COMPARATIVE STUDY OF ATTENUATION OF CARDIOVASCULAR RESPONSE TO LARYNGOSCOPY AND INTUBATION WITH IV DEXMEDETOMIDINE VS. IV LIGNOCAINE

P. V. Shiva[1], M. Pavani[2], B. Deepraj Singh[3]

[1]Associate Professor, Department of Anaesthesiology, Government Medical College, Nizamabad, Telangana.
[2]Consultant Anaesthesiologist, Department of Anaesthesiology, Secunderabad Hospital, Secunderabad, Telangana.
[3]Professor, Department of Anaesthesiology, Osmania Medical College, Hyderabad, Telangana.

ABSTRACT

Endotracheal intubation is one of the most commonly performed procedures, where the role of the anaesthesiologists in patient care is noteworthy. Endotracheal intubation is translaryngeal placement of endotracheal tube into the trachea via the nose or mouth. General anaesthesia procedures involve stressful events at various stages. The most stressful situations are seen during the period of induction, intubation and extubation. A 25% to 50% increase in mean arterial pressure and heart rate is seen during induction followed by laryngoscopy and intubation peaking at 1-2 minutes and returning to baseline within 10-15 minutes.

AIMS AND OBJECTIVES

A comparative study of attenuation of cardiovascular response to laryngoscopy and intubation with IV dexmedetomidine vs IV lignocaine, to compare changes in HR, systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) in patients premedicated with dexmedetomidine and lidocaine and to observe for any significant difference in the anaesthetic requirement and intraoperative complications if any in both the groups.

METHODOLOGY

A total of 60 patients undergoing elective surgeries were selected. Group D consists of 30 patients, who received IV dexmedetomidine loading dose, 1 mcg/kg diluted in 50 mL NS for 10 minutes prior to laryngoscopy. Group –L consists of 30 patients who received plain preservative-free Lidocaine 2%, 1.5 mg/kg body weight IV bolus ninety seconds prior to laryngoscopy.

RESULTS

Dexmedetomidine in a bolus dose of 1 ug/kg IV attenuates heart rate response to laryngoscopy and intubation effectively than plain preservative-free lignocaine. The basal values of heart rate were reached within 1 min after intubation in case of dexmedetomidine group. Dexmedetomidine blunts the increase in systolic, diastolic and mean arterial pressure effectively than Plain preservative-free Lignocaine.

KEYWORDS

Inj. Dexmedetomidine, Inj. Lignocaine.

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INTRODUCTION: Since 1950s hypertension and tachycardia have been recognised as commonly associated with intubation under light anaesthesia.[1] The mechanical stimulation of four areas of upper respiratory tract - the nose, the epipharynx, the laryngopharynx and tracheobronchial tree induces reflex cardiovascular response which is associated with enhanced neuronal activity in cervical sympathetic fibres. This response is accompanied by raised plasma adrenaline concentration. These changes are probably of little consequence in healthy patients. But this cardiovascular response to intubation is of a serious concern in patients with hypertension, raised intracranial pressure, diseased cerebral vasculature or with ischaemic heart disease where increase in myocardial oxygen consumption can lead to myocardial infarction.[2] Failure to blunt the responses to intubation may have disastrous consequences like acute left ventricular failure, intracranial haemorrhage and pulmonary oedema. Convulsions may be precipitated in eclamptic patients. Herniation of intracranial contents and cerebral ischaemia can occur in patients with raised intracranial pressure. Arrhythmias (sinus tachycardia and sinus bradycardia, atrial and ventricular extra systoles and pulsus alternans, less commonly multifocal extra systoles, pulsus bigeminy and atrial fibrillation) are reported. Heart block, ventricular tachycardia and ventricular fibrillation are fortunately rare. Different techniques with different drugs have been suggested to attenuate these responses. The stress response can also be minimised by decreasing the duration of laryngoscopy to less than 15 seconds. Some

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Corresponding Author:
Dr. B. Deepraj Singh,
#1-7-145/15, Musheerabad-500020, Hyderabad, Telangana.
E-mail: drdeepraj@gmail.com
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of these are topical and intravenous lignocaine, deep inhalational anaesthesia, ganglion blockers, precurarisation, narcotics (fentanyl, alfentanyl, etc.), vasodilators, IV nitroglycerin, and calcium channel blockers, alpha 2 agonists like clonidine and dexmedetomidine. Lidocaine is easily available drug used for attenuation of pressor response.

