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COMPARISON OF INTUBATION RESPONSE FOLLOWING INTRAVENOUS AND INHALED LIGNOCAINE IN ELECTIVE SURGERIES

Anesthesiology

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ABSTRACT

Introduction: General anaesthesia with endotracheal intubation and IPPV/ spontaneous ventilation is commonly practised by anaesthesiologist for many surgical procedures. Tracheal intubation is one of the most painful procedures. So adequate blunting of the intubation response is required to avoid hemodynamic instability following intubation. Our aim was to compare the response to intubation following intravenous and nebulisation of lignocaine.

Methodology: A prospective, observational, double blinded, randomized controlled trial using a sample size of 60 patients of either sex who comes under ASA I & II admitted for elective surgeries under general anaesthesia was conducted in Father Muller medical college, Mangalore after institutional ethical clearance. Patients were divided into two groups. Group A received intravenous 2% Lignocaine 1.5 mg/kg and the group B received 4% Lignocaine nebulisation. Data were recorded as means, standard deviation and percentages. The categorical co varieties (sex, ASA grade) were analysed using the Chi-Square test and Fishers exact test. The intergroup comparison of the parametric data (age, weight, height, hemodynamic response) were done using student t- test.

Result: The maximum increase in Heart rate was 109.73±15.34 bpm in Group A and 95.00±32.334 bpm in Group B at 1 minute after endotracheal intubation. The maximum increase in systolic BP was 166.7±20.5 mm of Hg in Group A and 144.67±17.43 mm of Hg in Group B at 1 minute after endotracheal intubation. The maximum increase in diastolic BP was 105.13±11.78 mm of Hg in Group A and 97.27±12.2 mm of Hg in Group B at 1 minute after endotracheal intubation. The maximum increase in mean arterial BP was 125.71±8.19 mm of Hg in Group A and 114.67±12.83 mm of Hg in Group B at 1 minute after endotracheal intubation.

Conclusion: Our study concludes that inhaled 4% Lignocaine can control the hemodynamic changes of intubation more effectively than IV 2% Lignocaine.

KEYWORDS

Intubation Response, Intravenous Lignocaine, Inhaled Lignocaine.

INTRODUCTION

Pressor response with laryngoscopy is an important concern for anaesthesiologist. Laryngoscopy and intubation causes exaggerated hemodynamic response causing tachycardia, hypertension, dysrhythmias and it can increase intracranial, intraocular pressures which might be detrimental in patients at risk of myocardial infarction, cerebrovascular accidents, glaucoma etc.[1] Ideally all the patients who needs endotracheal intubation measures should be taken to blunt the pressor response. There are various factors influencing the pressor response like types of laryngoscope blade, induction drugs etc. There are studies which says that usage of video laryngoscopes, Mccoy blade reduces the pressor response when compared to the traditional Macintosh blades.[2,13] We can also use drugs like opioids, clonidine, dexmedetomidine, lignocaine, alpha and beta blockers, magnesium sulphate, calcium channel blockers to blunt the pressor response.[3-6,9,12] Lignocaine can be used in various routes like intravenous, sprays, aerosols and it is found to be effective in controlling the pressor response. The added advantage of lignocaine is its bronchodilatation and anti arrhythmic property.[7] So we conducted a study to compare the effects of intravenous and nebulised Lignocaine in controlling the intubation response.

METHODOLOGY

After approval from the institutional ethics committee, 60 patients were selected for the study. Randomization was done by sealed envelope technique. Group A received injection Lignocaine 2% 1.5mg/kg 90 seconds prior to intubation. Group B received nebulization with Lignocaine 4% (4 ml) 15 min prior to intubation. All the patients underwent a thorough pre- anaesthetic evaluation on the previous day of the surgery and an informed written consent will be taken from parents/guardian. Basic lab investigations like CBC, Bleeding time, Clotting time, Coagulation profile(if needed), Chest X-ray(if needed) and ECG(if needed) were done. In the preoperative room group B received nebulization 15 minutes prior to the procedure. Monitors like pulse oxymeter, ECG leads and NIBP(non-invasive blood pressure) cuff were attached and baseline values were recorded. On arrival into the operation theatre, standard ASA monitors were connected. Both the group patients were premedicated with injection Midazolam 1mg iv, induced with injection Propofol 1.5mg/kg and paralysed with injection Vecuronium 0.1mg/kg and intubated with appropriate sized cuffed endotracheal tubes, injection Fentanyl 1.5mcg/kg was given post intubation. Adequate depth of anaesthesia was maintained with N_2O, O_2 and Isoflurane and intermittent dose of Vecuronium. Baseline hemodynamic parameters heart rate, SBP, DBP, MAP every minute interval for the first 5minutes post intubation and every 2 minutes for the next 10 minutes were noted. Post procedure patients were reversed with neostigmine and glycopyrolate and extubated.

