Comparison of Effects of Intravenous Lignocaine and Magnesium Sulphate on the Cardiovascular Response to Laryngoscopy and Intubation

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ABSTRACT

Background and Objective: The patient's heart rate, blood pressure, pulmonary arterial pressure, and capillary wedge pressure will all rise during laryngoscopy and tracheal intubation. These hemodynamic alterations are less noticeable in those with normal blood pressure but have been linked to several diseases. They are well tolerated by those with normal blood pressure. The study's goal is to compare the cardiovascular effects of lignocaine and magnesium sulfate during laryngoscopy and intubation.

Methodology: The objective of this randomized controlled trial was to examine the effects of lignocaine and magnesium sulphate on cardiovascular and respiratory responses after endotracheal intubation in ASA I patients. The 60 participants' ages ranged from 15 to 45, and they were all classed as "grade I" by the ASA. Each of these patients underwent elective surgery under general anesthesia voluntarily. The sixty people were divided into two groups of thirty using a random selection method. Neither group was significantly different from the other in terms of demographics or blood-flow parameters. Group L was administered 1.5 mg/kg intravenously of lidocaine three minutes before to induction. Group M received an intravenous injection of magnesium sulphate at a rate of 40 mg/kg over the course of one minute.

Results: In both the Magnesium and Lignocaine groups, heart rates increased, but the Lignocaine group's increase was far more dramatic (p value 0.01). Both groups experienced an increase in systolic blood pressure following medication administration (p value 0.05). The results of Group M remained statistically significant for the first minute after the insertion of the tube, but by the fifth minute, they had returned to pre-insertion levels. In Group L, blood pressure increased significantly at 1 and 3 minutes after tube insertion, but by 5 minutes, blood pressure had returned to pre-insertion levels. The SBP of group L individuals increased significantly greater than that of group M participants. (p 0.05). Immediately following intubation, there was a statistically significant (p 0.01) increase in DBP in both groups; however, three minutes later, DBP had returned to pre-intubation levels in both groups (p 0.01). It is five percent as important. (p 0.05). The results revealed that the growth rate of group L members was significantly higher than that of group M members. In the presented situation, p equals 0.01

Conclusion: When it comes to preventing a rise in heart rate, blood pressure, and blood pressure during laryngoscopy and tracheal intubation for patients with an ASA grade I, magnesium sulphate is more beneficial than lidocaine.

INTRODUCTION

One of the steps in administering general anesthesia is performing an endotracheal intubation and laryngoscopy. It is common knowledge among doctors that laryngoscopy and endotracheal intubation are trigger hemodynamic effects. When doing a laryngoscopy, the autonomic nervous system is aroused, causing the larynx, pharynx, and trachea to respond. These modifications may have unfavorable effects on people, especially those who are already vulnerable due to preexisting diseases including ischemic heart disease, cerebrovascular sickness, high blood pressure, old age, or type 2 diabetes. Many studies have been conducted to find ways to stop this stress reaction, but none of them have been wholly successful. Magnesium sulphate and lignocaine were examined in this study for their abilities to reduce the detrimental effects of laryngoscopy and tracheal intubation on blood flow. Individuals who had undergone these treatments were studied for this study.

METHODOLOGY

This prospective, randomized trial was approved by the Ethical Committee, and all participants supplied written informed permission. Sixty people, ages 15 to 45, all with ASA physical status I, agreed to be surgical subjects for the study. Patients with an ASA respiratory status of 2, 3, or 4, women who are pregnant, patients undergoing emergency surgery, patients who are allergic to the study drugs, and patients whose airways are difficult to open and require more than 30 seconds of laryngoscopy and more than one attempt were excluded from the study. The study also did not include patients with an ASA respiratory status of 2, 3, or 4.

These people were arbitrarily separated into two categories (30 in each group). Participants in Group M received a dose of 40 mg/kg of magnesium sulphate 50%, while those in Group L received a dose of 1.5 mg/kg of lidocaine. Patients received complete physicals and any additional diagnostic testing that was considered essential before undergoing any type of surgical operation. Before surgical treatments, patients were instructed to fast for at least six hours. Each subject had 0.2 milligrams of glycopyrrolate injected into a muscle prior to receiving the general anesthesia. It was necessary to do this before the operation.

