ATTENUATION OF PRESSOR RESPONSE DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION WITH INTRAVENOUS DEXMEDETOMIDINE- A CLINICAL STUDY

Balasubramanyam V[1], Sunku Prasad[2], Talisetti Jamuna[3]

[1]Associate Professor, Department of Anaesthesiology and Critical Care, S.V. Medical College / SVRR Government General Hospital, Tirupati, Andhra Pradesh.
[2]Postgraduate Student, Department of Anaesthesiology and Critical Care, S.V. Medical College / SVRR Government General Hospital, Tirupati, Andhra Pradesh.
[3]Professor, Department of Anaesthesiology and Critical Care, S.V. Medical College / SVRR Government General Hospital, Tirupati, Andhra Pradesh.

ABSTRACT

BACKGROUND
Laryngoscopic manipulation and endotracheal intubation are very painful stimuli capable of producing tachycardia, arrhythmia and hypertension. The aim of the study was to evaluate the efficacy of single premedication dose of I. V dexmedetomidine in attenuating pressor response to laryngoscopy & endotracheal intubation.

MATERIALS AND METHODS
This study was carried out at S.V. Medical College, Tirupati for one year period. The study was undertaken after obtaining ethical committee clearance as well as written informed consent from all patients. 60 patients in the age group 20-40 yrs. of either sex, belonging to ASA grade I and II scheduled for elective surgical procedures under General anaesthesia were included. 60 patients aged between 20 to 40 years belonging to ASA grade I & II were randomly divided into 2 groups, each group consists of 30 patients Group I (Saline group) 100 ml normal saline infused, Group II (Dexmedetomidine group) 1 mcg/kg in 100 ml normal saline infused over 15 min. The hemodynamic parameters were monitored from baseline upto 10 min after intubation.

RESULTS
Demographic data were analysed by student's t test. Analysis of variance for repeated measures (ANOVA) was used to analyse changes over time. The statistical software SPSS version 16.0 was used for the analysis of the data and Microsoft Word and Excel have been used to generate graphs, tables etc. There was statistically significant difference ($p<0.05$) between dexmedetomidine and normal saline in heart rate, systolic, diastolic, mean arterial pressure at all time points after tracheal intubation with dexmedetomidine.

CONCLUSION
Dexmedetomidine in the dose of 1 µg/kg as IV infusion, given 15 minutes before induction can be used safely to attenuate the pressor response to laryngoscopy and intubation without significant side effects.

KEYWORDS
General anaesthesia, Dexmedetomidine, Intubation, Laryngoscopy.

HOW TO CITE THIS ARTICLE: Balasubramanyam V, Prasad S, Jamuna T. Attenuation of pressor response during laryngoscopy and endotracheal intubation with intravenous dexmedetomidine- a clinical study. J. Evid. Based Med. Healthc. 2017; 4(94), 5841-5848. DOI: 10.18410/jebmh/2017/1176

BACKGROUND

Endotracheal intubation is the translaryngeal placement of a tube into the trachea via the nose or mouth. Endotracheal intubation includes laryngoscopy & intubation. The process of laryngoscopy & intubation are noxious stimuli & therefore constitute a period of extreme haemodynamic stress and is associated with intense sympathetic activity marked by tachycardia & hypertension.[1]

The cardiovascular response is a reflex phenomenon. This is mediated by vagus (X) & Glossopharyngeal (IX) cranial nerves. Vagus & Glossopharyngeal nerves carry the afferent stimulus from epiglottis & infraglottic region & activate the vasomotor centre to cause a peripheral sympathetic adrenal response to release adrenaline & noradrenaline. The increase in Pulse rate, Blood pressure are usually transitory, variable & unpredictable. Normal, healthy persons tolerate this response, but in susceptible individuals, this transient sympathetic response can evoke life–threatening condition. Alpha-2 agonists have been used for attenuating the sympathetic response.[2] and among α-2 agonists both clonidine and dexmedetomidine appear to fulfil all the above criteria. Both Clonidine and dexmedetomidine have actions on both α-1 and α-2 receptors but Dexmedetomidine is highly specific and selective α-2 adrenoceptor agonist with α2:α1 binding selectivity ratio of 1620:1 compared to 220:1 for clonidine.

Financial or Other, Competing Interest: None.
Submission 25-11-2017, Peer Review 07-12-2017,
Acceptance 14-12-2017, Published 16-12-2017.
Corresponding Author:
Dr. Balasubramanyam V,
#20-48-S3-299, Rayalavari, Street, Yerramitta, Tirupati- 517501,
E-mail: balasubramanyamvallem@gmail.com
DOI: 10.18410/jebmh/2017/1176

Various studies have also found that dexmedetomidine can decrease the hemodynamic response to laryngoscopy and intubation.[3,4,5,6]

The study was undertaken to evaluate the effects of recently introduced alpha-2 agonist, dexmedetomidine as premedication in attenuating pressor response during Laryngoscopy & Endotracheal intubation.

