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To study the hemodynamic variation to laryngoscopy and endotracheal intubation while comparing the effectiveness of nebulised VS intra-venous form of 2% lidocaine to attenuate the sympathetic response to laryngoscopy in Indian population

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Abstract
Various clinical publications have already established tachycardia and hypertension as potential side effects of laryngoscopy and endotracheal intubation.
Notably, the exaggerated sympathetic response during laryngoscopy has been found to be a key factor in causing this more common than not desired complication. Although transient; such an exaggerated sympathetic response may lead to cardiac as well as CNS catastrophe in the sub-set of patients with known or unknown pre-existing cardiac or CNS disease. Not only such adverse events increase morbidity and mortality amongst patients they also increase the burden on health care infrastructure.
Various methods to attenuate this response including but not restricted to use of both intravenous and nebulized lidocaine have already been established in literature. However not many studies have been carried out in the Indian sub set population to compare the effect of intra venous lidocaine vs nebulized lidocaine. In view of study to compare the effect of 2 % preservative free intravenous lidocaine with 2% nebulized lidocaine in ASA I and II patients listed for elective surgeries under general anaesthesia in a service hospital of the armed forces was carried out between Jul 2022 to Jun 2023.
Materials and Methods: After taking approval of the institute ethical committee and informed consent we included seventy five ASA I & II patients of both the genders between ages 20-65 year who were posted for elective surgeries under general anaesthesia. Patients were then divided in three groups equally keeping the confounders of anthropology, ASA grade and obvious difficult airway same. In group A, standard anaesthesia technique was used to facilitate laryngoscopy and endotracheal intubation using Fentanyl @2 mcg/kg, Propofol 2mg/kg and Atracurium @ 0.5mg/kg Group B received nebulization with 2% lignocaine @ 1.5 mg/kg 10 minutes prior to the endotracheal intubation and Group C received 2% lignocaine @ 1.5 mg/kg intravenous 90 sec before induction.
The variables included in the study are heart rate, systolic and diastolic blood pressure and mean arterial pressure. Basal values and values subsequently at 1st, 3rd, 5th, and 10th minute after intubation were recorded.
Those patients who required laryngoscopy for more than 30 secs or requiring more than two attempts were excluded from the study.
Results: In all the 3 groups post endotracheal intubation demonstrated an increase in heart rate, systolic BP, Diastolic BP and mean arterial BP. However the minimum change in hemodynamic and thus most effective way of attenuating the sympathetic response to laryngoscopy and intubation was seen in the subgroup where lidocaine was administered intravenously.
Conclusion: In the present study we conclude that the use of lignocaine when used in combination with opioid for laryngoscopy and endotracheal intubations reduces the increase in heart rate and blood Pressure. However the attenuation of exaggerated sympathetic response was much more in the subset of patients where intravenous lidocaine was used.

Keywords: Laryngoscopy, endotracheal intubation, cardiovascular response, nebulized lidocaine

Introduction
Balanced anaesthesia techniques mandate minimum hemodynamic disturbances during the peri-operative period. Although transient, laryngoscopy and endotracheal intubation by far have been described as the most important causes in increasing the heart rate and blood pressure peri-operatively [1].

Tachycardia and hypertension due to laryngoscopy and endotracheal intubation can increase systolic blood pressure by 40-50% and heart rate to 20 per minute [2]. More common than not these responses are well tolerated in healthy individuals, nonetheless they may lead to various complications including but not restricted to myocardial ischemia, cerebral haemorrhage in susceptible subset of patients [3]. The cardio-vascular response is mediated vide the Xth and XIth Cranial nerves via the afferent impulses carried from epiglottis and infra-glottic regions which then activate the vasomotor centres leading to intense sympathetic response causing hypertension, tachycardia and arrhythmias [4, 5]. In patients with pre-existing sub-optimal cardiovascular and cerebro-vascular conditions it may lead to pulmonary oedema [6], myocardial ischemia [7] and cerebrovascular accidents [8]. Hence it is imperative to block all unwanted response that can increase the peri-operative morbidity and mortality.

Various non-pharmacological as well as pharmacological techniques to attenuate this unwanted response have already been published in literature. Use of supra glottic airway devices in the form of laryngeal mask airways, i-gel, decreasing the duration of laryngoscopy and blocking of glossopharyngeal & superior laryngeal nerves are such non pharmacological modalities that have established themselves as an effective tool in decreasing the exaggerated sympathetic response during airway intervention [9-12].

