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ATTENUATION OF HAEMODYNAMIC RESPONSE TO LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION WITH INTRAVENOUS DEXMEDETOMIDINE AND LIGNOCAINE: A COMPARATIVE STUDY

Anaesthesiology

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ABSTRACT

Introduction: Direct laryngoscopy and endotracheal intubation are stressful conditions and is almost always associated with haemodynamic changes. These haemodynamic responses are transient and harmless in healthy individuals but may be harmful to patients having reactive airways, hypertension, coronary artery disease, myocardial insufficiency and cerebrovascular diseases. **Aims:** The study aimed to compare the efficacy of 0.75 mcg/kg of IV dexmedetomidine and 1.5 mg/kg of IV lignocaine in attenuating the haemodynamic response to laryngoscopy and endotracheal intubation. **Materials and Methods:** After obtaining approval from the Institutional Ethis Committee, 64 ASA I and II patients of both sexes, between the age group 18 to 60 years, scheduled for elective surgeries under general anaesthesia were enrolled in the study. Patients were randomly divided into two groups Group L and Group D with 32 patients each. Group D received dexmedetomidine 0.75 mcg/kg IV diluted to 5 ml , Group L received lignocaine 1.5 mg/kg IV diluted to 5 ml with normal saline 5 min prior to induction. Haemodynamic parameters were recorded during the basal period, during intubation, 1 min, 2 min, 3 min and 5 min after intubation. **Results:** The demographic parameters were comparable in the two groups. The HR, SBP, DBP and MAP decreased significantly in Group D while there was an increase in these parameters in Group L. **Conclusion:** Dexmedetomidine IV 0.75 mcg/kg effectively attenuated the haemodynamic response to laryngoscopy and direct laryngoscopy when compared to Lignocaine IV 1.5 mg/kg.

KEYWORDS

Dexmedetomidine, Lignocaine, Haemodynamic response, Laryngoscopy and intubation

INTRODUCTION

Laryngoscopy and endotracheal intubation are frequently performed procedures in the practice of anaesthesia and are most stressful conditions to which the patient is subjected. Direct laryngoscopy and endotracheal intubation following induction of anaesthesia is almost always associated with haemodynamic changes due to reflex sympathetic discharge caused by stimulation of larynx, pharynx, epipharynx and trachea which are extensively innervated by autonomic nervous system. This increased sympathoadrenal activity may result in hypertension, tachycardia and arrhythmias.[1,2] These haemodynamic responses are transient and usually do not produce any consequence in healthy individuals but may be harmful to patients having reactive airways, hypertension, coronary artery disease, myocardial insufficiency and cerebrovascular diseases,[3] which may precipitate myocardial infarction, acute heart failure and even cerebrovascular accidents[.4] Extensive research work has been done to prevent or at least attenuate these responses.

Several drugs and manoeuvres have been used for mitigating the stress response with variable benefits and side effects.[5] Dexmedetomidine, introduced in 1999 for human use is a selective α2 adrenergic agonist. Its sedative, hypnotic and antinociceptive properties are due to its agonism of the presynaptic α2 adrenergic receptor which blocks the release of norepinephrine, thus terminating the pain signals and inhibits sympathetic activity which contributes in decreasing the blood pressure and heart rate. It has 8 times more affinity for α2 adrenergic receptors compared to clonidine and possesses all the properties of α2 agonist without respiratory depression.

Few authors have used dexmedetomidine in doses of 0.5 and 1 mcg/kg and found it to be effective in attenuation of stress response to laryngoscopy and endotracheal intubation.[6,7] Although they found promising results, the higher dose of 1 mcg/kg of dexmedetomidine was associated with increased incidence of hypotension and bradycardia.[6,7] It was found that dexmedetomidine in a dose of 0.5 mcg/kg was effective in blunting the tachycardic response to intubation but incompletely attenuate the increase in systolic and diastolic blood pressure, while lignocaine 1.5 mg/kg is more effective than dexmedetomidine 0.5 mcg/kg in attenuating the increase in systolic and diastolic blood pressure.[8] Hence, this study was taken up to compare the efficacy of 0.75 mcg/kg of IV dexmedetomidine and 1.5 mg/kg of IV lignocaine in attenuating the haemodynamic response to laryngoscopy and endotracheal intubation.

