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Analysis of the effect of perioperative magnesium sulphate on minimal alveolar concentration of desflurane using bispectral index monitoring

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Abstract. Background. In this study we aimed to analyze the effect of perioperative magnesium sulphate (MgSO₄) on minimal alveolar concentration (MAC) of desflurane using bispectral index (BIS) monitoring. Patients and methods. Sixty patients undergoing abdominal surgery under general anesthesia were randomized into two groups: Mg – receiving perioperative MgSO₄ supplementation and C – control. Anesthesia was titrated to maintain the BIS value between 45-55. Results. MAC values, tachycardia and hypertension during intubation was found to be lower in group Mg compared to group C (p<0.001). Time to extubation, verbal cooperation and eye opening was longer in patients receiving infusion of MgSO₄ (p<0.001). Conclusion. We concluded that perioperative MgSO₄ infusion may be used as an adjunct as it decreases MAC of desflurane and suppresses the hemodynamic response to intubation.

 ${\bf Key}$ words: magnesium, desflurane, minimal alveolar consant ration, bispectral index

Magnesium (Mg) ion is the second most important intracellular cation and is the 4th cation in the whole human body [1]. Mg is involved in energy metabolism, protein and nucleic acid synthesis and directly influences muscle contraction, neuronal activity, vasomotor tonus control, cardiac excitability and transmitter release. It is also the physiological antagonist of calcium [2]. Mg is mainly found in endoplasmic reticulum and mitochondrial membranes. In the cell, the free form of Mg and the membrane bound form are in equilibrium. Mg acts on cell metabolism through regulation of intracellular calcium concentration. This regulation is achieved by activation of cytosolic calcium channels and competition with calcium at the transmembraneous calcium channels or protein kinases. Mg also regulates neurotransmitter release triggered by calcium [3].

Mg decreases the vascular resistance, pulmonary and systemic arterial pressures. Intubation induced stress and resultant tachycardia and hypertension results due to catecholamine release. Mg also decreases catecholamine release from adrenal medulla and adrenergic nerve endings [1]. Lack of Mg increases the risk of perioperative arrythmia [2, 3]. Additionally it has an antiaggregant effect by decreasing thrombocyte adhesion, aggregation and release of thromboxane [2]. It is used as an adjuvant in perioperative analgesia[1]. In a rat study, Mg infusion has resulted in a 60% decrease of the minimal alveolar concentration (MAC) value of halothane [4]. Perioperative addition of MgSO₄ to total intravenous anesthesia (TIVA) resulted in a marked decrease in the amount of drugs [5]. In another study, perioperative MgSO₄ resulted in a decrease

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in propofol consumption, suppression of the hemodynamical response to intubation and increase in time to extubation, spontaneous eye opening and cooperation [6].

To our knowledge no study investigated the effect of $MgSO_4$ over MAC value of desflurane. MAC is the dose of an inhaled anesthetic agent that prevents movement in 50% of patients in response to surgical stimulus. MAC is a useful measure because it mirrors partial pressure of inhaled anesthetic in brain tissue, allows comparisons of potency between agents, and provides a standard for experimental evaluations [7].

Desflurane is a fluorinated methyl ethyl ether. It has the lowest tissue/gas partition coefficient among the potent volatile anesthetics, resulting in a decreased potency. Due to its low fat solubility, desflurane requires less downward titration in long surgical procedures. Desflurane is associated with tachycardia and hypertension [8].

In this study we aimed to determine the effects of perioperative use of $MgSO_4$ on MAC value of desflurane and hemodynamic parameters using BIS monitoring.

