ORIGINAL RESEARCH PAPER

Anaesthesiology

KEY WORDS: Intravenous Lignocaine, Dexmedetomidine, Attenuation Haemodynamic, Laryngoscopy, Tracheal Intubation, General Anaesthesia

COMPARATIVE STUDY BETWEEN INTRAVENOUS LIGNOCAINE AND DEXMEDETOMIDINE FOR ATTENUATION OF HAEMODYNAMIC RESPONSE TO LARYNGOSCOPY AND TRACHEAL INTUBATION IN PATIENTS UNDERGOING SURGERY UNDER GENERAL ANAESTHESIA

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ABSTRACT

Background Laryngoscopy and endotracheal intubation are undesirable stimuli that trigger the sympathetic nervous system. Various anaesthetic techniques and drugs have been tried to reduce the stress response. The aim of the study was to compare the efficacy of intravenous lignocaine 1.5 mg/kg and intravenous dexmedetomidine 0.5 μg/kg in attenuating the hemodynamic stress response (heart rate, systolic blood pressure) to laryngoscopy and intubation in patients undergoing general anaesthesia for elective surgeries. **Materials And Methods** A total of 100 patients of ASA grade I undergoing elective surgical procedures under general anesthesia requiring endotracheal intubation were randomly divided into two groups. Group lignocaine (Group L) received inj. Lignocaine 1.5mg/kg, 3 minutes before intubation and Group dexmedetomidine (Group D) received Inj. Dexmedetomidine 0.5 μg/kg 20 minutes before induction. The changes in heart rate, systolic blood pressure were assessed at baseline, pre-inducton, post-induction and at various time intervals of 0,1,3,5,7,10 minutes from the onset of laryngoscopy and intubation. **Results** The changes in heart rate, systolic blood pressure at post-induction and at various time intervals of 0, 1, 2, 3, 5, 7, 10 min from the onset of laryngoscopy and intubation in L and D group were statistically significant (P<0.05). At intubation heart rate and systolic blood pressure increased transiently but it came down gradually in both the groups with dexmedetomidine group having better control. None of the patient had bradycardia or hypotension. **Conclusion** 0.5 μg/kg dexmedetomidine given ten minutes before intubation can be effectively used to reduce the haemodynamic changes associated with laryngoscopy and endotracheal intubation.

INTRODUCTION

The gold standard for patient care in general anaesthesia is endotracheal intubation. Laryngoscopy and endotracheal intubation are undesirable stimuli that trigger the sympathetic nervous system, which might have a negative impact on the cardiovascular systems.[1] Tachycardia, hypertension, and arrhythmias occurred due to reflex sympathetic discharge brought on by mechanical stimulation of both the pharynx and larynx during direct laryngoscopy and intubation.[2,3] The cardiovascular reaction is directly influenced by the force and length of the laryngoscopy.[4] The sympathoadrenal response begins immediately after laryngoscopy and intubation and returns to baseline in 5 to 10 minutes. In healthy people, the brief rise in blood pressure and heart rate might not be hazardous. However, these reactions might result in consequences including pulmonary oedema, myocardial infarction, ventricular dysrhythmias or cerebrovascular accidents in individuals with hypertension, myocardial insufficiency or cerebrovascular illness.[5,6] Even in individuals with normal blood pressure, reflex sympathoadrenal response caused myocardial ischemia shortly after laryngoscopy and intubation.[3] Various anaesthetic techniques like gentle intubation, deeper plane of anaesthesia[7], topical and intravenous (IV) lignocaine[8,9] and drugs like hydralazine, SNP[10], NTG[11], esmolol[12], Ca channel blockers[13], Fentanyl, morphine, pethidine[14,15,16] have been tried to reduce the hemodynamic reaction to direct laryngoscopy and intubation.

Lignocaine (2 % preservative free) is an amide local anaesthetic. When lignocaine reaches the cell membrane and binds to the voltage-gated sodium channel, the action potential conduction is blocked. Intravenous lignocaine is the drug frequently used for reducing haemodynamic response following laryngoscopy and intubation.[17,18] Due to their haemodynamic stabilising effects, alpha-2 (α2) adrenoceptor agonists such as clonidine and dexmedetomidine have been considered to reduce the stress response. A newer imidazoline derivative - dexmedetomidine is a highly selective agonist at the α2 adrenergic receptor. Compared to other alpha 2 agonists like clonidine, its duration of action is short. It causes analgesia, sympatholysis, hypnosis as well as anxiolysis.[19] It decreases sympathetic discharge from the CNS in a dose-dependent manner. Due to haemodynamic stability and very few adverse effects, it has been reported to produce sedation without respiratory depression and might even prove to be a valuable adjuvant during general anaesthesia. Dexmedetomidine is nowadays used in the ICU to provide sedation and analgesia for mechanically ventilated patients and for patients undergoing surgery under monitored anaesthesia care.[20] So we compared intravenous dexmedetomedine 0.5 μg/kg and intravenous lignocaine 1.5 mg/kg for attenuating haemodynamic response to laryngoscopy and intubation.

