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ORIGINAL ARTICLE

Comparing Intravenous versus Nebulized either Dexmedetomidine or Lidocaine for Attenuation of the Hemodynamic Responses to Laryngoscopy and Intubation

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ABSTRACT

Background: To reduce the laryngoscopy effects and the risks of hemodynamic perturbations, nebulized dexmedetomidine (Neb-Dex) could be a useful alternative to conventional intravenous medications.This study compared the effects of intravenous versus nebulized, either dexmedetomidine or lidocaine, on attenuation of the hemodynamic responses that follow laryngoscopy and endotracheal intubation.

Methods: In a prospective double-blinded clinical study, 92 cases were allocated randomly into four groups, 23 cases in each group:Group1: (ND) Nebulized Dexmedetomidine, Group 2: (NL) Nebulized Lidocaine, Group 3: (VD) IV Dexmedetomidine, and Group 4: (VL) IV Lidocaine.Changes in patients' serum cortisol levels before and after laryngoscopy were the primary outcome, while secondary outcome parameters included changes in systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rates (HR), as well as mean arterial pressure (MAP) basally and after laryngoscopy and endotracheal intubation.**Results:**There was a significant decrease of serum Cortisol level in groups (VD) & (ND) at ten minutes13.35±1.7 &13.59±2mcg/dlin comparison to basal time13.91±1.4 &13.76±1.9mcg/dl respectively, $p<0.05$. In comparison, there was a significant increase in serum Cortisol ug/dl in group (NL)&group (VL) at ten minutes13.79±1.22 &14.01±1.42mcg/dl in comparison to basal time13.49±1.07 & 13.66±1.52mcg/dlrespectively, $p<0.05$.There was a significant decline in heart rates systolic, diastolic and mean arterial pressures in group VD compared with groups NL, ND, and VL at different time intervals ($p<0.05$ in each).**Conclusion:**
Thehemodynamic response to laryngoscopy and intubation can be mitigated, to varying degrees, by administering either intravenous or nebulized lidocaine or dexmedetomidine.

Nebulized dexmedetomidine with the best profile outcome could be a useful alternative to conventional intravenous medications.The decline in systolic and diastolic blood pressure, mean arterial pressure and heart rates before and after laryngoscopy were more prominent in the intravenous dexmedetomidine than in other groups.

Keywords: Nebulized,Dexmedetomidine,Lidocaine, Laryngoscopy, Intubation.

INTRODUCTION

Laryngoscopy and endotracheal intubation are the most stimulating procedures that cause acute hemodynamic responses that endure for at least ten minutes and are accompanied by sympathetic reactions. These brief reactions manifest as a rise inheart rate and blood pressure just after intubation. Increased stress hormone levels such as catecholamine and cortisol in the plasma cause these reactions. Medications used to lessen sympathetic reactions to laryngoscopy and intubation include fentanyl, esmolol, lidocaine and α2-agonists, such as clonidine and dexmedetomidine [1].

Every year, over 310 million surgeries are carried out worldwideand many of them embrace the successful endotracheal intubation of the patient to conduct these procedures successfully.Medications such as fentanyl, esmolol, lidocaine and α2-agonists, including clonidine and dexmedetomidine, have been used to alter sympathetic responses to laryngoscopy and intubation[2].

Intranasal dexmedetomidine has been studied for its effectiveness, safety and high patient acceptance. Since nebulized dexmedetomidine (Neb-Dex) spreads to a larger surface area (the nasal, buccal and respiratory mucosa), it may be a good substitute because it improves systemic absorption, reduces the risk of postoperative sore throat and lessens the effects of sedation, analgesia and laryngoscopy response. Selective alpha-2 agonists delivered intravenously (IV) have been shown to obstruct sympathoadrenal responses related to laryngeal intubation [3].

Because topical anesthetic directly inhibits airway reflexes, it can help minimize hemodynamic reaction during laryngoscopy and endotracheal intubation. An α2 receptor agonist, dexmedetomidine, has analgesic, sedative and sympatholytic effects without causing clinically significant respiratory depression. It has a systemic effect by preventing norepinephrine from being released and lowering sympathetic activity[4].

Lidocaine is a form of local anesthetic in the amide family; it is an aminoethyl amide. Hemodynamic reactions to laryngoscopy and intubation were blunted by administering a

single intravenous dose of lidocaine (1.5 mg/kg) three minutes before intubation [5].

