Original Article

A study of lignocaine in attenuation of cardiovascular response to laryngoscopy and endotracheal intubation

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Abstract

Introduction: Increase in heart rate and blood pressure is well documented sequelae of direct laryngoscopy and endotracheal intubation in normotensive individuals. Direct laryngoscopy and endotracheal intubation frequently induces a cardiovascular stress response characterized by hypertension, tachycardia due to reflex sympathetic stimulation which in turn leads to increased plasma catecholamine concentration **Aims and Objectives:** To study Lignocaine in attenuation of cardiovascular response to Laryngoscopy and Endotracheal intubation **Materials and Methods:** The study was approved by the Ethics Committee of Kidwai Memorial Institute of Oncology, Bangalore and all patients gave valid written informed consent. One Sixty inpatients, 20 – 60 years of age, of either sex undergoing elective surgical procedures at Kidwai Memorial Institute of Oncology, Bangalore requiring general anaesthesia with endotracheal intubation were selected randomly. The study was conducted in the Department Of Anaesthesia and Pain Relief, Kidwai Memorial Institute of Oncology, Bangalore for a period of one year 01-01-2013 to 01-01-2014. Descriptive and inferential statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean ± SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5 % level of significance. **Result:** The increase in mean HR immediately after intubation, at 1and 5 minutes after intubation compared to basal HR was statistically significant($p<0.001$).The decrease in mean SBP observed at 1, 3, 5 and 10 minutes after intubation when compared with basal SBP was statistically significant ($p<0.05$). The decrease in mean DBP observed at 3, 5 and 10 minutes after intubation when compared with basal DBP was statistically significant ($p<0.05$). The decrease in MAP observed at 3, 5 and 10 minutes after intubation when compared with basal MAP was statistically highly significant ($p<0.01$). **Conclusion:** Lignocaine significantly attenuates the cardiovascular responses to laryngoscopy and intubation.

Keywords: Lignocaine, cardiovascular response to Laryngoscopy and Endotracheal intubation.

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INTRODUCTION

Increase in heart rate and blood pressure are well documented sequelae of direct laryngoscopy and endotracheal intubation in normotensive individuals.[1,2,3,4] Direct laryngoscopy and endotracheal intubation frequently induces a cardiovascular stress response characterized by hypertension, tachycardia due to reflex sympathetic stimulation which in turn leads to increased plasma catecholamine concentration. This transient, self-limiting hypertension and tachycardia are innocuous in healthy individuals but either or both may be hazardous to patients with hypertension, coronary insufficiency or with cerebro-vascular disease.[5] Pressor response to intubation is exaggerated in hypertensive patients even though rendered normotensive preoperatively by antihypertensive medications.[6] Pressor response may result in intra-operative myocardial infarction,[7] acute

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L.V.F,[7] dysarrhythmias[8] and intracerebral bleed[7] in individuals with end organ decompensation. Intravenous anesthetic induction agents do not adequately or predictably suppress the circulatory responses evolved by endotracheal intubation.[5] So prior to initiating laryngoscopy additional pharmacological measures like use of volatile anaesthetics,[2] topical and intravenous lidocaine,[9] opioids[10,11,12], vasodilators – SNP[13], NTG[14], calcium channel blockers[15, 16, 17] and β-blockers[18, 19] have been tried by various authors. These measures attenuate but do not completely abolish the pressor response. Each technique has its own disadvantage which suggests lack of an ideal measure. Minimum alveolar concentration of Halothane, Isoflurane and Enflurane required to attenuate the pressor response is very difficult to attain during the short period available for anaesthetic induction.[5] If at all attained, volatile anaesthetic may cause unacceptable mycocardial depression[2, 21] hazardous to hypertensive patient with ischaemic heart disease. Intravenous lidocaine in varying doses has been shown to attenuate the stress response to laryngoscopy and intubation.[20, 22] Sodium Nitroprusside requires special administration technique and invasive arterial BP monitoring and undue hypotension can still occur. Recommendations for attenuating reflex tachycardia and hypertension are therefore manifold. The technique besides minimizing the cardiovascular responses to laryngoscopy and intubation for a patient at risk must also satisfy the following requirements, It must be applicable regardless of patient's collaboration, It should prevent impairment of cerebral blood flow and avoid arousal of the patient, It should neither be time consuming nor effect the duration or modality of ensuing anaesthesia. Tomoriand Widdicombel[23] demonstrated that mechanical stimulation of upper respiratory tract caused cardiovascular response associated with increased activity in cervical sympathetic chain. Studies of some authors concluded that β blockers[18,19]attenuated the pressor response to laryngoscopy and intubation. Marked elevation in plasma catecholamine levels following laryngoscopy and intubation was observed consistently by various authors.[24, 25]This confirmed that pressor response was mediated by sympathetic nervous system. But the pharmacological action of the then available beta blockers – propranolol[18, 19], metoprolol[26, 27]outlasted the duration of pressor response. Also non cardio-selective propranolol was contraindicated in COPD patients. Lidocaine was synthesized in 1943 in Sweden by Lofgren and it was introduced into clinical practice by Gordh in the year 1948. [8]It suppresses the automaticity in ectopic foci by antagonizing phase IV depolarization in purkinje fibers and ventricular muscles by blocking sodium channels. It does not depress SA node automaticity. The rate of phase-0 depolarization is not decreased except in presence of hyperkalemia. Lidocaine markedly decreases the action potential duration and effective refractory period in purkinje fibers and ventricular muscles. Conduction velocity is not decreased. It has practically no effect on action potential duration and effective refractory period of atrial fibers. Atrial re-entry is not affected. It can suppress the re-entrant ventricular arrhythmias either by abolishing one way block or by producing two way block. At therapeutic plasma concentration of 3-5µg/ml, it causes little depression of cardiac contractility. There are no significant autonomic actions. All cardiac effects are direct actions. Lidocaine is widely is used in the management of ventricular dysrhythmias in a dose of 1 to 2 mg/kg bolus intravenously and 2.4 mg/min as infusion. It acts by its membrane stabilizing effect and depression of automaticity at atrio-ventricular node. It has been used effectively in the management of ventricular arrhythmias following myocardial infarction and cardiac surgery.

