Original Research Article

Indian Journal of Anesthesia and Analgesia
2018; 5(6): 969-976
DOI: http://dx.doi.org/10.21088/ijaa.2349.8471.5618.15

To Study the Changes in Intraocular Pressure during various Steps of General Anesthesia using Thiopentone Sodium or Ketamine for Induction and Succinylcholine for Neuromuscular Blockade

Manjiri V. Deshpande

Assistant Professor, Dept. of Anaesthesiology, Dr. V M Govt. Medical College, Solapur, Maharashtra 413003, India.

Abstract

Background: Several general and local anesthetic techniques have been employed to try and prevent increase in intraocular pressure during surgery. Interest in general anesthesia for intraocular surgery is relatively recent. Many surgeons now prefer full anesthesia and indeed the increasing scope of ophthalmic surgery demands it. This study was therefore undertaken to know the changes in intraocular pressure, during various steps of general anesthesia using thiopentone sodium or ketamine for induction and suxamethonium (succinylcholine) for neuromuscular blockade. *Material and Methods:* Sixty patients between the age group of 1-75 years were selected for this study. These patients were divided into two groups –I and II. The patients in groups were given general anesthesia and were operated for either ophthalmic or non – ophthalmic indications. A detailed general examination (including measurement of blood pressure) and systemic examination was done to rule out any systemic disorder which would directly or indirectly affect the measurement of intraocular pressure during the course of the study. Routine blood and urine investigations were done. *Results:* The mean fall in intraocular pressure after induction as compared to mean baseline intraocular pressure was 2.48±1.32 in group – I. The mean rise in intraocular pressure after induction as compared to mean baseline intraocular pressure was 2.67±2.08 in group-II. This difference in the intraocular pressure after induction in group -I was statistically highly significant as compared to group -II ($p<0.01$). *Conclusions:* Thiopentone sodium can be safely used as an inducing agent in any intraocular surgery done under general anesthesia. Ketamine, as far as possible, should be restricted to extraocular ophthalmic surgeries.

Keywords: Thiopentone Sodium; Intraocular Pressure; Suxamethonium; Ketamine.

Introduction

The maintenance of intraocular pressure, along with analgesia and akinesia, is very important during an intraocular surgery. Any increase in the intraocular pressure while the globe is open, may cause expulsion of vitreous and subsequent loss of vision. Several general and local anesthetic techniques have been employed to try and prevent increase in intraocular pressure during surgery. Whether the patient is operated under general or local anesthesia, the desirability of a soft globe for anterior segment surgery is generally an accepted surgical principal. A reduced intraocular pressure with a concave vitreous face can help the procedure to be carried out smoothly. Preoperative intraocular pressure reduction is thought to minimize the risk of vitreous loss and expulsive haemorrhage during cataract surgery. To obtain a soft eye preoperatively is quite difficult in patients being operated under general anesthesia, whereas in local anesthesia, there is a major advantage of obtaining this with the help of various methods [1].

Interest in general anesthesia for intraocular surgery is relatively recent. Many surgeons now prefer full anesthesia and indeed the increasing scope of ophthalmic surgery demands it. A large series of intraocular operations carried out under

Corresponding Author: Manjiri V. Deshpande, MD, Dept. of Anaesthesiology, Dr. V M Govt. Medical College, Solapur, Maharashtra 413003.
E-mail: mhsur16@gmail.com

Received on 09.02.2018, **Accepted on** 08.03.2018

general anesthesia suggest that the surgical results are neither better nor worst than those done under local, but there may be an irreversible gain, firstly in that the patients are spared a trying ordeal and secondly that patients previously refuse surgery because their cooperation was in doubt, are now having sight restored by operations performed under general anesthesia.

General anesthesia may vary from a technique which utilizes anesthesia, muscle paralysis and ventilation with intubation on one hand to reliance on spontaneous breathing and a laryngeal mask on the other [2].

