Compare the Effects of Dexmedetomidine Infusion versus Normal Saline as Placebo on Haemodynamic Response in Elective Laparoscopic Surgery Under General Anaesthesia

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Abstract

Background: Throughout the present medical age, laparoscopic surgery is the most effective diagnostic and therapeutic methods. But it is associated with potentially harmful neuroendocrine response. Subjects and Methods: In our study we used dexmedetomidine infusion in group D and normal saline at a rate of 0.4mcg / kg / hr as placebo in group N beginning from 10 minutes before induction before pneumoperitoneum release. Patients in both groups were alike in age, gender, BMI, grade of ASA and anaesthesia. Results: After 10 min infusion of dexmedetomidine and normal saline in group D and group N respectively, fall in HR, DBP and Mean arterial pressure was significantly increased in group D compared to group N. Difference found in both the groups was statistically significant. Group N showed significant increase in HR, SBP, DBP and MAP during laryngoscopy, intubation and at 2 min, 5 min and 10 min after creation of pneumoperitoneum. Difference found was statistically significant in both groups. Thus attenuation in HR, SBP, DBP and MAP was seen in group D as compared to group N during laryngoscopy, tracheal intubation and pneumoperitoneum which were statistically significant. Group N showed statically significant increase in HR, DBP and MAP during extubation compared to group D. Difference found in both the groups was statistically significant. Thus attenuation in HR, DBP and MAP was seen in group D as compared to group N during extubation which was statistically significant. Postoperative sedation score was more in group D compared to group N which was statistically significant. Post-extubation, rescue analgesia was required early in group N compared to group D. Adverse effect in form of bradycardia was seen in 2 patients in group D which did not require atropine administration. Conclusion: Dexmedetomidine infusion at a rate of 0.4 mcg / kg / hr apparently started 10 minutes before the induction of anesthesia before release of pneumoperitoneum without any bolus dose. Useful adjuvant anesthesia to diminish the response of haemodynamic stress to intubation, Pneumoperitoneum, and extubation in laparoscopic surgical patients.

Keywords: Dexmedetomidine, Laparoscopy, Placebo

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Introduction

There has been a relentless quest for new surgical modalities, procedures and equipment since the beginning of medicine’s surgical period. The twentieth century saw the dawn of a modern surgical method, which was broadly accepted by both the patients and surgical fraternity. Minimally invasive surgery nearly revolutionized surgical care across a wide range of diseases.

The importance of performing an internal examination of the many compartments of the human body has been recognized for several centuries. He realised that pneumoperitoneum was important for exposure and hence used room air for insufflation of the peritoneal cavity.

Throughout the present medical age, laparoscopic medical procedure is one of the best demonstrative and helpful methods. Laparoscopy advantages include shorter hospital stay, faster return to daily life, reduced discomfort and fewer incisions.[1] New surgical methods, however, turn into additional anaesthetic problems that include improvements in anaesthetic techniques. Although it has certain benefits over traditional surgery, it comes at the expense of potentially adverse neuroendocrine reaction.
Pneumoperitoneum is produced by carbon dioxide (CO2) insufflation during laparoscopic surgical procedures. Both pneumoperitoneum and CO2 trigger side effects of the cardiovascular system. Some of these symptoms are CO2 associated, and others are due to higher intrabdominal pressure. Immediately after pneumoperitoneum, the activity of plasma norepinephrine, epinephrine, and plasma renin rises. The elevated level of catecholamine also stimulates the renin-angiotensin - aldosterone pathway. Both of these progressions will in general lead to increased arterial pressure increase systemic and pneumonic vascular opposition, and reduced cardiac output, tachycardia, and once in a while arrhythmia. For high-risk patients particularly those with cardiopulmonary conditions these insults can be devastating. Hence, recognizing pathophysiology and preserving haemodynamic control in these patients is of utmost importance.

