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Prevention of Pressor response to Laryngoscopy: A Comparison of Lignocaine with Dexmedetomidine

Ayesha Shahid, Muhammad Salman Maqbool, Sidra Shabbir, Fahd Mudassar Hameed, Nargis Shabana, Fareeha Tayyab

ABSTRACT:

Objective: To compare intravenous Lignocaine with Dexmedetomidine for prevention of pressor response to laryngoscopy.

Study design and setting: Randomized Clinical Trial at Anesthesia Department, RIHS Islamabad. (1st May2019 To 30th October 2019)

Methodology: 68 patients with age 20-60 years, ASA status(I or II), planned for elective surgeries under GA were included in this study and randomly divided into groups A and B by lottery method. Patients with history of hypertension, heart blocks, beta-blockers were excluded from study. Group-A patients were given injection Lignocaine 2% 1mg/kg 1min before induction and Group-B patients were given injection Dexmedetomidine 1ug/kg 15 min before induction of anesthesia. After premedication with injection Midazolam, Nalbuphine, and Ondansetron, Induction of anesthesia was done with injection Propofol and Cisatracurium. Patients were intubated. Use of stylet,BURP maneuver, incidence of laryngospasm was noted. Vitals before and after intubation were noted.

Results: There was no significant difference in demographic profile,use of a stylet, BURP maneuver and laryngospasm in both groups. ANOVA test shows significant decrease in change in Systolic BP in Dexmedetomidine group at 2 and 3 minutes after intubation as compared to Lignocaine group however Diastolic BP and Heart rate was comparable in both groups. Paired t test showed a significant decrease in Systolic and Diastolic BP in both groups(more in Dexmedetomidine group).Regarding heart rate paired t test showed insignificant difference in Lignocaine group and significant difference in Dexmedetomidine group.

Conclusion: Intravenous Dexmedetomidine is superior to Lignocaine to prevent pressor response to laryngoscopy.

Keywords: Dexmedetomidine, Laryngoscopy, Lignocaine, Pressor response

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INTRODUCTION:

Pressor response to laryngoscopy was first described by Reid and Brace in 1940. Both laryngoscopy and intubation are essential part of general anesthesia[1]. Intubation not only helps us to maintain patency of airway, it also assures that adequate ventilation is being delivered to patient. It reduces risk of aspiration of gastric contents and facilitates delivery of anesthetic gases to patient. During laryngoscopy, extension of head at the atlantooccipital joint not only brings oral, laryngeal and pharyngeal axes into alignment, but also improves glottic view. The stretching of oropharyngeal tissues during laryngoscopy result in activation of sympathetic nervous system.[2] The reflex tachycardia and hypertensive effects of laryngoscopy are greater than that of tracheal intubation.[2] During laryngoscopy and intubation, afferent signals from glottis and epiglottis are carried by vagus and glossopharyngeal nerves to vasomotor centers in brain stem .Activation of vasomotor centers cause intense sympathetic discharge in the body.[3] Elevated catecholamines levels in the body results in 20-27% increase in heart rate and 30-50% increase in blood pressure.[4]

This hemodynamic response is directly proportional to duration of laryngoscopy and intubation. This response begins in 15 seconds, reaches its peak at 45 seconds and

may last for 5 minutes after intubation.[5] Although this pressor response is transient in most of the patients, it can be detrimental to those having history of hypertension, ischemic heart disease and cerebrovascular accidents.[6] The underlying mechanism involves vasoconstriction due to sympathetic stimulation. This results in increase in myocardial work and demand for coronary blood flow also increases. If coronary arteries are already narrowed, than they can't accommodate increase in blood flow and some parts of myocardium may get under perfused.[2] Perioperative myocardial infarctions have been reported after laryngoscopy and intubation that increases the mortality and morbidity by 12-40%.[7] Light anesthesia, prolonged laryngoscopy, anatomically difficult airway, multiple intubation attempts, use of miller blade causing more pressure on posterior part of tongue, excessive force applied during intubation are the various factors that affect hemodynamics at time of intubation.[8]