Most of the agents commonly used for induction in general anesthesia reduce intraocular pressure [3]. Thiopentone sodium, for example, significantly reduces intraocular pressure [3-7]. Ketamine, a useful inducing agent for paesiatric ophthalmic examination and short procedures on the other hand, causes a rise in intraocular pressure [3,6,7]. Syxamethonum, a muscle relaxant, used before intubation also causes a rise in the intraocular pressure [3,6,7].

Some investigators have reported that ketamine, but not propofol, provides additional effects on decreasing the incidence of EA in pediatric patients [8], and ketamine induction provides less EA when compared to thiopental induction for desflurane anesthesia for a tonsillectomy and adenoidectomy without delayed recovery[9]. However, another report showed that the EA after sevoflurane anesthesia was significantly reduced by propofol induction, as compared with thiopental sodium [10].

However, general anesthesia may produce problems in many elderly patients and especially in those with concomitant systemic disease. Further, straining, coughing and vomiting associated with it may cause serious increase in intraocular pressure which may be disastrous [11]. Local anesthesia, on the other hand, has become increasingly popular for a variety of surgical procedures. The trend towards local anesthesia in preference to general anesthesia in eye surgery increases apace for several reasons, not least of which is the move towards day care surgery [12].

There are advantages and disadvantages of both the local anesthetic techniques. Since all these procedure appear to work well in experienced hands, most surgeons, however, continue to use the techniques they know best.

It is seen that though both these techniques of local anesthesia provide a good amount of akinesia and anesthesia, they produce a rise in the intraocular pressure, after their injection, which might be hazardous after opening the globe [13]. This study was therefore undertaken to know the changes in intraocular pressure, during various steps of general anesthesia using thiopentone sodium or ketamine for induction and suxamethonium (succinylcholine) for neuromuscular blockade.

Objectives

To study the changes in intraocular pressure during various steps of general anesthesia using thiopentone sodium or ketamine for induction and succinylcholine for neuromuscular blockade.

Materials and Methods

Sixty patients between the age group of 1-75 years were selected for this study. These patients were divided into two groups –I and II. The patients in groups were given general anesthesia and were operated for either ophthalmic or non – ophthalmic indications.

Group – I contained 30 patients who were given Inj. Thiopentone sodium intravenously for induction of anesthesia. Group – II contained 30 patients who were given Inj. Ketamine intravenously for induction of anesthesia. In both the groups, succinylcholine (suxamethonium) was used for neuromuscular blockade before intubation.

A detailed history was taken which included history of any systemic disorders like diabetes mellitus, hypertension, bronchial asthma, ischaemic heart disease etc.

A detailed general examination (including measurement of blood pressure) and systemic examination was done to rule out any systemic disorder which would directly or indirectly affect the measurement of intraocular pressure during the course of the study. Routine blood and urine investigations were done.

All the patients were started on systemic and local antiobiotics preoperatively and were given Inj. Atropine 0.6 mg intramuscularly half prior to surgery. Lignocaine sensitivity test was done in patients who were to be operated under local anesthesia. Written consent was taken for surgery, anesthesia and for relevant examination.

Materials

And all the materials necessary for induction of anesthesia and intubation.

General Anesthesia	
Inj. Thiopentone sodium	500 mg or 1 gm vial
Inj. Ketamine	50 mg/cc 10 cc vial
Inj. Succinylcholine	50 mg/cc 10 cc vial
Syringes	2 cc

Schiotz Tonometer [14,15]

In this study the intraocular pressure was measured with the help of Schiotz tonometer.

This is a prototype of indentation instruments. With the help of this instrument, the deformation or indentation of the globe in response to a standard weight applied to the cornea is measured.

The instrument consists of a metal plunger that slides through a hole in a concave metal foot plate. The plunger supports a hammer device that is connected to a needle that crosses a scale. The plunger, hammer and needle weigh 5.5 gm. This can be increased to 7.5 gm, 10 gm or 15 gm by addition of appropriate weights. The more the plunger indents the cornea, higher is he scale reading and lower is the intraocular pressure. Each scale unit represents a 0.05 mm protrusion of the plunger.

