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COMPARISON OF LIGNOCAINE AND FENTANYL FOR ATTENUATION OF CARDIOVASCULAR STRESS RESPONSE TO LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION

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ABSTRACT
BACKGROUND AND AIM
Intravenous Fentanyl Citrate and Lignocaine hydrochloride is being used as premedication for general anaesthesia with an intent to attenuate cardiovascular reflexes of laryngoscopy and endotracheal intubation. Present study was aimed to compare efficacy of both drugs as premedication to attenuate cardiovascular reflexes of laryngoscopy.
METHODOLOGY
Present study was carried out on randomly selected 50 patients of ASA Grade 1 and 2, aged 20 to 50 years with mallam pati airway assessment grade 1 scheduled for elective surgery requiring general anaesthesia with endotracheal intubation. All routine non-invasive monitors were applied and iv line secured. Glycopyrrolate 0.004 mg/kg and injection midazolam 0.02 mg/kg was given intravenously and IV infusion of ringer lactate was started.
Patients were divided in two groups.
Group-1: -Lignocaine hydrochloride group. Here patients received injection lignocaine hydrochloride 1.5 mg / kg IV bolus 3 minutes prior to induction.
Group-2: -Fentanyl citrate group. Here patients received injection fentanyl citrate 2 µg / kg IV bolus 3 minutes prior to induction.
After preoxygenation general anaesthesia with Thiopentone Sodium 5mg/kg and Succinyl choline 2mg/kg was given and endotracheal intubation was done. Heart rate, SBP, DBP, MAP and ECG were recorded in all patients pre operatively, after premedication, before study drug, after induction, after laryngoscopy and intubation and for every 1 minute up to 6 minutes after intubation during which no stimulus was given to patients. Laryngoscopy done and endotracheal intubation was done.
An observation was made related to adverse effects of drugs and laryngoscopy related problems and were attended to appropriately.
RESULTS
In our study, injection fentanyl citrate 2 µg/kg attenuated the haemodynamic response better than Lignocaine 1.5 mg/kg. Heart rate, systolic blood pressure, diastolic blood presuure and mean arterial pressure did not rise significantly after intubation with Fentanyl compared to Lignocaine and reached basal value within study time.
CONCLUSION
Fentanyl citrate is safe and effective in attenuation of hemodynamic response to laryngoscopy without significant side effects as compared to lignocaine hydrochloride.

KEYWORDS: LIGNOCAINE, FENTANYL, ATTENUATION, CARDIOVASCULAR

INTRODUCTION

General anaesthesia is a medically induced coma with loss of consciousness and protective reflexes. A variety of medications can be administered, with the overall aim of sleep, amnesia, analgesia, relaxation of skeletal muscles and loss of control of reflexes of the autonomic nervous system. Airway maintenance is essential following induction of anaesthesia, as nearly all general anaesthetics reduce or eliminate both the ventilator drive and the reflexes that maintain the airway patency. Stimulation of upper airway is associated with a reflex increase in

Sympathetic activity and increased catecholamine secretion. This sympathetic stimulation results in tachycardia and elevation of blood pressure.

In normotensives, this rise is 20 to 25 mmHg. Rise in arterial pressure begins after about 15 seconds and peaks approximately within 30-45 seconds after laryngoscopy. It is associated with significant increase in heart rate during endotracheal intubation. However, it returns to baseline within 5 to 10 minutes after intubation.

They may have detrimental effects in high-risk patients, especially those with cardiovascular diseases, increased intracranial pressure or anomalies of cerebral vessels. Although the response may be transient, it is invariable, significant, often persistent, and of great concern.

Many drugs and techniques have been used to prevent the hemodynamic responses induced by laryngoscopy and endotracheal intubation like

- Block of the peripheral sensory receptors and afferent input- topical application and infiltration of superior laryngeal nerve.
- Block of the central mechanisms of integration of sensory input- fentanyl, morphine, droperidol, etc.
- Block of the efferent pathway and effector sites – Intravenous Lignocaine, BetaBlockers, Calcium Channel Blockers, and Hydralazine etc.

The present study is undertaken to determine the efficacy of bolus doses of injection Lignocaine hydrochloride1.5 mg/kg and injection Fentanyl citrate 2μg/kg intravenous before induction in attenuating the sympathetic response to laryngoscopy and tracheal intubation.

