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EFFICACY OF LIGNOCAINE VS DEXMEDETOMIDINE FOR ATTENUATION OF HEMODYNAMIC STRESS RESPONSE TO LARYNGOSCOPY AND INTUBATION

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Abstract

Background: Direct laryngoscopy and endotracheal intubation following induction of anesthesia may trigger sympathetic and sympathoandrenal responses causing profound variation in cardiovascular physiology, and may cause serious complications in patients with underlying coronary artery disease, hypertension, or intracranial neuropathology. Present study was planned to evaluate the effect of IV dexmedetomidine infusion and IV lignocaine in attenuating the hemodynamic responses during laryngoscopy and intubation. **Methodology:** On approval from institutional ethical committee, total 70 ASA grade I and II, normotensive patients in the age group of 18-60 years, planned for elective surgery under general anesthesia were enrolled in the study. Patients were divided into 2 equal groups of 35each; Group L (Lignocaine group) received lignocaine 1.5 mg/kg IV 3 min before induction, Group D (dexmedetomidine group) received dexmedetomidine 0.5 µ/kg IV in 100 ml NS 10 min before induction. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), oxygen saturation of arterial blood (SpO2) were monitored and recorded during the Baseline on OT table(TB), After study drug given(TA),Before Intubation (T0), and at 1 min(T1),3 min (T3),5 min(T5), 7 min (T7) and 10 min (T10) after intubation. Total dose requirement of induction agent was recorded in both groups. **Results:** A better control of stress response was observed in patients receiving inj dexmedetomidine infusion than inj lignocaine. Maximum increase in heart rate was around 22% in group L and 9% in group D (P = 0.002). Maximum increase in mean arterial pressure was more in group L than group D (22% vs 11%) respectively. (P = 0.001). Total propofol dose requirement was 16% less in group D compared to group L (P < 0.001). **Conclusion:** Dexmedetomidine 0.5 µg/kg IV is more effective in attenuates the hemodynamic stress response to laryngoscopy and endotracheal intubation as compared to lignocaine 1.5 mg/kg

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IV without any deleterious effect. Moreover, dexmedetomidine lowers the overall dosage of propofol used in induction.

Keywords: Dexmedetomidine; Hemodynamic stess response; Lignocaine; Laryngoscopy; Intubations.

Introduction

Laryngoscopy and endotracheal intubation are frequently performed procedures in the practice of general anaesthesia. These are most stressful conditions to which the patient is subjected. Laryngoscopy and tracheal intubation may trigger sympathetic and sympathoadrenal response which result in profound variation in cardiovascular physiology and may cause serious complications in patients having coronary artery disease, hypertension or intracranial neuropathology. So, anesthesiologist is always worried about this pressor response which leads to abnormal circulatory reaction which may be severe or prolonged.[1] Various drugs have been used to attenuate these responses, but none have been entirely successfull[2,3] Hence, there is a constant search to attenuate the hemodynamic stress response to laryngoscopy and endotracheal intubation. Therefore, the goal in modern anaesthesia techniques is to maintain haemodynamic stability during the surgical procedure and avoid sympathetic discharge.

As intravenous induction agents are not able to obtund stress response to laryngoscopy and endotracheal intubation, we have to add other pharmacological agents like beta blocker, sodium nitroprusside nitroglycerine, lidocaine, calcium channel blockers, inhalational anaesthetic agents, ACE inhibitors etc. Intravenous lignocaine reduces the cardiovascular response to endotracheal intubation by enhancing the depth of anaesthesia, peripheral vasodilation, and direct cardiac depression. Lignocaine has been used to blunt pressor response to intubation,[4,5] because of its short duration, antiarrythmic effects and its effect on synaptic transmission.

In last few years, a great enthusiasm has been shown toward the use of α2 agonists in anesthesia practice because of their sympatholytic, sedative, anxiolytic, and analgesic-sparing properties lacks opioid-related side effects.[6,7] Dexmedetomidine, the pharmacologically active d-isomer of medetomidine, is a selective α2 agonist with 8 times more affinity for α2 adrenergic receptors compared to clonidine. Its short half-life makes it an ideal drug for intravenous (IV) titration.[8] Intravenous use of dexmedetomidine in the perioperative period had been found to decrease serum catecholamine levels by 90%[9] to blunt the hemodynamic response to laryngoscopy, tracheal intubation, pneumoperitoneum and extubation[10] to provide sedation without respiratory depression and to decrease post operative analgesic requirements.[11]

Various studies have evaluated its hypnotic, analgesic, and anxiolytic properties in the intraoperative period and critical care setting.[12-15] However, there are limited data on its effect on attenuation of pressor response to direct laryngoscopy and intubation response. The purpose of this study was therefore, to evaluate the effects of a single preinduction IV dose of 0.5μg/kg dexmedetomidine vs lignocaine 1.5mg/kg on hemodynamic response to laryngoscopy and intubation, dose requirements of propofol for induction and its adverse effects in adult patients undergoing surgery under general anesthesia.

