Clinical and Experimental Vision and Eye Research (2021), 4, 17–24

ORIGINAL ARTICLE

Ups and Downs of Intraocular Pressure under Anesthesia

Prateek Upadhyay[1], Parul Ichhpujani[2], Shibal Bhartiya[3]

[1]Department of Anaesthesia and Intensive Care, Government Medical College and Hospital, Chandigarh, India, [2]Department of Ophthalmology, Government Medical College and Hospital, Chandigarh, India, [3]Department of Ophthalmology, Fortis Memorial Research Institute, Gurugram, India

Key words:

Intraocular pressure, Anesthesia, Suprachoroidal hemorrhage

Corresponding Author:
Parul Ichhpujani, Department of Ophthalmology, Government Medical College and Hospital, Sector 32, Chandigarh - 160 030, India.
E-mail: parul77@rediffmail.com

Received: 12-06-2021
Accepted: 29-06-2021
doi: 10.15713/ins.clever.62

Abstract

Even though local anesthetic techniques are the cornerstone of ophthalmic anesthesia, the clinical and scientific impact of other kinds of anesthesia cannot be ignored. These include the rapid fluctuations of intraocular pressure (IOP), as well as ocular perfusion imbalance, and can even result in retinal ischemia, visual field defects, and associated ocular morbidity. This becomes especially relevant in patients of glaucoma, open-globe injuries, and retinal detachment. Especially vulnerable is those patients with coexistent diabetes, hypertension, atherosclerosis, and other diseases of vascular dysregulation. Strategies for anesthetic management, therefore, must be three pronged: Control IOP fluctuations to minimize the risk of ischemia as well as expulsive choroidal hemorrhage, safeguard retinal, and ONH perfusion, while providing adequate anesthesia and akinesia. This review aims to critically evaluate the impact of both, anesthetic procedures (including general, regional, and neuraxial anesthesia) and anesthetic agents, on IOP and ocular perfusion. It also endeavors to elucidate the strategies for IOP management during various kinds of anesthesia.

Introduction

Maintenance of intraocular pressure (IOP) in the physiological range during anesthesia is of utmost importance, both for the patient and the doctor. Inadvertent and unnoticed rise in IOP in an anesthetized patient can lead to compromise to optic nerve head. Any form of surgical penetration in the globe with high IOP can lead to catastrophic hemorrhage. Once the ocular chambers are exposed/open, IOP equates to atmospheric pressure and prolapse of the iris and/or lens and loss of vitreous can ensue with any abrupt pressure change. In an eye with already high IOP, any further increase can precipitate acute glaucoma.[1] It is crucial to avoid fluctuations in IOP in an open eye, glaucoma, and retinal detachment to lessen further damage.

When a patient is being administered general anesthesia (GA), induction and emergence are the most critical phases in which substantial rise in IOP. Even a tightly positioned anesthesia mask over patient's eyes is enough to reduce blood flow to zero. Moreover, repeated laryngoscopy and intubation attempts are associated with higher and more detrimental IOP.[2] Hence, vigilance must be maintained in induction with a patient with compromised ocular perfusion or open eye.

Measures to Prevent IOP Elevation at Induction

Video-guided laryngoscopy should be employed in all cases of difficult airway since it is proven to causes a smaller increase in IOP than direct laryngoscopy.[3] Use of supraglottic airway (SGA) causes far less increase in IOP during insertion as well as removal as compared with endotracheal tube.[4,5] Its safety and efficacy are well established. SGAs have been increasingly accepted as a means to secure the airway in patients with minimal risk for aspiration who are having eye surgery with GA.[6] It allows for a smooth emergence.[7] Care must be taken to combat intraoperative laryngospasm, especially in pediatric patients. Correct placement and well securing of SGA must be confirmed before surgery is allowed to proceed since airway is not easily accessible to anesthesiologists in ophthalmic procedures and it warrants for the procedure to be interrupted in case of inadequate ventilation secondary to SGA dislodgment. Bucking/coughing on emergence can be avoided by smooth extubation and aggressive management of post-operative nausea and vomiting (PONV).

Apart from that, inadequate ventilation, hypoxia, hypercapnia, and hypertension are anesthetic concerns that can cause a rise

in IOP.[8] However, the overall effect is IOP reduction in spite of increase in sympathetic outflow secondary to induction.[9] In a recent crosssectional study comparing pre-anesthesia IOP measurements with post-induction IOP measurements, it was noted that IOPs were significantly underestimated for patients under GA. The authors concluded that the actual IOP must be taken as approximately 4–6 mmHg higher than the recorded value. This becomes very relevant while planning treatment for children with glaucoma based on IOP values. The authors also proposed that the normal range for IOP must be considered different under GA.[10]

For measuring IOP of children under anesthesia, inhalational agents (sevoflurane or desflurane) are preferred. It is important that a gap of 3–5 min is maintained between intubation and IOP measurement. Applanation tonometry by Perkins, Tono-Pen, or rebound tonometry should be used for measurement as per availability and performed before pachymetry. It is to be noted that if serial measurements are to be taken, it should be performed by same device as for previous measurement with similar anesthesia protocols. There needs to be avoidance of undue pressure on the globe while indentation and speculum use must be avoided.[11] Teamwork with excellent communication and coordination is essential. The ophthalmologist should be ready with instruments before anesthesiologist starts with induction. For an easy access to eye, anesthesiologist must position the anesthesia equipment such as masks and breathing circuit away from eyes. Mask may be briefly removed for IOP measurement.

