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Original Research Article

A Comparative Study of Nebulised Dexmedetomidine Versusnebulised Lignocaine in Blunting The haemodynamic Responses to Laryngoscopy and Intubation in Elective Surgeries Under General Anesthesia

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Abstract

Background: Endotracheal intubation is a procedure where placement of endotracheal tube into the trachea through mouth or nose to maintain airway patency and it is associated with various hemodynamic stress responses. This response is mainly mediated by raised plasma adrenaline concentration. In patients with hypertension, raised intracranial pressure, cerebral vascular disease or with ischaemic heart disease, this cardiovascular response to intubation is more life-threatening in which increase in myocardial oxygen consumption can lead to myocardial infarction.

Aims: This study is undertaken to compare dexmedetomidine to lidocaine as regards to itsefficacy on attenuation of intubation response.

Materials and Methods: It is a randomized control study conducted on 100 American society of Anesthesiologists physical status I and II patients aged between 18 to 45 years with Mallampati grade I and II. This study was undertaken in 2 groups Group D and Group L to compare HR, SBP, and DBP from the time of intubation till 10 minutes. The results were analysedusing an unpaired t test, Chi-square test and ANOVA.

Results: In present study decrease was seen in Systolic blood pressure in both groups.
Group L had more decrease (21.2 mean) in systolic blood pressure than Group D
(Mean 11.5). Decrease in Diastolic blood pressure was observed in both groups. Group L had more decrease (16.92 mean) in Diastolic blood pressure than Group D (mean 4.5).
Decrease in heart rate was observed at 10 mins from pre-Induction. Statistical significance was observed on pre induction, induction, at the time of intubation, at 3 mins and 5 mins.

Conclusion: Hemodynamic parameters in present study shows decrease in SBP, DBP and MAP was seen in both group. When compared Group L cases had more rate of decrease than Group D cases in SBP, DBP and MAP.

Introduction

General anaesthesia with endotracheal intubation and intermittent positive pressure ventilation is frequently performed in the practice of anaesthesia. Endotracheal intubation is a procedure where placement of endotracheal tube into the trachea through mouth or nose to maintain airway patency and it is associated with various hemodynamic responses. The most stressful situations are seen during the period of induction, intubation and extubation. These cardiovascular responses are mainly due to enhanced neuronal activity in cervical sympathetic fibers caused by the mechanical stimulation of upper respiratory tract areas- the nose, the epipharynx, the laryngopharynx and tracheobronchial tree during endotracheal intubation. In healthy individuals upto

50% increase in mean arterial pressure and heart rate is seen and peaking at 1-2 minutes and returning to baseline within 15 minutes during intubation. This response is mainly mediated by raised plasma adrenaline concentration. To reduce these responses, various techniques have been suggested. The stress response can also be minimized by decreasing the duration of laryngoscopy to less than 15 seconds. Some of these includes administration of lignocaine via intravenous, inhalational, endotracheal routes, deep inhalational anaesthetics, ganglion blockers, precurarization, narcotics (fentanyl, alfentanyl etc;), vasodilators, nitroglycerine, and calcium channel blockers, alpha 2 agonists like clonidine & dexmedetomidine. Lidocaine is easily available and most commonly used drug for attenuation of pressor response. [1,2] Dexmedetomidine a new generation highlyselective alpha 2 agonists, attenuate the pressor response by decreasing the sympathetic tone, and it also has analgesic properties which makes it more favorable drug to be used to blunt these responses, as itcan be used as an adjunct to anaesthesia. Nebulization is a process by which medications are added to inspired air and converted into a mist that is then inhaled by the patient into their respiratory system. Before the inhaled drug can be absorbed into the blood from the lung periphery, it has several barriers to overcome: lung surfactant, surface lining fluid, epithelium, interstitium and basement membrane and the endothelium. Drug absorption is regulated by a thin alveolar–vascular permeable barrier. Nebulization offers the potential to achieve both a high local drug concentration and a lower systemic toxicity. Hence in this study nebulization route has been used to compare the efficacies of lignocaine and dexmedetomidine.

