Original Article

A study of variations in sympathetic response to laryngoscopy and intubation observed with intravenous magnesium sulphate, esmolol and lignocaine

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Abstract

Introduction: 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. Increase in heart rate and blood pressure is well documented sequelae of direct laryngoscopy and endotracheal intubation in normotensive individuals. **Aims and Objectives:** To Study Variations in Sympathetic Response to Laryngoscopy and Intubation Observed With Intravenous Magnesium Sulphate, Esmolol and Lignocaine. **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:** Statistical evaluation between the groups showed that the decrease in mean HR observed in group E was statistically significant when compared to decrease in mean HR in the group L, M ($p<0.001$) at 1min after drug admin, immediately after intubation and 1, 3, 5 and10 minutes following intubation ; also mean SBP observed in group L was statistically highly significant when compared to decrease in mean SBP in the group E, M ($p<0.05$) immediately after intubation and 1, 3, 5 and10 minutes following intubation. The decrease in mean SBP at 10min after intubation was also maximum in group Lin comparison to the baseline value and the decrease in mean DBP observed in group E was statistically significant when compared to decrease in mean DBP in the group L, M ($p<0.05$) immediately after intubation and 1 minute following intubation. The decrease in mean DBP observed in group L was statistically significant when compared to decrease in mean SBP in the group E, M ($p<0.05$) at 3 and 5 minutes following intubation. The decrease in mean DBP at 10min after intubation was not significant in any of the groups in comparison to the baseline value. **Conclusion:** Esmolol is more effective when compared to both lignocaine and magnesium sulphate in attenuating the cardiovascular response i.e. Sympathetic Responseto laryngoscopy and intubation.

Keywords: Magnesium Sulphate, Esmolol, Lignocaine,Sympathetic Response to Laryngoscopy and Intubation.

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INTRODUCTION

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. Increase in heart rate and blood pressure are well documented sequelae of direct laryngoscopy and endotracheal intubation in normotensive individuals.[1,2,3,4] This transient, self-limiting hypertension and tachycardia are innocuous in healthy

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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 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. In year 1982.[28] a new intravenous cardio-selective β blocker Esmolol Hydrochloride became available for clinical use. Many authors used esmolol to blunt the short lived haemodynamicsequelae associated with laryngoscopy and intubation because of its unique properties such as rapid onset of action, peak effect within minutes and short elimination half-life after intravenous administration. Intravenous esmolol has been extensively tried in patients[29,30] by many authors to blunt the pressor response to tracheal intubation and found to be effective and safe. Intravenous Magnesium Sulphate has been extensively tried with reasonable margin of safety in management of Pregnancy induced hypertension[35.]Inhibition of catecholamine release[32] and vasodilatory properties[33] of Magnesium sulphate prompted us to study its effect on pressor response to laryngoscopy and intubation. There is not much literature comparing intravenous lidocaine, esmolol and magnesium sulphate in attenuating the cardiovascular stress response to laryngoscopy and intubation. This prompted us to conduct this PROSPECTIVE RANDOMISED PLACEBO CONTROLLED STUDY to determine which among intravenous lidocaine 1.5mg/kg or intravenous esmolol 2mg/kg or intravenous magnesium sulphate 30mg/kg given prior to laryngoscopy and intubation is superior in attenuating the cardiovascularstress response to laryngoscopy and 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.Group C –received normal saline 3ml i.v 3 minutes before intubation. Group E – received esmolol 2mg/kg i.v. bolus 3 minutes before intubation. Group L – received lignocaine 1.5mg/kgi.v bolus 3 minutes before intubation. Group M – received magnesium sulphate 30mg/kg iv 3 minutes before intubation. 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

In Table 1:Statistical evaluation between the groups showed that the decrease in mean HR observed in group E was statistically significant when compared to decrease in mean HR in the group L, M ($p<0.001$) at 1min after drug admin, immediately after intubation and 1, 3, 5 and10 minutes following intubation. In Table 2:Statistical evaluation between the groups showed that the decrease in mean SBP observed in group L was statistically highly significant when compared to decrease in mean SBP in the group E, M ($p<0.05$) immediately after intubation and 1, 3, 5 and10 minutes following intubation. The decrease in mean SBP at 10min after intubation was also maximum in group Lin comparison to the baseline value. In Table 3:Statistical evaluation between the groups showed that the decrease in mean DBP observed in group E was statistically significant when compared to decrease in mean DBP in the group L, M ($p<0.05$) immediately after intubation and 1 minute following intubation. The decrease in mean DBP observed in group L was statistically significant when compared to decrease in mean SBP in the group E, M ($p<0.05$) at 3 and 5 minutes following intubation. The decrease in mean DBP at 10min after intubation was not significant in any of the groups in comparison to the baseline value.

