administered in a bolus 2 min before ETI. Systolic arterial pressure (SAP), diastolic arterial pressure (DAP), mean arterial pressure (MAP), and heart rate (HR) were recorded at four time points: before anaesthetic induction, 1 min after lidocaine administration, immediately after ETI, 5 min after ETI. The incidences of hypotension, hypertension, bradycardia, and tachycardia were also recorded.

Results: The SAP, DAP, MAP, and HR at baseline were not significantly different among the three groups ($P = 0.620$, $P = 0.575$, $P = 0.433$, $P = 0.537$, respectively). Immediately after ETI, the SAP in Group L1 was significantly lower than Group S ($P = 0.024$), while the DAP, MAP, and HR were comparable among the three groups at the same time points ($P > 0.05$). There were no significant differences in the incidences of hypotension, hypertension, bradycardia and tachycardia among the three groups ($P > 0.200$).

Conclusions: Intravenous lidocaine could attenuate the increase of blood pressure but not HR after ETI during sufentanil-based induction of anaesthesia without increased incidence of side-effects.

Key words: intubation, lidocaine, induction, intravenous, sufentanil.

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Endotracheal intubation (ETI) during induction of general anaesthesia can cause a temporary significant hemodynamic response [1]. It could be potentially harmful, and increases the risk of cardiovascular events, especially in elderly patients or patients with cardiovascular diseases. As an analogue of fentanyl, sufentanil has been widely used for blunting the hemodynamic response induced by ETI. It could reduce the dose of propofol or etomidate taken for loss of consciousness and provide a relatively stable hemodynamic profile during anaesthetic induction [2, 3]. However, a low dose of sufentanil may be insufficient to suppress the hemodynamic response induced by ETI [2, 4]. On the other hand, a large dose of sufentanil may lead to hypotension during anaesthetic induction, prolong emergence from anaesthesia, and cause respiratory depression after a short operation [5, 6]. Therefore, adding a suitable agent to suppress the stimulation induced by ETI without causing cardiovascular compromise during sufentanil-based anaesthetic induction may be worthwhile.

Lidocaine is a local anaesthetic and class IB antidysrhythmic agent. It was reported that intravenous administration of lidocaine could effectively suppress the hemodynamic response to laryngoscopy and ETI [7]. A study from animals showed that both intravenous and topical laryngeal lidocaine could attenuate the pressor response to ETI, whereas intravenous lidocaine further reduced the cough response [8]. In addition, a clinical study showed that intravenous lidocaine was able to diminish the hemodynamic response induced by ETI and maintain the baseline conditions of patients during anaesthetic induction [9]. Based on the evidence regarding the efficacy of sufentanil and lidocaine,

we hypothesized that the combination of the two agents may provide better hemodynamic stability during induction of anaesthesia and ETI than sufentanil alone. To the best of our knowledge, relevant research regarding this issue is still inadequate. Therefore, the current study was designed to investigate the effects of additional intravenous lidocaine on sufentanil-based induction of general anaesthesia and ETI.

METHODS

The randomized controlled trial was approved by the Institutional Committee of Ethics of the hospital (2019-12). After written informed consent was obtained, 90 patients aged 18–65 years with American Society of Anesthesiologists (ASA) physical status I or II scheduled for elective surgery between March 2019 and June 2019 were recruited. Exclusion criteria were allergy to lidocaine, history of cardiovascular disease, cardiac arrhythmia, tachycardia, respiratory disease, cerebral disease, and predicted difficult airway. No premedication was given to any of the patients before surgery. On arrival in the operating room, the patients were routinely monitored by electrocardiography, non-invasive blood pressure (BP), and pulse oximetry. Ringer's solution was infused at a rate of 4–6 mL min^{-1} via a peripheral intravenous line. The patients were preoxygenated via a facemask with a fresh gas flow of 6 L min^{-1}. Induction of anaesthesia was initiated by intravenous administration of sufentanil (Yichang Humanwell Pharmaceutical Co, Ltd, Hubei, China) 0.4 μg kg^{-1}; after 30 seconds, midazolam 0.04 mg kg^{-1} and cisatracurium 0.2 mg kg^{-1} were administered in sequence during 30 seconds; then, propofol 0.5 mg kg^{-1} was administered followed by 10–30 mg to the loss of consciousness. Manual ventilation was performed during induction of anaesthesia. The patients were randomized to 3 groups according to computer-generated random numbers (n = 30 per group):