Dexmedetomidine a newly introduced alpha 2 agonist, decreases the sympathetic tone, hence blunts the pressor response, and also has analgesic properties. This makes it more favourable drug to be used for attenuating pressor response, as it can be used as an adjunct to anaesthesia.

Hence, a study is undertaken to compare dexmedetomidine to lidocaine as regards to its efficacy on attenuation of intubation response.

Assessment of Cardiovascular Response: Cardiovascular responses can be assessed with the changes in heart rate and blood pressure. Typically, BP begins to rise after about 15 sec of laryngoscopy and becomes maximal after 30-45 sec. King et al observed that the average rise in BP after laryngoscopy and intubation was 45 sec which remains for 1-2 min, followed by a gradual fall to prelaryngeal level within 5 min. HR and BP responses are minimal when laryngoscopy is brief (< 15 sec).

Urban et al[3] examined the relationship between several intraoperative haemodynamic variables, fixed cardiac risk factors, intraoperative myocardial ischemia and postoperative MI in a group of patients undergoing CABG. They found that RPP> 12000 beats/min. mmHg exhibited a significant association (p=0.04) with pre-cardiopulmonary bypass myocardial ischaemia. Thus, a more rational approach is to use the patients' preoperative levels as a guide and to ensure that these do not fluctuate beyond 20% in the perioperative period, during the anaesthetic management of susceptible patients.

AIMS AND OBJECTIVES: A comparative study of attenuation of cardiovascular response to laryngoscopy and intubation with IV dexmedetomidine vs. IV lignocaine.

To compare changes in HR, systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) in patients premedicated with dexmedetomidine and Lidocaine. To observe for any significant difference in the anaesthetic requirement and intraoperative complications if any in both the groups.

MATERIALS & METHODOLOGY: A randomised controlled study was conducted in Secunderabad Hospital, Secunderabad, after approval from the Hospital Ethics Committee, and obtaining written informed consent from the patients during the period between May 2012 and October 2014. A total of 60 patients undergoing elective surgeries were selected.

This study was undertaken in 2 groups.

Group-D consists of 30 patients, who received IV dexmedetomidine loading dose, 1 mcg/kg diluted in 50 mL NS for 10 minutes prior to laryngoscopy.

Group –L consists of 30 patients, who received plain preservative-free Lidocaine 2%, 1.5 mg/kg body weight IV bolus 90 seconds prior to laryngoscopy.

Inclusion Criteria:

- Age range 18-55 of both sex.
- ASA grade I & II.
- Mallampati class I & II.

Exclusion Criteria: ASA grade III & IV, Mallampati class III & IV, intubation duration lasting longer than 20 seconds or more than one attempt of laryngoscopy, predicted difficult intubation, patients with morbid obesity, BMI above 30 kg/sq. m, patient undergoing procedures requiring head and neck manipulation.

Complete haemogram, urine analysis, blood chemistry, electrocardiogram, and x-ray chest [PA view] were carried out in every case before surgery.

Venous Cannulation: Intravenous cannulation with 18G cannula was inserted and a drip was started with Ringer's lactate solution.

Monitors: Multipara monitor showing the following: NIBP, SpO2, ECG, Heart Rate, ETCO2, respiratory rate were monitored.

Anaesthetic Technique: After Institutional Ethical Committee approval, and obtaining informed consent from patients, a randomised double blinded prospective study involving 60 patients of both sexes requiring endotracheal intubation and general anaesthesia will be included. No sedation was given on the night before surgery. All patients will be premedicated with Inj. glycopyrrolate, 0.2 mg IV, Inj. ondansetron 4 mg IV, Inj. midazolam 0.05 mg/kg IV, Inj. fentanyl 1.5 µg/kg IV.

The D group received a total dose of 1 mcg/kg dexmedetomidine diluted in 50 mL NS in 10 minutes by infusion using infusion pump and after 7 min of starting the infusion, patient was induced with 5 mg/kg thiopentone Sodium through intravenous route. After confirming ventilation, injection vecuronium 0.1 mg/kg was administered intravenously. After 3 min of ventilation with bag and mask, patient was intubated with gentle laryngoscopy.

