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EFFECT OF DEXMEDETOMIDINE AND MAGNESIUM SULPHATE ON HAEMODYNAMIC AND RECOVERY RESPONSES DURING TRACHEAL EXTUBATION : A PROSPECTIVE RANDOMISED, DOUBLE BLINDED, COMPARATIVE STUDY.

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BACKGROUND:

Tracheal extubation is as important as intubation in general anaesthesia which causes a moderate to severeincrease in blood pressure and heart rate even though transient is unwarranted in high risk patients. Hence a smooth emergence is preferable for all patients[1,2]. In this study we aimedto compare the effect of intravenous dexmedetomidine and magnesium sulphate in facilitating smooth endotracheal extubation in terms of haemodynamics, airway reflexes and sedation in patients undergoing elective surgical and gynecological procedures under GA[3]

MATERIALS AND METHODS:

60 patients posted for elective surgical and gynecological procedures aged between 20 - 45 years with ASA grades I and II wereincluded in the study.Patients were randomly divided into two groups of 30 each. At the time of skin closure, Group D and Group M each received an infusion of dexmedetomidine (0.5 µg/kg) and magnesium sulfate (30 mg/kg), respectively, over a ten-minute period. Base line parameters and at 1 ,3 and 5 minutes after drug administration, during extubation, and 1, 3, 5, 10, 15minutes following extubation were recorded. point rating scale (quality of extubation) and a six-point Ramsay sedation scale were alsoanlysed.

RESULTS :

We observed that hemodynamic parameters (HR, SBP, DBP) in Group D were substantially more acceptable than those of Group M. Extubation quality scores ranged from one to two, with one being the majority for patients in group D (83.3%) and three being the majority for patients in group M (80%) . 77% of patients in group D had a sedative level of 3 (calm)while 53% of patients in group M had a score of 2 (anxious & agitated).

CONCLUSION :

Dexmedetomidine provides a smoother recovery profile as compared to MgSO4 when administered prior to extubation. Hence dexmedetomidine can be recommended for smooth recovery even in high risk patients.

KEYWORDS: Airway reflexes; Clonidine; Dexmedetomidine; Extubation; Haemodynamic responses.

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INTRODUCTION

Tracheal extubation is the discontinuation of an artificial airway when the indications for its placement like airway obstruction, protection of airway, suctioning, ventilatory failure and hypoxemia no longer exist[4] Tracheal extubation is an important event in course of general anaesthesia which causes a modest (10% to 30%) and transient (lasting approximately 5 to 15 min) increase in heart rate and blood pressure[5] Extubation is associated with reflex sympathetic discharge caused by epipharyngeal and laryngopharyngeal stimulation. These changes are transient and probably of no consequences in healthy individual going for general surgery, but has a major concern for patients with coronary artery disease, cerebrovascular disease and in hypertensive patients[6] For a smooth extubation, there should be no straining, movement, coughing, breath holding or laryngospasm. Extubation at light levels of anesthesia or sedation can stimulate reflex responses via tracheal and laryngeal irritation. Various agents like lidocaine, opioids, esmolol, calcium channel blockers[7], magnesium sulphate and propofol have been shown to attenuate these responses, but they all have limitations and side effects. Dexmedetomidine, an α2-adrenoreceptor agonist with a distribution half-life of approximately 6 minutes has been successfully used for attenuating the stress response to laryngoscopy. Magnesium is naturally occurring calcium antagonist and noncompetitive antagonist of N-methyl D-aspartate (NMDA) receptor[8] It inhibits many calcium mediated responses like the release of catecholamine from both adrenal glands and adrenergic nerve terminals in response to sympathetic stimulation[9] Intravenously administered magnesium sulphate is capable of attenuating the adverse haemodynamic response associated with endotracheal intubation[10] We designed a prospective, randomized, double blinded trial to determine the effectiveness of dexmedetomidine as an alternative for blunting the hemodynamic response to tracheal extubation. Extubation can be associated with several complications like coughing and respiratory and hemodynamic alterations. These changes are usually transient and well tolerated by most patients, but may be deleterious in certain subgroups of patients. easy extubation, provide a more comfortable recovery and allow early neurological examination following intracranial operations [11] Dexmedetomidine 0.5 mcg/kg, given 5 minutes before extubation

MATERIAL AND METHODS

This study was carried out on 60 patients posted for elective surgical and gynecological procedures aged between 20 - 45 years with ASA grades I and II were included in the study.

