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Original research article

A comparative study of the efficacy of intravenous lignocaine hydrochloride and intravenous esmolol hydrochloride in attenuating hemodynamic responses to laryngoscopy and tracheal intubation

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Abstract

Background and Objectives: High blood pressure and heart rate are common side effects of laryngoscopy and tracheal intubation, triggered by the body's reactive sympathetic pressor response. In patients at high risk, this could have a negative impact. The purpose of this research is to examine how lignocaine and esmolol affect this reaction and to draw comparisons between the two.

Methods: One hundred and fifty normotensive patients with ASA I or II status who were scheduled for elective surgery were selected at random and split into three groups of 50. All patients were premedicated with 0.2 milligrammes of pentazocine intravenously and 0.02 milligrammes of midazolam intravenously. All patients were given the same dose of intravenous thiopentone (5 mg/kg) for induction of anaesthesia. Succinylcholine 2 mg/kg i.v. with 0.01% glycopyrrolate mg/kg produced a calming effect. The initial batch did not get any dampening. Three minutes before laryngoscopy and intubation, the second group received an intravenous bolus of 1.5 mg/kg lignocaine, whereas the third group received an intravenous bolus of 2 mg/kg Esmolol. Noninvasive measurements of heart rate and systolic and diastolic blood pressure were taken at 0, 1, 3, 5, and 10 minutes after laryngoscopy had begun. Statistical significance was determined using the 'Z' test.

Results: Following intubation, the incidence of tachycardia with a heart rate of more than 100 beats per minute was substantially higher in the control and lignocaine groups than in the esmolol group ($z>1.96$, p0.05-0.001) The control group and the lignocaine group both showed a statistically significant rise in their blood pressure compared to the esmolol group ($z > 1.96$, p 0.05).

Conclusion: Both lignocaine and esmolol have been shown to have the effect of dampening the pressor response. When compared to lignocaine 1.5mg/kg i.v bolus, the attenuation provided by esmolol 2 mg/kg i.v bolus is more constant, dependable, and effective than that provided by lignocaine 1.5mg/kg i.v bolus.

Keywords: Tracheal intubation, tachycardia, lignocaine, anaesthesia, laryngoscopy

Introduction

Since Rowbotham and McGill first described endotracheal intubation in 1921, it has developed into an essential component of both the administration of anaesthesia and intensive medical care. Reflex sympatheticoadrenal stimulation is how King *et al*. (1951) characterised the circulatory responses that occurred after laryngoscopy and tracheal intubation in response to laryngeal and tracheal stimulation [1, 2, 3].

Even though the increases in blood pressure and heart rate that are caused by laryngoscopy and intubation only last for a short period of time, these procedures have the potential to cause serious complications in high-risk patients, such as myocardial infarction, cardiac failure, intracranial haemorrhage and increases in intracranial pressure [4].

Both laryngoscopy and tracheal intubation can cause considerable alterations in the quantities of catecholamines that are circulating in the blood. The levels of norepinephrine, epinephrine and dopamine all increase, but the increase in norepinephrine levels is the only one that is reliably linked to an increase in both blood pressure and heart rate [2, 3, 5, 6, 7].

In fact, some anaesthetists consider the intubation period to be one of the surgical patients with coronary artery disease and patients who have intracranial aneurysms who are at the greatest risk during this part of the procedure. In spite of the fact that the reaction could be fleeting, it is invariably significant, frequently enduring, and a source of major worry [7].

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The procedures of laryngoscopy and tracheal intubation are not just used in the operating room; rather, they are also utilised for a variety of other purposes outside of the realm of anaesthesia. In a few rare cases, such as during diagnostic laryngoscopy or fiberoptic bronchoscopy, intubation may be necessary. In other situations, such as during mechanical breathing in intensive care units or to prevent aspiration, intubation may also be necessary. All of these treatments have the potential to produce sympathetic responses, and it is important to keep in mind that the majority of these patients are in a critical condition and are at a higher risk.
As a result, it is essential to identify an efficient method for reducing sympathetic responses to laryngoscopy and tracheal intubation [7].