The group L received 5 mg/kg thiopentone Sodium through intravenous route. After confirming ventilation, injection vecuronium 0.1 mg/kg was administered intravenously and plain preservative-free Lidocaine 2%, 1.5 mg/kg body weight IV bolus 90 seconds prior to laryngoscopy.

Time taken for intubation did not exceed 15-20 seconds in both groups. The D group received 0.2 mcg/kg/hr of dexmedetomidine as maintenance dose. Anaesthesia was maintained with vecuronium bromide and

intermittent positive pressure ventilation using closed circuit system in both groups. Haemodynamic changes like Heart rate (HR), Systolic Blood Pressure(SBP), Mean Blood Pressure(MBP) and Diastolic Blood Pressure(DBP) were monitored before, during and 1, 3, and 5 minutes after endotracheal intubation.

Monitoring: Non-invasive Blood pressure monitoring – Systolic and Diastolic and Mean Arterial Pressure, Heart rate, Oxygen Saturation, Continuous ECG monitoring [limb lead II]and Adequacy of ventilation was monitored clinically.

OBSERVATIONS AND RESULTS: Sixty patients, undergoing elective non-cardiac surgery were selected for the study. The patients were randomly divided into two groups, of 30 patients each.

Group–L patients, who received plain preservative-free Lidocaine 2%, prior to laryngoscopy.

Group-D patients, who received IV dexmedetomidine prior to laryngoscopy.

STATISTICAL METHODS: Data were collected, tabulated, coded and analysed. Numerical variables were presented as mean and standard deviations (SD) while categorical variables were presented as frequency and percentage. For the numerical variables, Student t-test was used whenever appropriate, for comparison between the two groups. For the categorical variables, Chi-square test was used. A difference with P value <0.05 was considered statistically significant.

Statistical Software: The statistical software, Epi info 3.5.3 was used for the analysis of the data and Microsoft Word and Excel have been used to generate graphs and tables, etc.

Randomisation: Sample taken at random from a population when each member of the population has an equal chance of being chosen. The purpose is to produce groups that are as nearly similar as possible prior to the experimental procedure.

Mean: The mean of a collection of numbers is their arithmetic average, computed by adding them up and dividing by their number.

Standard Deviation (SD): It is a statistical measure of spread or variability. The standard deviation is the root mean square (RMS) deviation of the values from their arithmetic mean.

The range for ages was 18-55 years and 18-54 years in Lignocaine group and Dexmedetomidine group respectively. The range for weight was 37- 60 kg and 35-62 kg in Lignocaine group and Dexmedetomidine group respectively. There was no statistically significant difference between the groups. This implies that the patients for each group was selected randomly without showing any bias.

Parameters	Lignocaine		Dexmedeto-midine		P value
	Mean	SD	Mean	SD	
Heart Rate	85.88	±9.01	82.92	±8.05	0.294
Systolic BP (mmHg)	121.44	±5.78	121.00	±4.30	0.952
Diastolic BP (mmHg	79.52	±4.66	80.56	±5.49	0.736
MAP (mmHg)	93.2	±4.14	94.08	± 4.89	0.664

Table 1: Showing Haemodynamic parameters at Pre-anaesthetic evaluation

Tests of significance between groups were carried out by student's t-test or modified t-test. No significant difference was observed between Lignocaine group and Dexmedetomidine group values (P value> 0.05), in any parameters at pre anaesthetic check-ups.

Parameters	Lignocaine		Dexmedeto-midine		P value
	Mean	SD	Mean	SD	
Heart Rate	87.12	±9.12	83.52	±8.060	0.265
Systolic BP (mmHg)	120.2	±7.41	119.93	±6.62	0.996
Diastolic BP (mmHg	79.84	±4.65	76.83	±4.62	0.283
MAP (mmHg)	92.44	±6.07	91.67	± 6.67	0.685

Table 2: Comparison of Lignocaine group and Dexmedetomidine group with Haemodynamic parameters during Pre-Induction Period

Tests of significance were carried out by student t-test or modified t-test.

The difference between both groups is not statistically significant (P>0.05) for all the parameters. Therefore, Pre-induction readings are taken as basal values.

