ATTENUATION OF HAEMODYNAMIC RESPONSE TO LARYNGOSCOPY AND TRACHEAL INTUBATION IN ADULT PATIENTS WITH A SINGLE INTRAVENOUS BOLUS DOSE OF 0.6 µG/KG BODY WEIGHT OF DEXMEDETOMIDINE- A PROSPECTIVE, RANDOMISED, DOUBLE-BLIND CONTROLLED CLINICAL STUDY

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ABSTRACT

BACKGROUND

Laryngoscopy and endotracheal intubation is often associated with hypertension and tachycardia, which can produce deleterious effects in cardiovascular patients. Dexmedetomidine has been particularly effective in blunting the haemodynamic response to laryngoscopy and tracheal intubation in addition to reducing the anaesthetic drug requirements. The present study was undertaken to study the efficacy of 0.6 µg/kg body weight dexmedetomidine IV given 10 minutes before induction in obtunding the haemodynamic responses to laryngoscopy and tracheal intubation compared to control group.

MATERIALS AND METHODS

One hundred normotensive patients aged between 18-55 years belonging to ASA class I and II and Mallampati grade I and II were included and assigned randomly into two groups. Group C (n=50) received 10 mL normal saline intravenously over 10 minutes, 10 minutes before induction and Group D (n=50) received diluted dexmedetomidine intravenously over 10 minutes, 10 minutes before induction. Anaesthesia was induced with Inj. Thiopentone as 2.5% solution till loss of eyelash reflex occurred and dose of thiopentone required was noted followed by Inj. Succinylcholine. HR, SBP, DBP and MAP were recorded at various time intervals.

RESULTS

It was noted that in group C (control), following laryngoscopy and intubation, the rise in Heart Rate (HR), Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP) and Mean Arterial Pressure (MAP) were found to be 36.24 bpm, 30.02 mmHg, 22.34 mmHg and 26.42 mmHg respectively, one minute after intubation. In group D (dexmedetomidine), rise of HR, SBP, DBP and MAP were decreased by 2.86 bpm, 15.86 mmHg, 9.54 mmHg and 1.98 mmHg, respectively, which was statistically highly significant (p=0.000). Dexmedetomidine reduced the requirement of thiopentone and vecuronium bromide and produced arousable sedation after extubation.

CONCLUSION

Dexmedetomidine in the dose of 0.6 µg/kg body weight given intravenously 10 minutes before induction was seen to effectively attenuate the haemodynamic response to laryngoscopy and tracheal intubation without any side effects.

KEYWORDS

Laryngoscopy and Tracheal Intubation, Haemodynamic Response, IV Dexmedetomidine.

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BACKGROUND

Laryngoscopy and tracheal intubation in adults are commonly accompanied by increase in arterial blood pressure and heart rate, which is dependent on various factors such as depth of anaesthesia, measures taken prior to airway manipulation, the anaesthetic agent used, the duration of laryngoscopy and intubation.[1] The transitory hypertension and tachycardia is the sympathetic response, which maybe the result of increase in catecholamine activity and hazardous to those with hypertension, myocardial insufficiency or cerebrovascular diseases, which predispose to development of pulmonary oedema, myocardial insufficiency and cerebrovascular accident.[2]

Exaggerated pressor response hypertensive patients may result in intraoperative myocardial infarction, acute left

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ventricular failure, dysrhythmias and intracranial bleed in individuals with end-organ decompensation.[3,4]

To suppress the circulatory responses evolved by endotracheal intubation, additional pharmacological measures like use of volatile anaesthetics, topical and intravenous lidocaine, vasodilators, sodium nitroprusside, β-blockers, nitroglycerine, opioids, calcium channel blockers have been tried by various authors.[5-9] None of the drugs mentioned above have been found to be effective to attenuate the sympathetic response to intubation. Hence, there is a need of finding out the drugs, which can meet the requirements. Alpha-2 agonists have been used for attenuating the sympathetic response and among α-2 agonists both clonidine and dexmedetomidine appear to fulfil the above criteria. Both 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. Various studies have also found that dexmedetomidine can decrease the haemodynamic response to laryngoscopy and intubation.[10,11]