Materials & Methods
A clinical investigation comparing the attenuation of sympathetic response to laryngoscopy and intubation was carried out on 150 individuals who were scheduled to undergo elective procedures. The research was carried out at Department of Anaesthesia, Government Medical College, Kadapa, Andhra Pradesh, India, over a period of December 2021 to November 2022. All of the patients had general anaesthesia, and endotracheal intubation was performed on each of them.
Individuals who were scheduled to have laparoscopic surgery, orthopaedic surgery, ENT surgery, gynaecological surgery, general surgery, or neurological surgery were chosen for this study.
The selection of patients was determined by using the following criteria.

Inclusion criteria
- Patients who are set to have elective procedures
- The age range for both sexes is between 20 and 50 years old.
- Patients classified as having ASA grade I or II.
- An evaluation of Mallampati airways for grades I and II

Exclusive criteria
- Patients that are uncooperative.
- Patients with an ASA grade of III or higher.
- Patients with cardiovascular illness.
- Patients using beta-blockers or calcium channel blockers.
- Patients undergoing emergency surgery.
- Patients who are expected to have a difficult time intubating them.
- Those for whom the laryngoscopy and intubation process was found to be time-consuming or challenging.

Participants were chosen for the study after undergoing exhaustive preoperative evaluations and examinations.
All of the patients gave their consent after receiving information about the study.
There were a total of 150 cases, which were split evenly among three groups of 50 instances each.
This was the control group, Group I. In this group, no medication was given to the patients in order to dull their sympathetic response to the laryngoscopy and intubation procedures.
The lignocaine group was known as Group-II. Patients in this study were given lignocaine intravenously at a dose of 1.5 milligrammes per kilogramme three minutes before their laryngoscopy and intubation.
The esmolol group was considered Group III. Before beginning the laryngoscopy and intubation procedures, all of the patients in this group were given a bolus of 2 mg/KG administered intravenously.

Results
Statistical analysis
Data provided descriptively, with means and standard deviations, as well as in percentages Comparisons between the groups were made using the 'Z' statistic, and a value of 1.96 was deemed to be statistically significant.

Table 1: Age Distribution

Groups	Age in year (Mean ± SD)
Control group	31.19±0.43
Lignocaine group	32.18±0.84
Esmolon group	33.6±0.57

The mean and standard deviation of the control group's age, in years, is 31.19. The mean and standard deviation (in years) for the lignocaine group is 32.18. The mean and standard deviation of the age in years for the esmolon group is 33.6. It was determined that there was not a significant difference (p>0.05) in the sex-based distribution of the cases between the groups. On the page facing you is a

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graphical representation of the information.

Table 2: Sex Distribution

Sex	No. of cases	Percentage (%)
Male	24	48%
Female	26	52%
Total	50	100%

In the control group, male patients accounted for 48% of the total, while female patients made up 52% of the total. 52% of the patients in the lignocaine group were male, whereas the other 48% were female.
42% of the patients in the Esmolol group were male, while 58% of the patients were female. It was determined that there was not a significant difference (p>0.05) in the sex-based distribution of the cases between the groups.

Table 3: Weight Distribution

	Control	Lignocaine	Esmolol
Minimum	38	38	40
Maximum	65	66	80
Mean	51.04	52.82	52.24
Std. deviation	6.06	7.04	8.76

In the control group, the weight range is from 38 to 65 kilogrammes, and the mean value is 51.04 with a standard deviation of 6.06.
In the lignocaine group, the range of weights was 38–66 kg, with a mean of 52.82 and a standard deviation of 7.04 kg.
The Esmolol group had a weight distribution that ranged from 40 to 80 kgs, with a mean value of 52.24 and a standard deviation of 8.76.
By comparing the weights of the three groups, there was not a discernible difference seen (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 ±6.63	–	3.26	<0.001	0.66	0.745	3.88	<0.0001
Post Industion	91.48 ±10.69	8.7	81.76 ±6.27	32	82.96 ±6.09	5.7	5.55	<0.001	0.97	0.322	4.9	<0.0001
1 Minute	118.80 ±10.11	41.11	104.74 ±8.09	30.5	89.28 ±5.90	13.8	7.68	<0.001	10.92	<0.0001	17.83	<0.0001
3Minute	118.50 ±11.77	41	103.48 ±8.74	3.1	90.56 ±5.99	15.48	7.24	<0.001	8.62	<0.001	14.96	<0.0001
5Minute	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	<0.0001
7Minute	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	6.86	<0.0001
10Minute	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	5.21	0.9442

A statistical analysis of the changes in heart rate before and after the induction of anaesthesia as well as at various time intervals beginning with the beginning of laryngoscopy and intubation is reported for both the control group and the study group.