Parameters	Lignocaine		Dexmedetomidine	
	Mean	SD	Mean	SD
Heart Rate	104.68	±9.12	85.67	±4.90
Systolic BP (mmHg)	136.88	±6.90	125.03	±5.89
Diastolic BP (mmHg	89.36	±5.38	72.23	±4.78
MAP (mmHg)	104.16	±6.06	86.96	±5.46

Table 3: Comparison of Lignocaine group and Dexmedetomidine group with Haemodynamic parameters during Intubation Period

In Lignocaine group, the heart rate and arterial pressures showed a continuous rise compared to basal values (pre-induction values).

In the Dexmedetomidine group, diastolic pressure and mean arterial pressure showed a decline, compared to basal values (pre-induction values). The difference is statistically significant (P<0.05) in the Dexmedetomidine group compared to that of Lignocaine group.

But, the heart rate and SBP in the Dexmedetomidine group were increased during intubation period, compared to basal values (pre-induction values). But the rise is significantly small than that of rise in Lignocaine group (P<0.05).

Electrocardiographic changes were found to be within normal limits in all the groups.

Parameters	Lignocaine		Dexmedetomidine	
	Mean	SD	Mean	SD
Heart Rate	104.23	10.65	79.83	5.02
Systolic BP (mmHg)	135.53	7.12	115.5	6.25
Diastolic BP	89.23	5.505	70.2	4.62
MAP (mmHg)	104.35	4.68	85.13	4.13

Table 4: Comparison of Lignocaine group and Dexmedetomidine group with Haemodynamic parameters 1 min after Intubation

Parameters	Lignocaine		Dexmedetomidine	
	Mean	SD	Mean	SD
Heart Rate	101.7	9.80	74.53	4.16
Systolic BP	132.13	5.63	110.5	5.22
Diastolic BP	84.66	3.91	69.53	4.44
MAP (mmHg)	100.4	3.16	83.18	3.26

Table 5: Comparison of Lignocaine group and Dexmedetomidine group with Haemodynamic parameters 3 mins after Intubation

Parameters	Lignocaine		Dexmedetomidine	
	Mean	SD	Mean	SD
Heart Rate	99.03	9.48	70.16	4.91
Systolic BP	130.23	5.42	106.23	5.48
Diastolic BP	82.83	3.99	67.16	4.74
MAP (mmHg)	98.54	2.94	80.02	3.64

Table 6: Comparison of Lignocaine group and Dexmedetomidine group with Haemodynamic parameters 5 mins after Intubation

Time point	Lignocaine	Dexmedetomidine
	Mean	Mean
Pre-induction (basal)	87.12	83.52
During intubation	104.68	85.67
1 minute after intubation	104.23	79.83
3 minutes after intubation	101.7	74.53
5 minutes after intubation	99.03	70.16

Table 7: Comparison of heart rate at various stages

Time point	Lignocaine	Dexmedetomidine
	Mean	Mean
Pre-induction (basal)	120.2	119.93
During intubation	136.88	125.03
1 minute after	135.53	115.5
3 minutes after	132.13	110.50
5 minutes after	130.23	106.23

Table 8: Comparison of Systolic Blood Pressure at various stages

Both the groups have SBP in the same range in pre-induction stage, during intubation there is increase in SBP in both groups but after intubation there is attenuation of pressor response in Dexmedetomidine group, with statistical significant difference (P<0.05) between the groups which continued till 5 minutes post intubation.

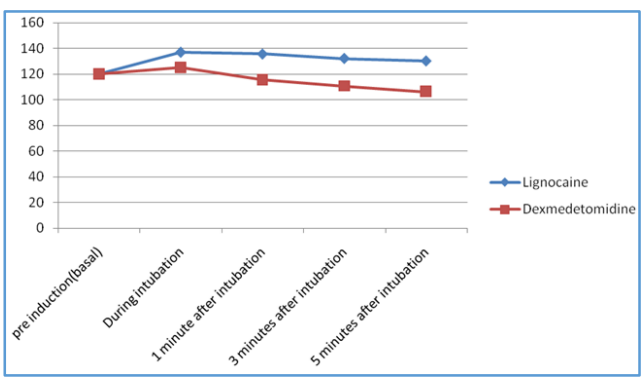

Fig. 1: Comparison of Systolic Blood Pressure at various stages

	Lignocaine	Dexmedetomidine
Pre-induction (basal) vs During intubation	P=0.0000*	P=0.0400*
Pre-induction (basal) vs 1 minute after intubation	P=0.0000*	P=0.0000
Pre-induction (basal) vs 3 minutes after intubation	P=0.0397*	P=0.0000*
Pre-induction (basal) vs 5 minutes after intubation	P=0.2959*	P=0.0000*

Table 9 : Significance of changes of Diastolic Blood Pressure from Pre-induction (basal) values with other stages

*p<0.05.

