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Research Article

Effect of intravenous dexmedetomidine (1µg/kg) in obtunding the pressor response to laryngoscopy and tracheal intubation compared to intravenous preservative free 2% lignocaine (1.5mg/kg)

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ABSTRACT

Background: Haemodynamic variation during layngoscopy/intubation is always a matter of concern for Anesthesiologists. A stable circulatory system is the wish of all performing general anaesthesia. Hence an acceptable and easy method needs to be established to prevent the haemodynamic variations. Our aim was to evaluate the effect of intravenous Dexmedetomidine (1µg/kg) infusion in obtunding the pressor response to laryngoscopy and tracheal intubation compared to intravenous preservative free 2% Lignocaine (1.5mg/kg).
Methods: In this study, 60 patients, aged between 18-60 years belonging to ASA I and II are included. They were randomly divided into 2 groups, each comprising of 30. In group D, patients were given Dexmedetomidine 1µg/kg IV infusion over 10min and in group L, patient were given Lignocaine 1.5mg/kg IV.
Results: In group D, the systolic, diastolic, mean arterial pressure and heart rate decreased significantly, from baseline, at first, second, third, fourth and fifth minute post intubation. In group L, the systolic, diastolic, mean arterial pressure and heart rate increased from baseline at first and second minute and then decreased at third, fourth and fifth minute post intubation.
Conclusions: When compared between the two groups all the hemodynamic parameters showed statistical significance. There are no significant side effects and severe haemodynamic variability like hypotension and bradycardia. Therefore we concluded that Dexmedetomidine is superior to lignocaine in blunting the hemodynamic response to laryngoscopy and endotracheal intubation without any significant side effects.

Keywords: Intubation, Haemodynamic changes, Dexmedetomedine, Lignocaine (Preservative free)

INTRODUCTION

Laryngoscopy and endotracheal intubation are frequently performed procedures in the practice of anaesthesia. These are most stressful conditions to which the patient is subjected. Standard technique of laryngoscopy and endotracheal intubation involves stimulation of larynx, pharynx, epipharynx and trachea, which are extensively innervated by autonomic nervous system, namely parasympathetic innervation via vagus and glossopharyngeal nerves and sympathetic via superior cervical ganglion. Direct laryngoscopy/tracheal intubation following induction of anaesthesia are almost always associated with hemodynamic changes due to reflex sympathetic discharge caused by epipharyngeal and laryngopharyngeal stimulation.[1] Circulatory response to laryngoscopy and intubation include increase in heart rate, arterial pressure, intracranial tension, intraocular pressure and cardiac dysrrhythmias.[2,3] In addition there can be cardiac asystole and coronary and cerebral infarction and haemorrhage. These adverse responses occur in a normal sequence of induction/intubation and

can further be aggravated by Light planes of Anaesthesia, Hypoxia, Hypercarbia, Anxiety and Reflex baroreceptor effect following induction agents like Thiopentone Sodium. Hypertensive patients are more prone to significant increases in blood pressure whether they have been treated before or not[4]. Haemodynamic changes are not serious enough in a normal individual but may be hazardous to patients with compromised circulatory system and cerebrovascular disorders.[5-7] Reid et al were first to report the circulatory responses to laryngeal and tracheal stimulation in anaesthetized man.[8] Extensive research work has been done to prevent or atleast attenuate these responses. Some of these are

Smooth, gentle intubation with shorter duration of laryngoscopy; Blocking glossopharyngeal, superior laryngeal nerves; Deep General Anaesthesia; Topical Lignocaine; Antihypertensive drugs like β-blockers, calcium channel blockers; Intravenous Lignocaine; Midazolam; Sodium Nitroprusside.[9]

The more recent techniques are
1. α_2 agoinst
I) Intra venous dexmedetomidine
II) Clonidine
- Oral
- Transdermal

III) Mivazerol
2. Nitroglycerine: Topical
3. I.V. Labetolol / Esmolol
- Ointment
- Transdermal patches

4. I.V. Fentanyl / Alfentanyl / Sufentanyl

This study was undertook to assess efficacy of intravenous dexmedetomidine premedication in attenuating the pressor response to laryngoscopy and intubation in Normotensive patients, compared to the control group with intravenous preservative free Lignocaine. Objectives of the study were to evaluate the effect of intravenous Dexmedetomidine (1µg/kg) infusion in obtunding the pressor response to laryngoscopy and tracheal intubation compared to intravenous preservative free 2% Lignocaine (1.5mg/kg) -A prospective, randomized, controlled and double blinded study.

