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Attenuation of Haemodynamic Response to Laryngoscopy and Tracheal Intubation Using Dexmedetomidine, Dexmedetomidine with Fentanyl and Lignocaine.

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ABSTRACT

Background: As no ideal drug has been found to blunt the stress response, our aim was to study the attenuation of haemodynamic stress response to laryngoscopy and tracheal intubation using Dexmedetomidine, Dexmedetomidine with Fentanyl and Lignocaine. **Methods:** 60 patients scheduled for various elective surgical procedures were included in the study. The study population was divided randomly into three groups.Group I –D (Dexmedetomidine group) 20 patients will receive IV Dexmedetomidine 1µm/Kg in 100 ml NS infused over 10-15 minutes before induction.Group II –DF (Dexmedetomidine with fentanyl group) 20 patients received IV Dexmedetomidine 1µgm/Kg combined with Fentanyl 1µgm/Kg in 100 ml NS infused over 10-15 minutes before induction. Group III – L (Lignocaine group) 20 patients received IV bolus lignocaine 1.5mg/Kg 90 seconds before intubation. All the patients were premedicated with injection glycopyrrolate 0.2mg, injection Ondansetron 4mg, injection Tramadol 2mg/kg and injection Midazolam 0.05mg/kg given slowly IV 15 minutes before induction. The HR, SBP, DBP, MAP was recorded at 1, 3, 5 minutes after intubation. In Group L-Lignocaine could not blunt the intubation response until 3 or 5 minutes after intubation. **Results:** In Group D, Dexmedetomidine showed marked decrease in hemodynamic response but the attenuation was highly significant in Group DF (Dexmedetomidine with Fentanyl) throughout the study period. **Conclusion:** IV Dexmedetomidine in the dose of 1µg/kg with Fentanyl 1µg/kg given 15 minutes before induction as infusion effectively attenuated the pressor response during Laryngoscopy and Intubation without any deleterious side effects.

Keywords: Haemodynamic response, laryngoscopy, intubation, Dexmedetomidine, Fentanyl, Lignocaine.

INTRODUCTION

The circulatory response to laryngeal and tracheal stimulation following laryngoscopy and intubation results from the increase in sympathetic and sympathoadrenal activity, as evidenced by increased plasma catecholamine concentration in patients undergoing surgery under general anaesthesia.[1] The increase in pulse rate and blood pressure are usually transitory, variable and unpredictable. Normal healthy persons tolerate this response, but in susceptible individuals this transient sympathetic response can evoke life-threatening conditions. Transitory hypertension and tachycardia may predispose to the development of pulmonary edema, and myocardial insufficiency. Here in days the rationale to continue the quest for an anaesthetic technique where the cardiovascular response can be attenuated. Various agents such as opioids, beta adrenergic blockers, calcium channel antagonists, and clonidine have been used to blunt the hemodynamic response to laryngoscopy and intubation, but they all had limitations.[2-4] Hence the search for an ideal agent to attenuate the hemodynamic response is still continuing. Intravenous (IV) lignocaine is one of the oldest, cheapest and most easily available drug used for attenuation of hemodynamic response to laryngoscopy and intubation. Dexmedetomidine a newly introduced highly selective alpha 2 adrenergic agonist (α2:α1 receptor activity 1620:1).[5] Dexmedetomidine, s-entanomer of meditomidine, possesses hypnotic, sedative, anxiolytic, sympatholytic, and opiod sparing, analgesic properties without producing significant respiratory depression. It has the ability to reduce both the anaesthetic and opioid analgesic requirement during the perioperative period. It's sympatholytic effect decreases mean arterial pressure (MAP) and heart

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rate (HR) by reducing norepinephrine release. These properties makes Dexmedetomidine more favorable drug to be used for attenuating pressor response, as it can be used as an adjunct to anaesthesia. Various studies have also found that Dexmedetomidine can decrease the hemodynamic response to laryngoscopy and intubation.[6-8]

Fentanyl was first synthesized in 1960 and found to be significantly more potent than commonly used opioids such as morphine or meperidine.[9,10] The large safety margin, relatively short duration of action, and minimal respiratory depression at analgesic doses observed for fentanyl soon made it the drug of choice for intravenous anaesthesia.[11] Its ability to provide cardiovascular stability and to block the stress response to surgical stimuli at high doses made it the mainstay of cardiac anaesthesia.[12] Fentanyl, a short acting synthetic opoid agonist with a rapid onset of action i.e 75-125 times potent than morphine and is one of the extensively studied drugs to attenuate the intubation response.[13] The aim of the study is to compare the efficacy of IV Dexmedetomidine, Dexmedetomidine with Fentanyl and Lignocaine as premedication to attenuate the haemodynamic responses to laryngoscopy and endotracheal intubation.