Therefore, modern anaesthesia procedures aim to minimize sympathetic discharge to have perioperative haemodynamic control. To order to accomplish this goal with varying effectiveness, multiple agents to the form of opioid analgesics, benzodiazepines, beta blockers, calcium channel blockers and vasodilators have been employed. Thanks to their effectiveness, multiple agents to the form of opioid analgesics, benzodiazepines, beta blockers, calcium channel blockers and vasodilators have been employed. Dexmedetomidine decreases sympathetic tone, blood pressure, pulse rate and causes sedation. This also reduces anaesthetic agents and analgesics requirements intraoperatively. In some study reports, dexmedetomidine infusion levels ranging from 0.1 to 10 mcg/kg/hr were used. Higher infusion levels trials have further incidences of adverse effects such as hypotension and bradycardia. In our study, dexmedetomidine at an infusion rate of 0.4 mcg/kg/hr should be used during elective laparoscopic surgery under general anaesthesia. This placebo treatment controlled, randomized forthcoming investigation is intended to assess the impacts of dexmedetomidine infusion on haemodynamic reaction, postoperative sedation and absence of pain necessities in patients of indian phenotype experiencing elective laparoscopic medical procedure under general anaesthesia.

Subjects and Methods

It is Prospective, randomized, double blind, placebo-controlled study conducted in the department of Anaesthesiology, Sri Venkeshwara Ramnarayan Ruia (SVRGGH) Hospital, Tirupathi. The study will be conducted in 60-100 ASA grade 1 and 2 patients between age group above 18 to 65 years, posted for elective laparoscopic surgery. The patients will be uniformly separated into 2 categories using a sealed-envelope system either to receive normal saline infusion (group N) or dexmedetomidine infusion (group D). Each group will be having 30-50 patients. Both groups will be comparable with regards to number, age, BMI, gender distribution and ASA grading. Sample size 27 cases (minimum in each intervention group, i.e. total 54 cases with Group N (Normal Saline) to Group D (Dexmedetomidin)

Inclusion Criteria:

ASA physical grade 1 and 2, Patients with age between above 18 to 65 years of either gender, who are willing to give written and informed consent for study and posted for elective laparoscopic surgery under general anaesthesia.

Exclusion Criteria:

ASA physical grade 3 and 4 patients, Patients who are not willing to give consent to participate, on alpha-2 agonists, Pregnant and lactating women and morbidly obese patients.

The data has collected in a pretested proforma meeting the objectives of the study upon receiving authorisation from the ethical institutional committee. Each patient was given a patient information sheet and informed written consent from the patient was obtained. A pre-anesthetic evaluation done on the evening before surgery, and patients were kept nil per oral 6 hours before surgery. The patients randomly allocated either to receive normal saline infusion (group N) or dexmedetomidine infusion (group D). Each group had 30 patients.

For two patient groups, two separate infusions were held available, based on the group allocated. The dexmedetomidine infusion, available at a 50 mcg concentration in 0.5 ml of the drug, was removed in a 50 ml syringe and was diluted up to 50 ml with normal saline resulting in a final 1 mcg / ml concentration. The syringe pump which was used to infuse the drugs. Depending on the patient’s weight, the targeted infusion rate of dexmedetomidine supplied by the pump is 0.4 mcg / kg / hr. Vital parameters such as baseline pulse rate (PR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and oxygen saturation were monitored and noted after the patient was taken on the operating table. A wide bore intravenous cannula was inserted for the intravenous IV fluids, and another venous line for the infusion was taken. Infusion of dexmedetomidine began at a rate of 0.4 mcg / kg / hr. Premedication given to all patients in the form of midazolam injection (0.05 mg / kg), fentanyl injection (2mcg / kg), and ondansetron injection (0.1mg / kg) IV before induction of anesthesia. 10 min after infusion with thiopentone injection begins at 5mg/kg IV, anesthesia was induced.