Since, it has been recently introduced in India and not many studies have been done in India regarding its usefulness in suppressing intubation response, there is a need to study its effectiveness. The advantages of intravenous dexmedetomidine as premedicant in anaesthesia setting include sedation, analgesia, anxiolysis and improved haemodynamic stability. Because of these beneficial properties, it has been found that the Minimum Alveolar Concentration (MAC) of volatile anaesthetics also decreases significantly up to 90% and hence decreases the requirement of anaesthetics.[12,13]

The present study is aimed at attenuation of haemodynamic response to laryngoscopy and intubation in adult patients posted for various surgeries under general anaesthesia with single intravenous bolus dose of 0.6 µg/kg body weight dexmedetomidine given 10 minutes prior to induction.

OBJECTIVES

Primary Objective- To evaluate the efficacy of intravenous dexmedetomidine in the dose of 0.6 µg/kg body weight in attenuating the haemodynamic responses to laryngoscopy and endotracheal intubation.

Secondary Objectives

1. To study the effects of dexmedetomidine on the dose requirement of thiopentone for induction of anaesthesia.
2. To study the effects of dexmedetomidine on the dose requirement of vecuronium bromide for muscle relaxation.
3. To study any adverse effects associated with dexmedetomidine administration such as increased sedation, prolonged recovery, hypotension and bradycardia.

MATERIALS AND METHODS

The present study, a prospective, randomised, double-blind controlled clinical study was undertaken at ACSR Medical College and Hospital, Nellore, Andhra Pradesh, for a period of one year from March 2015 to February 2016, after obtaining ethical committee clearance as well as informed consent from all the hundred patients.

Inclusion Criteria for the Study

- Adult patients aged between 18 and 55 years of both sex.
- Patients belonging to ASA class I and II.
- Mallampati grade I and II.
- Elective surgeries under general endotracheal anaesthesia.

Exclusion Criteria for the Study

- Patients with cardiac, coronary, renal, hepatic, cerebral diseases and peripheral vascular diseases.
- Patients with hypertension.
- Patients with heart rate less than 60 bpm, systolic blood pressure less than 100 mm of Hg.
- Presence of first, second or third-degree heart block.
- Patients with difficult airway and obese patients (BMI >30).
- Patients with endocrinal diseases like hyperthyroidism, hypothyroidism and diabetes mellitus.

The study population was randomly divided into two groups (C and D) with 50 patients in each.

Group C - Control group (n=50) received 10 mL of normal saline intravenously over 10 minutes, 10 minutes prior to induction using syringe pump.

Group D - dexmedetomidine (n=50) received diluted injection dexmedetomidine 0.6 µg/kg body weight (Dexem, Themis Medicare Limited, 200 µg in 2 mL ampoule) intravenously over 10 minutes, 10 minutes prior to induction using syringe pump.

Preanaesthetic evaluation and premedication with tablet alprazolam 0.5 mg and tablet ranitidine 150 mg orally at bedtime were done and were kept nil orally 10 p.m. onwards.

In the operating room, the patients were connected to Siemens SC-7000, multi-parameter monitor and baseline readings of heart rate, SBP, DBP, MAP and ETCO2 were recorded with continuous ECG monitoring, oxygen saturation, cardiac rate and rhythm.

After recording the baseline reading, patients in group D received diluted dexmedetomidine 0.6 µg/kg body weight, which was prepared by the senior anaesthesiologist who was not involved with the study and observer as well as patient was blinded for the study, intravenously over 10 minutes, 10 minutes before induction by syringe pump and patients in group C received normal saline 10 mL intravenously over 10 minutes, 10 minutes before induction using syringe pump.

All the patients were premedicated with Inj. Midazolam, Inj. Pentazocine after test drug administration and preoxygenated for 3 minutes via a facemask with Bain's circuit. Before intubation, all patients received IV lignocaine

and anaesthesia was induced with thiopentone as a 2.5% solution till loss of eyelash reflex occurred and dose of thiopentone recorded. Endotracheal intubation was facilitated with 1.5 mg/kg IV succinylcholine one minute prior to laryngoscopy and intubation and endotracheal tube was fixed. If time for laryngoscopy and intubation exceeds 15 seconds, such patients were excluded from the study.

