Original Article

Comparative study of cardiovascular response to laryngoscopy and tracheal intubation following intravenous lignocaine with lignocaine nebulization

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Abstract

Introduction: The major responsibility of an anaesthesiologist is the management of airway to provide adequate ventilation to the patient by securing airway during general anaesthesia. As such, no anaesthesia is safe unless diligent efforts are devoted to maintain an intact functional airway. **Aims and Objectives:** To Study of Cardiovascular Response to Laryngoscopy and Tracheal Intubation Following Intravenous Lignocaine with Lignocaine Nebulization. **Methodology:** It was a Comparative, undertaken in Victoria hospital and Bowring and Lady Curzon hospitals, Bangalore during November 2008 to 2010. Ethical clearance was obtained for the study. The study was conducted on 90 ASA grade I and II patients in the age group of 18 to 45 years of either sex scheduled for elective surgeries done under general anaesthesia. Patients were allocated into three groups with the sample size of 30 each.Group C (n=30) received no drug, as control. Group I (n=30) received 2% Lignocaine 2mg/kg slow intravenous. Group N (n=30) received 2% nebulization of Lignocaine 2mg/kg. Descriptive statistical analysis has been carried out in the present study. Analysis of variance (ANOVA) has been used to find the significance of study parameters between three or more groups of patients, Chi-square/ Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups. **Result:** The maximum rise in heart rate was noted at 1 min following intubation in all the three groups. The mean rise in Heart rate at 1 minute in control group was 23.4bpm compared to 18bpm and 24.86bpm in group I and group N respectively. The maximum rise in Systolic blood pressure was noted at 1 min following intubation in all the three groups. The mean rise in Systolic blood pressure in control group was 42.6mm Hg compared to 17.54mm Hg and 32.26mm Hg in group I and group N respectively. The maximum rise in Diastolic blood pressure was noted at 1 min following intubation in all the three groups. The mean rise in Diastolic blood pressure in control group was 25.36 mm Hg compared to 13.84 mm Hg and 24.83 mm Hg in group I and group N respectively. The maximum rise in Mean arterial blood pressure was noted at 1 min following intubation in all the three groups. The mean rise in Mean arterial blood pressure in control group was 29.44 mm Hg compared to 16.1 mm Hg and 27.3 mm Hg in group I and group N respectively. **Conclusion:** In our study though nebulization was not effective in blunting the haemodynamic responses to laryngoscopy and intubation, it is still a safe and a simple technique to be used.

Key Words: Cardiovascular Response to Laryngoscopy and Tracheal Intubation, Intravenous Lignocaine, Lignocaine Nebulization.

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INTRODUCTION

The major responsibility of an anaesthesiologist is the management of airway to provide adequate ventilation to the patient by securing airway during general anaesthesia. As such, no anaesthesia is safe unless diligent efforts are devoted to maintain an intact functional airway. Endotracheal intubation is the overall accepted *'Gold standard of securing* the airway and providing adequate ventilation. However, endotracheal intubation requires time, a skilled anaesthesiologist, appropriate instruments and adequate circumstances with respect to space and illumination. Direct laryngoscopy and endotracheal intubation following induction of anaesthesia is almost always associated with hemodynamic changes due to reflex sympathetic discharge caused by epipharyngeal and laryngopharyngeal stimulation.[1] This increased

