

A Comparative Study Between Intravenous Lignocaine And Intravenous Dexmedetomidine In Attenuating The Haemodynamic Responses During Laryngoscopy And Endotracheal Intubation

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Abstract

AIM: To compare the attenuation of hemodynamic changes during laryngoscopy and Endotracheal intubation with intravenous lignocaine versus intravenous dexmedetomidine.

MATERIALS AND METHODS: Study – randomized, double blind. Ethical committee approval - obtained from our institute. Written informed consent - obtained from all the patients.

SOURCE OF DATA: Sixty one patients of both sexes admitted for elective surgeries under general anaesthesia in various surgical disciplines of Government MK medical college, Salem.

OBSERVATION: From our study, we observed that Lignocaine attenuated but did not fully abolish the pressor response to laryngoscopy and intubation. Also we adequately established that Dexmedetomidine in the dose of 1 µg/kg was comparatively superior in attenuation of the haemodynamic changes during direct laryngoscopy.

CONCLUSION: We conclude that Dexmedetomidine in the dosage of 1 µg/kg over ten minutes before intubation efficiently attenuated the haemodynamic changes to laryngoscopy and endotracheal intubation. Lignocaine in the dosage of 1.5 mg/kg given 3 min before laryngoscopy and intubation was not fully effective in reducing the increase in heart rate and blood pressure. Hence Dexmedetomidine may be beneficial for cardiac patients where the haemodynamic response to laryngoscopy and intubation is highly detrimental.

Keywords: Catecholamine release; Dexmedetomidine; Hemodynamic response; Lignocaine; Pressor response

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I. Introduction

Direct laryngoscopy and endotracheal intubation form the vital steps for providing general anaesthesia. Laryngoscopy and intubation is an adverse stimulus, which can cause unaccepted response in the respiratory and cardiovascular systems. Tachycardia and hypertension in response to laryngo-tracheal stimulation are due to reflex sympathetic discharge, which in sequence causes increased plasma nor-epinephrine concentration. Though these sympathoadrenal responses are probably of little significance in healthy individuals, it is hazardous to those with hypertension, coronary artery heart disease, intracranial pathology and hyperactive airways. In such cases, these responses need to be suppressed.

Hence this study was designed to compare the efficacy of intravenous Lignocaine and intravenous Dexmedetomidine to effectively attenuate the haemodynamic responses accompanying laryngoscopy and intubation during anaesthetic induction.

After getting approval from the hospital ethical committee, we carried out this study in the Department of Anaesthesiology, Government Mohan Kumaramangalam Medical College, Salem during the time of August 2018 to August 2019.

II. Materials And Method

Type of Study: A prospective, randomised double blind study was conducted in the Department of Anaesthesiology, Government MK medical college, Salem from August 2018 to August 2019, after getting proper approval from the hospital ethical committee and getting prior written informed consent from all the 61 patients in the age group 18-60 of ASA PS I or II.

INCLUSION CRITERIA:

1. Patients in ASA grades I and II
2. Patients with modified Mallampatti scores I & II
3. Age 18-55years

EXCLUSION CRITERIA

1. Patients in ASA grades III and IV
2. Patients with modified Mallampatti scores III & IV
3. Patients with predicted difficult airway
4. Obese patients
5. Patients with Systemic Hypertension, CAD, H/O Cerebrovascular Accidents, CRF, Valvular Heart Diseases, patients on antihypertensive or cardiac drugs
6. Patients posted for emergency surgeries
7. Patients with full stomach
8. If the intubation time has exceeded 15 seconds
9. Age <20 and >55 years
10. Patient undergoing procedures requiring head & neck manipulation

ASSESSMENT:

All patients were assessed by a detailed physical examination supported by investigations like routine blood tests- Hb, blood sugar, blood urea, serum creatinine, serum electrolytes, chest X ray PA view, Electrocardiogram, etc.

