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A COMPARATIVE STUDY ON EFFICACY OF LIGNOCAINE AND DEXMEDETOMIDINE IN ATTENUATING THE PRESSOR RESPONSE TO LARYNGOSCOPY AND INTUBATION

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ABSTRACT The present study was designed to compare the efficacy of Lignocaine and Dexmedetomidine in attenuating the pressor response to larynoscopy and intubation. 120 ASA I and II adult patients scheduled for elective surgery under general anesthesia were randomly divided into 2 groups of 60 patients each irrespective of age and sex into group L (Lignocaine) and group D (Dexmedetomidine) to receive either intravenous lignocaine 1.5 mg/kg body weight 3 minutes prior to intubation (Group L) or to receive intravenous dexmedetomidine 0.75 μg/kg body weight as infusion started 10 minutes before induction (Group D).

The heart rate, systolic, diastolic and mean arterial pressures were recorded basal (on entering the operation theater) before administration of study drug, after administration of the study drug, after induction and at 0, 2, 5, and 10 minutes after intubation. In both the groups there was definite rise in heart rate and blood pressure after intubation. Following administration of the study drug there was decrease in heart rate and mean arterial pressure in both the groups. There was decrease in heart rate after induction of anaesthesia which was greater in dexmedetomidine group compared to lignocaine group whereas the decrease in mean arterial pressure was lesser in dexmedetomidine group compared to lignocaine group. There was wide variation in heart rate and mean arterial pressure in lignocaine group compared to dexmedetomidine group.

The patients belonging to dexmedetomidine group were sedated but could be aroused easily on verbal command as compared to patients belonging to lignocaine group who were not sedated.

KEYWORDS : Anaesthesia, Intubation, Pressor Response, Dexmedetomidine, Lignocaine.

INTRODUCTION

Most of the patients undergoing surgery under general anesthesia require laryngoscopy and tracheal intubation. It forms the basic integral part of balanced anesthesia wherein the airway is maintained, controlled ventilation of the patients and delivery of anesthetic gases is achieved.[1]

The circulatory response to laryngoscopy and intubation was first documented by Reid and Brace in 1940 and later by King et al in 1951 and consists of increase in arterial blood pressure and heart rate.[2] The magnitude of haemodynamic changes observed may be dependent on various factors such as depth of anaesthesia, whether any measures are taken prior to airway manipulation, the anaesthetic agent used, the duration of laryngoscopy and intubation. To date, exact mechanism of haemodynamic responses to laryngoscopy and intubation has not been fully understood. However the principle mechanism causing tachycardia and hypertension is the stimulation of sympathetic system[3-4] which results in increase in catecholamine activity.[5] The increase in the pulse rate and blood pressure are usually transitory, variable and unpredictable. Transitory hypertension and tachycardia are probably of no consequence in healthy individuals but either or both may be hazardous to those with cardiovascular or cerebrovascular diseases. Intravenous anaesthetic inducting agents do not adequately or predictably suppress the circulatory responses evolved by endotracheal intubation. Various pharmacological agents have being tried in past to attenuate this pressor response. They include topical anaesthesia of oropharynx (viscous lignocaine), laryngo-tracheal instillation of lignocaine, intravenous lignocaine, opioids, vasodilators like sodium nitroprusside and nitroglycerine, calcium channel blockers, adrenergic blocking drugs (either alpha or β-blockers).

Ideally the drug which is selected to attenuate this response should be safely usable in any patient, prevent impairment of cerebral blood flow and avoid awareness in the patient. It should neither be time consuming nor affect the duration or modality of the ensuing anaesthesia and should not have any effects on the recovery characteristics. Many methods to attenuate this stress response have been studied, among which intravenous lignocaine, one of the oldest and most easily available drug, has been popular, probably because of its theoretical advantages of suppressing cough reflex,[6] preventing increases in intracranial pressure,[7] attenuating circulatory responses[8] and its antiarrhythmic properties. Lignocaine is an aminoethylamide and prototype of amide local anesthetic group.[9] An intravenous dose of Lignocaine 1.5 mg/kg body weight given 3 min prior to intubation has shown near optimal results.[10,11]

