International Journal of Medical Research Professionals
P-ISSN: 2454-6356; E-ISSN: 2454-6364
DOI: 10.21276/ijmrp

IJMRP

Original Research Article.

Reduction in Dose of Induction Agent Propofol and Hemodynamics by A2 Agonists Dexmedetomidine Versus Clonidine in Patients Undergoing Elective Surgeries Under General Anaesthesia: A Prospective Randomised Double Blind Clinical Trial

Balwinder Kaur[1], Mandeep Kaur[2*], Sahil Garg[3]

[1]Professor, [2,3]Assistant Professor,
Department of Anaesthesia & Critical Care, Government Medical College, Patiala, Punjab, India.

ABSTRACT

Background: The use of α2 adrenoreceptor agonists, dexmedetomidine and clonidine as an anesthetic adjuvant is well documented in promoting haemodynamic stability and decreasing induction dose of i.v. anesthetics, intraoperative analgesic and volatile anaesthetic requirement for maintenance of anaesthesia. This study aims to compare the effects of Dexmedetomidine (0.5 µg/kg) and Clonidine (0.5 µg/kg) on effect on anaesthetic requirements, haemodynamic responses to endotracheal intubation, and effect on sedation.

Materials And Methods: In this prospective, randomised, clinical trial, 100 patients of either sex, aged 20 - 60 years of ASA grade I and II scheduled for elective surgeries under general anaesthesia were randomly divided into two groups Group D (Inj. Dexmedetomidine dose 0.5 µg/kg IV in 100 mL normal saline) and Group C (Inj. Clonidine dose 0.5 µg/kg IV in 100 mL normal saline). Haemodynamic parameters (HR, SBP, DBP, MAP, SpO2) were monitored continuously and recorded before the start of infusion, at the start of infusion, at 5 mins of start of infusion, at 10 mins of start of infusion, at intubation and then at 1, 3, 5 and 10 minutes after intubation.

Results: The mean dose of propofol used for induction in Group D was 54.800 ± 7.068 mg and in Group C was 67.200 ± 12.296 mg. There was statistically highly significant (p= 0.001) reduction in dose required for induction in Group D than in Group C.

Conclusion: The prior administration of α2 agonist especially Dexmedetomidine, not only decreases the sympathetic response of laryngoscopy and intubation but decreases the dose requirement of intravenous induction agent as well as intraoperative inhalational requirement.

Keywords: Dexmedetomidine; Clonidine; Reduction in Dose; Propofol; Haemodynamic; Laryngoscopy.

***Correspondence to:**

Dr. Mandeep Kaur,
Assistant Professor,
Department of Anaesthesia & Critical Care,
Government Medical College, Patiala, Punjab, India.

Article History:

Received: 11-06-2022, **Revised:** 04-07-2022, **Accepted:** 29-07-2022

Access this article online	
Website: www.ijmrp.com	Quick Response code
DOI: 10.21276/ijmrp.2022.8.4.008	

INTRODUCTION

General anaesthesia is a drug-induced reversible condition composed of four behavioral and physiologic states: Unconsciousness, amnesia, analgesia, immobility and stability of the physiologic systems including the autonomic, cardiovascular, respiratory and thermoregulatory systems.[1] Induction is a critical phase of general anaesthesia.[2]

Anaesthesia induction is commonly initiated by intravenous administration of hypnotics for abruptly bringing wakeful patients into unresponsiveness to strong adrenergic stimuli including endotracheal intubation and surgical procedures.[3] The use of α2 adrenoreceptor agonists, dexmedetomidine and clonidine as an anesthetic adjuvant is well documented in promoting haemodynamic stability and decreasing induction dose of i.v. anesthetics, intraoperative analgesic and volatile anaesthetic requirement for maintenance of anaesthesia.[4,5] Laryngoscopy and tracheal intubation after the induction of anaesthesia are nearly always associated with a sympathetic hyperactivity. To attenuate the pressor response various drugs have been tried, but these drugs were either partially effective or they produced undesirable effects.[6-9] α 2 – adrenoceptors are members of the G – protein – coupled family of transmembrane receptors, which are present in the central and peripheral nervous system at both pre- and post – synaptic autonomic ganglia. Binding of endogenous agonists (e.g. norepinephrine) or exogenous agonist (e.g. clonidine) results in G-protein coupling with the inhibition of both adenylyl cyclase and phospholipase C activity. Both dexmedetomidine and clonidine are imidazoline compounds. Dexmedetomidine displays an α2 : α1 selectivity of 1600:1 , which is eight times greater than that of