RANDOMIZATION:

The patients were randomly allocated to two groups of 30 with the help of a computer generated table of random numbers to receive following drugs:

Group L: 30 patients were given Inj Lignocaine 1.5 mg/kg body weight intravenously, 3 minutes before intubation.

Group D: 30 patients were given Inj Dexmedetomidine (1 mcg/kg body weight) intravenously over 10 minutes, given 10 minutes before intubation.

PREMEDICATION AND MONITORING

All patients were given Tab Diazepam 10 mg orally the night before surgery. Patients were kept nil orally 6-8 hours prior to surgery. Now patients were randomly divided by computer into two groups. In preoperative room baseline parameters were observed and documented. All patients were given Inj Glycopyrolate 10 mcg/kg body weight intramuscularly 45 minutes before surgery. Patients were shifted to the operating room and an 18-gauge intravenous cannula was inserted in the forearm and infusion of ringer lactate was started. Standard multipara monitor was connected - ECG, NIBP and pulse oximeter. NIBP was recorded every two minutes.

INDUCTION AND INTUBATION:

Patients were given Inj. Fentanyl 2mcg/kg body wt. Group D received 1 mcg/kg of Inj. Dexmedetomidine in 10 ml of normal saline over 10 minutes and 5ml of normal saline 3 minutes before induction. Group L received 10 ml of normal saline over 10 minutes and Inj. Lignocaine 1.5 mg/kg diluted in 5 ml of normal saline 3 minutes before induction. These solutions of 10 ml and 5 ml were prepared by first anaesthesiologist. The second anaesthesiologist, who was not aware of the groups, administered the drug and monitored the patients recording vital parameters before intubation and immediately after intubation and also 1min, 3min or 5 min after laryngoscopy and endotracheal intubation according to the group to which they were assigned.

The laryngoscopy and intubation were performed by the third anaesthesiologist who was also blinded to the drug given. Patients were preoxygenated for 5minutes. Patients were induced with Inj. Propofol 2 mg IV and followed by Inj. Suxamethonium 2mg IV. Thereafter all the patients were manually ventilated with bag and mask with 100% oxygen for 3minutes. Laryngoscopy and intubation was then done and the time taken for the same was noted.

Those that took > 15 seconds were excluded from the study. After confirming the position of the endotracheal tube, anaesthesia was maintained for the next 5 minutes with 67% nitrous oxide and 33% oxygen. No surgical stimulation was permitted for 5minutes after intubation. The baseline, before intubation, immediately after intubation (0 minutes), 1 minute, 3 minutes and 5 minutes after intubation values of circulatory variables such as HR, SBP, DBP and MBP were recorded.

III. Results

The demographic data was comparable in both the groups without any significant variation.

- The mean of PLHR in the group Lignocaine is 82.35 and 75.76 in the group Dexmedetomidine with the association stastically significant.
- The mean of PLSBP in the groups Lignocaine and Dexmedetomidine were 107.9, 101.9 and mean of PLDBP were 65.64 and 60.86 respectively with p value stastically significant.
- The mean of PLMBP of the groups Lignocaine & Dexmedetomidine were 79.8 and 74.53 respectively with association statistically significant.
- This difference in the pre laryngoscopy value is due to alpha two agonist action of dexmedetomidine, whereas lignocaine has membrane stabilizing action.

