



Anaesthesiology

ATTENUATION OF HAEMODYNAMIC RESPONSE FOR LARYNGOSCOPY AND ENDO TRACHEAL INTUBATION WITH 2% LIGNOCAINE VISCOUS VERSUS 10% LIGNOCAINE SPRAY A COMPARITIVE STUDY

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ABSTRACT To compare the efficacy of 10 ml of lignocaine 2% viscous and 5 puffs of oral lignocaine 10% spray in attenuation of haemodynamic response in terms of blood pressure and heart rate also oxygen saturation after laryngo scopy and endotracheal intubation in 60 patients divided into two groups aged between 20 to 60 years, ASA grade 1 and 2 undergoing various surgical procedures under general anaesthesia. experimental study reveals that both 2% lignocaine viscous and 10% lignocaine spray were effective in attenuating the haemodynamic response but oral lignocaine viscous 2% was more effective in blunting the haemodynamic response to laryngo scopy and endo tracheal intubation compared to 10% lignocaine spray.

KEYWORDS : Lignocaine Viscous 2%, Lignocaine Spray 10%, Attenuation Of Haemodynamic Response

INTRODUCTION

A crucial part of general anaesthesia is endotracheal intubation. It assists in maintaining the patency of the upper airway, ensuring proper ventilation, lowering the risk of aspiration, and giving patients access to inhalational anaesthetics through breathing circuits. The most important procedures during the induction of general anaesthesia are laryngoscopy and tracheal intubation, which activate somatic and visceral nociceptive afferent fibres that trigger reflex sympathoadrenal responses and are associated with increased neuronal activity of cervical sympathetic efferent fibres.

- The percentage of difficult intubations ranges from 1% to 6% of all intubations, and the incidence of unsuccessful intubations ranges from 0.1% to 0.3% of all intubations, according to an incomplete statistical analysis. The majority of the patients in the emergency room and intensive care unit are hospitalised for serious illnesses; as a result, medical personnel must quickly complete endotracheal intubation and, more importantly, provide airway management in circumstances where adequate emergency planning and facilities are not available.
- Patients undergoing general anaesthesia lose consciousness and the ability to control breathing and to protect their airway. Tracheal intubation is considered a vital procedure that secures the airway and provides the possibility of continued oxygenation. Direct laryngoscopy and endotracheal intubation are mostly associated with hemodynamic changes caused by reflex sympathetic discharge, caused by epi pharyngeal and laryngopharyngeal stimulation. Tachycardia, hypertension, and other symptoms like arrhythmias are caused by sympathoadrenal activity which are potentially dangerous. These changes are most pronounced at one minute after laryngoscopy and intubation and lasts for 5-10 minutes. This increase of Blood pressure and heart rate are typically transient, variable, and unpredictable.
- Whether or not they have been treated previously, patients with hypertension mostly to experience a significant rise in blood pressure. Patients with hypertension, myocardial disease, or cerebrovascular disease may be at risk from transient hypertension and tachycardia. In above patients the laryngoscopy response may predispose to pulmonary oedema, cerebrovascular accidents, intracerebral bleed, myocardial insufficiency, and acute left ventricular failure with end organ decompensation.
- A variety of agents have been used to decrease this response. The techniques like topical anaesthesia of oropharynx-viscous Lignocaine, lignocaine spray, laryngotracheal installation of Lignocaine just before intubation, Lignocaine intravenous adrenergic blocking drugs, either alpha or beta blockers, alpha2 agonist like Dexmedetomidine, Clonidine, vasodilators like Hydralazine, Sodium nitroprusside, Nitroglycerine, deep inhalational anaesthesia, intravenous Opioids, Magnesium sulfate etc. majority of these agents are not totally effective for this purpose.
- Lignocaine is an aminoethyl amide. it is a prototype of amide group

of local anaesthetics, introduced in 1948. It is most widely used local anaesthetic. Lignocaine has been used both topically and intravenously for attenuation of pressor response during laryngoscopy and endotracheal intubation. Lignocaine blocks sodium channels in the myocardium, thus reducing the rate of rise of action potential and altering conduction velocity throughout the His-Purkinje system and atrium and ventricular musculature.

