Original Article

Comparative Study of Attenuation of effects of Intubation on Blood Pressure Using IV Xylocard and IV Beta Blocker (Esmolol)

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Abstract

Background: The occurrence of cardio vascular reactions to laryngoscopy and tracheal intubation has attracted the attention of anaesthesiologists and methods to avoid these potentially harmful responses even though transitory have been sought, particularly in critically ill patients, hypertensive patients. Tracheal intubation under light general anaesthesia is consistently accompanied by a pressor response, tachycardia and in some instances by cardiac arrhythmias. This pressor response, which was recognised early as 1951 is due to sympathetic reflex provoked by stimulation of the epipharynx and laryngopharynx. **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. **Results:** The age of the patients varied from 10 to 60 years. The MAP in group A decreased after induction. There was a highly significant raise to 109+/-11 mm Hg during laryngoscopy and intubation. This decreased to 105+/-9mm Hg after five minutes which is not significant. **Conclusion:** These responses are transitory, variable and are much more marked in a hypertensive patient than in the normotensive patient. Once the laryngoscopy and endotracheal intubation is completed, the increase in pulse and blood pressure subside, but the dysrhythmia persists for more than 2-3 minutes.

Keywords: Intubation, Blood Pressure, Xylocard and IV Beta blocker

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Introduction

Induction of anaesthesia is recognised as a hazardous phase in the management of the patient during the operative procedure. The occurrence of cardio vascular reactions to laryngoscopy and tracheal intubation has attracted the attention of anaesthesiologists and methods to avoid these potentially harmful responses even though transitory have been sought, particularly in critically ill patients, hypertensive patients. Tracheal intubation under light general anaesthesia is consistently accompanied by a pressor response, tachycardia and in some instances by cardiac arrhythmias.[1] This pressor response, which was recognised early as 1951 is due to sympathetic reflex provoked by stimulation of the epipharynx and laryngopharynx. This is hazardous to those with hypertension, myocardial or cerebrovascular insufficiency in whom subarchnoid haemorrhage, myocardial ischemia, left ventricular failure or cardiac arrhythmias can occur. Several techniques have been used with varying degrees of success to obtund this pressor response to laryngoscopy and tracheal intubation. Including intravenous or topical lignocaine, thoracic epidural analgesia, by deepening general anaesthesia, intravenous opiods, alpha and beta adrenergic receptor blocker, and peripheral vasodilators.[2] But none of the pharmacological approaches has proved entirely satisfactory, as the reflex is not completely blocked. The agents used to block the response may themselves produce undesirable side effect. As stretching of the tissues of the epipharynx and laryngopharynx presumably trigger the reflex response, minimising stretching of tissues as during blind nasal intubation sounds more theoretical.[3,4]

The aim of the present study was to observe the occurrence of any increase in Blood Pressure, increase in Heart rate and / or cardiac arrhythmias during laryngoscopy and tracheal intubation. An attempt was made to minimise these responses by either intravenous 2% lignocaine hydrochloride (in dose of 1.5 mg Kg-1 body weight) because of its well established central depressant and antiarrhythmic effects or with intravenous esmolol hydrochloride (in a dose of 500 mcg/Kg body weight) which is a relatively new cardio selective intravenous beta blocker which has rapid onest of action exerts a peak hemodynamic effects within minutes and has a short elimination half life of 9.2 min, consequently it should prove ideal for control of the short lived haemodyamic sequele associated with laryngoscopy and tracheal intubation.

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.

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Pre Anaesthetic Assesment

Pre anaesthetic assessment was done on the day prior to surgery. The patients are posted for surgery for general surgical, orhpaedic, gynaecological, ENT problems. They had no associated diseases.

A routine pre-anaesthetic examination was conducted assessing:-

1. General condition of the patient
2. Nutritional status and weight of the patient.
3. A detailed examination of the cardio vascular system including recording of blood pressure.
4. An examination of the respiratory system.
5. Other associated diseases.

The following investigations were done:-

1. Routine Haemogram, including Haemoglobin estimation, total count, differential count, ESR.
2. Urine examination: Albumin, Sugar, and Microscopic Examination.
3. Blood sugar (Random)
4. Blood urea and serum creatinine
5. Electro cardiogram (EKG) and Chest X-ray was done in patients over 40 year of age and those patients who had cardiovascular and respiratory problems.

Premedication:-

All the patients included in the study were given anxiolytics Alprazolom 2.5mg orally on the previous night of the surgery. Thirty minutes Before induction each patient received pethedine 1-2 mg/kg and promethazine 1mg/kg intramuscularly.

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.

Results

In the present series seventy five patients hospitalised for undergoing various surgical procedures were selected for the attenuation of hemodynamic changes during laryngoscopy and oro-endotracheal intubation.