Aims and Objectives

Aim- This randomized prospective study was done to evaluate the efficacy of single premedication dose of I.V. dexmedetomidine in attenuating pressor response to laryngoscopy & endotracheal intubation.

Objectives- To evaluate the efficacy of single premedication dose of intravenous dexmedetomidine 1 µg/kg body weight in

1. Attenuating the pressor response to laryngoscopy and endotracheal intubation
2. Post anaesthesia Recovery characteristics
3. Side effects if any.

MATERIALS AND METHODS

Design-This study was carried out at S.V. Medical College, Tirupati for one year period. The study was undertaken after obtaining ethical committee clearance as well as written informed consent from all patients. 60 patients in the age group 20-40 yrs. of either sex, belonging to ASA grade I and II scheduled for elective surgical procedures under General anaesthesia were included.

Inclusion Criteria-

1. Patients aged between 20-40 yrs. of either sex, with ASA grade I & II scheduled for elective surgical procedures under general anaesthesia.

Exclusion Criteria-

1. Patients with anticipated difficult airway
2. Patients on anti-hypertensive drugs
3. Patients on sedatives, hypnotics& antidepressants
4. H/o cardiovascular, respiratory, hepatic, renal diseases.
5. Patients with ASA grade III & above.

Technique of Anesthesia/Procedure - 60 patients aged between 20 to 40 years belonging to ASA grade I & II were randomly divided into 2 groups, each group consists of 30 patients.

Group I (Saline group)

Group II (Dexmedetomidine group)

After recording the baseline vitals in the operating room,

Group I (Saline group) patients received 100 ml normal saline infused over 15 mins.

Group II (Dexmedetomidine group) patients received Intravenous dexmedetomidine 1 µg per kg in 100 ml normal saline infused over 15 mins.

After 5 mins of stabilizing period SBP, DBP, MAP, Heart rate, SpO_2 (T1) was recorded. Prior to induction, Inj. Glycopyrrolate 0.2 mg, Inj Ondansetron 4 mg, & Inj. Rantac 50 mg administered IV.

All patients were pre- Oxygenated for 4 mins & Anaesthesia induced with 5 mg/kg Thiopentone sodium (2.5%). After successful trial ventilation with 100% oxygen, Vecuronium 0.1 mg /kg given to facilitate laryngoscopy & intubation. Oxygenation continued by positive pressure mask ventilation using Bains circuit. Maintained with 50% O_2 and 50% N_2O. At 2mins after induction, SBP, DBP, MAP, Heart rate & SpO_2 was recorded (T2) At 3 min after induction, using laryngoscope with a Macintosh blade intubation was done with well lubricated, appropriate sized cuffed, disposable oral endotracheal tube by an experienced anaesthesiologist and accomplished within 20 sec. SBP, DBP, MAP, Heart rate, SpO_2 were recorded.

SBP, DBP, MAP, Heart rate, SpO_2 were recorded at 1 (T3), 3 (T4), 5 (T5), & 10min (T6) after laryngoscopy & intubation.

Sequence	SBP, DBP, MAP, Heart rate, SpO_2 Recording
Basal reading when the patient is shifted to OT	T0
At 5 min after infusion of dexmedetomidine/saline	T1
At Induction (2 min after Thiopentone Sodium + Vecuronium)	T2
At 1 min after intubation	T3
At 3 min after intubation	T4
At 5 min after intubation	T5
At 10 min after intubation	T6

Surgery commenced at the end of 10 min after laryngoscopy & intubation. No form of stimulus was applied during this period. After adequate clinical recovery patients shifted to post anaesthesia care unit, observed for 2 hrs. for Nausea vomiting, Bradycardia, Hypotension, & Sedation. Complications like bradycardia and hypotension if occurs the rescue drugs like atropine and ephedrine can be used. After assessing the Steward awakening score (Steward score >12), patient shifted to the ward.

Scores 12-14 indicating excellent recovery; scores 9- 12 indicating satisfactory recovery and scores <9 as poor recovery from general anaesthesia.

In PACU all Patients were monitored for HR, NIBP, ECG continuously. As per steward awakening score, recordings were done once in 15 min for two hours.

Statistical Method Employed- All data are presented as mean ± SD (standard deviation). Demographic data were analysed by student's t test. Analysis of variance for repeated measures (ANOVA) was used to analyse changes over time.

$p<0.01$- Statistically highly significant (HS).

$p<0.05$- Statistically significant (S).

$p>0.05$- Statistically not significant (NS).

Statistical Software- The statistical software SPSS version 16.0 was used for the analysis of the data and Microsoft Word and Excel have been used to generate graphs, tables etc.

OBSERVATION AND RESULTS

Type of Surgery	Group I (Saline) Number of Patients	Group II (Dexmedetomidine) Number of Patients
General surgeries	12	13
Plastic Surgeries	3	3
Ent surgeries	10	11
Neurosurgery	5	3
Total	**30**	**30**

Table 1. Showing the Type of Surgical Procedures

The duration of all the surgeries mention above were in the range of one to two hours.

