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A comparison between topical & intravenous administration of lignocaine to aid the insertion of laryngeal mask airway

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Abstract

Introduction: The cough reflex and laryngeal spasm & cardiovascular hazards were significant after insertion of laryngeal mask airway.
Objective: Aim of the study to determine the proper way to aid the insertion of Laryngeal mask airway.
Patients & methods: The study was performed between December 2007 and December 2008 in Al jumhori teaching hospital.
Results: Cough and laryngeal spasm due to insertion of LMA were statistically significant in group -A- (control group), but cough and laryngeal spasm in group -B- (Lignocaine i.v.) is statistically not significant, and in group -C- (topical lignocaine) there were no cough and no laryngeal spasm.
Conclusion: the use of topical lignocaine spray is the best aid for insertion of LMA.
Abbreviations: LMA = Laryngeal mask airway**.**

Keywords: laryngeal mask airway, cough reflex, laryngeal spasm.

Introduction

Laryngeal spasm is a very primitive reflex and is intended to protect the lungs from inhalation of noxious substances by sensory stimulation of superior laryngeal nerve [(1)].

Laryngeal spasm may be precipitated by surgical and visceral stimulation such as incision, peritoneal traction, anal stretch and cervical dilatation)) or the presence of secretions, blood or foreign bodies ((e. g: an oropharyngeal airway or laryngeal mask airway in the region of the pharynx and larynx, mainly occur during light anesthesia. Children are particularly more prone to laryngeal spasm than adults [(1)].

Therefore, laryngospasm is most common during induction and emergence from general anaesthesia [(1)]. Cardio vascular response to laryngoscopy, intubation, insertion of laryngeal mask airway and insertion of Guedel airway arranged according to severity of the response. Causes hypertension, tachycardia and arrhythmias [(2)]. The deflated cuff laryngeal mask airway is lubricated and inserted blindly in to the hypopharynx but LMA insertion under direct visualization with a laryngoscope or fibro optic bronchoscope may prove beneficial in difficult cases [(3)].

Treatment of laryngeal spasm includes:

Providing gentle positive pressure ventilation with 100% oxygen or administering i.v. lignocaine (1-1.5mg / kg) if laryngeal spasm persists and hypoxia develops succinylcholine (0.25 — 1 mg /kg) should be given in order to paralyze the laryngeal muscles and allow controlled ventilation [3].

The stimulation of laryngeal mask to upper airways is not severing like laryngoscopy and intubation so the cardio vascular response is much less.... When we use lignocaine i.v. there will be cardio vascular stability, so the response will be less, on the other hand the use of lignocaine spray in enough doses will abolish, the cardio vascular response completely to the insertion of the laryngeal mask airway.

Cardio vascular response occurs during light general anesthesia, hypoxia, hypercapnia or cough can cause this reflex in group A, of patients because of stimulation of nerve endings of vagus and trigeminal nerves [4,5] and sympathoadrenal response [5].

Consequent rise in rate — pressure product may result in myocardial oxygen demand which exceed the oxygen supply and under these circumstances, may induce myocardial ischemia [6]. An increase in heart rate is more likely than hypertension to produce signs of myocardial ischemia on the ECG [7]. Indeed, in an anaesthetized patient the incidence of myocardial ischemia sharply increases in patients who experiences heart rate greater than 110 beats/min. ((Ischemic threshold)) [7] when heart rate is less than 110 beats/min. the incidence of myocardial ischemia is random and silent being unrelated to heart rate [7].

Conceptually, rapid heart rate increases the myocardial oxygen requirements and decreases the time during diastole for coronary blood flow and thus delivery of oxygen.

Conversely increased myocardial oxygen requirements produced by hypertension tend to be off set by improved perfusion through pressure dependent atherosclerotic coronary arteries [7]. In healthy patients these responses are generally well tolerated. However, in patients with limited coronary or myocardial reserve. myocardial ischemia or failure may follow. The patients with vascular lesion at risk such as intracranial vascular anomaly or trauma of the thoracic aorta may also suffer serious sequel [2]. This response is sympathetically mediated ([6, 8, and 9]

Lignocaine

Lignocaine was synthesized in 1943 in Sweden, by Lofgren of AB Astra and it was introduced in to clinical practice in 1948 [12].

