ORIGINAL ARTICLE

A Randomized Comparative Study between Dexmedetomidine and Fentanyl on Attenuating Stress Response and Airway Response to Tracheal Extubation

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Abstract:

Background: Tracheal extubation and emergence is associated with major hemodynamic alterations and is poorly tolerated by patients of comorbid disorders. The efficacy of dexmedetomidine and fentanyl was contrasted in mitigating hemodynamic stress response in study groups and evaluated the quality of extubation. *Aim and Objectives:* To study the efficacy of dexmedetomidine and fentanyl on the attenuation of hemodynamic responses and airway reflexes during extubation following surgery under general anaesthesia. *Material and Methods:* A randomized comparative study was conducted in Shri B. M. Patil Medical College Hospital and Research Centre, Vijayapura. Total of 60 patients scheduled for various surgical procedures under general anaesthesia were allotted into two groups. Group D: Intravenous dexmedetomidine 0.4mcg/kg body weight. Group F: Intravenous fentanyl 0.5 mcg/kg body weight. *Results*: Peak rise in mean heart rate was 86.7 % in Group D and 117.5% in Group F, Peak rise in systolic blood pressure was 150.3% in Group F and 109.8% in Group D, peak rise in mean arterial blood pressure was 115.7% in Group F and 88.2% in Group D and diastolic blood pressure rise was 98.9% in Group F and 78.2% in Group D respectively. Dexmedetomidine group had better extubation quality than the fentanyl group. *Conclusion:* Intravenous injection of dexmedetomidine administered 15 minutes before tracheal extubation was better as compared to intravenous injection of fentanyl in attenuating airway and hemodynamic reflexes to a greater extent allowing smooth and easy tracheal extubation, thus providing comfortable recovery.

Keywords: Dexmedetomidine, Fentanyl, Tracheal Extubation, Hemodynamic Response

Introduction:

Tracheal extubation is the discontinuation of an artificial airway at the end of surgical procedures. It is not indicated in conditions like airway obstruction, ventilatory failure and hypoxemia. 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 [1-2]. This increase in Blood Pressure (BP) and Heart Rate (HR) are usually transient, variable and unpredictable. It is more hazardous to the patient with hypertension, myocardial insufficiency or cerebro-vascular diseases [3]. Significant decrease in ejection fractions (from 55%+7% to 45%+7%) after extubation without electrocardiographic signs of myocardial ischemia is demonstrated with coronary artery disease patients [4].

In the clinical practice respiratory complications 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 causes abrupt increase in intracavitary pressures (intra-ocular, intrathoracic, intraabdominal, intracranial) which could put patient at high risk [4]. Smooth

tracheal extubation requires the absence of straining, movement, coughing, breath holding or laryngospasm [5-6]. Various techniques and anti-hypertensive drugs are available to attenuate airway and circulatory reflexes during extubation but none have been successful [7-10].

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[11]. Studies have been carried out with the use of diltiazam [12-13], lignocaine [14], esmolol [15], labetalol [16], and opioids [17] as sole agent or in comparison with each other. Fentanyl, a synthetic opioid, has been reported to reduce the prevalence of cough during and after extubation and to suppress the sneezing reflex after abdominal hysterectomy and periocular injections [18]. Fentanyl has also been reported to attenuate the cardiovascular responses to tracheal extubation in elective gynecologic surgery[8]. Dexmedetomidine a highly selective alpha-2 adrenoceptor agonist has been studied as a single dose at the time of extubation [19]. It has a sympatholytic effect through decrease in concentration of norepinephrine [20]. This in turn decreases the blood pressure and heart rate [21-22]. Dexmedetomidine, therefore, is theoretically appropriate for reducing airway and circulatory reflexes during extubation.

Material and Methods:

The study protocol was reviewed and approved by the Institutional Ethics Committee of the Shri B. M. Patil Medical College, Hospital and Research Centre, Vijayapura. A randomized comparative study was conducted among 60 patients between 14st November 2018 and 14th September 2020. Purposive sampling technique was used to identify the subjects for the study. Inclusion criteria for the participants were male and female subjects those scheduled for various procedures under General Anaesthesia who were willing to participate and in the age group of 18-60 years with ASA status I and II. After obtaining informed and written consent, preanesthetic evaluation was done. Patients were randomized into two groups Group D (Dexmedetomidine) and Group F (Fentanyl).