Methods

Patients were enrolled in the study following local ethical committee approval (Medical Faculty of Yuzuncuyil University, Van, Turkey) and obtaining informed patient consents. ASA class I-II patients, 18-60 years of age scheduled for elective abdominal surgery (umbilical hernia, gastrointestinal, open cholecystectomy) were included in the study. Patients with cardiac insufficiency, ischemia, arrythmia, valvular heart disease, hypertension, renal failure, liver failure, obesity, pregnancy, electrolyte imbalance, alcohol abuse, sedative or antipsychotic drug use, untreated endocrine or metabolic disease, history of difficult intubation, asthma, chronic obstructive lung disease, allergy to drugs, opioid use or abuse were excluded. Sixty patients (24 male, 36 female) were randomised into two groups on the basis of the following baseline variables: age, sex, height and weight: Patients in group Mg received 30 mg/kg MgSO4 i.v loading dose in 10 minutes before induction of anesthesia and maintenance dose of 10 mg/kg/hour Mg until extubation. Patients in group C received the same amount of isotonic NaCl solution during the same period of time.

All patients received routine monitoring of electrocardiography (ECG), blood pressure (BP), heart rate (HR) and peripheral oxygen saturation (SpO_2) (Petaş-KMA 800 Model, Turkey). Depth of anesthesia was monitored using BIS values derived as average over every five min (A-2000 Aspect medical systems, USA). Forehead was cleaned with a sponge prior to application of BIS (Quatro) sensor. Preoperative serum total Mg levels were measured and 5% Dextrose in Ringer's lactate solution was started preoperatively.

All patients received propofol 2.5 mg/kg, fentanyl 2 µg/kg for induction without premedication. Muscle relaxation was obtained with single dose of vecuronium 0.1 mg/kg. After intubation, central body temperature, MAC and end tidal carbondioxide (ETCO₂) levels were additionally monitored and recorded throughout the operation every five minutes until extubation. During surgery, anesthesia was maintained with 40% $O_2+60\%$ N₂O and desflurane in all patients. Remifentanyl infusion was started five minutes after intubation at a dose of 0.15 µg/kg/min. 0.03 mg/kg vecuronium was administered every 30 minutes, taking care to administer last maintenance dose 30 minutes before the end of the operation. Desflurane dose was adjusted to keep the BIS value between 45-55 during surgery. The desflurane dose was started as calculated by the anesthesia device according to the age and body mass of each patient (Draeger primus SN: ARTL-0021, AG & CoKGaA, Germany). When BIS increased above 55, desflurane dose was raised 1% and when BIS fell below 45 desflurane dose was reduced 1%. When HR and mean arterial pressure decreased >20% of base value or when the systolic arterial pressure was < 90 mmHg, remifertanyl dose was gradually decreased. 0,01 mg/kg atropine was administered when bradycardia (HR <50/min) occured. The respiratory rate was set to 12/min, tidal volume to 8 ml/kg, PEEP to 3-4 cmH₂O in intermittant positive pressure ventilation (IPPV) mode.

Five minutes prior the end of the operation, desflurane, N₂O, MgSO₄ and remifentanyl infusion were stopped and the patient was ventilated manually with 100% O₂. The patients were decurarized with atropine and neostigmine when positive inspiratory pressure exceeded 10 cmH₂O and tidal volume exceeded 4 mL/kg. When the BIS value reached 80, the patients were extubated. Time to extubation, spontaneous eye opening and time to cooperation were recorded after stopping maintenance drugs. Patients in group Mg were neurologically examined to rule out hypermagnesemia before discharged from postanesthesia care unit. Serum total Mg levels were measured one hour and 24 hours after stopping MgSO₄ infusion.

Statistical analysis

The size of the sample was based on previous investigations that have been conducted about the effects of Mg sulphate and clonidine on propofol consumption, hemodynamics and postoperative recovery [7]. To determine a 15% difference in MAC of desflurane at an alpha error of 0.05 and a power of 80 %, we have calculated that 30 patients in each group would suffice. Results were analysed using SPSS for Windows version 15. The results were reported as mean±standard deviations (mean±SD) and median (range) or count. Distribution of test results was evaluated using the Kolmogorov-Smirnov test. Comparisons between groups were analysed using repeated measures analysis of variance (ANOVA). Post hoc analysis were done using independent t-test for between groups comparisons and paired t-test for within groups comparisons. Side effects and values expressed as percentile were analyzed using Chi-square test. P<0.05 was regarded as significant.