METHODOLOGY

A prospective, randomized double blind comparative study was carried out. Study was carried out on randomly selected 50 patients of ASA Grade 1 and 2, aged 20 to 50 years with mallam pati airway assessment grade 1 scheduled for elective surgery requiring general anaesthesia with endotracheal intubation. On the day before surgery, all the patients were examined thoroughly and investigated. After proper preanaesthetic counseling, a written and informed consent was taken.

Exclusion criteria were_Patients with history of known allergies to study drugs, Emergency Surgeries,_Anticipated difficult intubation, Patients with ASA Grade III or higher, Patients on beta blockers or Calcium Channel blockers or sympatholytic drugs, Active seizures.

Pre-Anaesthetic evaluation of all the patients consisted of detailed history, general and systemic examination, routine investigations.

All patients were kept nil by mouth for 6 hours. Vital signs noted in preoperative room were considered as control baseline values. Written and informed consent was taken in all patients. On the day of surgery in pre-operative room, pulse rate, SBP, DBP were recorded. On entering the operation theatre, IV line was secured with 18 G IV Cannula, and Pulse Oximeter, non-invasive BP, ECG monitor were applied. Heart rate, SBP, DBP and MAP were recorded and injection Glycopyrrolate 0.004 mg/kg and injection midazolam 0.02 mg/kg were given intravenously and IV infusion of ringer lactate was started.

Patients were divided into two groups and each group consisted of 25 patients. Group-1: -Lignocaine hydrochloride group. Here patients received injection lignocaine hydrochloride 1.5 mg / kg IV bolus 3 minutes prior to induction.
Group-2: -Fentanyl citrate group. Here patients received injection fentanyl citrate 2 µg / kg IV bolus 3 minutes prior to induction.

All the patients were preoxygenated with 100% oxygen by mask for 3 minutes before induction. Induction was achieved with injection Thiopentone sodium 5 mg/kg Intravenous (2.5% Solution) till loss of eyelid reflexes. Heart rate, SBP, DBP, MAP was recorded and injection Succinylcholine 2 mg/kg was given Intravenously. IPPV given with 100% O_2 and after adequate relaxation (≈60 seconds) laryngoscopy was done using standard Macintosh blade. Oral Intubation was done with appropriately sized, disposable, high volume low pressure, cuffed endotracheal tube. patients who needed laryngoscopy for more than 30 seconds were excluded from the study.

All the patients were ventilated with Bain's Circuit and anaesthesia was maintained with O_2 (33%), N_2O (67%), Isoflurane (0.5-1.0%) and injection vecuronium bromide. Intraoperative vitals were monitored using ECG, Pulse Oximeter, NIBP and Capnography. Heart rate, SBP, DBP, MAP and ECG were recorded in all patients pre operatively, after premedication, before study drug, after induction, after laryngoscopy and intubation and for every 1-minute up to 6 minutes after intubation during which no stimulus was given to patients.

An observation was made related to adverse effects of drugs and laryngoscopy related problems and were attended to appropriately.
Descriptive data of both Groups were compared by unpaired "t" test. For all the tests "p" value of <0.05 was considered for statistical significance.

OBSERVATION AND RESULTS

Demographic data, baseline pulse, blood pressure and type of surgery were comparable between both groups (Table 1, 2, 3, 4)

Table – 1 Shows the mean value of age and weight and baseline pattern of patients in both groups.

Measurable Parameters	Group-I (Lignocaine hydrochloride Group)	Group-II (Fentanyl citrate Group)
No. of patients	25	25
Age (in years)	31 ±7	31± 8
Male / Female	6/19	9/16
Weight (kgs.)	55± 5	56± 6
Pulse rate (bpm)	83±8.3	81±5.4
SBP (mm of Hg)	119±6.1	121±6.1
DBP (mm of Hg)	80±3.7	79±2.6
MAP (mm of Hg)	90±3.6	93±2.7

Table – 2 Patient's age distribution in different age groups.