- Usually, it is administered via the intravenous route at 1.5 mg kg⁻¹ bodyweight for 3 min before intubation to suppress the hemodynamic response. However, such suppression is not complete and a spike in SBP at 1-min and 3-min intervals post-intubation has been reported
- In our study we are comparing the efficacy of 2% lignocaine viscous with 10% lignocaine spray in attenuation of haemodynamic response for laryngo scopy and endo tracheal intubation
- This is a prospective randomized study and the study conducted in 60 ASA grade I and II adult patients. Divided into two groups.
- Group V receives 10 ml of 2% oral lignocaine viscous gargle for 5 minutes prior to induction.
- Group S receives five puffs of oral lignocaine spray prior to induction.
- 1. All the values are analysed and expressed as Mean +/- SD
- 2. Student test and ANOVA test for parametric data.
- 3. Chi-square test for non parametric data.
- 4. P<0.05 will be considered as statistically significant

MATERIALS AND METHODS

The study was taken up after the approval of the Ethical committee of the medical college. During the study, purpose of the study was explained to all study subjects in his/her own language and informed written consent was taken.

This is a prospective randomized study and the study will be conducted in 60 ASA grade I and II adult patients. Divided into two groups. Group V receives 10 ml of 2% oral lignocaine viscous gargle for 5 minutes prior to induction. Group S receives five puffs of oral lignocaine spray prior to induction

Inclusion Criteria

1. Belonging to ASA grade I and II.
2. Patients belonging to age 20 to 50 yrs
3. Patients giving informed written consent.
4. Patients scheduled to undergo elective surgeries under general anaesthesia

Exclusion Criteria

1. Patient refusal for procedure.
2. Patients belonging to ASA III & IV.
3. Active URTI and LRTI.
4. Patients with Asthma, Obstructive sleep apnea, Obesity,
5. Bleeding disorders,

6. Allergic to drugs.

Investigations: Blood: complete blood picture, Bleeding time, Clotting time, Blood grouping and Typing, liver unction tests renal function tests, ECG and Chest X- ray.

Procedure : All the patients on the day of surgery were premedicated with glycopyrrolate 0.2 mg/kg, tramadol hydrochloride 100 mg, ondansetron 4mg, midazolam 2mg intravenously 30 minutes before the expected time of induction.

On arrival to operation theatre the pulse rate, systolic and diastolic blood pressures were recorded in all patients

In group V, patients were instructed to gargle 10 ml of lignocaine viscous 5 min prior to induction

In group S, patients were administered with 5 puffs of 10 % lignocaine spray prior to induction

After preoxygenation for 3 minutes anesthesia was induced with Thiopentone sodium (2.5 solution) 5mg/kg in order to obtund the eyelash reflex ,followed by vecuronium bromide 0.1mg/kg was used to facilitate intubation .after the onset of apnoea and muscle relaxation intubation was done with standard Macintosh laryngoscope and a lubricated appropriate sized cuffed endotracheal tube was passed .the laryngoscopy was completed in 15-30seconds any patient who strained or required 2nd attempt were excluded from the study .the cuff inflated and tube was connected to work station through closed circuit.

No analgesic drugs or volatile agents were given and surgical stimulus was not allowed till 10 min after cuff inflation of endotracheal tube

Anesthesia was maintained with 33%oxygen 66% nitrous oxide,narcotic intermittent doses of vecuronium bromide and sevoflurane as inhalational agent with IPPV,residual effect of muscle relaxant reversed with 0.05mg/kg neostigmine and glycopyrrolate.

After laryngoscopy and endotracheal intubation

- Heart rate
- Systolic blood pressure
- Diastolic blood pressure
- Mean arterial blood pressure
- Oxygen saturation

Were recorded and computed for two groups at following instance
1.Pre induction (base line) 2. Immediately after laryngoscopy 3.1 minute after laryngoscopy 4.3minutes after laryngoscopy 5.5minutes after laryngoscopy and traced till 10 min.

Statistical Analysis

Appropriate statistical analysis of data was done using one of the following tests.