Results

Demographic data is shown in *table 1*. The groups showed no statistically significant difference in terms of age, weight, height, sex, ASA status, operation time and adverse events. Bradycardia was seen in 5 patients in group Mg and 4 in group C.

When groups were compared regarding mean arterial pressures (MAP), group C had significantly higher values following intubation (p < 0.001). In group Mg, preoperative MAP was higher than all other recordings (p < 0.001). Post Mg MAP was higher than the rest of the recordings (p < 0.01). In group C, the preoperative MAP was higher than the rest of the recordings except the immediate postintubation MAP (p < 0.001) (*table 2*).

When groups were compared regarding HR, group C had a higher increase after intubation and at 5 minutes compared to group Mg (p<0.001) (p<0.05). In group Mg, all intraoperative HR recordings were lower than the preoperative HR values (p<0.001). In group C, all intraoperative HR recordings were lower than the preoperative HR values except the one immedite post intubation (p<0.001) (table 2).

Although Mg levels at the first postoperative hour were higher compared to preoperative and postoperative 24^{th} hour values in group Mg (p<0.001), no patient showed clinical signs of hypermagnesemia (depression of respiration, weakness of deep tendon reflexes, hypotension, elongated PR interval in ECG). Mg levels at the first postoperative hour were higher in group Mg compared to group C (p<0.001) (table 3).

Perioperative MAC of desflurane was statistically significantly decreased in group Mg (p < 0.001). There was a significant decrease in the requirement of desflurane in group Mg (3.93% \pm 0.05%, mean \pm SD) compared to group

Table 1. Demographic data, duration of operations and side effects (mean±SD and count).

	Group Mg (n=30)	Group C (n=30)
Age (years)	$31.23{\pm}11.21$	$30.74{\pm}11.32$
Weight (kg)	$64.43{\pm}11.40$	$60.43{\pm}8.66$
Height (cm)	$166.10{\pm}8.44$	$164.17{\pm}5.70$
Sex (male/female)	17/13	19/11
Operation time(min)	$93.17{\pm}15.78$	$86.00{\pm}12.13$
ASA (I/II) Bradycardia	28/2 5	27/3 4

SD: standard deviation; kg: kilogram; cm: centimeter; min: minute.

Mg: Patients receiving perioperative MgSO₄.

C: Patients in the control group.

