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ATTENUATION OF HAEMODYNAMIC RESPONSE TO LARYNGOSCOPY AND INTUBATION WITH LIGNOCAINE AND NALBUPHINE: A COMPARATIVE STUDY

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ABSTRACT

Background: Previous studies done on Lignocaine and Nalbuphine individually found to attenuate the haemodynamic stress response but none of the study compared the two drugs. To compare the efficacy of intravenous Lignocaine versus intravenous Nalbuphine for attenuating the stress response to laryngoscopy and endotracheal intubation. **Material and method**: The present study was a hospital based prospective, randomized study conducted amongst adults aged 20 years to 60 years who were scheduled for elective surgery under general anaesthesia in the Department Of Anaesthesiology And CriticalCare. The patients were randomly allocated to GROUP L (n = 30) Patients received intravenous lignocaine 1.5 mg kg^{-1} ninety seconds before tracheal intubation and GROUP N (n =30) received intravenous nalbuphine 0.2 mg kg^{-1} one hundred and twenty seconds before tracheal intubation. **Results**: All the patients were monitored for heart rate, blood pressure(systolic, diastolic and mean BP) for haemodynamic responses during laryngoscopy, endotracheal intubation or side effects in the postoperative period. There was no statistically significant difference between both groups with regards to mean age and gender. The attenuation of pressor response to laryngoscopy and endotracheal intubation was significantly better with nalbuphine as compared to lignocaine.There was no side effect in both the groups. **Conclusion**: It is safe to assume that the dosage of 0.2 mg/kg of Nalbuphine is better at the attenuation of haemodynamic pressor response to laryngoscopy and endotracheal intubation than Lignocaine 1.5 mg kg -1.

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INTRODUCTION

Securing airway is the most important aspect of safe anaesthetic procedure. Endotracheal tube is gold standard for securing the airway during general anaesthesia and in critical care settings.

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When laryngoscopy and intubation is carried out, there is mechanical irritation of stretch receptors situated in the respiratory tract leading to reflex haemodynamic responses through a sympathetic reflex. Fibers forms by the vagus and gloss opharyngeal supply the heart, blood vessels and adrenal medulla, causing a stimulatory adrenosympathetic response resulting in increased blood pressure, heart rate and plasma catecholamines.[1-3] It may have deleterious respiratory, neurological and cardiovascular effects which are more marked in hypertensive patients.[1,4,5] In patients with coronary artery disease, leaking abdominal aneurysm, intracranial

aneurysm and recent myocardial infarction ,these transient changes can result in potentially deleterious effects such as myocardial ischaemia, left ventricular failure (as a result of increased myocardial oxygen demand) and cerebral haemorrhage.[5-7] Various drugs and techniques have been used in current anaesthesia practice to attenuate these adverse effects, including deep level of general anaesthesia before endotracheal intubation with inhalation agents like halothane, use of supraglottic intubating devices like laryngeal mask airway, use of drugs such as calcium channel blockers, ACE inhibitors, vasodilators, Alpha 2 agonists, Beta -adrenergic blocking agents, magnesium sulfate, local anaesthetics (lignocaine) and opioids.[8-18] None of the above techniques have been proved to be satisfactory as they all had limitations. Administration of potent analgesics prior to intubation has been shown to significantly attenuate the pressor response to laryngoscopy and intubation. Opioids specifically have been found to be useful in suppressing of this haemodynamic response but may produce respiratory depression, rigidity or may prolong the recovery time.