MATERIALS AND METHODS

This was a hospital based prospective randomized, double blind, comparative study conducted among 100 patients who underwent elective surgeries under general anaesthesia (GA) in a Tertiary Care Hospital, after approval from Institutional Ethics Committee. We included normotensive patients of ASA grade I of either sex, of age group 18 to 55 years, Mallampati grade 1 posted for elective surgeries under GA with endotracheal intubation. We excluded patients with anticipated difficult intubation, patients with history of drug allergy, pregnant and nursing females, patients with hypertension, cardiac arrhythmias, coronary artery disease, cerebrovascular disease, hepatic or renal dysfunction, patients having morbid obesity (BMI > 30 kg/m2). Patients requiring more than one attempt of intubation and intubation time exceeding 30 seconds were excluded. All the patients went through detailed pre anesthetic evaluation. All routine investigations were done. The patients who met the inclusion criteria were considered for the study. The anesthetic procedure was briefly explained to the patient. An informed written consent was obtained from the patient or his/her relatives. Patient were kept nil by mouth for 8 hours before surgery.

On the day of surgery intra venous line was secured and

Ringer lactate (2ml/kg/hr) was started. The patients were connected to a monitor (Phillips Intellivue MP50) which included heart rate, noninvasive blood pressure, EtCO2, pulse oximeter and continuous electrocardiogram. Baseline parameters like heart rate(HR), systolic blood pressure(SBP) were recorded. Patients were randomly divided into two group (n =50) using computer generated randomization table. The assigned group was enclosed in a sealed envelope to ensure concealment of allocation group. The allocation and preparation of study drug was done by an independent anaesthesiologist not involved in the recording of observation of patient. Group L received 10 ml of normal saline over 10 minutes, 20 minutes before induction by syringe pump and Inj. Lignocaine 1.5 mg/kg diluted to 6 ml with normal saline, 3 minutes before intubation and Group D received 0.5 µg/kg of Inj. Dexmedetomidine diluted to 10 ml with normal saline over 10 minutes by syringe pump, 20 minutes before induction and 6ml of normal saline 3 minutes before intubation. The anaesthesiologist who was blinded to the study drug, administered the drug, monitored the patients and recorded the vital parameters. All patients received premedication with injection glycopyrrolate 0.004 mg/kg, ondansetron 0.1mg/kg and iv fentanyl 2µg/kg. After preoxygenation with 100% oxygen for 3 minutes, anaesthesia was induced with propofol 2mg/kg (titrated till loss of eyelash reflex). After successful ventilation, vecuronium 0.1mg/kg was given and the patients were ventilated for 3 minutes. Laryngoscopy was performed using macintosh laryngoscope (blade no 3 or 4 according to patient size) by expert anaesthesiologist and patient's trachea was intubated with appropriate sized cuffed endotracheal tube. Bilateral equal air entry was confirmed and the tube was secured. The entire procedure of laryngoscopy and intubation was completed in less than 30 seconds and in a single attempt. Hemodynamics (HR, SBP) were monitored at 0, 1, 3, 5, 7 and 10 minutes of intubation. No stimulus was given during this study period. Surgery commenced after 10 minutes of intubation. Anaesthesia was maintained with 66% N2O, 33% O2, isoflurane, and incremental doses of vecuronium (0.025mg/kg) and fentanyl as required. Intraoperative monitoring of all the vital parameters was done every 10 minutes. Bradycardia (HR50 beats per minute(bpm), hypotension (SBP90mmHg) were noted and treated accordingly. At the end of surgery neuromuscular blockade was antagonized, patients were extubated and were shifted to post anaesthesia care unit for observation.

Statistical Methods

Data was analysed using Statistical Package for Social Sciences (SPSS) version 20. Quantitative data was presented as means ± standard deviations (SD) and qualitative data was presented as frequencies. The unpaired t test was used to compare normally distributed continuous variables between groups, P < 0.05 was considered significant.

