Management of Cardiovascular Stress Responses to Laryngoscopy and Tracheal Intubation using Esmolol Hydrochloride

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Results of prospective double blind randomized controlled study over one year period on 50 ASA status i & ii patients presented using an i.v. bolus of isotonic saline or Esmolol hydrochloride before induction of anesthesia and endotracheal intubation. Various haemodynamic changes were measured to assess the pressor response to laryngoscopy & tracheal intubation. Significant reduction in the pressor response was observed in esmolol group after five minutes of intubation. Changes in the heart rate remained significant even fifteen minutes after intubation. Esmolol hydrochloride can be regarded as a safe and effective agent to suppress the cardiovascular stress response to laryngoscopy and tracheal intubation.

Key Words Tracheal Intubation, Cardiovascular Sterss Response, B- Adrenergic Blocker's, Esmolol.

The circulatory responses to laryngeal and tracheal stimulation have been established since 1951[1]. This is the result of reflex sympathoadrenal stress response provoked by stimulation of epipharynx and hypopharynx.[2] The response is characterized by tachycardia, hypertension and raised serum concentrations of catecholamines and is usually transitory, variable and unpredictable [3]

The resulting increase in the cardiac workload in turn may result in peri-operative myocardial ischemia and acute heart failure in susceptible patients[4].

There is an additional danger of cerebral haemorrhage where as convulsions may be precipitated in pre-eclamptic patients. All these complications are more likely in hypertensive patients in the presence of coronary or cerebral atheroma. Several techniques have been employed to suppress this pressor response with variable success rates. Esmolol hydrochloride is an intravenous cardio selective beta adrenorecerptor antagonist with rapid onset of action exerting haemodynamic effects with in minutes and a short elimination half-life of nine minutes. This prospective study was performed to compare the suppression of haemodynamic effects of laryngoscopy and tracheal intubation in ASA i & ii patients using a bolus of Esmolol 100 mg or placebo (normal saline) before induction of anaesthesia.

Patients and Methods

The study was carried out at Mayo hospital, Lahore over 6 months period from Jan.94 to Jun. 94. After the approval from hospital ethical committee, a total of 50 patients of either sex, age between 15 to 45 years were included from different general surgical departments. All patients were of an ASA status i and ii. No drug was used as pre medication and no anticholinergic drug was given prior to induction. Various variables monitored were heart rate, systolic, diastolic and mean arterial blood pressure and continuos ECG monitoring using limb lead ii. Once the patients get stabilized in operating theater. An 18 gauge i.v cannula was inserted under local anaesthesia into peripheral vein and a small volume of 5% dextrose water was infused during study period. Base line measurements of systolic, diastolic and mean arterial blood pressure were obtained using non invasive automaterd sphygmomanometer (dinamap bp 103 n/rs) on contralteral arm. Heart rate was measured by using ECG lead ii. Patients were randomly divided in to two groups. Group-i patients received 5% dextrose water 20 ml as i.v. bolus (placebo). Group ii patients received 20 ml of i.v. solution containing 100mg of esmolol at time zero over 15 seconds. Immediately after thiopentone sodium 5mg/kg was given as i.v. inducing agent followed by suxamethonium 1.5mg/kg for laryngeal inlet relexation. Patients were ventilated with 33% oxygen, 66% N_2o and 0.5% halothane, using semi open system with nine-liter per minute fresh gas flow.

Laryngoscopy and orotracheal intubation was performed exactly after two minutes of bolus of esmolol using standard macintosh laryngoscope blade. All intubatons were done within 15 seconds smoothly by author himself. Anaesthesia was maintained by using 33% oxygen, 66% n2o and .5% of halothane. Atracurium besylate .3 to .6 mg/kg was given as i.v. bolus after restoration of spontaneous respiration. Position making, draping or surgical incision, were delayed upto 10 minutes after induction. Haemodymamic variables were monitored at 0, 5 and 15 minutes. Mean changes in heart rate, the systolic, diastolic and mean arterial blood pressure were analyzed by analysis of covariance, taking base line variables as covariants. Scheffe's test was used to compare the individual mean.

Results

There was no significant difference between age, sex, weight, ASA status and base line haemodynamic variables between group i. and group ii (TABLE-I).

Table 1. Comparison of base line variables between group I. & II.

Variable	Group-I (Mean)	Group-Ii. (Mean)	P.Value
Age	33.89	32.98	> 0.05
Weight	60.42	61.03	> 0.05
Heart rate	91.95	89.96	> 0.05
SAP	133.37	131.26	> 0.05
DAP	79.58	77.15	> 0.05
MAP	99.95	99.85	> 0.05

Several changes were observed during and 5 minutes after laryngoscopy. Both groups developed rise in heart rate, systolic, diastolic and mean arterial pressure. All these changes were significantly greater in control group-i. than those recorded in study group- ii. (Table-II.).

