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ORIGINAL ARTICLE

A RANDOMIZED CLINICAL TRIAL OF COMPARISON OF PRESSOR RESPONSE DURING AND AFTER TRACHIAL EXTUBATION AND LMA (LARYNGEAL MASK AIRWAY) REMOVAL IN CONTROLLED HYPERTENSIVE PATIENTS

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ABSTRACT: BACKGROUND: The primary goal of an anaesthesiologist is to maintain a patent airway during general anaesthesia. Endotracheal intubation is considered as gold standard for securing an unobstructed airway. Endotracheal intubation and extubation cause reflex cardiovascular responses due to mechanical stimulation of the upper respiratory tract. Several methods have been used for attenuating these cardiovascular responses. Each method or drug that is used for attenuating these pressor responses has its own associated sequelae or disadvantages. Most certain method of avoiding adrenergic responses would be to avoid both laryngoscopy and endotracheal intubation. Use of laryngeal mask airway has also shown lesser haemodynamic response as no laryngoscopy is required for its placement. **AIM:** To compare the haemodynamic responses associated with laryngeal mask airway removal and endotracheal extubation in controlled hypertensive patients undergoing elective surgical procedures. **DESIGN:** A prospective randomized comparative study. **METHODS:** The study was conducted in eighty-controlled hypertensive patients in the age group 35-65 years belongs to ASA grade 1&2 scheduled for elective surgical procedure. Patients were allocated in a randomized manner by the envelop method, into two groups one undergoing endotracheal tube intubation (Group T) and other group undergoing laryngeal mask airway insertion (Group L). At the end of the procedure. Endotracheal extubation and laryngeal mask airway removal was carried out when patient was completely conscious and responded to verbal commands. Pulse rate and blood pressure were recorded for the study at the following intervals; A). Pre induction., B). Just before extubation or laryngeal mask removal (Baseline value), C). 1, 2, 3, 5 and 10 minutes after extubation., Rate pressure product and mean arterial pressure at those intervals were calculated. **RESULTS:** After airway instrumentation, in patients of group T, mean pulse rate, mean of mean arterial pressure and mean rate pressure product increased to a statistically significant levels above baseline and did not come back to baseline values even after 10 minutes of extubation. In patients of group L even though the rise was significant after instrumentation it came back to baseline values within 5 minutes post instrumentation. The rise was more significant in group T compared to group L. **CONCLUSION:** The conclusions drawn from this study are that in controlled hypertensive patients, the haemodynamic responses produced by laryngeal mask airway removal are lesser and short lived as compared to endotracheal extubation. Use of Laryngeal mask airway is quite advantageous in hypertensive patients where in there is a concern about the pressor responses due to airway instrumentations.

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KEYWORDS: Controlled Hypertension, Laryngeal Mask Airway, Pressor Response, Trachiall Extubation.

INTRODUCTION: The primary goal of an anaesthesiologist is to maintain a patent airway during general anaesthesia. Endotracheal intubation is considered as gold standard for securing an unobstructed airway. But, as good things come with a price tag, endotracheal intubation is accomplished at the cost of pressor responses.

Endotracheal intubation and extubation cause reflex cardiovascular responses due to mechanical stimulation of the upper respiratory tract. The predominant response is tachycardia and systemic hypertension. These reflex responses are mediated by increased sympathetic nervous system activity.[1] Sympatho-adrenergic responses that occur at intubation as well as during extubation may lead to complications like myocardial infraction, left ventricular failure, cerebrovascular accidents, intracranial hypertension and a rise in intraocular pressure.

Several methods have been used for attenuating these cardiovascular responses viz, use of sodium nitroprusside, fentanyl, esmolol, calcium channel blockers and by using topical airway anaesthesia.[2,3,4,5,6]

Hypertensive patients have been shown to exhibit exaggerated cardiovascular responses during intubation and extubation than normotensive patients.

Yoshitaka Fuji, Hiderori Toyooka[7] carried out a study to evaluate the haemodynamic changes of tracheal extubation in normotensive and hypertensive patients. From their study, they concluded that hypertensive patients exhibit exaggerated pressor responses than normotensive patients.

Prys Robert et al[1] evaluated the pressor responses to laryngoscopy and intubation in controlled and uncontrolled hypertensive patients. They were surprised to find that patients on antihypertensive therapy with well controlled blood pressure, were equally prone to develop hypertensive responses to intubation.

