IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 17, Issue 12 Ver. 9 (December. 2018), PP 05-14
www.iosrjournals.org

Effect of Perioperative I.V. Infusion of Dexmedetomidine Vs Lignocaine on Haemodynamic Responses to Intubation, Extubation and Postoperative Analgesia– An Institutional Study

Dr. V. Prem Swarup, Dr. P.K. Subhashini , Dr. U.S.S.A. Vara Prasad, Dr. G. Arvind Anand & Dr.S.K.K.Chaitanya Veera

-Associate Professor, Department of Anaesthesia, Dr Pinnamaneni Siddhartha Institute of Medical Sciences, and Research Foundation, Chinnaoutpalli, A.P, India.
-Assistant Professor, Department of Pharmacology, Siddhartha Medical College, Vijayawada, A.P., India.
-H.O.D. Anesthesiology, Dr Pinnamaneni Siddhartha Institute of Medical Sciences, and Research Foundation, Chinnaoutpalli, A.P, India.
-Resident Post Graduates in Anaesthesiology & Critical Care, Dr PSIMS & RF.
Corresponding Author: Dr. V. Prem Swarup

Abstract:
Background: *Decades of clinical research have established that Acute Pain after surgery has a distinct Pathophysiology that reflects peripheral and central sensitisation as well as humeral factors contributing to pain at rest and during movement, this can impair functional ability and often cumulates in delayed recovery. General Anesthesia involves stressful events at various stages, the most stressful situations are seen during the period of induction, intubation and extubation.*
Since 1950 Hypertension, Tachycardia has been recognised as commonly associated under light planes of Anesthesia and stressful period.
Different techniques with different drugs have been suggested to attenuate the haemodynamic response to intubation, extubation and also provide effective postoperative analgesia — topical & IV Lignocaine deep inhalational anaesthesia, ganglion blockade, precurarisation, Narcotics, Vasodilators, IV Nitroglycerine, Calcium Channel Blockers and α2 agonists like clonidine.
Dexmedetomidine an α2 agonist is being used for many years as a premedication agent and as lignocaine is a freely available drug used for the pressor response. Both the drugs decrease the sympathetic tone, hence blunt the pressor response and also has analgesic properties to be used as an adjunct in anaesthesia.
Hence the primary objective of this present study is to observe whether a regimen of IV lignocaine Vs Dexmedetomidine in preoperative bolus doses followed by perioperative IV infusion can reduce the pressor effects and haemodynamic changes during tracheal intubation/extubation as well as attenuation of postoperative pain following elective surgeries.
Methods: *This is a randomised controlled study conducted during 2015-17 in the Department of Anesthesiology, Dr PSIMS & R.F. after approval from the ethics and drug committee.*
A total of 80 patients undergoing elective surgeries were selected. Group-D (Dexmedetomidine) 40 patients were given after diluting with 50 ml Normal Saline infused at 1 mcg/kg IV over 10 min prior to laryngoscopy followed by maintenance of 0.25 mcg/kg/hr. 40 patients as Group-L (lignocaine) 2% diluted to a volume of 24 ml given 1.5 mg/kg over 10 min prior to laryngoscopy and thereafter infused at 1.5mg/kg body weight/hr. Time taken for intubation did not exceed 10 minutes in both groups. Anaesthesia was maintained with Nitrous Oxide (N_2O) + Oxygen (O_2) and Vecuronium Bromide with IPPV in both groups of patients. Haemodynamic responses like Heart rate, Mean arterial pressor were monitored before & post induction, post intubation and also after 3 mts, 5 mts & 10 mts. Postoperatively the time of request for first analgesic was recorded. Pain-free period (NRS-4) was taken as the period from the conclusion of surgery to a requirement for first analgesic (NSAID), and assessment of Analgesia was done with Numerical Pain Scale.
Randomization: *Sample was taken at random from the population when each number of the population has the equal chance of being chosen. The purpose is to produce groups that are nearly similar as possible to the experimental procedure.*
Keywords: *Dexmedetomidine, Lignocaine, Pressor response, Hemodynamic changes, Narcotics, Rescue Analgesia, Numerical Pain Score and Pain-free period.*

Date of Submission: 08-12-2018 Date of acceptance: 24-12-2018

I. Introduction

The mechanical stimulation of four areas of upper respiratory tract – the nose, the epipharynx, the laryngopharynx and tracheobronchial trees induces reflex cardiovascular response which is associated with enhanced neuronal activity in cervical sympathetic fibres. A 25-50% increase in Mean Arterial Pressure and Heart Rate is seen during induction followed by laryngoscopy and intubation peaking at 1-2 min and returning to baseline within 10-15 min. This response is accompanied by raised plasma adrenaline concentration. But this cardiovascular response to intubation is of serious concern in patients with hypertension, raised ICP, diseased cerebral vasculature or with Ischemic Heart Disease where an increase in myocardial oxygen consumption can lead to Myocardial Infarction[2]. Failure to blunt the responses to intubation may have disastrous consequences like acute Left Ventricular Failure, intracranial haemorrhage and pulmonary oedema. Convulsions may be precipitated in eclamptic patients Herniation of intracranial contents, and cerebral ischemia can occur in a patient with raised intracranial pressure. Arrhythmias (sinus tachycardia and sinus bradycardia, atrial and ventricular extrasystoles and pulsus alternans, less commonly multifocal extrasystoles, pulsus bigeminy and atrial fibrillation) are reported. Heart book, ventricular tachycardia and ventricular fibrillation are rare.

