Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(4), 2043-2052 | ISSN:2251-6727

A Comparative Study of Intravenous Dexmedetomidine and Intravenous Clonidine in Assessing Hemodynamic Stability During Creation of Pneumoperitoneum and Postoperative Analgesia in Laparoscopic Surgery Under General Anesthesia: A Prospective Randomized Double-Blind Study

Dr Siddesh N Kadur

Associate Professor, Dept of Anaesthesia, KLE JGMM Medical College, Hubli KAHER University.

Dr Prokwana Burman

SDM Medical College and Hospital, Dharwad

corresponding author- Dr Siddesh N Kadur

(Received: 16 July 2025 Revised: 10 August 2025 Accepted: 30 August 2025)

KEYWORDS

Intravenous Dexmedetomidine, Intravenous Clonidine

ABSTRACT:

Introduction - During laparoscopic surgery, CO2 insufflation may produce significant hemodynamic changes in heart rate, increase in arterial blood pressure, SVR and PVR due to increased intra-abdominal pressure and hypercarbia. α2 adrenergic receptor agonists, clonidine and dexmedetomidine can be used to blunt these responses due to their sympatholytic and cardiovascular stabilizing actions.

Materials and methods – A randomized double blind comparative study in elective laparoscopic surgical patients of ASA grades I and II was conducted with 76 patients aged between 18-65 years, who were randomly divided into two groups. Patients were randomized by computer generated numbering. Group A patients received 2 µg/kg of clonidine and group B patients received 1µg/kg of dexmedetomidine both diluted in 10 ml of normal saline slow IV over 10 minutes before induction of general anesthesia. Systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MAP) and heart rate (HR) were monitored at baseline, at induction, intubation, creation of pneumoperitoneum, then at regular intervals of every 15 minutes till desufflation and extubation. Post operatively VAS scores were recorded for all patients at the end of surgery, 15 minutes, 30 minutes, 45 minutes, 60 minutes and 90 minutes. Patients were observed in the post-operative room till VAS score of 5. Inj. diclofenac sodium 75mg IV was given as first line and Inj.tramadol 2mg/kg IV was given as second line of rescue analgesia.

Results - The data was presented as Mean ± SD. Groups A and B were compared by independent unpaired student's t test at each timepoint. P value of <0.05 was considered statistically significant. The SBP measured at 30 minutes of pneumoperitoneum for group A vs group B was 109.42± 8.62 vs 112.58±10.65, p=0.160, for DBP 69.45±7.45 vs 71.68±7.29, p=0.190; for MAP 73.26±12.21 vs 76.71±11.43, p=0.208; for HR 72.11±13.37 vs 77.76±12.68, p=0.062. Findings were also recorded for all other timepoints and there was no statistical significance. The mean VAS scores in Group A was higher in all timepoints compared to Group B. The scores at 30 minutes and 45 minutes were significantly higher in Group A with p values of 0.018, 0.001 respectively.

Conclusion - Hence it can be concluded that both the drugs were found to be equally effective in attenuating the hemodynamic response to pneumoperitoneum. Also dexmedetomidine provided better post operative analgesia when compared to clonidine.

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(4), 2043-2052 | ISSN:2251-6727

Introduction

Laparoscopic surgeries are surgeries performed with smaller incisions for different pathologies in the abdomen and pelvic region with the help of pneumoperitoneum.

The use of optimal insufflating gas to create adequate space for the surgery is of paramount importance in a laparoscopic surgery. Over the years various gases like air, oxygen, nitrous oxide, nitrogen, carbon dioxide, argon and helium have been used for creating pneumoperitoneum. [1]

However, with the use of CO_2 for insufflation, serious complications leading to changes in acid-base balance, cardiovascular, pulmonary physiology and stress responses have been seen. These are characterized by an increase in arterial pressure, SVR and PVR and also decrease in cardiac output.[2][3][4][5].Pulmonary function changes are characterized by reduced compliance without large alterations in arterial saturation of oxygen (PaO2), but tissue oxygenation can be adversely affected due to reduced O2 delivery[3] . Hence , a major difficulty is in maintaining normocarbia due to decreased pulmonary compliance secondary to upward movement of the diaphragm due to pneumoperitoneum and the changes in CO2 homeostasis following absorption of insufflated CO2[6].To prevent these, various modes of anaesthesia like epidural or spinal or combined epidural and general anesthesia techniques using drugs such as opioids, esmolol, sodium nitroprusside, nitroglycerin and α2-adrenergic agonists are reported.[7]

