



# Intravenous lidocaine for attenuation of pressor response after endotracheal intubation. A randomized, double-blinded dose-finding study

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## ABSTRACT

**Introduction:** Endotracheal intubation (ETI) is one of the commonly used maneuvers in the daily anesthetic practice, it is commonly associated with hemodynamic stimulation leading to marked tachycardia, hypertension, and myocardial ischemia.

**Aim and objectives:** In this study, we compared three doses of lidocaine for prophylaxis against pressor response of ETI. We hypothesized that the larger dose (2 mg/Kg) will be more effective for attenuation of the pressor response.

**Methods:** After randomization, post induction of anesthesia patients were categorized into three groups according to the dose of lidocaine: group A received 1 mg/Kg, group B received 1.5 mg/Kg, and group C received 2 mg/Kg. To achieve blinding, the study drug was prepared by a research assistant and was diluted to 10 mL in all groups. Heart rate measurement after 1 minute of lidocaine injection was carried out. Heart rate, cardiac output and stroke volume were continuously measured and were recorded every 30 seconds starting from baseline pre-induction reading till 5 minutes after ETI, systolic and diastolic blood pressure were measured at 1-minute intervals starting from baseline reading till 5-minute after ETI.

**Results:** Pressor response was lower in group C receiving 2 mg/Kg with  $P$  value = 0.021 defined by an increase in the heart rate, cardiac output, or systolic blood pressure by 20% or more which was evaluated after ETI continuously for 5 minutes.

**Conclusion:** Lidocaine in the dose of (2mg/Kg) is more effective than lower doses in attenuation of the pressor response of the ETI.

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## 1. Introduction

Somato-visceral reflexes are thought to be the mechanism behind the pressor response, which is a reaction to laryngoscopy and orotracheal intubation [1]. When proprioceptors at the base of the tongue are stimulated during a laryngoscopy, systemic blood pressure, heart rate (HR), and plasma catecholamine concentrations rise in an impulse-dependent manner. Following oro-tracheal intubation, more receptors are recruited, increasing the hemodynamic and epinephrine responses as well as some vagal heart inhibition [2]. The related cardiovascular complications could be serious in patients with cardiac comorbidities [3–5]. Although the value of lidocaine during induction of anesthesia was well investigated, the optimum dose for lidocaine as an adjuvant to induction agents is not known. Previous reports had investigated different doses of lidocaine varying between 1 mg/Kg to 2 mg/Kg [6–8]. However, no studies to the best of our knowledge had compared different doses of lidocaine. Qi et al [9] had reported that lidocaine is an effective agent in attenuation of pressor response for ETI; however, they suggested that the optimum dose needs further research. Electrical cardiometry is recently introduced for assessment of many cardiovascular variables and continuously applicable method of cardiac output (CO), stroke volume

(SV), and other hemodynamic parameters monitoring. Its use is growing because it is non-invasive, shows reliability in CO measurements and can be used as a continuous bedside monitor.

In this study, we compared three doses of lidocaine for prophylaxis against pressor response of ETI. We hypothesized that the larger dose (2 mg/Kg) will be more effective for attenuation of the pressor response.

## 2. Material and method

From October 2018 to March 2019, 51 adult patients were enrolled in our dose finding study in Faculty of medicine, Cairo university hospitals. All participant gave written informed permission after being told of the study's purpose and methodology. A research assistant created random sequences using an Internet randomization tool (<http://www.randomizer.org>). A sealed, opaque envelope contained each code. A different research helper, unconnected to outcome evaluation, was in charge of removing the package and getting the study medication ready. The instructions for drug preparation were included in the envelope. The drug was prepared according to the patient weight and diluted to 10 mL in all patients.

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### 3. Inclusion criteria

- Patients aged above 18 years old, both sexes.
- Undergo general anesthesia

#### Exclusion criteria:

- Patients with suspected difficult airway {e.g., high neck circumference (above 42 cm), high body mass index (above 30 kg/m<sup>2</sup>), airway masses, mouth scars, neck scars, limited neck extension or history of snoring.
- Total duration of laryngoscopy will be noted and in cases where duration exceeded 15 sec the case will be excluded from the study.
- Any patient on regular intake of beta blockers or calcium channel blockers.