1. All the values were analysed and expressed as Mean +/- SD
2. Student test and ANOVA test for parametric data.
3. Chi-square test for non parametric data.
4. P<0.05 will be considered as statistically significant

RESULTS

Vital Parameter Data:

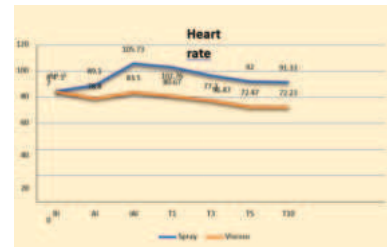
Heart Rate:

The table no.4 and graph no.4 show the trend of HEART RATE from baseline to over a period of 10 min after intubation. The basal (BL) mean heart rate of group S was 84.50±4.09 and group V was 84.17±7.64, and the difference in the mean heart rate at baseline among the groups was not statistically significant (p>0.05). After induction the mean heart rate starts increasing in group S and in group V mean heart rate start decreasing after induction and slightly raised after immediately after intubation and gradually decreasing till 10 min after intubation. In group S immediately after intubation highest mean heart rate was observed, and start decreasing at 1 minute, 3 minute, 5 minute and 10 minute after intubation but not reached baseline level, The mean heart rate between two groups from after induction to 10 minutes after intubation was significant. (p<0.05).

Table 4: Comparison Of Heart Rate (bpm) In The Study Groups At Different Time Intervals:

Time	Group	N	Heart rate		
			Mean	SD	P value

Baseline (BI)	S	30	84.50	4.09	0.834
	V	30	84.17	7.64	
After induction (AI)	S	30	89.10	5.43	<0.001
	V	30	78.80	7.38	
Immediately after intubation (IAI)	S	30	105.73	7.14	<0.001
	V	30	83.50	7.97	
1 min (T1)	S	30	102.76	7.24	<0.001
	V	30	80.67	6.32	
3 min(T3)	S	30	96.47	7.26	<0.001
	V	30	77.30	5.57	
5 min (T5)	S	30	92.00	5.79	<0.001
	V	30	72.47	4.91	
10 min (T10)	S	30	91.33	5.99	<0.001
	V	30	72.23	5.34	



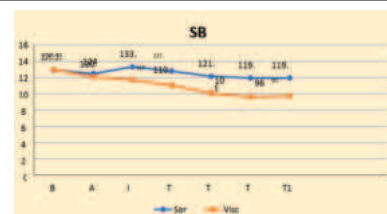
Graph 4: Comparison Of Heart Rate Variation Among The Study Groups At Different Time Intervals:

Systolic Blood Pressure

The table no.5 and graph no.5 shows the trend of SYSTOLIC BLOOD PRESSURE from baseline to over a period of 10 min after intubation. At baseline, the mean systolic blood pressure (SBP) of group S was 129.13± 7.51, group V was 129.60±7.38 and the difference in mean SBP at baseline among the groups is not statistically significant (p>0.05). After induction the mean SBP starts decreasing in both groups. However, in group S mean SBP start increasing immediately after intubation and gradually start decreasing from 1 minute after intubation to 10 minutes after intubation. The mean SBP between two groups from 1 minute after induction to 10 minutes after intubation was significant. (p<0.05).

Table 5: Comparison Of SBP (mm Hg) Among The Study Groups At Different Time Intervals

Time	Group	N	BP			
			Mean	SD		P value
Baseline	S	30	129.13	7.51	0.232	
	V	30	129.60	7.38		
After induction	S	30	124.80	9.49	0.064	
	V	30	120.93	5.93		
Immediately after intubation	S	30	133.37	5.25	<0.001	
	V	30	117.20	12.25		
1 min	S	30	127.97	6.35	<0.001	
	V	30	110.53	9.20		
3 min	S	30	121.57	6.06	<0.001	
	V	30	100.90	10.64		
5 min	S	30	119.47	5.33	<0.001	
	V	30	96.30	10.58		
10 min	S	30	119.47	6.72	<0.001	
	V	30	97.53	11.21		



Graph 5: Comparison Of SBP Variation Among The Study Groups At Different Time Intervals

