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Attenuation of Heaemodynamic Responses During Laryngoscopy and Endotracheal Intubation with Intraoral Glyceryl Trinitate Spray and Intravenous Lignocaine, a Comparative Study

KEYWORDS: Oral NTG spray, IV xylocaine, hemodynamic response attenuation

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ABSTRACT *To observe the cardiovascular responses during larygoscopy and intubation and to compare the efficacy of Glyceryl Trinatrate spray(400micro grams 1 minute before induction) and intravenous lignocaine (1.5 mg/kg, 2 minutes intubation) prior to laryngoscopoy and intubation in 75 patients belonging to ASA grade I and II and aged between 20-60 years, under going various surgical procedures. Experimental studies reveals that there was good hemodynamic response attenuation both with oral NTG spray and IV lignocaine when compared with control group and also much more attenuation with NTG spray when compared with IV lignocaine*

INTRODUCTION

The present study consist of 75 pateints aged about 20-60 years scheduled for general surgery orthopedic and neuro surgery,on whom attenuation of hemodynamic response following laryngoscopy and endotracheal intubation was attempted using oral spray of NTG and IV xylocaine.

METERIAL AND METHODS

To our experimental study we had taken 75 paients aged about 20-60 years of ASA GRADE 1&2 they were divided into 3 groups of 25 each

GROUP 1:control
GROUP 2:patients who received glyceryl trinitrare intra oral spray 1 minute before induction
GROUP 3:patients who received intravenous lingno caine.2 minutes after intubation

All patients were premedicated with oral diazepam 0.2mg/kg body weight on the night before surgery.on the day of suregy patients were premedicated with glycopyrrolate 0.2 mg, tramadol hydrochloride 100 mg,ondansetron hydrochloride 4mg midazolom 2mg intravenously 30 minutues before the expected time of induction,group1 was used as the control .group2 patients received glyceryl trinitrate oral spray, one metered doses(400micro grams)1 minute before induction. Group 3 paients received intravenous lingnocaine(1.5mg/kg)2 minutes before intubation.

on arrival to the operation theatre the pluse rate, the systolic and diastolic blood pressure were recorded in all patients.

After preoxygenation for 3 min, anaesthesia was induced with thiopentone (2.5 solution) 5mg/kg in order to obtund the eyelash reflex, followed by 66% nitrous oxide and 33% oxygen .vecuronium bromide 0.1mg/kg was used to facilitate intubation. Laryngoscopy and intubation were performed after the onset of apnoea using a Macintosh laryngoscope and a lubricated appropriate size cuffed endotracheal tube was passed. The laryngoscopy was completed in 15-30 secs any patient who strained or required a second attempt intubation was excluded from the study. The cuff inflated and the tube was connected to boyle's machine through a closed circuit . no analgesic drugs or volatile agents were given and surgical stimulation was not allowed till five minutes after the inflation of the cuff of the endotracheal tube .anaesthesia was maintained with nitrous oxide oxygen (2:1), narcotic and relaxant with IPPV. Residual effect of muscle relaxant was reversed at the end of the surgery with neostigmine 0.05mg/kg, atropine 0.02mg/kg was administered to neutralize the muscarinic effect .

The following five parameters viz **/1.pulse, rate,2. systolic bloodpressure,3. diastolic blood pressure,4 .mean arterial pressure and5. rate pressure product,** were computed at the following instances for all the three groups : **1.pre-induction,2. during larngoscopy (zero).**

3. 1 minute after larngoscopy.4. 3 minutes after laryngoscopy 5 .5 minutes after laryngoscopy.

Blood pressure was recorded with the help of a mercury manometer by auscultation the systolic pressure (SAP) was taken at the first sound and the diastolic pressure (DAP) at the first definite change in sound. Pulse rate was recorded manually by the palpatory method .the mean arterial pressure (MAP)

Was calculated by the formula.

MAP = DAP+ SAP-DAP/3

Rate pressure product was calculated by multiplying pulse rate with systolic arterial pressure

OBSERVATIOS;

MEAN PULSE RATE

Table 1i: mean pulse rate in various group at different time intervals.

