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TO STUDY THE EFFECT OF DEXMEDETOMIDINE ON THE HEMODYNAMIC AND RECOVERY RESPONSES DURING TRACHEAL EXTUBATION

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ABSTRACT

INTRODUCTION: Marked increases in arterial blood pressure and heart rate occur frequently at the time of extubation. These effects are alarming and, may cause detrimental increases in intracranial and intraocular pressure after neurologic and ophthalmic surgery. Dexmedetomidine is a highly selective α-2 adrenoceptor agonist that has sedative, anxiolytic ,hypnotic, analgesic and sympatholytic properties , shows a high ratio of specificity for the α2 receptor (α2/α1 1600:1) making it a complete α2 agonist and has a short half-life of 2-3 hours.

MATERIALS AND METHODS: The study was conducted on sixty patients (thirty in each group) undergoing surgery under general anaesthesia Patients in group A recieved Intravenous infusion of dexmedetomidine 0.5 mcg/kg body weight in 100ml normal saline.Patients in group B recieved Intravenous placebo with 100 ml normal saline 15 minutes prior to extubation.

RESULTS: dexmedetomidine as given in group A before extubation attenuates the hemodynamic response, enables smooth extubation and provides adequate sedation ,these results were statistically significant ($p<0.005$) Incidence of bradycardia and hypotension was seen in group A but was statistically insignificant.

KEYWORDS : Alpha-2 adrenoceptor agonist, dexmedetomidine, extubation, haemodynamics

INTRODUCTION

The tracheal tube (extubation) is removed when it is no longer needed for airway protection . Extubation is performed at different depths of anesthesia, Cardiovascular complications include arterial hypertension, tachycardia, and dysrhythmias. These effects are normally transient, however, they may cause detrimental increases in intracranial and intraocular pressure after neurologic and ophthalmic surgery. Many techniques can reduce the incidence of these adverse effects. Presynaptic α2 receptors may be of the greatest clinical importance, because they regulate the release of norepinephrine and adenosine triphosphate by negative feedback mechanism . Receptors for α2 are found in the peripheral and central nervous systems. Dexmedetomidine is a highly selective α-2 adrenoceptor agonist that has sedative, anxiolytic ,hypnotic, analgesic and sympatholytic properties. It exerts its sedative and anxiolytic effects through activation of α2- adrenoreceptors in the locus ceruleus, a site of noradrenergic innervation in the CNS and analgesic action at α2 receptors in the locus ceruleus and spinal cord , has minimal effects on ventilation. In CNS, activation of α2-adrenoreceptors leads to a reduction in sympathetic outflow and an increase in vagal activity, all these properties make it very suitable for use in attenuating hemodynamic responses during extubation.

MATERIALS AND METHOD

The study was conducted on sixty patients admitted to Dr. DY Patil medical college, Pune (thirty in each group A and B) in ASA grade I and II, of either sex between 18-65 years of age, undergoing elective surgery under general anaesthesia. Prior permission of institutional ethics committee was obtained. Informed written consent obtained and all patients were subjected to pre anaesthetic evaluation. Anesthetic technique employed using Propofol (2 mg/kg), pentazocine (0.3 mg/kg), vecuronium (0.08 mg/kg) nitrous oxide-oxygen (50% each) and isoflurane (0.6-1%). Standard monitoring with electro cardiography , pulse oximetry and noninvasive blood pressure was done. About fifteen minutes before the estimated time of end of surgery, inhalational agent was cut off and patients in each group received the specified solution intravenously over fifteen minutes. Group A received 0.5 mcg/kg body weight of dexmedetomidine in 100 ml normal saline I.V. and Group B 100 ml normal saline I.V. Patients were reversed with neostigmine 0.05 mg/kg and glycopyrrolate 0.008 mg/kg and extubated. Heart rate,mean arterial pressure were recorded at extubation and thereafter at 1, 5, 10 and 15 minutes. Occurrence of any event like laryngospasm, bronchospasm, desaturation, respiratory depression, vomiting, hypotension, bradycardia or undue sedation was noted.

RESULTS

On evaluation of age the mean was 42.80+12.88 in group A and in group B was 40.83+17.99 it was analyzed quantitatively within groups. The Z value was 0.48, which was statistically not significant (P value >0.05). Overall among 60 cases 37 were males and 23 were females.(P >0.05) shows statistically not significant. Patients belonging to Group A, the mean pulse rate during extubation was 79.3±11.17 beats/minute and at 1, 5,10 and 15 minutes after extubation were 76.4±9.21, 69.33±6.98, 66.47±6.45 and 62.37±4.88 respectively, in patients of group B, during and 1, 5, 10 and 15 minutes after extubation are 91.07±13.35, 95.70±13.50, 94.93±12.23, 90.90±11.16 and 87.60±11.60 respectively. This shows that there is significant difference in the hemodynamic parameters during and at 1,5,10 and 15 minutes after extubation which implies that sympathetic responses were lesser in group A as compared to patients of group B.($p<0.005$)

Table 1: Comparison of pulse rate in group A and group B

PR/min	Group A (n=30)		Group B (n=30)		Z Value	P Value
	Mean	SD	Mean	SD		
Start of drug	81.53	10.88	86.8	10.83	1.88	>0.05
During extubation	79.63	11.177	91.07	13.352	3.59	<0.001
1min post extubation	76.40	9.212	95.70	13.501	6.47	<0.0001
5min post extubation	69.33	6.989	94.93	12.23	9.95	<0.0001
10min post extubation	66.47	6.458	90.90	11.161	10.38	<0.0001
15min post extubation	62.37	4.888	87.60	11.069	11.42	<0.0001

