Effect Of Dexmedetomidine On Stress Response To Extubation
D Jain, R Khan, M Maroof

Citation

Abstract
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 extubation2. These responses may produce myocardial ischemia or infarction in susceptible patients. Various agents like lidocaine and esmolol have been proved to attenuate these responses3. Dexmedetomidine, an ß2 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.

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 extubation1. These responses may produce myocardial ischemia or infarction in susceptible patients. Various agents like lidocaine and esmolol have been proved to attenuate these responses3. Dexmedetomidine, an ß2 agonist has been successfully used for attenuating the stress response to laryngoscopy4. 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 2µg/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, 1µg/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, 7 minutes and 10 minutes. Residual neuromuscular blockade was reversed with inj. neostigmine(0.04mg/kg) and inj. atropine(0.02mg/kg). On achieving the TOF ratio of over 0.8, 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±S.D. Data was analysed statistically using...
Effect Of Dexmedetomidine On Stress Response To Extubation

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 1μg/kg of dexmedetomidine bolus dose.

Figure 1

Table

Table 2

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 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 1μg/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

Author Information

Divya Jain  
Assistant Professor, Department of Anaesthesiology, Maulana Azad Medical College

R.M. Khan  
Professor, Department of Anaesthesiology, Jawaharlal Nehru Medical college, AMU

M. Maroof  
Professor, Department of Anaesthesiology, University of Chapel Hill