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(RESEARCH ARTICLE)

The effect of magnesium sulphate pretreatment on suxamethonium-induced rise in intraocular pressure in patients undergoing surgery under general anaesthesia

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Abstract

Background: Patients with increased intraocular ocular pressure (IOP) or open eye with full stomach scheduled for surgery under general anaesthesia are at the of risk of extrusion of eye content and possible regurgitation and aspiration of gastric contents. Thus prevention or reducing the rise in IOP following the administration of suxamethonium in this group of patients is crucial.

Aim: This study aimed to evaluate the efficacy of MgSO4 pre-treatment on suxamethonium- induced rise in IOP during general anaesthesia in patients coming for elective general surgery.

Methodology: Ninety-six ASA1 or 2 patients aged 15-65 years were recruited into two equal groups after ethics committee approval. One group received MgSO4 and the other group received normal saline. The IOP was measured and recorded at specified times up to 15 minutes after tracheal intubation.

Data were analysed with the SPSS version 16. Continuous data were summarised as means and standard deviation (SD) and dichotomous data as counts and frequency. Parametric data were compared using student's t-test and categorical data analysed using chi-square and fisher's exact test. A P value of < 0.05 was considered as statistically significant. All statistical tests were two - sided.

Result: There were significant differences in IOP changes following suxamethonium, tracheal intubation, at 5th minute, 10thminute and 15th minute post intubation with p values 0.001, <0.001, <0.001, <0.001, 0.001 respectively (independent t-test).

The mean time taken to return to baseline/near baseline was shorter in the study group. There were statistical differences in the IOP, PR, SBP, DBP, and MAP after suxamethonium administration and laryngoscopy and tracheal intubation. The incidence of side effects and complications were minimal.

Conclusion: This study revealed that 30 mg/kg of magnesium sulphate pretreatment minimized the increase in IOP following suxamethonium administration. It also reduced the haemodynamic response associated with laryngoscopy and tracheal intubation.

Keywords: Intraocular Pressure; Suxamethonium; Magnesium Sulphate; Open Eye

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1. Introduction

Patients with penetrating eye injuries presenting for surgery pose challenges to the anaesthesiologist as they may require rapid sequence induction of anaesthesia due to full stomach.[3,4] Suxamethonium is the fastest (30-45seconds onset time) and shortest acting depolarising muscle relaxant (3-5minutes duration) in clinical use with profound muscle relaxtion.[6,7] These make it the muscle relaxant of choice during rapid sequence induction (RSI), difficult/failed intubation.[7] However it is associated with many side effects like increased intraocular, intracranial, intra-gastric pressures, fasciculation, myalgia, hyperkalaemia, malignant hyperthermia.[6,8]. The raised intraocular pressure predisposes to extrusion of eye contents in this group of patients.

The mechanisms of suxamethonium-induced raised intraocular pressure are poorly understood.[2,3.5] One possible mechanism is suxamethonium-induced muscle fasciculation (extra-ocular muscles inclusive)[5] which leads to increase blood flow to the eyes.[1] Another speculation is that it could be due to an increase in serum catecholamine level following suxamethonium administration which leads to increase in blood pressure with resultant increase in blood flow to the eyes.

Various studies have shown that by modifying anaesthetic techniques and use of some drugs, the side effects of suxamethonium can be attenuated or minimised.[4,9,10] These include self- taming with small doses of suxamethonium, pre-treatment with non-depolarising muscle relaxants, nifedipine, clonidine, dexmedetomidine, magnesium sulphate ($MgSO_4$).[2] In recent times, $MgSO_4$ has been used in clinical practice for treatment of various conditions such as seizure, arrhythmias, pain, hypertension, tocolysis, myocardial, neuronal ischemia and in anaesthetic practice.[11,12,13] This is due to its antagonistic effect on NMDA receptors and calcium ion channels.[14,15]

Studies on the effect of $MgSO_4$ pre-treatment of suxamethonium on IOP changes is scanty. This study therefore aimed to study the efficacy of $MgSO_4$ in mitigating rise in IOP suxamethonium administration during induction of GA for elective general surgery.