Patients were allocated, according to the study group into:

- Group A: this group received lidocaine 1 mg/kg after induction of anesthesia
- Group B: this group received lidocaine 1.5 mg/kg after induction of anesthesia
- - Group C: this group received lidocaine 2 mg/Kg after induction of anesthesia.

### 4. Intraoperative management

Upon arrival to the operating room, basic monitoring as Electrocardiography (ECG), Non-invasive Blood Pressure (NIBP) monitor and pulse oximetry was attached, nerve stimulator to ensure adequate muscle relaxation and Electrical cardiometry for cardiac output monitoring. Base line readings were documented. Ringer's lactate solution was infused when intravenous access was established. Prior to being moved to the operation room, the patients received pre-medicating IV infusions of ranitidine 50 mg and IV midazolam 0.05 mg/Kg over 30 seconds.

All patients were pre-oxygenated with four to five breaths of 100% oxygen and Intravenous induction of anesthesia was done using propofol (2 mg/Kg), fentanyl (1.5 mcg/Kg) and rocuronium (0.6 mg/Kg). After 3 minutes of mask ventilation with isoflurane (1.5%), intravenous lidocaine was administered according to the group allocation, then Endotracheal intubation (ETI) was performed.

Patients who experienced prolonged laryngoscopy (>15 seconds) were excluded from the study.

### 5. Measurement tools

Arterial blood pressure monitoring, Electrocardiogram and Pulse oximeter, Nerve stimulator to ensure adequate muscle relaxation and the ICONR monitor for electrical cardiometry (Osypka Medical, Inc., La Jolla,

California and Berlin). Germany) were utilized to track cardiac output. Patients' skin was covered with four ECG electrodes in the following positions: (1) on the left neck, just below the ear; (2) across the middle of the left clavicle; (3) along the left mid-axillary line, at the level of the xiphoid process; and (4) two inches below the third electrode.

### 6. Outcomes

#### Primary Outcome:

Heart rate measurement 1 min after intubation.

#### Secondary outcomes:

- Heart rate (it was continuously measured and was recorded every 30 seconds starting from baseline pre-induction reading till 5 minutes after ETI. Seventeen readings were recorded as follows: baseline pre-induction, six readings after induction and before laryngoscopy and 10 readings after ETI).
- Cardiac output (was measured continuously and was recorded every 30 seconds starting from baseline reading till 5-minute after ETI).

Seventeen readings were recorded as follows: baseline pre-induction, six readings after induction and before laryngoscopy and 10 readings after ETI.

- Stroke volume (was measured continuously and was recorded every 30 seconds starting from baseline reading till 5-minute after ETI).

Seventeen readings were recorded as follows: baseline pre-induction, six readings after induction and before laryngoscopy and 10 readings after ETI.

- Systolic blood pressure (was measured at 1-minute intervals starting from baseline reading till 5-minute after ETI).

Nine readings were recorded as follows: baseline pre-induction, three readings after induction and before laryngoscopy and five readings after ETI.

- Diastolic blood pressure (was monitored starting with the baseline reading and continuing for 5 minutes after ETI at 1-minute intervals).

Nine readings were recorded as follows: baseline pre-induction, three readings after induction and before laryngoscopy and five readings after ETI.

- Pressor response which is defined as an increase in the heart rate, cardiac output, or systolic blood pressure by 20% or more were evaluated after ETI for 5 minutes.

## 7. Sample size calculation

Our primary outcome was heart rate 1 minute after ETI. In a previous study [6], the pre-intubation heart rate in lidocaine group was ( $71 \pm 3$  bpm) which increased by 12% after intubation to be ( $80 \pm 4$  bpm).

Our sample size was determined to be  $3.3 \pm 3$  bpm, which is 5% less than the baseline measurement. A minimum of 46 patients (16 patients per group) were needed to achieve a research power of 95% and an alpha error of 0.025, according to the G power (14.10.2) programme. To account for potential drop-outs, the number was increased to 51 patients (17 patients each group).

