COMPARISON OF HEMODYNAMIC RESPONSE TO LARYNGOSCOPY AND INTUBATION WITH PROPOFOL AND PROPOFOL PLUS INTRAVENOUS LIGNOCAINE IN KNOWN HYPERTENSIVE PATIENTS

Aqib Usman[1], Abdus Salam[2], Hidayat Ur Rehman[3], Laila Khan[4], Mehreen Khalid[5], Raheel Ahmad[6]

ABSTRACT

INTRODUCTION: Endotracheal intubation is a stressful stimulus, which is responsible for the complications mostly in patient with high BP and cardiovascular diseases. Propofol is an ideal intravenous (I.V) anesthetic agent that decreases cardiovascular response to laryngoscopy and endotracheal intubation. However mere use of Propofol has not shown complete satisfactory results and a need of further attenuation is felt. Various studies have been conducted on Lignocaine to check its effects on hemodynamic response to intubation. Aim of this study was to see effects of Lignocaine on cardiovascular response to intubation, when used as adjunct to Propofol.

OBJECTIVE: To evaluate the attenuation of hemodynamic response to laryngoscopy and intubation in response to I.V lignocaine in known hypertensive patients.

MATERIAL AND METHODS: Following Institutional Review Board approval, data of 60 patients who went under general anesthesia was collected. Patients with ASA (American Society of Anesthesiologist) physical status grade II and III (known hypertensive) belonging to any gender ranging from 30 to 65 years of age were included. These Patients were randomly divided into two groups, group P and group PL. Patients in group P received injection of propofol with a dosage of 2mg/kg and group PL patients received injection of lignocaine 1.5mg/kg in addition to propofol dosed at 2mg/kg. Hemodynamic indicators (HR, SBP and DBP) were noted down before induction, three minutes before intubation, thirty seconds and 5 minutes after intubation followed by statistical analysis performed on the obtained data.

RESULTS: A significant increase in haemodynamic response (Increased HR, SBP, DBP and MAP) was seen after the insertion of ETT in group P. On the contrary, response to intubation was less significant, in fact quite diminished in group PL. It was also observed that the haemodynamic response to ETT insertion is significantly greater than that to laryngoscopy alone.

CONCLUSION: Lidocaine is an effective medication to minimize hemodynamic responses (HR, SBP and DBP) in patients undergoing intubation for general Anesthesia.

KEYWORDS: Hemodynamic response, Laryngoscopy, Intubation, Propofol, Propofol plus, Intravenous lignocaine, Hypertensive patients

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[1] Lecturer, Northwest Institute of Health Sciences, Peshawar - Pakistan.
[2] Lecturer, Ahmad Medical Institute, Peshawar - Pakistan.
[3] Lecturer, Northwest Institute of Health Sciences Peshawar - Pakistan.
[4] Lecturer, Northwest Institute of Health Sciences, Peshawar - Pakistan.
[5] Lecturer, Northwest Institute of Health Sciences, Peshawar - Pakistan.
[6] Lecturer, Northwest Institute of Health Sciences, Peshawar - Pakistan.

Address for correspondence:

Laila Khan
Lecturer, Northwest Institute of Health Sciences, Peshawar - Pakistan.
E-mail: lailakhan854@gmail.com
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INTRODUCTION

Securing a definitive airway is done best with an endotracheal tube placement following laryngoscopy.[1,2] To facilitate laryngoscopy, patient is first anesthetized using induction agents. These Induction agents include sedatives and some neuromuscular blocking agents. These drugs can cause hypotension, however as a response to laryngoscopy and tracheal intubation, hypertension and tachycardia can occur. In normotensive patients, laryngoscopy and insertion of an endotracheal tube is immediately followed by an average increase of mean arterial pressure of 25 mm

Hg.[3] Usually these changings are not harmful to normal patients. But it may be lethal for hypertensive patients and those who have coronary artery diseases.[4]

Propofol is an ideal IV anesthetic agent that decreases cardiovascular response to laryngoscopy and endotracheal intubation. However mere use of Propofol has not shown complete satisfactory results and a need of further attenuation is felt.

Lignocaine acts as a local anesthetic and anti arrythmic due to its sodium (Na+) channel blocking effect, however, when given intravenously before induction of anesthesia it can exert sedative and analgesic effects in the central nervous system due to its G protein-coupled receptors and NMDA receptors.[5] Due to these effects, lidocaine is thought to be helpful for and has widely been used intravenously for reducing blood pressure and heart rate increases durig general anesthesia induction.[6]

A pilot study done on the attenuating effects of lignocaine was done in 1977, states that the cardiovascular response to laryngoscopy and intubation could be dynamically maintained with this drug.[7]

Furthermore, It was concluded that the use of the lignocaine therefore offer some limited advantages over routine IV anesthetics to further blur the hypertensive response in the anesthetic management of patients where the avoidance of presser response is of particular concern.[8]

