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Original Research Article

A Comparative Study to Analyse the Effectiveness of Intravenous 50% Magnesium Sulphate and Dexmedetomidine for Attenuation of Cardiovascular Stress Response during Laryngoscopy and Endotracheal Intubation

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Abstract:

Background: Direct laryngoscopy and endotracheal intubation is almost always associated with hemodynamic changes due to reflex sympathetic discharge as hypertension, tachycardia, predisposition to cardiac arrhythmia and increased myocardial oxygen consumption.

Objectives: To compare the attenuation of cardiovascular stress responses during laryngoscopy and endotracheal intubation using intravenous 50% Magnesium sulphate and Dexmedetomidine.

Methods: A prospective randomized double blind comparative study of Dexmedetomidine versus 50% Magnesium sulfate for attenuation of cardiovascular stress responses was done in sixty patients aged between 20 to 60 years of physical status ASA grade I and ASA grade II undergoing elective surgery under general anaesthesia. All patients were divided into 2 groups of 30 patients each. Group-M received 30 mg/kg of Magnesium sulphate and Group-D received 1 µg/kg Dexmedetomidine intravenous infusion ten minutes before intubation. Both the groups were observed for changes in hemodynamic parameters i.e. heart rate (HR) systolic, diastolic blood pressure and mean arterial pressure at 0, 2 and 5 minutes post intubation.

Results: Statistical analysis was performed using SPSS version 20 (USA). Heart rate was increased at 0 minute that is immediately after intubation in group D by 2.86 ± 1.74 bpm (3.52%) from pre induction mean value, while in group M it was 12.26 ± 2.96 bpm (14.69%) (p = 0.0001), showing highly significant difference among both the groups. Similarly at 2 minutes after intubation rise in heart rate in group D is only 0.10 ± 1.90 bpm (0.12 %) while in group M it is 5.56 ± 2.18 bpm (1.79%) showing a statistically significant difference (p = 0.0021). At 5 minutes after intubation both groups shows mean heart rate near pre induction mean values in group D it was 78.3 ± 7.15 bpm with a decrease of 3.17 bpm (-3.89%) while in group M it was 83.30 ± 8.73 bpm with a difference of -0.20% from pre induction (p = 0.0184). Systolic blood pressure, diastolic blood pressure and mean arterial pressure remain comparable in both the groups at 0, 2 and 5 minutes after intubation i.e. statistically not significant (p > 0.05). Therefore both dexemedetomidine and $MgSO_4$ were effective in controlling the SBP, DBP & MAP after intubation.

Conclusion: Magnesium sulphate is as effective as dexmedetomidine in attenuating the cardiovascular stress response to laryngoscopy and endotracheal intubation. Dexmedetomidine (1µg/kg) provides fairly good and sustained control over hemodynamic responses to the stress of laryngoscopy and intubation and is significantly better than $MgSO_4$ (30mg/kg). So we conclude that dexmedetomidine is better alternative to $MgSO_4$ for attenuation of stress responses of laryngoscopy and intubation.

Keywords: Dexmedetomidine, Magnesium Sulfate, Hemodynamic Response, Laryngoscopy, Endotracheal Intubation, Stress Response.

Introduction

Direct laryngoscopy and endotracheal intubation is almost always associated with hemodynamic changes due to reflex sympathetic discharge caused by epipharyngeal and laryngopharyngeal stimulation. [1] The sympathetic response to laryngoscopy and intubation includes hypertension, tachycardia, predisposition to cardiac arrhythmia and increased myocardial oxygen consumption.

The sympathoadrenal responses result in an acute increase in plasma concentration of catecholamines (epinephrine and norepinephrine). [2]

These changes are maximum at 1 min after intubation and last for 5-10 min. The factors which influence the magnitude of hemodynamic changes are the duration of laryngoscopy and intubation, the type of blades, the anaesthetic agent used and the depth of anaesthesia. [3]

Various techniques are used to attenuate these intubation-related stress responses, all of which causes reduction in input stimuli or the blockade of adrenergic surge. There have been various anaesthetic drug combinations and hemodynamic-moderating efforts that researchers have trialed over the years in an effort to attenuate this sudden and often dramatic, hemodynamic response.

