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Comparison of the Efficacy of Clonidine and Dexemedetomidine Infusions Administered Preoperatively for Attenuation of the Hemodynamic Response Following Laryngoscopy and Endotracheal Intubation in a Placebo-Controlled Study

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Abstract

Background:

It is well established that Laryngoscopy and Endotracheal Intubation invariably cause increase in heart rate, Blood pressure and cardiac rhythm. In this placebo controlled, randomized, double blind, multicentric, prospective study an attempt to obseve to asses & compare the efficacy of preoperative Clonidine vs Dexmeditomidine infusion in attenuating the hemodynamic response following Endotracheal intubation&Laryngoscopy in three devided groups tried.

Method:

After taking ethical comitte permission,90 patients in the age group 25-50 years of either sex,ASA Gr I&II undergoing varius abdominal surgery under general Anaesthesia were randomly allocated into three equal groups.Gr C:receiving Clonidine µgm/kg,Gr D:receiving Dexmeditomedine 1µgr. N:receiving normal saline(control).The infusion were given 20 minutes before induction of Anaesthesia over a period of 15 minutes.Tracheal intubation performed within a period of 15 seconds.HR,SBP,DBP.MAP observed only in study drug in 1,2,3,5,10 minutes after intubation.

Results:

when the preoperative baseline HR was observed no statistical significance found(p value0.0953) .After induction of Anaesthesia a significant HR reduction observed in Gr D(P value 0.032).The increase in HR during laryngoscopy & Intubation at 1,2,3,5.10 minutes after intubation, were highly significant in Gr. N, compared to Gr.C &D.

Conclusion:

From This study, it can be presumed that Clonidine or Dexmeditomidine administered intravenously before laryngoscopy and intubation effectively attenuate the hemodynamic response and Dexmeditomidine found to provide better hemodynamic stability than Clonidine.

Keywords: Laryngoscopy Intubation, Diastolic blood pressure(DBP), Meanarterial pressure(MAP), Dexmeditomidine, Clonidine

Introduction

It is well established that laryngoscopy and endotracheal intubation invariably cause haemodynamic changes associated with increased heart rate, increased blood pressure and occasional disturbance in cardiac rhythm.These hemodynamic alterations are hazardous to the patients with

hypertension, myocardial insufficiency or cerebrovascular disease. In patients with coronary artery disease it may lead to myocardial ischaemia and dysrhythmia. In hypertensive patients these exaggerated haemodynamic responses may lead to left ventricular failure, pulmonary oedema and congestive cardiac failure.

Aim & Objective

In this placebo-controlled, randomized, double-blind, unicentric, prospective study an attempt has been made to observe, assess, and compare the efficacy of preoperative clonidine and dexmedetomidine infusions in attenuating the hemodynamic response following laryngoscopy and endotracheal intubation in three groups of adult patients of either sex undergoing various elective abdominal surgeries under general anaesthesia.

Exclusion Criteria

Patients with higher Mallmpati class (III and IV), Patients on antihypertensive drugs , patients with altered liver functions and renal functions, women of reproductive age group with a history of amenorrhoea and a positive urine test for pregnancy were excluded from study.

Materials And Method

Ninety patients in the age group between 25and 50 years, of either sex, of ASA physical status I and II, undergoing various elective abdominal surgeries under general anaesthesia were randomly allocated into three equal groups (n=30):Group-C (clonidine), Group-D (dexmedetomidine), and Group-N (normal saline or control). Group-C and Group-D received infusion of clonidine 3 µg kg-1 in normal saline and dexmedetomidine 1 µg kg-1 in normal saline respectively. Group-N (control) received only normal saline infusion. The infusions were given 20 minutes before induction of anaesthesia over a period of 15 minutes. In all patients general anaesthesia were , induced with 2.5% thiopental sodium 4-5 mg kg-1 and neuromuscular blockade with Vecuronium 0.1 mg kg-1 intravenously. Randomization was achieved by closed envelopes chosen by patients prior to the procedure.

Subsequently tracheal intubation with an appropriate size endotracheal tube was performed in less than 15 seconds. Anaesthesia was maintained with 66% nitrous oxide in oxygen and Isoflurane 1%. Hemodynamic parameters (HR, SBP, DBP and MAP) were recorded before study drug infusion, after infusion, after induction, during laryngoscopy and intubation, 1, 2, 3, 5 and 10 minutes after intubation. No surgical stimulus was allowed during the study period and hemodynamic changes beyond the study period were not taken into account. At the end of surgery the patients were adequately reversed. In postoperative period the patients were monitored in the recovery room for any complications and appropriately treated if required.

