Original Research Article

Effect of two different doses of Dexmedetomidine on hemodynamic responses during tracheal extubation

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Abstract

Background: Haemodynamic responses to tracheal extubation have always been of importance to the anaesthesiologist. Different concentrations of dexmedetomidine ranging from 0.25 µg/kg to 1.0 µg/kg intravenously as a bolus, have been studied for attenuation of pressor responses to extubation and intubation. **Aim:** To study the effects of two different doses of dexmedetomidine on hemodynamic responses during tracheal extubation. **Material and Methods:** A total of 66 patients undergoing surgery under general Anaesthesia in our hospital were randomly divided into 2 groups: Group-D1: Dexmedetomidine (0.25 µg/kg) group –33 patients; Group-D2: Dexmedetomidine (0.5 µg/kg) group – 33 patients. **Results:** The heart rate was significantly lower in D2 group at 10 minutes after starting of study drug, during extubation and till 15 minutes after extubation ($p<0.05$). The SBP and DBP was significantly lower in D2 group during extubation and till 15 minutes after extubation ($p<0.05$). **Conclusion:** Dexmedetomidine in 0.5 mcg/kg dose is optimum to attenuate the extubation response as compared to low dose i.e., 0.25 mcg/kg.

Key Word: Dexmedetomidine, tracheal extubation, hemodynamic changes, dosage

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INTRODUCTION

Endotracheal intubation is frequently performed in the operating room to secure the airway in patients undergoing a surgical procedure under general anaesthesia. Laryngoscopy and tracheal intubation cause significant haemodynamic changes in apatient.[1] Tracheal extubation is also associated with haemodynamic changes due to reflex sympathetic discharge caused by epipharyngeal and laryngopharyngeal stimulation.[2] This increase in sympathoadrenal activity may result in hypertension, tachycardia, arrhythmias and increased myocardial oxygen consumption. These metabolic and cardiovascular responses are usually transitory, variable, and unpredictable. However, they may adversely affect the balance between myocardial oxygen supply and demand, causing significant myocardial ischemia and haemodynamic compromise. Thus, modulation of the post-operative sympathetic response may afford haemodynamic stability and decrease morbidity in high risk surgical patients with hypertension, myocardial insufficiency or cerebrovascular diseases.[3] Therefore, these haemodynamic responses to tracheal extubation have always been of importance to the anaesthesiologist. Dexmedetomidine is a selective adrenergic α2 agonist and has sedative, analgesic and anaesthetics paring effects and it decreases heart rate, blood pressure and circulating plasma catecholamines in a dose dependent fashion.[4-7]It sedates patients by decreasing central sympathetic activity and they are easily roused to full consciousness.[8] Lack of respiratory depression, arousable sedation and hemodynamic stability makes dexmedetomidine a better choice to attenuate the pressor response to extubation with an added advantage of preventing emergence delirium.[9,10] Different concentrations of dexmedetomidine ranging from 0.25 µg/kg to 1.0µg/kg intravenously as a bolus, have been studied for attenuation of pressor responses to extubation and intubation.[11-13]The present

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study was conducted to study the effects of two different doses of dexmedetomidine on hemodynamic responses during tracheal extubation.

MATERIAL AND METHODS

This prospective, randomized, double blind study was conducted on patients undergoing surgery under general anesthesia in our hospital.

Sample Size

From previous study[12] it was found that, mean and standard deviation of systolic BP after 1min of extubation in Group-1was 125+/-15.71 and mean and standard deviation of systolic BP after 1min of extubation in Group-2 was 134.9+/-12.61.So, sample size was calculated by the formula:$(n1=n2) = <(b1^2 + b2^2/k) (Z_{1-a/2} + Z_{1-b})>/delta^2$ where, b1 - std. deviation of group 1=15.71; b2 - std. deviation of group 2=12.61; delta - difference in group means =134.9-125=9.9 k=n1/n2 = 1 $Z_{1-a/2}$ = two sided z value=1.96; Z_{1-b} =power of study=1.84. So, sample size; n1=n2=33; Sample size of group1=D1= 33; Sample size of group2=D2= 33. Thus, after obtaining informed written consent from patients, patients wererandomly divided into 2 groups: Group-D1: Dexmedetomidine (0.25mcg/kg) group–33 patients Group-D2: Dexmedetomidine (0.5mcg/kg) group – 33 patients

Inclusion Criteria

- Patients with ASA grade I or II Either gender with age between 18-45 years
- Patients who give informed written consent.

