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Study Comparing the Effects of Fentanyl and Dexmedetomidine for Attenuation of the Haemodynamic Response During Endotrcheal Extubation in Patients Undergoing Elective Surgeries

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Abstract

Introduction: Tracheal extubation is almost always associated with hemodynamic changes due to reflex sympathetic discharge caused by epipharyngeal and laryngo-pharyngeal stimulation. This increase in sympatho–adrenal activity may result in hypertension, tachycardia and arrhythmias *Aims*: The purpose of the study is to compare the effect of intravenous Fentanyl 1µg/kg with dexmedetomidine 0.7µg/kg on the hemodynamic and recovery responses during extubation. *Materials and methods*: Prospective, randomized, double blind, controlled study was conducted in 60 patients of either sex between 20 and 50 yrs of age belonging to ASA-I and II, undergoing general procedures and urological procedures were selected for the study *Results:* The heart rate increased in both the groups during extubation but the increase was more in Group-F patients. The MAP increased for the initial 1min after drug administration in Group-D. However, dexmedetomidine attenuated the increase in blood pressure to a greater degree than lignocaine. The airway response (coughing) was better attenuated in Group-D than Group-F. The patients in Group-D were drowsy but responding to verbal commands when compared to Group-F.The incidence of bradycardia and hypotension though minimally present in Group-D, which was easily managed. *Conclusion:* Compared to Fentanyl 1µg/kg, dexmedetomidine 0.7µg/kg administered I.V. before extubation attenuates airway and hemodynamic reflexes to a greater extent allowing smooth and easy tracheal extubation, thereby providing comfortable recovery

Keywords: Fentanyl; Dexmedetomidine; Haemodynamic Response; Endotrcheal Extubation

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Introduction

Tracheal extubation is the discontinuation of an artificial airway when indications for its placement like airway obstruction, protection of airway, suctioning, ventilatory failure and hypoxemia no longer exist. Tracheal extubation is almost always associated with hemodynamic changes due to reflex sympathetic discharge caused by epipharyngeal and laryngo-pharyngeal stimulation. This increase

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in sympatho–adrenal activity may result in hypertension, tachycardia and arrhythmias. This increase in B.P and HR are usually transitory, variable and unpredictable. It is more hazardous to the patient with HTN, myocardial insufficiency or cerebrovascular diseases.[1] Significant decreases in ejection fractions (from 55% + 7% to 45% + 7%) after extubation without electrocardiographic signs of myocardial ischemia is demonstrated with coronary artery disease patients. In the clinical practice respiratory complications[4] like coughing, laryngospasm, bronchospasm are three times more common during extubation than during tracheal intubation and induction of anaesthesia (12.6% vs. 4.6%).coughing cause abrupt increases in intracavitary pressures (intraocular, intrathoracic, intraabdominal, intracranial) which could put patient at high risk. Smooth tracheal extubation requires the absence of straining, movement, coughing, breath holding or laryngospasm. Various techinques and anti-hypertensive drugs are available to attenuate airway and circulatory reflexes during extubation but none have been successful.[2] Attempts have been made to attenuate the pressor response by the use of drugs such as narcotic analgesics, deep anaesthesia induced by inhalational anaesthetics, local anaesthetics, adrenoceptor blockers and vasodilator drugs.[3] Studies have been carried out with use of diltiazam, lignocaine, esmololand labetalol, as sole agent or in comparison with each other. To attenuate airway and pressor response during tracheal extubation, Fentanyl, a synthetic opioid, has been reported to reduce the prevalence of coughing during and after extubation and to suppress the sneezing reflex after abdominal hysterectomy and periocular injections. Fentanyl has also been reported to attenuate the cardiovascular responses to tracheal extubation in elective gynecological surgeries. Dexmedetomidine a highly selective alpha2 adrenoceptor agonist has been studied as single dose at the time of extubation. It has a sympatholytic effect through decrease in concentration of norepinephrine[22]. This in turn decreases the blood pressure and heart rate. Dexmedetomidine therefore is theoretically appropriate for reducing airway and circulatory reflexes during extubation.

