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COMPARISON OF DEXMEDETOMIDINE AND CLONIDINE IN ATTENUATION OF HEMODYNAMIC RESPONSE IN LARYNGOSCOPY AND INTUBATION

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ABSTRACT **Introduction:** Tracheal intubation and laryngoscopy are examples of unpleasant stimuli that cause a brief but noticeable sympathetic reaction. By preventing the production of noradrenaline, alpha-2 adrenoceptor agonists reduce sympathoadrenal responses. **Aims:** The purpose of this research is to assess and contrast the impact of intravenous clonidine and dexmedetomidine on the cardiovascular response that follows endotracheal intubation and laryngoscopy. **Materials and Method:** The present study was a Comparative Study. This Study was conducted over a period of 1 year at Department of Anaesthesia, ESI Hospital Sealdah and Department of Critical Care Medicine, AMRI Dhakuria, Kolkata. Total 150 patients were included in this study. **Result:** In Group clonidine, the mean Systolic Blood Pressure (SBP) (mmHg) of patients was 117.9 ± 5.9. In Group dexmedetomidine, the mean SBP (mmHg) of patients was 120.1 ± 13.5.In Group Control, the mean SBP (mmHg) of patients was 123.1 ± 4.8. Distribution of mean SBP (mmHg) with the Demographic data Group was not statistically significant (p=0.17). **Conclusion:** In both the study groups attenuating response to haemodynamic changes were observed. When compared amongst each other, both Dexmedetomidine and Clonidine groups came out to be statistically similar in attenuating haemodynamic responses.

KEYWORDS : Dexmedetomidine, Clonidine, Attenuation

INTRODUCTION

Excessive stimulation such as tracheal intubation and laryngoscopy cause a brief but noticeable sympathetic reaction that raises blood pressure and heart rate (BP). These modifications peak five to ten minutes following intubation. These hemodynamic alterations in individuals with cardiovascular disease can result in potentially fatal consequences such myocardial ischemia, abrupt heart failure, and cerebrovascular accidents.[1]

Topical lignocaine sprays, deeper planes of anaesthesia provided by opioids or inhaled/intravenous (IV) medications, calcium channel blockers, and vasodilators like nitroglycerine and sodium nitroprusside are examples of conventional therapeutic techniques. While there are a number of approaches, research on how to lessen the pressor response to laryngoscopy and intubation is still ongoing.[2]

The benefits of alpha-2 (α2)-adrenoceptor agonists, which stabilise hemodynamics and spare anaesthesia, may make them a viable alternative to the supplementary anaesthetic drugs now in use.[3]

It has been demonstrated lately that premedication with the α2-adrenergic agonist clonidine can lessen the need for anaesthesia and narcotics by reducing the stress reaction to the surgical stimuli.[4] Furthermore, clonidine stabilises blood pressure by heightening the sensitivity of the heart baroreceptor reaction to an increase in systolic blood pressure (SBP). Its lengthy half-life and low selectivity to α2 adrenoceptors have, however, restricted its usage. A more recent imidazole derivative that selectively binds to α2-adrenergic receptors is called dexmedetomidine. Reduced systemic noradrenaline release results from α2-agonists' hyperpolarization of noradrenergic neurons and inhibition of neuronal activity in the locus coeruleus. This causes hemodynamic stability and sympathoadrenal responses to be attenuated during tracheal intubation and laryngoscopy.[5]

Research has been conducted to assess how clonidine and dexmedetomidine affect the hemodynamic response that occurs during tracheal intubation and laryngoscopy. Although α2-adrenergic agonists have been demonstrated to be helpful in reducing cardiac problems, a recent meta-analysis found no effect in terms of mortality in individuals with heart conditions.[6]

MATERIALS AND METHOD

Study Design: Comparative Study.
Study Place: Department of Anaesthesia, ESI Sealdah
Department of Critical Care Medicine, AMRI Dhakuria, Kolkata.
Study Duration: 1 year
Sample Size: 150

- Group-I-50 patients with Group clonidine
- Group-II- 50 patients with Group dexmedetomidine
- Group-I-50 patients with Control

Inclusion Criteria

- Patients aged 18 to 65 years old.
- Patients scheduled for elective surgery under general anaesthesia requiring endotracheal intubation.
- American Society of Anaesthesiologists (ASA) physical status I or II.
- Patients with Mallampati Class I or II.
- Patients able to provide informed consent or with consent provided by their legal guardian.
- Patients willing to comply with study procedures and follow-up.

