Attenuation of Haemodynamic Responses to Laryngoscopy & Intubation following Nitroglycerin and Esmolol infusion

P Gupta, B Panda, R Verma, P Ranjan, S Mathur, G Sinha

Citation

Abstract

Background: Adrenergic stress response induced by laryngoscopy and tracheal intubation appears to be attenuated by esmolol and nitroglycerine but their potential clinical benefits have not been fully weighed against possible adverse effects. Objective: The objective of the present study was to compare the efficacy of intravenous infusions of nitroglycerin and esmolol in attenuating hemodynamic responses to laryngoscopy and endotracheal intubation. Methods: Sixty ASA 1 and 2 patients scheduled for elective surgical procedures were randomly divided into three groups of 20 patients each. The patients of groups C, E and N received intravenous infusions of normal saline, esmolol hydrochloride (100 μg/kg/min) and nitroglycerin (0.5 mgm/kg/min) respectively. Infusions were started 5 minutes before induction and continued till 5 minutes after intubation. Anesthesia was induced with thiopentone sodium and succinylcholine and maintained with nitrous oxide-oxygen and vecuronium bromide. Results: It was observed that nitroglycerin prevented a rise in diastolic blood pressure and attenuated the rise in systolic blood pressure, but failed to attenuate increase in the heart rate, while esmolol effectively controlled the increase in systolic BP, diastolic BP and heart rate following intubation. Conclusion: It was concluded that esmolol infusion is more effective in attenuating hemodynamic responses to intubation as compared to nitroglycerin infusion.

INTRODUCTION

In 1940, Reid and Brace first described hemodynamic response to laryngoscopy and intubation. Laryngoscopy and tracheal intubation are known to cause sympathoadrenal stimulation. This manifests as hypertension and tachycardia. Usually these transient changes have no deleterious consequences in healthy individual, but in some patients they can provoke left ventricular failure, myocardial ischemia and cerebral hemorrhage. These complications are more likely to occur in the presence of coronary or cerebral atheroma or pre-existing hypertension. The present study was conducted to study the efficacy of intravenous infusions of nitroglycerin and esmolol in attenuating these responses.

MATERIALS AND METHODS

The study was approved by the ethical committee of the hospital and was conducted in 60 ASA grade I and II patients of both sex, posted for surgery under general anaesthesia. Patients suffering from hypertension or other cardiovascular diseases were not included in the study. After obtaining informed consent they were randomly allocated to one of the following three groups of 20 patients each, depending on the infusion of the hypotensive agent through a syringe pump.

Group C - Normal saline infusion (control group)
Group E - Esmolol hydrochloride infusion 100 μg/kg/min
Group N - Nitroglycerin infusion 0.5 μg/kg/min

All patients were premedicated with alprazolam 0.5 mg two hours before operation. On arrival in the operation theatre patients’ baseline heart rate, systolic, diastolic and mean blood pressures were recorded and 0.2 mg glycopyrrolate and 0.5 mg/kg of pentazocine were administered intravenously. The infusion of normal saline/ study drug was started. The induction was done with thiopentone sodium 1-2 mg/kg and neuromuscular blockade was achieved by succinylcholine 1.5 mg/kg. Direct laryngoscopy was done and tracheal intubation was completed within 30 sec in all the patients. Serial measurement of heart rate, systolic blood
Attenuation of Haemodynamic Responses to Laryngoscopy & Intubation following Nitroglycerin and Esmolol infusion

pressure, diastolic blood pressure and mean arterial pressure were done with a bioview monitor. The infusion (study drug/saline) was stopped 5 minutes after intubation. Anaesthesia was maintained with 66% nitrous oxide in oxygen and vecuronium bromide. Data was analyzed by Chi square test and Students’t’ test as applicable.

RESULTS
The three groups were comparable with respect to age, weight and gender (Table 1).

Figure 1
Table 1: Mean Age, body weight and sex of patients in the three groups

<table>
<thead>
<tr>
<th>Groups</th>
<th>Group C</th>
<th>Group N</th>
<th>Group E</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age *(year)</td>
<td>35.31 ± 11.31</td>
<td>35 ± 7.78</td>
<td>35.18 ± 9.88</td>
</tr>
<tr>
<td>Body weight*(kg)</td>
<td>49.92 ± 7.78</td>
<td>50.45 ± 6.88</td>
<td>54.9 ± 8.89</td>
</tr>
<tr>
<td>Sex Ratio(M:F)</td>
<td>7.13</td>
<td>4.16</td>
<td>2.18</td>
</tr>
</tbody>
</table>

* Mean ± SD

No significant difference in mean age, weight and sex ratio in the three groups.