So, we aimedinthis study to compare the effects of intravenous versus nebulized either dexmedetomidine or lidocaine for attenuation of the hemodynamic responses that follow laryngoscopy and endotracheal intubation.

METHODS

We conducted thisprospective clinical studyat Zagazig University Hospitals from January 2023 to August2023. It is a double-blinded study as the patient and the outcomes observer were blinded to the patient group and the intervention.After institutional review board approval of (IRB#10666/2–4-2023) and registration on clinicaltrials.gov ID (NCT05941767, date of registration: 7-4–2023), written informed consent was obtained from all participants. The study was done according to The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Inclusion criteria comprised individuals aged from 18 to 50 years from both sexes who were scheduled to undergo elective surgery under general anesthesia, “the first case of the morning operative list,” those who were American Society of Anesthesiologists(ASA) I & ASA II, patients with BMI: ≤ 35 kg/m^2 and those with Mallampati grade I,II. Conversely, exclusion criteria were used to exclude hemodynamically unstable patientsand patients who had an allergy to anyof the used drugs.Additionally, the study did not include individuals with renal or hepatic dysfunction or hypertension.Complete medical histories, physical examinations (the patient's general fitness, airway by Mallampati grading, nutritional state, body weight and a thorough check of the circulatory and respiratory systems were all evaluated before the anesthesia was administered) and laboratory investigations were performed on all study participants. These tests included a complete blood count (CBC), random blood glucose, kidney function test, liver function test and coagulation profile.

Six hours before surgery, all trial participants were kept nil per os.A blood sample was taken for basal cortisol level measurement 45 minutes before surgery. When patients were escorted into the preoperative room, they had an intravenous line placed, a maintenance fluid started, and their baseline systolic, diastolic and mean arterial pressures,as well as heart rates,were recorded. On admission to the operating room, patients were connected to monitor those tracks vitals (heart rate, non-invasive blood pressure measures (systolic, diastolic, mean arterial pressure), continuous electrocardiogram (ECG) and oxygen saturation).

Patients were randomized by computer-generated randomization table according to

the technique used into four equal groups ((23 cases)) in each group: Group (ND) N.=23 patients (Nebulized dexmedetomidine (200mcg/2ml))،the patient was given 2 milliliters of 0.9% normal saline in a face mask nebulizer and one microgram per kilogram (mcg/kg) of body weight of dexmedetomidine 10 minutes before induction. Twenty minutes before induction, a 50 ml syringe pump of normal saline was started, and a bolus of 10 ml of normal saline 0.9 percent was administered ninety seconds prior to the laryngoscopy.

Group (NL) N.=23 patients (Nebulized Lidocaine): Ten minutes before induction, the patients were given a nebulized mixture of 3 milligrams per kilogram of body weight (mg/kg) of lidocaine 4% and 2 milliliters of normal saline (0.9%). Twenty minutes prior to induction, a 50 ml syringe pump of normal saline was flushed by an infusion, and a bolus of 10 ml of 0.9 percent normal saline was administered ninety seconds before the laryngoscopy procedure.

Group (VD) N.=23 patients (Intravenous Dexmedetomidine): patients were given an intravenous infusion of 1mcg/kg of dexmedetomidine((200 mcg/2ml)) added to 48 ml of normal saline 0.9 percent ((1 ml= 4mcg)) 20 minutes prior to induction. For unbiased results, 3 ml of 0.9% normal saline via face mask nebulization was accomplished ten minutes before induction.Additionally, 90 seconds before laryngoscopy, a 10 ml of 0.9% normal saline bolus was injected intravenously.

Group (VL) N.=23 patients (Intravenous Lidocaine): Patients were administered an intravenous bolus of 2% lidocaine (1.5 mg/kg) (1 ml = 20 mg) completed to 10 ml with 0.9% normal saline and 90 seconds prior to the induction, for avoiding the bias ten minutes prior to induction, 3 ml of 0.9% normal saline via face mask nebulization was accomplished.Twenty minutes before the laryngoscopy, a 50 ml syringe pump of normal saline was flushed by an infusion.Three minutes of preoxygenation at 100% oxygen were followed by the induction of anesthesia with propofol (2 mg/kg), fentanyl (1 mcg/kg) and then atracurium (0.5 mg/kg) to facilitate endotracheal intubation, 1.5% isoflurane in 100% O_2 was used to maintain anesthesia. Tidal volumes of 8 ml/kg and respiratory rates of 12 breaths/minute were used to initiate mechanical ventilation, which was then adjusted to keep the patient in the normocapnic range of 35-45 mmHg.Neostigmine 0.04mg/kg and atropine 0.02mg/kg were administered to reverse the effects of the muscle relaxant after surgery. The same anesthesia team conducted all procedures with the same policy.