### 7.1. Statistical analysis

Data analysis was carried out using the SPSS programme, version 15 for Microsoft Windows (SPSS Inc., Chicago, IL, USA). The frequency (%) of categorical data was reported, and the relevant chi-squared test or Fisher's exact test was used to analyse it.

Shapiro-Wilk test was used to determine the normality of continuous data, which was then reported as the mean (standard deviation) or median (quartiles) as necessary. Unpaired t-tests (for normally distributed data) and Mann-Whitney tests on ranks were used to analyse continuous data (for skewed data). A mixed ANOVA was performed for repeated measurements using a generic linear model (within-between subject factors). The Bonferroni test was used to make a post-hoc pairwise comparison. Statistical significance was defined as a *P*-value 0.05.

## 8. Results

Fifty-one adult patients scheduled for general anaesthesia were enrolled in the study. Patients were randomly

allocated by a computer-generated table into one of the study groups according to the dose of lidocaine. The randomization sequence had been concealed in sealed opaque envelopes. Group A ( $n = 17$ ): received 1 mg/kg, Group B ( $n = 17$ ): received 1.5 mg/kg and Group C ( $n = 17$ ): received 2 mg/kg.

## 9. The results showed the following

As regards age, weight and sex, there was no statistically significant differences found between three groups (*P*-value > 0.05) (Tables 1,2).

## 10. Data monitoring

### 10.1. Heart rate

Heart rate was monitored every 30 seconds beginning with the pre-induction baseline data every 30 s for 3 min till ETI insertion and continuing for 5 min following endotracheal intubation (ETI). Mean and standard deviation are used to show the data (SD). Group B and group C had a statistically significant difference at (HR 0.5), and there was a statistical difference between group A and group C at (HR 0.5, 2.5, 3), but there was no statistical significance between the other two groups (Table 3).

### 10.2. Stroke volume

Stroke volume was monitored every 30 seconds beginning with the pre-induction baseline data every 30 s for 3 min till ETI insertion and continuing for 5 min following endotracheal intubation (ETI). Group A and group C showed a statistical difference at (SV 1, 2, 5), as did group B and group C at (SV 1, 2, 5), but there was no statistical difference between the other two groups (Table 4).

**Table 1.** Gender of the patients in three groups. Represented as count and percentage (%). Group A: Lidocaine dose 1 mg/kg, Group B: Lidocaine dose 1.5 mg/kg, Group C: Lidocaine dose 2 mg/kg.

		group						P value
		Group A (n=17)		Group B(n=17)		Group C (n=17)		
		Count	%	Count	%	Count	%	
Gender	male	10	58.8	12	70.6	11	64.7	0.773
	female	7	41.2	5	29.4	6	35.3	

**Table 2.** Age and weight of the patients in three groups. Data are represented as mean and standard deviation (SD). Group A: lidocaine dose 1 mg/kg, Group B: lidocaine dose 1.5 mg/kg, Group C: lidocaine dose 2 mg/kg.

	group						P value
	Group A (n=17)		Group B (n=17)		Group C (n=17)		
	Mean	SD	Mean	SD	Mean	SD	
Age(years)	35.8	11.3	32.7	6.7	34	7.2	0.565
Weight(kg)	76.1	7.9	77.4	10.4	74.9	10.9	0.766

**Table 3.** Heart rate (beats per minute) in three groups, presented as mean± SD. *Group A:* lidocaine dose 1 mg/kg, *Group B:* lidocaine dose 1.5 mg/kg, *Group C:* lidocaine dose 2 mg/kg. \*denotes significance between Group a and Group C \*\*denotes significance between Group B and Group C.

