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Original research paper

Lignocaine and fentanyl in laryngoscopy and intubation: Comparison of changes in heart rate

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Abstract

Lignocaine is useful to decrease sympathetic response to laryngoscopy and intubation when applied topically or sprayed or nebulised or gargled providing sensory blockade in the airway. Intravenous administration is advantageous. Lignocaine at 1.5 mg/kg intravenous has been recommended before laryngoscopy and intubation. Optimal time of administration is 3 min before laryngoscopy and intubation[16]. Therapeutic concentration is achieved earlier than topical use. A clinical comparative prospective study of attenuation of sympathetic response to laryngoscopy and intubation was done in 150 patients posted for elective surgeries. General anaesthesia was provided with endotracheal intubation for all the patients.
Patients undergoing various Orthopaedic, Ear, Nose and Throat surgeries, Gynaecological, Neurosurgical and Laparoscopic procedures were selected. Following criteria's were adopted for selecting patients. There appeared no significant difference in heart rate at pre and post induction levels between lignocaine and fentanyl groups ($p=0.42$ and 0.97). The heart rate response between lignocaine and fentanyl groups was statistically highly significant at 1 and 3 minutes ($p<0.001$) and significant at 5 minutes ($p<0.01$) after the onset of laryngoscopy and intubation.

Keywords: Lignocaine, fentanyl, heart rate

Introduction

Plasma lignocaine level less than 5 µg/ml decrease the rate of spontaneous phase 4 depolarisation. At concentrations of 5-10 µg/ml there is myocardial depression and arteriolar relaxation resulting in profound hypotension, decrease in cardiac output and reduced systemic vascular resistance. At excessive plasma level cardiac conduction is slowed resulting in

increase in PR interval and QRS complex. Cardiovascular collapse and central nervous system toxicity dosage ratio CC/CNS is 7.1 for lignocaine i.e., seven times more lignocaine is required to induce cardiovascular collapse as is needed to produce convulsions. Ventricular arrhythmias are rare. CC/CNS ratio is less for pregnant woman [1, 2].

Lignocaine is useful to decrease sympathetic response to laryngoscopy and intubation when applied topically or sprayed or nebulised or gargled providing sensory blockade in the airway. Intravenous administration is advantageous. Lignocaine at 1.5 mg/kg intravenous has been recommended before laryngoscopy and intubation. Optimal time of administration is 3 min before laryngoscopy and intubation. Therapeutic concentration is achieved earlier than topical use. In addition to circulatory stability it suppresses cough, rise in intracranial pressure and rise in intraocular pressure due to intubation. It controls extubation related cough, laryngospasm and circulatory changes. Lignocaine also protects against arrhythmias [3, 4].

Fentanyl brings haemodynamic stability during peri operative period by its action on cardiovascular and autonomic regulatory areas. It decreases sympathetic tone and increases parasympathetic tone. There is no change in myocardial contractility. Atrioventricular node conduction is slowed with prolongation of R-R interval, AV node refractory period and action potential of purkinje fibres. There is no effect on coronary circulation.

Fentanyl inhibits pituitary adrenal response directly or indirectly via hypothalamus. Opioid receptors are distributed in cardiovascular and autonomic regulatory areas. These receptors are also found in myocardium and conducting system. Fentanyl decreases sympathetic outflow, controls secretion of vasopressin, suppresses somatosensory reflexes transmitted by unmyelinated 'C' fibres and modulates in spinal cord, brain stem and other sites. It increases central vagal tone and results in bradycardia. Its efficacy in controlling hormonal manifestations of stress response is dose dependent. It can also blunt the cardiovascular stimulation induced by inhalational agent like desflurane [5].

Blunting of sympathetic response is dose dependent. Fentanyl given intravenously at 2 µg/kg attenuates the response to laryngoscopy and intubation. Optimal time of administration is 5 minutes before laryngoscopy and intubation [6].

Methodology

A clinical comparative prospective study of attenuation of sympathetic response to laryngoscopy and intubation was done in 150 patients posted for elective surgeries. General anaesthesia was provided with endotracheal intubation for all the patients.

