Journal of Cardiovascular Disease Research

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 06, 2023

Evaluation of Effects of Premedication dose of Dexmedetomidine on Pressor Response to Laryngoscopy and Endotracheal Intubation During Elective Intrabdominal Surgery Under General Anaesthesia

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Abstract

Background: Laryngoscopy and endotracheal intubation are frequently linked to hypertension and tachycardia because of the sympathoadrenal stimulation. To combat the pressor response several medications like local anaesthetic, B blockers and opioids. Alpha 2 agonist, Dexmedetomidine (1ug/kg) has been used as premedication 10minutes prior to induction of general anaesthesia in order to assess the pressor response of laryngoscopy and endotracheal intubation. **Material and Methods:** After ethical clearance 60 patients aged 18-60 years old were randomised and divided into two groups, Group A(n=30) and group B(n=30). Group A received IV normal saline 20ml and group B received IV Dexmedetomidine, 1μg/kg diluted in 20ml of normal saline which was administered over 10 minutes and started 10 minutes prior to induction of general anaesthesia. Hemodynamic parameters were were recorded at preinduction, after induction of anaesthesia, HR, SBP, DBP and MAP were recorded at. base line (T0) after 10 minutes of the administration of the study drug (T1), after 3 minutes of the administration of the neuromuscular blocker T2, T3, T4, T5, T6, T7 and T8 recorded after 1min,2 min,3 min,5min,10min,15min and 20min after intubation. **Results:** There was significant difference in heart rate, systolic, diastolic and mean blood pressre at T1, T2, T3, T4, T5, T6, T7 and T8. ($P<0.05$). There was significant decrease in hemodynamic parametres at different time interval post laryngoscopy and induction of general anaesthesia. **Conclusion:** The pressor response to tracheal intubation and laryngoscopy was observed to be successfully attenuated by dexmedetomidine (1 μg/kg) IV, given 10 min before induction, with no major adverse effects.

Keywords: Laryngoscopy, Induction, Pressor Response, Intubation, Dexmedetomidine.

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INTRODUCTION

A period of extremely high hemodynamic stress is seen during laryngoscopy and intubation because of release of mechanical and chemical stimuli during the procedure.[1,2] In patients with end organ decompensation, pressor response can lead to intra-operative myocardial infarction, abrupt left ventricular failure, dysrhythmias, and intracranial bleeding. These responses hardly take one minute of intubation to occur and the reaction reaches its peak intensity and lasts for five to ten minutes.

These responses can be minimised gentle laryngoscopy, keeping intubation time within 15seconds and blind nasal intubations. Various pharmacological medicines, such as topical, nebulized, or systemic lidocaine, opioids, beta adrenoceptor blocking agents like metoprolol are used to decrease the pressor response.[3,4]

Alfa 2 agonists had sympatholytic action and it attenuates the hemodynamic response following laryngoscopy and endotracheal intubation. The other advantages being sedation, anxiolysis and analgesia,[2,5] and it also decreases minimum alveolar concentration (MAC) of volatile anaesthetics during general anaesthesia.[6,7]

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We hypothesized that Dexmedetomidine an alpha 2 agonist has no effect on hemodynamic effects after laryngoscopy and intubation when given 10 minutes before induction of anaesthesia.
The primary outcome was to record hemodynamic responses at various time interval during laryngoscopy and intubation.