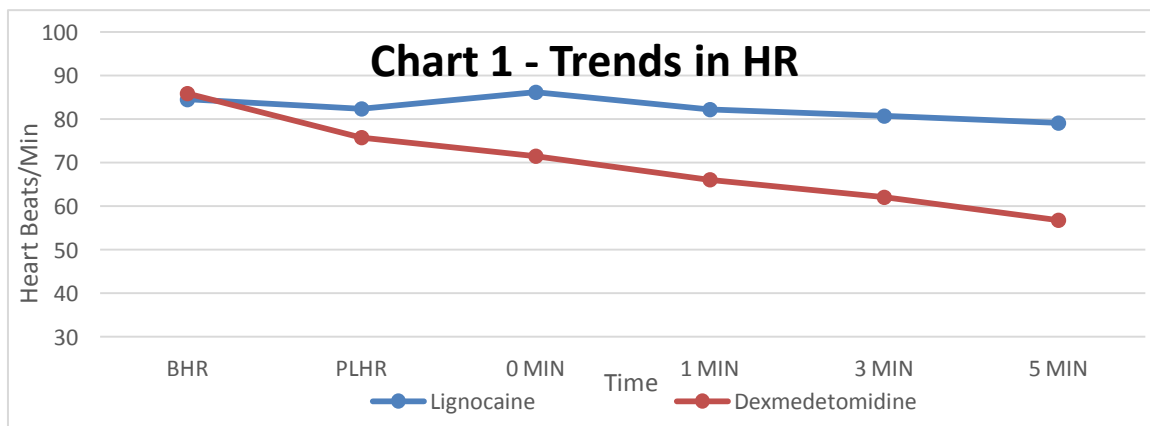
TABLE: 1 - Independent Sample T Test of Parameters PLHR, PLSBP, PLDBP, PLMBP.

| Parameter | Mean ± STD | | P value |
|-----------|---------------|-----------------|---------|
| | Lignocaine | Dexmedetomidine | |
| PLHR | 82.35 ± 11.19 | 75.76 ± 8.46 | 0.0121 |
| PLSBP | 107.9 ± 11.64 | 101.9 ± 5.19 | 0.0122 |
| PLDBP | 65.64 ± 6.12 | 60.86 ± 3.69 | <0.0001 |
| PLMBP | 79.8 ± 7.73 | 74.53 ± 4.17 | <0.0001 |

The mean values of BHR in the groups Lignocaine and Dexmedetomidine were 84.48 and 85.86 respectively and p value stastically non-significant. The mean of PLHR in the group Lignocaine is 82.35 and 75.76 in the group Dexmedetomidine with the association stastically significant.

TABLE: 2 - Independent Sample T Test of Heart Rate

| Parameter | Mean±STD | | P value |
|--------------|---------------|-----------------|---------|
| | Lignocaine | Dexmedetomidine | |
| BHR | 84.48 ± 11.16 | 85.86 ± 9.5 | 0.6048 |
| PLHR | 82.35 ± 11.19 | 75.76 ± 8.46 | 0.0121 |
| 0-Heart Rate | 86.16 ± 11.52 | 71.5 ± 7.46 | <0.0001 |
| 1-Heart Rate | 82.19 ± 9.74 | 66.1 ± 6.143 | <0.0001 |
| 3-Heart Rate | 80.70 ± 8.691 | 62.06 ± 5.13 | <0.0001 |
| 5-Heart Rate | 79.09 ± 7.828 | 56.8 ± 4.080 | <0.0001 |