Time	Saline	Dexmedetomidine	p- value	Remarks
T_0 (Basal)	89.4 ± 12.02	90.6 ± 13.26	0.57	NS
T_1 (Pre-induction)	87.2 ± 11.91	73.83 ± 12.38	0.002	HS
T_2 (Induction)	87.3 ± 11.66	73.2 ± 12.95	0.000	HS
T_3 (1 min)	101.6 ± 11.05	83.06 ± 11.84	0.000	HS
T_4 (3 min)	99.06 ± 11.26	80.13 ± 11.07	0.000	HS
T_5 (5 min)	95.26 ± 11.02	77.83 ± 10.34	0.000	HS
T_6 (10 min)	90.53 ± 9.91	76.16 ± 10.54	0.000	HS

Table 2. Showing the Intergroup Comparison of Mean Heart Rate (BPM) Changes in Response to Laryngoscopy and Intubation between Saline Group and Dexmedetomidine Group

(p<0.01)– Statistically highly significant, (p<0.05) – Statistically significant, (p>0.05)– Statistically Not Significant (NS).

In the Group I (Saline), the basal mean HR was 89.4 ± 12.02 bpm, at preinduction it was 87.2 ± 11.91 bpm and at induction it was 87.3 ± 11.66 bpm. The mean heart rate one minute after intubation was 101.6 ± 11.05, representing a rise of 12.2 bpm from the basal heart rate (13.6%). By 3, 5 and 10 minutes mean HR were 99.06 ± 11.26 bpm, 95.26 ± 11.02 bpm, 90.53 ± 9.91 bpm respectively. The persistent increase in HR was observed even at 10th minute after intubation. The increase in mean HR at 1 min after intubation compared to basal value was statistically HS (p=0.000).

In Group II (Dexmedetomidine), the basal mean HR was 90.6 ± 13.26 bpm, at preinduction it was 73.83 ± 12.38 bpm and at induction it was 73.2 ± 12.95 bpm. The mean heart rate 1 min after intubation was 83.06 ± 11.84 bpm, representing a decrease of 7 bpm from the basal mean heart rate (8.4%). By 3, 5, and 10 minutes the mean HR values were 80.13 ± 11.07 bpm, 77.83 ± 10.34 bpm, and 76.16 ± 10.54 bpm respectively. The decrease in mean HR at 1 min after intubation compared to basal value was statistically HS (p= 0.009).

Statistical evaluation between the groups showed that the basal mean HR between Group I and Group II was statistically not significant (p=0.57), at pre- induction, induction, 1, 3, 5 and 10 minutes after intubation the HR changes were statistically highly significant (p=0.000). Maximum HR changes were observed at 1 min after intubation in both the Groups. In Group II there was 8.4% decrease in HR compared to basal. In Group I there was 13.6% increase in mean HR compared to basal. In Group II there was a constant decrease in HR from the time of pre-induction until 10th min of intubation which when compared to that of Group I was statistically highly significant (p=0.000).

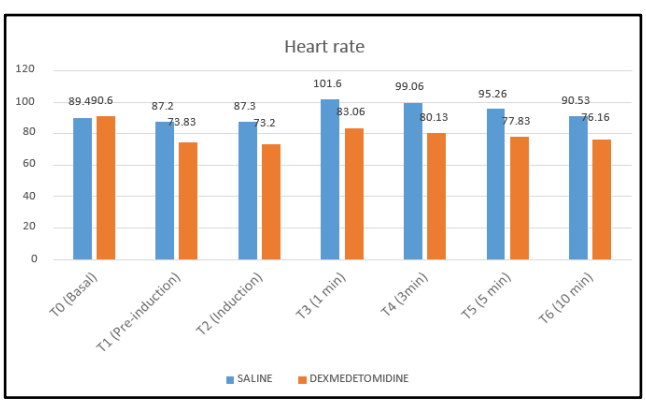

Figure 1. Showing the Intergroup Comparison of Mean Heart Rate (BPM) Changes in Response to Laryngoscopy and Intubation between Saline Group and Dexmedetomidine Group

Time	Saline	Dexmedetomidine	p- value	Remarks
T_0 (Basal)	122.43 ± 8.67	123.46 ± 10.89	0.79	NS
T_1 (Pre-Induction)	119.3 ± 8.45	112.1 ± 11.61	0.02	S
T_2 (Induction)	120.3 ± 7.75	108.5 ± 10.72	0.01	S
T_3 (1 min)	127.9± 7.63	111.9 ± 9.26	0.000	HS
T_4 (3 min)	124.9 ± 7.03	111.06 ± 8.97	0.000	HS
T_5 (5 min)	121.1 ± 6.63	109.2 ± 8.13	0.0001	HS
T_6 (10 min)	118 ± 7.10	106.6 ±8.75	0.0004	HS

Table 3. Showing Intergroup Comparison of Mean Systolic Blood Pressure (SBP in mmHg) Changes in Response to Laryngoscopy and Intubation between Saline Group and Dexmedetomidine Group

(p<0.01) – Statistically highly significant, (p<0.05) – Statistically significant, (p>0.05) - Statistically Not Significant (NS).