Time interval	Group-1	Group-2	Group-3
Pre-operative	80.08+- 4.44	82.84+-5.38	85.72+-6.43
Pre-induction	80.92+-4.15	83.44+-4.84	86.40+-6.86
During laryngoscopy	102.24+-4.12	100.96+-6.43	87.20+-6.63
1st min after laryngoscopy and ETI	108.28+-3.93	103.64+-4.36	86.92+-6.94
3rd min after laryngoscopy and ETI	107.52+-4.23	101.56+-5.57	88.80+-6.83
5th min after laryngoscopy and ETI	101.08+-6.26	97.20+-4.27	89.80+-6.58

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Table 2 : inter group comparison of changes in pulse rate.

Time interval	Group 1&2	Group 1&3	Group 2&3
Pre-operative	P>0.05	P>0.05	P>0.05
pre-inductive	P>0.05	P>0.05	P>0.05
During laryngoscopy	P>0.05	P<0.001	P<0.001
1st min after laryngoscopy and ETI	P<0.001	P<0.001	P<0.001
3rd min after laryngoscopy and ETI	P<0.001	P<0.001	P<0.001
5th min after laryngoscopy and ETI	P<0.05	P<0.001	P<0.001

NOTE: p<0.05 significant P<0.001 highly significant p>0.05 not significant

Pulse rate

the mean pulse rate was increased in all three groups.

The highest increased was seen in group1 (control) and group2 (nitroglycerine).the least increase was seen in group3(lingnocaine)

Systolic Blood Pressure

Table 3 : mean systolic blood pressure in various group at different time intervals .

Time interval	Gropu1	Group2	Group3
Pre-operative	118.72+-3.40	120.84+-3.00	121.72+-4.09
Pre-induction	122.16+-3.60	118.96+-1.81	128.40+-6.70
During laryngoscopy	134.24+-4.87	98.52+-5.66	115.88+-5.54
1st min after laryngoscopy	145.84+-6.58	94.40+-5.22	113.80+-5.62
3rd min after laryngoscopy	135.84+-3.60	93.68+-4.60	115.04+-5.13
5th min after laryngoscopy	127.36+-4.02	96.28+-5.25	116.68+-5.31

Table 4 : inter group comparision of changes in systolic blood pressure.

Time interval	Group 1&2	Group 1&3	Group 2& 3
Pre-operative	P>0.05	P>0.05	P>0.05
pre-induction	P<0.001	p>0.05	p>0.05
During laryngoscopy	P<0.001	P<0.001	P>0.005
1st min after laryngoscope and ETI	P<0.001	P<0.001	P>0.005
3rd min after laryngoscope and ETI	P<0.001	P<0.001	P>0.005
5th min after laryngoscope and ETI	P<0.001	P<0.001	P>0.005

NOTE: P<0.05 significant P<0.001 highly significant p>0.05 not significant

the mean systolic blood pressure was as follows in all the groups

the highest increase was in group 1(control)

the least increase was in group3(lignocaine) followed by decrese in group2(nitroglycerine).

During the 5th min after laryngoscope and ETI there was decrease in mean systolic blood pressure in group 2&3 when comparde to group1.

Diastolic blood pressure

Table 5 : mean diastolic blood pressure in various group at different time intervals.

Time interval	Group1	Group2	Group3
Pre-operative	81.92+-5.17	86.16+-6.27	83.40+-6.15
Pre-induction during laryngoscopy	84.04+-6.05	82.52+-4.92	91.48+-6.94
1st min after laryngoscopy and ETI	101.84+-4.35	72.12+-6.68	78.00+-7.38
3rd min after laryngoscopy and ETI	101.84+-4.35	72.60+-5.30	77.72+-6.70
5th min after laryngoscopy and ETI	88.96+-5.07	72.36+-6.52	78.76+-7.00

Table 6 : inter group comparisions of changes in diastolic blood pressure.

Time interval	Group1	Group2	Group3
Pre-operative	P>0.05	P>0.05	P>0.05
Pre-induction during laryngoscopy	p>0.05	P>0.05	P>0.05
During laryngoscope	P<0.001	P<0.001	P>0.05
1st min after laryngoscopy and ETI	P<0.001	P<0.001	P>0.05
3rd min after laryngoscopy and ETI	P<0.001	P<0.001	P>0.05
5th min afterlaryngoscopy and ETI	P<0.001	P<0.001	P>0.05

NOTE: p<0.05 significant

P<0.001 highly significant p>0.05 not significant

the mean diastolic blood pressure was as follows in all the three groups

the highest increase was in group1(control)

the highest decrease was in group2(nitroglycerine)followed by decrease in group3(lignocaine)

during the 5th min after laryngoscope and ETI there was decrease in mean diastolic blood pressure in group 2 and 3 when compared to group1.