Lignocaine is an aminoethylamide prototype of amide local anaesthetic group with antiarrhythmic properties which has been used for topical anaesthesia of upper respiratory tract in form of spray, gargle, intratracheal and translaryngeal nerve blocks prior to laryngoscopy.[13] Lignocaine has also been used intravenously for attenuating these stress responses. In1961 Bromage showed that intravenous lignocaine blunted pressure response to intubation.[27] Its onset of action after i/v administration is 45-90 seconds with distribution half- life of around 8 minutes. Elimination half -life of lignocaine is around 90 to 110 minutes. It is metabolized in liver and then excreted by the kidney. Lignocaine is one of the cheapest and safest drug used widely to attenuate stress response to intubation.[6,13] Besides pure mu receptor agonists, partial agonists or agonist-antagonists are being used now as agents to attenuate pressor response. Nalbuphine is a semi-synthetic opioid agonist-antagonist analgesic of phenanthrene series. It has a potency equivalent to that of morphine on milligram basis. It is a partial mu antagonist analgesic. It binds to mu, kappa, and delta receptors. Its onset of action after i/v administration is 2-3 minutes with half -life of 3 to 6 hours. Nalbuphine exibits ceiling effect such that increase in dose greater than 30 mg does not produce further respiratory depression in the absence of other medications affecting respiration. Nalbuphine may partially reverse or block opioid - induced respiratory depression from mu agonist analgesic.[19,20]It does not require special narcotics license and is a safe analgesic even in children.[21] After extensive Medline search it was found that no work has been done to compare the effects of lignocaine versus nalbuphine for attenuating the haemodynamic stress response to laryngoscopy and endotracheal intubation. Therefore, the present study was planned to assess the effect of iv nalbuphine on attenuating stress responses during laryngoscopy and endotracheal intubation and to compare it with iv lignocaine as both these drugs are easily available, inexpensive and are used daily in operating room.

MATERIAL AND METHODOLOGY

The present study was conducted in the Department Of Anaesthesiology and Critical Care after approval from the Institutional Ethical Committee. This was a prospective randomized study carried out over a period of one and half years i.e. from December 2018 to March 2020.A total of Sixty patients aged 20-60 years of either sex belonging to American Society of Anesthesiologist (ASA) physical status I or II, presenting for elective surgery under general anaesthesia requiring endotracheal intubation were included in the present study. Patient showing Mallampatti score III or more, allergic to study drugs, who had anticipated difficult intubation, not consenting for the participation in the study and on beta - blockers or any other antihypertensive drugs were excluded from the study. Informed consent was taken from all the patients and were subjected to preanaesthetic check- up and all the routine investigations a day before the surgery. They were also advised to be fasting for 8 hours before surgery.

Anaesthetic technique: On the day of surgery, base line values of heart rate, blood pressure e.g. systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial blood pressures (MAP) were recorded (T_0). ECG and SPO_2 was also monitored continuously. An intravenous line was established with IV 18 G cannula and all the patients were randomly allocated to one of the following two groups by slip draw method:

GROUP L :(n = 30) Patients belonging to this group received intravenous lignocaine 1.5 mg kg^{-1} ninety seconds before tracheal intubation. GROUP N :(n =30) patients belonging to this group received intravenous nalbuphine 0.2 mg kg^{-1} one hundred and twenty seconds before tracheal intubation. Patients belonging to group L received intravenous lignocaine, while group N received intravenous nalbuphine hydrochloride. All patients were premedicated with glycopyrrolate 0.2 mg kg^{-1} iv. Patients were pre-oxygenated with 100% oxygen for 3 to 5 minutes and then induced with i/v propofol 2 mg kg^{-1}at the rate of 1mlsec^{-1} end point of induction was lack of response to verbal command followed by loss of eyelash reflex. This was followed by inj. vecuronium bromide 0.1 mg kg^{-1} intravenously. Patients belonging to group L were administered intravenous lignocaine 1.5 mg kg^{-1} ninety seconds before intubation and patients belonging to group N received intravenous nalbuphine 0.2 mg kg^{-1} one hundred and twenty seconds before intubation. Three minutes after vecuronium administration laryngoscopy and tracheal intubation was done using an appropriately sized cuffed endotracheal tube. Patients in whom endotracheal intubation was prolonged for more than 20 seconds or failure of intubation at first attempt were excluded from the study. Readings of heart rate, blood pressure (systolic, diastolic and mean arterial pressure) were measured at following intervals and labelled as

T_0 - base line
T_1 - just before intubation
T_2 - just after intubation
T_3 -Two minutes after intubation
T_4 -Four minutes after intubation
T_5 -Six minutes after intubation
T_6 -Eight minutes after intubation
T_7 -Ten minutes after intubation
T_8 -Fifteen minutes after intubation as primary variables.

