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Research Article
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Comparison of efficacy of Lignocaine 1.5 mg/kg & Dexmedetomidine 1 µg/kg in attenuating the hemodynamic pressure response to laryngoscopy & intubation

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Background: Dexmedetomidine attenuates the hemodynamic stress response tolaryngoscopy and intubation more effectively compared with Lignocaine without any deleterious effects. To study the efficacy of Lignocaine 1.5 mg/kg & Dexmedetomidine 1 µg/kg in attenuating the hemodynamic pressure response to laryngoscopy & intubation. **Material and Methods:** A total of 100 American Society of Anesthesiologists (ASA) physical status I and II patients aged between 18 and 50 years undergoing elective surgery were enrolled in the study. Patients posted for elective surgery were randomly selected and divided into two groups. Group L = 50 Patients had given 1.5mg/kg Lignocaine; Group D = 50 Patients had given 1µg / kg Dexmedetomidin. **Results:** In Dexmedetomidine group, HR, SBP, DBP and MAP showed significant decrease throughout the study period, as compared to Lignocaine group. The statistical analysis shows that, in Dexmedetomidine group, HR, SBP, DBP and MAP showed significant decrease throughout the study period, as compared to Lignocaine group. **Conclusion:** Newer agents like Dexmedetomidine, a centrally acting alpha-2 agonist suppresses reflex sympathetic stimulation caused by laryngoscopy & intubation more effectively than Lignocaine. Thus, it is concluded that Dexmeditoedine is a better drug compared to Lignocaine in attenuating pressure response to laryngoscopy & intubation.

Keywords: Dexmeditoedine, Lignocaine, Pressure response, Laryngoscopy, Intubation

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Introduction

Endotracheal intubation has become an integral part of anaesthetic management and critical care of the patient. Direct laryngoscopy and endotracheal intubation are almost always associated with haemodynamic changes due to reflex sympathetic discharge, caused by epipharyngeal and laryngopharyngeal stimulation. This increased sympathoadrenal activity results in hypertension, tachycardia and arrhythmias which are usually transitory, variable and unpredictable. Hypertensive patients are more prone to have significant increase in BP, whether they have been treated beforehand or not. Transitory hypertension and tachycardia may be hazardous to those with hypertension, myocardial insufficiency and cerebro-vascular diseases. The laryngoscopic reactions in such individuals may predispose to pulmonary edema, myocardial insufficiency and cerebrovascular accidents [1,2]. Many pharmacological methods have been devised to reduce the extent of haemodynamic events, e.g. with high dose of opioids, alpha1 agonists and beta blockers, local anaesthetics like Lignocaine and vasodilator drugs like nitroglycerine. Intravenous Lignocaine with its well established centrally depressant and anti-arrhythmic effect was found to minimize pressor response [3-7]. Laryngoscopy and tracheal intubation are known to increase sympathetic activity that may be detrimental to patients with pre-existing ischemic or hypertensive heart diseases. In patients with cardiovascular or cerebral disease, there is increased risk of morbidity and mortality from the tachycardia and hypertension resulting from this stress. Recommendations for attenuation of reflex hypertension and tachycardia are therefore manifold. Since there were many studies and many drugs used to attenuate the reflex of laryngoscopy and intubation and the scope of Dexmetomidine needed to be more elaborately discussed. Therefore, this study comparison of efficacy of Lignocaine 1.5 mg/kg & Dexmedetomidine 1 µg/kg in attenuating the hemodynamic pressure response to laryngoscopy & intubation.

Materials and Methods

Study design/Type of study: This was the prospective randomized study conducted in patients posted for elective surgery. It was conducted at one of the tertiary care centre, Government Medical College of India.

Sample size & duration of study: A total of 100 American Society of Anesthesiologists (ASA) physical status I and II patients aged between 18 and 50 years undergoing elective surgery were enrolled in the study, after obtaining the approval of ethical committee of the institute. The study was conducted from March 2017 - October 2018. Patients were divided into two groups with the help of computer-generated coded envelopes.