On the other hand use of topical or intravenous lignocaine, high dose opioids, α & β adrenergic blockers, calcium channel antagonists and vasodilators like nitro-glycerine are some of the pharmacological techniques which are used for the same [13-15].

Various clinical publications have established the use of topical anaesthesia with lignocaine in forms of viscous gargles, aerosols and oropharyngeal sprays and also of intravenous lidocaine are found to be popular methods in reducing the stress response to laryngoscopy and endotracheal intubation when used alone or in combination with other drugs [17-20]. Since most of these studies have been conducted in the western countries, a need was felt to conduct a study in the Indian sub set of patients and also to compare the attenuation of sympathetic response to laryngoscopy and endotracheal intubation while using lidocaine in its aerosolized form and intra-venous form and establish a safer and better route of administration of the drug.

Material and Methods

After taking approval of the institute ethical committee and informed consent we included seventy five ASA I & II patients of both the genders who were posted for elective surgeries under general anaesthesia. Patients in all the three groups were equally distributed in variables of age (20-65 years), ASA grades and obvious difficult airway. Group A received standard anaesthesia techniques using Ondansetron 4mg iv, Fentanyl @2mcg/Kg, Propofol @2mg/Kg and Atracurium @ 0.5mg/kg. In addition to the standard anaesthesia techniques Group B received nebulization with 2% lignocaine @ 1.5 mg/kg 10 minutes prior to the endotracheal intubation and Group C received 2% lignocaine @ 1.5 mg/kg intravenous 90 sec before induction. Standard anaesthesia monitoring techniques were ensured in all the 3 groups. The variables we included in the study are heart rate, systolic and diastolic blood pressure and mean arterial pressure. Basal and subsequent values of the included variables were recorded at 1st, 3rd, 5th, and 10th minute after intubation were recorded (Table 1 to 4 and Fig A to D).

Patients who were either unwilling to participate in the study or already on beta/ alpha blocking agents and those requiring laryngoscopy for more than 30 secs or more than 2 attempts were excluded from the study.

Group B received nebulization with 1.5 mg/kg of 2% Lidocaine diluted in 5 ml of 0.9% normal saline using Compressor Nebulizer (DeVilbiss-3655I) face mask10 min before induction.

Group C received 1.5 mg/kg of intra venous preservative free Lidocaine (Loxicard) 90 seconds before starting intra venous induction.

A conventionally trained anaesthesiologist performed Laryngoscopy and endotracheal intubation in all the subgroups using Macintosh size 3 blade and Gum elastic bougie to prevent airway manipulation and decrease the duration of laryngoscopy after loss of verbal response.

Anaesthesia was maintained using combination 50% nitrous oxide with 50% of oxygen and 1% sevoflurane. Minimum monitoring as per ASA standards were used in all the subgroups.

Statistical methods

Data was compiled in EXCEL sheet and Master sheet was prepared. For comparison of Quantitative variables of three groups unpaired t-test was used. p- Value < 0.05 was considered to be statistically significant (S) while p Value > 0.05 was considered Not significant (NS).

Results

Samples were then matched anthropometrically in terms of age, gender and weight (Group A mean 32.20±10.63, Group B mean 33.80±9.45 and Group C of 31.10±9.950) with t = 1.70 and p = 0.097. On the basis of weight (Group A mean 55.00±10.65, Group B mean 59.63±9.90 and Group C 57.00±10.01) with t = 1.08 and p = 0.24. There was no significant difference in age, gender and weight distribution in the two groups.

Table 1: Table depicting variations in Mean Heart Rate HR Group A Mean ± SD Group B Mean ± SD and Group c Mean ± SD t-value P-value Basal 84.97±10.23, 86.97±11.23 and 87.30±13.09 t=0.151 and P=0.91 NS

Post-intubation 1 Minute 138.71±17.3, 11.83±15.91 and 99.0±12.25 t=3.50 P=0.001 which was considered statistically significant.

Post intubations at 3 Minute 134.31±18.2, 105.87±16.46, and 96.90±15.01 t= 2.41 P=0.031 and found to be statistically significant.

At 5 Minute post intubation 121.2±19.4, 95.33±14.81and 93.36±12.91with t value 0.743 P=0.477. These result were not found to be statistically significant At 10 Minutes 81.74±9.1, 88.33±12.56 and 87.93±12.41 T value.124 and p value of 0.902 which was found to be statistically not significant.

