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# A Study On Effect Of Intravenous Infusion Of Dexmedetomidine On Perioperative Hemodynamics In General Anesthesia

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***Abstract: Dexmedetomidine is a new intravenous drug gaining popularity in anesthesia because it offers a unique cooperative sedation,anxiolysis and analgesia with no respiratory depression.Our objectives for current study were to evaluate the efficacyof dexmedetomidinein providing better hemodynamic stability intraoperatively without causing sedation. We recruited 50 patients undergoing elective laproscopic surgery, divided them randomly into group D and group C with 25 patients in each group. D group was given dexmedetomidine and control group received 0.9% saline. We find heart rate and mean blood pressure among D group individuals remained stable throughout intraoperative period. (p<0.05). Sedation was minimal and it did not affect the recovery time. So we conclude dexmedetomidine provides excellent hemodynamic stability.***

## INTRODUCTION

Dexmedetomidine is a relatively new intravenous drug gaining popularity in anesthesiology and critical care practice. Dexmedetomidine produces dose-dependent sedation, anxiolytic, and analgesia (involving spinal and supraspinal sites) without respiratory depression.[1, 2]

In recent studies, dexmedetomidine has been shown to have clinically significant effects on anesthetic requirements and hemodynamic responses induced by anesthesia and surgery in patients.[3]Dexmedetomidine significantly attenuated the haemodynamic responses to intubation and the emergence from anaesthesia.[4] In addition, it maintained intraoperative cardiovascular stability. Patients receiving dexmedetomidine had their tracheal tubes removed faster than those in the placebo group, indicating preserved respiratory function. Continuous infusion of dexmedetomidine had been shown to improve hemodynamic stability in patients undergoing surgery without increasing the incidence of hypotensive episodes or bradycardia. In addition, patients treated with dexmedetomidine were discharged from the post anesthesia care unit earlier.[5]

So we decided to evaluate the usefulness of the anti-nociceptive and sympatholytic properties of dexmedetomidine.

## AIMS AND OBJECTIVES

- To assess the effect of dexmedetomidine on the maintenance of hemodynamic stability.
- To evaluate sedation and postoperative recovery characteristics when dexmedetomidine is used as an anesthetic adjuvant.
- To evaluate the provision of condition that facilitates surgical exposure.

## MATERIAL AND METHODOLOGY

❖ SOURCE OF DATA AND METHOD OF COLLECTION OF DATA (INCLUDING SAMPLING PROCEDURE IF ANY):

Present study conducted on 50 patients aged 35 to 65 who were scheduled for laparoscopic abdominal surgery in Civil Hospital, Ahmedabad, attached to B.J. Medical College, Ahmedabad, Gujarat.

50 patients were randomized to one of the two arms based on computer generated random numbers after taking informed written consent from their relatives. Written approval from institutional ethics committee was also taken before commencement of the study.

- Group D to receive dexmedetomidine infusion (n =25)
- Group C to receive 0.9% sodium chloride as control (n =25)

❖ INCLUSION CRITERIA:

- Patients aged 35 to 65 years
- American society of anesthesiology (ASA) l &ll
- Scheduled for laparoscopic abdominal surgery.
- Preoperative Glasgow coma scale (GCS) 15

❖ EXCLUSION CRITERIA:

- Patients using α2-adrenergic receptors antagonists or any other antihypertensive drugs.
- Patients having dysrhythmia by ECG.
- Body weight more than 100 Kg
- Height less than 150
- Heart rate <50/min, first or second- or third degree heart block.
- Patients having cardiac dysfunction, hepatic or renal disease,
- Known allergy to dexmedetomidine.
- Participation in another drug study during the preceding 1 month.
- Patient's refusal

❖ STUDY DESIGN:

It is prospective randomized blinded placebo controlled trial.

❖ METHODOLOGY :

All the patients underwent a detailed pre anesthetic check-up before surgery and all the routine and specific investigations were noted. The patients were electively kept nil per oral for 6 hours before surgery and prior to operation patients were explained about the procedure. After the patient was shifted to the theatre, standard monitors like ECG, NIBP, and pulse oximetery were applied. Intravenous line secured with one 18 gauge cannula and another 20 gauge for infusion of study drug in all the patients and intravenous fluid was started.

- MATERIAL:

Precede ™, 2 ml ampoule (Manufactured by Hospira Wellness Limited).
Each ml contains Dexmedetomidine hydrochloride injection equivalent to Dexmedetomidine……..100µg. This 2ml volume was diluted with 98 mL of normal saline to yield a final concentration of 2 µg/mL. For each patient in Group P, 100 mL volume of 0.9% saline solution was prepared.

