International Journal of Health Sciences and Research
www.ijhsr.org ISSN: 2249-9571

Original Research Article

Effect of Dexmedetomidine on Airway Reflexes and Haemodynamic Responses to Tracheal Extubation

Ashem Jack Meitei[1], L. Pradipkumar Singh[2], Haorangbam Shanti Singh[3], Takhelmayum Hemjit Singh[4], Ningthoujam Anita Devi[2], Biswajit Debbarma[5]

[1]Senior Registrar, [2]Associate Professor, [3]Professor, [4]Asstistant Professor, [5]PGT,
Department of Anaesthesiology and Critical Care, Regional Institute of Medical Sciences, Imphal-4, Manipur, India.

Corresponding Author: Ashem Jack Meitei

Received: 21/10/2015 *Revised: 19/11/2015* *Accepted: 21/11/2015*

ABSTRACT

Background: Tracheal extubation significantly alter haemodynamic responses and produces unwanted airway reflexes. Aim is to study the effect of dexmedetomidine on attenuation of these changes and its side effects.
Methods: Fifty patients with ASA I & II aged 18 to 60 years were selected for elective surgeries under general anaesthesia. Ten minutes before reversing the neuromuscular blockade, patients were randomly allocated to receive either dexmedetomidine 0.5 mcg/kg body weight diluted in 10 ml saline (Group D, n=25) and Normal Saline 10 ml intravenously (Group C, n=25) on double blind design. Haemodynamic parameters were assessed before, during and after extubation. Time to extubation and eye opening, laryngospasm, bronchospasm and bouts of cough were recorded. Extubation quality was rated using 5-point scale. Sedation was rated using Ramsay Sedation Scale.
Results: Haemodynamic changes and incidence of coughing were significantly lower in dexmedetomidine group compared to Saline group ($P < 0.001$). Time to extubation and eye opening were prolonged in Group D ($p< 0.001$). Episodes of tachycardia ($p < 0.001$), hypertension ($p = 0.001$), coughing ($p = 0.001$) and agitation ($p < 0.001$) were more in Group C. Significant number of patients in Group D were drowsy (5/25; 20%, score of 3) and asleep (1/25; 4%, score of 4) but responded to verbal commands following extubation as compared to Group C (15/25; 60%, score of 2) who were co-operative and oriented. Extubation quality was better in Group D ($p < 0.001$).
Conclusion: Infusion of dexmedetomidine 0.5 µg/kg over 10 minutes before extubation attenuates the airway and haemodynamic reflexes during emergence from anaesthesia and facilitates smooth extubation without causing respiratory depression and undue sedation. But may prolonged time to extubation and eye opening.

Key words: Dexmedetomidine, Tracheal extubation, Hemodynamic responses, Airway reflexes.

INTRODUCTION

Tracheal extubation (a translaryngeal removal of a tube from the trachea via the nose or mouth) usually accompanied with significant rise in heart rate, myocardial contractility and increased systemic vascular resistance. [1-3] Though these rises are transitory and tolerable by majority, it may produce myocardial ischemia or infarction in susceptible patients. [4,5] It also occasionally produces unwanted airway reflexes leading to coughing, laryngospasm and bronchospasm which are another life threatening conditions. Various agents

have been studied for the protection of these responses. [6-12]

Dexmedetomidine (Dex) an alpha2 agonist with the properties of sedative, anxiolytic and analgesic action is known to exhibit attenuation of stress response to intubation. [13] The study was conducted with the dose of 0.5 µg/kg body weight to determine the degree of attenuation of hemodynamic responses and airway reflexes to tracheal extubation.

MATERIALS AND METHODS

Following institutional ethical committee approval, the study was conducted on fifty patients of ASA grade I-II aged between 18- 60 years of either sex undergoing elective surgeries of duration 90 minutes or less under general anaesthesia after obtaining informed written consent. Patients suffering from cardiac and pulmonary diseases or any endocrine disorder, surgeries on neck and oral cavities, history of drug abuse or psychological disorder, difficult airway or sleep apnoea and refuse to give inform consent were excluded.

