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A COMPARATIVE STUDY TO ASSESS THE EFFECT OF TWO DIFFERENT DOSES OF INTRAVENOUS DEXMEDETOMIDINE ON ATTENUATION OF AIRWAY REFLEXES AND STRESS RESPONSE TO EXTUBATION AFTER GENERAL ENDOTRACHEAL ANAESTHESIA

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ABSTRACT

Background: Smooth emergence and extubation after general anaesthesia requires the attenuation of hemodynamic stress response and airway reflexes to prevent adverse postoperative outcomes. Dexmedetomidine is a highly selective alpha2 -adrenergic agonist with sedative, hypnotic and analgesic properties which can counteract the extubation response.

Aims and Objectives: We aimed to study and compare the effect of bolus dose of 0.5µg/ kg and 1µg/ kg of intravenous Dexmedetomidine on attenuation of hemodynamic responses and airway reflexes during extubation after general anaesthesia.

Materials and Methods: This double blinded randomized controlled study was conducted on 90 patients who were ASA physical status I and II and aged between 20 to 60 years. The patients were randomly divided into 3 groups (n=30 in each). All patients were administered general endotracheal anaesthesia. Group C, the control group received 10 ml normal saline intravenous injection as placebo prior to extubation. Group D 5 received 0.5 µg/ kg body weight of Dexmedetomidine intravenous injection and Group D 1 received 1 µg/ kg body weight of Dexmedetomidine intravenous injection. HR, SBP, DBP, SpO2 readings prior to drug or placebo infusion; 1, 2,5, 7 and 10 minutes during infusion; following reversal administration; post extubation every 5 min for 15 min, thereafter every 15 min for next 2 hours were noted down. Time to extubation, time to emergence and sedation score as per Ramsay scale were also documented.

Results: The study showed that intravenous Dexmedetomidine in the dose of 0.5 µg/ kg and 1 µg/ kg caused statistically significant attenuation of hemodynamic stress response to extubation in camparison to the control group. Although in group D1, the blood pressure showed a biphasic response with initial increase in blood pressure followed by gradual decline in blood pressure values. Time to extubation and emergence were significantly prolonged in Group D1 when compared to Group D5 and C ($p< 0.001$). Patients in group D1 (1µg/kg) had the highest sedation scores at all time periods as compared to group C and group D5 ($p<0.001$).

Conclusion: Intravenous Dexmedetomidine in the dose of 0.5 µg/ kg body weight and 1 µg/ kg body weight attenuates hemodynamic stress response to extubation, thereby facilitating smoother emergence from anaesthesia. But in the higher dosage group, the recovery times were significantly prolonged and sedation scores were significantly higher at all times. Therefore, we conclude that intravenous Dexmedetomidine in the dose of 0.5 µg/ kg body weight is better with less complication rates.

KEY WORDS: Dexmedetomidine; Hemodynamic stress response; Extubation response; Smooth emergence

Introduction:

Endotracheal intubation has become an integral part of balanced anaesthesia since its introduction by Ivan. W. Magill and Rowbotham in 1921 because of its ability to secure the airway and also isolate the airway. Cardiovascular responses in the form of tachycardia and hypertension to endotracheal intubation are well documented.[1] Emergence from general anaesthesia and tracheal extubation are also often accompanied by adverse hemodynamic response and is of equal concern as intubation response.[2]

Majority of the patients tolerate adverse cardiovascular responses without any significant clinical consequences but patients suffering from diseases like hypertension, diabetes, ischaemic heart disease may be unable to do so. A frequent recommendation is to maintain heart rate and blood pressure within 20% of normal awake value of that patient to prevent myocardial ischaemia. Perioperative ischemic insults have a causal relationship to post-operative myocardial infarction.[3,4] It thus becomes imperative to prevent these responses for a better postoperative outcome.