	group						P value
	Group A (n=17)		Group B (n=17)		Group C (n=17)		
	Mean	SD	Mean	SD	Mean	SD	
HR0(bpm)	81.1	14.7	82.2	11.9	90.9	16.9	0.114
HR0.5(bpm)	83.9*	13.8	83.1**	12.2	85.9	18.7	0.03*
HR1(bpm)	86.8	12.4	89.5	10.8	98.3	20.1	0.076
HR1.5(bpm)	85.8	11.2	86.2	11.6	93.5	17.9	0.203
HR2(bpm)	83.2	9.5	84.8	11.5	93.5	16.8	0.052
HR2.5(bpm)	82.4*	9.9	82.9	10.1	91.4	12.9	0.038*
HR3(bpm)	81.6*	11.6	82.4	9.4	91	12.1	0.029*
HR3.5(bpm)	99.2	11.4	92.8	16.2	93.4	16.9	0.388
HR4(bpm)	101.4	13.4	95.4	16.1	94.1	16.1	0.332
HR4.5(bpm)	100.8	12.6	94.4	15.3	93.1	13	0.218
HR5(bpm)	100	13.1	94.7	14.4	91.8	12.4	0.198
HR5.5(bpm)	97	11.7	93.4	12	90.9	11.9	0.334
HR6(bpm)	94.2	12.3	92.5	11.9	89	13	0.467
HR6.5(bpm)	91.9	12	90.6	10.8	87.8	11.5	0.577
HR7(bpm)	90.7	11.1	90.2	11.7	85.8	10.1	0.368
HR7.5(bpm)	87.7	10.2	87.8	11	85.8	10.5	0.836
HR8(bpm)	87.1	9.9	86	10.7	86.2	9.9	0.943

**Table 4.** Stroke volume for the three groups. Data is presented as mean and standard deviation (SD) *Group A:* lidocaine dose 1 mg/kg, *Group B:* lidocaine dose 1.5 mg/kg, *Group C:* lidocaine dose 2 mg/kg.\*denotes significance between Group a and Group C \*\*denotes significance between Group B and Group C.

	group						P value
	Group A (n=17)		Group B (n=17)		Group C (n=17)		
	Mean	SD	Mean	SD	Mean	SD	
SV0	62.4	10.9	59.3	11.8	68	15.7	0.150
SV0.5	63.1	10.9	60.4	11.6	70.2	15.1	0.072
SV1	61.6	10.4	59.6**	11.8	69.8	13.1	0.037*
SV1.5	61.4	11.2	58.4	11.5	68	14.2	0.078
SV2	60.8	10.3	57.8**	11.9	68.9	11.7	0.017*
SV2.5	61.1	9.7	57***	11.8	67.2	11.7	0.033*
SV3	59.5	10.3	57.7	13.4	67.7	14.6	0.064
SV3.5	69.9	10.6	67.2	13.5	69.8	13.3	0.785
SV4	71.5	10.6	68.4	13.7	69.1	15.3	0.770
SV4.5	69.8	10.2	68	12.8	69.6	13.7	0.897
SV5	69.7	10.5	66.8	12.4	68.2	14.5	0.807
SV5.5	69.2	11	65	10.9	68.9	13.9	0.528
SV6	69	13.3	64.9	11.7	69.4	13.4	0.531
SV6.5	66.8	12.1	64.4	10.9	68.4	12.4	0.604
SV7	66.3	12	63.5	11.1	66.7	12.9	0.702
SV7.5	66.9	11.9	62.6	10.5	66.6	12.6	0.567
SV8	59.9	23.8	62.7	10.9	66.7	11.6	0.483

### 10.3. Cardiac output

CO was recorded every 30 seconds starting from baseline reading for 3 min till ETI insertion continuing till 5-min after ETI; Data are presented as mean and standard deviation (SD), there was no statistical significance between three groups (Figure 1) (Table 5).

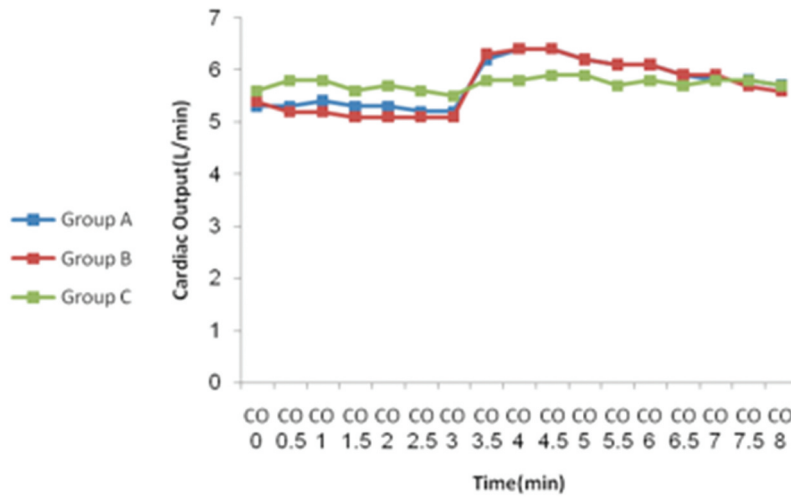
### 10.4. Systolic arterial blood pressure (SBP)