Thus, even though the blood pressure of a patient is very well controlled preoperatively, pressor responses are just same as that of uncontrolled hypertensive patients and are exaggerated than normotensive patients.

Each method or drug that is used for attenuating these pressor responses has its own associated sequelae or disadvantages.

Most certain method of avoiding adrenergic responses would be to avoid both laryngoscopy and endotracheal intubation.

The laryngeal mask airway which was first described by Brain A. I. J in 1983, can be used for maintenance of a clear and secure airway and for its placement no laryngoscopy is required.[8]

Use of laryngeal mask airway has also shown lesser haemodynamic response as no laryngoscopy is required for its placement.[9,10]

N. Brande et al (1989) conducted a study to compare the pressor responses of tracheal intubation with that of laryngeal mask insertion in healthy patients. From their study they concluded that insertion of the laryngeal mask airway is accompanied by lesser cardiovascular responses.

In a similar study I.G Wilson et al (1992), compared the cardiovascular responses induced by laryngoscopy and intubation with those produced by insertion of a laryngeal mask. From their

study, they concluded that laryngeal mask airway insertion is associated with lesser cardiovascular response.

Although the endotracheal intubation and the problems associated with it have received much attention, endotracheal extubation has been relatively less emphasized. The score and significance of these problems are real. The endotracheal extubation also causes a transient increase in blood pressure and heart rate.[11]

Not much literature is available on haemodynamic responses to laryngeal mask airway removal in hypertensive patients and there is a knowledge gap in this aspect.

Hence, here an attempt was made to compare the pressor responses at endotracheal extubation and laryngeal mask airway removal in controlled hypertensive patients undergoing elective surgical procedures.

METHODS: This study was designed to evaluate the cardiovascular changes related to laryngeal mask airway removal and to compare those with endotracheal extubation in controlled hypertensive patients.

After obtaining approval of the ethical committee, this study was carried out on patients at MGM hospital, warangal.

Patient selection and pre anaesthetic evaluation;

The study was conducted in eighty-controlled hypertensive patients in the age group of 35-65 years belonging to ASA Grade II scheduled for elective surgical procedures. A controlled hypertensive patient is a known hypertensive patient on antihypertensives with systolic blood pressure less than 140mmHg diastolic blood pressure less than 90mmHg or a recently diagnosed case, diagnosed at least 2 weeks before surgery on antihypertensives with systolic blood pressure <140 and diastolic blood pressure less than 90mmHg.[12]

Patients were allocated in a randomized manner by the envelop method, into two groups one undergoing endotracheal tube intubation (Group T) and other group undergoing laryngeal mask airway insertion (Group L). Each group had 40 patients.

Informed consent was obtained from all the patients during pre anaesthetic check-up which was carried out one day prior to surgery. A meticulous history and a thorough clinical examination, pulse rate, blood pressure, respiratory rate, weight, airway assessment and systemic examination including cardiovascular system, respiratory system and per abdomen was carried out.

The exclusion criteria of this study consisted of;

1. History of chronic obstructive pulmonary diseases.
2. Emergency surgical procedures
3. Pregnant women
4. Morbid obese patient
5. Patients with pharyngeal mass

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All the patients were investigated pre operatively and following investigations were done.

1. Haemoglobin estimation.
2. Urine examination; albumin, sugar, microscopic examination.
3. Random blood sugar.
4. Blood urea and serum creatinine.
5. Chest X-ray.
6. ECG and Echo if necessary.

Premedications: All the patients received 5mg alprazolam orally night before surger y. Inj On d an setr on 4mg IV inj glycopyrrolate 0.005mgkg^{-1}, and inj ranitidine 50mg I.V were administered 60minutes before surgery.

Procedure: Once the patient was shifted to the operation theatre, pulse rate and blood pressure were noted. pre-oxygenation done with 100% oxygen for 5 minutes, inj. Fentanyl 1µg/kg i.v. given. Intravenous induction was carried out with injection thiopentone sodium 5mgkg-1, Inj. Lignocaine 1.5mg/kg i.v. given. After the loss of eyelash reflex, injection succinylcholine 1.5mgkg-1 administered and positive pressure ventilation was carried through a face mask using 100% oxygen. After one minute either laryngoscopy and endotracheal intubation or blind laryngeal mask insertion size 3 or size 4 was carried out as per the groups allocated to them. Air was injected into the cuff of endotracheal tube or laryngeal mask cuff until a tactile seal was achieved.