- Group L1: immediately after propofol administration, a bolus of lidocaine (1 mg kg^{-1}, Suicheng Pharmaceutical Co, Ltd, Henan, China) was administered over 5 seconds,
- Group L1.5: immediately after propofol administration, a bolus of lidocaine (1.5 mg kg^{-1}) was administered over 5 seconds,
- Group S: immediately after propofol administration, an equal volume of normal saline (NS) was administered over 5 seconds.

Two minutes after administration of lidocaine or NS, oral ETI assisted by a video-laryngoscope was performed by an experienced anaesthesiologist in 30 seconds. The cuff of the tube was inflated immediately after intubation. Anaesthesia was maintained by inhalation of sevoflurane with an end-tidal concentration of 0.6–1% depending on the patient's age.

The vital signs including systolic arterial pressure (SAP), diastolic arterial pressure (DAP), mean arterial pressure (MAP), and heart rate (HR) were recorded at 4 time points:

- T0: immediately before administration of sufentanil,
- T1: 1 min after administration of lidocaine,
- T2: Immediately after cuff inflation of endotracheal tube,
- T3: 5 min after cuff inflation of endotracheal tube.

During anaesthetic induction and maintenance, atropine 0.2–0.5 mg was administered for HR < 45 bpm, and dopamine 1–2 mg was administered for SAP < 80 mm Hg with or without HR < 45 bpm. SAP ≥ 180 mm Hg or HR ≥ 100 bpm after ETI was considered to be inadequate anaesthesia; propofol 30–50 mg was administered for this condition. The incidences of hypotension (SAP < 80 mm Hg or MAP < 55 mm Hg), hypertension (SAP ≥ 180 mm Hg), bradycardia (HR < 45 bpm), and tachycardia (HR ≥ 100 bpm) in each group at each time points were also recorded.

Data were expressed as mean ±standard deviation (SD), number, and percentage. The normality of data distribution was tested by the Shapiro-Wilk test. Comparison of age and weight was analysed with Student's t-test. Gender, ASA physical status, and incidences of hypotension, hypertension, bradycardia, and tachycardia were compared using the χ^2 test or Fisher's exact test. Comparison of SAP, DAP, MAP, and HR was analysed with repeated-measures two-way analysis of variance (ANOVA). Multiple comparisons were performed using Tukey's test. SPSS 22.0 (SPSS Inc, Chicago, IL, USA) was used for statistical analysis. A P value of < 0.05 was considered statistically significant.

RESULTS

The gender, age, weight, and ASA physical status of the patients among the three groups were comparable (Table 1).

As shown in Table 2, the baseline values of SAP, DAP, MAP, and HR at T0 were comparable among the groups (P = 0.620, P = 0.575, P = 0.433, P = 0.537, respectively). In each group, the SAP, DAP, MAP, and HR decreased significantly after anaesthetic induction at T1 (P < 0.001), and then increased significantly immediately after ETI at T2 (P < 0.001), and decreased again after 5 min of ETI at T3 (P < 0.001). At T2, the SAP in Group L1 was significantly lower than Group S (P = 0.024). Even though the SAP in Group L1.5 was also slightly lower than Group S at T2, the difference was not significant (P = 0.184). In addi-

tion, in Group L1 and Group L1.5, the SAP values at T2 were significantly lower than the baseline at T0 ($P = 0.001$ and $P = 0.003$); however, in Group S, the SAP at T2 was not significantly different compared to T0 ($P = 0.999$). Meanwhile, in Group L1 and Group L1.5, the MAP values at T2 were also significantly lower than T0 ($P = 0.005$ and $P = 0.021$). Moreover, in each group, the HR at T2 was significantly higher than the baseline at T0 ($P = 0.036$ in Group L1, $P = 0.000$ in Group L1.5, $P = 0.001$ in Group S), and the HR at T2 was not significantly different between the three groups ($P = 0.921$). There were no other significant differences in DAP and MAP among the three groups at the same time points ($P > 0.05$).

