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Original Article

Attenuation of Hemodynamic Changes during Laryngoscopy and Endotracheal Intubation with Intravenous Lidocaine versus Intravenous Dexmedetomidine Given as Premedication: A Randomized Prospective Clinical Study

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Abstract

Background: Both laryngoscopy and endotracheal intubations are important tools in the hands of an anesthesiologist in securing and maintaining the airway and are the most stressful events during anesthesia induction. The extent of hemodynamic changes observed may be dependent on several factors like depth of anesthesia, whether any measures are taken before manipulation of airway, the anesthetic agent used, the duration of laryngoscopy and intubation. These events can lead to hypertension, tachycardia, arrhythmias, and myocardial ischemia. An alpha-2 adrenoreceptor agonist, dexmedetomidine, is gaining its name for its sympatholytic, anesthetic sparing, sedative, and also properties like hemodynamic stabilization without any significant respiratory depression. Hemodynamic changes during endotracheal intubation are major concerns in general anesthesia.

Aim of the Study: This study aims to compare the efficacy of intravenous (IV) dexmedetomidine and IV lidocaine in attenuating hemodynamic response to laryngoscopy and endotracheal intubation.

Materials and Methods: A study involving 60 patients of both sexes requiring endotracheal intubation and general anesthesia was included. All patients were pre-medicated with Inj. midazolam 1 mg IV, induced with Inj. propofol 2 mg/kg IV, analgesia with Inj. fentanyl 1.5 μg/kg IV, and muscle relaxation produced with Inj. vecuronium 0.1 mg/kg IV. Patients were alternately allocated to Group L (lidocaine group) or D (dexmedetomidine). Plain preservative-free lidocaine 2%, 1.5 mg/kg body weight IV bolus in Group L (n=30) 90 s before laryngoscopy and the other Group D (n=30) received dexmedetomidine 1 μg/kg body weight slow IV, 10 min before laryngoscopy. Hemodynamic changes such as heart rate (HR), systolic blood pressure (SBP), mean blood pressure, and diastolic blood pressure (DBP) were monitored before induction, that is, baseline and after anesthesia induction, at laryngoscopy and 1, 3, 5, and 10 min after endotracheal intubation.

Results: The hemodynamic parameters recorded include HR, SBP, DBP, and mean arterial pressure (MAP) before induction, that is, baseline and after anesthesia induction, at laryngoscopy and at 1, 3, and 5 min after laryngoscopic intubation. In lignocaine group, HR, SBP, DBP, and MAP increased maximally and gradually returned to baseline value. The dexmedetomidine group showed highly significant attenuation of sympathetic response to laryngoscopy and intubation compared to lignocaine groups.

Conclusion: The study results shows to demonstrate that dexmedetomidine infusion is better at attenuating the pressor response to intubation than preservative-free IV lignocaine when given before induction.

Key words: Dexmedetomidine, Hemodynamic stress response, Intravenous dexmedetomidine, Intravenous lignocaine, Intubation, Laryngoscopy, Premedication

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INTRODUCTION

Both laryngoscopy and endotracheal intubations are important tools in the hands of an anesthesiologist in securing and maintaining the airway. Moderate to high rise in serum catecholamines is seen as a result of sympathetic response of both laryngoscopy

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and endotracheal intubation. The rise in serum concentrations of norepinephrine is approximately up to 147% and that of epinephrine level which is up to 60% when performing laryngoscopy and endotracheal intubation.[1] It leads to an increase in heart rate (HR) and blood pressure (BP).[2]

The extent of hemodynamic changes observed may be dependent on several factors like depth of anesthesia, whether any measures are taken before manipulation of airway, the anesthetic agent used, the duration of laryngoscopy and intubation. Till date, the exact mechanism of hemodynamic responses to laryngoscopy and intubation has not been clarified yet. The principle mechanism in tachycardia and hypertension is the sympathetic responses[2,3] which may be the result of high catecholamine activity.[4] This laryngoscopic effect in those people may predispose to the development of pulmonary edema, cerebrovascular accident, and myocardial insufficiency[5,6]

