Original Paper

ATTENUATION OF CARDIOVASCULAR RESPONSE DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION BY USING PETHIDINE WITH LIGNOCAINE

Mollick MT[1], Hossain MD[2], Ali NP[3]

Abstract

A prospective comparative study was done on lignocaine versus lignocaine with pethidine to observe the effect on cardiovascular response to laryngoscopy and endotracheal intubation. One hundred such elective surgical patients of active age group (16 - 60 years) having American Society of Anaesthesiologist (ASA) physical status I & II irrespective of surgical procedure were randomly assigned to one of the two groups of 50 each. Group I received injection lignocaine 1 mg/kg intravenously 02 minutes before induction of general anaesthesia. Patients in group II received injection pethidine 1 mg/kg body weight and injection lignocaine 1 mg/kg body weight intravenously 02 minutes before induction of general anaesthesia. Haemodynamic parameter i.e. blood pressures (systolic blood pressure, diastolic blood pressure and mean blood pressure), heart rate, rate pressure product were monitored after 1st, 3rd, 5th minutes following intubation. There were statistically significant ($p<0.001$) increase in blood pressures, heart rate and rate pressure product in group I i.e. pretreatment with 1 mg/kg body weight intravenous lignocaine and remained so after 5 minutes. On the other hand there were no statistically significant ($p>0.05$) increase in heart rate, blood pressures and rate pressure product in group II, where pre-treatment done with pethidine 1 mg/kg body weight with lignocaine 1 mg/kg body weight and the values returned control level before 5 minutes. The study showed that pethidine 1 mg/kg body weight with lignocaine 1 mg/kg body weight pretreatment suppresses the cardiovascular response due to laryngoscopy and intubation.

Key words : **Cardiovascular response, laryngoscopy, lignocaine with pethidine.**

Introduction

The stress response to laryngoscopy and endotracheal intubation activates the sympathetic nervous system, which may increase myocardial oxygen demand by increasing heart rate and arterial blood pressure. Activation of the sympathetic nervous system may also cause coronary artery vasoconstriction reducing the supply of oxygen to the myocardium, which in turn would predispose to myocardial ischaemia. Of course intubations using Glide Scope videolaryngoscope causes lesser stress response in comparison to intubation with a Macintosh laryngoscope[1,2].

Intravenous and inhalational anaesthetic agents have no appreciable effects on stress response. Lignocaine has some proven effect on attenuation of stress response during laryngoscopy and endotracheal intubation[3,4]. Various other methods have also been tried by different authos[5,6]. High dose opioid analgesia may completely inhibit the stress response. These high doses of opioids are impractical. In this study low dose of lignocaine and low dose opioid was used to observe the cardiovascular response after laryngoscopy and endotracheal intubation.

Materials and Methods

One hundred patients of both the sex had been selected between the ages of 16-60 years having American Society of Anaesthesiologist (ASA) physical status I & II without any cardiovascular disease. They were divided into group I and group II irrespective of their age, sex and type of surgery. Both groups were treated with tablet diazepam 0.15 mg/kg body weight orally at night (2200 hours) before operation. After arrival into the operating room cardiovascular parameter of all the patients were recorded. The cardiovascular parameters were heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean blood pressure (MBP) and rate pressure product (RPP) and were continuously recorded by the non invasive cardiac monitor. The cardiovascular parameter also recorded during pre-oxygenation and before induction of anaesthesia as control value.

To the patients of group I, intravenous (IV) lignocaine 1 mg/kg body weight was administered 02 minutes before induction with thiopentone 5 mg/kg body weight and suxamethonium 1.5 mg/kg body weight. Intubation condition was assessed clinically and by neuromuscular stimulator by observing train of four ratio. Then laryngoscopy and endotracheal intubation were done. Subsequently, patient had been ventilated with oxygen (O_2), Nitrous oxide (N_2O) and halothane. The

1. Lt Col Md Torab Mollick MBBS, FCPS, CMH, Jessore; 2. Lt Col Md Delwar Hossain MBBS, FCPS, CMH Dhaka; 3. Maj Nadeem Parvez Ali MBBS, DA, FCPS, Graded Specialist in Anaesthesiology, CMH, Bogra.

laryngoscopy and endotracheal intubation were done within 20 seconds. The cardiovascular parameters were recorded on 1st, 3rd, 5th minute after laryngoscopy and endotracheal intubation. In case of group II patients same procedure was followed except that pethidine 1 mg/kg body weight was administered in addition to lignocaine.