Any adverse effects and complications were recorded as secondary end points. During this period anaesthesia was maintained with 33% oxygen: 67% nitrous oxide and isoflurane 1 MAC, with muscle relaxation being provided by

injection vecuronium. Hypotension, bradycardia or arrythemia if any during surgery were recorded and treated accordingly. No surgical stimulus was allowed during study period. At the end of surgery, the neuromuscular blockade was reversed with i/v neostigmine 0.05mgkg^{-1} and i/v glycopyrrolate 0.01mgkg^{-1}. Patients were extubated after extubation criteria were met and were shifted to recovery room. In the post - anaesthesia care unit, they were monitored for any evidence of complications or adverse events like nausea, vomiting, respiratory depression, etc for four hours.

STATISTICAL ANALYSIS

The results of variables were expressed in mean and standard deviations. Independent t-test was performed for comparing mean of the two groups, paired t-test was performed for comparing mean percentage of improvement in the groups of p value of < 0.05 was taken as statistically significant.

RESULTS

This prospective randomized study to compare the effect of lignocaine and nalbuphine for attenuating the pressor response to intubation was done on 60 patients aged between 20-60 year. In group L patients were received intravenous lignocaine 1.5 mg kg^{-1} ninety seconds before intubation and patients belonging to group N received intravenous nalbuphine 0.2 mg kg^{-1} one hundred and twenty seconds prior to intubation and following observations were found.

Table 1 shows both groups were comparable with respect to their demographic profile with no significant difference in age and gender

Table 1. Showing demographic distribution in two groups

Group	Gender			Total
	Male	Female	Mean Age	
Group L	8	37.43	37.43+8.406	30
Group N	10	36.10	36.10+10.240	30
P value	0.573		0.584	

Table 2 shows that the baseline heart rate was comparable between the two groups. Rise of mean heart rate at just after intubation was high in lignocaine group as compared to nalbuphine group. These values indicated better control of heart rate in both the groups

Table 2. Intergroup comparison of the mean heart rate

	Group L		Group N		
Heart rate	Mean	Std. Deviation	Mean	Std. Deviation	P value
Base Line(T_0)	82.87	9.769	81.93	12.051	0.743
Just before Intubation(T_1)	84.80	10.270	78.63	10.581	0.026*
Just after Intubation(T_2)	101.40	12.447	94.33	13.407	0.039*
2min after intubation(T_3)	98.27	12.897	89.77	15.213	0.023*
4min after intubation(T_4)	94.60	12.748	86.10	16.670	0.030*
6min after intubation(T_5)	92.67	12.344	84.03	16.059	0.023*
8min after intubation(T_6)	90.10	11.909	82.47	15.558	0.037*
10min after intubation(T_7)	87.83	12.100	79.87	13.516	0.019*
15min after intubation(T_8)	85.57	11.242	78.57	12.139	0.024*

* - (0.05) statistically significant when compared to their respective value in two groups

**-(0.01) statistically highly significant when compared to their respective value in two groups

Table 3 shows inter group comparison of SBP. At induction a decrease in SBP was noticed in both groups as compare to baseline which was not statistically significant.

