

Attenuation of Cardiovascular Responses to Laryngoscopy and Intubation-A Comparative Study between IV Esmolol hydrochloride and Lignocaine Hydrochloride.

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ABSTRACT

Background: Laryngoscopy and tracheal intubation is invariably associated with a reflex Sympathetic pressor response resulting in elevated heart rate and blood pressures. This may prove detrimental in high-risk patients. Objective of this study is to compare the effects of lignocaine and esmolol in attenuation of this response. **Methods:** 150 ASA I - II status normotensive patients scheduled for elective surgical procedures were selected randomly and divided into three groups of 50 each. All patients received premedication with pentazocine 0.5mg/kg i.m and midazolam 0.05 mg/kg i.m. Induction of anesthesia was standardized for all patients who received, thiopentone 5-mg/kg i.v. and Glycopyrrolate 0.01. mg/kg and were relaxed with succinylcholine 2mg/kg i.v. First group did not receive any attenuation. The second group received 2mg/kg lignocaine i.v. bolus and the third group received 1mg/kg Esmolol iv. bolus, 3 minutes before laryngoscopy and intubation. HR, systolic, diastolic blood pressure was recorded noninvasively before induction, post induction-1,3,5, 7 and 10 minutes from the onset of laryngoscopy. 'z' test was used for statistical analysis. **Results:** After intubation incidence of tachycardia (HR>100/min) was significantly greater in control and lignocaine group than in esmolol group ($z > 1.96$, $p < 0.05-0.001$). Rise in SBP and DBP were also statistically significant in control and lignocaine group than in esmolol group ($z > 1.96$, $p < 0.05$). **Conclusion:** Attenuation of pressor response is seen both with lignocaine and with esmolol. Of the two drugs Esmolol 1mg/kg i.v. bolus provides a consistent, reliable and effective attenuation as compared to lignocaine 2mg/kg iv. bolus.

Keywords: Attenuation, pressor response, laryngoscopy, intubation, lignocaine, esmolol.

INTRODUCTION

Endotracheal intubation has become an integral part of anaesthetic management and critical care since its description in 1921 by Rowbotham and Magill. King et al. (1951) described the circulatory responses to laryngeal and tracheal stimulation following laryngoscopy and tracheal intubation as reflex sympatho-adrenal stimulation.^[1-3]

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Even though the elevation in blood pressure and heart rate due to laryngoscopy and intubation are brief, they may have detrimental effects in high risk patients including myocardial infarction, cardiac failure, intracranial hemorrhage and increases in intracranial pressure.^[4]

Laryngoscopy and tracheal intubation induces rise in nor-epinephrine, epinephrine and dopamine levels rise, but the raise in nor epinephrine levels is

consistently associated with elevation of blood pressure and heart rate.^[2,3,5-7]

Although the response may be transient, it is invariable, significant, often persistent and of great concern.^[7] Hence it is important to find an effective means of attenuating sympathetic response to laryngoscopy and endotracheal intubation.

Many strategies have been advocated to minimize these hemodynamic adverse responses and aimed at different levels of the reflex arc.^[7]

- Block of the peripheral sensory receptors and afferent input – topical application and infiltration of local anaesthetic to superior laryngeal nerve.
- Block of central mechanism of integration and sensory input – fentanyl, morphine etc.
- Block of efferent pathway and effector sites i.v. lignocaine, β blockers, calcium channel blockers, hydralazine etc.

No single drug or technique is 100% efficient.^[7]

Recommendations for attenuating the reflex hypertension and tachycardia are therefore manifold. The technique besides minimizing the cardiovascular responses to anaesthesia for patients at risk must also satisfy the following requirements.

1. It must be applicable regardless of patient's co-operation.
2. It should prevent impairment of cerebral blood flow and avoid arousal of the patient.
3. It should neither be time consuming nor affect the duration or modality of ensuing anaesthesia

Among the recommended procedures i.v. lignocaine, fentanyl and esmolol appear to fulfill the above mentioned criteria. Large doses of fentanyl may cause unwanted side effects like respiratory depression. Intravenous lignocaine has shown variable results. In one study conducted by Miller and Warren i.v lignocaine failed to attenuate the cardiovascular responses to laryngoscopy and intubation.

