



E-ISSN: 2706-9575
P-ISSN: 2706-9567
IJARM 2021; 3(2): 631-634
Received: 10-07-2021
Accepted: 12-08-2021

Dr. Raghunath Ravirala
Assistant Professor,
Department of Anaesthesiology,
Dr. Patnam Mahender Reddy
Institute of Medical Sciences,
Chevella, Telangana, India

Dr. Ershad Mohammed Sohail
Assistant Professor,
Department of Anaesthesiology,
Meenakshi Medical College
Hospital & Research Institute,
Kancheepuram, Tamil Nadu,
India

Corresponding Author:
Dr. Ershad Mohammed Sohail
Assistant Professor,
Department of Anaesthesiology,
Meenakshi Medical College
Hospital & Research Institute,
Kancheepuram, Tamil Nadu,
India

Effectiveness and safety of dexmedetomidine in patients undergoing general anaesthesia: A randomized, single-blind trial

Dr. Raghunath Ravirala and Dr. Ershad Mohammed Sohail

DOI: <https://doi.org/10.22271/27069567.2021.v3.i2i.440>

Abstract

Background: During the administration of general anaesthesia, laryngoscopy and endotracheal intubation must be performed. Just before intubation, succinyl choline is given. Intraocular pressure, heart rate, and blood pressure may all rise as a result of this. Many medications have been tried and utilised to mitigate this stress reaction. Dexmedetomidine is a sedative, anxiolytic, and analgesic since it is an alpha₂ adrenergic agonist.

Methods: For this study, researchers utilised a randomised, single-blind, placebo-controlled design. Treatment was provided by the Anaesthesiology Division at Meenakshi Medical College Hospital & Research Institute, Kancheepuram, Tamil Nadu, India between July 2020 to June 2021. A no of patients used was 60 was utilised, with 30 participants in each of two groups.

Results: Adjustments to IOP, HR, MAP, and sedation were evaluated between the two groups in this single-blind randomised trial. In the group that received the optimal dosage of dexmedetomidine, intraocular pressure (IOP) was significantly lowered, and the expected increase in IOP due to intubation was avoided. Additionally, the pressor response to laryngoscopy and endotracheal intubation was greatly diminished.

Conclusion: With the current trial design, premedication with intravenous Dexmedetomidine reduces the increase in intraocular pressure that occurs after succinylcholine and intubation. Where intubation and succinylcholine lead to increases in intraocular pressure that are harmful to patients.

Keywords: General anaesthesia, intraocular pressure, and dexmedetomidine

Introduction

The anesthetist's job is made more difficult during emergency ocular surgery because patients with penetrating eye injuries commonly come with a full stomach. When the eye is open, increased intraocular pressure (IOP) may cause the lens to be rejected and the vitreous humour to be expelled, putting both the eye and the eyesight at risk^[1, 2]. In order to perform neurosurgeries safely and quickly, consistent hemodynamics, minimal fluctuations in intracranial pressure (ICP), and a rapid postoperative recovery from anaesthesia are all necessities. Laryngoscopy and intubation elicit a significant stress reaction^[3, 4]. There are a number of medications that may dampen these responses. Dexmedetomidine, a highly selective alpha₂-adrenergic agonist, has calming, cardioprotective, and neuroprotective properties^[5]. As such, it may find use in the field of neuroanaesthesia. The purpose of this randomised clinical trial using a single-blind design is to determine whether or if pre-intubation administration of Dexmedetomidine reduces hemodynamic responses, intraocular pressure, and the need for an induction dose of thiopentone. To avoid increasing intraocular pressure, these patients need a quick sequence induction and intubation. Rapid sequence induction with suxamethonium is typical, although it causes an increase in intraocular pressure. Pretreatment with a non-depolarizing muscle relaxant, usage of nifedipine and nitroglycerin, etc. have all been utilised to reduce the side effects of suxamethonium. However, there were flaws in every single one of them^[6, 7].