Data collection: the primary outcome was the change in serumCortisol levelfrom a baseline blood sample taken 45 minutes before surgery

and 10 minutes after intubation. The secondary outcomes included the durationit took to perform the laryngoscopy and intubation, the changes inthe patient's heart rate and other hemodynamic parameters such as systolic, diastolic andmean arterial blood pressuresat the baseline before the use of the study drugs and then at 1,3,5,7,and 10 minutes after the endotracheal intubation.Any reported side effectsin the first 24 hours were documented.

Statistical analysis : Sample size: Assuming the mean MAP was 92,86 ± 15.5 mmHg vs 103.7 ± 10.6 mmHg in the DL group "intravenous dexmedetomidine and nebulized lidocaine" versus the D group "intravenous dexmedetomidine." At 80% power and 95% CI, the estimated sample was 92 cases allocated randomly into four groups, 23 cases in each group using Open Epi program. IBM's statistical analysis software, SPSS, version 20.0, was used to process the data. Qualitative data was represented with numerical and percentage-based language. Quantitative information was summarized by means and standard deviations. An ANOVA was performed to test for statistical significance between more than two groups.The existence of a statistically significant difference between the groups was determined using a student t-test. The correlation between two qualitative factors was analyzed using the chi-square test.

RESULTS

In this study, 100 individuals were assessed for eligibility, 8 cases were excluded (3 refused to participate, and 5 cases didn't meet inclusion criteria) and specific eligibility criteria were applied to the participants (Figure 1).

Age, sex, BMI, ASA, Mallampati and duration of laryngoscopydid not show any statistically significant difference among the four groups (P> 0.05); There was a significant decrease in serum Cortisol level in groups (VD) & (ND) at ten minutes13.35±1.7 &13.59±2mcg/dl in comparison to basal time13.91±1.4 &13.76±1.9mcg/dl respectively, p<0.05. In comparison, there was a significant increase in serum Cortisol ug/dl in group (NL)&group (VL) at ten minutes13.79±1.22 &14.01±1.42mcg/dl in comparison to basal time13.49±1.07 & 13.66±1.52mcg/dlrespectively, p<0.05. (Table 1).

As regards the heart rate changes before and after laryngoscopy and intubation, there were significant reductions in heart rate in group VD in comparison to other groups at 3, 5,7,10 minutespost- intubation (P<0.05 for each) and at seven and 10 minutes, it was found that there were significant lower heart rates in group ND in comparison to group VL (P<0.05)(Table 2 and Figure 2).

As regardsthe mean arterial pressure changes before and after laryngoscopy and intubation,

there were significant reductions in Mean arterial pressure in group VD in comparison to other groups at 3,5,7,10 minutespost-intubation (P<0.05 for each), also, at seven and 10 minutes, it was found that there was significant lower mean arterial pressure in group ND in comparison to theNL and VL groups (p<0.05 for each) (Table 2 and Figure 3).

As regardsthe systolic blood pressure changes before and after laryngoscopy and intubation, there were significant reductions in the systolic blood pressure of group VD in comparison to other groups at 3,5,7,10 minutespost-intubation (P<0.05 for each), also at seven and 10 minutes, the systolic blood pressure was observed to be significantly lower in group ND compared to groups NL and VL (P<0.05 for each) (Figure 4).

As regards the diastolic blood pressure changes before and after laryngoscopy and intubation, there were significant reductions of the diastolic blood pressure in group VD in comparison to other groups at 3,5,7,10 minutespost-intubation (P<0.05 for each), also at three, five, seven and 10 minutes, it was found that there was significant lower diastolic blood pressure in group ND compared to group NL (P<0.05) (Figure 5).Worth mentioning that there were no reported clinically significant complications in the first 24 hours postoperative.

Figure (1): Flow chart.

Figure (2): Mean Heart rate (beat/min), before induction of anesthesia, at 1,3,5,7 and10 minutes after endotracheal intubation for studied groups.

