ISSN:0975-3583,0976-2833 VOL13, ISSUE05, 2022

ORIGINAL RESEARCH ARTICLE

COMPARISON OF CHANGES IN MEAN ARTERIAL PRESSURE BETWEEN LIGNOCAINE AND FENTANYL: LARYNGOSCOPY AND INTUBATION

[1]Dr.Dinesh M,[2]Dr.Mahalingappa,[3]Dr.Goolappa M Chikkanargund,[4]Dr.Deepak Dhummansure

[1]Resident, Narayana Hrudayalaya, Bangalore, Karnataka, India

[2]Resident, Rural Development Trust Hospital, Bathalapalli, Ananthapuramu, Andhra Pradesh

[3]Resident, Meenakshi Mission Hospital and Research Centre, Madurai, Tamil Nadu, India

[4]ESIC Medical College and Hospital, Kalaburagi, Karnataka, India

Corresponding Author:

Dr.Deepak Dhummansure

Abstract

Different studies have shown rise of mean blood pressure of 25-35 mmHg during laryngoscopy and intubation when compared with pre-intubation valuesand elevation of plasma noradrenaline and adrenaline by 45% and 40% respectively. A correlation between changes in mean arterial pressure and noradrenaline and pulse pressure and heart rate and adrenaline is found. A clinical comparative prospective study of attenuation of sympatheticresponse to laryngoscopy and intubation was done in 150 patientsposted for elective surgeries. General anaesthesia was provided withendotracheal intubation for all the patients.Patients undergoing various Orthopaedic, Ear, Nose and Throat surgeries, Gynaecological, Neurosurgical and Laparoscopic procedures were selected. A significant difference was seen in all the groups at 1, 3, 5 and 7 minutes interval ($p<0.01$). Attenuation of pressure response by lignocaine when compared with control group was significant at 1, 3, 5 and 7 minutes interval ($p<0.01$). The maximum rise was 13.5% in lignocaine group where as it was 18.8% in control group at 1 minute interval.

Keywords: Mean arterial pressure, lignocaine, fentanyl

Introduction

Laryngoscopy and tracheal intubation are frequently associated with sympathetic response. Diagnostic laryngoscopy under anaesthesiaand tracheal suctioningare also associated with adverse regulatory changes. Severe hypertension, tachycardia, increase in intracranial pressurecan also be seen[1].

Supraglottic traction during laryngoscopy or superficial stimulation of airway or passage of tracheal tube into trachea may be associated with reflex sympathetic changes.

Other contributory factors to hypertension and tachycardia like anxiety, baroreceptor

ISSN:0975-3583,0976-2833 VOL13, ISSUE05, 2022

mediated reflex after induction etc are less important than laryngotracheal stimulation.The tracheal intubation following laryngoscopy is not only accompanied by increased sympathetic activity but also increased sympathoadrenal activity. Increased hypothalamic activity and increased traffic in sympathetic efferent tracts are observed. Release of trophic hormones from hypothalamus can stimulate release of ACTH, TSH, GH, FSH, LH and prolactin in addition to ADH from the pituitary gland[2, 3].

Afferent impulses are carried through trigeminal, glossopharyngeal, vagus and sympathetic nerve from the airway. These impulses are relayed in cranial nerve nuclei, vasomotor and autonomic regulatory areas[4].

Key areas that integrate cardiovascular responses and maintain cardiovascular homeostasis are nucleus tractus solitarius, dorsal vagal nucleus, nucleus ambiguus and parabrachial nucleus. The nucleus solitarius is the area of primary central synapse for baroreceptor mediated reflexes and relay station for peripheral information to hypothalamic sympathetic control centres. It projects directly to intermediolateral nucleus of the spinal cord, the common pathway for pre-ganglionic sympathetic outflow. Hypothalamus along with nucleus ambiguus plays an important role in control of secretion of vasopressin[5].

Increase in sympathetic and hypothalamo pituitary adrenal activity is responsible for cardiovascular changes seen with laryngoscopy and tracheal intubation.

Different studies have shown rise of mean blood pressure of 25-35 mmHg during laryngoscopy and intubation when compared with pre-intubation valuesand elevation of plasma noradrenaline and adrenaline by 45% and 40% respectively. A correlation between changes in mean arterial pressure and noradrenaline and pulse pressure and heart rate and adrenaline is found[6].