RESULTS

In this study, 60 patients were randomly allocated to two treatment groups (30 patients/group) and analyzed. The demographic data like age, weight and height were compared in both the groups and there was no significant difference (P value = 1). This indicates that the randomization process had created two comparative groups.

Table 1: Comparison of Heart rate

HR	GROUP A(n=30)	GROUP B(n=30)	P VALUE
	Mean ± SD	Mean ± SD	
BASELINE	87±11.24	87.90±21.50	0.84
1 MIN	109.73±15.34	95.00±32.334	0.001
2 MIN	103.93±19.3	85±15.342	0.029
3 MIN	100.8±15.39	80.670±22.576	0.001
4 MIN	96.6±14.23	79.670±15.320	<0.001
5 MIN	92.8±14.49	80.330±13.425	<0.001
7 MIN	92.37±13.85	85.330±13.654	0.001
9 MIN	88.33±12.46	83.330±15.698	0.071
11 MIN	85.07±12.29	78.000±15.544	0.175
13 MIN	85.77±10.24	78.670±14.408	0.045
15 MIN	85.77±10.24	77.000±15.544	0.042

Table 1 shows the comparison of heart rate at different time intervals in both the groups. In group A, the basal HR was 87±11.24 bpm, 1 minute after intubation, it was 109.73±15.3. Subsequently, the elevated heart rate started settling down 4 minutes. By 3 and 4 minutes it was 100.8±15.39 bpm and 96.6±14.23bpm respectively. In group B, the basal HR was 87.90±21.50bpm, 1 minute after intubation, it was 95.00±32.334. Subsequently, the elevated heart rate started settling down by 3 minute. By 3 and 4 minutes it was 80.670±22.576 and

79.670±15.320 bpm respectively. There was significant difference in the heart rate between two groups between 1st to 7th minute.

Table 2: Comparison of Systolic BP

SBP	GROUP A(n=30)	GROUP B(n=30)	P VALUE
	Mean ± SD	Mean ± SD	
BASELINE	123.53±2.54	123.17±10.84	0.91982
1 MIN	166.7±20.5	144.67±17.43	0.0002
2 MIN	158.4±15.46	138.73±18.93	0.00136
3 MIN	145.1±6.34	134±20.49	0.034
4 MIN	138.53±4.87	130.4±16.51	0.11733
5 MIN	136.33±14.76	127.97±14.33	0.0729
7 MIN	133.93±22.85	128.93±12.95	0.1433
9 MIN	131.33±19.52	125.37±10.53	0.0857
11 MIN	127.93±14.48	128.13±13.4	0.9583352
13 MIN	130.6±13.53	129±11.09	0.67429
15 MIN	126.46±12.24	129±11.09	0.44301

Table 2 showing changes in Mean Systolic Blood Pressure (SBP).In group A the basal value of SBP was 123.53±2.54 mm Hg, 1 minute following intubation, the SBP increased by 166.7±20.5mm Hg. This elevated pressure started coming down by 5 minutes. By 4 minutes and 5 minutes it was 138.53±4.87 mm Hg and 136.33±14.76 mm Hg respectively. In group B the basal value of SBP was 123.17±10.84 mm Hg, 1 minute following intubation, the SBP increased by 144.67±17.43 mm Hg. This elevated pressure started coming downby 3 minutes. By 3 minutes and 4 minutes it was 134±20.49 mm Hg and 130.4±16.51 mm Hg respectively. There was statistical significance between two groups in the 1st, 2nd and 3rd minute.

