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Effect of Dexmedetomidine Premedication on the Intraocular Pressure Changes after Succinylcholine and Intubation

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Abstract*: succinylcholine is still recommended for some situations in open globe injuries. However its use is associated with an increase in intraocular pressure (IOP) which may be deleterious in open globe injuries. We investigated whether dexmedetomidine, an alpha-2 agonist, could attenuate this increase in IOP after succinylcholine and intubation. Forty patients with no pre-existing eye disease undergoing general anesthesia were randomly premedicated by i.v dexmedetomidine 0.3mg/kg or saline. Heart rate(HR), mean arterial pressure(MAP),and IOP (using Schioetz tonometer) were measured before, after the premedication, after thiopental, after succinylcholine, immediately after intubation, and then every 3 min for 9 min succinylcholine and intubation increased IOP in both groups. 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 base line value(P,0.001). The HR also showed less fluctuation in the dexmedetomidine group than in the saline group. We conclude that dexmedetomidine premedication (0.3mg/kg) could be beneficial in open globe injuries.*

Keywords*: Dexmedetomidine, General anaesthesia, intraocular pressure, succinylcholine*

I. Introduction

Emergency ophthalmic surgery poses a challenge to the anaesthesiologist 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) . When the eye globe is open, any factor that increases the intra-ocular pressure (IOP) may possibly cause drainage of the aqueous humour or extrusion of the vitreous humour through the wound, which can permanently damage vision [1]

Suxamethonium the most commonly used depolarising muscle relaxant for rapid sequence airway management in high risk patients for aspiration because of its fast onset time and excellent intubating conditions, however it increases the IOP[2,3]. Laryngoscopy and tracheal intubation further aggravate the rise in IOP [4] .Various methods have been used to attenuate the effects of succinylcholine on IOP. They included 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[1] However, no modality was devoid of drawbacks and limitations.

Dexmedetomidine is a highly selective alpha-2 adrenergic agonist that has sedative and analgesic effects. [5 ,6]Alpha-2 agonists provide potentially beneficial effects in ophthalmic surgery because of their IOP lowering proper-ties.[7] The aim of this study is to investigate the effects of dexmedetomidine premedication on the IOP changes after succinylcholine and endotracheal intubation.

II. Patients and Methods

This prospective, randomized, double blind , placebo controlled study included 40 patients aged 20-50 years, belonging to either ASA I or II class, undergoing elective non-ophthalmic surgeries under general anaesthesia, were included in the study. A proper approval from the local ethics committee and informed consent was taken from the patients included in the study. Patients were randomly allocated into two equal groups (20 patients each) to receive either single bolus i.v. dose of dexmedetomidine 0.3mg/kg as premedication (dexmedetomidine group) or saline (control group). Surgery was performed early in the morning to avoid diurnal variations in IOP. No other sedative premedication was given to patients. Patients were excluded if they were older than 50 yr, had a body weight more than 150% of their ideal body weight using Broca's index, had acute or chronic eye disease, had any contraindication to the study drugs, or were receiving any medication known to alter IOP.

Standard intraoperative monitoring including three-lead ECG, pulse oximeter, capnometry, and non-invasive arterial pressure was performed. IOP was measured with a Schioetz tonometer by an ophthalmologist

who was unaware of the anaesthetic technique. For this procedure, topical proparacaine hydrochloride 0.5% was applied to the cornea before measurement.

Dexmedetomidine was prepared by diluting 1 ml of dexmedetomidine 100 mg/ ml with 49 ml of normal saline to a concentration of 2 mg/ ml. Syringes containing aqueous solutions of either dexmedetomidine or saline were prepared in a double-blind fashion by a team member who was not involved in data recording. Before induction of anaesthesia, a single dose of dexmedetomidine 0.3mg/ kg was administered i.v over 10 min. The same amount of saline was given to the patients in the control group.

Anaesthesia was standardized in all patients. After pre-oxygenation for 3 min, anaesthesia was induced with thiopental 5 mg/ kg and fentanyl 1 mg/ kg. Succinylcholine was then administered at a dose of 1.5 mg/ kg. When the fasciculation's had ceased, the trachea was intubated under direct vision laryngoscopy and the correct position of the tracheal tube was verified by auscultation and by capnometry. If the trachea could not be intubated at the first attempt with direct laryngoscopy, the patient was excluded from the study. Rocuronium 0.6 mg/ kg provided the intraoperative neuromuscular block. Anaesthesia was maintained in both groups with isoflurane in a mixture of with 66% nitrous oxide in oxygen. Fluid administration of lactated Ringer solution was standardized at 4 ml/ kg/ h. Mean arterial pressure (MAP), heart rate (HR), and IOP were recorded at the following time points:

- T1: 5 min after arrival to the operating room, before premedication (baseline).
- T2: 10 min after premedication.
- T3: 45 s after thiopental.
- T4: 45 s after succinylcholine.
- T5: immediately after intubation.
- T6 – 8: every 3 min for 9 min after intubation.