Smooth extubation also requires the absence of straining, movement, coughing, breath holding, and laryngospasm.[5] Many techniques and drugs have been proposed to attenuate airway and cardiovascular responses, but none have been completely successful. Various options in vogue to attenuate extubation response include: deeper planes of anaesthesia, topical anaesthesia, use of intravenous local anaesthetics (Lignocaine), calcium channel blockers (Verapamil, Nicardipine), opioids (Fentanyl, Alfentanil), sympathetic blockers (beta blockers, alpha2 –agonists) etc .[5]

Alpha2 -agonists simultaneously potentiate the effects of general anaesthetics, reduce their dose requirements, and attenuate sympathoadrenal responses to noxious stimuli encountered during anaesthesia and surgery, thus providing improved hemodynamic, metabolic, and hormonal stability.[4] Clonidine has been widely studied in this aspect. Due to its prolonged duration of action and undue sedation we decided to use Dexmedetomidine, a newer drug in this class which is a highly selective alpha2 - adrenergic agonist. It has sedative, anxiolytic and analgesic actions as well. It is known to exhibit dose dependent attenuation of stress response to intubation. Not enough literature is available with regard to hemodynamic response to extubation using Dexmedetomidine. Hence a study was conducted to assess the degree of attenuation of hemodynamic responses and airway reflexes to extubation by Dexmedetomidine.[6]

Objective:

To study the effect of two different dose of intravenous Dexmedetomidine on attenuation of hemodynamic responses and airway reflexes during extubation following surgery under general anaesthesia.

Materials and Methods:

This double- blinded randomized controlled study was carried out in a tertiary hospital after approval from institutional ethics committee. A total of 90 study subjects aged between 20 to 60 years and belonging to ASA physical status I and II who were admitted for the various surgeries

under general anaesthesia were included in the study. Patients suffering from significant cardiac, pulmonary or endocrine disorder, those with history of drug abuse or psychological disorder and obese patients, with difficult airway or history of sleep apnoea were excluded from the study.

After obtaining informed written consent, patients were randomly divided into 3 groups based on computer generated random allocation. Enrolling investigator prepared the drug and had no role in drug administration or patient assessment.

- Group C: Control group consisting 30 patients received 10 ml normal saline intravenous injection as placebo prior to extubation.
- Group D 5: consisting 30 patients received 0.5 µg/ kg body weight of Dexmedetomidine intravenous injection diluted to 10 ml in normal saline.
- Group D 1: consisting 30 patients received 1 µg/ kg body weight of Dexmedetomidine intravenous injection diluted to 10 ml in normal saline.

All patients were monitored with electrocardiography (ECG), percentage saturation of oxygen (SpO2), non- invasive blood pressure (NIBP), end tidal carbondioxide (EtCO2). Trachea was intubated with a soft seal cuffed sterile poly vinyl chloride endotracheal tube of appropriate size. Endotracheal tube cuff was inflated with air with just enough volume to prevent an audible leak.
Anaesthesia was maintained with 50% nitrous oxide in oxygen and Isoflurane. Muscle paralysis was maintained with Inj.Vecuronium bromide 0.08mg/kg bolus dose and 0.02 mg/kg intermittent dose. Fifteen minutes before the estimated time of completion of surgery Isoflurane was turned off. Patients received Dexmedetomidine bolus or placebo intravenous infusion over 10 minutes depending on their group allocation. Nitrous oxide was stopped following end of infusion.
Residual neuromuscular blockade was reversed using injection Neostigmine 0.05 mg/ kg and injection Glycopyrrolate 0.008 mg/ kg iv when patient's spontaneous respiratory efforts were appreciated as flickers on the reservoir bag of Bain's circuit. Patient was extubated when respiration was considered adequate in terms of rate, rhythm and tidal volume. Oxygen supplementation at 5 l/ min was provided with Hudson mask for upto 2hrs postoperatively.
HR, SBP, DBP, SpO2 readings prior to drug or placebo infusion; 1, 2,5, 7 and 10 minutes during infusion; following reversal administration; post extubation every 5 min for 15 min, thereafter every 15 min for next 2 hours were noted down. Time to extubation, time to emergence and sedation scores were also documented.

Descriptive and inferential statistical analysis was carried out in the present study. Results on continuous measurements were presented on Mean±SD (Min-Max) and results on categorical measurements were presented in Number (%). Microsoft word and Excel have been used to generate graphs, tables etc.
Student t test (two tailed, independent) was used to find the significance of study parameters on continuous scale between two groups (Inter group analysis) on metric parameters.

