

Comparative Study of Fentanyl and Fentanyl Plus Lidocaine on Attenuation of Hemodynamic Responses to Tracheal Intubation in Controlled Hypertensive Patients Undergoing General Anaesthesia

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Abstract

Aim: Comparative analysis of fentanyl and fentanyl plus lidocaine on attenuation of hemodynamic responses to tracheal intubation in controlled hypertensive patients undergoing general anaesthesia.

Methodology: This prospective randomized observational study was conducted in the Department of Anaesthesiology, Govt. Medical College, Barmer from Jan-2021 to Dec-2021 at Rajasthan after getting approval from ethical committee 90 patients aged between 30 years to 60 years of either sex belonging to ASA class II (controlled hypertensives) posted for various elective surgeries under general anesthesia at our institute were randomly selected for the study. Study population (90 patients) were randomly divided by computer generated numbers into 3 groups with 30 patients in each group. Group A: will receive Inj Fentanyl 2 µg/kg before induction. Group B: will receive Inj. Fentanyl 2µg/kg plus inj Lignocaine 1.5mg/kg before induction. Group C: will receive normal saline before induction.

Results: We found that our study suggests that combination of fentanyl and lignocaine provides more significant attenuation of heart rate, MAP, controls more effectively rise in SBP and DBP than fentanyl alone after laryngoscopy and tracheal intubation. No clinically relevant side effects were observed in all three groups.

Conclusion: Fentanyl and combination of Fentanyl and lignocaine, both effectively decreased hemodynamic response to tracheal intubation. However, neither fentanyl nor fentanyl plus lignocaine could inhibit all hemodynamic responses. Fentanyl plus lignocaine was more effective in attenuating hemodynamic responses than fentanyl alone.

Keywords: Endotracheal Intubation, Fentanyl, Lignocaine/Lidocaine, Hemodynamic Responses.

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Introduction

Laryngoscopy, endotracheal intubation, and other airway manipulations (e.g., placement of a nasopharyngeal or oropharyngeal supralaryngeal airway) are noxious stimuli that may induce profound changes in cardiovascular physiology, primarily through reflex responses. These changes are even more profound in hypertensive patients. Direct Laryngoscopy and endotracheal intubation are invariably associated with certain cardiovascular changes such as hypertension, tachycardia and wide variety of cardiac arrhythmias.[1]

Although these responses may be of short duration, variable, unpredictable and of little consequence in healthy individuals; serious complications can occur in patients with underlying coronary artery disease, myocardial insufficiency, hypertensive, reactive airways, intracranial neuropathology, cerebral hemorrhage, pre-eclamptic and eclamptic patients.

Regardless of the preoperative blood pressure control in hypertensive patients, there is an exaggerated fall in BP following induction (due to depleted intravascular volume status) and an excessive rise in BP following endotracheal intubation.

A variety of drugs and methods have been tried to attenuate this stress response[2,3] considering their ability to block the intense sympathetic discharge during airway stimulation.

- Premedicating patient with antihypertensive drugs
 1. vasodilator (eg. hydralazine),
 2. beta blocker(eg. Esmolol, labetalol),
 3. calcium channel blocker (eg. nifedipine)
 4. α -2 agonist(clonidine, dexmedetomidine),
 5. nitroglycerine (intravenous, intranasal spray or sublingual)
 6. ACE inhibitor(eg. captopril, enalapril)
- Opioids (fentanyl, alfentanyl, sufentanyl)

- Lignocaine (intravenous, spray or gargles)
- Deepen plane of anaesthesia by intravenous induction agent or increasing concentration of volatile anaesthetic during mask ventilation.
- Decreasing laryngoscopy time to less than 15 seconds.

Fentanyl, a synthetic opioid agonist (μ receptor) is popularly used as an IV analgesic and a component of balanced anaesthesia. In appropriate dose, it controls both heart rate and blood pressure responses.[4] It is about one hundred times more potent than morphine as an analgesic.