Patients with cardiovascular or respiratory disorders, diabetes, hypertension, obesity, difficult airway, medications that effect heart rate (HR) or blood pressure (BP), pregnant, currently breast feeding women, history of sleep apnea and those for emergency procedures were excluded. Institutional board approval was taken and written informed consent was taken from each patient. Pre-anesthetic check up was conducted and a detailed history and complete physical examination recorded. Routine investigations like complete blood picture, blood grouping/typing, blood urea and serum creatinine were done. Routine anesthetic technique was used using propofol, fentanyl, vecuronium, nitrous oxide-oxygen and sevoflurane. Standard monitoring with electrocardiography (EKG), pulse oximetry (SpO2) and noninvasive BP was done. Patients were randomly divided into two groups of 30 each. At the time of skin closure, Group D and Group M each received an infusion of dexmedetomidine (0.5 µg/kg) and magnesium sulfate (30 mg/kg), respectively, over a ten-minute period. Base line parameters and at 1 ,3 and 5 minutes after drug administration,

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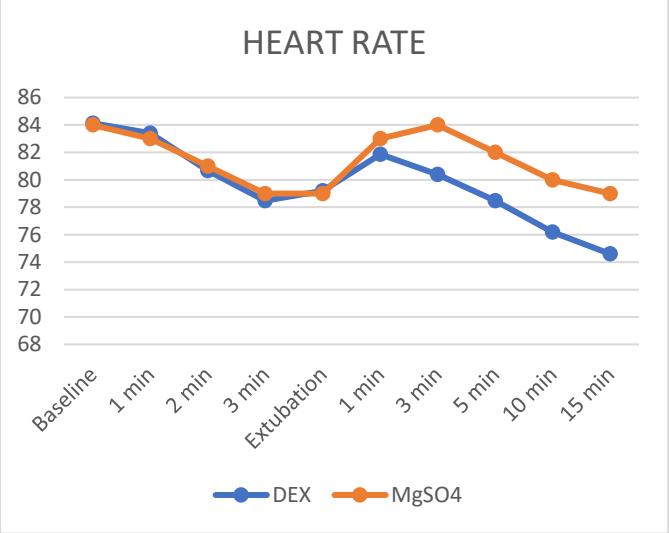
during extubation, and 1, 3, 5, 10, 15 minutes following extubation were recorded. point rating scale (quality of extubation) and a six-point Ramsay sedation scale were also anlysed. Residual neuromuscular blockade was reversed with neostigmine 0.05 mg/kg and atropine 0.02 mg/kg IV. When patients' spontaneous respirations were considered sufficient and patients were able to obey simple commands, suction of throat was done and trachea was extubated. Occurrence of any event like laryngospasm, bronchospasm, desaturation, respiratory depression, vomiting, hypotension, bradycardia or undue sedation was noted. Hypotension was defined as a decrease in systolic BP of more than 30 mmHg from baseline and was corrected with IV fluids and if required, with small dose of mephentermine 6 mg IV. Bradycardia was defined as a HR of less than 60/minute and was corrected, if associated with hemodynamic instability, with atropine 0.6 mg IV. Quality of extubation was evaluated based on cough immediately after extubation, using a 5 point rating scale (ExtubationQuality Score): 1 = no coughing, 2 = smooth extubation, minimal coughing (1 or 2 times), 3 = moderate coughing (3 or 4 times), 4 = severe coughing (5-10 times) and straining, 5 = poor extubation, very uncomfortable (laryngospasm and coughing >10 times). Postoperative sedation was evaluated on a 6 point scale(Ramsay Scale):[7] 1 = Anxious or agitated and restless or both, 2 = Cooperative, oriented and tranquil, 3 = Drowsy but responds to commands, 4 = Asleep, brisk response to light glabellar tap or loud auditory stimulus, 5 = Asleep, sluggish response to light glabellar tap or loud auditory stimulus, 6 = Asleep and unarousable.