Patients undergoing various Orthopaedic, Ear, Nose and Throat surgeries, Gynaecological, Neurosurgical and Laparoscopic procedures were selected. Following criteria's were adopted for selecting patients.

Inclusion criteria

- Patients scheduled for elective surgeries.
- Age between 20 to 50 years of both the sexes.
- Patients with ASA class I and II.
- Mallampati airway assessment of class I and II.

Exclusion criteria

- Unwilling patients.
- Emergency surgeries.

- Anticipated difficult airway.
- Patients with ASA class III or higher.
- Patients with asthma, hypertension and other cardiovascular diseases.
- Patients on antihypertensive medications like beta blockers, calcium channel blockers and angiotensin converting enzyme inhibitors.
- Patients in whom laryngoscopy and intubation proved to be prolonged (more than 20 seconds) or difficult.

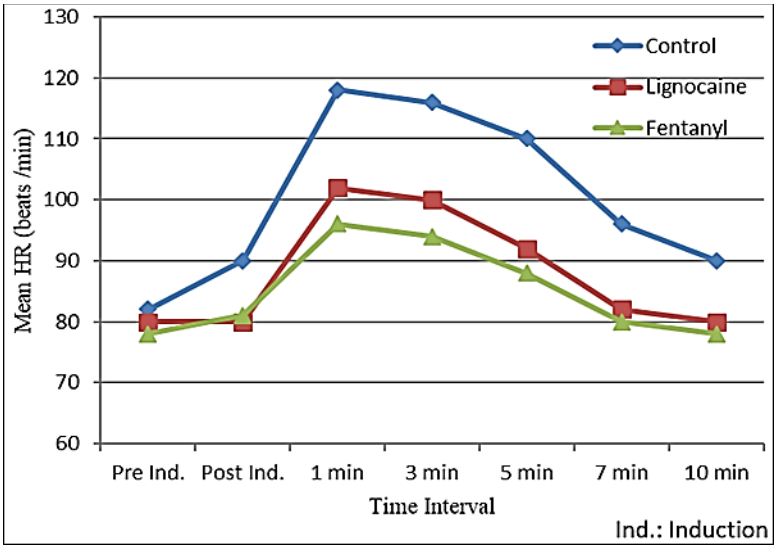
Patients were selected after thorough pre-anaesthetic assessment and investigations. An informed consent was taken with all the patients.
150 cases were divided into three groups of 50 each by *double blind randomization using a chit method.*
Group-I was Control group: In this group no drug was administered for attenuating sympathetic response to laryngoscopy and intubation.
Group-II was Lignocaine group: Here patients received 1.5 mg/kg of lignocaine intravenously 3 minutes before laryngoscopy and intubation.
Group-III was Fentanyl group: All the patients in this group received 2 µg/kg of fentanyl intravenously 5 minutes before laryngoscopy and intubation.

Results

Table 1: Comparison of Changes in Heart Rate

Time of Assessment	Control (I)		Lignocaine (II)		Fentanyl (III)		Anova 'F'* p	Diff. between groups**		
	Mean ± SD	% diff	Mean ± SD	% diff	Mean ± SD	% diff		I-II	I-III	II-III
Pre-induction	77.1 ± 7.0	-	79.3 ± 6.8	-	78.2 ± 6.7	-	1.29 0.27	0.14	0.44	0.42
Post induction	84.2 ± 7.9	8.7	81.8 ± 6.3	3.1	81.7 ± 5.3	4.5	2.28 0.10	0.10	0.06	0.97
1 min	118.8 ± 10.1	41.1	104.7 ± 8.1	32.0	98.4 ± 8.2	25.8	58.4 <0.001	<0.001	<0.001	<0.001
3 min	118.5 ± 11.9	41.0	103.5 ± 8.7	30.5	95.6 ± 8.4	22.2	83.6 <0.001	<0.001	<0.001	<0.001
5 min	106.7 ± 13.3	26.7	90.6 ± 6.2	14.2	86.5 ± 8.1	10.6	62.1 <0.001	<0.001	<0.001	<0.01
7 min	93.7 ± 11.5	11.3	82.6 ± 6.7	4.1	80.6 ± 6.9	3.1	33.6 <0.001	<0.001	<0.001	0.13
10 min	86.0 ± 8.7	2.1	78.8 ± 7.4	- 0.6	78.2 ± 5.5	0	71.4 <0.001	<0.001	<0.001	0.64

-ve sign indicates decrease, *one-way ANOVA, **Unpaired 't' test, $p<0.05$ is significant, $p<0.001$ is highly significant, $p>0.05$ is not significant.