METHODOLOGY

This Prospective Double Blinded Randomized Controlled Trial was conducted at tertiary care hospital after seeking approval from institutional ethical committee and taking written informed consent from the patient. A total of 60 patients between the ages of 18–60 years of ASA grade I/II posted for various elective intra-abdominal surgeries under general anaesthesia were included in study. This study was conducted between September 2021 to September 2022.
Patients on preoperative β blockers, angiotensin receptor antagonist, ACE inhibitors, pregnant or nursing women, Patient refusal to participate in the study, drug allergy, patients with anticipated difficult intubation and more than one intubation attempt or any history of cardiovascular, respiratory, hepatic, renal diseases were excluded from study.
The patients were randomized using chit and box method into two groups of 35 patients each the allocation was concealed by utilising sealed, opaque envelopes.
Group A (n=35) received IV Normal saline 20ml administered over 10 minutes, ten minutes before induction of general anaesthesia.
Group B (n=35) received IV Dexmedetomidine, 1µg/kg diluted in 20ml of normal saline administered over 10 minutes, started 10 minutes before induction of anaesthesia.
All patients had a pre-anesthesia assessment prior to surgery. According to the institution's regulations, all patients were brought for surgery while under general anaesthetic. Patients were induced with propofol, 2 mg/kg, fentanyl, 2 mcg/kg, and vecuronium, 0.1 mg/kg, with all American Society of Anaesthesiologists (ASA) standard monitors attached. If there was a 20% rise in HR and SBP from baseline values, intravenous fentanyl 0.25 g/kg was administered as an analgesic during the maintenance period. The patient was kept on nitrous oxide, isoflurane, and vecuronium, and the residual blockage was reversed with an intravenous infusion of 50 mcg/kg neostigmine and an intravenous infusion of 10 mcg/kg glycopyrrolate. The hemodynamic parameters were noted at various time intervals i.e. base line (T0) after 10 minutes of the administration of the study drug (T1), after 3 minutes of the administration of the neuromuscular blocker (T2), T3, T4, T5, T6, T7 and T8 recorded after 1min,2 min,3 min,5min,10min,15min and 20min after intubation respectively.
Based on similar study by Vishwanath P et al,[8] the sample size was calculated using the formula
$n= (\sigma 12 + \sigma 22) [Z1-\alpha/2 + Z1-\beta]2 /(\bar{x}1 - \bar{x}2)2$
where n is sample in each group, σ1 and σ2 is standard deviation of the variable in group A and group B respectively, Z1-α/2 and Z1-β is normal deviate value at considered level of confidence (1.96 at 95% confidence interval) and normal deviate value at considered power (0.84 at 80% power) of the study for two sided tests respectively, $\bar{x}1$ and $\bar{x}2$ is mean of variable in group A and B respectively. The minimal sample size needed for the study to have an 80 percent power was determined to be 27 based on the calculations above and earlier studies, with 0.05 being chosen as the alpha error. The number was increased to 30 in both categories after taking into account any case cancellations or block failures.
Data are expressed as the mean (standard deviation) for normally distributed data. The student's t test was used nonparametric data. Analysis of variance for repeated measures (ANOVA) was used to evaluate hemodynamic changes over different time intervals. Haemodynamic variables were compared using repeated measure of ANOVA. $p<0.05$ was considered statistically significant.

RESULTS

The consort flow diagram shows the distribution of patients from randomisation to analysis [Figure 1].
The demographic profile was comparable in both the groups [Table 1]. Heart rate continued to increase from first minute till 20 minutes in group a while in group B there was sustained decrease in heart rate and was statistically significant. [Table 2]
There was significant decrease in systolic, diastolic and mean blood pressure at all time intervals in Group B as compared to Group A except baseline preinduction value which was comparable in both the groups. [Table 3-5]