Aim and Objectives

The aim of the study was to evaluate the effect of pre-treatment with Magnesium sulphate on suxamethonium-induced rise in IOP during induction of general anaesthesia, laryngoscopy and endotracheal intubation.

2. Methodology

This was a prospective randomised double blind study conducted at the University of Benin Teaching Hospital Benin City Edo state. Patients with American Society of Anesthesiology physical status, ASA I and II patients aged between 15-65years scheduled for general surgeries under general anaesthesia were recruited for the study.

The following categories of patients were excluded from the study: patients scheduled for other surgery apart from general surgery under GA, patients with raised IOP [glaucoma], patients with history of allergy to any of the study drugs, hypertensive patients, patients with anticipated difficult airway and patients who had more than one attempt at intubation.

Preoperative assessment and preparation of the patients was carried out by the researcher a day before surgery as per departmental protocol. The procedure was explained to the patient and an informed consent obtained. Every patient was fasted over night from 10pm. Each patient was given oral diazepam 5mg at 22.00hours of the night before and one hour before surgery.

Ninety-six patients were randomly assigned into one of the two groups (48 each), 1 or 2 using a random number table with allocation ratio 1:1. The serial number and group allocation were packed in a sealed envelope. The allocation sequence was concealed from the investigator enrolling and assessing the participants. Only the serial number was entered on the proforma sheet of the individual patients. The sealed papers were put in the same envelop with proforma and stapled back. These were reopened at the conclusion of the study. Group 1 comprised those that received intravenous $MgsSO_4$ and group 2 received saline.

After routine anaesthetic machine check and monitoring of baseline vital signs, group 1, received 30mg/kg of $MgSO_4$, (Magphate™; Lincoln Pharmaceuticals, Batch no EA-3802; NAFDAC Reg no: A4-2027). This was made up to 15ml with normal saline in a syringe and coded by another anaesthetist not involved in the study. The drug was given intravenously over 10 minutes and at least five minutes before induction of anaesthesia. The IOP, PR and BP (SP, DP,

and MAP) were measured and recorded after completing the injection. For the group 2 patients, 15ml of normal saline in a coded syringe was given intravenously over 10 minutes and fiveminutes before induction of anaesthesia.

The patients were then pre-oxygenated with 100% oxygen for four minutes using a tight fitting face mask, avoiding pressure on the eyes. Anaesthesia was induced with 0.15mg/kg midazolam then 2mg/kg of propofol. Following loss of consciousness, IOP was measured as well as the PR and BP and then recorded. The readings were taken by the investigator. Suxamethonium 1.5mg/kg was given to facilitate endotracheal intubation. The IOP, PR and BP were then measured after fasciculation and muscle paralysis. Laryngoscopy and tracheal intubation was then done. Another measurement of the IOP, PR, BP were done immediately after intubation and then after 2, 5, 10 minutes and 15 minutes.

Anaesthesia was maintained in all patients with 70% nitrous oxide 30% oxygen and 1-2% isoflurane. Analgesia was achieved using 1mg/kg of pethidine intravenously and 0.5mg/kg of ketorolac (NSAID) intravenously. Muscle relaxation was also achieved using atracurium at 0.4 mg/kg . Patient was then ventilated to normocarbia (35mmHg - 45mmHG) till end of surgery. Monitoring of SPO_2, PR, NIBP, ECG, $ETCO_2$, urine output and temperature continued till end of surgery.

At the end of surgery, the oropharynx was suctioned; residual neuromuscular blockade antagonised using neostigmine [0.04mg/kg] and glycopyrolate [0.005 mg/kg]. The inhalational agents were turned off. Following signs of adequate recovery from anaesthesia, the trachea was extubated. The patient was then transferred to the recovery room.

