Research Article

Haemodynamic changes during insertion and removal of LMA and ILMA

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Abstract

The laryngeal mask airway (LMA) is a new airway management device andhas become increasingly popular in anaesthesia to maintain airway patency during spontaneous and controlled ventilation.the standard LMA is not an ideal intubation aid as the airway tube is too narrow to accommodate an adult diameter tracheal tube, too long to ensure a normal length tracheal tube will reach the trachea and not sufficiently rigid to function as a guide to exact alignment of the mask with glottis. In addition, the mask aperture base may obstruct passage of the tracheal tube. In an attempt to overcome these limitations, a prototype intubating laryngeal mask airway (ILMA) has been developed.Presently there are contradictory reports regarding haemodynamic changes during laryngoscopy, intubation and extubation with or without ILMA. Hence this study was planned to evaluate the haemodynamic changes to insertion and removal of LMA in comparison with ILMA in healthy patients as reduced haemodynamic response may be beneficial to the patients with especially with cardiovascular and cerebral diseases.

Key Word: laryngeal mask airway (LMA), Intubating Laryngeal Mask Airway(ILMA).

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INTRODUCTION

The laryngeal mask airway (LMA) is a new airway management device that was invented by Dr. Archie Brain at London Hospital, Whitechapel, in 1981[1]. It was introduced into clinical practice in late 1992. It has become increasingly popular in anaesthesia to maintain airway patency during spontaneous and controlled ventilation.It is a novel device that fills the gap in airway management between tracheal intubation and in the use of facemask[2]. It has the advantage that it does not require laryngoscopy for insertion. The haemodynamic response to the insertion of the LMA is significantly less than after laryngoscopy and tracheal intubation. However, the standard LMA is not an ideal intubation aid as the airway tube is too narrow to accommodate an adult diameter tracheal tube, too long to ensure a normal length tracheal tube will reach the trachea and not sufficiently rigid to function as a guide to exact alignment of the mask with glottis. In addition, the mask aperture base may obstruct passage of the tracheal tube. In an attempt to overcome these limitations, a prototype intubating laryngeal mask airway (ILMA) has been developed[3]. The intubating laryngeal mask airway is the most recent trend of laryngeal mask airway to become available. This was introduced in clinical practice in 1997[4]. The new device is not intended as a replacement for the standard laryngeal mask airway but is an alternative means of intubating the trachea, even in cases of failed intubation under direct laryngoscopy. A potential advantage of intubating laryngeal mask guided intubation is that its placement does not require distortion of the pharyngeal structures and might be less stimulating with less haemodynamic alterations.Joo and colleagues showed that the haemodynamic response to ILM-guided intubation is less than laryngoscope-guided intubation and the incidence of postoperative pharyngolaryngeal morbidity, airway complications and overall intubation success was similar[5]. Presently there are contradictory reports regarding haemodynamic changes during laryngoscopy, intubation and extubation with or without ILMA. Hence this study

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was planned to evaluate the haemodynamic changes to insertion and removal of LMA in comparison with ILMA in healthy patients as reduced haemodynamic response may be beneficial to the patients with especially with cardiovascular and cerebral diseases.

MATERIALS AND METHODS

This study was conducted in the department of Anaesthesiology and Intensive Care, Himalayan Institute of Medical Sciences, Swami Ram Nagar, Dehradun. The study was undertaken in sixty patients of either sex, ranging from 20-70 yrs. of age, belonging to the ASA grade I and II scheduled to undergo elective surgical procedures under general anaesthesia. ASA grade III and IV, cardiorespiratory or cerebrovascular disease i.e. blood pressure >150/90 mmHg, sore throat within 10 days, risk of aspiration, head and neck surgery, known difficult airway or Mallampati grade IV, patient weighing 20% more or less than their ideal body weight were excluded from the study. Pre-anaesthetic checkup was done for each patient, a day prior to surgery. History was asked about the presence of hypertension, coronary artery disease, respiratory disease, drug intake and drug allergy. General physical examination and systemic examination of cardiovascular , respiratory and central nervous system were done. Airway assessment using Mallampati classification to predict the likelihood of difficult intubation was done. Basic routine investigations like haemoglobin (Hb), bleeding time (BT), clotting time (CT), urine analysis, X-ray chest and electrocardiogram (ECG) were carried out. Special investigations like blood glucose, blood urea nitrogen (BUN), serum creatinine and serum electrolytes were advised in specific patients wherever required, to rule out systemic disorders. If reports were within normal limits, the patients were taken up for the study and surgery.