To administer Ringer lactate intravenously, a cannula was placed in a vein in the patient's non-dominant hand. The patient's health was evaluated with the aid of a pulsoximeter, an electrocardiogram (ECG), and a noninvasive blood pressure monitor (NIBP).

Each patient was given three minutes of pure oxygen before surgery. Group L patients were given lignocaine intravenously at a rate of 1.5 mg/kg three minutes before their scheduled general anesthetic procedure. It was done before the general anesthetic so that everything would go well. Prior to undergoing general anesthesia, those in Group M were given 40 milligrams per kilogram of body weight (mg/kg) of magnesium sulphate intravenously at a pace of one minute. Then, an intravenous dose of thiopentone (5 mg/kg) was given to induce unconsciousness. A dose of 1.5 mg/kg of succinylcholine was administered, and then the patient was given a 100 percent oxygen mask. After the fasciculations stopped, a laryngoscopy with a Macintosh laryngoscope blade was done, and an endotracheal tube was

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placed. The patient was kept unconscious with a gas mixture consisting of 50% oxygen, 50% nitrous oxide, and 1% isoflurane.

**Statistical analysis:** The average clinical parameters of the two study groups were compared using the independent sample t-test to see if there was a statistically significant difference between the groups (such as HR, SBP, and DBP). Verification of the validity of the normality and variance assumptions allowed us to conclude that this was the case. We used the t-test, which does not need evenly paired samples, to compare the two groups' parameters. We used the paired t-test to compare each group before and after receiving the study medications (Lignocaine and Magnesium sulphate). A p-value of 0.05 or less is considered to be the cutoff for statistical significance. All testable hypotheses were crafted to be in agreement with or disagreement with the null hypothesis.

**RESULTS**

Table 1:

<table>
<thead>
<tr>
<th></th>
<th>Group L</th>
<th>Group M</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>29.2 ± 8.44</td>
<td>30.1 ± 9.19</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Gender M/F</td>
<td>15/15</td>
<td>15/15</td>
<td></td>
</tr>
<tr>
<td>Basal HR (bpm)</td>
<td>73.2 ± 3.94</td>
<td>72 ± 3.84</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Basal SBP (mmHg)</td>
<td>123.43 ± 6.73</td>
<td>122.27 ± 6.03</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Basal DBP (mmHg)</td>
<td>79.33 ± 5.31</td>
<td>77.27 ± 4.97</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>

Table 2:

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>after Lignocaine</th>
<th>1 min</th>
<th>3 min</th>
<th>5 min</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (bpm)</td>
<td>73.2 ± 3.73</td>
<td>70.99 ± 3.19</td>
<td>102.68 ± 18.65</td>
<td>96.10 ± 7.75</td>
<td>80 ± 6.14</td>
<td>0.59 &lt; 0.01 &lt; 0.01 &lt; 0.01</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>114.51 ± 5.63</td>
<td>127.19 ± 5.48</td>
<td>139 ± 7.26</td>
<td>141 ± 7.21</td>
<td>124.58 ± 5.01</td>
<td>0.81 &lt; 0.01 0.65</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>78.32 ± 6.11</td>
<td>81.24 ± 7.28</td>
<td>96.83 ± 7.73</td>
<td>124.58 ± 6.9</td>
<td>74.25 ± 8.02</td>
<td>0.29 &lt; 0.01 0.01 &lt; 0.05</td>
</tr>
</tbody>
</table>

Table 3:

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>after Magnesium sulphate</th>
<th>1 min</th>
<th>3 min</th>
<th>5 min</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (bpm)</td>
<td>72 ± 3.98</td>
<td>78.2 ± 4.39</td>
<td>82.3 ± 7.97</td>
<td>79.22 ± 4.58</td>
<td>78.2 ± 6.81</td>
<td>&lt; 0.01 &lt; 0.01 &lt; 0.01</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>119.19 ± 5.02</td>
<td>124.18 ± 5.39</td>
<td>129.57 ± 8.33</td>
<td>125.12 ± 6.44</td>
<td>117.24 ± 5.32</td>
<td>0.5 &lt; 0.01 0.19 &lt; 0.01</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>76.18 ± 5.01</td>
<td>77.3 ± 4.27</td>
<td>85.05 ± 6.71</td>
<td>85.69 ± 6.53</td>
<td>71.71 ± 8.62</td>
<td>0.18 &lt; 0.01 &lt; 0.01 &lt; 0.01</td>
</tr>
</tbody>
</table>