Methodology

A clinical comparative prospective study of attenuation of sympatheticresponse to laryngoscopy and intubation was done in 150 patientsposted for elective surgeries. General anaesthesia was provided withendotracheal intubation for all the patients.

Patients undergoing various Orthopaedic, Ear, Nose and Throat surgeries, Gynaecological, Neurosurgical and Laparoscopic procedures were selected. Following criteria's were adopted for selecting patients.

Inclusion criteria

- Patients scheduled for elective surgeries.
- Age between 20 to 50 years of both the sexes.
- Patients with ASA class I and II.
- Mallampati airway assessment of class I and II.

Exclusion criteria

- Unwilling patients.
- Emergency surgeries.
- Anticipated difficult airway.
- Patients with ASA class III or higher.
- Patients with asthma, hypertension and other cardiovascular diseases.
- Patients on antihypertensive medications like beta blockers, calcium channel

ISSN:0975-3583,0976-2833 VOL13, ISSUE05, 2022

blockers and angiotensin converting enzyme inhibitors.

- Patients in whom laryngoscopy and intubation proved to be prolonged (more than 20 seconds) or difficult.

Patients were selected after thorough pre-anaesthetic assessment and investigations. An informed consent was taken with all the patients.

150 cases were divided into three groups of 50 each by *double blind randomization using a chit method.*

Group-I was Control group: In this group no drug was administered for attenuating sympathetic response to laryngoscopy and intubation.

Group-II was Lignocaine group: Here patients received 1.5 mg/kg of lignocaine intravenously 3 minutes before laryngoscopy and intubation.

Group-III was Fentanyl group: All the patients in this group received 2 µg/kg of fentanyl intravenously 5 minutes before laryngoscopy and intubation.

Results

Table 1: Comparison of Changes in Mean Arterial Pressure

Time of Assessment	**Control (I)**		**Lignocaine (II)**		**Fentanyl (III)**		**Anova 'F'* p**	**Diff. between groups****		
	Mean ± SD	**% diff**	**Mean ± SD**	**% diff**	**Mean ± SD**	**% diff**		**I-II**	**I-III**	**II-III**
Pre-induction	94.3 ± 6.7	-	95.1 ± 6.5	-	93.6 ± 5.9	-	0.760.47	0.54	0.58	0.23
Post induction	92.2 ± 7.0	-2.2	94.2 ± 6.1	-1.0	92.4 ± 5.7	-1.3	1.450.24	0.13	0.87	0.30
1 min	112.0 ± 6.2	18.8	107.9 ± 70	13.5	99.5 ± 5.2	6.3	53.4 <0.001	<0.01	<0.001	<0.001
3 min	111.3 ± 6.2	18.0	106.1 ± 7.3	11.6	98.3 ± 5.5	5.0	53.1 <0.001	<0.001	<0.001	<0.001
5 min	104.3 ± 6.9	10.6	98.8 ± 5.2	3.9	94.0 ± 5.2	0.4	39.2 <0.001	<0.001	<0.001	<0.001
7 min	97.7 ± 6.4	3.6	94.2 ± 5.4	-1.0	91.8 ± 5.2	-1.9	13.7 <0.001	<0.01	<0.001	<0.05
10 min	94.4 ± 5.9	0.1	92.9 ± 5.5	-2.3	90.9 ± 6.2	-2.9	4.33 <0.05	0.20	<0.01	0.10

-ve sign indicates decrease, *one-way ANOVA, **Unpaired 't' test, $p<0.05$ is significant, $p<0.001$ is highly significant, $p>0.05$ is not significant.

