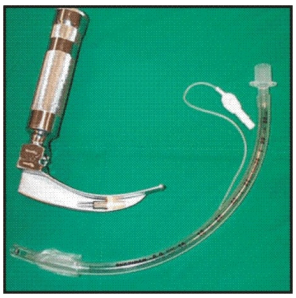
ORIGINAL PROF-890

LARYNGOSCOPY AND TRACHEAL INTUBATION;

EFFICACY OF IV LIGNOCAINE IN ATTENUATING HEMODYNAMIC RESPONSES



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ABSTRACT ... liaqut786@hotmsil.com **Objectives:** To assess the efficacy of IV Lignocaine in a dose of 1.5 mg/kg to attenuate the haemodynemic response of laryngoscopy and tracheal intubation. **Design of study:** A control interventional prospective study. **Setting:** CMH Gujranwala and CMH Muzaffarabad. **Period:** Jan 2004 to Dec 2004. **Method & materials:** 120 ASA I & II patients anaesthetized with thiopentone sodium and suxamethomium. Patients were allocated randomly to a control group or three treatment groups to receive lignocaine 1.5 mg/kg I. V, 3,2, minutes respectively before laryngoscopy. **Results:** Maximum increase in heart rate was 1 minute after laryngoscopy and intubation in all four groups (21-30%). There was no significant difference between the groups. Maximum rise in MAP was also 1 minute after laryngoscopy and intubation averaging 31.2%, 25%, 26.9%, 26.4% in group I, II, II, IV respectively. There was no significant (P>.05) difference in the rise of mean arterial pressure(MAP) in patients receiving IV lignocaine when compared with the control group. **Conclusions:** IV lignocaine in a dose of 1.5 mg/kg starting 3 minutes before laryngoscopy and intubation does not suppresses significantly, the increase in MAP and heart rate , during laryngoscopy and endotracheal intubation.

Key Words: Lignocaine, laryngoscopy. Hemodynamic response.

INTRODUCTION

Laryngoscopy and tracheal intubation after induction of anaesthesia are frequently associated with hypertension and tachycardia[1]. Mean arterial pressure may increases from 30 to 60 torr, when compared with awake pre-intubation levels[2]. The changes in blood pressure have been associated with increased intracranial pressure, cerebral haemorrhage and cardiac failure with pulmonary edema[3]. This post intubation response is potentially harmful in patients with preoperative hypertension, cardiovascular disease and raised intra-cranial pressure[4]. In an attempt to attenuate this response, various maneuvers and techniques have been recommended including, pretreatment with I/V lignocaine, topical local anaesthetics, vasodilators, low and high dose opioids, adrenoceptor blockers and magnesium sulphate. The aim of this study was to investigate the effect of I/V lignocaine, on the changes in arterial blood pressure and heart rate, during laryngoscopy for tracheal intubation.

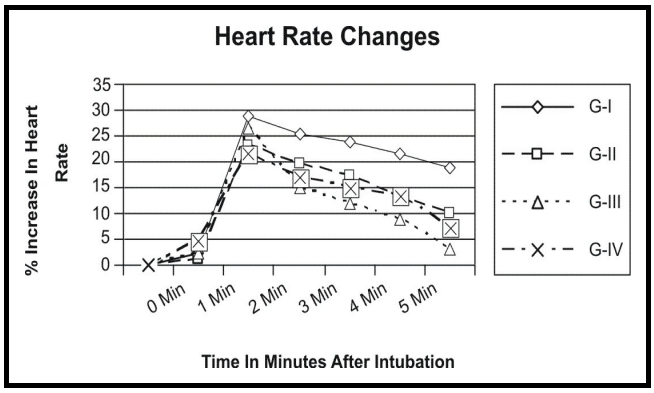
MATERIAL & METHODS

We selected 120 adult patients. None were taking drug with cardiovascular effects. All the patients were ASA I or

II and presented for elective (Non Cardiovascular) surgery. They were between the ages of 30-60 years. Only those patients were included in the study in which intubation was completed successfully without any difficulty (< 30 s) and without external displacement of the larynx. No pre-medication was given to these patients. On arrival of the patients in the operating room and before the induction of general anaesthesia, blood pressure (NIBP) and heart rate monitoring was commenced with the help of DINAMAP, electro-cardiography (ECG) monitoring was also started. A peripheral vein was cannulated in the ward, before sending the patients to the operating room. The patients were allocated to four groups randomly (Thirty patients in each group). Patients in group I (control) were given normal saline 4 ml I/V over 30 seconds beginning 2 min before laryngoscopy. In group 2,3,&4 patients were given I/V lignocaine 1.5 mg/kg over 30 seconds beginning 3, 2 and 1 min respectively, before laryngoscopy.

