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Original Research Paper

Cardiology

"EFFECT OF DEXMEDETOMIDINE ON CHANGES IN INTRAOCULAR PRESSURE FOLLOWING SUCCINYLCHOLINE AND ENDOTRACHEAL INTUBATION'

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ABSTRACT

Aims & Objectives: The aim of this study is to evaluate the efficacy of dexmedetomidine premedication for prevention of rise in IOP and effect on hemodynamic response following administration of succinylcholine and endotracheal intubation.

Methodology: In this study 60 patients were randomly allocated into two groups of 30 patients. Group D (Dexmedetomidine): 0.4 mcg/kg/hr. Dexmedetomidine IV infusion over a period of 10 min. Group C (Control): Similar volume and rate of infusion of saline. Intra Ocular Pressure (IOP), Heart rate (HR), Systolic BP (SBP), and Diastolic BP (DBP) were recorded at predefined time intervals from T1 to T8.

Results: There was no significant difference at the baseline IOP in both the groups. After succinylcholine and intubation in Group D there was significant fall in IOP as compared to Group C ($P<0.0001$). In Group D there was significant fall in Heart rate ($P<0.05$) and MAP ($P<0.0001$) as compared to group C.

Conclusion: Dexmedetomidine premedication (0.4µg/kg) effectively prevents the rise of IOP after succinylcholine and endotracheal intubation, causes fall in IOP, HR, SBP, DBP and MAP and cause attenuation of pressor response after succinylcholine, laryngoscopy and endotracheal intubation.

KEYWORDS :: Dexmedetomidine, Succinylcholine, Endotracheal Intubation, Laryngoscopy.

INTRODUCTION

General anesthesia has been in use for ophthalmic surgery since 1847. Precise control of intraocular tension is an accepted advantage of general anesthesia. The impact of anesthetic drugs on intraocular pressure (IOP) must be considered when ophthalmic surgery is to be carried out under general anesthesia. Intravenous anesthetics and volatile agents reduce IOP; Underlying mechanisms include a direct effect on cerebral IOP control centers and indirect effects mediated through the balance between production and drainage of aqueous humor, general circulation and ocular muscle tone. IOP is likely to be elevated during induction and recovery.

IOP between 7 and 21 mmHg is considered to be statistically normal. The upper cut-off value of 21 mmHg represents 2 standard deviations above the mean IOP of 16 mmHg in a population of white individuals. The normal IOP is pulsatile, reflecting its cardio-vascular origin.

Intraocular pressure (IOP) is influenced by several factors such as central venous pressure, choroidal blood volume changes, and extra ocular muscle tonicity. Sudden increase in blood pressure, as occurred after laryngoscopy and tracheal intubation during surgical practice, results in a 10 to 20 mmHg increase in IOP. This increase in IOP can be troublesome during ophthalmic surgeries, especially in the presence of glaucoma or open eye trauma.

Emergency ophthalmic surgery poses a challenge to the anesthesiologist as patients with penetrating eye injury often present with full stomach. These patients require rapid sequence induction and intubation without increasing the intraocular pressure (IOP). Succinylcholine, the most commonly used depolarizing muscle relaxant for rapid sequence airway management, increases the IOP. Various methods have been used to attenuate the effects of succinylcholine on IOP. They include self-taming with small dose of succinylcholine, pre-treatments with non-depolarizing muscle relaxant, use of nifedipine and nitroglycerin.

Dexmedetomidine is highly selective α2-adrenoceptor agonist that has sedative and analgesic effects. A2-adrenoceptor agonists provide potentially beneficial effects in ophthalmic surgery because of their IOP lowering properties. It has already been established that dexmedetomidine infusion produces ocular hypotension in rabbits with normal and elevated IOP. α2-adrenoceptor agonists in various studies have shown a significant reduction in IOP.

Based on these observations, the present study has been done to evaluate the attenuation of succinylcholine induced rise of IOP by dexmedetomidine premedication.

METHODOLOGY

After receiving the Institutional Ethics Committee approval and informed consent, 60 adult patients of both sexes between 18-60 years of age, belonging to ASA grade I and II, without any pre-existing ocular disease, hypertension and cardiovascular disease, undergoing routine surgeries under GA were randomly allocated in two groups of 30 patients each.

