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Laparoscopic surgeries- Effect of two different iv doses of Dexmedetomidine on hemodynamic response

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Abstract

Background: In laparoscopic surgeries, pneumoperitoneum results in tachycardia, hypertension, and increased myocardial oxygen demand. These changes are more pronounced in hypertensive patients. The intravenous administration of dexmedetomidine attenuates sympathoadrenal response and provides better hemodynamic stability intraoperatively.

Aims: To evaluate the hemodynamic stabilizing properties of two different doses of dexmedetomidine including 0.6 $\mu g.kg^{-1}.h^{-1}$ and 0.2 $\mu g.kg^{-1}.h^{-1}$ in patients undergoing laparoscopic surgeries.

Methods:A total of 60 patients posted for laparoscopic surgeries under general anesthesia were randomly assigned into two groups of 30 each. Group A and B received loading dose of dexmedetomidine 1 $\mu g.kg^{-1}$ over 10 min and maintenance dose at 0.2 and 0.6 $\mu g.kg^{-1}.h^{-1}$, respectively. Hemodynamic parameters (heart rate and mean arterial pressure) and intra operative propofol requirement were monitored.

Results: Fluctuations in the hemodynamics of patients were effectively attenuated by dexmedetomidine and there is no difference in the attenuation of these hemodynamic changes by maintenance dose of 0.2 or 0.6 $\mu g.kg^{-1}.h^{-1}$.

Conclusions: Dexmedetomidine administered as infusion in a maintenance dose of 0.6 $\mu g.kg^{-1}.h^{-1}$ serves as an ideal anesthetic adjuvant in patients undergoing laparoscopic surgeries.

Keywords: Anesthesia, dexmedetomidine, laparoscopic, hemodynamic

Introduction

Pneumoperitoneum can cause increased plasma levels of catecholamines and vasopressin. Elevation of intra abdominal pressure can cause various effects on cardiovascular system like elevated arterial pressure and increased systemic and pulmonary vascular resistance leading to hypertension and tachycardia.[1] Opioids, α2 agonist, beta blockers are used for reducing stress response induced by pneumoperitoneum.[2] Fentanyl, a narcotic drug which acts on 'μ' opioid receptor responsible for analgesia and sedation and is used in the operating room and sometimes in the ICU for anaesthetic and analgesic implications.[3]The α2 agonists modulate sympathetic nervous system by reducing the hemodynamic response produced by surgical stimuli thereby inhibiting stress response. Clonidine is α2 agonist and has the property of sympatholytic, whereas dexmedetomidine is selective α2 adrenergic agonist and its α2 sensitivity is 8 times that of α1.[4] Evidence has been found to associate prolonged laparoscopic procedures with increased stress responses. Dexmedetomidine has sedative, analgesic and sympatholytic effects which limits the release of renin and thus provides hemodynamic stability.[5,6,7] Dexmedetomidine has been shown to decrease the levels of cortisol.[8] This blunts many of the cardiovascular responses seen during the perioperative period and also attenuates the hemodynamic stress response to tracheal intubation and extubation.[9] The sedative and analgesics effects are mediated by α2 receptors in brain and spinal cord. When used intraoperatively, it reduces intravenous and volatile anaesthetic requirements, when used postoperatively it reduces concurrent analgesics and sedative requirements. Rapid administration may elevate blood pressure, but hypotension and bradycardia can occur during ongoing therapy. Hence, Dexmedetomidine may serve the role of an ideal pharmacological adjuvant to obtund these hemodynamic response to laryngoscopy, intubation, pneumoperitoneum without having any adverse effects and it also reduces opioid requirements and stress response to surgery ensuring a stable haemodynamic state.[10] Our aim was to compare the efficacy of two different doses of IV Dexmedetomidine- 0.2 μg/kg/hr infusion after 1μg/kg IV bolus and 0.6 μg/kg/hr infusion after 1μg/kg IV bolus in attenuating the hemodynamic stress responses and intra op propofol requirement in laparoscopic surgeries.