- Group L= 50 Patients had given 1.5mg/kg Lignocaine
- Group D= 50 Patients had given 1µg /kg Dexmedetomidin

Inclusion criteria

1) Written informed consent

2) Patients posted for elective surgery under general anesthesia

3) Age group 18 to 50

4) ASA Grade I or II

Exclusion criteria

1) Impaired renal & hepatic function

2)History of heart disease, hypertensive patients, pulmonary disease, pregnancy, morbid obesity, allergy to drug

3) Patients with anticipated difficult airway or in whom intubation attempt lasted longer than 15 sec

Study tools: Structured questionnaire, Inj. Lignocaine (1.5 mg/kg) vial, Inj. 1µg / kg Dexmedetomidine ampoule.

Data collection procedure: All patients included in the study were premedicated with tablet alprazolam 0.5 mg and tablet ranitidine 150 mg orally at bedtime the night before surgery.

They were kept nil orally 10 pm onwards on the previous night. On arrival of the patient in the operating room baseline parameters such as heart rate (HR), blood pressure (BP), respiratory rate, and oxygen saturation (SpO_2) were recorded. All patients were prehydrated with 500 ml of Ringer's lactate solution.

Group L received 100 ml of normal saline 20 min preoperatively over a period of 10 min, and the infusion was completed 10 min before induction and 1.5 mg/kg of Lignocaine was administered IV 3 min before intubation.

Group D received Dexmedetomidine 1 mcg/kg diluted in 100 ml of normal saline IV over a period of 10 min, and the infusion was completed 10 min before induction.

After preoxygenation, all patients were induced with injection thiopentone as 2.5% solution IV till loss of the eye lash reflex occurred. Endotracheal intubation was facilitated with 2 mg/kg of succinylcholine given IV 1 min prior to laryngoscopy and intubation. Laryngoscopy & tracheal intubation was performed using Macintosh laryngoscope & appropriate size endotracheal tube. No surgical or any other stimulus was applied during 10 min of study period and vecuronium was the only additional drug given during this period, was maintained using 33% oxygen and 66% nitrous oxide with isoflurane and vecuronium. At the end of the procedure, patients were reversed with injection neostigmine 0.05 mg/kg IV and injection glycopyrolate 0.08 mg/kg IV. Patients were extubated when they regained reflexes and consciousness.

Evaluation of the response to intervention: Hemodynamic parameters were recorded during the basal period, pre-induction, induction, during intubation, 1 min, 3 min, 5 min, and 10 min after intubation.

Data analysis: The statistical software SPSS version 21.0 was used for the analysis of the data and Microsoft word and excel have been used to generate tables etc.

Ethical approval: Taken

Statistical analysis: Statistical evaluation between the groups showed that the basal mean HR between Group I and Group II was statistically significant(p=0.001), at pre induction, induction, 1, 3, 5 and 10 minutes after intubation the HR changes were statistically highly significant (p=0.000). Maximum HR changes were observed at 1 min after intubation in both the Groups. In Group I there was 12.34% decrease in HR compared to basal. In Group II there was 6.84 % increase in mean HR compared to basal.

After getting the required information, the collected data were coded, tabulated and analysed. The various statistical techniques i.e. the mean, standard deviation and test of significance (t-test and chi-square-test) were used for drawing valid conclusions.

Statistical analysis done using student t-test. SPSS 13.0 software was used to calculate p value. P<0.05 was taken as statistically A descriptive analysis was done on all variables to obtain a frequency distribution. The mean + SD and ranges were calculated for quantitative variables. Continuous variables were compared by the Student t test. Proportions were analyzed with the chi-square test.

Results

- The study population consists of 100 patients posted for laryngoscopy. They were divided into two groups of 50 each. Group I received Dexmedetomidine and Group II received Lignocaine
- The following observations were made during the course of the study.

Table-1: Heart rate of patients at different times in two groups.