In group I (Control) the basal value of mean SBP was 122.43 ± 8.67 mmHg, at pre-induction it was 119.3 ± 8.95 mmHg and at induction it was 120.3 ± 7.75 mmHg. The mean systolic blood pressure 1 min after intubation was 127.9 ± 7.63 mmHg, representing an increase of 5.43 mmHg (4.46%) from the basal value. By 3 min mean SBP values were 124.9± 7.03 mmHg with a rise of 2.5 mmHg (2.04%) from the basal, and at 5 and 10 minutes mean SBP was 121.1 ± 6.63 mmHg and 118 ± 7.10 mmHg respectively. The increase in SBP at 1 min after intubation compared to basal value was statistically significant p=0.04).

In the group II (dexmedetomidine), the basal value of mean SBP was 123.46 ±10.89 mmHg, at pre-induction it was 112.1 ± 11.6 mmHg and at induction it was 108.5±10.72 mmHg representing a decrease in SBP. One minute following intubation the mean SBP was 111.9 ± 9.26 mmHg representing a fall of 11.5 mmHg from the basal value (10.27%). By 3, 5, and 10 minutes mean SBP values were 111.06 ± 8.97 mmHg, 109.2 ± 8.13 mmHg, and 106.6 ±8.75 mmHg respectively. The SBP continued to be below the basal value even after 10 minutes of intubation. The decrease in SBP at 1 min after intubation compared to basal value was statistically HS (p= 0.000).

Statistical evaluation between the groups showed that the basal mean SBP between Group I and Group II was statistically not significant (p=0.79) but the comparison of SBP changes between the 2 Groups at pre-induction and induction was statistically significant (p=0.02, p=0.01) and at 1, 3, 5 and 10 minutes after intubation it was statistically highly significant (p=0.000, p=0.001, p=0.004 respectively). In Group II SBP continued to remain below the basal value from the time of pre-induction until 10th min after intubation which was statistically significant.

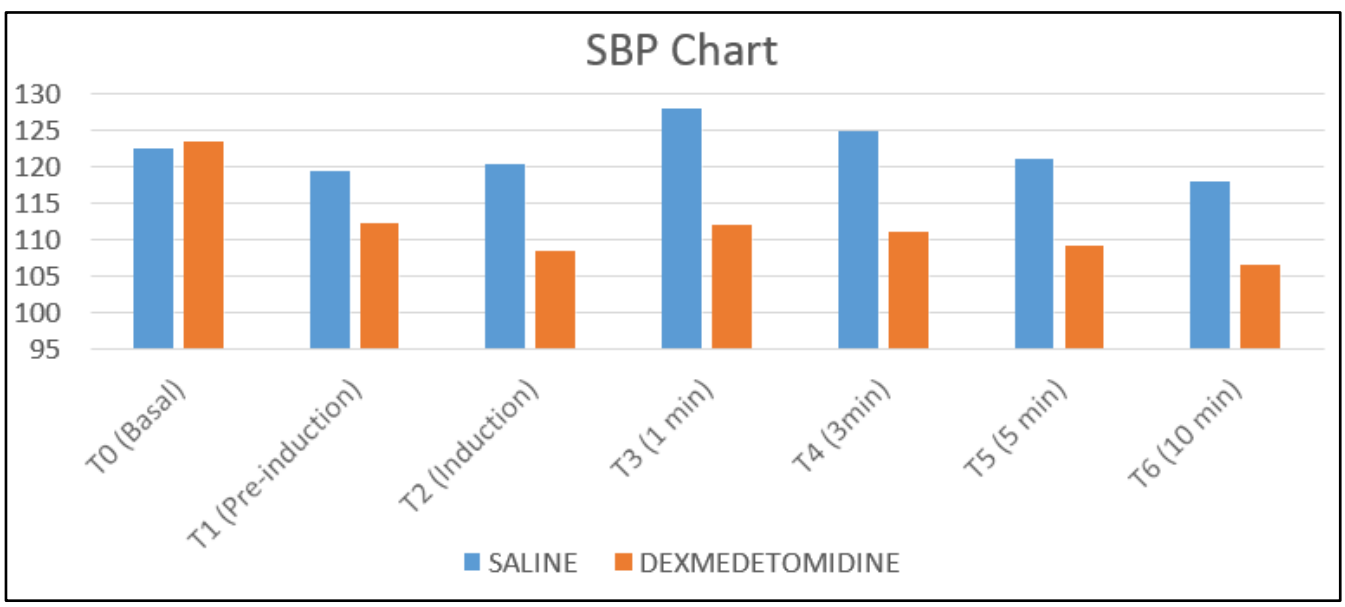

Figure 2. Showing the Intergroup Comparison of Mean SBP (mmHg) Changes in response to Laryngoscopy and Intubation between Saline Group and Dexmedetomidine Group