According to the data, neither group of patients was significantly different from the other. (Table 1). Changes in blood flow were measured in Group L after intravenous lidocaine was administered and compared to baseline values. As seen in Table II, lignocaine caused a transient and mild reduction in heart rate. Although the systolic blood pressure did not change immediately after lidocaine was injected, it did rise significantly one and three minutes after the tube was placed. For the first five minutes after the tube was put, there was little to no change in the subject's blood pressure. Diastolic blood pressure did not immediately change after intravenous lidocaine administration. One and three minutes after intubation, however, there was a notable increase compared to pre-intubation levels. The diastolic blood pressure of this group dramatically decreased after 5 minutes of intubation, relative to the pre-intubation value. Magnesium sulphate was given intravenously to Group M, and their blood flow was then monitored and compared to the initial levels. The heart rates of this group significantly increased when magnesium sulfate was given, and again one minute, three minutes, and five minutes following intubation, as compared to the values obtained at baseline (Table III). It remained high after three minutes had elapsed, but
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statistically speaking it was no different than when it had initially started. After intubation, this group experienced a significant reduction in systolic blood pressure five minutes later. It was immediately apparent after the magnesium sulfate injection, but it was noticeable one and three minutes later. After 5 minutes of intubation, this group’s DBP dropped significantly compared to its baseline levels.

The hemodynamic responses of both groups were measured and compared immediately after the administration of the study medication. In Group L, the average heart rate was 71.93 beats per minute, while in Group M it was 79.3 beats per minute. It was shown that Group M saw a much larger increase in heart rate than Group L. The average heart rates of both groups are shown changing over time in Figure 3. The average blood pressure of the study’s participants, immediately after receiving the study drug, was 123.27 6.44 mmHg. Group L had a mean systolic blood pressure of 80.13 8.17 mmHg, while Group M had a mean systolic blood pressure of 78.4 5.88 mmHg. Treatment with the drug had no noticeable effect on DBP (p value was greater than 0.05).

Blood pressure and pulse rate were monitored and compared before and after trachea implantation (Figure 3). The average heart rate in Group M was 81.4 5.88 beats per minute, while the average heart rate in Group L was 103.73 19.96 beats per minute. The heart rate in Group L increased noticeably greater than those in Group M after just one minute after intubation (p 0.05). SBP averaged 124 13.74 mmHg in Group M, compared to 140 8.19 mmHg in Groups L and M. SBP in Group L increased significantly more than in Group M three minutes after intubation. The significance level of 0.05.

Changes in blood flow were tracked and analyzed for five minutes. After a tracheal tube was placed in the patient’s windpipe, the average heart rate in Group L was 81.7 2.72 beats per minute, which was significantly higher than Group M’s average heart rate of 79.17 9.34 beats per minute. Nothing unusual happened in terms of heart rate (p value 0.16). The average systolic blood pressure was 123.87 4.03 mm Hg in Group L and 118.13 6.39 mm Hg in Group M. Group L’s SBP was significantly higher than Group M’s (p 0.01). (Table 4).

Group L had mean DBP of 75.27 9.04 mmHg, while Group M had mean DBP of 72.67 7.67 mmHg. Concentrations of DBP did not differ significantly between the two groups (p 0.23). (Fig. 5).