Patients were randomly allocated into two groups of 30 patients each. After getting permission from the ethical committee, informed consent was taken for patient's participation in the study. All patients were kept fasting since midnight day before surgery.

Premedication

All patients were given tab. diazepam 10mg orally at night (10 p.m.) before surgery and tab. diazepam 5mg was given with a sip of water in the morning (6 a.m) on the day of surgery.

Anaesthesia Technique

Consent and fasting status of the patient were confirmed on arrival of the patient in the preoperating room. Intravenous line was secured with 18G cannula and infusion commenced with Ringer lactate solution. After the patient was shifted to the operation table, ECG leads were fixed and a continuous heart rate, lead II ECG monitoring,, non-invasive blood pressure and pulse oximeter monitoring were done. Monitoring of Heart rate, Systolic blood pressure (SBP), Diastolic blood pressure (DBP), Mean arterial pressure (MAP), SpO_2 and ECG were done by using LandT (Lunar) multichannel monitor. Basal reading of heart rate, arterial pressure and SpO_2 were noted.

The patient was in a supine position with the head on a standard pillow 7cm in height. Preoxygenation was done with 100% oxygen by facemask delievered through a magill circuit for 3 minutes. Anaesthesia was induced with sleep dose of inj. thiopentone 4-7 mg/Kg IV slowly. To facilitate the insertion of LMA/ ILMA inj. Vecuronium 0.1mg/Kg IV was given and ventilated with 100% oxygen for another 3 minutes. Maintenance of anaesthesia was done with 66% N_2O in oxygen, muscle relaxant inj. vecuronium 0.015mg/Kg IV and inj morphine 0.1mg/Kg IV.

Insertion Technique

Before insertion of appropriate size LMA/ ILMA, the cuff of the LMA/ ILMA was deflated and the posterior surface of the LMA/ILMA was lubricated with 2% lignocaine jelly. In group I, LMA was inserted by using standard technique. The mouth may be allowed to fall open. The tube portion was grasped as if it was a pen, with the aperture facing anteriorly; the tip of the cuff was placed against the inner surface of the upper incisors or gums. The mask was pressed back against the hard palate to keep it flattened, as it was advanced in to the oral cavity, using the index finger to push upward against the palate. A change of direction could be sensed as the mask tip encountered the posterior pharyngeal wall and followed it downward. The cuff should then be inflated with enough air to obtain a seal. The correct placement was confirmed when no cuff was visible in the oral cavity, the presence of a smooth oval swelling in the neck around the thyroid and cricoid area and the slight outward movement of tube upon LMA inflation. Then gentle manual intermittent positive pressure ventilation (IPPV) was started via the LMA. In group II, ILMA was inserted using a one handed rotational technique with the head and neck in the neutral position. After insertion of ILMA, the cuff was inflated with air. Gentle manual intermittent positive pressure ventilation (IPPV) was started via the ILMA. The adequacy of ILMA placement was assessed as satisfactory if the peak inspiratory pressure was less than 20cm H_2O and end tidal CO_2 35-40 mmHg. The position was maintained for intubation by holding the handle firmly. The lubricated silicon tracheal tube was placed in the ILMA tube and advanced to 1cm beyond the epiglottic elevating bar. Intubation was then attempted by gently advancing the tube. If no resistance was felt after the tube was advanced by 8cm, the cuff was inflated.