Exclusion Criteria

- Patients with known hypersensitivity or contraindications to dexmedetomidine, clonidine, or any components of the study drugs.
- Patients with severe cardiovascular diseases such as unstable angina, congestive heart failure (New York Heart Association [NYHA] Class III or IV), severe valvular heart disease, or significant arrhythmias.
- Patients with severe hepatic impairment (Child-Pugh Class C) or severe renal impairment (creatinine clearance <30 mL/min).
- Patients with a history of significant respiratory compromise or chronic obstructive pulmonary disease (COPD).
- Patients with a history of drug or alcohol abuse within the past year.
- Patients with a history of psychiatric illness or cognitive impairment that may affect their ability to understand and comply with study procedures.
- Pregnant or breastfeeding women.
- Patients participating in another clinical trial within the past 30 days.
- Patients with anticipated difficult airway management (e.g., restricted mouth opening, neck mobility issues).
- Patients on medications known to interact with dexmedetomidine or clonidine (e.g., monoamine oxidase inhibitors, alpha-adrenergic agonists or antagonists).
- Patients with anticipated surgery duration exceeding 4 hours.
- Patients with significant intraoperative bleeding anticipated.
- Patients with any condition that, in the opinion of the investigator, would compromise their safety or ability to complete the study.

Study Variable:
Primary Study Variable:

- Hemodynamic Response: This includes changes in blood pressure (systolic, diastolic, mean arterial pressure) and heart rate before, during, and after laryngoscopy and intubation. The response may be assessed at various time points such as baseline, pre-induction, pre-intubation, immediately post-intubation, and at specific

intervals thereafter.

Secondary Study Variables:

- Adverse Events: Any adverse events related to the administration of dexmedetomidine or clonidine, such as bradycardia, hypotension, or other cardiovascular complications.
- Duration of Intubation: The time taken for successful endotracheal intubation.
- Intubation Conditions: Subjective assessment of intubation conditions by the anesthesiologist, which may include ease of laryngoscopy, vocal cord visualisation, and ease of passage of the endotracheal tube.
- Duration of Anaesthesia: The duration of time from induction of anaesthesia to emergence from anaesthesia.
- Postoperative Pain: Assessment of postoperative pain using a validated pain scale such as visual analog scale (VAS) or numerical rating scale (NRS).
- Postoperative Recovery: Assessment of recovery parameters such as time to extubation, time to achieve Aldrete score ≥ 9, and time to readiness for discharge from the post-anesthesia care unit (PACU).
- Hemodynamic Stability During Surgery: Hemodynamic parameters during the intraoperative period, including blood pressure and heart rate stability.
- Patient Satisfaction: Subjective assessment of patient satisfaction with the anaesthetic technique and perioperative care.

RESULT

Table 1: Demographic and Baseline Hemodynamic Data

	Variable	Group clonidine (n=50)	Group dexmede-tomidine (n=50)	Control (n=50)	P value
Demographic data	Male (%)	12 (40)	13 (43.3)	14 (46.7)	0.76
	ASA physical status I/II	25/25	28/22	32/18	0.67
	Age (years), mean±SD	29.5 ± 4.8	30.8 ± 5.5	32.6 ± 5.8	0.66
	Weight (kg), mean±SD	62.3 ±s 8.9	61.8 ± 11.2	64.8 ± 7.8	0.26
Hemodynamic data	HR (/min), mean±SD	77.5 ± 9.7	75.8 ± 17	76.1 ± 14.6	0.52
	SBP (mmHg), mean±SD	117.9 ± 5.9	120.1 ± 13.5	123.1 ± 4.8	0.17
	DBP (mmHg), mean±SD	73.1 ± 4.8	74.5 ± 9.8	76.9 ± 7.6	0.14
	MAP (mmHg), mean±SD	88.7 ± 5.1	89.4 ± 10.4	91.4 ± 6.5	0.33

Table 2: Distribution Based on General Examination Findings 10 min after Drug Administration

	Group	Group C	Group D	Group S	P (C vs D)	P (C vs S)	P (D vs S)
10 min after drug	HR (/min)	71.6± 8.7	60.8 ± 13.9	75.5± 15.3	0.005	0.45	<0.01
	SBP (mmHg)	107.7 ±7.6	109.8 ± 14.5	122.1 ±5.9	0.91	<0.001	<0.001
	DBP (mmHg)	64.1± 3.4	65.7 ± 13.0	75.3± 8.3	0.98	<0.001	<0.001
	MAP (mmHg)	78.8± 3.8	80.3 ± 12.7	90.8± 7.2	0.88	<0.001	<0.001

