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ATTENUATION OF HEMODYNAMIC STRESS RESPONSE DURING EMERGENCE FROM GENERAL ANAESTHESIA: A PROSPECTIVE RANDOMIZED CONTROLLED STUDY COMPARING FENTANYL AND DEXMEDETOMIDINE

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ABSTRACT: BACKGROUND: Tracheal extubation and emergence is associated with significant haemodynamic alterations and is poorly tolerated by patients with co-morbid conditions. We compared the efficacy of fentanyl and dexmedetomidine in mitigating haemodynamic stress response and assessed extubation quality in study groups. **MATERIALS AND METHODS:** One fifty patients of either sex, ASA grade I & II normotensive patients, aged 18-55 years undergoing elective surgeries under general anaesthesia were randomized into 3 equal groups. Anaesthetic technique was standardized. 10 minutes prior to extubation, patients in Group N, F and D received intravenous bolus infusion of 0.9% normal Saline, Fentanyl 1µg /kg and Dexmedetomidine 1µg /kg respectively over 10 minutes period. Heart Rate (HR), Systolic BP (SBP), Diastolic BP (DBP) and Mean Arterial Pressure (MAP) were noted at extubation, 2, 4, 6, 8, 10 min and at regular interval thereafter for a period of two hours. Extubation quality was evaluated on 5-point extubation quality scale [scale 1 = no coughing, 2 = smooth extubation, minimal coughing (1 or 2 times), 3 = moderate coughing (3 or 4 times), 4 = severe coughing (5-10 times) and straining, 5 = poor extubation, very uncomfortable (laryngospasm and coughing >10 times)]. Ramsay sedation score and Aldrete's recovery score were recorded. Any adverse events, use of rescue drugs and postoperative analgesics were noted. **RESULTS:** All the measured haemodynamic parameters were significantly elevated at extubation and at various points of observation in normal saline group than fentanyl and dexmedetomidine group (p=0.000). Tachycardia response was seen in 84% patients in group N, compared to 36% and 8% in group F & D respectively (p=0.000). Statistically significant hypertensive response was noticed in 43(86%) patients of group N, 9(18%) of group F and 3(6%) of group D (p=0.000). Duration of tachycardia and hypertensive response was significantly longer in control group. Three groups differed with regard to overall extubation quality (p<0.001). Groups D (1.50±0.58) and F (1.94±0.55) had lower scores compared to group N (2.68±0.47) implying smoother extubation. Use of rescue drugs to treat acute hypertensive response was more in group N (34%) than group F (2%) and group D (0%). Sedation and recovery scores were similar in all the three groups. **CONCLUSION:** Dexmedetomidine 1 µg/kg IV was most effective followed by fentanyl 1 µg/kg IV in attenuating haemodynamic stress responses during emergence with no clinically significant differences in sedation and recovery profile. Dexmedetomidine group had smoother and best extubation quality. **KEYWORDS:** Dexmedetomidine, fentanyl, emergence, extubation quality.

INTRODUCTION: Securing airway with endotracheal intubation forms the mainstay of standard general anesthetic practice. Similar to intubation, extubation is also associated with various

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circulatory and airway responses due to reflex sympathetic activity following stimulation of epipharyngeal and laryngo-pharyngeal structures leading to coughing, agitation, bronchospasm, tachycardia, hypertension, arrhythmias, myocardial ischemia and raised intracranial and intraocular pressures.¹

These transient but significant changes, which may be well tolerated by healthy individuals, may prove to be deleterious in patients with hypertension, coronary artery disease or intracranial pathologies.

Respiratory complications resulting in serious consequences (hypoxic brain injury and death) following tracheal extubation have been thrice more common than those occurring during intubation (4.6% versus 12.6%).^{2,3,4,5,6,7,8,9}

Clinical research is still on to find an ideal drug with good safety margin, which attenuates most of the hemodynamic alterations in response to airway manipulation at extubation without delaying recovery and causing adverse events (sedation, respiratory depression, hypotension etc).

Various pharmacological agents like fentanyl, clonidine, esmolol and lignocaine have been used to attenuate extubation response.^{10,11,12,13}

Fentanyl, an opioid agonist, may blunt cardiovascular and airway reflexes during emergence without prolonging the recovery.¹⁰ Dexmedetomidine an alpha-2 agonist, which has sedative, analgesic, sympatholytic and anxiolytic effects, mitigates most of the cardiovascular responses in the perioperative period.^{14,15,16}

We conducted a prospective randomized controlled clinical trial with primary aim to assess the efficacy of fentanyl and dexmedetomidine in attenuating emergence stress response and compared extubation quality in study groups under standard anaesthetic condition using entropy and neuromuscular monitors. We also assessed sedation score, recovery score and other complications amongst the three groups in post anesthesia care unit over a period of 2 hours.