Data collected was statistically evaluated and analyzed. Parametric data was expressed as mean±SD, thereby the intergroup comparison were made by student's t-test. The test was two sided and referred for p-value for its significance. P-value less than 0.05 ($p<0.05$) was taken to be statistically significant. For comparison of different observations within and between the groups, data were first analysed by repeated measures analysis of variance, and differences were then calculated by *post hoc* testing .The analysis was performed on SPSS version 22 for Mac (IBM Inc., Chicago U.S.A). Data were presented as mean $\pm$ SD in the text and in Table 1, and as mean in the graphs.

III. Results

There were no significant differences between the two groups with regard to age, weight, height, and gender. There were also no significant differences at baseline HR, MAP, and IOP (Table 1)

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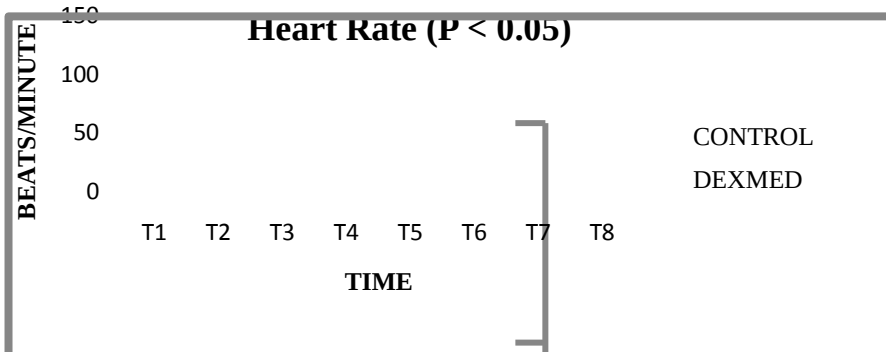
Table 1 Comparison of demographic data and other characteristics in two groups:

PARAMETER	CONTROL mean±SD	DEXMEDETOMIDINE mean±SD	P value
Age (years)	34.4 $\pm$ 6.4	35.3 $\pm$ 7.2	0.7832
Height (cm)	166 $\pm$ 8.2	168 $\pm$ 6.6	0.8894
Gender (M/F)	12/8	11/9	0.6324
Preop HR (bpm)	76.6 $\pm$ 6.8	78.6 $\pm$ 5.4	0.9856
Preop MAP (mmHg)	90.4 $\pm$ 4.8	88.3 $\pm$ 6.2	0.7364
Preop IOP (mmHg)	13.2 $\pm$ 1.1	12.9 $\pm$ 1.3	0.6939

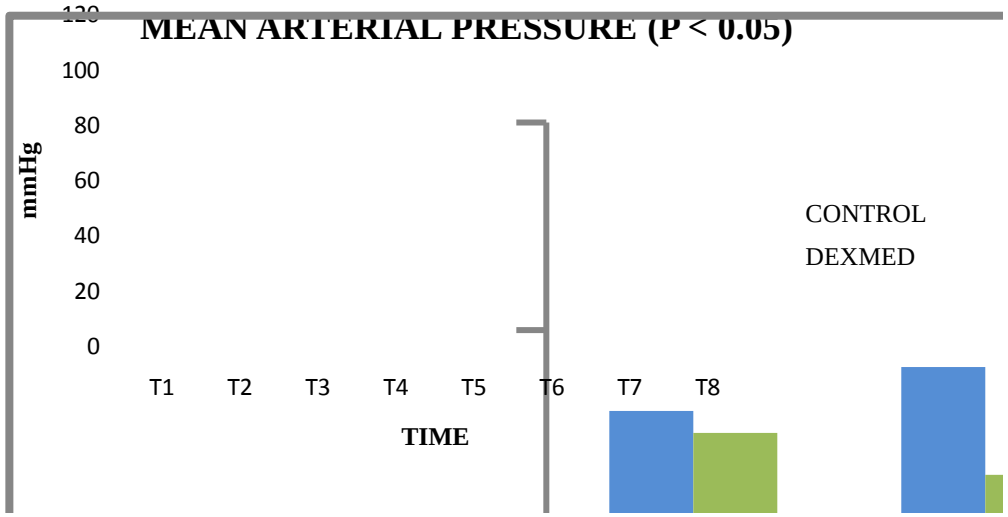
The values of HR, MAP, and IOP are shown in Figure 1,2,3. No significant differences in HR between the two groups were recorded at any time. However, in the control group, the HR increased significantly after injection of thiopental, succinylcholine, and intubation. These increases in HR were not observed in the dexmedetomidine group.