METHODS

This study was carried out in the Department of Anaesthesiology, Mamata Medical College, Khammam from August 2015- Februry 2016. A total number of 60 patients, 30 in each group were included in this study. Patients were placed in two groups, Group D (pts. Given Dexmeditomidine 1mcg/kg) and Group L (pts. received Lignocaine 1.5mg/kg.

Inclusion criteria

- ASA I and II.
- Age between 18-60yrs, belonging to both sexes.
- Laryngoscopy and intubation time <15 second.
- Single attempt at intubation.

Exclusion criteria

- ASA Grade ≥III.
- Patient allergic to Dexmedetomidine or Lignocaine
- Patients age <18 or >60.
- Laryngoscopy and intubation lasting for >15 seconds
- More than one attempt at intubation.
- Patients with suspected airway anomalies or anticipated difficult airway.

All patients were explained in detail about the study and informed consent was taken. Peripheral intravenous line secured in the pre-operative holding area. The Anaesthesiologist who prepared and administered the drug was not involved with the intra and post-operative management of the patient. Electrocardiogram, pulse oximeter and noninvasive blood pressure monitoring was done. Baseline systolic blood pressure, diastolic blood pressure, mean arterial blood pressure and heart rate were recorded before induction of anaesthesia. Patients of Group D, received injection Dexmedetomidine 1mcg/kg diluted to 50 ml in normal saline and given as an infusion over 15 minutes before induction and then 10ml normal saline 90 seconds before intubation. Group L received infusion of plain normal saline in a 50ml syringe over 15 minutes and 1.5mg/kg preservative free I.V Lignocaine diluted to 10ml with normal saline 90 seconds before intubation.

Patients were premedicated with injection glycopyrrolate 0.005mg/kg, injection fentanyl 1mcg/kg, induced with injection thiopentone 5mg/kg, they were intubated with an appropriate size oral cuffed endotracheal tube after giving injection vecuronium 0.1mg/kg. Anaesthesia was maintained intra-operatively with Oxygen, Nitrous Oxide and Sevoflurane at 33%, 66% and 1.5-2% respectively. Neuromuscular blockade was maintained with incremental doses of vecuronium. The parameters recorded and analyzed for the study were heart rate, systolic blood pressure, diastolic blood pressure and mean arterial blood pressure. Readings were recorded as follows

- Baseline- in the pre-operative holding area before securing intravenous access
- 1 minute, 2 minute, 3minute, 4minute and 5minute after intubation.

Parameters defined for the study were.

Hypotension-defined as systolic blood pressure <25% of baseline value or less than 90 mmHg, whichever is lower, hypertension-defined as systolic blood pressure >25% of baseline value or greater than 150 mmHg, whichever is higher, tachycardia-defined as heart rate >25% of baseline value, bradycardia-defined as heart rate <25% of baseline value, Arrhythmia-defined as any rhythm other than sinus as well as incidence of all these parameters was recorded in both the groups.

Statistical data

All the data was statistically analyzed using diagrammatic representation and descriptive data presented as mean±SD, continuous data are analyzed by sample "t" test and chi-square test to assess the statistical difference between the 2 groups

RESULTS

Age, sex and weight distribution were comparable in both groups and statistically not significant. (Table 1, Table 2 and Table 3.)

Table 1: Age distribution.

	Group D	Group L
18-30	4	7
31-40	7	8
41-50	11	10
51-60	8	5
Mean age	44.33	40.33
Maximum age	60	60
Minimum age	25	21

The age distribution in group-D and group l was from 18-60. When chi squared the value is 1.625 with two degrees of freedom with P value 0.654 which is considered statistically not significant. Both groups were comparable in terms of sex distribution.

Table 2: Sex distribution.

Sex	Group D	Group L	Total
Male	16	16	32
Female	14	14	28
Total	30	30	60

Chi squared equals 0.267 with 1 degree of freedom. The two-tailed P 0.606. The association between rows (groups) and columns (outcomes) is considered to be not statistically significant (Table 2).

Table 3: Weight distribution.

Weight in kgs	GROUP D	GROUP L
41-50	3	5
51-60	8	10
61-70	7	7
> 70	12	8
Mean weight	69.76	65.86
Maximum weight	86	83
Minimum weight	52	51

The mean weight of patients in both the groups was comparable. There is no statistical significance between the groups with P value being 0.3281.