Endotracheal intubation with a suitable sized cuff facilitated with injection vecuronium 0.1 mg/kg as a muscle relaxant. Anesthesia maintained with oxygen, nitrous oxide, sevoflurane. Furthermore, vecuronium top up (25% of the initial dose, if required). All patients mechanically ventilated using a circle system. Respiratory rate (RR) and tidal volume were adjusted according to body weight to keep the EtCO2 between 35 and
The age, gender, ASA and BMI distribution did not differ significantly between the two intervention groups (P-value>0.05).

The average heart rate at T0 did not differ significantly between the two intervention groups (P-value>0.05). The average heart rate at time points (T1 to T11) is significantly higher in Group N compared to Group D (P-value<0.05 for all).
- The average systolic BP at T0, T1, T4, T8, T9, and T10 did not differ significantly between two intervention groups (P-value>0.05 for all).
- The average systolic BP at T2, T3, T5, T6, T7, and T11 is significantly higher in Group N compared to Group D (P-value<0.05 for all).

Two intervention groups (P-value>0.05 for both) did not vary significantly from the normal
Diastolic BP at T0 and T4. In group N, the average diastolic BP at T1, T2, T3, T5, T6, T7, T8, T9, T10, and T11 is significantly higher than in group D (for both, the P-value<0.05).

The average peak arterial pressure at T0, T4, and T9 was not significantly different between the two intervention groups (for both, P-value>0.05). The mean blood pressure at T1, T2, T3, T5, T6, T7, T8, T9, and T11 in group B is significantly higher than in group D (for both, P-value<0.05).
- The average SpO2 at all-time points (T0 to T11) did not differ significantly between two intervention groups (P-value>0.05 for all).

The average sedation score at 5 min, 15 min, 30 min, and 60 min post extubation is significantly higher in group D compared to group N. (P-value<0.001 for all). The average pain score (VAS) after 5 min of extubation is significantly higher in group N compared to group D (P-value<0.001).

The average time to rescue analgesia is significantly higher in group N compared to group D (P-value<0.001).

Discussion

The study was conducted in 60 ASA grade I and II patients between the age group 18-65 years, posted for elective laparoscopic surgery. Patient’s randomly allocated either to receive a normal saline infusion (group N) or dexmedetomidine infusion (group D). Each group had 30 patients. Both groups were comparable, and there was no statistically significant difference with regards to mean age, BMI, gender distribution, and ASA grading. Dexmedetomidine infusion started at a rate of 0.4 mcg/kg/hr in group D and normal saline as a placebo in group N from 10 min before induction until the release of pneumoperitoneum in patients undergoing elective laparoscopic surgery. Parameters like HR, SBP, DBP, and MAP recorded at specific intervals. Patients were also observed for postoperative sedation and analgesiarequirement. In our study, after 10
Table 1: The demographic distribution of the cases studied between two intervention groups (n=60).

| Age Group (years) | Group D [Dexmed] (n=30) | Group N [Control] (n=30) | P-value (Group D vs Group N) |
|-------------------|--------------------------|--------------------------|------------------------------|
|                   | N | %  | N | %  |                            |
| 18.0 – 27.0       | 5 | 16.7 | 9 | 30.0 | 0.566NS                      |
| 28.0 – 37.0       | 10 | 33.3 | 5 | 16.7 |                            |
| 38.0 – 47.0       | 7 | 23.3 | 7 | 23.3 |                            |
| 48.0 – 57.0       | 5 | 16.7 | 5 | 16.7 |                            |
| >58.0             | 3 | 10.0 | 4 | 13.3 |                            |
| Total             | 30 | 100.0 | 30 | 100.0 |                            |

**Gender distribution**

|       | Group D [Dexmed] | Group N [Control] | P-value (Group D vs Group N) |
|-------|-----------------|-------------------|------------------------------|
| Male  | 13 | 43.3 | 14 | 46.7 | 0.795NS                      |
| Female| 17 | 56.7 | 16 | 53.3 |                            |