Esmolol is an ultra short acting β blocker and has been consistently associated with control of pressor response to laryngoscopy and intubation. The present study is undertaken to determine the efficacy of i.v. lignocaine 2 mg/kg bolus and i.v. esmolol 1mg/kg bolus in attenuating the sympathetic responses to laryngoscopy and tracheal intubation. The superiority of esmolol over lignocaine or vice versa will also be determined.

Objectives

The study of administration of i.v lignocaine or i.v esmolol before laryngoscopy and tracheal intubation has following objectives.

- 1) To observe the variations in sympathetic response to laryngoscopy and intubation.
- 2) To study the effectiveness of
 - a) Lignocaine 2 mg/kg i.v. administered 3min before laryngoscopy and intubation and
 - b) Esmolol 1mg/kg i.v. bolus administered 3min before laryngoscopy and intubation in attenuating the sympathetic response.
- 3) To ascertain the superiority of esmolol over lignocaine or vice versa in suppressing sympathetic response to laryngoscopy and tracheal intubation

Physiology of Sympathetic Response to laryngoscopy and Tracheal Intubation

Laryngoscopy and tracheal intubation are frequently associated with sympathetic response. Diagnostic laryngoscopy under anaesthesia and tracheal suctioning^[8] are also associated with adverse circulatory changes. Severe hypertension, tachycardia, increase in intracranial pressure can also be seen. Supraglottic traction during laryngoscopy^[9] or superficial stimulation of airway or passage of tracheal tube into trachea may be associated with reflex sympathetic changes.

Other contributory factors to hypertension and tachycardia like anxiety, are baroreceptor mediated

reflexes after induction than laryngotracheal stimulation^[1]. The tracheal intubation following laryngoscopy is not only accompanied by increased sympathetic activity but also increased sympathoadrenal activity. Increased hypothalamic activity and increased traffic in sympathetic efferent tracts are observed.

Different studies have shown rise of mean blood pressure of 25 mm Hg^[1], 20-40 torr when compared with awake control levels and 35-60 torr when compared with pre-intubation values and elevation of plasma noradrenaline and adrenaline by 45% and 40% respectively.^[7] A correlation between changes in mean arterial pressure and noradrenaline and pulse pressure or heart rate and adrenaline is found.^[2]

μ receptors are found in the CNS areas concerned with cardiovascular response. Neurons of this area contain an endogenous opioid called enkephalin. Opioids can also modulate the afferent impulses at spinal cord and brain stem. It can also modulate the activity of the hypothalamo-pituitary-adrenal axis i.e HPA axis.^[10]

Attenuation of sympathetic response to laryngoscopy and intubation:

Lignocaine is useful to decrease sympathetic response to laryngoscopy and intubation when applied topically or sprayed or nebulized or gargled providing sensory blockade in the airway. Intravenous administration is advantageous. Lignocaine at 1.5mg/kg IV has been recommended before laryngoscopy and intubation. Optimal time of administration is 3min 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 a rise in intraocular pressure due to intubation. It controls extubation related cough, laryngospasm and circulatory changes. Lignocaine also protects against arrhythmias

MATERIALS AND METHODS

A clinical comparative study of attenuation of sympathetic response to laryngoscopy and intubation was done in 150 patients posted for elective surgeries. Study was conducted in Fatima Institute of medical Sciences, Cuddapah. General anaesthesia was provided with endotracheal intubation for all the patients. Patients undergoing various orthopaedic, ENT, General Surgical, Neurosurgical,dental 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 grade I or II.
- Mallampati airway assessment of grade I.

Exclusion criteria:

- Unwilling patients
- Emergency surgeries
- Anticipated difficult intubation
- Patients with ASA grade III or higher .
- Patients with cardiovascular diseases
- Patients on beta-blockers or calcium channel blockers.
- Patients in whom laryngoscopy and intubation proved to be prolonged or difficult.