Table 1: Gender Distribution

Groups	Agents For Attenuation	No.Of Patients	Male	Female
A	Control	25	12	13
B	IV Xylocard 1.5 mg/kg	25	15	10
C	IV Esmolol HCL 500 mcg/kg	25	16	09
Total		75	43	32

As seen in [Table 1], of the 75 patients 43 (57.3%) were males and 32 (42.65%) were females. In Group "A" there were 12 (48%) Males and 13 (52%) females. Group "B" had 15 (60%) Males and 10(40%) Females. While group "C" had 16(54%) males and 9(36%) Females.

Table 2: The age distribution.

Age In Years	A	B	C	Total	%
11-20	4	5	2	11	14.64%
21-30	9	7	8	24	32%
31-40	6	5	4	15	20%
41-50	4	4	7	15	20%
51-60	2	4	4	10	13.36%
Total	25	25	25	75	100%

The youngest patient included in this study was 11 years boy, oldest was fifty nine (59) years. Most of the patients i.e. 72% belonged to the age group 11-20 years while 14.64% belong to age group 11 to 20 years while only 13.36% were above 50 years.

Table 3: The systolic B.P. during various time intervals

Timing	A	B	C
Basal	119+/-12	127+/-15	124+/-12
Preinduction	116+/-9	122+/-13	124+/-12
Just Before Laryngoscopy	102+/-10	108+/-21	109+/-13
Laryngoscopy + ETI	149+/-19	144+/-22	129+/-20
1st minute after ETI	146+/-16	140+/-21	126+/-19
2nd minute after ETI	143+/-16	133+/-18	122+/-18
5th minute after ETI	142+/16	132+/-19	120+/-20

Table 4: Systolic BP before induction and after induction and intubation

Sl. No		A	B	C
Just Before Laryncoscopy		-14	-14	-15
P		>0.05	>0.05	>0.5
Remarks		NS	NS	NS
Laryncoscopy + ETI		+33	+36	+5
P		<0.001	<0.001	<0.005
Remarks		VHS	VHS	NS

Table 5: A comparison between the systolic BP at intubation and five minutes

	A	B	C
Mean Of The Difference	-7+/-5	-14+/-6	-9+/-9
P	>0.05	<0.001	<0.001
Remarks	NS	VHS	VHS

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In group A the systolic pressure decreased from a preinduction value of 116+/-9 to 102+/-10. This was followed by an increase to 149+/-17 following intubation which is highly significant. There was no significant decrease in BP at the end of the five minutes (P>0.005).

In group B, the decrease in systolic blood pressure following induction was 14mm Hg. There is no significant increase during laryngoscopy and ETI. The fall in BP at the end of five minutes following intubation was highly significant (P<0.001).

The fall in systolic blood pressure in group C following induction was 15 mm Hg. The increase in blood pressure from the induction level was 20 mm Hg, at the time of laryngoscopy, which was highly significant. At the end of five minutes the blood pressure decreased by an average of 9 mm Hg which is once again statistically highly significant (P<0.001)

Table 6: Mean arterial Pressure

Timing	A	B	C
Basal	94+/-6	96+/-7	95+/-6
Preinduction	92+/-4	95+/-8	96+/-5
Just before laryngoscopy	86+/-6	87+/-10	89+/-6
Laryngoscopy + ETI	109+/-11	106+/-13	97+/-10
1st minute after ETI	107+/-11	103+/-13	95+/-10
2nd minute after ETI	105+/-9	99+/-10	93+/-11
5th minute after ETI	105+/-9	98+/-10	93+/-10

Table 7: The changes in MAP following induction and intubation as compared to pre induction values

Sl. No	A	B	C
Just Before Laryncoscopy	-6	-8	-7
P	>0.05	>0.05	>0.5
Remarks	NS	NS	NS
Laryncoscopy + ETI	+17	+11	+1
P	<0.001	<0.001	>0.05
Remarks	VHS	VHS	NS

Table 8: The increase in MAP at intubation as compared to that following induction

	A	B	C
Increase IN mm Hg	+23	+19	+8
P	<0.001	<0.001	<0.001
Remarks	VHS	VHS	VHS

Table 9: The decrease in MAP after five minutes of intubation as compared to that following endotracheal intubation

	A	B	C
Mean of the Difference	-4+/-5	-8+/-6	-4+/-4
P	>0.05	<0.001	<0.01
Remarks	NS	VHS	NS

The MAP in group A decreased after induction. There was a highly significant raise to 109+/-11 mm Hg during laryngoscopy and intubation. This decreased to 105+/-9mm Hg after five minutes which is not significant.