Dexmedetomidine is a potent alpha2-adrenoceptor agonist with high affinity for the alpha2-adrenoceptor. Dexmedetomidine has shown sedative, analgesic and anxiolytic effects after intravenous administration. [4] Due to its central sympatholytic effect, dexmedetomidine is useful in blunting hemodynamic responses in perioperative period. It is successfully used in intravenous doses varying from 0.25 to 1 mcg/kg for attenuating intubation response. [5,6] Optimal dose for attenuating pressure response seems to be 1 mcg/kg with lesser doses not being that effective. [7]

Magnesium sulphate which is a known anti-convulsant agent in eclampsia have been tried for attenuating the stress responses and reducing cardiovascular effects associated with laryngoscopy and tracheal intubation, when used in relatively small doses.

The present study was undertaken to compare the attenuation of cardiovascular stress response during laryngoscopy and endotracheal intubation using intravenous 50% Magnesium sulphate and Dexmedetomidine.

Material and Methods

This prospective randomized double blind comparative study was conducted after appropriate institutional human research scientific committee and ethics committee approval and written, informed consent of patients in language they understand.

Inclusion Criteria: 60 patients aged 20 to 60 years with physical status ASA I & II of both genders undergoing elective non cardiac surgery under general anaesthesia with endotracheal intubation through oral route.

Exclusion Criteria: Patients with body weight < 55 kg & > 90 kg, ASA Grade III, IV & V, MPG III & IV, predicted difficulty in intubation, history of allergy/ contraindications to study drugs either Dexmedetomidine or Magnesium sulfate, laryngoscopy and intubation time > 20 seconds, pregnant or nursing women, emergency surgeries, patients with hypertension, coronary artery disease, ischemic heart disease, history of heart blocks, patients with heart rate < 60 bpm and >120 bpm, systolic blood pressure <100 mmHg and >150 mmHg and patient refusal or patient unable to understand the consent / not giving consent were excluded from the study.

All patients were evaluated a day before surgery and were kept fasting overnight.

Randomization, Group allocation and Blinding:

Patients were randomly allocated into two groups each of thirty using computer-generated random number table. For double-blinding, the study drug was prepared by some other person, and the person administering the drug and the patients both were unaware of the allocated group. Patients of Group D were received Dexemedetomidine 1 µg/kg and of Group M 50% Magnesium sulfate 30 mg/kg diluted to 100 ml with normal saline, intravenously over 10 minutes, 10 minutes before intubation.

Anaesthetic Technique:

All patients were monitored with ECG, pulse oximetry and non-invasive blood pressure after arrival to preoperative room. Monitors were connected and Baseline parameters as heart rate, systolic blood pressure, diastolic blood pressure and mean blood pressure were recorded. An IV line was secured and the patients were administered IV fluid Ringer's lactate in the ward before shifting the patient to preoperative room. All patients were received Inj. Midazolam 1 mg IV as premedication. Patients of Group D were received Dexemedetomidine 1µg/kg diluted to 100 ml normal saline, intravenously over 10 minutes, 10 minutes before intubation. Group M patients were administered 50% Magnesium sulfate 30mg/kg diluted to 100ml with normal saline, intravenously over 10 minutes, 10 minutes before intubation. Patients were monitored throughout the period study drug infused in the preoperative room and anaesthesia was induced in operating room after 10 minutes.

Standardised general anaesthesia technique was used for both the groups. All the patients were pre-oxygenated for 3 minutes and were induced with Inj. Fentanyl 1.5 µg/kg, propofol 2 mg/kg till loss of verbal response, followed by Inj.Vecuronium 0.1 mg/kg. Three minutes later, laryngoscopy and orotracheal intubation was done with appropriate sized MacIntosh blade and endotracheal tube. Laryngoscopy and intubation time were noted for all cases and in patients where laryngoscopy and intubation time was > 20 seconds were excluded from the study. Heart rate, systolic blood pressure,

diastolic blood pressure and mean blood pressure were recorded immediately before anaesthesia induction, before endotracheal intubation, immediately after endotracheal intubation (0 minute), at 2 minutes and 5 minutes after endotracheal intubation. Any side effects like bradycardia, hypotension, hypertension and tachycardia were monitored and managed according to standard protocol.