Lignocaine, an aminoethylamide and prototype of amide local anesthetic group. The use of lignocaine is well known in treatment of patients with ventricular dysarrhythmias. In 1961, Bromage showed that its intravenous (IV) use blunted pressure response to intubation. An IV dose of lignocaine 1.5 mg/kg given 3 min prior to intubation has shown near optimal results.[5]

Both the drugs modulate nociceptive input by a different pharmacological mechanism for control of cardiovascular response to intubation. Thus, their combination may prove to be more efficacious than either agent alone. More so, hypertensive patients show an exaggerated changes in hemodynamics in response to such noxious stimuli. The potential benefit and safety of combination of Fentanyl and Lignocaine has been suggested by previous investigations.

Therefore, this study was designed to evaluate and compare the effectiveness of intravenous fentanyl and a combination of fentanyl and lignocaine in attenuating hemodynamic response to direct laryngoscopy and endotracheal intubation in controlled hypertensive patients undergoing surgical procedures under general anaesthesia.

Materials and Methods

Source of Data

This prospective randomized observational study was conducted in the Department of Anaesthesiology, Govt. Medical College, Barmer from Jan-2021 to Dec-2021 at Rajasthan after getting approval from ethical committee 90 patients aged between 30 years to 60 years of either sex belonging to ASA class II (controlled hypertensives) posted for various elective surgeries under general anesthesia at our institute were randomly selected for the study.

Data was collected in study proforma meeting the aims and objectives of the study. Study population (90 patients) were randomly divided by computer generated numbers into 3 groups with 30 patients in each group.

Group A: will receive Inj Fentanyl 2 µg/kg before induction

Group B: will receive Inj. Fentanyl 2µg/kg plus inj Lignocaine 1.5mg/kg before induction

Group C: will receive normal saline before induction

A written and informed consent was taken from the patient after explaining the procedure to the patient.

Inclusion Criteria:

- Age 30 to 60 years.
- ASA II
- Undergoing elective surgery of longer than one hour duration.

Exclusion criteria:

- ASA grade I, III, IV and V.
- Known case of bronchial asthma, IHD.
- Patients with atrial/ventricular arrhythmias, second/third degree A-V conduction block.
- Patients with Congestive Heart Failure and terminal valvular insufficiency.
- Patients with severe hemodynamic instability like severe anaemia, hypotension.

- Patients on beta adrenergic antagonist therapy/calcium channel blockers, hypnotics, narcotic analgesics
- Patients with anticipated difficult airway.
- Patients requiring more than one attempt at intubation.
- Patients known to have allergy to anaesthetic drugs used in study
- Psychiatric patients

Procedure:

Preoperative assessment (PAC)

All the patients underwent a detailed pre anaesthetic check-up on the day before surgery and all the routine and specific investigations like Hemoglobin, Total leucocyte count, Differential leucocyte count, Liver function test, Renal function test, ECG, X-Ray chest (PA view), Fasting/ Random Blood Sugar, Platelet count were done. Whenever necessary special tests were carried out.

The patients were electively kept nil by mouth for 6 hours before surgery and prior to operation patients were explained about the procedure and informed consent were taken from patients' relatives. After the patient was shifted to the operation theatre, standard monitors like ECG, NIBP, and pulse oximetry were applied and baseline parameters [Spo₂, Heart rate (HR), Systolic blood pressure (SBP), Diastolic blood pressure (DBP), Mean arterial pressure (MAP)] were recorded. Two intravenous lines with 18/20-gauge cannula were secured and intravenous fluid was started.