clonidine, has an elimination half-life, which is 4 times less and distribution half-life, which is 2 times less than clonidine making dexmedetomidine more desirable.[6]

Adrenergic receptors have attracted increasing interest as an adjuvant to anaesthesia. Clinical studies with clonidine have demonstrated, among other effects, reduced anaesthetic requirements and improved cardiovascular and adrenergic stability during surgery.[7]

MATERIALS AND METHODS

This prospective randomised double blind clinical trial was carried out after obtaining ethics committee clearance as well as written informed consent from all patients. 100 patients of either sex, aged 20 - 60 years of ASA grade I and II scheduled for elective surgeries under general anaesthesia at Govt. Medical College, Rajindra Hospital, Patiala were included. The patients were randomly divided into 2 groups (Group D and Group C) of 50 each by simple randomisation was done using lottery method. (Group D received dexmedetomidine and Group C received clonidine).

Inclusion criteria were age 20 - 60 years, ASA grade I and II, elective surgical procedure under general anaesthesia, Mallampati grade 1 and 2, patient willing to participate in this study. Exclusion criteria were patient refusal, history of bradycardia (Heart Rate < 50 bpm), history of renal or liver dysfunction, history of previous cerebrovascular accident, history of coronary artery disease, pregnant and lactating patients.

A written informed consent was obtained from each patient after explaining the anaesthetic technique prior to inclusion in this study in their own vernacular language. Patients were randomly divided into 2 groups (Group D and Group C) of 50 each. Group D patients received 0.5 µg/kg of IV Dexmedetomidine in 100 mL normal saline infused over 10 mins before laryngoscopy and intubation. Group C patients received 0.5 µg/kg of IV Clonidine in 100 mL normal saline infused over 10 mins before laryngoscopy and intubation.

A complete protocol of pre-anaesthetic check-up was followed, and relevant routine investigations were done. Each patient was kept fasting for at least six hours pre-operative and tablet lorazepam 1mg at 6 am on the day of surgery was given as premedicant.

After routine check-up of anaesthesia machine, circuit and resuscitation equipment, fasting patients were shifted to OT and were connected to multichannel monitor. Two IV lines were secured with 18-G cannula and preloading with 500 mL ringer lactate was done over 30 mins for all the patients. Basal Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), Heart Rate (HR) and SpO2 were recorded after 5 mins of settling in OT (T0). Rhythm monitoring from a continuous visual display of ECG along with continuous monitoring of the vital parameters was done.

Following this, patients of Group D received IV dexmedetomidine 0.5 µg/kg in 100 mL NS to be infused over 10 mins. Patients of Group C received IV clonidine 0.5 µg/kg in 100 mL NS to be infused over 10 mins. HR, SBP, DBP, MAP and SpO2 were monitored continuously, but recorded/documented at the start of infusion (T1) at 5 mins of start of infusion (T2) and at 10 mins of start of infusion i.e. completion of infusion (T3) in both groups.

Prior to induction Inj. Glycopyrrolate 0.2 mg, inj Butorphenol 2 mg, inj Ondansetron 4 mg and inj. Ranitidine 50 mg was given intravenously. After pre-oxygenation with 100% oxygen, all patients were induced with IV anaesthetic agent propofol and inhalational agent isoflurane. The dose of propofol was controlled by loss of phonation followed by succinylcholine 2 mg/kg to facilitate endotracheal intubation. Patients were intubated with an appropriately sized, orally cuffed, disposable endotracheal tube.