Table 3. Intergroup comparison of the mean systolic blood pressure

	Group L		Group N		
Systolic blood pressure in mm Hg	Mean	Std. Deviation	Mean	Std. Deviation	P value
Base Line(T_0)	127.17	14.499	128.07	14.353	0.810
Just before Intubation(T_1)	102.87	12.902	102.47	15.126	0.913
Just after Intubation(T_2)	149.93	17.213	137.73	19.846	0.014*
After 2min(T_3)	125.30	14.900	124.80	19.105	0.910
After 4min(T_4)	116.87	12.697	116.90	18.905	0.994
After 6min(T_5)	110.37	12.178	110.43	16.513	0.986
After 8min(T_6)	105.57	15.545	103.57	13.190	0.593
After 10min(T_7)	102.87	16.145	100.27	10.419	0.462
After 15min(T_8)	100.73	14.558	98.50	11.029	0.506

*- statistically Significant when compared to their respective value in two groups.

Table 4 shows intergroup comparison of DBP. Comparison in the two groups revealed that the DBP were comparable at the baseline was not statistically significant.(p>0.05).

The table 5 shows intergroup comparison of MAP. Comparison in the two groups revealed no statistically significant difference between the baseline MAP (p>0.05).

DISCUSSION

In 1940, Reid and Brace first described hemodynamic response to laryngoscopy and intubation.[1]Induction of anaesthesia, endotracheal intubation and surgical stimulation often invoke cardiovascular responses characterized by increase in arterial blood pressure, heart rate changes and disturbances in cardiac rhythmas a result of intense sympathetic nervous system stimulation.[13,22] Laryngoscopy and Endotracheal intubation can lead to eminent changes in hemodynamic and intracranial pressure which could be transient and highly variable occurring probably .These changes are maximum immediately after intubation and lasts for 5-10 min, which may be well-tolerated by normal individual but they may have detrimental effects in high risk patients including myocardial infarction, cardiac failure, intracranial haemorrhage and increase in intracranial pressure.[16,17] These changes need to be attended by an anaesthesiologist to decrease haemodynamic response to laryngoscopy and tracheal intubation. Various studies with different techniques and drugs have been carried out, aiming at attenuation of stress response but are only partially effective. Drugs being used include intravenous local anesthetics, opioids, calcium channel blockers, clonidine, gabapentin, and β - adrenergic blockers (esmolol and metoprolol) and their combinations.[23,24] Lignocaine is an effective, safe and established agent, having a suppressive effect on the circulatory responses in patients undergoing laryngoscopy and tracheal intubation because of its short duration, antiarrythmic effects and its effect on synaptic transmission. Lignocaine blocks the sodium channels in the cell membranes of the heart and reduces the rate of the rise of the action potential and hence the conduction velocity. Possible mechanism behind the response of intravenous lignocaine in blunting rise in pulse, blood pressure, intracranial and intraocular pressure could be due to direct myocardial depressant effect, a peripheral vasodilating effect and an effect on synaptic transmission.[24]

Table 4. Intergroup comparison of the mean diastolic blood pressure

	Group L		Group N		
Mean Diastolic BP in mmHg	Mean	Std. Deviation	Mean	Std. Deviation	P value
Base Line(T_0)	79.67	8.409	80.63	9.141	0.671
Before Intubation(T_1)	63.77	9.940	64.77	14.219	0.753
Just after Intubation(T_2)	98.23	9.947	92.53	9.888	0.030*
2min after intubation(T_3)	85.07	10.174	78.67	10.256	0.018*
4min after intubation(T_4)	77.57	9.811	72.20	11.078	0.052*
6min after intubation(T_5)	70.80	9.796	68.77	12.467	0.485
8min after intubation(T_6)	68.13	12.014	66.77	8.935	0.619
10min after intubation(T_7)	66.07	12.988	64.70	9.308	0.641
15min after intubation(T_8)	63.67	11.672	62.37	9.390	0.636