Study group	20-30 Years	31-40 Years	41-50 Years

Group-I	14	56%	8	32%	3	12%
Group-II	14	56%	8	32%	3	12%
Whole Study	28	56%	16	32%	6	12%

Table-3 Sex distribution in both the groups

Study Group	Male		Female		Total no. of patients
	No. of pts.	%	No. of pts.	%	
Group-I	6	24%	19	76%	25
Group-II	9	36%	16	64%	25

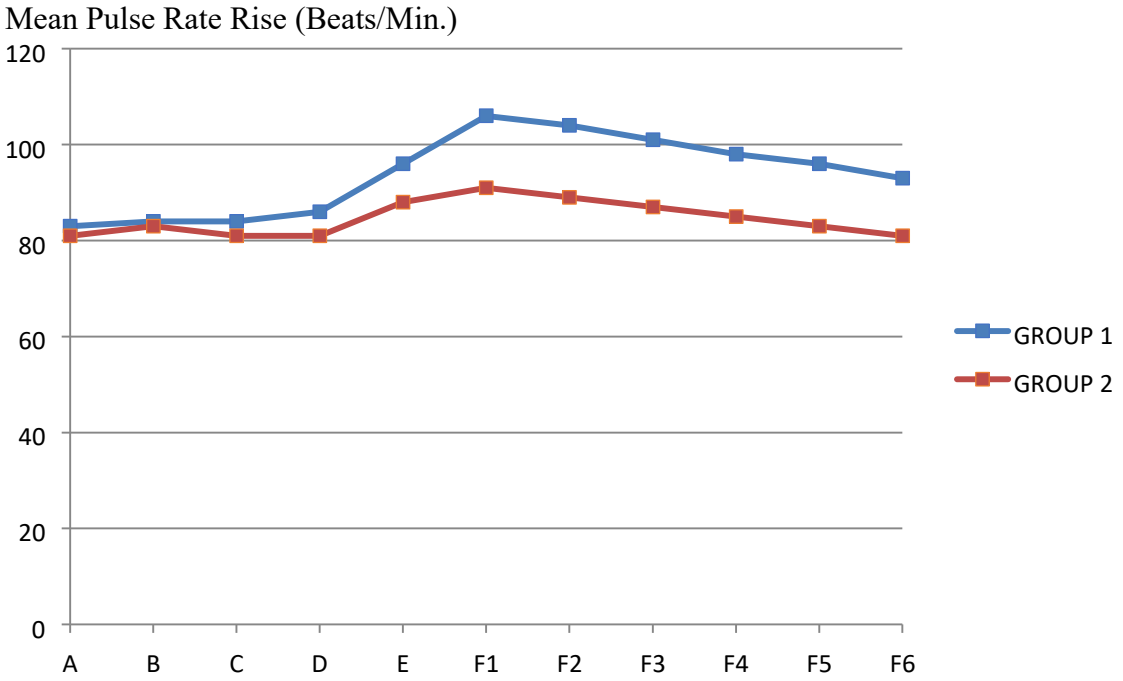
Table – 4 Surgery distribution in both groups

Surgery	Group – I		Group – II	
	No. of pts.	%	No. of pts.	%
Lap. Cholecystectomy	2	8	3	12
Open cholecystectomy	0	0	1	2
Lap. Appendicectomy	5	20	4	16
Diagnostic laparoscopy	2	8	3	12
MTP+Lap TL	4	16	6	24
Pyelolithotomy	1	4	1	4
Nephrectomy	2	8	0	0
Excision biopsy	5	20	5	20
Salpingectomy	1	4	0	
Cystolitholapexy + ureterolithotmy	2	8	0	
Laparoscopic cyst removal	1	4	0	
Thyroidectomy	0		1	4
Breast abscess I&D	0		1	4

Table 5 show a rise in mean pulse rate after laryngoscopy.

Recording interval	Group – I		Group – II	
	Beats/min	%	Beats/min	%
A	83±8.3	-	81±5.4	-
B	84±8.5	1.2	83±5.4	2.4
C	84±7.9	1.2	81±5.2	-
D	86±9	3.6	81±6.2	-
E	96±8.8	15.6	88±6.3	8.6
F1	106±11	27.7	91±5.9	12.3
F2	104±8.7	25.3	89±6.1	9.8
F3	101±8.7	21.6	87±5.8	7.4
F4	98±8.1	18.0	85±5.8	4.9
F5	96±8.4	15.6	83±5.6	2.4
F6	93±7.4	12.0	81±5.3	-

Mean Pulse Rate Rise (Beats/min)



A - Basal Value
B - At 0 min, Inj. Midazolam 0.02% mg/kg Intravenous given
C - Study drug given
D - Induction
E - Laryngoscopy and tracheal intubation
F1 to F6 - Every 1-minute up to 6 minutes after intubation.