Time	Saline	Dexmedetomidine	p- value	Remarks
T_0 (Basal)	79.2 ± 7.61	79.46 ± 9.39	0.51	NS
T_1 (Pre-induction)	76.63 ± 8.12	68.6 ± 9.53	0.004	HS
T_2 (Induction)	76.9 ± 6.95	66.03 ± 9.08	0.000	HS
T_3 (1 min)	84 ± 5.75	69.9 ± 9.18	0.000	HS
T_4 (3 min)	81.3 ± 6.28	67.43 ± 8.56	0.001	HS
T_5 (5 min)	78.8 ± 6.15	66.4 ± 7.95	0.000	HS
T_6 (10 min)	76 ± 6.05	64.06 ± 6.74	0.003	HS

Table 4. Showing intergroup Comparison of Mean Diastolic Blood Pressure (DBP in mmHg) Changes in Response to Laryngoscopy and Intubation between Saline and Dexmedetomidine Group

(p<0.01)– Statistically highly significant, (p<0.05)– Statistically significant, (p> 0.05)– Statistically Not Significant (NS).

In group I (Saline), the basal mean DBP was 79.2 ± 7.61 mmHg, at preinduction it was 76.63 ± 8.12 mmHg and at induction it was 76.9 ± 6.95 mmHg. The mean diastolic blood pressure at 1 min after intubation was 84 ± 5.75 mmHg, representing an increase of 4.8 mmHg from basal value (6.06%). At 3, 5 and 10 minutes mean DBP values were 81.3 ± 6.28 mmHg, 78.8 ± 6.15 mmHg and 76 ± 6.05 mmHg respectively. The increase in DBP at 1 min after intubation compared to basal value was statistically HS (p=0.000).

In group II (dexmedetomidine), the basal mean DBP was 79.46 ± 9.39 mmHg, at preinduction it was 68.6 ± 9.53 mmHg and at induction it was 66.03 ± 9.08 mmHg representing a decrease in DBP from basal. The mean diastolic blood pressure 1 min after intubation was 69.9 ± 9.18 mmHg representing a decrease of 9.56 mmHg from the basal value (13.6%). At 3, 5, and 10 minutes mean DBP values were 67.43 ± 8.56 mmHg, 66.4 ± 7.95 mmHg, and 64.06 ± 6.74 mmHg respectively. The DBP continued to be below basal value even after 10 minutes of intubation. The decrease in DBP at 1 min after intubation compared to basal value was statistically HS (p=0.009).

Statistical evaluation between the groups showed that the basal mean DBP between Group I and Group II was statistically not significant (p=0.51). The comparison between the 2 groups at pre-induction, induction, 1, 3, 5 and 10 minutes after intubation was statistically highly significant (p=0.000).

Maximum DBP changes were observed at 1 min after intubation in both the Groups, compared to basal there was a 13.6% decrease in Group II in DBP and in Group I there was 6.06% increase in DBP. In Group II there was a steady decrease in DBP from the time of pre-induction until 10th min of intubation which when compared to that of Group I was statistically highly significant.