Anesthesia

Patients were premedicated with:

- Inj. Ondansetron 0.15 mg/kg i.v.
- Inj. Glycopyrrolate 4µg/kg i.v.
- Group A will receive Inj. Fentanyl 2 µg/kg before induction
- Group B will receive Inj. Fentanyl 2µg/kg plus inj lidocaine 1.5 mg/kg before induction
- Group C will receive Inj. Normal saline

Preoxygenation

The patient will be pre oxygenated for 5 mins using 100% oxygen with Bain's circuit with O₂ flow at 8L/min

Induction

Induction of anaesthesia will be carried out using Inj. Propofol 2mg/kg iv and inj vecuronium bromide 0.1 mg/kg iv

Intubation

Intubation was done with an appropriate portex cuffed endotracheal tube after direct laryngoscopy using macintosh blade. After checking bilateral air entry equal, endotracheal tube was fixed and positive pressure ventilation was started. Throat packing, positioning and surgical incision will be withheld till completion of recording

Maintenance

Anaesthesia will be maintained using 50% oxygen, 50% nitrous oxide, 1% sevoflurane and Inj. Vecuronium Bromide 0.08mg/kg IV.

Monitoring

- Heart rate (HR)
- Systolic blood pressure (SBP)
- Diastolic blood pressure (DBP)
- Mean arterial blood pressure (MAP)
- Pulse oximetry (SpO₂)

All parameters were recorded at following stages:

- Baseline
- After pre-medication
- Before induction
- After induction.

- After intubation.
- At 1,2,3,5 and 10 mins after intubation.

Reversal

All patients were reversed after onset of spontaneous respiration using Inj. Glycopyrrolate 8µg/kg i.v. and Inj. Neostigmine 0.05 mg/kg i.v.

Extubation

After satisfied criteria for extubation, thorough oral and endotracheal suction was done, and patients were extubated. Any prevalence of laryngospasm, bronchospasm or desaturation were recorded and managed according to standard protocols. Any intraoperative complication was recorded and managed accordingly. Patients were shifted to recovery room and any immediate postoperative complication e.g. nausea, vomiting, shivering, respiratory depression, sedation, restlessness, hypotension, bradycardia etc were recorded and managed accordingly.

Statistical Analysis

All patients' data were recorded in proforma of study. Data was expressed as mean values \pm standard deviation (SD). Quantitative data was analyzed using t-test and qualitative by chi square test. Statistical calculations were carried out using Microsoft Office Excel 2016. Changes in hemodynamic variables from baseline and a comparison of means were analyzed by paired t-test for each time interval. A P-value <0.05 was considered statistically significant.

Results:

Table 1: Comparison of changes in Mean HEART RATE \pm S.D. between three group

	A		B		C		P VALUE		
	MEA N	\pm SD	MEA N	\pm SD	MEA N	\pm SD	AB	BC	AC
BASELINE	89.83	12.5 3	86.76	13.9	88.43	14.2 9	0.374 9	0.649 8	0.688 2
AFTER PREMED	84.16	11.6 1	80.93	11.5 2	86.56	13.5 3	0.283 5	0.087 9	0.464 1

BEFORE INDUCTION	89.83	10.8 7	78.73	11.6 6	85.7	13.1 3	0.001	0.034	0.189 5
AFTER INDUCTION	87.5	11.6 5	86.6	11.9 8	96.1	13.0 3	0.777 5	0.007	0.009 2
AFTER INTUBATION	98.96	11.4 3	92.83	11.2 3	105.5 3	12.8 2	0.040 5	0.000 1	0.040 7
1 MIN AFTER INTUBATION	103.8	10.5	96.53	10.4 7	110.6	11.7 3	0.009 8	0.000 1	0.020 8
2 MINS AFTER INTUBATION	104.8	9.43	93	10.2 5	112.7 6	10.8 3	0.000 1	0.000 1	0.003 9
3 MINS AFTER INTUBATION	101	9.26	89.46	10.3 4	108.8 3	10.6 4	0.000 1	0.000 1	0.003 7
5 MINS AFTER INTUBATION	91.96	9.27	84.03	10.3 7	101.7 3	10.3 2	0.002 8	0.000 1	0.000 3
10 MINS AFTER INTUBATION	86.13	9.81	80.63	10.0 6	94.8	10.0 6	0.036 3	0.000 1	0.000 6

This table shows the comparison of changes in mean heart rate at various predetermined time interval and P value of group AB, group BC and group AC to determine the significance of the changes in heart rate between three groups.