Table 1: Means of heart rate at basal, 1, 3, 5 and 10 minutes

Heart Rate	Group A Mean ± SD	Group B Mean ± SD	Group C Mean ± SD	t- value	P value
Pre Anaesthesia	84.97±10.23	86.97±11.23	87.30±13.09	0.151	P=0.91 Not significant
1 Mins	138.71±17.3	111.83±15.91	99.00±12.25	3.50	P=0.001 Significant
3 Mins	134.31±18.2	105.87±16.46	96.90±15.01	2.41	P=0.031 Significant
5 mins	121.2±19.4	95.33±14.81	93.36±12.91	0.743	P=0.477 Not Significant
10 mins	81.74±9.1	88.33±12.56	87.93±12.41	0.124	P=0.902 Not Significant

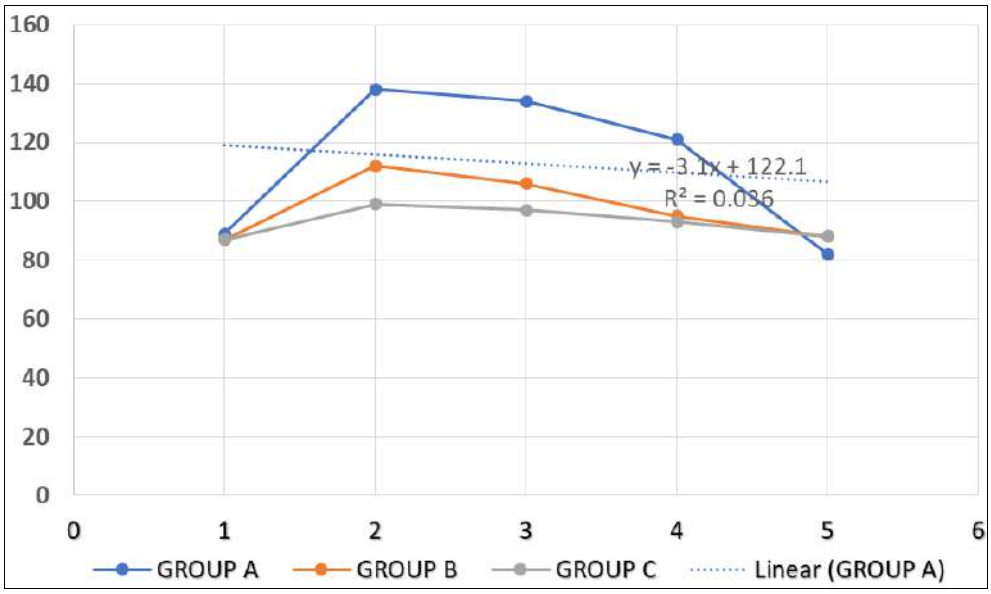

Fig 1: Mean heart rate in group a, b and c

Table 2: Table showing changes in Mean Systolic Blood Pressure (SBP) Group A Mean ± SD Group B Mean ± SD t-value P-value Base Line 112.1±12.2, 130.17±11.13, 132.10±15.18 0.247 P=0.629 Not significant
Post-intubation 1 Minute 148.71±22.3, 139.77±13.39, 135.10±14.68 t 2.09 P=0.039 Statistically significant
3Minute 151.31±19.2, 130.13±19.53, 121.97±17.851.98 P=0.031 S
5 Minute 150.2±19.4, 125.73±18.84, 114.23±17.08 t value2.44 P=0.016 S
10 Minute 81.74±9.1, 116.66±11.56, 113.73±17.35 t value 1.26 P=0.102 NS

Table 2: Means of diastolic Bp at basal, 1, 3, 5 and 10 minutes

Mean Systolic BP	Group A Mean ± SD	Group B Mean ± SD	Group C Mean ± SD	t- value	P value
Pre Anaesthesia	112.1±12.2	130.17±11.13	132.10±15.18	2.47	P=0.629 Not Significant
1 Mins	148.71±22.3	139.77±13.39	135.10±14.68	2.09	P=0.039 Significant
3 Mins	151.31±19.2	130.13±19.53	121.97±17.85	1.98	P=0.031 Significant
5 mins	150.2±19.4	125.73±18.84	114.23±17.08	2.44	P=0.016 Significant
10 mins	81.74±9.1	116.66±11.56	113.73±17.35	1.26	P=0.102 Not Significant