No hypertension or bradycardia occurred in any of the groups. There were no significant differences in the incidence of hypotension ($P = 0.749$ at T1 and $P = 0.200$ at T3) or tachycardia ($P = 0.713$ at T2) among the three groups (Table 3).

TABLE 1. Patients' demographic details

Parameters	Group L1 ($n = 30$)	Group L1.5 ($n = 30$)	Group S ($n = 30$)	*P*-value
Gender (M/F)	13/17	17/13	14/16	0.56
Age (years)	44 ±14	48 ±9	45 ±14	0.46
Body mass (kg)	62 ±9	64 ±10	62 ±7	0.55
ASA status (I/II)	7/23	9/21	9/21	0.80

Data are number or mean ±standard deviation (SD)
There were no significant differences in any parameters among the groups

DISCUSSION

The current study showed that sufentanil 0.4 μg kg^{-1} effectively attenuated the increase of BP induced by ETI; immediately after ETI, the SAP, DAP, and MAP were not significantly different from the baseline. Moreover, the additional intravenous lidocaine 1 mg kg^{-1} or 1.5 mg kg^{-1} further attenuated the increase of BP induced by ETI without an increased incidence of hypotension or bradycardia during anaesthetic induction. However, sufentanil 0.4 μg kg^{-1} with or without the additional intravenous lidocaine failed to suppress the dramatic increase of HR induced by ETI.

Hemodynamic instability is not uncommon during anaesthetic induction and ETI [1, 10]. Propofol is widely used for induction of anaesthesia; however, either a bispectral index (BIS)-guided or body weight-adjusted dose of propofol (2 mg kg^{-1}) could lead to hypotension with an incidence up to 45% [11]. It was reported that midazolam could lower propofol consumption and reduce hemodynamic variations during anaesthetic induction [12]. In addition, both of the drugs possess hypnotic and amnesic effects [13, 14]. Therefore in the current study, midazolam and propofol were used in combination to avoid cardiovascular compromise during anaesthetic induction. The doses of midazolam and propofol used in the study were based on the previous studies and the loss of patient consciousness [12, 15].

Studies showed that sufentanil could maintain the stability of circulation and inhibit the stress response during induction of general anaesthesia and ETI [2, 3]. However, an adequate dose of sufentanil for blunting the hemodynamic response may induce hypotension during anaesthetic induction, prolong emergence from anaesthesia or cause respiratory depression after surgery [5, 6]. On the other hand, the commonly recommended dose (0.3 μg kg^{-1}) was still inadequate to suppress the hemodynamic response induced by ETI [16]. Hence, in the current study, the induction dose of sufentanil

TABLE 2. Vital signs in each group at baseline (T0), before endotracheal intubation (ETI) (T1), immediately after ETI (T2), and 5 min after ETI (T3)

Vital signs	Group ($n = 30$)	T0	T1	T2	T3
SAP (mm Hg)	Group L1	125.7 ±13.7	92.8 ±13.5^{a}	113.5 ±19.5^{a,b,d}	98.5 ±16.0^{a,c}
	Group L1.5	128.0 ±13.0	94.9 ±10.7^{a}	117.1 ±23.0^{a,b}	103.4 ±11.7^{a,b,c}
	Group S	124.8 ±12.2	97.8 ±13.3^{a}	124.4 ±24.1^{b}	101.9 ±13.2^{a,c}
DAP (mm Hg)	Group L1	76.5 ±9.4	53.0 ±8.3^{a}	70.9 ±13.3^{ab}	58.1 ±9.3^{a,c}
	Group L1.5	76.9 ±11.3	56.7 ±10.0^{a}	75.2 ±15.5b	63.0 ±9.4^{a,b,c}
	Group S	74.2 ±10.5	54.2 ±9.5^{a}	74.2 ±15.5^{b}	56.4 ±10.7^{a,c}
MAP (mm Hg)	Group L1	91.8 ±11.3	63.5 ±7.9^{a}	83.9 ±13.9^{a,b}	70.6 ±12.1^{a,b,c}
	Group L1.5	92.7 ±10.5	66.5 ±10.2^{a}	85.8 ±15.8^{a,b}	75.2 ±10.3^{a,b,c}
	Group S	89.1 ±11.8	67.5 ±10.5^{a}	90.6 ±18.5^{b}	71.3 ±11.8^{a,c}
HR (bpm)	Group L1	77.8 ±10.9	67.2 ±11.2^{a}	83.2 ±13.4^{a,b}	64.5 ±10.8^{a,c}
	Group L1.5	74.8 ±11.3	66.3 ±11.1^{a}	84.6 ±17.5^{a,b}	64.6 ±11.4^{a,c}
	Group S	75.1 ±11.3	64.9 ±11.6^{a}	83.1 ±16.4^{a,b}	62.8 ±10.6^{a,c}