Different techniques with different drugs have been suggested to attenuate the hemodynamic response to intubation, extubation and also to provide effective postoperative analgesia. Lignocaine, deep inhalational anaesthesia, ganglion blockers, pre-curarization, narcotics (fentanyl) vasodilators, i.v. Nitroglycerine and calcium channel blockers, alpha 2 agonists like clonidine and Dexmedetomidine. All the studies so far have taken into consideration only a single parameter of Lignocaine or Dexmedetomidine utility, i.e. either attenuation of hemodynamic changes secondary to intubation/extubation or postoperative analgesic efficacy.

Hence, the primary objective of the present study is to observe whether a regimen of i.v. Lignocaine versus i.v dexmedetomidine of pre-operative bolus doses followed by peri-operative infusion can reduce the hemodynamic changes during tracheal intubation and extubation as well as attenuation of postoperative pain following elective surgeries.

II. Methodology

A randomised controlled study was conducted in Pinnamaneni Siddhartha Institute of Medical Sciences and Research Foundation, Chinaoutapalli after approval from the hospital ethics committee, and obtaining written informed consent from the patients during the period between November 2015 and May 2017. A total of 80 patients undergoing elective surgeries were selected.

This study was undertaken in 2 groups:

- Group-D consists of 40 patients, who received IV Dexmedetomidine diluted in 50 ml NS at loading dose 1 mcg/kg infusion over a period of 10 min prior to laryngoscopy followed by a maintenance dose of 0.25 mcg/kg/hr.
- Group-L consists of 40 patients, who received plain preservative free lignocaine 2% diluted to a volume of 24 ml, 1.5 mg/kg body weight IV over a period of 10 min prior to laryngoscopy followed by an infusion of 1.5 mg/kg/hr.

Inclusion Criteria

1) ASA Grade I & II, 2) Age 18-50 years of both sexes. 3) Weight between 40-70 kgs.
4) Mallampatti Class I & II

III. Observations And Results

Eighty patients, undergoing elective non-cardiac surgery were selected for the study. The patients were randomly divided into two groups, of 40 patients each.

Dexmedetomidine provides better protection against untoward hemodynamic side effects of laryngoscopy, intubation & extubation as compared to lignocaine. Lignocaine also blunts the response but not as effectively as dexmedetomidine. When it comes to post-operative analgesia, Lignocaine offers prolonged pain-free period as compared to dexmedetomidine.

Dexmedetomidine provides and augments analgesia and diminishes shivering as well as agitation postoperatively. The safety record of dexmedetomidine suggests that it can be used effectively and safely, with appropriate monitoring and interventions to manage cardiovascular sequelae.

Statistical Methods

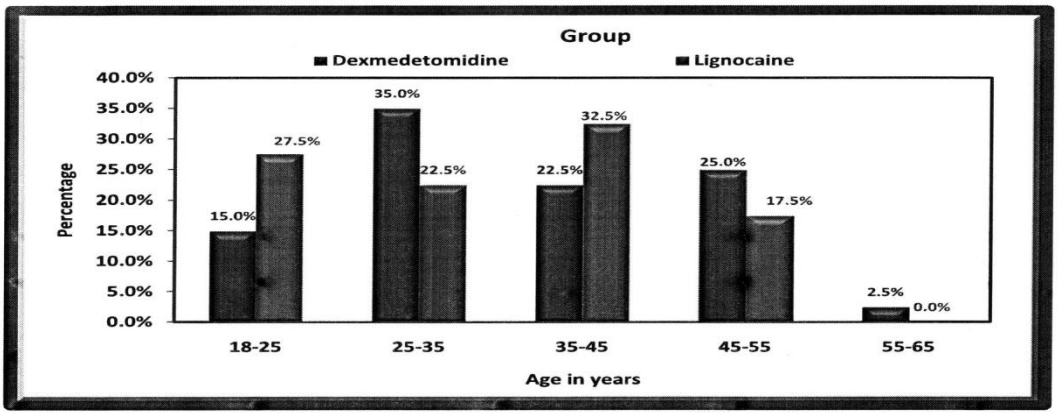
Data collected, tabulated, coded and analysed. Numerical values variables were presented, as mean and standard deviation while categorical variables were presented as frequency and percentage. For the numerical values students test ‘t’ was used whether appropriate for comparison between the two groups. For the categorical variables, a chi-square test was used. A difference with a P value <0.05 was considered statistically

significant. The statistical software Epi Info 3.5, 3 was used for the analysis of data and Microsoft Word and Excel have been used to generate graphs and tables.

Age							
Group	**N**	**Minimum**	**Maximum**	**Mean**	**SD**	**t-value**	**P-value**
Dexmedetomidine	40	19.0	56.0	37.5	10.3	0.99	033
Lignocaine	40	18	55.0	35.00	11.6		

Table No.1: Mean Age Distribution in Both Groups

The range for ages was 19-56 years for the Dexmedetomidine group and 18-55 years for the Lignocaine group. Both the groups had a similar weight distribution with overweight and obese people left out of the study.



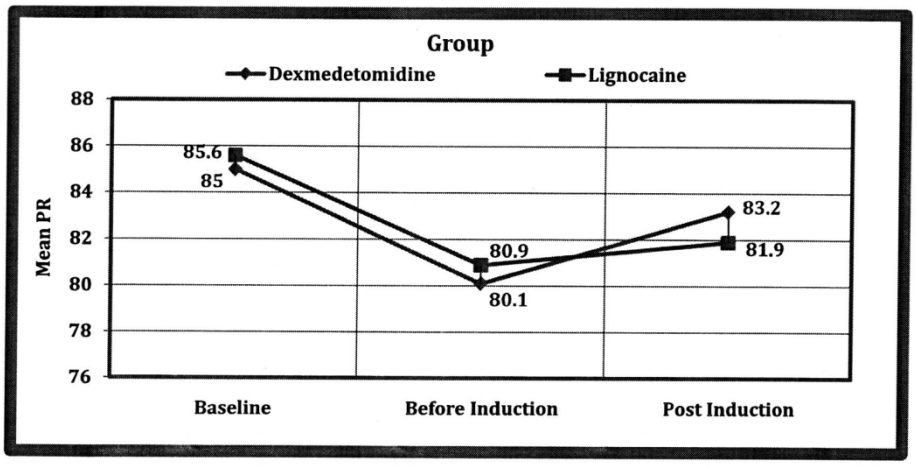
Graph 1: Showing the Graphical Representation of Age Wise Distribution of The Patients Involved in Both the Groups.