Anaesthesia was maintained using 66% nitrous oxide and 33% of oxygen with 0.5% halothane and neuromuscular blockade was maintained with vecuronium 0.05 mg/kg body weight total dose of vecuronium required for the surgery recorded and patients were reversed with neostigmine 0.05 mg/kg body weight and atropine 0.02 mg/kg body weight.

MONITORING

Cardiovascular parameters, Heart Rate (HR), Systolic blood Pressure (SBP), Diastolic Blood Pressure (DBP) and Mean Arterial Pressure (MAP) were recorded in all patients. Basal readings before giving study drug and at intervals of 2 minutes, 5 minutes and 8 minutes after study drug and before and after induction followed by 1, 3, 5, 10 minutes after laryngoscopy and intubation. The side effects of the study drug like hypotension, bradycardia and sedation were noted.[14,15,16] Sedation scoring noted as per Ramsay sedation scale.[17]

Statistical Methods Employed

SPSS for windows (version 17.0) was employed for data analysis $p<0.05$ was considered as significant and $p<0.01$ was considered as highly significant.

RESULTS

The minimum age in groups C and D were 20 and 18 years and maximum was 55 years. Mean age in group C and D were 36.8 ± 9.7 and 36.42 ± 9.36, respectively. Males accounted for 49 and females 56 in the study. The mean body weight (kgs) in Group C was 56.12 ± 6.15, and in Group D, it was 55.34 ± 7.56.

In group C, the basal mean HR was 87.36 ± 9.96 bpm. The mean HR after study drug administration at 2nd, 5th and 8th minute and before induction were 88.18 ± 9.77, 86.62 ± 9.08, 84.82 ± 9.92 and 84.52 ± 10.81, respectively, and a statistically significant increase (P=0.000) was noted after induction in HR compared to basal value. The mean HR at one minute after intubation was 123.6 ± 10.46 representing a rise of 36.24 bpm from the basal HR, which was statistically highly significant (p=0.000). By 3, 5 and 10 minutes of intubation, the mean HR were 116.4 ± 9.16, 109.62 ± 9.17 and 98.30 ± 9.82, respectively.

In group D (dexmedetomidine), the basal mean HR was 87.36 ± 13.58. After drug administration, the mean HR at 2, 5 and 8 minutes and before induction were 81.28 ± 14.25, 78.64 ± 13.47, 75.26 ± 12.62 and 74.08 ± 11.09, respectively, which was statistically highly significant (p=0.000). The mean HR after induction was 81.26 ± 12.08, which is statistically highly significant (p=0.000), but after intubation at 1 minute was 84.50 ± 11.41, which was not significant compared to basal value (p=0.079). By 3, 5 and 10 minutes after intubation, the mean HR remained low compared to basal value (p=0.000) and this is statistically highly significant (p=0.000).

The basal heart rate was comparable in both groups (p=1.000) and showed a significant fall in HR in group D at 2, 5 and 8 minutes of drug administration and before and after induction. The mean HR increase observed at 1, 3, 5 and 10 minutes after intubation in group C was statistically highly significant compared to mean HR in group D (p=0.000) (Table 2).

In group C (control), the basal mean SBP was 128.00 ± 6.09 mmHg. After drug administration at 2, 5 and 8 minutes and before induction, mean SBP were 127.72 ± 6.65, 127.76 ± 5.62, 127.18 ± 5.84 and 127.90 ± 6.86, respectively. There was a significant fall in SBP after induction (p=0.000) compared to basal value. The mean SBP one minute after intubation was 158.02 ± 4.41 representing a rise of 30.02 mmHg and by 3, 5 and 10 minutes after intubation, the mean values were 149.02 ± 8.14, 138.70 ± 8.26 and 128.78 ± 6.35, respectively, which was statistically highly significant (p=0.000).

In group D (dexmedetomidine), the basal value of mean SBP was 127.38 ± 11.30. Statistically significant change in SBP was noted by 5 and 8 minutes of drug administration and before and after induction. The mean SBP values were 116.92 ± 11.99, 111.32 ± 11.80, 110.02 ± 10.03 and 103.68 ± 11.15, respectively, representing a significant fall in SBP (p=0.000). One minute after intubation, the mean SBP was 111.52 ± 9.95 representing an increase in SBP and at 1, 3, 5 and 10 minutes after intubation are statistically highly significant (p=0.000).