Table 2: Comparison of changes in Mean SBP (SYSTOLIC BLOOD PRESSURE) \pm S.D. between three groups

	A		B		C		P VALUE		
	MEAN	\pm SD	MEAN	\pm SD	MEAN	\pm SD	AB	BC	AC
BASELINE	125.8	9.87	126.13	10.72	127.23	9.27	0.9008	0.6725	0.5645
AFTER PREMED	123.1	8.88	124.16	9.021	126.3	8.03	0.6462	0.3373	0.1487
BEFORE INDUCTION	122.2	9.02	124.13	9.59	125.93	7.68	0.4249	0.4259	0.0898
AFTER INDUCTION	116.1	8.76	116.56	9.19	117.46	8.22	0.8412	0.6909	0.5358
AFTER INTUBATION	134.7	6.78	129.46	9.32	143.43	7.91	0.0158	0.0001	0.0001
1 MIN AFTER INTUBATION	136.03	6.74	130.73	9.23	147.3	7.53	0.0138	0.0001	0.0001
2 MINS AFTER INTUBATION	133.33	6.46	127.7	9.15	147.96	6.4	0.0079	0.0001	0.0001
3 MINS AFTER INTUBATION	129.26	6.09	125.03	8.79	144.86	6.12	0.0343	0.0001	0.0001
5 MINS AFTER INTUBATION	124.8	6.49	123.23	8.5	139.36	6.02	0.4258	0.0001	0.0001
10 MINS AFTER INTUBATION	121.73	6.54	121.23	8.33	132.96	5.92	0.7969	0.0001	0.0001

This table shows the comparison of changes in mean SBP (Systolic Blood Pressure) at various predetermined time interval and P value of group AB, group BC and group AC to determine the significance of the changes in SBP between three groups.

Table 3: Comparison of changes in Mean DBP (DIASTOLIC BLOOD PRESSURE) \pm S.D. between three groups

	A		B		C		P VALUE		
	MEAN	\pm SD	MEAN	\pm SD	MEAN	\pm SD	AB	BC	AC
BASELINE	80.13	9.52	82.06	8.44	81.26	6.90	0.409	0.6893	0.5998
AFTER PREMED	78.23	7.45	80.46	7.58	81.2	5.026	0.2548	0.6605	0.076

BEFORE INDUCTION	77.93	7.14	79.43	7.21	80.83	4.511	0.4219	0.3712	0.0653
AFTER INDUCTION	73.66	6.96	74.5	6.54	75.5	3.91	0.6347	0.4756	0.214
AFTER INTUBATION	85.53	6.4	81.86	7	89.8	3.791	0.0386	0.0001	0.0027
1 MIN AFTER INTUBATION	87.2	6.75	80.7	6.88	91.63	3.61	0.0005	0.0001	0.0024
2 MINS AFTER INTUBATION	85.86	6.83	80	6.69	94.06	2.80	0.0014	0.0001	0.0001
3 MINS AFTER INTUBATION	83.6	6.64	78.66	6.83	91.33	2.83	0.0063	0.0001	0.0001
5 MINS AFTER INTUBATION	80.2	6.86	77.9	5.99	88.66	2.46	0.1719	0.0001	0.0001
10 MINS AFTER INTUBATION	77.6	6.83	75.86	5.55	84.8	2.86	0.2854	0.0001	0.0001

This table shows the comparison of changes in mean DBP (Diastolic Blood Pressure) at various predetermined time interval and P value of group AB, group BC and group AC to determine the significance of the changes in DBP between three groups.