	Heart rates		Mean arterial pres	sures
Time	Group Mg (n=30)	Group C (n=30)	Group Mg (n=30)	Group C (n=30)
Preoperative	$87.26{\pm}13.42$	$82.96{\pm}8.86$	$93.53{\pm}7.90$	90.26 ± 10.26
After MgSO ₄ inf.	$88.80{\pm}13.03$	$82.96{\pm}8.86$	$88.06{\pm}12.20$	90.26 ± 10.26
Postinduction	$69.46{\pm}8.91$	$66.03{\pm}6.89$	$67.53{\pm}8.20$	66.40 ± 6.10
Postentubation	$76.76{\pm}9.51$	$90.40{\pm}8.64^{**}$	$86.10 {\pm} 9.72$	$96.56 \pm 9.55^{**}$
5 th min	$67.90{\pm}9.68$	$72.46{\pm}7.53{*}$	$75.53{\pm}13.14$	80.46 ± 11.46
10 th min	$65.70 {\pm} 9.24$	$68.50{\pm}7.30$	$72.56{\pm}14.39$	74.40 ± 10.94
15 th min	$66.30{\pm}10.23$	$67.63 {\pm} 8.42$	$73.80{\pm}13.87$	74.96 ± 9.75
20 th min	$64.66{\pm}10.61$	$68.10{\pm}10.61$	$74.83{\pm}14.79$	78.73 ± 11.39
25 th min	$67.16{\pm}10.91$	$67.53{\pm}11.02$	$78.20{\pm}14.49$	77.53 ± 11.66
30 th min	$67.40{\pm}11.38$	$67.56{\pm}10.99$	$77.83{\pm}15.20$	80.03 ± 11.79
35 th min	$68.33{\pm}11.41$	$68.60{\pm}11.82$	$78.73{\pm}13.07$	80.40 ± 14.07
40 th min	67.23 ± 10.90	$69.46{\pm}11.00$	$79.10{\pm}11.29$	81.70 ± 13.15
$45^{ m th}$ min	$67.10{\pm}10.02$	$68.66{\pm}10.48$	$79.56{\pm}12.70$	81.23 ± 12.98
50 th min	$67.00 {\pm} 9.86$	$68.73{\pm}9.71$	$80.43{\pm}14.88$	79.80 ± 11.50
55^{th} min	$66.66 {\pm} 9.17$	$68.36{\pm}9.75$	$77.86{\pm}14.06$	78.90 ± 9.75
60 th min	$66.60{\pm}8.83$	$68.23{\pm}9.46$	$77.83{\pm}12.51$	79.93 ± 11.38
$65^{ m th}~{ m min}$	$67.43 {\pm} 9.67$	$68.03{\pm}9.51$	$79.10{\pm}12.62$	79.10 ± 9.03
70 th min	$66.76{\pm}8.83$	$68.03 {\pm} 8.76$	$78.26{\pm}12.97$	$78.10{\pm}10.14$
75 th min	$66.66 {\pm} 8.85$	$68.50{\pm}7.76$	$78.59{\pm}12.47$	$81.46{\pm}10.49$
80 th min	$66.70 {\pm} 9.33$	$67.00{\pm}7.27$	$79.29{\pm}12.16$	$79.41{\pm}10.23$
85 th min	$65.80{\pm}9.68$	$65.92{\pm}8.73$	$79.04{\pm}12.21$	$80.14{\pm}8.71$
90 th min	$64.72{\pm}8.75$	$67.20 {\pm} 9.82$	$76.66{\pm}11.73$	$83.10{\pm}9.17$
95 th min	$64.56{\pm}8.80$	$67.33{\pm}4.63$	$76.31{\pm}13.78$	$81.50 {\pm} 9.77$

Table 2.	Mean	arterial	pressures	and	heart	rates of	f the	groups	$(mean \pm SD).$
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* p<0.05, ** p<0.001. Min: minute; SD: standard deviation.

Table 3. M	Iagnesium	concentrations	(mg/dL)	of the patients.
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Time	Group Mg (n=30)	Group C (n=30)
Preopetrative	1.82(1.45-2.50)	1.92(1.42 - 2.58)
Postoperative 1 st hour	$3.18\left(2.38\text{-}3.90 ight)^{**}$	1.80(1.41-2.35)
Postoperative 24 th hour	1.91 (1.60-2.89)	1.84(1.58-2.45)

Data are expressed as median (range). ** p<0.001.

C (4.89% \pm 0.02%, mean \pm SD) (p<0.001) (table 4). Administering these amounts, there was no difference between groups regarding BIS values (group Mg: 48.64 \pm 0.35, group C: 48.10 \pm 0.54, mean \pm SD) (p>0.05) (*table 4*). When groups were compared regarding esophageal temperature, there was a statistically significant difference at 85^{th} minute only (p>0.05).

Esophageal temperatures decreased in both groups as time passed (p < 0.05). The time to extubation, cooperation and eye opening was increased in group Mg (p < 0.001) (table 5).