MATERIALS AND METHODS

This randomized clinical trial was conducted in the Department of Anaesthesiology, at a Tertiary care centre, Imphal, Manipur from September 2019 to August 2021. After obtaining approval from the Institutional Ethics Committee and Clinical Registry of India, 64 ASA I and II [9] patients of both sexes between the age group 18 to 60 years scheduled for elective surgeries under general anaesthesia were enrolled in the study. Patients with uncontrolled hypertension and diabetes, cardiac, coronary, renal, hepatic, cerebral diseases, peripheral vascular diseases, bradycardia, morbid obesity, history of allergy to the study drugs, patients in whom intubation attempts lasted longer than 15 seconds and anticipated difficult airway were excluded from the study.

Patients were randomly divided into two groups Group L and Group D with 32 patients each using a computer generated randomization. Group L received Lignocaine 1.5 mg/kg IV diluted to 5 ml with normal saline and Group D received Dexmedetomidine 0.75 mcg/kg IV diluted to 5 ml with normal saline, 5 min prior to induction.

After securing intravenous access in preoperative room, Ringer's Lactate solution (10 ml/kg) was given. Patients were pre-medicated with Inj. Glycopyrolate 0.004 mg/kg IV and Inj. Ondansetron 0.1 mg/kg IV. In the operation theatre, baseline parameters such as heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and oxygen saturation (SpO2)

were recorded.

Group L received Inj. Lignocaine 1.5 mg/kg IV diluted to 5 ml with normal saline and Group D received Inj. Dexmedetomidine 0.75 mcg/kg IV diluted to 5 ml with normal saline 5 min prior to induction, while preoxygenation was started in the last 3 min. Inj. Mephentermine 3 mg IV increment was given when systolic blood pressure (SBP) decreased by 20% of baseline. Bradycardia (heart rate less than 50/min) was treated with Inj. Atropine 0.6 mg.

Anaesthesia was induced with Inj. Propofol(1%) 2 mg/kg and endotracheal intubation was facilitated with Inj. Succinylcholine 1.5 mg/kg. Inj. Fentanyl 2 mcg/kg was given after 5 min of observation of stress response to intubation. Anaesthesia was maintained using 40% Oxygen and 60% Nitrous oxide, Sevoflurane and Inj. Atracurium. At the end of the surgery, patients were reversed with Inj. Neostigmine 0.05 mg/kg IV and Inj. Glycopyrolate 0.008 mg/kg IV. Patients were extubated after regaining consciousness and protective airway reflexes. Haemodynamic parameters were recorded during the basal period, during intubation, 1 min, 2 min, 3 min and 5 min after intubation and were analysed. Other associated side effects were also recorded.

Sample size was calculated based on a study[17] by Prasadh SR et al where it was 30 participants for each group at α value of 0.05 and power of 80%. Considering 5% drop out rate, we took 32 patients in each group with the total sample size of 64. Data collected were entered in IBM SPSS statistics version 21 for windows [IBM Corp.1995,2012]. Appropriate statistical method was used for analysis of data. Quantitative data were analysed using Student's t-test, qualitative data were analysed using Chi-square test. P value <0.05 was considered as statistically significant.

RESULTS:

The two groups were comparable in patient characteristics with respect to age, weight, sex and ASA physical status and statistically not significant (p value >0.05). [Table 1]

Table 1: Demographic Parameters

Parameter	Group D	Group L	P value
Age	39.81±10.66	37.66±12.28	0.456
Weight	62.66±7.005	60.56±9.15	0.308
Sex (M/F)	16/16	15/17	0.802
ASA (I/II)	26/6	29/3	0.281

P<0.05 is significant

It was observed that in group D, heart rate fell significantly during intubation and continued to fall below the baseline till 5 min after intubation. However in group L, heart rate increased significantly during intubation and 1 min, 2 min, 3 min and 5 min after intubation as shown in (Table 2).