Table 4: Comparison of changes in Mean of MAP (MEAN ARTERIAL PRESSURE) \pm S.D. between three groups

	A		B		C		P VALUE		
	MEAN	\pm SD	MEAN	\pm SD	MEAN	\pm SD	AB	BC	AC
BASELINE	95.35	9.09	96.75	9.00	96.58	7.41	0.5512	0.9379	0.5669
AFTER PREMED	93.18	7.42	95.03	7.79	96.23	5.75	0.3519	0.5001	0.081
BEFORE INDUCTION	92.68	7.14	94.33	7.70	95.86	5.08	0.3949	0.3669	0.0518
AFTER INDUCTION	87.811	6.72	88.52	7.07	89.48	4.76	0.6913	0.5371	0.2694
AFTER INTUBATION	101.92	5.45	97.73	7.47	107.67	3.78	0.0161	0.0001	0.0001
1 MIN AFTER INTUBATION	103.47	5.61	97.37	7.38	110.18	3.78	0.0007	0.0001	0.0001
2 MINS AFTER INTUBATION	101.68	5.47	95.9	7.21	112.03	3.15	0.0009	0.0001	0.0001
3 MINS AFTER INTUBATION	98.82	5.24	94.12	7.142	109.17	3.01	0.0052	0.0001	0.0001
5 MINS AFTER INTUBATION	95.06	5.78	93.01	6.47	105.56	2.83	0.2002	0.0001	0.0001
10 MINS AFTER INTUBATION	92.31	5.90	90.98	6.07	100.85	3.04	0.3961	0.0001	0.0001

This table shows the comparison of changes in mean of MAP (Mean Arterial Pressure) at various predetermined time interval and P value of group AB, group BC and group AC to determine the significance of the changes in MAP between three groups.

Table 5: Comparison of changes in Mean Oxygen saturation \pm S.D. between three groups

	A		B		C		P VALUE		
	MEAN	\pm SD	MEAN	\pm SD	MEAN	\pm SD	AB	BC	AC
BASELINE	98.83	0.91	99.03	0.76	98.76	0.72	0.3615	0.1719	0.7556
AFTER PREMED	98.7	0.83	98.76	0.93	98.8	0.84	0.7721	0.8855	0.6472
BEFORE INDUCTION	98.76	0.91	98.7	0.91	98.76	0.77	0.7812	0.7617	1
AFTER INDUCTION	98.9	0.80	98.73	0.91	99	0.74	0.4558	0.2196	0.6185

AFTER INTUBATION	99	0.74	98.83	0.91	99.03	0.76	0.4411	0.3615	0.8646
1 MIN AFTER INTUBATION	98.7	0.91	98.7	0.83	99.1	0.60	1	0.0584	0.0508
2 MINS AFTER INTUBATION	98.73	0.78	98.76	0.72	98.73	0.69	0.8651	0.8563	0.862
3 MINS AFTER INTUBATION	99.1	0.60	98.8	0.84	98.9	0.80	0.1203	0.6406	0.2811
5 MINS AFTER INTUBATION	98.73	0.69	98.76	0.77	99.03	0.93	0.8609	0.2338	0.1631
10 MINS AFTER INTUBATION	98.76	0.72	99	0.74	98.76	0.93	0.2241	0.289	1

Discussion:

Cardiovascular response to laryngoscopy and endotracheal intubation has always become a challenge for anaesthetists. Cardiovascular response may occur in form of hypertension, tachycardia and different types of arrhythmias. These effects may prove disastrous in patients of hypertension, myocardial insufficiency, pre-eclampsia, eclampsia, cerebral hemorrhage etc. C. Prys-Roberts et al (1971)[6] reported high incidence of cardiac arrhythmia, myocardial ischemia-infarction, acute LVF and cerebrovascular accidents following intubation in patients with hypertension.

It would seem prudent therefore to adopt preventive measures to attenuate this cardiovascular response which otherwise may lead to dangerous complications or even sudden death. Hence it becomes the moral obligation of anaesthesiologist towards any patient to ensure attenuation of this cardiovascular response to intubation.