Materials and Methods

A randomised controlled study was conducted among 100 patients after obtaining consent. A total of 100 patients undergoing elective surgeries were selected. This study was undertaken in 2 groups. Group-D consists of 50 patients, who received nebulized Dexmedetomidine with the dose of 1 mcg/kg diluted up to 5 ml NS for 10 minutes prior to induction. Group –L consists of 50 patients, who received Lignocaine 4%, at the dose of 3mg/kg body weight for 10 mins before induction.

Inclusion Criteria

Age range 18-45 years of age, ASA grade I & II, Mallampatti class I &II

Exclusion Criteria

ASA grade III & IV, Mallampatti class III & IV, Patients who are known cases of COPD, Cardiovascular illnesses, Cerebro vascular diseases, psychiatric illness and liver disorder, Patients who have known allergy to either lignoacaine and other anesthesia drugs, Patients with history of laryngeal, tracheal surgery or pathology and Refusal or inability to understand the procedure.

No sedation was given on the night before surgery. Nebulisation was done with either dexmedetomidine for or lignocaine with a nebulizer facemask and a continuous flow of oxygen at the rate of 6 litres per min for 10 mins before induction of anesthesia in sitting position. All patients were premedicated with Inj. Glycopyrrolate, 0.2mg I.V, Inj Fentanyl 2µg/kg IV. Induction agent was Propofol 1-2 mg /kg/body weight titrated to theloss of verbal response and the amount of drug administered was noted Inj Atracurium was used as a muscle relaxant at the dose of 0.5 mg/kg/ body weight. The D Group received nebulisation dexmedetomidine at the dose of 1 mcg /kg and diluted upto 5ml NS.

The group L received Lignocaine 4% nebulization at the dose of 3 mg/kg 10 mins prior induction. Time taken for intubation did not exceed 15-20 seconds in both groups and the patient was connected to the ventilators post intubation and was undisturbed for a period of 10 mins after intubation Haemodynamic changes like Heart rate (HR), Systolic Blood Pressure (SBP), Mean Blood Pressure (MBP) and Diastolic Blood Pressure (DBP), SPO2, were monitored before, during and 1, 3, and 5, 7 and 10 mins after endotracheal intubation.

Results

Table 1: Heart rate at various intervals in present study

Intervals	Group-D	Group-L	P-Value
Pre Induction	84.54±12.70	92.80±16.08	0.0053**
Induction	82.82±11.24	92.40±13.18	0.0002***
Intubation	81.84±11.82	88.72±12.73	0.0062**
1 min	86.70±16.29	85.04±13.23	0.577
3 mins	91.48±23.22	82.76±12.78	0.022*
5 mins	92.20±25.01	81.28±9.78	0.004**
7 mins	82.78±16	80.92±11.16	0.5
10 mins	80.32±13.29	79.24±10.26	0.65

In present study in both group there was decrease in heart rate at 10 mins from pre-Induction. Statistical significance was observed on pre induction, induction, at the time of intubation, at 3 mins and 5 mins. No

significance was seen at 1 min, 7 mins and 10 mins.

Table 2: Systolic BP at various intervals in present study

Intervals	Group-D	Group-L	P-Value
Pre Induction	129.8±16.86	126.6±13.15	0.29
Induction	122.6±12.23	124.5±11.67	0.42
Intubation	121.2±10.70	121.4±13.01	0.92
1 min	132.5±18.79	109.7±12.92	<0.0001***
3 mins	133.4±23.36	106.0±22.95	<0.0001***
5 mins	125.9±14.98	110.6±12.26	<0.0001***
7 mins	120.4±15.53	105.5±11.68	<0.0001***
10 mins	118.3±15.49	105.4±11.99	<0.0001***

In present study decrease was seen in Systolic blood pressure in both groups. From preinduction to 10 mins there was statistical difference between two groups. Group L had more decrease (21.2 mean) in systolic blood pressure than Group D (mean 11.5).

