ORIGINAL ARTICLE

Attenuation of Rate Pulse Throughout Endotrachial Intubation Utilizing Tracheal Lidocaine

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ABSTRACT

Aim: Assess pulse rate variations throughout endotrachial intubation.
Methodologies: Reflection of pulse rate variations throughout endotrachial intubation have been applied upon 80 patients separated into two sets: Set A (controlling set): usually has been provided anesthesia involving succinylcholine and thiopentone before to intubation. Set B: Tracheal Lidocaine (2%) was given 180 second prioranesthesia induction.
Results: The fallowing phenomena have been observed; Endotrachial intubation caused a noticeable increase in the pulse rate. There was a variance in the response among the selectedsets; in set B, it will be recorded a greatlysubstantiallower magnitudes of pulse rate than the controllingset A (P less than 0.001).
Conclusion: It has been concluded from the analysis that throughout endotrachial intubation, certain patients who obtained Tracheal Lidocaine 180 seconds before anesthesia induction displayed limited pulse rate variations.
Keyword: Anesthesia, Rate of Pulse, Tracheal Lidocaine, intubation.

INTRODUCTION

With the advent of endotracheal intubation, the revolutionary changes have taken place only in the advancement of surgery contemplating for long hours with safety, but at the same time the field of anesthesiology has become quite safe for the patient, unfortunately little attention has been paid toward the harmful effects of endotracheal intubation which has got a significant contribution toward the patient mortality & morbidity[1].The sudden rise in the pulse rate observed in almost all the patients undergoing endotracheal intubation cause left ventricular failure, cerebral hemorrhage & myocardial ischemia[2].

Thus the anesthesiologists should be aware of these complications although they are usually so engrossed in the technical aspect of tracheal intubation which seems to be simple, so they pay little attention to the abnormal changes taking place in the patient at the time of this procedure.

Innervation Of The Larynx & Trachea

Nerve supply of the larynx: The upper half of the larynx is innervated with visual and secretive motor fibers by the inner laryngeal division of the upper vagus nerve branch of the laryngeal.

The lower half of the larynx is innervated with sensory & secretomotor fibers by the recurrent the vagus branch of laryngeal that innervate also all the intrinsic muscles of the larynx except the circo-thyroid muscle thatwas provided by the externally branch of laryngeal for the superior laryngeal nerve.

The sympathetic supply comes in a long the arteries.

Above the cords, the fibers run with the superior laryngeal artery from the superior cervical ganglion, while below the cords , the inferior laryngeal artery brings fibers from the middle cervical ganglion[3,4].

Nerve supply of the trachea: Recurrent vagus nerve branch of the laryngeal suppliesthe trachea with sensory &secreto-motor fibers.

Sympathetic fibers reach the trachea on the inferior thyroid artery from the middle cervical ganglion[3].

Pharmacology Of Lidocaine: Lidocaine is diethyl-amino-aceto2,6-xylidide. It is one of the local anesthetics with a partition coefficient 2.9 &PKa of 7.9 . It is 58-75% protein bound in plasma. Lidocaine has a rapid onset of action & intermediate potency & duration of action[5].

In the cardiac muscle cell, functioning by blocking fast channels of sodium in the cell membrane, it often suppresses excessive spontaneous cell membrane contraction by growing the external conductivity of potassium in the ventricular tissue. As a consequence, the diastolic threshold for ventricular stimulation raises the current needed to cause premature ventricular contracting leading to dysrhythmias inhibition induced by intelligent productivity or ectopic foci[6]..

The upper limit of safe dosage of lidocaine in adults is 200-400 mg(3mg/kg B.W.) without epinephrine & 500mg(7mg/kg) with epinephrine[6].

The usual I.V. dosage of 1-2mg/kg which is used in the management of cardiac dysrhythmias will not reach the toxic blood level[7].

Toxic symptoms occurs at blood levels above 5.3 microgram/ml & include circumoral& tongue numbness, tinnitus, visual disturbance, muscular twitching, convulsions, coma, respiratory arrest & cardiac ventricular depression [8].

PATIENTS &METHODOLOGIES

For this study, all the patients were provided an informed consent at the preoperative visit. Fourty patients of Anesthesiologist American Society (ASA) physical status Class I and Class II with normal cardiovascular function, arranged for elective surgery needing endotracheal intubation are randomly assigned into twosets;

Set A: The control set.

Set B: AssumedTracheal Lidocaine (2%)180 second before anesthesia induction.

The pulse rate responses have been measured as fallows:

1. Base line documented on the arrival in the operating room .
2. Immediately during endotrachial intubation.
3. Three hundred seconds after intubation.
4. The rate of pulse has been measured by the pulse oximeter.

Anesthesia Technique: After Pre-Oxygenation with pure $O_2$for 3 minutes using face mask, anesthesia was induced by sleeping dose of 2.5% sodium thiopentone based on the absence of eye lid response &suxamethonium in a dose of 1mg/kg I.V. Patients were ventilated with 100% O_2 , followed by endotracheal intubation using the cuffed endotracheal tube. All patients were intubated without difficulty by using the direct laryngoscope (Macintosh laryngoscope). No surgical stimulus was permitted during the course of this study. Anesthesia was maintained with inhalational agents under relaxation & controlled ventilation. The patients have beenseparated into twosets A&B,each set contained 40 patients.