Table 3: Distribution Based on General Examination Findings after Intubation

	Group	Group C	Group D	Group S	P (C vs D)	P (C vs S)	P (D vs S)
After 1 min	HR (/min)	82.4 ± 6.3	75.5± 13.7	98.7± 15.3	0.83	<0.001	<0.001
	SBP (mmHg)	117.3 ± 7.2	110.8± 8.4	131.1±5 .7	0.002	<0.001	<0.001
	DBP (mmHg)	74.1± 5.7	70.3± 6.1	84.3± 9.2	0.1	<0.001	<0.001
	MAP (mmHg)	88.4 ± 4.7	83.7± 5.6	99.9± 7.7	0.01	<0.001	<0.001
After 3 min	HR (/min)	78.8± 6.3	72.5± 11.5	95.8± 15.4	0.12	<0.001	<0.001
	SBP (mmHg)	112.1± 8.6	106.6±8 .3	127.9±6 .1	0.017	<0.001	<0.001
	DBP (mmHg)	69.5± 5.4	64.8± 11.1	82.3± 7.8	0.1	<0.001	<0.001
	MAP (mmHg)	83.4± 5.7	78.6± 7.5	97.6± 7.1	0.014	<0.001	<0.001
After 5 min	HR (/min)	74.3± 6.2	69.8± 12.7	94.3± 15.7	0.49	<0.001	<0.001
	SBP (mmHg)	113.2± 7.9	103.5±6 .9	126.3+ 6.1	0.001	<0.001	<0.001
	DBP (mmHg)	67.9± 5.1	61.9± 9.7	80.3+ 8.5	0.011	<0.001	<0.001
	MAP (mmHg)	82.8± 4.7	75.7± 7.9	95.7± 7.4	0.003	<0.001	<0.001
After 10 min	HR (/min)	74.9± 8.5	67.8± 11.5	91.4± 15.5	0.08	<0.001	<0.001
	SBP (mmHg)	110.3± 7.6	99.3± 8.8	124.2±6 .2	<0.001	<0.001	<0.001
	DBP (mmHg)	65.5± 7.4	58.5+ 10.6	78.8± 7.6	0.006	<0.001	<0.001
	MAP (mmHg)	80.5± 6.8	71.8± 9.9	93.7± 7.3	0.0004	<0.001	<0.001

Demographic Data

Sex

In Group clonidine, 12 (40%) patients were male. In Group dexmedetomidine, 13 (43.3%) patients were male.In Group Control, 14(46.7%) patients were male. Association of sex with Demographic data Group was not statistically significant (p=0.76

ASA Physical Status I/II

In Group clonidine, 25/25 patients had ASA physical status I/II. In Group dexmedetomidine, 28/22 patients had ASA physical status I/II. In Group Control, 32/18 patients had ASA physical status I/II. Association of sex with Demographic data Group was not statistically significant (p=0.67).

Age (years)

In Group clonidine, the mean Age (mean± s.d.) of patients was 29.5 ± 4.8 In Group dexmedetomidine, the mean Age (mean± s.d.) of patients was 30.8 ± 5.5. In Group Control, the mean Age (mean± s.d.) of patients was 32.6 ± 5.8. Distribution of mean Age (years) with the Demographic data Group was not statistically significant (p=0.66).

Weight (kg)

In Group clonidine, the mean Weight (kg) (mean± s.d.) of patients was 62.3 ± 8.9. In Group dexmedetomidine, the mean Weight (kg) (mean± s.d.) of patients was 61.8 ± 11.2. In Group Control, the mean Weight (kg) (mean± s.d.) of patients was 64.8 ± 7.8. Distribution of mean Weight (kg) with the Demographic data Group was not statistically significant (p=0.26).

Hemodynamic Data

HR (/min)

In Group clonidine, the mean HR (/min) (mean± s.d.) of patients was 62.3 ± 8.9 In Group dexmedetomidine, the mean HR (/min) (mean± s.d.) of patients was 61.8 ± 11.2. In Group Control, the mean HR (/min) (mean± s.d.) of patients was 64.8 ± 7.8. Distribution of mean HR (/min) with the Demographic data Group was not statistically significant (p=0.26).

