EFFECT OF INTRAVENOUS MAGNESIUM SULPHATE ON CARDIOVASCULAR RESPONSES DURING TRACHEAL EXTUBATION IN PATIENT UNDERGOING CRANIOTOMIES

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ABSTRACT

BACKGROUND
During emergence of general anaesthesia, hypertension and tachycardia caused by tracheal extubation may lead to serious complications during craniotomies. This study was designed to assess the effect of magnesium sulphate on these cardiovascular responses during extubation following craniotomies under general anaesthesia.

MATERIALS AND METHODS
Sixty patients of ASA Grade I and II, aged 18 - 50 yrs. undergoing craniotomies for nonvascular ICSOL were randomised into 2 groups with 30 patients in each group. Patients in Group M received an IV infusion of magnesium sulphate 30 mg/kg and Group C received 100 mL normal saline at the time of skin closure over 10 mins. Heart rate, systolic and diastolic blood pressure were recorded prior to drug infusion, 3 and 5 mins after drug infusion, during extubation and at 3, 5, 10 and 15 minutes after extubation. Side effects like bradycardia, hypotension, nausea, vomiting, shivering and desaturation also noted. Statistical analysis was performed by the use of Student’s ‘T’ test and chi-square test.

RESULTS
As compared with baseline heart rate and blood pressure increase in both groups during and after extubation, heart rate was lower in Group M than Group C during extubation till 15 minutes after extubation (p < 0.05). In Group M, systolic blood pressure was lower from 3 mins after study drug infusion to 10 mins after extubation and diastolic blood pressure was lower during extubation till 10 mins after extubation (p < 0.05). The groups did not differ significantly in regard to the prevalence of adverse effects.

CONCLUSION
We conclude that using of magnesium sulphate before extubation decreases the undesirable cardiovascular responses during extubation period.

KEYWORDS
Cardiovascular Response, Extubation, Magnesium Sulphate, Craniotomy.

MATERIALS AND METHODS
After obtaining Ethics Committee approval this prospective, randomised, double-blind clinical study was carried out on 60 patients after getting written informed consent from the patients.

Inclusion Criteria
- Patients of ASA Grade I and II.
- Patients of age group 18 to 50 years of either sex.
- Patients undergoing craniotomies for nonvascular ICSOL under GA.

Exclusion Criteria
- Patients with cardiopulmonary diseases.
- Patients on antihypertensive, antiarrhythmic, adrenoreceptor agonist and antagonist.
- Patients with renal dysfunction and hepatic dysfunction.
- Pregnant and lactating patients.
- Any patient who required postoperative ventilation.
- Patients with psychiatric illness.

Pre-anaesthetic assessments of all the selected patients were done with complete history and physical examination. Routine investigations like complete blood count, blood sugar, blood urea, serum creatinine, chest x-ray and ECG were done.

Randomisation
Based on a computer generated random number table using Microsoft Excel, all the patients were randomised into 2 groups (Group M and Group C) of 30 patients each.

All the patients were kept nil orally for 6 hours before procedure. All patients were uniformly pre-medicated with Inj. glycopyrrolate 0.2 mg IM 30 mins before shifting to operation theatre.

Upon arrival of the patient in the operation theatre, intravenous access with 18-G cannula was established. Crystalloid (Normal saline) infusion was started. Various monitoring devices like NIBP, pulse oximeter, 3 leads ECG were connected and basal heart rate (beats per minute), systolic and diastolic blood pressure (SBP and DBP) (mmHg), SpO2 (%) were recorded.

Patients were medicated with Inj. pentazocine IV 0.5 mg/kg followed by preoxygenation with 100% oxygen for 3 minutes. Induction of general anaesthesia was done with Inj. thiopentone sodium 5 mg/kg BW. Endotracheal intubation was facilitated with intravenous succinylcholine 1.5 mg/kg BW and ventilation with 100% oxygen for 1 minute.

General anaesthesia was maintained with nitrous oxide and oxygen (66:33) and isoflurane (0.5 - 1%) given by Bain’s circuit with intermittent dosage of non-depolarising muscle relaxant IV vecuronium loading dose- 0.04 mg/kg and intermittent dose- 0.01 mg/kg throughout surgical procedure.

At the time of skin closure, isoflurane was discontinued. In Group M 30 mg/kg inj. magnesium sulphate in 100 mL normal saline and in Group C normal saline 100 mL were given over a period of 10 minutes. Residual muscle paralysis was reversed with inj. neostigmine (0.05 mg/kg) and inj. glycopyrrolate (0.01 mg/kg) IV. Once patient was conscious and responded to verbal command, extubation was performed and all patients were given O2 by face mask during recovery period. Values for heart rate, SBP and DBP were recorded just before the study drug administration (A0), which was taken as baseline value for comparison and 3, 5 mins after the study drug administration (A3 and A5), at extubation (E) and at 3, 5, 10 and 15 mins after extubation (E3, E5, E10, E15).