Patients were selected after thorough preanaesthetic assessment and investigations. An informed consent was taken in all the patients.

150 cases were divided into three groups with 50 cases in each group.

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 2mg/kg lignocaine i.v., 3 minutes before laryngoscopy and intubation.

Group-III was esmolol group. All the patients in this group received 1mg/kg i.v. bolus 3 minutes before laryngoscopy and intubation.

Investigations: Included - Hb%, TC, DC, and ESR; FBS; Blood urea and serum creatinine, ECG, Chest x- ray.

Premedication: All patients received Diazepam 10mg orally at night on the day before surgery. On the day of surgery intravenous line was secured and following pre-medications were given 45 minutes before induction - Inj. Midazolam 0.05mg/kg i.m, Inj. Pentazocine 0.5mg/kg i.m (maximum 30mg), Patients were monitored by pulse oximeter.

On entering the OT pulse oximeter, non-invasive blood pressure and ECG monitors were connected. A preinduction heart rate, systolic and diastolic blood pressures were recorded. i.v. Infusion of DNS solution was started.

Anaesthesia technique: All the patients were pre-oxygenated with 100% oxygen for 3 minutes before induction. Induction was achieved with Inj. Thiopentone sodium 5mg/kg i.v. given in 2.5%

solution. Inj. Glycopyrrolate 0.2 mg i.v. was given along with Thiopentone.

After induction of anaesthesia (loss of eyelash reflex), heart rate, systolic and diastolic blood pressures were recorded.

Succinylcholine was administered at a dose of 2mg/kg i.v.. Laryngoscopy was done using rigid laryngoscope with standard Macintosh blade. Intubation was done with an appropriate sized, disposable, high volume low-pressure cuffed endotracheal tube.

Laryngoscopy and intubation was done within 15 to 20 seconds.

Heart rate, systolic and diastolic blood pressure were recorded at 1,3,5,7 and 10 minute intervals from the onset of laryngoscopy.

In group-II, i.v. lignocaine 2mg/kg was administered 3 minutes before laryngoscopy and intubation.

In group-III, i.v. esmolol 1mg/kg was administered 3 minutes before laryngoscopy and intubation.

Patients were connected to Bain’s circuit and anaesthesia was maintained with oxygen (33%), N₂O (67%), halothane 0.5% and non-depolarising muscle relaxant vecuronium bromide at a dose of 0.05 mg/kg i.v. and IPPV. Adequacy of ventilation was monitored clinically and S_PO₂ was maintained at 99-100%. Positioning, epinephrine infiltration throat packing and surgery were withheld till the completion of recording. At the end of the surgery reversal was done with inj. Neostigmine 0.05 mg/kg and inj. Glycopyrrolate 0.01mg/kg i.v..

An observation was made related to adverse effects of drugs and anesthesia related problems and was attended to appropriately.

Statistical analysis: Descriptive data presents as Mean ± SD and in percentage. Pair wise comparison between the groups was done by ‘z’ test .For all tests a ‘z’ value of 1.96 was considered significant and a ‘p’ value of ≤ 005 was considered significant.

RESULTS

Table 1: Age Distribution.

	Control	Lignocaine	Esmolol
Minimum	20	20	20
Maximum	50	50	50
Mean	31.18	32.18	33.66
Std. deviation	9.79	10.64	10.83

The above table shows the age distribution in control and the two study groups. There was no significant difference between the three groups (p>0.05).

Table 2: Sex Distribution.

Sex	Group C		Group L		Group E	
	No.of cases	Percentage	No of cases	Percentage	No of cases	Percentage
Male	24	48	26	52	21	42
Female	26	52	24	48	29	58
Total	50	100	50	100	50	100

No significant difference was observed in sex wise distribution of the cases between the three groups ($p>0.05$).