The control group is comprised of
This group's pre-induction mean heart rate was 84.22 beats per minute, and their standard deviations ranged from 8.22 to 8.23. The mean results saw a rise of 8.7% after the induction of anaesthesia had been performed. After induction, the mean values were 91.48 with a standard deviation of 10.69. A 41.1% increase in the mean heart rate was detected with values of 118.8010.11 at one minute from the beginning of laryngoscopy, and it stayed higher with a mean heart rate of 118.8011.77 after three minutes. This was observed one minute after the beginning of laryngoscopy. After that, a pattern of the heart rate gradually slowing down was observed beginning five minutes to ten minutes after the laryngoscopy was completed. After five minutes, the average heart rate was 106.74 13.18, which was 26.7% higher than the data before the induction. After ten minutes, there was not a discernible increase in the heart rate compared to the values taken before the induction.
The lignocaine group had a mean heart rate of 79.30 beats per minute and a standard deviation of 6.80 beats per minute at the time of pre-induction. Following induction, there was an increase that accounted

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for 3.1%, with a mean value of 81.76 6.27. When one minute had passed since the beginning of the laryngoscopy, the patient's heart rate had increased to 104.74 8.09, representing an increase of 32.0% from the readings obtained before the induction. At three minutes, the observer recorded a heart rate of 103.48 + 8.74. After then, the mean heart rate continued its downward trend, reaching 90.6 6.2 (13.8%) at 5 minutes, and then 85.58 5.86 (7.91%) at 7 minutes. After ten minutes, the patient's heart rate was recorded at 81.96 5.78, which was 3.35 percentage points higher than the baseline during preinduction.

Esmolol group

This sample had a mean preinduction heart rate of 78.426.63, with a standard deviation of 6.63. After the induction, the patient's heart rate climbed by 5.7%, coming in at 82.966.89. At one minute following the laryngoscopy, there was an additional increase of 13.8%, resulting in a mean value of 89.28 5.90. It was observed that the patient's heart rate had increased slightly more, reaching 90.56 + 5.99. The mean heart rate was 87.486.55 at the end of the fifth minute, and it continued to drop to 81.963.92 and 79.103.39 at the end of the seventh and tenth minutes, respectively. The decline in heart rate began at the fifth minute.

At each and every point of time during the study, the changes in heart rate between the control group and the lignocaine group were shown to be statistically significant (z > 1.96 and p 0.001).

The control group experienced a maximum rise in heart rate of 41.1%, whereas the lignocaine group experienced an increase of 32%. It has a very high level of statistical significance (p 0.001). Until for the tenth minute, when it becomes statistically insignificant, these differences in heart rate between the control group and the esmolol group remain statistically very significant at all times. The difference between 1 and 3 minutes after laryngoscopy is statistically significant (p 0.001) and clinically meaningful (z > 1.96). The maximum rise in heart rte seen in the esmolol group was 15.48% at 3 minutes after laryngoscopy. This was much lower than the increases seen in the control (41.1%), as well as the lignocaine (32.0%) groups.

Increases in heart rates were clinically significant until the end of 7 minutes in the control group and until the end of 5 minutes in the lignocaine group. However, increases in heart rates in the esmolol group were not clinically significant at any time.

Analyses using the z test revealed statistically significant shifts in heart rate both before and after induction, as well as at intervals of 1, 3, 5, 7, and 10 minutes after the beginning of laryngoscopy and intubation.

The lignocaine group and the esmolol group did not significantly differ from one another in terms of their heart rates before and after the induction. (z-0.66, p=0.75 and z=0.97, p=0.97).