Anaesthesia was maintained with O_2 and N_2O in a ratio of 50% : 50% and 1% sevoflurane. Muscle relaxation was maintained with IV vecuronium top ups of 0.04 mg/kg. After surgery, IV neostigmine 0.05 mg/kg and IV glycopyrrolate 0.01 mg/kg were given for reversal. After adequate recovery, patients were shifted to post-anaesthesia care unit and monitored there and later on shifted to ward. Any complications occurring in postoperative period were monitored and noted. Any side effects like bradycardia, hypotension, hypertension and tachycardia were monitored and managed according to standard protocol

Statistical Analysis:

Statistical software SPSS 20 was used and outcome measured and compared using unpaired t test. A p value of < 0.05 was considered statistically significant and a p value of < 0.005 was considered statistically highly significant.

Results

Both the groups were comparable to each other in terms of demographic data (Table 1).

Table 1: Demographic data among groups

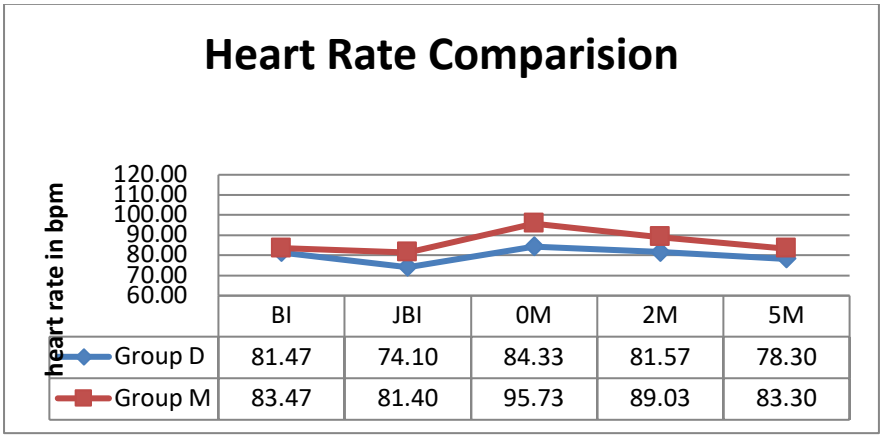
	Group D (n=30)	Group M (n=30)
Mean age (yrs)	40.43 ± 9.59	39.63 ± 9.87
Gender (M/F)	17/13	15/15
Mean weight (kg)	69.83	69.50

Heart rate was increased at 0 minute that is immediately after intubation in group D by 2.86 ± 1.74 bpm (3.52%) from pre induction mean value, while in group M it was 12.26 ± 2.96 bpm (14.69%) with a p value 0.0001, showing highly significant difference among both the groups. Similarly at 2 minutes after intubation rise in heart rate in group D is only 0.10 ± 1.90 bpm (0.12 %) while in group M it is 5.56 ± 2.18 bpm (1.79%) showing a statistically significant difference with a p value of 0.0021. At 5 minutes after intubation both groups shows mean heart rate near pre induction mean values in group D which was 78.3 ± 7.15 bpm with a decrease of 3.17 bpm (-3.89%) while in group M it was 83.30 ± 8.73 bpm with a difference of -0.20% from pre induction mean value, with a p value of 0.0184. (Table 2 and Graph 1)

Table 2: Comparision of changes in heart rate

Time of Assessment	Group D	% Diff	Group M	% Diff	p value
BI	81.47 ± 9.32		83.47 ± 8.14		0.3797
JBI	74.10 ± 7.72	-9.05%	81.40 ± 9.83	-2.48%	0.0004
0 min after intubation	84.33 ± 7.58	3.52%	95.73 ± 12.10	14.69%	0.0001
2 min after intubation	81.57 ± 7.42	0.12%	89.03 ± 10.32	6.66%	0.0021
5 min after intubation	78.30 ± 7.15	-3.89%	83.30 ± 8.73	-0.20%	0.0184

BI- Before Induction, JBI- Just Before Intubation



Graph 1: Comparision of changes in heart rate

There was rise in systolic blood pressure immediately after intubation at 0 minute in both the groups. The rise in group D from pre induction mean value was 6.54 mm Hg (5.18 %) while in group M it was 5.46 mm Hg (4.25 %) with a p value of 0.7031, showing no statistically significant difference in both the groups. In both the groups systolic blood pressure decreases at 2 minutes and 5 minutes after the intubation from pre induction values, showing no statistically significant difference in the both the groups, p value was > 0.05. (Table 3)