Methods

After obtaining institutional ethical committee clearance and written informed consent from the patients,study was conducted in 60 patients of 18-60 years age group with ASA I and ASA II grade of either sex undergoing laparoscopic surgeries under general anaesthesia at a tertiary care hospital.This is a hospital based prospective single blinded randomized comparative study conducted between November 2020-November 2021. 60 Patients are randomly divided into 2 groups of 30 patients each by using computer formulated randomization technique (www.sealedenvelope.com) **Group A**: Patients received Dexmedetomidine infusion at 0.2 μg/kg/hr infusion after 1μ/kg IV bolus **Group B:** Patients received Dexmedetomidine infusion at 0.6 μg/kg/hr infusion after 1μ/kg IV bolus. Pre anaesthetic evaluation was done and written informed consent was obtained 1 day prior to the surgery. 60 patients satisfying inclusion criteria were randomly allocated into two groups by computerized randomization table. All patients were fasted for 8 hours before the planned surgical procedure. On the day of surgery all patients were shifted to the operating room and a peripheral intravenous line was secured using 18 G IV cannula. Infusion of the study medication was prepared (100 μg of Dexmedetomidine added to saline to achieve a total volume of 50ml – 2 μg / ml preparation). Bolus of 1μg/kg given to all patient and then infusion of 0.2μg/kg/hr for Group A and 0.6μg/kg/hr for Group B was

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started.Preoxygenation with 100% oxygen for 3 minutes was started 7 minutes after the commencement of infusion medication. Anaesthesia was induced with propofol 2mg/kg IV till the loss of verbal response as the end point. Inj. succinylcholine 2mg/kg IV given to facilitate tracheal intubation. After one minute of manual ventilation, patients was intubated.After confirmation by 5 points auscultation and Endtidal carbon dioxide (ETCO2),isoflurane 0.6 % end-tidal concentrations level along with nitrous oxide 66% and oxygen 33% mixture was given to maintain adequate depth of anaesthesia and muscle relaxation was achieved by Inj. vecuronium 0.6mg/kg IV loading dose. Anaesthesia was maintained with nitrous oxide 66%, oxygen 33% mixture with isoflurane. With controlled Ventilation was adjusted to maintain EtCO2 between 30 and 40 mmHg. Muscle relaxation was achieved by administration of Inj. Vecuronium ¼ of loading dose during operation as needed.MAP and heart rate values were maintained within 20% of baseline values. Hypotension (defined as MAP < 20 % of baseline values on two consecutive readings within 2 – 3 min), was first treated by decreasing the isoflurane end tidal concentration and by administering fluid bolus of 200ml. Hypotension not responding to the above two interventions, if required than it was treated with Inj. Ephedrine in bolus doses of 6 mg IV. The infusion of study medication was stopped if hypotension persists > 3 min after these interventions. In the presence of hypertension (defined as MAP value > 20 % of baseline values on two consecutive readings within 2 – 3 min) and / or tachycardia (defined as HR > 20 % of baseline values > 2 min). Hypertension and tachycardia were treated by increasing the end tidal concentrations of isoflurane in steps of 0.2% up to a maximum end tidal concentration of 2%. Hypertension and tachycardia not responding to the above interventions were treated by administration of propofol in bolus doses of 25mg-50mg IV.During the surgical procedure patients received IV crystalloid solutions calculated according to the Holliday Segar formula (4-2-1 formula).Infusion of Dexmedetomidine was discontinued upon desufflation of abdomen. Isoflurane was terminated at the start of fascial layer closure and
nitrous oxide was discontinued after skin closure. After wound closure residual neuromuscular block was reversed with Inj. neostigmine 0.04 mg / kg IV and glycopyrrolate 0.01 mg / kg IV, once the patients started breathing spontaneously. When spontaneous respirations were adequate and patients were able to obey simple commands, oropharyngeal suctioning was done and tracheal extubation was performed. Heart rate (HR), mean arterial pressure (MAP), and no of times intermittent propofol bolus required for stabilization of haemodynamic response were measured.

Results

Maximal increase in heart rate in both the groups occurred following laryngoscopy and endotracheal intubation. The increase in mean heart rate in Group A was from 85 to 101 beats per minute while in Group B mean heart rate increased from 91 to 95 beats per minute during endotracheal intubation with $p= 0.014$ i.e. $p<0.05$(using independent t-test). Hence, it was found statistically significant. It indicates that attenuation of HR was superior in Group B as