Anaesthesia was maintained with oxygen (50%) + nitrous oxide(50%) + Sevoflurane (0.5%) and vecuronium bromide 0.1mgkg^{-1}.

At the end of procedures patients were reversed with Inj glycopyrrolate 0.01mgkg^{-1} and neostigmine 0.05mgkg^{-1}i.v. Endotracheal extubation and laryngeal mask airway removal was carried out when patient was completely conscious and responded to verbal commands.

Pulse rate and blood pressure were recorded for the study at the following intervals;

1. Pre induction.
2. Just before extubation or laryngeal mask removal (Baseline value).
3. 1, 2, 3, 5 and 10 minutes after extubation.
4. Rate pressure product and mean arterial pressure at those intervals were calculated.

RESULTS: This study was carried out in 80 controlled hypertensive patients undergoing elective surgical procedures under general anaesthesia. The patients in group T (Endotracheal extubation) were compared with group L (Laryngeal mask airway removal). The results of this study were as follows;

Group T	Group L
n=40	n=40

Table 1: Shows the distribution of patients in two groups

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Age group	Group T	%	Group L	%
35-50	28	70%	30	75%
51-65	12	30%	10	25%

Table 2: Shows the distribution of age in years in two groups. Mean age in group T and group L were 47.08 and 46.48 years respectively

Range of weight (Kgs)	Group T		Group L	
	No. of patients	%	No. of patients	%
36-50	12	30%	18	45%
51-65	20	50%	18	45%
66-80	8	20%	4	10%

Table 3: Shows the distribution of weight in kgs, in two groups. Mean weight in patients of group T and L were 56.30 and 52.08kgs respectively

Sex	Group T	%	Group L	%
Males	11	27	12	30
Females	29	73	28	70

Table 4: Shows distribution of sex in both groups

All haemodynamic values are expressed as mean±SD (HS-Highly significant, NS-non significant).

Mean	Group T	Group L	't' value	'p' value
Pulse rate beats/min	88±11.5	88.5±9.7	0.01	0.99 (NS)
Mean of MAP	100.2±6.764	99±5.773	0.88	0.38 (NS)
Mean rate Pressure Product	12261.7±1825.51	12042.6±1146.32	0.60	0.55(NS)

Table 5

Table 5 shows that baseline haemodynamic values of mean pulse rate, mean of mean arterial pressure and mean of rate pressure product were comparable in both the groups (i.e. there was no significant variation between endotracheal tube extubation group and laryngeal mask removal group).

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	Group T				Group L			
	Base line	**After 1 minute**	**'t' value**	**'p' value**	**Base line**	**After 1 minute**	**'t' value**	**'p' value**
Mean Pulse rate	88.00 ±11.5	110.63 ±12.87	29.01	0.0000052 (HS)	88.5±9.7	94.00±10.02	12.23	0.0000063 (HS)
Mean of Mean arterial pressure	± 6.76	± 8.72		15 (HS)	± 5.77	± 10.77		0.0000015 (HS)
Mean rate pressure product	12261.7 ± 1825.51	17571.8 ± 2101.82	51.78	0.0000014 (HS)	12042.6± 1146.32	13421.1± 1629.74	12.77	0.0000016 (HS)

Table 6.1: Intra group comparison after one minute

Table 6.1 shows that mean haemodynamic values one minute after airway instrumentation in both the groups were significantly high when compared to baseline.

	Group T	**Group L**	**'t' values**	**'p' values**
Mean pulse rate	110.63± 12.874	94±10.021	6.25	0.00003 (HS)
Mean of mean arterial pressure	119.567± 8.72701	105.133± 10.7764	6.66	0.000004 (HS)
Mean rate pressure product	17571.8± 2101.82	13421.1± 1629.74	9.99	0.000002 (HS)

Table 6.2: Inter group comparison after one minute

Table 6.2 shows that the rise in haemodynamic values in group T was significantly more than that of group L.