Pulse Rate

In present study, pre-operative baseline mean pulse rate in Group-I was 83 beats/minute and in Group-II was 81 beats/minute.

During laryngoscopy and endotracheal intubation mean pulse rate increased by 13 beats/minute in Group-I and 7 beats/minute in Group-II.

In Group-I, mean pulse rate following intubation at F1, F2, F3, F4, F5, F6 at every oneminute interval increased by 23 beats/minute, beats/minute, 21 beats/minute, 15 beats/minute, 13 beats/minute, 10 beats/minute respectively from basal value.

In Group-II, mean pulse rate following intubation at F1, F2, F3, F4, F5, F6 at every one-minute interval increased by 10 beats/minute, 8 beats/minute, 6 beats/minute, 4 beats/minute, 2 beats/minute respectively and at F6 pulse rate comes to near normal. This shows that there was no significant rise in pulse rate after laryngoscopy and intubation in Group-II and it comes to the baseline level within 5-6 minutes' post intubation.

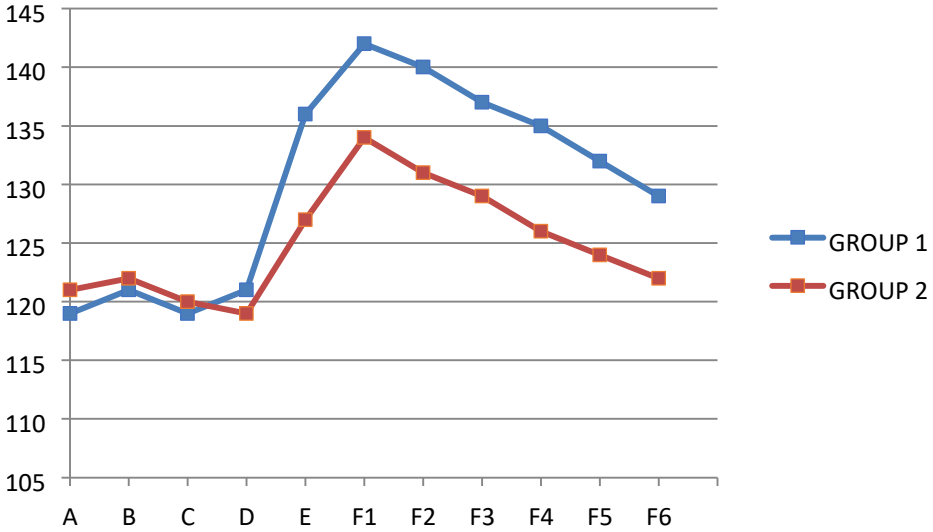
Table 6 and 7 shows rise in systolic and diastolic blood pressure and table 8 shows rise in mean arterial pressure.

Table – 6 Rise in Systolic Blood Pressure (SBP in mm of Hg)

	Group – I		Group – II	
Recording interval	mm of Hg	%	mm of Hg	%

A	119 ± 6.1	-	121±6.1	-
B	121±5.2	1.6	122±6	-
C	119±6.6	-	120±6	-
D	121±6.9	1.6	119±6.2	-
E	136±8.7	14.2	127±6.1	4.9
F1	142±6.7	19.3	134±5.8	10.7
F2	140±6.2	17.6	131±6	8.2
F3	137±6.4	15.1	129±5.7	6.1
F4	135±5.6	13.4	126±5.5	4.1
F5	132±6.1	10.9	124±5.6	2.4
F6	129±7.3	8.4	122±5.7	-

Rise in Systolic Blood Pressure (SBP in mm of Hg)



Systolic Blood Pressure (SBP)

In present study, pre-operative baseline SBP in Group-I was 119 mm of Hg and in Group-II was 121 mm of Hg.

During laryngoscopy and endotracheal intubation mean SBP increased by 17 mm of Hg in Group-I and increased by 6 mm of Hg in Group-II.

In Group-I, following intubation mean SBP at F1, F2, F3, F4, F5, and F6 at every oneminute interval increased by 23 mm of Hg, 21 mm of Hg, 18 mm of Hg, 16 mm of Hg, 13 mm of Hg, 10 mm of Hg respectively.