Mean arterial blood pressure

Table 7 : mean arterial blood pressure in various groups at different time intervals.

Time interval	Group1	Group2	Group3
Pre-operative	94.22+-3.36	97.63+-4.35	96.00+-4.72
Pre-induction	97.11+-4.14	94.76+-3.51	103.65+-5.97
During laryngoscope	111.54+-5.47	81.37+-5.17	91.42+-5.37
1st min after laryngoscopy and ETI	117.95+-3.83	79.74+-5.72	89.84+-4.26
3rd min after laryngoscopy and ETI	112.92+-3.01	79.87+-4.13	90.57+-5.10
5th min after laryngoscopy and ETI	101.51+-4.01	80.42+-5.75	91.43+-5.30

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Table 8: intra group comparision in mean arterial blood pressure.

Time interval	Group 1&2	Group1 &3	Group 1 &3
Pre-operative	P>0.05	P>0.05	P>0.05
Pre-induction	p>0.05	p>0.05	P>0.05
During laryngo-scope	P<0.001	P<0.001	P>0.05
1st min after laryn-goscopy and ETI	P<0.001	P<0.001	P>0.05
3rd min after laryn-goscopy and ETI	P<0.001	P<0.001	P>0.05
5th min after laryn-goscopy and ETI	P<0.001	P<0.001	P>0.05

The Mean arterial blood pressure changes are as follows

The heighest increase was seen in group I

The heighest decrease was een in group II(nitro glycerine) followed by decrease n group III

During the 5th min after laryngoscopy and ETT there was decrease in mean arterial pressure in groupie when compared to grop I.

Rate pressure product

Table 8 : mean rate pressure product in various group at different time intervals .

Time interval	Group1	Group2	Group3
Pre-operative	9508.88+-623.03	10012.76+-723.20	10432.56+-845.68
Pre-induction	9885.60+-587.37	9926.40+-603.02	1109028+-1016.67
During laryn-goscope	13.722.16+-692.23	9948.08+-875.22	10102.88+-897.49
1st min after laryngoscopy and ETI	15793.20+-940.25	9776.40+-558.02	9888.32+-912.32
3rd min after laryngoscopy and ETI	14606.80+-719.20	9512.08+-673.94	10226.40+-1020.75
5th min after laryngoscopy and ETI	12879.04+-974.06	9354.08+-588.59	10480.64+-931.78

Table 10: intra group comparision of changes in mean rate pressure product.

Time interval	Group 1&2	Group1&3	Group 2 &3
Pre-operative	P>0.05	P>0.05	P>0.05
Pre-induction	p>0.05	p>0.05	P>0.05
During laryngoscope	P<0.001	P<0.001	P>0.05
1st min after laryngos-copy and ETI	P<0.001	P<0.001	P>0.05
3rd min after laryngos-copy and ETI	P<0.001	P<0.001	P>0.05
5th min after laryngos-copy and ETI	P<0.001	P<0.001	

HEAMODYNAMIC RESPONSE

In group 1 (control)the increase in mean pulse rate ,systolic, diastolic blood pressure, mean arterial pressure product that occurred at laryngoscopy and

intubation, remained persistently above the preoperative values even after 5 minutes.

In group 2 (glyceryl trinitrate oral spray) the mean pulse rate remained above the pre- operative values even at the end of 5 minutes, after laryngoscopy and ETI. the systolic blood pressure ,diastolic blood pressure, mean arterial pressure and rate pressure product remained below the pre-operative value even at the end of 5 minutes, after laryngoscopy and ETI.

In group 3 (intravenous lingnocaine) the mean pulse rate remained above the pre-operative levels even at the end of 5 minutes, after laryngoscopy and ETI, the systolic blood pressure ,diastolic blood pressure and mean arterial pressure remained below the pre-operative values even at the end of 5 minutes, after laryngoscopy and ETI ,except for the rate pressure product the value remained below the pre-operative levels at the end of 3 minutes, after laryngoscopy and ETI.