Haemodynamic changes before and after intubation are shown in tables 2 - 5. Heart rate increased significantly in group C, 1 min after intubation (p < 0.01) and increased further 3 and 4 min after intubation (p < 0.001). In group N the heart rate increased one minute after intubation and continued to increase at subsequent intervals. The increase was statistically significant 3, 4 and 5 minutes after intubation (p < 0.05). In group E the changes in heart rate following intubation were statistically not significant (fig 1).

Figure 2
Fig 1: Showing Heart Rate per minute

Five minutes after the start of infusion and just before intubation systolic blood pressure decreased in all the three groups but the change was statistically significant in group N (p<0.05) and group E (p<0.01). Following intubation it increased significantly, 1, 3 and 5 minutes after intubation in group C (p<0.001) and 3 and 5 minutes after in group N (p<0.01 and <0.05 respectively). This rise in the group N was significantly less than group C (p<0.05). In group E systolic blood pressure did not increase following intubation (figure 2).

Figure 3
Fig.2: Showing Systolic Blood Pressure

There was a significant rise in diastolic blood pressure, 1 and 3 minutes after intubation in group C (p<0.01). Diastolic blood pressure did not change significantly in group N and group E (Figure 3).

Figure 4
Figure 3: Showing Diastolic Blood Pressure

Mean arterial pressure increased significantly in the control group following intubation after 1 min (p<0.01) and after 3 and 5 min (<0.001). In group N the rise in mean arterial pressure was statistically significant only 3 min after intubation (p<0.001). In the esmolol group mean arterial pressure decreased significantly (p<0.05) 1 minute after intubation. It increased subsequently but was not significantly different from the initial value (Figure 4).
DISCUSSION

Hypertension and tachycardia subsequent to tracheal intubation have been well documented. In susceptible patients even this short period (2-7 minutes) of hypertension and tachycardia can result in myocardial ischaemia or increased intracranial pressure. Complications resulting from these haemodynamic events after intubation include left ventricular dysfunction, hypertensive crises, pulmonary oedema, cardiac dysrhythmias, myocardial ischaemia, and myocardial necrosis.

Many agents have been used to attenuate undesirable haemodynamic responses to laryngoscopy and intubation with varying success. These include intravenous opioids, vasodilators, calcium channel blockers, intravenous and topical lignocaine and adrenoceptor blocking drugs alone or in combination with other drugs. In the present study esmolol and nitroglycerin were selected because of their similar pharmacokinetic profile i.e. rapid onset of action, short duration of action, rapid elimination and termination of action on discontinuation of infusion. Many studies have been done to assess the effect of these drugs when given as a bolus injection, but very few studies have been done with intravenous infusion. We preferred to use nitroglycerin and esmolol in the form of an infusion as it would be less likely to cause acute fluctuations in heart rate and blood pressure.

In the present study nitroglycerin prevented a rise in diastolic blood pressure and attenuated the increase in systolic blood pressure following intubation. It failed to attenuate ionotropic response to intubation. The reason for this could have been the tendency of nitroglycerin to cause tachycardia. This finding is similar to that of Singh et al who also reported failure of nitroglycerin to attenuate increase in heart rate following intubation. Esmolol on the other hand effectively controlled increase in heart rate, systolic blood pressure, diastolic blood pressure and Mean arterial pressure following intubation.

These findings are similar to those of other studies where a continuous infusion of esmolol was administered. Figueredo et al in a meta-analysis, found that esmolol given as continuous infusion was more effective and had minimum side effects as compared to esmolol bolus.

CONCLUSION

It can be concluded from the present study that esmolol (100 μgm/kg/min) was superior to nitroglycerin (0.5 μgm/kg/min) in attenuating the hemodynamic responses to laryngoscopy and intubation.

References

1. Reid LC, Brace DE. Irritation of the respiratory tract and its reflex effect upon heart. Surg Gynaec & Obst; 1940; 70: 157-62.
Attenuation of Haemodynamic Responses to Laryngoscopy & Intubation following Nitroglycerin and Esmolol infusion

Author Information

PK Gupta, MD, DA
Prof. & HOD, Department of Anaesthesiology, Institute of Medical Sciences

BK Panda, MD
Lecturer, Department of Anaesthesiology, Institute of Medical Sciences

RK Verma, MD
Reader, Department of Anaesthesiology, Institute of Medical Sciences

P Ranjan, MD
Reader, Department of Anaesthesiology, Institute of Medical Sciences

SK Mathur, MD
Reader, Department of Anaesthesiology, Institute of Medical Sciences

GK Sinha, MD
Reader, Department of Anaesthesiology, Institute of Medical Sciences