ISSN:0975-3583,0976-2833 VOL13, ISSUE05, 2022

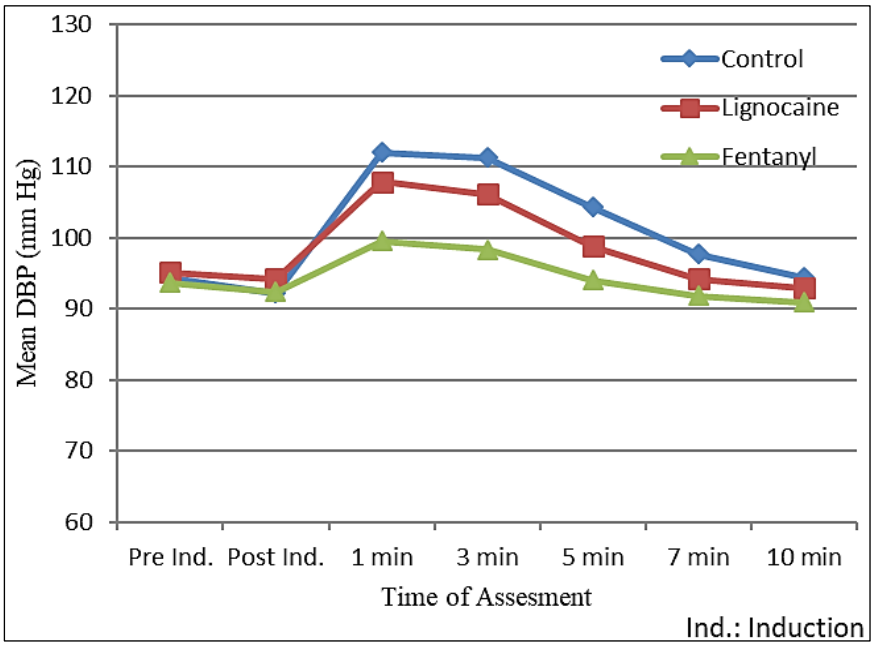

Fig 1: Mean Arterial Pressure

The changes in mean arterial pressure assessed at pre and post induction and at various time intervals from the onset of laryngoscopy and intubation in control, lignocaine and fentanyl groups.

Control group

The pre-induction mean arterial pressure was 94.3 ± 6.7. It showed 2.2% decrease to 92.2 ± 7.0 after induction of anaesthesia. It increased by 18.8% above preinduction level to 112.0 ± 6.2 at 1 minute after the onset of laryngoscopy and intubation, which was highly significant ($p<0.001$). It remained at similar level at the end of 3 minutes. It decreased in subsequent intervals and was almost equal to basal level at 10 minutes.

Lignocaine group

The pre-induction mean arterial pressure was 95.1 ± 6.5. With induction of anaesthesia it decreased by 1% below basal value to 94.2 ± 6.1. A maximum rise of mean arterial pressure was found at 1 minute from the onset of laryngoscopy and intubation, to 107.9 ±7.0 (13.5% above preinduction level). It further reduced to 106.1 ± 7.3 (11.6% above preinduction level) at 3 minutes and 98.8 ± 5.2 (3.9% above preinduction level) at 5 minutes. A small decrease below preinduction values was seen at 7 and 10 minutes.

Fentanyl group

The pre-induction mean arterial pressure in this group was 93.6 ± 5.9. A small decrease below basal value of 1.3% (92.4 ± 5.7) was seen with induction of anaesthesia. The maximum rise in mean arterial pressure was at 1 minute to 99.5 ± 5.2 (6.3% above preinduction level). At 3 minutes it was 98.3 ± 5.5 (5.0% above preinduction level). Subsequently it reached near basal values after 5 minutes. A small decrease below basal value was found at 7 and 10 minutes.

One-way ANOVA shows no significant difference among three groups before and after induction ($p=0.47$ and $p=0.24$).

ISSN:0975-3583,0976-2833 VOL13, ISSUE05, 2022

A significant difference was seen in all the groups at 1, 3, 5- and 7-minutes interval ($p<0.01$).

Attenuation of pressure response by lignocaine when compared with control group was significant at 1, 3, 5 and 7 minutes interval ($p<0.01$). The maximum rise was 13.5% in lignocaine group where as it was 18.8% in control group at 1 minute interval.

When compared with control group, fentanyl group was highly significant in attenuating the pressure response ($p<0.001$). i.e. The fentanyl group shows only 6.3% increase above basal value while control group shows 18.8% increase beyond basal value at 1 minute interval.

Among lignocaine and fentanyl groups, fentanyl group was highly significant in attenuating the pressure response. Lignocaine group shows 13.5% increase in mean arterial pressure, but fentanyl group shows only 6.3% increase ($P<0.001$).

Discussion

The laryngotracheal stimulation is known to cause reflex sympatho adrenal response with marked increase in heart rate and blood pressure. Arrhythmias can be precipitated. The harmful nature of this response has been noted in patients at risk. Various techniques and drugs have been advocated to decrease the haemodynamic response and none of them is totally satisfactory. Hence there is a need to find a simple and satisfactory method[7, 8].

The present clinical comparative study was done in 150 normotensive, ASA grade I and II patients scheduled for various elective surgical procedures under general anaesthesia with endotracheal intubation. The objectives of this study were to observe the variations of sympathetic response to laryngoscopy and intubation in control group and to study the effectiveness of lignocaine and fentanyl in attenuating this sympathetic response and to ascertain the superiority of one drug over the other.