Time point	Lignocaine	Dexmedetomidine
	Mean	Mean
Pre-induction (basal)	79.84	76.83
During intubation	89.36	72.23
1 minute after intubation	89.23	70.20
3 minute after intubation	84.66	69.53
5 minute after intubation	82.83	67.16

Table 10: Comparison of Diastolic Blood Pressure at various stages

In Lignocaine group, the diastolic blood pressures showed a continuous rise compared to basal values (pre-induction values). In the Dexmedetomidine group, diastolic pressures showed a decline, compared to basal values (pre-induction values). The difference is statistically significant (P<0.05) in the Dexmedetomidine group.

Time point	Lignocaine	Dexmedetomidine
	Mean	Mean
Pre-induction (basal)	92.44	91.67
During intubation	104.16	86.96
1 minutes after intubation	104.35	85.13
3 minutes after intubation	100.40	85.13
5 minutes after intubation	98.54	80.20

Table 11: Comparison of Mean arterial pressure at various stages

	Lignocaine	Dexmedetomidine
Pre-induction (basal) vs. During intubation	P=0.0000*	P=0.0399*
Pre-induction (basal) vs. 1 minute after intubation	P=0.0000*	P=0.0000
Pre-induction (basal) vs. 3 minutes after intubation	P=0.0000*	P=0.0000*
Pre-induction (basal) vs. 5 minutes after intubation	P=0.0000*	P=0.0000*

Table 12: Significance of changes of Mean arterial pressure from Pre-induction (basal) values with other stages

*p<0.05.

Both the groups have Mean arterial pressure in the same range in Pre-induction stage. But during and after Intubation there is a significant difference between the groups which continued till 5 minutes post intubation. Attenuation of reflex mean arterial pressure responses to intubation was significant in the Dexmedetomidine group, (P<0.05).

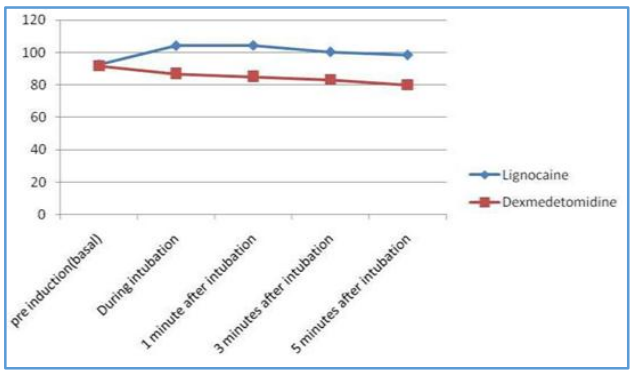

Fig. 2: Comparison of Mean arterial pressure at various stages

	Lignocaine gr	Dexmedetomidine gr
Hypotension	2(7%)	10(33%)
Bradycardia	1(3%)	11(%37)
Apnoea	1(3%)	6(20%)
Intra-operative hypertension	19(63%)	2(7%)
Intra-operative tachycardia	20(67%)	2(7%)

Table 13: Complications noted during the study

DISCUSSION: The haemodynamic response to laryngoscopy and endotracheal intubation has been a topic of discussion right since 1940. These hemodynamic responses were first recognised in 1940 by Reid and Bruce et al,[4] Burstein et al,[1] found that the pressor response occurring at laryngoscopy and endotracheal intubation was due to augmented sympathetic response, provoked by stimulation of epipharynx and laryngopharynx. These factors were further confirmed by Prys-Roberts. The efferent sympathetic outflow to the heart is T1-T4, while that to adrenal medulla is from T3-L3.

The elevation in arterial pressure in response to tracheal intubation that typically starts within 5 seconds of laryngoscopy peaks in 1 to 2 minutes, and returns to control levels within 5 minutes. Such hemodynamic changes seem to cause little harm to most patients though in compromised patients, there may be a risk of cardiovascular decompensation leading to fatal myocardial infarction.