**DISCUSSION**

The patient may be at danger for a cerebral hemorrhage, left ventricular failure, and other complications as a result of the reflex sympathetico adrenal discharge because of these responses. Lignocaine acts in this way to immediately slow down the heart rate and enlarge the blood vessels that are surrounding the heart. These two medications have been investigated by researchers to see whether or not they can minimize the response to intubation, this group’s systolic and diastolic blood pressures. This held true whether group got injection first. Magnesium-treated patients did not experience an increase in systolic blood pressure after tube implantation. Even the “control” group experienced this. Both groups’ worldviews were different. Diastolic blood pressure also changed. Puri et al. [11] found that magnesium alone lowered pre-induction mean arterial pressure (P 0.001). 0.05 Only the control group changed following anesthetic injection (P 0.01). Before intubation, the two groups couldn’t be statistically differentiated. Both pre- and post-intubation (3 minutes apart) lignocaine revealed greater MAP than magnesium (P 0.01). In two minutes, air pressure normalized. Compared to baseline, lignocaine reduced mean arterial pressure by 10%. After the operation, something happened. After intubation, the treatment group’s mean arterial pressure rose 30% and stayed high for 5 minutes. Systolic and diastolic blood pressure reduced fast after magnesium injection [13] (Sharma J., et al.). Navid Nooraei et al. [15] found that magnesium sulfate regulates hemodynamics better than lignocaine. Despite increasing HR, this was true. Compared to the lignocaine group, 30 mg/kg of magnesium sulphate significantly reduced HR and MAP (P 0.05). Lignocaine isn’t as effective as magnesium sulfate at reducing heart rate during laryngoscopy and intubation. Intravenous magnesium sulfate is given one minute before induction. Five minutes in, there were no evidence of a rising SBP. Group M’s systolic blood pressure increased after taking magnesium sulfate, however the increase was not statistically significant. The significance of this increase became obvious once the patient was given a breathing tube. Lignocaine was not as effective as magnesium sulfate at reducing heart rate during intubation. Group M’s blood pressure dropped significantly. SBP’s blood pressure didn’t change after taking experimental medications. Group L participants showed greater blood pressure
one, three, and five minutes after the tube was introduced. Group L's diastolic blood pressure did not change after lidocaine administration, but it increased one and three minutes after intubation compared to pre-intubation values. Although there was no immediate difference, this was the result. Group M's DBP rose after taking magnesium sulfate, although not much.

When calcium is present, sympathetic activation of adrenergic nerve terminals stimulates adren sympathetic release. This rivalry disrupts calcium-dependent processes. Magnesium prevents intubation-induced catecholamine release. This reduces heart problems. This study indicated that the heart was most affected. Magnesium reduces calcium-driven depolarizing current in pacemaker tissue, delaying animal atrial beats. This affects pacemaker tissue. We got ideas from an animal. In healthy animals, magnesium can prevent vague nerve acetylcholine release [18]. This raised heart rate. Before tubes were implanted, the lignocaine group's heart rate was lower than the magnesium group's. After intubation, lignocaine-treated patients had higher heart rates than magnesium-treated patients. Compared to magnesium, lignocaine raised epinephrine levels higher. The magnesium group presumably had better blood pressure regulation due to vasodilatory effects and reduced catecholamine production. Because magnesium inhibits catecholamine release, Studies demonstrate that magnesium affects these processes. Even at high blood levels, magnesium has no neurological effects. Magnesium probably doesn't work this way. The blood-brain barrier prevents magnesium from entering the brain. High dosages of magnesium have a modest effect on the CNS. [20]

Magnesium may help in this situation. Magnesium levels between 2 and 4 mmo/L may increase a pregnant woman's survival after endotracheal intubation. Magnesium reduces succinylcholine-induced fasciculations [22] and potassium release [23]. This could be useful because magnesium affects heart pumping. Magnesium doesn't seem to prolong succinylcholine's effects. [13,24]. Magnesium and other relaxants that don't lower heart rate should be used with these medicines. This combination requires a lower dose of the relaxant. [23,24] Magnesium sulfate's effects on non-depolarizing relaxant block duration and strength are unknown. Magnesium sulfate aids intubation. [23,24]

CONCLUSION

Magnesium sulfate reduces systolic and diastolic blood pressure following tracheal intubation but not heart rate. Lignocaine doesn't impact heart rate or systolic blood pressure. Magnesium sulphate is superior to lidocaine in ASA grade I patients during laryngoscopy and tracheal intubation.

REFERENCES

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