SBP (mmHg)

In Group clonidine, the mean SBP (mmHg) (mean± s.d.) of patients was 117.9 ± 5.9. In Group dexmedetomidine, the mean SBP (mmHg) (mean± s.d.) of patients was 120.1 ± 13.5.In Group Control, the mean SBP (mmHg) (mean± s.d.) of patients was 123.1 ± 4.8. Distribution of mean SBP (mmHg) with the Demographic data Group was not statistically significant (p=0.17).

DBP (mmHg)

In Group clonidine, the mean SBP (mmHg) (mean± s.d.) of patients was 73.1 ± 4.8. In Group dexmedetomidine, the mean SBP (mmHg) (mean± s.d.) of patients was 74.5 ± 9.8. In Group Control, the mean SBP (mmHg) (mean± s.d.) of patients was 76.9 ± 7.6. Distribution of mean SBP (mmHg) with the Demographic data Group was not statistically significant (p=0.14).

MAP (mmHg)

In Group clonidine, the mean MAP (mmHg) (mean± s.d.) of patients was 88.7 ± 5.1. In Group dexmedetomidine the mean MAP (mmHg) (mean± s.d.) of patients was 89.4 ± 10.4. In Group Control, the mean

MAP (mmHg) (mean± s.d.) of patients was 91.4± 6.5. Distribution of mean MAP (mmHg) with the Demographic data Group was not statistically significant (p=0.33).

10 min after Drug Administration

HR (/min)

In Group C, the mean HR (/min) (mean± s.d.) of patients was 71.6±8.7. In Group D, the mean HR (/min) (mean± s.d.) of patients was 60.8 ± 13.9. In Group S, the mean HR (/min) (mean± s.d.) of patients was 75.5±15.3. Distribution of mean HR (/min) with After 1 min Group(C vs D) was statistically significant (p=0.005). Distribution of mean HR (/min) with After 1 min Group(C vs S) was not statistically significant (p=0.45). Distribution of mean HR (/min) with After 1 min Group (D vs S) was statistically significant (p<0.01).

SBP (mmHg)

In Group C, the mean SBP (mmHg) (mean± s.d.) of patients was 107.7±7.6. In Group D, the mean SBP (mmHg) (mean± s.d.) of patients was 109.8 ± 14.5. In Group S, the mean SBP (mmHg) (mean± s.d.) of patients was 122.1±5.9. Distribution of mean SBP (mmHg) with After 1 min Group(C vs D) was not statistically significant (p=0.91). Distribution of mean SBP (mmHg) with After 1 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean SBP (mmHg) with After 1 min Group (D vs S) was statistically significant (p<0.001).

DBP (mmHg)

In Group C, the mean DBP (mmHg) (mean± s.d.) of patients was 64.1±3.4. In Group D, the mean DBP (mmHg) (mean± s.d.) of patients was 65.7 ± 13.0. In Group S, the mean DBP (mmHg) (mean± s.d.) of patients was 75.3±8.3. Distribution of mean DBP (mmHg) with After 1 min Group(C vs D) was not statistically significant (p=0.98). Distribution of mean DBP (mmHg) with After 1 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean DBP (mmHg) with After 1 min Group (D vs S) was statistically significant (p<0.001).

MAP (mmHg)

In Group C, the mean MAP (mmHg) (mean± s.d.) of patients was 78.8±3.8. In Group D, the mean MAP (mmHg) (mean± s.d.) of patients was 80.3 ± 12.7. In Group S, the mean MAP (mmHg) (mean± s.d.) of patients was 90.8±7.2. Distribution of mean MAP (mmHg) with After 1 min Group(C vs D) was not statistically significant (p=0.88). Distribution of mean MAP (mmHg) with After 1 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean MAP (mmHg) with After 1 min Group (D vs S) was statistically significant (p<0.001).

After 1 min Intubation

HR (/min)

In Group C, the mean HR (/min) (mean± s.d.) of patients was 82.4 ± 6.3 In Group D, the mean HR (/min) (mean± s.d.) of patients was 75.5±13.7 In Group S, the mean HR (/min) (mean± s.d.) of patients was 98.7±15.3 Distribution of mean HR (/min) with After 1 min Group(C vs D) was not statistically significant (p=0.83). Distribution of mean HR (/min) with After 1 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean HR (/min) with After 1 min Group (D vs S) was statistically significant (p<0.001).

SBP (mmHg)

In Group C, the mean SBP (mmHg) (mean± s.d.) of patients was 117.3 ± 7.2. In Group D, the mean SBP (mmHg) (mean± s.d.) of patients was 110.8±8.4. In Group S, the mean SBP (mmHg) (mean± s.d.) of patients was 131.1±5.7. Distribution of mean SBP (mmHg) with After 1 min Group(C vs D) was statistically significant (p=0.002). Distribution of mean SBP (mmHg) with After 1 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean SBP (mmHg) with After 1 min Group (D vs S) was statistically significant (p<0.001).