Exclusion Criteria

Patients with:

- Allergy to adrenergic agonists.
- History of uncontrolled hypertension, Obesity.
- Heart block greater than first degree. History of uncontrolled hypertension.
- History of alcohol or drug abuse.
- Clinically significant neurologic, cardiovascular, renal, hepatic, gastrointestinal diseases.

METHODOLOGY

With a minimum fasting state of 6-8 hours before anesthesia, IV access was obtained and standard monitoring consisted of Electrocardiography (ECG), pulse Oximetry, Non-invasive Blood pressure (NIBP) and End tidal carbon dioxide monitoring (ETCO2). All patients were pre-oxygenated with 100% oxygen for 3 minutes and pre-medicated with injection ranitidine 1mg/kg; injection Ondansetron 4mg; injection Glycopyrrolate 5mcg/kg; injection Midazolam 0.03mg/kg and Injection Pentazocine 0.5mg/kg intravenously. They were induced with injection Propofol 2mg/kg and intubation facilitated with injection Atracurium 0.5mg/kg intravenously. Patients were maintained on66% nitrous oxide in oxygen and isoflurane 1%-2%. Atracurium was used for maintenance of muscle paralysis. In Group-D1 patients, Dexmedetomidine 0.25mcg/kg body weight diluted to 10 mlin normal saline was infused over 10 minutes, approximately 10 minutes prior to reversal. In Group-D2 patients, Dexmedetomidine 0.5 mcg/kg kg body weight diluted to 10ml in normal saline was infused over 10 minutes, approximately 10 minutes prior toreversal. At the beginning of closure of skin incision or approximately 10 minutes prior toreversal, isoflurane was discontinued which is followed by beginning of intravenous dexmedetomidine; and Nitrous oxide was discontinued at the end of completion of dressing. Residual neuromuscular blockade was reversed using injection neostigmine0.05mg/kg and injection glycopyrrolate 0.01mg/kg intravenously.

Patients were extubated when the following extubation criteria were fulfilled:

1) Sustained head lift for 5 seconds.
2) Sustained hand grip for 5 seconds.
3) Adequate level of consciousness.

All patients were given oxygen by face mask during the recovery period. Awakening time: Discontinuation of nitrous oxide to eye opening. Extubation time: completion of reversal to extubation. Values for HR, SBP, DBP and MAP was recorded just before (A0) and 1, 3, 5,10(A1, A3, A5, A10) min after the study drug administration and at extubation (E0) and 1, 3,5, 10, 15 min after extubation (E1, E3, E5, E10, E15).Respiratory rate, SpO 2 and airway responses like coughing, breath holding, laryngospasm or bronchospasm was recorded at extubation (E) and 1, 3, 5, 10 and 15 min after extubation (E1, E3, E5, E10, E15). At the end of extubation, quality of extubation was recorded with five-point scale.[7]After extubation, all these patients were observed for sedation by Modified Ramsey sedation score. Time taken for eye opening after Nitrous oxide is discontinued was recorded. Time taken for extubation, after completion of injection of neuromuscular reversal was recorded. Any change in Heart rate (HR) and blood pressure (BP) (±20% of pre drugadministration value), if occurred was recorded and treated with appropriate drugs, if required. Any other side-effect of study drugs if occurs, were also recorded.

Statistical Analysis

The quantitative data was represented as their mean ± SD. Categorical and nominal data was expressed in percentage. The t-test was used for analysing quantitative data, or else non parametric data was analysed by Mann Whitney test. Categorical data was analysed by using chi-square test. The significance threshold of p-value was set at <0.05. All analysis was carried out by using SPSS software version 21.