Measurement of Intraocular Pressure [14,15]

Following method and guidelines were followed for measuring intraocular pressure in both the groups.

- The patient was given appropriate anesthesia depending on the group of anesthesia under study.
- A drop of topical anesthetic (4% xylocaine) was instilled in eye after explaining the nature of the test to the patient.
- Patient's lids were retracted without placing any tension on the globe.
- The tonometer was placed directly over the eye and when the patient relaxed, the tonometer was lowered gently on to the cornea.
- The intraocular pressure measurement was repeated until three consecutive readings were within 0.5 scale units.
- The average scale reading was converted to intraocular pressure in millimeters of Mercury using a conversion chart.
- After each use, the tonometer plunger and foot plate were cleaned with spirit followed by normal saline and then wiped dry lint free material.

Pre - Medication

Inj. Pentazocine 0.3 mg/kg and Inj. Glycopyrolate 0.004 mg/kg was given intramuscularly 30 minutes prior to induction.

Induction

- After taking the patient on the operation table, intraocular pressure was measured after putting 1-2 drops of 4% xylocaine in the eye.
- Prior to and during the procedure, special care was taken to avoid any pressure on the patients eye and to prevent any respiratory disturbances e.g. coughing, straining, laryngospasm etc. since these disturbances produce a rise in intraocular pressure due to rise in venous pressure.
- Pre – oxygenation was done with 100% oxygen for 3 minutes.
- In group A-I, induction was done with thiopentone sodium 5 mg/kg intravenously and succinylcholine 2 mg/kg intravenously was given to obtain muscular paralysis.
- In group A–II, induction was done with ketamine 2 mg/kg intaravenously and succinylcholine 2 mg/kg was used to facilitate endotracheal intubation.
- Intubation was done after adequate muscle relaxation.
- Anesthesia was maintained with O_2-NO_2-halothane combination.
- Patient was reversed with neostigmine 0.04 -0.06 mg/kg and Inj. Glycopyrolate 0.008 mg/kg intravenously and extubation was done.

Recording of Intraocular Pressure

Intraocular pressure was measured at following stages:

1. Before induction
2. After the patient was induced with thiopentone sodium i.e. before giving succinylcholine
3. Immediately after tracheal intubation.
4. After the patient was extubated.

Statistical Analysis

Descriptive statistics such as mean, SD and percentage was used. Comparison between two groups was done by using t-test. A p-value less than 0.05 were considered as significant. Statistical analysis was performed by using software SPSS v16.0

Results

Group – I consisted of 30 cases in which there were 11 males (36.7%) and 19 females (63.3%). Group – II also consisted of 30 cases in which there were 9 males (30%) and 21 females (70%).

There were more females in group A because most of the cases selected in this group were being operated for either obstetrical or gynecological indications.

In group – I, there were 17 cases (56.7%) which belonged to age group 21-30 years followed by 5 cases (16.7%) in the group 0-10 years and 4 cases (13.3%) in the age group 41-50 years.

In group – II, there were 10 cases (33.3%) which belonged to 21-30 years followed by 7 cases (23.3%) each in the age group 11-20 years and 31-40 years.

In the present study, it was seen that the mean pre-induction intraocular pressure was 13.84+2.75 and the mean post – induction intraocular pressure was 11.35+2.52. After applying the student 't' test the difference between these 2 values was found to be statistically highly significant ($p<0.01$).

The mean fall in intraocular pressure after induction as compared to mean baseline intraocular pressure was 2.48+1.32.