Topical lignocaine sprays, deeper planes of anesthesia by both inhalational/intravenous (IV) agents and narcotics, calcium channel blockers such as verapamil, nicardipine, and diltiazem, and vasodilators such as sodium-nitroprusside, nitroglycerin, esmolol, magnesium sulfate, and gabapentin[4] have been tried to reduce the pressor response and still remain as treatment modalities. The drugs mentioned above have been found to be ineffective to attenuate the sympathetic response to intubation and also not able to meet all the criteria required. Hence, there is a need of finding out the drugs which can meet both requirements.

Alpha-2 adrenergic agonists like dexmedetomidine reduce sympathetic tone and blunt the hemodynamic responses to noxious stimulation and prevent the hemodynamic variability overall and also reduce the need for anesthetics. Therefore, dexmedetomidine is a more specific and selective alpha-2 adrenergic agonist than clonidine which has a shorter duration of action.[7] The advantages of IV dexmedetomidine as premedication in anesthesia setting include analgesia, sedation, anxiolysis, and improved hemodynamic stability. Because of these beneficial properties, it has been found that minimum alveolar concentration of volatile anesthetics also decreases significantly up to 90% and so decreases the requirement of anesthetics.[8,9] The present study is aimed at comparison of attenuation of hemodynamic response to laryngoscopy and endotracheal intubation in adult patients posted for various surgeries under general anesthesia and also to assess the incidence of side effects, that is, rebound hypertension, bradycardia and hypotension, etc., that are associated with the use of dexmedetomidine.

MATERIALS AND METHODS

After the institutional ethical committee clearance of Viswabharathi Medical College and Hospital, Penchikalapadu, RT Nagar, Kurnool, the study was carried out from January 2018 to February 2019. A total of 60 patients with American Society of Anaesthesiologists (ASA) physical status I and II patients and aged between 18 and 60 years who are undergoing surgery on elective basis under general anesthesia were enrolled in the study. The research plan is carried out safely and methodologically and probable side effects of the drugs used were explained to the patients in their native language and were included in the study after obtaining written informed consent from all patients.

Patients with cardiac, hypertension, coronary, hepatic, cardiac, hepatic, cerebral and peripheral vascular diseases, bradycardia, obese patients, anticipated difficult airway, nasogastric tube insertion, need of neck manipulations, Mallampati Grade III and IV, history suggestive of sensitivity to drugs used during the study, and difficult ventilation and/or difficult intubation (more than 15 s) after induction were excluded from the study.

The patients were randomly allocated to one of the two groups: Group L (lidocaine group) or Group D (dexmedetomidine). Plain preservative-free lidocaine 2%, 1.5 mg/kg body weight IV bolus in Group L (n=30) 90 s before laryngoscopy, and the other Group D (n=30) received dexmedetomidine 1 μg/kg body weight slow IV using syringe pump, 10 min before induction.

None of the above patients were on any significant drug therapy preoperatively. All patients included in the study were pre-medicated with tablet ranitidine 150 mg orally and tablet alprazolam 0.5 mg on the night before surgery and nil orally from night 10 pm onward. All the said patients were connected to multipara monitors and basal readings of HR and BP were noted cautiously.

An 18G IV cannulation was inserted in the operation theater and before pre-oxygenation, patients were pre-medicated with midazolam 0.02 mg/kg, ondansetron 0.08 mg/kg, glycopyrrolate 0.01 mg/kg, and analgesia with fentanyl 0.5 mg/kg intravenously. After 3 min of pre-oxygenation, all patients were induced with injection propofol 2 mg/kg IV till loss of the eye lash reflex.

Laryngoscopy and intubation were performed using Macintosh no. 3 or 4 blade lasting for not more than 15 s and after confirmation of bilateral equal air entry, the endotracheal tube was fixed. Anesthesia was maintained using 66% nitrous oxide and 33% of oxygen with 0.5% halothane.