MATERIALS AND METHODS

A study entitled "Attenuation of haemodynamic response to laryngoscopy and tracheal intubation in adult patients by using Dexmedetomidine, Dexmedetomidine with Fentanyl and Lignocaine – a prospective randomized double blind clinical study" was carried out. The study was undertaken after obtaining ethical committee clearance as well as informed consent from all patients.Sixty patients, scheduled for various elective surgical procedures belonging to ASA class I were included in the study. The patients were normotensive with age varying from 18 to 45 years. Materials: Dexmedetomidine 100µg/ml- 2ml ampoule, Fentanyl 50µg/ml- 2 ml ampoule, Lignocaine 2% in 50 ml vial, Normal saline- 100ml

Inclusion criteria

1. Adult patients aged between 18 and 45 years of both the sex
2. Patients belonging to ASA class I
3. Mallampatti grade I and II
4. Elective surgeries under general anaesthesia

Exclusion criteria

1. Patients of ASA grade II and aove.
2. Patients with anticipated difficult airway.
3. Patients on antihypertensive, sedatives, hypnotics and antidepressants.
4. Time taken for laryngoscopy >20 secs
5. H/O Cardiovascular, Respiratory, Hepatic, Renal and endocrinal diseases like hyperthyroidism, hypothyroidism and diabetes mellitus.

The study population was randomly divided into three groups with 20 patients in each group. Group I – D (Dexmedetomidine group) 20 patients will receive IV Dexmedetomidine 1µm/Kg in 100 ml NS infused over 10-15 minutes. Group II – DF (Dexmedetomidine with fentanyl group) 20 patients received IV Dexmedetomidine 1µgm/Kg combined with Fentanyl 1µgm/Kg in 100 ml NS infused over 10-15 minutes. Group III – L(Lignocaine group) 20 patients received IV bolus lignocaine 1.5mg/Kg 90 seconds before intubation. Pre-anaesthetic evaluation was done on the day before surgery. A routine pre-anaesthetic examination was conducted assessing; General condition of the patient ,Airway assessment by Mallampatti grading and rule of 1- 2- 3 ,A detailed examination of the Cardiovascular system, A detailed examination of the Respiratory system. The following investigations were done in all patients. All Patients were explained about the anaesthetic technique and written informed consent was obtained. Patients were kept nil per oral for 8hours prior to surgery. All patients included in the study were premedicated with tab alprazolam 0.5 mg and tab ranitidine 150 mg orally at bed time the previous night before surgery. They were kept nil orally 10 pm onwards on the previous night. On arrival of the patient in the operating room, an 18-gauge intravenous cannula was inserted under local anaesthetic infiltration and an infusion of normal saline was started. The patients were connected to multichannel monitor which records Heart rate, non-invasive measurements of SBP, DBP, MAP, EtCO2 and continuous ECG monitoring and oxygen saturation. The baseline systolic, diastolic blood pressure, mean arterial pressure and heart rate were recorded. The cardiac rate and rhythm were also monitored from a continuous visual display of electrocardiogram from lead II. Parameters like SBP, DBP, MAP, HR, SPO2 recorded 1, 3, 5 minutes after the administering drugs which are included in study. All the patients were premedicated with injection Glycopyrrolate 0.2mg, injection Ondansetron 4mg, injection Tramadol 2mg/kg and injection Midazolam 0.05mg/kg given slowly IV 15 minutes before induction. Then patients were preoxygenated with 100% oxygen for 3 minutes. Anaesthesia was induced with propofol (2mg/kg). Endotracheal intubation was facilitated with 1.5 mg kgIV succinylcholine.. Intubation was achieved with an appropriate size oral, cuffed, portex endotracheal tube by aid of Macintosh laryngoscopy blade. The time taken for intubation should not exceed 20 seconds (intubation that needed more than 20 seconds excluded from study). Anaesthesia was maintained with a loading dose of 0.1 mg/kg