Results:

A total of 30 study subjects were included in each group and analysed

HEART RATE	**GROUP D5**		**P VALUE**	**GROUP C**		**P VALUE**	**GROUP D1**	
(BEATS/MIN)	***MEAN***	***SD***		***MEAN***	***SD***		***MEAN***	***SD***
BASAL	88.13	13.22	0.27	84.93	8.43	0.49	83.57	6.35
T_1	85.87	12.83	0.58	87.43	8.31	<0.001**	77.43	6.25
T_2	82.67	12.74	0.02^+	89.77	9.63	<0.001**	69.63	6.90
T_5	74.03	11.11	<0.001**	92.50	10.68	<0.001**	60.93	6.36
T_7	76.47	11.61	<0.001**	95.70	10.73	<0.001**	62.80	5.27
T_{10}	75.90	12.34	<0.001**	96.73	11.37	<0.001**	62.17	5.50
T_R	80.30	12.03	<0.001**	101.77	9.68	<0.001**	64.50	5.44
T_E	87.83	12.75	<0.001**	112.53	12.47	<0.001**	74.47	6.40
PE_5	79.53	13.32	<0.001**	103.43	10.79	<0.001**	66.03	6.57
PE_{10}	75.07	13.16	<0.001**	96.10	10.46	<0.001**	65.23	6.93
PE_{15}	75.23	12.15	<0.001**	92.13	8.47	<0.001**	66.03	6.95
PE_{30}	75.47	11.25	<0.001**	88.13	7.71	<0.001**	68.57	5.36
PE_{60}	78.27	10.52	0.01^+	85.33	8.38	<0.001**	72.87	4.39
PE_{90}	81.47	10.55	0.77	82.17	7.53	0.02^+	78.00	6.04
PE_{120}	82.23	8.79	0.15	79.03	8.31	0.87	78.73	6.08

Demographic data and duration of surgery and anaesthesia were comparable in the three study groups.

Our study showed that basal heart rate of groups C, D5 and D1 were comparable. But it was statistically lower from the 2nd minute of drug infusion up to 60 minutes in group D5 (0.5µg/kg) and from 1st min of infusion up to 90 minutes post extubation in D1 as compared to group C. Maximum fall in heart rate was at 5th min after infusion and was 16 % and 27% respectively in group D5 and D1. Post extubation reduction in heart rate was maximum at 10 min- 15% and 22% respectively in group D5 and D1 as compared to baseline.

Table 1: Comparison of Heart Rate among the three study groups

'+' – SIGNIFICANT '**'-HIGHLY SIGNIFICANT

Table 2: Comparison of Systolic BP among the Three Study Groups

SYSTOLIC BP	GROUP D5		P VALUE	GROUP C		P VALUE	GROUP D1	
(mmHg)	*MEAN*	*SD*		*MEAN*	*SD*		*MEAN*	*SD*
BASAL	125.57	12.53	0.53	127.50	11.02	0.43	125.40	8.81
T_1	123.63	11.41	<0.001**	132.80	9.31	0.36	130.57	8.96
T_2	122.07	9.65	<0.001**	139.53	9.84	0.95	139.70	8.65
T_5	119.80	11.07	<0.001**	145.17	11.27	0.03^+	150.60	6.65
T_7	117.40	9.32	<0.001**	146.73	11.12	<0.001**	133.30	7.11
T_{10}	114.97	8.93	<0.001**	148.50	13.42	<0.001**	122.43	6.72
T_R	119.47	10.44	<0.001**	153.03	16.11	<0.001**	116.10	5.95
T_E	124.10	11.54	<0.001**	165.37	19.31	<0.001**	126.03	7.61
PE_5	114.70	10.30	<0.001**	151.00	11.39	<0.001**	117.77	6.48
PE_{10}	111.30	9.81	<0.001**	144.13	10.07	<0.001**	114.43	5.77
PE_{15}	111.33	9.81	<0.001**	139.33	8.35	<0.001**	112.93	6.62
PE_{30}	112.37	10.09	<0.001**	134.93	8.16	<0.001**	108.13	6.84
PE_{60}	118.63	8.67	<0.001**	130.37	7.31	<0.001**	115.53	6.41
PE_{90}	122.97	8.35	0.01^+	127.73	5.69	<0.001**	120.30	7.06
PE_{120}	123.47	8.85	0.05	127.03	5.99	0.04^+	123.40	7.62