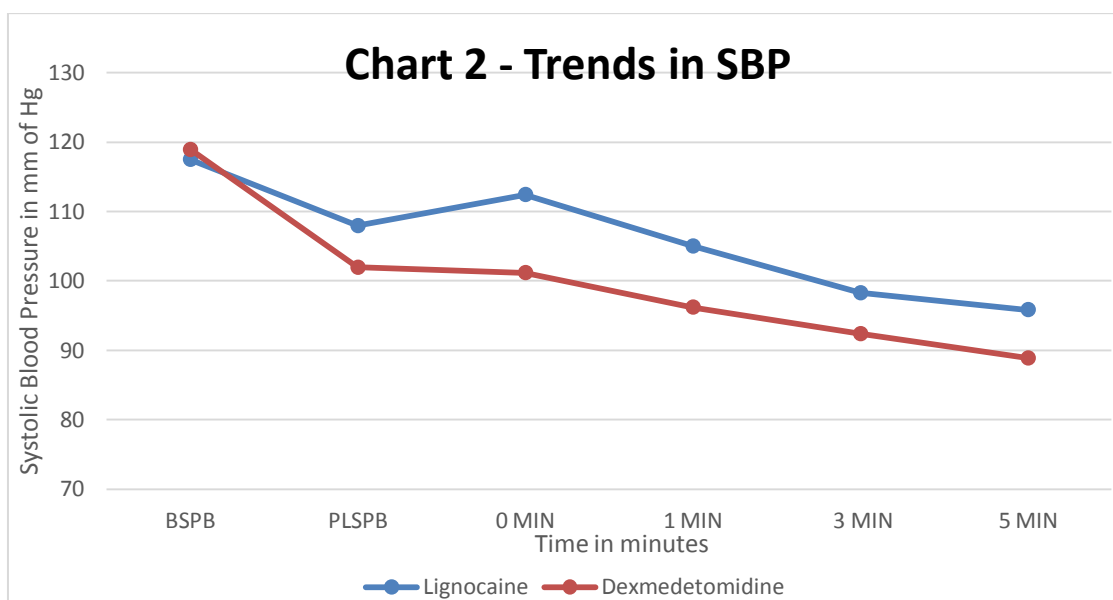
The mean of the heart rate at 0 minute in the group Lignocaine was 86.16 and in the group Dexmedetomidine was 71.5 and the association with stastically significant. The mean of the heart rate at 1 minute in the groups Lignocaine and Dexmedetomidine were 82.19 and 66.1 respectively and p value is stastically significant. The mean of the heart rate at 3 minutes in the group Lignocaine was 80.70 and in the

group Dexmedetomidine was 62.06 and the association with stastically significant. The mean of the heart rate at 5 minutes in the groups Lignocaine& Dexmedetomidine were 79.09 & 56.8 respectively and p value is stastically significant.

The mean of BSBP in the group Lignocaine is 117.4 and in the group Dexmedetomidine is 118.9 with p-value stastically not significant. The mean of PLSPB in the groups Lignocaine and Dexmedetomidine were 107.9 and 101.9 respectively with p value stastically significant. The mean of the Systolic Blood Pressure at 0 minute in the group Lignocaine was 112.4 and in the group Dexmedetomidine was 101.1 and the association with stastically significant. The mean of the Systolic Blood Pressure at 1 minute in the groups Lignocaine & Dexmedetomidine were 105 and 96.2 respectively and p value is stastically significant. The mean of the Systolic Blood Pressure at 3 minutes in the group Lignocaine was 98.25 and in the group Dexmedetomidine was 92.36 and the association with stastically significant. The mean of the Systolic Blood Pressure at 5 minutes in the groups Lignocaine& Dexmedetomidine were 95.83& 88.86 respectively and p value is stastically significant.

TABLE: 3 - Independent Sample T Test of Systolic Blood Pressure

| Parameter | Mean ± STD | | p value |
|-----------|---------------|-----------------|---------|
| | Lignocaine | Dexmedetomidine | |
| BSPB | 117.4 ± 13.34 | 118.9 ± 11.28 | 0.6565 |
| PLSPB | 107.9 ± 11.64 | 101.9 ± 5.19 | 0.0122 |
| 0-SBP | 112.4 ± 7.29 | 101.1 ± 3.98 | <0.0001 |
| 1-SBP | 105 ± 6.76 | 96.2 ± 3.54 | <0.0001 |
| 3-SBP | 98.25 ± 6.37 | 92.36 ± 3.25 | <0.0001 |
| 5-SBP | 95.83 ± 4.194 | 88.86 ± 4.342 | <0.0001 |