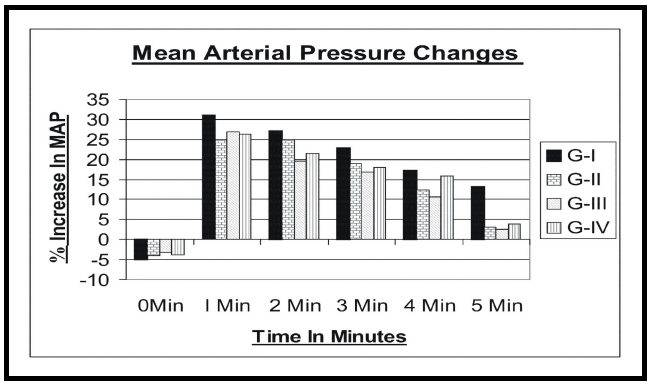
Anaesthesia was induced with thiopentone Sodium 5 mg/kg given over 30 seconds, beginning 2.5 min before laryngoscopy followed by suxamethonium 1.5 mg/kg I/V. At the same time as the initial injection of thiopentone was started the patients began to breath 60% Nitrous oxide in Oxygen via a face mask. When the patient's breathing stoped, he was manually ventilated until tracheal intubation, after which, anaesthesia was maintained with isoflurane in 60% Nitrous oxide in Oxygen, via the tracheal tube. All laryngoscopies and subsequent tracheal intubation were performed by senior anaesthetist, using a Macintosh laryngoscope and cuffed tracheal tube (portex).

Patient's heart rate, systolic blood pressure (SBP), mean arterial pressure (MAP) and diastolic blood pressure (DBP) were recorded. Measurements were made at preinduction control reading (0 min), after induction & immediately before laryngoscopy (3 min), then after one minute interval for five minutes. The data at each of the measurement points were compared and analyzed using analysis of variance and student's t test. $P < 0.05$ was taken as significant.



RESULTS

All the groups were comparable in age, weight and physical status (Table I). There was no significant inter-group difference for baseline mean arterial pressure and heart rate (Table II). There was no evidence of lignocaine toxicity. Only those patients were included in the study in which intubation was completed successfully without any difficulty in less than 30 seconds. Heart rate increased above the base level after induction i.e. before laryngoscopy but the magnitude of this increase was not significant (Fig. 1). Subsequently maximal increases in heart rate (21-30%) occurred 01 minute after laryngoscopy and intubation. There was no significant difference between the groups. Systolic Arterial Pressure (SAP), diastolic Arterial Pressure (DAP) mean arterial pressure (MAP) increased above the base line levels after laryngoscopy in all the groups (Table II).



The rise in SAP, DAP and MAP, in group I , recorded after the laryngoscopy and intubation was higher than the groups ,II, III & IV but the difference was not

significant.

Table I. Demographic Data				
Parameters	**G-I**	**G-II**	**G-III**	**G-IV**
Age (Average in years)	49	46	47	48
Weight (Average in Kgs)	67.4	63.6	65.4	61.5
Physical Status				
ASA-I	22	26	27	25
ASA-II	08	04	03	05
Mean duration of Laryngoscopy (Sec)	23	26	25	28
Sex F/M	9/21	10/20	12/18	16/14

Table II. Haemodynamic Changes								
Parameter	**Group**	**B/Line**	**0 Min**	**1 Min**	**2 Min**	**3 Min**	**4 Min**	**5 Min**
Heart rate (Beats/min)	G-I	79±6.2	81±5.3	103±2.0	99±7.5	98±8.0	96±6.4	94±5.3
	G-II	83±2.0	84±3.6	102±8.4	99±2.4	97±7.3	93±4.3	91±3.5
	G-III	81±3.0	83±2.2	101±7.2	93±4.0	90±2.5	88±3.5	83±3.8
	G-IV	84±1.5	88±3.5	102±0.3	98±5.0	96±5.3	95±4.4	90±7.5
Systolic BP (mm Hg)	G-I	136±3.6	131± 4.2	160±6.8	156±4.7	148±43	142±2.0	140±6.5
	G-II	142±7.6	136±6.5	174±5.0	158±5.5	151±3.6	145±7.7	144±7.0
	G-III	134±1.2	133±2.4	155±6.5	154±7.4	149±3.1	140±6.6	136±3.6
	G-IV	148±5.5	140±7.5	176±2.7	166±3.2	158±2.8	160±1.0	158±2.8
Diastolic BP (mm Hg)	G-I	81±3.1	80±3.0	99±2.2	96±3.3	92±1.6	87±2.9	84±6.5
	G-II	84±4.6	85±2.5	95±6.7	90±2.3	88±7.2	86±9.0	86±5.0
	G-III	78±6.8	72±7.6	97±9.1	91±4.5	86±6.6	82±2.5	80±2.0
	G-IV	82±2.7	78±6.2	107±1.2	102±4.1	99±3.2	92±3.8	88±3.5
Mean arterial BP(mm Hg)	G-I	96±0.5	91±5.2	126±7.4	122±6.2	118±2.7	103±3.0	99±6.4
	G-II	100±7.1	96±2.5	125±8.3	125±3.3	119±7.3	107±3.6	92±7.3
	G-III	93±2.4	90±8.4	118±7.5	108±7.7	105±1.5	100±4.6	90±1.6
	G-IV	102±6.8	98±9.8	129±2.1	120±3.6	114±1.2	108±6.6	106±3.5

Maximal increases in MAP above base line occurred 1 minute after tracheal intubation averaging 31.2% i.e. 20.7±1.8 torr (mean ±SD) in control patients where as rise in MAP after 1 minute was 25% i.e. 24±5 torr , 26.9 % i.e 25± torr and 26.4% i.e 27.8±6 torr (mean±D), in groups II, III and IV respectively (Fig 2).