Maximum	65	66	80
Mean	51.04	52.82	52.24
Std. deviation	6.06	7.04	8.76

Table 3: Weight Distribution.

	Control	Lignocaine	Esmolol
Minimum	38	38	40

No significant differences were observed weight wise between the three groups ($p>0.05$).

Table 4: Comparison of Heart Rates.

	Heart rate						Z test					
	Control		Lignocaine		Esmolol		I-II		II-III		I-III	
	Mean±SD	% Diff	mean±SD	% Diff	mean±SD	% Diff	Z value	P value	Z value	P value	Z value	P value
Pre induction	84.22±8.23	-	79.30±6.80	-	78.42±6.68	-	3.26	<0.001	0.66	0.745	3.88	<0.001
Post induction	91.48±10.69	8.7	81.76±6.27	3.1	82.96±6.09	5.7	5.55	<0.001	0.97	0.322	4.90	<0.001
1 min	118.80±10.11	41.1	104.74±8.09	32.0	89.28±5.90	13.8	7.68	<0.001	10.92	<0.0001	17.83	<0.0001
3 min	118.50±11.77	41.0	103.48±8.74	30.5	90.56±5.99	15.48	7.24	<0.001	8.62	<0.001	14.96	<0.0001
5 min	106.74±13.18	26.7	90.60±6.20	13.8	87.44±4.96	11.5	7.84	<0.001	2.81	<0.001	9.69	<0.001
7 min	93.72±11.47	11.3	85.58±5.86	7.91	81.96±3.92	4.51	4.47	<0.001	3.63	<0.001	5.24	<0.001
10 min	85.98±8.64	2.1	81.96±5.78	3.35	79.10±3.39	0.86	3.02	<0.001	2.73	<0.001	5.24	0.9442

Analysis of Heart Rate:

Statistical analysis of changes in heart rate at pre induction, post induction and at different time intervals from the onset of laryngoscopy and intubation in control and study groups are presented.

Control group: The pre- induction mean heart rate and standard deviations in this group were 84.22±8.23 respectively. After induction of anaesthesia there was 8.7% increase in the mean values. The mean values at post induction were 91.48±10.69. At one minute from the onset of laryngoscopy a 41.1% increase in the mean heart rate was observed with values of 118.80±10.11 and remained higher with a mean heart rate of 118.50±11.77 at 3 minutes. Subsequently a decreasing trend in the heart rate was noted starting from 5 minutes to 10 minutes after laryngoscopy. Mean heart rate at 5 minutes was 106.74±13.18 which was 26.7% higher than pre induction values. The heart rate at the end of 10 minutes was not significantly higher than the pre induction values.

Lignocaine group: In this group the mean heart rate and standard deviation at pre- induction were 79.30±6.80. After induction, there was an increase of 3.1% with mean of 81.76±6.27. At 1 minute from the onset of laryngoscopy, the heart rate rose to 104.74±8.09 with an increase of 32.0% from pre induction values. At 3 minutes, heart rate was observed to be 103.48 ± 8.74. Subsequently the mean heart rate decreased to 90.6±6.2 (13.8%) and 85.58±5.86 (7.91%) at 5 and 7 minutes respectively. At the end of 10 minutes, heart rate was 81.96±5.78,

which was 3.35% above the base line at preinduction.

Esmolol group: The mean preinduction heart rate in this group was 78.42±6.63. Post induction heart rate increased by 5.7% to 82.96±6.89. There was a further increase by 13.8% at 1-minute post laryngoscopy with a mean value of 89.28±5.90. A small further rise in the heart rate to 90.56±5.99 was noted. Heart rate began to decline from the 5th minute with a mean of 87.48±6.55 and it further decreased to 81.96±3.92 and 79.10±3.39 at 7th and 10th minutes respectively.

The difference in the heart rate between control and lignocaine groups remains very significant at all times of assessment ($z > 1.96$ and $p < 0.001$) except at 10th minute where it is statistically insignificant.

Maximum increase in heart rate in esmolol group was 15.48% at 3 minutes post laryngoscopy which was far less than control (41.1%) and lignocaine (32.0%).