Anaesthesia was maintained with intermittent positive pressure ventilation using Bain's circuit with appropriate mixture of N2O and O2, Isoflurane and using Inj. vecuronium bromide 0.08 mg/kg to 0.1 mg/kg IV bolus followed by maintenance dose 1/4th of the initial dose depending upon requirement. Cardiovascular parameters (HR, SBP, DBP, MAP, SpO2, EtCo2) were recorded during laryngoscopy and intubation (T4) and at 1, 3, 5 and 10 mins after laryngoscopy and intubation (T5 to T8) and then after every 10 mins interval intraoperative till the end of surgery.

Table 1: Response to Vital Parameters HR, SBP, DBP, MAP, SpO2, EtCo2 during Infusion of Dexmedetomidine/ Clonidine

Basal reading after 5 mins of patient being shifted to OT	T0
At the start of infusion of dexmedetomidine/ clonidine	T1
At 5 mins after infusion of dexmedetomidine/ clonidine	T2
At 10 mins/ completion after infusion of dexmedetomidine/ clonidine	T3

Table 2: Response to Vital Parameters HR, SBP, DBP, MAP, SpO2, EtCo2 after Infusion of Dexmedetomidine/ Clonidine

During laryngoscopy and intubation	T4
At 1 min after laryngoscopy and intubation	T5
At 3 mins after laryngoscopy and intubation	T6
At 5 mins after laryngoscopy and intubation	T7
At 10 mins after laryngoscopy and intubation	T8

The concentration of isoflurane was adjusted to maintain systolic blood pressure (SBP) within 20% of the preoperative values.

At the end of surgery, neuromuscular blockade was reversed with neostigmine 50 µg/kg and glycopyrrolate 10µg/kg intravenously. After satisfying the extubation criteria, patients were extubated and transferred to post-anaesthesia care unit (PACU).

In PACU, HR, SBP, DBP, MAP, SpO2, sedation score and any incidence of complications/ adverse event was monitored for next 90 mins at interval of 10 mins. Once the patient was shifted to PACU, first reading was taken as 0 min and then after every 10 mins till 90 mins. Modified Aldrete scoring > 9 was considered criteria for shifting the patients to ward from PACU.

Fall in BP 20% below baseline was considered as hypotension and was managed appropriately. Pulse rate lower than 50 beats per minute (bpm) was regarded as bradycardia and was managed with atropine (0.3 - 0.6 mg). Fall in saturation was managed meticulously depending upon the cause. Rise or fall in EtCo2 was managed accordingly depending upon the cause.

Sedation scoring was done as per Ramsay sedation scale after completion of drug infusion. Adverse effects (hypotension, bradycardia, arrhythmia) if any were treated and recorded.

Descriptive statistics was done for all data and suitable statistical tests of comparison were done. Continuous variables were analysed with unpaired t-test and Mann- Whitney U test. Categorical variables were analysed with the Chi-square test. Statistical significance was taken as P value <0.05, statistical highly significant was taken as P value <0.001, statistical non-significant was taken as P value >0.05. The observations were depicted in tables. The data was analysed using IBMM SPSS statistics (22.00 version) and Microsoft Excel 2007.

Sample Size Calculation

Sample size was estimated based on pilot study. We see that mean difference in heart rate in 2 groups was 4.06 with SD of 7.05. With this our sample size n= 48 per group at a power of 80% and confidence interval of 95%. For possible dropouts, it was decided to include 50 patients per group.

$N= 2\sigma2 (Z1-\alpha/2 + Z1-\beta)2 / \Delta2$ where $Z1-\alpha/2$ is the critical value of the Normal distribution at $1-\alpha/2$, $Z1-\beta$ is the critical value of the Normal distribution at $1-\beta$, $\sigma2$ is the population variance and Δ is difference between 2 means.