HR	Dexmedetomidine		Lignocaine		t-test	Significant
	Mean	SD	Mean	SD		
Baseline	79.76	9.027	84.92	7.777	-3.062	0.003**
Pre Induction	71.78	10.14	80.88	8.911	-3.65	0.00**
Induction	74.2	11.853	81.76	8.575	-4.76	0.00**
Intubation	75.62	7.767	82.82	7.461	-4.72	0.00**
1 Min	69.84	7.495	78.08	10.815	-4.42	0.00**
3 Min	67.4	6.902	80.8	10.128	-4.73	0.00**
5 Min	67.34	6.906	81.86	6.996	-10.44	0.00**
10 Min	68.2	8.337	82.34	8.019	-8.64	0.00**

*P<0.05 significant at 5% level of Significant, **P<0.01 Highly Significant at 1%

Table-2: Comparison of SBP in the both groups at different instances of time.

SBP	Dexmedetomidine		Lignocaine		t-test	Significant
	Mean	SD	Mean	SD		
Baseline	136.16	8.934	130.3	13.001	2.62	0.01
Pre Induction	126.9	13.989	126.54	11.189	0.14	0.88
Induction	120.9	12.218	122	12.392	-0.44	0.65
Intubation	115	14.463	117.44	19.551	1.9	0.59
1 Min	110.82	12.202	115.9	15.197	-1.84	0.068
3 Min	109.08	10.421	113.6	12.501	-1.96	0.05*
5 Min	108.88	6.384	116.52	10.023	-4.54	0.00**
10 Min	107.08	6.417	114.24	9.048	-4.56	0.00**

*P<0.05 significant at 5% level of significant, **P<0.01 highly significant at 1% LOS, p>0.05 not significant at 5% LOS

Table-3: Comparison of DBP in the both groups at different instances of time.

DBP	Dexmedetomidine		Lignocaine		t-test	Significant
	Mean	SD	Mean	SD		
Baseline	86.16	5.726	79.08	8.144	5.029	0.00*
Pre Induction	80.5	8.2	79.62	7.925	0.546	0.58
Induction	76.36	9.182	76.1	9.272	0.14	0.88
Intubation	74.18	12.834	80.3	12.068	-2.45	0.016*
1 Min	73.96	10.513	78.5	10.664	-2.14	0.035*
3 Min	73.06	10.895	66.28	9.143	3.37	0.001**
5 Min	67.22	7.04	71.3	12.236	-2.04	0.04*
10 Min	67.24	6.817	70.64	9.703	-2.02	0.046*

*P<0.05 significant at 5% level of Significant, **P<0.01 highly significant at LOS, p>0.05 not significant at 5% LOS

Table-4: Comparison of SPO_2 in the both groups at different instances of time.

SPO2	Dexmedetomidine		Lignocaine		t-test	Significant
	Mean	SD	Mean	SD		
Baseline	98	0.13	99	0.14	--	--
Pre Induction	98.04	0.198	98.14	0.351	-1.75	0.082
Induction	99	0.21	98.02	0.141	49	0.84
Intubation	97.94	0.373	98	0.14	-1.13	0.26
1 Min	98	0.32	98.02	0.473	-0.29	0.76
3 Min	99	0	99	0	--	--
5 Min	98	0	98	0	--	--
10 Min	99	0.21	97.96	0.198	-0.32	0.856

*P<0.05 significant at 5% level of Significant, **P<0.01 Highly Significant at 1%LOS, p>0.05 not significant at 5%LOS.

Table-5: Comparison of map in the both groups at different instances of time.

MAP	Dexmedetomidine		Lignocaine		t-test	Significant
	Mean	SD	Mean	SD		
Baseline	102.86	4.721	96.18	7.116	5.53	0.00**
Pre Induction	95.92	6.779	95.24	6.808	0.5	0.61
Induction	91.14	7.567	91.44	6.753	-2.09	0.83
Intubation	90.74	8.499	92.68	10.649	-1.007	0.31
1 Min	86.24	7.566	90.98	9.516	-2.75	0.007**
3 Min	85.04	8.478	82.04	7.486	1.87	0.064
5 Min	81.08	5.345	86.44	8.622	-3.73	0.00**
10 Min	80.56	4.811	85.14	7.451	-3.65	0.00**

*P<0.05 significant at 5% level of significant, **P<0.01 highly significant at 1% LOS, p>0.05 not significant at 5% LOS