Successful intubation was confirmed by detection of CO_2 in the expired gases. We excluded patients from further study in whom intubation failed at the first trial. Surgeons were requested not to clean, drape or position the patients till 5 minutes after placement of LMA/ILMA, so as to avoid any stimuli likely to interfere with the findings. Serial heart rate, arterial pressure, SpO_2 and ECG recording were done at the time of insertion, 1, 3, 5 minutes following insertion. In group II after 5 minutes of insertion of ILMA, cuff of ILMA was deflated and tracheal tube connector was removed. The ILMA was removed using a 25cm length tube, cut from an 8mm silicon tracheal tube (TT) as a pusher to prevent accidental extubation while the device was being withdrawn. At the time of removal and 1 minute after that, heart rate, arterial pressure, SpO_2 ECG recording were done. At the end of surgical procedure, reversal of the neuromuscular blockage was done by inj. Neostigmine 40 µg/Kg IV and inj. Glycopyrrolate 10 µg/Kg IV and gentle assisted ventilation was done to allow the patient to start breathing. Oxygen was continuously administered through the anaesthetic circuit. When reflexes were restored and the patient was able to open the mouth on command, cuff was deflated and LMA was removed. At the time of removal of LMA and 1 minute after that, heart rate arterial pressure, SpO_2 and ECG recording were done. Oral suctioning was done and airway patency and respiratory depth was verified.

RESULTS AND OBSERVATIONS

Table 1: Heart rate per min at different intervals of time in Group I and Group II

Variables	Group I	Group II	p-value	Remarks
Basal	80.47 ± 3.13	76.00 ± 8.37	>0.05	NS
Insertion	84.33 ± 1.65	87.10 ± 9.68	>0.05	NS
1 min	79.47 ± 3.31	83.50 ± 6.55	< 0.01	HS
3 min	75.53 ± 3.88	77.27 ± 5.72	>0.05	NS
5 min	72.80 ± 3.66	73.27 ± 5.72	>0.05	NS
Removal	82.30 ± 1.97	83.23 ± 9.69	>0.05	NS
1 min	82.97 ± 1.71	81.37 ± 9.05	>0.05	NS

Table 2: Systolic blood pressure at different intervals of time in Group I and Group II

Variables	Group I	Group II	p-value	Remarks
Basal	128.57 ± 5.94	126.20 ± 8.09	> 0.05	NS
Insertion	142.83 ± 6.91	163.77 ± 10.59	< 0.01	HS
1 min	134.77 ± 6.78	152.63 ± 9.77	> 0.05	NS
3 min	133.17 ± 6.63	142.33 ± 9.29	< 0.01	HS
5 min	125.20 ± 5.27	126.90 ± 7.99	> 0.05	NS
Removal	129.40 ± 5.28	139.73 ± 9.18	> 0.05	NS
1 min	127.87 ± 5.97	144.40 ± 9.51	< 0.01	HS

Table 3: Diastolic blood pressure at different intervals of time in Group I and Group II

Variables	Group I	Group II	p-value	Remarks
Basal	75.17 ± 5.79	75.40 ± 5.31	> 0.05	NS
Insertion	88.40 ± 13.74	95.00 ± 6.74	< 0.01	HS
1 min	86.57 ± 8.66	87.50 ± 6.12	> 0.05	NS
3 min	80.67 ± 6.17	83.93 ± 6.01	> 0.05	NS
5 min	77.97 ± 5.15	76.47 ± 4.99	> 0.05	NS
Removal	81.17 ± 10.37	85.73 ± 5.71	> 0.05	NS
1 min	77.97 ± 10.95	85.87 ± 4.89	> 0.05	NS

Table 4: Mean arterial pressure at different intervals of time in Group I and Group II

Variables	Group I	Group II	p-value	Remarks
Basal	93.03 ± 3.76	92.27 ± 5.34	> 0.05	NS
Insertion	106.37 ± 8.95	117.57 ± 6.67	< 0.05	S
1 min	102.73± 4.90	109.30 ± 6.26	> 0.05	NS
3 min	98.37 ± 2.67	103.40 ± 6.08	> 0.05	NS
5 min	93.40 ± 2.24	93.83 ± 5.61	> 0.05	NS
Removal	97.23 ± 6.41	103.73 ± 5.84	> 0.05	NS
1 min	94.50 ± 6.97	105.50 ± 5.35	< 0.01	HS

Table 5: Showing comparison of mean heart rate, mean systolic blood pressure, mean diastolic blood pressure and mean arterial pressure between Basal and Insertion of LMA and ILMA