Table 3: Demographic Parameters and Surgical Time

Variable	Group D		Group C		P value	Significance
	Mean	S.D.	Mean	S.D.		
Age (yrs.) {Unpaired T test}	41.30	10.90	41.36	11.98	0.979	NS
Sex (M/F) {Chi-square}	10/40		12/38		0.629	NS
ASA grade (I/II) {Chi-square}	26/24		25/25		0.841	NS
Body weight (kg) {Unpaired T test}	67.42	7.33	68.00	4.96	0.644	NS
Duration of surgery (mins) {Unpaired T test}	77.32	17.68	75.76	31.02	0.758	NS

NS: Not Significant; S.D: Standard Deviation

Table 4: Comparison of Changes in Mean Heart Rate

HR (bpm)	Group D		Group C		P value	Significance
	Mean	S.D.	Mean	S.D.		
T0	87.66	11.793	83.72	10.325	0.059	NS
T1	87.92	12.270	84.32	9.999	0.066	NS
T2	85.32	12.188	83.58	10.033	0.242	NS
T3	83.92	11.733	82.24	14.767	0.492	NS
T4	95.14	9.600	100.06	8.049	0.013	S
T5	88.26	8.898	93.00	7.466	0.005	HS
T6	87.92	8.231	88.98	8.193	0.451	NS
T7	86.98	8.277	86.88	7.148	0.830	NS
T8	86.46	10.448	86.08	6.442	0.849	NS

HR: Heart Rate; NS: Not Significant; S: Significant; HS: Highly Significant; S.D: Standard Deviation.

Table 5: Comparison of Changes in Systolic Blood Pressure

SBP (mmHg)	Group D		Group C		P value	Significance
	Mean	S.D	Mean	S.D		
T0	126.00	9.897	127.72	9.781	0.354	NS
T1	124.88	9.766	127.04	9.243	0.258	NS
T2	121.08	9.357	124.56	9.537	0.067	NS
T3	117.60	11.766	122.24	8.756	0.085	NS
T4	135.80	10.095	141.76	8.530	<0.001	HS
T5	124.96	9.118	131.04	7.343	<0.001	HS
T6	122.64	7.199	128.56	7.835	<0.001	HS
T7	121.32	5.563	124.96	6.184	0.003	HS
T8	120.92	6.327	124.04	5.763	0.011	S

Table 6: Comparison of Changes in Diastolic Blood Pressure

DBP (mmHg)	Group D		Group C		P value	Significance
	Mean	S.D	Mean	S.D		
T0	83.72	5.361	83.68	6.790	0.654	NS
T1	82.88	6.236	84.76	6.784	0.283	NS
T2	81.12	5.844	82.54	6.938	0.410	NS
T3	78.98	5.730	80.66	6.589	0.110	NS
T4	91.72	5.345	96.00	5.686	<0.001	HS
T5	84.56	7.451	89.28	6.151	0.001	HS
T6	81.48	6.487	85.48	12.066	0.042	S
T7	82.68	6.864	85.64	5.153	0.017	S
T8	81.38	6.746	83.96	4.857	0.031	S

DBP: Diastolic Blood Pressure; NS: Not Significant; S: Significant; HS: Highly Significant; S.D: Standard Deviation.

Table 7: Comparison of Changes in Mean Arterial Blood Pressure

MAP (mmHg)	Group D		Group C		P value	Significance
	Mean	S.D	Mean	S.D		
T0	97.81	6.283	98.36	7.090	0.882	NS
T1	96.88	6.736	98.85	6.916	0.164	NS
T2	94.44	6.135	96.55	7.239	0.143	NS
T3	91.85	6.894	94.52	6.687	0.075	NS
T4	106.41	6.058	111.25	5.720	<0.001	HS
T5	98.12	6.740	103.20	5.774	<0.001	HS
T6	95.20	5.835	99.84	8.673	0.002	HS
T7	95.56	5.820	98.74	4.478	0.003	HS
T8	94.56	5.654	97.32	4.232	0.007	HS

MAP: Mean arterial blood pressure; NS: Not Significant; HS: Highly Significant; S.D: Standard Deviation.