The difference in heart rate response between lignocaine and esmolol was highly significant at all times starting from 1 to 10 minutes ('z' > 1.96 and p 0.001), with esmolol displaying a favourable reaction 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 Industion	128.74 ±11.70	-1.4	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 Minute	156.44 ±11.48	19.8	151.12 ±13.74	14.5	133.86 ±10.55	6.6	2.1	0.036	7.05	<0.0001	10.24	<0.0001
3Minute	155.10 ±11.64	19.8	148.46 ± 14.17	12.6	134.62 ±10.13	6.57	2.56	0.011	5.62	<0.001	9.38	<0.0001
5Minute	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	<0.0001
7Minute	134.88 ±11.13	3.4	130.52 ±10.72	-1.1	131.14 ±9.49	-1.5	2	0.05	0.31	0.76	1.81	0.07.3
10Minute	129.72 ±10.01	-0.6	128.48 ±10.63	-2.6	130.04 ±9.40	-0.8	0.6	0.55	0.78	0.44	0.16	0.8729

The differences in the systolic blood pressure changes that were detected between the control group and the study group before and after the induction as well as at various time intervals are provided.

The control group had a mean preinduction systolic blood pressure of 130.46 10.86, which was significantly lower than the results of the experimental group. After the induction, the patient's systolic blood pressure dropped to a reading of 128.74 mm Hg and 11.70. After one minute had passed since the laryngoscopy, the patient's systolic blood pressure was found to have increased by 19.8%, with a mean reading of 156.4411.48. Starting at three minutes in, there was a drop in systolic blood pressure to 19.2% of the pre-induction value at three minutes, 10.1% of the pre-induction value at five minutes, and at the end of ten minutes, systolic blood pressure fell down to 0.6% below baseline with a mean value of

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129.7210.01.
In the lignocaine group, the pre-induction systolic blood pressure read 131.94 with a standard deviation of 11.50. After the induction, a slight decline of 0.7% was seen, with a mean of 131.04 being recorded.
One minute after laryngoscopy resulted in an increase in systolic blood pressure of 14.5%, with a mean value of 151.1213.74. The value was taken immediately after the procedure. After three minutes, the systolic blood pressure had dropped to 148.4614.17 from its previous level. After then, the systolic blood pressure started to drop, and at the end of the 10 minutes, it had reached a value that was 2.6% lower than the baseline systolic blood pressure, with a mean value of 128.4810.63.
In the group that received esmolol, the pre-induction systolic blood pressure reading was 128.86 minus 11.65. After the induction, the patient's systolic blood pressure dropped by 2.5%, coming in at 125.52 minus 10.98. After one minute of laryngoscopy, the patient's systolic blood pressure rose by 3.9% to a mean of 133.86 10.55. This reading was taken immediately after the procedure. At three minutes after the laryngoscopy, the patient's systolic blood pressure had somewhat increased to a mean of 134.621013. From that point, a progressive decrease in systolic blood pressure was seen, with readings of 133.12 minus 9.83 at 5 minutes and 131.14 minus 9.49 at 7 minutes. Within 10 minutes following the laryngoscopy, the patient's systolic blood pressure had nearly recovered to its baseline value, with a mean of 130.04 9.40.
Before and after the induction, the systolic blood pressure of any of the groups studied did not show any significant differences. When compared to the control group and the lignocaine group, the attenuation of systolic blood pressure that occurred in the lignocaine group was significantly greater. When compared to the control group's 19.8% increase in systolic blood pressure, the 14.5% increase seen in the lignocaine group was statistically significant (p 0.05).
The rise in systolic blood pressure in the esmolol group was only 3.9%, which is statistically highly significant (z>1.96, p0.001) when compared to the control group.
When compared to the lignocaine group, the esmolol group displayed a more significant attenuation in systolic blood pressure until three minutes after the laryngoscopy. At one minute immediately following laryngoscopy, the difference was found to be statistically significant (z > 1.96, p 0.001). In terms of statistics, there was not much of a significant difference between the two groups after 5.7 and 10 minutes.