*- Significant when compared to their respective value in two groups

Table 5. Intergroup comparison of the mean arterial pressure

	Group L		Group N		
Mean arterial pressure in mmHg	Mean	Std. Deviation	Mean	Std. Deviation	P value
Base Line(T_0)	96.47	9.926	95.63	9.901	0.746
Just before Intubation(T_1)	78.30	10.256	76.30	13.644	0.524
Just after Intubation(T_2)	114.70	10.021	106.33	13.471	0.008*
2min after intubation(T_3)	99.67	10.993	94.67	15.439	0.154
4min after intubation(T_4)	90.93	10.415	88.73	16.962	0.547
6min after intubation(T_5)	84.67	9.977	83.00	16.563	0.639
8min after intubation(T_6)	80.43	12.199	77.30	9.966	0.280
10min after intubation(T_7)	79.23	12.610	75.03	9.290	0.147
15min after intubation(T_8)	77.57	11.921	73.63	8.954	0.154

*- statistically Significant when compared to their respective value in two groups

Several studies have found that 1.5 $mgkg^{-1}$ of lignocaine suppresses stress response to intubation when given 1-3 min before intubation.[22,24,25] Nalbuphine is an opioid with agonist antagonist action, weak antagonist at mu and agonist at kappa opioid receptors. Nalbuphine has a comparable analgesic potential to morphine. Unlike morphine, nalbuphine has a plateau effect on respiratory depression and also has been used to reverse the respiratory depression due to morphine.[19,20] It is safe, inexpensive and easily available. Being cardiovascular stable having longer duration of action, it has been used in major surgeries intra operatively. Ahsan et al. and Chawda et al. and found that Nalbuphine given in dose of 0.2mg/kg before laryngoscopy and endotracheal intubation prevented stress response which was comparable to our study in attenuating haemodynamic response by Nalbuphine to intubation.[26,27]

In the present study, Lignocaine and Nalbuphine were compared in regards to their efficacy to attenuate stress responses during laryngoscopy and endotracheal intubation. In this study, patients belonging to group L were administered intravenous Lignocaine 1.5 mg kg^{-1} ninety seconds before intubation and patients belonging to group N received intravenous Nalbuphine 0.2 mg kg^{-1} one hundred and twenty seconds before intubation. These doses were found to be adequate to control haemodynamic response to intubation with minimal side effects in our patients. Mean age of our patients in group L and N were 37.43±8.40 and 36.10±10.24 years respectively which was comparable. As per gender distribution there were 27% male and 73% female in group L whereas in group N males were 33% and females 67%. The results are statistically not significant in between males and females in both groups L and N (P=0.573). hence gender was not also the confounding factor. In Lignocaine group, heart rate at baseline was 82.87±9.76. There was sudden rise in heart rate after intubation to the value of 101.40±12.44 which gradually touched to baseline values at 15 minutes (85.57±11.24).

In Nalbuphine group, baseline heart rate was 81.93±12.051. Following 0.2mgkg ¯1 nalbuphine infusion at 8 minutes after intubation heart rate values touches the base line and after that there is slight decrease in heart rate reaching upto 78.57±12.139 at 15 minutes. Baseline heart rates were comparable between the groups(p=0.743). At all time intervals following intubation, the mean heart rate was suppressed maximum at 15 min interval with heart rate of 78.57±12.13 in group N whereas the heart rate was suppressed to lesser extent in group L nearing baseline values at 15 min interval at 85.57±11.24. The heart rate between the groups was statistically significant ($p \leq 0.05$) before intubation, after intubation upto the 15 min intraoperatively with superior effect obtained from the Nalbuphine. Gurulingappa et al. compared the effect of lidocaine, fentanyl and placebo on stress response and found that heart rate was increased more in case of lidocaine and placebo.[28] Similar results were found in the study of Thippeswamy et al, Valeshabad, Mohmmadi et al.[29-31] Muhammed Ahsan has compared nalbuphine 0.2mg kg^{-1} with placebo. He noticed increases in HR just after intubation which was significant i.e., more than 20% rise from baseline in placebo group. Our result is comparable to this study. This difference can be explained by the adequate sedation effect of the premedication drug given 10 minutes before induction of anaesthesia. Rise in HR occurs due to elevations in plasma catecholamines levels which occurred markedly in placebo group while lesser increase in HR occurred following intubation in patients receiving nalbuphine.[26] Khan had compared effects of nalbuphine versus fentanyl on haemodynamic response and showed HR response after tracheal intubation was significantly higher in the Nalbuphine group as compared to fentanyl group.[32] According Begum I et al. immediately after drug administration, the heart rate increased in Nalbuphine Group and decreased in Dexmedetomidine Group (P-value < 0.0001), the heart rate gradually stabilized at near or below the baseline value during the intra operative period in both the groups. Intraoperatively, there was no significant difference in the heart rate of both the