Figure (3): Mean arterial pressure (MAP: mmHg), before induction of anesthesia, at 1,3,5,7 and10 minutes after endotracheal intubation for studied groups.

Figure (4) Mean systolic blood pressure (SBP: mmHg), before induction of anesthesia, at 1,3,5,7 and10 minutes after endotracheal intubation for studied groups.

Figure (5) Mean diastolic blood pressure (DBP: mmHg), before induction of anesthesia, at 1,3,5,7 and10 minutes after endotracheal intubation for studied groups.

Table (1): Demographic data of the studied groups and Serum Cortisol ug/dl, before induction of anesthesia, and 10 minutes after endotracheal intubation for studied groups:

Variables	Studied Groups				f	p-value
	Group ND n=23	Group NL n=23	Group VD n=23	Group VL n=23		
Age (year) Mean ± SD (range)	32.26±8.66 (18-49)	37.30±11.49 (22-50)	36.82±8.75 (23-45)	38.91±8.22 (22-50)	0.592	0.622
BMI (kg/m^2) Mean ± SD (range)	25.51±2.29 21.7-30	24.38±2.76 18.9-30.2	24.44±3.14 21.4-29.3	25.81±2.65 21.6-29.9	1.688	0.180
	n.(%)	n.(%)	n.(%)	n.(%)	χ^2	p-value
Sex						
Females	9(39.1)	11(47.8)	12(52.2)	10(43.5)	0.876	0.831
Males	14(60.9)	12(52.2)	11(47. 8)	13(56.5)		
ASA I II	16(69.6) 7(30.4)	15(65.2) 8(34.8)	13(56.5) 10(43.5)	14(60.9) 9(39.1)	0.933	0.817
Mallampati I II	16(69.6) 7(30.4)	15(65.2) 8(34.8)	14(60.9) 9(39.1)	11(47.8) 12(52.2)	2.556	0.465

Variables		Studied Groups				f	p-value
		Group ND n=23	Group NL n=23	Group VD n=23	Group VL n=23		
Serum CORTISOL ug/dl Mean ± SD (range)	Basal time	13.76±1.9 10.4-17.3	13.49±1.07 10.8-15.8	13.91±1.4 11.2-16.1	13.66±1.52 10.5-15.6	0.331	0.803
	10 minutes after intubation	13.59±2 9.7-17.2	13.79±1.22 11.1-16.1	13.35±1.7 10.5-15.8	14.01±1.42 10.7-15.8	0.715	0.545
	Paired t p	1.37 0.184	**2.4 0.023**	**3.61 .002**	**6.15 0.0001**		

BMI: body mass index. ASA: The American Society of Anesthesiologists Data were expressed as number and percent, or mean ±SD [SD=standard deviation, range, f= anova test, χ 2 Chisquare test , f= anova test, paired t compare Cortisol within group basal & 10 minute after .p>0.05 was considered no significant, p<0.05 was considered significant

Table (2): Heart rate (HR: beat/min) and Mean Arterial Pressure (MAP: mmHg), before induction of anesthesia, at 1,3,5,7 and10 minutes after endotracheal intubation for studied groups