1. Group D: 0.4 mcg/kg/hr. of Dexmedetomidine IV infusion over a period of 10 min.
2. Group C: Similar volume and rate of infusion of saline.

Intra Ocular Pressure (IOP), Heart rate (HR), Systolic BP (SBP), and Diastolic BP (DBP), and MAP were recorded at fixed time intervals. T1: Before premedication with Dexmedetomidine, T2: Five minutes after premedication with Dexmedetomidine, T3: Thirty seconds after thiopentone sodium, T4: Thirty seconds after succinylcholine, T5: One minute after intubation, T6: Two minutes after intubation, T7: Four minutes after intubation, T8: Six minutes after intubation.

Intra Ocular Pressure readings were recorded with Schiotz Tonometer (Riester Germany) and the scale readings thus obtained were converted to IOP with the help of Freidenwald Nomogram. Blood pressure both systolic and diastolic was recorded by non-invasive method with the help of standard size BP cuff attached to monitor. Patients were continuously monitored for arterial oxygen saturation. Any period of desaturation, gross fluctuation of blood pressure and any abnormality in the pulse rhythm was noted and recorded. Patients were continuously monitored for hypotension and bradycardia.

Exclusion criteria that were kept for these study patients were as following;

1. Patients more than 60 years of age.
2. Having hypertension with systolic BP more than 140 mm Hg.
3. Any degree of atrio-ventricular conduction abnormality.
4. With raised IOP or any other ophthalmic disease.
5. On alpha-2 blocker in the preceding 30 days.
6. Having any contraindication to dexmedetomidine, like hemodynamic instability.
7. Receiving any drug known to alter IOP.
8. Having hepatic impairment and renal impairment.
9. Pregnant and lactating women.
10. Those unable to give informed consent.

OBSERVATION TABLES

Table 1 Age wise distribution of studied cases in both groups

Age groups	Dexmedetomidine group (group D)	Control group (Group C)		
	No.	%	No.	%
15-25	3	10.0	10	33.3
26-35	13	43.3	4	13.3
36-45	6	20.0	11	36.7
46-55	6	20.0	4	13.3
56-65	2	6.7	1	3.3
Total	30	100.0	30	100.0
Mean ± SD	37.7 ± 11.9	35.5±11.8		

The mean age of the patients in group D and group C was 37.7±11.9 and 35.5±11.8 respectively. P value>0.05, both the groups are comparable.

Table 2 Changes observed in mean IOP (mm of Hg) of both the study groups

Time of observation	Group D Mean IOP ± SD	Group C Mean IOP ± SD	P value	Inference
T1	13.9 ± 2.4	12.3 ± 2.5	0.014	Significant
T2	13.8 ± 2.5	12.7 ± 2.4	0.087	Not significant
T3	14 ± 3.8	14.4 ± 2.3	0.624	Not significant
T4	12.2 ± 3.5	15.1 ± 2.0	0.0003	Highly significant
T5	9.4 ± 3.4	15.7 ± 1.8	< 0.0001	Highly significant
T6	8.5 ± 2.6	16.5 ± 1.6	< 0.0001	Highly significant
T7	8.3 ± 2.6	16.4 ± 1.5	< 0.0001	Highly significant
T8	8.1 ± 2.5	16.4 ± 1.6	< 0.0001	Highly significant

The above table shows the mean change in IOP. After premedication with Dexmedetomidine there was 41.7% drop in the IOP (95% confidence interval, 36-51%, p value <0.001).