Adrenergic receptors were originally differentiated into α and β receptors by Ahlquist[8][9]. Activation of either α- or β-adrenergic receptors produced excitatory effects in some tissues and inhibitory effects in others. Later the α receptors were further subdivided into α1 and α2 subtypes[10,11,12]. α1 receptors are further classified into α 1a, α 1b and α 1d. Similarly α2 receptors are also further classified into α2a, α2b and α2c, which are presynaptic, post synaptic and extrasynaptic[11][12][13]. β receptors are classified into β1, β2 and β3 types. All of these are linked to G proteins. α1 receptors are post synaptic receptors located in the smooth muscles of the eyes, lungs, blood vessels, uterus and gut. α2 receptors are usually found on presynaptic nerve terminals.[13][14][15][16] β1 receptors are foundpost synaptically in the heart while β2 receptors are found largely postsynaptically in the smooth muscles and gland cells. β3 receptors are found in the gall bladder and brain adipose tissue. Functionally even though α 1 receptors are always excitatory, it has been found that α 2 receptors can be either inhibitory or excitatory.

These receptor subtypes are distributed vastly, and each are exclusively responsible for some, but not all, of the actions of α2 agonists; for instance, the α2B-adrenoceptor subtype mediates the short-term hypertensive response to α2 agonists[17][18], whereas the α2A adrenoceptor is responsible for the anesthetic and sympatholytic responses. [19]

Aim -

To study the effectiveness of intravenous dexmedetomidine and clonidine in attenuating the hemodynamic response to pneumoperitoneum in patients undergoing elective laparoscopic surgeries in a comparative study.

Objectives-

Primary objective :

To compare the efficacy of IV dexmedetomidine and IV clonidine in blunting the hemodynamic response during CO_2 insufflation.

Secondary objective :

a) To study postoperative analgesia of these two drugs and the need for rescue analgesic.
b) To study the adverse effects if any.

Need for study –

Unfavorable hemodynamic reactions being common during pneumoperitoneum can be detrimental in cardiovascular compromised patients. Therefore it is preferred to attenuate adverse hemodynamic response by using different pharmacological agents. Hence to blunt these hemodynamic response during pneumoperitoneum which can complicate to cardiac dysrhythmias, myocardial ischemia, infarction, cerebrovascular accidents with higher risk in patients with preexisting essential hypertension, ischemic heart disease, or increased intracranial pressure conditions and to provide postoperative analgesia and sedation, i.v dexmedetomidine and i.v clonidine have been studied due to their alpha 2 agonist actions. However, limited

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(4), 2043-2052 | ISSN:2251-6727

study is available on the comparison of the potency and adverse effects of both the drugs during lapaoscopic surgeries.

Methodology -

A prospective randomised double blind control study was conducted with all patients between 18 to 60 yrs age,belonging to the ASA Grade I/II undergoing elective laparoscopic surgery with endotracheal intubation were included in the study. Procedures lasting for more than 120 minutes, patients with anticipated difficult airway, with neurological , cardiovascular, renal or hepatic disorders, pregnant or breast feeding females, patients on antihypertensives, antipsychotics, analgesics or sedative medications were excluded.

Study period was from April 2021 to January 2023
Sample size calculation:

$$n=\frac{2\times S^2\times(Z\alpha+Z\beta)^2}{d^2}$$

Where, $S=(S_1+S_2)^2$

S_1 is standard deviation of one parameter prior to drug administration.

S_2 is standard deviation of same parameter after drug administration.

d = difference between two means

$Z\alpha = 1.96$

$Z\beta = 1.24$

From this we derived n=30.

Hence we considered dropout rates and included 38 patients in each group.

Study design :

After obtaining institutional ethical committee clearance and written informed consent from 76 patients fulfilling the inclusion criteria were randomly divided into two groups, group A and group B of 38 each using computer generated random numbering table. After detailed preoperative evaluation, all the patients were kept nil per oral for solids for 8 hours,premedicated with tab. pantoprazole 40mg and tab alprazolam 0.25 mg on the night before the surgery and VAS score for postoperative pain was explained to patient.

On the day of surgery after NPO status and patient consent was reconfirmed, a wide bore IV line was secured in the preoperative room. Patient was shifted to operating room and all standard ASA monitors were attached and baseline readingswere recorded for both the groups. Group A received 2µg/kg of clonidine diluted in 10 ml normal saline over 10 minutes slow IV and Group B received 1µg/kg of dexmedetomidine diluted in 10 ml normal saline slow IV over 10minutes before induction of general anesthesia.

After preoxygenation for 5 mins, general anaesthesia was induced with fentanyl 2µg/kg and propofol 2mg/kg by weight and endotracheal intubation was facilitated by vecuronium bromide 0.1mg/kg iv and anaesthesia was maintained with oxygen and nitrous oxide in the ratio 33:66 and with 1-1.2% isoflurane. Muscle relaxation was maintained by vecuronium bromide 0.02mg/kg intermittently thereafter.