In group D Before induction 129.8 mean SBP was observed where after 10 mins it was decreased to 118.3. In Group L 126.6 mean SBP was observed before induction and at 10 minsit was decreased by 21.2 mean. Statistically significant was observed between both groups.

Table 3: Diastolic BP at various intervals in present study

Intervals	Group-D	Group-L	P-Value
Pre-Induction	81.06±10.04	83.20±7.80	0.23
Induction	81.30±10.11	77.12±16.10	0.12
Intubation	79.24±9.15	79.64±7.33	0.80
1 min	82.40±12.01	71.04±11.82	<0.0001***
3 mins	86.08±17.24	69.40±11.11	<0.0001***
5 mins	84.04±11.39	67.84±8.46	<0.0001***
7 mins	77.72±11.93	65.68±7.90	<0.0001***
10 mins	76.56±11.58	66.28±8.87	<0.0001***

In present study decrease was seen in Diastolic blood pressure in both groups. From pre induction to 10 mins there was statistical difference between two groups. Group L hadmore decrease (16.92 mean) in Diastolic blood pressure than Group D (mean 4.5). In group D Before induction 81.06 mean DBP was observed where after 10 mins it wasdecreased to 76.56. In Group L 83.20 mean DBP was observed before induction and at 10 mins it was decreased by 16.92 mean. Statistically significant was observed between both groups P<0.0001.

Table 4: Mean arterial BP at various intervals in present study.

Intervals	Group-D	Group-L	P-Value
Pre-Induction	116.8±37.92	97.60±7.86	0.0007***
Induction	112.9±33.48	93.04±12.96	0.0002***
Intubation	110.9±32.59	93.56±7.91	0.0004***
1 min	118±37.62	83.96±11.39	<0.0001***
3 mins	120.6±37.84	81.68±11.90	<0.0001***
5 mins	116.5±37.03	82.08±8.92	<0.0001***
7 mins	110.5±39.67	78.96±8.37	<0.0001***
10 mins	109±40.01	79.32±9.21	<0.0001***

In present study decrease was seen in MAP in both groups. From pre induction to 10 mins there was statistical difference between two groups. Group L had more decrease (18.28 mean)in Diastolic blood pressure than Group D (mean 7.8). In group D Before induction 116.8 mean MAP was observed where after 10 mins it was decreased to 109. In Group L 97.60 mean MAP was observed before induction and at 10 mins it was decreased by 18.28 mean. Statistically significant was observed between both groups P<0.0001.

Discussion

Laryngoscopy and endotracheal intubation are known to cause an undesired noxious stimulus to produce undue pressor responses like raised

pulse rate and mean blood pressure, laryngospasm, bronchospasm, increased intracranial pressure and intraocular pressure. Stimulating the mechanoreceptors that are present in the pharyngeal wall, epiglottis and vocal cords, is thought to be the cause for this undesired hemodynamic response. In present study our aim was to compare the efficacy of nebulized dexmedetomidine with the dose of 1 mcg/ kg versus nebulized lignocaine 4% with the dose of 3mg /kg on hemodynamic responses during laryngoscopy and intubation. In present study in Group D most of the cases 30% were seen in 31 – 40 years of age. group followed by 28% between 41 – 50 years of age group, 26% in 21-30 years of age group, 12% cases hadage more than 50 years of age and 4% cases had age less than or equal to 20 years of age. In Group D 37.42 years mean age was observed. In Group L most cases 38% had age between 41-50 years of age followed by 26% cases in 21-30 years of age, 18% cases had age between 31 – 40 years of age, 10% cases had age more than 50 years where 8% cases were seen having age less than or equal to 20 years of age. 37.98years mean age was observed. No statistically significance was seen between two groups.