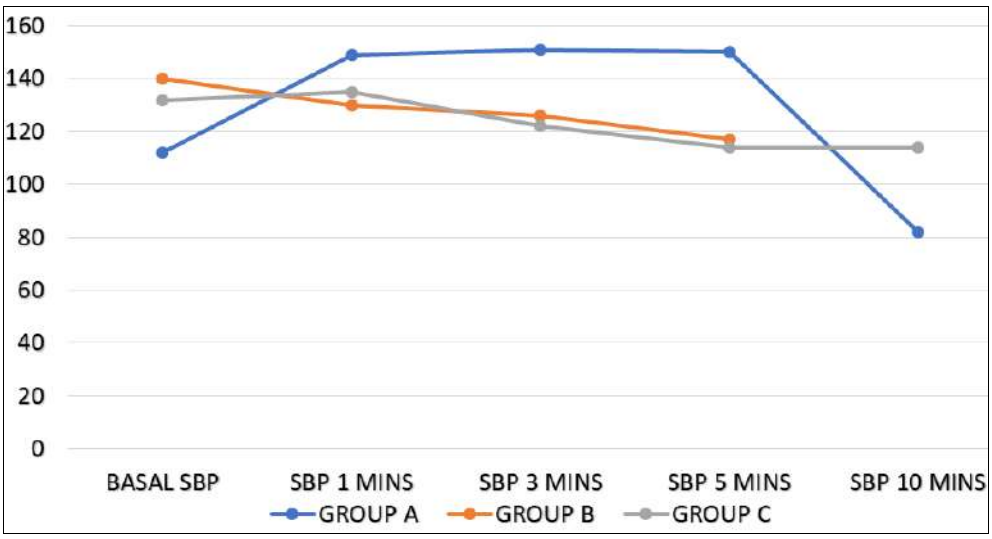

Fig 2: Means of systolic blood pressure at basal,1, 3, 5 and 10 minutes

Table 3: Table showing changes in Mean Diastolic Blood Pressure (SBP) Group A Mean ± SD Group B Mean ± SD t-value P-value Base Line 78.12±9.02, 83.26±7.83, 84.46±8.92 tvalue 0.553 and p value 0.582 which is statistically not significant.

At 1 minute post intubation 103±10.24, 87.0±11.20, 87.07±18.69, t value 4.18 and p of 0.0 which was statistically significant

At 3 mins post intubation 102±12.91, 89.90±10.79, 80.26±11.32 t value 3.37 and p of 0.001 which was statistically significant

At 5 minutes ost intubation 99±10.10, 81.90±11.27, 75.80±12.04 t value 2.25 and p value of 0.028 which was statistically significant

At 10 minutes post intubation 74±6.65, 78.93±5.36, 75.17±10.85 and t value 1.70 with p value of 0.094 which was statistically non-significant

Table 3: Means of diastolic Bp at basal,1, 3, 5 and 10 minutes

Mean Diastolic BP	Group A Mean ± SD	Group B Mean ± SD	Group C Mean ± SD	t- value	P value
Pre Anaesthesia	78.12±9.02	83.26±7.83	84.46±8.92	0.553	P=0.582 Not Significant
1 Mins	103±10.24	87.0±11.20	87.07±18.69	4.18	P=0.000 Significant
3 Mins	102±12.91	89.90±10.79	80.26±11.32	3.37	P=0.001 Significant
5 mins	99±10.10	81.90±11.27	75.80±12.04	2.25	P=0.028 Significant
10 mins	74±6.65	78.93±5.36	75.17±10.85	1.70	P=0.094 Not Significant