	Group T				Group L			
	Base line	**After 2 minutes**	**'t' value**	**'p' value**	**Base line**	**After 2 minutes**	**'t' value**	**'p' value**
Mean pulse rate	88±11.5	107 ±12.14	24.97	0.0000013 (HS)	88.5±9.7	92.72 ±10.14	8.17	0.0000056 (H.S)

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Mean of mean arterial pressure	100.2± 6.76	115.33 ±5.64	18.97	0.0000027 (H.S)	99±5.77	102.28 ±6.27	4.81	0.0000023 (H.S)
Mean rate pressure product	12261.7 ± 1825.51	16689.65 ± 2215.9	24.88	0.0000015 (H.S)	12042.6 ± 1146.32	13043.45 ± 1704.69	6.83	0.0000036 (H.S)

Table 7.1: Inter group comparison after two minutes

	Group T	**Group L**	**'t' value**	**'p' value**
Mean pulse rate (beats/min)	107±12.147	92.72±10.145	5.7774	0.000002 (H.S)
Mean of mean arterial pressure	115.33±5.6427	102.28±6.726	9.5	0.000002 (H.S)
Mean rate pressure product	16689.65±2215.9	13043.45±1704.696	8.353	0.0000031 (H.S)

Table 7.2: Shows that the rise in group T was significantly more than that of group L

Table 7.1 shows that mean haemodynamic values two minutes after airway instrumentation in both the groups were significantly high when compared to baseline.

	Group T				**Group L**			
	Base line	**After 3 minutes**	**'t' value**	**'p' value**	**Base line**	**After 3 minutes**	**'t' Value**	**'p' value**
Mean pulse rate	88±11.5	105.98 ±14.78	11.06	0.0000016 (H.S)	88.5 ±9.7	90±10.82	1.94	0.0009 (H.S)
Mean of mean arterial pressure	100.2 ±6.76	112±5.57	18.95	0.0000031 (H.S)	99± 5.77	99.55±10.82	1.20	0.23 (N.S)
Mean rate pressure product	12261.7± 1825.51	16076.6 ±2233.22	15.99	0.000001 (H.S)	12042.6 ± 1146.32	12427.1± 167.46	1.92	0.001 (S)

Table 8.1: Intra group comparison after three minutes

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	Group T	Group L	't' value	'p' value
Mean pulse rate (beats/min)	105.98± 14.786	90±10.82	5.3923	0.000009 (HS)
Mean of mean arterial pressure	112 ± 5.57			0.0000081 (HS)
Mean rate pressure product	16074.6± 2233.22	12427.1± 167.46	8.36817	0.0000029 (HS)

Table 8.2: Shows that the rise in haemodynamic values in group

Table 8.1 shows mean haemodynamic values three minutes after airway instrumentation. Values in group T were significantly higher compared to baseline. In group L heart rate was significantly high compared to baseline, MAP reached baseline and RPP was near base line.

T were significantly more than in group L.

	Group T				Group L			
	Base line	After 5 minutes	't' value	'p' value	Base line	After 5 minutes	't' value	'p' value
Mean pulse rate	88±11.5	100.9± 10.75	4.32	0.00000 65 (HS)	88.5±9.7	88.1± 10.77	0.65	0.54 (NS)
Mean of mean arterial pressure	100.2± 6.76	110.15 ±5.43	8.33	0.00000 27 (HS)	99±5.77	98.35± 5.62	-2.36	0.54 (NS)
Mean rate pressure product	12261.7± 1825.51	15076.5± 1599.91	4.75	0.00000 36 (HS)	12042.6± 1146.32	11964.4± 1574.21	0.30	0.40 (NS)

Table 9.1: Intra group comparison after five minutes

	Group T	Group L	't' value	'p' value
Mean pulse rate (beats/min)	100.9±10.758	88.1±10.777	5.38	0.000001 (HS)

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Mean of mean arterial pessure	110.15± 5.43	98.35±5.62	9.66	0.0000097 (HS)
Mean rate pressure product	15076.5 ± 1599.91	11964.4±1574.21	8.88	0.000003 (HS)

Table 9.2: Inter group comparison after five minutes

Table 9.1 shows mean haemodynamic values five minutes after airway instrumentation. Values in group T were significantly higher compared to baseline. In group L, haemodynamic values had reached to baseline.

Table 9.2 shows that these haemodynamic values were significantly more in group T compared to group L.