Sample Size Calculation:

Sixty patients are required (thirty per group) to have a 95% chance of detecting, as significant at the 1% level, an increase in the primary outcome measure from 38% in the fentanyl group to 85% in the dexmedetomidine group.

Calculation was based on the formula:

$$\mathbf{n = f(\alpha/2, \beta) \times [p_1 \times (100 - p_1) + p_2 \times (100 - p_2)] / (p_2 - p_1)^2}$$

where p_1 and p_2 are the percent 'success' in the fentanyl and dexmedetomidine groups respectively.

α (Type 1 error) = 1% level of significance.

β (Type 2 error) = 5 % level of significance (95% power)

f = Distribution function of standard variate at type 1 error and type 2 error.

A total of 60 patients were enrolled in the study who were willing to participate by giving a written informed consent. Inclusion criteria were the patients who were belonging to American Society of Anaesthesiologists (ASA) classification for Physical Status I and II of either sex in the age range of 18 to 60 years undergoing surgery under general anaesthesia. They were randomized into two groups of 30 each.

The ethical clearance for the study was obtained from the Institutional Ethics Committee, Shri B. M. Patil Medical College Hospital and Research Centre, Vijayapura.

A preanesthetic evaluation with detailed medical history and systemic examination was done and relevant investigations were advised and reviewed on the previous day and on the day of surgery. Patients were randomized into two groups:

Group D (Dexmedetomidine):

Intravenous dexmedetomidine 0.4 mcg/kg body weight diluted to 20 ml in normal saline was infused over 15 minutes prior to completion of surgery using infusion pump.

Group F (Fentanyl):

Intravenous fentanyl 0.5 mcg/kg body weight diluted to 20 ml in normal saline infused over 15 minutes prior to completion of surgery using infusion pump.

Standard monitoring with Electrocardiography (ECG), pulseoximetry (SpO_2), End-tidal CO_2 ($ETCO_2$) and noninvasive blood pressure was done in the operation theatre. Intravenous line was established using 18 gauge intravenous cannula. After preoxygenation, patients were preinduced with injection midazolam 0.025 mg/kg and fentanyl 1-2 mcg/kg. They were induced with injection. propofol 2 mg/kg intravenously and intubation facilitated with injection atracurium 0.8 mg/kg intravenously. Patients were maintained on 60% nitrous oxide in oxygen and isoflurane percentage was adjusted to maintain hemodynamics within the normal range.

Atracurium initial bolus and intermittent thereafter was used for maintenance of muscle paralysis. Intraoperative patients were ventilated to maintain partial pressure of $ETCO_2$ between 30-35 mmHg. About 15 minutes prior to extubation, inhalational agent was stopped and the infusion was started over a period of 15 minutes by the anaesthesia resident (who was unaware of the contents of the infusion). After onset of spontaneous breathing, intravenous neostigmine 0.05 mg/kg and glycopyrrolate 0.01 mg/kg was administered to antagonize the effect of muscle relaxants. Patients were extubated when the extubation criteria were fulfilled. HR, systolic BP, diastolic BP and mean arterial pressure were recorded just before reversal, during extubation, 1 min of tracheal extubation and thereafter 5 min, 10 min and 15 min of tracheal extubation.

Adverse events like bradycardia, hypotension, vomiting, laryngospasm, respiratory depression and delayed arousal were noted and treated accordingly.

Results:

Demographic Analysis:

The mean age of the study population in Group D was 42.6 ±10.4 years and in Group F was 39.6 ± 7.1 years which was comparable with P value of 0.207. The gender distribution was also comparable among the Groups with P value of 0.796.