GA

Premedication

The chief rationale for premedication is to avoid anxiety and risk of aspiration. Midazolam is commonly administered as anxiolytic. It has a little effect on IOP and hence used for the evaluation of IOP when anxiolysis or mild sedation is indicated.[12] As far as pediatric age group is concerned, premedication with oral midazolam promises an optimal induction with minimal change in IOP by avoidance of agitation and crying.

The goal of IOP measured under anesthesia is to get an adequate idea of the awake IOP. There is neither a significant difference between the IOPs of awake children and those sedated with oral midazolam nor a difference in IOP fluctuation from baseline between those who received midazolam and those who received placebo.[13] Hence, it is a well-tolerated and safe method for IOP measurements in children.

Alprazolam is commonly prescribed to adults as an oral premedication for anxiolysis. Benzodiazepines are known to cause bilateral angle closure as a rare adverse effect which is observed mostly in the elderly.[14] A few nonelderly patients have also presented with bilateral acute angle closure after a single use of two long half-life benzodiazepines, clonazepam, and alprazolam.[15]

Other premedication strategy includes alpha-2 agonists (clonidine and dexmedetomidine) and gabapentin. Both of them attenuate the IOP increase secondary to laryngoscopy, however, there is a decrease in blood pressure (BP) by these drugs which can compromise ocular perfusion.[16,17]

H2 receptor antagonists are commonly prescribed premedication to counter acidity. They are included the classes of medications that have the potential to induce angle closure.[18] Cimetidine and ranitidine by the virtue of their weak anticholinergic adverse effects have potential to precipitate angle closure glaucoma in susceptible people, especially those on prolonged therapy like for duodenal ulcer.[19] Contrarily, Cohen *et al*. studied the effect of intravenous infusion of cimetidine on IOP in medically controlled glaucoma patients and observed no significant change.[[20]

Opioids

The commonly used opioids during induction of anesthesia such as fentanyl and alfentanil not only significantly reduce IOP on induction but also cause a sustained reduction in IOP after succinylcholine (SCh) administration and laryngoscopy. Remifentanil is also as effective as fentanyl and alfentanil in IOP changes.[21-23] The effects of sufentanil and fentanyl on IOP after easy and difficult intubations were studied and it was observed that even though both drugs blunt the increased IOP during laryngoscopy and tracheal intubations, in difficult intubation, sufentanil offered enhanced safety.[23]

Induction agents

The commonly used intravenous induction anesthetics reduce IOP in general. The mechanism is postulated to be a depression of central nervous system ocular centers that result in relaxation of extraocular muscle tone. Propofol, thiopental, and etomidate reduce IOP by up to 40%, 27%, and 30%, respectively.[24,25]

Propofol causes a moderate reduction in IOP even when used as low-dose intravenous sedation has the most profound effect even as sedation agent, causing an IOP reduction of 17–27%.[26] It reduces the rate of aqueous humor formation to a greater extent than it decreases trabecular outflow facility.[27] Propofol prevented a rise in IOP after induction as compared with other drugs (etomidate and thiopental) administered during phacoemulsification (PE) cataract extraction. Furthermore, decrease in BP and heart rate (HR) after induction and LMA insertion were remarkable with propofol used as inducing agent. Thiopental, on the other hand, is observed to have most control on cardiovascular parameters (especially HR) and it also prevents IOP rise.[28] However, as compared with propofol and etomidate, when thiopental is used as inducing agent, it results in a higher IOP and BP post-intubation.[29]

Ketamine is not known to alter IOP. Initially, it was believed to increase IOP significantly when indentation tonometry was used.[30,31] It was due to nystagmus (a side effect of ketamine) that made tonometry result incorrect. When it is employed as a sedative agent for pediatric age group, there is no significant

increase in IOP in patients without eye injuries.[32] Similarly, clinically meaningful associations of ketamine with elevation of IOP are not observed at dosages of 4 mg/kg or less.[33] Peuler *et al*. studied ketamine (2 mg/kg i.v.) in adults and observed no significant effect on IOP.[34] Similarly, another study observed no increase in IOP with ketamine (8 mg/kg intramuscular). In fact, values obtained were similar to those reported with halothane and isoflurane.[35,36] Applanation tonometry has replaced indentation tonometry for measuring IOP. Even though ketamine has no effect on IOP, its adverse effects such as nystagmus, blepharospasm, and agitation/hallucinations during recovery have led to limited usefulness in ophthalmic surgery.

Intraocular pressure decreases significantly by etomidate despite painful intravenous injection and myoclonus, thereby, making it a useful anesthetic agent to preserve IOP during induction.[25,37] Although both etomidate and thiopentone decrease IOP significantly, a far greater decrease in IOP is seen with etomidate.[38] However, the myoclonus after etomidate injection can be dangerous for a patient with a ruptured globe.

Sevoflurane, a volatile anesthetic agent, is commonly used as an induction agent in pediatric patients and is associated with a reduction in IOP within minutes.

Muscle relaxant

Depolarizing agent SCh increases IOP by about 8–10 mmHg.[39] The postulated mechanisms for increase in IOP being reduction in aqueous humor outflow, choroidal blood volume expansion, and increased central venous pressure.[7,40] The rise in IOP is transient ranging over a few minutes. After a rapid injection, within 1–4 min, a peak IOP of approx. 9 mmHg is reached. It returns to baseline with termination of action at around 7 min.[41] This effect can be reduced by administration of lidocaine and sufentanil by a mean of 5 mmHg.[42] Furthermore, alfentanil pre-treatment can prevent the increase in IOP following suxamethonium administration.[43]

It has been shown that pre-treatment with lidocaine before induction with thiopental and SCh shields against IOP rise and may, therefore, be of value in rapid sequence induction for open eye injuries.[44] Despite fears that this SCh-induced increase in IOP may induce extrusion of ocular contents in patients with an open-globe injury, clinical practice in thousands of patients has not reported this complication.[45,46] Still, it is not an ideal agent for the same.