vecuronium bromide followed by 0.08 mg/kg top up doses and IPPV with N2O and O2 in the ratio of 66% and 33% and isoflurane using circle absorber system connected to anaesthesia machine. Isoflurane used in lower possible concentration necessary to keep the BP and HR within 20% of the patient's pre-op baseline values. SBP, DBP, HR, SPO2 recorded at 1, 3, 5 minutes after Laryngoscopy and intubation. At the end of the surgery patients were reversed with Neostigmine 0.05 mg kg-1 IV and Glycopyrrolate 0.01 mg kg-1 IV. Total dose of vecuronium required for the surgery recorded. Recovery assessed and extubation done after thorough throat suction. After adequate clinical recovery patients were shifted to PACU, observed for 2hours for nausea, vomiting, bradycardia, hypotension and sedation. All patients were followed up in the post operative period.

Statistical methods employed-Independent sample t-test(to measure difference between two groups i.e intergroup comparison).Paired sample t-test(to measure difference within the group i.e intra group comparison).Analysis of variance(ANOVA) has been used to find the significance of study paramaters between three or more groups of patient, Post- HOC Tukey test has been used to find the significance pairwise. Chisquare/Fischer exact test has been used to find the significance of study parameters on categorical scale between two or more groups. Contingency table analysis (for association between the rows and columns) $p<0.005$ was considered as significant and $p<0.001$ was considered as highly significant

RESULTS

In this study 60 patients were included, They were randomly assigned into three Groups of 20 patients each. Group I patients received IV Dexmedetomidine 1µg/kg in 100 ml NS infused over 10-15 minutes before induction. Group II patients received IV Dexmedetomidine 1µg/kg with Fentanyl 1µg/kg in 100 ml NS infused over 10-15 minutes 10-15 minutes before induction. Group III patients received IV lignocaine 1.5mg/kg 90 seconds before intubation.

All patients put in two age groups, one group of 18-32 years and other group of 33-45 years. In group D 10 patients was under 18-32 years and 10 patients was under 33-45 years. In group DF 9 patients was under 18-32years and 11 patients was under 33-45 years. In group L 11 patients was under 18-32 years and 9 patients was under 33-45 year.

In group I (D) the basal mean rate was 93.3±12.54. At 1 min post intubation the mean HR was 84±11.61, a fall from basal Heart rate. By 3 and 5 minute there was a significant fall which is statistically significant($p<0.001$).

In group II (DF) the mean preintubation HR was 90.25±6.19. There was decrease in HR in 1, 3, 5 minutes respectively. Attenuation of tachycardia at 1, 3 ,5 was significant with this group DF compared to Group D.

Table 1: Comparison of HR (min) in 3 groups studied Pair wise significance

	D Mean (SD)	DF MEAN (SD)	L MEANS (SD)	P VALUE	D-DF	DF-L	D-L
BASAL	93.3± 12.54	90.25 ±6.19	94.7±10.35	.365	0.335	0.107	0.702
1 MT	84±11 .61	75.1± 5.99	95.6±9 .1	<.001	0.009	<0. 001	0.001
3 MT	79.8± 11.47	71.25 ±4.56	90.15± 9.72	<.001	0.012	<0. 001	0.002
5 MT	79.8± 11	70.65 ±4.92	87.65± 12.52	<.001	0.014	<0. 001	0.043

In group III (L) the mean pre intubation basal HR was 94.7±10.35. At 1 min post intubation the HR was 95.6±9.1, But at 3 and 5 minute there was decrease in HR.

Statistical evaluation between the 3 groups showed that the basal mean HR was statistically not significant($p=0.365$). 1, 3, 5 minutes after intubation HR changes were statistically highly significant ($p<0.001$). Attenuation of tachycardia was better with Group DF compared to other 2 Groups D and L.

Table 2: Comparison of SBP (mmHg) in 3 Groups studied.