	Studied Groups				f	p	P2	P3	P5	P6
	Group ND n=23	Group NL n=23	Group VD n=23	Group VL n=23						
HR basal	88±14.13 70-113	88.26±12.58 68-106	91.26±13.79 70-116	87.44±8.79 73-105	.433	.730	-	-	-	-
HR min1	87.17±13.15 70-110	87.78±12.79 68-103	81.61±8.05 64-97	87.44±8.91 73-105	1.652	.183	-	-	-	-
HR min3	80.91±15.85 66-107	84.39±13.21 67-103	76.61±8.05 59-92	86.35±8.099 72-100	3.024	.034	0.21	0.57	0.122	0.028
	P1=0.32		P4=0.006							
HR min5	78.04±14.53 64-105	82.22±12.66 65-101	72.61±8.05 55-88	83.61±7.19 70-95	4.607	.005	.09	.67	0.091	0.004
	P1=0.22		0.001							
HR min7	74.17±13.45 61-97	79.13±11.89 62-97	67.30±7.58 50-82	80.39±7.38 62-90	7.473	.0001	.028	.68	0.046	0.0001
	P1=0.11		P4=0.0001							
HR min10	71.61±12.53 59-93	76.65±11.04 60-93	62.44±6.795 48-78	78.304±7.28 60-88	12.39	.0001	.002	.57	0.022	0.0001
	P1=0.082		P4=0.0001							
	Group ND n=23	**Group NL n=23**	**Group VD n=23**	**Group VL n=23**	**f**	**p**	**P2**	**P3**	**P5**	**P6**
MAP Basal	81.22±3.95 73-87	80.96±4.39 73-87	80.65±4.04 73-86	80.695±2.88 75-85	.106	.956				
MAP min1	80.44±3.62 73-85	80.65±4.24 73-87	77.57±3.63 71-83	80.61±2.74 75-85	4.017	.010	.008	.96	٠,٨٧	٠,٠٠٥
	P1=0.83		P4=0.005							
MAP min3	76.74±3.57 70-82	77.65±4.24 70-84	71.61±3.23 65-78	77.74±2.68 72-82	16.218	.0001	.0001	.933	٠,٣٣	٠,٠٠٠١
	P1=0.37		P4=0.0001							
MAP min5	73.78±3.37 68-80	75.48±4.21 67-81	68.39±4.43 62-75	75.78±3.23 70-82	18.265	.0001	.0001	.789	٠,٠٨١	٠,٠٠٠١
	P1=0.138		P4=0.0001							
MAP min7	70.09±2.73 65-76	72.83±4.24 64-78	65.87±4.04 60-72	72.35±2.76 67-77	18.809	.0001	.0001	0.64	٠,٠٣٢	.0001

	Studied Groups				f	p	P2	P3	P5	P6
	Group ND n=23	Group NL n=23	Group VD n=23	Group VL n=23						
	P1=0.01		P4=0.0001							
MAP min10	67.22±2.47 63-71	70.35±3.90 62-76	59.52±3.42 53-66	70.44±2.81 65-75	59.127	.0001	.0001	0.92	.001	.0001
	P1=0.001		P4=0.0001							

P1 compare: group (ND)& (NL). P2 compare: group (ND)& (VD)

P3 compare: group (NL)& (VL) .P4 compare: group (VD)& (VL)

P5 compare group (ND)& (VL) .P6 compare: group (NL)& (VD)

Data were expressed as Mean ±SD [SD=standard deviation, range,

f= anova test, p>0.05 was considered no significant, p<0.05 was considered significant,