On the contrary, non-depolarizing drugs at equipotent paralyzing doses directly reduce the IOP by relaxing the extraocular muscles.

A significant decrease in IOP by 22.6% is seen with vecuronium bolus.[47] Atracurium or vecuronium alone has no adverse effects on IOP.[48] Rocuronium is associated with a greater reduction in IOP as compared with atracurium and it offers equally favorable intubating conditions as SCh without IOP rise.[49,50] Rocuronium is, therefore, a viable alternative to SCh in an open-globe injury setting. In addition to rocuronium, cisatracurium and mivacurium also result in no changes on IOP in patients undergoing ophthalmic surgery under GA. Therefore, cisatracurium, rocuronium, and mivacurium are safe to be used in ophthalmic anesthesia practice.[51]

Maintenance agents

Total intravenous anesthesia (TIVA) is an emerging concept gaining widespread acceptance in practice to allay atmospheric pollution and greenhouse effect of conventionally used volatile anesthetics. TIVA is promising in ophthalmic anesthesia since accomplishes the basic goals. Not only it reduces BP and IOP but also circumvents PONV to maintain reduced IOP. Peak inspiratory pressure (PIP) in patients under GA being mechanically ventilated has been shown to raise IOP. An increased intrathoracic pressure results in higher PIP which raises central venous pressure. This results in a reduced outflow of aqueous humor through the episcleral venous system and hence an increased IOP.[52] It is found to be significantly lower with propofol-based TIVA than with the conventional volatile anesthesia. Furthermore, the IOP is maintained significantly lower with propofol-TIVA group in IOP raising surgical positions and after pneumoperitoneum creation. It is hence suggested that propofol-based TIVA should be considered as the regimen of choice during anesthesia maintenance, especially in at-risk patients.[53]

The volatile anesthetics, namely, isoflurane, sevoflurane, halothane, desflurane, and nitrous oxide (N_2O) are commonly used for maintenance of anesthesia after intravenous induction and less frequently used for induction when intravenous access is difficult to obtain, for example pediatric patients. It is well known that the volatile anesthetic agents cause a reduction in IOP.[54] They reduce IOP in proportion to anesthetic depth by reducing choroidal volume, relaxing extraocular muscles, and facilitating aqueous humor outflow.[55] It is postulated that this drop in IOP is due to reduction of aqueous humor production, enhancement of aqueous humor outflow, or relaxation of the extraocular muscles. A depression of a central nervous system control center in the diencephalon is also conjectured.[56,57]

Sevoflurane as a maintenance anesthetic agent reduces IOP to the same degree as propofol.[58] However, irrespective of the concentration of sevoflurane (0.5–5%) or the order of administration (from low to high concentration or opposite), this fall in IOP is not significant enough as compared with the awake state. Hence, propofol and sevoflurane are effective anesthetic agents for the evaluation of IOP in adults under anesthesia.[59] The only exception is when remifentanil is used during induction. In that case, IOP reduction is more pronounced with propofol.[60] Clinical studies evaluating the effects of propofol and sevoflurane on respiratory mechanics during surgery found no significant difference in PIP.[61,62]

Desflurane has similar effect on IOP as with propofol in the reverse Trendelenburg position.[63] Yet, when it comes to alleviation of IOP in Trendelenburg position or pneumoperitoneum, it is less effective.[64-66] It has been seen that the total inspiratory resistance of desflurane is significantly

higher than sevoflurane and isoflurane at a 1.5 minimum alveolar concentration.[67]

N_2O has no effect on IOP, even when combined with sevoflurane and remifentanil.[68,69] Although, it is contraindicated in vitreoretinal surgery involving gas tamponade with expandable gases such as sulfur hexafluoride into the vitreous.[70] When nitrous is being administered, any injected bubble can cause a dramatic rise in IOP peaking around 20 min.[71,72] The subsequent rise in IOP can diminish retinal perfusion. It has been noted that N_2O although enhances the internal tamponade effect of the perfluorocarbon intraoperatively, it is followed by a dramatic drop in IOP and volume on discontinuation of N_2O. It is recommended N_2O administration 15 min before gas injection.[72] If a patient with intravitreous gas injection happens to be put under anesthesia, N_2O must be omitted for 5 days after an air injection and for 10 days after sulfur hexafluoride injection. After perfluoropropane injection, N_2O should be prescribed for at least 70 days.[1]

Emergence

About 30% of eye injuries under anesthesia are reported to be related to unplanned patient movement during surgery,[73,74] coughing, or bucking on the ETT during emergence from anesthesia that is accompanied by increase in IOP of 40 mmHg.[75] This can be prevented by extubation in deeper plane, a "*no-touch*" extubation technique, and the use of sympatholytic (such as remifentanil, fentanyl, intravenous lidocaine, and dexmedetomidine).[75-78] Caution needs to be maintained for a smooth emergence, extubation in deeper anesthesia plane remains a viable option. Awake extubation can also be performed safely by a remifentanil-based technique. PONV needs to be avoided since it may cause elevation of IOP.