There was no significant difference (p> 0.5)in the rise of Mean Arterial Pressure in patients receiving I/V lignocaine, when compared with the control group.

DISCUSSION

Reflex circulatory responses to direct laryngoscopy and tracheal intubation during general anaesthesia were first described about 55 years ago[5]. Pressure responses during intubation result from both stimulation by direct laryngoscopy and placement of the tracheal tube. This response is potentially harmful in patients with cardiac disease or increased intra cranial pressure. Roy and Edelist[6] found that there is a high incidence of myocardial ischemia during non-cardiac surgical procedures in patients with coronary artery disease.

The highest risk interval during anesthesia and operation, is the period in which intubation is performed, particularly when it was associated with an increased rate pressure product[7]. Post intubation pressure responses have been associated with ST segment changes, ventricular arrhythmias, pulmonary edema and rupture of cerebral aneurysm[8].

Various methods to attenuate the sympathetic response to laryngoscopy and intubation have been studied. King et al[5] found that deep Ether anaesthesia abolished this response. Stoelting[9] effectively used a bolus of sodium Nitroprusside to limit the increase in mean blood pressure after intubation. However this technique did not block the development of tachycardia.

Davies and Cowie[10] used 0.4 mg/kg of intravenous hydralazine and found it effective to control the intubation hypertension. The effects of pretreatment with 60 mg/kg body weight magnesium sulfate intravenously and cardiovascular responses, catecholamine release associated with tracheal intubation were measured by James et al[11]. They found that magnesium Sulfate attenuates the catecholamine mediated responses after tracheal intubation.

Beta blockers also prevent the haemodynamic responses to laryngoscopy and intubation. Esmolol was tried successfully by Gold and Harrington[12]. Narcotics may block afferent nerve impulses resulting from stimulation of the pharynx and larynx during intubation. Alfentanil was tried to blunt the pressor responses to tracheal intubation by Crawford and Smith[13]. They found that alfantanil 10 ugm/kg prevented any increase in heart rate and arterial pressure after tracheal intubation.

Lignocaine was advocated widely and was a standard agent for attenuation of the press or response at various centers. There are many reports[14,15] on the antitussive effects of I/V lignocaine. Lignocaine has also been shown to attenuate the increase in heart rate[14] arterial pressure[16] and intra cranial pressure associated with laryngoscopy and intubation. In this study we used 1.5 mg/kg of lignocaine, higher doses can produce tinnitus, circumoral paraesthesia and dizziness. The optimal time of injection of I/V lignocaine for the maximal attenuation of circulatory response was studied by Tam, Chung and Campbell [17]. They concluded that a dose of 1.5 mg/kg offered complete attenuation against post intubation increases in heart rate and arterial blood pressure when given 3 min prior to intubation and partial attenuation when given 2 min prior to intubation. Andrew Levitt and colleagues[18] found that Esmalol and Lignocaine have similar efficacies to attenuate haemodynamic response to intubation.

In contrast Miller & Warren[19] showed that I/V lignocaine fails to attenuate the cardiovascular response to laryngoscopy and tracheal intubation. Laurito and co-authors[20] gave nebulized lignocaine 4 mg/kg over 15 minutes and Chara-Emmer-Jorgensen and Coworkers[21] gave IV lignocaine 1.5 mg/kg beginning 2 min before laryngoscopy, both these studies showed that lignocaine had no significant effect in the cardiovascular effects of

intubation and laryngoscopy. In this study, we used IV Lignocaine 1.5 mg kg^{-1}within 3 min of laryngoscopy and intubation. IV lignocaine failed to suppress the intubation response. The results in this study are comparable with other recent studies. Helfman et al [22] compared Lignocaine, Fentanyl and Esmolol to prevent tachycardia and hypertension associated with tracheal intubation. They concluded that only Esmolol provided consistent and reliable protection against increases in both heart rate and systolic blood pressure accompanying laryngoscopy and intubation.

Durrani and Piotre Janicki[4] compared chloroprocaine with lignocaine and found that chloroprocaine blunted the cardiovascular response of laryngoscopy and tracheal intubation while lignocaine did not. Chaudhary and Arora[23] found that pre-treatment with Ketamine 1.0 mg/kg is superior to 1,5 mg/kg of lignocaine. There is currently no evidence to support the use of intravenous Lignocaine as a pretreatment to suppress the haemodynamic responses to laryngoscopy and tracheal intubation.

CONCLUSION

We conclude that I/V lignocaine in a dose of 1.5 mg/kg, starting 3 minutes before laryngoscopy and intubation, does not suppress significantly, the increases in MAP and heart rate, during endotracheal intubation.

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Don't hate your enemy; Sort him out

Awais Shuja