'+' – SIGNIFICANT '**'-HIGHLY SIGNIFICANT

SBP was statistically lower in group D5 from 1st minute of drug infusion up to 90 minutes post extubation as compared to group C. Whereas, the values in higher dosage group (group D1) were comparable to group C up to 5 minutes of drug infusion due to an initial increase in SBP in the study group. From 7th minute of drug infusion there was persistent fall in SBP values up to 120 minutes post extubation which was statistically significant ($p<0.001$). This suggests a BIPHASIC RESPONSE of the SBP in higher dosage group (group D1). Maximum reduction in SBP post extubation was between 10th to 30th min and was 11% in group D5 and 14% in group D1 as compared to baseline. P value was significant at all measurement points during drug infusion, with statistically lower values in group D5 as compared to D1 due to biphasic response

Table 3: Comparison of Diastolic BP among the three study groups

DIASTOLIC BP	GROUP D5		P VALUE	GROUP C		P VALUE	GROUP D1	
(mmHg)	*MEAN*	*SD*		*MEAN*	*SD*		*MEAN*	*SD*
BASAL	77.87	10.01	0.41	81.60	10.05	0.03^{+}	76.73	5.60
T_1	77.60	9.00	0.26	84.57	9.54	0.03^{+}	80.00	5.51
T_2	75.73	9.02	<0.001**	87.90	9.65	0.02^{+}	82.73	6.21
T_5	74.97	11.78	<0.001**	90.27	11.28	0.11	86.43	5.96
T_7	72.77	8.07	<0.001**	92.07	10.51	<0.001**	76.93	4.86
T_{10}	71.53	8.22	<0.001**	92.77	12.28	<0.001**	72.57	5.55
T_R	75.07	7.37	0.03^{+}	95.50	12.11	<0.001**	67.67	5.73
T_E	78.80	10.75	0.03^{+}	101.17	12.00	<0.001**	78.30	7.14
PE_5	70.70	9.02	0.01^{+}	91.57	9.79	<0.001**	72.43	7.70
PE_{10}	67.77	9.12	<0.001**	89.67	8.59	<0.001**	70.10	7.65
PE_{15}	67.50	6.72	<0.001**	86.90	8.31	<0.001**	67.67	5.49
PE_{30}	69.40	6.56	0.27	83.60	7.80	<0.001**	64.87	5.56
PE_{60}	74.23	8.28	0.89	81.53	9.56	<0.001**	72.33	6.63
PE_{90}	76.00	6.76	0.57	80.03	7.78	0.01^{+}	74.80	6.26
PE_{120}	78.53	7.05	0.52	79.50	8.77	0.48	78.10	6.00

'+' – SIGNIFICANT '**'-HIGHLY SIGNIFICANT

Table 3 shows that basal DBP of groups C, D5 and D1 were comparable. DBP began to fall in group D5 (0.5µg/kg) at 2nd min of infusion with the onset of drug effect, while in group D1 (1µg/kg) there was a BIPHASIC RESPONSE with an initial rise starting at 1st minute of infusion and reaching peak at 5th minute (12% above baseline), followed by gradual decline to baseline value at 7th min of infusion. Maximum DBP reduction after extubation was observed between 15th to 30th min and was 13% in group D5 and 16% in group D1 as compared to baseline. P value was highly significant at reversal administration and 30 min post extubation with statistically lower values in group D1.

Table 4: Comparison of Extubation Parameters in the three groups studied

EXTUBATION PARAMETERS	GROUP D5	GROUP C	GROUP D1
TIME TO EXTUBATION (IN MIN)	17.43±3.63^{+}	13.97±1.77	23.17±4.29*

TIME TO EMERGENCE (IN MIN)	19.0± 3.90^{+}	15.23±1.83	24.33±4.34*

* Time to extubation and emergence were significantly prolonged in Group D1 when compared to Group D5 and C ($p < 0.001$).