Reversal

It has been observed that IOP increases from 13.5 to 21 mmHg after neostigmine/glycopyrrolate administration. With sugammadex though, there was no change in IOP. Although IOP was raised post extubation after employing either of the drugs, but it was significantly lower in the sugammadex group as the anticholinergic effect of glycopyrrolate may reduce the aqueous outflow.[79,80]

Special Considerations: Special Conditions under GA

Surgical positioning and IOP

In surgeries requiring steep Trendelenburg positioning such as robotic prostatectomy surgery, IOP increases significantly. This is due to raised CVP positioning and increases in choroidal blood volume from absorbed CO_2 during insufflation. Fortunately, post-operative visual function has been reported to remain unchanged in most of the cases.[81] There are only a few reported incidences of post-operative blindness after prolonged steep Trendelenburg positioning in laparoscopic prostatectomy[82] and colorectal surgery.[83]

Pre-existing comorbid conditions such as atherosclerotic disease, diabetes, and glaucoma may lower the threshold of physiologic tolerance to acute intraocular derangements during laparoscopic surgery. It has been observed that IOP after pneumoperitoneum and Trendelenburg positioning is significantly low when propofol-based TIVA is used. It's because of the inhibitory effect of propofol on arginine vasopressin (AVP).[84-86] AVP and its synthetic derivative desmopressin have been shown to increase IOP.[87,88] AVP concentration remains unaltered with volatile anesthetics.[89] Propofol also inhibits magnocellular neuron excitability in the paraventricular nucleus and supraoptic nucleus through GABA-A-mediated inhibitory currents.[90,91]

LDP is associated with an increase the IOP of the dependent eye[92,93] due to the increased episcleral venous pressure and choroidal volume resulting from gravity or a shift of body fluid and jugular vein compression.[93] Here also, IOP is significantly low when propofol-based TIVA is used, due to the fact that the IOP reducing effect of propofol was greater than the IOP increasing effect of LDP. This does not occur with volatile agents.[66] It has been reported that IOP of the dependent eye increased by 5 mmHg when sevoflurane was used in patients undergoing lung surgery by the end of procedure.[92] In another study, it was noted that IOP increased by around 8 mmHg with sevoflurane and 3.6 mmHg with propofol after 1 h of lateral decubitus positioning.[66]

Hypotensive anesthesia

There is a reduction in IOP and OPP under hypotensive anesthesia. In LDP, IOP reduction can be achieved by a controlled hypotension of 30–40% lower MAP than baseline values. Declines in IOP due to hypotensive anesthesia reduce the difference in the IOP values of dependent and non-dependent eyes to 2–3 mmHg regardless of propofol TIVA or sevoflurane. Propofol can decrease IOP more effectively than desflurane during hypotensive anesthesia since IOP is significantly lower in patients receiving propofol TIVA when compared to desflurane.[94]

Valsalva maneuver employed in spine surgery/ENT procedures increase IOP significantly.[95] Inadequate depth of anesthesia and neuromuscular block allows the patient to cough and perform Valsalva. It generates larger increases in IOP and thus must be avoided.[96]

Regional Anesthesia

The orbital cavity is a closed space with a volume of 30 mL and IOP is bound to increase with injection into or around it.[97] Ocular blocks are reported to initially increase IOP by 5–10 mmHg which lasts for 5 min. Due to a large volume of local anesthetic injection which further has tendency to cause proptosis, peribulbar blocks cause the greatest increase in IOP.[98] The initial increase in IOP is most marked in the peribulbar group due to injection of higher volume compared with the retrobulbar and sub-Tenon's technique.[99] Retrobulbar anesthesia has potential to cause ocular ischemia since it reduces

retrobulbar blood flow velocity by compression of the posterior ciliary arteries or disturbing the autoregulation.[100] Mechanical compression causes a decrease in vitreous volume, increase in systemic absorption of orbital extracellular fluid, and increase in aqueous humor outflow which further reduces the IOP.[101]

This increase in IOP becomes problematic in open-globe repair surgery and can be countered using hyaluronidase mixed into the drug which acts swiftly with its dissipation. Gentle non-continuous digital pressure or an ocular compression device can be used at a set pressure (25 mmHg) to reduce the volume of blood and aqueous in the eye.[102] These measures of preventing IOP rise must be avoided in hypertensive eyes because induced pressures can exceed the central retinal artery perfusion pressure.[103] It should be known that after removal of such device, the eye is expected to be transiently hypotonic until blood and aqueous volumes are reestablished. Ocular compression is not needed for IOP reduction when using local anesthesia for cataract surgery.[104]

Interscalene block (ISB) is a commonly applied nerve block by anesthesiologists. It is known to be associated with Horner's syndrome due to spread of local anesthetic to the cervical sympathetic chain. The decrease in sympathetic tone is feared to raise IOP. However, it is reported that ISB decreases IOP in the blocked side and it is a safe regional technique of choice in elderly patients at high risk for developing glaucoma.[105]

IOP Under Neuraxial Anesthesia

Spinal anesthesia alone has no acute and subacute effects on IOP.[106] Moreover, it has been seen that IOP increase is significantly less in patients who undergo lumbar disc surgery in the prone position under SA compared with GA.[107]

Conclusion

Perioperative IOP fluctuations can result in decreased ocular perfusion causing decreased ONH and retinal perfusion (decreased IOP), as well as an increased risk of expulsive suprachoroidal hemorrhage (increased IOP). Both of these eventualities can adversely impact the visual outcomes of the surgery. The challenge, therefore, is to optimize both, surgical conditions and outcomes, by choice of the kind of anesthesia and anesthetic agents. The Holy Grail for ophthalmic anesthesia, therefore, is a management strategy that ensures globe akinesia, no perioperative pain, low to normal IOP, ocular perfusion pressure control, attenuation of the oculocardiac reflex, minimal bleeding, adequate oxygenation and normocarbia, as well as a smooth reversal of the anesthesia.