3. Results

Ninety-six ASA I or II patients aged between 15 to 65 years were enrolled for the study and the data obtained from all patients were analysed as no patient was lost to protocol violation. There was no difference between the two groups with regard to age, ASA physical status, and intercurrent medical status when compared with Chi square test as shown in table 1

Table 2 shows the type of surgery (elective versus emergency) and surgical procedures performed. There were 35 elective patients (72.9%) and 13emergency cases (27.1%) in the study group while in the control group, there were 37 patients (77.1%) for elective and 11 (22.9%) patients for emergency surgery. There was no statistical significance when compared with independent t-test. (P=0.637). The surgical procedures were similar in both groups with appendectomy and exploratory laparotomy accounting for 22.9% each in the study and control group respectively.

In table 3, the duration of surgery, time intervals from administration of study drug to induction of anesthesia; time from administration of study drug to laryngoscopy and tracheal intubation and from induction of anaesthesia to laryngoscopy and tracheal intubation were shown. The mean duration of surgery was similar in both groups being 127.94+_94+_ 39.69 min and 124.63+_43.98 minutes for study and control group respectively with p value of 0.699. The other time intervals were comparable in both groups with no statistical significance with p values of 0.793, 0.403 and 0.417 respectively using independent t-test.

Table 4 shows the mean intraocular pressure (IOP) variations in both groups at different times of the study. The reading in the recovery room, baseline, post study drug administration and induction of anaesthesia showed no statistical significant difference, though the degree of change (decrease) in IOP after the study drug administration was more in the study group than the control group. However, there were significant differences in IOP change (increase) following suxamethonium administration, tracheal intubation, at 5th, 10th and 15th minute post intubation with p values of 0.001, < 0.001, <0.001, <0.001, <0.001 and 0.001 respectively (independent t-test).The mean time taken to return to baseline/near baseline was shorter in the study group being between five to 10 minutes and more than 15 minutes in the control group.

Figure 1 shows the incidence of complications in both groups. Three patients (6.3%) in the study group and nine patients (18.8%) in the control group had some complications. Table v shows the type of intraoperative complications in both groups. In the study group, 6.3% (3) patients had hypotension while in the control group, a total of nine patients had complication namely, bradycardia (4.2%), hypotension (10.4%), stridor at extubation (2.1%) and tachycardia (2.1%). There was no statistical significant difference (Fisher exact test). The complications were managed by atropine, fluid and re-intubation.

Table 6 shows the variations in pulse rate intraoperatively. The pulse rate readings showed a significant difference in the recovery room, baseline, at 5th, 10th and 15th minutes post intubation with p values of 0.004, 0.001, 0.001,<0.001 and <0.001 respectively. There was a decrease in pulse rate from baseline after study drug administration which was more in study group and also a comparable decrease in pulse rate after induction of anaesthesia in both groups.

Following suxamethonium administration and laryngoscopy and tracheal intubation, there was an increase in pulse rate in both groups but the increase was remarkable after intubation in both groups.

Table 1 Patient's characteristics in both groups

Parameter	Study groups		p-value*	Level of significance
	Group A (n = 48)	Group B (n = 48)		
Age (mean ± SD) years	39.94 ± 13.57	41.75 ± 14.38	0.527	NS
ASA				
1	24 (50.0)	22 (45.8)	0.683	NS
II	24 (50.0)	26 (54.2)		
Intercurrent medical conditions				
Asthma	6 (12.5)	2 (4.2)	0.372+	NS
Diabetis	4 (8.3)	3 (6.3)		
Epilepsy	1 (2.1)	0 (0.0)		
Hypertension	1 (2.1)	0 (0.0)		
PUD	2 (4.2)	1 (2.1)		
None	34 (70.8)	42 (87.5)		