Lignocaine belongs to amide group of local anesthetic and have been widely used to blunt pressor response to laryngoscopy. Bromage reported that an intravenous dose of Lignocaine given 3 minutes before intubation effectively blunts pressor response.[9] Dexmedetomidine is highly selective α-2 adrenoceptor agonist, having sedative, analgesic, anxiolytic, sympatholytic, and opioid-sparing properties. Dexmedetomidine has got unique sedative ability that allows patient to be cooperative and communicative when stimulated, hence it allows slow and easy transition from sleep to wakefulness. It has got sympatholytic properties that help to achieve stable hemodynamic in perioperative period. It not only decrease myocardial oxygen consumption but also decreases heart rate. All these effects help to reduce cardiac complications perioperatively.[10]

The rationale of this study was to compare intravenous Lignocaine with Dexmedetomidine for prevention of pressor response to laryngoscopy so that better management plans can be improvised to avoid detrimental effects of sympathetic discharge at the time of laryngoscopy and intubation. This study would be helpful to improve anesthesia plans in patients with history of hypertension and ischemic heart diseases.

METHODOLOGY:

The study approval was given by Institutional Dean and Head Research Ethical Committee, Rawal Institute of Health Sciences, vide letter No. RIHS-REC/039/19 dated 15.04.2019. Non probability consecutive sampling technique was well-thought-out for this prospective interventional study. This study was done at Anesthesia Department of Rawal Institute of Health Sciences Islamabad from 1st May 2019 to 30th October 2019. 68 patients with age 20-60 years, ASA[11] physical status class I or II, planned for elective surgeries under general anesthesia were included in this study. Patients with history of hypertension, heart blocks or taking beta blockers were excluded from study. Informed written consent was taken from all the patients included in the study. Sample size was calculated by using WHO sample size calculator with assumptions (confidence level=95%, alpha error=5%, mean heart rate in Lignocaine group=82.2[12], mean heart rate in Dexmedetomidine group=71.9[12], common sigma= 15) the sample size came out to be 68 (34 cases placed by lottery method into each group). Group A patients were given injection Lignocaine plain 2% 1mg/kg 1min before induction of anesthesia. Group B patients were given injection Dexmedetomidine 1ug/kg 15 min before induction of anesthesia. Informed written consent was taken from all the patients included in this study. Patients fasted and pre-medicated according to ASA guidelines. Electrocardiograph, pulse oximetry, end-tidal CO_2, blood pressure monitors attached and baseline readings noted. Patients were divided in group A and B by lottery method. All patients were premedicated with injection Midazolam 2 mg intravenously, injection Nalbuphine 0.1 mg/kg and inj Ondensetron 4mg. Induction of anesthesia was done with injection Propofol 2mg/kg and Cisatracurium 0.15 mg/kg. Patients were ventilated for 3 minutes and intubated with ETT of appropriate size. Vitals were noted on arrival in OT, before induction and 5 minutes after intubation (1 minute interval). Anesthesia was maintained by mixture of 50% N_2O, 50% O_2 and 0.8% Isoflurane. Injection Cisatracuium was used for maintenance during procedure. Use of stylet or BURP maneuver was noted during intubation. Any incidence of laryngospasm was also noted. At the end of surgery, on return of muscle power, residual neuromuscular blockade was reversed by injection Neostigmine 30µg/kg along with Atropine 15µg/kg intravenously. Patients were extubated and after complete recovery from anesthesia patients were shifted to recovery room. All the data was recorded on forms and confidentiality of patients was maintained.