RESULTS



The mean HR was lower in dexmedetomidine as compared to magnesium sulphate but none of patients suffered from bradycardia and did not required dose reduction. Aksu R et al21 in their study observed that dexmedetomidine was superior than fentanyl for blunting haemodynamic response. Our results are in accordance with Guler G et al.18 Dexmedetomidine activates receptors in the medullary vasomotor centre, reducing noradrenaline turnover and decreasing central sympathetic outflow resulting in alteration in sympathetic function and decreased heart rate and blood pressure.22,23

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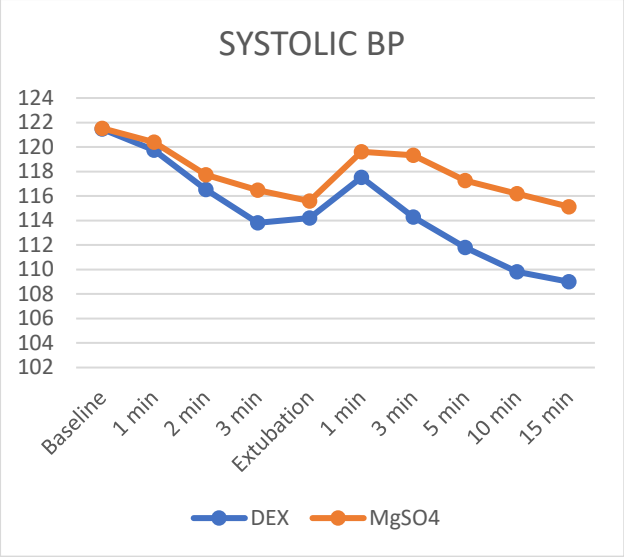
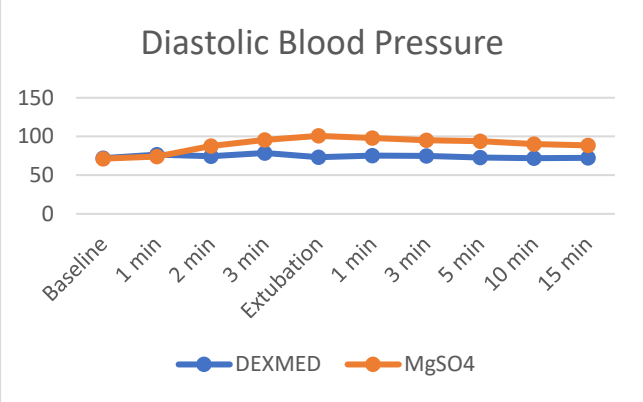
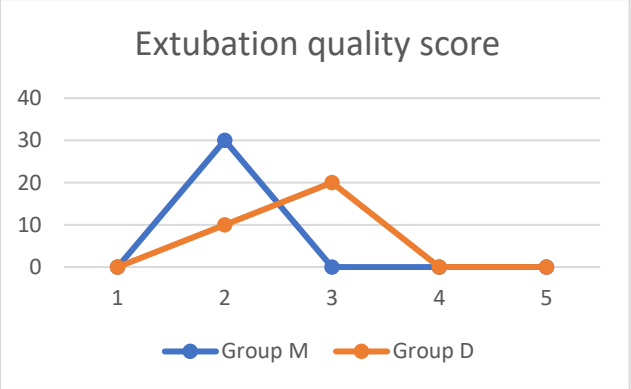




Results in our study has clearly shown that tracheal extubation led to significant increase of SBP and DBP in group C whereas in group M and group D, SBP and DBP remains near to baseline value during extubation. Our results related to dexmedetomidine are in accordance with Turan G et al26 and Jain D et al.12 In their study it has been shown a significant reduction in blood pressure during extubation with use of dexmedetomidine. Nooraei N et al27 supported the fact that use of magnesium sulphate provides better arterial pressure control than lignocaine during intubation. Panda NB et al28 studied similar dose as used in our study and found significant reduction in BP during intubation.