Results were reported as mean ± standard deviation (SD) and data were compiled in tabulated form. Unpaired Student's 't' test was done as applicable.

Results

In both the groups, the cardiovascular parameter that is heart rate, systolic blood pressure, diastolic blood pressure and mean blood pressure increased significantly after 1 minute of endotracheal intubation. But increase was relatively less in group II ($p<0.001$). In group I the parameter did not return to control level after 5 minute of endotracheal intubation ($p<0.05$ or <0.001). But in group II parameters return to close to control level after 5 minutes of endotracheal intubation and in all cases mean value fall below the control value ($p<0.01$ or <0.001) remaining within normal range.

Table-I : Comparative changed in heart rate (number/minute) between group I and II.

Group of Patients	Control Mean ± SD	After 1 minute Mean ± SD	After 3 minute Mean ± SD	After5 minute Mean ± SD	Control vs after 5 min
Group –I (n=50)	89.1 ± 8.2	107.2 ± 10.05	103 ±9.5	99.2 ±8.6	p <0.001
Group –II (n=50)	88 ± 7.39	91 ± 7.6	85.5 ± 6.8	83.6 ± 3.76	p <0.01
p value	p > 0.05	p <0.001	p <0.001	p <0.001	

Table-II: Comparative changed in Systolic blood pressure (in mm of Hg) between group I and II.

Group of Patients	Control Mean ± SD	After 1 minute Mean ± SD	After 3 minute Mean ± SD	After 5 minute Mean ± SD	Control vs after 5 min
Group –I (n=50)	119.5 ± 6.7	137 ± 6.3	131 ± 4.9	124.2 ± 3.32	p <0.01
Group –II (n=50)	122.1 ± 5.57	125.6 ± 4.6	121.2 ± 2.86	116 ± 2.35	p <0.001
	p > 0.05	p <0.001	p <0.001	p <0.001	

Table-III : Comparative changed in diastolic blood pressure (in mm of Hg) between group I and II.

Group of Patients	Control Mean± SD	After 1 minute Mean ± SD	After 3 minutes Mean ± SD	After 5 minutes Mean ± SD	Control vs after 5 min
Group-I (n=50)	77.5 ± 4.9	89.5 ± 4.6	84.2 ± 5.3	81.2 ± 3.5	p <0.01
Group-II (n=50)	78.5 ± 3.8	81.3 ± 3.3	79.5 ± 3.6	75.8 ± 2.7	p <0.01
	p > 0.05	p <0.001	p <0.001	p <0.001	

Table-IV : Comparative changed in mean blood pressure (in mm of Hg) between group I & II.

Group of Patients	Control Mean ± SD	After1 minutes Mean ± SD	After 3 minutes Mean ± SD	After 5 minutes Mean ± SD
Group-I (n=50)	91.98 ± 5.8	104.8 ± 7.2	100.3 ± 6.6	95.2 ± 5.3
Group-II (n=50)	92.8 ± 6.12	96.2 ± 6.21	91.7 ± 5.3	87.5 ± 3.9
	p > 0.05	p <0.001	p <0.001	p <0.001

Discussion

Laryngoscopy and endotracheal intubation can cause striking changes in haemodynamics and intracranial pressure probably as a result of intense sympathetic nervous system stimulation[2]. In patients who are at risk of developing increased intracranial pressure, arterial hypertension, myocardial ischemia, these changes may be life threatening. They may lead to cerebral hemorrhage, left ventricular failure and life threatening cardiac arrhythmias. Various techniques were tried to attenuate these cardio-vascular responses[5,6]. One of them is deep inhalational anaesthesia which may cause intracranial hypertension. Administration of a large dose of thiopental sodium can effectively prevent arterial and intracranial hypertension, but in this case there is a risk of severe cardiac depression. Potent vasoactive drugs need larger doses to attenuate arterial blood pressure and fail to prevent tachycardia caused by laryngoscopy and intubation[6]. Vasoactive drugs cause cerebral hypertension. Some of them cause hypertension with reflex tachycardia and others depress the myocardium severely in patients with preexisting left ventricular dysfunction or those receiving beta-adrenergic antagonists. These effects are not desirable and limit their usefulness.