By computer generated randomization, patients were divided into two groups of 25 patients each and received either dexmedetomidine (Group D), or Saline (Group C) in a double blind design. Preanaesthetic check up was conducted and a detailed history with complete physical and systemic examination including Mallampati assessment was recorded. All routine investigations were also done. At 10 pm on the night, prior to the day of surgery, all the patients were given oral Ranitidine 150 mg (Aciloc) and Alprazolam 0.5 mg (Zolam). On the day of surgery intravenous canulation with 20G to all the patients were established for drug and continuous fluid administration. Thirty minutes before the induction of anaesthesia, patients were premedicated with injection glycopyrrolate 4µg/kg body weight intramuscularly and injection ondansetron 4mg intravenously. On arrival in the operating room, standard monitoring devices including train of four (TOF) watch were connected to the patient and after settling in, patient's baseline heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), rate pressure product (RPP), percentage of oxygen saturation ($SPO_{2)}$ were recorded. Three minutes before induction of anaesthesia, patient received intravenous fentanyl (Troyfentil) 2 µg/kg body weight followed by preoxygenation with 100% oxygen for 3 minutes subsequently anaesthesia was induced with intravenous propofol 2 mg/kg and intubation facilitated with injection atracurium (Artacil) 0.5mg/kg body weight. Trachea was intubated using poly vinyl chloride (PVC) endotracheal tube of appropriate size. Anaesthesia was maintained with nitrous oxide in oxygen (60:40), trace isoflurane (<1%). Muscle paralysis was maintained with intermittent infusion of atracurium 10 µg/kg guided by TOF count. In both the groups, additional adjuvant was provided with injection diclofenac sodium 75 mg (JustIn) intramuscularly at 30 minutes following induction of anaesthesia. The idea was to avoid additional sedative effect and respiratory depression by opioid analgesics.

At the end of the surgery, 10 minutes before reversing the neuromuscular blockade, isoflurane was turned off then Group D patients received infusion of dex 0.5 µg/kg diluted in 10 ml saline over 10 minute while Group C patients received 10 ml Normal Saline over 10 minutes by using infusion pump (Perfusor Compact). Nitrous oxide was stopped following end of infusion of study drug.

Recording of HR, SBP, DBP, MAP, RPP, $SPO_{2,}$ $EtCO_{2,}$ TOF, ECG started immediately before infusion of study drug then at 1, 3, 5, 7 and 10 minutes respectively during infusion. Residual

neuromuscular blockade was reversed using injection neostigmine 0.05 mg/kg and glycopyrrolate 0.01 mg/kg intravenously when TOF ratio was ≥ 0.7. Another recording of same parameters as above was done following completion of reversal injection. Then tracheal extubation was performed when the extubation criteria (spontaneous respiration preserved, obeys commands, sustained hand grip for ≥ 5 seconds, tidal volume >6 ml/kg, TOF ratio ≥ 0.9) was fulfilled during which same parameters excluding TOF and $EtCO_2$ were recorded at extubation, 1 minute, thereafter every 5 minutes till 15 minutes following extubation. Bradycardia and tachycardia were defined as heart rate less than 60 or more than 90 beats per minute respectively. Hypotension was defined as 30% decrease from base line or mean arterial pressure (MAP) less than 60 mmHg whereas hypertension was define as 30% increase from base line or SBP more than 180 mmHg.

Extubation quality was monitored and assessed for 15 minutes following extubation based on cough immediately after extubation, using 5-points scale. [14] Score 1= no cough, 2= smooth extubation (1-2 cough), minimal coughing, 3= moderate coughing (3-4 times), 4= severe coughing (5-10 times) and straining, 5= poor extubation, very uncomfortable (laryngospasm and coughing more than 10 times). Similarly assessment of desaturation following extubation was also recorded for 15 minutes.

Time to extubation and time to eye opening, i.e. interval between termination of nitrous oxide to extubation and eye opening respectively were noted.

Post extubation sedation was evaluated (on 6 points scale) using Ramsay sedation scale. [15] 1= Anxious or agitated and restless or both, 2= Co-operative, oriented, tranquil, 3=Responsive to verbal commands, drowsy, 4=Asleep, responsive to light stimulation, 5=Asleep, slow response to stimulation, 6=No response to stimulation.