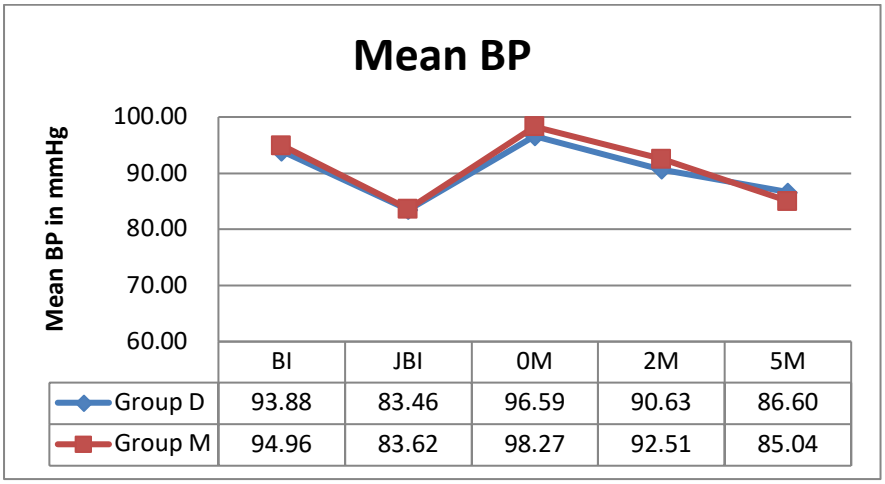
There was rise in diastolic blood pressure immediately after intubation at 0 minute in both the groups. The rise in group D from pre induction mean value was 0.8 mm Hg while in group M it was 2.23 mm Hg with a p value of 0.6935, showing no statistically significant difference in both the groups. In both the groups diastolic blood pressure decreases at 2 minutes and 5 minutes after the intubation from pre induction values, showing no statistically significant difference in the both the groups, p value was > 0.05. (Table 3)

Table 3: Comparision of changes in systolic and diastolic blood pressure

Time of Assessment	Group D	% Diff	Group M	% Diff	p value
SBP (mm Hg)					
BI	126.23 ± 9.17		128.67 ± 7.25		0.2591
JBI	110.43 ± 12.04	-12.52%	113.6 ± 7.31	-11.71%	0.2227
0 min after intubation	132.77 ± 8.51	5.18%	134.13 ± 17.49	4.25%	0.7031
2 min after intubation	121.5 ± 8.74	-3.75%	125.73 ± 14.83	-2.28%	0.1836
5 min after intubation	115 ± 10.39	-8.90%	115.13 ± 13.46	-10.52%	0.9667
DBP (mm Hg)					
BI	77.7 ± 5.06		78.1 ± 4.65		0.751
JBI	69.97 ± 5.74	-9.95%	68.63 ± 5.45	-12.13%	0.3576
0 min after intubation	78.5 ± 5.02	1.03%	80.33 ± 10.32	2.86%	0.6935
2 min after intubation	75.2 ± 5.99	-3.22%	75.9 ± 11.23	-2.82%	0.7643
5 min after intubation	72.4 ± 6.17	-6.82%	70 ± 8.3	-10.37%	0.2088

BI- Before Induction, JBI- Just Before Intubation

There was rise in mean arterial pressure immediately after intubation at 0 minute in both the groups (p = 0.4695), showing no statistically significant difference in both the groups. MAP at 2 min and 5 min after the intubation was comparable in both the groups (p > 0.05). (Graph 2)



Graph 2: Comparision of changes in mean arterial pressure

None of the patients in both the groups experienced any complications.

Discussion

Laryngoscopy and endotracheal intubation are noxious stimuli. Both of them are responsible for a huge spectrum of stress responses such as hypertension, tachycardia, laryngospasm, bronchospasm, raised intraocular pressure and intracranial pressure. King and colleagues in 1951 first described the reflex circulatory responses to direct laryngoscopy and tracheal intubation. [8]

Within seconds of direct laryngoscopy, haemodynamic responses are initiated and further increases with the passage of the endotracheal tube. The response is initiated within 5 seconds of laryngoscopy, peaks in 1–2 min and returns to normal levels by 5 min. [9] These changes are usually short lived and well tolerated by normal

patients. In patients with cardiovascular disease, it can result in harmful effects such as myocardial ischaemia, ventricular failure, ventricular dysrrhythmias and pulmonary edema. It can also lead to cerebrovascular accidents in cerebrovascular disease patients. [10] Elbert et al [11] noted that hemodynamic response is proportional to the duration of laryngoscopy. The response starts at 15 seconds and peaks at 45 seconds. Hence it is recommended that duration of laryngoscopy and intubation should be limited to 30 seconds. In our study we have excluded those patients where duration of laryngoscopy and intubation was more than 30 seconds.