Discussion

The effect of laryngoscopy and tracheal intubation on the cardiovascular system can be over looked because the anesthesiologist may become engrossed only in the technical aspects of intubation . laryngoscopy and tracheal intubation are associated with a rise in heart rate, arterial pressure and dysrhythmia (king et al.,1951, de vault et al., 1960,wycoff 1960,takeshima et al, 1964,forbes and dally 1970,prys Roberts etal., 1971,Derbyshire et al.,1983 ,low et al., 1986, forbers and dally 1970). These changes were interrupted as a results of reflux sympathoadrenal stimulation (king etal.,1951,forbes and dally 1970), rather than a 'vago-vagal' phenomenon, as suggested by ried and brace in 1940.

The predominant sympathoadrenal response in man is tachycardia and hypertension, the later being due to a increased cardiac output rather than increased sympathetic vascular resistance and is associated with a transient rise in CVP (Corbett et.,al.,1969).

These responses are usually transitory, variable and unpredictable. These are much more marked in a hypertensive patient than in the normotensive patient. In hypertensive patients there is an exaggerated response to many forms of stress, in the conscious as well as in the anaesthetised state. The frequency and duration of this response in the normotensive are less certain . once the laryngoscope and endotracheal tube are in position and the laryngoscope withdrawn , the increase in pulse rate and blood pressure subside but the dysrhthmia persists for more than 2-3 minutes.

The haemodynamic changes are not of much consequences in mormotensive patients but may be hazardous in patients with medical problems like hypertension, ischaemic heart disease and cerebrovascular diease where circulation is already jeopardized. The sympatho adrenal response may be hazardous as it increases the work load of myocardium and may result in left ventricular failure(masson 1964),myocardial ischaemia,pulmonary edema (E. J. FOX 1977).ventricular arthythmias (prys Roberts 1971).cerebral hemorrahage has also been reported. Convulsion may be precipitated in a preeclamptic patient.

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The other factors contributing to pressor response are suggested to be a. anxiety, b. atropine premedication, c.reflex baroreceptor effect upon fall in the arterial pressure after induction with thiopentone, d.hypoxia. e.hypercarbia and f.cough.

Many attempts have been made to attenuate the pressor response by 1.deep anesthesia (king et al.,1951) 2. Narcotic agents like buprenorphine (khan and kamal 1989). Fentanyl (black and kay 1984), sufentanil (kay and Nolan 1987)3. I.V. lingnocaine and lignocaine spray (stoelting1979). 4. Beta adrenergic blockers like metaprolol (coleman and Jordan 1980), esmolol (vacevic et ., al., 1992), 5.vasodilator drugs like sodium nitroprusside (stoelting 1979). Hydralazine (davies and Cronin 1981)

6. topical and intranasal nitroglycerine (karma et al., 1986), (fassoulaki and kaniaris 1983),7. Hypnotic drugs like I.V Droperidol (curran and Crowley 1980),8 antiarrhythmic agents like I.V Mexiletine (kolli and mishra 1987) and 9.celcium channel blockers like verapamil (yaku et.,al.,1992) nifedioine (puri and batra 1988).but none of these methods were entirely satisfactory.

The present study was done to observe the cardiovascular response to laryngoscopy and tracheal intubation in normotensive patients and to find out the efficary of infra oral glyceryl trinitrate spray and intravenous lingnocaine hydrochloride attenuating the haemodynamic response.

The study was conducted in 75 patients . all of the were adults belonging to ASA I & II. . Patients udergoing general surgery, orthopedic and neurosurgical prodedures were include in the study.

These patients were randomly divded into three groups of 25 each,patients belonging to group 1 were used as the control, while those belonging to group2 received 400 micro.grams glyceryl trinatrate spray one minute before induction. In patients belonging to group 3, 1.5mg/kg intravenous xylocaine given 2 minutes before intubation.

Patients were premedicated with inj.glycopyrrolate 0.2mg and inj.ondansetron hydrochloride 4mg,injmidazolam 1mg, inj.tramadal hydrochloride(100mg) IV 30minutes before induction.

Hypoxia and hypoercarbia have been implicated as causes for increases in [pluse and blood pressure(king et .,al.,1951). In this study, co2 retention could be ruled out as an etiologic factor in the production of rise in pluse in pluse and blood pressure since adequate eliminating of co2 was ensured by controlled ventilation technique with soda lime circle absorber. Hypoxia was avoided by preoxygenation with 100 percent oxygen and ventilation continued with 66% nitrous oxide in 33% oxygen in all groups after induction.