groups (P-value >0.05)[33]. Bhandari et al. also compared the effects of nalbuphine versus fentanyl on haemodynamic response and found increase in the heart rate after intubation in both the groups which was statistically significant. Similar results were found in the study done by Kay et al and Sharma et al.[34-36] None of the earlier studies compared the lidocaine with nalbuphine, so intergroup results of our study can't be compared to previous study. Upon overall comparison between two groups in present study, the heart rate was better controlled with Nalbuphine.

In lignocaine group baseline systolic blood pressure was 127.17±14.49 . There was fall in SBP following drug infusion but immediately following intubation there was hike in the mean SBP (149.93±17.21) and again then there was gradual fall in SBP below baseline from 2 minutes to 15 minutes after intubation (100.73±14.55). In nalbuphine group the baseline SBP was 128.07±14.35. Mean systolic blood pressure increased just after intubation (137.73±19.84) followed by decreased SBP below baseline by 2 min after intubation (98.50±11.029). The reason behind elevation of the mean SBP could be due to the fact that both lidocaine and nalbuphine have minimal depressive effect on the cardiovascular system and cannot completely attenuate response to endotracheal intubation. The mean SBP was in both the groups comparable (p= 0.81). There was transient rise in the SBP immediately after intubation. Subsequently mean SBP revealed a downward trend in both groups. On intergroup comparison results were statistically non-significant except just after intubation where result were statistically significant ($p \leq 0.05$) suggesting equivalent hemodynamic stability with both the drugs. Valeshabad et al. compared the attenuation of the stress response of lidocaine and propacetamol, found that none of them was effective in attenuating blood pressure responses after laryngoscopy, whereas in our study lignocaine and nalbuphine both attenuate SBP after intubation.[29] In contrast to the present study, Gurulingappa et al and Bachofen reported that fentanyl showed a significant pressure-lowering action persisting over the whole observation period in all patients while no significant effect of lidocaine on the pressure response could be observed.[28,37] In concordance to the present study Bhandari et al. observed significant attenuation of the hemodynamic changes in patients of nalbuphine as compared to fentanyl group.[34] Contrary to the present study, Khan and Hameedullah observed that Nalbuphine provided lesser haemodynamic stability in comparison to Fentanyl when used as an intraoperative analgesic in TIVA with propofol.[32] Aftab et al compared Fentanyl/Isoflurane and Nalbuphine/Isoflurane in patients undergoing elective coronary artery bypass surgery. In contradiction to the present study, they showed that Fentanyl/Isoflurane provided better haemodynamic stability than Nalbuphine/ Isoflurane.[38]

None of the earlier studies compared the lidocaine with nalbuphine, so intergroup results of our study can't be compared to previous study. Upon overall comparison between two groups in present study, the SBP was attenuated by both the drugs. In our study intergroup comparison revealed that the DBP was comparable at the baseline and was not statistically significant.(p>0.05). However, there was statistically significant difference in the DBP just after intubation, at 2 and 4 minutes .In nalbuphine group DBP rise from baseline 80.63±9.14 to 92.53±9.88 just after intubation but reverted below base line at 2 min after intubation and stayed below base line throughout study period .Whereas in lignocaine group DBP rises from baseline from 79.67±8.40 to 98.23 ±9.94 just after intubation but reverted below base line at 4 minutes after intubation and then remains below baseline. Singh G et al. and Gulabani et al. studied dexmedetomidine and lignocaine in attenuating the hemodynamic responses during laryngoscopy and endotracheal intubation and found that lidocaine had less attenuating effect on DBP than dexmedetomidine .[39,40]