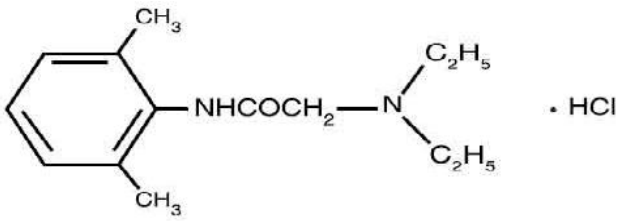

Figure1: Structure of lignocaine

Mechanism of action

It has quinidine like action (cell membrane stabilizing effect) which causes:

1- Reduce automaticity of pacemaker cells: suppress premature beats or an ectopic pacemaker.
2- Reduce responsiveness of cardiac cells to excitation.
3- Reduce conduction velocity of an impulse, both 2&3 of help to limit the speed of abnormal impulse. In normal therapeutic doses there is no change in myocardial pump action [12].

It does not alter the PR, QRS or QT intervals on ECG and has minimal negative inotropic effect. An excessive plasma concentration may decrease the conduction of cardiac impulse through the AV node and His - Purkinje system, and evoke seizures followed by coma. Large doses (5 mg / kg) administered to animal my increase the intensity and duration of neuromuscular blockade produced by non - depolarizing muscle relaxants .

Mode of action of lignocaine as a local anaesthetic

Three distinct sites have been proposed where local anaesthetic might exert their effect on Sodium conductance across the nerve membrane these are: -

- On the membrane surface involving alteration of the fixed negative charge and hence trans membrane potential without change in resting intra cellular potential.
- Within the membrane matrix, involving its lateral expansion. There by causing distortion of the sodium channel
- Specific receptors within the sodium channel.

Half life

After single i.v. injection, the plasma concentration declines in two phases: the first is rapid (8 min), and reflects dilution of the drug in the blood and distribution to tissue with rapid blood flow within half an hour the rate of fall in plasma concentration change to a second phase (90-100min) which reflects removal of the drug from the circulation by metabolism.

Dose and route of administration:

- **Loading dose**

Is 1-2 mg / kg as an i.v. bolus, followed by infusion for 12-48 hours, at a rate of 4 mg / min. for 30 min., 2 mg / min. for the next 2hrs. And then at 1 mg / min. For treatment of ventricular ectopic.

- **Through endotracheal tube to got a systemic effect.**

- **Oral**

It has high hepatic first pass metabolism, require too frequent dosing to be practicable.

- **Topical**

Due to its high lipid solubility and rapid route of absorption, therapeutic plasma concentration may occur following laryngeal spray '[14], peritoneal dialysis, after topical application to the epicardium during open heart surgery and following subcutaneous infiltration during neurosurgery [14].

Metabolism:

Lignocaine is metabolized in the liver by removal of one or both ethyl groups from the molecule. The resulting metabolites monoethylglycin exylidide (MEGX) and glycinxilldine (GX) still have pharmacological activity and may contribute to the CNS toxicity [12].

Clinical uses:

- As local anaesthetic agent
- Treatment of ventricular premature beats [7].
- Treatment of recurrent ventricular fibrillation [7].
- To prevent reflexes by giving 1-1.5 mg /kg 4 min. before laryngoscopy.

Adverse effects:

- Drowsiness, convulsion and coma.
- In very large doses AV conduction depression and negative inotropic effect
- On neuromuscular transmission, depress ganglionic transmission and neuromuscular transmission; enhance the action of neuromuscular blocking agent.
- Systemic toxicity from local administration causes, with increasing plasma concentration, sedation, anxiety, restlessness, tremors, parasthesia severe hypoxia and acidosis which can occur compound the drug - induced medullary depression and aggravate depressed myocardial function, so that profound cardio vascular collapse ensures toxicity appears with decreased hepatic blood flow as occurs with reduced cardiac output.

Patients and Methods

Sixty patients were selected, following the selection criteria below, divided into equal groups, each group of (20) patients:

1. Group A: control group (not given any drug) and the insertion of LMA after 5 minutes from induction.

2. Group B: given lignocaine 1 mg / kg, slow i.v. injection immediately before induction and 5 minutes before the insertion of LMA.