Nitroglycernine originally used as antianginal agent, was found to reduce the blood pressure by preferentially dilating the venous capacitance vessels in low doses.the vascular effects if nitrates profoundly affects cardic performance, myocardial oxygen demand and coronary blood flow, thus reduction in myocardial ischaemia, improves myocardial contractility (Kaplan 1993)

There was asignificant rise in pluse rate in group 1 and2 during laryngoscopy and endotracheal intubation (table vi) which was similar to the findings of (puri and batra 1988, karma, wig and sapru 1986, fassoulaki and kaniaris 1983, kotak etal., 1986, mahajan et al., 1993, jayashree m.thakker et al., 1994). The maximum rate was seen at the first minute after laryngoscopy. (mary ollappally and iris MC 1992). In the control group the mean pulse rate was increased by 28.28+-4.18 at the first minute after laryngoscopy . in the group2 it was 20.08+-6.73 in group3 it was 1.2+-7.62 suggesting that there is no significant increase. In all the group there was a small increase before induction which may be due to pressure but not the heart rate response to intubation.

Systolic blood pressure raised significantly in the control group (table 3). mean systolic blood pressure showed in increase of 27.12+-6.58 in the control group,at the first minute after laryngoscopy. In the group2 the corresponding decrease was -26.44+-5.89 and in the group3 it was -7.92+-5.80. among the various groups, there was a highly significant difference in the increase in systolic blood pressure between groups 1 and 2 and 1 and 3($p<0.01$) . This was similar to the finding of desai N and desai K (1993). Between groups 2 and 3, in group2 there was significant decrease in the systolic blood pressure from the preperative value during intubation and at the first minute after laryngoscopy followed by group 3. At the third minute and fifth minute there was a highly significant difference ($p<0.01$) . This was due to a statistically highly significant fall in the systolic blood pressure in group2 in the third and fifth minute following intubation which was similar to the findings of fassoulaki and kaniaris(1983) they used intranasal nitroglycerine to attenuate the pressor response to laryngoscopy and tracheal intubation.

The systolic blood pressure was significantly elevated even at the fifth minute after laryngoscopy in the group 3 at laryngoscopy and after 1 minute , 3rd and 5th minute of intubation.

The above observation suggest that the pressor response was attenuated and prevented by glyceyl trinitrate oral spray and intravenous lignocaine.

Pramer and sabnis (1989) studied 100 cases of treated and untreated hypertensive patients divided into four groups. Nitroglycerine was given prior to induction. In response to laryngoscopy they founded a statistically significant increase in systolic blood pressure in patient who were not treated with nitroglycerine when compared to those treated with nitroglycerine. In my study we had excluded hypertrnsive individuals,but in normotensives response to laryngoscopy and intubation founded to be statically significant decreses in systolic and diastolic blood pressure in who were treated with nitroglycerne oral spray.

The diastolic blood pressure showed its maximum value at the first minute after laryngoscopy in control group . The diastolic blood pressure increased by 23.44+-4.52 in the 1st group,at the first minute after laryngoscopy. The corresponding decreases in group 2 and 3 were -14.04+-4.52 and -5.4+-8.10 respectively. The increase in diastolic pressure persisted even at the fifth minute after laryngoscopy in the first group, while in group2 and 3 decrease in diastolic blood pressure persisted even at the was a statistically highly significant difference in the elevation of diastolic blood pressure caused by laryngoscopy and intubation ($p<0.01$ among groups 2 and group3, in group 2 there was a significant difference in the fall of diastolic blood pressure caused by laryngoscopy and intubation.

Glyceryl trinitrate oral spray and IV lingnocaine effectively

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attenuated the increase in mean arterial pressure during laryngoscopy and endotracheal intubation compred to the control group where mean arterial pressure showed an increase of 23.73+-3.36 at the first minute after intubation corresponding value in group it was -17.88+-4.08 In group 3 it was -6.15+-5.94.

Karma s, wig j, sapra RA (1986) studied the effect of nitroglycerine ointement in attenunating the pressure response to tracheal intubation in 56 pts. The maximum rise in systolic blood pressure was significantly lower (7.66mm hg,6.2%) as compare to the control group (25.7mm hg,20.2%) but in my study with nitroglycerine spray showed significantly decreased in systolic and diastolic blood pressure . the pulse rate however increased in both groups to an equal extent .