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sympatho adrenal activity may result in hypertension, tachycardia and arrhythmias.[2,3,4] This increase in blood pressure and heart rate are usually transitory, variable and unpredictable. Transitory hypertension and tachycardia are probably of no consequence in healthy individuals[5] but either or both may be hazardous to patients with hypertension, myocardial insufficiency, penetrating eye injuries, intracranial lesion, or cerebrovascular diseases. This laryngoscopic reaction in such individuals may predispose to development of pulmonary oedema[6] myocardial insufficiency[7] and cerebrovascular accident.[8]At least in such individuals there is a necessity to blunt these harmful laryngoscopic reactions. Attenuation of pressor responses to manipulation of the airway has been practiced either by deepening the plane of anaesthesia,[9, 10] by the use of drugs known to obtund them or by using advanced airway devices.[11, 12] Many methods have been devised to reduce the extent of hemodynamic events including high dose of opioids,[5,13]alpha and beta adrenergic blockers,[14,15] calcium channel antagonist like diltiazem, verapamil[16]and vasodilatation drugs like nitroglycerine.[17] α_2 – agonist like Clonidine[18] and Dexmedetomidine are used[19] Various studies have reviewed the effect of Lignocaine in forms like viscous lignocaine[20] aerosols,[21]oropharyngeal sprays [22] and intravenous[23, 24]route to blunt these responses. Topical anaesthesia with lignocaine applied to the larynx and trachea in a variety of ways remains a popular method used alone or in combination with other techniques. Intravenous lignocaine has been used to supress cough during tracheal intubation,[25] laryngospasm and cough during extubation.[26] It has also been used to suppress airway hyperactivity and mitigate bronchoconstriction after tracheal intubation.[27] In a study using intravenous and inhaled lignocaine, lignocaine in both the routes attenuated reflex bronchoconstriction significantly. Lignocaine plasma concentrations were significantly lower in the group where lignocaine was used via inhalational route.[28] Intravenous lignocaine with its well established centrally depressant and anti-arrhythmic effect was found to be a more suitable alternate method to minimize this pressor response.[23, 24] The present study was undertaken to compare the effect of Intravenous lignocaine and nebulization of lignocaine on blunting the haemodynamic responses to endotracheal intubation.

METHODOLOGY

A Study entitled Comparative study of lignocaine nebulization with intravenous lignocaine on stress response to laryngoscopy and tracheal intubation was undertaken in Victoria hospital and Bowring and Lady Curzon hospitals, Bangalore during November 2008 to 2010. Ethical clearance was obtained for the study.The study was conducted on 90 ASA grade I and II patients in the age group of 18 to 45 years of either sex scheduled for elective surgeries done under general anaesthesia. Patients were allocated into three groups with the sample size of 30 each. Group C (n=30) received no drug, as control.Group I (n=30) received 2% Lignocaine 2mg/kg slow intravenous. Group N (n=30) received 2% nebulization of Lignocaine 2mg/kg. Patients with chronic obstructive lung disease, cerebrovascular disease, cardiovascular diseases, psychiatric illness, liver disorders. Patients with known allergy to Lignocaine and its preservatives .Patients coming for emergency surgical procedures. Patients with history of laryngeal or tracheal surgery or any pathology.A detailed pre-anaesthetic evaluation including history of previous illness, previous surgeries, general physical examination, and detailed examination of Cardiovascular system, Respiratory system and other relevant systems were done. Baseline investigations were carried out and recorded in the proforma. The following cardiovascular parameters were recorded in all patients: Heart rate (HR) in beats per minutes (bpm) ,Systolic blood pressure (SBP) in mm Hg, Diastolic blood pressure (DBP) in mm Hg, Mean arterial pressure (MAP) in mm Hg. Descriptive statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean ± SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5 % level of significance. Analysis of variance (ANOVA) has been used to find the significance of study parameters between three or more groups of patients, Chi-square/ Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups. The Statistical software namely SAS 9.2, SPSS 15.0, Stata 10.1, MedCalc 9.0.1, Systat 12.0 and R environment ver.2.11.1 were used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables etc**.**

RESULT

Table 1: Table showing changes in Mean Heart Rate

HR (bpm)	Group C	Group I	Group N	Significant value		
				Group C-Group I	Group C-Group N	Group I-Group N
Basal	85.50±10.30	86.13±10.27	86.97±11.24	0.971	0.854	0.950
Post intubations						
1 min	108.90±14.13	104.13±11.85	111.83±15.91	0.392	0.699	0.092+
2 min	104.87±14.73	103.53±12.42	109.73±15.34	0.930	0.385	0.215
3 min	100.10±13.93	101.03±15.07	105.87±16.47	0.969	0.310	0.438
4 min	95.80±12.79	93.63±13.34	100.8±15.39	0.818	0.348	0.119
5 min	95.07±10.85	93.60±11.79	95.93±14.81	0.894	0.962	0.754
7 min	94.57±11.48	89.47±10.17	92.30±14.94	0.252	0.758	0.650
9 min	91.33±12.12	87.00±7.72	91.60±14.71	0.338	0.996	0.296
11 min	89.13±10.95	87.27±12.4	88.33±12.57	0.819	0.964	0.937
13 min	87.57±8.53	86.00±9.87	85.53±12.50	0.830	0.732	0.984
15 min	85.13±9.36	84.20±12.53	86.63±12.04	0.946	0.867	0.687