MATERIALS AND METHODS

The study was approved by the Ethics Committee of Kidwai Memorial Institute of Oncology, Bangalore and all patients gave valid written informed consent. One Sixty inpatients, 20 – 60 years of age, of either sex undergoing elective surgical procedures at Kidwai Memorial Institute of Oncology, Bangalore requiring general anaesthesia with endotracheal intubation were selected randomly. The study was conducted in the Department Of Anaesthesia and Pain Relief, Kidwai Memorial Institute of Oncology, Bangalore for a period of one year 01-01-2013 to 01-01-2014. Patients aged between 20 - 60 years of age posted for elective non cardiac surgical procedures under general anaesthesia, ASA grade I and II patients, Patients with Mallampatti grade I and II were Included into study. Whereas Patients with any known medical comorbidities like hypertension, ischemic heart diseases, diabetes, asthma etc, Basal Heart Rate < 60 and> 100, Basal Systolic Blood Pressure < 100mmHg, Predicted difficult intubation If patient is allergic to any of the study drugs, More than 1 attempt at intubation and attempt lasting more than 40 seconds, Cormack – Lehane Laryngoscopic Grading > 2, On any medications with cardiovascular effects, If patient is allergic to any of the study drugs, More than 1 attempt at intubation and attempt lasting more than 40 seconds Cormack – Lehane Laryngoscopic Grading > 2. On any medications with cardiovascular effects were excluded from the study. The patients were connected to multichannel monitor which records Heart Rate, non-invasive measurements of SBP, DBP, MAP, EtCO2 and continuous ECG monitoring and oxygen saturation. The baseline systolic, diastolic blood pressure, mean arterial

pressure and heart rate were recorded. The cardiac rate and rhythm were also monitored from a continuous visual display of electrocardiogram from lead II. Descriptive and inferential statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean ± SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5 % level of significance.

RESULTS

Table 1: Heart rate (bpm) changes – intragroup comparison - Lignocaine

HR	Mean	Std. Dev	SE of Mean	Mean Difference	t	P-Value
Baseline	87.35	10.11	1.60	---	---	---
1 Min after drug administration	93.45	11.31	1.79	6.100	-5.151	<0.001*
Immediately after intubation	98.38	12.29	1.94	11.025	-6.895	<0.001*
1 Min post intubation	96.60	11.41	1.80	9.250	-6.581	<0.001*
3 Min post intubation	91.00	16.85	2.66	3.650	-1.406	0.168
5 Min post intubation	91.13	11.70	1.85	3.775	-2.302	0.027*
10 Min post intubation	90.45	11.81	1.87	3.100	-1.997	0.053

p <0.05 is significant; p<0.001 highly significant

In the Group L, the pre-induction mean HR was 87.3±10 bpm. The mean heart rate after 1min of study drug administration was 93.45±11.3 bpm, immediately after intubation was 98.3±12.2 bpm, one minute after intubation it was 96.6±11.4 bpm representing a rise of 9.3 bpm from the pre-induction heart rate. By 3, 5 and 10 minutes mean HR were 91±16.8 bpm, 91.1±11.7 bpm and 90.4±11.8 bpm respectively. The mean heart rate did not come to the pre-induction levels even by 10th minute. The increase in mean HR immediately after intubation, at 1and 5 minutes after intubation compared to basal HR was statistically significant($p<0.001$).