3. Group C given topical lignocaine spray 10% 40 mg to the larynx and posterior pharyngeal wall before induction and 5 minutes before insertion of LMA.

Selection criteria:

1- All patients were adult males, any patients below 18 years or above 45 years of age were excluded.

2- All patients were of class 1 - ASA, of average weight and height.

3- Only patients with peripheral surgery were included (relatively short operations not need muscle relaxants. upper and lower limb surgery, varicocele perianal abscess ....).

4-Any patient with temporomandibular joint dysfunction, pharyngeal obstruction and pathology like: pharyngeal abscess was excluded because it needs to open the mouth widely enough to insert the LMA.

5- Any case of full stomach was excluded from the study.

Method:

The patients were not premedicated. Induction of anaesthesia by thiopentone sodium 6 mg / kg. most of the patients needed manual assisted ventilation because of the apnea of thiopentone until return of spontaneous breathing. Then maintenance of anaesthesia by halothane 3%, l00% oxygen.

After 5 minutes from induction. Insertion of LMA size 3 were made in all cases. All patients monitored which include ECG (Lead II), noninvasive blood pressure (NIB) and pulse oximetry. Any case with difficult insertion of LMA or need laryngoscopy for insertion of LMA was excluded from the study. Observation of cough, laryngeal spasm during insertion of LMA.

Heart rate and mean blood pressure (MBP) readings taken at pre induction, after insertion of LMA and after 3 minutes from insertion of LMA.

Results

The three groups were comparable in age, weight, heart rate and mean blood pressure:

Cough and laryngeal spasm due to insertion of LMA were statistically significant in group -A- (control group) by using Z test; P value < 0.05, but cough and laryngeal spasm in group -B- (Lignocaine i.v.) is statistically not significant P value > 0.05, and in group -C- (topical lignocaine) there were no cough and no laryngeal spasm. Mean blood pressure (MBP) and heart rate (H.R) pre induction considered as the base line values.

After insertion of LMA the mean blood pressure and heart rate statistically significantly increased for a group -A- and group -B- (by using paired t. test); P value < 0.05, but in group -C-: mean blood pressure and heart rate statistically not significantly increased, P value > 0.05. By using the student's t. test.

When comparison of the increase in mean blood pressure and heart rate among the three groups, there were no significant difference between lignocaine i.v. and control groups; P value > 0.05 also there were no significant difference between topical lignocaine & control group, P value > 0.05 and no significant difference between topical and i.v. lignocaine; P value > 0.05.

Table 2, Data of patient in group –A

No	L. Spasm	Cough	Pre induction		After insertion of LMA		After 3 min.	
			Heart rate	MBP	Heart rate	MBP	Heart rate	MBP
•	+	-	90	93	62	110	85	110
•	+	-	82	95	110	113	102	108
•	-	-	95	102	116	107	110	100
•	-	+	70	83	113	99	107	102
•	-	-	85	76	100	82	90	85
•	-	-	88	80	122	90	115	85
•	+	-	105	84	81	115	86	115
•	-	+	105	84	81	115	86	115
•	+	-	92	98	106	101	90	93
•	-	-	86	91	107	85	102	85
•	-	+	80	105	97	98	97	98
•	-	-	75	97	90	110	85	107
•	-	-	85	89	102	93	98	93
•	-	-	93	86	110	97	95	95
•	-	+	99	78	125	95	115	93
•	-	-	110	75	123	82	118	82
•	-	+	91	105	115	115	110	115
•	-	-	75	93	95	104	95	104
•	-	-	84	80	99	92	95	93
•	-	-	78	95	92	109	90	110
Mean	4	5	88	89	105	100	100	99
SD	*		10	10	16	11	11	11

- The percentage of laryngeal spasm 20%, cough 25 %

The percentage of increase after insertion of LMA from the base line are:

- Mean blood pressure 12 %
- Heart rates 18.9 %
- SD= Standard deviation
- The laryngeal spasm in 3 patients ended by injection of 25 mg of succinylcholine and the other patient ended within 90 sec. After providing positive pressure ventilation. Three patient developed ventricular ectopic beats which ended spontaneously after proper positioning of the LMA.