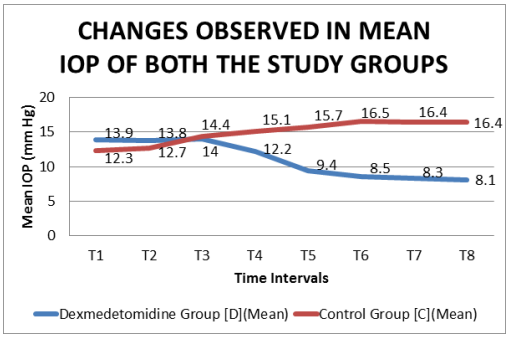


Table 3 Changes observed in Mean Heart rate (bpm) in both study groups

Time of observation	Group D Mean HR ± SD	Group C Mean HR ± SD	P value	Inference
T1	82 ± 12.9	82±10.8	> 0.99	Not significant
T2	86 ± 13.3	86 ± 11.05	> 0.99	Not significant
T3	89 ± 12.3	90 ± 11.06	0.74	Not significant
T4	86 ± 10.7	91 ± 10.24	0.069	Not significant
T5	81 ± 9.6	88 ± 11.09	0.011	Significant
T6	78 ± 10.4	88 ± 11.09	0.002	Highly significant
T7	75 ± 10.3	84 ± 11.13	0.0019	Highly significant
T8	73 ± 9.5	81 ± 10.83	0.0035	Highly significant

The above table shows the mean change in Heart Rate (HR). After Dexmedetomidine there was 10.9% drop in the Heart rate (95% confidence interval, 8-13%, p value=0.003)

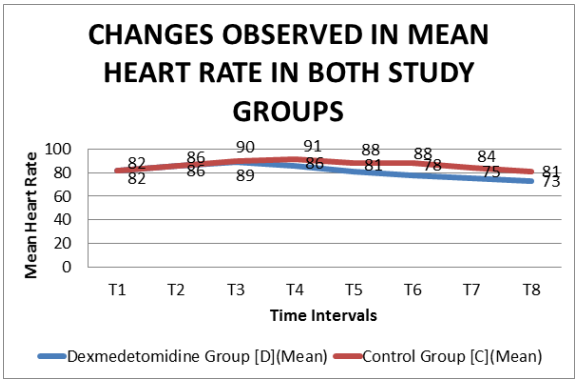
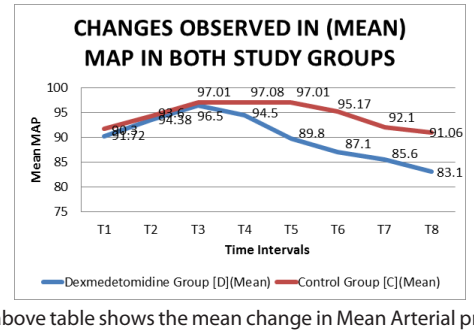


Table 4 Changes observed in (mean) MAP (mm of Hg) in both study groups

Time of observation	Group D Mean MAP ± SD	Group C Mean MAP ± SD	P value	Inference
T1	90.3 ± 7.0	91.72 ± 6.79	0.435	Not significant
T2	93.6 ± 7.6	94.38 ± 5.85	0.69	Not significant
T3	96.5 ± 7.8	97.01 ± 6.96	0.793	Not significant
T4	94.5 ± 7.4	97.08 ± 6.35	0.164	Not significant
T5	89.8 ± 6.9	97.01 ± 5.07	< 0.0001	Highly significant
T6	87.1 ± 6.7	95.17 ± 5.93	< 0.0001	Highly significant
T7	85.6 ± 6.6	92.1 ± 6.42	0.0003	Highly significant
T8	83.1 ±6.7	91.06 ± 7.11	< 0.0001	Highly significant



The above table shows the mean change in Mean Arterial pressure (MAP). After Dexmedetomidine there was 7.9% drop in the Mean

MAP (95% confidence interval, 7.7-8.3%, p value=0.0002)

RESULTS

In present study, of the total patients, 28 were males (46.67%) and 32 were females (53.33%). In group D 50% of the cases were male and the rest 50% of were females. In group C 43.3% of the cases are male and 56.7% were females. There was no significant difference between number of male and female patients in group D and group C. The male to female ratio in both the groups did not differ significantly. Thus the mean age, weight and distribution of gender and ASA class were similar in both the groups, and both the study groups were comparable.