- PREMEDICATION:

All patients were preoxygenated with 100% oxygen for 5 min using Bain's circuit and received,

Inj. Ondansetron 0.15 mg/kg IV
Inj. Glycopyrrolate 0.004 mg/kg IV
Inj. Fentanyl 1 µg/kg IV to group D and 1 µg/kg IV to group P

- Induction:

Achieved with thiopentone 3 to5 mg/kg IV. Vecuronium0.1 mg/kg IVwas given to facilitate tracheal intubation.

- Maintenance:

It was achieved with 0.5 to 2% (end-tidal concentration) sevoflurane in 60% nitrous oxide and 40% oxygen. In each case, the aim was to maintain mean arterial blood pressure (MAP) within 80–120% of baseline values. Mean arterial blood pressure rise of more than 20% above baseline was treated by administering a 2 µg/kg IVbolus of fentanyl and raising the end-tidal sevoflurane concentration to 2%. Mean arterial blood pressure drop of more than 20% below baseline was treated initially with reduction of the end-tidal sevoflurane concentration to 0.5%. Supplemental boluses of vecuronium 0.05 mg/kg

IVwere administered as required maintaining muscle relaxation during surgery. Each Group D patient received an initial loading dose of dexmedetomidine 1 µg/kg over 30 min prior to induction, followed by an infusion started at 0.5 µg/kg/hr. The infusion was discontinued when surgery ended. Group C patients received the same volume of 0.9% saline as a sham loading dose, followed by a saline infusion. Heart rate, SpO2, and MAP were recorded at specific time points (zero, five, 15, 30, 60, 90 and 120 min) during the surgical procedure. The total amount of fentanyl administered during each operation was also recorded.

Upon completion of surgery, each patient was extubated when they were able to execute simple verbal commands. All subjects were transferred to the post anesthesia care unit (PACU), where they were monitored and received nasal O2 supplementation.

Each patient remained in the PACU for two hours, and was transferred thereafter to the ward. Data for heart rate, MAP, and sedation scores were recorded at 10, 20, 30, 40, 50, 60, 90 and 120 min in the PACU, at arrival on the ward, and at 3, 4, 8, 12, 16 and 24 hour postoperatively. Each subject's mean SpO2 value in the PACU was also recorded. Observers who recorded data were blinded with respect to patients' group allocation. The observer was never the anesthesiologist providing clinical care of the patient. The degree of sedation was assessed using the 'Ramsay sedation scale.' In this system,

1 = agitated and uncomfortable,
2 = cooperative and orientated,
3 = can follow simple directions,
4 = asleep but strong response to stimulation,
5 = asleep and slow response to stimulation, and
6 = asleep and no response to stimulation.

Times to extubation of the trachea, and side effects possibly related to opioid or dexmedetomidine administration [nausea and vomiting, urinary retention, bradypnea (fewer than 8 breaths/min), and itching] were recorded for each case.

## STASTICAL ANALYSIS:

Results obtained from the study were expressed in mean +/- SD, for numeric data 'T' test was applied. A "p" value less than 0.05 was considered as statistically significant

## RESULTS

After studying 50 cases, the observations and results were summarized in tabulated form. The patients were divided into two groups with 25 patients in each group (n=25).

**Table 1: Demographic profile of the Dexmedetomidine (D group) and Control (C group) Groups:**

| Parameter | D group (n=25) (mean ± SD) | C group (n=25) (mean ± SD) |
|---|---|---|
| Age (years) | 46.96 ± 6.09 | 45.68 ± 6.46* |
| Weight (Kg) | 60.36 ± 7.06 | 62.28 ± 6.47* |
| Sex (male: female) | 11:14 | 12:13* |
| ASA I/ ASA II | 12:13 | 11:14* |
| Duration of surgery( min) | 149.16 ± 12.82 | 153.92 ± 14.40* |