The mean SBP were comparable in both groups (p=0.734). After 2 minutes of drug administration, the change in SBP was not significant (0.456). The mean SBP values at 5 and 8 minutes of drug administration, before and after induction were significantly low (p=0.000) compared to group C. The increase in SBP in group C at 1, 3, 5 and 10 minutes after intubation was statistically highly significant (p=0.000) compared to group D (Table 3).

In group C, the basal mean DBP was 76.64 ± 6.08. After drug administration at 2, 5 and 8 minutes and before induction, the mean DBP was statistically not significant (p>0.05). Within one minute after intubation, the mean DBP was 98.98 ± 3.72 representing a rise of 22.34 mmHg, 3, 5 and 10 minutes after intubation, the DBP values were 92.06 ± 7.39, 86.18 ± 8.04 and 79.76 ± 8.12, respectively, and the increase in mean DBP at 1, 3, 5 and 10 minutes was statistically highly significant (p=0.000) compared to basal value. In group D (dexmedetomidine), the basal mean DBP was 78.32 ± 7.53. The mean DBP at 2 minutes of drug administration was statistically not significant and by 5 and 8 minutes of drug administration and before and after induction, the mean DBP values were 69.14 ± 6.81, 68.16 ± 7.00, 64.32 ± 9.85 and 60.32 ± 10.63, respectively, representing a significant fall in DBP (p=0.000). After intubation, the mean DBP values at 1, 3, 5 and 10 minutes were 68.78 ± 9.41, 62.20 ± 10.70, 68.68 ± 9.48 and 60.60 ± 8.93, respectively, which was statistically highly significant (p=0.000). The DBP continued to be lower than the basal

value even 10 minutes after intubation. The mean basal DBP are comparable in both groups (p=0.223). The mean DBP values at 5 and 8 minutes of drug administration, before and after induction were significantly low (p=0.000) in group D compared to group C. The increase in DBP in group C at 1, 3, 5 and 10 minutes after intubation was statistically highly significant (p=0.000) compared to group D (Table 4).

In group C (control), the basal mean MAP was 91.84 ± 3.44. After intubation, the mean MAP was 84.80 ± 4.85 representing a significant fall in MAP (P=0.000) compared to basal value. The mean MAP one minute after intubation was 118.26 ± 3.42 representing a rise of 26.42 mmHg, which was statistically significant (p=0.000). The mean MAP remained high at 3, 5 and 10 minutes after intubation, which was statistically highly significant (p=0.000). In group D, the basal mean MAP was 90.84 ± 3.44. The mean MAP after drug administration at second mins. was 91.88 ± 3.40 (p=0.942) and at 5 and 8 minutes of drug administration, before and after induction were 86.24 ± 4.47, 81.70 ± 6.02, 83.32 ± 7.00 and 81.02 ± 8.11, respectively, which was statistically highly significant (p=0.000). After intubation, the mean MAP value at 1, 3, 5 and 10 minutes continued to be at lower levels (p=0.000) compared to basal value. The mean basal MAP are comparable in both groups (p=1.000) with significant difference in MAP values at 5th minute, 8th minute after drug administration and before and after induction (p=0.000). The increase in MAP in group C was statistically highly significant at 1 minute and 3, 5 and 10 minutes after intubation (p=0.000) compared to group D (Table 6).

The mean dose of thiopentone sodium required for loss of eyelash reflex in group C and group D were 278 ± 34.49 and 170.5 ± 30.17, respectively, showing reduction in dose required for induction and was highly significant (p=0.000). The mean dose of vecuronium bromide required for muscle relaxation in group C and group D were 4.70 ± 1.36 and 3.74 ± 1.22, respectively, showing a statistical significant reduction in dose of vecuronium bromide for muscle relaxation (p=0.000). In group C, sedation score was 2.62 ± 0.49, and in group D, the score was 2.52 ± 0.43 and was not statistically significant (p=0.087) (Figure 1 and 2). In group C, none of the patients had side effects like bradycardia and hypotension. In group D, 4 patients had bradycardia, 3 had hypotension and 1 patient had both bradycardia and hypotension.

