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Effects of Intubation on heart rate: Xylocard and Esmolol

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Abstract

Background: Lignocaine is a local anaesthetic of moderate potency and duration with good penetrative power and rapid onset of action carbonated lignocaine has remarkable penetrative power, rapid onset of action a high incidence of motor block and a reduced incidence of missed segments (When used for epidural anaesthesia) when compared to hydrochloride of lignocaine. **Subjects and Methods:** Seventy five (75) patients belonging to ASA grade 1 & 2 scheduled for general surgical, orthopaedic surgical, ENT, gynaecological surgical producers were studied. **Results:** The age of the patients varied from 10 to 60 years. In our study, the heart rate in controls (Group A) before induction was 86+/-10 rose to 93+/-12 and 107+/-13 after induction and after laryngoscopy + ETI respectively. The changes seen after endotracheal intubation alone was statistically very highly significant (<0.001). In Group B, the pre induction heart rate was 94+/-17, which increased to 99+/-13, after induction.This increase not significant (P>0.05). There was an increases of 15 at laryngoscopy + ETI which was very highly significant (P<0.001). **Conclusion:** In Group C, the pre induction heart rate of 96+/-15 increased to 97+/-11 at induction and to 108+/-15 following endotracheal intubation.

Keywords: Effects of Intubation, heart rate, Xylocard and Esmolol.

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Introduction

Lignocaine was synthesized in 1943 by Lofgren and lundqvist in 1943 at A. B. Astra, Sweden. It was introduces into clinical practice by Gordh in 1948. Introduced as an antiarrhythmic agent in 1962. Chemically it is an amide of xylidinedietyl amino 2:6 dimethy 1 acetanilide. It is very stable, not decomposed by boiling, acids or alkalies and withstands repeated autoclaving. The pKa of lignocaine is 7.90. At the normal tissue pH of 7.4 approximately 75% of lignocaine exists in the charged cationic form, while 25% exists in the nonionized form. At concentration of 2 microgram per milli litre, approximately 70% of the drug is bound to the plasma proteins. Lipid solubility is 2.9.

Lignocaine is a local anaesthetic of moderate potency and duration with good penetrative power and rapid onset of action carbonated lignocaine has remarkable penetrative power, rapid onset of action a high incidence of motor block and a reduced incidence of missed segments (When used for epidural anaesthesia) when compared to hydrochloride of lignocaine. Tachyphylaxisoccus with repeated injections. Lignocaine causes vasodilation. Epinephrine prolongs the action of lignocaine and reduces its rate of systemic absorption.[1]

Lignocaine is used in the treatment of ventricular dysrhythmias after myocardial infarction or cardic surgery. It depresses the rate of rapid depolarization without any effect on slow depolarization. This shortens the duration of action potential and effective refractory period in purkinje fibres and ventricular muscle, shortening of ERP in an area of tissue damage will result in impdulse being conducted in antegrade fashion, in case of unidirectional block. Hence retrograde conduction and re-entry fails to occur and therefore dysrhythmias ceases.[2,3]

Though true hypersensitivity to lignocaine has been reported, methylparaben a derivative of benzoic acid, which is used as a preservative, being highly antigenic, is the usual cause for hypersensitivity. Other side effects like nausea, drowsiness or dizziness can occur. At higher plasma levels, tremors, convulsions, bradycardia, decreased respiration with hypoxia, and cardiovascular collapse can ensue.[4]

Esmolol hydrochloride is chemically methyl 3-{4-{2-hydroxy-2-(isopophylamino) propoxy) phyenyl} propionate Hcl.

Structurally esmolol is very similar to metaprolol. Para substitute onaromatic ring imparts cardioselectivity to esmolol as it does to metoropolol. The presence of ester group in the para position imparts metabolic lability to esmolol and is responsible for it's short duration of action. The ester is rapidly hydrolysed by erythrocyte esterases.[5]

Clinical Pharmacology studies in normal volunteers have confirmed the beta blocking activity of Esmolol showing reduction of Heart rate at rest and during exercise, and attenuation of isoproterenol induced tachycardia. Blood levels of Esmolol have been shown to correlate with extent of beta blockade. After termination of infusion, substantial recovery from beta blockade is observed in 10-20 min.