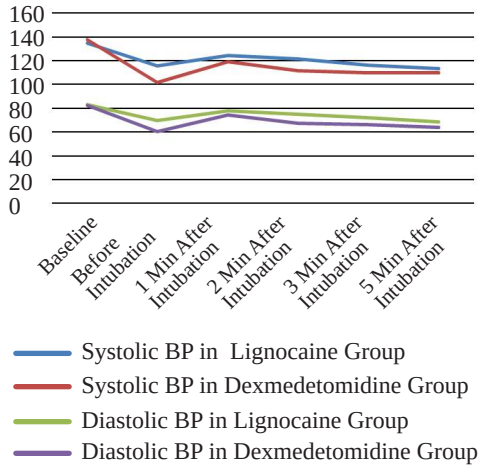
RESULTS:

Data was entered and analyzed in SPSS 22. Demographic profile is shown in table-1. There was no significant difference in demographic profile in both groups. Factors that can effect pressor response like use of stylet, BURP maneuver and evidence of laryngospasm was also compared among both groups and was found to be insignificant. Systolic BP, diastolic BP in two groups are shown in graph 1 and heart rate are shown in graph 2.

Table-1: Demographic data (n=68)

	Group-A (Lignocaine plain)	Group-B (Dexmedeto-midine)	P value
Age(mean in years)	37.70± 12.97	37.32 ±13.36	0.90
Male(percentage)	14	9	0.20
Female(percentage)	20	25	
Bronchospasm (%)	0(0)	2(5.9)	0.151
Stylet (%)	3(8.8)	4(11.8)	0.69
Burp (%)	11(32.4)	12(35.3)	0.79

Graph 1: Systolic and Diastolic BP in Two Groups



Graph 2: Heart Rate in Two Groups

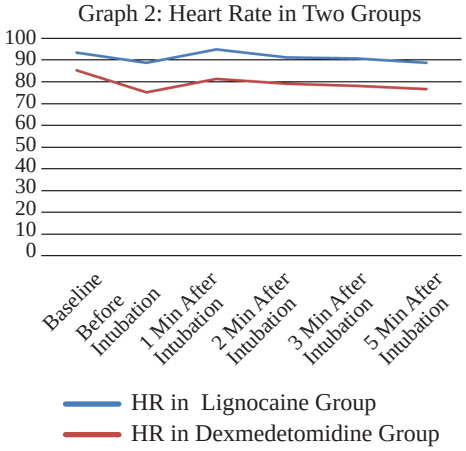


Table 2: ANOVA test to compare % of change in Systolic BP, Diastolic BP and Heart Rate in both groups

	A	B	p-value
% of Change in systolic BP at 1 min	-6.74 ±17.8	-12.44 ±18.4	0.199
% of Change in systolic BP at 2 min	-9.44 ± 14.5	-17.9 ± 16.4	0.028
% of Change in systolic BP at 3 min	-13.54 ± 14.4	-19.9 ± 11.9	0.049
% of Change in systolic BP at 5 min	-15.7± 14.5	-19.4± 13.3	0.278
% of change in diastolic BP at 1 min	-7.73± 18.89	-8.83± 19.30	0.813
% of change in diastolic BP at 2 min	-10.66 ± 20.18	-17.40± 17.45	0.145
% of change in diastolic BP at 3 min	-14.09 ± 22.65	-18.91 ± 16.47	0.320
% of change in diastolic BP at 5 min	-18.8 ± 22.46	-21.5 ± 18.18	0.579
% of change in heart rate after I min	2.31± 12.23	-3.76 ± 17.55	0.102
% of change in heart rate after 2 min	-1.71 ± 11.27	-6.37 ± 16.02	0.173
% of change in heart rate after 3 min	-2.4± 11.80	-7.02 ± 19.07	0.233
% of change in heart rate after 5 min	-3.91 ± 14.47	-8.63± 18.1	0.240

ANOVA test was applied to compare change in systolic BP, diastolic BP and heart rate at 1, 2, 3 and 5 minutes after intubation among two groups keeping baseline value as reference.

Table 3: Paired T test to compare mean of Systolic BP, Diastolic BP and Heart Rateamong same groups.