In our study, plain preservative-free Lidocaine 2%, 1.5 mg/kg IV bolus was given 90 seconds prior to laryngoscopy. The results were in accordance to 2 min. group of Stanley Tam et al study i.e. cardiovascular responses were significantly above baseline levels.

Aleem et al[5] compared the effects of lignocaine and fentanyl in attenuation of pressor response to laryngoscopy and tracheal intubation. After intubation incidence of tachycardia (HR>100/min) was significantly greater in lignocaine group than in fentanyl group ($p<0.05$). Rise in SBP and DBP were also statistically significant in lignocaine group than in fentanyl group ($p<0.05$). Attenuation of pressor response is seen both with lignocaine and fentanyl. Of the two drugs, fentanyl 4 mcg IV bolus provides a consistent, reliable and effective attenuation as compared to lignocaine 1.5 mg/kg IV bolus. In our study, plain preservative-free Lidocaine 2%, 1.5 mg/kg IV bolus was given 90 seconds prior to laryngoscopy. The results were similar to that of above study.

Abou-Modi, Keszer and Yacoub[6] (1977) who had used intravenous lignocaine in 1.5 mg/kg and 0.7 mg/kg doses. They had found that intravenous lignocaine in 1.5 mg/kg dose provided complete protection against arrhythmias, but only a borderline protection against increase in blood pressure and heart rate, while the dose 0.7 mg/kg was inadequate in providing protection against arrhythmias, only preventing blood pressure elevation. They have described the possible mechanisms of action of lignocaine as- direct myocardial depressant effect, peripheral vasodilating effect and effect of synaptic transmission. Robert Stoelting[7] (1977) found that lignocaine in 1.5 mg/kg intravenously can attenuate the pressor response effectively in a study conducted on 36 known heart disease patients scheduled for noncardiac major surgeries.

D. G. Clayton et al[8] (1983) studied effects of pretreatment with intravenous lignocaine 1.5 mg/kg given 1 min. prior to induction of anaesthesia. It was shown to reduce significantly, the incidence of dysrhythmias during dental anaesthesia and also to reduce the rise in blood pressor associated with endotracheal intubation.

Gupta et al[9] compare effectiveness of intravenous esmolol and lignocaine in suppressing the cardiovascular stress response. Patients were divided into three groups of 20 patients each. Group-L received lignocaine and Group-E received esmolol three minutes before intubation, Group-C did not receive any drug under study. It was found that patients given esmolol had better attenuation of stress response to laryngoscopy and intubation than patients given lignocaine. Our results were similar to that of above study.

Kim JT et al[10] conducted to determine whether lignocaine or remifentanil effectively attenuate the response to endotracheal intubation during rapid sequence induction. Forty-eight patients were randomly divided into three groups: Group NS(n=16) received normal saline 0.1 mL/kg, Group L(n=16) received 1.5 mg/kg of lignocaine, and Group R(n=16) received remifentanil 1 mcg/kg. Anaesthesia was induced with propofol 2 mg/kg after glycopyrrolate 0.2 mg IV. Each study drug was given intravenously over 30 seconds after loss of consciousness. Cricoid pressure was applied until intubation. Succinylcholine 1.0 mg/kg was administered to facilitate tracheal intubation. The results indicate that remifentanil 1 mcg/kg, but not lignocaine 1.5 mg/kg, effectively attenuates the haemodynamic response to endotracheal intubation during rapid sequence induction using propofol.

Prakash et al[11] compared intubating conditions after induction of anaesthesia with propofol, midazolam and fentanyl with those after propofol, lignocaine and fentanyl. In 80 ASA I/II adult patients undergoing elective gynaecological surgery, intubating conditions were compared after induction of anaesthesia with a fentanyl 2 µg/kg, midazolam 0.03 mg/kg, propofol 2.5 mg/kg combination (group FMP) vs. a fentanyl 2 µg/kg, lignocaine 1.5 mg/kg, propofol 2.5 mg/kg combination. They concluded that the fentanyl, midazolam, propofol combination more reliably provides acceptable conditions for intubation than the fentanyl, lignocaine, propofol combination in the absence of neuromuscular blocking agents.

In our study, only 7 out of 30 patients in the dexmedetomidine group showed elevated BP at 2-3 minutes after starting of dexmedetomidine infusion.