Time	MAC values		BIS values	
	Group Mg (n=30)	Group C (n=30)	Group Mg (n=30)	Group C (n=30)
Preoperative	-	-	97.23±0.72	96.63 ± 5.44
After MgSO ₄ inf.	-	-	$95.63{\pm}2.35$	$96.63{\pm}5.44$
Postinduction	-	-	$41.60{\pm}3.21$	$41.73{\pm}2.66$
Postentubation	-	-	$49.90{\pm}2.24$	$50.16{\pm}1.83$
5 th min	$0.82{\pm}0.04^{**}$	$0.96{\pm}0.08$	$48.90{\pm}1.86$	$49.13{\pm}2.02$
10 th min	$0.82{\pm}0.06^{**}$	$0.97{\pm}0.07$	$48.63{\pm}2.09$	$48.30{\pm}1.87$
15 th min	$0.83{\pm}0.07{**}$	0.98 ± 0.06	$48.86{\pm}1.88$	$48.10{\pm}1.64$
20 th min	$0.84{\pm}0.06^{**}$	$0.98{\pm}0.05$	$48.70{\pm}1.66$	$48.53{\pm}2.21$
25 th min	$0.82{\pm}0.06^{**}$	$0.97{\pm}0.05$	$48.53{\pm}2.21$	$48.40{\pm}2.01$
30 th min	$0.82{\pm}0.07{**}$	$0.97{\pm}0.05$	$48.50{\pm}2.02$	$48.23{\pm}2.29$
35 th min	$0.83{\pm}0.05{^{**}}$	$0.98{\pm}0.05$	$49.46{\pm}1.81$	$48.73{\pm}1.68$
40 th min	$0.83{\pm}0.06{**}$	$0.99{\pm}0.05$	$49.13{\pm}2.04$	$48.56{\pm}1.77$
45 th min	$0.84{\pm}0.05^{**}$	$0.98{\pm}0.04$	$48.86{\pm}1.94$	$48.63{\pm}2.04$
50 th min	$0.83{\pm}0.05{**}$	$0.98{\pm}0.05$	$48.53{\pm}1.45$	$48.20{\pm}1.88$
55 th min	$0.83{\pm}0.05{**}$	$0.99{\pm}0.05$	$48.46{\pm}1.67$	$48.16{\pm}1.41$
60 th min	$0.83{\pm}0.05{**}$	$0.98{\pm}0.05$	$48.26{\pm}1.65$	$48.03{\pm}1.51$
65 th min	$0.83{\pm}0.05{**}$	$0.98{\pm}0.06$	$48.96{\pm}1.65$	$48.30{\pm}1.29$
70 th min	$0.83{\pm}0.05{**}$	$0.97{\pm}0.06$	$48.53{\pm}1.88$	$47.90{\pm}1.49$
75 th min	$0.82{\pm}0.05{**}$	$0.97{\pm}0.05$	$48.51{\pm}1.71$	$48.21{\pm}1.61$
80 th min	$0.82{\pm}0.06^{**}$	$0.96{\pm}0.05$	$48.79{\pm}1.74$	$48.00{\pm}1.61$
85 th min	$0.82{\pm}0.07^{**}$	$0.97{\pm}0.05$	$48.09{\pm}2.09$	$47.71{\pm}1.58$
90 th min	$0.81{\pm}0.08^{**}$	$0.99{\pm}0.05$	$48.05{\pm}2.09$	$46.90{\pm}1.52$
95 th min	$0.83{\pm}0.07{**}$	$0.98{\pm}0.04$	$48.62{\pm}1.78$	$47.16{\pm}1.60$

Table 4. Mean perioperative MAC values of desflurane and BIS values (mean±SD).

** p<0.001. Min: minute; SD: standard deviation.

Table 5. Times of extubation, spontaneous eye opening and cooperation (mean±SD).