In set A, the patients were intubated without drug premedication & so called the control set,while in set B the Tracheal Lidocaine (2%) 80 mg used 180 seconds before anesthesia induction**.**

RESULTS

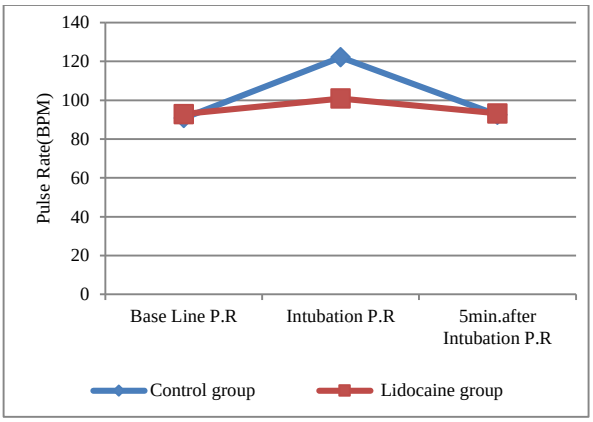
The results in table (3-1) and Fig.(3-1) shows a striking increase in pulse rate (P.R) .The pulse rate had significant increase ($P < 0.001$) immediately after endotracheal intubation compared with the base line recordings.The mean value of pulse rate (P.R) increased to 122.1BPM (31.3 BPM above the base line) of control group (group A) and the mean value of pulse rate (P.R) was 100.9 BPM (8.05 BPM above the base line) of Tracheal Lidocaine Group (group B) . It is important to mention that we have noticed a decline in the pulse rate in group B in comparison with group A above the base line reading & 5 minutes after intubation.

Table 1: The mean value of pulse rate

Categories	Mean ± SE		
	Base Line P.R	**Intubation P.R**	**5min.after Intubation P.R**
Control group(n=40)	90.800±2.887	122.100±4.108	92.450±3.429
Lidocaine group (n=40)	92.850±2.569	100.900±2.961	93.250±2.961
	ns	**	ns

** Referring to significant differences at (P lessthan 0.001). ns referring to insignificant

Fig.1: The mean value of pulse rate



DISCUSSION

Endotrachial intubation have been reported to cause significant increase in blood B.P. & heart rate[9,10].In our research, we found a substantial rise in P.R. In the reference set (A) immediately after endotrachial intubation. Cardiovascular response to tracheal intubation has been presumed to be a responsive the response of sympathetic to mechanical stimulating of trachea and larynx[10].

Because sympathetic stimulation during stress could cause angina in a conscious patient, it is likely that reflex sympathetic stimulation in an anaesthetized patient can cause myocardial ischemia by can myocardial function[10].

Various strategies were utilized to reduce the adverse cardiovascular reaction to endotracheal intubation by growing the depth of anesthesia prior to intubation. The use of alfa sympathetic blockers, beta sympathetic blockers , the use of vasodilators such as sodium nitroprusside or isosorbiddinitrate[11,12].

The efficacy of I.V. lidocaine to protect against cardiovascular reactions related with laryngoscopy & endotracheal intubation has been investigated by Abou-Madi[13],he found that 0.75 mg/kg dosage of lidocaine afford protection against hypertension responses to intubation & a lesser degree to &against cardiac dysrhythmias & tachycardia, but with the use of 1.5 mg/kg I.V. they had a more protection against the cardiovascular responses to intubation[14].

In our study, we have use Tracheal Lidocaine (2%). We have proved from our results that Tracheal Lidocaine (2%) is effective in attenuation of pulse rate during endotracheal intubation. We think that this is related to the fact that using of tracheal Lidocaine will perform anesthesia to the mucous membrane of the trachea & the larynx so this will lead to less sympathetic reflex & less increasing in pulse rate, this will lead to attenuate the cardiovascular response to the maneuver[11].

Also the mild cardiovascular depression of lidocaine shared in this phenomenon, this will lead to decrease the effect of sympathetic over stimulation by endotracheal intubation on the pulse rate during induction of anesthesia[7].

CONCLUSIONS

We convey three important information from our study to the anesthesiologist:

- The maneuver of endotracheal intubation carries a high in view of accompanying tachycardia& highly significant incidence of cardiac complications.
- Critical monitoring of pulse rate during induction & endotracheal intubation is mandatory.
- It is absolutely necessary to prevent the mentioned complications of laryngoscopy & endotracheal intubation by using Tracheal Lidocaine (2%). prior to induction of anesthesia.

RECOMMENDATION

It is advised that Tracheal Lidocaine (2 percent) be used prior to anesthesia's induction to reduce the side effects of pulse irregularities such as ischaemic cardiac failure or stroke.

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