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Comparison of Intravenous Lignocaine and Dexmedetomidine for Attenuation of Hemodynamic Stress Response to Laryngoscopy and Endotracheal Intubation

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Abstract

Objectives: To compare the safety and efficacy of lignocaine versus dexmedetomidine in attenuation of cardiovascular response to laryngoscopy. *Study design*: Randomized controlled trial. Sixtypatients of ASA I & II category posted for elective surgery under general Anesthesia were enrolled in the study. Patients were randomly divided into two Groups: Group L(Lignocaine) and Group D (Dexmedetomidine) with 30 patients in each group.

Materials and Methods: Group L received 1.5 mg/kg of lignocaine intravenous (IV) and Group D received 1 mcg/kg of dexmedetomidine as IV infusion. Thiopentone was given until eyelash reflex was lost, and intubation was facilitated with succinylcholine. Anesthesia was maintained with 33:66 oxygen, nitrous oxide, and titrated doses of inhalation agents and vecuronium was given. Hemodynamic parameters were recorded as baseline vitals, at preinduction, after induction, during intubation, 1 min, 3 mins, 5 mins, and 10 mins after intubation.

SPSS version 16 was used for analysis.

Results: All the demographic variables were well matched between groups. There was a statistically significant difference ($p < 0.001$) between dexmedetomidine and lignocaine in parameters like heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure at all time intervals after tracheal intubation, with dexmedetomidine being the most effective. Sedation scores were more with dexmedetomidine.No adverse effects were noticed in patients of both groups.

Conclusion: Dexmedetomidine attenuates the hemodynamic stress response to laryngoscopy and intubation more effectively when compared with lignocaine 1.5 mg/kg IV, without any adverse effects.

Keywords: Lignocaine, Dexmedetomidine, Laryngoscopy, Intubation, Hemodynamic stress response.

Introduction

The introduction of general anesthetics into clinical practice lead to the development of modern surgery and spawned the speciality of anesthesiology. Airway management and patient safety is the most important aspect of patient management in general Anesthesia. Endotracheal intubation protects airway, delivers anesthetic gases and protects against aspiration.[1,2] During intubation, stimulation of laryngeal and tracheal tissues causes catecholamine discharge which results in a increase in heart rate and systemic arterial pressure.[3,4] Control of heart rate and blood pressure response

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to intubation is essential in preventing the adverse cardiovascular events, because rate pressure product acts as an indicator of oxygen demand by the heart.[5] There are a wide array of drugs such as opioids, beta blockers, calcium channel blockers, nitroglycerine, alpha agonist, lidocaine which blunts this response.[6-13] Dexmedetomidine is a highly selective centrally acting α_2 agonist, more selective and potent than clonidine ($\alpha_2 : \alpha_1$, 1620:1 for dexmedetomidine, 220:1 for clonidine) Lidocaine is one of the cheapest and easily available drug for attenuation of hemodynamic response.[14,15] On this background, we compared the safety and efficacy of lignocaine versus dexmedetomidine in attenuating the cardiovascular response to laryngoscopy and intubation.

Materials and Methods

It was a randomised controlled study, conducted after approval from institution ethical committee and obtaining valid written informed consent from the patients. Sixty patients, aged 18-60 years, weighing between 40 and 75 kg, belonging to ASA grade I and II undergoing elective surgeries under general Anesthesia were subjected in the study. Patient's refusal, known allergic to study drug and with comorbidities like hypertension, cardiac, renal, cerebral, hepatic, cerebral disease, obese patients, anticipated difficult airway, and in whom intubation attempts lasted longer than 15 second were excluded.

Patients were randomised using a computer generated randomization and divided into Groups L (Lignocaine) and group D (Dexmedetomidine) with 30 patients in each group. All patients received preoperative night sedation with T. Alprazolam 0.5 mg P.O and T. Ranitidine 150 mg P.O at bed time on the previous night. After shifting the patient to operating table, baseline parameters such as heart rate, blood pressure, SpO_2, respiratory rate were recorded.

Group L received 100 ml of normal saline over a period of 10 mins and completed 10 mins before induction and 1.5 mg/kg of lignocaine was administered IV 3 min before intubation. Group D received dexmedetomidine 1 mcg/kg diluted in 100 ml of normal saline IV over a period of 10 min, and completed 10 mins before induction.