Systolic BP in Lignocaine group		p-value
Systolic BP at baseline	134.97	
Systolic BP at 1 min after intubation	124.44	0.014
SystolicBP at 2 min after intubation	121.38	0.000
SystolicBP at 3 min after intubation	116.32	0.000
SystolicBP at 5 min after intubation	113.18	0.000
SystolicBP in Dexmedetomidine group		
SystolicBP at baseline	137.85	
SystolicBP at 1 min after intubation	119.32	0.000
SystolicBP at 2 min after intubation	111.71	0.000
SystolicBP at 3 min after intubation	109.53	0.000
SystolicBP at 5 min after intubation	109.85	0.000
Diastolic BP in Lignocaine group		
Diastolic BP at baseline	82.29	
Diastolic BP at 1 min after intubation	74.18	0.006
Diastolic BP at 2 min after intubation	67.44	0.00
Diastolic BP at 3 min after intubation	66.12	0.000
Diastolic BP at 5 min after intubation	63.76	0.000
Diastolic BP in Dexmedetomidine group		
Diastolic BP at baseline	85.15	
Diastolic BP at 1 min after intubation	77.79	0.014
Diastolic BP at 2 min after intubation	75.06	0.005
Diastolic BP at 3 min after intubation	72.24	0.001
Diastolic BP at 5 min after intubation	68.53	0.000
Heart Rate in Lignocaine group		
Heart Rate at baseline	93.53	
Heart Rate at 1 min after intubation	94.97	0.453
Heart Rate at 2 min after intubation	91.15	0.205
Heart Rate at 3 min after intubation	90.74	0.174
Heart Rate at 5 min after intubation	88.82	0.054
Heart Rate in Dexmedetomidine group		
HR at baseline	85.32	
Heart Rate at 1 min after intubation	81.24	0.123
Heart Rate at 2 min after intubation	79.09	0.016
Heart Rate at 3 min after intubation	78.26	0.021
Heart Rate at 5 min after intubation	76.74	0.004

When % of change in systolic BP was compared among two groups, there was a significant difference at 2 and 3 minutes after intubation (p value less than 0.05) with more drop in systolic BP in Dexmedetomidine group. However this difference was not significant at 1 and 5 minutes after intubation. As shown in table 2.

When % of change in diastolic BP and heart rate was compared among both groups, no significant difference was observed at 1, 2 3 and 5 minutes after intubation. As shown in table 2. Paired t test was applied to compare systolic BP, diastolic BP and heart rate in same groups keeping baseline value as referenced value. When systolic BP was compared

in Lignocaine group, there was significant decrease in systolic BP after intubation (p value less than 0.05) but this decrease was not significant at 1 minute after intubation. In Dexmedetomidine group there was significant decrease in systolic BP after intubation (more than Lignocaine group) as shown in table 3. When paired t test was applied to diastolic BP, there was significant reduction in both groups except at 1 minute after intubation in Dexmedetomidine group.

When paired t test was applied to heart rate, there was no significant difference in Lignocaine group, however in Dexmedetomidine group there was significant decrease in heart rate at 2, 3 and 5 minutes, but no significant difference was observed at 1 minute after intubation as shown in table3.

DISCUSSION:

Review of literature showed various drugs that can be used to blunt pressor response to laryngoscopy. These include Opioids, Beta blockers, Calcium channel blockers, Local anesthetics, Benzodiazepines, Barbiturates, Alpha adrenergic antagonists, Angiotensin converting enzyme inhibitors, Pregabalin, nerve blocks and inhalational anesthetics. [13]

In last few years Dexmedetomidine has been used through various routes as an adjuvant. [14]It causes suppression of neuronal activation at locus coeruleus. This leads to blunting of sympathetic discharge, and thus it stabilizes hemodynamics during laryngoscopy and intubation. [15]In addition to its use as intravenous route.[16] Dexmedetomidine has also been used as nebulization to prevent hemodynamic response to laryngoscopy.[17]

Gulabani M [12] conducted a study to compare Lignocaine 1.5mg/kg with Dexmedetomidine 0.5 ug/kg and 1 ug/kg for attenuation of hemodynamic pressor response to laryngoscopy and intubation. When Systolic BP was compared among three groups, Paired t test showed significant reduction in SBP after intubation in all three groups however maximum reduction was observed in Dexmedetomidine1 ug/kg group, as noted in our study.