References

1. Barash PG. Clinical Anesthesia. 7th ed. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams and Wilkins; 2013.
2. Bithal PK, Mohanty B, Reddy TS, Prabhakar H. Effect of repeat laryngoscopy on intraocular pressure. Eur J Anaesthesiol 2004;21:496-503.
3. Ahmad N, Zahoor A, Riad W, Al Motowa S. Influence of GlideScope assisted endotracheal intubation on intraocular pressure in ophthalmic patients. Saudi J Anaesth 2015;9:195-8.
4. Obsa MS, Kanche ZZ, Olana Fite R, Tura TS, Adema BG, Kinfe AA, *et al*. Effect of laryngeal mask airway insertion on intraocular pressure response: Systematic review and meta-analysis. Anesthesiol Res Pract 2020;2020:7858434.
5. Wainwright AC. Positive pressure ventilation and the laryngeal mask airway in ophthalmic anaesthesia. Br J Anaesth 1995;75:249-50.
6. Lamb K, James MF, Janicki PK. The laryngeal mask airway for intraocular surgery: Effects on intraocular pressure and stress responses. Br J Anaesth 1992;69:143-7.
7. Thomson KD. The effect of the laryngeal mask airway on coughing after eye surgery under general anesthesia. Ophthalmic Surg 1992;23:630-1.
8. Kelly DJ, Farrell SM. Physiology and role of intraocular pressure in contemporary anesthesia. Anesth Analg 2018;126:1551-62.
9. Shribman AJ, Smith G, Achola KJ. Cardiovascular and catecholamine responses to laryngoscopy with and without tracheal intubation. Br J Anaesth 1987;59:295-9.
10. Senthil S, Nakka M, Rout U, Ali H, Choudhari N, Badakere S, *et al*. Changes in intraocular pressures associated with inhalational and mixed anesthetic agents currently used in ophthalmic surgery. Indian J Ophthalmol 2021;69:1808-14.
11. Mikhail M, Sabri K, Levin AV. Effect of anesthesia on intraocular pressure measurement in children. Surv Ophthalmol 2017;62:648-58.
12. Carter K, Faberowski LK, Sherwood MB, Berman LS, McGorray S. A randomized trial of the effect of midazolam on intraocular pressure. J Glaucoma 1999;8:204-7.
13. Oberacher-Velten I, Prasser C, Rochon J, Ittner KP, Helbig H, Lorenz B. The effects of midazolam on intraocular pressure in children during examination under sedation. Br J Ophthalmol 2011;95:1102-5.
14. Park MY, Kim WJ, Lee E, Kim C, Son SJ, Yoon JS, *et al*. Association between use of benzodiazepines and occurrence of acute angle-closure glaucoma in the elderly: A population-based study. J Psychosom Res 2019;122:1-5.
15. Matos AG, Castillo PD, Bisneto JA, Paula JS. Acute angle closure triggered by oral benzodiazepines. Arq Bras Oftalmol 2021;84:170-3.
16. Jaakola ML, Ali-Melkkilä T, Kanto J, Kallio A, Scheinin H, Scheinin M. Dexmedetomidine reduces intraocular pressure, intubation responses and anaesthetic requirements in patients undergoing ophthalmic surgery. Br J Anaesth 1992;68:570-5.
17. Kaya FN, Yavascaoglu B, Baykara M, Altun GT, Gülhan N, Ata F. Effect of oral gabapentin on the intraocular pressure and haemodynamic responses induced by tracheal intubation. Acta Anaesthesiol Scand 2008;52:1076-80.
18. Razeghinejad MR, Pro MJ, Katz LJ. Non-steroidal drug-induced glaucoma. Eye (Lond) 2011;25:971-80.
19. Dobrilla G, Felder M, Chilovi F, de Pretis G. Exacerbation of glaucoma associated with both cimetidine and ranitidine. Lancet 1982;1:1078.
20. Cohen MM, Feldman F, Clark L, Hudy D. Effect of cimetidine on intraocular pressure in patients with glaucoma. Can J Ophthalmol 1984;19:212-4.
21. Sweeney J, Underhill S, Dowd T, Mostafa SM. Modification