Statistical analysis

Base on a previous study a minimal sample size of 24 patients in each group were required though this study conducted on 25 patients in each group. Descriptive and inferential statistical analysis was conducted with the software SPSS 16.0, MS Word and Excel for Windows using student t test (two tailed, independent) to find significance of study parameters on continuous scale between two groups presented on mean ± SD and on categorical measurements presented in number (%), chi square/Fisher Exact test was used to find the significance of study parameters on categorical scale between two or more groups. P value < 0.005 was considered significant.

RESULTS

The patients in the study groups were comparable for age, height, weight, sex (male:female), ASA physical status, duration of surgery which was not statistically significant (p > 0.05) [Table-1].

Table-1: Demographic profile of the two study groups

Parameters	Group D n= 25, Mean±SD	Group C n= 25, Mean±SD	Test Statistic T	P Value
Age	37.24 ±10.997	36.08 ±10.551	0.381	.705
Height	156.32±4.589	156.80±6.880	.290	.773
Weight	56.80±5.530	56.20±7.687	.317	.753
Sex (male:female)	2:23	6:19	X^2=0.218	0.123
ASA (1:2)	22:3	21:4	X^2=0.058	0.684
Duration of surgery	64.56±18.423	57.72±14.825		0.155

Heart rate was significantly lower (p < 0.05) in group D from 7 minute of drug infusion till the end of observation (i.e. till 15 minute following extubation) [Table-2a].

Table-2a. Haemodynamic Parameters of the two study groups

Time Intervals	HR Variation		
	† Dex Mean ±SD	Saline Mean ± SD	P value
Baseline	91.48±5.745	93.28±8.429	.382
†† PTI	80.36±8.543	77.48±5.973	.174
* A1 min	80.88±9.787	78.16±7.140	.267
*A3 min	81.84±11.687	78.36±8.793	.291
*A5 min	79.80±13.641	81.80±8.813	.541
*A7 min	75.72±11.957	84.96±10.593	.006
*A10 min	72.92±12.546	89.84±12.037	.000
Reversal	71.40±13.928	93.24±15.743	.000
Extubation	85.12±12.401	109.72±17.719	.000
**B1 min	83.72±8.839	110.32±18.064	.000
**B5 min	80.00±8.865	97.88±12.531	.000
**B10 min	76.28±7.203	86.64±9.376	.000
**B15 min	74.68±8.102	80.96±7.575	.007

*A=During drug infusion period; **B=Following Extubation, †Dex=Dexmedetomidine, †† PTI= prior to drug infusion

A statistically significant difference was observed in SBP, DBP, and MAP ($p < 0.05$) from 10 minute of drug infusion till the end of observation [Table-2b].

Table-2b. Haemodynamic Parameters of the two study groups

Time Intervals	SBP variation			DBP variation			MAP variation		
	† **Dex Mean ±SD**	Saline Mean ± SD	P value	† Dex Mean ±SD	Saline Mean ± SD	P value	† Dex Mean ±SD	Saline Mean ± SD	P value
Baseline	128.88±6.300	129.52±7.264	.471	83.52±5.896	82.64±5.407	.585	98.84±5.444	98.28±5.319	.715
†† PTI	125.80±13.614	122.04±8.178	.242	85.44±10.568	81.64±8.755	.173	98.84±10.660	95.16±7.543	.165
* A1 min	131.72±15.694	126.24±9.243	.139	87.20±11.747	82.64±7.831	.141	102.04±12.229	97.00±7.354	.084
*A3 min	132.56±17.144	127.56±10.469	.219	88.92±13.162	82.72±11.066	.078	103.36±14.103	97.52±10.162	.099
*A5 min	134.28±16.794	130.96±10.118	.401	88.96±12.445	85.60±8.026	.262	104.00±13.444	100.72±8.8.147	.302
*A7 min	131.36±15.615	133.68±8.586	.518	87.40±14.315	87.56±7.119	.960	102.16±14.366	103.04±7.115	.785
*A10 min	127.84±12.970	135.64±9.504	.019	84.16±11.764	89.96±7.156	.040	98.72±11.603	105.12±7.748	.026
Reversal	125.76±13.965	139.24±10.349	.000	81.84±11.183	92.96±7.097	.000	96.52±11.544	108.24±7.584	.000
Extubation	133.48±12.530	150.68±9.512	.000	86.48±10.239	99.64±5.663	.000	102.16±10.168	116.60±6.110	.000
**B1 min	133.48±12.176	148.08±12.413	.000	86.92±9.827	98.60±9.958	.000	102.40±9.785	115.04±9.902	.000
**B5 min	125.36±10.492	142.64±12.649	.000	81.80±9.657	94.04±8.075	.000	96.40±8.665	110.20±9.005	.000
**B10 min	121.88±8.447	133.72±12.300	.000	80.36±9.313	88.76±6.685	.001	94.28±8.409	103.76±7.535	.000
**B15 min	122.60±6.583	128.68±11.120	.023	79.56±8.221	83.80±6.487	.049	94.00±7.059	98.84±7.341	.022