DBP (mmHg)

In Group C, the mean DBP (mmHg) (mean± s.d.) of patients was 74.1±5.7. In Group D, the mean DBP (mmHg) (mean± s.d.) of patients was 70.3±6.1. In Group S, the mean DBP (mmHg) (mean± s.d.) of patients was 84.3±9.2. Distribution of mean DBP (mmHg) with After 1 min Group(C vs D) was not statistically significant (p=0.1). Distribution of mean DBP (mmHg) with After 1 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean DBP (mmHg) with After 1 min Group (D vs S) was statistically significant (p<0.001).

MAP (mmHg)

In Group C, the mean MAP (mmHg) (mean± s.d.) of patients was 88.4 ± 4.7. In Group D, the mean MAP (mmHg) (mean± s.d.) of patients was 83.7±5.6. In Group S, the mean MAP (mmHg) (mean± s.d.) of patients was 99.9±7.7. Distribution of mean MAP (mmHg) with After 1 min Group(C vs D) was statistically significant (p=0.01). Distribution of mean MAP (mmHg) with After 1 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean MAP (mmHg) with After 1 min Group (D vs S) was statistically significant (p<0.001).

After 3 min Intubation

HR (/min)

In Group C, the mean HR (/min) (mean± s.d.) of patients was 78.8±6.3. In Group D, the mean HR (/min) (mean± s.d.) of patients was 72.5±11.5. In Group S, the mean HR (/min) (mean± s.d.) of patients was 95.8±15.4. Distribution of mean HR (/min) with After 3 min Group(C vs D) was not statistically significant (p=0.12). Distribution of mean HR (/min) with After 3 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean HR (/min) with After 3 min Group (D vs S) was statistically significant (p<0.001).

SBP (mmHg)

In Group C, the mean SBP (mmHg) (mean± s.d.) of patients was 112.1±8.6. In Group D, the mean SBP (mmHg) (mean± s.d.) of patients was 106.6±8.3. In Group S, the mean SBP (mmHg) (mean± s.d.) of patients was 127.9±6.1. Distribution of mean SBP (mmHg) with After 3 min Group(C vs D) was statistically significant (p=0.017). Distribution of mean SBP (mmHg) with After 3 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean SBP (mmHg) with After 3 min Group (D vs S) was statistically significant (p<0.001).

DBP (mmHg)

In Group C, the mean DBP (mmHg) (mean± s.d.) of patients was 69.5±5.4. In Group D, the mean DBP (mmHg) (mean± s.d.) of patients was 64.8±11.1. In Group S, the mean DBP (mmHg) (mean± s.d.) of patients was 82.3±7.8. Distribution of mean DBP (mmHg) with After 3 min Group(C vs D) was not statistically significant (p=0.1). Distribution of mean DBP (mmHg) with After 3 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean DBP (mmHg) with After 3 min Group (D vs S) was statistically significant (p<0.001).

MAP (mmHg)

In Group C, the mean MAP (mmHg) (mean± s.d.) of patients was 83.4±5.7. In Group D, the mean MAP (mmHg) (mean± s.d.) of patients was 78.6±7.5. In Group S, the mean MAP (mmHg) (mean± s.d.) of patients was 97.6±7.1. Distribution of mean MAP (mmHg) with After 3 min Group(C vs D) was statistically significant (p=0.014). Distribution of mean MAP (mmHg) with After 3 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean MAP (mmHg) with After 3 min Group (D vs S) was statistically significant (p<0.001).

After 5 min Intubation

HR (/min)

In Group C, the mean HR (/min) (mean± s.d.) of patients was 74.3±6.2. In Group D, the mean HR (/min) (mean± s.d.) of patients was 69.8±12.7. In Group S, the mean HR (/min) (mean± s.d.) of patients was 94.3±15.7. Distribution of mean HR (/min) with After 5 min Group(C vs D) was not statistically significant (p=0.49).

Distribution of mean HR (/min) with After 5 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean HR (/min) with After 5 min Group (D vs S) was statistically significant (p<0.001).