ASA grade

Both the groups are similar with respect to ASA Grade. When chi squared the value is 0.164 with one degree of freedom with P value >0.05 indicating that there is no statistical significance (Table 4). Both the groups are similar with respect to ASA Grade as is evident by the statistics below.

Table 4: ASA grade.

Grade	Group-D	Group-L	Total
Gr I	25	23	48
Gr II	5	7	12
Total	30	30	60

When chi squared the value is 0.164 with one degree of freedom with P value >0.05 indicating that there is no statistical significance.

Table 5: Comparison of systolic blood pressure between the two groups.

	Group	N	Mean	Std. deviation	P value
SBP.B	Group D Group L	30 30	136.27 138.13	14.246 15.040	0.6247
SBP.1M	Group D Group L	30 30	91.83 140.27	5.173 18.308	0.0001
SBP.2M	Group D Group L	30 30	89.13 135.38	4.64 17.41	0.0001
SBP.3M	Group D Group L	30 30	86.53 129.23	5.184 17.368	0.0001
SBP.4M	Group D Group L	30 30	84.83 125.7	4.09 16.54	0.0001
SBP.5M	Group D Group L	30 30	83.70 123.47	4.244 17.909	0.0001

SBP- Systolic blood pressure. b – baseline. 1m- first minute. 2m- second minute. 3m- third minute. 4m- fourth minute. 5m- fifth minute

Table 6: Comparison of diastolic blood pressure between the groups.

	Group	N	Mean	Std. deviation	P value
DBP.B	Group A Group B	30 30	80.40 78.23	7.942 8.597	0.3141
DBP.1M	Group A Group B	30 30	60.30 81.07	8.860 4.891	0.0001
DBP.2M	Group A Group B	30 30	58.13 78.30	7.62 5.91	0.0001
DBP.3M	Group A Group B	30 30	56.13 75.90	7.300 8.389	0.0001
DBP.4M	Group A Group B	30 30	54.73 75.43	6.36 7.36	0.0001
DBP.5M	Group A Group B	30 30	53.60 74.90	6.117 7.251	0.0001

DBP- Diastolic blood pressure. b- baseline. 1m- first minute. 2m- second minute. 3m- third minute. 4m- fourth minute. 5m- fifth minute.

Table 7: Comparison of mean arterial pressure between the groups.

	Group	N	Mean	Std. deviation	P value
MAP.B	Group A Group B	30 30	98.67 97.10	7.526 10.685	0.5132
MAP.1M	Group A Group B	30 30	70.57 100.63	6.882 8.438	0.0001
MAP.2M	Group A Group B	30 30	68.03 96.30	6.08 7.87	0.0001
MAP.3M	Group A Group B	30 30	65.97 92.03	5.798 9.554	0.0001
MAP.4M	Group A Group B	30 30	64.53 92.10	4.80 8.57	0.0001
MAP.5M	Group A Group B	30 30	63.33 92.77	4.490 9.662	0.0001

MAP- Mean arterial pressure**.** b- Baseline**.** 1m- first minute**.** 2m- second minute**.** 3m- third minute**.** 4m- fourth minute**.** 5m- fifth minute

Table 8: Comparison of heart rate between the groups.

	Group	N	Mean	Std. deviation	P Value
HR.B	Group A Group B	30 30	81.17 77.97	14.370 10.417	0.3275
HR.1M	Group A Group B	30 30	67.40 85.43	9.605 11.082	0.0001
HR.2M	Group A Group B	30 30	65.33 81.31	8.34 10.47	0.0001
HR.3M	Group A Group B	30 30	63.70 77.37	7.953 10.568	0.0001
HR.4M	Group A Group B	30 30	61.47 77.54	7.54 10.08	0.0001
HR.5M	Group A Group B	30 30	59.63 76.07	7.294 10.432	0.0001

HR- heart rate B- baseline. 1M- first minute. 2M- Second minute. 3M- Third minute. 4M- Fourth minute. 5M- Fifth minute.