	D Mean (SD)	DF MEAN(SD)	L MEANS(SD)	P VALUE	D-DF	DF-L	D-L
BASAL	124.5 ±5.41	124±7. 19	123.75 ±6.10	.92	0.805	0.906	0.683
1 MT	114.35±5.79	107.9± 6.52	121.45 ±6.53	<.001	0.002	<0. 001	0.003
3 MT	110.9 ±5.78	104.45 ±6.15	117.45 ±6.29	<.001	0.001	<0. 001	0.003
5 MT	112.1 ±5.24	102.35 ±6.16	115.75 ±7.06	<.001	<0. 001	<0. 001	0.158

In group D, the mean SBP value deceased by 10.15% at 1 minute post intubation whereas the fall was 13.6% and 12.4% at 3 and 5 minute respectively compared to basal value of 124.5±5.41. Fall in SBP was maximum at 3 minute. In Group DF the mean SBP fall at 1, 3, 5 minutes post intubation was 16%, 19.64% and 21.76% respectively, maximum fall seen in 5 minute compared to basal SBP. In Group L the mean SBP not decreased significantly at 1 minute whereas the attenuation was 6.4% at 3 minute and 8% at 5 minute post intubation. Attenuation of SBP is better with Group DF than compared to the other two groups D and L which is statistically significant.($p<0.001$)

Table 3: Comparison of DBP (mmHg) in 3 groups studied.

	D Mean (SD)	DF MEAN (SD)	L MEANS (SD)	P VALUE	D-DF	DF-L	D-L
BASAL	81.3±6.24	81±8.92	86.35±4.23	.0242	0.902	0.020	0.004
1 MT	71.2±4.99	66.4±7.64	82.7±6.83	<.001	0.240	<0.001	<0.001
3 MT	67.75±5.75	62.5±8.12	78.35±5.35	<.001	0.023	<0.001	<0.001
5 MT	69±6.44	64.35±6.45	76.35±5.92	<.001	0.028	<0.001	0.002

In group D, the basal mean DBP decreased from 81.3±6.24 to 10% at 1minute and 13.54% and 12.4% at 3 and 5 minutes post intubation. In group DF, the attenuation of DBP at 1 minute was 14.6% which is statistically significant (p<0.001). In Group L there is no significant fall of mean DBP at 1 minute from basal mean, while a fall of 8% and 10% is noticed at 3 and 5 minutes respectively. Post intubation response attenuation is significant with the Group DF than Group L.

Table 4: Comparison of MAP (mmHg) in 3 groups studied.

	D Mean (SD)	DF MEAN (SD)	L MEANS (SD)	P VALUE	D-DF	DF-L	D-L
BASAL	94.55±6.01	95.55±11	98.95±4.23	.0372	0.605	0.047	0.109
1 MT	85.55±4.26	79.05±67	95.55±6.38	<.001	0.003	<0.001	<0.001
3 MT	82.35±4.60	76.05±6.52	91.4±5.18	<.001	0.002	<0.001	<0.001
5 MT	83.45±5.14	77.15±5.29	88.6±6.75	<.001	0.003	<0.001	<0.001

In group D at 1 minute post intubation there was a 9% decrease in MAP. The fall in MAP is better at 3 minute (12.1%) and 5 minute (11.1%). In Group DF there is a decrease of 16.6% at 1 minute whereas a decrease of 19.5% and 18.4% decrease at 3 and 5 minutes respectively. In-group L the maximum decrease in basal MAP is seen at 5 minute (10%) post intubation but no significant fall at 1 minute (3.5%) compared to Group D and Group DF. Attenuation in MAP is better with Group D but is more significant with Group DF when compared to Group D and Group L.