Table 6: Diastolic Blood Pressure

	Diastolic blood peessure						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 Industion	74.00 ±6.40	-3	75.74 ±5.37	-1	74.12 ±4.37	-2.9	1.47	0.14	1.65	0.1	0.11	0.9124
1 Minute	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
3Minute	89.40 ±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
5Minute	84.60 ±6.11	10.9	79.78 ±4.28	4	80.24 ±3.93	-1.8	4.57	<0.001	0.56	0.58	4.24	0.001
7Minute	79.18 ±5.52	3.8	76.10 ±4.70	-0.8	78.96 ±4.04	-1.6	3	<0.001	3.26	<0.001	0.23	0.8181
10Minute	76.68 ±5.47	0.5	75.10 ±4.43	-2.1	78.02 ±3.85	-1.2	1.59	0.1	3.52	<0.001	1.42	0.1556

The differences in diastolic blood pressure that were measured before and after induction, as well as at various time intervals, are compared between the control group and the study group.

The control group is comprised of
This group's pre-induction diastolic blood pressure averaged out to 76.28 6.12 mm Hg, according to the findings. After the beginning of the anaesthetic, the patient's vital signs dropped by 3% to 74.006.40. At one minute, it rose to 89.845.20 having increased by 17.7%. At three minutes, the mean value was 89.40 5.18, and the increase of 17.2% was still maintained. At five minutes, it was 84.60 6.11, and then at seven minutes, it was 79.18 5.52. After a period of 10 minutes, the diastolic blood pressure had reverted to its initial value of 76.68 5.27, which was the mean value.
Before the induction, the participants in the lignocaine group had a mean diastolic blood pressure of 76.72 5.77. After the introduction, a drop of 1% was observed, bringing the total to 75.74 5.37. After one minute after the laryngoscopy, it rose to 86.24 5.22, an increase of 12.4%.
It dropped to 84.925.29 after three minutes, and it continued to drop after five and seven minutes, reaching 79.784.28 and 76.104.70 correspondingly. After ten minutes, the diastolic blood pressure had

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dropped to 75.10+4.43, which is a 2% decrease from the initial reading.
Esmolol group:
Before the induction, the diastolic blood pressure of those receiving esmolol was 76.40 5.08. After induction 2.9% fall to 74.12±4.37 was noted. After one minute, the value increased 6.0%, reaching 81.024.49; at three minutes, the value increased 6.9%, reaching 81.743.85. When five and seven minutes had passed, the diastolic blood pressure had dropped to 80.24 minus 3.93 and 78.96 plus 4.04, respectively. At the end of 10 minutes it was 2% above the baseline with a mean of 78.02±3.85.
Maximum increase in the lignocaine group was 12.4%, while the largest increase in the control group was 17.7%. Diastolic blood pressure is significantly reduced by lignocaine compared to the control group for up to 7 minutes (z>1.96, p0.001).
The esmolol group saw a significantly lower maximal increase in diastolic blood pressure (z>1.96, p0.001).
Diastolic blood pressure was significantly lower in the esmolol group compared to the lignocaine group for the first five minutes (z>1.96, p0.001).

Table 7: Mean Arterial Blood Pressure (Mean arterial pressure)

	Mean arterial 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 Industion	92.25 ±6.92	-2.2	94.17 ±6.05	-1	91.17 ±5.16	-2.8	1.48	0.1389	2.67	0.0076	0.89	0.3735
1 Minute	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
3Minute	111.30 ±6.17	18	106.10 ±7.27	11.6	99.34 ±4.72	0.74	3.86	<0.001	5.51	<0.001	10.89	<0.0001
5Minute	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	5.5	<0.001
7Minute	97.75 ±6.33	3.6	94.24 ±5.45	-1	96.29 ±4.42	-1.6	2.97	0.003	2.07	0.0385	1.34	0.1802
10Minute	94.36 ±5.89	0.1	92.89 ±5.46	-2.3	95.29 ±4.17	-1	1.29	0.1971	2.47	0.01435	0.91	0.3628

Changes in mean arterial pressure at pre induction, post induction and at various time intervals after the commencement of laryngoscopy in control and study groups are shown.