In the control group, the basal HR was 85.50 bpm. One minute after intubation, it was 108.90, representing a rise of 23.4bpm. Subsequently, the elevated heart rate started settling down by 9 min By 3and 5 min it was 100 and 95.07 bpm respectively. The increase in HR at 1 minute after intubation compared. In group I, the basal HR was 86.13 bpm, 1 minute after intubation, it was 104.13 representing a rise of 18 bpm. Subsequently, the elevated heart rate started settling down 9minute. By 3 and by 5 minutes it was 101.03 and 93.6 bpm respectively. In group N, the basal HR was 86.97 bpm, 1 minute after intubation, it was 111.83 representing a rise of 24.86 bpm. Subsequently, the elevated heart rate started settling down by 11 minute. By 3 and by 5 minutes it was 105.87 and 95.93bpm respectively. When mean change in heart rate in first minute in group I and group N were compared with control (group C) group independently, there was no clinical or statistical significance.(group C v/s group I P = 0.392 , group C v/s group N p = 0.699)Intergroup comparison of change in heart rate in first minute between the study groups (group N and group I) showed no clinical or statistical significance (p = 0.092).

Table 2: Changes in the Mean Systolic Blood Pressure (SBP)

SBP (mm Hg)	Group C	Group I	Group N	Significant values		
				Group C-Group I	Group C-Group N	Group I-Group N
Basal	121.73±15.84	119.23±11.62	123.17±10.84	0.736	0.904	0.471
Post intubations						
1 min	164.33±22.17	139.77±13.40	155.43±14.89	<0.001**	0.119	0.002**
2 min	156.63±24.57	139.70±17.54	149.37±17.29	0.004**	0.345	0.155
3 min	148.00±18.20	130.13±19.53	138.40±18.67	0.001**	0.124	0.210
4 min	143.63±19.25	125.60±20.92	133.07±19.67	0.002**	0.106	0.321
5 min	139.63±17.58	125.73±18.84	129.67±15.63	0.007**	0.074+	0.657
7 min	134.73±15.30	126.53±15.25	127.47±13.65	0.085+	0.143	0.967
9 min	130.53±14.57	122.57±12.67	127.97±12.44	0.057+	0.735	0.261
11 min	128.63±12.95	124.67±11.24	125.37±10.53	0.387	0.524	0.970
13 min	130.60±13.64	124.20±13.12	128.00±12.51	0.147	0.723	0.502
15 min	127.50±13.37	125.60±13.41	128.60±11.25	0.832	0.940	0.633

Table showing changes in Mean Systolic Blood Pressure (SBP).In the control group the basal value of SBP was 121.73 mm Hg, 1 minute following intubation, the SBP increased by 164.33 mm Hg, representing a rise of 42.6 mm Hg. This elevated pressure started coming down by 3 minutes. By 3 minutes and by 5 minutes it was 148 mm Hg and 139.63mm Hg respectively. In group I the basal value of SBP was 119.23 mm Hg, 1 minute following intubation, the SBP increased by 139.77 mm Hg, representing a rise of 17.54 mm Hg. This elevated pressure started coming down by 3 minutes. By 3 minutes and by 5 minutes it was 130.13 mm Hg and 125.73 mm Hg respectively. In group N the basal value of SBP was 123.17 mm Hg, 1 minute following intubation, the SBP increased by 155.43 mm Hg, representing a rise of 32.26 mm Hg. This elevated pressure started coming down by 3 minutes. By 3 minutes and by 5 minutes it was 138.40 mm Hg and 129.67 mm Hg respectively. Statistical evaluation between the groups showed that the increase in SBP observed in control group was statistically highly significant when compared to increase in SBP in group I and N. The increase in SBP in group C and group I were statistically highly significant compared to increase in SBP in group N($p < 0.001$) and remained significant even up to 5minute post intubation. Between group C and group N was no statistical significance. Between group I and group N, the increase in SBP in group N was statistically significant compared to increase in SBP in group I ($p < 0.002$).