Result Analysis

The results of the observations thus obtained in each group of patients were tabulated, compiled and statistically analyzed using Microsoft™ Excel™ 2007 for Mac (version 12.0), StatPlus®:Mac 2009 (version 5.8.3.8) and SPSS version 13.0. Hemodynamic parameters within group at different time intervals were compared with baseline value with repeated measures by ANOVA. A p value < 0.05 was considered as statistically significant and < 0.01 was considered as highly significant.

Demographic Variables

Table 1. Comparison of demographic variables between three study groups

Demographic Variables	Group C (n =30) (Mean ± SD)	Group D (n =30) (Mean ± SD)	Group N (n =30) (Mean ± SD)	*p* value

Volume 4, Issue 6; November-December 2021; Page No 483-493

Sex (M : F)	12 : 18	10 : 20	10 : 20	0.8237
Age (years)	38.03 ± 8.16	39.07 ± 8.39	39.13 ± 8.37	0.8487
Body weight (kg)	56.07 ± 9.68	56.90 ± 9.94	55.83 ± 8.91	0.9029
Height (cm)	161.63 ± 9.39	160.3 ± 10.39	159.63 ± 9.57	0.7043
ASA grade (I : II)	24 : 6	24 : 6	23 : 7	0.9355

All the three groups were statistically comparable with respect to sex, age, body weight, height and ASA grading. No significant differences were observed between the groups (p value > 0.05) [Table 1].

Table 2. Types of operative procedures in three study groups

Operative procedures	Group C (n =30)	Group D (n =30)	Group N (n =30)	Statistical Analysis
TAH + BSO	5	4	6	
LAVH	3	3	3	
Diagnostic laparoscopy	2	3	2	Chi-Square
Excision of tubo-ovarian mass	1	2	1	(χ^2) value
Myomectomy	1	1	2	4.6584
Cholecystectomy	8	7	8	
Laparoscopic cholecystectomy	5	3	4	*p* value
Appendicectomy	2	3	2	0.9993
Laparoscopic appendicectomy	2	1	1	

Volume 4, Issue 6; November-December 2021; Page No 483-493

Incisional hernia repair	1	3	1	

TAH + BSO: Trans-abdominal hysterectomy with bilateral salpingo-öophorectomy

LAVH: Laparoscopy assisted vaginal hysterectomy

HAEMODYNAMIC PARAMETERS HEART RATE

Table 3. Comparison of heart rates between and within the study groups at different points of time

Time interval	HEART RATES (beats per minute)			*p* value
	Group C (n =30) (Mean ± SD)	Group D (n =30) (Mean ± SD)	Group N (n =30) (Mean ± SD)	
Before study drug infusion (baseline) (T1)	83.13 ± 9.24	84.03 ± 9.14	83.27 ± 9.49	0.9213
After study drug infusion (T2)	80.60 ± 8.52	79.17 ± 8.66 *	83.93 ± 8.79	0.0953
After induction of anaesthesia (T3)	78.03 ± 8.51 *	76.10 ± 8.18 **	81.80 ± 8.57	0.0320
During laryngoscopy and intubation (T4)	87.90 ± 6.98 *	83.63 ± 6.74	98.47 ± 7.77 **	< 0.0001
1 minute after intubation (T5)	93.63 ± 7.06 **	87.63 ± 7.55	107.67 ± 6.38 **	< 0.0001
2 minutes after intubation (T6)	91.43 ± 7.09 **	86.07 ± 7.32	103.13 ± 6.76 **	< 0.0001
3 minutes after intubation (T7)	84.50 ± 7.29	81.83 ± 6.72	94.70 ± 8.41 **	< 0.0001

Volume 4, Issue 6; November-December 2021; Page No 483-493

5 minutes after intubation (T8)	80.27 ± 6.48	78.00 ± 6.95 **	86.63 ± 6.63	< 0.0001
10 minutes after intubation (T9)	77.17 ± 6.69 **	76.83 ± 7.03 **	81.50 ± 7.48	0.0200

SD : standard deviation

Comparison Between Groups

When the preoperative baseline HR was compared between three groups, no statistically significant difference was found (p value 0.9213). HR was also similar in all groups after study drug infusion (p value 0.0953). After induction of anaesthesia, a significant reduction in HR was noted in Group D (p value 0.032). The increases in HR during laryngoscopy and intubation, at 1, 2, 3 and 5 minutes after intubation were highly significant in Group N compared to Group C and Group D (p value < 0.01). After 10 minutes of intubation, it was also significant in Group N (p value 0.02) [Table 3].