RESULTS

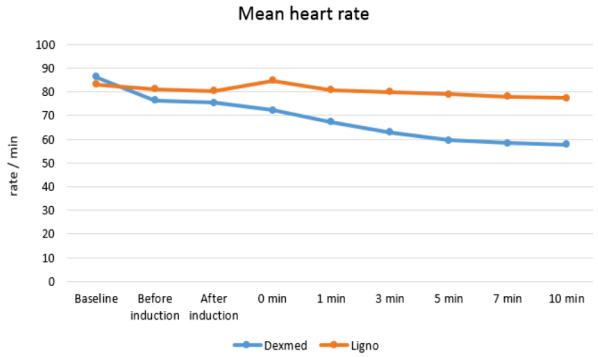
Age, sex and other demographic data was comparable in both groups.

The mean values of baseline HR in the groups L and D were comparable (p = 0.136). The mean values of HR at 0 minute of intubation in the groups L and D were 84.64 ± 11.789 bpm and 72.26 ± 8.875 bpm respectively and p value was statistically significant (p = 0.000), representing a rise of 1.42 bpm in L group and fall of 14.14 bpm in D group from the baseline HR. The mean values of HR at 1, 3 and 5 minute of intubation in the groups L and group D were statistically significant (p = 0.000). At 10 minute of intubation there was fall of 5.74 bpm in L group and fall of 28.70 bpm in D group from the baseline HR which was statistically significant(p=0.000).

Group Statistics						
Time Interval	Group	N	Mean	Std. Deviation	Std. Error Mean	P Value
Baseline	D	50	86.40	9.760	1.380	0.136
	L	50	83.22	11.359	1.606	
Before induction	D	50	76.42	9.515	1.346	0.029
	L	50	81.06	11.376	1.609	
After induction	D	50	75.46	9.489	1.342	0.021
	L	50	80.32	11.116	1.572	
0 minute	D	50	72.26	8.875	1.255	0.000
	L	50	84.64	11.789	1.667	
1 minute	D	50	67.22	8.660	1.225	0.000
	L	50	80.76	9.449	1.336	
3 minute	D	50	62.90	8.006	1.132	0.000
	L	50	79.92	8.559	1.210	
5 minute	D	50	59.46	7.693	1.088	0.000
	L	50	78.94	7.723	1.092	
7 minute	D	50	58.40	7.660	1.083	0.000
	L	50	78.02	7.455	1.054	
10 minute	D	50	57.70	7.660	1.083	0.000
	L	50	77.48	7.217	1.021	

Haemodynamic Parameters Heart rate

Table 1

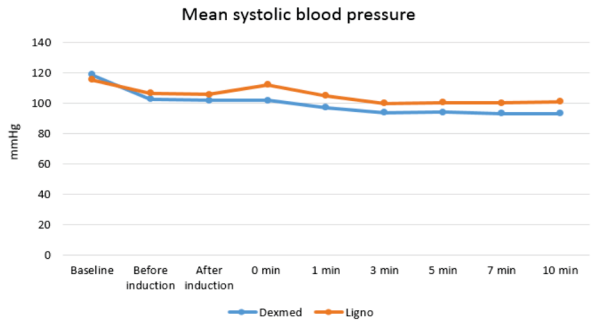


The values of baseline mean SBP in the groups L and D were comparable (p = 0.179). The mean values of SBP at 0 minute of intubation in the groups L and D were 111.90 ± 7.683 mmHg and 101.80 ± 5.171 mmHg respectively and p value was statistically significant (p = 0.000), representing a fall of 3.46 mmHg in L group and fall of 16.88 mmHg in D group from the baseline SBP. The mean values of SBP at 1, 3 and 5 minute of intubation in the groups L and group D were statistically significant (p = 0.000). At 10 minute of intubation there was fall of 22.26 mmHg in group L and fall of 25.58 mmHg in group D from the baseline SBP which was statistically significant (p=0.000).

Group Statistics						
Time Interval	Group	N	Mean	Std. Deviation	Std. Error Mean	P Value
Baseline	D	50	118.68	11.324	1.601	0.179
	L	50	115.36	13.160	1.861	
Before induction	D	50	102.60	6.181	1.874	0.046
	L	50	106.44	11.877	1.680	
After induction	D	50	101.74	6.197	1.876	0.041
	L	50	105.52	11.254	1.592	
0 minute	D	50	101.80	5.171	1.731	0.000
	L	50	111.90	7.683	1.087	
1 minute	D	50	96.86	4.300	1.608	0.000
	L	50	104.70	7.141	1.010	
3 minute	D	50	93.72	3.239	1.458	0.000
	L	50	99.82	6.945	1.982	
5 minute	D	50	93.84	3.076	1.435	0.000
	L	50	100.26	7.009	1.991	
7 minute	D	50	93.08	2.966	1.419	0.000
	L	50	100.12	6.495	1.919	
10 minute	D	50	93.10	3.015	1.426	0.000
	L	50	100.76	6.299	1.891	

Systolic Blood Pressure

Table 2



DISCUSSION

Various authors have used dexmedetomidine in different doses. Among the literature, there were few studies evaluating the role of lower doses of dexmedetomidine (0.5 μg/kg) in attenuation of pressor responses.