A similar study in the same year, found that the differences between the two groups with respect to mean heart rate and systolic arterial pressure were not significant. However, maximum individual increases in systolic arterial pressure over control values were significantly greater in individuals without lignocaine.[9]

A comparison done in healthy patients and known hypertensive patients, compared the cardiovascular responses induced by laryngoscopy and intubation with those produced by prior lignocaine administration. The mean maximum increase in systolic arterial pressure after laryngoscopy and tracheal intubation was 51.3% compared with 22.9% for prior lignocaine administration group.[10]

The aim of this study was to evaluate the attenuation of hemodynamic response to laryngoscopy and intubation in response to I.V lignocaine in known hypertensive patients.

METHODOLOGY

This is prospective randomized comparative study. The study was conducted following Institutional Review Board and obtaining informed patient consent. 60 Patients selected by Non-probability purposive sampling technique were randomly divided into two groups each group containing 30 patients, Group P and Group PL. The study recruited known hypertensive adult between the age of 30 to 65 years who were ASAII and ASAIII patients of both sexes presenting for elective surgeries at IGH (Irfan general hospital). Any patient with coexisting morbidity and physiological derangement such as renal or hepatic failure, history of recent myocardial ischemia less than six weeks, congestive heart failure, symptomatic valvular or stenotic heart disease, history of asthma, morbid obesity (body mass index (BMI) > 35, history of gastro esophageal reflux and presence of cardiac pacemakers, patients with predictable difficult intubation, prolonged airway handling and delayed intubation, were excluded from our study.

Patients in group P received injection of Propofol with a dosage of 2mg/kg and group PL patients received injection of lignocaine 1.5mg/kg in addition to Propofol dosed at 2mg/kg. Hemodynamic indicators (HR, SBP and DBP) were noted down before induction, three minutes before intubation, thirty seconds and 5 minutes after intubation The data was collected in organized data sheet.

RESULTS

The group P had 16 males and 14 females and the group PL had 16 males and 14 females. Age ranged from 30 to 65 years in both groups. The range for weight was 45 to 85 kg and 46 to 84 kg in the group P and PL respectively. The two groups were comparable in terms of demographic data as there were no significant differences between the two groups in terms of age, sex, weight and ASA classification (Table 1).

Pre insertion values are taken as baseline values. The heart rates of the two groups were compared at induction. At insertion, the heart rate increased significantly in both groups, but the elevation was substantially higher in group P as compared to group PL. The elevation of heart rate significantly persisted for a longer period of time in group P, where it returned to the baseline value by 5 minutes as compared to the group PL where it returned by 3 minutes. By 5 minutes there was no significant difference between the groups.

Pre insertion values are taken as baseline values. The systolic blood pressure in the two groups was compared at baseline. An increase in SBP was noted just after insertion in both the groups, but the increase elicited by the group P was significantly higher($p<0.0001$) and persisted for a longer period of time as compared to that elicited by the group PL. It took 5 minutes for the group P values to return to baseline and 3 minutes for the group PL values to do so.

Pre insertion values are taken as baseline values.

The diastolic blood pressure was compared between the two groups at baseline. After insertion, both groups showed an increase in DBP that was statistically significant within and between the groups. The values returned to baseline by 3 minutes in the group PL and by 5 minutes in the group P. The difference between the groups was lost by 5 minutes.

Pre insertion values are taken as baseline values. In terms of MAP, the two groups were not statistically different at baseline. After instrumentation, the group P had an increase in MAP that was significantly higher

Table 1: Demographic and clinical characteristics of study participants.

		GROUP P	GROUP PL	P value
MEAN AGE (years)		47.5	48.5	0.278
Gender	Male	16	16	1.00
	Female	14	14	
ASA I		15	15	1.00
ASA II		15	15	

Table 2: Mean heart rate at different times among Group P and Group PL.

	Group P Mean±SD	P value for difference within group P	Group PL Mean±SD	P value for difference within group PL	P value for difference between group P and group PL
T0	93.0±13.5	-	90.8±11.8	-	0.383
T1	111.9±13.6	<0.0001	106.9±11.1	<0.0001	0.047
T2	106.5 ±13.4	<0.0001	97.8±9.2	<0.0001	0.0001
T3	99.5±13.1	<0.0001	88.5±6.8	0.592	0.0001
T4	92.2±11.4	0.664	85.4±6.5	0.059	0.066

Table 3: Mean systolic blood pressure at different time points among the study participants.