Hemodynamic Parameters:

Table 1 showed comparison of mean HR between Group D and Group F. The mean HR in Group D at 1 min and 5 min indicated a steady rise in HR before extubation, whereas mean HR at 1 min and 5 min indicated steady fall of HR before extubation in Group F. The difference in the HR was statistically significant (P=0.001). The mean HR at extubation in Group F was 113.2 which was significantly more than mean HR in Group D, 83.7 (P=0.001). The peak raise in mean HR was at 1 min

after extubation in both Group D (86.7) and Group F (117.5) respectively and the difference was statistically significant. At 5, 10, 15 min after drug administration the HR in Group F remained significantly high as compared to Group D.

Table 2 showed comparison of SBP between Group D and Group F. After extubation mean SBP in Group F was 148 which was significantly more than mean SBP 101.7 in Group D. Peak rise in SBP at 1 min after extubation was more in Group F (150.3) as compared to Group D (109.8) and the difference was statistically significant (p= 0.001). Mean SBP at 1, 5, 10, 15 min after extubation in Group F was comparatively more than Group D and it was statistically significant (p=0.001).

Table 1: Showing the Comparison of Heart Rate among the Study Groups

HR	Group D	Group F	Mean Difference	*P*
	Mean ± SD	Mean ± SD		
JBR	75.2 ± 10.8	92.5 ± 7.2	-17.4	<0.001*
AEX	83.7 ± 12.2	113.2 ± 7.7	-29.5	<0.001*
1min	86.7 ± 11.8	117.5 ± 8.0	-30.8	<0.001*
5min	82.0 ± 10.3	103.9 ± 7.3	-21.8	<0.001*
10min	78.8 ± 9.0	94.6 ± 6.4	-15.8	<0.001*
15min	79.3 ± 8.4	88.9 ± 5.9	-9.5	<0.001*

JBR- just before reversal, AEX- at extubation

Table 2: Showing the Comparison of Systolic Blood Pressure among the Study Groups

SBP	Group D	Group F	Mean Difference	*P*
	Mean ± SD	Mean ± SD		
JBR	108.8 ± 10.8	127.5 ± 7.1	-18.6	<0.001*
AEX	101.7 ± 12.2	148.0 ± 7.8	-46.3	<0.001*
1min	109.8 ± 10.9	150.3 ± 7.8	-40.5	<0.001*
5min	103.4 ± 11.8	125.6 ± 4.0	-22.2	<0.001*
10min	101.9 ± 11.8	118.5 ± 5.2	-16.6	<0.001*
15min	104.2 ± 9.6	116.0 ± 4.8	-11.8	<0.001*

JBR- just before reversal, AEX- at extubation

Table 3 showed the comparison of DBP between both Group F and Group D. The mean DBP at extubation in Group F was significantly high as compared to mean DBP in Group D. Peak rise in DBP occurred at 1 min after extubation was more in Group F (98.9) as compared to Group D (78.2) and the difference was statistically significant. The mean DBP at 5, 10, 15 min in Group F was significantly more as compared to mean DBP in Group D and the difference was statistically significant (p=0.001).

Table 3: Showing the Comparison of Diastolic Blood Pressure among the Study Groups

DBP	Group D	Group F	Mean Difference	*P*
	Mean ± SD	Mean ± SD		
JBR	71.7 ± 10.6	82.8 ± 7.0	-11.1	<0.001*
AEX	70.0 ± 11.9	98.3 ± 7.0	-28.3	<0.001*
1min	78.2 ± 8.7	98.9 ± 5.2	-20.7	<0.001*
5min	70.3 ± 9.0	81.5 ± 4.7	-11.2	<0.001*
10min	70.6 ± 8.1	77.6 ± 4.9	-7.1	<0.001*
15min	71.6 ± 7.7	78.6 ± 5.4	-7.0	<0.001*

JBR- just before reversal, AEX- at extubation

Table 4: Showing the Comparison of Mean Arterial Blood Pressure among the Study Groups