Comparison Within Group

Group C: The change in HR after study drug infusion was notstatistically significant (p value 0.274). But, a significant fall in HR was observed after induction of anaesthesia (p value 0.03). HR increased significantly during laryngoscopy and intubation (p value 0.0279). Highly significant rise in HR occurred at 1 and 2 minutes after intubation (p value < 0.01). Thereafter, HR decreased gradually and remained around the baseline value. No significant difference was observed at 3 and 5 minutes after intubation (p value 0.5271 and 0.1693 respectively). After 10 minutes of intubation, HR decreased further and became highly significant (p value < 0.01).

Group D: HR decreased significantly after study drug infusion comparedto the baseline value (p value 0.0385). A highly significant fall in HR occurred after induction of anaesthesia (p value < 0.01). HR remained around the baseline value during laryngoscopy and intubation, at 1, 2 and 3 minutes after intubation and no significant difference was observed (p value > 0.05). Thereafter, HR decreased again from the baseline value and became highly significant at 5 and 10 minutes after intubation (p value < 0.01).

Group N: When compared with the baseline HR, no significantdifference was noted after study drug infusion (p value 0.7788) and induction of anaesthesia (p value 0.5324). HR increased and remained persistently high during laryngoscopy and intubation, at 1, 2 and 3 minutes after intubation. Statistically highly significant values were noted throughout this period (p value < 0.01). Thereafter, HR decreased gradually and remained around the baseline value. No significant difference was observed at 5 and 10 minutes after intubation (p value 0.1166 and 0.4266 respectively).

Systolic Blood Pressure

Table 4. Comparison of systolic blood pressures between and within the study groups at different points of time

Time interval	SYSTOLIC BLOOD PRESSURE (mm of Hg)			*p* value
	Group C (n =30) (Mean ± SD)	Group D (n =30) (Mean ± SD)	Group N (n =30) (Mean ± SD)	

Volume 4, Issue 6; November-December 2021; Page No 483-493

Before study drug infusion (baseline) (T1)	121.60 ± 11.76	122.47 ± 12.22	120.63 ± 12.37	0.8433
After study drug infusion (T2)	115.77 ± 10.93	109.70 ± 11.97 **	118.57 ± 11.12	0.0104
After induction of anaesthesia (T3)	106.87 ± 10.76 **	101.33 ± 10.96 **	112.83 ± 11.37 *	< 0.0001
During laryngoscopy and intubation (T4)	123.33 ± 9.69	118.77 ± 8.15	137.60 ± 8.05 **	< 0.0001
1 minute after intubation (T5)	130.77 ± 8.11 **	123.50 ± 9.10	148.00 ± 7.60 **	< 0.0001
2 minutes after intubation (T6)	127.47 ± 8.69 *	122.53 ± 8.02	142.47 ± 7.52 **	< 0.0001
3 minutes after intubation (T7)	117.67 ± 9.66	113.73 ± 8.51 **	130.23 ± 7.97 **	< 0.0001
5 minutes after intubation (T8)	110.53 ± 9.84 **	110.30 ± 8.66 **	119.97 ± 7.87	< 0.0001
10 minutes after intubation (T9)	107.73 ± 9.36 **	109.07 ± 8.85 **	113.47 ± 8.44 *	0.0363

SD : standard deviation

Comparison Between Groups

When the preoperative baseline SBP was compared between three groups, no statistically significant difference was found (p value 0.8433). After study drug infusion, a significant reduction in SBP was noted in Group D (p value 0.0104). After induction of anaesthesia, this reduction in SBP became highly significant in Group D (p value < 0.01). The increases in SBP during laryngoscopy and intubation, at 1, 2, 3 and 5 minutes after intubation were highly significant in Group N compared to Group C and Group D (p value < 0.01). After 10 minutes of intubation, it was also significant in Group N (p value 0.0363) [Table 4].

Volume 4, Issue 6; November-December 2021; Page No 483-493

Comparison Within Group

Group C: The change in SBP after study drug infusion was notstatistically significant (p value 0.0514). But, a highly significant fall in SBP was observed after induction of anaesthesia (p value < 0.01). SBP increased during laryngoscopy and intubation but it was statistically insignificant (p value 0.5358). The increase in SBP was highly significant at 1 minute after intubation (p value < 0.01) and significant at 2 minutes after intubation (p value 0.032). Thereafter, SBP decreased near the baseline value and no significant difference was observed at 3 minutes after intubation (p value 0.1623). At 5 and 10 minutes after intubation, SBP decreased further and became highly significant (p value < 0.01).