Analysis by ‘z’ test showed significant variations 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. There was no significant difference in heart rate at pre and post induction levels between lignocaine and esmolol groups. ($z=0.66, p=0.75$ and $z = 0.97, p=0.97$).

The heart rate response between lignocaine and esmolol was very significant at all times starting from 1 to 10 minutes ($z > 1.96$ and $p < 0.001$) with esmolol showing a favourable response towards attenuation of heart rate.

Table 5: Comparison Of Systolic Blood Pressure.

	Systolic blood pressure						Z-test					
	Control		Lignocaine		Esmolol		I -II		II-III		I-III	
	Mean± SD	% Diff	mean± SD	% Diff	mean± SD	% Diff	Z value	P value	Z value	P value	Z value	P value
Pre induction	130.46±10.86	-	131.94±11.50	-	128.86±11.66	-	0.66	0.745	1.33	0.18	0.71	0.4777
Post induction	128.74±11.70	-	131.04±11.44	-0.7	125.52±10.98	-2.5	0.99	0.322	2.46	0.01	1.42	0.1556
1 min	156.44±11.48	19.8	151.12±13.74	14.5	133.86±10.55	6.6	2.10	0.036	7.05	<0.001	10.24	<0.0001
3 min	155.10±11.64	19.2	148.46±14.7	12.6	134.62±10.13	6.57	2.56	0.011	5.62	<0.0001	9.38	<0.0001
5 min	143.72±13.09	10.1	136.84±11.28	3.7	133.12±9.83	-1.1	2.82	0.005	1.76	0.08	4.58	<0.001
7 min	134.88±11.13	3.4	130.52±10.72	-1.1	131.14±9.49	-1.5	2.00	0.05	0.31	0.76	1.81	0.0703
10 min	129.72±10.01	-0.6	128.48±10.63	-2.6	130.04±9.40	-0.8	0.60	0.55	0.78	0.44	0.16	0.8729

Analysis of systolic blood pressure:

Control group: The mean pre-induction systolic blood pressure in this group was 130.46±10.86. After induction, there was a 1.4% fall in systolic blood pressure to 128.74±11.70. At 1 minute following laryngoscopy, a 19.8% increase in systolic blood pressure was noted with a mean value of 156.44±11.48. Starting from 3 minutes there was a fall in systolic blood pressure to 19.2% the pre induction value at 3 minutes, 10.1% pre-induction value at 5 minutes and at the end of 10 minutes systolic blood pressure came down to 0.6% below baseline with a mean value of 129.72±10.01.

Lignocaine group: Pre-induction systolic blood pressure in this group was 131.94±11.50. Post induction a marginal fall of 0.7% with a mean of 131.04 was observed. There was a 14.5% increase in systolic blood pressure following one minute after laryngoscopy with a mean value of 151.12±13.74. At 3 minutes a decrease in systolic blood pressure to 148.46±14.17 was noted. The systolic blood pressure then decreased and at the end of 10 minutes to 2.6% below the base line systolic blood pressure was observed with a mean value of 128.48±10.63.

Esmolol group: In this group, pre- induction systolic blood pressure was 128.86±11.65. On induction, the systolic blood pressure decreased by 2.5% to 125.52±10.98. A 3.9% increase in systolic blood pressure with a mean of 133.86±10.55 was noted at 1 minute following laryngoscopy. Systolic blood pressure increased slightly with a mean of 134.62±10.13 at 3 minutes post laryngoscopy. From there on a gradual fall in systolic blood pressure was noted as 133.12±9.83 at 5 minutes, 131.14±9.49 at 7 minutes. At 10 minutes, post laryngoscopy the systolic blood pressure almost returned to base line with a mean value of 130.04±9.40.

No significant variations were noted in all groups in systolic blood pressure pre and post induction. In comparison to control group and lignocaine group attenuation of systolic blood pressure is significant in lignocaine group (p <0.05). In comparison to control group, the rise in systolic blood pressure was only 3.9% in esmolol group, which is statistically highly significant (z >1.96, p<0.001).