DISCUSSION

General anaesthesia has almost become synonymous with endotracheal anaesthesia. Laryngoscopy and endotracheal intubation are considered as the most critical events during general anesthesia. They provoke a transient, but marked sympathetic and sympathoadrenal response. In our study we compared intravenous Lignocaine, a drug which has been successfully and widely used to blunt the hemodynamic response to intubation, with dexmedetomidine a newer α_2 agonist with additional properties such as sedation, anxiolysis and sympatholysis

for attenuating the hemodynamic response to laryngoscopy and tracheal intubation. All the baseline values systolic blood pressure, diastolic blood pressure, mean arterial pressure and heart rate are comparable between the two groups. There is statistically no significant difference in the baseline values between the two groups.

In Group D (Dexmedetomidine group) the systolic, diastolic, mean arterial pressure and heart rate decreased significantly from baseline at first, second, third, fourth and fifth minute post intubation. The change in systolic, diastolic, mean arterial pressure and heart rate from base line in group D is statistically significant and is comparable with the findings of Mendal F et al in their study.[10] They found that in dexmedetomidine group systolic, diastolic and mean arterial pressures were lower at all times i.e. after induction of general anesthesia, one, three and five minutes after endotracheal intubation. In our study, dexmedetomidine effectively attenuated the intubation response.

Scheinin B et al studied the effect of dexmedetomidine on intubation response.[11] They had similar findings correlating to our observation. In addition they also observed that the thiopentone dose required for induction is lesser in the dexmedetomidine group. In the present study they did not measure the dose required for induction. We have measured the clinical effect, whereas they have measured the biochemical effect.

Jaakola et al studied the effect of dexmedetomidine infusion on intraocular pressure, hemodynamic and sympathoadrenal responses to intubation in ophthalmic surgical patients.[12] The dosage used was 0.6 mcg/kg which was lesser than the dosage we used in our study. The dosage we used in our study was 1 mcg/kg. Their findings also correlated with present study/ observation.

Yildiz et al in their study found that need for Thiopental and Sevoflurane concentration were decreased by 39% and 92%, respectively, in the dexmedetomidine group compared with the placebo group.[13]

In all groups, blood pressure and heart rate increased after tracheal intubation; but both were significantly lower in the dexmedetomidine group than in the placebo group. This observation is comparable to our study. They also found that the arterial blood pressure and heart rate in post-operative period is significantly lower in the dexmedetomidine group.

Divya jain et al studied the effect of Dexmedetomidine in attenuating extubation response.[14] They also found similar findings correlating to our observation. Pekka et al studied the effect of dexmedetomidine on perioperative hemodynamic in vascular surgical patients.[15] The change in hemodynamic is less affected in Dexmedetomidine group. In the present study they had similar perioperative findings as Yildiz, Divya Jain and Pekka however they did not monitor extubation response and post-operative period.

In Group L (Lignocaine group), the systolic, diastolic, mean arterial pressure and heart rate increased from baseline at first and second minute and then decreased at third, fourth and fifth minute post intubation. This increase in systolic, diastolic, mean arterial pressure and heart rate at the first and second minute was statistically significant. The decrease in systolic, diastolic, mean arterial pressure and heart rate at third, fourth and fifth minute was statistically significant. These findings correlated with the findings of Malde AD et al.

Thus in group L, the systolic, diastolic, mean arterial pressure and heart rate increased from the baseline value at first and second minute after intubation but it is not significant. Malde AD et al compared the efficacy of fentanyl versus lignocaine in attenuating the pressure response to intubation.[16] They have used glycopyrrolate 0.2 mg as premedication prior to surgery. They had used halothane as the maintenance agent. They had similar observation in the lignocaine group.

Wilson et al showed that irrespective of timing of administration of injection of lignocaine at second, third or fourth minute before tracheal intubation, there was a significant increase in heart rate in all the groups.[17]

When comparing the hemodynamic parameters between the Dexmedetomidine Group (Group D) and Lignocaine Group (Group L), systolic, diastolic, mean arterial pressure and heart rate decreased from the baseline value after intubation in dexmedetomidine group, whereas in the lignocaine group there was an increase in the first and second minute, it was a significant change and decreased at third, fourth and fifth minute after intubation. All the hemodynamic parameters showed significant difference when comparing between the two groups. There are no significant side effects like severe hypotension and bradycardia.

CONCLUSION

Study concludes that dexmedetomidine and Lignocaine are effective in blunting the hemodynamic response to intubation, but dexmedetomidine is superior to Lignocaine in blunting the hemodynamic response to laryngoscopy and endotracheal intubation without any significant side effects.

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