In our study, we have attempted the combination of fentanyl and lignocaine to attenuate hemodynamic response and also compared its efficacy with fentanyl alone. Fentanyl, an opioid analgesic (μ receptor agonist) significantly attenuates the stress response to laryngoscopy and intubation and also seem to provide more stable hemodynamic profile prior to laryngoscopy and tracheal intubation. Fentanyl has been used in various doses varying from 2-15 $\mu\text{g}/\text{kg}$ for blunting stress response but doses greater than or equal to 5 $\mu\text{g}/\text{kg}$ may cause

excessive sedation, apnoea, and chest wall rigidity preoperatively, and nausea, vomiting and prolonged respiratory depression post operatively especially in surgeries with duration of less than 2 hours.

The beneficial effect of lignocaine may be due to direct myocardial depression and peripheral vasodilatation. The advantage of combining Fentanyl and lignocaine is considered to be based on their different mechanism of action.

Hemodynamic Parameters

(A) Heart Rate (HR): As shown in table 1, Baseline values of mean Heart rate were comparable between three groups with no statistically significant difference ($P>0.05$). Changes in heart rate after giving study drug and after induction were also not statistically significant between any of the group ($P>0.05$). Heart rate increased in all groups after intubation and increase was maximum in group C (105.53 ± 12.82), i.e when no drug was given. In comparison between group A where heart rate increased by 10.16% (from 89.83 ± 12.53 at baseline to 98.96 ± 11.43 after intubation). The increase in heart rate in Group B after intubation was 6.99% (from 86.76 ± 13.9 at baseline to 92.83 ± 11.23 after intubation). In controls, heart rate continued to rise till 3 minutes after intubation (108.83 ± 10.6). Maximum heart rate in Group A (104.86 ± 9.43) and in Group B (96.53 ± 10.47). Heart rate started to return to baseline after 5 minutes in group A and after 3 minutes of intubation in group B. Whereas in Group C heart rate started

returning to baseline only after 10 minutes. Changes in heart rate remained significant between all the 3 groups starting from intubation up till 10 minutes (P value <0.05). Thus our study suggests that combination of fentanyl and lignocaine provides more significant attenuation of heart rate than fentanyl alone after laryngoscopy and tracheal intubation.

This is in contradiction to Valiallah et al 2013[7] and siddharth et al 2015[8] who found that fentanyl and lignocaine combined was not more effective than fentanyl alone in reducing the heart rate. Our study was also in line with study by Ali et al. in 2010[9] revealed that pre-treatment with xylocard improves intra- and post-operative hemodynamic stability during laparoscopic surgery without prolonging recovery.

Additionally, Malde and Sarode in a 2007[10] study compared lignocaine and fentanyl efficacy on hemodynamic stability and revealed that lignocaine and fentanyl both attenuated the rise in heart rate; however, fentanyl produced better results.

(B) Systolic Blood Pressure (SBP):

As shown in table 2, Baseline values of mean SBP were comparable between three groups with no statistically significant difference ($P>0.05$). Changes in SBP after giving study drug and after induction were also not statistically significant between any of the group ($P>0.05$). SBP increased in all groups after intubation and increase was maximum in group C, 12.7% (from 127.23 ± 9.27 at baseline to 143.43 ± 7.91 after intubation). In group A there was 7.07% (125.8 ± 9.87 to 134.7 ± 6.78) increase in heart rate compared to 2.6% in Group B (126.13 ± 10.72 to 143.43 ± 7.91) after intubation. In control group C systolic BP continued to rise till 2 minutes after intubation and remained elevated throughout the study period. It started to return to baseline only after 10 minutes. In group A and group B SBP was maximum at