MATERIALS AND METHODS: STUDY DESIGN AND PARTICIPATION: One hundred and fifty patients (both male and female) of American Society of Anesthesiologists grades 1 or 2, aged 18–55 years, coming for elective surgeries lasting for 90-240 minutes, under general anesthesia requiring endotracheal intubation were enrolled for the study. Written informed consent was taken from patients. Data was collected over a period of 6 months (Nov 2012–May 2013) at a corporate tertiary care center with postgraduate training facility. The study was approved by the institutional review board (Hospital Ethics Committee for Human Research), which supervised the data collection and safety issues.

Exclusion criteria were patients with cardiorespiratory abnormalities (Hypertension, New York Heart Association heart failure grades 3 and 4, bronchial asthma, chronic obstructive pulmonary disease, and restrictive lung disease), renal insufficiency (serum creatinine more than 1.6 mg/dl) and liver dysfunction (liver enzymes-serum glutamic oxaloacetic transaminase/ serum glutamic pyruvic transaminase values elevated by more than 50% of normal). Patients with difficult airway, severe obesity (body mass index greater than 35), and undergoing surgeries on neck and oral cavity and emergency procedures were excluded from the study.

SAMPLE SIZE ESTIMATION: Sample size was calculated on the basis of an earlier research which studied the effect of dexmedetomidine on extubation response conducted by D Jain and coworkers,¹⁷

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who observed an increase in SBP of 32.3 ± 11 mmHg in the control group during extubation. We hypothesized that 20% reduction in extubation stress response in the study drug group would be clinically significant. Group size was determined by using the sample size estimation “for two group mean method” with 90 % power and 1 % significance, based on which we needed to study 43 patients in each group. Adding 10% compensation for ‘loss to follow up’ and for inclusion of a third group into the study, we recruited 50 patients in each group.

RANDOMIZATION: Based on a computer generated random number table using Microsoft Excel (created using Microsoft Excel 2003 software, Redmond, WA), all the patients were randomized into 3 groups, with 50 patients in each group. Group N, F and D received intravenous bolus infusion (over a period of 10 minutes) of 0.9% Normal Saline, $1 \mu\text{g} / \text{kg}$ Fentanyl and $1 \mu\text{g} / \text{kg}$ Dexmedetomidine respectively, 10 minutes prior to extubation.

PREOPERATIVE MANAGEMENT: Patients were assessed and evaluated as per the routine preoperative protocol. All the patients were kept nil by mouth 6 hours before the surgery.

INTRAOPERATIVE PERIOD: In addition to standard monitors (Electrocardiography, non-invasive blood pressure, pulse oximetry and temperature probe), neuromuscular monitor TOF-watch (organon Teknika Corporation) and entropy monitors (GE health care) were used during the intraoperative period. Pre-induction heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MAP) and oxygen saturation were noted.

After pre-oxygenation for 3 minutes, all the patients were induced with intravenous propofol 2 mg/kg, fentanyl $2 \mu\text{g} / \text{kg}$ and lignocaine 1.5mg/kg. Muscle relaxation was achieved with atracurium 0.5mg/kg. Intravenous paracetamol 1 gm infusion was given to all the patients. After achieving TOF count of zero, a gentle laryngoscopy and endotracheal intubation was performed with appropriate size endotracheal tube. Anaesthesia was maintained with isoflurane (0.8-1.5%) in oxygen and air (50:50). Muscle relaxation was maintained with atracurium 0.3mg/kg/hr infusion. Intra-operative analgesia was maintained by using fentanyl 0.5-1 $\mu\text{g} / \text{kg} / \text{hr}$ IV infusion.

Entropy was maintained between 40-60 by titrating isoflurane between 0.8-1.5 percent. Vital parameters were maintained within 20% of baseline values. Atracurium and fentanyl infusion were stopped 20-30 minutes before expected time of extubation. Isoflurane was discontinued at skin closure and HR, SBP, DBP and MAP were recorded as ‘Baseline’ value.