		Basal	Insertion	p-value	Remarks
Group I	Heart Rate	80.47 ± 3.13	84.33 ± 1.65	< 0.05	S
	Systolic Blood Pressure	128.57 ± 5.94	142.83 ± 6.91	< 0.05	S
	Diastolic Blood Pressure	70.17 ± 5.79	88.40 ± 13.74	> 0.05	NS
	Mean Arterial Pressure	93.03 ± 3.76	106.37 ± 8.95	> 0.05	NS
Group II	Heart Rate	76.00 ± 8.37	87.10 ± 9.68	> 0.05	NS
	Systolic Blood Pressure	126.20 ± 8.09	163.77 ± 10.59	< 0.01	HS
	Diastolic Blood Pressure	75.40 ± 5.31	95.00 ± 6.74	< 0.01	HS
	Mean Arterial Pressure	92.27 ± 5.34	117.57 ± 6.67	< 0.01	HS

Table 6: Showing comparison of mean heart rate, mean systolic blood pressure, mean diastolic blood pressure and mean arterial pressure between Basal and Removal of LMA and ILMA

		Basal	Removal	p-value	Remarks
Group I	Heart Rate	80.47 ± 3.13	82.30 ± 1.97	> 0.05	NS
	Systolic Blood Pressure	128.57 ± 5.94	129.40 ± 5.26	> 0.05	NS
	Diastolic Blood Pressure	70.17 ± 5.79	81.17 ± 10.37	> 0.05	NS
	Mean Arterial Pressure	93.03 ± 3.76	97.23 ± 6.41	> 0.05	NS
Group II	Heart Rate	76.00 ± 8.37	82.23 ± 9.69	< 0.01	HS
	Systolic Blood Pressure	126.20 ± 8.09	139.73 ± 9.18	< 0.05	S
	Diastolic Blood Pressure	75.40 ± 5.31	85.73 ± 5.71	< 0.05	S
	Mean Arterial Pressure	92.27 ± 5.34	103.73 ± 5.84	< 0.01	HS

DISCUSSION

We observed that in group I during insertion and removal, there was increase in heart rate as compared to basal value but non-significantly. In group I, there was non-significant increase in mean arterial pressure during insertion and removal compared with basal value. The findings of our study closely correlate with those of Hollande *et al.*[6] and Naqib *et al.*[7] who also have observed that during insertion and removal of LMA there was non-significant increase in heart rate and mean arterial pressure. Braude*et al.*[8] and Wilson *et al.*[9] observed that there was significant increase in haemodynamic variables after LMA insertion. Our results are contrary to their study. The significant increase in heamodynamic may be due to lack of proper analgesia. During insertion of LMA, pressure responses i.e. increase in heart rate and arterial pressure may be induced by the passage of the LMA through the oral and pharyngeal spaces, pressure on the larynx and the pharynx by an inflated cuff and the dome of the LMA. The signals are transferred to the brain through the trigeminal, glossopharyngeal and vagus nerves. These nerves carry the afferent impulses to the vasomotor centre which in turn activate sympatho adrenal system to release catecholamines resulting in increase of the heart rate and blood pressure. The increase in blood pressure is due to increased cardiac output rather than due to increased systemic vascular resistance. The cardio vascular response is maximum during the stimulation of epipharynx, where as those arising from stimulation of tracheo-bronchial tree is least marked. During removal of LMA, the haemodynamic response is probably triggered by pharyngeal stimulation during reverse rotation of cuff. In group II, during insertion/intubation there was non-significant increase in heart rate as compared to basal value, but during removal, there was highly significant increase in heart rate as compared to basal value. In group II, there were highly significant increase in mean arterial pressure during insertion and removal as compared to basal value. Kihara *et al.*[10](2000) observed that compared to pre-insertion value, there were significant increase in mean arterial pressure and heart rate, but compared with base line value there were no change in mean arterial pressure but there was increase in heart rate. General anaesthesia was induced with intravenous lidocaine 0.5 mg/kg, propofol 2.5 mg/kg and vecuronium 0.1 mg/kg. This suggests that the haemodynamic response produced by ILMA insertion/intubation is at least matched by the hypotensive response to anaesthesia. Our findings were in contrast with Kihara*et al.*[10], who showed that during insertion/intubation there was non-significant increase in mean arterial pressure and significant increase in heart rate. This interstudy difference may be related to their use of intravenous lidocaine and propofol at induction, that causes decrease in mean arterial pressure and reflex increase in heart rate. During removal mean arterial pressure did not exceed pre induction value but heart rate was higher than pre induction value. This may be due to a greater depth of anaesthesia during maintenance with 2% sevoflurane in oxygen 33% and nitrous oxide, which