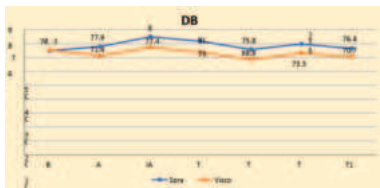
Diastolic Blood Pressure:

The table no.6 and graph no.6 shows the trend of DIASTOLIC BLOOD PRESSURE from baseline to over a period of 10 min after intubation. At baseline, the mean Diastolic blood pressure (DBP) of

group S was 74.93 ± 3.99 , group V was 75.50 ± 3.69 , and the difference in mean DBP at baseline among the groups is not statistically significant ($p > 0.05$). After induction the mean DBP starts increasing in group S till immediately after intubation and gradually start decreasing from 1 minute after intubation to 10 minutes after intubation but never reached baseline level. However, in group V mean DBP start decreasing immediately after intubation and slightly increased immediately after intubation and gradually decreasing till 10 min after intubation. The mean DBP between two groups from after induction to 10 minutes after intubation was significant. ($p < 0.05$).

Table 6: Comparison Of DBP (mm Hg) Among The Study Groups At Different Time Intervals

Time	Group	N	Mean	SD	P value
Baseline	S	30	74.93	3.99	0.571
	V	30	75.50	3.69	
After induction	S	30	77.97	5.96	<0.001
	V	30	71.43	3.35	
Immediately after intubation	S	30	85.00	4.59	<0.001
	V	30	77.47	9.18	
1 min	S	30	81.80	4.81	<0.001
	V	30	73.90	5.09	
3 min	S	30	75.87	4.47	<0.001
	V	30	68.83	4.48	
5 min	S	30	79.90	3.72	<0.001
	V	30	73.33	4.91	
10 min	S	30	76.47	5.18	<0.001
	V	30	70.77	6.64	



Graph 6: Comparison Of DBP Variation Among The Study Group At Different Time Intervals

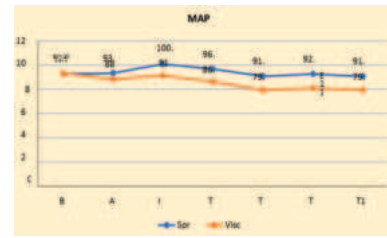
Mean Arterial Pressure

The table no. 7 and graph no.7 shows the trend of MEAN ARTERIAL PRESSURE from baseline to over a period of 10 min after intubation. At baseline, the average mean arterial pressures (MAP) in group S was 92.90 ± 4.35 , group S was 93.23 ± 4.03 and the difference in mean MAP at baseline among the groups is not statistically significant ($p > 0.05$). After induction the mean MAP starts increasing in group S till immediately after intubation and gradually start decreasing from 1 minute after intubation to 10 minutes after intubation but reached baseline level at 3 minutes. However, in group V mean MAP start decreasing immediately after intubation and slightly increased immediately after intubation and gradually decreasing till 10 min after intubation. The mean MAP between two groups from after induction to 10 minutes after intubation was significant. ($p < 0.05$).

Table 7: Comparison Of MAP (mm Hg) In The Study Groups At Different Time Intervals:

Time	M		AP		P value
	Group	N	Mean	SD	
Baseline	S	30	92.90	4.35	0.759
	V	30	93.23	4.03	
After induction	S	30	93.67	6.18	0.001
	V	30	88.50	4.92	
Immediately after intubation	S	30	100.99	3.40	<0.001
	V	30	91.50	9.09	
1 min	S	30	96.91	5.48	<0.001
	V	30	86.43	5.81	
3 min	S	30	91.13	4.36	<0.001
	V	30	79.63	4.96	
5 min	S	30	92.93	3.32	<0.001
	V	30	81.10	6.31	

10 min	S	30	91.07	5.98	<0.001
	V	30	79.87	7.78	



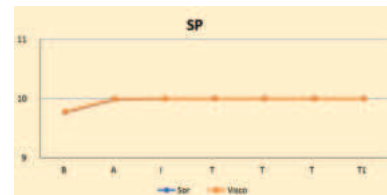
Graph 7: Comparison Of MAP Variation Among The Study Groups At Different Time Intervals

Mean Spo2

The table no. 8 and graph no.8 shows the trend of MEAN SPO2 from baseline to over a period of 10 min after intubation. At baseline, the average MEAN SPO2 in group S was 97.70 ± 0.75 , group S was 97.73 ± 0.83 and the difference in mean MAP at baseline among the groups is not statistically significant ($p > 0.05$). After induction the mean SPO2 starts increasing in both groups and reached to 100% in both groups.