The control group is comprised of
Mean arterial pressure before induction in this group 94.34±6.68. After the induction, it dropped to 92.25 6.92, a 2.2% reduction. During one minute following laryngoscopy and intubation, it showed a rise of 18.8%, coming in at 112.04 6.17. In three minutes, there was a slight drop of 18.0% compared to the data before the induction, and it continued to fall after that. It was 0.1% higher than baseline ten minutes after the laryngoscopy was completed.
In the lignocaine group, the pre-induction mean value was 95.136.55 and after induction it fell by 1% to 94.176.05. At one minute, it reached 107.877.00, representing a 13.5% increase; but, at three minutes, it had dropped slightly to 106.107.27. It went down even lower after 5, 7, and 10 minutes had passed. After 10 minutes following the laryngoscopy, the reading revealed a 2.3% drop below the baseline, coming in at 92.89 5.46.
The Esmolol group had a pre-induction mean arterial blood pressure of 93.80 with a standard deviation of 5.75. About three minutes after the laryngoscopy, the most significant increase of 5.9% was seen, bringing the total to 99.34 4.72. After then, it proceeded to go down more. After a period of 10 minutes, the mean value of arterial blood pressure was 1.5% higher than the baseline, coming in at 95.29 4.17.
In the 1, 3, 5, and 7-minute intervals, there is a significant difference between all of the groups (z>1.96, p0.01); additionally, there is a substantial attenuation of the pressor response by lignocaine in comparison to the control group (z>1.96, p0.001). The control group saw a maximum increase of 18.8%, whereas the lignocaine group saw a maximum increase of 13.5%. When contrasted with the control group, the attenuation seen in the esmolol group is extremely statistically significant (p 0.001). In the comparison of the two study groups, esmolol was found to be significantly more effective than lignocaine in dampening the pressor response (z > 1.96, p 0.05).

Discussion
When performed in sequence, induction of an aesthesia, laryngoscopy, and tracheal intubation are linked with considerable changes in hemodynamics and autonomic reflex activity, which may be a cause for

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worry in many patients who are considered to be at high risk [8].

Laryngoscopy and intubation are both related with an increase in the likelihood of cardiac arrhythmias as well as an increase in heart rate and blood pressure. Within five minutes of the beginning of the laryngoscopy procedure, these potentially harmful modifications vanish. 40 Despite the fact that these responses of blood pressure and heart rate are momentary and have a short lifespan, they have the potential to be harmful in patients who are at high risk, particularly those who have cardiovascular disease, increased intracranial pressure, or anomalies in the cerebral blood vessels [9].

There has been recorded evidence of a rise in mean arterial pressure of between 25 and 47.7 mm Hg on average. 41 After the placement of an endotracheal tube, it has been reported that there is an increase in the patient's mean arterial pressure of 26.5 mm Hg and 20 to 40 torr when compared with awake control levels and an increase of 35 to 60 torr when compared with values before the endotracheal tube was inserted [2, 10, 11]. In addition to this, a rise in mean heart rate of 29.9 beats per minute was seen [12].

The alterations in cardiovascular function that are linked with laryngoscopy and intubation are influenced by a number of different factors. The pressor response can be affected by factors such as age, medications, the nature and length of procedures, the level of anaesthesia administered, the depth of anaesthesia, hypoxia, hypercarbia, and so on.

As one gets older, the likelihood of experiencing irregular heart rates drops. Those under the age of 30 have more severe alterations [13]. Geriatric individuals frequently exhibit substantial changes in their hemodynamic responses [14, 15]. [Citation needed] Throughout the course of our research, we determined that the ideal age range to be between 20 and 50 years old.

Individuals who are taking medication to treat high blood pressure may experience a diminished pressor response. Those who were already taking medication for hypertension were not included in our research.

The sympathetic response to laryngoscopy and intubation can be affected by a variety of drug combinations that are employed at various stages of the anaesthetic process, including premedication, induction, relaxation, and maintenance.

When administered intravenously at a dose of 0.2 milligrammes per kilogramme, midazolam has the opposite effect of thiopentone, lowering blood pressure while speeding up the heart rate [16, 17]. On the other hand, its effect on sympathetic response to laryngoscopy and intubation was modest and was only slightly altered [16]. Pentazocine, an opioid agonist antagonist, has the potential to raise catecholamine levels while also elevating blood pressure and heart rate. A premedication with glycopyrrolate may cause a slight acceleration of the heart rate.