causes non-significant increase in mean arterial pressure during removal as compared to pre induction value. Shimoda *et al.*[11](2002) observed that removal of ILMA produced a larger response than insertion and intubation of ILMA. During insertion/intubation there were, non-significant change in heart rate and mean arterial pressure, while removal causes significant increase in heart rate and mean arterial pressure as compared to basal value. General anaesthesia was induced with intravenous fentanyl 3 µg/kg, midazolam 0.1 mg/kg and vecuronium 0.1 mg/kg. The distribution half-life ($t_{½}$ α) of fentanyl and midazolam are reported to be 13.4 minute and 12 minute, respectively. The effect site concentration of two drugs would be expected to show slower changes than plasma concentration. Changes in drug effects over the duration of this study would thus not be expected to account for the observed difference in responses. To prevent accidental extubation during removal of ILMA, we tend to advance a tracheal tube towards the carina by pushing with the stabilizing rod. Movement of the tracheal tube probably provides the stimulus[12], which produces the different magnitudes of haemodynamic responses to removal versus insertion of the ILMA. In our study insertion/intubation of ILMA produced larger response then removal of ILMA. This may be due to removal of ILMA after 1 minute of successful intubation. Kihara *et al.*[10] observed that the impact of ILMA removal on haemodynamic response depends on its timing. If ILMA removal is accomplished 1-2 minutes after ILMA insertion / Intubation, arterial pressure and heart rate are raised but if removal occurs after more than 3 min after insertion / intubation the effect is less pronounced. This could either be due to summation of the two stimuli or changes in depth of anaesthesia over time. We observed that during insertion and removal of LMA, there are non-significant increase in heart rate and mean arterial pressure, while insertion/intubation of ILMA causes non-significant increase in heart rate, but significant increase in arterial pressure and its removal causes significant increase in heart rate and mean arterial pressure as compared with base line values. Bennett *et al.*[13] observed that insertion of LMA causes non-significant change in heart rate and mean arterial pressure as compared to base line value respectively. While there was non-significant increase in heart rate during intubation with ILMA but significant increase in mean arterial pressure. Our observations are in conformity with their study. Bennett *et al.*[13] observed that removal of the LMA and ILMA was not associated with heamodynamic changes, suggesting that this is less stressful than airway insertion. Our results are contrary to their study, this may be due to greater depth of anaesthesia by using sevoflurane, fentanyl and midazolam intermittently in their study. We observed that base line values of haemodynamic parameters in both groups were comparable. There was non-significant increase in heart rate in both group during insertion, during removal in group II there was highly significant increase in heart rate while in group I this was non-significant. However the increase in heart rate was more in group II and this was significant after 1 minute of insertion. There was non-significant increase in arterial pressure in group I during insertion and removal, however in group II this was highly significant. The increase in arterial pressure was more in group II and this increase was significant during insertion and 1 minute after removal.Kellar and calleagues[14]demonstrated that insertion of ILMA provides a more effective seal than the LMA but pharyngeal mucosal pressure for the ILMA are 3-70 times higher than for LMA and exceed capillary perfusion pressure at most location. The highest mucosal pressure for the ILMA were in the distal oropharynx where the rigid tube is firmly wedged against the bone of the anterior cervical vertebrae. Their reports and our findings indicated that insertion/intubation and removal of ILMA produce significant nociceptive stimuli to the upper airway compared to LMA and causes greater haemodynamicpressor response.

CONCLUSION

It is concluded from our study that LMA could be useful in situation where minimal changes in haemodynamics are desirable like patients with coronary artery disease, cerebral vascular disease.

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