RESULTS

In present study, mean age of the study groups was 34.56 and 36.79 years in D1 and D2 group respectively (p-0.31). Overall out of 66 cases, there were 56.1% males and 43.9% females (p-1.0). Of the 66 cases, 51 (77.3%) belonged to ASA grade I and 15 (22.7%) belonged to ASA grade II (p-1.0). The mean duration of surgery was 120.57 minutes and 130.17 minutes in D1 and D2 group (p-0.14).

Table 1: Comparison of change in heart rate before and after extubation

Heart rate		D1		D2		
		Mean	SD	Mean	SD	p-value
Baseline	A0	83.2	11	82.93	15.25	0.50
Start of study drug infusion	A1	75.77	6.52	75.83	8.87	0.89
	A3	75.93	6.79	74.07	8.18	0.34
	A5	74.83	6.54	72.4	8.07	0.35
	A10	74.5	6.58	69.6	9.13	0.06
	A15	74.17	6.56	68.03	8.19	<0.05
At Extubation	E0	73.4	6.74	68.93	7.43	<0.05
	E1	73.3	6.88	67.33	7.81	<0.05
After extubation	E3	72.97	7.16	67.17	7.61	<0.05
	E5	72.33	6.44	66.87	7.13	<0.05
	E10	71.67	6.98	65.63	8.06	<0.05
	E15	71.37	7.05	64.67	8.14	<0.05

The baseline mean heart rate was comparable between study groups at the baseline (p-0.5). The heart rate was significantly lower in D2group at 10 minutes after starting of study drug, during extubation and till 15 minutes after extubation (p<0.05).

Table 2: Comparison of systolic blood pressure before and after extubation

SBP		D1		D2		
		Mean	SD	Mean	SD	p-value
Baseline	A0	122.23	9.94	123.83	13.55	0.60
Start of study drug infusion	A1	119.63	7.85	120.17	9.85	0.82
	A3	119.07	7.7	117.63	9.05	0.51
	A5	118.93	7.63	116.63	9.28	0.30
	A10	118.67	8.21	115.37	10.67	0.19
	A15	117.03	8.06	114.2	9.43	0.22
At Extubation	E0	116.57	7.55	111.9	8.52	<0.05
	E1	116.27	7.61	110.97	8.67	<0.05
After extubation	E3	115.9	7.74	110.13	9.35	<0.05
	E5	115.4	7.86	109.13	9.19	<0.05
	E10	113.97	8.44	109.03	7.23	<0.05
	E15	113.57	8.44	109.77	8.25	<0.05

Baseline Mean systolic blood pressure was comparable between study groups at the baseline (p-0.6). The SBP was significantly lower in D2 group during extubation and till 15 minutes after extubation (p<0.05).

Table 3: Comparison of diastolic blood pressure before and after extubation

DBP		D1		D2		
		Mean	SD	Mean	SD	p-value
Baseline	A0	75.07	7.03	76.3	9.49	0.57
Start of study drug infusion	A1	74.2	6.32	73.9	8.45	0.88
	A3	73.63	6.35	73.33	8.84	0.88
	A5	73.03	6.51	71.23	9.12	0.38
	A10	72.4	6.54	70.7	8.18	0.38
	A15	71.77	6.53	69.13	8	0.17
At Extubation	E0	71.7	6.15	69.27	8.23	<0.05
	E1	71.67	6.5	68.5	7.91	<0.05
After extubation	E3	71.27	6.23	67.23	7.57	<0.05
	E5	70.73	6.62	67.1	7.64	<0.05
	E10	70.3	6.23	66.13	7.91	<0.05
	E15	70.17	7.05	66.2	8.39	<0.05

Mean diastolic blood pressure was comparable between study groups at the baseline (p-0.57). The DBP was significantly lower in D2 group during extubation and till 15 minutes after extubation (p<0.05).