**ASA distribution**

| Grade | Group D [Dexmed] | Group N [Control] | P-value (Group D vs Group N) |
|-------|-----------------|-------------------|------------------------------|
| I     | 23 | 76.7 | 21 | 70.0 | 0.559NS                      |
| II    | 7 | 23.3 | 9 | 30.0 |                            |

**BMI (kg/m²)**

|       | Group D [Dexmed] | Group N [Control] | P-value (Group D vs Group N) |
|-------|-----------------|-------------------|------------------------------|
|       | 21.98 | 2.38 | 21.80 | 2.23 | 0.759NS                      |

Table 2: Group Heart rate (Per min) average

| Heart Rate (Per min) | Group D [Dexmed] (n=30) | Group N [Control] (n=30) | P-value (Group D vs Group N) |
|----------------------|--------------------------|--------------------------|------------------------------|
| T0                   | 84.9 | 8.21 | 84.9 | 2.85 | 0.983NS                      |
| T1                   | 78.5 | 5.45 | 89.3 | 6.64 | 0.0001***                    |
| T2                   | 81.8 | 8.22 | 108.0 | 8.00 | 0.0001***                    |
| T3                   | 74.5 | 6.16 | 94.8 | 4.22 | 0.0001***                    |
| T4                   | 74.0 | 5.79 | 82.2 | 5.72 | 0.0001***                    |
| T5                   | 76.5 | 5.45 | 90.8 | 7.97 | 0.0001***                    |
| T6                   | 76.1 | 5.13 | 91.9 | 5.62 | 0.0001***                    |
| T7                   | 76.1 | 4.60 | 91.5 | 5.14 | 0.0001***                    |
| T8                   | 75.7 | 4.39 | 87.0 | 5.88 | 0.0001***                    |
| T9                   | 73.1 | 5.13 | 84.9 | 5.62 | 0.0001***                    |
| T10                  | 79.4 | 6.95 | 101.2 | 3.24 | 0.0001***                    |
| T11                  | 69.9 | 7.37 | 96.3 | 3.59 | 0.0001***                    |

Gogus N et al. (2014) studied the effects of dexmedetomidine, fentanyl, and esmolol on hemodynamic response to intubation. Group I received one mcg/kg dexmedetomidine as an infusion over 10 min, Group II received two mcg/kg fentanyl, Group III received 2mg/kg esmolol 2 min before induction. They found that dexmedetomidine was superior in the prevention of tachycardia following intubation. Our results are consistent with this study. In our study, after 10 min infusion of dexmedetomidine in group D and normal saline in group N, there was a fall in SBP, DBP, and MAP by 8.8%, 16.8%, and 13.6% respectively in group D which was statistically signifi-
Table 3: The inter-group comparison of Systolic BP at each time interval (n=60).

| Systolic BP (mmHg) | Group D [Dexmed] (n=30) | Group N [Control] (n=30) | P-value (Group D vs Group N) |
|-------------------|--------------------------|--------------------------|-----------------------------|
|                   | Mean                     | SD                       | Mean                       | SD          |                         |
| T0                | 135.9                    | 7.53                     | 132.9                      | 9.42        | 0.164 NS                 |
| T1                | 123.9                    | 7.53                     | 128.3                      | 15.26       | 0.172 NS                 |
| T2                | 131.0                    | 6.71                     | 146.6                      | 9.49        | 0.001 ***                |
| T3                | 121.0                    | 6.71                     | 131.1                      | 13.9        | 0.001 ***                |
| T4                | 122.2                    | 4.76                     | 118.8                      | 11.9        | 0.152 NS                 |
| T5                | 127.4                    | 4.34                     | 137.9                      | 15.71       | 0.001 ***                |
| T6                | 128.3                    | 4.15                     | 138.3                      | 15.04       | 0.001 ***                |
| T7                | 130.4                    | 4.25                     | 141.0                      | 15.05       | 0.001 ***                |
| T8                | 128.4                    | 4.25                     | 130.7                      | 14.09       | 0.382 NS                 |
| T9                | 120.4                    | 4.25                     | 121.0                      | 15.93       | 0.834 NS                 |
| T10               | 133.0                    | 6.71                     | 137.4                      | 11.20       | 0.070 NS                 |
| T11               | 120.3                    | 4.76                     | 127.8                      | 9.16        | 0.001 ***                |