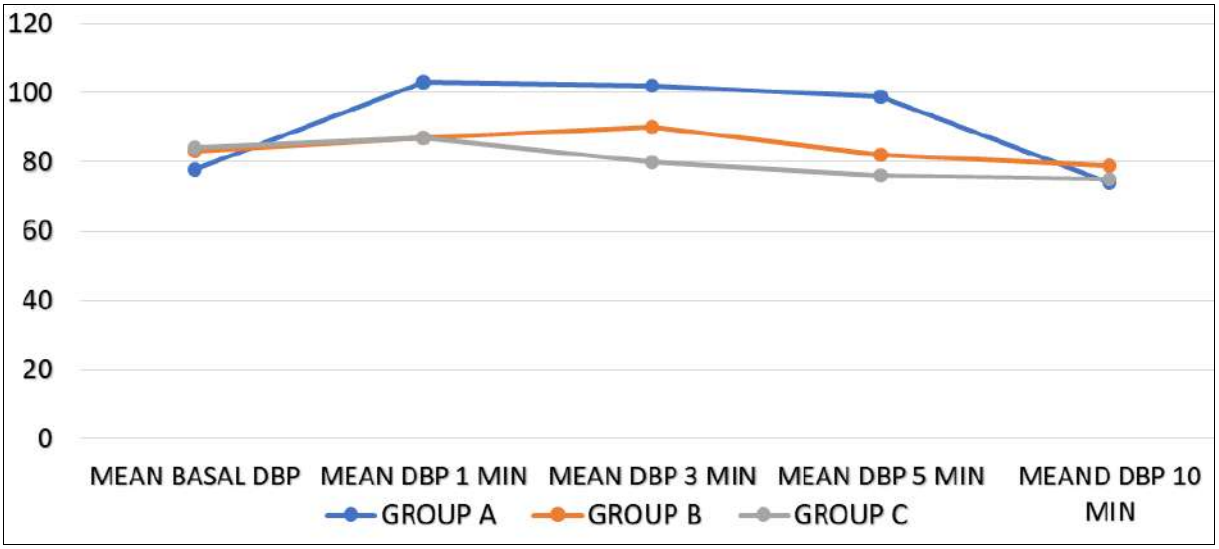

Fig 3: Means of diastolic blood pressure at basal, 1, 3, 5 and 10 minutes

Table 4: Table showing changes in Mean of Mean Arterial Pressure Group A Mean ± SD Group B Mean ± SD t-value P-value Base Line 99±7.99, 98.63±7.83, 100.23±10.05 tvalue 0.763 p value 0.642 which is statistically not significant.

At 1 minute post intubation 128±12.31,120.93±11.64, 105.00±11.01, t value 0.763 and p=0.642 NS which was statistically not significant

At 3 mins post intubation 108.91±10.31, 105.86±12.4494.23±12.23 t value 3.65 and p of 0.001 which was statistically significant.

At 5 minutes post intubation 111.34±9.91, 95.90±12.43, 88.16±12.77 t value 3.29 and p value of 0.001 which was statistically significant

At 10 minutes post intubation 97±5.51, 90.86±7.9, 87.96±12.3 and t value 1.1 with p value of p=0.128 which was statistically non-significant

Table 4: Means of mean arterial pressure at basal, 1, 3, 5 and 10 minutes

Mean MAP	Group A Mean ± SD	Group B Mean ± SD	Group C Mean ± SD	t- value	P value
Pre Anaesthesia	99±7.99	98.63±7.83	100.23±10.05	0.763	p=0.642 Not Significant
1 Mins	128±12.31	120.93±11.64	105.00±11.01	5.44	p=0.000 Significant
3 Mins	108.91±10.31	105.86±12.44	94.23±12.23	3.65	p=0.001 Significant
5 mins	111.34±9.91	95.90±12.43	88.16±12.77	3.29	p=0.001 Significant
10 mins	97±5.51	90.86±7.9	87.96±12.3	1.1	p=0.128 Not Significant