In Group B a'fall in MAP , similar to that of group A occurred on induction. The MAP rose to 106+/-12 mm Hg at laryngoscopy which is highly significant. At the end of five minutes the MAP was 98+/- 10mm Hg. This decrease is statistically very highly significant. In group C MAP decreased from 96+/5mm Hg (preinduction) to 89+/-6 mm Hg at induction. This increased to 97+/-10 mm Hg following intubation, a significant change. The MAP decreased significantly to level of 93+/-10 after five minutes.

Table 10: The variations in RPP at various time intervals

Timing	A	B	C
Basal	9720+/-2480	10799+/-2068	10675+/-2119
Preinduction	9953+/-1477	11414+/-2063	11972+/-2468
Just before laryngoscopy	9426+/-1634	10608+/-2313	10643+/-1894
LARYNGOSCOPY + ETI	1590+/-2744	15719+/-2599	14131+/-4196
1ST MINUTE AFTER ETI	15443+/-2476	14784+/-2639	13524+/-4114
2ND MINUTE AFTER ETI	14645+/-2204	13329+/-2255	12704+/-3774
5TH MINUTE AFTER ETI	14122+/-2335	12838+/-2382	12061+/-3891

Table 11: The changes in RPP occurring at induction and endotracheal intubation

Sl. No	A	B	C
Just Before Laryncoscopy	-527	-806	-1329
P	>0.05	>0.05	>0.5
Remarks	NS	NS	NS
Laryncoscopy + ETI	+5957	+4305	+2157
P	<0.001	<0.001	>0.05
Remarks	VHS	VHS	NS

Table 12: the increase in RPP following intubation when compared to RPP following induction

	A	B	C
Increase in mm Hg	+6484	+5111	+3488
P	<0.001	<0.001	<0.001
Remarks	VHS	VHS	VHS

Discussion

The haemodynamic responses to laryngeal and tracheal stimulation in anaesthetised human are tachycardia, a rise in blood pressure and arrhythmias. These facts are derived from studies during different forms of anaesthesia (Raid and Brace – 1940, king Harris and Griefenstin 1950, Noda and Higachi 1964) and are interpreted as a result of reflex sympathoadrenal stimulation.[5]

These responses are transitory, variable and are much more marked in a hypertensive patient than in the normotensive patient. Once the laryngoscopy and endotracheal intubation is completed, the increase in pulse and blood pressure subside, but the dysrhythmia persists for more than 2-3 minutes.

The sympathoadrenal response may be hazardous as it compromises the ventricular performance (Giles R.W.1982) lead to myocardial ischemia, pulmonary oedema, cerebral haemorrhage (Fox E.J. 1977) and ventricular arrhythmias (Pry-Roberts, 1971). The complications are more likely in the presence of coronary artery disease 9Larence W Roy – 1979). Cerebral atheroma or hypertension (Prys-Roberts, 1971).[6]

The other factors contributing to the presser response are (a) anxiety, (b) Atropine premedication (c) Reflex baroreceptor effect upon fall in the arterial pressure after induction with Thiopentone (d) Hypoxia € Hypercarbia and (f) Cough

Attempts to attenuate the presser response have been made using various approaches. The response may be diminished or modified locally, centrally or peripherally. Different workers have used various modes of attenuation, like topical

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anaesthesia of phrynx and larynx (Wycoff,C,C-1960 ShujiDohi - 1982) i.v. lignocaine (Stoeliting R.K – 1978, Abou-Madi-1977). Adrenergic blocking drugs (Mikawa K. 1990), deep general anaesthesia (king B.D. – 1951, Paul R.Knight 1980) vasodilators 9Stoelting R.K. 1979), and non-depolarizing muscle relaxants (Singhal - 1989).[7]

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.

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 mceg/ml when circulatory depression become obvious. In our study, the dose of lignocaine used was therefore considered to be within safe limits. No untoward toxic response occurred in any case in dosage used. Regarding Esmolol toxicity occurred at the dosage of 5000-6250 mg/kg/min. over 1-2 min infusion resulted in Brady cardia, 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 Forber1970) and sedtivelypnotic and analgesics. They are not known to attenuate or augment the hemodynamic response to laryngoscopy (Devault 1960) Atropine was totally avoided in our study.[8]

Murthy S, James D, Laddu (1986) observed after intubation the peak RPP was lowered in esmolol group than controls, As the RPP is a reliable indicator of Myocardial oxygen demand, attenuation of intubation induced increase in RPP with esmolol supports the cardio protective effect of such treatment.