In Human electrophysiology studies Esmolol produced effects typical of a beta blocker: a decrease in the heart rate

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(R), increase I sinus cycle length, prolongation of the sinus node recovery time, prolongation of A-V interval during sinus rhythm and actual pacing and an increase in antegradewenckebach length. Esmolol in dosage of >200 mcg/kg/min. produced reduction in heart rate (R), systlic Blood pressure (P) rate pressure product (RPR) Right and left ventricular ejection fraction, and cardiac index at rest.[6] Which is similar to propranolol 4mg. same results are reproduced during exercise also.
The relative cardio selectivity of esmolol is studied in comparison with propranolol. In asthmatic patents requiring no broncho dilator therarpy in esmolol group in therapeutic dosage.

Subjects and Methods

Seventy five (75) patients belonging to ASA grade 1 & 2 scheduled for general surgical, orthopaedic surgical, ENT, gynocological surgical producers were studied. The age of the patients varied from 10 to 60 years.

Procedure
The patients were studied in groups A,B, and C.

Group "A" Patients served as control.
Group "B" in this group patients received intra-venous xylocard 1.5 mg/kg
Group "C" – This group received intravenous esmolol hydrochloride 500mcg kg-1.

Technique:-
All the patients in the groups A,B and C were induced with thiopentone sodium 5mg kg-1 and succinylcholine 1.5 mg kg-1. All the patients were pre-oxygenated for 3 minutes before induction.

In group A, laryngoscopy was performed after the fasciculation's subsided using Macintosh laryngoscope and intubation performed with a suitable endotracheal tube, anaesthesia was the maintained with 60% Nitrous oxide and 40% oxygen.

In group B, laryngoscopy was performed after one minute of administering intravenous xylocard 1.5 mg/kg. Xylocard was administered immediately after succinylcholine injected. Anaesthesia was then maintained with 60% Nitrous oxide ad 40% oxygen.

In group C, laryngoscopy and intubation was performed after three minutes of administering intravenous esmolol hydrochloride 500 mcg kg-1. Esmolol hydrochloride was given just after injection of Thiopentoe sodium anaesthesia was maintained with 60% Nitrous oxide and 40$ oxygen.

In all the three groups 0.5% Halothane was started just after intubation to prevet the patients from becoming lighter.

Monitoring devices used in the study were.
Pulse oximeter to observe changes in Heart rate
Sphygmo monometer with cuff (manually operated) to check Blood Pressure changes
Continuous ECG monitor occurrence of Arrythmias in lead 2.

Results

Table 1: AGE and SEX distribution

Age In Year	A		B		C	
	M	F	M	F	M	F
11-20	2	2	3	2	1	1
21-30	5	4	4	3	5	3
31-40	2	4	4	1	2	2
41-50	2	2	2	2	5	2
51-60	1	1	2	2	3	1
Total	12+	13=25	15+	10=25	16+	9=25

Table 2: Nature of Surgery.

Sl.No.	Nature Of Surgery	A	B	C
1	Exploratory laparotomy, Cholecystemctomy, Herniorrhaphy, G-J with vagotomy, Splenectomy.	13	4	9
2	Thoracic ad lumbar Laminectomy	2	3	2
3	Thyroidectomy Thoracotomy	6	10	8
4	Phylolithotomy Nephrectomy Vescical calculus	0	1	2
5	Abdominal and Vaginal hysterectomies	0	1	2
6	Upper limb orthopaedic Procedures.	0	2	0
7	Tonsilectomes Nasal Polypectomy	0	3	0
8	Miscellaneous	4	1	2

General surgical cases constituted 66.67% (50) of the cases selected for the study. In group "A" 88% (22) in group "B" 44% (11) and in groups "C" 68%(17) of the cases underwent general surgical procedures. About 18 (Eighteen) patients in group "A",B and "C". where hypertensive with blood pressure more than 140/90 mm Hg.
All patients belonged to ASA grade 1 and 2

Table 3: The changes in heart rate during various stages

Timing	A	B	C
Basal	78+/-9	85+/-15	86+/-15
Pre-induction	86+/-10	94+/-17	96+/-15
Just before laryngoscopy	93+/-12	99+/-13	97+/-11
LARYNGOSCOPY + ETI	107+/-13	109+/-12	108+/-15
1ST MIN AFTER ETI	106+/-10	106+/-11	106+/-15
2ND MIN AFTER ETI	108+/-10	100+/-12	103+/-15
5TH MIN AFTER ETI	102+/-11	98+/-12	99+/-14

ETI=Endotracheal intubation

Table 4: The difference in heart rate after induction and endotracheal intubation compared to pre induction levels.