Table 8: Comparison Of Spo2 (%) In The Study Groups At Different Time Intervals:

TIME	SPO2				
	Group	N	Mean	SD	P value
Baseline	S	30	97.70	0.75	0.871
	V	30	97.73	0.83	
After induction	S	30	99.87	0.35	0.398
	V	30	99.93	0.25	
Immediately after intubation	S	30	100	0	-
	V	30	100	0	
1 min	S	30	100	0	-
	V	30	100	0	
3 min	S	30	100	0	-
	V	30	100	0	
5 min	S	30	100	0	-
	V	30	100	0	
10 min	S	30	100	0	-
	V	30	100	0	



Graph 8: Comparison Of Spo2 (%) Variation Among The Study Groups At Different Time Intervals

DISCUSSION

Heart Rate

The trend of HEART RATE from baseline to over a period of 10 min after intubation. The basal (BL) mean heart rate of group S was 84.50 ± 4.09 and group V was 84.17 ± 7.64 , and the difference in the mean heart rate at baseline among the groups was not statistically significant ($p > 0.05$). After induction the mean heart rate starts increasing in group S and in group V mean heart rate start decreasing after induction and slightly raised after immediately after intubation and gradually decreasing till 10 min after intubation. In group S immediately after intubation highest mean heart rate was observed, and start decreasing at 1 minute, 3 minute, 5 minute and 10 minute after intubation but not reached baseline level, The mean heart rate between two groups from after induction to 10 minutes after intubation was significant. ($p < 0.05$).

Systolic Blood Pressure

At baseline, the mean systolic blood pressure (SBP) of group S was 129.13 ± 7.51 , group V was 129.60 ± 7.38 and the difference in mean SBP at baseline among the groups is not statistically significant ($p > 0.05$). After induction the mean SBP starts decreasing in both groups. However, in group S mean SBP start increasing immediately

after intubation and gradually start decreasing from 1 minute after intubation to 10 minutes after intubation. The mean SBP between two groups from 1 minute after induction to 10 minutes after intubation was significant. ($p<0.05$).

Diastolic Blood Pressure

At baseline, the mean Diastolic blood pressure (DBP) of group S was 74.93 ± 3.99 , group V was 75.50 ± 3.69 , and the difference in mean DBP at baseline among the groups is not statistically significant ($p>0.05$). After induction the mean DBP starts increasing in group S till immediately after intubation and gradually start decreasing from 1 minute after intubation to 10 minutes after intubation but never reached baseline level. However, in group V mean DBP start decreasing immediately after intubation and slightly increased immediately after intubation and gradually decreasing till 10 min after intubation. The mean DBP between two groups from after induction to 10 minutes after intubation was significant. ($p<0.05$).

Mean Arterial Pressure

At baseline, the average mean arterial pressures (MAP) in group S was 92.90 ± 4.35 , group V was 93.23 ± 4.03 and the difference in mean MAP at baseline among the groups is not statistically significant ($p>0.05$). After induction the mean MAP starts increasing in group S till immediately after intubation and gradually start decreasing from 1 minute after intubation to 10 minutes after intubation but reached baseline level at 3 minutes. However, in group V mean MAP start decreasing immediately after intubation and slightly increased immediately after intubation and gradually decreasing till 10 min after intubation. The mean MAP between two groups from after induction to 10 minutes after intubation was significant. ($p<0.05$).

Mean SPO2

At baseline, the average MEAN SPO2 in group S was 97.70 ± 0.75 , group V was 97.73 ± 0.83 and the difference in mean MAP at baseline among the groups is not statistically significant ($p>0.05$). After induction the mean SPO2 starts increasing in both groups and reached to 100% in both groups.