	Group T				**Group L**			
	Base line	**After 10 minutes**	**'t' value**	**'p' value**	**Base line**	**After 10 minutes**	**'t' value**	**'p' value**
Mean pulse rate	88±11.5	98.87± 14.61	5.56	0.0000027 (HS)	88.5±9.7	87.32± 10.83	-2.97	0.08 (NS)
Mean of mean arterial pressure	100.2±6.76	108.77± 5.48	9.86	0.0000038 (HS)	99±5.77	99.28± 5.48	-3.38	0.051 (NS)
Mean rate pressure product	12261.7± 1825.51	14614± 2303.8	7.00	0.0000026 (HS)	12042.6± 1146.32	11868± 1580	-4.07	0.06 (NS)

Table 10.1: Intra group comparison after ten minutes

	Group T	**Group L**	**'t' value**	**'p' value**
Pulse rate (beats/ min)	98.875± 14.612	83.225± 10.831	4.06	0.00014 (HS)
Mean of arterial pressure	108.77± 5.4882	99.283± 5.4827	8.65	0.00008 (HS)
Mean rate pressure product	14614± 2303.8	11868±1580	6.29	0.00002 (HS)

Table 10.2: Inter group comparison after ten minutes

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Table 10.1 shows haemodynamic response at 10 minutes after airway instrumentation. In group T, haemodynamic values were significantly higher when compared to baseline values, whereas these values had come to baseline in patients of group L.

Table 10.2 shows that these haemodynamic values were significantly higher in group T compare to group L.

DISCUSSION: Laryngoscopy and endotracheal extubation provoke a transient sympathetic response which manifests as tachycardia and hypertension. These are probably of no consequence in healthy individuals but these responses are a matter of concern in patients with cardiovascular disease like hypertension or coronary artery disease. Hypertensive patients have been shown to exhibit exaggerated cardiovascular response at extubation than normotensive patients. Sympatho-adrenergic responses that occur at intubation as well as during extubation may leads to complications like myocardial infraction, left ventricular failure, cerebrovascular accidents, intracranial hypertension and rise in intraocular pressure. Several methods have been used for attenuating these cardiovascular responses such as deepening the level of anaesthesia, use of drugs like opioids, lignocaine, beta blockers, calcium channel blockers or using topical airway anaesthesia. Each method or drug has its own associated sequelae or disadvantages.

Laryngeal mask airway which was first described by Brain A.I.J in 1983 can be used for maintaining a clear and secure airway[8].

We have studied 80 ASA Grade II controlled hypertensive adult patients, who were randomly allocated into 2 groups of 40 each.

Endotracheal extubation was performed in patients of group T, whereas size 3 or size 4 laryngeal mask airway removal was carried in group L.

The premedication was administrated in all the patients preoperatively, which consisted of I.V. glycopyrrolate 0.005 $mgkg^{-1}$, i.v. inj.rantidine 50mg i.v., inj.Ondansetron 4mg i.v.and inj. Fentanyl 1µg/kg. Intravenous induction of anaesthesia was carried out with inj thiopentone sodium $5mgkg^{-1}$, inj.lignocaine 1.5mg/kg and succinylcholine $1.5mgkg^{-1}$ i.v. given. Patients were maintained on O_2 (50%)+N_2O(50%) + Sevoflurane (0.5%) and vecuronium bromide $0.1mgkg^{-1}$ and IPPV.

Preinduction heart rate and blood pressure recorded. Subsequent measurements for the study were recorded immediately before extubation or removal of LMA (served as baseline value) and at 1, 2, 3, 5 and 10 minutes after extubation or LMA removal. The results were analyzed statistically using unpaired students „t" test for intergroup comparison and paired „t" test for intragroup comparison.

There was no difference in the demographic data of age, sex and weight in the patients in both the groups.

Pulse rate: In patients belonging to Group T, the mean pulse rate at the baseline (immediately before extubation) was 88±11.5 beats/minutes whereas it was raised to 110.60±12.87 beats/minutes at one minute after instrumentation. The values of pulse rate at 2, 3, 5 and 10 minutes after instrumentation were 107±12.14, 105.93±14.78, 100.9±10.75 and 98.87±14.61 respectively. These results indicates that laryngoscopy and extubation caused instantaneous and

significant increase in mean pulse rate which did not came back to baseline level even after 10 minutes.