Controlled mechanical ventilation was done to maintain $ETCO_2$ between 30-40 mmHg. Intra-abdominal pressure during pneumoperitoneum was maintained between 12-14 mmHg.

Vitals were recorded throughout the procedure, at baseline (T_0mins) , while giving study drug (T_5,T_{10} mins) ,at induction (T_i mins), at intubation (T_{it} mins), at skin incision (T_{sc}mins), carbondioxide insufflation (T_{pn} mins), thereafter at (T_{pn+15}, T_{pn+30}, T_{pn+45}mins) during procedure and at extubation (T_{ex}mins). Recordings were then tabulated and analysed using SPSS software.Any hypotension (MAP <20% preoperative) was treated with a fluid bolus of normal saline 250–300 mL if hypotension did not respond to fluid administration, Inj. Ephedrine 6 mg I.V. was the second line of management. Intra operative bradycardia i.e. heart rate of below 40bpm was managed with Inj. Atropine 0.6mg I.V.At the end of surgery, residual neuromuscular block was reversed by neostigmine in dose of 0.05mg/kg and glycopyrrolate in dose of 0.01mg/kg IV. Patients were extubated after complete reversal of neuromuscular blockade and were then transferred to recovery room.

Pain was assessed on 10 point visual analogue score (VAS) at the end of surgery,15 minutes,30 minutes,45minutes,60 minutes and 90 minutes postoperatively. Patients were observed in the post-operative room till VAS score of 5. Inj. diclofenac sodium 75mg I.V was given as first line and Inj. tramadol 2mg/kg IV was given as second line of rescue analgesia.

Journal of Chemical Health Risks

www.jchr.org

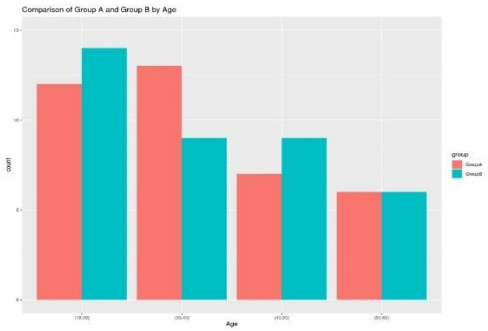
JCHR (2025) 15(4), 2043-2052 | ISSN:2251-6727

Results

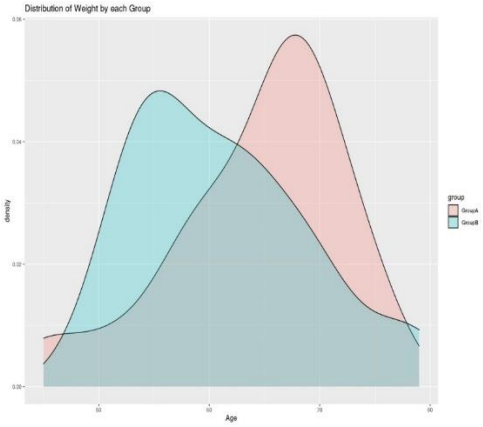
The demographic data such as age, sex, weight of patients in both the groups are shown in Table 1.

TABLE 1 – DEMOGRAPHIC DATA

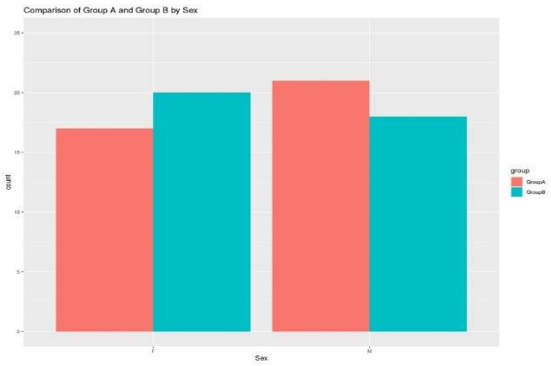
DEMOGRAPHICS	GROUP A	GROUP B	TOTAL	P VALUE
AGE				0.957
MEAN	37.737 (10.847)	37.605 (10.492)	37.671 (10.600)	
RANGE	23.000 - 61.000	20.000 - 58.000	20.000 - 61.000	
WEIGHT				0.079
MEAN	64.368 (7.754)	61.184 (7.839)	62.776 (7.909)	
RANGE	45.000 - 76.000	49.000 - 79.000	45.000 - 79.000	
SEX				0.491
MALE	17 (44.7 %)	20 (52.6 %)	37 (48.7 %)	
FEMALE	21 (55.3 %)	18 (47.4 %)	39 (51.3 %)	



GRAPH 1 – COMPARISON OF AGE BETWEEN TWO GROUPS



GRAPH 2 – DISTRIBUTION OF WEIGHT BETWEEN TWO GROUPS



GRAPH 3 – DISTRIBUTION OF SEX IN THE TWO GROUPS

Statistically no difference was found between the two groups in demographics.