Table 4: Comparison of mean arterial pressure before and after extubation

MAP		D1		D2		
		Mean	SD	Mean	SD	p-value
Baseline	A0	90.79	7.01	92.14	10.03	0.55
Start of study drug infusion	A1	89.34	6.14	89.32	8.05	0.99
	A3	88.78	5.98	88.1	7.69	0.71
	A5	88.33	6.11	86.37	8.23	0.30
	A10	87.82	6.16	85.59	7.71	0.22
	A15	86.86	6.21	84.16	7.42	0.13
At Extubation	E0	86.66	5.76	83.48	7.09	<0.05
After extubation	E1	86.53	6.1	82.66	6.97	<0.05
	E3	86.14	5.87	81.53	6.89	<0.05
	E5	85.62	6.11	81.11	7.01	<0.05
	E10	84.86	6.27	80.43	6.7	<0.05
	E15	84.63	6.69	80.72	7.18	<0.05

Baseline Mean arterial pressure was comparable between study groups at the baseline (p-0.57). The MAP was significantly lower in D2 group during extubation and till 15 minutes after extubation (p<0.05).

Table 5: Comparison of SpO2 before and after extubation

SpO2		D1		D2		
		Mean	SD	Mean	SD	p-value
Baseline	A0	98.67	0.84	98.67	0.84	1.00
Start of study drug infusion	A1	98.73	0.74	98.67	0.8	0.68
	A3	98.73	0.64	98.87	0.73	0.32
	A5	98.83	0.79	98.67	1.03	0.67
	A10	98.87	0.51	98.67	0.84	0.35
	A15	98.7	0.95	98.73	0.87	0.99
At Extubation	E0	98.73	0.87	98.7	1.09	0.82
After extubation	E1	98.97	0.77	98.63	0.93	0.13
	E3	98.9	0.85	98.8	0.66	0.51
	E5	98.87	0.9	98.87	0.82	0.80
	E10	98.5	0.82	98.57	0.97	0.69
	E15	98.6	0.97	98.63	0.67	0.77

No difference was observed between the study groups with respect to partial pressure of oxygen at baseline, during extubation and post-extubation.

Table 6: Comparison of quality of extubation

Quality of Extubation	Group		Total
	D1	D2	
Smooth (Grade 1/2)	27	31	58
	81.8%	93.9%	87.9%
Rough (Grade 3/4)	6	2	8
	18.2%	6.1%	12.1%
Poor (Grade 5)	0	0	0
	0.0%	0.0%	0.0%
Total	33	33	66
	100%	100%	100%
p- value - 0.25			

Smooth extubation was reported in 93.9% cases of D2 group as compared to 81.8% in D1 group. Rough experience was seen in 18.2% cases of D1 group as compared to 6.1% in D2 group. The difference was statistically non-significant (p-0.25). Post-extubation, mean sedation score were observed to be comparable in both the groups till 30 mins. Mean time to awaken was significantly higher in D2 group (310.2 sec 179.7 sec; p<0.05) while time for extubation was significantly lower (136.7 vs 224.5 sec; p<0.05). Incidence of adverse reactions like bradycardia and nausea was comparable between both groups with 4 and 6 cases of bradycardia and 2 and 3 cases of vomiting being observed in group D1 and D2 respectively (p-0.73; 1.0).