*A=During drug infusion period; **B=Following Extubation; †Dex=Dexmedetomidine
†† PTI = prior to drug infusion

We also observed a statistically significant difference ($p < 0.05$) in RPP between two groups from 7 minute of drug infusion onwards. It exceeds 12000 from 10 minute after administration of the drug [Table-2c].

Table-2c: Rate pressure product variation in two groups

RPP Variation			
Time Intervals	†Dex Mean ±SD	Saline Mean ± SD	P value
Baseline	11789.942±1150.774	12081.626±1305.382	.401
Prior to infusion	10232.56±1960.888	9469.48±1101.32	.096
*A1	10725.4±2219.968	9885.52±1316.845	.110
*A3	10902.56±2626.976	10037.3±1763.728	.178
*A5	10878.6±3099.744	10751.2±1804.835	.860
*A7	10044.08±2684.448	11408.6±1930.656	.045
*A10	9416.28±2513.217	12273.1±2340.097	.000
Reversal	9032.60±2445.169	13101.8±2932.317	.000
Extubation	11393.9±2137.764	16603.8±3189.244	.000
**B1	11127.3±1650.348	16561.5±3572.197	.000
**B5	10058.68±1567.474	13927.3±2613.570	.000
**B10	8941.88±2032.884	11603±1762.547	.000
**B15	9167.44±1184.696	11603±1439.921	.001

*A=During drug infusion period; **B=Following Extubation; †Dex=Dexmedetomidine

SPO_2 were comparable in two groups except at 1 minute following extubation which had shown suggestive of significance but it was clinically insignificant. We observed that $EtCO_2$ in two groups were comparable and found statistically insignificant.

Extubation quality 5-point scale was superior in group D implying smoother extubation as compared to group C ($p < 0.001$) [Table-3]. Time to extubation and time to eye opening were significantly prolonged in dexmedetomidine group compared to Saline group ($p \leq 0.001$) [Table-3].

Table-3: Showing Extubation parameters and extubation quality 5-points scale in two groups

Extuabtion parameters	Group D Mean±SD	Group C Mean±SD	P value	Extubation quality 5-points scale	Group D		Group C	
Time to Extubation	18.88±3.734	16.00±1.756	.001		No. of Pts	%	No. of pts	%
Time to eye opening	17.20±3.403	14.28±1.768	.000	Scale 1	23	92.0	3	12.0
Ext Quality 5 points Scale	1.08±0.277	2.32±0.802	.000	Scale 2	2	8.0	13	52.0
No. of bouts of cough	0.32±0.748	2.28±1.061	.000	Scale 3	0	-	7	28.0
				Scale 4	0	-	2	8.0
				Scale 5	0	-	0	-
				Total	25	100.0	25	100.0

It also observed that patients in dex group were significantly sedated compared to Saline group from extubation time till 5 minute post extubation [Table-4].