Materials and Methods

This prospective, randomized, double blind, controlled study was conducted in the Department of Anesthesiology and Critical care at Gandhi Medical Collage, Secundrabad during 2017-2018. After Institutional ethical committee approval, 60 patients of either sex between 20 and 50 yrs of age belonging to ASA-I and II, undergoing general procedures and urological procedures were selected for the study .

Excluding criteria: Patients with ischemic heart disease, with 2nd and 3rd degree heart block, uncontrolled hypertension, uncontrolled diabetes, any medication that affect heart rate or blood pressure, Pregnant and breast feeding women, expected difficult intubation and history of allergy to study drugs.

During the pre-operative visit, all patients were clinically evaluated, assessed and investigated as per the proforma (Proforma-II). The study protocol was explained to the patients and a written informed consent was taken. No patient was given any premedication. 60 cases are randomly divided into two groups 30 in each group. They were named as-

Group-F was Fentanyl group. In this group, 1mcg/kg of Fentanyl was administered 10min before extubation.

Group-D was Dexmedetomidine group. In this group, 0.7mcg/kg of Dexmedetomidine with infusion was administered for 10 min before extubation.

All basic investigations are done and Standard monitoring with electrocardiography (ECG), pulse oximetry (SpO_2), $ETCO_2$ and non- invasive blood pressure was done in the operation theatre. Intravenous line was established using 18 guage intravenous canula. In order to attain double blinding, the person who was not involved in recording the date prepared Fentanyl 1µg/kg. or Dexmedetomidine 0.7 µg/kg Patients were randomly allocated to two equal groups of 30 each by means of a computer generated table of random number to receive either Fentanyl 1µg/ kg (Group F) or Dexmedetomidine 0.7 µg/kg (Group D) over a period of 10 minutes, 10 min prior to extubation.For all the patients anesthesia was induced by using propofol 2mg/kg and fentanyl 2 µg/kg. Atracurium 0.5mg/kg was used to relax muscles for insertion of endotracheal tube. Anesthesia was maintained on O_2 (33%) + N_2O (66%)+ Sevoflurane (1-2%), with muscle relaxant as and when required. Intraoperatively analgesia was maintained with Paracetamol 15mg/kg infuse over 30 minutes. Intraoperatively patients were ventilated to maintain partial pressure of $ETCO_2$ between 30-35 mmHg. About 10 minutes prior to extubation, inhalational agent was stopped and the infusion was started over a period of 10 minutes by the anaesthesia resident (who was unaware of the

contents of the infusion). Residual neuromuscular blockade was reversed with neostigmine 0.05mg/ kg and glycopyrolate 10 µg/kg. Trachea was extubated after the patient resumed spontaneous respiration and obeyed verbal commands.Heart rate, systolic blood pressure, diastolic blood pressure, SpO_2 was recorded at base line / at the start of drug injection, at 1 and 5 minutes after the start of drug infusion, and thereafter at the time of extubation at the 1, 5, 10, 15, 20 minutes after extubation.

Hypotension was defined as a decrease in systolic blood pressure >20% from baseline or a mean arterial pressure of <60 mmHg and was corrected with intravenous fluids and if required, with small dose of mephenteramine 3mg IV. Bradycardia was defined as a heart rate of <60 beats/min and was corrected, if associated with hemodynamic instability, with atropine 0.5 mg IV.

Quality of extubation was evaluated based on cough immediately after extubation using 5 pt rating scale (extubation quality score).[4]

1. = No coughing
2. = Smooth extubation, minimal coughing (1 or 2 times)
3. = Moderate coughing (3-4 times)
4. = Severe coughing and straining (5-10 times)
5. = Poor extubation, very uncomfortable (laryngospasm and cough 10 times).

Post-operative sedation was evaluated on a Ramsay sedation scale (6 point scale)[5] at extubation and thereafter at every 15 minutes for 1 hr.

1. =Anxious or agitated and restless or both.
2. =Cooperative, oriented and tranquil.
3. =Drowsy but responds to commands.
4. =Asleep, brisk response to light glabellar tap or loud auditory stimulus.
5. =Asleep, sluggish response to light glabeller tap or loud auditory stimulus.
6. =Asleep and un arousabe.