Jain et al. found that both esmolol and lignocaine are effective in attenuating the stress response due to laryngoscopy and intubation, but with esmolol haemodynamic variables remain more stable.[18] Similarly, in the present study nalbuphine was found superior to be than lignocaine. The results of the present study are in accordance with that of Kothari et al. who found that non-significant fall ($P > 0.05$) up to 3 min and thereafter a significant rise ($P < 0.05$) in all the parameters were observed throughout the remaining study period with nalbuphine, whereas a continuous and significant ($P < 0.05$) rise in these parameters were observed with pentazocine. They concluded that nalbuphine effectively reduced the DBP.[41] The results of our study are contrary to the study done by Khan and Hameedullah. Sharma et al in their study comparing the effects of nalbuphine versus fentanyl.

They found that the increase in blood pressure was more in nalbuphine group than in the fentanyl group.[32,35] As none of the previous studies compared the lignocaine with nalbuphine, so intergroup results of our study can't be compared to any previous study. Upon overall comparison between two groups in present study, the DBP was attenuated by both the drugs but in initial 2 minutes more by nalbuphine. Mean arterial pressure is a derived value and is important in relation to the auto-regulatory responses of the heart, brain and kidneys. In the present study intergroup comparison of MAP revealed no statistically significant difference between the baseline MAP (p>0.05). However there was statistically significant difference in MAP at just after intubation (p=0.008). In nalbuphine group MAP rose just after intubation above baseline from 95.63±9.90 to 106.33±13.47 but it reverted back to below baseline at 2 minutes after intubation. While in lignocaine group MAP rises just after intubation from 96.47±9.92 to 114.7±10.02 and falls back below baseline at 4 minutes after intubation. There was decrease in MAP in both the groups, however, nalbuphine exhibited more decrease as compared to the lignocaine group. These results are supported by the study done by Ahsan et al, Chawda et al, Bhandari R et al where they found nalbuphine more effective in reducing the MAP than the other compared drugs eg.fentanyl.[26,27,34]

None of the earlier studies compared the lignocaine with nalbuphine, so intergroup results of our study can't be compared to any study as such. Overall, HR, SBP, DBP, and MAP were significantly elevated after the endotracheal intubation in both the groups, and higher values were noted in lignocaine group as compared to the nalbuphine group. The elevation persisted mostly for 2 min in nalbuphine group and 4 min in lidocaine group and subsequently, the parameters returned to the baseline values in both the groups. These results are similar to those stated by Miller. The initial fall in all the haemodynamic parameters in N group is because of its strong and predominant kappa agonistic action. Rise in hemodynamic parameters after intubation is due to sympathoadrenal stimulation.[42] None of the patients involved in the study had any complication such as respiratory depression, bradycardia, arrhythmias, nausea, vomiting, or

pruritus. Strength of the study was its randomization Limitations were the small sample size, lack of control group, the study was not blinded which might have led to observational bias, plasma catecholamine levels were not measured in our study, which is an objective means of measuring hemodynamic stress response and the effect was not seen in hypertensive and cardiac patients. It will be more useful to study in high-risk hypertensive and cardiac patients. Hence, further studies required to know the effective, accurate plasma concentration of the drug to prevent pressor response during laryngoscopy and intubation in risk patients.

CONCLUSION

Based on the findings of the present study, it is safe to assume that the dosage of 0.2 mg/kg of Nalbuphine is better at the attenuation of haemodynamic pressor response to laryngoscopy and endotracheal intubation than Lignocaine 1.5 mg kg -1. It also determines the efficacy and safety of the drugs at this dosage as none of the patients had any side effects due to the drugs. Our study favours Nalbuphine as the drug of choice for blunting of pressor response in such patients.
Further studies with a larger sample size and which will overcome our limitations may be required to generalize the results and strengthen the literature.

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