Table 2: Comparison Of Mean Heart Rate In Between The Two Groups:

HR	Group D	Group L	p value
HRb	85.03±10.688	83.75±11.601	0.647
HRi	83.72±14.315	94.88±13.013	0.002
HR1	81.56±12.664	95.16±12.242	0.000
HR2	79.84±11.857	92.00±12.197	0.000
HR3	77.94±11.219	89.03±11.378	0.000
HR5	75.59±10.210	84.97±9.386	0.000

P<0.05 is significant

In group D, there was significant decrease in SBP during intubation and after intubation. SBP remained below the baseline during intubation, 1 min, 2 min, 3 min and 5 min after intubation. In group L, there was significant increase in SBP during intubation and remained above the baseline till 3 min after intubation. The rise in SBP was maximum during intubation (Table 3). DBP (Table 4) and MAP (Table 4) also showed similar trend.

Table 3: Comparison Of SBP In Between The Two Groups

SBP	Group D N=32	Group L N=32	p value
SBPb	124.28±5.056	125.22±8.983	0.609
SBPi	122.25±11.573	149.91±12.475	0.000
SBP1	120.34±13.420	142.81±12.235	0.000
SBP2	116.44±11.276	133.56±13.60	0.000
SBP3	112.34±8.705	126.44±13.645	0.000
SBP5	109.03±7.490	119.59±12.165	0.000

P<0.05 is significant

Table 4: Comparison Of DBP In Between The Two Groups

DBP	Group D N=32	Group L N=32	p value
DBPb	82.56±4.064	82.22±4.689	0.755
DBPi	82.91±9.323	97.03±9.160	0.000
DBP1	79.25±11.514	93.34±10.133	0.000
DBP2	75.78±9.648	87.69±12.556	0.000
DBP3	72.03±6.944	82.47±10.776	0.000
DBP5	69.44±4.272	75.59±11.600	0.008

P<0.05 is significant

Table 5: Comparison Of MAP In Between The Two Groups

MAP	Group D	Group L	p value
MAPb	96.41±4.055	95.88±6.349	0.691
MAPi	95.50±9.308	112.16±9.436	0.000
MAP1	92.47±12.287	108.19±10.391	0.000
MAP2	89.91±9.888	102.56±12.420	0.000
MAP3	85.25±6.834	96.56±11.924	0.000
MAP5	81.94±5.092	90.06±10.839	0.000

P<0.05 is significant

DISCUSSION

Laryngoscopy and endotracheal intubation are considered as the most critical events during general anaesthesia as they provoke transient but marked sympathoadrenal response. The response is due to reflex sympathetic discharge caused by stimulation of larynx, pharynx, epipharynx and trachea which are extensively innervated by autonomic nervous system.[1,2] This stimulation can cause increase in heart rate, blood pressure, intraocular pressure and intracranial pressure. Though well tolerated in healthy adult patients it can have catastrophic consequences in patients with coronary artery disease and cerebrovascular diseases.[10] Extensive research work has been done to prevent or at least attenuate these responses.

Lignocaine was used in many studies for attenuation of haemodynamic stress response. This drug is presumed to cause myocardial depression, peripheral vasodilatation and by increasing depth of anaesthesia it may help in suppressing the cardiovascular response to laryngoscopy and intubation.[11] Of all the various routes of administration, intravenous administration is acclaimed to produce maximum suppression. The usual dose ranges from 1 to 2 mg/kg and 1.5 mg/kg is used commonly in most of the studies.[12,13]