Table II.

Variable	Group-I. (Mean)	Group-Ii .(Mean)	P.Value
Heart Rate	118.84	91.40	> 0.05
SAP	154.63	135.32	> 0.05
DAP	91.10	78.16	> 0.05
MAP	110.14	99.50	> 0.05

Where as after 15 minutes all the haemodynamic changes reverted back to near the base line values, group-ii patients reflected a relatively smooth decline as against the group-i patients with delayed and variable responses (Table-III). Comparison of haemodynamic variables after 15 minutes between group 'I' and 'II'.

Table-III.

Variable	Group-I.(Mean)	Group-Ii. (Mean)	P .Value
Heart Rate	106.05	83.60	< 0.05
SAP	125.05	122..60	> 0.05
DAP	83.56	80.52	> 0.05
MAP	95.68	95.15	> 0.05

The changes in the heart rate remained statistically significant between control group-i and study group-ii patients even after 15 minutes. On no occasions were significant hypotension, hypertension or changes in the heart rate encountered which could have necessitated discontinuation of the study.

Discussion

Laryngoscopy and tracheal intubation frequently induce a cardiovascular stress response, characterized by hypertension, tachycardia and increased serum concentration of catecholamine [3]

This sympathoadrenal response to laryngoscopy results in an increase in cardiac work load, this increase in cardiac work load may result in perioperative myocardial ischaemia and acute heart failure in susceptible patients [4].

This response is undesirable in any patient with heart disease undergoing surgery, irrespective of the nature of the surgery.

Several techniques have been used with varying degree of success to suppress the response to laryngoscopy and tracheal intubation such as:

a). Intervenous or topical lignocine
b). Thoracic extradural analgesia
c). Intra-venous opiods
d). Peripheral vasodilators

However, non of these above techniques have gained widespread acceptance. Commonly used beta blockers have relatively long duration of action and they lack cardio- selectivity in some cases. Esmolol hydrochloride is a relatively new cardio-selective intravenous beta blocker. It has a rapid onset of action, exerts peak haemodynamic effect within minutes and possesses a short elimination half life of nine minutes [5].

So esmolol possesses following properties which suggest that it might be valuable in suppressing responses to laryngoscopy and tracheal intubation:

i). Esmolol is highly cardio selective agent. it is analogous to metoprolol and this agent does not cause bronchospasm.
ii). Esmolol undergoes rapid esterase mediated metabolism characterized by an elimination half life of 9.2 min culminating in a rapid offset of action when infusion is stopped.
iii). More over significant drug interactions have not yet been reported with esmolol. The only side effects, which have been reported to esmolol, are hypotension and thrombophlebitis at the site of injection. Careful dilution of the agent (5mg/ml) and the judicious use of doses may avoid these side effects.

Esmolol has been administered with success as bolus injection to suppress the response to laryngoscopy and tracheal intubation. In our study, we used bolus of esmolol.

In our study, there was no significant difference in age, sex, weight, ASA status and baseline haemodynamic variables between group I & II. After the administration of esmolol in group-ii patients, haemodynamic variables were measured at 0, 5 and 15 minutes. Several changes were observed during and '5' minutes after laryngoscopy. Both groups developed rise in heart rate, systolic, diastolic and mean arterial blood pressure. All these changes were significantly greater in control group-i than those recorded in group-ii patients, where as after 15 minutes all the haemodynamic changes reverted back to near the base line values. The group-ii patients reflected relatively smooth decline as against the group-i patients with delayed and variable response.

The changes in heart rate remained statistically significant between control group i. and ii. Patients even after 15 minutes. The results in our study are in agreement with other workers. Shepperad studied the esmolol in 100 and 200 mg bolus doses for laryngoscopy and intubation. The difference in heart rate between control and both esmolol groups was significant before intubation but after intubaion only in 200 mg group was found. There was a significant difference in SAP between 100 mg and 200 mg group. He recommended esmolol '200'mg bolus to attenuate the pressor response to laryngoscopy [6]

Same doses (100 & 200 mg) were used by Ebert and Parnass. To attenuate the laryngoscopic response, both studies showed that both doses were collectively effective in controlling the heart rate and systolic pressure. No significant difference was found in both doses [7]

Conclusion

We concluded that bolus injection of esmolol 100 mg is safe and effective technique for suppressing the

cardiovascular stress responses associated with laryngoscopy and intubation.

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