	Group Mg (n=30)	Group C (n=30)
Extubation time (min)	$6.83{\pm}1.01^{**}$	$5.89{\pm}1.14$
Time to eye opening (min)	$7.92{\pm}1.58^{**}$	$6.95{\pm}1.30$
Cooperation time (min)	$9.78{\pm}1.68{}^{**}$	$8.24{\pm}1.34$

Min: minute; SD: standard deviation. ** p<0.001.

Discussion

Mg has been used in anesthesia and intensive care units because of its suppressive effect on hypertension and tachycardia which may occur after intubation and pheochromocytoma surgery and its antiarrythmic properties [9]. It also decreases the use of anesthetic and analgesic drugs [1]. In our study, the aim was not to evaluate Mg as an anesthetic agent but to find out whether Mg could reduce MAC of desflurane during general anesthesia. Mg itself is not an anesthetic agent but is an adjunct to anesthesia. Theoretically, Mg can modulate anesthesia with several mechanisms. One of these is the antagonizing effect on NMDA receptors in the central nervous system [5, 10, 11]. Many NMDA antagonists (ketamine, phencyclidine, etc.) reduce the MAC value of inhalation anesthetics *in vivo* [12]. Volatile anesthetic effect on NMDA receptors can be potentiated significantly by Mg, ketamine, or most profoundly by both [1]. The analgesic effects of ketamine and Mg are likely to be enhanced in the presence of volatile anesthetics [13]. Another mechanism is the suppressive effect on surgical or anesthetic stress or peripheral nociceptive sensitivity which leads to decreased catecholamine release [5]. Intrathecal administration of NMDA receptor antagonists inhibit nociceptive stimuli but these mechanisms fail to explain why there is a decreased need for anesthetic and analgesic drugs [12].

 $MgSO_4$ can also pass through the blood brain barrier in small quantitites. Choi and colleagues showed correlation between Mg concentration in the cerebrospinal fluid and plasma in preeclemptic case managed with 6 g MgSO₄ i.v. bolus and 2 g/h infusion [12]. There are two interesting studies in the literature commenting about ability of Mg to enter the brain tissue. Wong and colleagues studied 22 subarachnoidal hemorrhage cases with or without Mg infusion to compare cerebrospinal fluid Mg levels.

They showed that the cerebrospinal fluid Mg level was increased by 11-21%, which was sustained for nine days. Although the investigators were expecting a doubling in the concentration, the administered Mg may pass to the brain tissue, therefore not reflecting the intracellular Mg levels [14]. A later study by Wong and colleagues studied intracellular free Mg of brain tissue levels using ³¹P-Magnetic Resonance Spectroscopy. They showed that hypermagnesemic treatment produced a significant elevation in intracellular free Mg levels [15]. These studies showed that administration of intravenous Mg may penetrate the brain tissue through cerebrospinal fluid. To determine specific dosage requirements, dose response curve studies will be required.

Lately BIS monitoring has been used to detect depth of anesthesia and level of sedation [16-18]. Gant *et al.* [19] have reported that balanced anesthesia and BIS monitoring led to a decrease in propofol use and that BIS monitoring may be useful in intraoperative propofol dose adjustment. Telci and colleagues [5] applied total intravenous anesthesia using propofol, keeping BIS between 45-60, and administered 30 mg/kg MgSO₄ 10 minutes before induction and infused 10 mg/kg/hr MgSO₄ during the operation. They found a marked decrease in the amounts of propofol, remifentanyl and vecuronium used during the operation. Thompson *et al.* [4] showed a 60 % decrease of the MAC value of halothane in relation to the plasma level of Mg (without hypoxia, hypotension and hypothermia) in their rat study and concluded that the use of Mg resulted in a decrease in the need for anesthetic drugs. Additionally, prolonged hypomagnesemia was shown to increase volatile anesthetic MAC in rats [20].