In Group-II, following intubation mean SBP at F1, F2, F3, F4, F5, F6 at everyone minute interval increased by 13 mm of Hg, 10 mm of Hg, 8 mm of Hg,5 mm of Hg,3 mm of Hg and comes to near normal at F6.

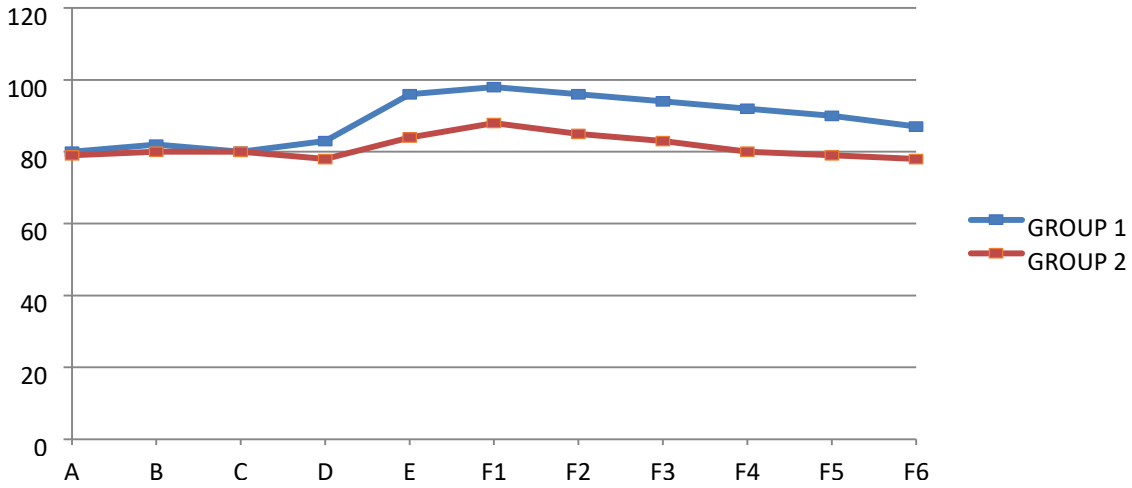
This suggests that in Group-II there was no significant rise in SBP after intubation and it comes to baseline within 5-6 minutes.

Table – 7 Rise in Diastolic Blood Pressure (in mm of Hg)

Recording interval	Group – I		Group – II	
	mm of Hg	%	mm of Hg	%
A	80±3.7	-	79±2.6	-
B	82±4.3	2.5	80±2.7	1.2

C	80±5.4	-	80±2.1	1.2
D	83±5.1	3.7	78±4.4	-
E	96±6.6	20	84±2.9	6.3
F1	98±6.7	22.5	88±3.7	11.3
F2	96±6.7	20	85±3.8	7.5
F3	94±6.4	17.5	83±3.8	5.0
F4	92±7.2	15	80±3.5	1.2
F5	90±6.9	12.5	79±3.2	-
F6	87±6.4	8.7	78±2.9	-

Rise in Diastolic Blood Pressure (in mm of Hg)



Diastolic Blood Pressure (DBP)

In present study, pre-operative baseline DBP in Group-| was 80 mm of Hg and in Group-|| was 79 mm of Hg.

During laryngoscopy and endotracheal intubation mean DBP increased by 16 mm of Hg in Group-| and increased by 5 mm of Hg in Group-||.

In Group-|, following intubation mean DBP at F1, F2, F3, F4, F5, F6 at every oneminute interval increased by 18 mm of Hg, 16 mm of Hg, 14 mm of Hg, 12 mm of Hg, 10 mm of Hg, 7 mm of Hg respectively.