Table 8: Comparison of Induction Dose of Propofol and Ramsay Sedation Scale

Variable	Group D		Group C		P value	Significance
	Mean	S.D.	Mean	S.D.		
IV Propofol Induction dose (mg) {T test}	54.800	7.068	67.200	12.296	0.001	HS
Ramsay Sedation Scale	2.06	0.239	2.04	0.197	0.648	NS

Table 9: Comparison of Modified Aldrete Score

MAS	Group D		Group C		P value	Significance
	Mean	S.D.	Mean	S.D.		
0 min	9.82	.388	9.86	.351	0.587	NS
10 min	9.98	.141	10.00	0.00	0.317	NS
20 min	10.00	0.00	10.00	0.00	-	-

MAS: Modified Aldrete Score; NS: Not Significant; S.D: Standard Deviation.

OBSERVATIONS

The present study has been designed to compare the effect of dexmedetomidine and clonidine on reduction in dose of induction dose of propofol and hemodynamics in patients undergoing elective surgeries under general anaesthesia at Government Medical College, Rajindra Hospital, Patiala. The study has been conducted in 100 patients randomly divided into two groups Group D (Dexmedetomidine 0.5 µg/kg) and Group C (Clonidine 0.5 µg/kg) of 50 patients each comparable in terms of demographic parameters, ASA grading (Table 3) and baseline haemodynamic parameters

DISCUSSION

For induction of anaesthesia various i/v induction agents propofol, intimidate, ketamine, thiopentone have been in practice and propofol is most commonly used IV induction agent. Beauty of propofol is that the context sensitivity half-life is very less. Like other induction agents, propofol is not free from adverse effects e.g. hypotension, bradycardia and putting life in danger. Several studies have reported α2 agonist dexmedetomidine and propofol pharmacodynamic interaction, leading to a reduction in the propofol dosage.[8] The haemodynamic response to laryngoscopy has been a topic of discussion since 1940 and these responses

can be detrimental in elderly and haemodynamically compromised patients due to increase in arterial pressure, heart rate, dysrhythmia and oxygen consumption. Therefore, controlling this perioperative stress response is an important goal of modern anaesthesia so various nonpharmacological (smooth and gentle intubation) and pharmacological methods (pre-treatment with IV lidocaine, narcotics, topical anaesthesia, beta blockers, calcium channel blockers, ACE inhibitors, vasodilators etc.) have been tried by various authors to attenuate the cardiovascular response to laryngoscopy.[9,10]

DOSE OF ANAESTHETIC AGENT

In our study, the mean dose of propofol used for induction in Group D was 54.800 ± 7.068 mg and in Group C was 67.200 ± 12.296 mg. There was statistically highly significant (p= 0.001) reduction in dose required for induction in Group D than in Group C (Table 8). In concordance to our study Shipra Singh et al also found that the requirement of propofol was reduced after pretreatment with dexmedetomidine (57.5±9.1mg propofol) as compared to clonidine (68±6.9 mg propofol).[4]

Naz Anjum et al found that average propofol mg/kg/hr requirement was reduced by adding α2 agonist by 45% (1.94±0.44) and 40% (2.1±0.42) with simultaneous administration of clonidine (group C) and dexmedetomidine (group D) respectively with propofol, which was statistically significant compared to 3.50±0.5 propofol alone (group P). But here in contrast to our study propofol dosage is reduced more in group C most likely explanation to this is that they have used higher dose of clonidine ie 3µg/kg as compared to our study (0.5 µg/kg).[5] Amitabh et al observed a statistically significant lowering of propofol requirement by 15% (ie.0.91±0.26 mg/kg from 1.07±0.23 mg/kg) in dexmedetomidine group for induction of anaesthesia and maintenance of GA as compared to plain group. Dexmedetomidine, by its action on central nervous system, is known to reduce anesthesia requirement.[6]

Suvadeep et al also concluded that the mean induction dose of propofol is significantly decreased 48.08% in group D (66.86±12.549 mg) from group P (124±16.033 mg) when dexmedetomidine infusion was used as adjuvant in group D.[11]

From the review of literature, it has been found that prior administration of α2 agonist especially dexmedetomidine not only reduced dose of propofol but the requirement of inhalational is also reduced as discussed in the following studies.