Table 4: The inter-group comparison of Diastolic BP at each time interval (n=60).

| Diastolic BP (mmHg) | Group D [Dexmed] (n=30) | Group N [Control] (n=30) | P-value (Group D vs Group N) |
|-------------------|--------------------------|--------------------------|-----------------------------|
|                   | Mean                     | SD                       | Mean                       | SD          |                         |
| T0                | 85.9                     | 4.81                     | 82.7                       | 8.21        | 0.073 NS                 |
| T1                | 71.4                     | 6.89                     | 78.5                       | 8.14        | 0.001 ***                |
| T2                | 71.9                     | 6.49                     | 94.4                       | 6.69        | 0.001 ***                |
| T3                | 64.6                     | 4.79                     | 88.9                       | 13.00       | 0.001 ***                |
| T4                | 72.3                     | 4.20                     | 78.1                       | 15.85       | 0.059 NS                 |
| T5                | 75.4                     | 4.56                     | 86.1                       | 11.20       | 0.001 ***                |
| T6                | 74.8                     | 4.11                     | 82.7                       | 10.55       | 0.001 ***                |
| T7                | 73.7                     | 4.10                     | 80.8                       | 11.55       | 0.003 **                 |
| T8                | 69.4                     | 6.11                     | 78.4                       | 13.78       | 0.002 **                 |
| T9                | 67.2                     | 8.62                     | 73.9                       | 15.04       | 0.038*                   |
| T10               | 83.9                     | 4.81                     | 88.7                       | 7.58        | 0.005**                  |
| T11               | 71.2                     | 4.07                     | 75.2                       | 8.29        | 0.022*                   |

Patel Chirag et al. (2012),[9] observed a significant fall in SBP and DBP of 6% and 9% respectively from baseline in patients 10 min after administration of 1mcg/kg of dexmedetomidine over 10 min as compared to control group. Our results are consistent with this study. Sukhmaninder Jit Singh Bajwa et al (2012),[10] also observed a transient increase in HR and MAP initially for 3-5 min after starting dexmedetomidine infusion of 1mcg/kg over 20 min. Biphasic responses found in this study might be due to the use of a higher dose of dexmedetomidine.

We did not observe a biphasic response in our study. This response might be due to the lower dose of dexmedetomidine used in our study, 0.4 mcg/kg/hr, that too, given in the form of

cant compared to group N.

In our study average systolic BP at T2, T3, T5, T6, T7, and T11 is statistically significantly higher in group N compared to group D and in group N, the average diastolic BP at T1, T2, T3, T5, T6, T7, T8, T9, T10, and T11 is significantly higher than in group D, which correlates with Kallio et al. (1989),[8] a dose-dependent decrease in systolic and diastolic blood pressure following administration of dexmedetomidine in single intravenous doses of 12.5, 25, 50, and 75mcg over 30 seconds to healthy male volunteers as part of a placebo-controlled study. Our results are consistent with this study.
Table 5: The inter-group comparison of SpO2 at each time interval (n=60).