The mean pulse rate, in patients of group L, at baseline (just before LMA removal) and 1, 2, 3, 5 and 10 minutes after LMA removal are as follows 88.5±9.7, 94±10.02, 92.72±10.14, 90±10.8, 88.1±10.77 and 87.32±10.83 respectively. This shows that laryngeal mask removal was also associated with significant increase in mean pulse rate compared to baseline and which returned back to baseline at 5minutes after the removal but the rise in mean pulse rate after extubation in group T was significantly higher than in group L. This shows that the sympathetic responses were lesser in patients of group L as compared to patients of group T.

Rate Pressure Product: In this study, mean rate pressure product in patients of group T at base line (just before extubation) and 1, 2, 3, 5 and 10 minutes after extubation were 12261.7± 1825.51, 17571.8±2101.82, 16689.65±2215.6, 16076.6±2233.22, 15076.5±1599.91 and 14614 ±2303.8 respectively. This shows that extubation caused a significant rise in mean rate pressure product from baseline value, which failed to reach baseline value even after 10 minutes of extubation.

The mean rate pressure product in patients of group L at base line (just before LMA removal), 1 minute, 2, 3, 5 and 10 minutes after (LMA removal) were 12042.6±1146.32, 13421.1±1629.74, 13043.45±1704.69, 12427.1±167.46, 11964.4±1574.21 and 11868±1580 respectively. This shows that LMA removal caused a significant rise in the rate pressure product when compared to baseline and the values came down to baseline value at 5 minutes post instrumentation.

This rise in rate pressure product was significantly higher in group T than in group L.

Mean Arterial Pressure: In this study mean arterial pressure in patients of group T, at baseline (just before extubation) and 1, 2, 3, 5 and 10 minutes after extubation were 100.2 ± 6.76, 119.56±8.72, 115.33±5.64, 112±5.57, 110.15±5.43 and 108.77±5.48 respectively. This shows a significant rise in mean arterial pressure after extubation which did not reached baseline values even after 10 minutes post extubation.

The mean arterial pressure in patients of group L at baseline (just before LMA removal) and 1, 2 3, 5 and 10minutes after LMA removal were 99±5.77, 105.13±10.77, 102.28±6.27, 99.55±10.82, 98.35±5.62 and 99.28±5.48 respectively. This shows a significant rise in mean arterial pressure after LMA removal which reached baseline values at 5 minutes after LMA removal.

This rise in mean arterial pressure was significantly higher in group T than in group L.

From this study we have observed that:

a. Both endotracheal extubation and laryngeal mask airway removal were associated with a statistically significant rise in pulse rate, rate pressure product and mean arterial pressure when compared with baseline values.
b. The rise was significantly higher in the extubation group as compared to laryngeal mask airway removal group. The values did not returned to baseline values even after 10minutes of extubation, whereas in laryngeal mask group the haemodynamic values returned to baseline levels, 5 minutes after LMA removal.

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Endotracheal extubation causes rise in haemodynamic values because of mechanical stimulation of respiratory tract including vocal cords causing increased sympathetic nervous system activity in the cervical sympathetic fibres, whereas the lesser cardiovascular response associated with laryngeal mask removal may be due to the fact that the laryngoscopy was avoided and vocal cords were not stimulated.

Roofa Mushtaq et al[12] conducted a study to observe the circulatory responses to laryngeal mask airway removal and compared it with endotracheal extubation in controlled hypertensive patients. They found that mean pulse rate in extubation group at baseline (just before extubation) was 100±12 beats/minutes, which was increased to 134±11 beats/minutes post extubation. This rise was significant even after 5minutes of extubation (114.0±11 beats/minutes). The mean rate pressure product at baseline was 15847±1836, which increased to 22454±2284 post extubation and was 17544.15±2088, 5minutes after extubation. The mean systolic and diastolic pressures in extubation group at baseline was 157±7/101±6mmHg, was increased to 167±6/108±4 mmHg post-extubation and was 158±8/102±4mmHg at 3 minutes after extubation.

The pulse rate in laryngeal mask airway removal group before LMA removal was 85±16 beats/minute, immediately after LMA removal and three minutes after LMA removal were 99±15 and 94±15 beats/minute respectively. The rate pressure product before LMA removal, 1 and 3 minutes after LMA removal was 13535±2892, 14893.7±2949 and 13805.65±2650.50 respectively.

