

Effect Of Dexmedetomidine On Stress Response To Extubation

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Citation

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Abstract

Laryngoscopy as well as tracheal intubation cause significant changes in the hemodynamics of the patient.⁽¹⁾
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).These responses may produce myocardial ischemia or infarction in susceptible patients. Various agents like lidocaine and esmolol have been proved to attenuate these responses(³
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)We conducted a double blind study to examine the role of Dexmedetomidine on hemodynamic changes during extubation of trachea.

INTRODUCTION

Laryngoscopy as well as tracheal intubation cause significant changes in the hemodynamics of the patient.⁽¹⁾A similar set of hemodynamic derangements have been noticed by various workers during tracheal extubation(²).These responses may produce myocardial ischemia or infarction in susceptible patients. Various agents like lidocaine and esmolol have been proved to attenuate these responses(³).Dexmedetomidine, an α_2 agonist has been successfully used for attenuating the stress response to laryngoscopy.⁽⁴⁾We conducted a double blind study to examine the role of Dexmedetomidine on hemodynamic changes during extubation of trachea.

METHODS

After being approved by the board of studies, informed consent was obtained from 30 ASA I and II patients scheduled for elective lower abdominal infraumbilical surgery. All the patients were randomly divided into 2 groups according to (chit in a box technique).They were premedicated with inj. metaclopramide 0.15mg/kg, inj. midazolam 0.025mg/kg and inj. Fentanyl 2 μ g/kg. After preoxygenation for 3 minutes, all patients were induced with injection propofol 2mg/kg. Relaxation was achieved with

inj. vecuronium bromide 0.1mg/kg. Following the placement of endotracheal tube, patients were maintained on 66%N₂O inO₂ with vecuronium bromide and propofol infusion in step down fashion(10mg/kg for 15 minutes, followed by 8mg/kg and thereafter at 5-6mg/kg) 10 minutes prior to administration of reversal the study drug and the placebo were given according to the group allocation

Group I (n=15) received a bolus of dexmedetomidine, 1 μ g/kg diluted to 10 ml in normal saline, over 10 min

Group II (n=15) received a bolus of 10 ml normal saline over 10 min

Pulse and systolic blood pressure were monitored at the start of bolus drug injection and subsequently at 3 minutes, 5 minutes, 7minutes and 10minutes.Residual neuromuscular blockade was reversed with inj.neostigmine(0.04mg/kg) and inj. atropine(0.02mg/kg).On achieving the TOF ratio of over0. 8, extubation of the trachea was performed. Pulse and blood pressure were recorded following administration of reversal agent, post extubation for 15 minutes, every 5 minutes. after extubation and thereafter every 5 minutes for 15 minutes. A fall in pulse rate <50bpm was treated by a rescue dose of 0.06mg atropine. Data in the table are expressed as mean \pm S.D.Data was analysed statistically using

paired t-test for comparison within group data and unpaired t-test for comparison between group data. $p < 0.05$ was considered significant.

RESULTS

Patients in group I were comparable with group II with regards to age (47 ± 3 vs 50 ± 4 yrs respectively) and weight (55 ± 2 vs 52 ± 4 kg respectively). In group I there was a significant fall in the pulse rate 7-10 minutes after the start of the bolus dose of dexmedetomidine ($p < 0.05$), but no intervention was required as this fall in pulse rate was transient and did not affect the blood pressure. The pulse rate in group I remained below the pre-dexmedetomidine values (baseline value), at all time intervals following extubation. On the contrary pulse rate rose significantly ($p < 0.05$) in group II following extubation. There was no significant change ($p < 0.05$) in the systolic blood pressure in group I throughout the study period. None of the patients were sedated after receiving $1 \mu\text{g/kg}$ of dexmedetomidine bolus dose.

Figure 1

Table

Time	0min	3min	5min	7min	10min	PR	PE	5min	10min	15min
Pulse (bpm)	90.46 ± 5.6	78.5 ± 7.7	75.5 ± 6.1	70.6 ± 7.3	66.13 ± 6.7	78.8 ± 7.6	84.0 ± 8.1	78.4 ± 8.1	78.5 ± 8.7	80.4 ± 9.2
SBP (mmHg)	127.3 ± 12.3	122.4 ± 10.5	121.4 ± 10.1	119.6 ± 10.3	120.8 ± 10.6	128 ± 10.8	133 ± 11.2	129.2 ± 9.3	127.2 ± 11.1	123.2 ± 8.4

Figure 2

Table 2

Time	0 min	3min	5min	7min	10min	PR	PE	5min	10min	15min
Pulse (bpm)	88.1 ± 7	87.8 ± 6.9	86.8 ± 7.3	86.5 ± 6.5	87.9 ± 6.16	108.2 ± 6	126.2 ± 9.7	99.4 ± 9.5	94.6 ± 8.9	90.7 ± 8.5
SBP (mmHg)	124.6 ± 10.8	122.9 ± 9.3	123.6 ± 10.7	122 ± 9.2	123.3 ± 9.2	137.2 ± 10.19	156.9 ± 12	130 ± 9.9	125.2 ± 10.8	124.8 ± 10.3

Recovering from anaesthesia often results in elevating

catecholamine concentration following withdrawal of anaesthetics at the culmination of surgery.

This response is further aggravated by the laryngeal manipulations occurring at the time of extubation.

Alpha 2 agonists decrease the sympathetic outflow and noradrenergic activity, thereby counteracting the haemodynamic fluctuations occurring at the time of extubation due to increased sympathetic stimulation.

Dexmedetomidine is a supraselective newer prototype of alpha 2 agonist. Various studies have been done to study the anxiolytic and analgesic properties of dexmedetomidine. In one study it was successfully used to blunt the stress response to laryngoscopy. In our study we made use of these properties of dexmedetomidine for providing a smooth transition from the pre-extubation to the post-extubation phase by minimizing the haemodynamic fluctuations.

CONCLUSION

A bolus dose of DEX $1 \mu\text{g/kg}$ over 10 minutes, prior to administration of reversal provided hemodynamic stability associated with extubation. This can prove beneficial for cardiac patients where the stress response to extubation is highly undesirable.

References

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