There was no statistically significant difference between the groups. This implies that the patients for each group were selected randomly without showing any bias.

PR (per min)	Group										t-value	p-value
	D					L						
	N	Minimum	Maximum	Mean	SD	N	Minimum	Maximum	Mean	SD		
Baseline	40	67	120	85	10.9	40	65	105	85.6	8.9	-0.28	0.78
Before Induction	40	50	105	80.1	11.3	40	60	99	80.9	8.3	-0.35	0.728
Post Induction	40	58	108	83.2	106	40	60	98	81.9	7.9	-0.56	0.576

Table 2: Showing Changes In Pulse Rate (P.R) Prior To Intubation

Tests of significance between groups were carried out by student's t-test or modified t-test. For the changes in Pulse Rate, No significant difference was observed between lignocaine and dexmedetomidine group values (p value> 0.05), prior to intubation.



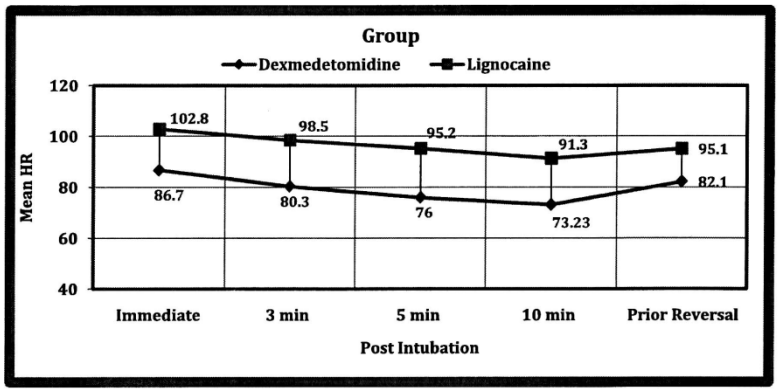
Graph 2: Showing The Changes in P.R Prior To Intubation

PR (per min)	Group										t-value	p-value
	D					L						
	N	Minimum	Maximum	Mean	SD	N	Minimum	Maximum	Mean	SD		
Immediate	40	67	108	86.7	8.7	40	80	130	102.8	11.4	-7.14	<0.001
3 min	40	58	102	80.3	8.3	40	75	121	98.5	12	-7.91	<0.001
5 min	40	57	100	76	7.7	40	72	118	95.2	12.2	-8.41	<0.001
10 min	40	60	98	73.23	7.1	40	69	114	91.3	12.3	-8.04	<0.001
Prior Reversal	40	64	96	82.1	6.5	40	75	110	95.1	9.5	-7.11	<0.001

Table 3 :Comparison of PR of Dexmedetomidine and lignocaine groups for the post-intubation response.

In Lignocaine group the pulse rate showed a continuous rise compared to the baseline; i.e. pre intubation values.

In the Dexmedetomidine group, the pulse rate was increased, compared to baseline; i.e. pre intubation values. But the rise is significantly small than that of the rise in lignocaine group ($p<0.05$). The difference is statistically significant ($p<0.05$) in the dexmedetomidine group compared to lignocaine group.

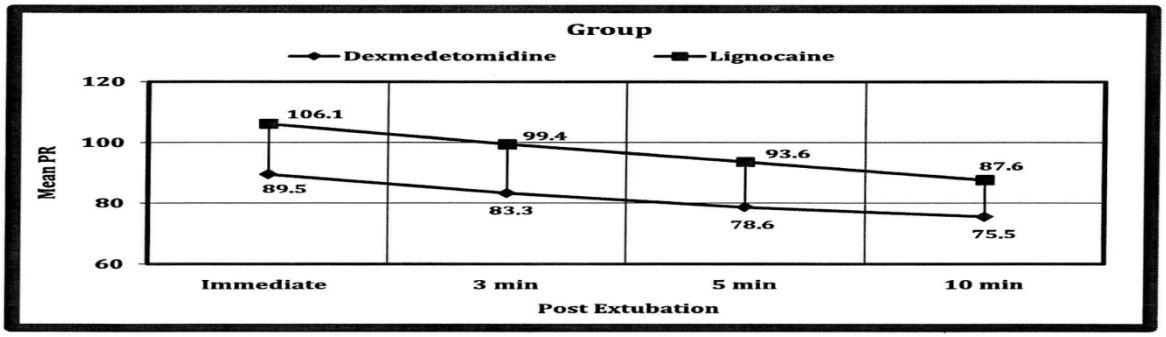


Graph 3: Showing The Changes in P.R Post Intubation With Both The Groups.

PR (per min)	Group										t-value	p-value
	D					L						
	N	Minimum	Maximum	Mean	SD	N	Minimum	Maximum	Mean	SD		
Immediate	40	72	120	89.5	8.8	40	82	125	106.1	9.3	-8.21	<0.001
3 min	40	66	104	83.3	6.6	40	80	118	99.4	9	-9.12	<0.001
5 min	40	66	97	78.6	5.8	40	72	110	93.6	9.2	-8.75	<0.001
10 min	40	62	92	75.5	5.5	40	66	106	87.6	9	-7.23	<0.001

Table 4: Showing Changes In P.R Post Extubation With Both The Groups

Both lignocaine and Dexmedetomidine showed attenuation of Pulse Rate response to extubation. But post-extubation, the mean pulse rate was much less with Inj Dexmedetomidine in comparison with Inj Lignocaine.