All patients were premedicated with Inj. Glycopyrolate 10 mcg/kg intramuscularly, 20 mins before induction, Inj. Midazolam 0.04 mg/kg and Inj.Fentanyl 2 µg/kg intravenously 5 minutes before induction. Patients were preoxygenated with 100% oxygen for 3 minutes. Level of sedation was assessed using Ramsay sedation score before induction of Anesthesia in both the groups. All patients were induced with Inj.Thiopentone 2.5% solution intravenously till loss of eyelash reflex occurred. Endotracheal intubation was facilitated with Succinylcholine 2 mg/kg given IV 1 min prior to laryngoscopy and intubation. Laryngoscopy was performed using Macintosh laryngoscope, trachea intubated with appropriate size endotracheal tube, confirmed with bilateral equal air entry and tube was fixed and secured. Anesthesia was maintained with Oxygen and Nitrous oxide in the ratio of 33:66, with titrated doses of volatile anesthetics and inj. vecuronium. At the end of surgery, all anesthetic agents were stopped 100% oxygen was given, reversed with inj.neostigmine 50 mcg/kg and inj.glycopyrrolate 8 mcg/kg and extubated after adequate neuromuscular efforts.

Hemodynamic parameters were recorded at baseline, preinduction, after induction, during intubation, 1 min, 3 mins, 5 mins, and 10 mins after intubation.

At the end of study, the data were compiled and subjected to statistical analysis using students paired "t" test and Fisher's exact test. SPSS version 16 was used for analysis. A statistical value of ($p < 0.001$) was considered significant.

Results

Both the groups were comparable with respect to demographic variables (Age, Height, Weight)

Table 1: Patient demographics

	Dexmedetomidine		Lignocaine		p value
	Mean	SD	Mean	SD	
Age	31.03	12.53	32.20	11.90	0.713
Weight	55.60	7.73	54.40	8.50	0.591
Height	159.93	5.70877	159.23	5.82549	0.672

Table 2: Gender

Gender	Group				p value
	Dexmedetomidine		Lignocaine		
Male	14	46.70	15	50.00	0.999
Female	16	53.30	15	50.00	
Total	30	100.00	30	100.00	

Table 3: Mallampatti Grading

MPG	Group				p value
	Dexmedetomidine		Lignocaine		
I	18	60.00	16	53.30	0.999
II	12	40.00	14	46.70	
Total	30	100.00	30	100.00	

Table 4: ASA Grading

ASA	Group				p value
	Dexmedetomidine		Lignocaine		
I	20	66.70	14	46.70	0.192
II	10	33.30	16	53.30	
Total	30	100.00	30	100.00	

Table 5: Duration of Laryngoscopy(seconds)

Dexmedetomidine		Lignocaine		p value
Mean	SD	Mean	SD	
13.90	0.90	14.30	0.80	0.109

Table 6: Induction Dose of Thiopentone (milligram)

Dexmedetomidine		Lignocaine		p value
Mean	SD	Mean	SD	
199.1	24.1	253.1	19.2	<0.0001

Table 7: Comparison of Side Effects

Side Effect	Group B	Group R
Nausea/Vomiting/Bradycardia/ Hypotension/Apnea	Nil	Nil

Hemodynamics

On comparing, the changes in heart rate between Group L and Group D is statistically significant at the time of induction and until 10 minutes after intubation (p <0.001) (Fig. 1).

The changes in systolic pressurein both the groups is statistically significant at the time of induction and until 10 minutes after intubation (p <0.001) (Fig. 2).

The difference in mean diastolic blood pressure between both the groups is statistically significant at the time of intubation and 1 minute, 3 minutes post intubation (Fig. 3).

The difference in mean arterial blood pressure between both the groups is statistically significant at intubation, 1 min, 3 min p <0.001), 5 min (p <0.001) and 10 min p <0.001) post intubation (Fig. 4).