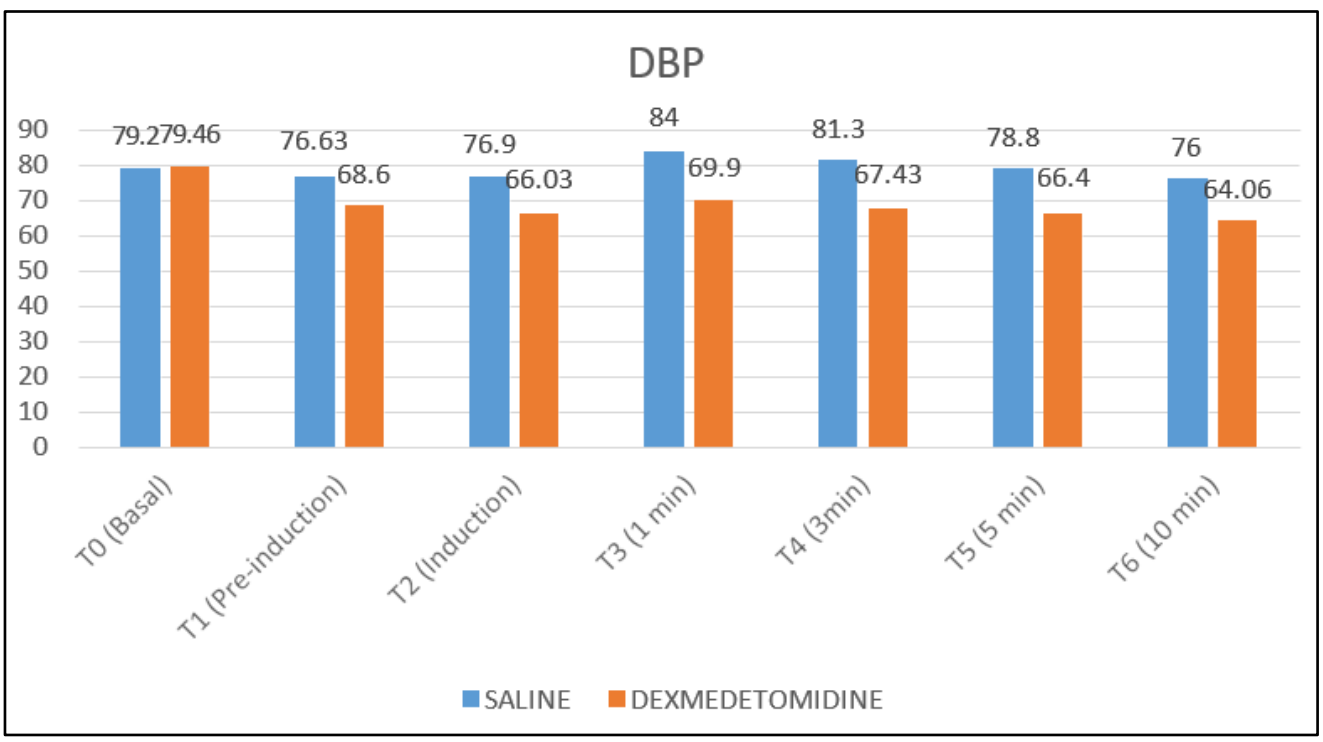

Figure 3. Showing the Intergroup Comparison of Mean DBP (mmHg) Changes in Response to Laryngoscopy and Intubation between Saline Group and Dexmedetomidine Group

Time	Saline	Dexmedetomidine	p- value	Remarks
T0 (Basal)	92.56 ± 7.22	93.96 ± 8.88	0.89	NS
T1 (Pre-induction)	90.26 ± 7.42	83.53 ± 9.64	0.003	HS
T2 (Induction)	90.46 ± 6.77	80.6 ± 9.06	0.001	HS
T3 (1 min)	98.06 ± 5.91	84.26 ± 8.74	0.000	HS
T4 (3 min)	94.96 ± 5.51	82.2 ± 8.19	0.000	HS
T5 (5 min)	92.16 ± 5.45	81.1 ± 7.59	0.000	HS
T6 (10 min)	89.3 ± 5.84	78.7 ± 7.56	0.000	HS

Table 5. Showing Intergroup Comparison of Mean Arterial Pressure (MAP in mmHg) Changes in Response to Laryngoscopy and Intubation between Control Group and Dexmedetomidine Group

(p<0.01)– Statistically highly significant, (p<0.05)– Statistically significant, (p> 0.05)– Statistically Not Significant (NS).

In group I (Saline), the basal mean MAP was 92.56 ± 7.22 mmHg, at preinduction it was 90.26 ± 7.42 mmHg and at induction it was 90.46 ± 6.77 mmHg. The mean arterial pressure 1 min after intubation was 98.06 ± 5.91 mmHg representing an increase of 5.5 mmHg from the basal mean MAP (5.9%). At 3, 5 and 10 minutes mean MAP values were 94.96 ± 5.51 mmHg, 92.16 ± 5.45 mmHg, and 89.3 ± 5.84 mmHg respectively. The increase in MAP at 1 min after intubation compared to basal value was statistically significant (p= 0.00).

In group II (dexmedetomidine), the basal mean MAP was 93.96±8.88 mmHg, at preinduction it was 85.53 ± 9.64 mmHg and at induction it was 80.6 ±9.06 mmHg representing a decrease in MAP. The mean arterial pressure one min after intubation was 84.26 ± 8.74 mmHg

representing a decrease of 9.7 mmHg from the basal value (11.5%). At 3, 5, and 10 minutes mean MAP values were 82.2 ± 8.19 mmHg, 81.1 ± 7.59 mmHg, and 78.7 ± 7.56 mmHg respectively. The MAP continued to be below the basal value even after 10 minutes of intubation. The decrease in MAP at 1 min after intubation compared to basal value was statistically HS (p=0.00).

Statistical evaluation between the groups showed that the basal mean MAP between Group I and Group II was statistically not significant (p=0.89) but the changes observed at pre-induction, induction, 1, 3, 5 and 10 minutes after intubation was statistically highly significant (p=0.003, p=0.001, p=0.000, p=0.000, p=0.000, p=0.000 respectively). Maximum MAP changes were observed at 1 min after intubation in both the Groups, compared to basal there was a 11.5% decrease in MAP in Group II and 5.9% increase in MAP in Group I. In Group II there was a constant decrease in MAP from the time of pre-induction until 10th min of intubation which when compared to that of Group I was statistically highly significant.