SBP was measured at 1-minute intervals starting from baseline reading for 3 min till ETI insertion continuing till 5-minute after ETI, Data is presented as mean and

standard deviation (SD). There was statistical difference between group A and group C at (SBP 1,5,6,7,8), otherwise there was no statistical significance between three groups (Table 6).

### 10.5. Diastolic arterial blood pressure (DBP)

DBP was measured at 1-minute intervals starting from baseline reading for 3 min till ETI insertion continuing till 5-minute after ETI. Data are presented as mean and standard deviation (SD). There was no statistical significance between three groups (Table 7).



**Figure 1.** Cardiac output for the three groups. Group A: lidocaine dose 1mg/kg, Group B: lidocaine dose 1.5mg/kg, Group C: lidocaine dose 2mg/kg.

**Table 5.** Cardiac output for the three groups. Data is presented as mean and standard deviation (SD). Group A: lidocaine dose 1 mg/kg, Group B: lidocaine dose 1.5 mg/kg, Group C: lidocaine dose 2 mg/kg.

	group						P value
	Group A (n=17)		Group B (n=17)		Group C (n=17)		
	Mean	SD	Mean	SD	Mean	SD	
CO 0 (L/m)	5.3	1.1	5.4	1.1	5.6	1.5	0.826
CO 0.5 (L/m)	5.3	1	5.2	1.2	5.8	1.6	0.349
CO 1 (L/m)	5.4	1	5.2	1.1	5.8	1.5	0.317
CO 1.5 (L/m)	5.3	1.1	5.1	1	5.6	1.3	0.356
CO 2 (L/m)	5.3	1.1	5.1	1	5.7	1.4	0.288
CO 2.5 (L/m)	5.2	1.1	5.1	1.1	5.6	1.3	0.412
CO 3 (L/m)	5.2	1.1	5.1	1.1	5.5	1.4	0.608
CO 3.5 (L/m)	6.2	1	6.3	1.3	5.8	1.6	0.484
CO 4 (L/m)	6.4	1.1	6.4	1.4	5.8	1.5	0.355
CO 4.5 (L/m)	6.4	1.1	6.4	1.5	5.9	1.5	0.432
CO 5 (L/m)	6.2	1.1	6.2	1.4	5.9	1.3	0.659
CO 5.5 (L/m)	6.1	1.2	6.1	1.4	5.7	1.4	0.602
CO 6 (L/m)	6.1	1.2	6.1	1.3	5.8	1.2	0.755
CO 6.5 (L/m)	5.9	1.1	5.9	1.3	5.7	1.2	0.801
CO 7 (L/m)	5.8	1.1	5.9	1.3	5.8	1.3	0.922
CO 7.5 (L/m)	5.8	1.2	5.7	1.1	5.8	1.4	0.960
CO 8 (L/m)	5.7	1.2	5.6	1.1	5.7	1.3	0.973

**10.6. Occurrence of pressor response**

Pressor response is presented with percentage, it is statistically significant between three groups, where its lower in group c than group B than group A. (Figure 2)

**11. Discussion**

In this study, we compared three doses of lidocaine for prophylaxis against pressor response of ETI and we found that the highest dose (2 mg/kg) is better than the two other doses. We found that the heart rate was comparable

**Table 6.** Systolic arterial blood pressure for the three groups. Data is presented as mean and standard deviation (SD). *Group A:* lidocaine dose 1 mg/kg, *Group B:* lidocaine dose 1.5 mg/kg, *Group C:* lidocaine dose 2 mg/kg. \*denotes significance between Group a and Group C.