Table 2: Systolic blood pressure changes – intragroup comparison –Lignocaine

SBP	Mean	Std. Dev	SE of Mean	Mean Difference	t	P-Value
Baseline	128.15	9.99	1.58	---	---	---
1 Min after drug administration	112.40	10.77	1.70	-15.750	9.049	<0.001*
Immediately after intubation	129.38	13.40	2.12	1.225	-0.484	0.631
1 Min post intubation	122.68	13.05	2.06	-5.475	2.259	0.030*
3 Min post intubation	109.25	10.09	1.60	-18.900	9.593	<0.001*
5 Min post intubation	103.75	10.84	1.71	-24.400	12.692	<0.001*
10 Min post intubation	112.50	13.80	2.18	-15.650	6.956	<0.001*

p<0.01–highly significant, p<0.05–significant, p>0.05–Not Significant.

In the group L (Lidocaine) the baseline value of mean SBP was 128.15 ± 9.99 mmHg and at immediately after intubation was 129.38 ± 13.40 mmHg. The mean systolic blood pressure one minute after intubation was 122.68 ± 13.05 mmHg, representing a fall of 5.47 mmHg. By 3, 5 and 10 minutes mean SBP values were 109.25 ± 10.09 mmHg, 103.75 ± 10.84 mmHg, 112.50±13.80 mmHg and with a fall of 18.9, 24.4 mmHg and 15.65 mmHg respectively compared to baseline values. The decrease in mean SBP observed at 1, 3, 5 and 10 minutes after intubation when compared with basal SBP was statistically significant ($p<0.05$).

Table 3: Diastolic blood pressure – intragroup comparison –Lignocaine

DBP	Mean	Std. Dev	SE of Mean	Mean Difference	t	P-Value
Baseline	79.63	9.38	1.48	---	---	---
1 Min after drug administration	73.25	9.26	1.46	-6.375	3.981	<0.001*
Immediately after intubation	86.70	13.24	2.09	7.075	-2.961	0.005*
1 Min post intubation	79.45	11.57	1.83	-0.175	0.077	0.939
3 Min post intubation	67.05	10.87	1.72	-12.575	6.922	<0.001*
5 Min post intubation	64.13	13.58	2.15	-15.500	6.144	<0.001*
10 Min post intubation	71.63	11.96	1.89	-8.000	3.577	0.001*

p<0.01–highly significant, p<0.05–significant, p>0.05–Not Significant

In the group L (Lidocaine) the baseline value of mean DBP was 79.63 ± 9.38 mmHg and at immediately after intubation was 86.70 ± 13.24 mmHg, a significant rise. The mean diastolic blood pressure one minute after intubation was 79.45 ± 11.57 mmHg, representing a fall of 0.18mmHg which is not statistically significant. By 3, 5 and 10 minutes mean DBP

values were 67.05 ± 10.87 mmHg, 64.13 ± 13.58 mmHg, 71.63±11.96 mmHg and with a fall of 12.58, 15.5 and 8 mmHg respectively compared to baseline values. The decrease in mean DBP observed at 3, 5 and 10 minutes after intubation when compared with basal DBP was statistically significant ($p<0.05$).

Table 4: Mean Arterial blood pressure changes – intragroup comparison - Lignocaine

MAP	Mean	Std. Dev	SE of Mean	Mean Difference	t	P-Value
Baseline	95.75	8.23	1.30	---	---	---
1 Min after drug administration	86.28	8.79	1.39	-9.475	6.767	<0.001*
Immediately after intubation	101.33	12.01	1.90	5.575	-2.456	0.019*
1 Min post intubation	93.48	11.00	1.74	-2.275	1.003	0.322
3 Min post intubation	81.10	9.92	1.57	-14.650	8.784	<0.001*
5 Min post intubation	78.10	9.63	1.52	-17.650	10.120	<0.001*
10 Min post intubation	85.25	12.10	1.91	-10.500	5.006	<0.001*