Variable	Group C (No.) (%)	Group D (No.) (%)	P valve
Age (Yrs.)			
18-28	12 (24)	13 (26)	0.835
29-38	13 (26)	12 (24)	
39-48	15 (30)	20 (40)	
49-55	10 (20)	5 (10)	
Mean age ± SD	36.8 ± 9.7	36.42 ± 9.36	
Gender			
Male	27 (54%)	22 (44%)	
Female	23 (46%)	28 (56%)	
Body Weight (KGS)			
40-44	0	3 (6%)	0.189
45-49	10 (20%)	8 (4%)	
50-54	10 (20%)	14 (28%)	
55-59	9 (18%)	13 (26%)	
60-64	18 (36%)	6 (12%)	
65-69	2 (4%)	4 (8%)	
70+	1 (2%)	2 (4%)	
Mean body weight in kg ± SD	56.12 ± 6.15	55.34 ± 7.56	

Table 1. Demographic Variables

	Group C	Group D	p-value
Basal	87.36 ± 9.96	87.36 ± 13.58	1.000 (NS)
AD - 2nd min.	88.18 ± 9.77	81.28 ± 14.25	0.006 (HS)
AD - 5th min.	86.62 ± 9.08	78.64 ± 13.47	0.001 (HS)
AD - 8th min.	84.82 ± 9.92	75.26 ± 12.62	0.000 (HS)
Before Induction (BI)	85.30 ± 9.81	74.08 ± 11.09	0.000 (HS)
After induction	96.24 ± 9.76	81.26 ± 12.08	0.000 (HS)
AI - 1st min.	123.6 ± 10.46	84.50 ± 11.41	0.000 (HS)
AI - 3rd min.	116.4 ± 9.16	82.38 ± 11.28	0.000 (HS)
AI - 5th min.	109.62 ± 9.17	79.88 ± 11.93	0.000 (HS)
AI - 10th min.	98.30 ± 9.82	76.90 ± 10.77	0.000 (HS)

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

(p<0.01)- Highly Significant (HS); (p<0.05)- Significant (S); (p>0.05)- Not Significant (NS); AD- After drug administration; AI- After intubation.

Time	Group C	Group D	p-value
Basal	128.00 ± 6.09	127.38 ± 11.30	0.734 (NS)
AD - 2nd min.	127.72 ± 6.65	129.26 ± 12.95	0.456 (NS)
AD - 5th min.	127.76 ± 5.62	116.92 ± 11.99	0.000 (HS)
AD - 8th min.	127.18 ± 5.84	111.32 ± 11.80	0.000 (HS)
Before Induction (BI)	127.90 ± 6.86	110.02 ± 10.03	0.000 (HS)
After induction	121.30 ± 5.11	103.68 ± 11.15	0.000 (HS)
AI - 1st min.	158.02 ± 4.41	111.52 ± 9.95	0.000 (HS)
AI - 3rd min.	149.02 ± 8.14	105.04 ± 11.13	0.000 (HS)
AI - 5th min.	138.70 ± 8.26	103.04 ± 12.38	0.000 (HS)
AI - 10th min.	128.78 ± 6.35	101.30 ± 11.12	0.000 (HS)

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

(p<0.01)- Highly Significant (HS); (p<0.05)- Significant (S); (p>0.05)- Not Significant (NS); AD- After drug administration; AI-After intubation.

Time	Group C	Group D	p-value
Basal	76.64 ± 6.08	78.32 ± 7.53	0.223 (NS)
AD - 2nd min.	76.78 ± 5.99	78.88 ± 6.65	0.674 (NS)
AD – 5th min.	75.32 ± 7.64	69.14 ± 6.81	0.015 (S)
AD – 8th min.	76.78 ± 5.99	68.16 ± 7.00	0.000 (HS)
Before Induction (BI)	76.58 ± 5.45	64.32 ± 9.85	0.009 (HS)

After induction	78.84 ± 6.64	60.32 ± 10.63	0.000 (HS)
AI - 1st min.	98.98 ± 3.72	68.78 ± 9.41	0.000 (HS)
AI - 3rd min.	92.06 ± 7.39	62.20 ± 10.70	0.000 (HS)
AI - 5th min.	86.8 ± 8.04	68.68 ± 9.48	0.000 (HS)
AI - 10th min.	79.76 ± 8.12	60.60 ± 8.93	0.000 (HS)

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

(p<0.01) - Highly Significant (HS); (p<0.05) - Significant (S); (p>0.05)- Not Significant (NS); AD- After drug administration; AI- After intubation.