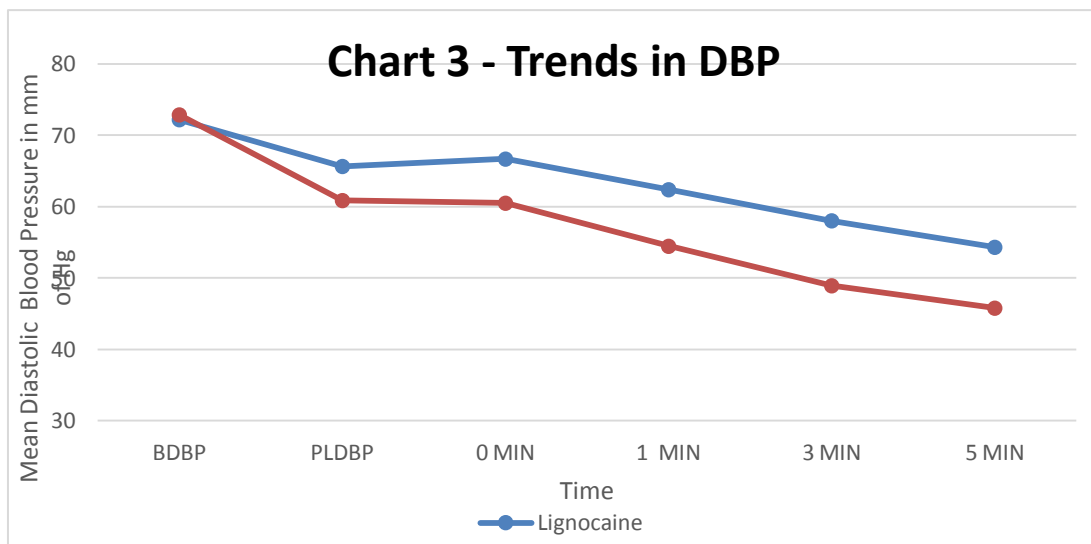
The mean of DBP in the group Lignocaine is 72.19 and in the group Dexmedetomidine is 72.86 with p-value stastically significant. The mean of PLDBP in the groups Lignocaine and Dexmedetomidine were 65.64 and 60.86 respectively with association statistically significant. The mean of the Diastolic Blood Pressure at 0 minutes in the group Lignocaine was 66.67 and in the group Dexmedetomidine was 60.5 and the association with stastically significant.

The mean of the Diastolic Blood Pressure at 1 minute in the groups Lignocaine& Dexmedetomidine were 62.38 & 54.46 respectively and p value is stastically significant. The mean of the Diastolic Blood Pressure at 3 minutes in the group Lignocaine was 58.03 and in the group Dexmedetomidine was 48.93 and the

association with stastically significant. The mean of the Diastolic Blood Pressure at 5 minutes in the groups Lignocaine, Dexmedetomidine were 54.32, 45.8 respectively and p value is stastically significant.

TABLE: 4 - Independent Sample T Test of Diastolic Blood Pressure

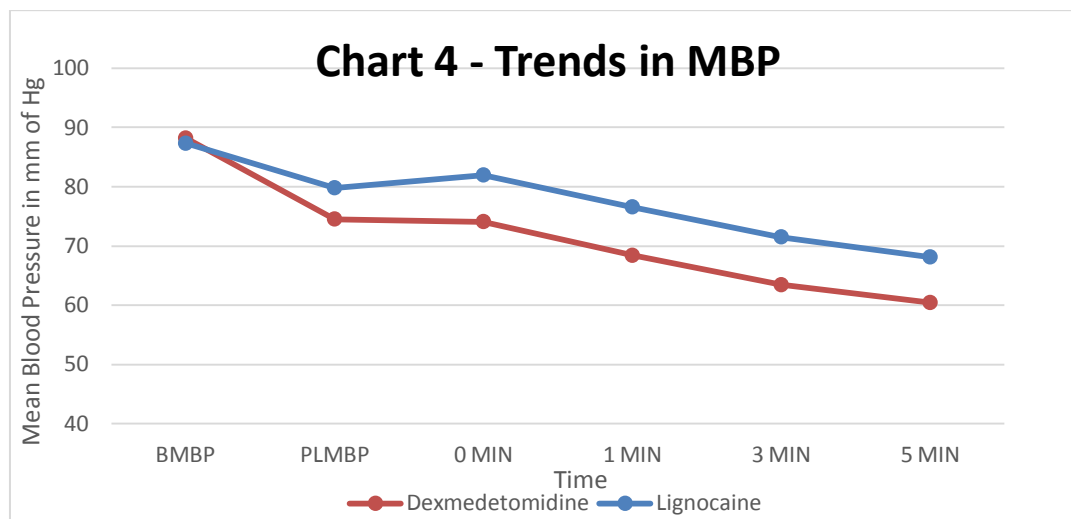
| Parameter | Mean + STD | | p value |
|-----------|--------------|-----------------|---------|
| | Lignocaine | Dexmedetomidine | |
| BDBP | 72.19 + 8.79 | 72.86 + 7.3 | 0.7466 |
| PLDBP | 65.64 + 6.12 | 60.86 + 3.69 | <0.0001 |
| 0-DBP | 66.67 + 4.55 | 60.5 + 2.73 | <0.0001 |
| 1-DBP | 62.38 + 5.84 | 54.46 + 2.85 | <0.0001 |
| 3-DBP | 58.03 + 5.55 | 48.93 + 1.66 | <0.0001 |
| 5-DBP | 54.32 + 4.23 | 45.8 + 1.49 | <0.0001 |