Dexmedetomidine,[12,13] a newer alpha2 agonist agent introduced in India in 2009, is highly specific and selective for alpha2 adrenoceptor agonist with alpha2:alpha1 binding selectivity ratio of 1620:1.[14] Dexmedetomidine has been used in various dose ranging from 0.5mcg/kg to 1 mcg/kg body weight and is becoming popular because it effectively decrease the haemodynamic response to laryngoscopy and intubation.[15-16] Bradycardia is a well-known side effect of dexmedetomidine Most of the studies have used dexmede to midine in a dose of 1 mcg/kg body weight but there are studies using lower doses as well quite effectively. We wanted to use a lower dose of dexmedetomidine (0.75 mcg/kg body weight) and check whether it is effective in suppressing the pressor response to laryngoscopy and intubation with lower incidence of bradycardia.

We designed a study to know the to compare the efficacy (along with side effects) of intravenous dexmedetomidine in the dose of 0.75 mcg/kg body weight compared with intravenous lignocaine 1.5 mg/kg body weight in attenuating the pressor responses to laryngoscopy and endotracheal intubation. We also wanted to study the effect of both drugs on the level of sedation before induction of anaesthesia.

MATERIAL AND METHOD

In 2015, Prasad et al.[17], conducted a prospective randomized study on 100 ASA I and II patients posted for elective surgery

under general anaesthesia, comparing the efficacy of lignocaine and dexmedetomidine in attenuating the hemodynamic response to laryngoscopy and endotracheal intubation. Sample size for this study has been calculated for assessing the difference between the two means at power 90% and confidence interval of 95% (alpha 0.05) with ratio of sample size in each group to be 1:1. The value of mean arterial pressure (MAP) at 5 minutes after intubation as found in the study conducted by Prasad et al.[17], was used to calculate sample size for the present study. The values were entered into Open Epi Online Calculator Version 3.03[18] and the sample size was found to be 51 in each group. Therefore 60 patients were selected for each group (total of 120 patients studied).

With institutional ethics committee approval and written, informed consent, 120 patients (ASA 1-2, aged 18-60 yr, having airway Mallampatti grade I and II) undergoing elective surgery under general anaesthesia with endotracheal intubation were randomly allocated (based on a computer generated random table) into two group, group L and group D. Patients in group L received intravenous Lignocaine 1.5 mg/kg body weight and patients in group D received Dexmed e tomidine 0.7 mcg/kg body weight. Patients were excluded if they were not willing to take part in the study, having anticipated difficult airway, with cardiovascular, renal, hepatic, central nervous system and peripheral vascular diseases.

A standard anaesthesia protocol was followed and routine monitoring applied. A baseline value (on entering the operation theater) of the heart rate, systolic, diastolic and mean arterial pressures were recorded before administration of study drug. After recording the baseline value, another reading was taken before administering any drug. All the patients were administered intravenous ondansetron 0.1 mg/kg body weight. Patients in group L received lignocaine 1.5m/kg body weight diluted in 10 ml normal saline 3 minutes before induction. Patients in group D received dexmed et omidine 0.75 mcg/kg body weight diluted in 10 ml normal saline intravenously over 10 min, prior to induction.

Patients were oxygenated during administration of study drug. Then patients received fentanyl 2 mcg/kg, and anaesthesia was induced with propofol 1% till loss of eyelash reflex. Muscle relaxation was achieved with atracurium 0.5 mg/kg after confirming that mask ventilation was possible. Laryngoscopy and intubation was done by a senior anaesthesia consultant using Macintosh blade, 3 minutes after administration of atracurium. All measures were taken so that the laryngoscopy did not last for more than 15 seconds. If time for laryngoscopy and intubation exceeded 15 seconds, such patients were excluded from the study.