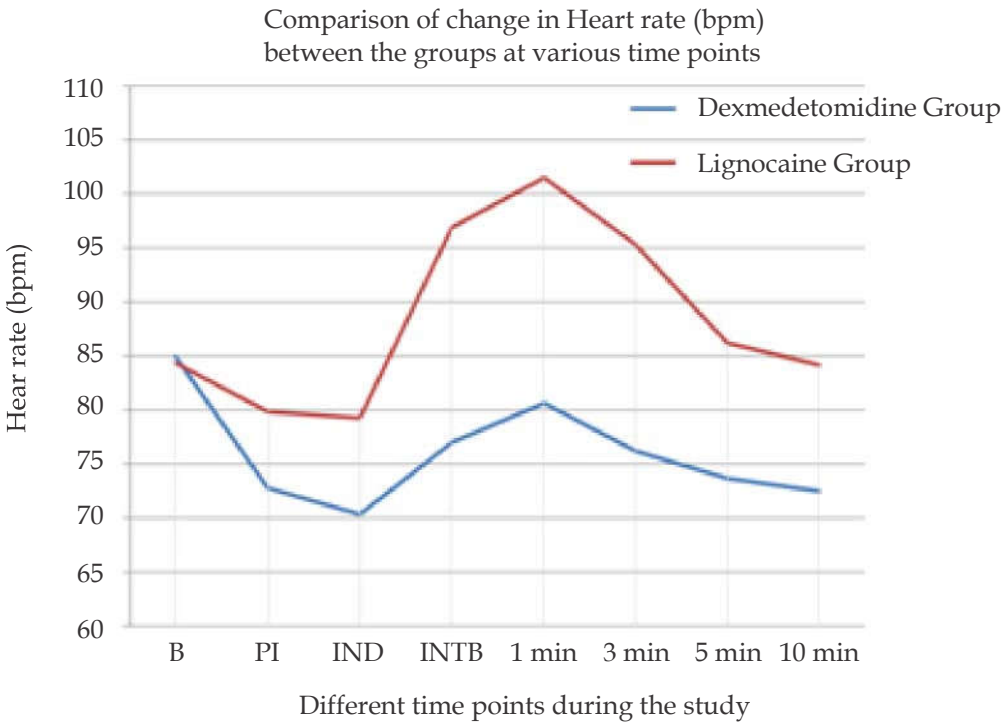

Fig. 1: Comparison of Heart rate in both groups.

B= Baseline; PI= Pre-induction; IND= Induction; INTB= During intubation; 1 min, 3 min, 5 min & 10 min represents readings after 1, 3, 5 and 10 minutes of intubation.

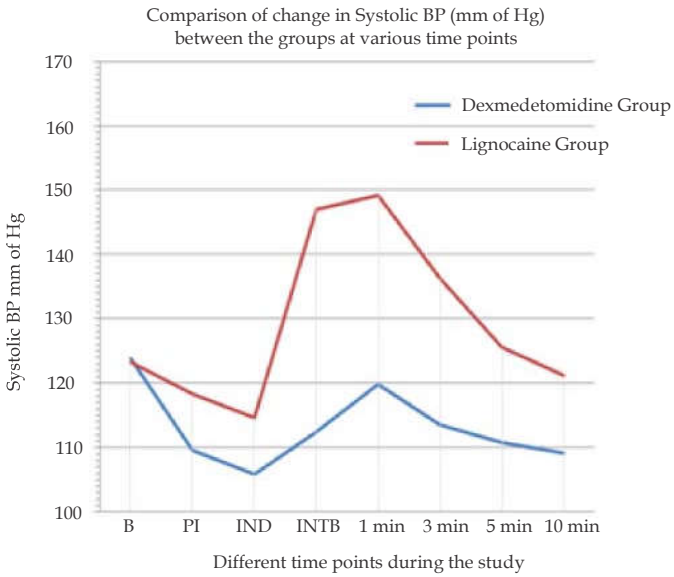

Fig. 2: Comparison of Systolic blood pressure in both groups.

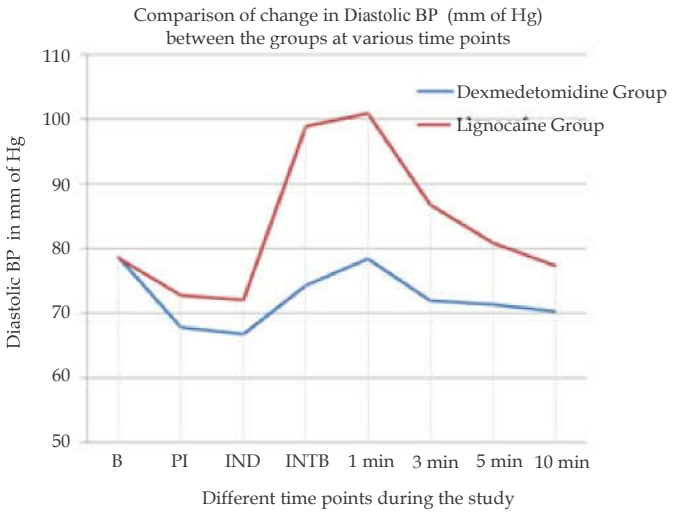

Fig. 3: Comparison of Diastolic blood pressure in both groups.

