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Original Research Article

Analytical study of effects of intravenous lignocaine on pressor response during laryngoscopy and intubation

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Abstract

Induction of general anaesthesia and endotracheal intubation for maintenance of airway and prevention of aspiration is carried out frequently by the anaesthesiologist in day to day practice. This study was aimed to study the effects of lignocaine on pressor response during laryngoscopy and intubation. It was carried out in 50 patients of both sexes, between the age group of 20-60 years, belonging to ASA physical status grade I and II. 2% preservative free lignocaine 1.5mgKg-1 diluted to 10ml over a period of one minute given intravenously before inbutation resulted in tachycardia after intubation. But at the end of 5 minutes, the heart rate was still higher in lignocaine group. We found out that intravenous lignocaine is less effective to attenuate the pressor response and a better alternative should be used after further comparative studies.

Keywords: Laryngoscopy, intubation, intravenous lignocaine, pressor response

1. Introduction

Laryngoscopy and endrotracheal intubation is an integral part of anaesthesia. Induction of general anaesthesia and endotracheal intubation for maintenance of airway and prevention of aspiration is carried out frequently by the anaesthesiologist in day to day practice. Although endotracheal intubation adds tremendous safety to the administration of general anaesthesia, it has deleterious effects especially on cardiovascular system. These responses are due to intense sympathetic discharge caused by stimulation of upper respiratory tract both during laryngoscopy and tracheal tube insertion. This pressor response is associated with the release of catecholamines in large amount, in the body.[1][2]

A normal healthy patient may be able to deal with these responses. In patients with various diseases like coronary artery disease, hypertension, intracranial aneurysms, dissecting aneurysm, the response to laryngoscopy and intubation may prove potentially lethal. In order to attenuate this pressor response various techniques have been tried by many workers. All of these depend upon the blockade of adrenergic response.

The purpose of this study is to evaluate the effects of lignocaine (2%) for attenuating pressor response to laryngoscopy and endotracheal intubation.

2. Materials and Methods

The Present study was carried out in 50 patients of both sexes, between the age group of 20-60 years, belonging to ASA physical status grade I and II. The patients were scheduled to undergo various elective operative procedures at Kidwai Memorial Institute Oncology, Bangalore.

The personal and medical history was obtained by interview and hospital indoor record sheet. Patients with history of hypertension, diabetes mellitus, respiratory or cardiovascular diseases are not included in the study. Patients posted for emergency surgery were excluded from study. Written consent was obtained from all the patients.

The selected 50 patients were preoxygenated with 100% oxygen for three minutes. Anaesthesia was induced with thiopentone (2.5%) 5mgKg^{-1} intravenously. Following this, patients received 2% preservative free lignocaine 1.5mgKg-1 diluted to 10ml over a period of one minute. Then succinyicholine 1.5 mg Kg^{-1} was given intravenously for facilitation of intubation. After 1 minute patients were intubated orally with suitable sized cuffed endotracheal tubes under laryngoscopic vision by the anaesthesiologists who were unaware of the drug used.

Heart rate and blood pressure were recorded at before induction, after induction, after trial drug administration, immediately after intubation, 2 minutes after intubation and 5 minutes after intubation. Mean arterial pressure and rate pressure product at different stages of the study were calculated.

3. Results

50 patients belonging to ASA grade – 1 and II were studied. The following are the observations of the study.

Table 1: Age and Weight distribution

Group	Age (in years) (Mean ± SD)	Weight (in Kilograms) (Mean± SD)
Lignocaine (L)	38.7 ±9.22	58.08±8.17

Table 2: Sex Distribution of cases

Group	Male	Female	Total
Lignocaine	15	35	50

Table 3: Heart rate (beats/minutes) changes

	Lignocaine (Mean±SD)
Pre-induction	81.80±6.74
After induction	86.54±8.00
After trial drug	89.88 ±8.11
Immediately after intubation	102.66±7.11
2 min after intubation	96.82±6.45
5 min after intubation	90.38± 5.98

After administration of trial drug there was a rise in heart rate. Immediately after intubation rise in heart rate from preinduction value (Preinduction 81.80±6.74, immediately after intubation 102.66 ± 7.11)

Table 4: Systolic Blood Pressure (mmHg) changes

	Lignocaine (Mean ±SD)
Pre-induction	121.20 ±8.72
After induction	120.96 ±8.37
After trial drug	119.96 ±8.99
Immediately after intubation	153.92±10.25
2 min after intubation	142.40±11.07
5 min after intubation	132.80±9.06

There was marginal fall in SBP following induction and after trial drug administration. Immediately after intubation increase in SBP was observed in lignocaine group (Preinduction 121.20 ± 8.72, immediately after intubation 153.92 ± 10.25)

Table 5: Mean Arterial Pressure (mmHg) changes

	Lignocaine (Mean ±SD)
Pre-induction	90.59 ±5.74
After induction	90.63 ± 568
After trial drug	90.50 ±5.66
Immediately after intubation	107.47±6.36
2 min after intuation	101.41±6.51
5 min after intubation	95.69±5.62

Immediately after intubation there was rise in MAP. 5 minutes after intubation MAP was well above (95.69± 5.62) the Preinduction value.