Table 3: Table showing changes in Mean Diastolic Blood Pressure (DBP)

DBP (mm Hg)	Group C	Group I	Group N	Significant values		
				Group C-Group I	Group C-Group N	Group I-Group N
Basal	78.27±8.75	77.93±9.72	78.87±7.89	0.988	0.962	0.912
Post intubations						
1 min	103.63±11.71	91.77±11.12	103.70±11.21	<0.001**	1.000	<0.001**
2 min	96.63±14.44	89.17±14.47	97.27±12.2	0.095+	0.983	0.064+
3 min	90.83±12.09	85.03±13.04	89.57±10.65	0.152	0.912	0.312
4 min	89.57±12.01	81.93±14.68	85.2±11.75	0.062+	0.392	0.590
5 min	88.00±11.06	79.63±11.91	83.47±12.05	0.018*	0.294	0.415
7 min	84.80±11.13	82.40±12.59	83.30±10.21	0.692	0.866	0.949
9 min	85.60±8.63	80.30±11.20	84.83±11.01	0.122	0.956	0.212
11 min	83.97±9.13	81.70±8.38	82.73±7.62	0.551	0.837	0.883
13 min	86.03±8.89	81.83±14.07	82.37±9.22	0.305	0.403	0.981
15 min	84.03±8.05	81.57±12.18	83.83±7.41	0.572	0.996	0.624

In control group the basal value of DBP was 78.27 mm Hg, at I minute following intubation, the DBP increased by 103.63 mm Hg, representing a rise of 25.36 mm Hg. This elevated pressure started coming down by 3 minutes. By 3 minutes and by 5 minutes it was 90.83 mm Hg and 88.00 mm Hg respectively. In group I the basal value of DBP was 77.93 mm Hg, at 1 minute following intubation, the DBP increased by 91.77 mm Hg, representing a rise of 13.84 mm Hg. This elevated pressure started coming down by 3 minutes. By 3 minutes and by 5 minutes it was 85.03 mm Hg and 79.63 mm Hg respectively. In group N the basal value of DBP was 78.87 mm Hg, at 1 minute following intubation, the DBP increased by 103.70 mm Hg, representing a rise of 24.83 mm Hg. This elevated pressure started coming down by 3 minutes. By 3 minutes and by 5 minutes it was 89.57 mm Hg and 83.47 mm Hg respectively. Statistical evaluation between the groups showed that the increase in DBP observed in control group was statistically highly significant when compared to increase in DBP in group I but not group N. The increase in DBP in group C and group I were statistically highly significant compared to increase in DBP in group N ($p < 0.001$).Between group C and group N there was no statistical significance. Between group I and group N, the increase in DBP in group N was statistically significant compared to increase in DBP in group I ($p < 0.001$).

DISCUSSION

The frequent occurrence of cardiovascular reactions to laryngoscopy and endotracheal intubation has attracted the attention of anesthesiologists for years. The reason being the reports of sudden death immediately following intubation and its association with tachycardia, hypertension and arrhythmia.[2,3,4] This response is primarily because of sympatho-adrenal stimulation, associated with laryngoscopy and endotracheal intubation.[1]A year later Burstein *et al*[2]totally contradicting Reid's statement, found that the pressor response occurring at laryngoscopy and endotracheal intubation was due to augmented sympathetic response, provoked by stimulation of epipharynx and laryngopharynx. These factors were further confirmed by Prys-Roberts.[4] The most important indications for attenuation of hemodynamic responses to laryngoscopy and endotracheal intubation becomes mandatory in patients with cardiovascular compromise like Ischemic heart disease (IHD) and Hypertension, patients with cerebrovascular diseases and in patients with intracranial aneurysms, even these transient changes can result in potentially deleterious effects like left ventricular failure,[6]pulmonary oedema,[6]myocardial ischemia[7]and ventricular dysrhythmias[4]and cerebral haemorrhage.[8] Control group: The base line value of heart rate (HR) was 85.50 bpm. One min following laryngoscopy and intubation, the heart rate (HR) increased to 108.9 bpm, representing a rise of 23.4 bpm, above the baseline value. Thus the maximal rise of heart rate (HR) seen in the control group was by an average of 23.4bpm. It was seen that the elevated heart rate (HR) started settling down towards the base line value by 7 to11 min, but it was still above the base line value even at 7 min. MounirAbou-Madi *et al*[21] noticed the increase in Heart rate (HR) in the control group following the laryngoscopy and intubation to be by 28bpm. In group I, where 2% Inj. Lignocaine 2mg/kg iv was administered to attenuate the hemodynamic response to laryngoscopy and intubation, the base line value of Heart rate (HR) was 86.13 bpm. One minute following laryngoscopy and intubation, the heart rate (HR) increased to 104.13 bpm, representing a rise of 18 bpm, above the base line value. Thus the maximal rise of Heart rate (HR) seen in the group 1 was by an average of 18 bpm. It was seen that the elevated Heart rate (HR) started settling down towards the baseline value by 7 min. Mounir Abou-Madi[23]noticed the increase in the heart rate (HR) by 21.1 & 8.5 bpm using 1% lignocaine 0.75mg/kg iv and 2% lignocaine 1.5mg/kg iv respectively following laryngoscopy and intubation. Stanlay*et al*[24]noticed a rise in heart rate (HR) of 12 bpm.