by fentanyl and alfentanil of the intraocular pressure response to suxamethonium and tracheal intubation. Br J Anaesth 1989;63:688-91.
22. Domi RQ. A comparison of the effects of sufentanil and fentanyl on intraocular pressure changes due to easy and difficult tracheal intubations. Saudi Med J 2010;31:29-31.
23. Sator-Katzenschlager SM, Oehmke MJ, Deusch E, Dolezal S, Heinze G, Wedrich A. Effects of remifentanil and fentanyl on intraocular pressure during the maintenance and recovery of anaesthesia in patients undergoing non-ophthalmic surgery. Eur J Anaesthesiol 2004;21:95-100.
24. Mirakhur RK, Shepherd WF, Darrah WC. Propofol or thiopentone: Effects on intraocular pressure associated with induction of anaesthesia and tracheal intubation (facilitated with suxamethonium). Br J Anaesth 1987;59:431-6.
25. Famewo CE, Odugbesan CO, Osuntokun OO. Effect of etomidate on intra-ocular pressure. Can Anaesth Soc J 1977;24:712-6.
26. Neel S, Deitch R Jr., Moorthy SS, Dierdorf S, Yee R. Changes in intraocular pressure during low dose intravenous sedation with propofol before cataract surgery. Br J Ophthalmol 1995;79:1093-7.
27. Artru AA. Trabecular outflow facility and formation rate of aqueous humor during propofol, nitrous oxide, and halothane anesthesia in rabbits. Anesth Analg 1993;77:564-9.
28. Alipour M, Derakhshan A, Pourmazar R, Abrishami M, Ghanbarabadi VG. Effects of propofol, etomidate, and thiopental on intraocular pressure and hemodynamic responses in phacoemulsification by insertion of laryngeal mask airway. J Ocul Pharmacol Ther 2014;30:665-9.
29. Sahraei R, Mohsen A, Navid K, Fatemeh E. The comparison of the influence of thiopental and propofol on intraocular pressure during induction of anesthesia in intubated patients under cataracct surgery. Int J Med Res Health Sci 2016;5:147-51.
30. Yoshikawa K, Murai Y. Effect of ketamine on intraocular pressure in children. Anesth Analg 1971;50:199-202.
31. Corssen G, Hoy JE. A new parenteral anesthetic-CI581: Its effect on intraocular pressure. J Pediatr Ophthalmol 1967;4:20.
32. Halstead SM, Deakyne SJ, Bajaj L, Enzenauer R, Roosevelt GE. The effect of ketamine on intraocular pressure in pediatric patients during procedural sedation. Acad Emerg Med 2012;19:1145-50.
33. Drayna PC, Estrada C, Wang W, Saville BR, Arnold DH. Ketamine sedation is not associated with clinically meaningful elevation of intraocular pressure. Am J Emerg Med 2012;30:1215-8.
34. Peuler M, Glass DD, Arens JF. Ketamine and intraocular pressure. Anesthesiology 1975;43:575-8.
35. Ausinsch B, Rayburn RL, Munson ES, Levy NS. Ketamine and intraocular pressure in children. Anesth Analg 1976;55:773-5.
36. Ausinsch B, Graves SA, Munson ES, Levy NS. Intraocular pressure in children during isoflurane and halothane anesthesia. Anesthesiology 1975;42:167-72.
37. Thompson MF, Brock-Utne 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.
38. Calla S, Gupta A, Sen N, Garg IP. Comparison of the effects of etomidate and thiopentone on intraocular pressure. Br J Anaesth 1987;59:437-9.
39. Cook JH. The effect of suxamethonium on intraocular pressure. Anaesthesia 1981;36:359-65.
40. Adams AK, Barnett KC. Anaesthesia and intraocular pressure. Anaesthesia 1966;21:202-10.
41. Kelly RE, Dinner M, Turner LS, Haik B, Abramson DH, Daines P. Succinylcholine increases intraocular pressure in the human eye with the extraocular muscles detached. Anesthesiology 1993;79:948-52.
42. Moeini HA, Soltani HA, Gholami AR, Masoudpour H. The effect of lidocaine and sufentanil in preventing intraocular pressure increase due to succinylcholine and endotracheal intubation. Eur J Anaesthesiol 2006;23:739-42.
43. Polarz H, Böhrer H, Fleischer F, Huster T, Bauer H, Wolfrum J. Effects of thiopentone/suxamethonium on intraocular pressure after pretreatment with alfentanil. Eur J Clin Pharmacol 1992;43:311-3.
44. Grover VK, Lata K, Sharma S, Kaushik S, Gupta A. Efficacy of lignocaine in the suppression of the intraocular pressure response to suxamethonium and tracheal intubation. Anaesthesia 1989;44:22-5.
45. Libonati MM, Leahy JJ, Ellison N. The use of succinylcholine in open eye surgery. Anesthesiology 1985;62:637-40.
46. Vachon CA, Warner DO, Bacon DR. Succinylcholine and the open globe. Tracing the teaching. Anesthesiology 2003;99:220-3.
47. Jantzen JP, Hackett GH, Erdmann K, Earnshaw G. Effect of vecuronium on intraocular pressure. Br J Anaesth 1986;58:433-6.
48. Schneider MJ, Stirt JA, Finholt DA. Atracurium, vecuronium, and intraocular pressure in humans. Anesth Analg 1986;65:877-82.
49. Vinik HR. Intraocular pressure changes during rapid sequence induction and intubation: A comparison of rocuronium, atracurium, and succinylcholine. J Clin Anesth 1999;11:95-100.
50. Chiu CL, Jaais F, Wang CY. Effect of rocuronium compared with succinylcholine on intraocular pressure during rapid sequence induction of anaesthesia. Br J Anaesth 1999;82:757-60.
51. Li S, Hu X, Tan F, Li W. Effects of cisatracurium, rocuronium, and mivacurium on intraocular pressure during induction of general anesthesia in ophthalmic surgery. Drug Des Devel Ther 2020;14:1203-8.
52. Awad H, Santilli S, Ohr M, Roth A, Yan W, Fernandez S, *et al.* The effects of steep trendelenburg positioning on intraocular pressure during robotic radical prostatectomy. Anesth Analg 2009;109:473-8.
53. Chang CY, Chien YJ, Wu MY. Attenuation of increased intraocular pressure with propofol anesthesia: A systematic review with meta-analysis and trial sequential analysis. J Adv Res 2020;24:223-38.
54. Mirakhur RK, Elliott P, Shepherd WF, McGalliard JN. Comparison of the effects of isoflurane and halothane on intraocular pressure. Acta Anaesthesiol Scand 1990;34:282-5.
55. McGraw Hill Education, Butterworth JF, Mackey DC, Wasnick JD. Anesthesia for ophthalmic surgery. In: Morgan and Mikhail's Clinical Anesthesiology. Ch. 36. United States: McGraw Hill Education; 2018. p. 773-85.
56. Duncalf D, Foldes FF. Effect of anesthetic drugs and muscle relaxants on intraocular pressure. In: Smith RB, editor. Anesthesia in Ophthalmology. Boston, MA: Little Brown; 1973. p. 21.
57. Park JT, Lim HK, Jang KY, Um DJ. The effects of desflurane and sevoflurane on the intraocular pressure associated with endotracheal intubation in pediatric ophthalmic surgery. Korean J Anesthesiol 2013;64:117-21.