*p>0.05

**Table 2: Comparison of HEART RATE between two groups at baseline, induction, after intubation, during surgery and after surgery:**

| Timings | | D group (mean ± SD) | C group (mean ± SD) |
|---|---|---|---|
| Baseline | | 76.84 ± 8.234 | 71.2 ± 7.405 |
| Induction | | 76 ± 8.06 | 75.2 ± 7.67 |
| Intubation | 1min | 76.12 ± 7.90 | 86.48 ± 3.41* |
| | 5min | 71.28 ± 8.08 | 86.2 ± 3.66* |
| Intraoperative | 15min | 69.4 ± 7.93 | 79.36 ± 3.70* |
| | 30 | 68.76 ± 7.94 | 79.96 ± 4.49* |
| | 60 | 67.56 ± 8.13 | 81.84 ± 3.62* |
| | 90 | 67.24 ± 7.46 | 82.36 ± 3.01* |

| | | | |
|---|---|---|---|
| | 120 | 67.20 ± 8.88 | 82.04 ± 2.86* |
| | 150 | 66.88 ± 9.0 | 82.6 ± 2.66* |
| | 180 | 66.56 ± 8.45 | 82.08 ± 2.05* |
| Postoperative | 10min | 78 ± 8.64 | 75.2 ± 7.65 |
| | 30min | 77.9 ± 7.62 | 75.6 ± 7.59 |
| | 60 min | 78.08 ± 8.36 | 75.9 ± 7.99 |
| | 90 min | 77.76 ± 7.31 | 77.16 ± 8.17 |
| | 120min | 79.96 ± 8.57 | 76.68 ± 8.33 |
| | 6hr | 81.48 ± 9.12 | 76.04 ± 7.97 |
| | 12hr | 82.6 ± 9.30 | 77.2 ± 8.99 |
| | 18hr | 82.7 ± 8.19 | 76.64 ± 8.0 |
| | 24hr | 85 ± 7.88 | 77.2 ± 8.36 |

*p<0.05

**Table 3: Comparison of MEAN BLOOD PRESSURE (MBP) between two groups at baseline, induction, after intubation, during surgery and after surgery:**

| **Timings** | | **D group (mean ± SD)** | **C group (mean ± SD)** |
|---|---|---|---|
| Baseline | | 76.84 ± 8.234 | 71.2 ± 7.40 |
| Induction | | 72.48 ± 9.83 | 74.56 ± 8.37 |
| Intubation | 1min | 73.96 ± 9.88 | 81.12 ± 8.58* |
| | 5min | 73 ± 9.09 | 79.04 ± 7.44* |
| Intraoperative | 15min | 68.92 ± 9.51 | 78.76 ± 7.99* |
| | 30 | 69.32 ± 8.94 | 78.32 ± 8.11* |
| | 60 | 68.6 ± 9.18 | 77.48 ± 8.27* |
| | 90 | 68.72 ± 9.01 | 79.96 ± 7.97* |
| | 120 | 68.76 ± 9.59 | 79.76 ± 8.08* |
| | 150 | 68.92 ± 9.78 | 79.84 ± 7.94* |
| | 180 | 71.64 ± 8.39 | 84.48 ± 7.80* |
| Postoperative | 10min | 75.24 ± 7.41 | 76.4 ± 7.77 |
| | 30min | 75.68 ± 7.36 | 77.76 ± 7.64 |
| | 60 min | 74.88 ± 7.87 | 77.76 ± 8.27 |
| | 90 min | 75.96 ± 6.91 | 78.12 ± 8.41 |
| | 120min | 77.56 ± 6.80 | 78.96 ± 7.67 |
| | 6hr | 77.8 ± 7.09 | 79.36 ± 7.93 |
| | 12hr | 78.24 ± 6.88 | 78.96 ± 7.3 |
| | 18hr | 77.84 ± 6.36 | 79.76 ± 7.42 |
| | 24hr | 77.76 ± 6.75 | 80.12 ± 7.97 |

*p<0.05

**Table 4: Postoperative Ramsay sedation score (RSS):**

| **Timings (postoperative)** | **D Group RSS score (mean ± SD) (n=25)** | **C group RSS score (mean ± SD) (n=25)** |
|---|---|---|
| 10min | 4.88 ± 0.37 | 3.44 ± 0.65* |
| 30min | 4.48 ± 0.47 | 3.72 ± 0.54* |
| 60min | 4.08 ± 0.64 | 2.52 ± 0.51* |
| 90min | 3.56 ± 0.58 | 2.08 ± 0.27* |
| 120min | 2.56 ± 0.50 | 2.04 ± 0.2* |
| 6hr | 2.44 ± 0.82 | 1.4 ± 0.5* |
| 12hr | 1.92 ± 0.49 | 1.2 ± 0.40* |
| 18hr | 1.32 ± 0.50 | 1.24 ± 0.43 |
| 24hr | 1.16 ± 0.72 | 1.44 ± 0.50 |