In group N, where nebulization of 2% Inj. Lignocaine 2mg/kg before laryngoscopy and intubation was used to blunt the pressor response, the base line value of Heart rate (HR) was 86.97 bpm. One minute following laryngoscopy and intubation, the heart rate (HR) increased to 111.83 bpm, representing a rise of 24.86 bpm above the base line value. Thus the maximal rise in heart rate (HR) seen was by an average of 24.86 bpm. It was seen that the elevated heart rate (HR) started settling down towards base line value by 11 min. MounirAbou-Madi *et al*[21] observed in their study a rise of 13 bpm following 1min of laryngoscopy and intubation in the pre-treatment group with lignocaine nebulization. The rise of heart rate in our study compared to the above study is more, which can be attributed to the lesser percentage of the drug used for nebulization, and also the wastage of drug during exhalation in the processes of nebulization. The maximum rise in heart rate was noted at 1 min following intubation in all the three groups which concurs well mentioned studies above. The mean rise in Heart rate at 1 min in control group was 23.4bpm compared to 18bpm and 24.86bpm in group I and group N respectively. The mean rise in the heart rate was comparatively lesser in the intravenous group but not statistically significant when compared to the group N and group C. There were no episodes of bradycardia in any of our study groups which was clinically significant. In the control group the basal value of Systolic blood pressure (SBP), Diastolic blood pressure (DBP), and Mean arterial pressure (MAP) was 121.73 mm Hg, 78.27 mm Hg, and 92.73 mm Hg respectively. Following laryngoscopy and intubation, the maximal rise in Systolic blood pressure (SBP) was found to be 42.6 mm Hg, that of Diastolic blood pressure (DBP) was 25.36 mm Hg and that of Mean arterial pressure (MAP) was 29.44 mm Hg. These elevated pressure readings started coming down by 5 minutes. MounirAbou-Madi *et al*[21]noticed that, in control group SBP, DBP and MAP, increased by 60 mm Hg and 37 mm Hg respectively Stanley Tam *et al*[24]noticed that, in control group SBP, DBP and MAP, increased by 38 mm Hg, 26 mm Hg, and 32 mm Hg respectively. Miller CD[49]found that in control group, SBP and DBP increased by 31 mm Hg and 28 mm Hg respectively. MounirAbou-Madi*et al*[23]found the rise in SBP and DBP to be 42 mm Hg and 30.7mm Hg respectively. Hence the results obtained in our study were similar to those obtained by the above mentioned authors.
In group I, where 2% Lignocaine 2mg/kg was employed 90sec before laryngoscopy and intubation to blunt the pressor responses, the maximal increase in the SBP, DBP and MAP was found to be 20.54 mm Hg, 13.84mm Hg and 16.1mm Hg respectively. MounirAbou- Madi *et al*[23] noticed the change in SBP by 17.6 and 30.4 mm Hg, change in the DBP was 20.1 and 21.8 mm Hg in lignocaine used in the doses of 0.75mg/kg and 1.5mg/kg IV respectively which does not concur as the dose used in the above study was lesser. Stanley Tam *et al*[24]employing Lignocaine 1.5 mg/kg IV observed maximal increase in SBP of 12 mm Hg, DBP of 9 mm Hg and MAP to be 11 mm Hg. Miller CD[49] noticed rise in SBP to be 33 mm Hg, DBP to be 37 mm Hg. Splinter *et al*[48] noticed a change in SBP to be 26 mm Hg, DBP to be 41 mm Hg and change in MAP to be 44 mm Hg. In group N, where nebulization of 2% Inj. Lignocaine 2mg/kg before laryngoscopy and intubation to blunt the pressor response, the maximal increase in the SBP, DBP and MAP was found to be 32.26 mm Hg, 24.83mm Hg and 27.3mm Hg respectively. MounirAbou-Madi *et al*[21] observed the changes in SBP, DBP following 1minute of laryngoscopy and intubation in the pre-treatment group with lignocaine nebulization to be 12mm Hg and 11mm Hg. Bahaman Venus noticed increase in the SBP, DBP and MAP to be 2.7mm Hg, 4mm Hg and 3.4mm Hg respectively. The rise of pressor response in our study compared to the above study is more, which can be attributed to the lesser percentage of only 2% being used when compared to above studies of the drug used for nebulization. In our study the pressor response was highly significant in the intravenous group than the control which concurs well with MounirAbou- Madi*et al*[23] as the dosage used by them was 1.5mg/kg when compared to 2mg/kg. In study conducted by Sklar BZ *et al*[29] the maximum rise in mean arterial pressure of 21.2 mm Hg was noted with intravenous group and minimum with nebulized lignocaine of 120mg of 10.1 mm Hg which did not concur with our study as the pressor response was much better statistically significant in group I when compared with the group N. There were not much of significant changes in blood pressure in control and nebulization group in the current study as it can be attributed to the fact that a simple face mask was used for administration of nebulization, lesser concentration of drug used and the wastage of drug during exhalation.