Occurrence of any event like laryngospasm, bronchospasm, desaturation, respiratory depression, vomiting was noted.

Statistical Methods

Data was entered in Microsoft excel and analysis was done using SPSS version 20.Descriptive statistical analysis was done. Results on continuous measurements are presented as Mean & Standard Deviation. Results oncategorical measurements are presented as Percentages. Significance is assessed at 5 % level of significance. Student t test (independent, two tailed) has been used to find out the. Significance of study parameters on a continuous scale between two groups .

Chi square test is used to find out the significance of study parameterson a categorical scale between two groups

Results

The minimum age in Group D & F were 18 & 20yrs respectively. The maximum age in both groups was 55yrs. The mean age in Group F & D were 39.6333 + 7.131 & 42.56 + 10.377 respectively. There was no significant difference in the age of patient's between the Group F & D [p = 0.208]

Table 1: Demographic data in present study

Age in intervals	Fentanyl	Dexmed	Total
18-28	3 (10%)	4 (13.3%)	7 (11.7%)
29-38	10 (33%)	7 (23.3%)	17 (28.3%)
39-48	15 (50%)	11 (36.7%)	26 (43.3%)
49-55	2 (6.7%)	8 (26.7%)	10 (16.7%)
Mean Age	39.6 ± 7.13	42.5 ± 10.37	0.208
Gender distribution			
Male	15 (50%)	14 (46.7%)	29 (48.3%)
Female	15 (50%)	16 (51.7%)	31 (51.7%)
Weight distribution in kgs			
50-59	10 (33%)	14 (46.7%)	24 (40%)
60-64	8 (26.7%)	7 (23.3%)	15 (25%)
65-69	9 (30%)	7 (23.3%)	16 (26.7%)
70 and above	3 (10%)	2 (6.7%)	5 (8.3%)
Mean Body weight	61.9 ± 6.104	60.63 ± 5.78	0.413

50% of Group F and 46.7% of Group D were males. Females are 50% in Group F and 53.3% in Group D. The sex distribution did not have any statistically significant difference [p = 0.796]

Body weight distribution of the patients. The mean body weight in Group F & D were 61.9 + 6.104 and 60.633 + 5.78. There was no statistically difference in the body weights between the two groups [p = 0.413] (Table 1).

The basal HR were comparable in both groups and the difference was not statistically significant (p = 0.736). The mean HR was compared at different time intervals and it was observed that, after administration of drug in Group-F, the mean HR at 1min and 5min were 89.466 ± 7.247 and 92.533 ± 11.64 respectively showing continuous raise of HR before extubation, While in Group-D mean HR at 1min and 5min was 83.433 ± 12.57 and 75.166 ± 1.81