Group D: SBP decreased from the baseline value after study druginfusion and induction of anaesthesia, which was highly significant (p value < 0.01). SBP remained around the baseline value during laryngoscopy and intubation, at 1 and 2 minutes after intubation and no significant difference was observed (p value > 0.05). Thereafter, SBP decreased again from the baseline value and became highly significant at 3, 5 and 10 minutes after intubation (p value < 0.01).

Group N: When compared with the baseline SBP, no significantdifference was noted after study drug infusion (p value 0.4988). A significant fall in SBP occurred after induction of anaesthesia (p value 0.0137). SBP increased and remained persistently high during laryngoscopy and intubation, at 1, 2 and 3 minutes after intubation. Statistically highly significant values were noted throughout this period (p value < 0.01). Thereafter, SBP decreased near the baseline value and no significant difference was observed at 5 minutes after intubation (p value 0.8041). At 10 minutes after intubation, SBP decreased significantly from the baseline value (p value < 0.0111)

Diastolic Blood Pressure

Table 5. Comparison of diastolic blood pressures between and within the study groupsat different points of time

Time interval	DIASTOLIC BLOOD PRESSURE (mm of Hg)			*p* value
	Group C (n =30) (Mean ± SD)	Group D (n =30) (Mean ± SD)	Group N (n =30) (Mean ± SD)	
Before study drug infusion (baseline) (T1)	80.83 ± 9.44	79.73 ± 9.47	79.27 ± 9.67	0.8082
After study drug infusion (T2)	75.67 ± 9.09 *	72.10 ± 8.25 **	78.00 ± 9.17	0.0374
After induction of anaesthesia (T3)	69.97 ± 8.36 **	67.67 ± 7.69 **	73.53 ± 9.46 *	0.0307
During laryngoscopy and intubation (T4)	82.60 ± 9.07	78.37 ± 7.42	87.83 ± 6.65 **	< 0.0001

Volume 4, Issue 6; November-December 2021; Page No 483-493

1 minute after intubation (T5)	86.37 ± 9.21 *	81.83 ± 7.55	95.87 ± 7.21 **	< 0.0001
2 minutes after intubation (T6)	85.00 ± 8.88	80.47 ± 7.15	92.33 ± 6.79 **	< 0.0001
3 minutes after intubation (T7)	78.67 ± 9.30	75.93 ± 7.58	83.47 ± 7.21	0.0019
5 minutes after intubation (T8)	74.17 ± 9.24 **	73.13 ± 7.41 **	78.83 ± 6.52	0.0129
10 minutes after intubation (T9)	71.20 ± 9.08 **	72.37 ± 7.88 **	73.23 ± 7.27 **	0.6217

SD : standard deviation

Comparison Between Groups

When the preoperative baseline DBP was compared between three groups, no statistically significant difference was found (p value 0.8082). Significant reductions in DBP were noted in Group D after study drug infusion (p value 0.0374) and after induction of anaesthesia (p value 0.0307). The increases in DBP during laryngoscopy and intubation, at 1, 2, and 3 minutes after intubation were highly significant in Group N compared to Group C and Group D (p value < 0.01). After 5 minutes of intubation, it was also significant in Group N (p value 0.0129). DBP became similar in all groups after 10 minutes of intubation (p value 0.6217) [Table 5].

Comparison Within Group

Group C: DBP decreased from the baseline value initially, which wasstatistically significant after study drug infusion (p value 0.0349) and highly significant after induction of anaesthesia (p value < 0.01). DBP increased during laryngoscopy and intubation but it was statistically insignificant (p value 0.4626). The increase in DBP was statistically significant at 1 minute after intubation (p value 0.0251). Thereafter, DBP decreased gradually and remained around the baseline value. No significant difference was observed at 2 and 3 minutes after intubation (p value 0.0835 and 0.3741 respectively). At 5 and 10 minutes after intubation, DBP decreased further and became highly significant (p value < 0.01).

Group D: DBP decreased from the baseline value after study druginfusion and induction of anaesthesia, which was highly significant (p value < 0.01). DBP remained around the baseline value during laryngoscopy and intubation, at 1, 2 and 3 minutes after intubation and no significant difference was observed (p value > 0.05). Thereafter, DBP decreased again from the baseline value and became highly significant at 5 and 10 minutes after intubation (p value < 0.01).