*p<0.05

**Table 5:Comparison of $SPO_2$ between two groups at baseline, induction, after intubation and during surgery:**

| Timings | | D group (mean ± SD) | C group (mean ± SD) |
|---|---|---|---|
| Baseline | | 98.2 ± 0.76 | 98.04 ± 0.88* |
| Induction | | 98.2 ± 0.81 | 98.44 ± 0.65* |
| Intubation | 1min | 98.32 ± 0.80 | 98.16 ± 0.74* |
| | 5min | 98.44 ± 0.71 | 98.44 ± 0.71* |
| Intraoperative | 15min | 98.52 ± 0.77 | 98.44 ± 0.82* |
| | 30 | 98.16 ± 0.80 | 98.16 ± 0.80* |
| | 60 | 98.04 ± 0.61 | 98.04 ± 0.61* |
| | 90 | 98.20 ± 0.70 | 98.24 ± 0.66* |
| | 120 | 98.48 ± 0.71 | 98.48 ± 0.71* |
| | 150 | 98.48 ± 0.71 | 98.48 ± 0.71* |
| | 180 | 98.64 ± 0.56 | 98.60 ± 0.57* |

*p>0.05

## DISCUSSION

In this prospective, randomized, placebo control study, as the table 1 shows both groups were comparable to various demographical parameters. There was no significant difference in age, sex, weight, ASA and duration of surgery.

From observation it can be seen that baseline hemodynamic parameters (pulse rate and mean blood pressure) were comparable in both the groups.

After administration of loading infusion (induction) there is a decrease in pulse rate and blood pressure in Group D but no such decrease is seen after saline infusion. This reduction in pulse rate and mean blood pressure from baseline values after induction is statistically significant. Statistically significant Difference in the pulse rate and mean blood pressure between the two groups was also found during intraoperative period. (Table 2, 3). However no significant difference found during postoperative period.

Nevriye Salman, Sennur Uzun, Fehmi Coskun, (2009)[6] and Vaddinenijagadish (2014)[7] have observed similar effects of dexmedetomidine on hemodynamics.

Bloor BC, Ward DS [8] et al have observed a biphasic effect on hemodynamics after intravenous dexmedetomidine in humans, an immediate increase in blood pressure(mediated by stimulation of peripheral stimulation of peripheral α-2 adrenoreceptors) followed by a longer lasting reduction in pressure caused by stimulation of α-2 adrenoceptors in central nervous system. Initial pressure effect is influenced by rate of intravenous infusion. They have observed this effect after giving 2μg/kg over 2 min period. However, we did not get any such biphasic response by giving dexmedetomidine over 5min.

Neusa Maria H. Bulow, Nilda Vargas Barbosa et al [9] also shows better hemodynamic stability with demedetomidine which enhance surgical outcome. Another study by Aanta R, Scheinin M. [10] and Hayashi Y, Maze M[11] shows centrally acting α2-adrenergic agonists including dexmedetomidine activate receptors in the medullary vasomotor center, reducing norepinephrine turnover and decreasing central sympathetic outflow, resulting in alterations in sympathetic function. it provide better hemodynamic and adrenergic stability via sympatholytic action, sedation, anxiolysis, decreased anesthetics and analgesic consumption and attenuation of opioid-induced muscle stiffness, without marked ventilatory depressing effects. Sympatholytic action of α2-adrenergic agonists is not related to changes in neurotransmitter synthesis, storage or metabolism, and is reversible with vasoactive agents, antagonists of these receptorsor simply by withdrawing the drug. Further dexmedetomidine decreases the need for anesthetics as well as attenuates adrenergic response to tracheal intubation.

Postoperative sedation score was assessed at regular intervals using Ramsay Sedation Scale. From table 4 observations, we can infer that Dexmedetomidine did cause some amount of sedation, but the sedation was not significantly different from the saline group. It did not cause

any delay in recovery of the patient from anesthesia. The sedation was also not that severe so as to warrant any interference from our side.

The results in our study were similar to one study of D.P.Bhattacharjee, SushilNayek et al [(12)] where they found that Dexmedetomidine caused sedation but did not cause any delay in recovery time. In another study of Judith E. Hall, toniUhrich et al[(13)] it was found that small dose of Dexmedetomidine infusions caused sedation, impairment of memory and psychomotor performance. While we did find some amount of sedation by Dexmedetomidine in the course of our study, we did not find any memory loss or psychomotor impairment in any of our patients. Oxygen saturation was maintained throughout the surgical period among both the groups and no difference found. (Table 5)

## SUMMARY AND CONCLUSION

- Intravenous infusion of dexmedetomidine doesattenuate the sympathetic hemodynamic response and stabilizes it, thereby facilitate surgical exposure.

- Dexmedetomidine produces some amount of sedation in the patients, but the sedation was not significant enough to delay recovery from anesthesia

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