Few authors have used dexmedetomidine doses of 0.5 and 1 mcg/kg and found it to be effective in attenuation of stress response to laryngoscopy and endotracheal intubation[.6,7] Although they found promising results, the higher dose of 1 mcg/kg of dexmedetomidine was associated with increased incidence of hypotension and bradycardia.[6,7] It was found that dexmedetomidine in a dose of 0.5 mcg/kg was effective in blunting the tachycardic response to intubation but incompletely attenuate the increase in systolic and diastolic blood pressure. Haemodynamic responses following dexmedetomidine infusion depends on dose and speed of infusion.[14] A sequence of transient hypertension with reflex bradycardia followed by hypotension is seen with higher dose and rapid infusion.[15] The subsequent decrease in heart rate and blood pressure may be due to decrease in central sympathetic outflow.[16]

The present study was taken up to compare the efficacy of these two drugs in attenuating haemodynamic response to laryngoscopy and endotracheal intubation. In our study, we used 1.5 mg/ kg lignocaine in Group L and found that there was statistically significant increase in mean heart rate during intubation and till 5 min after intubation and this was in accordance with the study conducted by Prasad SR et al.[17] It was also found that there was increased in mean SBP, DBP and MAP during intubation, 1 min, 2 min and 3 min after intubation and then decreased at 5 min after intubation. This increase in SBP, DBP and MAP during intubation, 1 min, 2 min and 3 min after intubation was significant. The decrease in SBP, DBP and MAP at 5 min after intubation is also significant. These findings correlated with the

findings of Samala S et al.[18]

From these observations we can say that lignocaine did not effectively attenuate haemodynamic response to laryngoscopy and intubation and this is in accordance with the study conducted by Prasad SR et al.[17] This in in contrast with some investigators[19,20] who found lignocaine 1.5 mg/kg IV to effectively attenuate the stress response to laryngoscopy and endotracheal intubation.

In a study conducted by Kalakeri S et al[21], they used dexmedetomidine 1 mcg/kg to attenuate cardiovascular stress response to endotracheal intubation in patients undergoing cardiac surgery and found that there was statistically significant decrease in heart rate, SBP, DBP and MAP compared to baseline value after drug administration and before induction. These variables remained below the baseline even after intubation. The incidence of bradycardia requiring active intervention was 4.34%. In the present study, dexmedetomidine 0.75 mcg/kg was given in Group D and we found that there was statistically significant decrease in mean heart rate during intubation and remained below the baseline after intubation till the end of our observation, ie. 5 min after intubation. There was no patient who required treatment for bradycardia. SBP, DBP and MAP also decreased during intubation and remained below the baseline till the end of our observation. These changes could be due to α2 agonist effect of dexmedetomidine which decreases central sympathetic outflow.

Sebastian B et al[22] compared two doses of dexmedetomidine 0.5mcg/kg and 0.75 mcg/kg in attenuating haemodynamic stress response and found that a dose of 0.75 mcg/kg was more effective. The HR, SBP, DBP and MAP fell below the baseline by 3 and 5 min after intubation with a dose of 0.75 mcg/kg. Their findings correlated with the findings of our study. In some studies[6,7,21] dexmedetomidine in a dose of 1 mcg/kg was found to be associated with increased incidence of bradycardia and hypotension. No untoward side effects were observed in both the study groups and no patient required active treatment.

Thus, dexmedetomidine 0.75 mcg/kg IV effectively attenuated haemodynamic stress response to direct laryngoscopy and endotracheal intubation without any adverse effects compared to lignocaine 1.5 mg/kg IV.

LIMITATIONS

The study had some inherent limitations. Our study would have been more useful if we had included high risk hypertensive and cardiac patients. We could not measure plasma catecholamine level which could have demonstrated the effectiveness of this dosage of dexmedetomidine in decreasing the sympathetic nervous system activity. Lastly, we terminated our study at 5 min after intubation, but the effects of the study drugs particularly that of dexmedetomidine can persist for long which can have important clinical implications.

CONCLUSION

Intravenous dexmedetomidine at the dose of 0.75 mcg/kg effectively attenuates the haemodynamic response to direct laryngoscopy and endotracheal intubation when compared to intravenous lignocaine at the dose of 1.5 mg/kg.

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