MAP	Group D	Group F	Mean Difference	*P*
	Mean ± SD	Mean ± SD		
JBR	82.3 ± 8.0	97.4 ± 5.2	-15.1	<0.001*
AEX	80.1 ± 8.7	114.5 ± 5.4	-34.4	<0.001*
1min	88.2 ± 7.9	115.7 ± 5.1	-27.5	<0.001*
5min	80.4 ± 7.6	96.5 ± 3.9	-16.1	<0.001*
10min	80.8 ± 8.4	91.0 ± 3.9	-10.3	<0.001*
15min	81.7 ± 6.7	87.5 ± 4.2	-5.9	<0.001*

JBR- just before reversal, AEX- at extubation

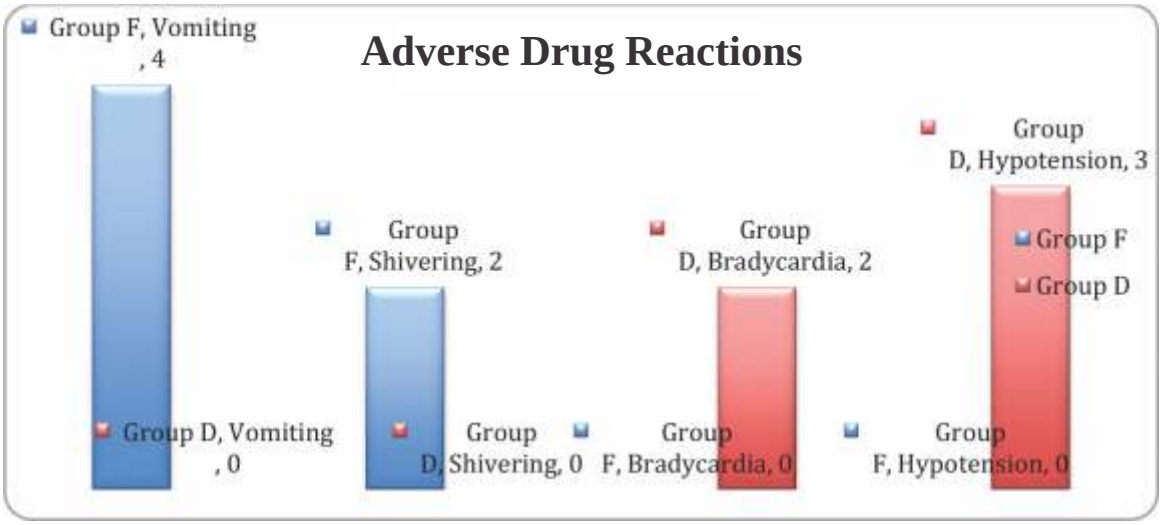

Fig. 1: Showing the Adverse Drug Reactions

Table 4 showed comparison of MAP between Group F and Group D. After extubation the MAP continued to increase in Group F, while there was a decrease in MAP in Group D and the difference was statistically significant (p=0.001). The peak rise in mean MAP occurred at 1 min after extubation was more in Group F (115.7) as compared to Group D (88.2). The mean MAP at 5, 10, 15 min in Group F was significantly more as compared to Group D.

Adverse Drug Reactions:

In Group F, four patients had vomiting and two had shivering. In Group D, two patients developed bradycardia and three patients developed hypotension (Fig.1).

Discussion:

The present study was undertaken to compare the effect of intravenous fentanyl 0.5 µg/kg with dexmedetomidine 0.4 µg/kg on attenuation of hemodynamic responses and airway reflexes during extubation. HR in Group D did not show a significant rise at extubation as compared to 1 min of drug administration, and any time in post extubation period. Though there was a rise in HR at extubation and 1 min after extubation, the rise in HR was significantly below the baseline HR. This observation was in concurrence with the study done by Rani *et al.* [24], where the HR in the dexmedetomidine group remained below the baseline value at all the time intervals following extubation. The rise in the HR that occurred during extubation and 1 min after extubation in Group D was less as compared to the raise in HR in Group F. In Group F there was a significant rise in HR as compared to baseline value. The rise in HR in Group F was more persistent than Group D.