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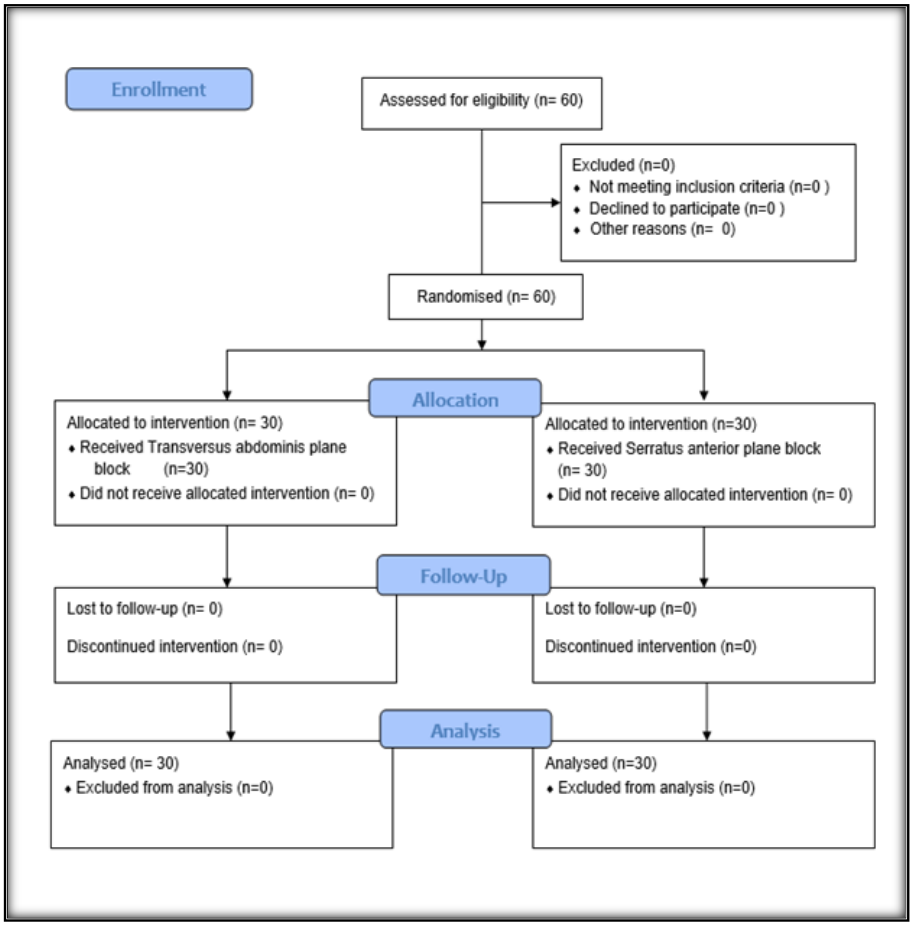

Table 1: Comparison of demographic data and of both groups

Parameter	Group Q (n=30)	Group T (n=30)	P value
Age (Years)	51.47±6.88	49.96±7.49	>0.05
Height (cm)	162.57±8.98	161.17±8.89	>0.05
Weight (kg)	58.9±4.33	59.2±6.23	>0.05
BMI (Kg/m2)	22.10±0.21	21.99±0.63	>0.05
P value <0.05 – Significant, P value >0.05—Non significant Mean±SD(Standard Deviation)			

Table 2: The distribution of mean heart rate.

Heart rate	Group A(Mean±SD)	Group B(Mean±SD)	P Value
T0	81.97±13.01	82.6±9.82	>0.05
T1	83.34±9.80	79.6±7.22	<0.05
T2	83.89±5.86	78.69±6.72	<0.05
T3	81.49±5.54	77.89±5.21	<0.05
T4	81.66±9.11	79.2±5.47	<0.05
T5	82.89±8.78	76.11±4.98	<0.05
T6	83.29±7.66	76.43±6.22	<0.05
T7	84.67±8.91	77.11±5.78	<0.05
T8	84.91±6.98	76.71±4.57	<0.05
P value <0.05 – Significant, P value >0.05—Non significant SD- Standard Deviation			

Table 3: The distribution of Systolic Blood Pressure.

Systolic Blood Pressure	Group A(Mean±SD)	Group B(Mean±SD)	P Value
T0	124.2±4.9	123.3±5.45	>0.05

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T1	121.2±7.89	125.9±5.61	<0.05
T2	119.5±9.55	115.3±5.27	<0.05
T3	120.4±8.67	115.3±7.31	<0.05
T4	118.6±8.23	113.7±7.45	<0.05
T5	118.2±5.12	112.6±5.54	<0.05
T6	115.3±9.42	110.5±6.26	<0.05
T7	117.6±8.98	111.4±6.78	<0.05
T8	118.7±9.87	113.3±5.98	<0.05
P value <0.05 – Significant, P value >0.05—Non significant SD- Standard Deviation			

Table 4: The distribution of Diastolic Blood Pressure.