When DBP was compared in three groups, paired t test showed that in Lignocaine there was no significant reduction in Diastolic BP at 1 minute after intubation however there was significant reduction in DBP at 2 and 5 minutes after intubation. However both Dexmedetomidine groups showed significant reduction in DBP after intubation with maximum reduction seen in Dexmedetomidine 1 ug/kg group. In our study there was significant reduction in Diastolic BP in both groups (almost equal) after intubation.

When heart rate was compared in three groups, paired t test showed that in lignocaine group there was no significant change in heart rate at 1 minute after intubation, however at 3 and 5 minutes there was significant reduction in heart rate. While in both Dexmedetomidine groups there was significant reduction in heart rate at 1, 3 and 5 minutes after intubation with maximum decrease noted in Dexmedetomidine 1 ug/kg group. This is in contrast to our study where we didn't noticed any significant decrease in heart rate in Lignocaine group, however in Dexmedetomidine group except at 1 min after intubation significant decrease in heart rate was noted.

Boksh SZ compared Lignocaine with Dexmedetomidine for prevention of sympathetic response to laryngoscopy. He observed that changes in Systolic BP , diastolic BP and heart rate was more smooth in Dexmedetomidine group as compared to Lignocaine group however statistically the difference in two groups were not significant. This is in contrast to our study where significant decrease in Systolic BP and Diastolic BP was observed before intubation in Dexmedetomidine group. However when two groups were compared than a statistically significant difference was observed in SBP at 2 and 3 minutes after intubation in Dexmedetomidine group, however Diastolic BP and Heart rate showed no significant difference when ANOVA test was applied.[18]

Rattaphol Seangrung compared Dexmedetomidine with Lignocaine to blunt hemodynamic response to laryngoscopy and found that there was decrease in SBP, DBP and HR after intubation that was more pronounced in Dexmedetomidine group, so findings of this study were similar to our trial.[19]

Chauhan Et al compared Dexmedetomidine, Fentanyl and Lignocaine to prevent pressor response to laryngoscopy. When systolic BP was compared in Dexmedetomidine group there was decrease in Systolic BP after intubation, while in Lignocaine group a rise in Systolic BP was observed after intubation.When Diastolic BP was compared, there was increase in Diastolic BP in both Dexmedetomidine and Lignocaine group , however this increase was more marked in Lignocaine group than in Dexmedetomidine group. Heart rate was increased in both Dexmedetomidine and Lignocaine group, however this increase was more significant in Lignocaine group than in Dexmedetomidine group. The results of this study vary from our trial that may be explained by fact that dose of Dexmedetomidine was 0.6mic/kg that was much less our dose 1mic/kg.[20]

Silpa AR, compared Dexmedetomidine 5ug versus 1ug/kg for prevention of hemodynamic response to laryngoscopy. He found that although there was no difference in sedation score in two groups, there was significant hypertensive response in 5ug/kg group as compared to 1ug/kg Dexmedetomidine group. so results of this study comply with our trial regarding dose of Dexmedetomidine.[21]

CONCLUSION :

Dexmedetomidine is superior to Lignocaine for prevention of pressor response to laryngoscopy.

Authors Contribution:
Ayesha Shahid: Concept & Design of Study, Drafting, Revisiting Critically, Data Analysis, Final Approval of version.
Muhammad Salman Maqbool: Concept & Design of Study, Drafting, Revisiting Critically, Final Approval of version.
Sidra Shabbir: Concept & Design of Study, Revisiting Critically, Final Approval of version.
Fahd Mudassar Hameed: Drafting,
Revisiting Critically, Final Approval of version.
Nargis Shabana: Revisiting Critically, Data Analysis, Final Approval of version
Fareeha Tayyab: Revisiting Critically, Data Analysis, Final Approval of version

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