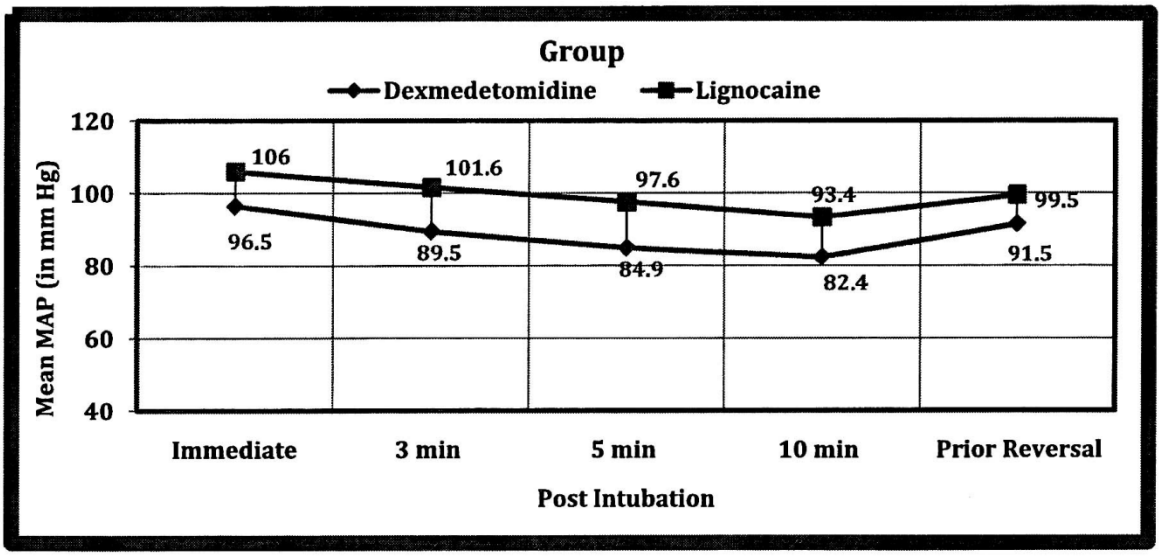


Graph 4: Showing Changes in P.R Post Extubation With Both The Groups

MAP (in mm Hg)	Group										t-value	p-value
	D					L						
	N	Minimum	Maximum	Mean	SD	N	Minimum	Maximum	Mean	SD		
Baseline	40	77	115	94.5	8.4	40	78	115	93	6.5	0.85	0.401
Before Induction	40	59	108	88.2	8.8	40	76	102	90.2	5.9	-1.16	0.25
Post Induction	40	68	105	86.5	8.6	40	76	101	88.1	7	-0.89	0.37

Table 5: Showing Changes in Mean Arterial Pressure (MAP) Prior to Intubation

During intubation, there is an increase in MAP in both the groups but after intubation, there is attenuation of pressor response in Dexmedetomidine group, with significant statistical difference ($p<0.05$) between the groups which continued till 10 minutes post intubation.



Graph 5: Showing MAP of Dexmedetomidine and Lignocaine Groups for Post Intubation Response.

MAP (in mm Hg) Post Extubation	Group										t-value	p-value
	D					L						
	N	Minimum	Maximum	Mean	SD	N	Minimum	Maximum	Mean	SD		
Immediate	40	86	153	97.6	12.6	40	93	125	107.6	7.1	-4.34	<0.001
3 min	40	82	113	91.9	6.9	40	88	126	103.1	7.4	-7.06	<0.001
5 min	40	76	110	87.9	6.8	40	82	115	97	5.6	-6.54	<0.001
10 min	40	68	104	85.1	7.5	40	77	113	94.3	6.6	-5.81	<0.001

Table 6 : Showing Changes in MAP Post Extubation With Both The Groups

IV. Discussion

The haemodynamic response to laryngoscopy and endotracheal intubation has been a topic of discussion right since 1940.These hemodynamic responses were first recognised in 1940 by Reid and Bruce et al. [1].Burstein et al. 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. The efferent sympathetic outflow to the heart is T1-T4, while that to adrenal medulla is from T3-L3

Stanley Tam, Frances Chung, Michael Campbell [4] made a study for determining optimal time of injection of IV lignocaine for the attenuation of circulatory responses secondary to endotracheal intubation. Four groups received lignocaine, 1.5 mg/kg IV single bolus over a period of less than 5 seconds 1,2,3 and 5 min before intubation. The fifth group served as the control and received no IV lignocaine. They observed that cardiovascular responses were significantly above baseline levels in patients given lignocaine 1 min group ($p < 0.05$), 2 min group ($p < 0.05$), and 5 min group ($p < 0.05$) before intubation or in controls ($p < 0.05$) compared to IV lignocaine 3 min before intubation group.

In our study Plain preservative free Lignocaine 2%, 1.5 mg/kg IV bolus was given over a period of 10 minutes prior to laryngoscopy. The results were in accordance with 1 min, 2 min & 5 min group of Stanley Tam et al. study, i.e., cardiovascular responses were significantly above baseline levels.

Robert Stoelting[2] found that lignocaine in 1.5 mg/kg intravenously can attenuate the pressor response effectively in a study conducted on 36 known heart disease patients scheduled for non-cardiac major surgeries.