DISCUSSION

Attenuation of the stress response during laryngoscopy, intubation, surgery and extubation has been one of the most researched topics in anesthesia, but only a few methods have been proved satisfactory. Numerous drugs and their combinations, in varying doses have been tried, to suppress the hemodynamic stress response to intubation and extubation. Dexmedetomidine is a selective adrenergic α2 agonist with sedative, analgesic and anesthetic sparing effects and it decreases heart rate, blood pressure and circulating plasma catecholamines in a dose dependent fashion.[4-7] Different concentrations of dexmedetomidine ranging from 0.25 µg/kg to 1.0 µg/kg intravenous as a bolus, have been studied for attenuation of pressor responses to extubation and intubation.[12,13]A total of 66 patients undergoing surgery under general Anaesthesia in our hospital were randomly divided into 2 groups: **Group-D1**: Dexmedetomidine (0.25 µg/kg) group – 33 patients; **Group-D2**: Dexmedetomidine (0.5 µg/kg) group – 33 patients. Both the groups were comparable with respect to baseline parameters like age, gender and ASA grade. The duration and nature of surgery, and time interval from study drug infusion to extubation were also comparable, as there was no statistical difference between the two groups (p value > 0.05). Hemodynamic changes associated with tracheal extubation may be due to pain, emergence from anesthesia, changes in plasma catecholamine levels or tracheal irritation. These hemodynamic changes normally do not constitute a major problem; but could be deleterious in patients with comorbidities. Dexmedetomidine activates the medullary vasomotor centre receptors, reducing central sympathetic outflow, resulting in decreased heart rate and blood pressure. Different studies have shown desirable as well as undesirable hemodynamic changes with doses ranging from 0.25µg/kg -1.0µg/kg as intravascular infusion.[14] In present study, we observed that mean heart rate and blood pressure readings was comparable between study groups before injection of study drug. However, the heart rate was significantly lower in D2 group (0.5 µg/kg) as compared to D1 group (0.2 µg/kg)at 10 minutes after starting of injection, during reversal and till 15 minutes after extubation ($p<0.05$). The systolic and diastolic blood pressure were also significantly lower in D2 (0.5 µg/kg) group before extubation and till 15 minutes after extubation ($p<0.05$). Antony D *et al*.[14]also observed a significant reduction in HR from 5 minutes after starting infusion in both 0.5 µg/kg and 1.0 µg/kg groups; with attenuation of rise in HR during extubation. Both doses effectively controlled responses in HR to extubation but the mean HR was significantly lower in higher dose group (1µg/kg) from 5 minutes after extubation till 30 minutes when compared with low dose group (0.5µg/kg). The mean SBP, DBP and MAP were significantly stable during extubation with both 0.5 µg/kg and 1.0 µg/kg groups when compared with the placebo group. They concluded that both doses were found to be effective in attenuating the hemodynamic responses to extubation. In present study, we observed better hemodynamic control with 0.5 µg/kg dose as compared to a lower dose of 0.25 µg/kg without any significant hypotension episodes. Ibacache *et al*.[15] in their study also reported no reduction in HR and blood pressure with lower dose of dexmedetomidine (0.3µg/kg). The observations in the present study are also comparable with the results of the study done by Guler G *et al*[16] suggesting a less significant increase in HR, SBP and DBP in response to extubation with 0.5µg/kg single bolus dose of, given as a slow IV infusion 5 minutes before the end of the surgery. Rao S *et al*.[17] also observed mean heart rate, systolic and diastolic blood pressure and mean arterial pressure were increased less in dexmedetomidine group. Similar findings have been made by Shruthi AH *et al*[18] where 0.5 µg/kg intravenous bolus infusion of dexmedetomidine given at the end of surgery effectively attenuated airway reflex responses to extubation maintaining good hemodynamic stability. Incidence of adverse reactions like bradycardia and nausea was comparable between both groups with 4 and 6 cases of bradycardia and 2 and 3 cases of vomiting being observed in group D1 and D2 respectively (p-0.73; 1.0). None of the complications were associated with hemodynamic instability and required any treatment. Antony D *et al*.[14] observed significant bradycardia in patients who received dexmedetomidine, the incidence being higher with 1µg/kg dose. None of the cases were hemodynamically unstable and required no treatment. The incidence of hypotension was 10% and 13.3% in patients who received dexmedetomidine 1µg/kg and 0.5µg/kg respectively; without anyone in the placebo group developinghypotension. In present study, none of the cases developed hypotension while bradycardia incidence was comparable between the groups. Rao S *et al*.[17] in their study observed bradycardia in only 1 patient (3%) in study group (0.5 µg/kg dexmedetomidine). Guler G *et al*[16] also observed that 0.5µg/kg of dexmedetomidine provides better hemodynamic stability compared to placebo without adverse effects like hypotension or bradycardia.

CONCLUSION

In present study, we observed that higher dose dexmedetomidine group i.e. 0.5mcg/kg, there was significant fall of blood pressure and heart rate during the procedure ascompared to the low dose dexmedetomidine group (0.25 mcg/kg) with no difference in theincidence of

adverse reactions. The extubation was relatively more smooth and time forextubation was less in the higher dose group (0.5mcg/kg) as there was better post-operative arousable sedation. We thus conclude that 0.5 mcg/kg dose of Dexmedetomidine is optimum to attenuate the extubation response as compared to low dose i.e. 0.25 mcg/kg.

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