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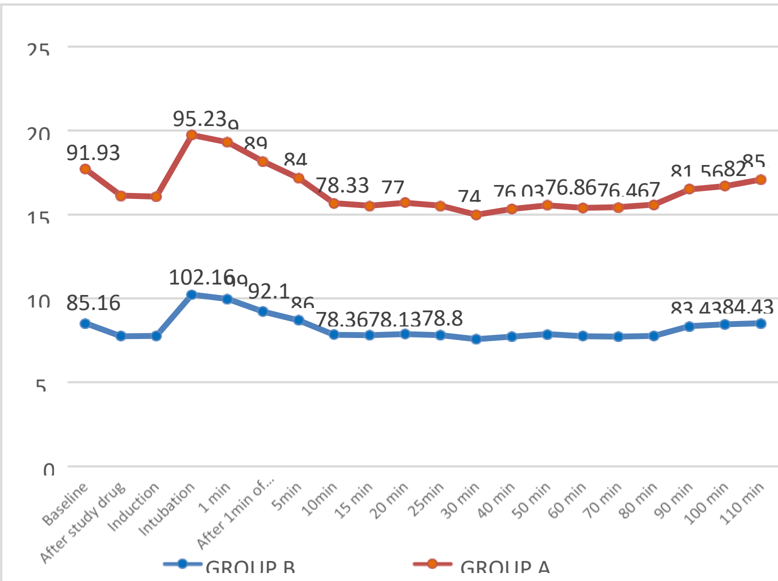
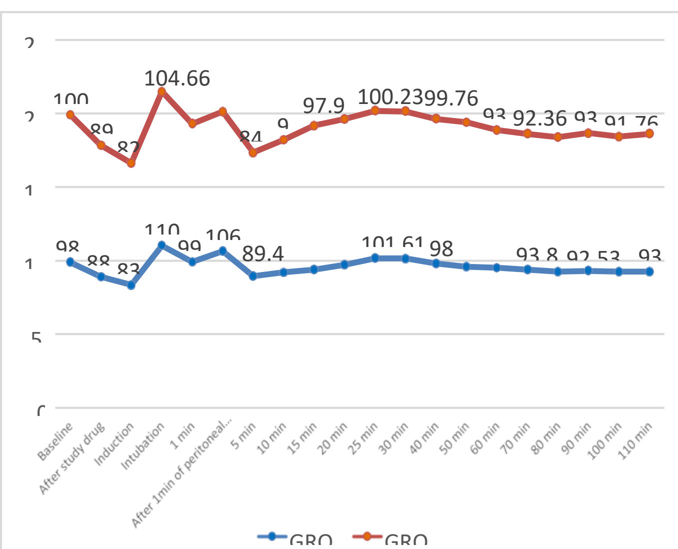
compared to Group A.(fig 1)

Fig 1 : Comparison of Heart Rate between Group A and Group B



Maximal increase in MAP occurred following endotracheal intubation and peritoneal insufflation with p=0.29 and p=0.00 respectively i.e. p<0.05 (using independent). Hence, the increase in MAP was statistically significant only during endotracheal intubation and peritoneal insufflation.It indicates that attenuation of MAP was superior in Group B as compared to Group A.(fig 2)

Fig 2: Comparison of mean arterial pressure (mmHg) between Group A and B



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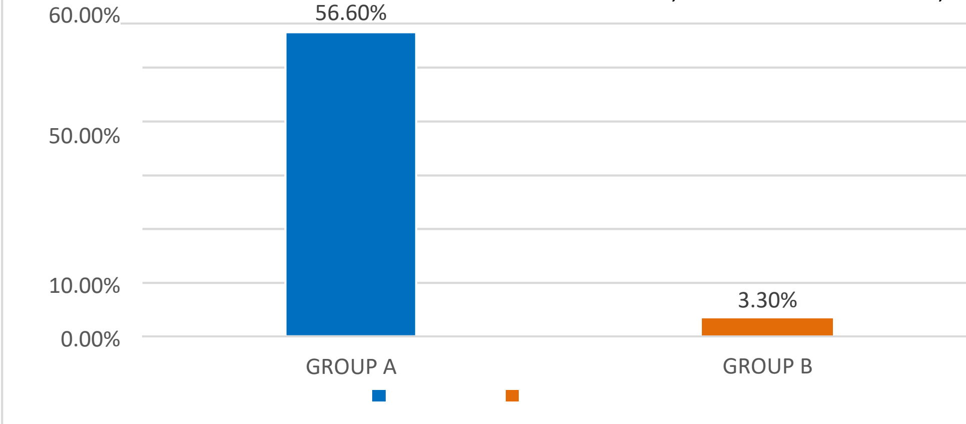

Fig 3: No. of times Intermittent Propofol bolus required in group A and B

The total number of times bolus doses of propofol required in Group A was 17.The total number of times bolus doses of propofol required in Group B was 1. On analysing the data statistically, the p value was calculated as p=0.000(usingChi-square test) for total number of bolus doses of propofol required in GroupA and Group B.(fig 3)