The mean value of BMBP in the groups Lignocaine and Dexmedetomidine were 87.32 and 88.2 respectively and its p value is stastically not significant. The mean of PLMBP of the groups Lignocaine and Dexmedetomidine were 79.8 and 74.53 respectively with association statistically significant. The mean of the Mean Blood Pressure at 0 hour in the group Lignocaine was 81.96 and in the group Dexmedetomidine was 74.1 and the association with stastically significant. The mean of the Mean Blood Pressure at 1 minute in the groups Lignocaine& Dexmedetomidine were 76.58 and 68.4 respectively and p value is stastically significant.

TABLE: 5 - Independent Sample T Test of Mean Blood Pressure

| Parameter | Mean ± STD | | p value |
|-----------|---------------|-----------------|---------|
| | Lignocaine | Dexmedetomidine | |
| BMBP | 87.32 ± 10.27 | 88.2 ± 8.54 | 0.7186 |
| PLMBP | 79.80 ± 7.73 | 74.53 ± 4.17 | <0.0001 |
| 0-MBP | 81.96 ± 5.27 | 74.1 ± 2.92 | <0.0001 |
| 1-MBP | 76.58 ± 5.98 | 68.4 ± 2.79 | <0.0001 |
| 3-MBP | 71.51 ± 5.61 | 63.43 ± 1.87 | <0.0001 |
| 5-MBP | 68.12 ± 4.32 | 60.43 ± 1.41 | <0.0001 |



The mean of the Mean Blood Pressure at 3 minutes in the group Lignocaine was 71.51 and in the group Dexmedetomidine was 63.43 and the association with statically significant. The mean of the Mean Blood Pressure at 5 minutes in the groups Lignocaine and Dexmedetomidine were 68.12 and 60.43 respectively and p value is statically significant.

IV. Discussion

The hemodynamic response is characterized by tachycardia and hypertension during handling in the larynx, by means of laryngoscopy and intubation. Stimulation of mechanoreceptors in the wall of pharynx, epiglottis and vocal cords, is thought to be the cause for this hemodynamic response. Shribman *et al* found that laryngoscopy only or followed by endotracheal intubation raises the HR, BP and catecholamine levels. These changes were reported to be greatest at 1 minute after intubation of the trachea that lasts for 5-10min. HR may rise from 26% to 66% and the SBP may rise from 36% to 45%. Myocardial ischemia may happen during the laryngoscopy & intubation in patients with CAD. There is a higher chance of progress of intraoperative ischemia into perioperative myocardial infarction in patients with limited cardiac reserve.