Kumari K et al. investigated the effect of a single pre-induction intravenous dose of dexmedetomidine of 0.5 g/kg on hemodynamic responses to tracheal intubation, as well as adverse effects and the dose requirements of anaesthetic drugs for induction. Eighty adult patients were randomly assigned to one of two groups (n = 40 each): dexme detomidine (0.5 μg/kg) and placebo. The increase in heart rate after intubation in the dexmedetomidine group was 19.6 % lower than in the placebo group. The increases in systolic, diastolic and mean blood pressure after intubation were significantly lower in the dexmedetomidine group than in the placebo group (12.38 % vs. 45.63 %, 19.36 % vs. 60.36 %, and 15.34 % vs. 50.33 % respectively). They concluded that a single pre-induction intravenous dose of 0.5 μg/kg dexmedetomidine resulted in a significant reduction in the rise in heart rate, systolic blood pressure, diastolic blood pressure, and mean blood pressure until 5 minutes post intubation. It significantly reduced propofol induction dose requirements while causing minimal side effects.[21]

Gangappa RC et al. did a clinical study of intravenous dexmedetomidine (1 μg/kg) versus lignocaine (1.5 mg/kg) as premedication for attenuation of hemodynamic responses to laryngoscopy and endotracheal intubation in sixty patients. Dexmedetomidine group had mean heart rate of 80.88±16.14 while lignocaine group had 105.04 ± 12.96 at intubation ($p < 0.001$). Dexmedetomidine group had mean systolic blood pressure of 126.22 ± 18.40 while lignocaine group had 166.56 ± 13.40 at intubation ($p < 0.001$).[22]

Haemodynamic Changes

Heart rate

Our study results showed that the mean heart rate at 0 minute of intubation in the group L and D were 84.64 ± 11.789 bpm and 72.26 ± 8.875 bpm respectively and p value was statistically significant ($p = 0.000$), representing a rise of 1.42 bpm in group L and fall of 14.14 bpm in group D from the baseline heart rate.

In the group L, there was increase in the mean HR at intubation by 5.37 % from the after induction value. On the contrary, in the group D, we observed 4.24 % decrease in the mean HR at intubation from the after induction value. Thereafter, the HR remained persistently lower in the group L and group D (fall of 5.74 bpm in group L and fall of 28.70 bpm in group D at 10 minutes post intubation from the baseline mean heart rate). On intergroup comparison, the mean HR was statistically significantly lower in the Group D values ($p = 0.000$) at all time points except baseline values. Thus we observed that heart rate control was better in group D. Our study results were similar to study results of Gangappa RC et al.[22]

Systolic blood pressure (SBP)

Our study findings showed that the mean values of systolic blood pressure at 0 minute of intubation in the group L and D were 111.90 ± 7.683 mmHg and 101.80 ± 5.171 mmHg respectively and p value statistically significant ($p = 0.000$), representing a fall of 3.46 mmHg in group L and fall of 16.88 mmHg in group D from the baseline mean SBP.

In the group D, 0.058 % increase was observed in mean SBP at intubation when compared to after induction mean values; however, when compared to baseline, there was a significant decrease in mean SBP post-intubation. In the group L, there was 6.04 % increase in mean SBP at intubation compared to after induction mean values. Thereafter, the mean SBP remained persistently lower in the group L and group D (fall of 22.26 mmHg in group L and fall of 25.58 mmHg in group D at 10 minutes post intubation from the baseline mean SBP). On intergroup comparison, a significant difference in the values of mean SBP was observed between the two groups at all-time intervals except baseline mean values, which was on lower side in group D ($p = 0.000$).

Thus SBP control was better in group D. Our study results were similar to study results of P Eniya et al.[23] None of the patient had bradycardia or hypotension.

CONCLUSION

- Lignocaine at a dose of 1.5 mg/kg given 3 minutes before laryngoscopy and intubation was not effective in lowering heart rate changes associated with laryngoscopy and endotracheal intubation.
- Dexmedetomidine 0.5 g/kg was more effective than lignocaine in maintaining stable haemodynamics during laryngoscopy and intubation without significant side effects. Hence, we conclude that 0.5 g/kg dexme detomidine given ten minutes before intubation can be effectively used to reduce the haemodynamic changes associated with laryngoscopy and endotracheal intubation.

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