Anaesthesia was maintained using 66% nitrous oxide and 33% of oxygen and 1% isoflurane. No surgical or any other stimulus was applied during the 10 min study period. At the end of the procedure muscle relaxation was reversed with neostigmine 0.05 mg/kg body weight and glycopyrrolate 0.01 mg/ kg body weight. The patients were extubated when they were fully awake and with good respiratory efforts.

The cardiovascular parameters like HR, SBP, DBP and MAP, along with sedation score (Ramsay Sedation Score) were monitored on entering at the operation theatre, before administration of drug, after administration of study drug, after induction of anaesthesia, immediately after laryngoscopy and intubation and 2, 5, 10 minutes after laryngoscopy and intubation.

Hypotension was defined as SBP ≤ 20% of baseline value. Tachycardia was defined as HR > 25% of baseline value. Bradycardia was defined as HR < 45 beats/ minute. Any dysrrhythmia was defined as any ventricular or supra ventricular beat or any rhythm other than sinus rhythm seen on electro cardiogram was recorded in both the groups. Ramsay Sedation Score was checked as per following scale.

Score 1: Anxious or restless or both
Score 2: Cooperative, oriented and tranquil
Score 3: Responding to commands
Score 4: Brisk response to stimulus
Score 5: Sluggish response to stimulus
Score 6: No response to stimulus

DATA ANALYSIS

Descriptive study was used to calculate percentage, mean and standard deviation of heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure and sedation score. Age and body weight were compared between the study groups using Independent sample t test. Crosstabs were used mainly for analyzing gender distribution. Repeated measure ANOVA was used to compare variables like heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure and sedation score between the groups for p value. $p<0.05$ was considered as significant and $p<0.001$ was considered as highly significant.

OBSERVATIONS AND RESULT

The present study was conducted on 120 ASA I and II adult patients scheduled for elective surgery under general anesthesia. These patients were randomly divided into 2 groups of 60 patients each irrespective of age and sex into group L (lignocaine) and group D (dexmedetomidine) by a computer generated random table. Group L patients received Lignocaine 1.5 mg/k of body weight whereas group D patients received Dexmedetomidine 0.75 mcg/kg of body weight to attenuate the pressor response of laryngoscopy and intubation.

The patient demographic data in terms of age, gender, weight, height and BMI were comparable in both groups. [Table 1-3, Fig. 1].

Table 1: Age distribution in study groups

Age (years)	Group L (Lignocaine)		Group D (Dexmedetomidine)	
	No. of patients	Percentage	No. of patients	Percentage
16-25	13	21.67	08	13.33
26-35	16	26.67	16	26.67
36-45	16	26.67	13	21.67
46-55	11	18.33	14	23.33
56-60	04	6.66	09	15.00
Total	**60**	**100**	**60**	**100**

Figure 1: Simple bar chart representing Age-distribution of the patients

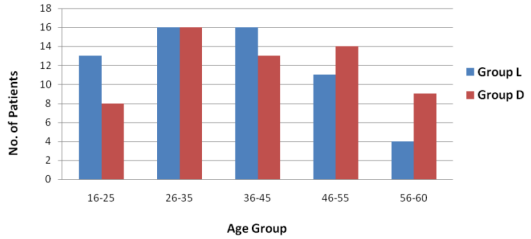


Table 2: Gender distribution in study groups

Sex	Group L (Lignocaine)		Group D (Dexmedetomidine)		p - value
	No. of patients	Percentage	No. of patients	Percentage	

Male	27	45	26	43	0.854
Female	33	55	34	57	
Total	60	100	60	100	

Table 3: Weight and Height distribution in study groups

	Group L (Lignocaine)	Group D (Dexmedetomidine)	p - value
Mean Weight (kg)	61.62 ± 11.085	58.22 ± 11.895	0.1080
Mean Height (cm)	161.4 ± 4.49	160.45 ± 4.3	0.2389

Table 4: Comparison of mean Heart Rate (bpm) changes after starting the drug infusion and in response to laryng oscopy and intubation between study groups