Table 3: Comparison of Diastolic BP

DBP	GROUP A(n=30)	GROUP B(n=30)	P VALUE
	Mean ± SD	Mean ± SD	
BASELINE	77.4±18.52	78.87±7.89	0.5369
1 MIN	105.13±11.78	97.27±12.2	0.021
2 MIN	96±10.81	89.57±10.65	0.0734
3 MIN	89.3±26.77	85.2±11.75	0.19
4 MIN	89 ±15.56	83.47±12.05	0.1
5 MIN	87±8.4	83.3±10.21	0.2
7 MIN	82.5±9.84	84.83±11.01	0.44
9 MIN	85.5±10.71	82.47±8.14	0.15
11 MIN	85 ±9.79	82.23±9.24	0.2
13 MIN	84±11.06	83.57±7.7	0.8
15 MIN	84±12.71	83.57±7.7	0.7

Table 3 shows the changes in diastolic BP in both the groups at various time intervals. In group A the basal value of DBP was 77.4±18.52 mm Hg, at 1 minute following intubation, the DBP increased by 105.13±11.78mm Hg. This elevated pressure started coming down by 3 minutes. By 3 minutes and 4 minutes it was 89.3±26.77 mm Hg and 89 ±15.56 mm Hg respectively. In group B the basal value of DBP was 78.87±7.89 mm Hg, at 1 minute following intubation, the DBP increased by 97.27±12.2 mm Hg. This elevated pressure started coming down by 3 minutes. By 3 minutes and 4 minutes it was 85.2±11.75 mm Hg and 83.47±12.05mm Hg respectively. There was statistical significant difference between two groups in 1st minute (P = 0.021).

Table 4 shows the changes in Mean arterial pressures in both the groups at various time intervals. The baseline MAP was 92.8±11.15 mm of Hg and 93.63±8.07 mm of Hg in Group A and Group B respectively. It increased to 125.71±8.19 mm of Hg in Group A and 114.67±12.83 mm of Hg in Group B at 1 minute after intubation which is statistically very significant (P=0.0048). The elevated MAP started to settle by 5 minutes to 103.3±4.62 in Group A and 98.03±10.4 in Group B.

Table 4: Comparison of Mean arterial pressure

MAP	GROUP A(n=30)	GROUP B(n=30)	P VALUE
	Mean ± sd	Mean ± sd	
BASELINE	92.8±11.15	93.63±8.07	0.742
1 MIN	125.71±8.19	114.67±12.83	0.0048
2 MIN	116.8±18.67	105.87±12.44	0.003
3 MIN	108±21.53	101.13±13.71	0.05
4 MIN	105.78±16.82	98.9±12.43	0.033
5 MIN	103.3±4.62	98.03±10.4	0.062
7 MIN	99.67±11.78	99.2±10.58	0.861
9 MIN	101±5.53	96.87±7.9	0.08
11 MIN	99.27±1.27	97.47±9.2	0.458
13 MIN	99.58±3.14	98.83±7.72	0.764
15 MIN	98.47±0.48	98.83±7.72	0.868