Various studies have shown that intravenous lignocaine is effective in preventing or attenuating the arterial hypertension and tachycardia in response to endotracheal intubation[7]. A few publications have shown the lack of effect of intravenous lignocaine on haemodynamic response[8,9]. Kobayashi carried out a randomized open study on 36 ASA I & II adult surgical patients to assess the effect of intravenous lignocaine and fentanyl on circulatory responses to laryngoscopy and tracheal intubation[10]. The three groups included: group-L (intravenous lignocaine 1.5 mg/kg 2 minute before laryngoscopy), group-F (intravenous fentanyl 4 mierogram/kg) and group-C (no treatment). Before induction anaesthesia, there were no significant differences among the three groups in mean arterial pressure (MAP), heart rate (HR) and rate pressure product (RPP). After laryngoscopy and intubation, the three haemodynamic variables increase significantly from control values in group-L and group-C. In group-F, these haemodynamic variables showed no significant charges after laryngoscopy and intubation and were significantly less than those in group-L and group-C. Intubation conditions were better in the fentanyl group than in the lignocaine group.

Fujii carried out a study on sixty hypertensive patients of ASA II under going elective surgery received in a randomized, double blind manner, 0.3 mg/kg diltiazem, 1.5 mg/kg lignocaine or 0.3 mg/kg diltiazem plus 1.5 mg/kg lignocaine intravenously (n=20 of each) before the initiation of laryngoscopy[11]. Anaesthesia was induced with 5 mg/kg thiopentone IV, and tracheal intubation was facilitated with 2 mg/kg succinylcholine IV after precurarizaion with 0.02 mg/kg vecuroneum IV. Changes in heart rate (HR), mean arterial pressure (MAP) and rate pressure product (RPP) were measured before and at immediate, 1, 2, 3, 5 and 10 minute after tracheal intubation. The inhibitory effects of diltiazem-lignocaine combination on cardiovascular response to tracheal intubation was greater than those of diltiazem or lignocaine [RPP; 10602 ± 1448 (combination) versus 11787 ± 1345 (diltiazem), 15428 ± 1756 (lignocaine), $p<0.05$].

To compare the safety and efficacy of lignocaine, esmolol and nitroglycerin in modifying the heamodynamic response to laryngoscopy and intubation, Singh[12] carried out a randomized, placebo controlled double blind study. 40 ASA plysical status I and II patients were selected under going elective surgery with general endotracheal anaesthesia. Anasthesia was induced with thiopental sodium 5 mg/kg and intubation was facilitated with vecuroneum 0.15 mg/kg. In addition, patients received one of the following four study drugs I.V prior to laryngoscopy: Group-I (control= saline 5 ml; group-II = lignocaine 1.5 mg/kg ; group-III = esmolol 1.4 mg/kg; group-IV nitroglycerin 2 microgram / kg.

Mean arterial pressure and heart rate were recorded every minute for 20 minutes following induction of anaesthesia. MAP increase significantly in all four treatment groups (control 49% ± 19%, lignocaine 55% ± 26 %, esmolol 25% ± 11%, nitroglycerin 45% ± 21%) compared with pre-induction baseline values. In the esmolol pretreatment patients, the increase in HR was significantly lower (20% ± 3%) compared with the nitroglycerin (37% ± 8%), lignocaine (52% ± 8%) and control (29% ± 4%) groups.

Safavi and Honarmand carried out a study on 60 patients of ASA-I and ASA-II under going elective abdominal surgery[13]. They divided the patients group-p who received pethidine 1.5 mg/kg and group-S received sufentanil 0.1 micro gm/kg. General anaesthesia induced 60 Seconds later by thiopental 4 mg/kg. Atracurium 0.6 mg/kg was given as an intravenous bolus to facilitate tracheal intubation, which was performed 5 minutes after induction. Patients lungs were ventilated for 4 minutes with 100% oxygen before tracheal intubation. Laryngoscopy was carried out at peak effects of pethidine and sufentanil (at 6th minute after injection) and tracheal intubation was accomplished within 30 seconds. The patient lungs were then mechanically ventilated with a tidal volume of 10 ml/kg and respiratory rate of 12 / minute to maintain end-tidal $PaCO_2$ at around 38 mmHg. Anaesthesia was maintained with isoflurane 1.2% and 50% nitrous oxide in oxygen. In each patient, the BP (SAP, DAP, map) and HR were measured at three time points. Baseline (3 minute before induction), pre-intubation (after induction of Anaesthesia) and Post intubation (1st, 3rd, 5th and 7th minute after start of laryngoscopy). Result of this prospective, randomized, double blind study demonstrated that adequate timing in opioid administration is warranted. The time of peak effect of each opioid drug and small doses of sufentanil or pethidine provide similar effective control of the inotropic response induced by laryngoscopy and tracheal intubation.