Dexmedetomidine has the ability to lower intraocular pressure. Its sedative effects make it ideal for usage in intensive care settings with patients who need mechanical ventilation^[8]. Dexmedetomidine has a number of positive benefits, including reduced anxiety and pain perception, sedation, parasympathetic activity, and sleepiness with little respiratory depression. It takes 2-3 hours for 50% of the initial plasma concentration of dexmedetomidine to reach equilibrium after a 10-minute infusion, and it takes 4 minutes and

250 minutes, respectively, after an 8-hour infusion. The increase in intraocular pressure caused by suxamethonium may be mitigated by administering dexmedetomidine beforehand, as has been seen [9, 10].

The goal of this research is to determine whether or not intravenous dexmedetomidine is effective in counteracting the effects of suxamethonium and intubation on the patient's heart rate, mean arterial pressure, and intraocular pressure under general anaesthesia. We used a multi parameter and a Schiötz to see whether dexmedetomidine might counteract the effects of succinyl choline and intubation on increasing heart rate, mean arterial blood pressure, and intraocular pressure. Examine the potential for sedation, hypotension, and bradycardia, among other possible adverse reactions [11, 12].

Methods

For this study, researchers utilised a randomised, single-blind, placebo-controlled design. Treatment was provided by the Anaesthesiology Division at Meenakshi Medical College Hospital & Research Institute, Kancheepuram, Tamil Nadu, India between July 2020 to June 2021. A no of patients used was 60 was utilised, with 30 participants in each of two groups.

Each patient will provide written informed permission once our institutional committee has approved the research plan. Subjects who match the inclusion criteria and agree to participate will be randomly assigned to one of two groups of 35 patients to receive either 0.6 mcg/kg or normal saline i.v. as premedication. Intraoperatively, using a syringe pump, 0.6 mcg/kg of the study medication was delivered over the course of 10 minutes, and IOP was recorded using a Schiötz tonometer. The patient in this trial is the only one who will be kept in the dark.

After 15 minutes, the patient's level of sedation will be evaluated using the Ramsay Sedation Score; pre-oxygenation will be performed for 3 minutes; fentanyl(1 mcg/kg) will be administered; and finally, thiopentone sodium will be administered to induce sleep. In order to produce muscular relaxation for intubation, 1.5 mg /kg of suxamethonium will be given.

Results

The data is presented as a mean +/- SD. The unpaired t test was used to compare demographic data and procedure type. The chi square test was used to analyse the differences between the sexes. Independent sample tests were used to examine the rates of change in intraocular pressure, heart rate, and mean arterial pressure (MAP) between the two groups at various times. Mann Whitney U test was used to compare sedation levels between the two groups. Regular SPSS 17.0 for Windows was used to analyse the data.

Table 1: Distribution of age

Age	Group 1		Group 2	
	Count	Percent	Count	Percent
<40	22	73.33	12	40.0
40 – 49	3	10	6	20.0
50 – 59	2	6.66	8	26.66
>=60	3	10	4	13.33
Mean ± SD	39.1±8.3		42.2±11.0	

Table 1 includes a breakdown of the patients in terms of their ages, along with the proportion of each age group that

makes up each of the two groups.

Table 2: Gender wise Distribution

Sex	Group 1		Group 2	
	Count	Percent	Count	Percent
Male	12	40.00	16	53.33
Female	18	60.00	14	46.66

Table 2 includes an analysis of the gender breakdown of the patients, along with a breakdown of their relative percentages in each of the two groups.

Table 3: Analyzing differences in body mass across groups

Group	Mean	SD	N
Group 1	65.9	8.9	30
Group 2	63.7	8.9	30

You can see the differences between the two groups in terms of mean and standard deviation in Table 3.

Table 4: Procedure allocation by social group

Procedure	Group 1		Group 2	
	Count	Percent	Count	Percent
Cholecystectomy	5	16.6	7	23.33
Excision	4	13.33	4	13.33
Thyroidectomy	16	53.33	17	55.87
Hemithyroidectomy	1	3.33	0	00.0
Parotidectomy	2	6.66	1	3.33
Septoplasty	2	6.66	1	3.33

The distribution of procedures by patient group, together with their relative percentages in the two groups, are shown in Table 4.