*Chi-square test, +Fisher's exact test

Table 2 Types of surgery and surgical procedure in both groups

Parameter	Study groups		p-value	Level of significance
	Group A n (%)	Group B n (%)		
Type of surgery				
EL	35 (72.9)	37 (77.1)	0.637*	NS
EM	13 (27.1)	11 (22.9)		
Types of surgical procedure				
Appendectomy	11 (22.9)	8 (16.7)	0.557**	NS
Cholesydtectomy	5 (10.4)	3 (6.3)		
Colostomy	0 (0.0)	2 (4.2)		
Exploratory laparotomy	10 (20.8)	11 (22.9)		
Hemioplasty	2 (4.2)	8 (16.7)		
Incisional hem	2 (4.2)	0 (0.0)		
Mastectomy	8 (16.7)	7 (14.6)		
Thyroidectomy	9 (18.8)	6 (12.5)		
Thyroglosal cyst	1 (2.1)	1 (2.1)		
Triple bypass	0 (0.0)	2 (4.2)		

*Chi-square t-test, **Fisher's exact test

The mean systolic blood pressure variations were shown in table vii. There were significant differences in the recovery room reading, baseline reading, after suxamethonium administration, tracheal intubation, at two and five minutes post

intubation readings with p values of< 0.001,<0.001,<0.001,0.001and 0.001 respectively while SBP in both groups after study drug, induction, 10 and 15 minutes post intubation showed no significance (independent t- test)

Table 8 displayed the mean diastolic blood pressure. There was statistical significant difference in the recovery room reading with p value of 0.004 The DBP increased after suxamethonium administration and after laryngoscopy and tracheal intubation. The p values were 0.029 and 0.001 respectively while the readings at other measuring times were comparable in both groups using independent t test.

Table 3 Duration of surgery, and surgical time duration in both groups

Parameter	Study groups (mean ± SD)		p-value*	Level of significance
	Group A (n = 48)	Group B (n = 48)		
Duration of surgery (hours)	127.94 ± 39.69	124.63 ± 43.98	0.699	NS
Time interval (administration of study drug to induction of anaesthesia) mins	0.062 ± 0.014	0.061 ± 0.008	0.793	NS
Time interval (administration of study drug to tracheal intubation) mins	0.073 ± 0.011	0.071 ± 0.008	0.403	NS
Time interval (induction of anaesthesia to tracheal intubation) mins	0.011 ± 0.009	0.010 ± 0.000	0.417	NS

*Independent samples t-test

Table 4 Variations in intraoperative IOP (mmHg) in both groups

Parameter	Study groups (mean ± SD)		p-value*	Level of significance
	Group A (n = 48)	Group B (n = 48)		
In recovery room	16.58 ± 1.72	16.33 ± 1.46	0.446	NS
Baseline	16.79 ± 2.17	16.35 ± 1.45	0.249	NS
After study drug administration	15.90 ± 1.51	16.17 ± 2.01	0.457	NS
After induction	12.90 ± 1.53	13.38 ± 1.48	0.123	NS
After Suxamethonium administration	16.19 ± 1.61	17.27 ± 1.43	0.001	S
After tracheal intubation	17.37 ± 1.61	21.06 ± 1.92	<0.001	S
2 mins	17.42 ± 1.66	20.13 ± 1.61	<0.001	S
5 mins	16.44 ± 1.66	19.23 ± 1.77	<0.001	S
10 mins	16.33 ± 1.52	18.00 ± 1.99	<0.001	S
15 mins	15.75 ± 1.52	16.96 ± 1.82	0.001	S

*Independent sample t-test

Table 5 Types of intraoperative complications in both groups

Intraoperative complications*	Study groups		p-value	Level of significance
	Group A n = 3 (%)	Group B n = 9 (%)		
Bradycardia	0 (0.0)	2 (4.2)	0.210**	NS
Hypotension	3 (6.3)	5 (10.4)		
Stridor at extubation	0 (0.0)	1 (2.1)		
Tachycardia	0 (0.0)	1 (2.1)		

*Management of complications: Group A; fluids, Group B; fluids, atropine and re-intubation. **Fisher's exact test.