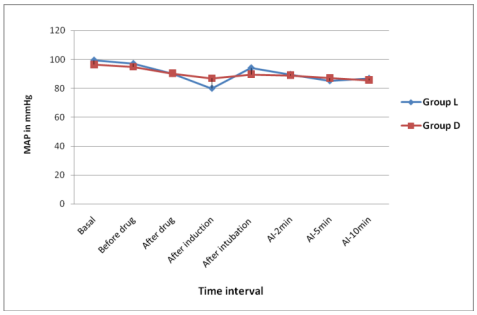
TIME	Group L (Lignocaine)	Group D (Dexmedetomidine)	p-value
	Mean HR ± SD	Mean HR ± SD	
Basal	82.13 ± 9.04	79.25 ± 10.23	0.105
Before drug administration	81.38 ± 9.05	77.65 ± 10.68	0.041
After drug administration	78.28 ± 9.95	70.03 ± 12.89	0.0001
After induction	77.37 ± 10.97	69.30 ± 11.39	0.0001
After intubation (AI)	86.13 ± 13.28	73.02 ± 11.85	0.0001
2 min AI	82.60 ± 14.48	74.25 ± 12.34	0.0010
5 min AI	78.25 ± 14.45	73.40 ± 12.53	0.052
10 min AI	76.78 ± 12.39	72.50 ± 12.30	0.060

The basal heart rate was comparable in both the groups (p =0.105). Statistical evaluation between the groups showed a significant fall in heart rate in both the groups after administration of the study drug and after induction which was highly significant compared to basal value. The mean heart rate increased in both the groups after intubation but the rise in heart rate was more in group L compared to group D which was highly significant (p=0001). In group L mean heart rate increased above the basal mean heart rate whereas in group D the rise in mean heart rate was below the basal mean heart rate. The heart rate observed at 2 minutes after intubation in group L was highly significant compared to mean heart rate in group D (p=0.001). The heart rate observed at 5 minutes after intubation in group L was significant compared to mean heart rate in group D (p=0.049). The heart rate observed at 10 minutes after intubation in group L was not significant compared to mean heart rate in group D (p=0.060). The heart rate observed at 0, 2, 5 and 10 min after intubation remained nearly constant in group D and did not showed much variation.

Table 5: Comparison of mean of Mean Arterial Pressure (MAP in mmHg) changes after administration of study drug and in response to laryngoscopy and intubation in study groups

TIME	Group L (Lignocaine) Mean MAP ± SD	Group D (Dexmedetomidine) Mean MAP ± SD	p-value
Basal	99.42±9.92	96.52±8.81	0.093
Before drug administration	97.08±9.53	94.93±9.13	0.209
After drug administration	90.22±10.39	90.43±9.00	0.906
After induction	80.02±12.88	86.92±8.98	0.001
After intubation (AI)	94.08±14.20	89.73±9.09	0.048
2 min AI	89.58±14.93	89.08±10.16	0.831
5 min AI	85.20±12.06	87.18±10.16	0.333
10 min AI	86.78 ±11.48	85.68±10.00	0.577

Figure 2: Comparison of mean arterial pressure (MAP in mmHg) changes after starting drug infusion and in response to laryngoscopy and intubation in study groups

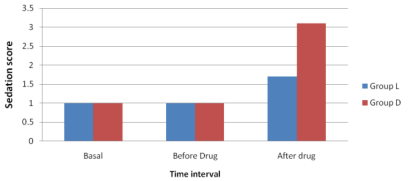


The mean basal MAP are comparable in both groups (p=0.093). The mean MAP decreased in both the groups after administration of the study drug and was statistically not significant (p= 0.906). After induction there was significant drop in mean MAP in group L compared to group D which was highly significant (p<0.001). After intubation there was significant increase in mean MAP in group L compared to group D from the induction level which was significant (p=0.048). At 2, 5 and 10 min after intubation mean MAP in both groups were lower than basal mean MAP and not significant (p=0.831), (p=0.333) and (p=0.577). There was a wide fluctuations in mean MAP in group L compared to group D.