Alpha-2 stimulation causes smooth muscle relaxation thereby preventing bronchoconstriction. Extubation score 1 (no coughing) was found in 90% patient of group D. Incidence of coughing was more in group C and group M than group D.

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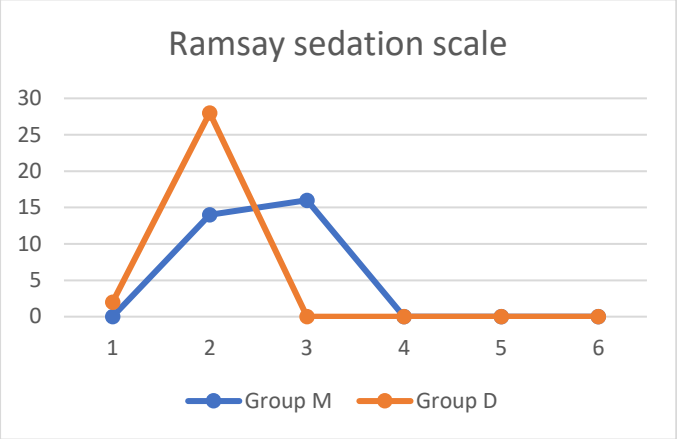

This is in accordance with study done by Sharma VB et al8 and Guler G et al.18 Significant number of the patients in group D had sedation score 3 while in group C and group M most of the patients belong to sedation score 1. Central stimulation of parasympathetic outflow and inhibition of sympathetic outflow from the locus coeruleus in the brainstem plays an important role in the sedation and anxiolysis produced by dexmedetomidine. Decreased noradrenergic output from the locus coeruleus facilitate for increased discharge of inhibitory neurons including the gammaamino butyric acid system resulting in anxiolysis and sedation.

DISCUSSION

Tracheal intubation and extubation are associated with marked elevation in heart rate and arterial pressure. Coughing is the most common reflex response which occurred due to the presence of endotracheal tube[12] It can produce increase heart rate, increase blood pressure, increase intraocular pressure, increase intracranial pressure, myocardial infarction and surgical bleeding. Systemic hypertension associated with tracheal extubation may lead to postoperative intracranial haematoma following craniotomies which is a devastating consequence and has an incidence of 0.8% -2.2%[13]

In our study, most patients in the Dexmedetomidine study group could be extubated smoothly with minimal coughing (Extubation Quality Score 2) when compared to $MgSo_4$ group, where most patients had moderate cough (Extubation Quality Score 3).

Dexmedetomidine 0.5 mcg/kg given as a single-dose bolus before tracheal extubation has been shown to attenuate airway-circulatory reflexes during extubation with no difference between the groups in the incidence of breath holding or desaturation[14] 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. We found that most patients in Dexmedetomidine study group were drowsy but responding to verbal commands (Ramsay Sedation Scale 3) after extubation when compared to $MgSo_4$ group, where most patients belonged to Ramsay Sedation Scale 2. The quality of sedation is better and the need for rescue sedation is less with dexmedetomidine and there is no significant adverse effect on hemodynamic or respiratory function[9] The activation of α2 adrenoceptors, imidazoline-preferring receptors, or both in the ventrolateral medulla and especially in the solitarius nucleus tract by dexmedetomidine causes

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bradycardia. In our study, the incidence of bradycardia and hypotension was higher in study group than in $MgSo_4$ group. In our study, none of the patients in either group developed respiratory depression, laryngospasm, bronchospasm, undue sedation or desaturation. Similar findings have been made by Guler et al[12]

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 not cause side effects like respiratory depression, laryngospasm, bronchospasm, undue sedation and desaturation.

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