Graph 1: Heart Rate

Changes in heart rate assessed at pre and post induction and at different time intervals from the onset of laryngoscopy and intubation in control and study groups and their comparative statistics are presented.

Control group

The mean heart rate in this group before induction of anaesthesia was 77.1 ± 7.0. Following induction, it increased by 9.2% with mean value of 84.2 ± 7.9. At one-minute interval from the onset of laryngoscopy and intubation, heart rate increased to 118.8 ± 10.1 (54.0% above the preinduction level) and remained at a significantly higher level with mean heart rate of 118.5 ± 11.9 (53.6% above preinduction level) at the end of 3 minutes ($p<0.001$).
A decreasing trend was noticed after 5 minutes, mean heart rate of 106.7 ± 13.3 which is 38.3% higher than pre-induction value.
Subsequently it decreased to 93.7±11.5 (21.5% above preinduction level) at the end of 7 minutes and 86.0±8.7 (11.5% above preinduction level) at the end of 10 minutes. It remained statistically significant even at the end of 10 minutes ($p<0.05$).

Lignocaine group

This study group shows the mean heart rate of 79.3 ± 6.8 before induction of anaesthesia. After induction of anaesthesia it increased by 3.1% with the mean of 81.8 ± 6.3. At 1 minute from the onset of laryngoscopy and intubation, the mean heart rate increased to 104.7 ± 8.1(32.0% above preinduction level). It decreased to 103.5 ± 8.7 (30.5% above preinduction level) after 3 minutes. Further it decreased to 90.6 ± 6.2 (14.2% above preinduction level) and 82.6 ± 6.7 (4.1% above pre-induction level) at the end of 5 and 7 minutes respectively. At the end of 10 minutes heart rate was 0.6% below pre-induction level.

Fentanyl group

The mean pre-induction heart rate in this group of patients was 78.2 ± 6.7. There was 4.5% increase in heart rate to 81.7 ± 5.3 after induction. A 25.8% increase in heart rate from preinduction level to 98.4 ± 8.2 was noticed at the end of 1 minute from the onset of laryngoscopy and intubation. A decrease in heart rate to 95.6 ± 8.4 (22.2% above preinduction level) was observed at the end of 3 minutes. Further it decreased to 86.5 ± 8.1 (10.6% above preinduction level) and 80.6± 6.9 (3.1% above preinduction level) at 5 and 7 minutes respectively. At the end of 10 minutes the heart rate was equal to the pre-induction level.
One-way ANOVA study showed that changes in heart rate before and after induction, and at time intervals of 1, 3, 5, 7 and 10 minutes from the onset of laryngoscopy and intubation was significant in all groups ($p<0.01$).
The difference in heart rate between control and lignocaine groups remained highly significant at all times of assessment ($p<0.001$).
Maximum increase in heart rate was 41.1% in control group and 32% in lignocaine group. It is statistically highly significant ($p<0.001$). It reached to a level which was clinically less significant by the end of 7 minutes in control group and 5 minutes in lignocaine group. The difference in heart rate between control and fentanyl groups remained statistically significant at all times of assessment ($p<0.001$).
The maximum increase in heart rate was 41.1% in control group and 25.8% in fentanyl group. Attenuation of heart rate by fentanyl when compared with control group is highly significant

($p<0.001$). Increase in heart rate remained clinically significant till the end of 5 minutes in control group and 3 minutes in the fentanyl group from the onset of laryngoscopy and intubation.