DISCUSSION

In the present study Dexmedetomidine was given IV (infusion) 1µg/kg in 100 ml NS infused over 10-15 minutes. Administration of bolus dose of Dexmedetomidine rapidly initially results in transient increase in BP and reflex decrease in HR. The initial reaction is due to peripheral α-2 adrenoceptor stimulation of vascular smooth muscle and can be attenuated by a slow infusion over 15 minutes.14 Kunisawa et al and Ferdi et al have employed Dexmedetomidine in 100 ml NS 15 minutes before induction.Attenuation of heart rate post intubation at 1 minute is significant with Dexmedetomidine and Fentanyl Group.15 Lignocaine does not decrease the HR at 1minute but does so at 3 minutes post intubation. Attenuation of HR is better with Group Dexemedetomidine but superior with Group Dexmedetomidine and Fentanyl. The decrease in HR is because of the inhibition of the central sympathetic outflow overriding the direct stimulant effects and stimulation of presynaptic α-2 adrenoceptors, leading to decrease in norepinephrine release.Kunisawa et al used 1µg/kg of Dexmedetomidine with Fentanyl and found out that though there was decrease in HR, the decrease in BP was supressed. The authors opined that vasoconstrictive effects of Group D through α-2 adrenoceptors which are located in vascular smooth muscle might be responsible for this suppression. They noted that following Laryngoscopy and Intubation , HR at 1min decreased by 7 bpm in Group D and increase by 12 bpm in control group which was statistically significant.16Viswanath et al in the year 2015 conducted study, comparision between Dexmedetomidine and Fentanyl for endotracheal intubation and found out that after induction and intubation HR, SBP, DBP and MAP were significantly lower in Group D than Group F (P<0.004, P=0.00, P<0.04, P<0.006 respectively). The need for thiopentone was decreased by 9% in the dexmedetomidine Group as compared to the fentanyl Group.[17] Post-operative sedation and pain scores were comparably less in Group D than Group F. They concluded, Preoperative infusion of 1µg/kg of both dexmedetomidine and fentanyl are effective in attenuating the sympathetic responses to laryngoscopy and tracheal intubation however, dexmedetomidine blunts this response more effectively than fentanyl. In addition dexmedetomidine has significant anaesthetic sparing effect.In the year 2015 Saya Rahavendra Prasad conducted the study on, comparision of intravenous Lignocaine and intravenous Dexmedetomidine for attenuation of hemodynamic stress response to laryngoscopy and intubation and found out that dexmedetomidine attenuates the hemodynamic stress response to laryngoscopy and intubation more effectively compared with lignocaine without any deletorious effect.[18] Attenuation of mean SBP at 1min is better with Dexmedetomidine and Fentanyl combination than plain Dexmedetomidine. Fall of mean SBP is more significant at 3 and 5 mins post intubation in all the 3 groups but marked in Group Dexmedetomidine and Fentanyl. Lignocaine has a minimal effect decrease on post intubation response at 1min.The mean DBP decrease is more significant at 3mins post intubation. Attenuation of mean DBP at 1 min is superior with Dexmedetomidine and

Fentanyl combination than plain Dexmedetomidine but minimal with Lignocaine group.There was a steady decrease in MAP from pre induction in Group Dexmedetomidine and Fentanyl at 1 min, whereas only plain Dexmedetomidine showed a fall of MAP but lesser than Dexmedetomidine and Fentanyl group. Attenuation of MAP is better in Group Dexmedetomidine but superior in Group Dexmedetomidine and Fentanyl combination, whereas Group Lignocaine showed minimal decrease compared to Group Dexmedetomidine and Dexmedetomidine and Fentanyl. The MAP values did not return to pre induction level even after 5 mins post intubation.There was no evidence of desaturation in all the 3 groups. All patients were followed for 24 hours. Except sinus bradycardia (52 bpm) in 2 patients for which no intervention was required no other complication was observed in patients belonging to any groups.

CONCLUSION

From the present study it can be concluded that Dexmedetomidine in the dose of 1µg/kg infused before 10-15 minutes before induction can blunt stress response to laryngoscopy and intubation markedly.IV Dexmedetomidine in the dose of 1µg/kg with Fentanyl 1µg/kg infused before 10-15 minutes blunt stress response to laryngoscopy and intubation more significantly than plain Dexmedetomidine without any deleterious side effects.Lignocaine could not blunt the intubation response until 3 or 5 minutes after intubation.