Laryngoscopy and endotracheal intubation (with appropriate size ET tube) were performed using Macintosh laryngoscope blade 3 or 4 after IV injection of 2 mg/kg of succinylcholine given over 1 min. After the auscultatory confirmation of bilateral equal air entry, the ET tube is fixed and connected to closed circuit. Laryngoscopy and intubation were limited to 15–20 s in all patients and failure to intubate within this limited period was excluded from this study. No surgical or any other stimulus was applied during 10 min of study period and vecuronium was only other additional drug given and used during this period.

All vital parameters such as HR, systolic BP (SBP), diastolic BP (DBP), and mean arterial pressure (MAP) were recorded before induction, at baseline, and after induction at laryngoscopy and immediately at 1, 3, 5, and 10 min after intubation and every 5 min thereafter using multipara monitor.

Anesthesia was maintained with 66% N_2O in 33% oxygen and 0.5% halothane. Bolus IV dose of 0.08 mg/kg vecuronium followed by intermittent dose of 0.02 mg/kg vecuronium was used for muscle relaxation as maintenance and after completion of surgical procedure, patients were reversed with injection neostigmine 0.05 mg/kg and injection glycopyrrolate 0.01 mg/kg intravenously. Patients were then extubated after they regained consciousness. An observation was made related to adverse effects of drugs and anesthesia-related problems and was attended appropriately.

RESULTS

The two groups were comparable in age, sex, weight, and types of surgeries [Tables 1 and 2].

DISCUSSION

Most of the general anesthetic procedures in the modern anesthetic practice are carried out with endotracheal intubation. Laryngoscopy and tracheal intubation are considered as the most critical events during administration of general anesthesia as they provoke transient but marked sympathoadrenal response manifesting as hypertension and tachycardia.[5] The responses are transitory, variable, and may not be significant in otherwise normal individuals. However, in patients with cardiovascular compromise such as hypertension, ischemic heart disease, cerebrovascular disease, and in patients with intracranial aneurysms, even these transient changes in hemodynamics can result in potentially harmful effects such as left ventricular failure,[10] pulmonary edema, myocardial ischemia,[10] ventricular dysrhythmias,[9] and cerebral hemorrhage.[10] This is by far the most important indication for attenuation of hemodynamic response to laryngoscopy and tracheal intubation.[11] Many methods such as use of inhalational anesthetic agents, lidocaine,[12-14] opioids,[15-17] direct-acting vasodilators,[18,19] calcium channel blockers,[20-22] and β-blockers[23,24] have been tried by various authors for blunting hemodynamic responses to laryngoscopy and intubation. However, all such maneuvers had their own limitations. For example, with opioids, respiratory depression and chest wall rigidity were potential problems, use of halothane was associated with dysrhythmias, calcium channel blockers produced reflex tachycardia, direct-acting vasodilators needed invasive hemodynamic monitoring, and lidocaine did not give consistent results in blunting the hemodynamic responses to laryngoscopy and intubation. Beta-blockers are also one group of pharmacological agents employed for blunting hemodynamic response to laryngoscopy and intubation. However, they blunt the HR response better than BP response.[5,23]

Table 1: Demographic profile in dexmedetomidine and lignocaine groups

Patients characteristics	Group D	Group L
Age (years)	40±13.4	40±12.9
Sex (M/F)	16/14	17/13
Weight (kg)	55±10.1	53±9.1