CONCLUSION

Oral lignocaine viscous 2% and lignocaine spray both were effective in blunting haemodynamic response to laryngoscopy and endotracheal intubation in patients undergoing surgical procedures under general anesthesia

Oral lignocaine 2% was MORE EFFECTIVE in attenuating the haemodynamic response to laryngoscopy and endotracheal intubation. Oral lignocaine 10% spray was also effective in attenuating haemodynamic response to laryngoscopy and intubation, but not effective as oral viscous lignocaine.

REFERENCES

1. Edward Morgan MSM G. Clinical Anesthesiology United States of America: McGraw-Hill; 2013. P320-2
2. PRYS-ROBERTS LTG C., MELOCHE R. and FOEX P. haemodynamic consequences of induction and endotracheal intubation. British journal of anaesthesia. 1971; 43:531-47. <https://doi.org/10.1093/bja/43.6.531> PMID: 5089931
3. Crosby ET, Cooper RM, Douglas MJ, Doyle DJ, Hung OR, Labrecque P, Muir H, Murphy MF, Preston RP, Rose DK and Roy L. The unanticipated difficult airway with recommendations for management. Can J Anaesth 1998; 45: 757-776.
4. Shiga T, Wajima Z, Inoue T and Sakamoto A. Predicting difficult intubation in apparently normal patients: a meta-analysis of bedside screening test performance. Anesthesiology 2005; 103: 429-437.
5. Carlson JN and Wang HE. Updates in emergency airway management. Curr Opin Crit Care 2018; 24: 525-530.
6. Reid LC, Brace DE. Irritation of the respiratory tract and its reflex effect upon the heart. Surg Gynecol Obstet. 1940 Feb;70:157-62.
7. King BD, Harris LC, Greifenstein FE, Elder JD, Dripps RD. Reflex circulatory responses to direct laryngoscopy and tracheal intubation performed during general anesthesia. Anesthesiology: The Journal of the American Society of Anesthesiologists. 1951 Sep 1;12(5):556-66.
8. Takashima Noda K: Cardiovascular responses to Endotracheal intubation. Anaesthesia Analgesia. 1964;43:201. 50
9. Forbes AM, Dally FG. Acute hypertension during induction of anaesthesia and endotracheal intubation in normotensive man. BJA: British Journal of Anaesthesia. 1970 Jul 1;42(7):618-24.
10. Reema Goel, Raka Rani, O.P. Singh, Deepak Malviya, S.K. Arya et al. Attenuation of cardiovascular responses to laryngoscopy and intubation by various drugs in normotensive patients, Hospital Today. 2000; 9.
11. Stoelting RK. Blood pressure and heart rate changes during short-duration laryngoscopy for tracheal intubation: influence of viscous or intravenous lidocaine. Anesthesia & Analgesia. 1978 Mar 1;57(2):197-9.
12. Fujii Y, Saitoh Y, Takahashi S, Toyooka H. RETRACTED ARTICLE: Diltiazemlidocaine combination for the attenuation of cardiovascular responses to tracheal intubation in hypertensive patients. Canadian Journal of Anesthesia/Journal canadien d'anesthésie. 1998 Oct 1;45(10):933-7.
13. Elisabeth J Fox, Garry S Sklar, Constance H Hill, Raymond Villanueva, Benton D King. Complication related to the pressor response to endotracheal intubation. Anaesthesiology. 1977; 47: 524-25.

14. Dalton B, Guiney T. Myocardial ischemia from tachycardia and hypertension in coronary heart disease—Patients undergoing anaesthesia. Boston: Ann Mtg American Society of Anaesthesiologists. 1972;20:1-2.
15. Denlinger JK, Ellison N, Ominsky AJ. Effects of intratracheal lidocaine on circulatory responses to tracheal intubation. Anesthesiology: The Journal of the American Society of Anesthesiologists. 1974 Oct 1;41(4):409-12.
16. Tam S, Chung F, Campbell M. Intravenous lidocaine: optimal time of injection before tracheal intubation. Anesth Analg. 1987 Oct 1;66(10):1036-8.