Table 5: Comparison of Sedation scores (Ramsay score) changes in response to the drug between the study groups

Time	Group L	Group D	p-value
Basal	1	1	
Before drug administration	1	1	
After drug administration	1.7	3.1	<0.001

Figure 3: Comparison of Sedation Scores (Ramsay score) ch anges in response to administration of the drug between the study groups

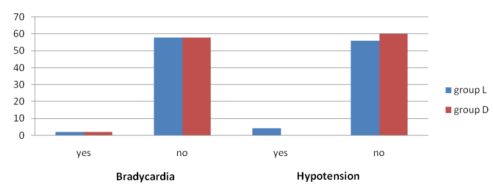


The mean basal sedation score are comparable in both groups. After administration of the study drug the change in sedation score was statistically highly significant with the p value of <0.001.

Table 7: Comparison of side effects between study groups

Group	Bradycardia		Total	Hypotension		Total
	Yes (%)	No (%)		Yes (%)	No (%)	
Group L	2 (3.33%)	58 (96.67%)	60	4 (6.67%)	56 (93.33%)	60
Group D	2 (3.33%)	58 (96.67%)	60	0 (0 %)	60 (100.0%)	60
Total	4	116	120	4	116	120
Chi-square	0.2586			2.3276		
p-value	> 0.05			> 0.05		

Figure 4: Bar- chart representing Complications in each Gro up of patients



In group L, 2 and 4 patients had side effects like bradycardia and hypotension respectively, whereas in group D, 2 patients had bradycardia and no incidence of hypotension was seen. The incidence of complication was insignificant.

DISCUSSION

Many of the surgical procedures in the modern day practice need to be managed under general anaesthesia with endotracheal intubation. It has long been observed that induction of anaesthesia and tracheal intubation may induce profound alteration of the haemodynamic state of the patient subsequent to both the effects of anaesthetic agents administered pre-operatively and the adrenergic state of the patient.[19]

Laryngoscopy and tracheal intubation are considered as the most critical events during administration of general anaesthesia as they provoke transient but marked sympatho-adrenal response manifesting as hypertension and tachycardia. Reflex changes in cardiovascular system after laryngoscopy and intubation leads to an average increase in blood pressure by 36-45% and heart rate by 26-66%.[20]

The present study confirms that there is a definite increase in heart rate and mean arterial pressure following laryngoscopy and tracheal intubation in both the groups but the increase is attenuated following premedication by study drugs.

In 1988, Basu and Pramanik observed that these circulatory changes are maximum from 1st to 3rd minute following laryngoscopy and intubation and lasts up to 10 minutes. The present study confirms this and shows that the maximum increase in the pulse rate and blood pressure was immediately after laryngoscopy to 3 minutes following laryngoscopy and intubation.

In our study both the groups were comparable and there was no statistically significant difference with regards to mean age, sex, weight and height. Several authors, Abou-Madi et al., Splinter et al., Tam et al., Wilson et al., have concluded that intravenous 1.5 mg/kg of lignocaine suppresses the haemodynamic response to laryngoscopy and intubation when given 3 minutes before laryngoscopy and intubation. However, different dosages of intravenous dexmedetomidine have been in use for suppressing the haemodynamic responses to laryngoscopy and intubation. Since various authors found intravenous dexmedetomidine effective in a dose range from 0.5 mcg/kg to 1 mcg/kg body weight, we decided to use 0.75 mcg/kg dexmedetomidine as a dose for our study. In the present study, lignocaine was diluted in 10 ml of normal saline and administered intravenously as a bolus over 5 sec (given approximately 3 minutes before laryngoscopy) whereas dexmedetomidine was diluted in 10 ml of normal saline and administered intravenously over 10 minutes (infusion was started 10 minutes before induction).