Esmolol group showed a better attenuation compared to lignocaine group in systolic blood pressure until 3 minutes post laryngoscopy.

Table 6: Comparison Of Diastolic Blood Pressure

	Diastolic blood pressure						Z test					
	Control		Lignocaine		Esmolol		I -II		II-III		I-III	
	Mean± SD	% Diff	mean± SD	% Diff	mean± SD	% Diff	Z value	P value	Z value	P value	Z value	P value
Pre induction	76.28±6.12	-	76.72±5.77	-	76.40±5.08	-	0.37	0.71	0.29	0.77	0.11	0.9124
Post induction	74.00±6.40	-3.0	75.74±5.37	-1.0	74.12±4.37	-2.9	1.47	0.14	1.65	0.10	0.11	0.9124
1 min	89.84±5.20	17.7	86.24±5.22	12.4	81.02±4.49	9.3	3.45	<0.001	5.36	<0.001	9.08	<0.0001
3 min	89.80±5.18	17.2	84.92±5.29	10.7	81.74±3.85	0.9	4.28	<0.001	3.44	<0.001	8.39	<0.0001
5 min	84.60±5.18	10.9	136.84±11.28	4.0	80.24±3.93	-1.8	4.57	<0.001	0.56	0.58	4.24	<0.001
7 min	79.18±5.52	3.8	76.10±4.70	-0.8	78.96±4.04	-1.6	3.00	<0.001	3.26	<0.001	0.23	0.8181
10 min	76.68±5.47	0.5	75.10±4.43	-2.1	78.02±3.85	-1.2	1.59	0.11	3.52	<0.001	1.42	0.1556

Analysis of Diastolic Blood Pressure

Control group: Mean pre- induction diastolic blood pressure in this group was found to be 76.28±6.12. A

3% fall to 74.00±6.40 was noted after induction of anaesthesia. It increased by 17.7% to 89.84±5.20 at 1 minute. A 17.2% increase was still maintained with mean of 89.40±5.18 at 3 minutes. It decreased to 84.60±6.11 and then to 79.18±5.52 at 5 and 7 minutes respectively. At the end of 10 minutes the diastolic blood pressure returned to baseline with a mean of 76.68±5.27.

Lignocaine group: In this group mean diastolic blood pressure was 76.72±5.77 before induction. A decrease to by 1% to 75.74±5.37 was noted after induction. It increased by 12.4% to 86.24±5.22 at 1 minute post laryngoscopy. It came down to 84.92±5.29 at 3 minutes and continued to fall at 5 and 7 minutes to 79.78±4.28 and 76.10±4.70 respectively. By the end of 10 minutes the diastolic blood pressure was at 75.10±4.43 a 2% below the baseline.

Esmolol group: Diastolic blood pressure in esmolol group before induction was 76.40±5.08. After induction 2.9% fall to 74.12±4.37 was noted. An increase in 6.0% to 81.02±4.49 and 6.9% to 81.74±3.85 was noted at 1 and 3 minutes respectively. Over 5, and 7 minutes the diastolic blood pressure decreased to 80.24±3.93 and 78.96±4.04 respectively. At the end of 10 minutes it was 2% above the baseline with a mean of 78.02±3.85.

A maximum rise of 12.4% as compared to 17.7% was noted between lignocaine and control groups. Attenuation of diastolic blood pressure by lignocaine as compared to control group is very significant until 7 minutes ($z > 1.96, p < 0.001$).

A maximum rise of only 6.9% of diastolic blood pressure was seen in esmolol group, which is statistically highly significant ($z > 1.96, p < 0.001$).

Attenuation of diastolic blood pressure was very significant with esmolol than with lignocaine group until 5 minutes ($z > 1.96, p < 0.001$).

Table 7: Comparison of Mean Arterial Blood Pressure.