Table 5: Group-based analysis of initial intraocular pressure

Group	Mean	SD	N
Group 1	14.1	3.0	30
Group 2	14.3	3.1	30

Loss of eyelid response for 30 seconds showed no statistically significant change in IOP between the study and control groups. There is no statistically significant difference between the study population and the control group in terms of mean IOP. The statistically meaningful time period begins at T2 (30 seconds after succinylcholine delivery) and continues until T4. Both groups see an increase in IOP when succinylcholine is given and intubation is performed, although the increase in the study group is much less compared to the control group. At T3, the IOP reaches its maximum.

Significantly higher heart rates were seen in the study's control group (mean of 76 bpm with SD 8 at baseline to mean 96 bpm with SD 7.3 at 1 min after intubation). This effect, however, was blunted in premedicated dexmedetomidine individuals. At 2, 4, and 6 minutes after intubation, the study group's heart rate continues to decline at a statistically significant rate compared to the control group. After premedication and succinylcholine induction, MAP drops by about 7 mm Hg in the study group, whereas it rises by about 2 mm Hg in the control group. However, mean arterial pressure increases from pre-succinylcholine levels following intubation in both the study and control groups. However, the stress-induced rise in MAP shown in

the study population is significantly mitigated.

Discussion

Within the first five seconds of the laryngoscope touching the base of the tongue, the sympathetic nervous system is expected to kick in, triggering an increase in intraocular pressure and a stress reaction in the hemodynamic system. About 2 minutes after intubation, it reaches its highest point. After then, it begins to drop and returns to pre-intubation levels anywhere between 5 and 10 minutes. We hypothesised that administering 0.6 micrograms per kilogramme of body weight of dexmedetomidine intravenously 15 minutes prior to the administration of succinyl choline and intubation would reduce these stress reactions, and we set out to test this hypothesis in the current investigation [13-15].

Drugs like alpha-2 adrenergic agonists dexmedetomidine and clonidine have been studied in the past for their impact on blood pressure. Both were helpful in mitigating the rise in intraocular pressure that followed succinylcholine injection. Dexmedetomidine's effects on intraocular pressure were investigated separately across a variety of contexts. Dexmedetomidine was reported to reduce intraocular pressure (IOP) by 34% in a trial when it was administered as a sedative during eye surgery under local anaesthesia. Using the medication for cataract operations in the elderly allowed researchers to reach the same findings. In their investigation, Lee *et al.* monitored the intraocular pressure (IOP) before and after receiving premedication with dexmedetomidine and maintaining anaesthesia with isoflurane [16-18]. Despite their expectations, they saw no significant decrease in IOP. Dexmedetomidine's hypotensive effects may be a result of its sympathoplegic effect on vessels, which increases aqueous humour drainage via the canal of Schlemm; its constriction of choroidal vessels, which decreases aqueous humour formation; or its direct activation of alpha 2 adrenergic receptors, which decreases the cardiovascular response by decreasing the catecholamine release and thus centrally mediates the sympatholytic effect. Dexmedetomidine's hypotensive effect has been shown in a number of previous investigations. Those who were given dexmedetomidine before laryngoscopy and intubation had significantly lower increases in IOP and heart rate after these procedures compared to patients in the control group [19-21].

Previous research using the same dosage of Dexmedetomidine as in the current trial demonstrated a decrease in intraocular pressure and eliminated the stress response to intubation. It has also been shown, via studies comparing the attenuation in stress response to various doses of dexmedetomidine that greater doses of dexmedetomidine aided in decreasing the stress reaction to intubation even more, but without causing a further decrease in IOP [22, 23]. Because anesthesiologists were divided on whether or not to employ succinylcholine for open eye globe injuries, non-depolarizing muscle relaxants were often utilised instead. Priming with a modest dosage before induction and delivering large doses of medication were thus used to shorten the onset time of non-depolarizing relaxants [24-26]. All of these approaches had drawbacks, including extended post-operative breathing, an uncertain airway, and an increase in intraocular pressure (IOP) from leaving the mask on a conscious paralysed patient. Although the development of Sugammadex has solved the issue of

muscle relaxants like vecuronium having a lengthy duration of action, the drug's high price and limited availability are major drawbacks. Despite the ongoing debate, many anesthesiologists continue to utilise succinylcholine when dealing with challenging airway circumstances when the visual implications might be outweighed by the benefits [27-30].