	group						P value
	Group A (n=17)		Group B (n=17)		Group C (n=17)		
	Mean	SD	Mean	SD	Mean	SD	
Baseline SBP (mmHg)	126.4	17.6	126.2	10.6	120.6	15.9	0.442
SBP (mmHg)	127.4*	19.2	125.2	10.5	115	14.5	0.049*
SBP2 (mmHg)	122.8	19	119.5	15.2	115.5	19	0.493
SBP3 (mmHg)	117.1	17.5	113.1	11.9	110.2	17.7	0.462
SBP4 (mmHg)	139.9	18.2	132	19.6	125.9	24.8	0.164
SBP5 (mmHg)	136.7*	15.3	123.9	22.4	116.3	18.7	0.011*
SBP6 (mmHg)	127.5*	20.1	117.5	16.7	112.2	15.6	0.045*
SBP7 (mmHg)	122.5*	17.7	114.3	16.4	107.7	14.2	0.035*
SBP8 (mmHg)	119.8*	17.2	107.8	15.2	105.7	14.5	0.024*

**Table 7.** Diastolic arterial blood pressure for the three groups. Data is presented as mean and standard deviation (SD). *Group A:* lidocaine dose 1 mg/kg, *Group B:* lidocaine dose 1.5 mg/kg, *Group C:* lidocaine dose 2 mg/kg.

	group						P value
	Group A (n=17)		Group B (n=17)		Group C (n=17)		
	Mean	SD	Mean	SD	Mean	SD	
Baseline DBP (mmHg)	74.6	10	76	8.9	77.3	10.3	0.721
DBP1 min (mmHg)	75.9	12.2	75.8	8.4	69.7	7.2	0.102
DBP2 min (mmHg)	74.8	13.4	74.6	12.6	72.9	11	0.883
DBP3 min (mmHg)	70.4	13.1	71.5	12.6	69.8	12.3	0.918
DBP4 min (mmHg)	85.7	16.9	86.9	18.2	77.2	11.6	0.162
DBP5 min (mmHg)	79.1	12.8	77.8	17	70.4	12.3	0.170
DBP6 min (mmHg)	75.4	13.8	74.7	14.2	69	12.2	0.324
DBP7 min (mmHg)	71.3	12	71.1	13.3	66.4	11.9	0.439
DBP8 min (mmHg)	70.9	10.3	67.9	13	64.1	10.3	0.221

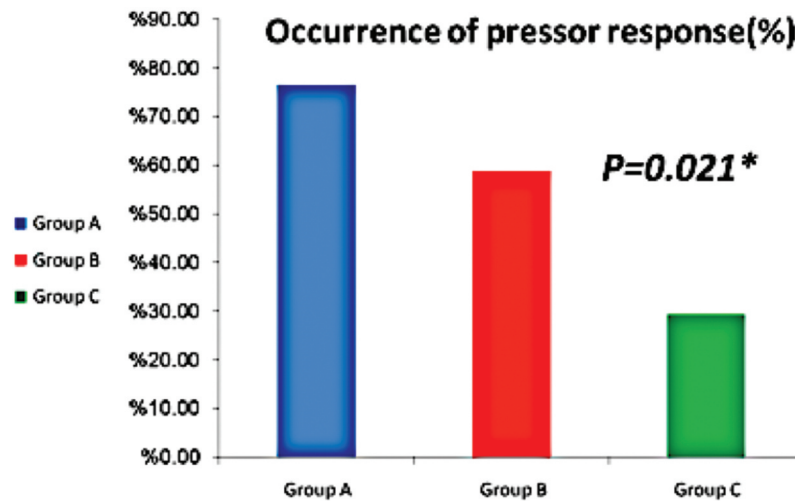
between the three groups; however, the incidence of pressor response was lowest in the 2 mg/kg group. The discrepancy between the heart rate and the incidence of the pressor response is explained by the broader definition of the pressor response which included any increase in the heart rate, blood pressure or cardiac output over 5 min.