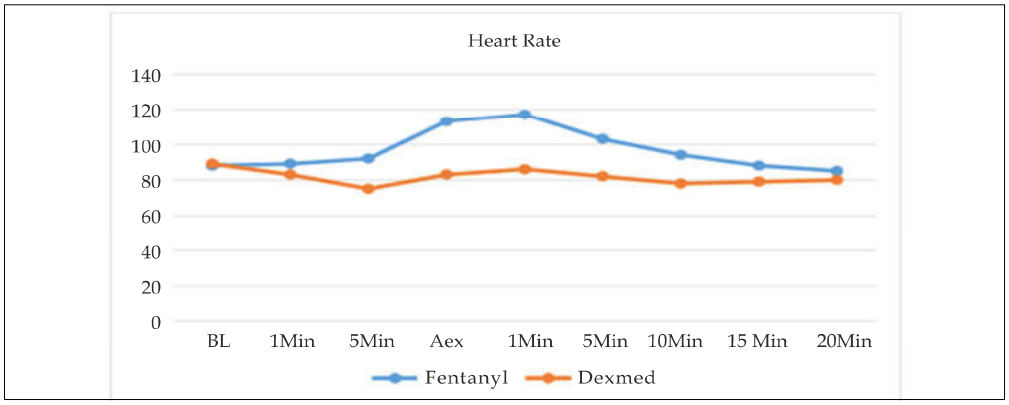

Fig. 1. Comparison of heart rate in two groups of drugs

respectively showing continuous fall in HR. The difference in the HR is statistically significant (p = 0.0001).The mean HR at extubation in Group-F was 113.233 ± 7.71 is significantly more than mean HR in group-D, 83.733 ± 12.23 (p = 0.0001). The peak raise in mean HR was at 1min after extubation in both Group- F and Group-D 117.5 ± 8.029 and 86.7 ± 11.79 respectively and the difference is statistically significant. At 3, 5, 10, 15. min after drug administration the HR in Group-F remained significantly high compared to Group-D (Fig. 1).

In Group-F, the basal mean SBP was 122.266 ± 7.80 and 125.1 ± 9.67 in Group-D, The difference was statistically not significant (p = 0.217). Mean SBP at 1min in Group-F increased minimally, 123.66 ± 7.14 and in Group-D increased minimally 129.33 ± 12.60 and the difference is statistically not significant (p = 0.036). Mean SBP at 5 min in Group-F was 127.46 ± 7.08 which is significantly more than mean SBP 108.83 ± 10.79 in Group-D, the difference is statistically significant (p = 0.001). At extubation mean SBP in Group-F was 147.96 ± 7.83 which was

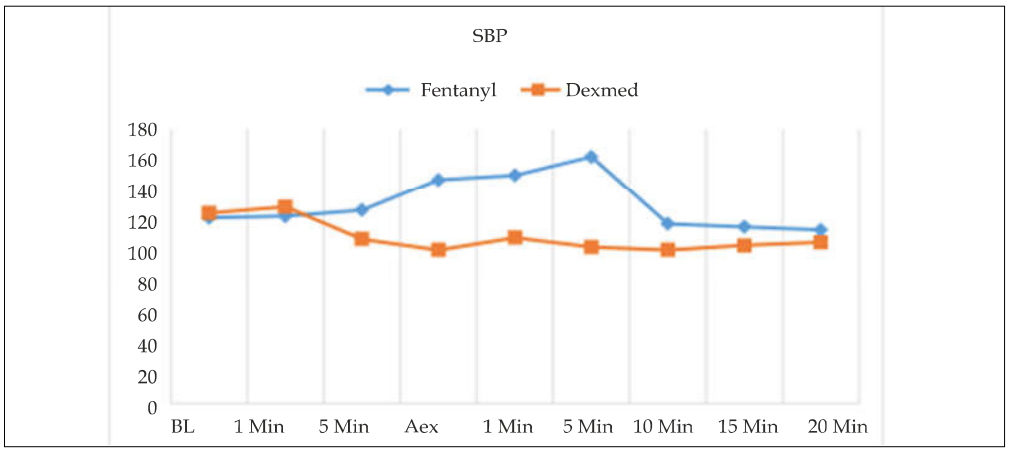

Fig. 2. Shows comparison of SBP between Group-F and Group-D

significantly more than mean SBP 101.7 ± 12.17 in Group-D. Peak raise in SBP occurred at 1min after extubation in both Group-F and Group-D (150.26 ± 7.77) and 109.76 ± 10.87 respectively, but the peak raise was more in Group-F compared to Group-D and the difference is statistically significant (p = 0.001). Mean SBP at 1, 5, 10, 15, 20 min after extubation in Group-F was comparatively more than Group-D and it is statistically significant (p = 0.001) (Fig. 2).