Discussion

In our study, comparison of intravenous dexmedetomidine (0.2µg/kg/hr) infusion after 1µg/kg IV bolus with dexmedetomidine (0.6µg/kg/hr) infusion after 1µg/kg IV bolus, was done in attenuating hemodynamic responses in laparoscopic surgeries. The result of the present study demonstrated that dexmedetomidine (0.6µg/kg/hr) infusion after 1µg/kg IV bolus over 10 minutes prior to intubation attenuated the rise in Heart rate and blood pressure better than dexmedetomidine (0.2µg/kg/hr) infusion after 1µg/kg IV bolus. The study revealed that there was statistically significant difference in the mean heart rate at intubation and 1min after intubation with p = 0.014 and p = 0.016 respectively. Once endotracheal intubation was performed, there was a rise in heart rate in both the groups, of which the rise in heart rate in group B was found to be least when compared to other group A and it was statistically significant as (p value < 0.05). None of the patients in both the groups encountered a significant bradycardia with a heart rate of less than 50bpm.Shamim et al[11] showed that a preinduction intravenous dose of dexmedetomidine 1 µg/kg, decreased the need for thiopental and sevoflurane by 39% and 92% respectively, and effectively blunted the hemodynamic responses to laryngoscopy. However our study also include a preinduction bolus dose of dexmedetomidine and better attenuation of hemodynamic response to laryngoscopy and intubation were observed in Dexmedetomidine at an infusion rate of 0.6 µg/kg/hr. The study findings revealed that there was statistically significant difference in the systolic and diastolic blood pressure and mean arterial pressure over time after study drug administration i.e. immediately after intubation and peritoneal insufflation having p value (<0.05). The rise in MAP was from 98 mm Hg to 110 mm Hg in Group A while that in Group B was from 100 mmHg to 104 mmHg during intubation. Even though the increase in SBP, DBP, and MAP was observed in both the groups during endotracheal intubation, the degree of increase was lesser in Dexmedetomidine 0.6 µg/kg/hr infusion group when compared to the Dexmedetomidine 0.2 µg/kg/hr infusion group.

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Dexmedetomidine in doses of 0.6 μg/kg/hr infusion had greater hemodynamic attenuation properties during endotracheal intubation when compared with doses of 0.2 μg/kg/hr infusion.

The use of intraoperative dexmedetomidine may provide hemodynamic stability due to the attenuation of stress-induced hemodynamic response to intubation, peritoneal insufflation and emergence from anaesthesia. The maximum decrease in SBP, DBP and MAP in both the groups was observed around 10 minutes following intubation and before peritoneal insufflations. More than 50 % of patients in dexmedetomidine 0.6 μg/kg/hr infusion group showed a decrease in SBP, DBP and MAP greater than 20 % from the baseline values before peritoneal insufflations, requiring administration of a fluid bolus and a decrease in volatile concentration.Ghodki et al[12] studied the hemodynamic responses to increasing concentrations of dexmedetomidine and concluded that increasing concentrations of dexmedetomidine in humans resulted in progressive increases in sedation and analgesia and progressive decreases in heart rate, cardiac output and memory. The results of this study are analogous to our study where infusion doses of 0.6 μg/kg/hr produced greater episodes of decrease in MAP when compared to an infusion rate of 0.2 μg/kg/hr. Hypertensive episodes with SBP,DBP and MAP values rising to above 20% of baseline values were observed in 17 patients in our study with 14 patients in dexmedetomidine 0.2 μg/kg/hr infusion group and 3 patients in Dexmedetomidine 0.6 μg/kg/hr infusion group .These hypertensive episodes were treated with increase in volatile concentration or propofol supplementation. The number of hypertensive episodes in dexmedetomidine 0.2 μg/kg/hr infusion group was significantly higher when compared to dexmedetomidine 0.6 μg/kg/hr infusion group. Those 14 patients in Group A who encountered intraoperative hypertension out of which 11 patients were given single bolus dose of propofol and 3 patient were given twice of bolus dose. Hypertensive episode that occurred in 3 patients in Group B out of which only 1 patient was administered with a single bolus dose of propofol. Thus concluding that dexmedetomidine in doses of 0.6 μg/kg/hr infusion has better hemodynamic stress response attenuating properties when compared to dexmedetomidine at a dose of 0.2 μg/kg/hr infusion group. Again the observations in our study are analogous to the study done by Manne et al [13] and Masoori et al [14] where they showed that progressively increasing concentrations of dexmedetomidine resulted in progressive increase in sedation and analgesia and progressive decreases in heart rate, cardiac output and memory. Thus dexmedetomidine at 0.6 μg/kg/hr infusion after bolus of 1μg/kg IV has better sympatholytic properties when compared to dexmedetomidine at doses of 0.2 μg/kg/hr infusion after bolus1μg/kg IV. The mean of total propofol required in Group A and Group B was 34mg and 30mg respectively. On analysing the data statistically, the p was significant. It indicate that Group B was superior than Group A in attenuating stress response which was also found by Vora et al [15] and Hall et al [16].