The main aim of the present study was to evaluate the efficacy of Dexmedetomidine for prevention of rise in IOP associated with the administration of succinylcholine and endotracheal intubation & to evaluate the effect of Dexmedetomidine premedication on hemodynamic response after laryngoscopy and endotracheal intubation. Intra Ocular Pressure (IOP), Heart rate (HR), Systolic BP (SBP), and Diastolic BP (DBP) and MAP were recorded at different times. The observations were made before premedication with Dexmedetomidine (T1), Five minutes after premedication with Dexmedetomidine (T2), Thirty seconds after thiopentone sodium (T3), Thirty seconds after succinylcholine (T4) and subsequently One minute (T5),Two minutes (T6), Four minutes (T7), and Six minutes(T8) after intubation.

The mean IOP observed in Group D(in mm Hg) was (T1) 13.9 ± 2.4, (T2) 13.8 ± 2.5, (T3) 14 ± 3.8, (T4) 12.2 ± 3.5, (T5) 9.4 ± 3.4, (T6) 8.5 ± 2.6, (T7) 8.3 ± 2.6 and (T8) 8.1 ± 2.5 and the mean IOP observed in Group C was (T1) 12.3 ± 2.5, (T2) 12.7 ± 2.4, (T3) 14.4 ± 2.3, (T4) 15.1 ± 2.0, (T5) 15.7 ± 1.8, (T6) 16.5 ± 1.6, (T7) 16.4 ± 1.5 and (T8)16.4 ± 1.6 respectively. There was no significant difference at the baseline IOP in both the groups.

There was increase in the mean IOP in the Group C at T3 while in group D, the mean IOP did not change significantly from baseline at T3. At time intervals T5, T6, T7 and T8 the mean IOP progressively decreased from the baseline value in group D while in group C there was a progressive rise in the IOP. In the Group C IOP remained above the baseline even after 6 min of intubation. The mean fall in IOP in Group D when compared to Group C at T5, T6, T7 and T8 time frames was highly significant.

After succinylcholine and intubation in Group D there was significant fall in IOP as compared to Group C ($P<0.0001$). In Group D there was significant fall in Heart rate ($P<0.05$) and MAP ($P<0.0001$) as compared to group C.

STATISTICAL ANALYSIS

All the relevant data was recorded on proforma prepared for the study. The data that was recorded, after its proper validation, checked for error, compiled and analyzed using the software IBM SPSS 20 for windows. Student's t test (two tailed) and one way ANOVA was used to test the difference in various variables like Age, Sex, Weight, IOP, SBP, DBP, MAP and HR. For all the statistical analysis, p value less than 0.05 was taken as statistically significant.

DISCUSSION

Laryngoscopy and tracheal intubation is an integral part of modern anesthetic practice. This procedure is invariably associated with rise in heart rate and blood pressure .Succinylcholine is used to facilitate rapid tracheal intubation in high-risk patients for aspiration because of its fast onset time and excellent intubating conditions. It is, however, associated with an increase in the IOP. Laryngoscopy and tracheal intubation further aggravate the rise in IOP. Various methods have been used to attenuate the effects of succinylcholine on IOP. They include self-taming, where a small dose of succinylcholine is given initially followed by the remaining amount of succinylcholine and pre-treatment with non-depolarizing neuromuscular blocking agents, lidocaine, narcotics, nifedipine and nitroglycerin.

Kovac AL et al did their work on controlling the hemodynamic response to laryngoscopy and endotracheal intubation. They found that the hemodynamic response to the stress of laryngoscopy and endotracheal intubation does not present a problem for most patients. However, patients with cardiovascular or cerebral disease may be at increased risk of morbidity and mortality from the tachycardia and hypertension resulting from this stress. These hemodynamic effects gained notice after the introduction and use of muscle relaxants at the time of anesthesia induction. A variety of anesthetic techniques and drugs are available to control the hemodynamic response to laryngoscopy and intubation.

The method or drug of choice depends on many factors, including the urgency and length of surgery, choice of anesthetic technique, route of administration, medical condition of the patient, and individual preference. The possible solutions number as many as the medications and techniques available and depend on the individual patient and anesthesia care provider. This paper reviewed these medications and techniques to guide the clinician in choosing the best methods. [1]

Kaya FN, Yavascaoglu B et al studied the effect of oral gabapentin on the intraocular pressure and hemodynamic responses induced by tracheal intubation. Similar study was done by Domi RQ who did a comparison of the effects of sufentanil and fentanyl on intraocular pressure changes due to easy and difficult tracheal intubations.