Choi et al. [12] used propofol-N₂O anesthesia and infused 50 mg/kg bolus and 8 mg/kg/h MgSO₄ and found a significant decrease in propofol use. Seyhan et al. [21] studied 80 women who underwent elective hysterectomy and compared different doses of MgSO₄ in propofol anesthesia keeping BIS between 45-55. They reported that the group receiving 40 mg/kg bolus and 10 mg/kg/h MgSO₄ infusion needed less intraoperative propofol. atracurium and postoperative morphine. The increase in Mg dose did not decrease the need for drugs but further suppressed the hemodynamical response. Durmus et al. [22] induced their patients with %7 sevoflurane and 6 L/minutes 100% O₂ and analyzed the effects of different doses of $MgSO_4$ on the hemodynamical response and the MAC values during endotracheal intubation and surgical incision. They found that tachycardia and hypertension during intubation and surgical incision was less in MgSO₄ group. The MgSO₄ group had higher MAC values only during surgical incision. In our study MgSO₄ administration led to a significant decrease in MAC values of desflurane.

Excess Mg is rapidly eliminated by kidneys. Therefore Mg should be given *via* parenteral route to obtain therapeutic serum levels typically required in the control of preeclampsia (2-4 mmol/L) The required initial dose of MgSO₄ is in the order of 40-60 mg/kg i.v. and continuous infusion should be at a rate of 15-30 mg/kg/h i.v.[3]. The patients receiving a lesser dose of MgSO₄ in our study reached serum concentrations of 2.38-3.90 mg/dL and showed no sign of hypermagnesemia. Remifentanyl decreases blood pressure and heart rate in a dose related manner [23]. In our study we used atropine in 5 patients in group Mg and in 4 patients in group C.

In all stages of surgical anesthesia the increase in heart rate and blood pressure resulting from catecholamine secretion may be a sign of pain. Mg is an important regulator of blood catecholamine levels, particularly epinephrine and norepinephrine [24].

In a comparative study in which the patients were induced with tiopenthal and succinylcholine,

the group which received 60 mg/kg MgSO₄ had less increase in blood pressure and heart rate than group C which received the same volume of %0.09 NaCl [25]. Atlan and colleagues [6], showed that preinduction 30 mg/kg bolus and intraoperative 10 mg/kg/h MgSO₄ suppressed the hemodynamical response to intubation. We used the same dose and got similar results. Also we found that the SpO2 levels following bolus mangesium injection before intubation were lower than the controls. This may be attributed to the sedative effect of Mg [22].

Mg has minimal postsynaptic effect at the neuromuscular junction but it potentiates the effect of nondepolarizing neuromuscular blockers by competing with calcium ions in the synaptic area and blocking acethylcholine release [3]. Fuchs-Buder and colleagues [26] reported that 40 mg/kg MgSO₄ i.v. decreases the effective dose of vecuronium by 25%, decreases the effecting time by half and increases the recovery period twice due to its presynaptic effect. Altan and colleagues [6] used 30 mg/kg bolus and 10 mg/kg/hr infusion of i.v. MgSO₄ in patients who were anesthetized with propofol and cisatracurium. They reported that the time to extubation, verbal response and orientation were longer in group Mg compared to group C. We also found increased time to extubation, spontaneous eye opening and cooperation in group Mg and attributed this to the potentiating effect of Mg on the effect of nondepolarizing neuromuscular blockers. In our study the MAC values of desflurane were found lower in group Mg.

Main limitation of the study was lack of serial perioperative Mg measurements. This was due to short and uniform operation durations. Future studies should include frequent perioperative measurements of Mg levels to better correlate the hemodynamic status and anesthesia level of the patient.

In conclusion, perioperative i.v. $MgSO_4$ may be used as an adjunct to general anesthesia as it decreases the MAC value of desflurane and stabilizes hemodynamic response during induction. Hovewer, the depth of anesthesia should be monitored by BIS and it should be kept in mind that $MgSO_4$ may prolong recovery from anesthesia.

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Disclosure

None of the authors has any conflict of interest or financial support to disclose.

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