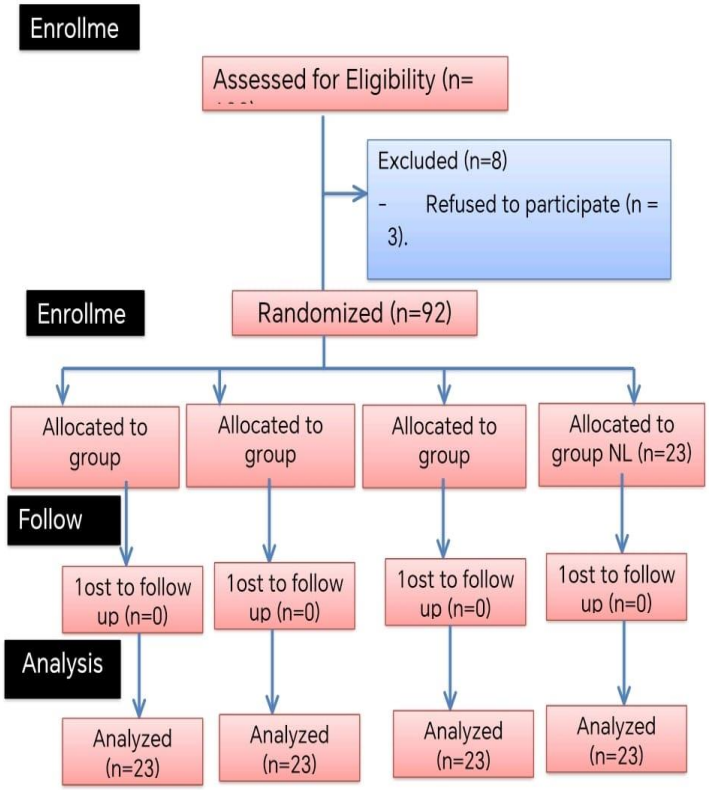

Figure (1): Study flow chart

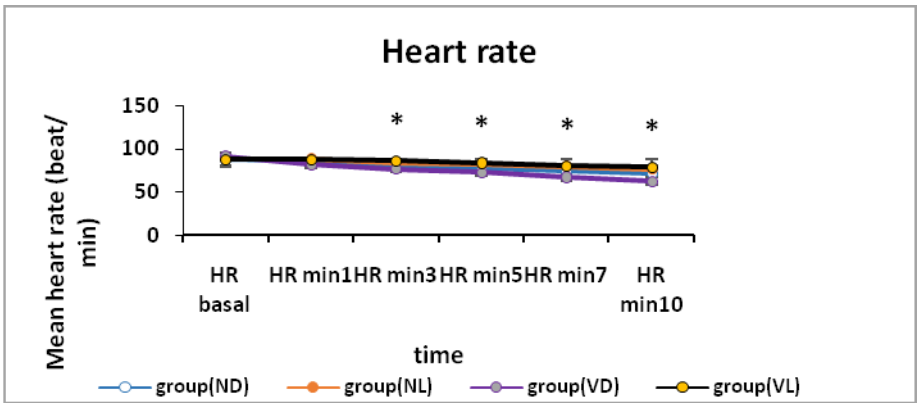

Figure (2): Mean Heart rate (beat/min), before induction of anesthesia, at 1,3,5,7 and10 minutes after endotracheal intubation for studied groups. (* significant)

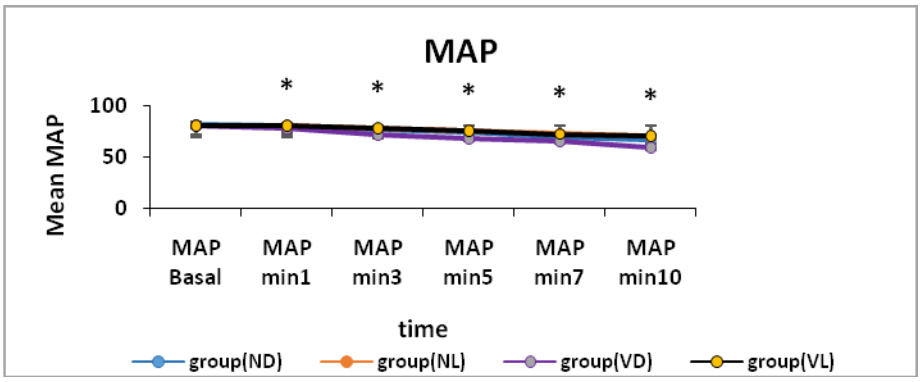

Figure (3): Mean arterial pressure (MAP: mmHg), before induction of anesthesia, at 1,3,5,7 and10 minutes after endotracheal intubation for studied groups. (* significant)

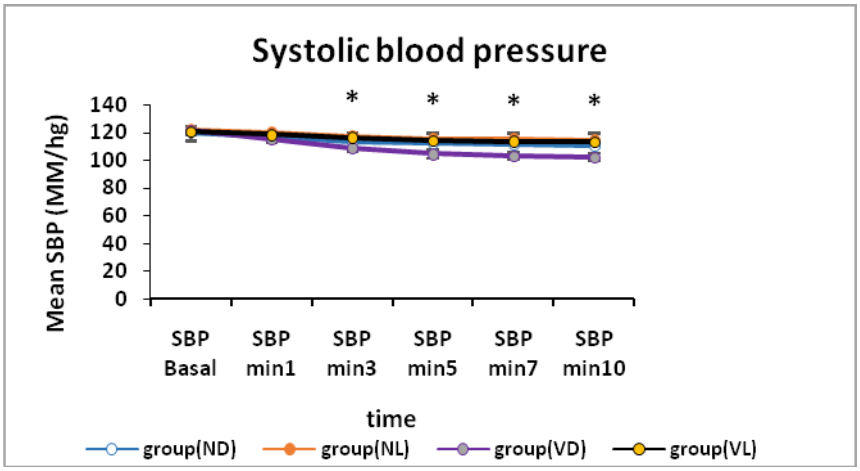

Figure (4) Mean systolic blood pressure (SBP: mmHg), before induction of anesthesia, at 1,3,5,7 and10 minutes after endotracheal intubation for studied groups. (* significant)