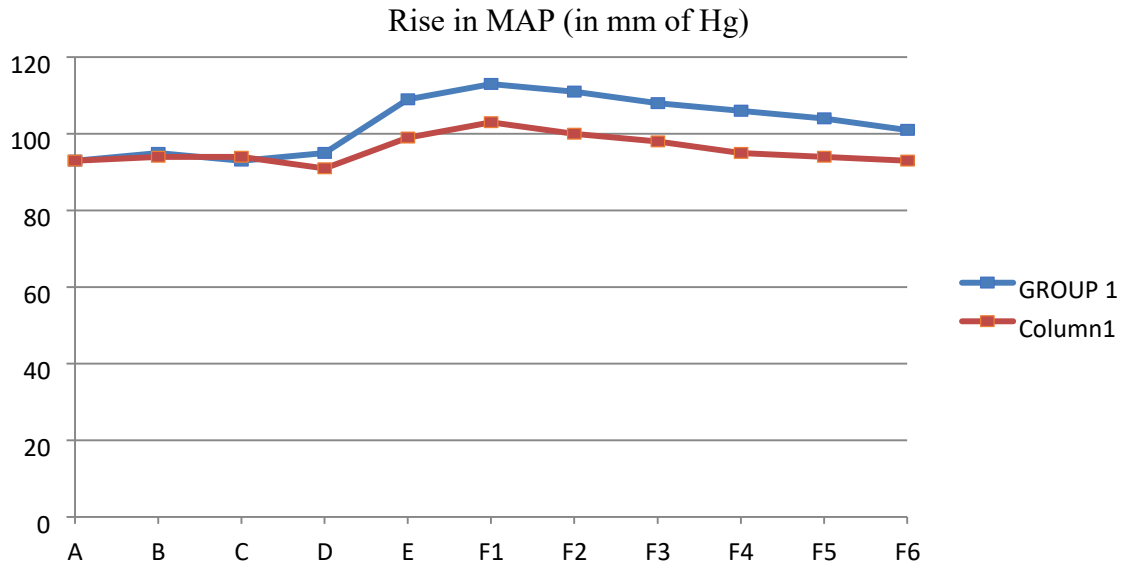
In Group-||, following intubation mean SBP at F1, F2, F3, F4, F5, F6 at every oneminute interval increased by 9 mm of Hg, 6 mm of Hg, 4 mm of Hg and comes to near normal at F4.

This suggests that in Group-|| there was no significant rise in DBP after intubation and it comes to baseline within 4-5 minutes.

Table – 8 Rise in MAP (in mm of Hg)

Recording interval	Group – I		Group – II	
	mm of Hg	%	mm of Hg	%
A	93±3.6	-	93±2.7	-
B	95±4	2.1	94±2.9	1.7
C	93±5.2	-	94±2.6	1.7
D	95±4.7	2.1	91±3.8	-

E	109±6.4	17.2	99±2.5	6.4
F1	113±6.1	21.5	103±3.1	10.7
F2	111±5.5	19.5	100±3.1	7.5
F3	108±5.5	16.1	98±2.9	5.3
F4	106±5.9	13.9	95±2.6	2.1
F5	104±5.9	11.8	94±2.9	1.0
F6	101±5.8	8.6	93±2.8	-



Mean Arterial Pressure (mm of Hg)

In present study, pre-operative baseline MAP in Group-| was 90 mm of Hg and in Group-|| was 90 mm of Hg.

During laryngoscopy and endotracheal intubation mean MAP increased by 16 mm of Hg in Group-1and increased by 6 mm of Hg in Group-2

In Group-|, following intubation mean MAP at F1, F2, F3, F4, F5, F6 at every oneminute interval increased by 20 mm of Hg, 18 mm of Hg, 15 mm of Hg, 13 mm of Hg, 11 mm of Hg, 8 mm of Hg respectively.

In Group-2 following intubation mean MAP at F1, F2, F3, F4, F5, F6 at every oneminute interval increased by 10 mm of Hg, 7 mm of Hg, 5 mm of Hg,2 mm of Hg and comes to near normal at F5.

This suggests that in Group-|| there was no significant rise in MAP after intubation and it comes to baseline within 4-5 minutes.

Table 9 shows no complications occurred during study.

Table 9 Complications

Complication	Group 1	Group 2
Trauma to lip, tongue and nose	0	0
Arrhythmia	0	0
Hypotension	0	0

Raised intracranial and intraocular tension	0	0
Laryngospasm	0	0
Bronchospasm	0	0
Trauma to pharynx, larynx, uvula, oesophagus	0	0
Oesophageal intubation	0	0
Bronchial intubation	0	0
Allergic reaction to drug	0	0

DISCUSSION

Endotracheal intubation and laryngoscopy are associated with rise in blood pressure, heart rate and cardiac dysrhythmias.