DURATION OF PNEUMOPERITONEUM –

TABLE 2 – DURATION OF PNEUMOPERITONEUM

Minutes	Group A mean (N=38)	Group B mean (N=38)	Total (N=76)	P value
Mean (SD)	61.211(3.378)	60.263(5.134)	60.737(4.343)	0.345
Range	45.00-68.00	43.00-68.00	43.00-68.00	

One- way ANOVA test was run to check for significant association between pneumoperitoneum time and the two groups which showed pneumoperitoneum duration was not a confounding factor in the study.

Journal of Chemical Health Risks

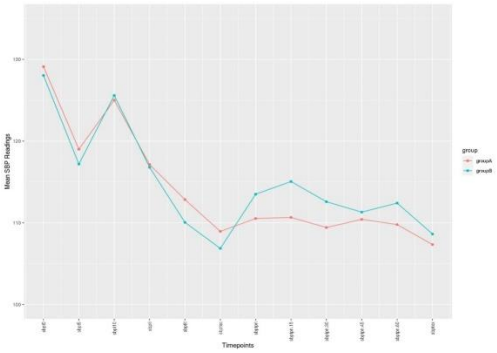
www.jchr.org

JCHR (2025) 15(4), 2043-2052 | ISSN:2251-6727

SYSTOLIC BLOOD PRESSURE

TABLE 3– SYSTOLIC BLOOD PRESSURE

TIMEPOINT	GROUP A MEAN	GROUP A SD	GROUP B MEAN	GROUP B SD	P VALUE
0 MINUTES	129.11	10.47	128.03	13.23	0.697
5 MINUTES	125.00	11.59	125.58	12.24	0.833
10 MINUTES	119.00	11.72	117.18	13.49	0.533
INDUCTION	112.84	13.50	110.05	14.84	0.394
INTUBATION	108.95	12.37	106.87	16.18	0.531
SKIN INCISION	107.34	11.82	108.63	15.02	0.679
PNEUMOPERITONEUM	110.53	10.39	113.50	12.13	0.255
15MINUTES OF PNEUMO	110.66	9.04	115.05	10.62	0.056
30MINUTES OF PNEUMO	109.42	8.62	112.58	10.65	0.160
45 MINUTES OF PNEUMO	110.42	9.44	111.31	11.70	0.722
60 MINUTES OF PNUEMO	109.78	7.88	112.41	13.58	0.359
EXTUBATION	117.13	13.41	116.79	12.02	0.907



GRAPH 4- MEAN SBP READINGS IN GROUP A AND GROUP B

The mean SBP drop in Group A was from 129.11±10.47 at 0 minutes to 110.53±10.39 at the time of pneumoperitoneum and in Group B was from 128.03±13.23 at 0 minutes to 113.50±12.13 at time of pneumoperitoneum (p value – 0.697 vs 0.255) . The mean SBP values at 30 mins of pneumoperitoneum in Group A vs B was 109.42 ±8.62 vs 112.58 ±10.65 with a p value of 0.160 which was statistically not significant.

DIASTOLIC BLOOD PRESSURE -

TABLE 4 – DIASTOLIC BLOOD PRESSURE

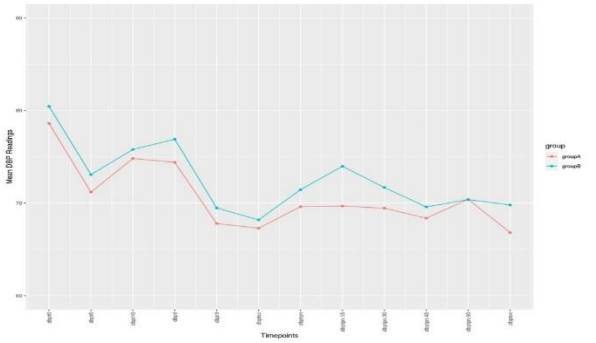
TIMEPOINT	Group A Mean	GroupA SD	Group B Mean	GroupB SD	p.value
0 MINUTES	78.61	7.26	80.45	11.64	0.411
5 MINUTES	74.82	7.37	75.79	9.61	0.622
10 MINUTES	71.18	7.13	73.08	8.82	0.307
INDUCTION	67.79	8.91	69.47	10.62	0.456
INTUBATION	67.29	9.14	68.18	11.58	0.71
SKIN INCISION	66.82	7.53	69.79	11.06	0.175
PNEUMOPERITONEUM	69.61	9.26	71.45	8.28	0.364
15MINUTES OF PNEUMO	69.68	7.67	73.97	7.42	0.015
30MINUTES OF PNEUMO	69.45	7.45	71.68	7.29	0.19
45 MINUTES OF PNEUMO	68.37	6.85	69.58	8.11	0.49
60	70.41	6.34	70.38	7.66	0.98