Koppert W et al. conducted a study [6]to determine the time course of the analgesic and antihyperalgesic mechanisms of perioperative lignocaine administration. Forty patients undergoing major abdominal surgery participated in this randomized and double-blinded study. Twenty patients received lignocaine 2% (bolus injection of 1.5 mg/kg in 10 min followed by an IV infusion of 1.5 mg/kg/h), and 20 patients received saline placebo. The infusion started 30 min before skin incision and was stopped 1 h after the end of surgery. Lignocaine blood concentrations were measured. Postoperative pain ratings (numeric rating scale of 0-10) and morphine consumption (patient-controlled analgesia) were assessed up to 72 h after surgery.

Mean lignocaine levels during surgery were 1.9 +/- 0.7 mcg/ml. Patient-controlled analgesia with morphine produced good postoperative analgesia (numeric rating scale at rest, <or=3; 90%-95%; no group differences). Patients who received lignocaine reported less pain during movement and needed less morphine during the first 72 h after surgery (103.1 +/- 72.0 mg versus 159.0 +/- 73.3 mg; Student's t-test; $P < 0.05$).These results are in accordance with results of the present study.

Cassuto J et al. conducted a study [7]to determine the efficacy of a continuous low-dose (2 mg/kg) intravenous infusion of lignocaine, postoperative pain (visual analogue pain scale) and the requirements for postoperative analgesics were measured in a double-blind, randomized trial in 20 patients after cholecystectomy. Lignocaine infusion was started 30 min before the operation and continued for 24 hr after surgery (n = 10). Saline was infused in a comparable group of ten patients.

McCarthy GC et al. conducted a systematic review[10] to determine the overall efficacy of intravenous lignocaine infusion on postoperative analgesia and recovery from surgery in patients undergoing various surgical procedures. They searched for randomized controlled comparisons of lignocaine infusion with placebo in the surgical setting and reporting on postoperative analgesia and other aspects of patient recovery from surgery. The quality of all included studies was assessed using the Modified Oxford Scale. Information on postoperative pain intensity and analgesic requirements were extracted from the trials and compared qualitatively.

Opioid consumption was reduced by up to 85% in lignocaine-treated patients when compared with controls. Infusion of lignocaine also resulted in the earlier return of bowel function, allowing for earlier rehabilitation and shorter duration of hospital stay. First flatus occurred up to 23 hours earlier, while the first bowel movement occurred up to 28 hours earlier in the lignocaine-treated patients. Duration of hospital stay was reduced by an average of 1.1 days in the lignocaine-treated patients. Administration of intravenous lignocaine infusion did not result in toxicity or clinically significant adverse events.

Stimulation of alpha-2b receptor in vascular smooth muscle seems to be responsible for the initial rise in the blood pressure in various dexmedetomidine studies, which can be attenuated by a slow infusion. However, even at slower infusion rates, the increase in mean arterial pressure over the first 10 minutes was shown to be in the range of 7 % with a decrease in H.R between 16 % and 18 %.

In our study, only 2 out of 40 patients in the dexmedetomidine group showed elevated BP at 2-3 minutes after starting of dexmedetomidine infusion. Apart from that, there was a fall of Heart Rate by 7 % and fall of MAP by 8 % at 10 min after starting dexmedetomidine. Arterial BP decrease has shown to be varied in different studies in accordance with varying dexmedetomidine doses, infusion speed, premedication and fluid infusion before drug administration.

Also when compared to the lignocaine group, there is a significant difference ($p<0.05$), and this difference continues up to 10 min post intubation. Regarding DBP, our study differs from other studies as it is decreased from the baseline values at all the recordings. Adverse effects related to dexmedetomidine are hypotension and bradycardia in several studies. But after their protocols were modified by slow infusion of a loading dose of dexmedetomidine, these side effects were hardly observed.

But clinically all the patients were stable and maintained MAP > 65 mmHg and HR > 50/min; thus none of these patients was intervened with a vasopressor. Literature is very few regarding stabilisation of blood pressure after a fall noticed due to dexmedetomidine and the use of the ideal drug with its dose.

Anaesthetic sparing effects of dexmedetomidine may be due to its effects of CNS [12] . Clonidine has a ceiling effect in this aspect because of its alpha 1 agonistic activity, but ceiling effect of dexmedetomidine is not yet observed. The mechanism is still to be found out. However, it has been seen that in rats, dexmedetomidine decreased MAC of halothane by >90% [11], in patients with abdominal hysterectomy, dexmedetomidine decreased the MAC of isoflurane by 90% [12,] and in another study, it decreased the MAC of sevoflurane by 92 %. In our study, though it was not recorded, decreased requirement of inhalational anaesthesia was felt clinically.

In our study, the induction agent requirement was lessened by 30% in the dexmedetomidine group in comparison with lignocaine group. Earlier reports quote that with dexmedetomidine 1mcg/kg the thiopentone requirement is decreased by 55% and with 0.5 mcg/kg it is decreased by 37% [12]. The disparity in results in our study may be due to different doses of premedication administered to the patients, e.g. in our study; we used 1 mcg/kg fentanyl and 0.05 mg/kg midazolam in both the groups.

Sulaiman S et al. [13] studied the efficacy of intravenous dexmedetomidine for attenuation of the cardiovascular response to laryngoscopy and endotracheal intubation in patients with coronary artery disease. Dexmedetomidine at a dose of 0.5 mcg/kg as 10 min infusion was administered prior to induction of general anaesthesia attenuated the sympathetic response to laryngoscopy and intubation in patients undergoing myocardial revascularization.

Thoma BN [14] et al. conducted a retrospective cohort study on patients undergoing isolated, elective CABG surgery. The patients who were sedated with either propofol or dexmedetomidine during the study period were included. The primary outcome was the number of patients achieving a postoperative duration of mechanical ventilation </= 6 hrs. Secondary outcomes were post-operative intensive care unit length of stay </= 48 hrs, total postoperative length of stay </= 5 days, the need for adjunctive opioid therapy and associated cost savings.