Stoelting (1978) reported in his study of 24 patients for coronary artery by pass graft that a laryngoscopy less than 15 seconds is an effective method of minimizing the pressor response.

Arrixica,Murcia etal., (1987) studied the effects of intravenous lingocaine on the cardiovascular response to laryngoscopy and intubation in normotensive patients they founded that intravenous lignocaine was significantly effective in attenunatng the pressor response. Similar response was noted in my study.

Woods AW, Grant S, Harten J, Noblkes JS, Davidson JA, (1998), studied 60ASA1 or 2 patient for haemodynamic response for intubation the pressor response similar seen in this study.

The rate pressure product correlates with myocardial oxygen consumption and bears a fairly constant relationship to the onset of angina pectoris in any one patient with ischaemic heart disease. If the RPP for the development of angina or ischaemic change in the ECG is known for a particular patient, the anesthesiogist should maintain a lower RPP during the perioperative period . in the absence of specific information, it is desirable to keep the RPP at the less than 15000.

In this study the peak value of RPP in the control group was 15793.20+-940.35 at the first minute of laryngoscopy in group 2 and 3 there was a best attenunation as tha corresponding values in these group were 9776.40+- 558.02 and 9888.32+-912.32 respectively. Between group 1 and 2 and 1 and 3 there was a statistically highly significant difference in the rise of rate pressure product from the preperative value at all the times of study ($p<0.01$) .

Among glycerylTrinitrate and lingnocaine ,glyceryl trinitrate produce a best attenunation of the rise in RPP. In all the stages of recording both glyceryl trinitrate and lignocaline produced statistically better attenauation of the rise in RPP (table XXIII).

In the study with glyceryl trinitrate found out that although tachycardia was not prevented there was statistically significant fall in RPP.

Mary ollappally and iris MC rajiva (1992) compared the efficacy of intravenous linocaine and topical nitroglycerine in attenunating the cardiovascular response to laryngoscopy and intubation in twenty hypertensive patients. They found out that topical nitroglycernine offers an advantage over intravenous in hypertensive patients.

In my study also nitroglycerine is found to be better than intravenous lignocaine in attenuating the cardiovascular response to laryngoscopy and intubation.

The first investigation of the effect of endotracheal intubation on electrocardiogram was reported in 1940 . numerous reports have followed which indicates that cardiographic changes including changes in the direction of electrical block, venricular tachycardia , premature ventricular contractions, depression of P wave, increase or shortening of the P-R interval, decreases in the QRS voltage shortening or prolongation of Q-T interval, decreases in the voltage of T wave and depression of the S- T Segment.(takeshima 1964).

The etiology of intubation arrhythmias has been disputed. Reid and brace suggested that it was due to a vagovagal type of reflex while Burstein and his co-workers observed that the efferent limb was the cardioaccelerator nerve. Kind et al., thought that arrhythmias were due to relative sympathetic predominance induced by decreased parasympathetic or increased sympathetic activity.

Burstein (1951) in their study of 109 cases reported 31 cases of cardiac arrhythmias . insufficient depth of anaesthesia, prolonged obstruction before intubation leading to accumulation of co2 and irritation have been suggested as causes for arrhythmias. No arrhythmias noted in my study.

It was observed form this study that laryngoscopy and endotracheal intubation is associated with a definite increase in pluse rate and systolic and diastolic blood pressures. Glyceryl trinitrate spray and lingnocaine effectively attenuated the pressor response, but there was rise in heart rate in group 2 (glyceryl trinitrate) but significant decreases in rate pressure product when compare to lingnocaine. Among the two, glyceryl trinitrate spray appeared to be better than intravenous lingnocaine in the attunation of presor response to laryngoscopy and endotracheal intubation.

SUMMARY; In this present study consisting of 75 pts between age groups 20 60 years scheduled for general surgery Orthopedic and neuro surgery ,attenuation of haemodynamic

Changes following laryngo scopy and ETI was attempted using glyceryl trinitrate spray and Intra venous xylocaine

The cases under taken for the study were divided into 3 groups I II III ,Group I served as control , Group II –received glyceryl tri nitrate spray ,1 min before induction .Group IIIpts

Received IV lignocaine 1.5 mg /kg 2 min before intubation

All the pts were sedated with oral Diazepam 0.2 mg/ kg on the night before surgery .They received Premedication on the day of surgery with inj Glycopyrrolate (0.2 mg)inj Ondensetron (4mg) Inj Midazolam (img),inj Tramadol hydro chloride(2mg/kg)30 min before induction Pulae rate ,systolic blood pressure, and diastolic blood pressure were recorded pre operatively And pre induction Laryngo scopy and ETI was performed following induction with pentothal sodium

In the dose of 5 mg/kg and vecuronium bromide in the dose of 0.1mg / kg to facilitate intubation

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Pulse rate ,systolic blood pressure , diastolc blood pressure were recorded during intubation ,1min

,3min,5min after intubation .