	Group P Mean±SD	P value for difference within group P	Group PL Mean±SD	P value for difference within group PL	P value for difference between group P and group PL
T0	76.4±7.2	-	75.7±7.1	-	0.618
T1	76.4±7.2	<0.0001	83.5±8.6	<0.0001	<0.001
T2	85.2±10.4	<0.0001	78.0±7.4	0.012	<0.0001
T3	80.7±10.0	0.007	75.5±8.0	0.793	0.005
T4	76.1±9.8	0.813	74.7±7.3	0.310	0.447

Table 4: Mean diastolic blood pressure at different times among Group P and Group PL

	Group P Mean±SD	P value for difference within group P	Group PL Mean±SD	P value for difference within group PL	P value for difference between group P and group PL
T0	121.2±10.8	-	117±11.9	-	0.067
T1	146.4±16.4	<0.0001	127.7±12.9	<0.0001	<0.0001
T2	135.4±12.8	<0.0001	121.5±11.4	<0.0001	<0.0001
T3	128.5±11.5	<0.0001	117.6±10.5	0.487	<0.0001
T4	122.0±11.8	0.665	115.4±9.1	0.147	0.002

Table 5: Mean arterial blood pressure at different times among group P and group PL.

	Group P Mean±SD	P value for difference within group P	Group PL Mean±SD	P value for difference within group PL	P value for difference between group P and group PL
T0	91.5±7.3	difference	89.7±7.2	-	0.225
T1	112.2±14.5	within group P	98.6±14.5	<0.0001	<0.0001
T2	104.3±10.6	<0.0001	93.3±8.1	<0.0001	<0.0001
T3	97.6±9.9	<0.0001	90.8±7.6	0.219	<0.0001
T4	93.2±9.7	0.228	89.2±7.0	0.536	0.019

and more persistent as compared to the group PL. These changes subsided to baseline values by 3 minutes and 5 minutes in the groups PL and P respectively.

DISCUSSION

Endotracheal intubation is a stressful noxious force stimulus, it stimulates laryngeal and tracheal sensory receptors, resulting in a slightly elevated the level of catecholamines which is responsible for the complications mostly in patient with cardiovascular diseases.

We found that lidocaine is effective medication to lower hemodynamic responses (heart rate, systolic blood pressure and diastolic blood pressure) of intubation. Significant difference between the efficacy of the two kinds of medication (propofol and propofol plus lidocaine) was observed.

Abou-Madi et al have discussed the possible mechanisms of action of IV lignocaine.[11] Their finding demonstrated that a preintubation lidocaine dose of 1.5 mg/kg given intravenously 3 minutes before intubation is safe and no harmful effects are seen,

Different studies show that lignocainestabilize arterial pressure, heart rate and cardiac output intra operatively and post operatively. The mechanism of lignocaine behind cardiovascular stability is due to direct myocardial depressant, effect on peripheral vasodilation and the effect on synaptic transmissions. Moreover, another study by Ali et al. in 2010 revealed that pre-treatment with xylocard improves intra- and post-operative hemodynamic stability during laparoscopic surgery without prolonging recovery.[12] Our study was in line with some previous studies such as Shin et al. that compared the effects of lidocaine, fentanyl, Nicardipine and Esmolol, on the hemodynamic response during intubation and that study revealed that all of these agents are effective in producing hemodynamic stability. Additionally, Malde and Sarode in a 2007 study compared lignocaine and fentanyl efficacy on hemodynamic stability and revealed that lignocaine and fentanyl both attenuated the rise in heart rate; however, lignocaine produced better results.[13] We noticed that lignocaine was effective on maintaining relative hemodynamic stability in hypertensive patients but it could not inhibit all hemodynamic responses to intubation. In this case our results were in agreement with Feng et al. and Salihoglu et al. studies that disclosed that lidocaine plus fentanyl were slightly more effective in controlling PR following endotracheal intubation. In conclusion, our study was well designed and we considered factors that could possibly have affected our results.[14] Propofol plus lidocaine is effective in decreasing the hemodynamic response to tracheal intubation, however, neither Propofol nor Propofol plus lidocaine could inhibit all hemodynamic responses.

A significant haemodynamic response consisting of an increase in Heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure was seen after the insertion of ETT in group P in this study but less significant, in fact quite diminished in group PL. It was also observed that the haemodynamic response to ETT insertion is significantly greater than that to laryngoscopy only. The response is also short lived in the PL group compared to group P. Since this response might be of no clinical importance in the healthy, normotensive patients, but might be harmful in patients with hypertension, aortic or cerebral aneurysm raised intracranial pressure or other cardiovascular diseases. In such cases, the attenuated response of the lignocaine might be desirable.

CONCLUSION

This study shows that hemodynamic responses to intubation in our patients were controlled effectively by using lidocaine, and not adding lidocaine to Propofol increases the hemodynamic instability more than propofol with lignocaine. In conjugation to this study, different studies show that lignocaine stabilize arterial pressure, heart rate and cardiac output intra operatively and post operatively.

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NOTES ON CONTRIBUTORS

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CONFLICT OF INTEREST

Authors declare no conflict of interest.

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