Table 6 Variations in intraoperative pulse rate (bpm) in both groups

Parameter	Study groups (mean ± SD)		p-value*	Level of significance
	Group A (n = 48)	Group B (n = 48)		
In recovery room	89.73 ± 19.79	80.63 ± 6.30	0.004	S
Baseline	87.21 ± 11.17	80.90 ± 7.17	0.001	S
After study drug administration	83.15 ± 10.09	81.00 ± 5.69	0.203	NS
After induction	77.15 ± 10.43	76.79 ± 5.79	0.838	NS
After Suxamethonium administration	84.10 ± 9.46	85.06 ± 6.10	0.557	NS
After tracheal intubation	96.04 ± 8.99	98.19 ± 7.11	0.198	NS
2 mins	89.71 ± 7.51	92.58 ± 6.79	0.052	NS
5 mins	84.15 ± 7.04	88.67 ± 5.09	0.001	S
10 mins	79.63 ± 5.87	85.79 ± 5.69	<0.001	S
15 mins	79.04 ± 4.99	83.75 ± 5.82	<0.001	S

*Independent samples t-test

Table 7 Variations in intraoperative systolic blood pressure (mmHg) in both groups

Parameter	Study groups (mean ± SD)		p-value*	Level of significance
	Group A (n = 48)	Group B (n = 48)		
In recovery room	128.60 ± 8.26	121.79 ± 7.77	<0.001	S
Baseline	127.94 ± 7.47	121.33 ± 7.68	<0.001	S
After study drug administration	123.27 ± 7.44	121.10 ± 7.92	0.170	NS
After induction	112.77 ± 6.10	111.56 ± 6.78	0.361	NS
After Suxamethonium administration	117.96 ± 5.57	122.81 ± 7.07	<0.001	S
After tracheal intubation	131.79 ± 6.80	140.06 ± 7.72	<0.001	S
2 mins	125.13 ± 6.35	130.00 ± 7.45	0.001	S
5 mins	120.75 ± 5.44	124.96 ± 6.56	0.001	S
10 mins	117.85 ± 5.18	119.38 ± 8.92	0.310	NS
15 mins	117.83 ± 4.97	118.29 ± 7.04	0.713	NS

*Independent samples t-test

Table 8 Variations in intraoperative diastolic blood pressure (mmHg) in both groups

Parameter	Study groups (mean ± SD)		p-value*	Level of significance
	Group A (n = 48)	Group B (n = 48)		
In recovery room	80.27 ± 6.26	76.04 ± 7.75	0.004	S
Baseline	79.50 ± 6.35	76.92 ± 9.34	0.116	NS
After study drug administration	76.13 ± 5.31	74.96 ± 7.73	0.391	NS
After induction	69.13 ± 4.97	68.02 ± 7.47	0.396	NS
After Suxamethonium administration	72.63 ± 4.77	75.42 ± 7.32	0.029	S

After tracheal intubation	82.54 ± 5.79	87.88 ± 9.38	0.001	S
2 mins	78.15 ± 5.12	79.25 ± 6.98	0.379	NS
5 mins	74.63 ± 5.62	75.33 ± 8.29	0.625	NS
10 mins	71.52 ± 4.39	73.00 ± 10.16	0.358	NS
15 mins	70.27 ± 5.72	73.10 ± 8.69	0.063	NS

*Independent t-test

Table 9 Variations in intraoperative mean arterial pressure (mmHg) in both groups

Parameter	**Study groups (mean ± SD)**		**p-value***	**Level of significance**
	Group A (n = 48)	**Group B (n = 48)**		
In recovery room	94.27 ± 7.04	87.81 ± 7.68	<0.001	S
Baseline	93.94 ± 6.82	88.33 ± 8.37	0.001	S
After study drug administration	89.94 ± 6.62	86.58 ± 8.03	0.028	S
After induction	81.58 ± 6.27	79.90 ± 6.33	0.193	NS
After Suxamethonium administration	86.31 ± 5.23	88.21 ± 6.60	0.122	NS
After tracheal intubation	97.48 ± 5.55	101.27 ± 9.22	0.017	S
2 mins	91.71 ± 5.63	92.17 ± 6.96	0.724	NS
5 mins	88.02 ± 5.26	89.23 ± 7.42	0.360	NS
10 mins	84.48 ± 5.35	86.73 ± 8.33	0.119	NS
15 mins	83.90 ± 4.46	84.81 ± 7.57	0.472	NS