Group N: When compared with the baseline DBP, no significantdifference was noted after study drug infusion (p value 0.6045). A significant fall in DBP occurred after induction of anaesthesia (p value 0.0238). DBP increased and remained persistently high during laryngoscopy and intubation, at 1 and 2 minutes after intubation. Statistically highly significant values were noted throughout this period

Volume 4, Issue 6; November-December 2021; Page No 483-493

(p value < 0.01). Thereafter, DBP decreased gradually and remained around the baseline value. No significant difference was observed at 3 and 5 minutes after intubation (p value 0.0615 and 0.8394 respectively). At 10 minutes after intubation, DBP decreased further and became highly significant (p value < 0.01).

Mean Arterial Pressure

Table 6. Comparison of mean arterial pressures between and within the study groups at different points of time

Time interval	MEAN ARTERIAL PRESSURE (mm of Hg)			*p* value
	Group C (n =30) (Mean ± SD)	Group D (n =30) (Mean ± SD)	Group N (n =30) (Mean ± SD)	
Before study drug infusion (baseline) (T1)	94.33 ± 10.19	93.90 ± 10.33	93.07 ± 10.57	0.8923
After study drug infusion (T2)	89.07 ± 9.59 *	84.70 ± 9.41 **	91.43 ± 9.66	0.0246
After induction of anaesthesia (T3)	82.03 ± 9.14 **	78.90 ± 8.68 **	86.67 ± 10.09 *	0.0069
During laryngoscopy and intubation (T4)	96.10 ± 9.11	91.83 ± 7.49	104.47 ± 6.88 **	< 0.0001
1 minute after intubation (T5)	101.13 ± 8.51 **	95.80 ± 7.91	113.20 ± 7.18 **	< 0.0001
2 minutes after intubation (T6)	99.23 ± 8.63 *	94.50 ± 7.33	109.07 ± 6.81 **	< 0.0001
3 minutes after intubation (T7)	91.73 ± 9.25	88.57 ± 7.61 *	99.00 ± 7.33 *	< 0.0001
5 minutes after	86.30 ± 9.21 **	85.50 ± 7.51	92.50 ± 6.80	0.0014

Volume 4, Issue 6; November-December 2021; Page No 483-493

intubation (T8)		**		
10 minutes after intubation (T9)	83.37 ± 8.98 **	84.60 ± 7.93 **	86.63 ± 7.58 **	0.2971

SD : standard deviation

Comparison Between Groups

When the preoperative baseline MAP was compared between three groups, no statistically significant difference was found (p value 0.8923). After study drug infusion, a significant reduction in MAP was noted in Group D (p value 0.0246). After induction of anaesthesia, this reduction in MAP became highly significant in Group D (p value < 0.0069). The increases in MAP during laryngoscopy and intubation, at 1, 2, 3 and 5 minutes after intubation were highly significant in Group N compared to Group C and Group D (p value < 0.01). MAP became similar in all groups after 10 minutes of intubation (p value 0.2971) [Table 6].

Comparison Within Group

Group C: MAP decreased from the baseline value initially, which wasstatistically significant after study drug infusion (p value 0.0437) and highly significant after induction of anaesthesia (p value < 0.01). MAP increased during laryngoscopy and intubation but it was statistically insignificant (p value 0.4816). The increase in MAP was highly significant at 1 minute after intubation (p value < 0.01) and significant at 2 minutes after intubation (p value 0.049). Thereafter, MAP decreased near the baseline value and no significant difference was observed at 3 minutes after intubation (p value 0.3051). At 5 and 10 minutes after intubation, MAP decreased further and became highly significant. (p value < 0.01).

Group D: MAP decreased from the baseline value after study druginfusion and induction of anaesthesia, which was highly significant (p value < 0.01). MAP remained around the baseline value during laryngoscopy and intubation, at 1 and 2 minutes after intubation and no significant difference was observed (p value > 0.05). Thereafter, MAP decreased again from the baseline value and became significant at 3 minutes after intubation (p value 0.0265) and highly significant at 5 and 10 minutes after intubation (p value < 0.01).

Group N: When compared with the baseline MAP, no significantdifference was noted after study drug infusion (p value 0.5345). A significant fall in MAP occurred after induction of anaesthesia (p value 0.0197). MAP increased and remained persistently high during laryngoscopy and intubation.

Conclusion

From these observations and analysis of the present study, it can be inferred that both clonidine and dexmedetomidine administered intravenously just before laryngoscopy and endotracheal intubation effectively attenuated the hemodynamic response by limiting the extent of rises in heart rate and blood pressure. Dexmedetomidine has been found to provide better hemodynamic stability than clonidine. Both the α2-agonists are devoid of any serious adverse effect and found safe in this study.

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Volume 4, Issue 6; November-December 2021; Page No 483-493

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Volume 4, Issue 6; November-December 2021; Page No 483-493