Apart from that there was a fall of HR by 7.2% and fall of SBP, MAP, and DBP 9.6%, 8.56% and 8.96% respectively at 5 min after starting dexmedetomidine. Arterial BP decrease has shown to be varied in different studies in accordance with varying dexmedetomidine doses, infusion speed, premedication and fluid infusion before drug administration.

Also when compared to the lignocaine group, there is a significant difference ($p<0.05$) and this difference continues up to 5 minutes post-intubation. Regarding DBP, our study differs from other studies as it is decreased from the baseline values at all the recordings. Adverse effects related to dexmedetomidine are hypotension and bradycardia in several studies. But after their protocols were modified by slow infusion of loading dose of dexmedetomidine, these side effects were hardly observed. But clinically all the patients were stable and maintained MAP >65 mmHg and HR >50/min; thus none of these patients were intervened with a vasopressor. Literatures are very few to recommend regarding stabilisation of blood pressure after a fall noticed due to dexmedetomidine and the use of ideal drug with its dose.

Alpha-2 adrenoceptors do not have an active role in the respiratory centre.[12] This leads to use the alpha-2 agonists as sedatives without affecting the ventilator efforts. Few of the studies have observed episodes of apnoea with dexmedetomidine infusion of 2 mcg/kg. This may be secondary to the profound sedation. In our study with dexmedetomidine 1 mcg/kg infused over 10 min, 6 patients developed apnoea requiring controlled bag and mask ventilation before administration of thiopentone.

This is in contrast to only 2 patients requiring support ventilation in the lignocaine group. This may be explained by the synergistic effects of sedative action of both midazolam and dexmedetomidine. SpO2 was never below 96% in both groups. However, in a study the lowest saturation noted was 92% and in another study the lowest saturation noted was 91%.

Anaesthetic sparing effects of dexmedetomidine may be due to its effects on CNS. Clonidine has a ceiling effect in this aspect because of its alpha- 1 agonistic activity; but ceiling effect of dexmedetomidine is not yet observed. The mechanism is still to be found out. But it has been seen that, in rats dexmedetomidine decreased MAC of halothane by >90%; in patients for abdominal hysterectomy, dexmedetomidine decreased the MAC of isoflurane by 90% and in another study it decreased the MAC of sevoflurane by 92%. In our study, though it was not recorded, decreased requirement of inhalational anaesthesia was felt clinically.

In our study, thiopentone requirement was lessened by 28% in comparison to the lignocaine group. Earlier reports quote that with dexmedetomidine 1 mcg/kg, the thiopentone requirement is decreased by 55% and with 0.5 mcg/kg it is decreased by 37%. Disparity in results from our study may be due to the different doses of premedication administered to the patient e.g. in our study, we used 1 mcg/kg fentanyl and 0.05 mg/kg midazolam in both the groups.

Findings suggest that use of dexmedetomidine as an alternative to propofol for sedation of CABG patients post-operatively contributes to reduced mechanical ventilation time, ICU length of stay and post-operative length of stay. Higher drug costs resulting from the propofol shortage were offset by savings in postoperative room and board costs. Additional savings may be possible by preventing medical complications to the extent possible. Sairaku A et al[13] tested the use of dexmedetomidine as a procedural sedative during ablation of atrial fibrillation (AF). Patients were randomised to be treated with dexmedetomidine (n= 43) or thiamylal (n = 44) as sedatives during AF ablation. Apnoeic and body movement events were monitored, during the procedure. The occurrence of hypotension [P=0.14] and bradycardia [P = 1.0] were similar in the patients with dexmedetomidine and thiamylal. Therefore, Dexmedetomidine is potential alternative for that with GABAergic anaesthetics.

Kim YS et al[14] investigated the optimum dosage of dexmedetomidine for prevention of post-anaesthetic shivering in patients scheduled for elective laparoscopic total hysterectomy. They concluded that dexmedetomidine 0.75 or 1.0 μg/kg provides effective prophylaxis against postoperative shivering as well as an analgesic effect.