Patients were closely observed for bradycardia (below 20% of basal value), hypotension (below 20% of basal value) and desaturation (< 85%) during intra- and post-operative period. During postoperative period, along with the above nausea, vomiting and shivering were also recorded if occurred. Any complication if occurred was treated with appropriate medications.

With the power of study being 80% and confidence limits at 95%, minimum sample size required to detect 30% reduction in haemodynamic parameters was 24 patients in each group. We conducted study with 30 patients in each group to make it more authentic. The observations recorded in all the groups were tabulated and statistical analysis carried out by using appropriate statistical software SPSS 17. To compare adverse effects, chi-square statistical test and to compare repeated haemodynamic parameters Student’s ‘t’ test was used. P value < 0.05 was taken statistically significant and p value < 0.001 was taken to be statistically highly significant.

RESULTS
The groups were well matched for age, male: female ratio, weight and duration of anaesthesia. The statistical difference was insignificant (p > 0.05).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group C</th>
<th>Group M</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs.)</td>
<td>38.26 ± 10.65</td>
<td>34.7 ± 9.01</td>
</tr>
<tr>
<td>Sex (male:female)</td>
<td>14:16</td>
<td>16:14</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>60.63 ± 9.74</td>
<td>63.4 ± 9.10</td>
</tr>
<tr>
<td>Duration of Anaesthesia (min)</td>
<td>176.66 ± 38.10</td>
<td>176.83 ± 35.82</td>
</tr>
</tbody>
</table>

Table 1. Demographic Profile of 2 Groups

Baseline heart rate, SBP and DBP were comparable in both groups. Heart rate was significantly higher in Group C than Group M during extubation till 15 mins after extubation (p < 0.05).

SBP was significantly higher in Group C than Group M at 3 and 5 mins after study drug administration, during extubation and at 3, 5 and 10 mins after extubation (p < 0.05).

DBP was higher in Group C during extubation and at 3, 5 and 10 mins after extubation (p < 0.05).

Nausea and shivering were found in 1 and 2 patients respectively in Group C. No other side effects or complications were observed in both groups. The difference was statistically insignificant (p > 0.05).

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Time in min.</th>
<th>Group C</th>
<th>Group M</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A0</td>
<td>80.83 ± 11.45</td>
<td>77.60 ± 9.49</td>
<td>.239</td>
<td></td>
</tr>
<tr>
<td>A3</td>
<td>81.63 ± 10.98</td>
<td>78.80 ± 8.76</td>
<td>.274</td>
<td></td>
</tr>
<tr>
<td>A5</td>
<td>83.10 ± 11.17</td>
<td>80.93 ± 8.68</td>
<td>.40</td>
<td></td>
</tr>
<tr>
<td>E</td>
<td>104.03 ± 17.65</td>
<td>94.26 ± 12.01</td>
<td>.015</td>
<td></td>
</tr>
<tr>
<td>E3</td>
<td>100.03 ± 17.02</td>
<td>89.83 ± 12.80</td>
<td>.011</td>
<td></td>
</tr>
<tr>
<td>E5</td>
<td>96.63 ± 16.08</td>
<td>87.40 ± 12.72</td>
<td>.017</td>
<td></td>
</tr>
<tr>
<td>E10</td>
<td>92.20 ± 14.39</td>
<td>84.86 ± 12.55</td>
<td>.04</td>
<td></td>
</tr>
<tr>
<td>E15</td>
<td>90.50 ± 11.24</td>
<td>82.66 ± 12.95</td>
<td>.015</td>
<td></td>
</tr>
</tbody>
</table>

Table 2. Haemodynamic Parameters in the Study Groups
Intravenously administered magnesium sulphate is capable of attenuating vasopressin stimulated vasoconstriction response to sympathetic stimulation. Although precise mechanism responsible for these changes are not known, possible stimuli may be emerging from anaesthesia, tracheal irritation and wound pain.1 There is release of catecholamine occurring during this stressful period. These haemodynamic changes has a major concern for patients with coronary artery disease,3 cerebrovascular disease12 and in hypertensive patients.13

Magnesium inhibits the release of catecholamine from both adrenal glands and adrenergic nerve terminals in response to sympathetic stimulation.14 It is also capable of attenuating vasopressin stimulated vasconstriction.15 Intravenously administered magnesium sulphate is capable of attenuating the adverse haemodynamic response associated with endotracheal intubation.10

In this study, we used magnesium sulphate before extubation to find out the attenuating effect on cardiovascular system response during tracheal extubation following craniotomies. We found in our study that heart rate was increased at 5 mins after magnesium sulphate infusion. This was probably due to the fact that Mg²⁺ inhibits the release of acetylcholine from the vagus nerve, so it produces tachycardia initially. Heart rate was higher in control group as compared to magnesium sulphate group during and after extubation. This was probably due to the fact that epinephrine levels in the magnesium sulphate group did not increase as significant, whereas in the control group there was a significant increase in epinephrine level.