Table 3, Data of patient in group –B

No	L.Spasm	Cough	Pre induction		After insertion of LMA		After 3 min.	
			Heart rate	MBP	Heart rate	MBP	Heart rate	MBP
•	-	-	92	84	105	90	100	93
•	-	-	72	102	85	110	92	105
•	-	-	85	105	102	100	97	92
•	-	-	83	98	90	108	95	105
•	-	-	90	110	85	103	90	100
•	-	+	97	85	110	112	105	105
•	-	-	105	90	108	95	108	95
•	-	-	97	83	95	88	90	85
•	-	-	88	92	93	95	95	92
•	-	-	93	105	98	115	100	110
•	-	+	78	107	92	110	92	115
•	-	-	81	97	85	93	90	93
•	-	-	87	93	93	91	92	90
•	-	-	85	98	100	102	95	100
•	-	-	110	103	105	107	105	107
•	-	-	94	100	97	97	97	96
•	-	-	86	92	96	102	88	101
•	-	-	102	87	112	92	110	93
•	-	-	107	93	105	106	105	102
•	-	-	100	101	115	92	108	92
Mean	0	2	92	96	98	101	98	99
SD	*		10	8	9	9	7	8

- The percentage of laryngeal spasm 0, cough 10 %
- The percentage of increase in:
- Mean blood pressure 5 %.
- Heart rate 7%.

Table 4, Data of patient in group –C

No	L.Spasm	Cough	Pre induction		After insertion of LMA		After 3 min.	
			Heart rate	MBP	Heart rate	MBP	Heart rate	MBP
•	-	-	75	82	83	86	85	90
•	-	-	87	93	96	95	95	87
•	-	-	85	94	103	93	102	92
•	-	-	92	97	98	101	98	101
•	-	-	105	110	115	102	110	96
•	-	-	90	102	85	105	85	103
•	-	-	98	105	95	103	96	105
•	-	-	102	111	105	113	105	108
•	-	-	110	120	107	112	100	101
•	-	-	70	85	88	93	90	95
•	-	-	83	92	97	95	94	93
•	-	-	115	122	104	112	102	110
•	-	-	97	108	108	102	105	103
•	-	-	102	105	95	113	92	91
•	-	-	120	104	110	106	110	105
•	-	-	85	93	77	98	80	96
•	-	-	84	101	91	106	93	104
•	-	-	92	85	99	97	97	97
•	-	-	99	92	92	98	90	85
•	-	-	105	91	110	96	108	98
Mean	0	0	95	100	98	101	97	98
SD			13	11	10	8	9	7

- There are no cough and no laryngeal spasm in this group,
- The percentage of increase in:
  1-Heart rate 3 %.
  2-mean blood pressure 1.3 %.

Discussion

Our objective in this study is to find the effective drug or route of administration of lignocaine to reduce the incidence and severity of cough, laryngeal spasm & cardiovascular response to insertion of LMA during thiopentone anesthesia. The respiratory tract is hypersensitive to stimuli arising during light thiopentone anaesthesia [(4)]. Laryngeal spasm is a forceful involuntary spasm of laryngeal musculature caused by a sensory stimulation of the superior laryngeal nerve [(3)].

Lignocaine spray in enough doses can cause adequate surface anaesthesia to the larynx and pharynx providing high level of stabilization of cell membrane of laryngeal and pharyngeal musculature and eliminating its sensitivity to stimulation due to insertion of LMA , but lignocaine i.v. can cause stabilization of cell membrane of nerves of larynx and pharynx decreasing their sensitivity to stimulation by LMA , but to a degree less than lignocaine spray depending on the dose of lignocaine i.v. (the higher the dose the higher degree of stabilization).

Conclusion and Recommendations

- Insertion of laryngeal mask requires depth of anaesthesia less than that with laryngoscopy and intubation, but a greater depth of anaesthesia than the insertion of guedel airway.
- The incidence of cough reflex and laryngeal spasm is significant in control group and less significant in lignocaine i.v. and neither cough nor spasm in topical lignocaine.
- There were no significant differences in cardio vascular response between the three groups.
- Providing good depth of anaesthesia the cardio vascular response to insertion of laryngeal mask airway will be minimal with the use of lignocaine.
- Regarding the cough & laryngeal spasm, the use of topical lignocaine spray is the best aid for insertion of LMA.

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