$^{+}$ Recovery times were significantly longer in Group D5 ($p<0.001$) as compared to Group C

Table 5: Comparison of Ramsay Sedation Scale among the three groups studied

RAMSAY SEDATION SCORE	**GROUP D5**	**GROUP C**	**GROUP D1**
	MEAN±SD	***MEAN±SD***	***MEAN±SD***
T_E	2.50±0.51^{+}	1.77±0.43	3.27±0.64*
PE_5	2.33±0.48^{+}	1.77±0.43	3.27±0.64*
PE_{10}	2.20±0.41^{+}	1.93±0.25	3.23±0.57*
PE_{15}	2.10±0.31^{+}	1.93±0.25	3.23±0.57*
PE_{30}	2.10±0.31	2.00±0.00	3.17±0.65*
PE_{60}	2.07±0.25	2.00±0.00	2.57±0.57*
PE_{90}	2.00±0.00	2.00±0.00	2.30±0.47*
PE_{120}	2.00±0.00	2.00±0.00	2.27±0.45*

* Patients in group D1 (1µg/kg) had the highest sedation scores at all time periods as compared to group C and group D5 ($p<0.001$).

$^{+}$ While patients in Group D5 (0.5µg/kg) had a statistically higher score at extubation and up to 15 minutes post extubation as compared to group C ($p<0.05$). SpO_2 of both the study groups were comparable to the control group.

Seven patients in group D5 and eight in group D1 had hypotension. In group D1, 2 patients developed bradycardia which responded to Inj. Atropine 0.6mg i.v. During extubation 7 patients in group C were agitated. 2 patients in group D5 had coughing post extubation as against 10 patients in control group. 2 patients in group D1 desaturated (SPO2< 90%) after extubation. There was no incidence of laryngospasm or bronchospasm in any of the groups.

DISCUSSION: Dexmedetomidine is a highly selective alpha2 -adrenergic agonist which causes a decrease in the sympathetic outflow and noradrenergic activity, thereby counteracting hemodynamic fluctuations occurring at the time of extubation due to increased sympathetic stimulation. It is known to produce sedation, hypnosis and analgesia while maintaining patient arousability and respiratory function.

Various studies have used bolus Dexmedetomidine in the dose of 0.5 µg/kg, 0.75µg/kg and 1µg/kg for this purpose. Previous studies have shown insignificant results with Dexmedetomidine at the dosage of 0.5µg/kg (Aksu R et al[7] and pilot study done by Barkha Bindu et al[8]). This prompted us to compare Dexmedetomidine 0.5 µg/kg dose with a higher dosage group. Our study was hence

designed to assess the degree of attenuation of hemodynamic responses and airway reflexes to extubation by administration of Dexmedetomidine at the dosage of 0.5 μg/ kg body weight and 1 μg/kg body weight and compare the response between the two groups. Dexmedetomidine was given as a bolus, 15 minutes prior to completion of surgery as it has peak action at around 15 minutes of intravenous administration. This was similar to study done by Barkha Bindu et al[8]. The infusion was given over 10 minutes in contrast to infusion over 5 minutes in the study done by Aksu R et al[7] as the attenuation of blood pressure was not significant in their study, presumably due to faster rate of infusion.

Technique of anaesthesia was similar in the three study groups. Parameters observed include HR, SBP, DBP, SPO2 readings prior to drug or placebo infusion (basal); 1,2,5,7 and 10 minutes during infusion; following reversal administration, post extubation every 5 minutes for 15 minutes, thereafter every 30 minutes for next 2 hours. Extubation quality, sedation, time to extubation and emergence were noted. In our study the three study groups were comparable with respect to age, sex, weight, duration of surgery and anaesthesia.

In our study we observed that, in Group D5 and D1, HR did not show a significant rise compared to basal value prior to extubation; at extubation or any period post extubation. Maximum fall in heart was at 5th min after infusion and was 16 % and 27% respectively in group D5 and D1. But in control group, there was a significant rise in HR compared to basal value at extubation ($p<0.001$). This observation is in concurrence with the study done by Jain D et al[9], where the pulse rate in study group remained below the baseline value at all time intervals prior to and following extubation. In our study, the attenuation of HR was better in the higher dosage group (1μg/kg of Dexmedetomidine).