| SPO2 (%) | Group D [Dexmed] (n=30) | Group N [Control] (n=30) | P-value (Group D vs Group N) |
|----------|-------------------------|--------------------------|-----------------------------|
|          | Mean        | SD        | Mean       | SD     |                      |
| T0       | 100.0       | 0.00      | 99.9       | 0.35   | 0.999<sup>NS</sup>   |
| T1       | 99.7        | 0.48      | 100.0      | 0.00   | 0.999<sup>NS</sup>   |
| T2       | 100.0       | 0.00      | 100.0      | 0.00   | 0.999<sup>NS</sup>   |
| T3       | 100.0       | 0.00      | 100.0      | 0.00   | 0.999<sup>NS</sup>   |
| T4       | 100.0       | 0.00      | 100.0      | 0.00   | 0.999<sup>NS</sup>   |
| T5       | 100.0       | 0.00      | 100.0      | 0.00   | 0.999<sup>NS</sup>   |
| T6       | 100.0       | 0.00      | 100.0      | 0.00   | 0.999<sup>NS</sup>   |
| T7       | 100.0       | 0.00      | 100.0      | 0.00   | 0.999<sup>NS</sup>   |
| T8       | 100.0       | 0.00      | 100.0      | 0.00   | 0.999<sup>NS</sup>   |
| T9       | 100.0       | 0.00      | 100.0      | 0.00   | 0.999<sup>NS</sup>   |
| T10      | 100.0       | 0.00      | 100.0      | 0.00   | 0.999<sup>NS</sup>   |
| T11      | 99.6        | 0.49      | 99.8       | 0.43   | 0.999<sup>NS</sup>   |

Table 6: The inter-group comparison of postoperative sedation, pain score and rescue analgesia at each time interval (n=60).

| Sedation Score after extubation | Group D [Dexmed] (n=30) | Group N [Control] (n=30) | P-value (Group D vs Group N) |
|--------------------------------|-------------------------|--------------------------|-----------------------------|
|                                | Mean        | SD        | Mean       | SD     |                      |
| 5 – Min                        | 2.43        | 0.50      | 1.57       | 0.73   | 0.001***             |
| 15 – Min                       | 2.20        | 0.48      | 1.43       | 0.50   | 0.001***             |
| 30 – Min                       | 2.10        | 0.30      | 1.40       | 0.49   | 0.001***             |
| 60 – Min                       | 1.80        | 0.41      | 1.33       | 0.48   | 0.001***             |
| Pain Score                     | 0.77        | 0.67      | 2.13       | 0.68   | 0.001***             |
| Time to rescue analgesia       | 80.8        | 15.98     | 33.8       | 7.73   | 0.001***             |

infusion. R. Saraf et al (2013),<sup>11</sup> also did not observe biphasic response with a dexmedetomidine dose of 0.6mcg/kg diluted to 10 ml NS given over 10 min before induction. This response might be due to the use of a lower dose of dexmedetomidine in their study. Our results are consistent with this study. Our study showed a maximal average increase of 5.7% and 14.26% in SBP and 0.7% and 20.25% in DBP in group D and group N, respectively, during laryngoscopy and tracheal intubation. This increase was significantly higher in group N compared to group D. Difference found in both the groups was statistically significant. Thus attenuation in SBP and DBP was seen in group D as compared to group N during laryngoscopy and tracheal intubation, which was statistically significant.

Yildiz M et al (2006),<sup>6</sup> observed that an increase in blood pressure after intubation was significantly low in the dexmedetomidine group (p<0.05) compared to the placebo group. Dexmedetomidine group had received a single pre-induction intravenous dose of dexmedetomidine 1mcg/kg. Our results are consistent with this study. Our study showed that an increase in HR, SBP, DBP, and MAP at 2 min, 5 min, and 10 min after the creation of pneumoperitoneum was significantly higher in group N compared to group D. Difference found in both the groups was statistically significant. Thus attenuation in HR, SBP, DBP, and MAP at 2 min, 5 min, and 10 min after the creation of pneumoperitoneum was seen in group D as compared to group N, which was statistically significant. In the study done by Shah Vandana et al (2015),<sup>12</sup> in the study done by Shah Vandana et al (2015)<sup>31</sup>, Group D (n=25) received dexmedetomidine 1mcg/kg/min loading dose followed by 0.2-0.7 mcg/kg/hr infusion titrated to maintain BIS value between 40-60 and vitals. Group P (n=25) received 25-75mcg/kg/min Propofol infusion titrated to maintain BIS value between 40-60 and vitals. They found that dexmedetomidine causes attenuation of hemodynamic response to laryngoscopy and pneumoperitoneum. Also, maintain the depth of anesthesia better than propofol. Our results are consistent with this study.
Group E received a bolus injection of 500 mcg/kg intravenous esmolol before pneumoperitoneum followed by an infusion of 100 mcg/kg/min in the study conducted by Dhrurjot Prosad Bhattacharjee et al. (2016).[13] Group D obtained 1 mcg/kg IV dexmedetomidine before pneumoperitoneum with a bolus injection accompanied by 0.2 mcg/kg/h infusion. Group S (control) provided 0.9 per cent saline. Our findings are in accordance with the study Group 1 received 2 mcg/kg of clonidine diluted in normal saline in the study performed by S Kumar et al. (2014), providing gradual intravenous infusion over 10 min until general anesthesia was induced. Group 2 obtained 1 mcg/kg of dexmedetomidine diluted in regular saline, given slow intravenous infusion more than 10 minutes before general anesthetic induction. They also found that dexmedetomidine and clonidine are effective in attenuating the pneumoperitoneum hemodynamic response with equal efficacy and without significant side effects in patients. Our conclusions are consistent with the report.