The only caveat of this study was that it was not possible to separate the effects of dexmedetomidine on intraocular pressure and its effects on cardiovascular response, since the two were intertwined. This means that the fact that IOP dropped due to a decrease in heart rate and BP was not the only reason for this phenomenon. However, its efficacy in preventing intraocular pressure drop due to succinylcholine and intubation cannot be denied. When administered intravenously as a premedication, dexmedetomidine not only prevented a drop in blood pressure during endotracheal intubation but also prevented a rise in intraocular pressure [31-33].

Conclusion

Prior intravenous the current research design found that low-dose dexmedetomidine prevented an increase in intraocular pressure induced by succinylcholine and intubation. Another benefit is a reduced hemodynamic stress response during laryngoscopy and intubation. Since a rise in intraocular pressure after succinylcholine and intubation may be harmful to patients, dexmedetomidine is sometimes administered as a premedicated in these circumstances.

Conflict of interest

None

Funding support

Nil

References

1. King BD, Harris LC Jr, Brefnstein FE *et al.* Reflex circulatory response to direct laryngoscopy and tracheal intubation performed during general anaesthesia. *Anaesthesiology*. 1951;12:5-56.
2. Wycoff CC. Endotracheal intubation: effect on blood pressure and pulse rate. *Anaesthesiology*. 1960;21:153.
3. Tomori Z, Widdicombe JG. Muscular, bronchomotor and cardiovascular reflexes elicited by mechanical stimulation of the respiratory tract. *J Physiol*. 1969;200:25-49.
4. F.N. Kaya B, Yavascaoglu, M, Baykara G.T. Altun: Effects of Gabapentin on the intraocular and hemodynamic responses induced by tracheal intubation. *Acta Anaesthesiol Scand*. 2008;52:1076-1080.
5. Bedford RF: Circulatory response to tracheal intubation, In: Erchnorm J H, Kirbv RB, Brown D L (Eds): *Problems in anaesthesia*. Philadelphia, JB Lippincott. 1998;200:25.
6. Derbyshire DR, Smith G, Achola KJ. Effect of topical lignocaine on the sympathoadrenal responses to tracheal intubation. *Br J Anaesth*. 1987;59:300-4.
7. Ovassapian A, Yelich SJ, Dykes MH, Brunner EE. Blood pressure and heart rate changes during awake fiberoptic nasotracheal intubation. *Anaesth Analg*. 1983; 62:951-4.
8. Shribman AJ, Smith G, Achola KJ. Cardiovascular and catecholamine responses to laryngoscopy with and