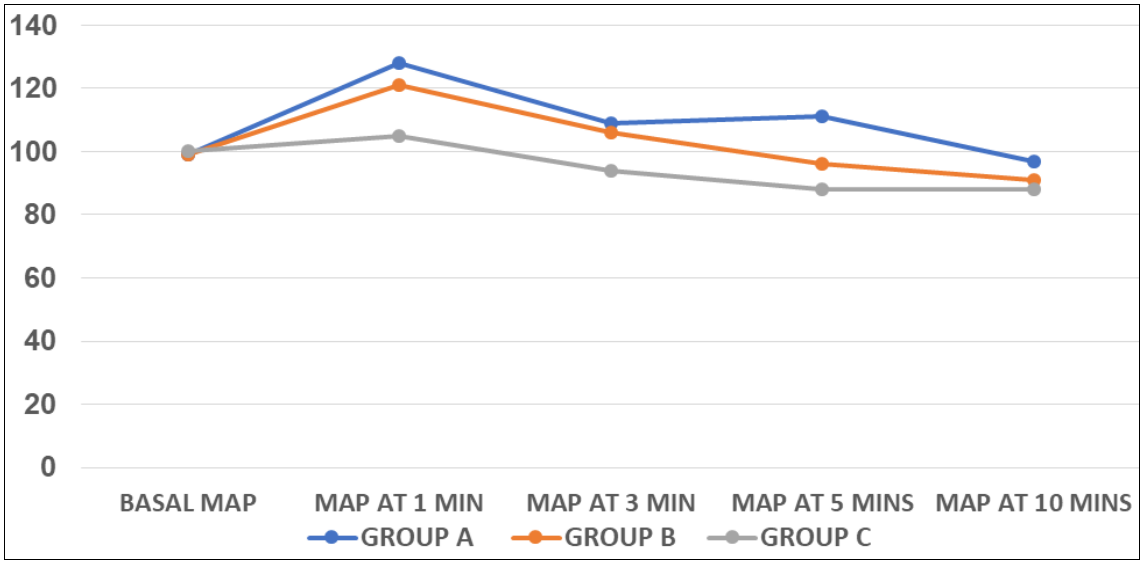

Fig 4: Means of mean arterial pressure at basal, 1, 3, 5 and 10 minutes

Discussion

Local anaesthetic agents like lignocaine has already established its efficacy and practicability to attenuate the hemodynamic response to laryngoscopy and endotracheal intubation. Various routes including but not restricted to gargle [17], aerosol [18] intravenous [19] lignocaine topical spray have been established to blunt the hemodynamic response to laryngoscopy have been published in literature.

Mechanisms including but not restricted to as follows have been prescribed in literature as the mode of action of lidocaine for the desired effect

a) Myocardial depression [22].
b) Peripheral vasodilatation [22].
c) Effective prophylaxis and treatment laryngospasm [23].
d) Analgesic properties when given intravenously [24].
e) Suppression of airway reflexes elicited by irritation of epi-pharyngeal and laryngopharyngeal mucosa [25].
f) Antiarrhythmic properties [26].
g) Depression of autonomic nervous system [27].

Gianelly *et al* established 2 to 5 mcg/ml plasma concentration of lidocaine as safe for preventing hemodynamic disturbance. However at concentration of greater than 9 mcg/ml the side effects may occur [28]. Adriani *et al* postulated that the absorption of aerosolized lidocaine is vide the pulmonary alveoli and this method is generally considered to be safe, simple, and effective [29].

The present study is aimed to to evaluate the efficacy of intravenously administered 2% lignocaine vs aerosolized 2% lidocaine to blunt the hemodynamic response to endotracheal intubation. Various other studies *viz*.

Mounir Abou-Madi *et al*. [18] and Stanlay Tarn *et al*. [30] and Mohan K, Mohana Rupa L observed that intravenous lignocaine at a dose of 1.5 mg/kg attenuated the increase in Heart rate (HR) and Arterial Blood Pressure (ABP), only when given 3 min, before intubation. Bahaman Venus, Ahmed M. *et al*., have already established the efficacy of 2% Lidocaine in the nebulized form as an effective method of delivery in preventing the pressor response and tachycardia when used in a patient 15 minutes prior to endotracheal intubation.

In the present study we also used 1.5 mg/ kg of 2% lignocaine intravenous for attenuation of pressor response and preferred to give 90 sec before induction and intubation while following standard anaesthesia techniques. We also observed that the use of nebulized Lidocaine along with fentanyl was not as effective in decreasing the pressor response to endotracheal intubation as the combination of intra venous form of lidocaine. One of the following reasons may be responsible for this sub optimal response.

a) Along with the pressure on mucous membrane the laryngoscope blade also applies pressure on deep proprio receptors located sub-mucosal region. Since these deep proprio receptors are not optimally blocked by aerosolized lidocaine the response is clinically sub optimal [31].
b) Up to 60 % of administered lignocaine can be lost via nebulized route in the patient's oral cavity leading to sub optimal anaesthesia in the Tracheal [32]
c) Aerosolized lidocaine partially inhibits vagal afferents leading to unopposed pressor response [33]

Results

Data Analysis of all the variables in all the 3 groups (as shown in Table 1-4 and Figure A -D) the present study shows that a maximum increase in heart rate, systolic, diastolic BP and MAP were seen in the group where lidocaine was not used in any form. Moreover it is also seen that intra-venous form of Lidocaine is safe, and more efficacious to attenuate the sympathetic response to laryngoscopy and intubation.

Recommendation

Based on the study it is recommended that the intra-venous form of Lidocaine in the dose of 1.5 mg/Kg may be considered safe and should be included unless contraindicated due to reasons including but not restricted to allergic response in patients to attenuate the hemodynamic response to laryngoscopy and endotracheal intubation. It is also recommended that intra venous lidocaine may be used as one of the drug in the armamentarium of anaesthesiologist while dealing with patients with known cardiac diseases.

Conflict of Interest

Not available

Financial Support
Not available

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