The basal mean DBP are comparable in both the groups and are statistically not significant (p = 0.348). 1min after drug administration the mean DBP in Group-F and Group-D are 79.2 ± 7.097 and 80.33 ± 5.90 respectively and the difference is not statistically significant (p = 0.504). The mean DBP at 5min and at extubation in Group F were significantly high compared to mean DBP in Group-D (p = 0.001). Peak raise in DBP occurred at 1min after extubation in both Group-F (98.9 ± 5.23) and Group D (78.23 ± 8.73) but the raise was more in Group-F compared to Group-D and

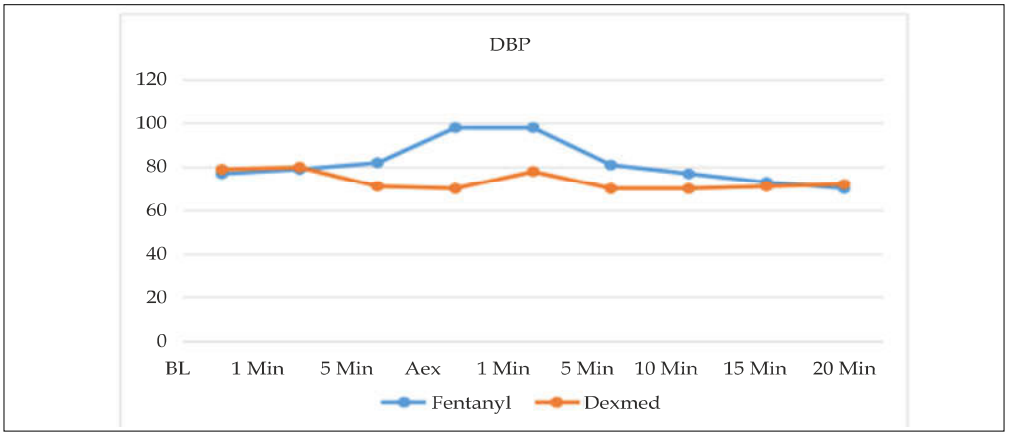

Fig. 3. Shows the comparison of DBP between Both Group-F and Group-D.

the difference is statistically significant. The mean DBP at, 5, 10 min in Group-F were significantly more compared to mean DBP in Group-D and the difference is statistically significant (p = 0.001).A t 15min,The mean DBP in Group-F and Group-D are 73.56 ± 5.43 and 71.6 ± 7.71 respectively and the difference is not statistically significant (p = 0.258). At 20min,The mean DBP in Group-F and Group-D are 70.46 ± 6.12 and 72 ± 6.44 respectively and the difference is not statisticallysignificant (p = 0.349) (Fig. 3).

The basal mean MAP are 91.96 ± 5.76 and 94.36 ± 6.44 respectively and are comparable in both groups (p = 0.134). AT 1min after the drug administration the change in mean MAP was statistically not significant (p = 0.766). At 5min after administration of drug and at extubation the MAP continued to increase in Group-F, while there is a decrease in MAP in Group-D and the difference is statistically significant (p = 0.001). The peak raise in mean MAP occurred at 1min after extubation in both Group-F (115.73 ± 5.11) and Group-D (88.23