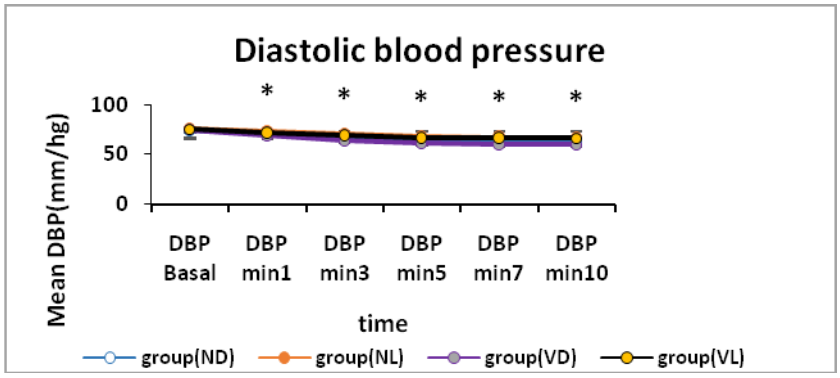

Figure (5) Mean diastolic blood pressure (DBP: mmHg), before induction of anesthesia,at 1,3,5,7 and10 minutes after endotracheal intubation for studied groups. (* significant)

DISCUSSION

Sympathetic reactions occur during laryngoscopy and intubation. Immediately upon intubation, these short-lived reactions manifest as a rise in blood pressure and heart rate. The release of extra catecholamines into the bloodstream causes these reactions. Sympathetic responses to laryngoscopy and intubation can be mitigated using drugs like fentanyl, esmolol, lidocaine and α2-agonists like dexmedetomidine and clonidine[6].Many individuals are put at risk for fatalities due to hemodynamic alterations, which might include cardiac ischemia, abrupt heart failure and cerebrovascular insults[7].This study was carried out to compare the effects of intravenous versus nebulized, either lidocaine or dexmedetomidine, on attenuation of the hemodynamic responses that follow laryngoscopy and endotracheal intubation.

Regarding serum cortisol levels before induction of anesthesia and at 10 min after endotracheal intubation for studied groups, the results showed that there was a significant decrease of the serum Cortisol ug/dlin group (VD) and group (ND) atthe 10^{th}minute compared to basal time. There is a significant increase in serum Cortisol ug/dl in group (NL)&group (VL) atthe 10^{th} minute compared to basal time. This indicated that stress hormonal response to laryngoscopy and intubation was better controlled in groups (VD) and (ND).

In agreement with this study, Patients having cardiac valve replacement were randomly assigned to either a control or Dex group in research by Wu et al. [8].Before anesthesia, the Dex group got 1.0 mcg/kg of Dex intravenously for 10 minutes, followed by 0.5 mcg/kg/h; the Control group received saline at the same rate as the Dex group.Cortisol, adrenaline, norepinephrine and serotonin (5-HT) levels in the plasma were measured at four points in time: immediately before injection (T0), immediately after sawing the sternum (T1), after the extracorporeal

circulation (T2) and 24 hours later (T3). The results showed that the Dex group had a significantly lower cortisol, adrenaline, norepinephrine and 5-HT increase than the control group.

Regarding heart rate changes, we showed significantly lower heart rates in group VD compared to group VL and group NL at the 3rd, 5th, 7th, and 10th minutes. Still, we didn't require intervention and it was clinically in significant. There were significantly lower heart rates in group VD compared to group ND.

The results of this study coincided with a study by Sriramka et al. [1].Before undergoing laryngoscopy, patients in Group DL were given IV Dexmedetomidine (1 microgram per kg-1) and nebulizedlidocaine 4% (3 mg kg^{-1}). Dexmedetomidine was administered intravenously to group D (1 microgram per kg), while lidocaine 4% was administered via nebulization to group L (3 mg kg^{-1}).After intubation, groups D and L saw a marginal rise in mean HR. However, the HR was lower in the DL group. Group DL had the best control over HR fluctuations after intubation and up to 3 minutes compared to groups D and L (P 0.01). Group D and L were equally active in HR at 5, 7 and 10 minutes, but Group D performed better. In agreement with this study by Mahjoubifard et al. [5], ninety cardiac surgery patients aged 30 to 70 participated. There were three distinct sets of participants. Dexmedetomidine (group D) received 1 mcg/kg IV, lidocaine (group L) received 1.5 mg/kg (1 %) intravenous 90 seconds before intubation and fentanyl (group F) received 2 mcg/kg IV. Pre-intubation and 1-, 3-, 5- and 10-minute post-intubation readings for HR, SBP, DBP and MAP were taken. The data showed that compared to the other groups, IV Dexmedetomidine significantly reduced HR in the third, fifth and tenth minutes after intervention. Another agreement with this studyis a study conducted by Rashmi & Kulama [9],where patients undergoing thyroid surgery were studied to see whether intravenous dexmedetomidine 0.6 microgram per kilogram affected their hemodynamic response to laryngoscopy and endotracheal intubation. At 1 minute, 5 minutes and 10 minutes after intubation, the mean HR in the dexamethasone group significantly decreased compared to the other group.