Findings suggest that use of dexmedetomidine as an alternative to propofol for sedation of CABG patients postoperatively contributes to reduced mechanical ventilation time, ICU length of stay and postoperative length of stay. Higher drug costs resulting from the propofol shortage were offset by savings in the postoperative room and board costs. Additional savings may be possible by preventing medical complications to the extent possible.

Sairaku A et al. [15] tested the use of dexmedetomidine as a procedural sedative during ablation of atrial fibrillation. Patients were randomized to be treated with dexmedetomidine(n=43) or thiamylal (n=44) as sedatives during atrial fibrillation ablation. Apnoeic and body movement events were monitored, during the procedure. The occurrence of hypotension [p=0.14] and bradycardia [p=1.0] was similar in patients with dexmedetomidine and thiamylal. Therefore, dexmedetomidine is a potential alternative for that with GABAergic anaesthetics.

Kim YS et al. [16] investigated the optimum dosage of dexmedetomidine for prevention of post-anaesthetic shivering in patients scheduled for an elective laparoscopic total hysterectomy. They concluded that dexmedetomidine 0.75 mcg/kg or 1 mcg/kg provides effective prophylaxis against post-operative shivering as well as an analgesic effect.

Hall JE et al. [18] conducted research which determined the safety and efficacy of two small-dose infusions of dexmedetomidine by evaluating sedation, analgesia, cognition, and cardiorespiratory function. 7 healthy young volunteers participated on three occasions with random assignment to drug or placebo. Heart rate, blood pressure, respiratory rate, ETCO2, O2 saturation, and processed electroencephalogram (bispectral analysis) were monitored. Baseline hemodynamic measurements were acquired, and psychometric tests were performed (visual analogue scale for sedation; observer's assessment of alertness/sedation scale; digit symbol substitution test; and memory).

The pain from a 1-min cold pressor test was quantified with a visual analogue scale. After a 10-min initial dose of saline or 1 mcg/Kg dexmedetomidine, volunteers received 50-min IV infusions of saline, or 0.2 or 0.6 microgram/kg/hr dexmedetomidine. Measurements were repeated at the end of infusion and during recovery. The two dexmedetomidine infusions resulted in similar and significant sedation (30%-60%), impairment of memory (approximately 50%), and psychomotor performance (28%-41%). Hemodynamics, oxygen saturation, $ETCO_2$, and respiratory rate were well preserved throughout the infusion and recovery periods. Pain to the cold pressor test was reduced by 30% during dexmedetomidine infusion. Small-dose dexmedetomidine provided sedation, analgesia, and memory and cognitive impairment.

Gurbet A et al. [19] conducted a prospective, randomized, double-blind study to assess whether intraoperative infusion of dexmedetomidine provides effective postoperative analgesia. Postoperative pain scores and morphine consumption were compared in a treated group and a placebo group, both of which received patient-controlled morphine after total abdominal hysterectomy. Fifty women were randomly assigned to two groups. Group D (n = 25) received a loading dose of dexmedetomidine 1 mcg/kg iv during induction of anaesthesia, followed by a continuous infusion at a rate of 0.5 mcg/kg/hr throughout the operation. Group P (n = 25) received a volume-matched bolus and infusion of placebo (0.9% saline).

For each case, heart rate, peripheral oxygen saturation, and systolic and diastolic blood pressure were recorded intra-operatively and for 48 hr postoperatively. Patients used a patient-controlled analgesia device to receive bolus doses of morphine after surgery. Total morphine consumption, pain scores, and sedation scores were recorded for the first 48 hrs (two hours in the postanaesthesia care unit and 46 hrs on the ward). The groups were similar with respect to mean times to extubation of the trachea. Pain and sedation scores were also similar between groups at all corresponding times throughout the 48-hr period of observation.

Group D patients consumed significantly less morphine in the postanaesthesia care unit and on the ward ($P < 0.05$ and $P < 0.01$, respectively). Fewer patients in Group D experienced itching or nausea/vomiting ($P < 0.05$). Thus, it was concluded that Continuous i.v Dexmedetomidine during abdominal surgery provides effective postoperative analgesia, and reduces postoperative morphine requirements without increasing the incidence of side effects. The results of our study were similar to the results noted during this study.

The pain-free interval and analgesic requirement in the first 24 h postoperatively was significantly less in the Group 'L' as compared to the Group 'D' of this study. A systemic review of randomised controlled trials on impact of IV lignocaine infusion on postoperative analgesia and recovery from surgery concluded that IV lignocaine infusion in the peri-operative period is safe and such patients had lower pain scores, reduced postoperative analgesic requirement, decreased intraoperative anaesthetic requirement, faster return of bowel function, and decreased length of hospital stay. [10]

The analgesic action was studied only till 24 h postoperatively and found IV lignocaine to be significant. Other studies have reported significant analgesic action varying from 2 to 48 hours postoperatively [5,7,8].

V. Conclusions And Summary

The following conclusions can be drawn from this study:

- Dexmedetomidine in a bolus dose of 1 mcg/kg followed by continuous infusion of 0.25 mcg/kg/hr attenuates pulse rate and mean arterial pressure response to laryngoscopy, intubation and extubation effectively than plain preservative free lignocaine.
- The peak response of pulse rate and MAP to intubation was observed during laryngoscopy and intubation in both the groups.
- Though there was a rise in pulse rate and MAP in the Dexmedetomidine group, the rise was not significant when compared with the same in the lignocaine group.
- However, the basal values of heart rate were reached within 3 min after intubation and extubation in Dexmedetomidine group.