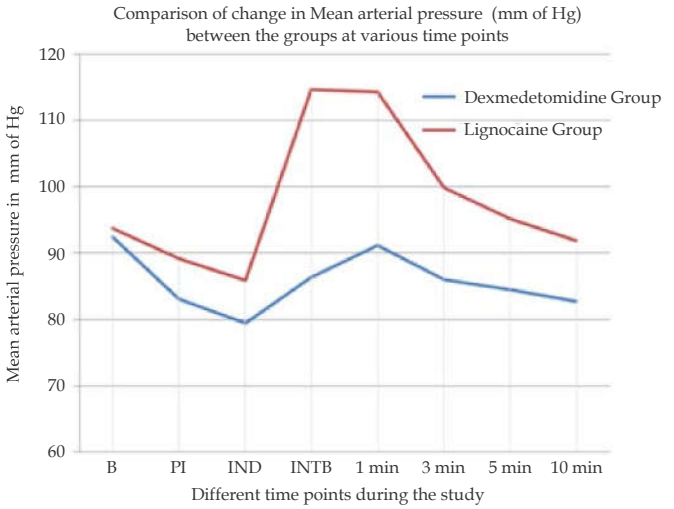

Fig. 4: Comparison of Mean arterial pressure in both groups.

Discussion

Hemodynamic response to laryngoscopy and endotracheal intubation was described by Reid and Brace in 1940.[16] The series of physiological changes following laryngoscopy and intubation occurs as a result of release of catecholamines and considered as the most critical event during general Anesthesia.[17] The response is transient occurring 30 seconds after intubation, peaks at 1 minute and lasting for less than 10 minutes. Usually these changes are transient and well coped by healthy individuals, but can be detrimental in cardiac and cerebrovascular patients.[5] This pressor response may predispose to development of pulmonary edema, acute ventricular failure, dysrrhythmias, intraoperative MI and cerebrovascular accident.[18,19] Various studies had been conducted to find an effective method to attenuate this pressor response. In our study, we had done comparative analysis between lignocaine and dexmedetomidine which drug attenuated the stress response better.

In our study, lignocaine 1.5 mg/kg IV given 3 min before intubation. Various studies[20-22] concluded that this dose is sufficient to debilitate the pressor response to intubation.

Dexmedetomidine used in our study was 1 mcg/kg diluted in 100 ml NS given over 10 min Various studies[23-25] proved that 0.5-1 mcg/kg of dexmedetomidine was sufficient to attenuate the hemodynamic response.

All patients in Group L had sedation score 2 and most of the patients in Group D had score of 3. Many authors[26,27] have reported that dexmedetomidine infusion produces sedation which are arousable to oral commands are in accordance with our study.

Scheinin et al.[28], L. keniya et al.[29], concluded that dexmedetomidine infusion given IV preinduction decreased the dose of thiopentone for induction of Anesthesia which was similar to our study.

Following infusion of dexmedetomidine, there was 14.25% reduction heart rate compared to Group L 5.33% and 16% reduction of mean arterial pressure in Group D compared to 4.9% in Group L which was statistically significant. Sukhminderjit Singh Bajwa et al.[30], Ozkose et al.[31], Aho et al.[32], Ferdi Menda et al.[33] concluded from their studies that dexmedetomidine (1 mcg/kg) attenuates the hemodynamic response significantly as of our study.

Miller CD et al., Wilson IG et al. reported that lignocaine fails to attenuate hemodynamic response significantly and our results are in accordance them.

None of the patients in our study developed hypotension, bradycardia, apnea.

Conclusion

From the present study, we conclude that dexmedetomidine 1 mcg/kg IV as infusion for 10 minutes attenuates the hemodynamic stress responseto laryngoscopy and intubation more effectively when compared with lignocaine 1.5 mg/kg IV, without any adverse effects.

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