DISCUSSION

According to the findings of this study, there is a significant difference in heart rate, systolic and diastolic BP between both the groups. In group A, the basal HR was 87±11.24 bpm, 1 minute after intubation, it was 109.73±15.3. There was a difference of approximately 30 bpm. Subsequently, the elevated heart rate started settling down 4 minutes. By 3 and 4 minutes it was 100.8±15.39 bpm and 96.6±14.23 bpm respectively. In a study conducted by Prasad SR et al.[10] the baseline heart rate was 83.80±10.62 bpm and it increased to 115.40±11.16 bpm after intubation, a difference of approximately 32 bpm which is similar to our study results. In another study conducted by Supriya s et al.[8] the average rise in heart rate was 18 bpm above the base line in the group which received intravenous lignocaine. Jokar A et al.[11] in their study found that the change in heart rate in intravenous group was from 88.11 bpm to 90.12 bpm after intubation. In group B, the basal HR was 87.90±21.50 bpm, 1 minute after intubation, it was 95.00±32.334. There was a difference of approximately 8 bpm. Subsequently, the elevated heart rate started settling down by 3 minute. By 3 and 4 minutes it was 80.670±22.576 and 79.670±15.320 bpm respectively. There was significant difference in the heart rate between two groups between 1st to 7th minute. In the study conducted by Supriya s et al.[8] the average rise in heart rate was 24.86 bpm above the base line in nebulisation group. The possible explanation could be we used higher percentage of lignocaine (4%) for nebulisation whereas they used 2% lignocaine. Jokar A et al.[11] in their study found that the change in heart rate in nebulisation group was from 89.14 bpm to 77.37 bpm after intubation which is similar to our study results. In group A, the basal SBP was 123.53±2.54 mm Hg, 1 minute following intubation, the SBP increased by 166.7±20.5mm Hg. The elevated pressure started coming down by 5 minutes. By 4 minutes and 5 minutes it was 138.53±4.87 mm Hg and 136.33±14.76 mm Hg respectively. Prasad SR et al.[10] in their study found that the baseline mean arterial pressure was 92.60±10.40 mm of Hg and it increased to 113.20±11.32 mm of Hg after intubation in the group which received intravenous lignocaine. In the study conducted by Supriya s et al.[8] the baseline SBP was 119.23±11.62 mm of Hg and the SBP after 1 minute after intubation was 139.77±13.40 mm of Hg in the group which received intravenous lignocaine which is similar to our results. In group B, the basal SBP was 123.17±10.84 mm Hg, 1 minute following intubation, the SBP increased by 144.67±17.43 mm Hg. This elevated pressure started coming down by 3 minutes. By 3 minutes and 4 minutes it was 134±20.49 mm Hg and 130.4±16.51 mm Hg respectively. In the study conducted by Supriya s et al.[8] the baseline SBP was 123.17±10.84 mm of Hg and the SBP after 1 minute after intubation was 155.43±14.89 mm of Hg in the group which received lignocaine nebulisation. The possible explanation for the difference in the results from our study could be the usage of 2% lignocaine for nebulisation. In group A, the basal DBP was 77.4±18.52 mm Hg, 1 minute following intubation, the DBP increased by 105.13±11.78 mm Hg. The elevated pressure started coming down by 5 minutes. By 4 minutes and 5 minutes it was 89 ±15.56 mm Hg and 87±8.4 mm Hg respectively. In the study conducted by Supriya s et al.[8] the baseline DBP was 77.93±9.72 mm of Hg and the DBP after 1 minute after intubation was 91.77±11.12 mm of Hg in the group which received intravenous Lignocaine which is similar to our results. In group B, the basal DBP was 78.87±7.89 mm Hg, 1 minute following intubation, the DBP increased by 97.27±12.2 mm Hg. This elevated pressure started coming down by 3 minutes. By 3 minutes and 4 minutes it was 85.2±11.75 mm Hg and 83.47±12.05 mm Hg respectively. In the study conducted by Supriya s et al.[8] the baseline DBP was 78.87±7.89 mm of Hg and the DBP after 1 minute after intubation was 103.70±11.21 mm of Hg in the group which received Lignocaine nebulisation. The possible explanation for the difference in the results from our study could be the usage of 2% Lignocaine for nebulisation. In our study the baseline MAP was 92.8±11.15 mm of Hg and 93.63±8.07 mm of Hg in Group A and Group B respectively. It increased to 125.71±8.19 mm of Hg in Group A and 114.67±12.83 mm of Hg in Group B at 1 minute after intubation. Jokar A et al.[11] in their study found that the change in MAP intravenous group was from 97.54 mm of Hg to 89.79 mm of Hg 2 minutes post intubation and in nebulisation group was from 97.06 mm of Hg to

87.15 mm of Hg 2 minutes post intubation which is similar to our results.

CONCLUSION

In our study we found that nebulisation with 4% Lignocaine 15 minutes prior to intubation was more effective in blunting the intubation responses to laryngoscopy when compared to 1.5 mg/kg intravenous 2% Lignocaine given 90 seconds prior to intubation in ASA I-II patients. We suggest the future studies should focus on evaluation of attenuation of intubation response by higher concentration of inhalational Lignocaine.

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