In case of Lignocaine group, the peak response, i.e. maximum increase in haemodynamic parameters to intubation was observed during laryngoscopy, intubation & extubation and they failed to return to basal values even 5 min after intubation and extubation.

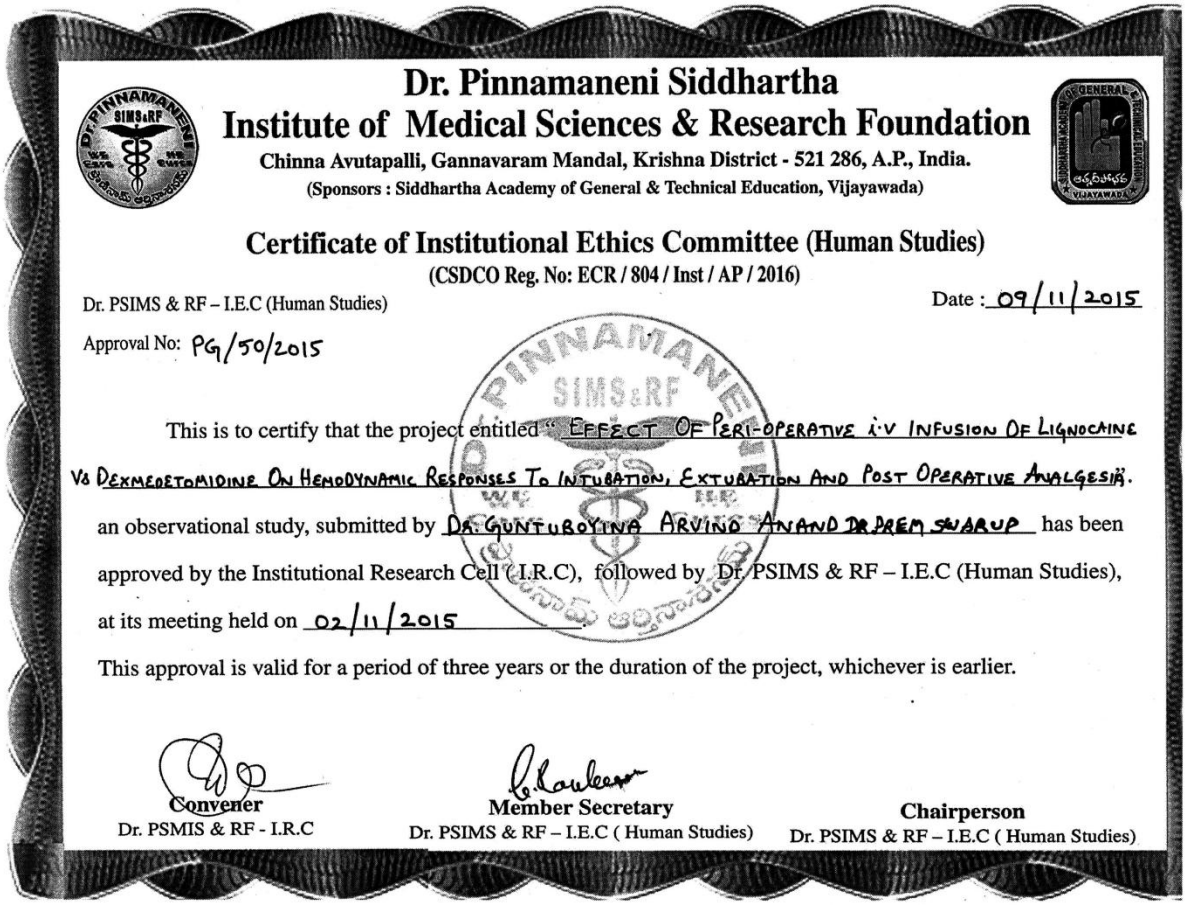
- No ECG changes were noted in all the groups.
- The pain-free interval was significantly more in the Lignocaine group as compared to the Dexmedetomidine group.

This study shows that Dexmedetomidine is superior to Lignocaine in attenuation of pressor response and ideal time for administration would be about 10 min prior to intubation.

This study also shows that Lignocaine is superior to Dexmedetomidine for the increased pain-free period and better postoperative analgesia.

Acknowledgement

We are thankful to the HOD of Anesthesia Dr U.S.S.A. Vara Prasad
moreover, Staff the Department of Anesthesia Dr P.S.I.M.S. & R.F.



Dr. Pinnamaneni Siddhartha
Institute of Medical Sciences & Research Foundation
Chinna Avutapalli, Gannavaram Mandal, Krishna District - 521 286, A.P., India.
(Sponsors : Siddhartha Academy of General & Technical Education, Vijayawada)

Certificate of Institutional Ethics Committee (Human Studies)
(CSDCO Reg. No: ECR / 804 / Inst / AP / 2016)

Dr. PSIMS & RF – I.E.C (Human Studies)

Date : 09/11/2015

Approval No: PG/50/2015

This is to certify that the project entitled " EFFECT OF PERI-OPERATIVE i.V INFUSION OF LIGNOCAINE Vs DEXMEDETOMIDINE ON HEMODYNAMIC RESPONSES TO INTUBATION, EXTUBATION AND POST OPERATIVE ANALGESIA".

an observational study, submitted by DR. GUNTUBOYINA ARVIND ANAND DR PREM SWARUP has been approved by the Institutional Research Cell (I.R.C), followed by Dr. PSIMS & RF – I.E.C (Human Studies), at its meeting held on 02/11/2015

This approval is valid for a period of three years or the duration of the project, whichever is earlier.