Groups N, F and D received intravenous bolus infusion of 0.9% Normal Saline, $1 \mu\text{g} / \text{kg}$ of Fentanyl, and $1 \mu\text{g} / \text{kg}$ of Dexmedetomidine respectively, infused over a period of 10 minutes starting 10 minutes prior to extubation. After onset of spontaneous breathing, intravenous neostigmine 0.04mg/kg and glycopyrrolate 0.01mg/kg was administered to antagonize the effect of muscle relaxants. Patients were extubated when the extubation criteria were fulfilled.¹⁸ HR, SBP, DBP and MAP were recorded at reversal, at extubation, every 2 min for 10 mins, every 5 mins for first 30 mins, and every 30 min for next 1 hour 30 minutes after extubation.

Extubation quality was evaluated on 5-point extubation quality scale^{10,19} [scale 1 = no coughing, 2 = smooth extubation, minimal coughing (1 or 2 times), 3 = moderate coughing (3 or 4 times), 4 = severe coughing (5-10 times) and straining, 5 = poor extubation, very uncomfortable (laryngospasm and coughing >10 times)]. Ramsay sedation score.^{20,21} Aldrete’s recovery score,^{22,23,24}

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were recorded every 15 min after extubation for 2 hours. Adverse events like hypertension, tachycardia, hypotension, bradycardia (< 30 % of base line value), desaturation, bronchospasm were noted and treated accordingly (Intravenous Esmolol 30 mg for hypertension). Postoperative pain was treated with combination of NSAIDS and opioids.

OPERATIONAL DEFINITIONS: Baseline value: Haemodynamic variables such as HR, SBP, DBP, and MAP measured prior to administration of study drug and after cessation of Isoflurane administration, was defined as baseline value.

- Peri-extubation period in our study was defined as period from administration of reversal agent to first 10 minutes after extubation.
- Tachycardia response in our study was defined as increase in heart rate \geq 30 beats per minute from baseline value.
- Hypertensive response was defined as increase in systolic blood pressure \geq 30 mmHg from baseline value.
- Bradycardia response was defined as decrease in heart rate \leq 30 beats per minute from baseline value.
- Hypotensive response was defined as decrease in systolic blood pressure \leq 30 mmHg from baseline value.

STATISTICAL ANALYSIS: Continuous data were expressed as mean SD. Categorical data were expressed as number (%). Analysis of variance (ANOVA) was used to compare study parameters among the three groups. Post-hoc Tukey test was used to find pair wise significance. Chi-square/ Fisher Exact test was used to compare categorical data among the three groups. $p < 0.05$ was considered statistically significant. Multiple paired t-test was used for within group data analysis and $p < 0.003$ was considered as significant after application of Bonferroni's correction.

RESULTS: We recruited total 150 patients, randomly divided into 3 groups of 50 each, receiving normal saline (group N), fentanyl (group F) and dexmedetomidine (group D) 10 minutes prior to extubation. One patient was excluded from analysis in group D because of incomplete collection of hemodynamic data. The three groups were demographically well matched with respect to age, sex, weight, ASA grade and duration of surgery. (Table 1)

Demography		Group N (n=50)	Group F (n=50)	Group D (n=49)	p-Value
Age (yrs)		35.46 \pm 10.07	35.80 \pm 10.06	35.86 \pm 9.73	0.977
Sex	Male: Female (%)	17:33 (34:66)	20:30 (40:30)	22:28 (44:56)	0.588
Weight (kg)		63.36 \pm 11.41	63.88 \pm 11.23	63.68 \pm 12.09	0.975
ASA-Grade	1:2(%)	43:7 (86:14)	48:2 (96:4)	41:9 (82:18)	>0.05
Duration of surgery (hrs)		2.13 \pm 0.63	2.15 \pm 0.63	2.29 \pm 0.62	0.414

Table 1: Demographics

HAEMODYNAMIC PARAMETERS (FIG-1, TABLE -2): Baseline value: Haemodynamic variables such as HR, SBP, DBP, and MAP measured prior to administration of study drug, after cessation of isoflurane. The 3 groups were well matched with respect to baseline HR, SBP, DBP and MAP.

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HEAT RATE: Maximum heart rate observed in Group N was 108 ± 10 bpm, which was significantly higher compared to other two groups ($p=0.000$). The average increase in HR above baseline noticed in group N (34 ± 11 bpm) was significantly more ($p=0.000$). Tachycardia response was seen in 84% patients in group N, compared to 36% and 8% in group F & D respectively ($p=0.000$). Duration of tachycardia was significantly longer (18 ± 20 min) in group N compared to group F (5 ± 4 min) and group D (3 ± 2 min) ($p=0.000$).