Conclusion

In this study maximum attenuating effects was observed by intravenous lignocaine with pethidine on cardiovascular system in response to laryngoscopy and endotracheal intubation. It was also observed that intravenous lignocaine with pethidine did attenuate the sympathetic responses to laryngoscopy and endotracheal intubation which came down to base line before 5 minute after intubation. But the group of patients which was treated only with lignocaine, their sympathetic responses did not come down to base line at 5 minute after laryngoscopy and endotracheal intubation.

References

1. Jakuoenko N, Kopeika U, Mihelsons M, Nagobade D, Putnioa AV, Pavârs A. Comparison Of Stress Response Performing Endotracheal Intubation by Direct Laryngoscopy, Fibreoptic Intubation and Intubation by the Glidescope Laryngoscope. Proceedings of the Latvian Academy of Sciences 2008; 62 (4/5) Section B: 176-181.
2. Shribman AJ. Smith G. Achola KJ. Cardiovscular and catecholmine response to laryngoscopy with and without tracheal intubation. British Journal of Anaesthesia 1986; 59:295-299.
3. You Mi Ki Y, Kim NS, Lim SH, Kong MH, Kim HZ. The Effect of Lidocaine Spray before Endotracheal Intubation on the Incidence of Cough and Hemodynamics during Emergence in Children. Korean J Anesthesiol 2007 November; 53(3): S1-S6.
4 Gupta A, Wakhloo R, Gupta V, Mehta A, Kapoor BB. Comparison of Esmolol and Lignocaine for Atttenuation of Cardiovascular Stress response to Laryngoscopy and Endotracheal Intubation. JK Science 2009 April - June; 11 (2): 78-81.
5. Safavi M, Honarmand A. Attenuation of Cardiovascular Responses to Laryngoscopy and Tracheal Intubation - Intravenous Sufentanil vs Pethidine. Middle East J Anesthesiol 2008 Oct; 19(6): 1349-59.
6. Fassoulaki A, Melemeni A, Paraskeva A, Petropoulos G. Gabapentin attenuates the pressor response to direct laryngoscopy and tracheal intubation. British Journal of Anaesthesia 2006; 96(6): 769-773.
7. Samaha T; Ravussin P; Claquin C; Ecoffey C: Prevention of increase blood pressure and intracanial pressure during endotracheal intubation in neurosurgery and sure during endotracheal intubation in neurosurgery and surgery : esmolol verus lodocaine. Ann Fr Anaesth Reanim. 1996; 15 (1) : 36-40.
8. Jorgemsen BC, Hoilund-Carlsen PF, Marving J, Christensen V. Lack of effect of intravenous lidocaine on haemodynamic responses to rapid sequence induction of general anesthesia. A double blind controlled clinical trial. Anesth Analg 1986; 65: 1037-41.
9. Laurito CE, Baughman VL, Polex VV, Riegier FX, Vadeboncouer TR. Aerosolized and intravenous lidocaine are no more effective than placebo for the control haemodynamic responses to intubation. Anesthesiology 1987; 67 (3A). ?
10. Kobayashi TL, Watanabe K, Ito T. Lack of effect of I.V. lignocaine on cardiovascular responses to laryngoscopy and intubation. Masui 1995; 44 (4): 579-82.
11. Fujii Y, Saitoh Y, Takahashi S, Toyooka H.Diltiazem lignocaine combination for the attenuation of cardiovascular responses to tracheal intubation in hypertensive patients. Can J Anaesth 1998; 45 (10) : 933-7.
12. Singh H, Vichitvejpaisal P, Gaines GY, White PF. Comparative effects of lignocaine, esmolol, and nitroglycerin in modifying the hemodynamic response to laryngoscopy and intubation. J Clin Anesth. 1995; 7(1): 5-8.
13. Safavi MR, Honarmand A. Attenuation of Cardiovascular response to laryngoscopy and tracheal intubation. M E J Anesth 2008; 19(6): 1349-59.