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(4), 2043-2052 | ISSN:2251-6727

MINUTES OF PNUEMO					8
EXTUBATION	74.39	8.79	76.89	7.17	0.179



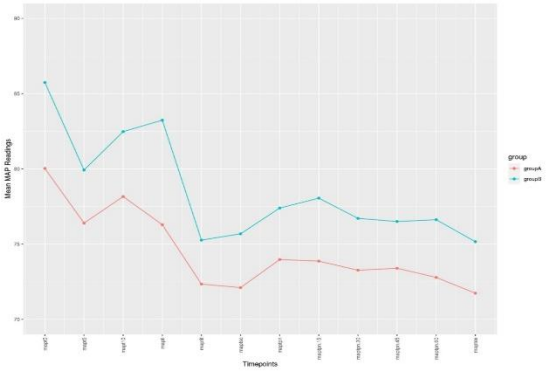
GRAPH 5 – MEAN DBP IN GROUP A AND GROUP B

The mean scores of DBP in Group A was lower compared to Group B over all timepoints with marginally being higher in Group A at 60 minutes of pneumoperitoneumtimepoint as compared to Group B. At 15 minutes of pneumoperitoneum , DBP reading of patients in Group A was significantly lower than Group B (p value = 0.015).

MEAN ARTERIAL PRESSURE (MAP)-

TABLE 5 – MEAN ARTERIAL PRESSURE

TIMEPOINT	GroupA Mean	GroupA SD	GroupB Mean	GroupB SD	p.value
0 MINUTES	80.03	17.93	85.74	17.2	0.161
5 MINUTES	78.16	16.24	82.47	15	0.233
10 MINUTES	76.39	15.45	79.92	13.84	0.298
INDUCTION	72.3	14.2	75.2	15.8	0.40
	74	9	76	2	1
INTUBATION	72.11	15.18	75.68	17.33	0.342
SKIN INCISION	71.74	13.29	75.16	14.38	0.285
PNEUMOPERITONEUM	73.97	14.26	77.39	13.26	0.282
15MINUTES OF PNEUMO	73.87	12.51	78.05	12.67	0.152
30MINUTES OF PNEUMO	73.26	12.21	76.71	11.43	0.208
45 MINUTES OF PNEUMO	73.39	11.84	76.5	11.54	0.257
60 MINUTES OF PNUEMO	72.78	9.4	76.62	8.36	0.085
EXTUBATION	76.29	15.3	83.24	13.11	0.037



GRAPH 6 – MEAN MAP IN GROUP A AND GROUP B

The mean MAP at 0 minutes vs time of pneumoperitoneum was 80.03±17.93 vs 73.97±14.26 in group A and 85.74±17.2 vs 77.39±13.36 in group B (p value – 0.161 vs 0.282)

The mean scores of MAP in Group A was lower compared to Group B across all timepoints. At time of extubation, Group A vs Group B MAP was 76.29±15.3 vs 83.24±13.11 which was significantly lower (p value =0.037).

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(4), 2043-2052 | ISSN:2251-6727

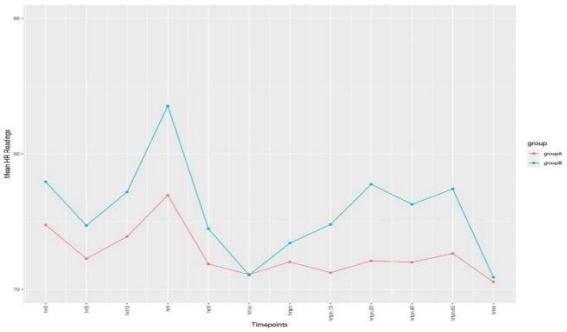
HEART RATE (HR)