Table-4: Comparison of Ramsay sedation scale in two groups

Ramsay Sedation Scale			
Time Interval	Group D (Mean ±SD)	Group C (Mean ±SD)	P - value
Extubation	2.28± .542	1.60±.500	.000
1 min	2.12±.322	1.64±.490	.000
5 min	2.00±.000	1.84±.374	.038
10 min	2.00±.000	1.96±.200	.322
15 min	2.00±.000	2.00±.000	-
20 min	2.00±.000	2.00±.000	-
25 min	2.00±.000	2.00±.000	-
30 min	2.00±.000	2.00±.000	-

Bradycardia was observed in 16% in Dex group requiring no rescue treatment otherwise episodes of tachycardia ($p < 0.001$), hypertension ($p = 0.011$), coughing ($p < 0.001$), and agitation ($P < 0.001$) were more in group C [Fig:1]

Incidence of coughing was significantly lower in dex group. Four (16%) patients in group D as compared to 22 (88%) patients in group C had cough beyond 15 minutes of following extubation [Fig:2].

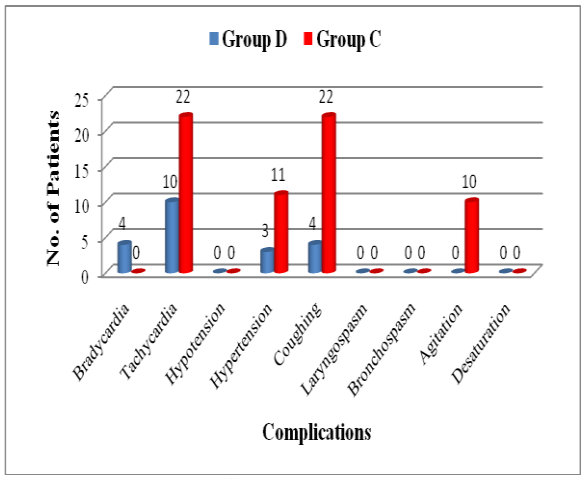

Figure-1: Comparison of complications in two groups

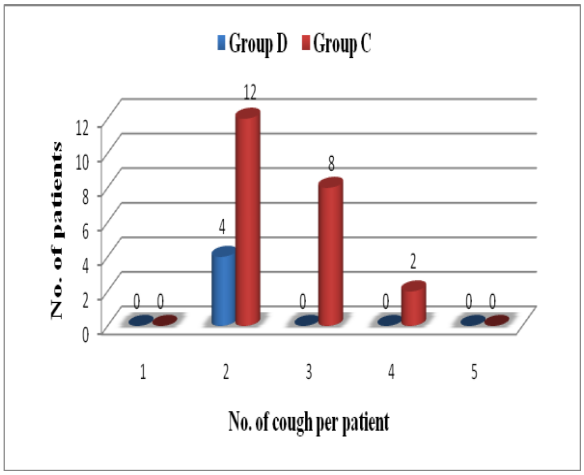

Figure-2: Number of cough per patient beyond 15 minute of post extubation in two groups

There were neither any observations of ECG changes nor desideration, bronchospasm or laryngospasm in both the groups.

DISCUSSION

Emergence from general anaesthesia and tracheal extubation frequently accompanied with several unwanted effects including respiratory and haemodynamic alterations which are transient and well tolerated by most of the patients but dangerous in susceptible patients. [12] Protection against these alterations may be a reasonable option to avoid its complications. Dex a highly selective alpha2 agonist has been shown to have anxiolytic, analgesic and sedative effects. It causes a dose dependent decrease in heart rate and blood pressure by decreasing in serum norepinephrine concentration.

We conducted this study on patients undergoing elective surgeries under general anaesthesia in an attempt to examine whether administration of dexmedetomidine 0.5 µg/kg was sufficient to attenuate haemodynamic alterations and airway reflexes to extubation without respiratory complications and sedation.