As shown in table no. 4, which describes the changes in mean heart rate in response to administration of lignocaine and to laryngoscopy and intubation, there was significant decrease in the mean heart rate on administration of intravenous lignocaine and on induction of anesthesia but on intubation there was significant increase in mean heart rate. Following intubation at 2, 5 and 10 minutes there was decrease in the mean heart rate. Also shown in table no. 5, which describes the changes in mean arterial pressure in response to administration of lignocaine and to laryngoscopy and intubation, there was significant decrease in the mean arterial pressure on administration of intravenous lignocaine and on induction of anesthesia but on intubation there was significant increase in mean arterial pressure from the after induction value but showed a decrease compared to basal mean arterial pressure. Following intubation at 2, 5 and 10 minutes there was decrease in the mean arterial pressure.

In our study following intubation there was rise in mean arterial pressure by 17% from the post induction values but it was still lower by 6% from the baseline values. Whereas there was increase in heart rate by 11% and 4% respectively from the post induction values and baseline values. We found that lignocaine partially attenuated the haemodynamic response to laryngoscopy and intubation.

As shown in table no 4, heart rate decreased by 9.22 bpm and 6.23 bpm after administration of dexmedetomidine and following laryngoscopy and intubation respectively compared to the baseline values which is highly significant. Various authors[13,15,17] have found similar response following administration of intravenous dexmedetomidine and to laryngoscopy and intubation.

As shown in table no 5, there was decrease in mean arterial pressure by 6.09 mmHg compared to basal value. There was further decrease in mean arterial pressure after induction. Following intubation there was increase in mean arterial pressure by 2.82 mmHg compared to post induction value but continued to remain below the basal value by 6.79 mmHg. At 2, 5 and 10 minutes after intubation also the mean arterial pressure remained below the basal value. It is seen that dexmedetomidine effectively blunts the increase in blood pressure after intubation (AI), at 2, 5 and 10 minutes following laryngoscopy and intubation compared to baseline. Even at 10th minute the mean arterial pressure did not reach the basal value. It was 10.84 mmHg below than the basal value.

COMPARATIVE ANALYSIS OF HAEMODYNAMIC DATA BETWEEN THE GROUPS

As shown in table no 4 and 5, basal heart rate and mean arterial pressure were comparable in both the groups. The heart rate and mean arterial pressure were significantly lower in group D (dexmedetomidine) compared to group L (lignocaine) after drug administration, immediately after intubation and at end of 2, 5 and 10 min. after intubation and was highly significant. Also in group D, less variation in heart rate and mean arterial pressure was seen compared to group L to various events involved in administration of general anaesthesia right from induction to intubation and thereafter. Prasad et al.,[17] Gulabani et al.,[21] and Gangappa et al.[22] in their studies also noted that the heart rate and mean arterial pressure were significantly lower in group D (dexmedet omidine) when compared to group L (lignocaine). Our study was in accordance with their finding.

SEDATION SCORING

In our study after administration of the study drug, patients in group D (dexmedetomidine) had a sedation score of 3.1 ± 0.57 compared to group L (lignocaine) where the sedation score was 1.7 ± 0.59 which was highly significant. The patients in group D (dexmedetomidine) were well sedated but arousal on verbal command.

SIDE EFFECTS

In lignocaine group, 2 patients developed bradycardia and 4 patients developed hypotension while in dexmedetomidine group, 2 patients developed bradycardia. The incidence of complication was not significant. Bradycardia was treated by injection atropine in both the groups. No patient required vasopressors for correction of hypotension it was managed by decreasing the concentration of inhalation agent and infusion of intravenous fluid.

CONCLUSION

We concluded from our study that Dexmedetomidine 0.75 mcg/kg body weight intravenous as 10 minutes infusion attenuates the pressor response to laryngoscopy and

intubation more effectively compared to lignocaine 1.5 mg/kg body weight intravenous without any deleterious effects. Dexmedetomidine infusion produces sedation on infusion which mimics normal sleep pattern, patients are easily arousable to verbal commands and it lacks respiratory depression. Patients receiving dexmedetomidine had less variation in heart rate and mean arterial pressure to various events involved in administering general anaesthesia. Providing a stable haemodynamic parameter throughout the procedure. There was also minimal complication on administration of dexmedetomidine.

Limitations of this study were that double blinding was not possible due to different pharmacokinetics profile of the drug resulting in different timing of the drug administration.

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