TABLE 6 – HEART RATE

TIMEPOINT	GroupA Mean	GroupA SD	GroupB mean	GroupB SD	p.value
0 MINUTES	74.76	14.14	77.95	14.52	0.336
5 MINUTES	73.89	13.95	77.18	14.54	0.318
10 MINUTES	72.26	14.3	74.71	14.01	0.454
INDUCTION	71.87	12.22	74.47	12.55	0.362
INTUBATION	71.11	10.19	71.05	8.84	0.981
SKIN INCISION	70.55	10.48	70.89	9.53	0.882
PNEUMOPERITONEUM	72.03	11.36	73.42	11.02	0.588
15MINUTES OF PNEUMO	71.24	11.39	74.79	11.6	0.182
30MINUTES OF PNEUMO	72.11	13.37	77.76	12.68	0.062
45 MINUTES OF PNEUMO	72	11.86	76.28	11.82	0.125
60 MINUTES OF PNUEMO	72.65	10.84	77.41	10.09	0.07
EXTUBATION	76.95	12.26	83.54	10.26	0.014

The HR in Group A vs Group B at 0 minutes and time of pneumoperitoneum was 74.76±14.14 vs 77.95±14.52 (p value 0.336) and 72.03±11.36 vs 73.42±11.02 (p value – 0.588) respectively.

The mean scores of HR in Group A was lower compared to Group B at all timepoints. The mean score of HR at extubation for group A vs group B was 76.95±12.26 vs 83.54±10.26 with a significant p value of 0.014.



GRAPH 7 – MEAN HR FOR GROUP A AND GROUP B

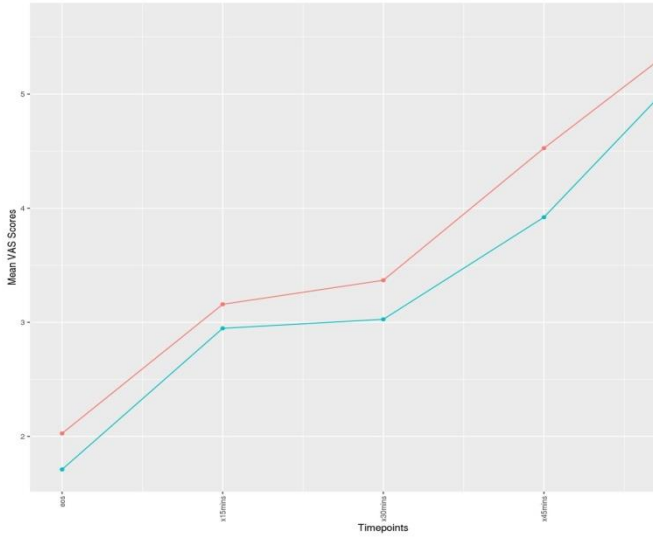
TABLE 7 – VAS SCORES

	TIMEPOINT	GroupA Mean	GroupA SD	GroupB mean	GroupB SD	p.value
1	End of surgery	2.03	0.72	1.71	0.73	0.061
2	after15minutes	3.16	0.49	2.95	0.46	0.059
3	after30minutes	3.37	0.67	3.03	0.54	0.018
4	after 45minutes	4.53	0.83	3.92	0.63	0.001
5	after90minutes	5.61	0.68	5.39	1.08	0.313

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(4), 2043-2052 | ISSN:2251-6727



GRAPH 8 – VAS SCORES FOR GROUP A AND GROUP B

The mean VAS scores in Group A was higher at all timepoints compared to Group B. The scores at 30 minutes and 45 minutes were significantly higher in Group A with p values of 0.018, 0.001 respectively. At 45minutes, the group difference in mean VAS score was highly significant (p value = 0.001).

TABLE 8 – RESCUE ANALGESIA

DRUG GIVEN	GROUP A(N=38)	GROUP B (N=38)	TOTAL(N=76)	P VALUE
				0.012
NO	2(5.3%)	10(26.3%)	12(15.8%)	
YES	36(94.7%)	28(73.7%)	64(84.2%)	

Pearsons chi-square test was used to check if there was any significant association between drug given in the two groups. Results showed significant association with a p value 0.012 which was <0.05. Rescue analgesia requirement was higher in Group A (94.7%) as compared to Group B (73.7%).

Discussion

Pneumoperitoneum creation and maintenance during laparoscopic surgeries causes detrimental hemodynamic and respiratory system changes in patients. Thus in our prospective comparative study , we studied two alpha 2 agonist drugs used to blunt these hemodynamic changes due to pneumoperitoneum during laparoscopic surgeries.

In the present study a total of 76 participants were recruited and then divided into two groups A and B with 38 participants in esch group. The age of the patients ranged from 20 to 61 years, with 49% patients being females and 51% patients being males. The weight ranged from 45 to 79 kgs. As per ASA grade, there were 36% cases of ASA grade 2 and 64% cases of grade 1. Comorbid illnesses present were hypothyroidism in 10%, type 2 DM in 21% and both of these in 4% cases.The demographic data of both the groups A and B such as age, gender, weight were presented as mean ± SD. Groups A and B were compared for HR, SBP,DBP, and MAP by independent unpaired student's t test at each timepoint.