Dexmedetomidine is a highly selective short-acting alpha2-adrenoceptor agonist having many desirable clinical benefits that make it suitable to use in the perioperative period. Dexmedetomidine provides a sedated patient in the preoperative period. Intraoperatively, in addition to its anaesthesia-sparing effects, it provides a stable hemodynamic profile by attenuating the stress responses during tracheal intubation, during surgery and emergence from anaesthesia. [12] Scheinin et al [13] showed in their study that dexmedetomidine attenuated the cardiovascular responses to laryngoscopy and endotracheal intubation and the concentration of noradrenaline in mixed venous plasma was smaller in the group of patients who received dexmedetomidine.

In the last few years, there has been sudden in-crease of interest in both the physiological and pharmacological properties of magnesium. Magnesium blocks the release of catecholamines from adrenergic nerve terminals and from the adrenal gland and reduces levels of serum epinephrine and cause a decrease in the atrial contraction, bradycardia and vasodilatation. [14,15] Catecholamine release and cardiovascular effects in response to tracheal intubation were found to be attenuated by intravenous magnesium in most clinical trials. [16] Puri GD et al [17] showed magnesium sulphate attenuates pressure response to laryngoscopy and intubation in patients with coronary artery disease.

In our study both the groups were comparable and there was no statistically significant difference with respect to mean age, weight and gender with a p value >0.05 among both the groups . It was in accordance with the previous studies conducted by other authors like Krishna Chaithanya et al [18] , Chhaya Joshi et al [19], Bidyut Borah et al [20].

Our study showed that dexmedetomidine in a dose of 1µg/kg was better in controlling heart rate at different intervals than $MgSO_4$ and heart rate values are statistically significant in dexmedetomidine group compared to magnesium sulphate group (p value <0.05). Krishna Chaithanya et al[18] observed that the decrease in heart rate was more with dexmedetomidine compared to magnesium sulphate. Chhaya Joshi et al [19] found heart rate stayed near or below baseline values at 2 minutes and 5 minutes showing that dexemedetomidine is effective in attenuating stress response. According to Chandrakala et al, [21] in controlling heart rate dexmedetomidine was more effective as compare to MgSO4. Also, the rise in heart rate was less in MgSO4 group which was not more than 10 to12 beats/min.

Systolic blood pressure, Diastolic blood pressure and mean arterial blood pressure remain comparable in both the groups at 0, 2 and 5 minutes after intubation i.e. statistically not significant (p > 0.05). Therefore both dexemedetomidine and $MgSO_4$ were effective in controlling the SBP, DBP & MAP after intubation. Krishna Chaithanya et al[18] and Scheinin B et al [13] both observed in their studies that both dexemedetomidine and $MgSO_4$ attenuated the rise in SBP, DBP & MAP. Padmawar et al [22] observed that SBP, DBP were increased significantly only at 1 minute after intubation in $MgSO_4$ group (40 mg/kg) and which came to base line at 5 minutes after intubation as compared to lignocaine (1.5 mg/kg). Keniya VM et al [23] and Mohan A et al [24] observed pre-treatment with dexemedetomidine 1 µg/kg attenuated cardiovascular response to tracheal intubation after induction of anaesthesia.

None out of 60 patients in our study experienced any complications like hypotension, bradycardia, tachycardia, hypertension, nausea and vomiting. Patients in both the groups were hemodynamically stable throughout the perioperative period. Recovery from anaesthesia was uneventful in both the groups. Our results were in accordance with studies of Krishna Chaithanya et al, [18] Bidyut Borah et al [20] & Mohan A et al [24].

Conclusion

The present study concluded that both dexmedetomidine 1µg/kg and $MgSO_4$ 30 mg/kg intravenous infusion over 10 minutes, 10 minutes before intubation are effective agents to attenuate the cardiovascular responses to laryngoscopy and intubation . According to our study dexmedetomidine (1µg/kg) provides fairly good and sustained control over hemodynamic responses to the stress of laryngoscopy and intubation and is significantly better than $MgSO_4$ (30 mg/kg). So we conclude that dexmedetomidine is better alternative to $MgSO_4$ for attenuation of stress responses of laryngoscopy and intubation.

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