We observed that there was no significant rise of heart rate compared to base line value from the time of dexmedetomidine administration till the end of observation. But in saline group, there was a significant rise in heart rate compared to dexmedetomidine group ($p < 0.001$). This observation was similar to those reported in earlier study where the pulse rate in the study group remained below the base line values at all time intervals following extubation. [5] Bradycardia was observed in 4 patients (16%) in group D which was transient and did not required treatment. This observation was supported by previous studies. [10, 16] SBP, DBP and MAP values were significantly lower in group D compared to group C starting from 10th minute of dexmedetomidine infusion till the end of study. This is comparable with the study conducted by Jain D et al [5] in which, patients in study group received dexmedetomidine 1 mcg/kg, and they did not observe any significant changes in the blood pressure in dexmedetomidine group throughout the study period. On contrary, SBP increased significantly in control group following extubation as we observed in our study which we achieved with dexmedetomidine 0.5 mcg/kg in our study only 3 (12%) patients in group D had hypertension as against 11 (44%) patients in group C [Fig:1] This observation is in contradiction with the study done by Aksu R et al [16] who observed significantly increased SBP at 1 and 5 minutes after extubation. Probably this is due to rapid infusion of dexmedetomidine over 5 minutes rather than slow infusion. It is shown with dexmedetomidine that there is a biphasic response with initial increase in blood pressure due to vasoconstrictive effects followed by reduction in blood pressure due to central sympatholysis. [17,18]

RPP which is a better indicator of myocardial oxygen requirement was observed significantly higher in saline group from 7th minute of drug infusion onwards. When compared with dexmedetomidine group, it exceeds 12000 starting from 10 minute of drug infusion till 5 minute post extubation. This shows the effectiveness of controlling the RPP with infusion of 0.5mcg/kg of Dex prior to extubation thereby preventing pathological changes in myocardial cells or tissues. This study is supported by Zuhua Ren et al. [11]

Dexmedetomidine being having the properties of analgesi**c** and sedative is known to blunt airway reflexes. Ninety two percent (92%) of the patients in group D had smooth extubation as compared to 12% in group C. Incidence of coughing was significantly higher in Saline group than when compared to Dex group (88%

vs 8%). This is in accordance with the study done by Aksu R et al. [16] Guler G et al [19] also noted the effect of dexmedetomidine on children undergoing adenotonsillectomy wherein dex group had significantly decreased incidence and severity of agitation and a smooth extubation without any side effects. The number of moderate to severe cough per patient was nil in group D when compared to group C. This further supports the observation in the present study. Hence, dex improves extubation quality.

There were no observations of desaturation, bronchospasm or laryngospasm in either of the groups. These observations are in concurrence with study conducted by Aksu R et al. [16]

Significant number of patients in group D (5/25;20%) were drowsy (score of 3) and (1/25;4%) was asleep (score of 4) but responded to verbal commands following extubation as against (15/25;60%) of patients in control group who were co-operative and oriented (score of 2). However, those patients who were drowsy and asleep maintained SpO2 with humidified oxygen administered by facemask at 4 liter/ minute which was routinely provided for all patients in Post Anaesthetic Care Unit (PACU). This observation is in agreement with the comparative study done between dexmedetomidine and fentanyl in those undergoing rhinoplasty by Aksu R et al. [16] But, in contrast to Jain D et al [5] who did not notice sedation in both the groups probably because of the difference in the anaesthetic technique employed by the authors.

Agitation was observed in 10 patients (40%) in Group C following extubation whereas none were agitated in Group D. This is statistically and clinically significant ($P < 0.001$). This observation is in conjunction with study done by Guler G et al [19] who conducted a study on the effect of single dose dexmedetomidine in reducing the agitation and providing smooth extubation after paediatric adenotonsillectomy.

In the present study, time to extubation and eye opening were significantly prolonged in dex group compared to saline group. This observation is in agreement with the study done by Guler G, Akin A et al [20] on emergence agitation in children undergoing adenotonsillectomy.

CONCLUSION

We conclude that infusion of dexmedetomidine 0.5 µg/ kg body weight given over 10 minutes before tracheal extubation attenuates the airway and hemodynamic reflexes during emergence from anesthesia without causing undue sedation. But, may slightly prolong in time to extubation and eye opening.