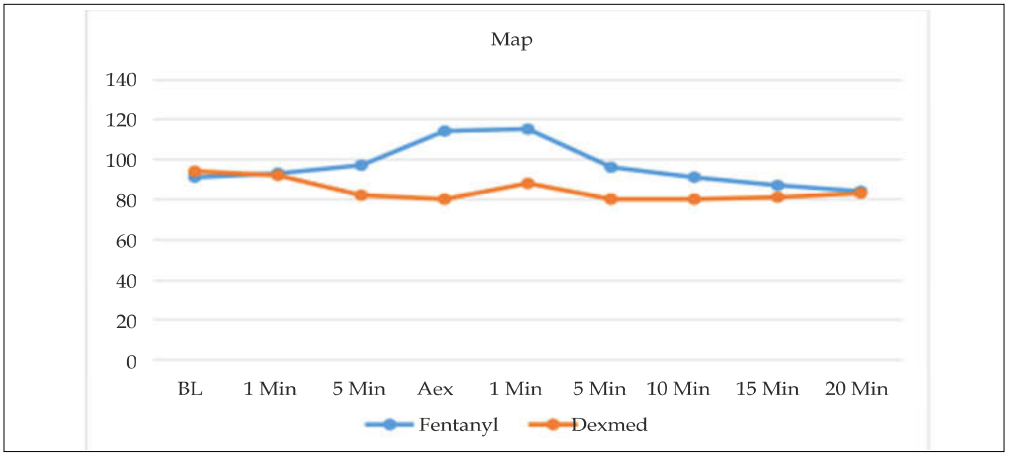

Fig. 4. Comparison of mean arterial pressure in twogroups of drugs

± 7.89) and the difference is statistically significant. The peak raise in MAP was more in Group-F compared to Group-D. The mean MAP at 5, 10, 15, min in Group-F is significantly more compared to Group-D. The mean MAP in Group-F and Group-D are 84.733 ± 4.37 and 83.3 ± 6.31 respectively and the difference is not statistically significant (p = 0.311). shows the mean SpO_2 distribution among two groups. The mean SpO_2 in Group-F was 99.6000 with standard deviation of 0.85 and in Group-D was 99.5000 with standard deviation of 0.90 and the difference is statistically not significant (p = 0.661) (Fig. 4).

There was a significant difference in the quality of extubation between the two groups (p = 0.125). 86.7% of the patients in Group-D could be extubated smoothly, where as 13.3% patients showed minimal coughing at the time of extubation. 66.7% of patients in Group-F could be extubated smoothly, 33.3% patients showed minimal coughing (Table 2).

There was a significant difference in the sedation score between the two groups (p = 0.001). In

Table 2: Distribution of extubation response

		Fentanyl	Dexmed	Total
Extubation response	1	20(66.7%)	26(86.7%)	46(76.7%)
	2	10(33.3%)	4(13.3%)	14(23.3%)
	3	0	0	0
	4	0	0	0
	5	0	0	0
Total		30	30	60

Group-F, 24 patients that is 80% have a score of 1, 6 patients (20%) have a score of 2. In Group- D, 0 patients have a score of 0, 20 patients that is 66.7% have a score of 2and 10 patients (33.3%) have score of 3 (Table 3).

In Group-F, 4 patients had vomiting and 2 had shivering. In Group-D 2 patients developed bradycardia and 3 patients developed hypotension. None had shivering or hypotension (Table 4).

Table 3: Distribution of ramsay sedation score

	Score	Fentanyl	Dexmed	Total
Ramsay sedation score	1	24(80%)	0	24(40%)
	2	6(20%)	20(66.7%)	26(43.3%)
	3	0	10(33.3%)	10(16.7%)
	4	0	0	0
	5	0	0	0
	6	0	0	0
Total		30	30	60

Table 4 : Distribution of side-effects among drug groups

Side Effects	Fentanyl		Dexmed	
	Number of Patients	%	Number of Patients	%
No	24	80	25	83.33
Bradycardia	0	0	2	6.66
Hypotension	0	0	3	10
Vomiting	4	13.33	0	0
Shivering	2	6.66	0	0
Total	30	100	30	100

Discussion

Most of the general anaesthetic procedures in the modern anaesthetic practice are carried out with endotracheal intubation. Laryngoscopy, tracheal intubation and extubation are considered as the most critical events during administration of general anaesthesia as they provoke transient but marked sympatho-adrenal response manifesting as hypertension and tachycardia. The increase in the pulse rate and blood pressure are usually transient, variable and unpredictable. Transient hypertension and tachycardia are probably of no consequence in healthy individuals but either or both may be hazardous to those with hypertension, myocardial insufficiency or cerebrovascular diseases. Pressor response is exaggerated in hypertensive patients even though rendered normotensive preoperatively by antihypertensive medication. Pressor response may result in post-operative myocardial infarction, acute left ventricular failure, intracranial bleed and dysrrythmias in individuals with end organ decompensation. Many methods like use of inhalational anaesthetic agents, lidocaine, opioids,direct acting vasodilators, calcium channel blockers and β-blockers have beentried by various authors for blunting haemodynamic responses to extubation. But all such manoeuvres had their own limitations. For example, use of halothane was associated with dysrrythmias, 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 extubationFentanyl, a synthetic opioid, has been reported to reduce the prevalence of coughing during and after extubation and to suppress the sneezing reflex after abdominal hysterectomy and periocular injections. Fentanyl has also been reported to attenuate the cardiovascular responses to tracheal extubation in elective gynecologic surgery.Alpha2- agonists like dexmedetomidine decrease the sympathetic outflow and noradrenergic activity, thereby counteracting hemodynamic fluctuations occurring at the time of extubation due to increased sympathetic stimulation.This study was undertaken to compare the effect of intravenous Fentanyl 1μg/kg with dexmedetomidine 0.7μg/kg on the hemodynamic and recovery responses during extubation.