The HR, SBP, DBP and MAP were recorded at 1 min, 3, 5 and 10 min after intubation. Heart rate in Dexmedetomidine at baseline was 79.76±9.027 BMP, after induction upto at 1 min, 3 min, 5 min and 10 min. It were 67.4±6.90 BPM, 67.34±6.90 BPM, 68.2±8.33 BPM respectively, lower as compared to Lignocaine and statistically significant (p = 0.00**)

SBP rate in Dexmedetomidine at Baseline was 136.16±8.94 bmp, after induction upto at 1 min, 3 min, 5 min and 10 min. It were 110.82±12.20 mmHg, 109.08±10.42 mmHg, 68.2±8.33 BPM, 108.88±6.38 mmHg and 107.08±6.41 mmHg respectively, lower as compared to Lignocaine and statistically significant (p = 0.01**) DBP rate in Dexmedetomidine at Baseline was 86.16±5.72 mmHg, after induction up to 3 min, 5 min and 10 min It were 73.06±1.89 mmHg, 67.22±7.04 mmHg and 67.24±6.8 mmHg respectively, lower as compared to Lignocaine and statistically significant (p = 0.01**)

MAP rate in Dexmedetomidine at Baseline was 102.86±4.72 mmHg, after induction up to 3 min, 5 min and 10 min It were 3, 5 and 10 minutes mean DBP values 85.04±8.47 mmHg, 81.08±5.34 mmHg, and 80.56±4.81 mmHg respectively, lower as compared to Lignocaine and statistically significant (p = 0.01**) The statistical analysis shows that, in Dexmedetomidine group, HR, SBP, DBP and MAP showed significant decrease throughout the study period, as compared to Lignocaine group.

Discussion

Direct laryngoscopy and endotracheal intubation following induction of anesthesia is almost always associated with hemodynamic stress response due to reflex sympathoadrenal discharge. Aim of the present study was to compare the efficacy of Lignocaine and Dexmedetomidine in attenuating the hemodynamic response to laryngoscopy and intubation.

A variety of anesthetic techniques and drugs are available to control the hemodynamic response to laryngoscopy and intubation. The method or drug of choice depends on many factors, including the urgency and length of surgery, choice of anesthetic technique, route of administration, medical condition of the patient, and individual preference. The possible solutions number as many as the medications and techniques available and depend on the individual patient and anesthesia care provider.

Kovac et al reviewed medications and techniques to guide the clinician in choosing the best methods to control the hemodynamic response to laryngoscopy and endotracheal intubation.

Shribman AJ et al also studied cardiovascular and catecholamine responses to laryngoscopy with and without tracheal intubation. Arterial pressure, heart rate and plasma noradrenaline and adrenaline concentrations were measured before and after induction and at 1, 3 and 5 min after laryngoscopy. There were significant and similar increases in arterial pressure and circulating catecholamine concentrations following laryngoscopy with or without intubation. Intubation, however, was associated with significant increases in heart rate which did not occur in the laryngoscopy-only group [1, 2]. Many pharmacological methods have been devised to reduce the extent of haemodynamic events, e.g. with high dose of opioids, alpha1 agonists and beta blockers, local anaesthetics like Lignocaine and vasodilator drugs like Nitroglycerine. Intravenous Lignocaine with its well established centrally depressant and anti-arrhythmic effect was found to minimize pressor response [3-7].

Similar studies were done by Stoelting RK et al who measured blood pressure and heart rate changes during short-duration laryngoscopy for tracheal intubation: influence of viscous or intravenous lidocaine and Adi MN et al Cardiovascular reactions to laryngoscopy and tracheal intubation following small and large intravenous doses of lidocaine. Following laryngoscopy and tracheal intubation, the 1.5 mg/kg dose afforded complete protection against cardiac arrhythmias of all types. The smaller dose was ineffectual in this respect. While the larger dose caused borderline protection against hypertension and tachycardia [8, 9].

Two similar studies on lidocaine were by Laurito CE et al who saw effects of aerosolized and/or intravenous lidocaine on hemodynamic responses to laryngoscopy and intubation. Wilson IG et al in their study saw sympathoadrenal responses to laryngoscopy and intubation: the effect of varying time of injection of lidocaine. There was a significant increase in heart rate of 21–26% in all groups. There was no significant increase in mean arterial pressure in response to intubation in any group of patients given Lignocaine before intubation, but in the placebo group, mean arterial pressure increased. Both studies elaborate the effect of lidocaine [10, 11]. Kindler CHetal did a double-blind, controlled clinical trial on effects of intravenous lidocaine and/or esmolol on hemodynamic responses to laryngoscopy and intubation, and to assess whether a combination of both drugs is more effective than either drug alone.