- without tracheal intubation. *Br J Anaesth.* 1987;59:295-9.
9. Finfer SR, Mackenzie SI, Saddler JM, Watkins TG. Cardiovascular responses to tracheal intubation: a comparison of direct laryngoscopy and fiberoptic intubation. *Anaesth Intensive Care.* 1989;17:44-48.
 10. Roy WL, Edelist G, Gilbert B. Myocardial ischemia during non-cardiac surgical procedures in patients with coronary artery disease. *Anesthesiology* 1979;51:393-7.
 11. Fox EJ, Sklar GS, Hill CH, Vilanueva P, King BD. Complications related to the pressor response to endotracheal intubation. *Anesthesiology.* 1977;47:524-5.
 12. Mangano DT, Browser WS, Hollenberg M, Li J, Tatoel M. Long term cardiac prognosis following non-cardiac surgery. The study of the operative Ischemia Research Group. *JAMA* 1992; 268: 233- 9.
 13. Ross AF, Tinker JH. Evaluation of adult patient with cardiac problems, In: Gogers MC, Timken JA, Covino BG, Langnecker DE (ECS). *Principals and practice of Anaesthesiology.* 1979;50:285- 92.
 14. Krupin T. Pestrich C, Bass I. Podos S. Becker B. Acidosis, alkalosis and aqueous humour dynamic in rabbits. *Invest Ophthalmol.* 1977;16:997-1001.
 15. Marcu F D, Krubin T, Podos S, Becker B. The effect of exercise on intraocular pressure. I. Human Beings. *Invest Ophthalmol.* 1970;9:749-52.
 16. Becker B. The mechanis of the fall in intraocular pressure induced by the carbonic anhydrase inhibitor, Diamox. *Am J Ophthalmol.* 1955;39:178-84.
 17. Saltzman HA. Anderson B, Hart L. Duffy E, Becker HO. The retinal vascular functional response to hyperbaric oxygenation. In: *Hyperbaric oxygenation. Proceedings of the Second International Congress.* Livingstone; London, 202, 1965.
 18. Van Sallmann L, Lowenstein O. Responses of intraocular pressure, blood pressure and cutaneous vessels to electrical stimulation in the diencephalon. *Am J Ophthalmol.* 1955;39:11-29.
 19. Holloway KB. Control of the eye during general anaesthesia for intraocular surgery. *Br Anaesth.* 1980;52:671-9.
 20. Stinson TW, Donlon IV. The interaction of intraocular air and sulfur hexafluoride with nitrous oxide: a computer simulation. *Anesthesiology.* 1983;59:547-8.
 21. Wolf GL. Capuano C, Harting J. Nitrous oxide increases intraocular pressure after intravitreal sulphur hexafluoride injection. *Anesthesiology.* 1983;59:547-8.
 22. Kornbueth W, Alademoff L. Magora F. Gabbay A. Influence of general anaesthesia on intraocular pressure in man. *Arch Ophthalmol.* 1959;61:84.
 23. Magora F, Collins VJ. The influence of general anaesthesia agents on intraocular pressure in man. *Arch Ophthalmol.* 1961;66:806-11.
 24. Al-Abrak MH. Samuel JR. Funher: observations on the effects of general anaesthesia on intraocular pressure in man. Halothane in nitrous oxide and oxygen. *Br J Anaesth.* 1974;46:756-9.
 25. Adams P. Freedman A. Henville JD. Normocapnic anaesthesia for intraocular surgery. *Br J Ophthalmol.* 1979;63:204-10.
 26. Al- Abrak MH. Samuel JR. Effects of general anaesthesia on intraocular pressure in man. Trichloroethylene in nitrous oxide and oxygen. *Br J Ophthalmol.* 1975;59:107-10.
 27. Rose NM. Adams AP. Normocapine anaesthesia with enflurane for intraocular surgery. *Anaesthesia* 1980;35:569-75.
 28. Runciman JC. Bowen-Wright RM. Welsh NH. Downing JW. Intraocular pressure changes during halothane and enflurane anaesthesia. *Br J Anaesth Analg.* 1975;54: 471-5.
 29. Au.rinsch B, Graves SA. Munson ES. Levy NS. Intraocular pressure in children during isoflurane and halothane anaesthesia. *Anesthesiology.* 1975;42:167-72.
 30. Thompson MF. Brock-Vme JG. Bean P, Welsh N. Downing JW. Anaesthesia and intraocular pressure: a comparison of total intravenous anaesthesia using etomidate with conventional inhalational anaesthesia. *Anaesthesia.* 1982;37:758-61.
 31. Yoshikawa K. Murai Y. The effect of ketamine on intraocular pressure in children. *Anesth Analg.* 1971;50:199-200.
 32. Peuler M, Glass DD, Arens JF. Ketamine and intraocular pressure. *Anesthesiology.* 1975;5:575-8.
 33. Ansinch B. Rayborn RL. Munsen ES. Levy NS. Ketamine and intraocular pressure in children. *Anesth Analg.* 1976;55:773-5.

How to Cite This Article

Ravirala R, Mohammed ES. Effectiveness and safety of dexmedetomidine in patients undergoing general anaesthesia: A randomized, single-blind trial. *International Journal of Advanced Research in Medicine.* 2021;3(2):631-634.

Creative Commons (CC) License

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 International (CC BY-NC-SA 4.0) License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.