	Mean arterial blood pressure						Z test					
	Control		Lignocaine		Esmolol		I-II		II-III		I-III	
	Mean±SD	% Diff	mean±SD	% Diff	mean±SD	% Diff	Z value	P value	Z value	P value	Z value	P value
Pre induction	94.34±6.68	-	95.13±6.55	-	93.80±5.75	-	0.59	0.5552	1.08	0.2801	0.44	0.6599
Post induction	92.25±6.92	-2.2	94.17±6.05	-1.0	91.17±5.16	-2.8	1.48	0.1389	2.67	0.0076	0.89	0.3735
1 min	112.04±6.17	18.8	107.87±7.00	13.5	98.61±5.24	8.1	3.16	<0.001	7.49	<0.001	11.73	<0.0001
3 min	111.30±6.17	18.0	106.10±7.27	11.6	99.34±4.72	0.74	3.86	<0.001	5.51	<0.001	10.89	<0.0001
5 min	104.31±6.86	10.6	98.80±5.19	3.9	97.86±4.66	-1.5	4.53	<0.001	0.96	0.3371	5.50	<0.001
7 min	97.75±6.33	3.6	94.24±5.45	-1.0	96.29±4.42	-1.6	2.97	0.003	2.07	0.0385	1.34	0.1802
10 min	94.36±5.89	0.1	92.89±5.46	-2.3	95.29±4.17	-1.0	1.29	0.1971	2.47	0.0135	0.91	0.3628

Analysis of Mean Arterial Blood Pressure:

Control group: Mean arterial pressure before induction in this group 94.34±6.68. It decreased by 2.2% to 92.25±6.92 after induction. It increased by 18.8% to 112.04±6.17 at 1-minute post laryngoscopy and intubation. It decreased marginally by 18.0% of pre induction values at 3 minutes and continued to decrease. At 10 minutes post laryngoscopy it was 0.1% above baseline.

Lignocaine group: Pre induction means value in this group was 95.13±6.55-post induction fall was 1% to 94.17±6.05. It increased by 13.5% to 107.87±7.00 at 1 minute and a marginal fall to 106.10±7.27 was noted at 3 minutes. It decreased further over 5,7 and 10 minutes. At 10 minutes post laryngoscopy it showed a decrease of 2.3% below base line to 92.89±5.46.

Esmolol group: Pre- induction mean arterial blood pressure was 93.80±5.75 in this group. The

maximum rise by 5.9% to 99.34±4.72 was observed at 3 minutes post laryngoscopy. From then on it continued to fall. At the end of 10 minutes mean arterial blood pressure was 1.5% above base line with mean value of 95.29±4.17.

Significant differences are seen in all groups at 1,3,5 and 7-minute intervals ($z > 1.96, p < 0.01$). Attenuation of pressor response by lignocaine when compared to control is significant ($z > 1.96, p < 0.001$). When compared to control group attenuation by esmolol group is very highly significant ($p < 0.001$). Among the two study groups esmolol is significant in attenuating pressor response compared to lignocaine ($z > 1.96, p < 0.05$).

DISCUSSION

The sequence of induction of anaesthesia, laryngoscopy and tracheal intubation are associated

with marked haemodynamic changes and autonomic reflex activity, which may be a cause of concern in many high risk patients.^[11] These potentially dangerous changes disappear within 5 minutes of onset of laryngoscopy.^[12]

An average rise in mean arterial pressure of 25mm Hg and 47.7 mmHg have been documented.^[13] An increase in mean arterial pressure of 26.5 mm Hg and 20 to 40 torr when compared with awake control levels and 35 to 60 torr when compared with pre-intubation values^[2,9,14] have been reported after placement of an endotracheal tube. A rise in mean heart rate of 29.9 beats/min has also been noted.^[13]

Many factors influence the cardiovascular changes associated with laryngoscopy and intubation. Age, drugs, type and duration of procedures, depth of anaesthesia, hypoxia, hypercarbia etc., influence the pressor response.

A variable combination of drugs used for premedication, induction, relaxation and maintenance of anaesthesia can influence the sympathetic response to laryngoscopy and intubation.