Table 1: Sex-wise distribution

Sex	Group – I Thiopentone sodium + succinylcholine		Group – II Ketamine + succinylcholine	
	No. of Cases	Percentage	No. of Cases	Percentage
Males	11	36.7	9	30.0
Females	19	63.3	21	70.0
Total	30	100.00	25	100.00

Table 2: Age-wise distribution

Age (years)	Group – I Thiopentone Sodium + Succinylcholine		Group – II Ketamine + Succinylcholine	
	No. of Cases	Percentage	No. of Cases	Percentage
0-10	05	16.7	----	----
11-20	02	6.7	7	23.3
21-30	17	56.7	10	33.3
31-40	01	3.3	7	23.3
41-50	04	13.3	03	10.00
Above 51	01	3.3	03	10.00
Total	30	100.00	30	100

Table 3: Mean intraocular pressure in group-I (thiopentone sodium + succinylcholine)

	Intraocular Pressure (IOP) Recording in mmHg (n=30)			
	Pre – Induction	Post – Induction	Post – Intubation	Post - Extubation
Mean ± SD	13.84 ± 2.75	11.35 ± 2.52	16.50 ± 2.10	12.56 ± 2.06
Range	8.5 – 18.9	8.5 – 18.9	12.2 – 20.6	7.1 – 18.9
Mean change in IOP as compared to mean baseline IOP	-----	2.48 ± 1.32	2.68 ± 1.60	1.74 ± 1.46

Table 4: Mean intraocular pressure in group-II (ketamine + succinylcholine)

	Intraocular Pressure (IOP) Recording in mmhg (N=30)			
	Pre – Induction	Post – Induction	Post – Intubation	Post - Extubation
Mean ± SD	12.17 ± 2.08	14.84 ± 1.94	17.18 ± 1.02	12.14 ± 1.72
Range	9.4 – 15.9	10.2 – 17.3	15.9 – 19.0	9.4 – 14.9
Mean change in IOP as compared to mean baseline IOP	-----	2.67 ± 2.08	5.16 ± 1.21	0.008 ± 0.65

Table 5: Comparison of intraocualr pressure (mmhg) between groups

Steps of Anesthesia	Group-I Mean ± SD	Group-Ii Mean ± SD	S.E.	t – value	P- value
Pre – Induction	13.84 ± 2.75	12.17 ± 2.08	----	----	----
Post – Induction	11.35 ± 2.52	14.84 ± 1.94	0.64	5.45	<0.01
Post – Intubation	16.50 ± 2.10	17.18 ± 1.02	0.47	1.44	>0.05
Post – Extubation	12.56 ± 2.06	12.14 ± 1.72	0.54	0.78	>0.05

In the present study, it was seen that the mean pre-induction intraocular pressure was 12.17+2.08 and the mean post – induction intraocular pressure was 14.84+1.94. The difference between these 2 values was found to be statistically highly significant ($p<0.01$).

The mean rise in intraocular pressure after induction as compared to mean baseline intraocular pressure was 2.67±2.08.

From the above table it was seen that there was a fall in intraocular pressure after induction in group-I (13.84+2.75 to 11.35+2.52) whereas in group– II there was a rise in intraocular pressure after induction (12.17+2.08 to 14.84+1.94).

This difference in the intraocular pressure after induction in group-I was statistically highly significant as compared to group -II ($p<0.01$)

Discussion

Intraocular pressure after induction with thiopentone (group -I):

Thiopentone is an ultrashortacting thiobarbiturate which even today is the most common intravenous inducing agent. While studying the changes in intraocular pressure due to thiopentone, we noted a fall in intraocular pressure.

In the present study, it was seen that the mean pre – induction intraocular pressure was 13.84+2.75 mmHg and mean post – induction intraocular pressure was 11.35+2.52 mmHg. After applying the student 't' test the difference between these two values was found to be statistically highly significant ($p<0.01$). The mean fall in intraocular pressure after induction as compared to mean baseline intraocular pressure was 2.48+1.32 mmHg. The fall in intraocular pressure ranged from 0.8–5.1 mmHg, the maximum fall being 5.1 mmHg.