The results of the study are 1.the patients receiving glyceryl trinatrate spray shows mild increase in

Puse rate ,but SBP,DBP,MAPand RPPvalues remained below the pre operative value at the end

Of 5 min after laryngoscopy and ETI when compared to control group 2.Lignocaine I.V has better

Control on heart rate when compared to glyceryl trinatrate oral spray and control groups

3.Among glyceryl trinatrate oral spray and I.V lignocaine ,glyceryl tri natrate oral spray showed

A better attenuation of pressor response than lignocaine group

CONCLUSIONS;

1 Glyceryl tri natrate oral spray and itravenous lignocaine hydro chloride are simple
Effective and comfortable means to attenuate the pressor response to laryngoscopy and ETI
2 Both glyceryl trinatrate oral spray and I.V lignocaine were significant in attenu tion
Of pressor responses compared to control group
3 Between glyceryl tri natrate intra oral spray and intra venous lignocaine group attenuation
Cardio vascular response and pressor response were more with glyceryl tri natrate spray.

REFERENCE

1. Katith G.allman and iain H. Wilson-oxford handbook of nanasthesia.drug formulary-glyceryl trinitrate spr ay sublingual does (400micro g pm) | 2. Robert k. stoelting-pharmacology and physiology in anaestesia practice 3rd edition | 3. Abou-madi. M,keszle H, yacoub JM.1977: "cardiovascular reaction to larynocopy and induction. | 4. Ajay kumar, bhatra YK,bhukal 1.1993 : "cardiovascular response to laryngoscopy and intubation.an evalution of nerve blocks and topical analgesia". ind.J.Anaesth. vol.41:20 | 5. Chandrasekhara PM and king r.1984: "attenuation of cardiovascular response to endotracheal intubartion". Ind.J.Anaesth. vol.32(5):358. | 6. Elkayan U and aronow ws.1982 : "glyceryl trinitrate(nitroglycerine ointement) | And isosorbide dinitrate : a review of their pharmacological properties and therapeutic use ". Drugs. Vol.23:165 | 7. Grover VK.etal., 1987 : "intranasal nitroglycerin attenuate pressor response to tracheal intubation in beta blocker treated hypertensive patients ". Anaesthesia . vol.42:884 | 8. Hill AB. Et al., 1981 : "intranasal administration of nitroglycerine". Anaesthesiology vol54:346 | 9. King BD etal., 1951: "reflex circulatory response to direct laryngoscopy and tracheal intubation performed during general anesthesia ". Anaesthesiology. Vol.12:556 | 10. Ollappaly m,iris MC> 1992 : "A comparative evaluation of sublingual nifedipine and topical nitroglycernine in attunating the haemodynamic response to laryngoscopy and intubation in hypertensive patients ". Ind.janaesth. vol.40:201 | 11. Parmer TK, sabnis SS.1989 : "topical nitroglycerine-A safeguard against pressor response to tracheal intubation . abstract of papers ". 38th annual conference.ind soc.of anaesthetists,manipal. | 12. Prys-roberts c. et al.,1971 : " studies of anaesthesia in relation to hypertension. Haemodynamics consequences of induction and endotracehal intubation". Br.j.anaesth, vol.43:531 | 13. Steolting RK. 1997: "circulatory changes during direct laryngoscopy and tracheal intubation. Influence of dusration of laryngoscopy with or without pripor lidocaine ".anaesthesiology. vol. 47:381 | 14. Stoelting RK 1978 "blood pressure and heart rate changes during short duration laryngoscopy and tracheal intubation : influence of viscous or intravenous lidocaine". Anaesth.analg. vol.57 197. | 15. Wilson IG,mieklejohn BH and smith G. 1991 : "intravenous lignocaine and sympatho adrenal responses to laryngoscopy, inhibition, the effect of varying time of injection ". Anaesthesia. Vol.46:177-180 |