It was found that the SBP was higher at all the timepoints of the study in group A compared to group B except for the time of creation and maintenance of pneumoperitoneum. However, none of these values were statistically significant.In our study dexmedetomidine group effectively showed more long lasting control over SBP during creation of pneumoperitoneum, its maintenance and at the time of extubation as compared to clonidine group. The DBP values showed a lower trend with both the drugs as compared to their pre medication values in our study.The group A participants recorded a lower DBP as compared to group B at all time points , with a statistically significant value showing at the mid time point of pneumoperitoneum maintenance (69.68±7.67 vs 73.97±7.42, p value – 0.015). Hence it can be concludedboth the drugsnot only lowered SBP and DBP recordings from their preoperative values but also were equipotent in controlling SBP and DBP during pneumoperitoneum.These findings are in accordance with the study done by Kumar et al[20]where they observed the mean trend of SBP in both groups were similar over the periods with slightly being higher in Group 1(clonidine group) at all periods as compared to

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(4), 2043-2052 | ISSN:2251-6727

Group 2(dexmedetomidine group).they also found that even though clonidine and dexmedetomidine reduces the DBP and prevents its rise during early periods of procedure, it does not suppress increase of DBP during extubation completely. In our study there was no increase of DBP during extubation in any of the groups.

The mean MAP values and the mean HR showed lower recordings in group A as compared to group B at all timepoints in our study with a statistically significant lower value at the time of extubation. However, during pneumoperitoneum both showed similar effects with no statistically significant values.

Hatti Prashant et al[21] in their randomized controlled trial observed that the MAP between the clonidine and dexmedetomidine groups showed values 92.28±7.00 vs 88.16±6.70 with a significant p value of 0.039 at 6 minutes of infusion of drug whereas rest of the timepoint showed no statistically significant difference in MAP between the two groups. So they inferred, clonidine or dexmedetomidine effectively attenuate the haemodynamic response in patients undergoing laparoscopic lower abdominal onco-surgeries when administered as IV bolus dose.These conferred to the findings of our study too.

For HR they found that both clonidine and dexmedetomidine groups showed reduction in the HR compared to basal values. Statistically significant reduction in the HR was noticed in dexmedetomidine group as compared to clonidine group at 12th minute of infusion (p=0.008), at the end of infusion (p=0.014), before intubation (p=0.005), 10 minutes after intubation (p=0.008) and immediately after pneumoperitoneum. (p=0.045). Hence they concluded that dexmedetomidine group showed greater hemodynamic stability than clonidine group as it is a highly selective potent and specific α2 agonist. However in the present study we observed that the HR was lower in the clonidine group when compared to dexmedetomidine group.

Our study showed the VAS scores to be higher in group A when compared to group B with a statistical significance. Mean VAS score in group A vs group B was 3.37±0.67 vs 3.03±0.54 (p value 0.018) at 30 minutes and 4.53±0.83vs 3.92±0.63(p value 0.001) at 45 minutes of post operative time. The requirement for rescue analgesia was higher in group A as compared to group B (94.7% vs 73.7%) with a statistically significant p value of 0.012. From the above findings it can be derived that dexmedetomidine provided better post operative analgesia than clonidine. Pooja Gautam[22] in her study found the mean VAS of the patients in clonidine was 3 at the end of procedure and all of the patients required analgesic after 60 minutes of surgery and 9/30 patients require rescue analgesia at extubation, while with dexmedetomidine, the mean VAS at the end of procedure was 1 and most patients had adequate analgesia up to 90 min. Thus, dexmedetomidine is far better analgesic as compared to clonidine regarding duration of analgesia.

There was no complication noted in the study.Thus, both the drugs were found to be safe.

Conclusion –

In our study comparing efficacy of clonidine and dexmedetomidine in blunting hemodynamic response to pneumoperitoneum we found that –

1) Both clonidine and dexmedetomidine were equipotent in blunting SBP, DBP and MAP.

2) Clonidine decreased HR from baseline values more than dexmedetomidine but there was no instances of bradycardia or hypotension.

3) Dexmedetomidine group had lower requirement of rescue analgesia.

4) Clonidine can be considered a more cost effective alternative to dexmedetomidine in laparoscopic surgeries.