Similarly, Norimasa Ohtani et al compared recovery profile from dexmedetomidine as a general anesthetic adjuvant in 4 groups. Group S (sevoflurane), group P (propofol), group SD (sevoflurane-dexmedetomidine) and group PD (propofol-dexmedetomidine). Dexmedetomidine reduced the anesthetic requirements required to maintain a BIS of 45 by 20-30% for both groups: decreasing sevoflurane 1.1±0.2% in group S to 0.8±0.2%in group SD, and propofol from 4.4±0.8 mg/kg/hr in group P to 3.1±1.0 mg/kg/hr in group PD.[12] Varshalli et al also found that dexmedetomidine also decreases intraoperative inhalational requirement as the average inspiratory concentration of isoflurane required during anaesthetic maintenance was 0.8% in control group (C) and 0.54% in dexmedetomidine group (D). So, a decrease of 32% was observed in group D compared to group C.[13]

In present study we also observed the same, but we did not calculate and document the reduction in the MAC and hence did not apply statistics.

HAEMODYNAMIC PARAMETERS

Heart Rate

Observations of our study demonstrated that there was increase in heart rate during laryngoscopy and intubation (T4) and after 1 min of laryngoscopy and intubation (T5) in both groups. However, magnitude of increase in heart rate at T4 and T5 was higher in Group C as compared to Group D and this was statistically significant (p= 0.013) at T4 and statistically highly significant (p= 0.005) at T5 (Table 4).

Similar to our study, Sameer Arora et al compared dexmedetomidine and clonidine to study haemodynamic responses to intubation and observed that during intubation there was rise in heart rate in both the groups. But it was more in Group C as compared to Group D and this rise in HR in Group C was statistically significant (p < 0.05) during intubation and after 1 min of intubation.[14]

Shirsendu et al compared dexmedetomidine and clonidine for attenuation of sympathoadrenal responses and anaesthetic requirements to laryngoscopy and endotracheal intubation in 60 patients divided into 3 groups of 20 patients each and demonstrated that there was statistically significant rise in heart rate during intubation in clonidine group compared to dexmedetomidine.[15]

Naz Anjum et al also found significant increase in the PR in group P (Saline) during laryngoscopy and extubation whereas a decrease was found in group C (clonidine)and group D (dexmedetomidine).[5]

These findings are in agreement with our results.

Blood Pressure/ Systolic Blood Pressure (SBP)

Our findings demonstrate that the mean SBP rises in both groups at T4 (during laryngoscopy and intubation), T5, T6, T7, T8 (1, 3, 5, 10 mins after laryngoscopy and intubation). But it rises more in Group C than Group D, which was highly statistically significant (p < 0.001) at T4, T5, T6, T7 and statistically significant (p < 0.05) at T8 between 2 groups (Table 5).

Diastolic Blood Pressure (DBP)

Mean DBP rises in both groups at T4, T5, T6, T7 and T8. But it rises more in Group C than Group D which was highly statistically significant (p < 0.001) at T4, T5 and statistically significant (p < 0.05) at T6, T7 and T8 (Table 6).

Mean Arterial Blood Pressure (MAP)

Mean Arterial Blood Pressure rises in both groups at T4, T5, T6, T7 and T8. But it rises more in Group C than Group D which was highly statistically significant at T4, T5, T6, T7, T8 (p<0.001) (Table 7).

No statistically significant differences were found in the mean systolic blood pressure, mean diastolic blood pressure and mean arterial blood pressure measurements between two groups and both groups were comparable at 20, 30, 40, 50, 60,70, 80, 90, 100, 110 and 120 mins.