Convener
Dr. PSMIS & RF - I.R.C

Member Secretary
Dr. PSIMS & RF – I.E.C (Human Studies)

Chairperson
Dr. PSIMS & RF – I.E.C (Human Studies)

References

[1]. Burstein Cl , Lopinto FJ and Newman W : Electrocardiographic studies during endotracheal intubation, Effects during usual routine technics, Anaesthesiology 1950 March, 11(2): 224-37.

[2]. Stoelting RK: Circulatory changes during direct laryngoscopy and tracheal intubation. Influence of duration of laryngoscopy with or without prior lidocaine. Anaesthesiology 1977, 47:381-383.

[3]. Clayton DG , Allt Graham J. Intravenous lignocaine in dental anaesthesia. The effect of pretreatment on the incidence of dysrhythmias. Anaesthesia 1983, 38: 1066-1070.

[4]. Stanley Tam MD FRCP, Frances Chung MD FRCP and Michael Campbell MD FRCP. Intravenous lignocaine: optimal time for injection before tracheal intubation. AnesthAnalg 1987;66: 1036-1038.

[5]. Wu CT, Borel CO, Lee MS, Yu JC, Liou HS, Yi HD, et al. The interaction effect of perioperative cotreatment with dextromethorphan and intravenous lidocaine on pain relief and recovery of bowel function after laparoscopic cholecystectomy. AnesthAnalg. 2005;100:448–53. [PubMed: 15673874]

[6]. Koppert W, Weigand M, Neumann F, Sittl R, Schuettler J, Schmelz M, et al. Perioperative intravenous lidocaine has preventive effects on postoperative pain and morphine consumption after major abdominal surgery. AnesthAnalg. 2004;98:1050–5. [PubMed: 15041597].

[7]. Cassuto J, Wallin G, Högström S, Faxén A, Rimbäck G. Inhibition of postoperative pain by continuous low-dose intravenous infusion of lidocaine. AnesthAnalg. 1985;64:971–4. [PubMed: 3898920]

[8]. Groudine SB, Fisher HA, Kaufman RP, Jr, Patel MK, Wilkins LJ, Mehta SA, et al. Intravenous lidocaine speeds the return of bowel function, decreases postoperative pain and shortens hospital stay in patients undergoing radical retropubic prostatectomy. AnesthAnalg. 1998;86:235–9. [PubMed: 9459225]

[9]. Nishina K, Mikawa K, Takao Y, Shiga M, Maekawa N, Obara H. Prostaglandin E1, lidocaine, and prostaglandin E1-lidocaine combination for attenuating cardiovascular responses to extubation. Can J Anaesth. 1997;44:1211–4. [PubMed: 9398965]

[10]. McCarthy GC, Megalla SA, Habib AS. Impact of intravenous lidocaine infusion on postoperative analgesia and recovery from surgery: A systematic review of randomised controlled trials. Drugs. 2010;70:1149–63. [PubMed: 20518581

[11]. Aho M, Lehtinen AM, ErkolaO , et al. The effects of Intravenously administered dexmedetomidine in perioperative haemodynamics and Isoflurane requirements in patients undergoing an abdominal hysterectomy. Anaesthesiology 1991; 74:997-1002

[12]. Aantaa R, Kanto J, Scheinin M. Dexmedetomidine Premedication in minor gynecologic surgery AnaesthAnalg 1990;70:407-13
[13]. Sulaiman S; Karthikeyan RB; Vakamudi M; Sundar AS; Ravullapalli H; Gandham R, Annals Of Cardiac Anaesthesia [Ann card Anaesth] 2012; 15: 39-43;
[14]. Thomas BN; Li J; McDaniel CM; Wordell CJ; Cavarocchi N; Pizzi LT, the Clinical and economic impact of substituting dexmedetomidine for propofol due to a US drug shortage: Examination of coronary artery bypass graft patients at an urban medical centre. Pharmacoeconomics , 2014 ; 32: 149-57
[15]. SairakuA; Yoshida Y; Hirayama H; Nakano Y; Ando M; Kihara Y, Procedural sedation with dexmedetomidine during ablation of atrial fibrillation: a randomized controlled trial. Europace: European pacing, Arrhythmias, And cardiac electrophysiology
[16]. Kim YS; Kim YI; Seo KH; Kang HR, Optimal Dose of Prophylactic dexmedetomidine for preventing postoperative shivering, Int J Med Sci, 2013; 10: 1327-32.
[17]. Le Guen M; Liu N; Tounou F; Auge M; Tuil O; Chazot T; Dardelle D; Laloe PA; Bonnet F ; Sessler DI; Fischler M, Dexmedetomidine reduces propofol and remifentanil requirements during bispectral index-guided closed-loop anesthesia : a double-blind, Placebo-controlled trial. AnesthAnalg , 2014, 118: 946-55;
[18]. Hall JE, Uhrich TD, Barney JA, Arain SR, Ebert TJ. Sedative, amnestic, and analgesic Properties of small-dose dexmedetomidine infusions. AnesthAnalg. 2000;90:699–705.
[19]. Gurbet A, Basagan-Mogol E, Turker G, Ugun F, Kaya FN, Ozcan B. Intraoperative Infusion of dexmedetomidine reduces perioperative analgesic requirements. Can J Anaesth. 2006 Jul;53(7):646-52.
[20]. Belleville JP, Ward DS, Bloor BC. Effects of intravenous dexmedetomidine in humans: Sedation, Ventilation and metabolic rate. Anaesthesiology 1992; T7:1125-33

Dr. V. Prem Swarup. "Effect of Perioperative I.V. Infusion of Dexmetidomidine Vs Lignocaine on Haemodynamic Responses to Intubation, Extubation and Postoperative Analgesia– An Institutional Study"." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 17, no. 12, 2018, pp 05-14.