Table 6: Rate Pressure Product (RPP) changes

	Lignocaine (Mean ±SD)
Pre-induction	9921.60 ±1148.51
After induction	10464.76 ± 1166.41
After trial drug	10787.96 ± 1292.75
Immediately after intubation	15824±1768.51
2 min after intubation	13468.80±2207.75
5 min after intubation	12014.64±1242.06

After induction and after trial drug administration there was rise in RPP. The RPP value at the end of 5 minutes after intubation was (approx.) 2093.04 more than the pre-induction value.

4. Discussion

Although endotracheal intubation adds on tremendous safety to the administration of general anaesthesia, it is associated with deleterious effects on cardiovascular system Viz tachycardia, hypertension[3] dysarrhythmias[4]. These responses are due to intense sympathetic discharge caused by stimulation of upper respiratory tract both during laryngoscopy and tracheal tube insertion. The pressor response is associated with the release of catecholamines in the body.[1][2]

These responses though well tolerated in normal patients, could be catastrophic in patients with cardiovascular or cerebrovascular diseases. It is therefore logical that attempts should be made to attenuate cardiovascular responses to laryngoscopy and intubation.

In order to attenuate the pressor responses various techniques have been tried by many workers. These methods include deep general anaesthesia, adrenoreceptor blockers[5][6], calcium channel blockers[7][8], opioids[9][10] and vasodilators[11]. These above mentioned drugs have their own side effects like excess sedation, respiratory depression

and hypotension. Hence anaesthesiologists were in a constant search for an ideal drug for attenuation of pressor responses.

Intravenous lignocaine has been used since many years to attenuate the pressor response to laryngoscopy and intubation and its effectiveness is controversial.

Abou-Madi *et al*[12] found out that 1.5mgKg^{-1} of intravenous lignocaine offered complete protection against arrhythmias, hypertension and tachycardia. Stoelting[13] found that laryngotracheal lignocaine is better than viscous or intravenous lignocaine for minimizing pressor response to laryngoscopy ad intubation.

Charaemmer *et al*[14] concluded that lidocaine 1.5mg/kg does not prevent or significantly blunt any of the hemodynamic reactions evoked by rapid sequence induction and intubation. Charles *et al*[15] and Miller *et al*[16] found that intravenous lignocaine is not effective for attenuation of pressor response.

Stanley *et al*[17] concluded that intravenous lidocaine at 1.5mg/kg attenuates increase in heart rate and arterial blood pressure only when given 3 minutes before intubation and offers no protection against post intubation haemodynamic changes when given at 1,2 or 5 minutes before intubation.

In lignocaine group the rise in heart rate from preinduction value immediately after intubation was nearly 20.86 beats/ min. At the end of 5 minutes it was well above the preinduction value. (Preinduction 81.80 ±6.74, 5 minutes after intubation 90.38 ± 5.98).

In lignocaine groups rise in SAP immediately after intubation from preinduction value was nearly 32.72 mm Hg, even after 5 minutes it was more than preinduction value. (Preinduction SAP 121.20 ± 8.72 mm Hg, 5 minutes after intubation 132.80 ±9.06 mm Hg). There was rise in MAP after intubation (lignocaine group preinduction 90.59 ± 5.75 mm Hg, immediately after intubation 107.47 ± 6.36 mm Hg). 5 minutes after intubation MAP was more than preinduction value in lignocaine group (95.69 ± 5.62). The rise in RPP immediately after intubation was increased nearly by 5903.24. At the end of 2 minutes RPP was higher than preinduction value. RPP at the end of 5 minutes showed that it was nearly 2093 more in lignocaine group.

Immediately after intubation there was tachycardia. But at the end of 5 minutes, the heart rate was still higher in lignocaine group.

5. Conclusion

We found out that intravenous lignocaine is less effective to attenuate the pressor response and a better alternative should be used after further comparative studies.

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