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DEXMEDETOMIDINE IN ATTENUATING EXTUBATION RESPONSE IN CHRONIC SMOKERS.

Anaesthesiology

Dr Aryan Guleria MD Anaesthesia, ZH Dharamshala, Himachal Pradesh, India
Dr Brish Bhanu Bhardwaj* MD Pathology, CH Nurpur, Himachal Pradesh, India *Corresponding Author

KEYWORDS

BACKGROUND
Smokers, because of their exaggerated upper airway refexes, have an increased risk of developing complications during extubation which may lead to laryngospasm, hypoventilation, hypoxemia, and reintubation. Tracheal extubation is often accompanied by a rise in plasma catecholamines leading to undesirable events which include, hypertension, and tachycardia.

METHODS
The prospective randomized triple-blinded controlled trial included 30 adults, current smokers aged between 40 and 60 years that were found to have used a minimum of 100 cigarettes. They currently smoke cigarettes daily or intermittently. These patients had an American Society of Anesthesiologists (ASA) physical status II and were kept for total extraperitoneal laparoscopic inguinal hernia repair under general anesthesia.

We obtained fresh written informed consent before surgery. On the night before surgery, tablet alprazolam 0.5 mg was given, and the patients remained fasting for 8 h. After premedication, smoking was not allowed. Patients under study were divided into three groups (n =10). The drug was prepared as an infusion with normal saline with a total volume of 10 ml. Group A received 0.5 µg/kg of dexmedetomidine in normal saline, group B received 0.75 µg/kg and group C 1 µg/kg.

In the OT an 18G i.v. cannula was secured, i.v. fluid was started and standard monitoring was attached. Pre oxygenation was done for 3 minutes.

Premedication was done using midazolam with fentanyl and patient was induced using propofol with Atracurium and an endotracheal tube was placed. Anesthesia was maintained using Oxygen:Nitrous: Isoflurane mixture along with intermitent doses of atracurium.

Isofurane was discontinued at the beginning of skin closure, and dexmedetomidine infusion was started only when patients showed initial signs of respiratory effort. In group A, infusion of 0.5 µg/kg dexmedetomidine diluted to 10 ml in normal saline was started and given over 10 min using an infusion pump. In group B, 0.75 µg/kg and in group C 1 µg/kg dose of dexmedetomidine was used respectively for infusion. After dexmedetomidine was infused over 10 min, all patients were extubated within 5 min of discontinuing the infusion. Before extubation, nitrous oxide was discontinued, and muscle relaxation was reversed using neostigmine and glycopyrolate.

The primary outcome included quality of extubation assessed by 5-point scale and any adverse event like hypotension, hypertension and bradycardia was noted.

RESULTS
In comparing group A with group B and group C, the quality of extubation was found to be signifcantly poorer in group A (P = 0.001).
On comparing the heart rate in various groups, we found that it was comparable in all three groups at the start of dexmedetomidine infusion. However, we found that the heart rate was lower in group B and group C as compared to group A. Although when a comparison was made between group B and group C, the heart rate was lower in group C.

When we compared the SBP, DBP, and MAP between group A and group B, it was found to be statistically insignifcant. The SBP, DBP, and MAP remained comparable between group B and group C from the start of infusion up to extubation.

In comparison, the mean SpO2 was comparable at the start of dexmedetomidine infusion up to extubation in all three groups.

The sedation score in group A was found to be the least of all the three groups. It was found to be the highest in group C patients.

DISCUSSION
In our study, the main objective was to fnd the optimal dose of dexmedetomidine which can be used to attenuate the extubation response in chronic smokers as we are well aware of the exaggerated upper airway refex among smokers. The optimal dose is defned as the one which provided stable hemodynamics both during and postextubation while improving the extubation quality.

Talke et al. (Talke et al. 2000)[1] concluded that the hemodynamic stress response to extubation was less, and the quality of extubation was improved when dexmedetomidine was used. Luthra et al. (Luthra et al. 2017)[2] and Khan et al. (Khan et al. 1999)[3] in their study using dexmedetomidine infusion (0.2 µg/kg/h and 0.4 µg/kg/h) found a signifcant reduction in MAP and HR. Similarly, in our study, dose of 1 µg/kg (group C) had a higher incidence of bradycardia.

It is important to maintain MAP within the accepted limits as chronic smokers are often associated with higher autoregulation values of cerebral and renal perfusion (Varon and Marik 2008)[4].

It was observed in our study that 60% of patients in group C had no coughing during extubation in comparison with 46.6% and 16.6% of patients in group B and group A.

Another study by Aksu et al. (Aksu et al. 2009)[5] done in patients undergoing rhinoplasty concluded that dexmedetomidine was better than fentanyl in controlling airway refex responses encountered during extubation and maintaining hemodynamic stability.

Fan et al. (Fan et al. 2015)[6] concluded that 0.7 µg/kg of dexmedetomidine resulted in smoother extubation when compared to a 0.5 µg/kg dose.

Bindu et al. (Bindu et al. 2013)[7] used a 0.75 µg/kg infusion of dexmedetomidine 15 min before extubation and found that it facilitates smooth extubation.

CONCLUSION
In our study, we conclude that dexmedetomidine when used in a dose of 0.75 µg/kg and 1 µg/kg more efectively attenuates the hemodynamic response to extubation. However, a higher incidence of bradycardia was recorded using a 1 µg/kg dose of dexmedetomidine. Hence, we conclude that dexmedetomidine intravenous infusion at the rate of 0.75 µg/kg can be safely started 10 min before extubation. This reduces the extubation stress response in chronic smokers while providing stable hemodynamics during and after extubation.

REFERENCES
1. Talke P, Chen R, Thomas B, Aggarwall A, Gottlieb A, Thorborg P, et al. The hemodynamic and adrenergic effects of perioperative dexmedetomidine infusion after vascular surgery. Anesth Analg. 2000;90:834-9.
2. Luthra A, Prabhakar H, Rath GP. Alleviating stress response to tracheal extubation in

neurosurgical patients: A comparative study of two infusion doses of dexmedetomidine. J Neurosci Rural PrACt 2017;8:49-56.
3. Khan ZP, Munday IT, Jones RM, Thornton C, Mant TG, Amin D. Effects of dexmedetomidine on isoflurane requirements in healthy volunteers. 1: Pharmacodynamic and pharmacokinetic interactions. Br J Anaesth. 1999;83: 372-80.
4. Varon J, Marik PE. Perioperative hypertension management. Vasc Health Risk Manag 2008;4:61527.
5. Aksu R, Akin A, Bicer C, Esmaoglu A, Tosun Z, Boyaci A. Comparison of the effects of dexmedetomidine versus fentanyl on airway reflexes and hemodynamic responses to tracheal extubation during rhinoplasty. Curr Ther Res Clin Exp. 2009;70:209–20.
6. Fan Q, Hu C, Ye M, Shen X. Dexmedetomidine for tracheal extubation in deeply anesthetized adult patients after otologic surgery: a comparison with remifentanil. BMC Anesthesiol. 2015;15:106.
7. Bindu B, Pasupuleti S, Gowd UP, Gorre V, Murthy RR, Laxmi M B. A double blind, randomized, controlled trial to study the effect of dexmedetomidine on hemodynamic and recovery response during tracheal extubation. J Anaesthesiol Clin Pharmacol. 2013;29:162-7.