Time	Group C	Group D	p-value
Basal	91.84 ± 3.44	90.84 ± 3.44	1.000 (NS)
AD - 2nd min.	92.08 ± 4.85	91.88 ± 3.40	0.812 (NS)
AD - 5th min.	91.54 ± 3.47	86.24 ± 4.47	0.000 (HS)
AD - 8th min.	91.48 ± 4.04	81.70 ± 6.02	0.000 (HS)
Before Induction (BI)	92.48 ± 3.29	83.32 ± 7.00	0.000 (HS)
After induction	84.80 ± 4.85	81.02 ± 8.11	0.000 (HS)
AI - 1st min.	118.26 ± 3.42	88.86 ± 7.65	0.000 (HS)
AI - 3rd min.	110.18 ± 6.94	80.44 ± 7.84	0.000 (HS)
AI - 5th min.	102.84 ± 7.24	79.34 ± 8.02	0.000 (HS)
AI - 10th min.	95.96 ± 6.61	80.54 ± 8.85	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) - Highly Significant (HS); (p<0.05) - Significant (S); (p>0.05)- Not Significant (NS); AD- After drug administration; AI- After intubation.

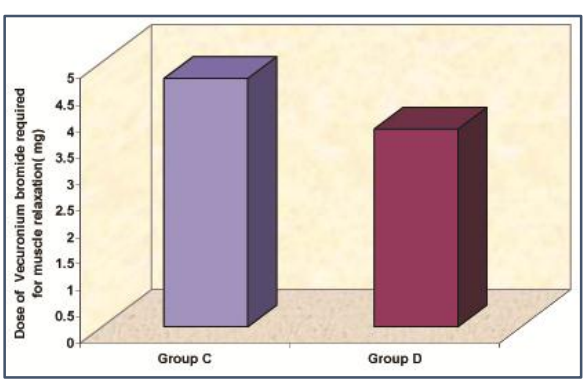

Figure 1. Showing the Total Dose of Vecuronium Bromide Required for Muscle Relaxation in Control and Dexmedetomidine Group

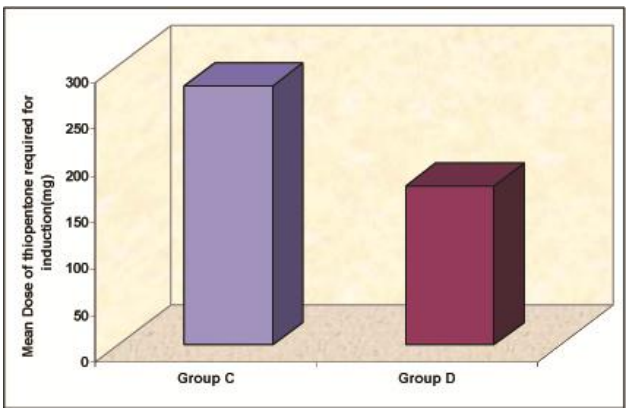

Figure 2. Showing the Dose of Thiopentone Required for Induction in Control and Dexmedetomidine Group