Arar C et al16 support our study, they also used magnesium sulphate during extubation and found that heart rate was significantly lower in magnesium sulphate group as compared to control group. Montazeri K and Fallah M17 compared 5 doses of magnesium sulphate (50, 40, 30, 20 and 10 mg/kg IV) and IV lignocaine 1.5 mg/kg for attenuation of haemodynamic response to endotracheal intubation. They concluded that 30 mg/kg dose is optimum dose for haemodynamic stability with less unexpected effects during intubation period.

Above mentioned studies supported the use of safe dose of IV magnesium sulphate (30 mg/kg), which was similar to our study with no incidence of adverse effects. James MFM et al18 Kiaee MM et al19 and Pipali G et al20 also used IV magnesium sulphate for stabilisation of heart rate during intubation and found similar result.

Mg²⁺ directly acts on blood vessels and also indirectly by sympathetic ganglia blockade, so it leads to decrease in arterial blood pressure.10 It reduces responsiveness of vascular smooth muscle to norepinephrine stimulation. It competes with calcium for membrane channels. From our study, it was observed that SBP was lower in Group M compared to control group at all time from 3 mins of study drug infusion to 10 mins after extubation, whereas DBP was lower during extubation till 10 mins after extubation.

In a study on hypertensive patients by Panda NB et al,21 magnesium sulphate infusion at a dose of 30, 40 and 50 mg/kg were given at induction of anaesthesia. Blood pressure was maintained within normal limit with 30 mg/kg magnesium sulphate and intervention required for hypotension in patients receiving 40 and 50 mg/kg of magnesium sulphate. They concluded that 30 mg/kg magnesium sulphate was better for stabilisation for haemodynamics during intubation.

Our results are in accordance with Arar C et al,16 Nooraei N et al22 and Puri GD et al.10

Shivering (6.66%) and nausea (3.33%) were observed only in control group patients. Beside these, no untoward side effects occurred in any group. The difference was statistically insignificant between groups (p > 0.05).

Pipali G20 also had done a study with magnesium sulphate and found similar results.

**CONCLUSION**

Magnesium sulphate administration before the extubation significantly decreases the cardiovascular response to tracheal extubation.

Table 3. Haemodynamic Parameters in the Study Groups

<table>
<thead>
<tr>
<th>Time in min.</th>
<th>Group C</th>
<th>Group M</th>
<th>P-value</th>
<th>Group C</th>
<th>Group M</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A0</td>
<td>123.83 ± 9.40</td>
<td>123.96 ± 11.08</td>
<td>.96</td>
<td>76.86 ± 8.90</td>
<td>78.70 ± 9.99</td>
<td>.456</td>
</tr>
<tr>
<td>A3</td>
<td>124.86 ± 9.53</td>
<td>118.63 ± 11.06</td>
<td>.023</td>
<td>77.96 ± 8.37</td>
<td>74.60 ± 9.70</td>
<td>.156</td>
</tr>
<tr>
<td>A5</td>
<td>126.60 ± 11.42</td>
<td>119.70 ± 11.35</td>
<td>.022</td>
<td>78.70 ± 8.22</td>
<td>76.10 ± 10.93</td>
<td>.302</td>
</tr>
<tr>
<td>E</td>
<td>145.93 ± 8.25</td>
<td>135.56 ± 12.10</td>
<td>.000</td>
<td>95.73 ± 9.36</td>
<td>88.40 ± 10.09</td>
<td>.005</td>
</tr>
<tr>
<td>E5</td>
<td>141.13 ± 7.41</td>
<td>132.43 ± 11.47</td>
<td>.001</td>
<td>92.26 ± 9.02</td>
<td>85.50 ± 10.47</td>
<td>.01</td>
</tr>
<tr>
<td>E10</td>
<td>136.73 ± 6.95</td>
<td>129.50 ± 11.37</td>
<td>.004</td>
<td>89.63 ± 7.71</td>
<td>83.63 ± 9.97</td>
<td>.012</td>
</tr>
<tr>
<td>E15</td>
<td>132.70 ± 6.75</td>
<td>127.10 ± 11.02</td>
<td>.021</td>
<td>86.03 ± 6.77</td>
<td>80.36 ± 9.75</td>
<td>.011</td>
</tr>
<tr>
<td>E10</td>
<td>128.96 ± 8.05</td>
<td>124.60 ± 11.08</td>
<td>.006</td>
<td>82.50 ± 6.80</td>
<td>78.30 ± 10.77</td>
<td>.07</td>
</tr>
</tbody>
</table>

Measurement points: A0: During study drug administration, A3: 3 minutes after drug administration, A5: 5 minutes after drug administration, E: At the time of extubation, E5: 3 minutes after extubation, E15: 5 minutes after extubation, E10: 10 minutes after extubation, E15: 15 minutes after extubation, M = Magnesium Sulphate, C = Control, SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure
REFERENCES