Diastolic Blood Pressure	Group A(Mean±SD)	Group B(Mean±SD)	P Value
T0	78.6±7.1	78.1±5.35	>0.05
T1	78.7±7.89	81.2±7.78	<0.05
T2	76.4±3.35	72.1±5.47	<0.05
T3	78.6±8.17	73.2±6.23	<0.05
T4	79.9±7.53	74.8±7.76	<0.05
T5	77.3±9.22	74.6±6.23	<0.05
T6	75.5±5.52	71.2±5.76	<0.05
T7	80.6±7.28	76.4±8.49	<0.05
T8	78.3±6.37	75.3±6.67	<0.05
P value <0.05 – Significant, P value >0.05—Non significant SD-Standard Deviation			

Table 5: The distribution of Mean Arterial Pressure

Mean Arterial Pressure	Group A(Mean±SD)	Group B(Mean±SD)	P Value
T0	89.9±4.9	90.3±5.45	>0.05
T1	91.3±7.89	85.9±6.65	<0.05
T2	90.7±9.55	86.3±5.47	<0.05
T3	89.7±8.67	87.4±6.43	<0.05
T4	90.8±8.23	82.9±7.56	<0.05
T5	91.2±10.12	84.2±5.78	<0.05
T6	89.8±9.42	86.7±7.21	<0.05
T7	90.2±8.98	84.1±7.56	<0.05
T8	90.1±9.87	82.4±6.98	<0.05
P value <0.05 – Significant, P value >0.05—Non significant SD- Standard Deviation			

DISCUSSION

In current study, we found that there was significant decrease in heart rate, systolic, diastolic and mean blood pressure in post induction period as compared to baseline hemodynamic parameters in preinduction period.

Endotracheal intubation during general anaesthesia leads to sympatho-adrenal stimulation caused by laryngoscopy and tracheal intubation which is linked with hypertension and tachycardia. Though thyese incidence is temporary and well tolerated in health individuals but in already compromised patients can be catastrophic and can lead to myocardial ischaemia or infarction, arrhythmias, cardiac failure, aortic dissection, raised ICP, and cerebral haemorrhage. The hemodynamic response to laryngoscopy and endotracheal intubation should be reduced, and this is by far the most significant rationale for doing for conducting the current study.

To maintain hemodynamic stability throughout the intraoperative phase, the majority of practising anaesthesiologists frequently utilise opioids, vasodilators, or beta blockers.[6] High dosages of inhalational/ intravenous anaesthetics and opioids may have inevitable adverse effects, such as respiratory depression, postoperative drowsiness, and delayed recovery.[9] So, to promote an early recovery, it would be advantageous

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 06, 2023

to utilise a medication or procedure that reduces intraoperative hemodynamic alterations and also reduces the need for high concentrations or dosages of anaesthetic drugs and opioids.

The alpha2 agonist dexmedetomidine is very selective and had advantage of maintaining sedation without causing much hemodynamic stability. The sedative effect of dexmedetomidine is increased when midazolam, fentanyl, propofol, or sevoflurane are also administered.[1,10]

Dexmedetomidine has been used effectively for providing conscious sedation. Studies have shown that loading dose (1 ug/kg) of intravenous dexmedetomidine provided conscious sedation without respiratory depression or upper airway obstruction for fibreoptic nasotracheal intubation. They have proven to be very successful at reducing the hemodynamic reaction to laryngoscopy and endotracheal intubation.[13,14]

When dexmedetomidine was infused 10 min before induction of patients in a dose of 1mcg/kg was assessed in our study. Various studies used 1mcg/kg dose of dexmedetomidine without significant adverse affect leading us to select this dose for current study.[11,15-17]

Though bradycardia is a noted complication in many previous studies, it was not observed in any of our patients further empowering the selection of dose of dexmedetomidine.[18,19]

Dexmedetomidine has added advantage of keeping hemodynamics stable besides providing optimum sedation.

There were some limitation of the study. We could not use invasive monitoring which could have provided us beat to beat variation of hemodynamic changes. Perioperative analgesic consumtion was also not assessed. Further research is needed to validate the findings with large sample size.

CONCLUSION

Intravenous dexmedetomidine whe given in infusion 10 minutes before induction can provide optimum hemodynamic stability and could aptly counter stress of laryngoscopy and intubation. Without causing much adverse effects.

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