SBP (mmHg)

In Group C, the mean SBP (mmHg) (mean± s.d.) of patients was 113.2±7.9. In Group D, the mean SBP (mmHg) (mean± s.d.) of patients was 103.5±6.9. In Group S, the mean SBP (mmHg) (mean± s.d.) of patients was 126.3+6.1. Distribution of mean SBP (mmHg) with After 5 min Group(C vs D) was statistically significant (p=0.001).

Distribution of mean SBP (mmHg) with After 5 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean SBP (mmHg) with After 5 min Group (D vs S) was statistically significant (p<0.001).

DBP (mmHg)

In Group C, the mean DBP (mmHg) (mean± s.d.) of patients was 67.9±5.1 In Group D, the mean DBP (mmHg) (mean± s.d.) of patients was 61.9±9.7. In Group S, the mean DBP (mmHg) (mean± s.d.) of patients was 80.3+8.5. Distribution of mean DBP (mmHg) with After 5 min Group(C vs D) was statistically significant (p=0.011). Distribution of mean DBP (mmHg) with After 5 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean DBP (mmHg) with After 5 min Group (D vs S) was statistically significant (p<0.001).

MAP (mmHg)

In Group C, the mean MAP (mmHg) (mean± s.d.) of patients was 82.8±4.7. In Group D, the mean MAP (mmHg) (mean± s.d.) of patients was 75.7±7.9. In Group S, the mean MAP (mmHg) (mean± s.d.) of patients was 95.7±7.4. Distribution of mean MAP (mmHg) with After 5 min Group(C vs D) was statistically significant (p=0.003). Distribution of mean MAP (mmHg) with After 5 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean MAP (mmHg) with After 5 min Group (D vs S) was statistically significant (p<0.001).

After 10 min Intubation

HR (/min)

In Group C, the mean HR (/min) (mean± s.d.) of patients was 74.9±8.5. In Group D, the mean HR (/min) (mean± s.d.) of patients was 67.8±11.5. In Group S, the mean HR (/min) (mean± s.d.) of patients was 91.4±15.5. Distribution of mean HR (/min) with After 10 min Group(C vs D) was not statistically significant (p=0.08). Distribution of mean HR (/min) with After 10 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean HR (/min) with After 10 min Group (D vs S) was statistically significant (p<0.001).

SBP (mmHg)

In Group C, the mean SBP (mmHg) (mean± s.d.) of patients was 110.3±7.6. In Group D, the mean SBP (mmHg) (mean± s.d.) of patients was 99.3±8.8. In Group S, the mean SBP (mmHg) (mean± s.d.) of patients was 124.2±6.2. Distribution of mean SBP (mmHg) with After 10 min Group(C vs D) was statistically significant (p<0.001). Distribution of mean SBP (mmHg) with After 10 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean SBP (mmHg) with After 10 min Group (D vs S) was statistically significant (p<0.001).

DBP (mmHg)

In Group C, the mean DBP (mmHg) (mean± s.d.) of patients was 65.5±7.4. In Group D, the mean DBP (mmHg) (mean± s.d.) of patients was 58.5+10.6. In Group S, the mean DBP (mmHg) (mean± s.d.) of patients was 78.8±7.6. Distribution of mean DBP (mmHg) with After 10 min Group(C vs D) was statistically significant (p=0.006). Distribution of mean DBP (mmHg) with After 10 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean DBP (mmHg) with After 10 min Group (D vs S) was statistically significant (p<0.001).

MAP (mmHg)

In Group C, the mean MAP (mmHg) (mean± s.d.) of patients was 80.5±6.8. In Group D, the mean MAP (mmHg) (mean± s.d.) of patients was 71.8±9.9. In Group S, the mean MAP (mmHg) (mean± s.d.) of patients was 93.7±7.3. Distribution of mean MAP (mmHg) with After 10 min Group(C vs D) was statistically significant (p=0.0004). Distribution of mean MAP (mmHg) with After 10 min Group(C vs S) was statistically significant (p<0.001). Distribution of mean MAP (mmHg) with After 10 min Group (D vs S) was statistically significant (p<0.001).

DISCUSSION

The present study was a Comparative Study. Increased stress is linked to anaesthesia and surgery, and this stress can be seen as tachycardia, hypertension, and elevated sympathetic activity. Stressful events like laryngoscopy and intubation can raise blood levels of catecholamines, cause tachycardia, and raise blood pressure. In this investigation, an equivalent amount of hypertensive response to laryngoscopy and intubation was reduced by administering 2 µg/kg of clonidine and 1 µg/kg of dexmedetomidine intravenously. 2 µg/kg of clonidine was utilised by Zalunardo et al. [7] and Ray et al. [8] to lessen the hemodynamic response after tracheal intubation. Because oral and intramuscular clonidine must be administered 90 minutes beforehand and has been demonstrated to be beneficial in attenuating the intubation reaction, they were not selected for this investigation.