Lidocaine is a multi-use drug in anesthesia and critical care. All local anesthetics exert their effect primarily by blocking voltage-gated sodium channels (VGSC) at the alpha-subunit inside the channel, which prevents sodium influx, subsequent depolarization, and action potential

generation [10]. This conduction block impedes pain transmission from neuronal cells to the cerebral cortex, thereby producing analgesia and anesthesia.

The use of lidocaine for preventing the pressor response to ETI had been previously reported in several studies and showed very good results compared to other agents such as magnesium, fentanyl, and beta adrenoceptor antagonists. A systematic review [11] supported the use of lidocaine in attenuation of the pressor response for ETI; however, the authors suggested that the optimum dose need further research. Previous reports had investigated different doses of lidocaine varying between 1 mg/





**Figure 2.** Occurrence of pressor response in percentage Group A: lidocaine dose 1mg/kg, Group B: lidocaine dose 1.5mg/kg, Group C: lidocaine dose 2mg/kg.

Kg to 2 mg/Kg; However, no studies to the best of our knowledge had compared different doses of lidocaine.

Gurulingappa, et al. in 2012 compared 75 patients who underwent general anaesthesia while receiving lignocaine (xylocaine), fentanyl, and a placebo to attenuate the cardiovascular response to direct laryngoscopy and intubation. They discovered that both lignocaine and fentanyl attenuated the pressor response. In comparison to lignocaine 1.5 mg/kg iv. bolus, fentanyl 4 microgram i. v. bolus offers a constant, dependable, and effective attenuation [12].

According to Sanjeev Singh et al., lidocaine 1.5 mg kg<sup>-1</sup> given two minutes before to intubation can successfully reduce the cardiovascular reactions to laryngoscopy and tracheal intubation. Esmolol 2 mg kg<sup>-1</sup> preventive treatment, when administered 2 minutes before to intubation, is considerably more effective than lidocaine at preventing hemodynamic alterations during tracheal intubation and laryngoscopy in normotensive black individuals. Further study is required to clarify the effects of various dosages of esmolol in the black population since dosage and time of medication administration are crucial elements that influence whether they will have a positive effect on laryngoscopy and tracheal intubation [13].

In 2017, Mendonça FT et al. investigated the effects of magnesium sulphate and lidocaine in reducing the hemodynamic response after tracheal intubation. The patients were divided into two groups; one got magnesium sulphate at a dose of 30 mg per kilogram, while the other received continuous infusions of lidocaine at a dose of 2 mg per kilogram just before to the induction of anaesthesia. At six different times related to the administration of the study drugs, blood pressure (BP), heart rate (HR), and bispectral index (BIS) were measured in both groups. It was discovered that magnesium sulphate and lidocaine have good efficacy and

safety for hemodynamic management in laryngoscopy and intubation [14].

Following laryngoscopy and intubation, there was an increase in HR and BP in both groups compared to baseline. After intubation, Group M's systolic and diastolic blood pressure levels increased statistically significantly, although this change had no clinical significance. The BIS values for the various groups were identical. Three (12%) of the individuals receiving magnesium sulphate experienced elevated blood pressure, compared to just one (4% of those receiving lidocaine, with no statistically significant difference [14].

Attenuating pressor response to endotracheal intubation has been an entity under thorough investigation, due to its impact on hemodynamic stability all through surgeries especially in high risk patients [9].

In selected populations, safety due to local anesthetic dosage could be compromised especially in extremes of age, end of organ failure, pregnancy and metabolic disturbances, thus it is desirable to use the least effective dose whenever possible [15].

## 12. Conclusion

Using Lidocaine in either of the three doses (1-, 1.5-, and 2 mg/Kg) produced comparable heart rate 1 min after ETI; however, the 2 mg/kg dose was associated with lower incidence of pressor response compared to the two lower doses.

## 13. Limitations

The study is a single-center study, including selected group of elective patients. Further studies are needed in other groups of patients and in emergency procedures. The study did not include a control group as we considered the use of lidocaine a standard-of-care practice

which should be provided to all patients, despite the lack of data about its best dose.

## Disclosure statement

No potential conflict of interest was reported by the authors.

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