Thiopentone was chosen as the induction agent due to the fact that it is, and will likely always be, the most widely used induction agent. Thiopentone at a dose of 5 mg./kg when administered intravenously can momentarily cause a drop in blood pressure of 10-20 mm Hg and an increase in heart rate of 15-20 beats per minute in normovolemic patients [18]. There is an increase in the levels of both noradrenaline and adrenaline, which are catecholamines [18].

The effects of succinyl choline on the inotropic and chronotropic systems are both unfavourable. It exerts its influence on the muscarinic receptors present in the SA node. When intubation was conducted while the patient was under the influence of succinylcholine, a significant noradrenergic response was observed [19].

There is some evidence that nitrous oxide can raise the sympathetic nervous system's tone. Nitrous oxide has a direct impact that is negative inotropism, although this is counteracted by a rise in sympathetic tone. Although halothane has the potential to slow down the heart rate, at the concentrations that are typically used for maintenance, it does not significantly affect the heart rate [20].

The process of nasotracheal intubation can be broken down into three distinct phases:

a) Nasopharyngeal intubation.
b) Direct laryngoscopy to identify the vocal cords.
c) Passage of the tracheal tube into the trachea.

The pressor response is significantly increased after nasopharyngeal intubation. The insertion of a tracheal tube into the larynx and trachea amplifies the response that is already present. According to the findings of a study that was carried out by Singh S and Smith JE [21]. The response did not considerably enhance when direct laryngoscopy was performed. In our research, we solely considered the procedures of orotracheal intubation and direct laryngoscopy. The majority of the cardiovascular responses that were recorded after laryngoscopy and tracheal intubation under anaesthesia may have been caused by laryngoscopy alone according to research published in [22].

The length of time spent doing the laryngoscopy is the factor that has been demonstrated to have the most significant impact on the cardiovascular responses [13]. Within the first minute and a half of the experiment, researchers noticed a linear increase in both the heart rate and the mean arterial pressure. Adding more time won't make much of a difference. While a laryngoscopy often lasts less than thirty seconds, the findings of studies in which it takes longer than this amount of time have little practical use in clinical settings. When doing a laryngoscopy, the amount of force used has very little impact [23]. Throughout the course of our research, we set a time constraint of twenty seconds for the entire

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laryngoscopy and intubation process.

It was ensured that adequate precautions were taken to obtain the necessary level of anaesthesia while avoiding hypoxia and hypercarbia, both of which can have an effect on the fluctuations in hemodynamics. Excluding hypoxia and hypercarbia, additional contributing causes of hypertension and tachycardia could include a continued manifestation of anxiety concerning anaesthesia and surgery, premedication with glycopyrrolate, a reflex baroreceptor effect after thiopentone, and the possible effect of suxamethonium.

Laryngotracheal stimulation during laryngoscopy and intubation seems to be of greater significance than these other factors, however.

Anesthesiologists are quite concerned with the suppression of sympathetic reactions during laryngoscopy and intubation, and this is especially true in high-risk patients, as was indicated before. Numerous strategies have been suggested, some of which are as follows: reducing the amount of time spent performing laryngoscopy to less than 20 seconds; applying topical anaesthetics locally [24, 22]; administering intravenous beta-blockers [24, 25, 26, 27, 28]; administering calcium channel blockers [29], clonidine [30], sodium nitroprusside 42; using lignocaine. There is no one substance or method that is completely effective; each method has its own set of benefits and drawbacks, the most notable of which is that the prevention typically outlasts the stimulus.

Bachofen M outlined the criteria for choosing the most appropriate medication to inhibit sympathetic response. It is necessary for the medication to work regardless of whether or not the patient is cooperating with the treatment, to protect cerebral blood flow, and to keep patients from becoming aroused. It shouldn't take too much time and it shouldn't have any impact on how long the anaesthesia lasts or how it's administered [31].

It would appear that intravenous bolus lignocaine and esmolol satisfy the requirements outlined above.

Nonetheless, other researchers have found that intravenous lignocaine is useful in decreasing cardiovascular responses to laryngoscopy and intubation [32]. Moreover, lignocaine is able to prevent the rise in intracranial and intraocular pressures that results from laryngotracheal stimulation. Moreover, it stifles coughing associated with extubation. It is recommended that a dose of 1.5 to 2 mg/kg administered intravenously be used [33, 34]. It is best to give the medication three minutes before beginning the laryngoscopy and intubation procedures.