Table 1: Heart rate (bpm) changes –INTERGROUP comparison

HR	Group	Mean	StdDev	SE of Mean	95% CI for Mean		F	p	Sig Diff Between
					Lower Bound	Upper Bound			
Baseline	L	87.35	10.11	1.60	84.12	90.58	4.016	0.009*	L vs M (P=0.006)
	E	85.28	8.76	1.38	82.47	88.08			
	M	79.78	11.10	1.75	76.23	83.32			
	C	84.40	10.28	1.63	81.11	87.69			
1 Min after drug	L	93.45	11.31	1.79	89.83	97.07	15.019	<0.001*	L vs E (P<0.001)
	E	80.38	8.97	1.44	77.48	83.29			L vs M (P=0.045)
	M	86.43	13.62	2.15	82.07	90.78			E vs N (P<0.001)
	C	96.25	11.91	1.88	92.44	100.06			M vs N (P=0.001)
Immediately after intubation	L	98.38	12.29	1.94	94.44	102.31	11.101	<0.001*	L vs E (P<0.001)
	E	85.03	8.82	1.41	82.17	87.89			E vs M (P<0.001)
	M	95.53	10.34	1.63	92.22	98.83			L vs N (P<0.001)
	C	95.25	12.16	1.92	91.36	99.14			
1 Min post intubation	L	96.60	11.41	1.80	92.95	100.25	11.453	<0.001*	L vs E (P<0.001)
	E	82.95	8.74	1.40	80.12	85.78			L vs M (P=0.004)
	M	88.30	10.86	1.72	84.83	91.77			E vs N (P=0.002)
	C	92.00	11.63	1.84	88.28	95.72			
3 Min post intubation	L	91.00	16.85	2.66	85.61	96.39	7.841	<0.001*	L vs E (P=0.001)
	E	80.03	8.57	1.37	77.25	82.80			E vs M (P=0.001)
	M	90.63	10.54	1.67	87.25	94.00			Evs N (P=0.001)
	C	90.90	10.76	1.70	87.46	94.34			
5 Min post intubation	L	91.13	11.70	1.85	87.38	94.87	15.349	<0.001*	L vs E (P<0.001)
	E	77.33	8.16	1.31	74.69	79.98			L vs M (P=0.001)
	M	82.78	7.64	1.21	80.33	85.22			E vs N (P<0.001)
	C	88.20	10.93	1.73	84.70	91.70			
10 Min post intubation	L	90.45	11.81	1.87	86.67	94.23	3.080	0.029*	L vs E (P=0.040)
	E	83.49	6.71	1.07	81.31	85.66			
	M	84.18	13.24	2.09	79.94	88.41			
	C	86.18	12.06	1.91	82.32	90.03			

Table 2: Systolic blood pressure changes – INTERGROUP comparison

SBP	Group	Mean	StdDev	SE of Mean	95% CI for Mean		F	P	Sig Diff Between
					Lower Bound	Upper Bound			
Baseline	L	128.15	9.99	1.58	124.96	131.34	0.844	0.472	
	E	127.78	10.98	1.74	124.26	131.29			
	M	129.68	11.77	1.86	125.91	133.44			
	C	131.30	11.49	1.82	127.63	134.97			
1 Min after drug	L	112.40	10.77	1.70	108.95	115.85	21.622	<0.001*	L vs E (P=0.001)
	E	122.36	11.35	1.82	118.68	126.04			L vs M (P<0.001)

	M	129.45	11.51	1.82	125.77	133.13			L vs N(P<0.001)
	C	131.48	13.10	2.07	127.28	135.67			E vs N (P=0.004)
	L	129.38	13.40	2.12	125.09	133.66			L vs M (P=0.024)
Immed. after intubation	E	130.77	11.16	1.79	127.15	134.39	6.083	0.001*	L vs N (P=0.004)
	M	138.75	12.00	1.90	134.91	142.59			E vs N (P=0.017)
	C	140.58	19.38	3.06	134.38	146.77			
	L	122.68	13.05	2.06	118.50	126.85			L vs N (P=0.004)
1 Min post intubation	E	124.74	9.75	1.56	121.58	127.90	4.552	0.004*	E vs N (P=0.032)
	M	127.43	17.14	2.71	121.94	132.91			
	C	134.13	17.57	2.78	128.51	139.74			
	L	109.25	10.09	1.60	106.02	112.48			L vs M(P=0.002)
3 Min post intubation	E	116.51	10.15	1.63	113.22	119.80	12.793	<0.001*	L vs N (P<0.001)
	M	120.55	14.33	2.27	115.97	125.13			E vs N (P=0.002)
	C	127.60	17.98	2.84	121.85	133.35			
	L	103.75	10.84	1.71	100.28	107.22			L vs M(P=0.001)
5 Min post intubation	E	110.33	8.35	1.34	107.63	113.04	12.618	<0.001*	L vs N (P<0.001)
	M	114.85	13.47	2.13	110.54	119.16			E vs N (P=0.002)
	C	120.73	16.76	2.65	115.36	126.09			
	L	112.50	13.80	2.18	108.09	116.91			L vs N (P=0.003)
10 Min post intubation	E	116.95	10.70	1.71	113.48	120.42	4.250	0.006*	
	M	117.03	18.19	2.88	111.21	122.84			
	C	123.70	12.83	2.03	119.60	127.80			