These above-mentioned effects may be short lived but they may have adverse effects in high-risk patients like, those with cardiovascular diseases, increased intracranial pressure or anomalies of cerebral vessels.

We selected the optimal age between 20 to 50 years. We excluded the patients taking anti-hypertensive drugs as these may decrease the pressor response. We included only laryngoscopy and orotracheal intubation in our study.

. A linear increase in heart rate and mean arterial pressure during first 45 seconds has been observed. Further prolongation has little effect. As duration of laryngoscopy is normally less than 30 seconds the result of studies in which it takes longer than this has less clinical relevance. The force applied during laryngoscopy has only minor effect. In our study the duration of laryngoscopy and intubation was limited to ≤30 seconds.

Criteria for selection of appropriate drug to prevent sympathetic response are: the drug must prevent impairment of cerebral blood flow, and avoid arousal of the patient. It should not prolong the duration of anaesthesia.

Fentanyl is advocated for attenuation of sympathetic response to laryngoscopy and intubation. Blunting of sympathetic response is dose dependent.

Fentanyl citrate is a primarily μ receptor agonist. Analgesia is produced principally through interaction with receptor at supraspinal sites.

It also binds, to a much lesser degree to the kappa opioid receptors, which mediates sedation and miosis, located within the spinal cord. They act by increasing K+ conductance into cells & inhibit calcium channel or both, thus decreasing the neurotransmitter release.

A single dose intravenous fentanyl citrate has rapid onset of action within 1 to 2 min & peak effect at 5 min.

Fentanyl suppresses the hemodynamic response by increasing depth of anaesthesia and decreasing sympathetic discharge. At high doses fentanyl produces tissue accumulation and thus longer lasting plasma and brain concentration of the drug. These patients may require mechanical respiratory support

Lignocaine hydrochloride a local anaesthetic. Action of local anaesthetic is on the cell membrane of the axon on which it produces electrical stabilization. It blocks the membrane permeability to sodium ion, required for nerve depolarization thus resting potential is maintained and depolarization response is inhibited, eventually propagation of impulse fails.

Local anaesthesia blocks the sodium conductance probably by the dual action on the cell membrane. After intravenous administration, onset of action occurs within 1 minute, peak is reached at 90 seconds and duration last for 20 minutes.

In our study maximum heart rate increase with Lignocaine hydrochloride was 27% and with Fentanyl citrate was 12%. Suppression of maximum rise in heart rate by Fentanyl citrate was statistically highly significant when compared with Lignocaine hydrochloride ($P < 0.001$). It remained significant throughout the study period.

In our study Systolic Blood Pressure increased maximally after laryngoscopy and intubation. With administration of Fentanyl citrate maximum increase compared to basal value was 10% and with Lignocaine hydrochloride was 19%. Among the two drugs Fentanyl citrate showed better result ($P < 0.001$). It remained significant throughout the study period.

Maximum increase in Diastolic Blood Pressure compared to basal value was 22% and 11% in Lignocaine hydrochloride and Fentanyl citrate groups respectively. Attenuation of Diastolic Blood Pressure by Fentanyl citrate was highly significant than Lignocaine hydrochloride ($P<0.001$). It remained significant throughout the study period.

Similarly, maximum increase in MAP compared to basal value was 21% and 10% in Lignocaine hydrochloride and Fentanyl citrate groups respectively. The attenuation of MAP by Fentanyl citrate was highly significant when compared with Lignocaine hydrochloride ($P<0.001$).

In 1991, Miller at al[1], showed that 100 mg bolus dose of esmolol is safe & effective for controlling the haemodynamic response to intubation. Esmolol was less effective in controlling the blood pressure than its combination with low dose fentanyl (2-3 µg/kg). Esmolol suppressed the systolic blood pressure response to tracheal intubation.

In 1991, Wilson I. G.[2], concluded that lignocaine 1.5 mg/kg given at least 4 min before laryngoscopy may completely attenuate the presser response but not the chronotropic response to laryngoscopy and intubation.

In 1991, Helfman and colleagues[3] compared attenuation of response to laryngoscopy and endotracheal intubation by Esmolol, Fentanyl and Lidocaine and found that Esmolol had best and consistent effect and Fentanyl was better than Lidocaine which is comparable to our study.