Lev and Rosen and Wilson *et al* in their study reviewed the use of prophylactic lignocaine as a preintubation medication. A dose of 1.5 mg/kg intravenously 3 min prior to intubation was employed and was found to be optimal for attenuating the sympathoadrenal response to laryngoscopy and intubation without any overt harmful effects. We also administered lignocaine 1.5 mg/kg 3 min before intubation in our study and observed a general decline in HR, SBP, and DBP. But there is spike in all the hemodynamic parameters HR, SBP, DBP and MBP immediately after intubation.

The decrease in HR and blood pressure in our study might also be attributed to the use of anaesthetic agents such as opioids (Fentanyl) and inhalational agents administered during the maintenance of anaesthesia. From our statistical analysis we also infer that though there is a general decline in HR after administration of lignocaine, but at the time interval corresponding to 0 min post-intubation, we observed an increase in HR. This shows that the pressure response was incompletely abolished by lignocaine.

Lignocaine attenuated the rise in blood pressure but not prevented it totally. The rise persisted for 1 min in the lignocaine group after intubation. In our study we found that lignocaine sufficiently attenuated the above mentioned hemodynamic response, but this attenuation was not complete and a spike in SBP was observed up to 1 min of post intubation.

We also noticed 2 spikes at 1 min and 3 min intervals post-intubation in the DBP recordings which are in concordance with the above study. In our study we also did not encounter any side effects like hypotension or bradycardia when lignocaine at a dose of 1.5 mg/kg was employed.

Recent studies however, doubted the efficacy of lignocaine. In studies by Singh *et al* and Kindler *et al*, intravenous lignocaine of dosage 1.5 mg/kg was not effective to decrease the acute haemodynamic response after intubation.

In a study conducted by Pathak *et al*, it was shown that lignocaine 1.5 mg/kg was not effective in blunting the responses during laryngoscopy and tracheal intubation when compared with two different doses of Alfentanil (15µg/kg and 30µg/kg). However in our study, we used Fentanyl universally in both the groups. From the interpretation of the results of our study we concluded that lignocaine attenuated but did not fully abolish the pressure response to laryngoscopy and intubation.

Alpha two adrenergic agonists decrease the sympathetic reflex and thereby attenuate the hemodynamic responses to laryngoscopy and intubation.

They also decrease the requirement of anaesthetic drugs and therefore can be used as an adjunct in general anaesthesia. Dexmedetomidine is a highly selective and specific alpha two adrenergic agonist. Therefore, it is increasingly being used as an agent to attenuate the pressor response.

Sagiroglu *et al.* concluded that the overall control of hemodynamic responses to tracheal intubation were better with Dexmedetomidine 1 µg/kg as compared to Dexmedetomidine 0.5 µg/kg. In our study we concluded that 1 µg/kg of Dexmedetomidine significantly reduced the increase in HR associated with laryngoscopy and intubation compared with Lignocaine. In the study conducted by Sagiroglu *et al.* the results of SBP, DBP and MAP were significantly lower in the group given Dexmedetomidine 1 µg/kg which was in agreement with our study as well.

On comparison we concluded that Dexmedetomidine 1 µg/kg brought upon a maximal reduction in SBP and DBPs at 0, 1, 3 and 5 min of post intubation. From our study, we adequately establish that Dexmedetomidine 1µg/kg was comparatively superior in attenuation of the haemodynamic changes during direct laryngoscopy.

V. Conclusion

We conclude that Dexmedetomidine in the dosage of 1 µg/kg over ten minutes before intubation efficiently attenuates the haemodynamic changes to laryngoscopy and endotracheal intubation.

Lignocaine in the dosage of 1.5 mg/kg given 3 min before laryngoscopy and intubation was not fully effective in reducing the increase in heart rate and blood pressure.

Dexmedetomidine 1 µg/kg has proved to keep the haemodynamics in stable manner during laryngoscopy & intubation. Hence Dexmedetomidine may be beneficial for cardiac patients where the haemodynamic response to laryngoscopy and intubation is highly detrimental.

In brief, Dexmedetomidine is a highly selective α₂ agonist that has many desirable clinical benefits that encourage its use in the perioperative period.

Bibilography

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