Systolic blood pressure and heart rate (HR) were recorded before induction, before injection of the first test drug, immediately before laryngoscopy, and 1, 2, and 5 minutes following intubation.

It was concluded that neither of the two doses of esmolol tested nor that of lidocaine affected the BP response. Only the combination of lidocaine and esmolol attenuated both HR and BP responses to tracheal intubation. In contrast to the present study, Miller CD et al found that i.v. Lignocaine fails to attenuate the cardiovascular response to laryngoscopy and tracheal intubation. Analysis of variance for measured and derived cardiovascular variables failed to show any significant difference between any of the groups [12, 13]. Effect of Dexmedetomidine on haemodynamic responses to laryngoscopy and intubation is studied by many .one such study by Yildiz M et al which postulates that Dexmedetomidine reduces the dose requirements for opioids and anaesthetic agents.

The purpose of this study was to evaluate the effect of a single pre-induction intravenous dose of Dexmedeto-midine 1 μ/kg on cardiovascular response resulting from laryngoscopy and endotracheal intubation, need for anaesthetic agent and perioperative haemodynamic stability. After this study and other similar studies, it was concluded that preoperative administration of a single dose of Dexmedetomidine resulted in progressive increases in sedation, blunted the haemodynamic responses during laryngoscopy, and reduced opioid and anaesthetic requirements. Furthermore, Dexmedeto-midine decreased blood pressure and heart rate as well as the recovery time after the operation [14-16].

In a similar study, Dexmedetomidine as an adjunct to anesthetic induction to attenuate hemodynamic response to endotracheal intubation in patients undergoing fast-track CABG was studied by Menda F et al During induction of general anesthesia hypertension and tachycardia caused by tracheal intubation may lead to cardiac ischemia and arrhythmias. In this prospective, randomized study, Dexmedetomidine has been used to attenuate the hemodynamic response to endotracheal intubation with low dose fentanyl and etomidate in patients undergoing myocardial revascularization receiving beta blocker treatment. It is concluded that Dexmedetomidine can safely be used to attenuate the hemodynamic response to endotracheal intubation in patients undergoing myocardial revascularization receiving beta blockers [17].

El-shmaa ns et al studied the efficacy of Labetalol vs Dexmedetomidine for attenuation of hemodynamic stress response to laryngoscopy and endotracheal intubation In a similar comparison of lidocaine, fentanyl, and esmolol for attenuation of cardiovascular response to laryngoscopy and tracheal intubation was done by Feng CK In order to alter the hyperdynamic consequences resulting from intubation during induction of general anesthesia, the authors chose esmolol, an ultra-short acting cardio selective beta-adrenergic blocker, to attenuate the cardiovascular responses during tracheal intubation in patients undergoing elective surgery.

Results of this study showed that only esmolol could reliably offer protection against the increase in both HR and SBP, low dose of fentanyl (3 micrograms/kg) prevented hypertension but not tachycardia, and 2 mg/kg lidocaine had no effect to blunt adverse hemodynamic responses during laryngoscopy and tracheal intubation [18, 19]. The study by Lee JH, Kim Hwas designed to compare the effect of Dexmedetomidine, and Remifentanil used in anesthetic induction on hemodynamic change after direct laryngoscopy and tracheal intubation. The systolic blood pressure, diastolic blood pressure and heart rate were recorded from entrance to operation room to 5 min after tracheal intubation. The percent increase in systolic and diastolic blood pressure due to tracheal intubation in group D and R were significantly lower than that of group C ($P < 0.05$).

The heart rate 1 min after tracheal intubation was lower in groups R and D than in the group C ($P < 0.05$). So, they concluded that in healthy normotensive patients, the use of Dexmedetomidine during anesthetic induction suppressed a decrease in blood pressure due to anesthetic induction and blunted the hemodynamic responses to endotracheal intubation [20].