There appeared no significant difference in heart rate at pre and post induction levels between lignocaine and fentanyl groups ($p=0.42$ and 0.97). The heart rate response between lignocaine and fentanyl groups was statistically highly significant at 1 and 3 minutes ($p<0.001$) and significant at 5 minutes ($p<0.01$) after the onset of laryngoscopy and intubation.

No statistical significance appears between lignocaine and fentanyl groups at 7 and 10 minutes ($p=0.13$ and 0.64).

Discussion

Variability of heart rate changes decreases with increasing age. Younger patients show more extreme changes. Marked fluctuations in haemodynamic responses are often seen in geriatric patients. So we selected the optimal age range of 20 to 50 years.

Antihypertensive drugs can decrease pressor response. We excluded the patients taking antihypertensive drugs from our study.

Different drugs used for premedication, induction, relaxation and maintenance of anaesthesia influence the sympathetic response to laryngoscopy and intubation. Midazolam at a dose of 0.2 mg/kg given intravenously decrease the blood pressure, and increase the heart rate similar to thiopentone. However, premedication with 0.05 mg/kg of intravenous midazolam has minimal effect. Midazolam has no effect on sympathetic response to laryngoscopy and intubation. Hence in our study all the patients received midazolam as a premedicant [7]. Propofol was selected for induction because it continues to be the most popular anaesthetic agent and has replaced thiopentone. Propofol has a fast onset of action, rapid effect site equilibration, rapid redistribution, high hepatic clearance and rapid recovery after a bolus dose. It causes myocardial depression in a dose dependent manner. Systemic vascular resistance is reduced up to 30% without a compensatory tachycardia. In normovoluemic patient's propofol 1.5-2 mg/kg given intravenously can transiently decrease 10-20 mmHg of blood pressure. Decrease in blood pressure is usually offset by increase in the catecholamine levels during intubation [8].

Isoflurane has a varied response on the heart rate but it does not appreciably change the heart rate if used in less than 1 MAC. Isoflurane lowers arterial blood pressure principally as a result of reduced systemic vascular resistance when used in more than 1 MAC. In our study we have used isoflurane upto 1 MAC [9].

Pressor response with direct laryngoscopy and intubation when compared to nasotracheal intubation was significantly lesser in study conducted by Singh S and Smith JE[10]. Due to these observed differences we included only laryngoscopy and orotracheal intubation in our study.

Laryngoscopy alone may produce most of the cardiovascular responses reported after laryngoscopy and tracheal intubation during anaesthesia. The most important factor influencing the cardiovascular response is found to be the duration of laryngoscopy. A linear increase in heart rate and mean arterial pressure during first 45 seconds of laryngoscopy has been observed. Further prolongation has little effect. As duration of laryngoscopy is normally less than 30 seconds the result of studies in which it takes longer than this have less clinical relevance. The force applied during laryngoscopy has only minor effect. In our study the duration of laryngoscopy and intubation was limited to 20 seconds and cases which exceeded this duration were excluded from the study.

In our study, heart rate increased by 54.0% when compared to the basal value in control group at 1 minute interval ($p<0.001$). Similar increase with lignocaine group 32% and fentanyl group 25.8% was observed at 1 minute interval. Both lignocaine and fentanyl attenuated the heart rate response significantly ($p<0.001$). In control group, even at the end of 10 minutes heart rate fails

to reach statistically significant levels. Suppression of maximum rise in hear rate by fentanyl is statistically significant when compared with lignocaine ($p<0.001$). It remains significant till 5 minutes.

Conclusion

One way ANOVA study showed that changes in heart rate before and after induction, and at time intervals of 1, 3, 5, 7 and 10 minutes from the onset of laryngoscopy and intubation was significant in all groups ($p<0.01$).
The difference in heart rate between control and lignocaine groups remained highly significant at all times of assessment ($p<0.001$).
Maximum increase in heart rate was 41.1% in control group and 32% in lignocaine group. It is statistically highly significant ($p<0.001$). It reached to a level which was clinically less significant by the end of 7 minutes in control group and 5 minutes in lignocaine group.

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