Mean arterial pressure changes in our study was similar to that of systolic blood pressure. It increased from awake levels by 17 mm Hg, in group A and by 11 and 1 mm Hg, in group B and C respectively following intubation. Curran.J.(1980) reported that MAP increased to 26.5 mm Hg in response to intubation in their control group. James F.Hamill (1981) observed that I.V. esmolol 500 mcg/kg is more effective that IV lignocaine 1.5mg/kg in reducing the MAP raise following intubation. Thus observations in our study compares with those of James F.Hamill.[9]

The Rate Pressure Product (RPP) correlates with myocardial oxygen consumption and bears fairly constant relationship to the onset of angina pectoris in any patient with ischemic heart disease. If the RPP for the development of angina or ischaemic chages in the E.C.G is known for a particular patient, the anaestheisiologist should maintain a lower R.P.P during the perioperative period. In the absence of specific information it is desirable to keep the R.P.P. at less than 15,000.

Lawrence Roy et al. (1979) noted that R.P.P in excess of 11,000 in patients with coronary artery disease was associated with 38% incidence of myocardial ishaemia. Bedford (1980) had got peak R.P.P values of 20,792+/-871 in mildly hypertensive patients. In our study the peak mean R.P.P was 15910+/-2744, 157129+/-2599, 14131+/-4196 in

group A,B and C respectively. Also the R.P.P returned to basal levels by 5 minutes in group C while it was nearly so in group B. In group A, R.P.P remained persistently high even after 5 minutes. Therefore the values suggest that there was a Better attenuation in Group C when compared to B. Our values are comparable to other techniques used. The R.P.P of 13286 mm Hg. Beat/min with triemthaphan (N.Saitoh - 1991), 17916 mm Hg. Beat/mm with hydrallazine and 15868 mm Hg. Beat/min with buprenorphine, 11914 mm Hg. Beats/min with dilitiazem (K.Mikawa - 1990) and over 11000 mm Hg. Beats/min with labetalol (Fishler.M - 1985).

The effect of endotracheal intubation on ECG was reported by Reid and Breace (1940). They suggested that the changes are reflex in origin. In our study all the 75 patients developed sinus tachycardia following induction and endortracheal intubation. Though the R.P.P in all the groups exceeded 14,000 mm Hg beat/min, no S-T segment or T wave changes were observed.

Burstien (1951) reported ECG changes in 68% of cases. They reported ST-T depression after large doses 0of thiopentone and was considered to be due to direct myocardial depression or reducedcoronary blood flow. They suggested that insufficient depth of anaesthesia, prolonged laryngoscopy, numerous attempts at intubation, respiratory obstruction before intubation leading to accumulation of CO2 of tracheal irritation have been suggested as cause for arrhythmias.[8]

Abou-Maid (1977) reports that a blood concentration of above 2 micro gram/ml are required for an antiarrhythmic effect. Stoelting (1977) observed blood level of 2 microgram / ml after a lignocaine spray of 2 mg/kg body weight. Therefore a direct myocardial depression in patients receiving a spray of4% lignocaine in the dose of 2 mg/kg body weight is unlikely. Oscar Viegas (1975) also concluded that a plasma concentration of 2-5 m8icrogram / ml of lignocaine is necessary for antiarrhythmic effect.

Nair G.L. (1985) reported that miaximum incidence of arrhythmias occurred in the thiopentone – relaxant technique which decreased by deepening the plane of anaesthesia using ether. Sharma U.C.(1984) reported that the commonest E.C.G finding following endortracheal intubation is tachycardia, which is comparable to our study. Haranth K.Baba (1974) opined That the E.C.G changes observed at intubation was due to a combination of effects of the inducing agent earlier and the effect or passing end tracheal tube later.[10]

Abou-Madi (1977) reported that a dose of 0.75 mg/kg of intravenous lignocaine was insufficient as antiarrhythmic while a dose of 1.5 mg/kg of i.v. lignocaine afforded complete protection against arrhythmias and also tachycardia, is similar to our finding of decreased tachycardia and absence of arrhythmias in group B.

The absence of arrhythmis and S.T segment changes in our study could be because of adequate level of anaesthesia, absence of hypoxia or hyupercarbia and rapid intubation and cardio protective antiarrythmic effect of both drugs.

The hypertensive patients in group B and in Group C did not differ from the other in a significant manner.

It was concluded in the study of attenuation of haemodynamics at larygoscopy and endotracheal intubation, that Esmolol hydrochloride was effective in attenuating the

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presser responses observed from the data. In the group where iv lingocaine was used, duration of tachycardia, hypertension and arrhythmias was reduced when compared to control. Intravenous Esmolol (500 mcg/kg) was found to be better in reducing the magnitude and duration of presser response.

Conclusion

In conclusion cardiovascular reaction of laryngoscopy and endotracheal intubation are potentially harmful and methods to obviate these, have been sought, particularly in critically ill patients. IV Esmolol was found to be the best, in attenuating the magnitude and duration of the presser response. IV lignocaine was found to be less effective in diminising magnitude and duration of the presser response when compared to IV esmolol.

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