Demographic Data

In our study, Age was higher in Control [32.6 ± 5.8] compared to Group dexmedetomidine [30.8± 5.5], Group clonidine [29.5 ± 4.8] but this was not statistically significant (p=0.66).

In our study, Weight (kg) was higher in Control [64.8±7.8] compared to Group dexmedetomidine [61.8 ± 11.2] and Group clonidine [62.3 ± 8.9] but this was not statistically significant (p=0.26).

α2-agonists are known to cause hypotension and bradycardia as their main adverse effects. Both clonidine and dexmedetomidine were shown in a research by Kakkar et al. to be efficacious in avoiding hemodynamic response to intubation; however, dexmedetomidine was linked to a higher incidence of adverse effects, including bradycardia and hypotension.[9]

Haemodynamic Data

In our study, HR (/min) was higher in Group clonidine [77.5 ± 9.7] compared to Group dexmedetomidine [75.8 ± 17] and Control [76.1 ± 14.6] but this was not statistically significant (p=0.52).

In our study, SBP (mmHg) was higher in Control [123.1 ± 4.8] compared to Group dexmedetomidine [120.1 ± 13.5] and Group clonidine [117.9 ± 5.9] but this was not statistically significant (p=0.17).

The effects of a single IV dosage of dexmedetomidine on the anaesthetic need, hemodynamic response to laryngoscopy, and tracheal intubation were investigated by Jaakola et al. Dexmedetomidine was linked to a reduction in the need for anaesthesia and a blunting of the hemodynamic response to intubation.[20]

In our study, DBP (mmHg) was higher in Control [76.9±7.6] compared to Group dexmedetomidine [74.5 ± 9.8] and Group clonidine [73.1 ± 4.8] but this was not statistically significant (p=0.14).

In our study, MAP (mmHg) was higher in Control [91.4± 6.5] compared to Group dexmedetomidine [89.4±10.4] and Group clonidine [88.7 ± 5.1] but this was not statistically significant (p=0.33).

10 min After Drug

In our study, HR (/min) was higher in Group C [71.6±8.7] compared to Group D [60.8 ±13.9] which was statistically significant (p=0.005).

In our study, HR (/min) was higher in Group S [75.5±15.3] compared to Group C [71.6±8.7] which was not statistically significant (p=0.45).

In our study, HR (/min) was higher in Group S [75.5±15.3] compared to Group D [60.8 ± 13.9] which was statistically significant (p<0.01).

In our study, SBP (mmHg) was higher in Group D [109.8 ± 14.5] compared to Group C [107.7±7.6] which was not statistically significant (p=0.91).

In our study, SBP (mmHg) was higher in Group S [122.1±5.9] compared to Group C [107.7±7.6] which was statistically significant (p<0.001).

In our study, SBP (mmHg) was higher in Group S [122.1±5.9] compared to Group D [109.8±14.5] which was statistically significant (p<0.001).

In our study, DBP (mmHg) was higher in Group D [65.7 ± 13.0] compared to Group C [64.1±3.4] which was not statistically significant (p=0.98).

In our study, DBP (mmHg) was higher in Group S [75.3±8.3] compared to Group C [64.1±3.4] which was statistically significant (p<0.001).

In our study, DBP (mmHg) was higher in Group S [75.3±8.3] compared to Group D [65.7 ± 13.0] which was statistically significant (p<0.001).

In our study, MAP (mmHg) was higher in Group D [80.3 ± 12.7] compared to Group C [78.8±3.8] which was not statistically significant (p=0.88).

In our study, MAP (mmHg) was higher in Group S [90.8±7.2] compared to Group C [78.8±3.8] which was statistically significant

(p<0.001).

In our study, MAP (mmHg) was higher in Group S [90.8±7.2] compared to Group D [80.3 ± 12.7] which was statistically significant (p<0.001).

After 1 min

In our study, SBP (mmHg) was higher in Group C [117.3 ± 7.2] compared to Group D [110.8±8.4] which was statistically significant (p=0.002).

In our study, SBP (mmHg) was higher in Group S [131.1±5.7] compared to Group C [117.3±7.2] which was statistically significant (p<0.001).

In our study, SBP (mmHg) was higher in Group S [131.1±5.7] compared to Group D [110.8±8.4] which was statistically significant (p<0.001).