REFERENCES

1. Riker RR, Fraser GL: Adverse events associated with sedatives, analgesics, & other drugs that provide patient comfort in the intensive care unit. Pharmacotherapy 2005;25:8s-18s
2. Asai T, Mapleson WW, Power I: Interactive effect of morphine and dexmedetomidine on gastric emptying and gastrointestinal transit in the rat. Br J Anaesth 1998; 80: 63-7
3. Herbert MK, Roth-Goldbrunner S, Holzer P, Roewer N: Clonidine and dexmedetomidine potently inhibit peristalsis in the Guinea pig ileum in vitro. Anesthesiology 2002; 97: 1491-9
4. Guler G, Akin A, Tosun Z, Eskitascoglu E, Mizrak A, Boyaci A: Single-dose dexmedetomidine attenuates airway and circulatory reflexes during extubation. ActaAnaesthesiolScand 2005; 49: 1088-91
5. Guler G, Akin A, Tosun Z, Ors S, Esmaoglu A, Boyaci A: Single-dose dexmedetomidine reduces agitation and provides smooth extubation after pediatricadenotonsillectomy. PaediatrAnaesth 2005; 15: 762
6. Unlugenc H, Gunduz M, Guler T, Yagmur O, Isik G: The effect of pre-anaesthetic administration of intravenous dexmedetomidine on postoperative pain in patients receiving patient-controlled morphine. Eur J Anaesthesiol 2005; 22: 386-91
7. Hofer RE, Sprung J, Sarr MG, Wedel DJ: Anesthesia for a patient with morbid obesity using dexmedetomidine without narcotics. Can J Anaesth 2005; 52: 176-80
8. Yushi U, Maiko S, Hideyuki H. Fentanyl attenuates the haemodynamic response to endotracheal intubation more than the response to laryngoscopy. AnesthAnalg. 2002;95:233–7.
9. Adachi YU, SatomotoM, Higuchi H, Watanabe K. Fentanyl Attenuates the Hemodynamic Response to Endotracheal Intubation More Than the response to laryngoscopy. AnesthAnalg. 2002 Jul; 95(1):233-7
10. HussainAM, Sultan ST .Efficacy of fentanyl and esmolol in the prevention of hemodynamic response to Laryngoscopy and endotracheal intubation. J Coll Physicians Surg Pak 2005; 15:454-7.
11. Yildiz M, Tavlan A, Tuncer S, Reisli R, Yosunkaya A, Otelcioglu S. Effect of dexmedetomidine on haemodynamic responses to laryngoscopy and intubation, perioperative haemodynamics and anaesthetic requirements. Drugs R D 2006;7:43-52
12. Malde AD, SarodeV.Attenuation of the Hemodynamic Response to Endotracheal Intubation: Fentanyl versus Lignocaine .The Int J of Anesth 2007. Volume 12 Number 1
13. Bakiye U, Mustafa O,Erdal G, NuriAO,Feray G. Effects of esmolol, lidocaine and fentanyl on hemodynamic responses to endotracheal Intubation: a comparative study .Clinical Drug Investigation 2007; 27:269-77
14. Basar H, Akpinar S, Doganci N, Buyukkocak U, Kaymak C et al. The effects of preanaesthetic, single-dose dexmedetomidine on induction, hemodynamic, and cardiovascular parameters. Journal of clinical anaesthesia,2008 Sep;20(6):431-436
15. Kunisawa T, Nagata O, Nagashima M. Dexmedetomidine supresses the decrease in blood pressure during anaesthetic induction and blunts the cardiovascular responses to tracheal intubation. J. of Clin. Anaesthesia 2009; 21: 194-9
16. Menda F, Koner O, Sayin M, Ture H, Imer P, Aykac B. Dexmedetomidine as an adjunct to anaesthetic induction to attenuate haemodynamic response to endotracheal intubation in patients undergoing fast track CABG. Ann Card Anasth 2010; 13: 16-21
17. Vishwanath R. Hiremath, N. Gnanasekar. "Dexmedetomidine and Fentanyl for Endotracheal Intubation: A Comparative Study". Journal of Evolution of Medical and Dental Sciences 2015; Vol. 4, Issue 83, October 15; Page: 14458-14468, DOI: 10.14260/jemds/2015/2057
18. Prasad SR, Matam UM, Ojili GP. Comparision of intravenous lignocaine and intravenous dexmedetomidine for attenuation of stress response to laryngoscopy and endotracheal intubation. J NTR Univ Health Sci 2015;4:86-90.

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