Maximum values of SBP, DBP and MAP attained in groups N, F and D was statistically significant ($p=0.000$). Magnitude of increase in SBP, DBP and MAP above baseline was highest in Group N which was statistically significant ($p=0.000$).

SYSTOLIC BLOOD PRESSURE: Maximum SBP observed in group N, F and D were 159 ± 11 , 136 ± 13 and 130 ± 9 mmHg which was statistically significant ($p=0.000$). Statistically significant Hypertensive response was noticed in 43(86%) patients of group N, 9(18%) of group F and 3(6%) of group D ($p=0.000$). Average duration of hypertension also remained significantly highest in group N ($p<0.05$).

DIASTOLIC BLOOD PRESSURE: Maximum rise in DBP in groups N, F, and D was 88 ± 10 , 83 ± 6 and 80 ± 8 mmHg respectively, which was statistically significant ($p=0.000$). Magnitude of increase in DBP above the baseline was 19 ± 11 mmHg (29%) in group N, 11 ± 8 mmHg (17 %) in group F and 11 ± 8 mmHg (16%) in group D, which was statistically significant ($p=0.000$).

Fig. 1: Graph showing changes in Heart Rate, Systolic Blood Pressure, Diastolic Blood Pressure, and Mean Arterial Blood Pressure during study period. * indicates maximum value recorded. ($p=0.000$)

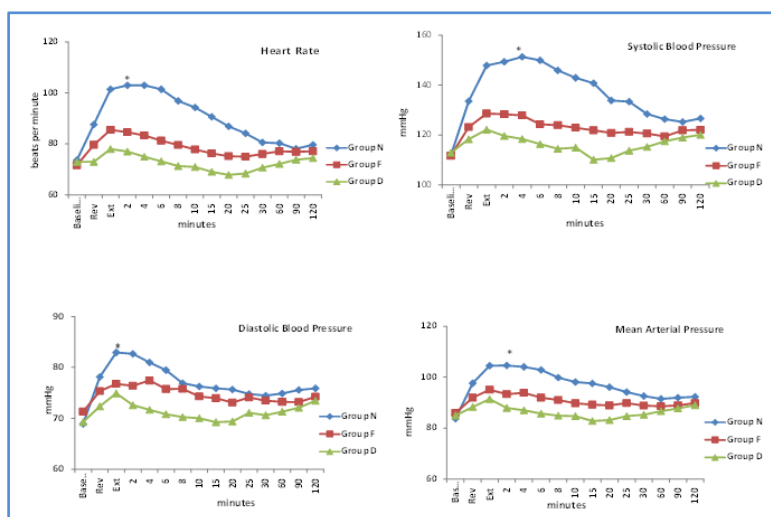


Fig. 1

MEAN ARTERIAL BLOOD PRESSURE: Maximum value of MAP observed in groups N, F, and D were 110 ± 10 , 99 ± 80 and 96 ± 9 mmHg respectively (p value). The magnitude of increase above baseline in groups N, F, and D was 27 ± 10 (32%), 13 ± 80 (16%) and 11 ± 9 (13%), mmHg respectively which was statistically significant. ($p=0.000$).

Acute haemodynamic perturbation during extubation necessitating the use of rescue drug (Esmolol 30 mg) as seen in Figure-3, was more in group N (34%) compared to group F (2%) and group D (0%) which was statistically significant ($p<0.001$).