Table 2: Type of surgeries in dexmedetomidine and lignocaine groups

General surgery	12	15
ENT\	8	6
Orthopedics	10	9

In our study, we used dexmedetomidine 1 mcg/kg diluted in 100 mL of normal saline and infused over 10 min for patients in Group D. Many authors[7,24] have used 0.5–1 mcg/kg of dexmedetomidine to attenuate stress response to intubation. The dose of lignocaine used in our study was 1.5 mg/kg IV given 3 min before intubation. To maintain uniformity and to prevent evaluator's bias, patients in Group D received 3 mL of normal saline 3 min before induction and patients in Group L received 100 ml of normal saline infusion 10 min before induction. We did not observed any significant differences in HR and arterial BP values between the baseline and post-intubation values in the dexmedetomidine group, the mean percentage variation analysis at the stated moments revealed an absence of any increase in HR, SBP, and DBP in dexmedetomidine group suggesting dexmedetomidine as an effective agent for blunting the hemodynamic response to laryngoscopy and tracheal intubation.

Bradycardia and hypotension have been reported in studies pertaining to the effect of dexmedetomidine

administration on perioperative hemodynamics.[25,26] We did not detect any excessive reduction in HR or systemic BP values in the dexmedetomidine group compared with other group. Moreover, in this study, neither bradycardia nor hypotension was observed in the patients.

All patients in Group L had sedation score 2 in pre-induction period. This was due to injection midazolam used as premedication. Most of the patients in Group D had sedation score 3 (responding to verbal commands). None of the patients in Group D had respiratory depression or fall in SpO2. Several authors[27,28] have reported that dexmedetomidine infusion produces sedation which mimics normal sleep, patients are arousable to verbal commands, and it lacks respiratory depression. These properties make dexmedetomidine a better choice of sedation for awake fiber-optic intubation, intensive care unit, post-anesthesia care unit, magnetic resonance imaging, and awake craniotomy.[29] Lignocaine failed to effectively attenuate hemodynamic response to laryngoscopy and intubation. Similar findings were found by many authors[30,31] who reported that the lignocaine fails to attenuate hemodynamic response and our observations are in accordance with them.

There were few limitations in our study. The effects of dexmedetomidine when used in hypertensive and cardiac patients were not studied as we did not have invasive BP monitoring and advanced cardiac setup in our institute. Furthermore, plasma catecholamine levels, which are an objective means of measuring hemodynamic stress response, were not measured in our study. Estimating depth of anesthesia by changes mediated by autonomic nervous system is difficult during dexmedetomidine infusion as it increases the hemodynamic stability. Intraoperative bispectral index monitoring would have been definitely more objective in deciding the depth of anesthesia and the requirement of anesthetic agent. Post-operative analgesic requirement of the patients was not studied in both the groups.