Materials And Methods

After obtaining institutional review board approval and written inform consent, this descriptive longitudinal study was carried out in a tertiary care teaching hospital. This study

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enrolled 70 patients ASA class I and II aged 18 to 60 years of age of either sex who were scheduled for surgery under general anaesthesia requiring endotracheal intubation and receiving dexmedetomidine or lignocaine as GA premedication. Patients with cardiac, coronary, renal, hepatic, cerebral diseases, and peripheral vascular diseases, endocrine disorder, obese patients, anticipated difficult airway, Patients on β blockers, calcium channel blockers, antihypertensive or antipsychotic drugs, pregnant or nursing women, with a history of drug sensitivity, or with intubation attempts lasting more than 15s were excluded from the study. Patients were divided into two groups: L (lignocaine) and D (dexmedetomidine), each with 35 patients for observation. Patients in group D were received iv dexmedetomidine 0.5mcg/kg and those in group L were received iv lignocaine 1.5mg/kg as premedication.

In all patients on arrival in the operating room baseline parameters such as heart rate (HR), mean arterial pressure (MAP), respiratory rate and oxygen saturation (SpO2) were recorded. Ringer's lactate solution (500 ml) was used to prehydrate all patients. Group L patients were given 100 ml of normal saline preoperatively over 10 minutes, with the infusion completed 10 minutes before induction and 1.5 mg/kg of lignocaine administered IV 3 minutes before intubation. Group D patients received dexmedetomidine 0.5 mcg/kg diluted in 100 ml of normal saline given IV over a period of 10 min and the infusion was completed 10 min before induction. Prior to preoxygenation, all patients received injections of glycopyrrolate 0.004 mg/kg IV, Ondansetron 0.08 mg/kg IV, midazolam 0.02 mg/kg IV, and fentanyl 2 mcg/kg IV. Ramsay sedation scale score were employed for assessing the sedation before induction of anesthesia in both the groups. Following preoxygenation, all patients were given an infusion of propofol until the eye lash reflex was lost. Endotracheal intubation was facilitated with 2 mg/kg succinylcholine given IV 1 min prior to laryngoscopy and intubation. Laryngoscopy was performed using Macintosh blade size 3 and intubation using the intratracheal tube (size 7.5-8 mm/cuffed) was carried out by a senior anesthesiologist or by a 2-year trained resident in anesthesiology. 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 then such patients were excluded from the study. After connecting the intubation tube to a closed circuit and confirming bilateral equal air entry, the endotracheal tube was secured. No surgical or any other stimulus were applied during 7 min of study period and vecuronium was the only additional drug given during this period. The anaesthesia was maintained using 33% oxygen and 66% nitrous oxide, along with isoflurane and vecuronium. At the end of the procedure, patients will be reversed with an injection neostigmine 0.05 mg/kg IV and injection glycopyrolate 0.08 mg/kg IV. Patient was extubated when they regained reflexes and consciousness. Haemodynamic parameters (HR, MAP) were recorded throughout the following time intervals:

TB: Baseline on OT table

TA: After study drug given

T0: Before intubation

T1: at 1 min after intubation.

T3: at 3 min after intubation

T5: at 5 min after intubation

T7: at 7 min after intubation

T10: at 10 min after intubation

Hypotension was defined as SBP < 20% of baseline. Tachycardia was defined as a heart rate that was greater than 25% of the baseline. Bradycardia was defined as HR < 45 beats/ minute.

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Statistical Analysis At the end of study all data were entered in Microsoft Excel& statistical analysis was done by SPSS (16.0) version. Descriptive data were presented as mean and standard deviation and continuous data were analyzed by paired student t- test. A chi-square test was used to assess statistical difference between groups. P value <0.05 was consider as significant and p value >0.05 was not significant.