Recep Aksu et al (2009)[14] used dexmedetomidine 0.5 mcg/kg and fentanyl 1 mcg/kg, 5 min before extubation. They found that dexmedetomidine is more effective than fentanyl in attenuating hemodynamic response to extubation. Our results are partially inconsistent with this study as attenuation in SBP was not statistically significant in group D compared to group N. which might be so because we stopped dexmedetomidine infusion at the time of the release of pneumoperitoneum, while in the above study, dexmedetomidine was given 5 min before extubation. In our study, the average sedation score at 5 min, 15 min, 30 min, and 60 min post-extubation was significantly higher in group D compared to group N. Difference found in both the groups was statistically significant. Postextubation, patients were more comfortable in group D compared to group N.

In our study average sedation score at 5 min, 15 min, 30 min, and 60 min post extubation is significantly higher in group D compared to group N. The average pain score (VAS) after 5 min of extubation is significantly higher in group N compared to group D. Yildiz et al (2006) found that patients in the dexmedetomidine group had sedation levels of 3 and 4 at 10 min. Our findings are consistent with this analysis as in most patients in group D at 5 and 15 min after extubation in our study a sedation level was 2 and 3. Our result The average time to rescue analgesia is significantly higher in group D compared to group B showing the benefits of the analgesic effect of dexmedetomidine is consistent with Varshali Keniya et al (2011),[15] observed that intraoperative fentanyl requirement was 100±10 mcg in the control group and 60±10 mcg in the dexmedetomidine group. Control group had received isoflurane-opioid and study group had received isoflurane-opioid-dexmedetomidine (1 mcg/kg dexmedetomidine was given over 10 min before the induction of anesthesia and was continued in a dose of 0.2-0.7 mcg/kg/hr to keep the hemodynamic parameters within acceptable range till the start of skin closure) in their study. S Kumar et al (2014),[16] also found that the mean sedation level of the dexmedetomidine group at the time of extubation (2.93±0.50) was significantly higher than that of the clonidine group (1.60±0.50), and patients were more comfortable during the postoperative period. Our result is consistent with the analysis.

**Conclusion**

Laparoscopic surgery is associated with haemodynamic stress response to intubation, pneumoperitoneum and extubation. These insults can be disastrous in high risk patients especially those with cardiopulmonary diseases. Hence, it is of utmost importance to maintain haemodynamic stability in these patients.

In our analysis, we concluded that 0.4 mcg/kg/hr infusion of dexmedetomidine started 10 minutes before activation of pneumoperitoneum anaesthesia without any dose of bolus; it serves as a very useful anaesthesia adjuvant to attenuate the response of haemodynamic stress to patients with intubation, pneumoperitoneum, and extubation patients undergoing laparoscopic surgery. This also increases postoperative sedation and reduces postoperative analgesic needs, without significant side effects.

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