Sl. No	A	B	C
Just Before Laryngoscopy	+17	+5	+1
P	>0.05	>0.05	>0.05
Remarks	N.S	N.S	N.S
Laryngoscopy + ETI	+11	+15	+12
P	<0.001	<0.001	<0.001
Remarks	V.H.S	V.H.S	V.H.S

N.S=Not Significant
V.H.S=Very Highly Significant.

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Heart rate at 5th minute after endotracheal intubation is compared with that of at laryngoscopy and end endotracheal intubation.

Table 5: Heart rate at 5th minute after endotracheal intubation is compared with that of at laryngoscopy and end endotracheal intubation

	A	B	C
Mean Of The Differece	-5+/-12	-11+/-7	-9+/-7
P	>0.05	<0.001	<0.001
REMARKS	NS	VHS	VHS

In our study, the heart rate in controls (Group A) before induction was 86+/-10 rose to 93+/-12 and 107+/-13 after induction and after laryngoscopy + ETI respectively. The changes seen after endotracheal intubation alone was statistically very highly significant (<0.001).
In Group B, the pre induction heart rate was 94+/-17, which increased to 99+/-13, after induction. This increase not significant (P>0.05). There was an increases of 15 at laryngoscopy + ETI which was very highly significant (P<0.001).
In Group C, the pre induction heart rate of 96+/-15 increased to 97+/-11 at induction and to 108+/-15 following endotracheal intubation.
The later change was very highly significant (P<0.001).
In all the three groups, the average increase of 12.67 beats / min was observed from pre induction levels following endotracheal intubation.
The increase in heart rate seen at laryngoscopy + ETI decreased after 5 minutes by 5 beats / min in groups A which was not significant (P>0.05) In group B and C the decrease was 11+/-7 and 9+/-7 respectively(beats/min) after 5 minutes. These changes were very highly significant (P<0.001).

Discussion

Hypoxia and hypercarbia have been implicated as causes for increase in pylse and blood pressure (king B.D., - 1951, Katz R.L., - 1970). In our study hypoxia and hypercarbia was avoided by controlled ventilation technique and by adequate pre oxygenation of high risk group. The pressure due to laryngoscopy ad intubation occurs 30-40 seconds after laryngoscopy (Steeling R.K.1977. The presser response due to CO2 retention is delayed and less abrupt.[7]
A significant rise in heart rate was observed during laryngoscopy, which was similar to the finding of stilting R.K. (1976 and 1977) and Den linger (1974). The heart rate increase was similar in all the groups A,B and C. it increase by an average of 4 beats/minute following induction. The present study was done to measure the cardiovascular response to laryngotracheal stimulation and to find a simple, safe and effective method for diminishing the cardiovascular reaction.[8]
In our study we used IV lignocaine and IV esmolol to attenceate the response. Lignocaine was used in dose of 1.5 mg/kg body weight and Esmolol 500mcg/kg. Bromage (1961) reports that the concentration of lignocaine will rise to 10 mcg/ml when 6 mg/kg given to patient In man toxic effect occur in conscious subjects at a plasma level of 5 mcg/ml and in anaesthetised patients at 10 mcg/ml when circulatory depression become obvious. In our study, the dose of ligncaine used was therefore considered to be within safe limits. No untoward toxic response occurred in any case in the dosage used. No untoward toxic response occurred in any case in the dosage used. Regarding esmolol toxicity occurred at the dosage of 5000-6250 mg/kg/min. Over 1-2 min infusion resulted in Brady cardio, Hypotension, loss of consciousness. In our study dosage is too less to cause any toxic untoward responses. Promethazine and ephedrine was used since they reduced the anaesthetic requirements (miller A Forbear 1970) and sedative hypnotic and analgesics. They are not known to attenuate or augment the hemodynamic response to laryngoscope (Devault 1960) Atropine was totally avoided in our study.[9]
Hypoxia and hypercarbia have been implicated as causes for increase in pulse and blood pressure (King B.D.-1951. Katz R.L.-1970). In our study hypoxia and hypercarbia was avoided by controlled ventilation technique and by adequate pre oxygenation of high risk group. The pressure due to larygoscopy and intubation occurs 30-40 seconds after larygoscopy (Stoelting R.K.1977). The presser response due to CO2 retention is delayed and less abrupt.[10]

Conclusion

There was an increase in the heart rate by an average of 4 beats/min following induction and intubation. However it was reduced by 5 minutes in group B and reached awake levels in group C while it was persistent in Group A.

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