Several authors[8,32] have concluded that 1.5 mg/kg of lignocaine suppresses stress response to intubation when given 3 min before intubation. The alpha adrenoceptors are involved in regulating the autonomic nervous system and cardiovascular systems. Alpha-2 adrenoceptors are located on blood vessels, where they mediate vasoconstriction and on sympathetic presynaptic terminals where they inhibit epinephrine and norepinephrine release.[11] Alpha-2 adrenoceptors are also located within the central nervous system and their activation leads to sedation, a reduction of tonic levels of sympathetic outflow, and an augmentation of vagal activity,[29,33] This can result in a decrease in HR and cardiac output. Alpha-2 adrenergic drugs, such as clonidine or dexmedetomidine, attenuate these potentially harmful cardiovascular reactions during laryngoscopy and intubation. Alpha-2 agonists produce hyperpolarization of noradrenergic neurons and suppression of neuronal firing in the locus coeruleus leads to decreased systemic noradrenaline release, results in attenuation of sympathoadrenal responses and hemodynamic stability during laryngoscopy and tracheal intubation.[30] In Singh *et al*.[34] study, IV lignocaine 1.5 mg/kg was ineffective in controlling the acute hemodynamic response following laryngoscopy and intubation. Hence, the need for a better alternative was initialized. Singh *et al*., recently, α-2 agonists such as clonidine and dexmedetomidine[34] have been tried for suppressing the response to intubation and have been found to have better effects compared to all the drugs mentioned above, without any of the side effects such as respiratory depression or increased incidence of PONV. Clonidine being less potent (α-1:α-2 = 1:220) compared to dexmedetomidine (α-1:α-2 = 1:1620) in its agonism to α-2 receptors.[35-37] Hence, dexmedetomidine may be a better drug among α-2 agonists for suppressing the hemodynamic responses to laryngoscopy and intubation. Kunisawa *et al*.[38] used 1 μg/kg body weight of dexmedetomidine with fentanyl and found that though there was a decrease in HR, the decrease in BP was suppressed and the authors opined that vasoconstrictive effects of dexmedetomidine through α-2 adrenoceptors which are located in vascular smooth muscle might be responsible for this suppression as a result of administration of higher dose. Dexmedetomidine has been found by various authors,[8,28] to blunt the hemodynamic response for laryngoscopy and intubation. Dexmedetomidine is recently introduced in India (only in 2009) and available as 200 μg/2 ml ampoule and not many studies have been done using dexmedetomidine for suppression of intubation response. Hence, the effects of dexmedetomidine for suppression of hemodynamic response to laryngoscopy and intubation were taken up as our study topic. Menda *et al*.[7] used 1 μg/kg body weight of dexmedetomidine for patients posted for CABG surgeries. They found that dexmedetomidine was effective in suppressing hemodynamic response to intubation. However, all the patients studied were on beta blockers. Esra *et al*.[39] used 0.5 and 1 μg/kg body weight of dexmedetomidine for suppression of intubation response and found that there was no significant change regarding HR in both doses. Hence, dexmedetomidine at the dose of 1 μg/kg body weight for attenuating pressor response to intubation was chosen for our study.

Stoelting[13] noted that the best way to prevent laryngoscopic reaction was to minimize the duration of laryngoscopy and intubation to 15 s. He also suggests that IV lignocaine in the doses of 1.5 mg/kg 3 min before laryngoscopy and intubation sufficiently attenuated the laryngoscopic

Table 3: Comparison of HR in patients of dexmedetomidine and lignocaine groups at different intervals

HR (beats/min)	Group D	Group L	*P*-Value	Significance
Basal	84.50±12.88	91.26±14.50	0.68	NS
Pre-induction	75.42±15.46	89.24±13.46	0.54	NS
Induction	81.46±13.62	105.20±15.96	0.001	HS
Intubation: 0 min	81.88±15.14	106.04±11.96	0.001	HS
Intubation: 1 min	81.44±11.38	107.08±10.84	0.001	HS
Intubation: 3 min	78.82±9.10	99.88±12.26	0.001	HS
Intubation: 5 min	71.98±10.92	92.16±13.60	0.001	HS
Intubation: 10 min	79.84±9.66	91.22±11.64	0.001	HS

NS: Not significant, HS: Highly significant, HR: Heart rate

Table 4: Comparison of SBP in patients of dexmedetomidine and lignocaine groups at different intervals

SBP (mmHg)	Group D	Group L	*P*-value	Significance
Basal	121.27±4.45	122.82±8.66	0.96	NS
Pre-induction	127.26±15.03	127.76±11.62	0.57	NS
Induction	121.80±14.62	116.44±4.48	0.001	HS
Intubation 0 min	127.22±17.40	167.56±12.40	0.001	HS
1 min	117.28±10.96	143.80±17.86	0.001	HS
3 min	113.06±10.82	131.64±15.66	0.001	HS
5 min	115.36±9.06	127.62±10.80	0.001	HS
10 min	115.28±13.21	127.62±11.48	0.001	HS

SBP: Systolic blood pressure, NS: Not significant, HS: Highly significant

Table 5: Comparison of DBP in patients of dexmedetomidine and lignocaine groups at different intervals