The present study was done in 60 patients, planned for various elective surgical procedures under general anaesthesia. Patients were selected after thorough preoperative evaluation. Patients with cardiac, renal, cerebrovascular diseases, 1st, 2nd, 3rd degree heart block, difficult airway and obese patients (BMI > 30) are excluded from the study. Patients were divided into two groups, Group-F and Group-D, 30 in each group. In both the groups there was no statistical difference with respect to their age, weight, sex, ASA grading, preoperative heart rate and blood pressure.The premedication, induction agent, muscle relaxant were standardized for both groups. At the last skin suture inhalational anaesthetic was discontinued,

on the return of spontaneous efforts Group-F patients received 1µg/kg fentanyl diluted to 10ml over 60 sec IV while Group-D received 0.7µg/ kgdexmedetomidine diluted to 10ml over 60 sec IV. HR, SBP, DBP, MAP are measured at 1min and 5min after the study drugs were administered. Neuromuscular block was reversed.Trachea was extubated 5min after study drug was administered and when the patents respirations are sufficient and obeying simple commands. HR, SBP, DBP, MAP, SpO_2, are measured at extubation,1min, 5min, 10min, 15min, 20min after extubation.The HR in Group-D did not show a significant raise compared to basal value from 1min of drug administration, at extubation and any time period post extubation. Though there is a raise in HR at extubation and 1min after extubation, the raise in HR was significantly below the base line HR. This observation is in concurrence with the study done by Rani P et al.[6], where the HR in the dexmedetomidine group remained below the baseline value at all the time intervals following extubation. The raise in the HR that occurred during extubation and 1min after extubation in Group-D is less compared to the raise in HR in Group-F. In Group-F there was a significant raise in HR compared to basal value. The raise HR in Group-F was more persistent than the Group-D. This is accordance with the study done by Rani P et al.[6]Bradycardia was observed in 2 patients at 1min and 2min after giving IV dexmedetomidine in Group-D, but none of the patients required treatment. No patients in Group-F developed bradycardia. These results correlate with the study done by Bindu et al.[7] The study done by Aksu R et al.[8] also found that the incidence of bradycardia was higher in Group-D compared to Group-F which correlates with our study.In our study the SBP increased in the 1st 1min after the dexmedetomidine was given and returned to normal after 5min. This is because the effect of α-2 agonists on the hemodynamics is biphasic, an immediate increase in systemic arterial pressure which is mediated by stimulation of peripheral α-2B receptor followed by a longer lasting reduction in pressure caused by stimulation of α-2 adrenoceptor in central nervous system. SBP decreased minimally after 1min in fentanyl group. Aksu R et al[8] and Rani p et al[6] observed similar increase in SBP after the initial administration of dexmedetomidine.We observed that at extubation SBP was significantly low in Group-D and is 24mmHg less than the basal SBP, While in Group-F SBP at extubation was significantly high and is 25mmHg greater than the basal SBP. Maximum increase in SBP occurred at 1min after extubation in both the groups. In Group-D though there is an increase in SBP at 1min after extubation it was 4mmHg less than the basal value. In Group-F the increase in SBP was 9mmHg greater than the basal value. Dexmedetomidine attenuated the ncrease in SBP to greater degree than fentanyl. The DBP increased at 1min after drug administration in both Group –D and Group-F and the difference is statistically not significant. In Group-D, DBP at extubation was significantly low and is 9mmHg less than the basal value. In Group-F at extubation the DBP was significantly high compared to Group-D. Maximum increase in DBP occurred at 1min after extubation in both groups but it was significantly high in Group-F compared to Group-D. These observations correlates with the observations made by Nishina et al.[9]In our study the MAP increased in the 1st 1min after the dexmedetomidine was given and returned to normal after 2min. This is because the effect of α-2 agonists on the hemodynamics is biphasic. MAP also increased after 1min in fentanyl group but the difference is statistically not significant.Similar observation was made by Rani P et al.[6] wherein they found initial transient raise in MAP in 20% of cases after IV dexmedetomidine. In another study Aksu R et al.[7] also observed similar increase in MAP after the initial administration dexmedetomidine. We observed that at extubation MAP was significantly low (80.13 ± 8.69) in Group-D and is 14mmHg less than the basal MAP, While in Group-F MAP at extubation was significantly high (114.53 ± 5.44) and is 23mmHg greater than the basal MAP. Maximum increase in MAP occurred at 1min after extubation in both the groups. In Group-D compared to the basal MAP the increase in MAP was 6mmHg and in Group-F It was 25mmHg. Dexmedetomidine attenuated the increase in MAP to greater degree than Fentanyl. MAP remained below the basal value till 20min after extubation in Group-D, while in Group-F it reached basal value 10min after extubation. These results correlate with the studies conducted by Turan et al[10] they found that dexmedetomidine 0.5µg/ kg administered 5min before the end of surgery stabilized hemodynamics. Jain et al.[11] carried out a study on the effect of dexmedetomidine on the stress response to extubation and inferred that bolus of drug administered before reversal provided hemodynamic stability that may prove beneficial for cardiac patients. In our study Hypotension was seen in 3 patients in dexmedetomidine group. Hypotension was managed with IV fluids. None of the patients required vasopressors for the correction of hypotension. In Fentanyl group no patients had hypotension. These results correlate