In present study in Group D 40% cases were male cases and 60% were female cases where in Group L 44% were male cases and 56% were female cases. No relation was seen between two groups. In present study in Group D 38% cases had weight between 51-60Kg, 32% cases had between 61-70, 24% cases had weight less than or equal to 50 and 6% cases had weight more than 70 Kg. 58.56Kg mean weight was observed in Group D. In Group L 46% cases had weight between 61-70 Kg, 30% cases had between 51-60, 18% cases had less than or equal to 50 and 6% cases had weight more than 70 Kg. 61.26Kg mean weight was seen. No statistical significance was seen in two groups. Sebastian et al.[3] compared the effect of 0.5 and 0.75mcg/kg with normal saline in attenuating pressor response. They showedthat the inter group comparison resulted in a statistically significant reduction in HR by dexmedetomidine than normal saline. These findings correlated with findings in our study in that it reduced HR significantly with dexmedetomedine 0.5mcg/kg.

Lee CW et al [4] defined Group C (cases who had normal saline and Group D cases who received dexamedetomine), in their study they observed the mean HR was significantly lower in group D than in group C at 3 and 5 min after intubation, but no intergroup difference was observed at 1 min. In group C, HR was significantly higher at 1 min (92.9 ± 13.5, P < 0.001) and 3 min (82.2 ± 10.3, P < 0.001) after intubation than at baseline (70.3 ± 7.4), and in group D, HR was significantly higher at 1 min (85.2 ± 12.6, P < 0.001) but lower at 5 min (65.7 ± 9.5, P = 0.007) after intubation than at baseline (72.1 ± 7.0). The maximum increase in HR was 32.1% in group C, and the minimum increase was 18.1% ingroup D at 1 min after intubation. The mean SBP was significantly lower in group D than ingroup C at 1, 3, and 5 min after intubation. In group C, SBP was significantly higher at 1 min (186.0 ± 26.6, P < 0.001) and 3 min (160.1 ± 23.2, P < 0.001) after intubation than at baseline (135.1 ± 13.4), and in group D, SBP was significantly higher at 1 min (165.2 ± 16.2, P < 0.001) but lower at 5 min (108.4 ± 12.6, P < 0.001) after intubation than at baseline (135.4 ± 23.8). The maximum increase in SBP was 37.7% in group C, and the minimum increase was 22% in group D at 1 min after intubation. The mean DBP was significantly lower in group D than in group C at 3 and 5 min after intubation, but no intergroup difference was observed at 1 min. In group C, DBP was significantly higherat 1 min (104.8 ± 14.9, P < 0.001) and 3 min (87.9 ± 15.5, P = 0.028) after intubation than at baseline (79.7 ± 9.8). In group D, DBP was significantly higher at 1 min (97.0 ± 14.5, P < 0.001) and lower at 5 min (66.5 ± 7.2, P < 0.001) after intubation than at baseline (76.7 ± 8.3). The maximum increase in DBP was 31.5% in group C, and the minimum increase was 26.4% in group D at 1 min after intubation.

Vishalakshi Patil et al.[5] study the average rise in HR in group III(4% lignocaine nebulisation) was 8.5% in group II(2% lignocaine nebulisation) was 10.7% and in control the rise was 23.75%. the average rise in SBP in group III was 4% in group II was 13.3% and in control 16.1% was noted. In our study we noted the average rise in HR in 4% lignocaine nebulisation 76 group to be 5% and the average raise in SBP was 2% results were comparable with the above study.