Stone H H has studied the effects of barbiturates and paraldehyde on aqueous humour dynamics in rabbits [16]. In this study he found a marked fall in intraocular pressure caused by these two drugs. Similar findings were also seen by Kornbleuth [4].

Many general anesthetic agents have been shown to reduce intraocular pressure. The exact mechanism is not known but may be related to the relaxation of the extraocular muscles and the depression of ocular centres in diencephalon, midbrain and hypothalamus. This was suggested by Von Sallman et al. [17]. This was supported by Kornbleuth et al [4], who also believed that increased facility for outflow drainage might also be an associated cause.

Sathe ND [18] has studied the fall in intraocular pressure after thiopentone induction in 30 patients between 18-60 years of age. In this study he found a fall in intraocular pressure which varied from 2-17 mmHg immediately after the sleep dose of thiopentone. In this case premedication was given; the drugs used were Inj. Atropine 0.6 mg and Inj. Pethidine 50 mg intramuscularly. In the present study, we also found a fall in intraocular pressure which varied between 0.8–5.1 mmHg. The increased fall in intraocular pressure in ND Sathe's study could be due to additional intraocular pressure lowering effect of pethidine.

Another study was carried out by Joshi C and Bruce DL [19]. They measured intraocular pressure in 18 patients and found a consistent fall in intraocular pressure after the sleep dose of pentothal (3 mg/kg). They found that the fall in intraocular pressure was statistically significant thus correlating well with the findings of the present study.

Col. Banerjee Sc et al. [20] studied the effects of general anesthesia on intraocular pressure. In this series they studied the effects of pentothal on intraocular pressure on 100 patients. The intraocular pressure reading was taken 3 minutes after the induction of anesthesia. They found a fall in intraocular pressure which ranged from 1-12 mmHg. The increased fall in this study might have been due to use of premedication agents like inj. Atropine and inj. Morphine.

Verma RS [21] studied 15 cases who were induced thiopentone. Premedication consisted of diazepam 10 mg intramuscular given 30-45 minutes before operation. Atropine 0.6 mg was given simultaneously with thiopentone. He found a significant reduction

in intraocular pressure after induction. Reductions ranged from 4.5-13 mmHg. Again the increased fall in his study might be due to the additive intraocular pressure lowering effect of the premedication agents. These findings were comparable with the findings of the present study.

Intraocular Pressure after Ketamine (group -II)

Ketamine was introduced by Corsen G and Domino EF [22]. It is related to phencyclidine and causes a dissociative type of anesthesia which is characterized by catalepsy, light sedation, amnesia and marked analgesia.

In the present study, it was seen that the mean pre – induction intraocular pressure was 12.17 + 2.08 mmHg and the mean post – induction intraocular pressure was 14.84+1.94 after applying the student 't' test the difference between the two values was found to be statistically highly significant ($p<0.01$). The mean rise after induction as compared to the mean baseline intraocular pressure was 2.67+2.08 mmHg, range being 0- 5.7 mmHg.

The factors which raise intraocular pressure under ketamine anesthesia may be systemic hypertension, hypercarbia and the use of suxamethonium as reviewed by Adams and Barnett [23].

Corssen G and Hoy JE [24] used this anesthetic n 46 patients in the age group 6 months to 77 years. They observed that the intravenous administration of 2-3 mg/kg of ketamine led to an increase of 2-7 mmHg in the intraocular pressure. There was no correlation between these changes, patients age or change in systemic blood pressure.

Yoshikawa K and Murai Y [25] studied 18 patients aged between 4-7 years of age and observed an increase of 18% in intraocular pressure as compared to the baseline values 5 minutes after the injection of ketamine and this value rose to 37% after 15 minutes. Ketamine was administered intramuscularly in a dose of 4-5 mg/kg.

Dave B et al. [26] studied 60 patients of both sexes undergoing different surgical procedures. They found rise in intraocular pressure ranging from 2-6 mmHg in 36 out of 60 patients. No fall in intraocular pressure was noted in any patient.