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Heart Rate	Group N	Group F	Group D	P value
Baseline	74±10	72±9	73±8	0.54
Max. value (bpm)	108±10	91±11	83±9	0.000
Magnitude of increase above baseline (%)	34±11 (49±19)	19±10 (27±14)	10±10 (15±15)	0.000
No of patients having Tachycardia & %	42 (84%)	18 (36%)	4 (8%)	0.000
Average Duration of Tachycardia (min)	18±20	5±4	3±2	0.000
Systolic Blood Pressure				
Baseline	112±10	112±13	113±11	0.88
Max. value (mmHg)	159±11	136±13	130±9	0.000
Magnitude of increase above baseline (%)	47±13 (43±13)	24±14 (23±13)	17±13 (16±14)	0.000
No of patients having Hypertension (%)	43(86%)	9(18%)	3(6%)	0.000
Average Duration of Hypertension (min)	20±27	17±39	6±5	0.032
Diastolic Blood Pressure				
Baseline	69±8	71±8	69±7	0.32
Max. value	88±10	83±6	80±8	0.000
Magnitude of increase above baseline (%)	19±11(29±22)	11±8(17±14)	11±8(16±12)	0.000
No of patients having value ≥30% of baseline (%)	15(30 %)	7(14%)	2(4%)	0.002
Average Duration of Hypertension (min)	33±48	22±45	6±4	0.002
Mean Arterial Blood Pressure				
Baseline	84±7	86±8	85±7	0.399
Max. value (mmHg)	110±10	99±8	96±9	0.000
Magnitude of increase above baseline (%)	27±10 (32±14)	13±8 (16±11)	11±9 (13±10)	0.000
No of patients having value ≥30% of baseline (%)	24(48%)	2(40%)	2(4%)	0.000
Average Duration of Hypertension (min)	16±33	6±6	3±1	0.003

Table 2: Hemodynamics

Values are expressed as mean±SD or Number (%). N-Normal saline, F-Fentanyl, D-Dexmedetomidine. Bpm- beats per minute,

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RECOVERY PROFILE: Lower the extubation quality scale smoother was the extubation. The groups differed with regard to overall extubation quality ($p < 0.001$). Groups D (1.50 ± 0.58) and F (1.94 ± 0.55) had lower scores compared to group N (2.68 ± 0.47) implying smoother extubation. Among group D and F, group D had best extubation quality (Fig-2)

There was a statistically significant difference among the groups with regard to Ramsay sedation score and Aldrete's Recovery Score ($p < 0.003$) for 25 min following arrival at PACU. Groups F (2.08 ± 0.34) and D (2.16 ± 0.42) had higher sedation scores compared to group N (1.92 ± 0.27). During later part of observation there were no statistically and clinically significant changes among study groups.

Fig. 2: Graph showing average extubation quality scale in 3 study groups.

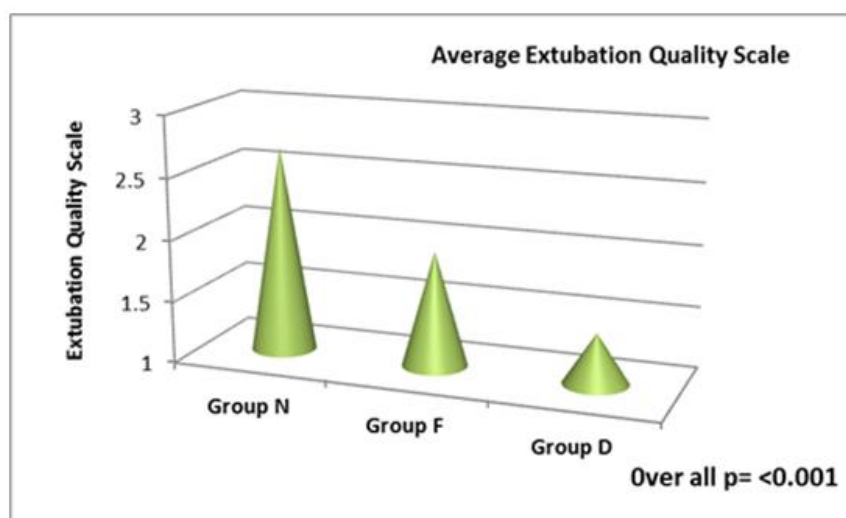


Fig. 2

DISCUSSION: Tracheal extubation is a critical step during emergence from general anaesthesia. Major airway complications, increased morbidity and mortality occur during extubation and emergence or during recovery in approximately one third of the reported closed claims cases relating to anaesthesia.^{25,26}

Fentanyl is used widely as an adjunct to general anaesthesia for attenuating hemodynamic responses to nociceptive stimuli induced by tracheal intubation, extubation and surgical procedures. Numerous studies,¹⁰ have shown that a bolus dose of intravenous fentanyl $2 \mu\text{g}/\text{kg}$ given at the end of surgery attenuates the cardiovascular changes associated with tracheal extubation and emergence from anaesthesia, without prolonging the recovery.