Result

Both the groups under study were comparable to each other with respect to age, gender, weight distribution, ASA physical status, time taken to laryngoscopy and intubation, Mallampati grade and type of surgery and anesthesisa.(Table.1) The change in HR and MAP in both groups at different time interval are shown in Fig 1. Base line (TB) HR and MAP was comparable in both groups. In group D, there was statistically significant initial fall in HR after giving study drug as compared to group L. In both groups there was rise in HR, after laryngoscopy and intubation, remained raised for 5 min postintubation (P<0.05). But this rise was statistically significantly more ingroup L from T1-T5 as compared to group D (P<0.05). Heart rate in both groups was almost near to the baseline values at T7(P>0.05). Maximum intubation response was seen at 1 min post-intubation in both groups. The maximum increases in HR in group L was 32.1% and 10.3% in group D at T1 time interval after intubation.

The group L had statistically higher values of HR, SBP, DBP and MAP at all time intervals up to 5min post-intubation when compared to Group D.Hence, it can be inferred that the haemodynamic response was better obtunded in Group D when compared with Group L. This indicates that dexmedetomidine in a dose of 0.5 µg/kg was completely attenuating the intubation response

No statistical significance was noted in MAP between groups at baseline,(TB), after study drug(TA) and before intubation (T0). There was a statistical significance noted in MAP at 1 min (T1), 3min (T3), and at 5min (T5) after intubation. The MAP was increased significantly compared with preoperative value after intubation in the group L (p=0.000) and was significantly higher than in group D (p=0.0001). In the group D, MAP was not significantly higher than the preoperative value at all times. Thegroup D had a better control of heart rate and blood pressure than the group L [Table 2]. No incidence of fall in Spo2, respiratory depression, hypotension and bradycardia requiring intervention was reported in both groups.

Table 1: Demographic profile of patient

DEMOGRAPHIC DATA	GROUP D	GROUP L	P VALUE
Age	34.21± 13.43	35.75 ±17.65	0.679
Male/ female	22/18	21/19	
Weight (kg)	71.42 ± 15.51	73.14 ± 9.49	0.578
Height (cm)	164.21 ± 9.812	165.140 ± 8.930	0.679
BMI (kg/m)2	26.23 ± 3.33	26.37 ± 4.37	0.881
ASA 1/2	28/12	30/10	
Time taken for intubation	13.75±22.29	14.12 1± 9.37	0.952

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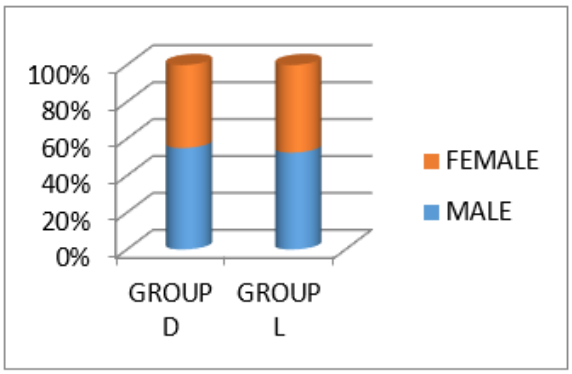
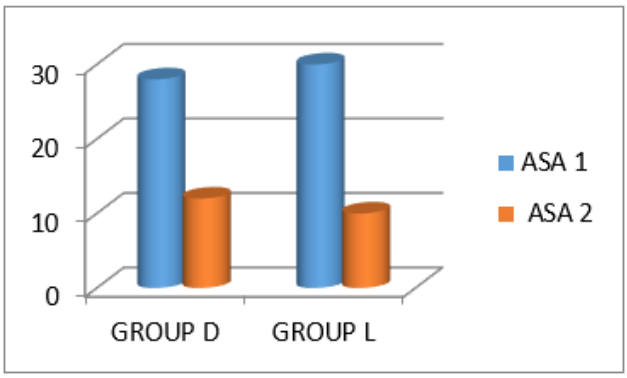
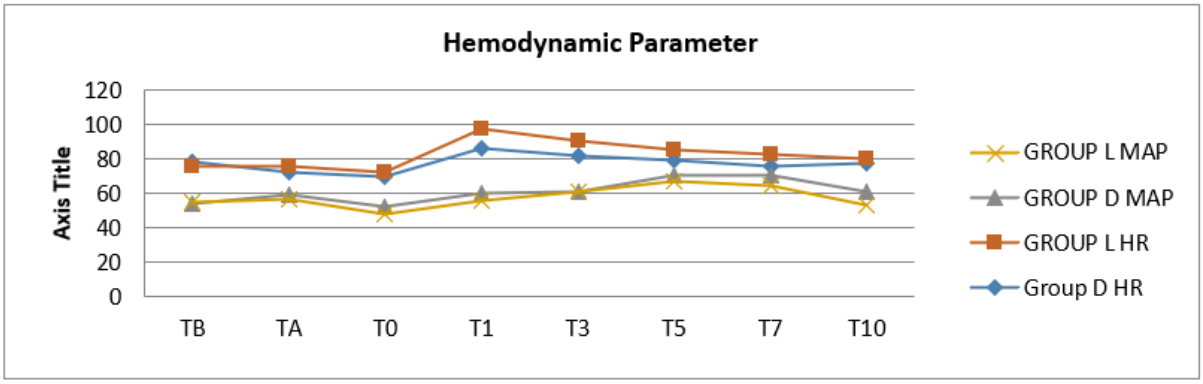