CONCLUSION

In our study though nebulization was not effective in blunting the haemodynamic responses to laryngoscopy and intubation, it is still a safe and a simple technique to be used. In our opinion it could be beneficial if: Proper nebulization could be administered minimizing its wastage during exhalation even though we did not asses the percentage wasted in the present study. A higher concentration of the drug could have better beneficial aspects. A longer latent period of time for complete establishment of the effects of nebulization on the airway as we have used a lesser concentration of lignocaine in our study.

REFERENCES

1. Shribman AJ, Smith G, Achola KJ: Cardiovascular and chatecholamine responses to laryngoscopy with or without tracheal intubation. *British Journal of Anaesthesia* 1987; 59:295
2. Burstein CL, Lo Pinto FJ and Newman W. Electrocardiographic studies during endotracheal intubation 1, effects during usual routine techniques. *Anaesthesiology*. 1950; 11:224.
3. Robert K. Stoelting. Blood pressure and heart rate changes during short-duration laryngoscopy for tracheal intubation. Influence of viscous or intravenous lidocaine. *Anaesthesia Analgesia*. 1978; 57: 197-199.
4. Prys-Roberts, Greene LT, Meloche R and Foex P. Studies of anaesthesia in relation to hypertension-II. Haemodynamic consequences of induction and endotracheal intubation. *British Journal of Anaesthesia*. 1971; 43: 541-547.
5. Forbes AM and Dally FG. Acute hypertension during induction of anaesthesia and endotracheal intubation in normotensive man. *British Journal of Anaesthesia*. 1970; 42: 618-622.
6. Fox EJ, Sklar GS, Hill CH, Villanueva R, King BD: Complications related to the pressor response to endotracheal intubation. *Anesthesiology* 1977; 47:524-525.
7. Dalton B and Guiney T *et al*. Myocardial ischaemia from tachycardia and hypertension in coronary heart disease – Patient's undergoing anaesthesia. *Ann. Mtg. American Society of Anaesthesiologists, Boston.* 1972; pp. 201-202.
8. Donegan MF and Bedford RF. Intravenously administered lignocaine prevents intracranial hypertension during endotracheal suctioning. *Anaesthesiology*, 1980; 52:516-518.
9. Nair GC, Hariwar Singh and Preetam Singh. Vital signs and electrocardiography during intubation. *Indian Journal of Anaesthesia*. 1965 Feb; 40-57.
10. Bedford RF and Lt Marshal K. Cardiovascular responses to endotracheal intubation during four anaesthetic techniques. *ActaAnaesthesiologica Scandinavia*. 1984; 28: 563-566.
11. Braude N, Clements EA, Hodges UM, and Andrews BP. The pressor response and laryngeal mask insertion. A comparison with tracheal intubation. *Anaesthesia* 1989; 44:551.
12. Wood ML, Forrest ET: The haemodynamic response to the insertion of the laryngeal mask airway: A comparison with laryngoscopy and tracheal intubation. *ActaAnaesthesiolScand* 1994; 38:510.