58. Sator-Katzenschlager S, Deusch E, Dolezal S, Michalek-Sauberer A, Grubmüller R, Heinze G, *et al*. Sevoflurane and propofol decrease intraocular pressure equally during non-ophthalmic surgery and recovery. Br J Anaesth 2002;89:764-6.
59. Gofman N, Cohen B, Matot I, Cattan A, Dotan G, Stolovitch C, Ela-Dalman N. Do intraocular pressure measurements under anesthesia reflect the awake condition? J Glaucoma 2017;26:299-302.
60. Schäfer R, Klett J, Auffarth G, Polarz H, Völcker HE, Martin E, *et al*. Intraocular pressure more reduced during anesthesia with propofol than with sevoflurane: Both combined with remifentanil. Acta Anaesthesiol Scand 2002;46:703-6.
61. Bang SR, Lee SE, Ahn HJ, Kim JA, Shin BS, Roe HJ, *et al*. Comparison of respiratory mechanics between sevoflurane and propofol-remifentanil anesthesia for laparoscopic colectomy. Korean J Anesthesiol 2014;66:131-5.
62. Salihoglu Z, Demiroluk S, Demirkiran O, Emin I, Kose Y. Effects of sevoflurane, propofol and position changes on respiratory mechanics. Middle East J Anaesthesiol 2004;17:811-8.
63. Asuman AO, Baris A, Bilge K, Bozkurt S, Nurullah B, Meliha K, *et al*. Changes in intraocular pressures during laparoscopy: A comparison of propofol total intravenous anesthesia to desflurane-thiopental anesthesia. Middle East J Anaesthesiol 2013;22:47-52.
64. Hwang JW, Oh AY, Hwang DW, Jeon YT, Kim YB, Park SH. Does intraocular pressure increase during laparoscopic surgeries? It depends on anesthetic drugs and the surgical position. Surg Laparosc Endosc Percutan Tech 2013;23:229-32.
65. Seo KH, Kim YS, Joo J, Choi JW, Jeong HS, Chung SW. Variation in intraocular pressure caused by repetitive positional changes during laparoscopic colorectal surgery: A prospective, randomized, controlled study comparing propofol and desflurane anesthesia. J Clin Monit Comput 2018;32:1101-9.
66. Yamada MH, Takazawa T, Iriuchijima N, Horiuchi T, Saito S. Changes in intraocular pressure during surgery in the lateral decubitus position under sevoflurane and propofol anesthesia. J Clin Monit Comput 2016;30:869-74.
67. Nyktari V, Papaioannou A, Volakakis N, Lappa A, Margaritsanaki P, Askitopoulou H. Respiratory resistance during anaesthesia with isoflurane, sevoflurane, and desflurane: A randomized clinical trial. Br J Anaesth 2011;107:454-61.
68. Lalwani K, Fox EB, Fu R, Edmunds B, Kelly LD. The effect of nitrous oxide on intra-ocular pressure in healthy adults. Anaesthesia 2012;67:256-60.
69. Goyagi T, Sato T, Horiguchi T, Nishikawa T. The effect of nitrous oxide on the intraocular pressure in patients undergoing abdominal surgery under sevoflurane and remifentanil anesthesia. Open J Anesthesiol 2016;6:85.
70. Stinson TW 3rd, Donlon JV Jr. Interaction of intraocular air and sulfur hexafluoride with nitrous oxide: A computer simulation. Anesthesiology 1982;56:385-8.
71. Wolf GL, Capriano C, Hartung J. Effects of nitrous oxide on gas bubble volume in the anterior chamber. Arch Ophthalmol 1985;103:418-9.
72. Stinson TW, Donlon JV. Interaction of SF6 and air with nitrous oxide. Anesthesiology 1979;51:S16.
73. Fagan C, Frizelle HP, Laffey J, Hannon V, Carey M. The effects of intracuff lidocaine on endotracheal-tube-induced emergence phenomena after general anesthesia. Anesth Analg 2000;91:201-5.
74. Kim ES, Bishop MJ. Cough during emergence from isoflurane anesthesia. Anesth Analg 1998;87:1170-4.
75. Macri FJ. Vascular pressure relationships and the intraocular pressure. Arch Ophthalmol 1961;65:571-4.
76. Aouad MT, Al-Alami AA, Nasr VG, Souki FG, Zbeidy RA, Siddik-Sayyid SM. The effect of low-dose remifentanil on responses to the endotracheal tube during emergence from general anesthesia. Anesth Analg 2009;108:1157-60.
77. Gonzalez RM, Bjerke RJ, Drobycki T, Stapelfeldt WH, Green JM, Janowitz MJ, *et al*. Prevention of endotracheal tube-induced coughing during emergence from general anesthesia. Anesth Analg 1994;79:792-5.
78. Sheta SA, Abdelhalim AA, Nada E. Evaluation of "no touch" extubation technique on airway-related complications during emergence from general anesthesia. Saudi J Anaesth 2011;5:125-31.
79. Yagan O, Karakahya RH, Tas N, Canakci E, Hanci V, Yurtlu BS. Intraocular pressure changes associated with tracheal extubation: Comparison of sugammadex with conventional reversal of neuromuscular blockade. J Pak Med Assoc 2015;65: 1219-25.
80. Greenstein SH, Abramson DH, Pitts WR 3rd. Systemic atropine and glaucoma. Bull N Y Acad Med 1984;60:961-8.
81. Hoshikawa Y, Tsutsumi N, Ohkoshi K, Serizawa S, Hamada M, Inagaki K, *et al*. The effect of steep Trendelenburg positioning on intraocular pressure and visual function during robotic-assisted radical prostatectomy. Br J Ophthalmol 2014;98: 305-8.
82. Weber ED, Colyer MH, Lesser RL, Subramanian PS. Posterior ischemic optic neuropathy after minimally invasive prostatectomy. J Neuroophthalmol 2007;27:285-7.
83. Kumar G, Vyakarnam P. Postoperative vision loss after colorectal laparoscopic surgery. Surg Laparosc Endosc Percutan Tech 2013;23:e87-8.
84. Viinamki O, Punnonen R. Vasopressin release during laparoscopy: Role of increased intra-abdominal pressure. Lancet 1982;1:175-6.
85. Joris JL, Chiche JD, Canivet JL, Jacquet NJ, Legros JJ, Lamy ML. Hemodynamic changes induced by laparoscopy and their endocrine correlates: Effects of clonidine. J Am Coll Cardiol 1998;32:1389-96.
86. Berg K, Wilhelm W, Grundmann U, Ladenburger A, Feifel G, Mertzlufft F. Laparoscopic cholecystectomy-effect of position changes and Co_2 pneumoperitoneum on hemodynamic, respiratory and endocrinologic parameters. Zentralbl Chir 1997;122:395-404.
87. Gondim EL, Liu JH, Costa VP, Weinreb RN. Exogenous vasopressin influences intraocular pressure via the V(1) receptors. Curr Eye Res 2001;22:295-303.
88. Wallace I, Moolchandani J, Krupin T, Wulc A, Stone RA. Effects of systemic desmopressin on aqueous humor dynamics in rabbits. Invest Ophthalmol Vis Sci 1988;29:406-10.
89. Leighton KM, Lim SL, Wilson N. Arginine vasopressin response to anaesthesia produced by halothane, enflurane and isoflurane. Can Anaesth Soc J 1982;29:563-6.
90. Shirasaka T, Yoshimura Y, Qiu DL, Takasaki M. The effects of propofol on hypothalamic paraventricular nucleus neurons in the rat. Anesth Analg 2004;98:1017-23.
91. Inoue Y, Shibuya I, Kabashima N, Noguchi J, Harayama N, Ueta Y. The mechanism of inhibitory actions of propofol on rat