REFERENCES

1. Hartley M,Vaughan RS. Problems with tracheal extubation. Br J Anaesth 1993;71:561-8.
2. Paulissian R, Salem MR, Joseph NJ, Braverman B, Cohen HC, Crystal GJ *et al*. Hemodynamic responses to endotracheal extubation after coronary artery bypass grafting. Anesth Analg 1991;73:10-15.
3. Lowrie A, Johnson PL, Fell D, Robinson SL. Cardiovascular and plasma catecholamine responses at tracheal extubation. Br J Anaesth 1992;68:261-3.
4. Elia S, Liu P, Chrusciel C, Hilgenberg A, Skourtis C, Lappas D. Effects of tracheal extubation on coronary blood flow, myocardial metabolism and systemic haemodynamic responses. Can J Anaesth 1989;36:2-8.
5. Jain D, Khan R, Maroof M. Effect of dexmedetomidine on stress response to extubation. The Internet Journal of Anesthesiology 2009;21:1
6. Jee D, Park SY. Lidocaine sprayed down the endotracheal tube attenuates the airway-circulatory reflexes by local anesthesia during emergence and extubation. Anesth Analg 2003;96: 293-7.

7. Mikawa K, Nishina K, Takao Y, Shiga M, Maekawa N, Obara H. Attenuation of cardiovascular responses to tracheal extubation. comparison of verapamil, lidocaine, and verapamil-lidocaine combination. Anesth Analg 1997;85: 1005-10.
8. Park SH, Do SH, Shin HY, Jeon YT, Hwang JW, Han SH. Nicardipine is more effective than esmolol at preventing blood pressure increases during emergence from total intravenous anesthesia. Korean J Anesthesiol 2009;57:597-603.
9. Sanjaya KG, Maniram KH, Ratan Singh N, Chaoba Singh L. Attenuation of cardiovascular response to extubation by lignocaine, esmolol and propofol. JMS 2009;23:15-19.
10. Ellis JE, Drijvers G, Pedlow S, Laff SP, Sorrentino MJ, Foss JF. Premedication with oral and transdermal clonidine provides safe and efficacious post operative sympatholysis. Anesth Analg 1994;79:1133-40.
11. Ren Z, Shao J, Zhang J, Liu Y. Effect of dexmedetomidine on myocardial oxygen consumption during extubation for old patients: A bispectral index-guided observation study. Afr J Pharm. Pharmacol 2013;7:1033-7
12. Bindu B, Pasupuleti S, Gowd UP, Gorre V, Murthy RR, Laxm MB. A double blind, randomized, control trial to study the effect of dexmedetomidine on hemodynamic and respiratory responses during tracheal extubation. Journal Of Anaesthesiology Clinical Pharmacology 2013;29:162-7.
13. Basar H, Akpinar S, Doganci N, Buyukkocak U, Kaymak C, Sert O *et al*. The effects of preanesthetic, single-dose dexmedetomidine on induction, hemodynamic, and cardiovascular parameters. J Clin Anesth 2008;20: 431-6.
14. Nishina K, Mikawa K, Maekawa N, Obara H. Fentanyl attenuates cardiovascular responses to tracheal extubation. Acta Anaesthesiol Scand 1995;39:85-9.
15. Ramsay M. Controlled Sedation With Alphaxalone-Alphadone. Br Med J 1974;2:656-9.
16. Aksu R, Akin A, Bicer C, Esmaoglu 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. Curr Ther Res Clin Exp 2009;70:209-20.
17. Hall JE, Uhrich TD, Barney JA, Arain SR, Ebert TJ. Sedative, amnestic, and analgesic properties of small-dose dexmedetomidine infusions. Anesth Analg 2000;90:699-705.
18. Bloor BC, Ward DS, Belleville JP, Maze M. Effects of intravenous dexmedetomidine in humans. II. Hemodynamic changes. Anesthesiology 1992;77:1134-42.
19. Guler G, Akin A, Tosun Z, Ors S, Esmaoglu A, Boyaci A. Single- dose dexmedetomidine reduces agitation and provides smooth extubation after pediatric adenotonsillectomy. Pediatr Anesth 2005;15:762-6.
20. Guler G, Akin A, Tosun Z, Eskitascoglu E, Mizrak A, Boyaci A. Single-dose dexmedetomidine attenuates airway and circulatory reflexes during extubation. Acta Anesthesiol Scand 2005;49:1088-91.

How to cite this article: Meitei AJ, Singh PL, Singh HS et al. Effect of dexmedetomidine on airway reflexes and haemodynamic responses to tracheal extubation. Int J Health Sci Res. 2015; 5(12):66-73.

****