Data are mean ±standard deviation (SD)
^{a} $P < 0.036$ compared with T0 in the same group, ^{b} $P < 0.032$ compared with T1 in the same group, ^{c} $P < 0.001$ compared with T2 in the same group, ^{d} $P < 0.024$ compared with group S at the same time point

TABLE 3. Incidences of hypotension (SAP < 80 mm Hg or MAP < 55 mm Hg), hypertension (SAP ≥ 180 mm Hg), bradycardia (HR < 45 bpm), and tachycardia (HR ≥ 100 bpm) in each group before induction of anaesthesia (T0), before endotracheal intubation (ETI) (T1), immediately after ETI (T2), and 5 min after ETI (T3)

Parameter	Group ($n = 30$)	T0	T1	T2	T3
Hypotension	Group L1	0 (0)	4 (13.3)	0 (0)	3 (10.0)
	Group L1.5	0 (0)	3 (10.0)	0 (0)	0 (0)
	Group S	0 (0)	5 (16.7)	0 (0)	3 (10.0)
Hypertension	Group L1	0 (0)	0 (0)	0 (0)	0 (0)
	Group L1.5	0 (0)	0 (0)	0 (0)	0 (0)
	Group S	0 (0)	0 (0)	0 (0)	0 (0)
Bradycardia	Group L1	0 (0)	0 (0)	0 (0)	0 (0)
	Group L1.5	0 (0)	0 (0)	0 (0)	0 (0)
	Group S	0 (0)	0 (0)	0 (0)	0 (0)
Tachycardia	Group L1	0 (0)	0 (0)	4 (13.3)	0 (0)
	Group L1.5	0 (0)	0 (0)	6 (20.0)	0 (0)
	Group S	0 (0)	0 (0)	4 (13.3)	0 (0)

Data are number (%)

There were no significant differences in the incidences of hypotension ($P = 0.749$ at T1 and $P = 0.200$ at T3), hypertension ($P = 1.0$), bradycardia ($P = 1.0$) or tachycardia ($P = 0.713$ at T2) among the groups

was 0.4 μg kg^{-1}, which was also recommended for induction of general anaesthesia and ETI in the previous study [17]. In addition, the maximum effect of sufentanil was achieved 3–6 min after an intravenous bolus [18]. Therefore, ETI was performed nearly 5 min after sufentanil administration.

The results showed that the SAP, DAP, MAP, and HR decreased significantly after induction of anaesthesia at T1 in Group S. However, the incidence of hypotension during anaesthetic induction was lower than the reported data [11], which may be due to the differences of the anaesthetic induction method and the definitions of hypotension. Immediately after ETI, the BP and HR increased significantly. However, the SAP, DAP, and MAP at T2 were not significantly different from T0 in Group S; these results indicated that sufentanil 0.4 μg kg^{-1} effectively attenuated the increase of BP induced by ETI. However, the HR at T2 was significantly higher than T0 in Group S; these results indicated that the hemodynamic response induced by ETI was still not adequately suppressed by sufentanil at 0.4 μg kg^{-1}. Therefore, adding an additional agent to suppress the stimulation of ETI is worthwhile.