13. Chen C: Fentanyl dosage for suppression of circulatory response to laryngoscopy and endotracheal intubation. *Anesthesiol Rev* 1986; 13:37.
14. Devault M, Greifenstein FE and Harris JR. LC. Circulatory responses to endotracheal intubation in light general anaesthesia; the effect of atropine and phentolamine. *Anaesthesiology*. 1960; 21: 360-362.
15. Prys-Roberts C, Foex P, Biro GP and Roberts JG. Studies of anaesthesia in relation to hypertension – V: Adrenergic beta-receptor blockade. *British Journal of Anaesthesia*. 1973; 45: 671-680.
16. Mikawa K, Nishina K, Maekawa N, Obara H: Comparison of nicardipine, diltiazem and verapamil for controlling the cardiovascular responses to tracheal intubation. *Br J Anaesth* 1996; 76:221.
17. Gallagher JD, Moore RA, Jose AB, *et al*: Prophylactic nitroglycerin infusions during coronary artery bypass surgery. *Anesthesiology* 1986; 64:785.
18. Mikawa K, Nishina K, Maekawa N, *et al*: Attenuation of the catecholamine response to tracheal intubation with oral clonidine in children. *Can J Anaesth* 1995; 42:869.
19. Aho M, Lehtinen AM, Erkola O, *et al*: The effects of intravenously administered dexmedetomidine on perioperative hemodynamiccs and isoflurane requirements in patients undergoing abdominal hysterectomy. *Anesthesiology* 1991; 74:997 – 1002.
20. Stoelting RK: Circulatory response to laryngoscopy and tracheal intubation with or without prior oropharyngeal viscous lidocaine. *AnesthAnalg* 1977; 56: 618
21. Abou-Madi M, Keszler H, and Yacoub O. A method for prevention of cardiovascular reactions to laryngoscopy and intubation. *Canad. Anaesth. Soc. J.* May (1975)22: 316(1975).
22. Williams KA, Barker GL, Harwood RJ, Woodall NM. Combined nebulization and spray–as–you-go topical local anaesthesia of the airway. *British Journal of Anaesthesia*. 2005; 95:549-553.
23. Abou-Madi M, Keszler H, and Yacoub JM. Cardiovascular reactions to laryngoscopy and tracheal intubation following small and large intravenous dose of lidocaine. *Canadian Society Anaesthesia Journal.* 1977; 24(1):12-18.
24. Tam S, Chung F, and Michael Campbell M. Intravenous lignocaine: Optimal time for injection before tracheal intubation. *Anesth Analg.*1987; 66: 1036-1038.
25. Yakioka H, Hayashi M, Yoshimoto N, *et al*. IV Lidocaine as a suppressant of coughing during tracheal intubation. *AnesthAnalg* 1985; 64: 1189-1192.
26. Baraka A. Intravenous lidocaine controls extubation laryngospasm in children. *AnesthAnalg* 1978; 57:506-507.
27. Adamizik M, Groeben H, Farahan R, Lehmann N, Peters J. Intravenous lidocaine after tracheal intubation mitigates bronchoconstriction in patients with asthma. *Anesth Analg.*1993; 77: 309 – 312.
28. Groeben H, Silvanus MT, Beste M, Peters J. Both intravenous and inhaled lidocaine attenuate reflex bronchoconstriction but at different plasma concentrations. *Am. J. Respir. Crit Care Med*. 1999; 159:530-535.

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