Thus the findings of the present study were in close correlation with the studied conducted by Corssen G and Hoy JE [24], Yoshikawa R and Murai Y [25] and Dave B et al. [26].

Whatever the cause of rise in intraocular pressure after ketamine induction, Adams [27] suggestion seems a valuable one. He says that ketamine is not contraindicated for ophthalmic examination solely on account of its effects on intraocular pressure provided the difference from baseline is remembered. He suggests that it would be wise to examine the child under the same agent if truly comparable readings are to be obtained. Ketamine, he says, is unlikely to be a satisfactory agent for major ophthalmic surgery.

Intraocular Pressure after Intubation

Succinylcholine is one of the most widely used muscle relaxants in anesthetic practice today. In the present study also we sued succinylcholine to facilitate intubation after induction with either thiopentone or ketamine and we found a definite rise in intraocular pressure after injection of succinylcholine for intubation.

In the present study, in group -I the mean post – intubation intraocular pressure was 16.50+2.10 mmHg and the mean pre – induction intraocular pressure was 13.84+2.75 mmHg, after applying the student 't' test it was found that this rise from baseline intraocular pressure was statistically highly significant ($p<0.01$). The mean rise in intraocular pressure after intubation as compared to the baseline value was 2.68+1.60 mmHg range being 0-7.1 mmHg.

In group – II, the mean pre – induction intraocular pressure was 12.17+2.08 mmHg and the mean post – intubation intraocular pressure was 17.18+1.02 mmHg, After applying the student 't' test it was found that the difference in these two values is statistically highly significant ($p<0.01$). The mean rise in intraocular pressure after intubation as compared to the baseline was 5.16+1.21 mmHg, range being 1.4–7.1 mmHg.

De Roeth A and Schwartz H [28] suggested that the transient rise in intraocular pressure may be caused by a peak depolarizing effect of succinylcholine.

Dillon JB et al. [29] have shown contracture of extraocular muscles in invitro study following succinylcholine injection. This, along with rise in internal jugular venous pressure and apnea due to succinylcholine, raised intraocular pressure.

Wynands JE and Crowell DE [30] showed even bigger and prolonged rise in intraocular pressure following succinylcholine and postulated that succinylcholine caused a rise in cerebrospinal fluid pressure due to increased cerebral blood flow and vascular dilatation. They have shown that intubation causes a further rise in intraocular pressure which is due to stimulation of sympathetic system causing tachycardia and hypertension.

Crythorne NWB et al. [31] used tonometry to measure intraocular pressure and found that following succinylcholine there was an average increase of 7.5 mmHg.

Taylor TH et al. [32] studied selected 50 adult patients presenting for ophthalmic surgery and anesthetised them by a simple technique in which succinylcholine was used to facilitate endotacheal intubation. They found that when intraocular pressure was measured within 1 minute of injection of succinylcholine, a rise in intraocular pressure was found in 20 of 29 patients. Maximum rise was 12 mmHg. The rise was transient and came down within 6 minutes before beginning the surgery.

Pandey K et al. [33] studied the intraocular hypertension action of succinylcholine. They found that the rise in intraocular pressure manifests in 1 minute. Endotracheal intubation following succinylcholine exaggerated the intraocular hypertension. Mean intraocular pressure before intubation was 17.96 mmHg which rose to a mean intraocular pressure of 21.17 mmHg after intubation.

Upadhyaya MR et al. [34] evaluated the efficacy of topical timolol to prevent the intraocular pressure response of succinylcholine and endotracheal intubation. In control group, in which timolol was not used, the intraocular pressure increased significantly and continued to remain above basal level even after 5 minutes (from basal value of 14.96 mmHg to 20.13 mmHg).

The results found in the present study were in close correlation to those conducted by Wynands JE and Crowell DE [30], Taylor TH et al. [32], Pandey K et al. [33] and Upadhyaya MR et al. [34].