After 3 min

In our study, SBP (mmHg) was higher in Group C [112.1±8.6] compared to Group D [106.6±8.3] which was statistically significant (p=0.017).

In our study, SBP (mmHg) was higher in Group S [127.9±6.1] compared to Group C [112.1±8.6] which was statistically significant (p<0.001).

In our study, SBP (mmHg) was higher in Group S [127.9±6.1] compared to Group D [106.6±8.3] which was statistically significant (p<0.001).

After 5 min

In our study, SBP (mmHg) was higher in Group C [113.2±7.9] compared to Group D [103.5±6.9] which was statistically significant (p=0.001).

In our study, SBP (mmHg) was higher in Group S [126.3+6.12] compared to Group C [113.2±7.9] which was statistically significant (p<0.001).

In our study, SBP (mmHg) was higher in Group S [126.3+6.1] compared to Group D [103.5±6.9] which was statistically significant (p<0.001).

After 10 min

In our study, SBP (mmHg) was higher in Group C [110.3±7.6] compared to Group D [99.3±8.8] which was statistically significant (p<0.001).

2] compared to Group C [110.3±7.6] which was statistically significant (p<0.001).

In our study, SBP (mmHg) was higher in Group S [124.2±6.2] compared to Group D [99.3±8.8] which was statistically significant (p<0.001).

CONCLUSION

As compared to the two study groups (Clonidine and Dexmedetomidine), in the placebo group all the haemodynamic parameters were higher than the baseline values.

In all the groups, a mean decrease in haemodynamic parameters as compared to after intubation values at all the follow up intervals.

In Dexmedetomidine group, the mean values for all haemodynamic parameters were significantly lower as compared to placebo groups at all after intubation time intervals.

When compared among the two groups (Dexmedetomedine and Clonidine), both came out to be statistically similar in attenuating haemodynamic responses.

No incidence of adverse side effects viz. Tachycardia, bradycardia or hypotension were observed in all the three groups under study.

The oxygen saturation was observed to be 98% in all three groups at all time intervals.

The early onset and promising response of Dexmedetomidine makes it a promising choice. However, before its routine use, we recommend larger studies on multiple dosages to find out optimum dosage at which Dexmedetomidine can be used. Fortunately, in the present study no adverse effect of the drug was noticed. However, this being a study with limited sample size, a study with larger sample size and different dose optimization is recommended.

REFERENCES

1. Shribman AJ, Smith G, Achola KJ. Cardiovascular and catecholamine responses to laryngoscopy with and without tracheal intubation. Br J Anaesth. 1987;59:295–9.
2. da Silva Neto WV, Azevedo GS, Coelho FO, Netto EM, Ladeia AM. Evaluation of hemodynamic variations during anaesthetic induction in treated hypertensive patients. Rev Bras Anestesiol. 2008;58:330–41.
3. Aho M, Lehtinen AM, Erkola O, Kallio A, Korttila K. The effect of intravenously administered dexmedetomidine on perioperative hemodynamics and isoflurane requirements in patients undergoing abdominal hysterectomy. Anesthesiology. 1991;74:997–1002.
4. Singh S, Arora K. Effect of oral clonidine premedication on perioperative haemodynamic response and postoperative analgesic requirement for patients undergoing laparoscopic cholecystectomy. Indian J Anaesth. 2011;55:26–30.
5. Grewal A. Dexmedetomidine: New avenues. J Anaesthesiol Clin Pharmacol. 2011;27:297–302.
6. Duncan D, Sankar A, Beattie WS, Wijeysundera DN. Alpha-2 adrenergic agonists for the prevention of cardiac complications among adults undergoing surgery. Cochrane Database Syst Rev. 2018;3:CD004126.
7. Zalunardo MP, Zollinger A, Spahn DR, Seifert B, Pasch T. Preoperative clonidine attenuates stress response during emergence from anaesthesia. J Clin Anesth. 2000;12:343–9.
8. Ray M, Bhattacharjee DP, Hajra B, Pal R, Chatterjee N. Effect of clonidine and magnesium sulphate on anaesthetic consumption, haemodynamics and postoperative recovery: A comparative study. Indian J Anaesth. 2010;54:137–41.
9. Kakkar A, Tyagi A, Nabi N, Sethi AK, Verma UC. Comparison of clonidine and dexmedetomidine for attenuation of laryngoscopy and intubation response – A randomised controlled trial. J Clin Anesth. 2016;33:283–8.