The alpha-2 agonist dexmedetomidine, a sedative and analgesic, reduces heart rate and blood pressure dose dependently. Guler G²⁷ and coworkers evaluated single-dose dexmedetomidine in attenuating airway and circulatory reflexes during extubation. They concluded that a single-dose bolus injection of dexmedetomidine administered 5 minutes before extubation attenuates airway-circulatory reflexes without prolonging the recovery. Recep Aksu et al²⁸ have found that a single-dose bolus injection of dexmedetomidine $0.5 \mu\text{g}/\text{kg}$ IV before tracheal extubation attenuates airway-circulatory reflexes better compared to fentanyl $1 \mu\text{g}/\text{kg}$ IV without prolonging recovery.

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Fig. 3: Graph showing use of rescue drug (Esmolol 30 mg) in study groups. $p < 0.001$

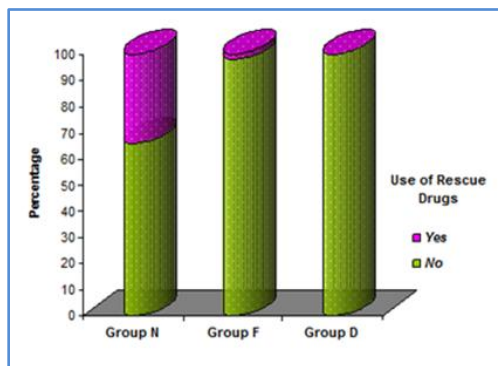


Fig. 3

Various studies have shown that dexmedetomidine in dosage 0.5-1 $\mu\text{g}/\text{kg}$ IV administered as bolus infusion 10-15 minutes prior to extubation stabilizes haemodynamics and facilitates smooth extubation compared to the control group.^{29,30,31,32,33}

In our study the control (normal saline) group, HR, SBP, DBP and MAP were significantly increased at peri-extubation period and thereafter ($p < 0.003$). All haemodynamic variables were around the baseline value at extubation in fentanyl group, and remained so throughout the study period. Whereas, in dexmedetomidine group, all haemodynamic variables were significantly decreased below the baseline value at extubation ($p < 0.003$) and remained so till the end of the study. Despite decreased haemodynamic parameters after extubation, none of the patients in the dexmedetomidine group required any clinical intervention.

Changes in HR, SBP, DBP, and MAP like maximum increase, magnitude of increase, number of patients having values $\geq 30\%$ above baseline and duration of this increased episode was more in control group compared to fentanyl and dexmedetomidine group. Tachycardia response was observed in 84% of control group compared to 36% of fentanyl group and 8% of dexmedetomidine group ($p = 0.000$). Hypertensive response was observed in 86% of patients in control group compared to 18% in fentanyl group and 6% in dexmedetomidine group which was statistically significant ($p = 0.000$). Duration of tachycardia ($p = 0.000$) and hypertensive response ($p = 0.032$) was higher in control group compared to other two groups.

Coughing and bucking (coughing on endotracheal tube) can result in hypertension, tachycardia, increased intraocular and intracranial pressure, myocardial ischemia, bronchospasm, surgical bleeding and wound dehiscence.³⁴ These adverse events are undesirable in patients with raised intracranial pressure, open globe injuries and aneurysmal surgeries etc.

We have used a 5 point extubation quality scale, where score 1 implies smooth extubation and score of 5 was poor. The average extubation quality in groups N, F and D was 2.68, 1.94 and 1.41 respectively. (Fig-2) 54% patients in dexmedetomidine group and 16% patients in fentanyl group had extubation quality scale of 1, while none in normal saline group. Dexmedetomidine group had smoother extubation quality than fentanyl group.

Sedation is a known and expected adverse effect of dexmedetomidine and fentanyl. We have followed our patients in the post-anaesthesia care unit for 2 hours following extubation, to ascertain whether study drug dosages of fentanyl or dexmedetomidine caused any significant sedation

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requiring clinical intervention. Initial 25 minutes of observation dexmedetomidine group had slightly higher Ramsay sedation score^{21,22} of 2.16 ± 0.42 compared to other groups which was statistically significant ($p=0.003$).

During later part of observation sedation score were comparable and none of patients in any group required any clinical intervention apart from routine post-operative monitoring. Aldrete's recovery score^{23,24} was used to assess the recovery and preparedness for discharge from post anaesthesia care unit (PACU) which was similar in the three groups studied.

All the patients, irrespective of their group, had a good recovery and were discharged from post anaesthesia care unit uneventfully. Studies done by Guler et al,²⁷ Recep Aksu et al²⁸ and Turan et al³¹ showed that dexmedetomidine was associated with smoother extubation quality without prolonging the recovery.