GRAPH 1:SEX WISE DISTRIBUTION IN PERCENTAGEGRAPH 2:COMPARISON OF ASA STATUS OF BOTH GROUPS.

TABE2: COMPARISION OF HAEMODYNAMIC PARAMETER AMONG BOTH GROUPS

Parameter	Group	TB	TA	T0	T1	T3	T5	T7	T10
SBP	**D**	136.7±12.4	124.4±14.5	122.4±13.3	145.6±16.4	136.8±11.7	133.4±12.39	127.1±11.2	124.2±11.2
	L	133.5±13.5	132.5±15.5	130.2±14.3	157.4±17.5	144.3±13.6	141.1±13.7	131.1±12.3	126.1±11.7
	P value	0.3054	0.02718	0.0210	0.0049	0.0103	0.0156	0.138	0.468
DBP	**D**	79.4±8.4	80.5±8.9	79.1±11.3	90.9±11.7	84.5±11.4	77.8±15.3	76.4±16.1	72.5±11.3
	L	78.7±9.4	79.7±10.7	78.7±12.3	98.7±11.9	92.4.±16.3	86.9±14.2	80.1±11.4	74.1±10.9
	P value	0.743	0.728	0.887	0.008	0.0185	0.0120	0.0717	0.548
MAP	**D**	98.4±5.5	95.2±5.4	93.6±5.9	109.2±6.7	102.3±5.1	96.4±6.9	93.4±7.6	90.3±6.2
	L	96.97±6.2	97.3±6.7	95.9±7.7	118.3±8.9	107.6±5.9	104.7±7.4	97.4.1±11.2	91.57±9.2
	P value	0.311	0.227	0.165	0.0001	0.0001	0.0001	0.095	0.490
HR	**D**	79.7±7.1	70.6±14.5	70.2±9.7	86.7±13.11	82.4±13.34	82.7±7.6	79.4±12.2	77.9±11.32
	L	80.5.5±7.8	75.2±9.6	74.5±8.9	97.7±11.2	90.5±15.78	88.4±11.45	82.3±10.2	79.4±12.31
	P value	0.695	0.532	0.057	0.0321	0.001	0.0170	0.286	0.795



GRAPH 3: COMPARISION OF MEAN ARTERIAL PRESSURE AND HEART RATE OF BOTH GROUPS.

TB: Baseline on OT table, TA: After drug, T0: Before intubation , T1: at 1 min after intubation, T3: at 3 min after intubation,T5: at 5 min after intubation, T7: at 7 min after intubations,T10: at 10 min after intubation.

Discussion

Laryngoscopy and endotracheal intubation are considered as the most critical events during general anesthesia as they lead to transient and variable hemodynamic responses due to afferent vagal stimulation and an efferent sympathoadrenal response. These transient and variable hemodynamic changes of tachyarrhythmia and hypertension may lead to life-

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threatening complications in high-risk patients. Many drugs have been tried by various authors for blunting hemodynamic responses to laryngoscopy and intubation[16-19] but all such maneuvers had their own limitations and choosing the ideal agent is still a controversial issue. Lignocaine was used in several studies for attenuation of the stress response.[20-25].lignocaine is a sodium channel blocker which attenuate the hemodynamic response to laryngoscopy and intubation by inhabiting sodium channel in neuronal cell membrane, decreasing the sensitivity of the heart muscle to electric impulse.