1 minute after intubation. SBP started to return to baseline values after 5 minutes in group and after 3 minutes in group. Between group A and group B changes in SBP was statistically significant after intubation and till 3 minutes after intubation. ($P<0.05$). Between group B- group C and group A- group C changes in SBP was statistically significant after intubation and till 10 minutes after intubation. ($P<0.05$). Thus, this data indicates that Fentanyl plus Lignocaine controls rise in SBP after laryngoscopy and tracheal intubation more effectively than Fentanyl alone. This result was in contrast to the findings of Valiallah et al.[7] and siddharth at al.[8], however our study showed similar results to study by ali et al.[9], Kautto et al.[11] and Malde and Sarode[10]

(C) Diastolic Blood Pressure (DBP):

As shown in table 3, Baseline values of mean DBP were comparable between three groups with no statistically significant difference ($P>0.05$). Changes in DBP after giving study drug and after induction were also not statistically significant between any of the group ($P>0.05$). DBP increased in all groups after intubation and increase was maximum in group C (94.06 ± 2.80) i.e. 15.7% increase. In group A maximum DBP was (87.2 ± 6.75) whereas rise in DBP in Group B was minimum (81.86 ± 7). Maximum rise in DBP was seen after intubation in all the groups. DBP started to return to baseline values after 3 minutes in group A, after 10 minutes in group C whereas in Group B DBP did not change much from baseline even immediately after intubation. Changes in DBP remained statistically significant between group A and group B till 3 minutes after intubation (P value <0.05). Thus, we conclude that fentanyl plus lignocaine effectively controls changes in DBP to intubation compared to fentanyl alone. This result was in contrast to the findings of Valiallah et al.[7] and siddharth at al.[8] our study showed similar results to study by ali et

al.[9], Kautto et al.[11] and Malde and Sarode.[10]

(D) Mean Arterial Pressure (MAP):

As shown in table 4, Baseline values of mean MAP were comparable between three groups with no statistically significant difference ($P>0.05$). Changes in MAP after giving study drug and after induction were also not statistically significant between any of the group ($P>0.05$). MAP increased in all groups after intubation and increase was maximum in group C (112.03 ± 3.15). In group A maximum increase in MAP was (103.47 ± 5.61) i.e. 8.5% and in group C MAP increases to (97.73 ± 7.47) i.e. 1.01%. Maximum rise in MAP was seen after intubation in all the groups. MAP started to return to baseline values after 3 minute in group A, after 2 minutes in group B and after 10 minutes in group C. Between group A and group B changes in MAP was statistically significant after intubation and till 10 minutes after intubation. ($P<0.05$). Between group B and group C changes in MAP was statistically significant after intubation and till 10 minutes after intubation. ($P<0.05$) Between group A and group C changes in MAP was statistically significant after intubation and till 10 minutes after intubation. ($P<0.05$) Hence this study demonstrates that fentanyl plus lignocaine is better than Fentanyl in attenuating rise in MAP after laryngoscopy and tracheal intubation. This result was in contrast to the findings of Valiallah et al.[7] and siddharth at al.[8] our study showed similar results to study by ali et al.[9], Kautto et al.[11] and Malde and Sarode.[10]

(E) Oxygen Saturation

As shown in table 5, Mean Oxygen saturation remained above 98% in all the groups. Changes in oxygen saturation was not statistically significant ($P>0.05$) between any of the groups at any point of time interval.

(F) Side Effects and Complication

In our study, there is no intraoperative bradycardia, hypotension, arrhythmias, bronchospasm or postoperative vomiting, respiratory depression, bronchospasm, bradycardia/tachycardia, hypotension/hypertension, arrhythmias or any other side effects or complication were observed in any of the groups. This result was similar to the findings of Valiallah et al.[7] and siddharth at al.[8]

Conclusion:

Fentanyl and combination of Fentanyl and lignocaine, both effectively decreased hemodynamic response to tracheal intubation. However, neither fentanyl nor fentanyl plus lignocaine could inhibit all hemodynamic responses. Fentanyl plus lignocaine was more effective in attenuating hemodynamic responses than fentanyl alone.

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