SBP, DBP values were significantly lower in Group D5 (Table 6 and Graph 2) compared to baseline values at all times from the time of Dexmedetomidine infusion to post extubation 60 minutes. This is in conjunction with the study conducted by Guler G et al[10] in which study group patients received 0.5μg/ kg of Dexmedetomidine and they did not observe any significant rise ($p<0.05$) in the blood pressure in Dexmedetomidine group throughout the study period.

However, blood pressure readings in Group D1 showed a BIPHASIC pattern in response to Dexmedetomidine infusion, with an initial increase starting at 1 minute of infusion lasting up to 5 minutes followed by a gradual decline prior to extubation. Infusion of 1μg/kg of Dexmedetomidine increased SBP, DBP by 19, 12 and 16% respectively. The transient increase in pressure peaked at around 5 minute after the start of infusion and was associated with reflex bradycardia at the same time (27% fall in HR from baseline). SBP, DBP readings at extubation and post extubation were below baseline values in Group D1. This is in accordance to study conducted by Bloor B C et al[11] who observed that healthy volunteers receiving higher dosage group Dexmedetomidine (1μg/kg and 2μg/kg) showed a biphasic response to the drug. But is in contrast to the study done by Jain D

et al[9]. The difference in results could be attributed to different anaesthetic technique employed by the authors who used Propofol infusion for maintenance of anaesthesia till the end of surgery.

The initial transient increase in BP is mediated by peripheral vasoconstriction (α 2B receptors); while the reduction in BP is mediated by both central and peripheral sympatholytic action. The incidence of biphasic response is influenced by drug dosing and rate of drug infusions with higher dosages (≥1μg/kg) and faster infusions resulting in greater incidence of biphasic response.

Guler G and Mizrak A et al[10] observed that time to extubation and emergence were prolonged significantly when compared to control group with $p<0.05$. (5.03± 2.3 vs 3.30± 1.3 minutes and 9.30± 2.9 vs 7.20± 2.7 minutes, respectively). This observation is in agreement with our study conducted, wherein time to extubation and emergence (i.e, interval between cut off of nitrous oxide to extubation and eye opening respectively) were significantly prolonged in Dexmedetomidine groups when compared to control group ($p <0.001$). Recovery times were significantly longer in higher dose group [Group D1 ($p<0.001$)] as compared to Group D5.

Sedation in our study was assessed using Ramsay Sedation Scale. Sedation score was significant post extubation for 15 minutes in Group D5 compared with control group. After this period sedation scores were comparable in both the groups. Where as in Group D1 the scores were significantly higher at all measurement points as compared to Group D5 and Group C.

Bradycardia was observed in 2 patients (7%) in Group D1 (1μg/kg) which responded to injection atropine. Seven patients in group D5 and eight in group D1 had hypotension. This is in conjunction with the observation made by Barkha Bindu et al[9]. Agitation was observed in 7 patients (23%) in Group C following extubation whereas none were agitated in Group D5 and D1. This is statistically and clinically significant ($p< 0.001$). Fall in SPO2 (<90%) was seen in 2 patients in Group D1. This observation is in conjunction with the study done by Guler G and Mizrak A et al[10]. Incidence of complications was thus higher in Group D1 as compared to Group D5.

Our study also had some limitations. We did not measure plasma catecholamine concentrations with which the pressor response best correlates. We also did not measure plasma Dexmedetomidine levels.

CONCLUSION

Intravenous Dexmedetomidine in the dose of 0.5 μg/ kg body weight and 1 μg/ kg body weight attenuates hemodynamic stress response to extubation, thereby facilitating smoother emergence from anaesthesia. But in the higher dosage group, the recovery times were significantly prolonged and sedation scores were significantly higher at all times. Therefore, we conclude that intravenous Dexmedetomidine in the dose of 0.5 μg/ kg body weight is better with less complication rates.

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