Table 3: Diastolic blood pressure changes – INTERGROUP comparison

DBP	Group	Mean	StdDev	SE of Mean	95% CI for Mean		F	P	Sig Diff Between
					Lower Bound	Upper Bound			
	L	79.63	9.38	1.48	76.63	82.62			
Baseline	E	77.43	6.93	1.10	75.21	79.64	2.571	0.056	
	M	82.25	10.50	1.66	78.89	85.61			
	C	81.63	7.04	1.11	79.37	83.88			
	L	73.25	9.26	1.46	70.29	76.21			L vs N (P<0.001)
1 Min after drug administration	E	74.77	7.61	1.22	72.30	77.24	22.359	<0.001*	E vs N (P<0.001)
	M	78.08	7.86	1.24	75.56	80.59			M vs N (P<0.001)
	C	87.78	9.89	1.56	84.61	90.94			
	L	86.70	13.24	2.09	82.46	90.94			
Immediately after intubation	E	82.44	7.03	1.13	80.16	84.72	6.651	<0.001*	E vs N (P<0.001)
	M	83.80	9.68	1.53	80.71	86.89			M vs N (P=0.003)
	C	92.05	10.64	1.68	88.65	95.45			
	L	79.45	11.57	1.83	75.75	83.15			
1 Min post intubation	E	76.62	6.35	1.02	74.56	78.67	5.306	0.002*	E vs N (P=0.001)
	M	80.05	9.67	1.53	76.96	83.14			
	C	85.43	11.62	1.84	81.71	89.14			
	L	67.05	10.87	1.72	63.57	70.53			L vs E (P =0.040)
3 Min post intubation	E	72.74	7.60	1.22	70.28	75.21	9.396	<0.001*	L vs M (P=0.005)
	M	74.00	8.04	1.27	71.43	76.57			L vs N (P<0.001)
	C	77.80	9.81	1.55	74.66	80.94			
	L	64.13	13.58	2.15	59.78	68.47			L vs E (P=0.029)
5 Min post intubation	E	70.67	6.62	1.06	68.52	72.81	8.521	<0.001*	L vs M (P=0.016)
	M	71.08	9.75	1.54	67.96	74.19			L vs N (P<0.001)
	C	75.53	9.45	1.49	72.50	78.55			
	L	71.63	11.96	1.89	67.80	75.45			
10 Min post intubation	E	77.62	8.05	1.29	75.00	80.23	2.538	0.059	
	M	76.20	13.66	2.16	71.83	80.57			
	C	77.30	9.35	1.48	74.31	80.29			

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.[34]There is increased release of catecholamines norepinephrine, epinephrine and vasopressin- the result of which is tachycardia and hypertension. It also causes a rise in intracranial pressure. 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. Toachieve 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. Various methods are employed to minimise the adverse hemodynamic responses to laryngoscopy and intubation- curtailing or reducing the duration of laryngoscopy, use of ACE inhibitors, sodium nitroprusside, lidocaine sprays and gargles, use of McCoy bladeinstead of Macintosh blade, use of thoracic epidural anaesthesia to blunt the stress response and use of beta blocker esmolol. Studies have shown that Magnesiumsulpahte is also effective in preventing the pressure response. The present clinical study was undertaken to compare the effects of three drugs – Esmolol, Lignocaine, Magnesium sulphate. In our study we have found Statistical evaluation between the groups showed that the decrease in mean HR observed in group E was statistically significant when compared to decrease in mean HR in the group L, M ($p<0.001$) at 1min after drug admin, immediately after intubation and 1,3,5 and10 minutes following intubation ; also mean SBP observed in group L was statistically highly significant when compared to decrease in mean SBP in the group E, M ($p<0.05$) immediately after intubation and 1,3,5 and10 minutes following intubation. The decrease in mean SBP at 10min after intubation was also maximum in group Lin comparison to the baseline value and the decrease in mean DBP observed in group E was statistically significant when compared to decrease in mean DBP in the group L, M ($p<0.05$) immediately after intubation and 1 minute following intubation. The decrease in mean DBP observed in group L was statistically significant when compared to decrease in mean SBP in the group E, M ($p<0.05$) at 3 and 5 minutes following intubation. The decrease in mean DBP at 10min after intubation was not significant in any of the groups in comparison to the baseline value. These findings are in agreement with study of Menkhaus *et al* [35] and Vucevic *et al* [36].

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