*Independent t-test

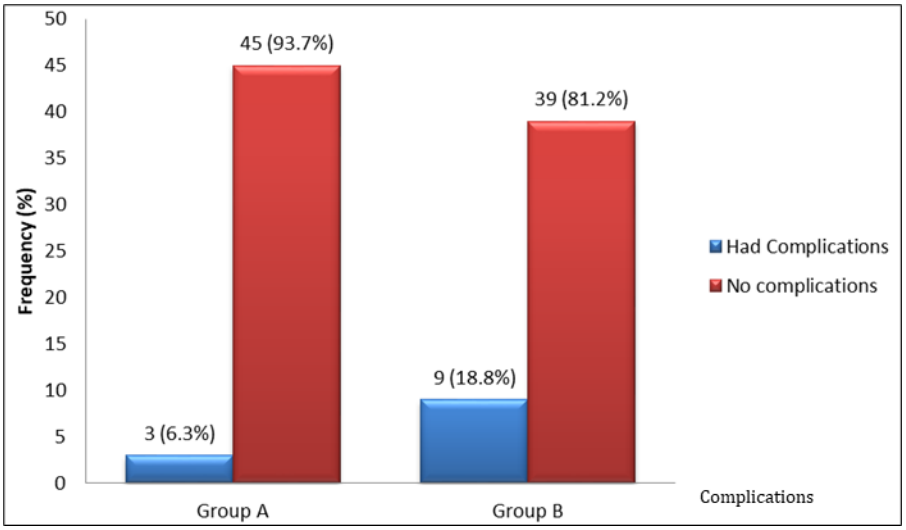

Figure 1 Incidence of complications following surgery in both groups

4. Discussion

Patients with penetrating eye injuries requiring emergency surgery may present with full stomach. The goal of anaesthesia is to secure the airway by rapid sequence induction (RSI) without increase in the IOP. However, suxamethonium, the gold standard for RSI increases IOP among other side effects. This study showed that pretreatment with magnesium sulphate resulted in a minimal increase in IOP following suxamethonium administration compared with the control group.

Increase in IOP following the administration of suxamethonium was marginal and comparatively less in the study group compared with the control group. In patients with an open eye/globe, any increase in IOP could lead to the extrusion of the intraocular content with resultant loss of vision.[28] Our findings are in agreement with a previous study by Sakuaba et al who demonstrated the effectiveness of $MgSO_4$ in reducing suxamethonium-induced rise in IOP in their patients.[23] The effect of $MgSO_4$ in minimizing IOP rise can be attributed to its antagonism of calcium ion release,[29] relaxation of smooth muscle,[30]inhibiting the release of catecholamine or combination of all[31,32] which in turn may lead to decrease blood flow to the eyes and subsequent decrease in IOP.

Chandum and colleagues,[3] studied the influence of dexmedetomidine on IOP changes following the administration of suxamethonium. They found out that IOP in the saline (control) group increased by 2.35mmHg above the base line while in this study, it increased by 0.92mmHg above the baseline in the control group. The disparity in the rise in IOP between the two studies could be the different induction agents used. While Chandum and colleagues used Sodium thiopentone (STP), propofol was used in this study. Propofol is known to maintain deeper planes of anaesthesia than STP and also depresses the cardiovascular system more.[33] However, the increase in IOP (0.09mmHg and 0.12mmHg) following the administration of suxamethonium were similar in the study groups of Chandum et al[3] and in our study.