Reddy SV et al did a randomized double-blind clinical study comparing Dexmedetomidine versus esmolol to attenuate the hemodynamic response to laryngoscopy and tracheal intubation. Heart rate (HR), systemic arterial pressures were recorded at baseline, after study drug infusion, after induction, immediately and 3, 5, 7, 10 min after intubation. Both the drugs attenuated the pressure response. It was concluded that of the two drugs administered, Dexmedetomidine 1.0 µg/kg provides a consistent, reliable and effective attenuation of pressure responses when compared to esmolol 2.0 mg/kg [21].

The aim of the study by Hancı V et al was to compare the effects of fentanyl or Dexmedetomidine when used in combination with propofol and lidocaine for tracheal intubation without using muscle relaxants. The intubation conditions were significantly more satisfactory in Group D than in Group F ($p= 0.01$). Heart rate was significantly lower in Group D than in Group F after the administration of the study drugs and intubation ($p< 0.05$).

Mean arterial pressure was significantly lower in Group F than in Group D after propofol injection and at 3 and 5 minutes after intubation ($p< 0.05$). After intubation, the rate pressure product values were significantly lower in Group D than in Group F ($p< 0.05$). Therefore, they concluded that endotracheal intubation was better with the Dexmedetomidine–lidocaine–propofol combination than with the Fentanyl–Lidocaine–Propofol combination. However, side effects such as bradycardia should be considered when using Dexmedetomidine [22].

Gulabani M et al did Comparative analysis of efficacy of Lignocaine 1.5 mg/kg and two different doses of Dexmedetomidine (0.5 µg/kg and 1 µg/kg) in attenuating the hemodynamic pressure response to laryngoscopy and intubation. There is no study comparing the efficacy of Lignocaine with two different doses of Dexmedetomidine for attenuating the pressor response. With this idea, the authors planned to conduct the present study. Dexmedetomidine 1µg/kg was more effective than 0.5µg/kg and Lignocaine 1.5mg/kg in attenuating the pressor response. It was concluded that Dexmedetomidine 1µg/kg adequately attenuates the hemodynamic response to laryngoscopy and endotracheal intubation when compared with Dexmedetomidine 0.5µg/kg and Lignocaine 1.5mg/kg [23].

Prasad SR et al did a study similar to the present study and did comparison of intravenous Lignocaine and intravenous Dexmedetomidine for attenuation of hemodynamic stress response to laryngoscopy and endotracheal intubation. Their prospective randomized study was carried out in a tertiary care teaching hospital Thiopentone dose requirement was 19% less in group D compared to group L ($P< 0.001$). So, they concluded that Dexmedetomidine attenuates the hemodynamic stress response to laryngoscopy and intubation more effectively compared with Lignocaine without any deleterious effects.

Furthermore, Dexmedetomidine decreases dose of Thiopentone for induction of anesthesia. Their findings are similar to the present study findings [24].

Limitation of the present study

01. Small sample size
02. Chances of bias
03. Single center trial

Conclusion

Dexmeditoedine has added advantages offered like its effect on reducing anxiety, produce sedation without any undesirable side effects like respiratory depression, ability to reduce overall anaesthetic requirements and property of potentiating post-operative analgesic regimens. It also has got significant antisialogogue effect. Thus, Dexmeditoedine is a better drug compared to Lignocaine in attenuating pressure response to laryngoscopy & intubation.

What this study adds to the existing knowledge?

Laryngoscopy and intubation cause an intense reflex increase in heart rate, blood pressure, due to sympathoadrenal pressor response. Lignocaine has shown blunting of pressor response to intubation. Dexmedetomidine attenuates central sympathetic outflow & hyperadrenergic response such as surgical stress response. It is seen that the decrease in reflex sympathetic stimulation is highly significant with Dexmedetomidine as compared to Lignocaine.

Author's contribution

Dr. Sujit Pingle: Concept and Data collection

Dr. Pallavi Sharma: Data Analysis and Discussion

Dr. Pradeep Dhumane: Supervised the study

Dr. Amrusha Raipure: Data collection

Dr. Abhay Sharma: Concept and Data collection

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