In present study in both group there was decrease in heart rate at 10 mins from pre-Induction. Statistical significance was observed on pre induction, induction, at the time ofintubation, at 3 mins and 5 mins. No significance was seen at 1 min, 7 mins and 10 mins. Decrease was seen in Systolic blood pressure in both groups. From pre induction to 10 mins there was statistical difference between two groups. Group L had more decrease (21.2 mean) in systolic blood pressure than Group D (mean 11.5). In group D Before induction 129.8 mean SBP was observed where after 10 mins it was decreased to 118.3 inGroup L 126.6 mean SBP was observed before induction and at 10 mins it was

decreased by 21.2 mean. Statistically significant was observed between both groups. Decrease was seen in Diastolic blood pressure in both groups. From pre induction to 10 mins there was statistical difference between two groups. Group L had more decrease (16.92 mean) in Diastolic blood pressure

than Group D (mean 4.5). In group D Before induction 81.06 mean DBP was observed where after 10 mins it was decreased to 76.56 inGroup L 83.20 mean DBP was observed before induction and at 10 mins it was decreased by 16.92 mean. Statistically significant was observed between both groupsP<0.0001.

Lee GT et al [6], study the mean MAP was significantly lower in group D than in groupC at 1, 3, and 5 min after intubation. In group C, MAP was significantly higher at 1 min (137.2 ± 18.3, P < 0.001) and 3 min (116.9 ± 16.2, P < 0.001) after intubation than at baseline (100.3 ± 11.9), and in group D, MAP was significantly higher at 1 min (123.4 ± 13.8,P < 0.001) and significantly lower at 5 min (83.4 ± 8.4, P < 0.001) after intubation than at baseline (97.7 ± 14.9). The maximum increase in MAP was 36.8% in group C, and the minimum increase was 26.4% in group D at 1 min after intubation.

In present study decrease was seen in MAP in both groups. From pre induction to 10 mins there was statistical difference between two groups. Group L had more decrease (18.28 mean) in Diastolic blood pressure than Group D (mean 7.8). In group D Before induction 116.8 mean MAP was observed where after 10 mins it was decreased to 109.

In Group L 97.60 mean MAP was observed before induction and at 10 mins it was decreased by 18.28 mean. Statistically significant was observed between both groupsP<0.0001.

The hemodynamic responses were first recognised in 1940 by Reid and Bruce et al. Burstein et al1, found that the pressor response occurring at laryngoscopy and endotracheal intubation was due to augmented sympathetic response, provoked by stimulation of epipharynx and laryngopharynx. These factors were further confirmed by Prys-Roberts. The efferent sympathetic outflow to the heart is T1-T4, while that to adrenal medulla.[7,8]

In patients undergoing total thoracolumbar anesthesia, Gurulingappa et al[9] observed that cardiovascular response to intubation is abolished. Thus, the cardiovascular responses to intubation may be due to diffuse activation of sympathetic system involving cardioaccelaratory fibers as well as the activation of adrenal medulla. It is well known thattopical application of local anesthesia in the form of sprays have been efficient in causing airway numbness and this has been used for regular outpatient procedures. Similarly, nebulization with lignocaine would have caused local anesthesia effect in the upper airway. In group L patients some of them have reported a sense of airway numbness after nebulization. [10,11,12]

Conclusion

In present study Group D cases received dexmedetomidine with the dose of 1 mcg/kg where Group L cases had received nebulized lignocaine 4% with the dose of 3mg /kg on hemodynamic responses during laryngoscopy and intubation. In present study most of the cases ranged between 31 – 40 years of age. In Group D 37.42years mean age was observed where 37.98years mean age was observed in Group L 58.56Kg mean weight was observed in Group D cases. 61.26Kg mean weight was seen in Group L cases. HR, SBP, DBP and MAP were recorded at interval of pre induction, induction, intubation, at 1 min, at 3 mins, at 5 mins, at 7 mins and at 10 mins. In present study decrease in SBP, DBP and MAP was seen in both groups. When compared both the group statistically significant was observed in drop of SBP, DBP and MAP Group L cases had more rate of decrease than Group D cases in SBP, DBP and MAP Lignocaine nebulization induction with general anaesthesia found to be more efficacious in attenuation of the haemodynamic changes to laryngoscopy and endotracheal intubation when compared with dexmedetomidine with the dose of 1 mcg/ kg.

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