Bijoy Kumar et al compared dexmedetomidine and clonidine for sympathoadrenal response and they also found comparatively more increase in SBP with clonidine than dexmedetomidine. These findings are consistent with our results.[16]

Similar to our study, Sameer Arora et al compared dexmedetomidine and clonidine to see haemodynamic responses to intubation and demonstrated that during intubation both groups had maximum rise in SBP, but this was more in Group C than in Group D which was statistically highly significant (p < 0.001).[14]

A Venkateswara et al compared dexmedetomidine and clonidine on induction, haemodynamic and cardiovascular parameters for intubation in general anaesthesia in 90 patients divided into 3 groups of 30 each and observed that after intubation, rise in SBP, DBP, MAP was present in all the 3 groups. But difference between Group NS and Group D was significant and difference between Group NS and Group C was also significant but difference between Group D and Group C was not significant.[17] In our study rise in SBP, DBP and MAP were present in both groups, but difference was statistically significant between Group D and Group C. This can be because they used high dose of clonidine than dexmedetomidine in their study and we used low dose of clonidine. Naz Anjum et al also found statistically significant increase in the MAP in group P (saline) whereas the increase was not significant in group C and group D.[5]

SpO_2

The mean SpO2 levels remain fairly constant above 95% in all patients in both the groups. The difference in SpO_2 was statistically insignificant at all times. These findings are in concordance with studies conducted by Sameer Arora et al.[14] and Mondal S et al.[15]

EtCO2

There was statistically insignificant difference in EtCO2 of patients in the 2 groups at all times.

RAMSAY SEDATION SCORE

Mean Ramsay sedation score in Group D was 2.06 ± 0.239 and in Group C was 2.04 ± 0.197. This was statistically insignificant (Table 8). This finding is not in concordance with Sameer Arora et al.[14] and Shirsendu et al.[15] also showed statistically significant difference between dexmedetomidine and clonidine group. This dissimilarity could be due to low dose of dexmedetomidine, and clonidine used in our study.

In post-operative period, no statistically significant difference was found in mean heart rate, mean systolic blood pressure, mean diastolic blood pressure, mean arterial pressure values, SpO2 measurements and Modified Aldrete Score between two groups (p value > 0.05).

ADVERSE EFFECTS

No patient in our study had bradycardia (HR < 50 bpm), hypotension (SBP < 90 mmHg or DBP < 60 mmHg or MAP <50 mmHg), arrhythmias. Vitals were also stable in Post-operative Anaesthesia Care Unit (PACU).

CONCLUSION

The present study corroborates with those of previous studies. Prior administration of α2 agonist especially Dexmedetomidine, it decreases the dose requirement of intravenous induction agent as well as intraoperative inhalational requirement. So indirectly it decreases financial burden or has economic benefits.

The sympathetic response of laryngoscopy and intubation is significantly attenuated by dexmedetomidine as compared to clonidine. So, IV bolus dose of dexmedetomidine 0.5 µg/kg administered 10 mins before laryngoscopy and intubation can be recommended to attenuate the sympathetic response to laryngoscopy and intubation without any side effects.

LIMITATIONS

Cost of drug is an important factor, and we did not conduct a cost-effectiveness analysis. We did not measure the drug levels in blood.