References –

1. Sood J, Jain A. K. Anaesthesia in laparoscopic surgery. New Delhi. Jaypee Brothers Medical Publishers (P) Ltd; 2007.
2. Kulkarni K, (2016) Effect of Intravenous Clonidine as Premedication on Haemodynamic Responses during Laparoscopic Cholecystectomy. J AnesthCrit Care Open Access 4(1): 00121.
3. Joris J (1994) Anesthetic management of laparoscopy. Miller RD (Ed.) Anesthesia Churchill Livingstone, USA, pp. 2011-2029.

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(4), 2043-2052 | ISSN:2251-6727

4. Wahba RWM, Beique F, Kleiman SJ (1995) Cardiopulmonary functionand laparoscopic choleystectomy. Can J Anaesth 42(1): 51-63.
5. Sharma KC, Brandstetter RD, Brensilver JM, Jung LD (1996) Cardiopulmonary physiology and pathophysiology as a consequence of laparoscopic surgery. Chest 110(3): 810-815.
6. Joris JL, Noirot DP, Legrand MJ, Jacquet NJ, Lamy ML (1993)Hemodynamic changes during laparoscopic cholecystectomyAnesthAnalg 76(5): 1067-1071.
7. Boussofara M, Mtaallah MH, Nefaa MN, KaddourC (2004) Clonidineand anesthesia. Tunis Med 82(3): 249-257.
8. Giovannitti JA Jr, Thoms SM, Crawford JJ. Alpha-2 adrenergic receptor agonists: a review of current clinical applications. Anesth Prog. 2015 Spring;62(1):31-9.
9. Ahlquist RP. A study of the adrenotropic receptors. Am J Physiol. 1948;153:586–600.
10. Van Meel JC, DeJonge A, Timmermans PBM, Van Zwieten, WM. Selectivity of some alpha adrenoceptors in the normotensive rat. J Pharmacol Exp Ther. 1981;219:760–767.
11. Gertler R, Brown HC, Mitchell DH, Silvius EN. Dexmedetomidine: a novel sedative-analgesic agent. Proc (BaylUniv Med Cent). 2001 Jan;14(1):13-21. doi: 10.1080/08998280.2001.11927725.
12. Langer SZ. Presynaptic regulation of catecholamine release. Biochem Pharmacol1974;23:1793–1800.
13. Bylund D. Subtypes of a1 and a2-adrenergic receptors. FASEB J. 1992;6:832–839.
14. Pichot C, Ghignone M, Quintin L. Dexmedtomidine and clonidine: from second- to first-line sedative agents in the critical care setting. J Intensive Care Med. 2012;27:219–237.
15. Carollo DS, Nossaman BD, Ramadhyani U. Dexmedetomidine: a review of clinical applications. CurrOpinAnaesthesiol. 2008;21:457–461.
16. Virtanen R, Savola JM, Saano V, et al. Characterization of the selectivity, specificity and potency of medetomidine as an a2-adrenoceptor agonist. Eur J Pharmacol. 1988;150:9–14.
17. Kamibayashi T, Maze M. Clinical uses of alpha2 -adrenergic agonists. Anesthesiology. 2000 Nov;93(5):1345-9. doi: 10.1097/00000542-200011000-00030. PMID: 11046225.
18. Link RE, Desai K, Hein L, Stevens ME, Chruscinski A, Bernstein D, Barsh GS, Kobilka BK: Cardiovascular regulation in mice lacking a2-adrenergic receptor subtypes b and c. Science 1996; 273:803–5
19. Lakhlani PP, MacMillan LB, Guo TZ, McCool BA, Lovinger DM, Maze M, Limbird LE: Substitution of a mutant a2A-adrenergic receptor via ‘hit and run’ gene targeting reveals the role of this subtype in sedative, analgesic, and anesthetic-sparing responses in vivo. Proc NatlAcad Sci U S A 1997; 94:9950–5
20. S Kumar, B B Kushwaha, R Prakash, S Jafa, A Malik, R Wahal, J Aggarwal, R Kapoor. Comparative Study Of Effects Of Dexmedetomidine And Clonidine Premedication In Perioperative Hemodynamic Stability And Postoperative Analgesia In Laparoscopic Cholecystectomy . The Internet Journal of Anesthesiology. 2014 Volume 33 Number 1.
21. HATTI P, MAMATHA H, SUGANDHARAJAPPA SG, GOWDA V. Effects of Intravenous Dexmedetomidine and Clonidine on Haemodynamic Response in Laparoscopic Lower Abdominal Oncosurgeries under General Anaesthesia: A Randomised Controlled Trial. Journal of Clinical & Diagnostic Research. 2021 Oct 1;15(10).
22. Pooja Gautam. Comparative study of clonidine vs dexmedetomidine for hemodynamic stability and postoperative analgesia during laproscopic surgery. International Journal of Contemporary Medical Research 2019;6(1):A1-A7.