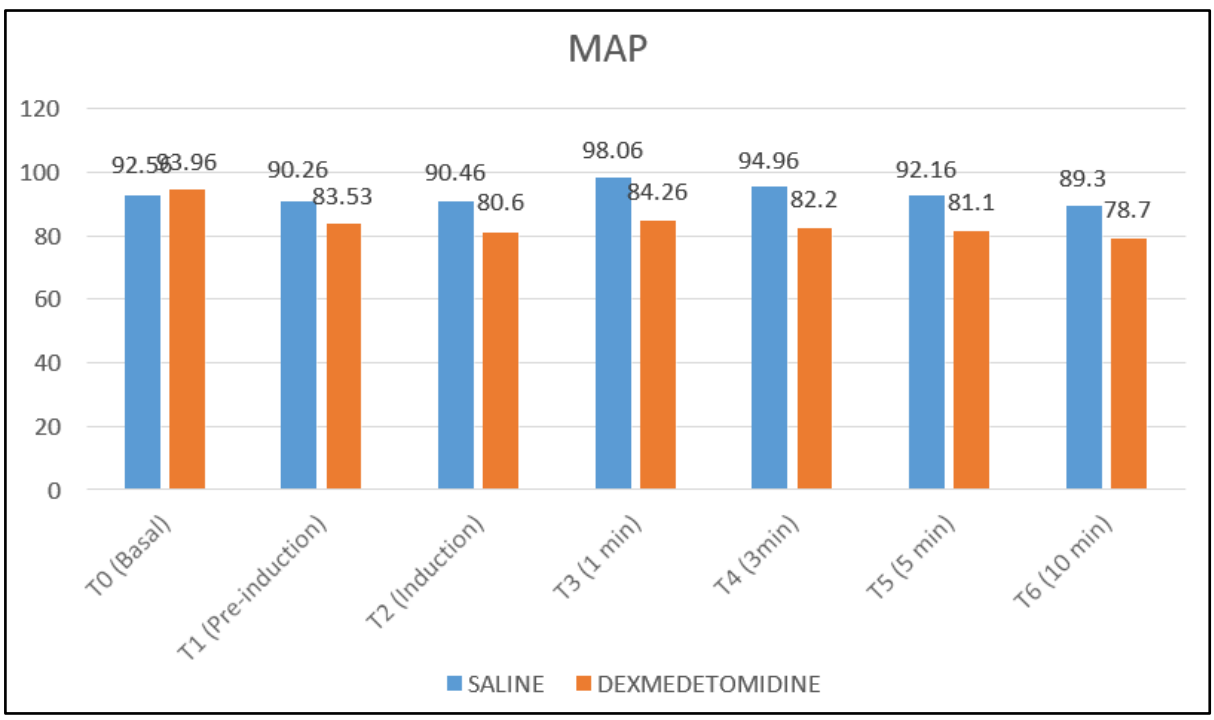

Figure 4. Showing the Intergroup Comparison of MAP (mmHg) Changes in Response to Laryngoscopy and Intubation between Saline Group and Dexmedetomidine Group

ECG- No significant ECG changes were observed in both the groups.

SPO_2- No significant SPO_2 changes were observed in both the Groups throughout the study period.

Group	No. of Patients	Steward Awakening Score	Percentage
Group – I (Saline)	30	12 – 14	100
	0	9 – 11	0
Group – II (Dexmedetomidine)	27	12 – 14	90
	3	9 – 11	10

Table 6. Steward Awakening Score

Side Effects- All patients were followed up for 24 hours post operatively. Side effects attributed to the study drug (Dexmedetomidine) like nausea, vomiting, dryness of mouth, sedation, were not observed. But side effects like bradycardia and hypotension may occur in some patients. For them the rescue drugs like atropine for bradycardia and ephedrine for hypotension may be used.

DISCUSSION

Laryngoscopy and tracheal intubation are considered as the most critical events during administration of general anaesthesia as they provoke transient but marked sympathoadrenal response manifesting as hypertension and tachycardia.[1]

These responses are transitory, variable and may not be significant in otherwise normal individuals. But in patients with cardiovascular compromise like hypertension, Ischemic heart disease, Cerebrovascular disease and in patients with intracranial aneurysms even these transient changes in haemodynamics can result in potentially harmful effects like left ventricular failure, pulmonary oedema, myocardial ischemia, ventricular dysrhythmias and cerebral haemorrhage. This is by far the most important indication for attenuation of haemodynamic response to laryngoscopy and tracheal intubation.[7]

Many methods like use of inhalational anaesthetic agents, lidocaine, opioids.[8] direct acting vasodilators, calcium channel blockers.[9] and β- blockers.[10] have been tried by various authors for blunting haemodynamic responses to laryngoscopy and intubation. But all such maneuvers had their own limitations. For example, with

opioids respiratory depression and chest wall rigidity were potential problems, use of halothane was associated with dysrhythmias, calcium channel blockers produced reflex tachycardia, direct acting vasodilators needed invasive haemodynamic monitoring and lidocaine showed in-consistent results in blunting the haemodynamic responses to laryngoscopy and intubation.