REFERENCES

1. Miller RD. Monitoring brain state during general anesthesia and sedation. Miller's anesthesia. 8th edn. Elsevier 2015; 1:1524-5.
2. Kaur S, Kataria AP, Kaur G, Kaur M. Comparison of induction characteristics of propofol-lipuro and etomidate –lipuro in cardiac patients in non-cardiac surgery. Int J Sci Study 2014; 2(6):66-72.
3. Yang HS, Kim TY, Bang S et al. Comparison of the impact of the anesthesia induction using thiopental and propofol on cardiac function for non-cardiac surgery. J Cardiovasc Ultrasound 2014; 22(2):58-64.
4. Singh S, Nazir N. A comparative study of effect of dexmedetomidine in dose general anaesthesia. Int J Res Med Sci 2016 Aug; 4(8): 3365-69.
5. Anjum N, Tabish H, Debdas S, Bani HP, Rajat C, Dastidar G et al. Effects of dexmedetomidine and clonidine as propofol adjuvants on intra-operative hemodynamics and recovery profiles in patients undergoing laparoscopic cholecystectomy: A prospective randomized comparative study. Avicenna J Med.2015 Jul-Sep; 5(3): 67-73.
6. Dutta A, Sethi N,Sood J, Panday BC, Gupta M, Choudhary P, Puri GD. The effect of dexmedetomidine on propofol requirements during anesthesia administered by bispectral index – guided closed loop anesthesia delivery system: A randomized controlled study. Anesth Analg 2019 July; 129(1):84-91.
7. Aantaa R, Scheinin M. Alpha 2 adrenergic agents in anaesthesia. Acta Anaesthesiol Scand 1993; 37(5): 433-48.
8. Guen ML, Liu N, Tounou F, Auge M, Tuil O, Chazot T et al. Dexmedetomidine reduces propofol and remifentanil requirements during bispectral index – guided closed loop anesthesia: A double – blind , placebo-controlled trial. Anesth Analg 2014 May; 118(5): 946-55.
9. Kaur B, Bariar H, Kumar P, Garg S, Sharma C, Kumar L. A comparative study of dexmedetomidine and clonidine to attenuate haemodynamic response during laryngoscopy and intubation. J Evolution Med Dent Sci 2018 May; 7(19): 2404-9.
10. Puri GD, Batra YK. Effects of nifedipine on cardiovascular response to laryngoscopy and intubation. Br J Anaesth 1988; 60(5): 579-81.
11. Sen S, Chakraborty J, Santra S, Mukherjee P, Das B. The effect of dexmedetomidine infusion on propofol requirement for maintainence of optimum depth of anaesthesia during elective spine surgery. Indian J Anaesth 2013 Jul-Aug; 57(4): 358-63.
12. Ohtani N, Kida K, Shoji K, Yasui Y, Masaki E. Recovery profiles from dexmedetomidine as a general anesthetic adjuvant in patients undergoing lower abdominal surgery. Anaesth Analg 2008; 107: 1871-4.
13. Keniya VM, Ladi S, Naphade R. Dexmedetomidine attenuates sympathoadrenal response to tracheal intubation and reduces perioperative anaesthetic requirement. Indian J Anaesth 2011 Jul-Aug; 55(4): 352-7.
14. Arora S, Parikh H, Karia S et al. A comparative study of haemodynamic responses to intubation: dexmedetomidine 1µg/kg vs clonidine 1µg/kg. International Journal of biomedical research 2014; 5(12): 721-4.

15. Mondal S, Mondal H, Sarkar R et al. Comparison of dexmedetomidine and clonidine for attenuation of sympathoadrenal responses and anaesthetic requirements to laryngoscopy and endotracheal intubation. Int J Basic Clin Pharmacol 2014; 3(3): 501-6.
16. Panda BK, Singh P, Marne S et al. A comparison study of dexmedetomidine vs clonidine for sympathoadrenal response, perioperative drug requirements and cost analysis. Asian Pacific journal of Tropical Disease2012; 2: 815-21.
17. Rao AV, Sreelatha V, Kumar MV. Dexmedetomidine and Clonidine on induction, hemodynamic and cardiovascular parameters for intubation in general anesthesia cases a comparative study. IOSR Journal of Dental and Medical Sciences 2015; 14(1): 57-68.

Source of Support: Nil. **Conflict of Interest:** None Declared.

Cite this article as: Balwinder Kaur, Mandeep Kaur, Sahil Garg. Reduction in Dose of Induction Agent Propofol and Hemodynamics by A2 Agonists Dexmedetomidine Versus Clonidine in Patients Undergoing Elective Surgeries Under General Anaesthesia: A Prospective Randomised Double Blind Clinical Trial. Int J Med Res Prof. 2022 July; 8(4): 32-38. DOI:10.21276/ijmrp.2022.8.4.008