Figure1: Comparision of heart rate during different time intervals between two groups



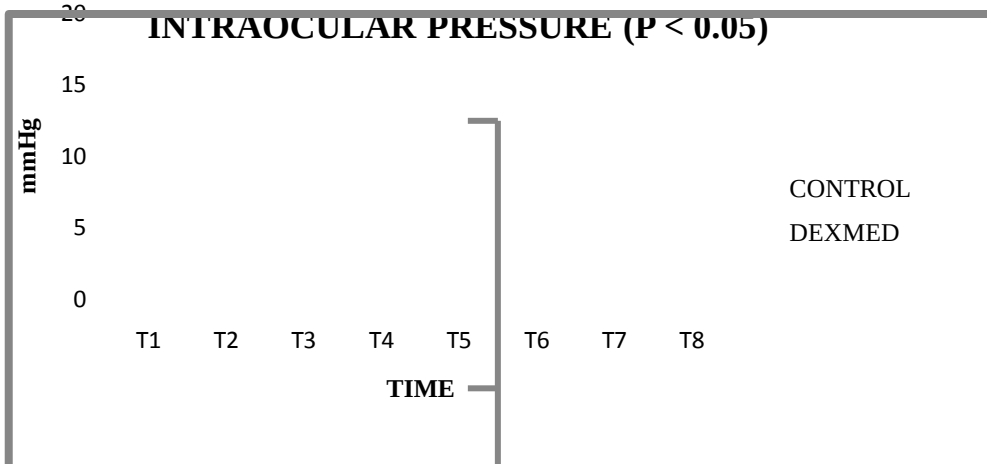
The MAP increased significantly compared with the preoperative value after intubation in the control group and was significantly higher than the MAP in the dexmedetomidine group (P<0.05). In the dexmedetomidine group, the MAP was not significantly higher than the preoperative value at all times. No incidence of hypotension or bradycardia requiring intervention was reported in both groups.

Figure2: Comparison of mean arterial pressure during different time intervals between two groups



There was no significant difference in the baseline IOP between both groups. After dexmedetomidine injection, there was a significant decrease in IOP, compared with baseline (P<0.05). Thiopental decreased IOP significantly in both groups (P, 0.001). Succinylcholine and intubation increased IOP in both groups. However, IOP in the dexmedetomidine group after intubation was not significantly different from that at baseline, unlike that in the control group.

Figure3: Comparison of intraocular pressure during different time intervals between two groups



IV. Discussion

Dexmedetomidine is a highly selective alpha-2 adrenergic agonist that has sedative and analgesic effects. In addition, dexmedetomidine attenuates the hemodynamic response to laryngoscopy and intubation [5, 6].

The intraocular hypotensive effect of dexmedetomidine in the present study is consistent with previous several researches on alpha-2 agonists. Clonidine was effective in preventing the rise of the IOP in response to succinylcholine and tracheal intubation.[8,9] Dexmedetomidine infusion as an adjunct to local analgesia in ophthalmic surgery was effective in reduction of the IOP significantly.[10] No previous study examined the effect of dexmedetomidine in such a low dose 0.3mg/kg on the succinylcholine-induced ocular hypertension.

The effect of dexmedetomidine on the IOP may be caused by a direct vasoconstrictor effect

on the afferent blood vessels of the ciliary body, which results in reduction of aqueous humour production [11]. Moreover, it could increase outflow of the aqueous humour caused by a reduction of the sympathetically mediated vasomotor tone of the ocular drainage system [12]. Additionally, its associated hemodynamic response could contribute to the IOP lowering effect [13].

In the present study, HR and MAP increased significantly after intubation in the control group. On the contrary, in patients who received dexmedetomidine pre- medication, this response was attenuated. Several previous studies have reported the blunting effect of dexmedetomidine on this sympathetic response to laryngoscopy and intubation [7, 14]. This could be due to the centrally mediated sympatholytic effects of alpha-2 agonists and by its decreasing nor-epinephrine release via peripheral pre- synaptic alpha-2 receptors [15, 16]

The dose of dexmedetomidine premedication administered in the present study (0.3 mg/ kg) is quite less than used in previous clinical study [7], where the selected dose resulted in a significant reduction in IOP and prevented the rise in the IOP in response to intubation. In addition, the pressor response to laryngoscopy and endotracheal intubation was also significantly attenuated. Higher doses of dexmedetomidine were associated with an additional reduction in the arterial pressure and HR without any further decrease in the IOP [17].

Some authors find that the use of succinylcholine in open ocular trauma is controversial and an alternative anaesthetic management based on the use of non- depolarizing neuromuscular blocking agents, despite its slower onset, was suggested [1]. Various methods have been tried to speed up this onset, including priming [16], administering the non-depolarizing relaxant before the induction agent [1], and high-dose regimen [14]. Despite these strategies, non-depolarizing neuromuscular blocking agents can still result in non-ideal intubation conditions: increases in the IOP from mask application and longer time with insecure airway and prolonged paralysis. Despite this debate about the use of succinylcholine in open globe injury, most authors still agree on its use in difficult airway cases with salvageable eye situations [1].

V. Conclusion

We conclude that the rise of IOP with succinylcholine and endotracheal intubation can be blunted with i.v. dexmedetomidine premedication at a dose of 0.3mg/kg which is quite less than most of the other studies. The haemodynamic stability is an additional advantage.

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