α-2 agonists like clonidine and dexmedetomidine.[11] have been tried for suppressing the response to intubation and have been found to have better effects compared to all the drugs mentioned above, without any of the side effects like respiratory depression or increased incidence of PONV. Clonidine being less potent (α-1: α-2=1:220) compared to dexmedetomidine (α-1: α-2=1:1620) in its agonism to α-2 receptors.[11] Hence dexmedetomidine may be a better drug among α-2 agonists for suppressing the haemodynamic responses to laryngoscopy and intubation.

Study was conducted in S. V. Medical College, Tirupati, Andhra Pradesh for one year period. Both the groups received inj. Glycopyrrolate 0.2 mg, inj Ondansetron 4 mg and inj Rantac 50mg as premedication before induction.

Haemodynamic Changes Heartrate- The basal mean HR in the present study in Group II and Group I were 90.6 bpm and 89.4 bpm respectively. 1 min after intubation in Group II there was 7.6 bpm decrease in mean HR compared to basal, whereas in Group I there was 12.23 bpm increase in mean HR which was statistically significant.

Aho et al.[12] studied the effect of 0.3 and 0.6 μg/kg body weight of dexmedetomidine on perioperative haemodynamics and concluded that dexmedetomidine at a dose of 0.6 μg/kg body weight had effectively blunted haemodynamic response to laryngoscopy and intubation than 0.3 μg/kg body weight dose.

Basar et al, and Kunisawa et al have found a statistically significant ($p<0.05$) obtundation of heart rate response to intubation at 1 min which is similar to our study.

At 3 min. and 5 min. after intubation the mean HR decreased in dexmedetomidine group was 10.5 & 12.8 bpm whereas in control group mean HR increased by 9.6 &5.8 bpm respectively.

At 10 mins, there was decrease in mean HR by 14.5 bpm in dexmedetomidine group compared to basal value whereas in control group group the HR increased by 1.07 bpm which was statistically highly significant ($p=0.000$).

The decrease in HR is because of the inhibition of the central sympathetic outflow overriding the direct stimulant effects and stimulation of presynaptic alpha-2 adrenoceptors, leading to a decrease in norepinephrine release.

Systolic Blood Pressure (SBP)- In the present study, the basal mean SBP in Group II and Group I were 123.46 mmHg and 122.43 mmHg respectively. 1 min after intubation in Group II there was 11.5 mmHg decrease in SBP compared to basal whereas in Group I there was 5.53 mmHg increase in SBP which was statistically significant. At 3, 5 and 10 min after intubation in Group II the decrease in SBP when compared to basal were 12.4 mmHg, 14.23 mmHg and 16.86 mmHg respectively. In group I there was increase in SBP at 1 and 3 min but decrease than basal at 5 min after intubation.

In our study SBP decreased from pre-induction and continued to remain below basal value until 10 min after intubation which was statistically significant. Many authors have observed a transient increase of the blood pressure and a reflex decrease in heart rate, especially in young healthy patients following dexmedetomidine bolus doses. The initial reaction can be explained by the peripheral alpha 2B adrenoceptors stimulation of vascular smooth muscles and can be attenuated by a slow infusion. We have not observed this transient increase in blood pressure probably because of slow infusion over 15 min and adequate preloading.

Diastolic Blood Pressure (DBP)- The basal mean DBP in the present study in Group II and Group I were 79.46 mmHg and 79.2 mmHg respectively. There was a steady decrease in DBP from pre-induction in Group II. At 1 min after intubation the DBP was 9.56 mmHg less compared to that of the basal value whereas in Group I there was 4.8 mmHg increase in DBP which was statistically significant. At 3 min, 5 min and 10 min after intubation the fall in DBP in Group II compared to basal value was 12.06 mmHg, 13.06 mmHg and 15.4 mmHg respectively.

Mean Arterial Pressure (MAP)- In the present study basal mean MAP in Group II and Group I were 93.96 mmHg and 92.56 mmHg respectively. There was a steady decrease in MAP from preinduction in Group II. At 1 min after intubation the MAP was 9.76 mmHg less compared to basal value whereas in Group I there was 5.5 mmHg increase in MAP which was statistically significant. At 3, 5 and 10 min the fall in MAP in Group II was 11.76 mmHg, 12.86 mmHg and 15.26 mmHg respectively.

In the present study, the decrease observed in MAP is because of the inhibition of central sympathetic outflow overriding the direct stimulant effects.

Recovery Score- After the patients were shifted to PACU, steward awakening score was monitored once in 15 min for one hour. 27 patients (90%) in Group II had a steward awakening score between 12-14 indicating excellent recovery and 3 patients (10%) had scores between 9-11 indicating satisfactory recovery and were fit for discharge from the post anaesthesia care unit. Scores <9, indicating poor recovery was not observed in any of the patients. Yildiz et al applied steward awakening score at 5 and 10 min after extubation and observed that at 10 min all patients had excellent recovery.

Side Effects- All the patients in the study were followed up for a period of 24 hours and side effects were observed.

CONCLUSION

From the present study dexmedetomidine in the dose of 1 µg/kg as IV infusion, given 15 minutes before induction can be used safely to attenuate the pressor response to laryngoscopy and intubation without significant side effects.

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