Dexmedetomidine, the highly selective alpha2-adrenergic agonist, has a unique pharmacological profile with sedation, sympatholysis, analgesia, opioid and anaesthetic sparing action, cardiovascular stability, and a significant advantage in avoiding respiratory depression.[12,26-29]

Three minutes before to intubation, an IV dosage of 1.5 mg/kg of lignocaine was administered in our study. Splinter WM *et al.*[20] Abou-Madi MN *et al.*[21]and Tam S *et al.*[25]concluded that 1.5 mg/kg of lignocaine suppresses stress response to intubation when given 3 min before intubation. Intravenous lignocaine prevent cardiovascular response to endotracheal intubation by increasing depth of anesthesia, peripheral vasodilatation and direct cardiac depression. The dose of dexmedetomidine used in our study was 0.5mcg/kg diluted in 100 ml of normal saline and infused over 10 min. Several authors[30-35] have used 0.5-1 mcg/kg of dexmedetomidine to attenuate stress response to intubation.

A biphasic cardiovascular response has been described after the administration of dexmedetomidine.[36] A bolus dose of 1 mcg/kg results in a transient increase in BP and reflex decrease in HR in young healthy patients. Initial response is due to α2 receptor stimulation of vascular smooth muscle. This response can be markedly decreased by slow infusion over 10 min. In our study, this effect was not noticed due to the slow infusion of the dexmedetomedine at the dose of 0.5 mcg over 10 min in 100ml NS.

Following infusion of dexmedetomidine, there was 12% reduction in HR and 5 reduction in MAP and the observations are similar to the observations of other studies and can be explained on the basis of decreased central nervous system sympathetic activity.[30,31,34,37]

Sulaiman S *et al.*[32]Pipanmekaporn T *et al.* [35] noted a significant fall in HR in the dexmedetomidine group and an insignificant fall in MAP in their study.

There was significant increase in heart rate and blood pressure from baseline after laryngoscopy and intubation in both groups, maximum rise in heart rate and blood pressure were noted at one minute after intubation but the rise in heart rate and blood pressure in dexmeditomidine group were significantly lower, less pronounced and shorter lasting as compared to lignocaine group. Dexmeditomidine has sympatholytic effect and also by activation of both α2 A and α2 C receptors in the spinal cord, it directly reduces pain transmission by reducing release of substance P. On comparison between the two groups, the heart rate and blood pressure was better controlled with dexmeditomidine than lignocaine after laryngoscopy and intubation. Similar result was obtain in previous study.

Compared to lignocaine, dexmedetomidine at a dose of 0.5mcg/kg significantly attenuated hemodynamic response to laryngoscopy and intubation but could not obtund it completely. Several authors[30-35] reported that dexmedetomidine at a dose of 0.5-1 μg/kg significantly attenuated hemodynamic response to intubation but did not obtund it completely, and our observations are in accordance with them.

Dexmedetomedine in locus ceruleus stimulation of α2 A and α2 C cause sedation. Several authors[8,12,32,35] have reported that dexmedetomedine infusion produces sedation which mimics normal sleep, patients are arousable to verbal commands, and it lacks respiratory

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depression. In our study, injection midazolam and injection fentanyl used as premedication pre induction period all patients in group L had sedation score 2 while in group while it was 3 in group D, which due to additional sedative action of dexmedetomedine. None of the patients in either group had respiratory depression or fall in SpO2.

Propofol is favorable induction agent with cardiovascular depressive property and is more effective at suppressing stress hormone release than thiopental. In our study propofol dose requirement was 16% less in group D as compared to group L ($P < 0.001$). In other study thiopental was used as induction agent and induction dose was significantly decreased ingroup D than group L.Keniya *et al.*,[31]Scheinin *et al.*,[33] and Bajwa *et al.*[34] reported decreased thiopentone requirement for induction of anesthesia in the dexmedetomidine group.

No serious side effects like hypotension and bradycardia were observed during study period in either group. This may be due to adequate plasma volume expansion and inj glycopyrrolate used as premedication. Also in our study Dexmedetomidine was administered as an infusion over 10 min to prevent bradycardia and hypotension associated with a bolus dose.[29] The change in hemodynamic parameter due to intubation stress response was less affected in group D than group L. Thusdexmedetomedine provided better protection against unwanted hemodynamic side effect of intubation and laryngoscopy by blunting the stress response than lignocaine.

Limitation

We did not measure the plasma not epinephrine levels nor extubation response and hemodynamic variations. This study need to be done in high risk cardiac patient and hypertensive group in which dexmedetomedine can be more beneficial.

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