In 1992, Chung K. S[4]. demonstrated that combination of low dose fentanyl (2 µg/kg) & esmolol (2 mg/kg) provides an alternative to a higher dose fentanyl (5 µg/kg) for blunting the pressor response to laryngoscopy & intubation.

Feng CK, Chan KH (1996)[5] et al studied comparison of fentanyl, Lignocaine and Esmolol for attenuation of cardio vascular response to laryngoscopy and tracheal intubation concluded that Esmolol reliably offered protection against in heart rate and blood pressure, low dose Fentanyl prevented hypertension but not tachycardia and Lignocaine had no effect to blunt hemodynamic response.

DK Baheti, Nipa Bhavsar (2001)[6] they compare lignocaine and fentanyl to attenuate haemodynamic response to laryngoscopy and intubation. They concluded that both are safe to use as bolus intravenous dose as none of the patients had any hypotension, bradycardia, arrhythmias, respiratory depression and muscular rigidity. As far as efficacy goes fentanyl (2µg/kg) was found to be better than lignocaine.

Yushi U. Adachi, Maiko S (2002)[7] had used 2µg/kg fentanyl IV immediately before the induction of anaesthesia. They concluded that fentanyl attenuated the haemodynamic response to endotracheal intubation more than the response to laryngoscopy.

Anila D. Malade, Vineet S (2007)[8] had used single bolus doses of fentanyl (2µg/kg) or lignocaine (1.5mg/kg) for attenuation of pressure response to laryngoscopy and endotracheal intubation. They concluded that lignocaine and fentanyl both attenuated the rise in pulse rate, though fentanyl was better. Our results are comparable to this study.

Similar to our observation Bachofen M (2011)[9] reported that Fentanyl showed a significant pressure lowering effect throughout in observation period while no significant effect of Lignocaine on pressure response.

Gupta and Tank (2011)[10] showed Fentanyl bolus 2µg/kg before induction of anaesthesia is effective in attenuating cardiovascular response. Our study is comparable to this response.

Gurulingappa, Aleem, Awati (2012)[11] studied comparison of i.v bolus fentanyl, Lignocaine and placebo and concluded that attenuation is seen with both the drugs but better with Fentanyl. Our study is comparable to it

Habib Bostan and colleagues (2012)[12] compared clinical efficacy of fentanyl, Lidocaine and Esmolol and found that Esmolol was consistent for attenuation than Fentanyl and Lidocaine. Fentanyl was better than Lidocaine which is comparable to our study.

Rangnathan S, Sarvandan D, Harrikumar (2013)[13] studied attenuation of cardio vascular response using Fentanyl and Lignocaine. Maximum effect was seen with fentanyl plus Lignocaine. No attenuation was seen with Lignocaine. Our result is comparable to this study.

ECG

In present study, ECG was monitored continuously throughout the procedure. No abnormal ECG changes were recorded in any patients.

Complications

No major complications like hypotension, arrhythmias, trauma, bronchospasm, laryngospasm, oesophageal intubation, and bronchial intubation, raised intracranial or intraocular pressure were found in any patients.

In present study of comparison between Inj. Lignocaine hydrochloride (1.5 mg/kg intravenous) and Inj. Fentanyl citrate (2 µg/kg intravenous), fentanyl citrate has proved its efficacy in reducing pulse rate, SBP, DBP and MAP.

Advantages of fentanyl citrate over lignocaine hydrochloride:

- Fentanyl citrate stabilized the Heart Rate and Blood Pressure during the laryngoscopy and intubation.
- This haemodynamic stability lasts even after intubation.
- Fentanyl citrate provides the haemodynamic stability by increasing sedation, analgesia and the depth of anaesthesia, thus blocking the reflex sympathoadrenal response to noxious stimulation of larynx.
- No respiratory, cardiovascular or central nervous system depression was seen.
- Minimal amount of drug interactions was encountered.

CONCLUSION

In our study, injection fentanyl citrate 2 µg/kg attenuated the haemodynamic response better than Lignocaine 1.5 mg/kg. Haemodynamic parameters did not rise significantly after intubation with Fentanyl and reached basal value within study time.

Among the two drugs Fentanyl citrate showed better attenuation of sympathetic response which is statistically highly significant.

So, the Fentanyl citrate is safe and effective in attenuation of hemodynamic response to laryngoscopy without significant side effects.

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