In this study, we also observed that IOP increased following laryngoscopy and tracheal intubation. However, the degree of increase was significantly lower in the study group, 0.58+1.45 versus 4.69±1.65mmHg from the baseline with p value < 0.001. The higher IOP reading in control group was also noted in a similar study by Agbamu and co workers.[34] They found an increase of 4.5mmHg in IOP following laryngoscopy and tracheal intubation following the administration of suxamethonium. The higher increase in IOP from laryngoscopy and tracheal intubation is likely due to the haemodynamic responses associated with laryngoscopy and tracheal intubation with resultant increase in BP. This leads to increase in blood flow to the ciliary vessels, thus increase in IOP.

The findings from the control group of this present study compares well with the findings of Murphy et al[28], where they employed lidocaine as pretreatment as against $MgSO_4$ we used in our study. They found no significant statistical difference between lidocaine group and placebo group. The explanation for their finding was that, they observed that tracheal intubation was performed under light anaesthesia induced by STP. The resultant haemodynamic responses may have contributed to the increase in IOP they noted in their study.

The degree of increase in IOP of 4.69mmHg in the control group of this study obviously would be deleterious in an open eye. In the case report by Amadasun and Isessele,[16] the authors opined that loss of eye content observed in their patient may be from laryngoscopy and tracheal intubation rather than from suxamethonium administration. This obvious increase of IOP from tracheal intubation may support the views of many authors that believe that the increase in IOP from suxamethonium cannot lead to extrusion of eye contents. However, considering the fact that any increase of IOP is a significant cause for extrusion of eye content in the open eye, it is pertinent to prevent IOP increase as much as possible.

In our study, $MgSO_4$ minimized but did not completely abolish increase in IOP increase from laryngoscopy and tracheal intubation. This may be due to the dose (30mg/kg body weight) of $MgSO_4$ used in this study. A higher dose may completely prevent the increase of IOP from laryngoscopy and intubation. However, a higher dose may be associated with side effects, although, some studies where 40- 60mg/kg of $MgSO_4$ were used did not report any sign of hypermagnesemia or clinically significant rise in serum magnesium levels.[35,36].

There was a mean decrease of 4.06b/m in the PR from the baseline after study drug administration and induction of anaesthesia in our study, although it was clinically insignificant. In a normal heart, pulse rate (heart rate) is generated from the SA node which also depends on calcium efflux during excitation.[37] One of the mechanisms of action of $MgSO_4$ is preventing the release of calcium.[38] Thus, it slows the rate of impulse formation at the SA node and prolongs impulse conduction at the SA node.[37,39] This could have accounted for the decrease in PR in the study group though clinically insignificant in this study.

This is in contrast to the finding of Dilip et[29] al where they noticed a clinically significant increase in PR from base line after injection of $MgSO_4$. It is noteworthy that in their study, $MgSO_4$ was injected over two minutes without being diluted. This could have caused some irritation/pain to the patient and thus reactionary increase in PR.

Following suxamethonium administration, the mean SBP increased in both groups. However, the increase was minimal in the study group compared to the control group. One of the mechanisms of actions of $MgSO_4$ is decrease in catecholamine release,[43,44] Suxamethonium on the other hand is known to increase catecholamine release.[42] It is thus expected that the effect of each drug on the release of catecholamine balances out, which may explain the minimal rise

in SBP observes in the study group. The decrease in SBP in the study group is both clinically and statistically significant with p value of 0.001.

The above finding has also been demonstrated by Yap and colleagues[45] when they studied the effect of magnesium sulphate pretreatment on succinylcholine facilitated tracheal intubation. They found out that $MgSO_4$ caused a decrease in SBP and attenuated the hypertensive response following suxamethonium administration and tracheal intubation. They used 60mg/kg of $MgSO_4$ to pretreat suxamethonium and also used fentanyl 2ug/kg and thiopentone for induction. These could have also combined to attenuate the rise in blood pressure.