Thiopentone was selected for induction since it still continues to be the most popular agent for induction. In normovolemic patients thiopentone 5mg/kg i.v can transiently decrease 10-20mm Hg of blood pressure and increase the heart rate by 15-20 beats/min.^[15] There is increase in catecholamine levels, both nor-adrenaline and adrenaline

Succinylcholine has negative inotropic and chronotropic effect. It acts on the muscarinic receptors of SA node. A marked noradrenergic response was noted when intubation was performed under Succinylcholine.^[16]

Nitrous oxide may increase the tone of sympathetic nervous system. The direct action of nitrous oxide is negative inotropism, which is offset by increased sympathetic tone.

Laryngoscopy alone may produce most of the cardiovascular responses reported after laryngoscopy and tracheal intubation during anaesthesia.^[17]

The most significant laryngoscopic factor influencing cardiovascular responses is found to be the duration of laryngoscopy. A linear increase in heart rate and mean arterial pressure during the first 45 seconds has been observed. Further prolongation has little effect. As the duration of laryngoscopy is normally less than 30 seconds, the results 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.

Adequate care was taken to achieve the required depth of anaesthesia avoiding hypoxia and hypercarbia, which can influence the hemodynamic variations.

Many strategies have been recommended which include minimizing the duration of laryngoscopy to

less than 20 seconds, topical application of local anaesthetics^[17,18] iv beta-blockers^[19-23], calcium channel blockers^[12], Clonidine^[24], Sodium Nitroprusside^[14], lignocaine. No single drug or technique is satisfactory.^[7] Each technique has advantages and disadvantages, the most obvious being that the prevention often outlasts the stimulus.

Though intravenous lignocaine failed to attenuate the cardiovascular responses to laryngoscopy and intubation in a study by Miller CD and Warren SJ²⁵, its efficacy was noted by others^[18,26-28] Lignocaine also prevents rise in intracranial pressure¹⁰, rise in intraocular pressure associated with laryngo tracheal stimulation. It also suppresses cough related to extubation. It is recommended to use at a dose of 1.5 to 2 mg/kg .iv.^[26,27]

Optimal time for administration is 3 minutes before laryngoscopy and intubation.^[27] Esmolol is a beta-blocking agent with several desirable properties. It is relatively cardioselective, ultrashort acting, with rapid onset of action.^[29-32]

1mg/kg bolus doses have been found to be more efficient and reliable in alternating both heart rate as well as blood pressure responses.^[20] In our study we used 1mg/kg i.v. bolus doses of esmolol.

In our study heart rate increased to a maximum of 41.1% when compared to pre induction value in the control group ($z > 1.96$, $p < 0.001$). Similar increases in lignocaine was 30.5% and in esmolol group by 15.48%. Both lignocaine and esmolol attenuated the heart rate significantly ($z > 1.96$, $p < .001$). It reaches a level, which is clinically less significant by the end of 7 minutes in control group and by the end of 5 minutes in lignocaine and esmolol group. Attenuation of maximum rise in the heart rate by esmolol is evident and statistically highly significant when compared with lignocaine ($z > 1.96$, $p < 0.001$).

The efficiency of esmolol over lignocaine in attenuation of cardiovascular responses similar to our study has been verified by many other studies.^[19,21,22,33] A combination of both lignocaine and esmolol has been recommended for better responses.^[22]

CONCLUSION

Based on the present clinical comparative study the following conclusions can be made.

In patients with no drugs to attenuate the sympathetic responses to laryngoscopy and intubation the maximum rises in heart rate, systolic, diastolic and mean arterial blood pressures were statistically and clinically very highly significant and can be detrimental in high-risk patients.

Lignocaine significantly attenuates the sympathetic responses to laryngoscopy and tracheal intubation.

Esmolol also very significantly attenuates the sympathetic responses.

Esmolol is more efficient than lignocaine in attenuating the sympathetic responses to laryngoscopy and intubation.

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