150 cases were divided into three groups of 50 each by double blind randomization using a chit method.

Group-I control group. These patients did not receive any drug for attenuation of sympathetic response.In group II lignocaine was administered at a dose of 1.5 mg/kg intravenously 3 minutes before laryngoscopy and intubation. In group III fentanyl 2 µg/kg was given intravenously 5 minutes before laryngoscopy and intubation.

In all study groups, patients received Diazepam 10 mg orally at night on the previous day before surgery. On the day of surgery, Tablet Diazepam 5 mg and Tablet Pantoprazole 40 mg was given with a sip of water at early morning.

On shifting the patient to operation theatre, 18 gauge intravenous cannula was secured and injection Midazolam 1 mg intravenously was administered 15 minutes before induction. A pre induction heart rate, systolic and diastolic blood pressures were recorded.

The anaesthesia was induced with injection Propofol 2 mg/kg intravenous, Vecuronium was used for muscle relaxation at a dose of 0.1 mg/kg intravenous. Laryngoscopy and intubation took maximum 20 seconds in all the cases. Anaesthesia was maintained with air oxygen mixture, maintaining FiO_2 of 0.4, Isoflurane 0.6% to 1% and ventilated with IPPV using a circle system.

The haemodynamic parameters recorded include heart rate, systolic, diastolic and mean arterial blood pressures at pre-induction, post induction and at 1, 3, 5, 7 and 10 minutes

ISSN:0975-3583,0976-2833 VOL13, ISSUE05, 2022

intervals from the onset of laryngoscopy and intubation.

Khan RM *et al*. demonstrated an average rise in mean arterial pressure of 25 mmHg[9]. An increase in mean arterial pressure up to 40-50 mmHg when compared with pre inductionvalues, after placement of an endotrachealtube was noted in some individuals. A rise in heart rate of upto 30 beats/min was also noticed in some patients[9].

Many factors like age, drugs, type and depth of anaesthesia, hypoxia, hypercarbia etc., affects the cardiovascular changes associated with laryngoscopy and intubation that influence the haemodynamics[10].

Conclusion

- One way ANOVA shows no significant difference in MAP among three groups before and after induction ($p=0.47$ and $p=0.24$).
- A significant difference was seen in all the groups at 1, 3, 5 and 7 minutes interval ($p<0.01$).

References

1. Pernerstorfer T, Kraffit P, Fitzgerald RD, *et al*. Stress response to tracheal intubation; direct laryngoscopy compared with blind oral intubation. Anaesthesia. 1995;50:17-22.
2. Feng CK, Chank H, Liu KN, Lee TY. A comparison of lidocaine, fentanyl and esmolol for attenuation of cardiovascular response to laryngoscopy and tracheal intubation. Acta AnaesthesioSin. 1996Sep;34(3):172.
3. Adachi YU, Sotomoto M, Higuchi H,Watanabe K. Fentanyl attenuate the haemodynamic response to endotracheal intubation more than the response to laryngoscopy. Anesth Analg. 2002July;95(1):233-37.
4. Singh S, Smith JE. Cardiovascular changes after the 3 stages of nasotracheal intubation. Br J Anaesth. 2003Nov;91(5):667-71.
5. Williams, Warwick, Gray's Anatomy. 36th Edn. Edinburgh: Churchill Livingstone, 1984.
6. Ronald D Miller. Anesthesia, Philadelphia: 5th Edn.Churchill Livingstone, 2000, 1-2.
7. Robert K. Stoelting, Pharmacology and physiology in anesthetic practice, 3rd Edn. Philadelphia: Lippincott-Raven, 1999.
8. Scott DB, Jebson PJR, Braid DP, Ortengren, and Frisch P. Factors affecting plasma levels of lignocaine and prilocaine. Br J Anaesth. 1972;44:1040-49.
9. Khan RM, KhanTZ,Iqbal Ahmed. Nifedipine and attenuation of blood pressure and pulse rate changes in response to laryngoscopy and tracheal intubation. Ind J Anaesth. 1987;35(5):346-49.
10. Curran J, Crowley M, O'Sullivan G. Droperidol and endotracheal intubation. Attenuation of pressure response to laryngoscopy and intubation. Anaesthesia. 1980;35:290-94.