Bradycardia was observed in two patients at 1 min and 2 min 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.* [25]. The study done by Aksu *et al.* [26] also found that the incidence of bradycardia was higher in Group D as compared to Group F which correlates with this study. In this

study the SBP increased in the first 1 min after the dexmedetomidine was given and returned to normal after 2 min. 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 1 min in fentanyl group. Aksu *et al*. [26] and Rani *et al.* [24] observed similar increase in SBP after the initial administration of dexmedetomidine. SBP was found to be significantly low in Group D at extubation and was 24 mmHg less than SBP estimated just before reversal. Although SBP at extubation was significantly high in Group F and was 25 mmHg higher than the SBP measured just before reversal. In both the groups, maximum increase in SBP occurred at 1 min after extubation. In Group D, after extubation, the SBP rise was 14 mmHg smaller than just before the reversal value at 1min. The rise in SBP in Group F was 29 mmHg higher than the value just before the reversal. The rise in SBP was attenuated by dexmedetomidine to a greater extent than that of fentanyl.

DBP was slightly low at extubation in Group D and is 9 mmHg lower than just before the reversal. Compared to Group D, DBP was extremely high in Group F at extubation. Maximum rise in DBP occurred in both groups at 1 min after extubation, but compared to Group D, it was significantly high in Group F. These observations correlate with the observations made by Nishina *et al*. [8].

In this study, MAP increased in the first 1 min after administration of dexmedetomidine and returned to normal after 2 min. This is because the effect on hemodynamics of the α-2 agonists is biphasic. After 1 min in the fentanyl group, MAP was increased, but the difference was not statistically significant. Similar findings were made by Rani *et al.* [24]. They found initial transient rise in MAP following IV dexmedetomidine in 20% of cases. A similar rise in MAP after initial administration of dexmedetomidine was also observed in study by Aksu *et al*. [26].

It was observed that at extubation MAP was significantly low (80.1) in Group D and was 14mmHg less than the baseline MAP. While in Group F, MAP at extubation was significantly high (114.5) and was 23 mmHg greater than MAP measured just before reversal. Maximum increase in MAP occurred at 1 min after extubation in both the groups. In Group D compared to the MAP measured just before reversal, the increase in MAP was 6 mmHg and in Group F, it was 25 mmHg. Dexmedetomidine attenuated the increase in MAP to greater degree than fentanyl. MAP remained below the just before reversal value till 15 min after extubation in Group D, while in Group F it reached just before reversal value 10 min after extubation. These results correlate with the studies conducted by Tao *et al.* [18] they found that dexmedetomidine 0.5µg/kg administered 5 min before the end of surgery stabilized hemodynamics. Jain *et al.* [27] 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.

Pathak *et al.* [28] compared 1mcg/kg (Group A) and 0.7 mcg/kg (Group B) dexmedetomidine in preinduction room 15min prior to surgery. They found that the heart rate was reduced to statistically

significant level at preinduction period in Group A and remained at lower levels throughout the operation. Compared to baseline values, reduction in the MABP was statistically insignificant in both the groups; maximum reduction was observed for 10 min in postintubation period. These findings were in favour of our study in view of reduction in heart rate and reduction in mean arterial blood pressure. In this study, hypotension was seen in three 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 correlated with the study of Guler *et al*. [19]. 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 one patient had bradycardia and three patients had hypotension.

Tendulkar *et al.* [29] compared three drugs for attenuation of stress response and airway relaxes. IV Esmolol 1.5 mg/kg (Group E) two minutes prior to extubation or IV Dexmedetomidine 0.5 mcg/kg (Group D) over ten minutes prior to extubation or no drug in the control group (Group C). Hemodynamic parameters were assessed before giving study drugs, before extubation and after extubation up to 15 minutes. This study is comparable to our present study in view of attenuation of stress response and smooth extubation quality.

Conclusion:

It was concluded that, as compared to fentanyl 0.5 µg/kg, dexmedetomidine 0.4 µg/kg administered intravenously before extubation attenuates hemodynamic stress response and airway reflexes to a greater extent allowing smooth and easy tracheal extubation, thereby providing comfortable recovery.

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