The use of $MgSO_4$ as pretreatment for suxamethonium led to a decrease in Mean Arterial Pressure (MAP). However, induction of anaesthesia, further decreased the MAP in both groups which was clinically remarkable and comparable both from the baseline and the post study drug administration. The decrease therefore can be explained by the prevention of catecholamine release by $MgSO_4$ and the vasodilation and slight myocardial depression caused by the induction agent (propofol). In a study by Nooraei et al[46] where 60mg/kg of $MgSO_4$ and 1.5mg/kg of lidocaine were given before anaesthetic induction, it was noted that $MgSO_4$ reduced MAP, SBP up to the first two minutes better than the lidocaine group. This was similar to the finding of this study though higher dose of magnesium sulphate was used in their work.

After laryngoscopy and tracheal intubation, it took relatively less time for the IOP and haemodynamic parameters to return to baseline or near baseline in the study group when compared with the control group. The IOP, SBP, DBP and MAP reached the baseline value in less than two minutes in the study group while in the control group, the SBP, and MAP returned to the baseline after 5minutes and DBP after 2 minutes. In comparing this study to that of Malaya et al,[43] the heamodynamic parameters showed a statistical significant increase immediately after intubation followed by a significant reduction in 3rd, 5th, and 7th minutes post intubation. The baseline values were reached around the 5th/7th minute post intubation which was similar to the findings in this study.

No side effects like sweating, palpitation, flushing and warmness were recorded in this study. The low incidence of side effects/complications observed in this study might be due to the careful selection of patients and low dose and concentration of the study drug chosen, administration of the drug over a long time and strict adherence to safe anaesthetic practice. Nevertheless, the complications noted in this study may not be due to the study drug but to surgical and anaesthetic practice. These include hypotension which could be due to surgical blood loss and was managed with intravenous fluid including blood. In the control group, bradycardia accounted for 4.2% (2patients) which could probably be as a result of surgical manipulation or undiagnosed cardiac condition. The bradycardia was treated with atropine. Tachycardia and stridor accounted for 2.1% (a patient) each. The stridor was due to suspected tracheomalacia in a thyroid patient. The trachea was re-intubated and managed accordingly while the tachycardia was managed with fluids/blood, analgesia and deepening the anaesthesia.

Many studies have shown the use of $MgSO_4$ in the dose of 40-60mg/kg without any significant rise in serum magnesium level after infusion of $MgSO_4$ for many hours[35] while others have shone side effects at such doses. [43] In a study by Chestnut et al, [50] patients pretreated with 60mg/kg of $MgSO_4$ experienced significant warm sensation. The cause of the side effect here was likely due to the rapid administration of the drug and the higher dose/concentration used compared to the longer time of administration and lower dose/concentration used in this present study.

5. Conclusion

This study revealed that 30mg/kg of magnesium sulphate pretreatment minimized the increase in IOP following suxamethonium administration. It also reduced the haemodynamic response associated with laryngoscopy and tracheal intubation.

Of greater significance was the fact that the increase in IOP following suxamethonium was not significant compared to that following laryngoscopy and tracheal intubation? Therefore further research should be carried on the prevention of IOP following laryngoscopy and tracheal intubation.

There were minimal side effects associated with the dose of $MgSO_4$ used in this study.

Study Limitations

The occasional agitation, squeezing of eyes or movement by patients before induction when taking the IOP reading may have slightly affected the reading.

The wide range of patient's age may also have affected the IOP and haemodynamic reading as it was observed that some older patients had slightly higher reading. But this covers the larger population.

The limitations notwithstanding, the strength of the observation of this study adds value to the available evidence of preventing suxamethonium and endotracheal intubation induced rise in IOP in patients that have open penetrating eye injury with full stomach coming for emergency surgery.

Compliance with ethical standards

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Disclosure of conflict of interest

We declare no conflict of interest and no external funding was received towards the conduct of the study.

Statement of ethical approval

Ethical approval for the conduct of the study was sought and obtained from the Research and Ethics Committee of the University of Benin Teaching Hospital, Benin City, Nigeria

Statement of informed consent

Informed consent was obtained verbally from all the patients who agreed to participate in the study.

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