Acute haemodynamic perturbations which required use of rescue drug (Esmolol 30 mg) was significantly higher in control group (34%) compared to fentanyl group (2%) and dexmedetomidine (0%).

Our study shows that extubation and emergence from general anaesthesia was associated with significant haemodynamic perturbations (hypertension and tachycardia) during peri-extubation period in healthy patients which needs to be controlled. Both fentanyl and dexmedetomidine were useful in mitigating this emergence stress response without prolonging recovery. Dexmedetomidine had overall good control of haemodynamics, smoother extubation quality, uneventful recovery and post anaesthesia discharge.

CONCLUSIONS: Dexmedetomidine 1 µg/kg intravenous or fentanyl 1 µg/kg intravenous bolus infusion administered 10 minutes prior to extubation was effective in attenuating haemodynamic stress response during extubation. Dexmedetomidine was efficacious compared to fentanyl in mitigating emergence response. Extubation quality was superior with dexmedetomidine compared to fentanyl or placebo. Sedation score and recovery profile were comparable. Attenuation of emergence stress response should be routine than an optional entity in day to day clinical practice.

REFERENCES:

1. Minogue SC, Ralph J, Lampa MJ. Laryngotracheal topicalization with lidocaine before intubation decreases the incidence of coughing on emergence from general anesthesia. *Anesth Analg* 2004; 99: 1253-7.
2. Cook TM, Scott S, Mihai R. Litigation related to airway and respiratory complications of anaesthesia: an analysis of claims against the NHS in England 1995-2007. *Anaesthesia* 2010; 65: 556-63.2.
3. Peskett MJ. Clinical indicators and other complications in the recovery room or postanesthetic care unit. *Anaesthesia* 1999; 54: 1143-9.3.
4. Rose DK, Cohen MM, Wigglesworth DF, De Boer DP. Critical respiratory events in the postanesthesia care unit. Patient, surgical, and anesthetic factors. *Anesthesiology* 1994; 81: 410-8.4.
5. Mhyre JM, Riesner MN, Polley LS, Naughton NN. A series of anesthesia-related maternal deaths in Michigan, 1985-2003. *Anesthesiology* 2007; 106: 1096-104.5.

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6. Auroy Y, Benhamou D, Péquignot F, Bovet M, Jouglu E, Lienhart A. Mortality related to anaesthesia in France: analysis of deaths related to airway complications. *Anaesthesia* 2009; 64: 366–70.
7. Lewis G. The Confidential Enquiry into Maternal and Child Health (CEMACH). Saving Mothers' Lives: Reviewing Maternal Deaths to make Motherhood Safer—2003–2005. The Seventh Report on Confidential Enquiries into Maternal Deaths in the United Kingdom. London: CEMACH, 2007.7.
8. Asai T, Koga K, Vaughan RS. Respiratory complications associated with tracheal intubation and extubation. *British Journal of Anaesthesia* 1998; 80: 767–75.
9. Asai T, Koga K, Vaughan R. Respiratory complications associated with tracheal intubation and extubation. *Br J Anaesthesia* 1998; 80: 767-75.
10. Nishina k, Mikawa k, Maekawa N, Obara H. Fentanyl attenuates cardiovascular responses to tracheal extubation. *Acta Anaesthesiol Scand* 1995; 39: 85-89.
11. Batra YK, Singh H, Singh SP. Blood pressure and Pulse rate changes during tracheal extubation, influence of topical or intravenous lidocaine. *Indian Journal of Anaesthesia* 1986; 34: 31-4.
12. Dwyer J P O, Yorukoglu D, and Harris M N E. The use of Esmolol to attenuate the haemodynamic response when extubating patients following cardiac surgery. *Eur Heart J* 1993; 14: 701-4.
13. Kim HD, Kim Sy, Do YW, Seok CH. Effects of Intra operative Intravenous clonidine on cardiovascular responses to extubation. *Korean J Anaesthesiol* 1994; 27: 20-8.
14. Lam SW, Alexander E, Sulsa GM. DRUG UPDATE: Dexmedetomidine Use in Critical Care; *AACN Advanced Critical Care* 2008; 19: 113-20.
15. Candiotti KA, Bergese SD, Bokesch PM, Feldman MA, Wisemandle W, Bekker AY. Monitored Anesthesia Care with Dexmedetomidine: A Prospective, Randomized, Double-Blind, Multicenter Trial. *Anesth Analg* 2010; 110: 47-56.
16. Ebert TJ, Hall JE, Barney JA, Uhrich TD, Colino MD. The effects of increasing plasma concentrations of dexmedetomidine in humans. *Anesthesiology* 2000; 93: 382-94.
17. Jain D, Khan R, Maroof M.: Effect of Dexmedetomidine on Stress Response to Extubation. *The Internet Journal of Anesthesiology*. 2009; 21(1).
18. Mogensen JV. Neuromuscular monitoring. In, Miller RD(ed). *Miller's Anesthesia*, 7th ed. New York, Churchill Livingstone, Elsevier Inc, 2010; 1529.
19. Turan G, Ozgultekin A, Turan C, Dincer E, Yuksel G. Advantageous effects of dexmedetomidine on haemodynamic and recovery responses during extubation for intracranial surgery. *Eur J Anaesthesiol* 2008; 25: 816-20.
20. Ramsay M. Controlled sedation with alphaxalone-alphadone. *Br Med J* 1974; 2: 656-59.
21. Ramsay MA, Huddleston P, Hamman B, Tai S, Matter G. The patient state index correlates well with the Ramsay sedation score in ICU patients. *Anesthesiology* 2004; 101: A338.
22. Aldrete JA. The post-anesthesia recovery score revisited. *J Clin; Anesth* 1995; 7: 89–91.
23. Aldrete J & Kroulik D, A postanesthetic recovery score. *Anesth Analg* 1970.
24. Scott I. Marshall, FRCA and Frances Chung, FRCPC. Discharge criteria and complications after ambulatory surgery; *Anesth Analg* March 1999 vol. 88 no. 3 508.
25. Cook TM, Woodall N, Frerk C. Royal College of Anaesthetists. 4th National Audit Project: Major Complications of Airway Management in the UK. Royal College of Anaesthetists, London, 2011: 62–70.