Esmolol is a beta-blocking drug that has various qualities that are considered to be desirable. It has a quick onset of action, is very cardioselective, and has an exceedingly short duration of action [35, 36, 37, 38].

Prior research [28, 39, 40] has demonstrated that due to esmolol's one-of-a-kind pharmacokinetic characteristics, the drug is ideally suited for managing the cardiovascular reactions that occur as a result of tracheal intubation when the technique of continuous infusion is utilised. On the other hand, the dosing schedule and the amount of time needed to prepare the infusion could add an additional layer of complexity. One option is to take bolus doses of esmolol, which have been the subject of numerous studies, all of which have reached the conclusion that this treatment method is effective [24, 11, 26, 27].

Investigations have been conducted using bolus dosages ranging from 100 to 200 mg administered intravenously. It was discovered that a bolus dose of 200 milligrammes was more effective and dependable in alternating both the reactions of the heart rate as well as the responses of the blood pressure [25]. Esmolol was administered intravenously in bolus doses of 2 mg/kg in our research.

When we compared the pre-induction heart rate of the control group to the value after the induction, we found that the heart rate had increased by a maximum of 41.1% ($z > 1.96$, p 0.001). The lignocaine group saw a rise of 30.5%, whereas the esmolol group saw an increase of 15.48%. Significant slowing of the heart rate was produced by the combination of lignocaine and esmolol ($z > 1.96$, p .001). At seven minutes in the control group and five minutes in the lignocaine and esmolol group, it reaches a level that is no longer clinically relevant. In the control group, this occurs after seven minutes. When compared with lignocaine, there is a clear and statistically highly significant difference between how esmolol attenuates the maximal rise in heart rate and how lignocaine does ($z>1.96$, p0.001).

In control group systolic blood pressure increased maximally after 1 minute from the onset of laryngoscopy and intubation. Over the course of 10 minutes, it went back to its pre-induction levels gradually. At the end of one minute, the greatest rise in systolic blood pressure associated with the lignocaine group was 14.5% higher than the values obtained before induction, but the rise associated with the esmolol group was only 6.6% higher than the values obtained before induction. Significant attenuation was seen with both medicines when compared to the control group ($p>1.96$, p0.05). Esmolol, one of the two medicines that were investigated, demonstrated superior attenuation to lignocaine up to five minutes after laryngoscopy ($z>1.96$, p0.001) in comparison to the other drug.

The greatest increase in diastolic blood pressure recorded in the control group was 17.7% ($z > 1.96$, p 0.001) when compared to values obtained before induction. Maximum increase in the lignocaine group over the control group over the course of 5 minutes was 12.4% ($z>1.96$, p0.001), whereas the esmolol group saw a similar rise of 12.4%. The diastolic blood pressure of the esmolol group was lower than that of the lignocaine group.

Similarly, one minute after laryngoscopy, the mean arterial pressure was 18.8% higher in the control

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group, 13.5% higher in the lignocaine group, and 8.1% lower in the esmolol group compared to pre-induction values. Esmolol significantly reduces mean arterial pressure compared to lignocaine and the control group ($z>1.96$, $p>0.05$).

Several investigations, including our own, have confirmed that esmolol is more effective than lignocaine at reducing cardiovascular reactions. Better results have been seen when lignocaine and esmolol are used together [27].

Conclusion

The following inferences can be drawn from the present clinical comparative analysis. Maximum increases in heart rate, systolic, diastolic, and mean arterial blood pressures during laryngoscopy and intubation were statistically and clinically very highly significant and can be deleterious in high-risk individuals who do not get medicines to reduce the sympathetic responses. Sympathetic responses to laryngoscopy and tracheal intubation are greatly reduced by lidocaine. The sympathetic responses are likewise greatly diminished by esmolol. When it comes to reducing sympathetic responses during laryngoscopy and intubation, esmolol is more effective than lignocaine. To reduce the sympathetic responses brought on by laryngoscopy and intubation, a bolus dosage of 2 mg/kg i.v. esmolol given 3 minutes beforehand may be advised.

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