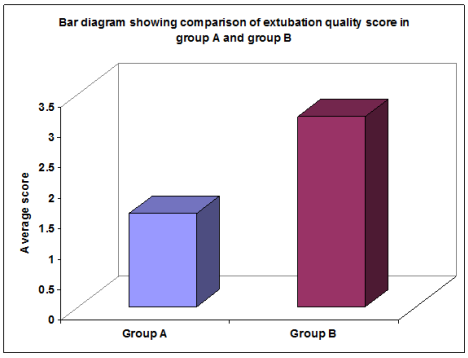
In Group A, the mean arterial blood pressure during extubation was 92.07±9.58. The values of mean blood pressure at 1, 5,10 and 15 minutes after extubation were 86.17±9.06, 86.13±7.01, 80.93±6.81 and 78.30±5.60 respectively. These results indicate that there is suppression of hemodynamic responses when the study drug is given 15 minutes prior to extubation. The mean blood pressure , in patients of group B, during and 1, 5, 10 and 15 minutes after extubation are , 103.03±8.42, 103.73±6.99, 100.07±7.50, 95.90±7.68 and 92.83±8.54 respectively. This shows there is significant difference in this hemodynamic parameter during and at 1,5,10 and 15 minutes after extubation which implies that sympathetic responses were lesser in group A as compared to patients of group B.

Table 2: Comparison of MAP in group A and group B

MAP (mmHg)	Group A (n=30)		Group B (n=30)		Z Value	P Value
	Mean	SD	Mean	SD		
Start of drug	105.23	8.520	104.10	8.43	0.52	>0.05
During extubation	92.07	9.584	103.03	8.422	4.71	<0.0001

1min post extubation	86.17	9.063	103.37	6.990	8.23	<0.0001
5min post extubation	86.13	7.011	100.07	7.501	7.43	<0.0001
10min post extubation	80.93	6.817	95.90	7.689	7.98	<0.0001
15min post extubation	78.30	5.603	92.83	8.546	7.79	<0.0001

In patients belonging to Group A, the mean extubation quality score was 1.53+0.57 and in group B, it was 3.13+0.57. There is a statistically significant difference in the scores in the two groups indicating that the quality of extubation was better in group A.



Bradycardia was seen in 2 patients, hypotension in 1 and vomiting also seen in 1 patient, and it proved to be statistically not significant.

Table 3: Side effects distribution of cases in group A and group

Side effects	Group A (n=30)	Group B (n=30)	Z Value	P Value
Bradycardia	2 (6.67)	0	1.46	>0.05
Hypotension	1 (3.33)	0	1.02	>0.05
Vomiting	1 (3.33)	0	1.02	>0.05

DISCUSSION

Extubation can be associated with several complications like coughing and respiratory and hemodynamic alterations. Dexmedetomidine has been successfully used to attenuate the hemodynamic responses to tracheal intubation. The dose of dexmedetomidine ranges from 0.5-1 mcg/kg. A pilot study, using three different doses (0.5 mcg/kg, 0.75 mcg/kg and 1 mcg/kg) of dexmedetomidine, was conducted. However in our study dose of 0.5 mcg/kg was used to attenuate the hemodynamic responses to tracheal extubation, although there is evidence in favour of 0.75 mcg/kg as well. Dexmedetomidine activates receptors in the medullary vasomotor center, reducing norepinephrine turnover and decreasing central sympathetic outflow, resulting in alterations in sympathetic function and decreased heart rate (HR) and blood pressure(BP).In the present study, the hemodynamic parameters in the study group were significantly stable during extubation when compared to the placebo group. Dexmedetomidine (plasma concentrations in the range of 0.18 to 0.35 ng/ml) attenuates the increase in HR and plasma norepinephrine concentrations during emergence from anaesthesia. Central stimulation of parasympathetic outflow and inhibition of sympathetic outflow from the locus coeruleus in the brainstem plays a prominent role in the sedation and anxiolysis produced by dexmedetomidine. Decreased noradrenergic output from the locus coeruleus allows for increased firing of inhibitory neurons including the g-amino butyric acid system resulting in anxiolysis and sedation. The activation of a2 adrenoceptors, imidazoline-preferring receptors, or both in the ventrolateral medulla and especially in the solitarius nucleus tract by dexmedetomidine causes bradycardia. Guler et al in 2005 conducted a study on single-dose dexmedetomidine attenuates airway and circulatory reflexes during extubation and he found that the increase in HR associated with emergence from anaesthesia were attenuated by i.v.dexmedetomidine 0.5 mg/kg. Bindu, et al in 2013 did a study on dexmedetomidine to prevent response to tracheal extubation using an intravenous infusion of dexmedetomidine 0.75 mcg/kg or placebo, over 15 minutes before anticipated time of end of surgery, they observed a statistically significant difference ($P < 0.05$) in HR between the two groups from 5 minutes after starting administration of the agent till 20 minutes after extubation. In the study conducted by Sharma, et al the MAP increased for the initial 3 minutes of drug administration in dexmedetomidine group. However, dexmedetomidine attenuated the increase in blood pressure to a greater degree than lignocaine.

With reference to the above studies and our observations we concluded that dexmedetomidine given as i.v. infusion15 minutes prior to extubation in a dose of 0.5 mcg/kg attenuates hemodynamic responses to extubation.

CONCLUSION

To conclude, use of dexmedetomidine before extubation attenuates the hemodynamic response to extubation. It enables smooth extubation of the trachea and provides adequate sedation postoperatively. Dexmedetomidine increases the incidence of bradycardia and hypotension, but does but does not cause side effects like respiratory depression, laryngospasm, bronchospasm, undue sedation and desaturation.

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