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26. Peterson GN, Domino KB, Caplan RA, Posner KL, Lee LA, Cheney F W. Management of the difficult airway: a closed claims analysis. *Anesthesiology* 2005; 103: 33-9.
27. Guler G, Akin A, Tosun Z, Eskitascoglu E, Mizrak A. and Boyaci A. Single dose dexmedetomidine attenuates airway and circulatory reflexes during extubation. *Acta Anaesthesiol Scand.* 2005; 49: 1088-91.
28. Aksu R, Akin A, Biçer C, Esmaoğlu A, Tosun Z, Boyaci A. Comparison of the effects of dexmedetomidine versus fentanyl on airway reflexes and hemodynamic responses to tracheal extubation during rhinoplasty: A double-blind, randomized, controlled study; *Current Therapeutic Research* - June 2009.
29. Sağiroğlu A, Celik M, Orhon Z, Yüzer S, Sen B: Different Doses of Dexmedetomidine on Controlling Haemodynamic Responses to Tracheal Intubation. *The Internet Journal of Anesthesiology.* 2010;27(2).
30. Wang BS, Yu JB, Wang F, Zhang L, Zhang Y, Li SQ. *Zhonghua Er Bi Yan Hou Tou Jing Wai Ke Za Zhi.* "Effect of dexmedetomidine on stress responses during extubation in patients undergoing uvulopalatopharyngoplasty". 2012 Jun; 47 (6): 498-501.
31. Turan G, Ozgultekin A, Turan C, Dincer E, Yuksel G. Advantageous effects of dexmedetomidine on haemodynamic and recovery responses during extubation for intracranial surgery. *Eur J Anaesthesiol* 2008; 25: 816-20.
32. Bindu B, Pasupuleti S, Gowd UP, Gorre V, Murthy RR, Laxmi M B. A double blind, randomized, controlled trial to study the effect of dexmedetomidine on hemodynamic and recovery responses during tracheal extubation. *J AnaesthesiolClinPharmacol* 2013; 29: 162-7.
33. Fox EJ, Sklar GS, Hill CH, Villanueva R, King BD. Complications related to the pressor response to endotracheal intubation. *Anesthesiology* 1977; 47: 524-5.
34. Stone DJ, Gal. Airway management. In: Miller RD, ed. *Anesthesia.* 5th ed. Philadelphia, Pa.: Churchill Livingstone Co.; 2000: 1414-51.

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