Intraocular Pressure after Extubation

In the present study, it was seen that in group -I the intraocular pressure after extubation was 12.56+2.06 mmHg and in group -II it was 12.14+1.72 mmHg. As all the cases in this study were extubated under deep levels of anesthesia so as to avoid any strenuous efforts like coughing, the mean intraocular pressure after extubation did not show any rise.

Comparison of Mean Intraocular Pressure Values between Groups

In the present study, it was seen that there was a fall in intraocular pressure in group -I after induction with thiopentone i.e. from a mean pre – induction intraocular pressure of 13.84+2.75 mmHg to 11.35+2.52 mmHg.

However there was a rise in intraocular pressure in group-II after induction with ketamine i.e. from a mean pre – induction intraocular pressure of 12.17+2.08 mmHg to 14.84+1.94 mmHg.

After applying the student 't' test it was found that the difference in intraocular pressure after induction in group A-1 was statistically highly significant as compared to the intraocular pressure after induction in group A-II ($p<0.01$).

The rise in intraocular pressure after intubation in both the groups was almost same and the difference in these values was not statistically significant ($p>0.05$). This similar rise in intraocular pressure after intubation in both the groups may be because of succinylcholine which was used in both the groups to facilitate intubation.

The intraocular pressure after extubation was similar in both groups and was found to be statistically insignificant ($p>0.05$). This was because in both the groups the patients were extubated under deep levels of anesthesia so as to avoid any strenuous effort.

Murphy DF [35] reviewed that with the exception of ketamine all the agents commonly used for induction of anesthesia, especially thiopentone and pentobarbital, decrease intraocular pressure.

Shaffer – Becker [36] reviewed that intraocular pressure reduced in proportion to the depth of anesthesia after induction with thiopental. They also reviewed that ketamine anesthesia increased intraocular pressure.

Thus, based on the observations cited in the present study, it can be said that thiopentone sodium significantly reduced intraocular pressure after induction whereas ketamine caused a significant rise in intraocular pressure after induction.

Conclusion

- Thiopentone sodium can be safely used as an inducing agent in any intraocular surgery done under general anesthesia.
- Ketamine, as far as possible, should be restricted to extraocular ophthalmic surgeries.

Reference

1. McDonnel PJ, Quigley HA, Maumenee EA, Stark WJ, Hutchins GM. The Honan intraocular pressure reducer – An experimental study. Arch Opthalmol 1985;103: 422–424.