In accordance with this study, Singh et al.[10],who randomized, compared intravenous dexmedetomidine (1microgram per kilogram over 10 minutes) and nebulized dexmedetomidine (1 microgram per kilogram in 3 - 4 mL of 0.9 % saline), administered 30 minutes before induction of anesthesia, in 120 adult patients undergoing elective operations requiring tracheal intubation. There was no discernible variation in hemodynamics across the groups until three minutes had passed. After that, there was a noticeable split because the intravenous group's heart rates dropped more dramatically in the intravenous group.

As regards the mean arterial pressure changes before and after laryngoscopy and intubation, there were significant reductions in Mean arterial pressure in group VD in comparison to other groups at 3,5,7,10 minutespost-intubation (P<0.05 for each). Also, at seven and 10 minutes, it was found that there was significant lower mean arterial pressure in group ND in comparison to the NL and VL groups (p<0.05 for each).In agreement with thisstudy by Sriramka et al. [1]post-intubation, group DL maintained better MAP control than groups D and L until minute 10.However, group L had not gained ground by the tenth minute (P value 0.221). Group D exhibited superior MAP control than Group L until 3 minutes (P 0.001). Still, there was no statistically significant difference in performance between groups D and L at the 5th and 7th minutes (P value 0.730, 0.978). At 10 minutes, group L had a significantly lower mean arterial pressure (MAP) than group D (P <0.001).

As regards the systolic blood pressure changes before and after laryngoscopy and intubation, there were significant reductions in the systolic blood pressure of group VD in comparison to other groups at 3,5,7 and 10 minutespost-intubation (P<0.05 for each), also at seven and 10 minutes, the systolic blood pressure was observed to be significantly lower in group ND compared to groups NL and VL (P<0.05 for each).

Following this study, a study by Mahjoubifardet al. [5] In the first, third, fifth and tenth minutes after administration, dexmedetomidine decreased systolic blood pressure. This one was noticeably larger compared to the decreases seen in the 5th and 10th minutes. In both the lidocaine and fentanyl groups, the average patient's systolic blood pressure increased slightly.

In another study carried out by Jokar et al. [11],one hundred patients between the ages of 18 and 65 who were scheduled to have elective surgery under general anesthesia were split evenly between two groups: those who would receive intravenous (IV) lignocaine (IVL) and those who would receive nebulized (NBL) lignocaine (NL). They revealed a significant difference in SBP between the groups at intubation. The SBP has increased significantly from the baseline in the group IVL compared to the group NL, which attained the baseline values by about the 3rd min post-intubation. At the 4th and 5th min, mean SBP was lower with NL than IVL. However, our study showed that the reduction in SBP DBP was higher in the VL group when compared to the NL group but was statically insignificant.

Limitations: The current study had limitations, includingthe subjective small sample size of cases that were included and being done in a single center.The room temperature and temperature of IV fluids were not tightly controlled. Another limitation was that we assessed the short-term outcomes only, so we suggest that future research include multicenter studies to validate our

findings, and to assess long-term outcomes accurately, studies should have a longer follow-up period.

CONCLUSIONS

Both intravenous or nebulized lidocaine and dexmedetomidine attenuated the hemodynamic response to laryngoscopy and intubation but with variable degrees withnebulized dexmedetomidine carried the best profile. While hormonal response (cortisol) to laryngoscopy was obliterated in intravenous and nebulized dexmedetomidine groups, neither nebulized nor intravenous lidocaine obliterated this response.The reductions in mean, systolic, and diastolic blood pressures and heart rate before and after laryngoscopy were more prominent in group intravenous dexmedetomidine than in other groups.

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