Le Guen M et al[15] conducted a double-blinded, randomised study used an automated dual closed-loop administration to maintain the Bispectral Index between 40 and 60. Dexmedetomidine (1 μg/kg over 10 minutes followed by a continuous infusion of 0.5 μg/kg/h throughout surgery) was compared with equal volumes of saline as a placebo. Propofol and remifentanil requirements were compared. They concluded that dexmedetomidine administration significantly reduced the requirement for both propofol and remifentanil during anaesthetic induction and reduced propofol use during maintenance of anaesthesia. In our study also Dexmedetomidine delayed postoperative analgesic use.

Dexmedetomidine is a useful adjuvant that reduces anaesthetic requirement and provides postoperative analgesia.

In our study, dexmedetomidine administration resulted in pleasant, relaxed patients in postoperative period. Dexmedetomidine provides and augments analgesia and diminishes shivering as well as agitation postoperatively. The safety record of dexmedetomidine suggests that it can be used effectively and safely in children, with appropriate monitoring and interventions to manage cardiovascular sequelae.

Though there is a chance of hypotension and bradycardia, this study revealed minimal such side effects, requiring intervention. It does not cause apnoea in any of the patients though the level of sedation is increased. It decreased the requirement of thiopentone when given in a sleeping dose for induction. Over a period of 3-4 hours of dexmedetomidine administration also results in a smoother extubation, and minimal requirement of intraoperative and early postoperative analgesics.

All effects of dexmedetomidine could be antagonised easily by administering the alpha-2 adrenoceptor antagonist, atipamezole, which reverses sedation and sympatholysis and has a half-life of 1.5 to 2 hours. The combination of dexmedetomidine and atipamezole might be the basis for a reversible intravenous anaesthetic technique that could provide timely independent recovery from anaesthesia and sedation in the future.

CONCLUSIONS AND SUMMARY: A study of plain preservative-free lignocaine and dexmedetomidine in attenuation of the cardiovascular response during Laryngoscopy and intubation was compared in 60 adult patients, undergoing elective surgeries under general anaesthesia. Patients of ASA Grade – I & II and Mallampati: grade – I & II, were selected and informed consent was taken for all cases. The patients were in the age group ranging from 18-55 years. The weight of patients in all groups ranges from 45-72 kg.

This study was taken in 2 groups.

Group-D consists of 30 patients, who received IV dexmedetomidine loading dose, 1 mcg/kg diluted in 50 mL NS for 10 minutes prior to laryngoscopy.

Group–L consists of 30 patients, who received plain preservative-free Lidocaine 2%, 1.5 mg/kg body weight IV bolus 90 seconds prior to laryngoscopy.

Premedication was given with Inj. glycopyrrolate, 0.2 mg IV, Inj. ondansetron 4 mg IV, Inj. midazolam 1 mg IV and Inj. fentanyl 1.5 mcg/kg IV. Induction of anaesthesia was achieved by intravenous thiopentone of 2.5% conc. (5 mg/kg.) Tracheal intubation was facilitated with IV vecuronium 0.1 mg/kg body weight. Haemodynamic parameters like blood pressure, heart rate, mean arterial pressure, ECG changes were recorded at the following intervals. Pre-induction (basal values), during laryngoscopy and endotracheal intubation, 1 minute, 3 minutes and 5 minutes after intubation. Patients who received dexmedetomidine did not show significant increase in the various haemodynamic parameters during laryngoscopy

and intubation. Patients who received plain preservative-free lignocaine 2% showed a significant increase in various parameters during laryngoscopy and intubation.

The following conclusions can be drawn from our study: Dexmedetomidine in a bolus dose of 1 µg/kg IV attenuates heart rate response to laryngoscopy and intubation effectively than plain preservative-free lignocaine.

The peak response of heart rate to intubation was observed during laryngoscopy and intubation in both groups. Though there was a rise in heart rate in the dexmedetomidine group, the rise was not significant when compared with the same in the lignocaine group.

But the basal values of heart rate were reached within 1 min after intubation in case of dexmedetomidine group.

Dexmedetomidine blunts the increase in mean arterial pressure effectively than plain preservative-free lignocaine. In case of lignocaine group, the peak response i.e. maximum increase in haemodynamic parameters to intubation was observed during laryngoscopy and intubation and they failed to return to basal values even 5 minutes after intubation.

No ECG changes were noted in all the groups.

This study shows that dexmedetomidine is superior to lignocaine in attenuation of pressor response and ideal time for administration would be about 10 minutes before laryngoscopy and intubation.

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