DBP (mmHg)	Group D	Group L	*P*-value	Significance
Basal	79.16±7.42	77.62±8.64	0.80	NS
Pre-induction	81.10±12.82	76.96±7.62	0.54	NS
Induction	79.06±12.96	78.10±4.18	0.001	HS
Intubation 0 min	75.36±5.24	91.66±18.90	0.002	HS
1 min	77.38±11.66	89.82±9.66	0.001	HS
3 min	72.24±11.08	83.16±8.96	0.001	HS
5 min	71.55±10.30	81.16±19.42	0.001	HS
10 min	72.67±11.85	76.44±8.20	0.001	HS

DBP: Diastolic blood pressure, NS: Not significant, HS: Highly significant

Table 6: Comparison of MAP in patients of dexmedetomidine and lignocaine groups at different intervals

Mean arterial pressure (mmHg)	Group D	Group L	*P*-value	Significance
Basal	95.06±9.24	93.96±10.92	1.00	NS
Pre-induction	81.02±10.38	85.70±10.08	0.656	NS
Induction	85.88±10.92	106.88±9.02	0.001	HS
Intubation 0 min	87.62±10.24	111.68±11.08	0.001	HS
1 min	83.64±11.08	107.66±13.98	0.001	HS
3 min	79.80±10.88	103.62±13.08	0.001	HS
5 min	77.46±8.62	97.60±11.78	0.001	HS
10 min	73.26±9.68	99.28±8.84	0.001	HS

NS: Not significant, HS: Highly significant, MAP: Mean arterial pressure

reactions. The advantage of IV lignocaine claimed by the author was that it depressed autonomic nervous system and in addition had anti-arrhythmic properties. Aho *et al*.[8] in 1977 studied cardiovascular response to laryngoscopy and tracheal intubation following small (0.75 mg/kg) and large (1.5 mg/kg) IV doses of lidocaine in eighty ASA Grade 1–4 patients and found that the higher dose was more effective in attenuating circulatory responses to tracheal intubation. Gangappa *et al*.[40] in his study noted that dexmedetomidine at the dose of 1 mcg/kg IV infusion for 10 min attenuates the hemodynamic stress response to laryngoscopy and endotracheal intubation more effectively when compared to lignocaine 1.5 mg/kg IV without any side effects. Hence, lidocaine at the dose of 1.5 mg/kg body weight for attenuation of pressor response to intubation was chosen for our study.

The two drugs were compared with respect to their efficacy in attenuating the pressor response to laryngoscopy and endotracheal intubation. In our study, the maximal average decrease was 24%, 28%, and 10% in SBP, DBP, and HR, respectively, in the dexmedetomidine group which varies from the above study probably because the dose of dexmedetomidine taken in our study was 1 μg/kg body weight and that fixed dose of 2 mg/kg propofol and 1.5 μg/kg body weight of fentanyl was used for induction.

In our study, dexmedetomidine (1 μg/kg body weight) showed better attenuation of pressor response to laryngoscopy and endotracheal intubation when compared to lignocaine 1.5 mg/kg. However, further studies are required to ascertain the same as our study was performed on a small population who belonged to the same race and hence cannot be extrapolated to the entire population [Tables 3-6].

CONCLUSION

This study concluded that dexmedetomidine at the dose of 1 mcg/kg IV infusion for 10 min attenuates the hemodynamic stress response to laryngoscopy and endotracheal intubation more effectively when compared to lignocaine 1.5 mg/kg IV without any side effects. The study was carried out to determine if IV dexmedetomidine at the dose of 1 μg/kg infusion over 10 min before induction is advantageous over IV preservative-free lignocaine 1.5 mg/kg 90 s before laryngoscopy to attenuate the pressor response to laryngoscopy and intubation. The results clearly confirm that both dexmedetomidine and lignocaine attenuate the pressor response to laryngoscopy and intubation. The attenuation of the pressor response is by far greater with dexmedetomidine than that by lignocaine. Therefore in patients whom pressor response to intubation

can be detrimental, dexmedetomidine appears to be a better alternative to preservative-free lignocaine.

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The authors report no conflicts of interest in this work.

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