with Guler et al.[12], study. They suggested that single dose of dexmedetomidine 0.5µg/kg given IV over 60 sec before tracheal extubation attenuated airway-circulatory reflexes during extubation .In the same study 1 patient had bradycardia and 3 had hypotension.Mean SpO_2 value in Group-D (99.5) and Group-F (99.6) are comparable. There is no incidence of desaturation in both the groups. This observation is in concurrence with study conducted by Aksu et al[8]. Sedation in our study was assessed using Ramsay sedation scale. Following extubation significant number (67%) of patients in Group-D are co-operative , oriented and tranquil (score of 2), 33% Patients were drowsy but responding to oral commands (score of 3) as against 80% of patients in Group-F are anxious or restless or both (score of 1). This observation is in agreement with the comparative study done between dexmedetomidine and lignocaine by Rani P et al.[6]. Quality of extubation was evaluated based on cough immediately after extubation, using 5 point score. Dexmedetomidine by virtue of its analgesic and sedative properties is known to blunt airway reflexes. In our study 86.7% of patients in the Group-D had smooth extubation (score 1) as against to only 67% patients in Group-F. Incidence of coughing was significantly higher inGroup-F than Group-D (33% VS 13%).This observation is in concurrence with the study done by Aksu R et al[8] where most Patients in dexmedetomidine group could be extubated smoothly with less coughing compared to fentanyl group. The results of Shrirang et al[13] study also correlates with our study. Guler et al[12]. noted the effect of dexmedetomidine on children undergoing adeno-tonsillectomy where in dexmedetomidine group had significantly decreased incidence and severity of agitation and smooth extubation without any increase in incidence of side effects.

In our study , insignificant number of patients in Group-F had vomiting's and shivering with none in Group-D. The absence of shivering among Group-D patients may be due to dexmedetomidine suppressing shivering, possibly by its activity at alpha2 B receptors in the hypothalamic thermoregulatory center of the brain. None of the patients in either groups developed undue sedation or desaturation and respiratory depression. Similar findings are have been made by Bindu et al.[7], Guler et al.[12] and Gosai ND et al.[14] studies also correlate with these findings.

Conclusion

From the data and statistical analysis we conclude that, compared to Fentanyl 1µg/kg, dexmedetomidine 0.7µg/kg administered I.V. before extubation attenuates airway and hemodynamic reflexes to a greater extent allowing smooth and easy tracheal extubation , thereby providing comfortable recovery.

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