Conclusion

Dexmedetomidine at an infusion rate of 0.6 μg/kg/hr after 1μg/kg IV bolus has a better analgesic and anaesthetic sparing property and provided better hemodynamic stabilization when compared to an infusion rate of 0.2 μg/kg/hr after 1μg/kg IV bolus with no serious side effects in laparoscopic surgery.Hence, we conclude that Dexmedetomidine administered at an infusion rate of 0.6 µg/kg/hr after 1µg/kg IV bolus may serve as an ideal anaesthetic adjuvant than 0.2 µg/kg/hr after 1µg/kg IV bolus, in patients undergoing laparoscopic surgeries.

References

1.Khan A, Khan F. Haemodynamic response to induction, laryngoscopy and tracheal intubation in diabetic and non-diabetic patients. Journal of the Pakistan Medical Association.2009;59:27-30.

2.NK S, Adhikari P. Impaired heart response to deep breathing in diabetic cardiac autonomic neuropathy: Importance of agerelated normal range. Int J of Biomed & Adv Res. 2014;5:429.

3.Keet S, Bulte C, Sivanathan A, Verhees L, Allaart C, Boer C et al. Cardiovascular autonomic function testing under nonstandardisedand standardised conditions in cardiovascularpatients with type-2 diabetes mellitus. Anaesthesia. 2014;69:476-483.

4. Oakley I, Emond L. Diabetic cardiac autonomic neuropathy and anaesthetic management: Review of literature. AmericanAssociation of Nurse Anesthetists. 2011;79:473-479.

5.Mustafa H, Fessel J, Barwise J, Shannon J, Raj S, Diedrich A etal. Dysautonomia. Survey of Anesthesiology. 2012;56:102-103.

6. Huang C, Kuok C, Kuo T, Hsu Y, Tsai P. Pre-operative measurement of heart rate variability predicts hypotension duringgeneral anesthesia. Acta Anaesthesiol Scand. 2006;50:542-548.

7.Makwana DK, Mehta DHB, Gokhle DPA, Shah DCJ,Manjulajameliya D, Kalasava DK. Role of Isometric Hand Grip Test and Resting Blood Pressure Measurement In Diagnosing Cardiovascular Autonomic Neuropathy In Patients with DiabetesMellitus. NJIRM. 2012;3(4):57-60.

8. Rehman S, Ahmad R, AH A. prevalence of orthostatic hypotension among diabetic patients in a community hospital ofpeshawar. Pak J Physiol. 2010;6(2):37-39.

9.Vinik AI, Maser RE, Mitchell BD, Freeman R. Diabetic autonomic neuropathy.Diabetes Care. 2003;26(5):1553-79.

10. Krahulec B, Mikes Z, Balazovjech I. The effect of cardiovascular autonomic neuropathy on resting ECG in type 1 diabetic patients. Bratisl Lek Listy.2002;103(2):54-8.

11. Shamim R, Srivastava S, Rastogi A, Kishore K, Srivastava A. Effect of two different doses of dexmedetomidine on stress response in laparoscopic pyeloplasty: A randomized prospective controlled study. Anesth Essays Res. 2017;11:1030–4.

12. Ghodki PS, Thombre SK, Sardesai SP, Harnagle KD. Dexmedetomidine as an anesthetic adjuvant in laparoscopic surgery: An observational study using entropy monitoring. J Anaesthesiol Clin Pharmacol. 2012;28:334–8.

13. Manne GR, Upadhyay MR, Swadia V. Effects of low dose dexmedetomidine infusion on haemodynamic stress response, sedation and post-operative analgesia requirement in patients undergoing laparoscopic cholecystectomy. Indian J Anaesth. 2014;58:726

14. Masoori TA, Gupta K, Agarwal S, Bansal M, Zuberi A, Samad A. Clinical efficacy of dexmedetomidine in two different doses to attenuate the hemodynamic changes during laparoscopic cholecystectomy. Int J Res Med Sci. 2018;6:959.

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15. Vora KS, Baranda U, Shah VR, Modi M, Parikh GP, Butala BP. The effects of dexmedetomidine on attenuation of hemodynamic changes and there effects as adjuvant in anesthesia during laparoscopic surgeries. Saudi J Anaesth. 2015;9:386–92.

16. Hall JE, Uhrich TD, Barney JA, Arain SR, Ebert TJ. Sedative, amnestic, and analgesic properties of small-dose dexmedetomidine infusions. Anesth Analg. 2000;90:699–705.