It was suggested that gabapentin is a useful adjuvant in order to prevent an increase in the IOP in response to laryngoscopy and tracheal intubation. Similarly both drugs fentanyl and sufentanyl blunt the increased intraocular pressure during laryngoscopy and tracheal intubations, but in difficult intubation, sufentanil presented better protection than fentanyl. [2, 3]

Similar studies were done by Duman A et al in paediatric cases in which the effect on intraocular pressure of tracheal intubation or laryngeal mask™ insertion was seen during sevoflurane without the use of muscle relaxants.IOPs were measured after induction, insertion of TT or LMA and at 1, 2 and 3.It was concluded that sevoflurane does not prevent the increase in IOP after intubation without muscle relaxants. LMA does not increase IOP in children after sevoflurane induction. [4]

Yildiz M, Tavlan A saw that dexmedetomidine reduces the dose requirements for opioids and anesthetic agents. The purpose of this study was to evaluate the effect of a single pre-induction intravenous dose of dexmedetomidine 1 μ/kg on cardiovascular response resulting from laryngoscopy and endotracheal intubation, need for anesthetic agent and perioperative hemodynamic stability. During intubation the need for thiopental and sevoflurane concentration were decreased by 39% and 92%, respectively, in the dexmedetomidine group compared with the placebo group.

In all groups, blood pressure and heart rate increased after tracheal intubation; both were significantly lower in the dexmedetomidine group than in the placebo group ($p < 0.05$) Arterial blood pressure and heart rate in the postoperative period were significantly lower in the dexmedetomidine group compared with the placebo group ($p < 0.05$).So it was concluded that preoperative administration of a single dose of dexmedetomidine resulted in progressive increases in sedation, blunted the hemodynamic responses during laryngoscopy, and reduced opioid and anesthetic requirements. Furthermore, dexmedetomidine decreased blood pressure and heart rate as well as the recovery time after the operation. [5]
Effect of dexmedetomidine premedication on the intraocular

pressure changes after succinylcholine and intubation was **also** studied by Mowafi HA, Aldossary N et al. The use of succinylcholine is associated with an increase in intraocular pressure (IOP). This may be deleterious in open globe injuries. Succinylcholine and intubation increased IOP. However, in the dexmedetomidine group, the IOP rise was not different from the baseline value (P=0.65) and was significantly lower than in the saline group (P=0.003). After intubation, the MAP in the control group was higher than that in the dexmedetomidine group (P=0.041) and exceeded the baseline value (P<0.001). The HR also showed less fluctuation in the dexmedetomidine group than in the saline group. It was concluded that dexmedetomidine could be a beneficial premedication in open globe injuries. [6]

Similar study was done by Pal CK, Ray M et al regarding changes in intraocular pressure following administration of suxamethonium and endotracheal intubation: The purpose of the study was to observe the efficacy of dexmedetomidine for prevention of rise in IOP associated with the administration of suxamethonium and endotracheal intubation. Fall in IOP was observed following administration of dexmedetomidine. [7]

Yavascaoglu B, Kaya FN et al did a comparison of esmolol and dexmedetomidine for attenuation of intraocular pressure and hemodynamic responses to laryngoscopy and tracheal intubation. They concluded that dexmedetomidine is more effective than esmolol in preventing the hemodynamic and IOP responses to tracheal intubation in ASA I–II patients. In order to further evaluate the effects of esmolol, additional studies should be planned to assess the optimum dose, mode and delivery timing of this drug. Furthermore, it should be noted that this study included only healthy patients and does not reflect the effects of these drugs on patients with a history of hypertension or glaucoma.[8]

CONCLUSION

1. Dexmedetomidine premedication (0.4µg/kg) effectively prevents the rise of IOP after succinylcholine and endotracheal intubation.
2. Dexmedetomidine premedication (0.4 µg/kg) causes fall in IOP, HR and MAP.
3. Dexmedetomidine premedication (0.4 µg/kg) cause attenuation of pressor response after succinylcholine, laryngoscopy and endotracheal intubation.

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