Mean systolic and diastolic blood pressure before LMA removal, 1 and 3 minutes after LMA removal was 140±10**/**90±10, 148 ±10**/**94±8 and 143±10**/**88.0±7 mmHg respectively.

They concluded that haemodynamic data in laryngeal mask group at baseline increased to statistically significant levels post removal, but came back to baseline values 3 minutes post removal, whereas these values were significantly higher even after 5minutes of extubation.

Thus the results of our study are consistent with those of above study. In a study conducted by Yoshitaka Fuji MD et al,[7] pressor response to tracheal extubation was compared with the response to laryngeal mask airway removal in normotensive and hypertensive patients. The mean pulse rate changed from 73±9 beats/minute to 95±14 beats/minute after extubation in normotensive patients, at 2minute it was 86±10 beats/minutes, and after 3minute it was 81±9. The pulse rate increased immediately after endotracheal extubation and remained elevated for two minutes. The MAP before extubation in normotensive patients immediately, 1, 3 and 5 minutes after extubation was 96±8, 124±18, 123±15, 112±12 and 106±11 respectively. MAP increased immediately following extubation and remained elevated for three minutes.

In LMA group in normotensive patients, pulse rate was 76±11 before LMA removal and immediately 1, 2 and 3 after LMA removal were 81±11, 82±14, 79±13 and 77±13. Pulse rate increased immediately after LMA removal and remained elevated for one minute after LMA removal.

MAP before LMA removal in normotensive patients, immediately, 1, 2, 3 and 5minutes after LMA removal were 93±10, 106±10, 101±12, 96±16, 94±10 respectively. MAP increased immediately following LMA removal and remained elevated for two minutes.

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The increases in these variables were less in LMA group than in endotracheal group. In hypertensive patients, the heart rate before extubation, immediately after extubation 1, 2, 3 and 5minutes after extubation were 77±8, 105±10, 100±10, 95±10, 96±9 and 84±9 respectively. The heart rate increased immediately after tracheal extubation and remained elevated for three minutes.

MAP before extubation, immediately after extubation, 1,2,3,5 and 10minutes after extubation were 96±8, 124±18, 123±15, 117±12, 112±12, 106±11 and 96±9.

MAP increased immediately after extubation and remained elevated for five minutes. In LMA group in hypertensive patients HR, before LMA removal, immediately 1, 2, 3 and 5minutes were 98±9, 119±12, 118±10, 108±6 and 104±8 respectively. Heart rate increased immediately after LMA removal and remained elevated for one minute. MAP also increased immediately after LMA removal and remained elevated for three minutes.

Thus the conclusion drawn from their study was that cardiovascular responses to extubation were greater than those related to removal of LMA in both normotensive and hypertensive patients. The changes in haemodynamic variables immediately following extubation or LMA removal from baseline levels were greater in hypertensive patients than in normotensive patients. This is in agreement with our study

In a similar study conducted by Bukhari S A et al,[13] the two devices for airway control were compared for pressor responses and intraocular pressure changes following insertion of laryngeal mask airway and endotracheal intubation. They observed a significant increase in heart rate in both the groups after insertion.

However the increase in heart rate was more in endotracheal group. Similarly both systolic and diastolic blood pressures and intraocular pressure increased significantly in endotracheal group. Although the increase in these parameters were observed in LMA group also, but this increase was significantly less than after endotracheal intubation. This is in agreement with our study.

In another study conducted by Brande N et al,[9] it was observed that there was a significant increase in systolic blood pressure immediately after tracheal intubation and for the subsequent 2 minutes. Mask insertion in the laryngeal mask group was associated with a systolic increase that achieved significance at one minute after insertion. The increase in mean blood pressure is both lesser and of shorter duration. This is in agreement with our study.

Thus from our study we confirm that laryngeal mask airway removal is associated with lesser cardiovascular responses as compared to endotracheal extubation in controlled hypertensive patients.

CONCLUSIONS: The conclusions drawn from this study are that in controlled hypertensive patients, the haemodynamic responses produced by laryngeal mask airway removal are lesser and short lived as compared to endotracheal extubation. Use of Laryngeal mask airway is quite advantageous in hypertensive patients where in there is a concern about the pressor responses due to airway instrumentations.

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