2. Rosen E. Anesthesia for ophthalmic surgery. Br. J. Ophthalmol 1993;77:542-543.
3. Murphy DF. Anesthesia and intraocular pressure. Anaesth – Analog 1985;64:520–30.
4. Kornbleuth W, Aladjemoff L, Magora F, Gabbay A. Influence of general anesthesia on intraocular pressure in man. Arch Ophthalmol 1959;61:84–87.
5. Couch JA, Eltringham RJ, Magauran DM. The effect of thiopentone sodium and fazadium on intraocular on intraocular pressure. Anaesth 1979;34:586–91.
6. Stanley W, Stead MD. General anesthesia for ophthalmic surgery. Anesthesia 1998;11:25–37.
7. Holloway KB. Control of the eye during general anesthesia for intraocular surgery. Br J Anaesth 1980; 52 : 671 – 679.
8. Cohen IT, Drewsen S, Hannallah RS. Propofol or midazolam do not reduce the incidence of emergence agitation associated with desflurane anaesthesia in children undergoing adenotonsillectomy. Paediatr Anaesth 2002;12:604-9.
9. Kim HS, Lee SK, Kang HS, Kim YM, Choi H, Moon HS. Comparison of thiopental and ketamine induction on emergence agitation after desflurane anesthesia in children undergoing a tonsillectomy. Korean J Anesthesiol 2007;53:356-60.
10. Hwang SM, Kim KS, Lim SY. Comparison of emergence agitation from sevoflurane anesthesia in propofol or thiopental sodium induction. Korean J Anesthesiol 2004;46:647-51.
11. Holloway KB. Control of the eye during general anesthesia for intraocular surgery. Br J Anaesth 1980; 52:671–679.
12. Forrester JV. Local anesthesia for eye surgery. Br J Anaesth 1992;76:705.
13. O' Donoghue E, Batterbury M, Lavy T. Effect on intraocular pressure of local anesthesia in eyes under going intraocular surgery. In J Opthalmol 1994;78: 605–07.
14. Allen F. Preoperative tension. Arch Ophthalmol 1963; 70:589.
15. Atkinson. Tonometry before cataract extraction. Arch Ophthalmol 1964;71:293.
16. Stone HH and Prijot EL. The effect of barbiturate and paraldehyde on aqueous humour dynamic in rabbit. Arch Ophthalmol 1955;54:834–40.
17. Von Sallman L, MAcri FJ, Wanko T and Grimes PA. Some mechanisms of centrally induced eye pressure responses. Am J Ophthalmokl 1956;42:130.
18. Sathe ND. Intraocular pressure variations following suxamethonium. In J Anaesth 1973;21:41.
19. Joshi C, Bruce DL. Thiopental and succinylcholine action on intraocular pressure. Anesth Analg 1975; 54:471–75.
20. Col. Banerjee SC and Wg Cdr. Balla GJ. Intraocular pressure profile during general anesthesia. In J Anaesth 1978;177–81.
21. Verma RS. Self taming of succinylcholine induced fasciculations and intraocular pressure. Anesthesiology 1979;50:245–47.
22. Corssen G, Domino EF. Dissociative anesthesia – Pharmacologic studies and first clinical experience withphenyclidine derivative CI – 581. Anaesth Analg 1966;45:29.
23. Adams AK, Barnett KC. Anesthesia and intraocular pressure. Anesthesia 1966;21:202–10.
24. Corsssen G and Hoy JE. A new parenteral anesthetic CI – 581 – its effect on intraocular pressure. Paed Ophthalmol 1967;4:20–23.
25. Yoshikawa K and Murai Y. The effect of ketamine on intraocular pressure in children. Anaesth Analg 1971;50:199–202.
26. Dave B, Chokshi JM and Bandopadhyaya AK. Ketamine and intraocular pressure. In J Ophthalmol 1976;24(II):5–8.
27. Adams A. Ketamine in pediatric ophthalmic practice. Anaesth 1973;28:212–13.
28. DeRoeth A and Schwartz H. Aqueous humour dynamics in glaucoma. Arch Opthalmol 1956;55:755.
29. Dillon JB, Sabawala P, Taylor dB and Gunter R. Depolarising neuromuscular blocking agent and intraocular pressure in vivo. Anesthesiology 1957; 18:439.
30. Wynands JE and Crowell DE. Intraocular tension in association with succinylcholine and endotracheal intubation –a preliminary report. Can An Soc J 1960; 7:39–43.
31. Craythorne NWB, Rottenstein MS and Dripps RD. Effect of succinylcholine on intraocular pressure in adults, in infants and children during ge=neral anesthesia. Anesthesiology 1960;21:59.
32. Taylor TH, Mulcahy M, Nightingale DA. Suxamethonium choloride in intraocular surgery. Br J Anaesth 1968;40:113–118.
33. Pandey K, Badola RP and Kumar S. Time course of intraocular bypertension production by suxamehtonium. Br J Aneas 1972;44:191.
34. Upadhaya MR, Sengupta S, Rawal HH. Efficacy of topical timolol to prevent intraocular pressure response to suxamethonium and endotracheal intubation. IJA 1988;42:38.
35. Murphy DF. Anesthesia and intraocular pressure. Anaesth – Analog 1985;64:520–30.
36. Shaffer – Becker. Diagnosis and therpy of glaucoma. Sixth edition.