As a frequently used local anaesthetic, lidocaine closes the Na^+ channels and prevents the signals from reaching the postsynaptic cell. On the other hand, intravenous lidocaine also blocks the sodium channels in the heart, which is used for treating ventricular arrhythmias. Studies also showed that intravenous lidocaine elevated the threshold of airway stimulation, directly depressed the hemodynamic response, and inhibited sympathetic transmission, thus suppressing the sympathetic response induced by ETI [7, 19]. A meta-analysis showed that intravenous lidocaine 0.5-2 mg kg^{-1} dose-dependently prevented intubation-, extubation-, and opioid-induced cough without any adverse effects [20]. Therefore, intravenous administration of lidocaine may be a promising choice for providing a stable hemodynamic profile in ETI. Qi *et al.* [7] summarized 37 clinical trials regarding intravenous lidocaine on attenuating the ETI-induced hemodynamic response; the authors reported that 1 mg kg^{-1} and 1.5 mg kg^{-1} dosages of lidocaine were effective in reducing systolic BP and HR changes in ETI. In consideration of the combination with sufentanil in the current study, the doses of lidocaine used in the current study were 1 mg kg^{-1} and 1.5 mg kg^{-1}. In addition, Tam *et al.* [21] reported that intravenous lidocaine 1.5 mg kg^{-1} attenuated ETI-induced increases of BP and HR only when given 3 min before ETI. Therefore, ETI was performed 2–3 min after lidocaine bolus in the current study.

The results showed that the additional intravenous lidocaine further diminished the increases of SAP and MAP after ETI. However, both of the two doses of lidocaine failed to suppress the increase of HR, which was not in line with the previous reports [7, 19, 21]. At least, the current study indicated that lidocaine at 1 mg kg^{-1} or 1.5 mg kg^{-1} could not further suppress the increase of HR induced by ETI when sufentanil was already used. The reason might be that the intravenous bolus of lidocaine 2–3 min before ETI could only slightly attenuate the hemodynamic response of ETI and the benefit could hardly be detected when combined with sufentanil. Nevertheless, similar results regarding the effects of intravenous lidocaine on the hemodynamic response during anaesthetic induction and ETI were reported previously. Qi *et al.* [7] found that intravenous lidocaine effectively reduced ETI-induced BP elevation but not HR changes in children; they speculated that children had a greater pressor response and higher baseline HR, making it harder to suppress the hemodynamic response to ETI. In addition, Hassani *et al.* [22] also found that fentanyl plus lidocaine was not more effective than fentanyl alone to suppress the ETI-induced hemodynamic response in controlled hypertensive patients. These differences may due to the different designs of the studies. However, the current study further supported the view that intravenous lidocaine might not be a good choice to suppress the ETI-induced increase of HR in sufentanil-based anaesthetic induction.

The incidences of hypotension, hypertension, bradycardia, and tachycardia in the three groups were not significantly different. These results in-

dicated that the addition of lidocaine would not induce further side-effects or adverse events, and could be safely used in sufentanil-based anaesthetic induction. However, based on the results of the current study, we found that there were no advantages to use of lidocaine as an adjunctive agent to sufentanil for blunting the hemodynamic response in ETI.

There were certain limitations of the study. First, the sample size in the study was small; a larger sample size would be more persuasive. Second, the BP was not monitored invasively; hence it was unable to detect the maximal and minimal BP during induction of anaesthesia. Third, although the recruited patients were similar in demographic details, potential selection bias might have been introduced such as the range of ages; this bias might have led to different tolerability to anaesthetics and different changes of vital signs during anaesthetic induction. Nevertheless, the study failed to prove the effect of intravenous lidocaine on suppressing the increase of HR induced by ETI.

CONCLUSIONS

As an adjunctive agent to sufentanil for induction of general anaesthesia, intravenous lidocaine 1 and 1.5 mg kg^{-1} could slightly attenuate the increase of BP after ETI without increased incidence of side-effects. However, it failed to suppress the increase of HR induced by ETI.

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