DISCUSSION

Endotracheal intubation and laryngoscopy are considered as critical events in anaesthetic practice as they caused transient sympathetoadrenal response manifested as HTN and tachycardia. To blunt these effects, various other drugs; e.g.- lidocaine, calcium channel blockers, etc. have been used and found to have their own limitations. Clonidine and dexmedetomidine have been introduced recently and found with better effects in suppression of haemodynamic response. 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. Hence, the effects of dexmedetomidine for suppression of haemodynamic response to laryngoscopy and intubation was taken up as our study topic and to assess the haemodynamic response during endotracheal intubation and laryngoscopy without any pretreatment. Two study groups with 50 each one as control C (receives IV saline) and other as study group D (receives dexmedetomidine 0.6 µg/kg body weight in 10 mL normal saline IV). Most of studies reported the effects of dexmedetomidine at various drug concentrations. Kallio et al[16] showed that the maximum inhibition of sympathetic nervous system activity occurred at 50 and 75 µg of dexmedetomidine dose and Kunisawa et al[18] used 1 µg/kg body weight of dexmedetomidine with fentanyl with same activity. Ferdi et al[19] used 1 µg/kg body weight, Esra et al[20] used 0.5 and 1 µg/kg body weight and Aho et al[12] studied the effect of 0.3 and 0.6 µg/kg body weight of dexmedetomidine on perioperative haemodynamics. Scheinin et al,[11] Jaakola et al[14] and Mowafi et al[21] found dexmedetomidine effective at the dose of 0.6 µg/kg body weight in attenuating stress response to intubation, hence 0.6 µg/kg body weight dose was chosen for our study. The administration of 10 mL in 10 minutes, 10 minutes prior induction is on like study of Basar et al[22] and Kunisawa et al.[18]

Changes in HR- Basar et al,[22] Kunisawa et al,[18] Ferdi et al[19] and Keniya et al[23] found that dexmedetomidine has decreased the HR at various intervals of 2, 5, 8 and 10 minutes, which coincides with findings of our study.

In comparison of mean HR changes between control and dexmedetomidine group, our finding was an increase of HR by 9 bpm in control group, whereas there is a decrease of 6

bpm in D group, which is on par with findings of Kunisawa et al.[18] In the present study, following laryngoscopy and intubation at 1 minute, the mean HR increased by 36.24 bpm in the control group, whereas in dexmedetomidine group, the mean HR decreased by 2.86 bpm, which is similar to findings of Aho et al,[12] Basar et al[22] and Mowafi et al.[21] At 5th minute in dexmedetomidine, there was further decrease in HR by 7.48 bpm, which was like findings of Scheinin et al[11] and Jaakola et al.[14] At 10th minute in dexmedetomidine group, the HR remained low by 10.46 bpm on par with findings of Basar et al.[22]

Changes in SBP- Aho et al,[12] Ralph Getler et al and Keniya et al[23] found a continuous gradual reduction of SBP from 5th minute onwards after dexmedetomidine administration, which is also observed in our study. After induction, a reduction of 24 mm of Hg was observed in dexmedetomidine group, which was also seen in study of Kunisawa et al.[18] After laryngoscopy and intubation in our study after dexmedetomidine administration at 10th minute, the SBP did not reach the basal value and it was 26 mmHg lower than the basal value, which coincides with findings of Jaakola et al and[14] Aho et al.[12]

Changes in DBP- After dexmedetomidine administration at second minute, a marginal increase of DBP followed by gradual fall till induction is noticed. After induction, a fall of 18 mm of Hg is noticed in Group D, which coincides with findings of Jaakola et al.[14] After laryngoscopy and intubation in our study after dexmedetomidine administration, DBP fall by 9 mmHg in 1st minute and 17 mmHg by 10th minute, which is on par with findings of Jaakola et al,[14] Kunisawa et al[18] and Ferdi et al.[19]

Changes in MAP- After dexmedetomidine administration in group D, continuous fall in MAP noted after 5th minute, which concurs with the findings of Basar et al[22] and Mowafi et al.[21] After induction, a fall of 9 mmHg is noticed in Group D. Findings of our study show in control group, there is an increase in MAP by 24 mmHg compared with 7 mmHg of increase in dexmedetomidine group. At 1st minute after intubation, the increase in MAP in control group 26 mmHg, whereas in dexmedetomidine group, there is a fall in MAP by 2 mmHg, which is statistically highly significant and concurs with the findings of Basar et al [22] and Mowafi et al.[21]

In our study, a reduction of 38% in dose of thiopentone was observed in Group D when compared with group C, which concurs with findings of Aanta et al.[24]

CONCLUSION

To conclude, dexmedetomidine at a dose of 0.6 µ/kg body weight diluted in 10 mL saline given 10 minutes before induction significantly obtunded the haemodynamic responses to laryngoscopy and tracheal intubation. It also decreased requirement of induction dose of thiopentone and the requirement of the total dose of vecuronium bromide for muscle relaxation without significant side effects.

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