p<0.01–highly significant, p<0.05–significant, p>0.05–Not Significant

In the group L (Lidocaine) the baseline value of MAP was 95.75 ± 8.23 mmHg and at immediately after intubation was 101.33 ± 12.01 mmHg. The MAP at one minute after intubation was 93.48 ± 11.00 mmHg, representing a fall of 2.27 mmHg which is not statistically significant. By 3, 5 and 10 minutes MAP values were 81.10 ± 9.92 mmHg, 78.10 ± 9.63 mmHg, 85.25 ± 12.10 mmHg and with a fall of 14.65, 17.65 and 10.5 mmHg respectively compared to baseline values. The decrease in MAP observed at 3, 5 and 10 minutes after intubation when compared with basal MAP was statistically highly significant ($p<0.01$).

DISCUSSION

Laryngoscopy and endotracheal intubation elicit a reflex cardiovascular response in the form of hypertension and tachycardia in adults. Though well tolerated in healthy adult patients it can have catastrophic consequences in patients with coronary artery disease and cerebrovascular diseases.[28]It is very much essential to minimize the hemodynamic response to laryngoscopy and intubation in high risk patients such as patients with history of coronary artery disease, hypertension and cerebrovascular diseases. To achieve this, it is important to understand the dynamic interactions between the drugs used and onset of drug effects. One should avoid over treating these responses which are usually short lived and well tolerated by most patients-one ounce of prevention is worth a pound of cure.[29] In present study In the Group L, the pre-induction mean HR was 87.3±10 bpm. The mean heart rate after 1min of study drug administration was 93.45±11.3 bpm, immediately after intubation was 98.3±12.2 bpm, one minute after intubation it was 96.6±11.4 bpm representing a rise of 9.3 bpm from the pre-induction heart rate. By 3, 5 and 10 minutes mean HR were 91±16.8 bpm, 91.1±11.7 bpm and 90.4±11.8 bpm respectively. The mean heart rate did not come to the pre-induction levels even by 10th minute. The increase in mean HR immediately after intubation, at 1and 5 minutes after intubation compared to basal HR was statistically significant($p<0.001$).These results match with that of Miller *et al*[30] In the group L (Lidocaine) the baseline value of mean SBP was 128.15 ± 9.99 mmHg and at immediately after intubation was 129.38 ± 13.40 mmHg. The mean systolic blood pressure one minute after intubation was 122.68 ± 13.05 mmHg, representing a fall of 5.47 mmHg. By 3, 5 and 10 minutes mean SBP values were 109.25 ± 10.09 mmHg, 103.75 ± 10.84 mmHg, 112.50±13.80 mmHg and with a fall of 18.9, 24.4 mmHg and 15.65 mmHg respectively compared to baseline values. The decrease in mean SBP observed at 1, 3, 5 and 10 minutes after intubation when compared with basal SBP was statistically significant ($p<0.05$). In the group L (Lidocaine) the baseline value of mean DBP was 79.63 ± 9.38 mmHg and at immediately after intubation was 86.70 ± 13.24 mmHg, a significant rise. The mean diastolic blood pressure one minute after intubation was 79.45 ± 11.57 mmHg, representing a fall of 0.18mmHg which is not statistically significant. By 3, 5 and 10 minutes mean DBP values were 67.05 ± 10.87 mmHg, 64.13 ± 13.58 mmHg, 71.63±11.96 mmHg and with a fall of 12.58, 15.5 and 8 mmHg respectively compared to baseline values. The decrease in mean DBP observed at 3, 5 and 10 minutes after intubation when compared with basal DBP was statistically significant ($p<0.05$). These results match with that of Stanley *et al*[31] and Hamill*et al*[32] In the group L (Lidocaine) the baseline value of MAP was 95.75 ± 8.23 mmHg and at immediately after intubation was 101.33 ± 12.01 mmHg. The MAP at one minute after intubation was 93.48 ± 11.00 mmHg, representing a fall of 2.27 mmHg which is not statistically significant. By 3, 5 and 10 minutes MAP values were 81.10 ± 9.92 mmHg, 78.10 ± 9.63 mmHg, 85.25 ± 12.10 mmHg and with a fall of 14.65, 17.65 and 10.5 mmHg respectively compared to baseline values. The decrease in

MAP observed at 3, 5 and 10 minutes after intubation when compared with basal MAP was statistically highly significant ($p<0.01$).

CONCLUSION

Lignocaine significantly attenuates the cardiovascular responses to laryngoscopy and intubation.

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