supraoptic neurons. Anesthesiology 1999;91:167-78.
92. Hwang JW, Jeon YT, Kim JH, Oh YS, Park HP. The effect of the lateral decubitus position on the intraocular pressure in anesthetized patients undergoing lung surgery. Acta Anaesthesiol Scand 2006;50:988-92.
93. Lee JY, Yoo C, Jung JH, Hwang YH, Kim YY. The effect of lateral decubitus position on intraocular pressure in healthy young subjects. Acta Ophthalmol 2012;90:e68-72.
94. Kim YS, Han NR, Seo KH. Changes of intraocular pressure and ocular perfusion pressure during controlled hypotension in patients undergoing arthroscopic shoulder surgery: A prospective, randomized, controlled study comparing propofol, and desflurane anesthesia. Medicine (Baltimore) 2019;98:e15461.
95. Kappmeyer K, Lanzl IM. Intra-ocular pressure during and after playing high and low resistance wind instruments. Ophthalmologe 2010;107:41-6.
96. Cunningham AJ, Barry P. Intraocular pressure-physiology and implications for anaesthetic management. Can Anaesth Soc J 1986;33:195-208.
97. Alwitry A, Koshy Z, Browning AC, Kiel W, Holden R. The effect of sub-Tenon's anaesthesia on intraocular pressure. Eye (Lond) 2001;15:733-5.
98. Bowman R, Liu C, Sarkies N. Intraocular pressure changes after peribulbar injections with and without ocular compression. Br J Ophthalmol 1996;80:394-7.
99. Patton N, Malik TY, Aslam TM, Vallance JH. Effect of volume used in sub-Tenon's anaesthesia on efficacy and intraocular pressure: A randomized clinical trial of 3 ml versus 5 ml. Clin Exp Ophthalmol 2004;32:488-91.
100. Huber KK, Remky A. Effect of retrobulbar versus subconjunctival anaesthesia on retrobulbar haemodynamics. Br J Ophthalmol 2005;89:719-23.
101. Robbins R, Blumenthal M, Galin MA. Reduction of vitreous weight by ocular massage. Am J Ophthalmol 1970;69:603-7.
102. Gayer S, Denham D, Alarakhia K, Bernal A, Cardenas G, Duncan R, *et al.* Ocular decompression devices: Liquid mercury balloon vs the tungsten powder balloon. Am J Ophthalmol 2006;142:500-1.
103. McDonnell PJ, Quigley HA, Maumenee AE, Stark WJ, Hutchins GM. The Honan intraocular pressure reducer. An experimental study. Arch Ophthalmol 1985;103:422-5.
104. Watkins R, Beigi B, Yates M, Chang B, Linardos E. Intraocular pressure and pulsatile ocular blood flow after retrobulbar and peribulbar anaesthesia. Br J Ophthalmol 2001;85:796-8.
105. Basaran B, Yilbas AA, Gultekin Z. Effect of interscalene block on intraocular pressure and ocular perfusion pressure. BMC Anesthesiol 2017;17:144.
106. Hatipoglu S, Abdullayev R, Kucukebe OB, Guler M, Hatipoglu F, Celik B, *et al.* Intraocular pressure changes after spinal anesthesia-acute and subacute effects on surgery patients. Adv Clin Exp Med 2015;24:857-61.
107. Pınar HU, Kaşdoğan ZE, Başaran B, Çöven İ, Karaca Ö, Doğan R. The effect of spinal versus general anesthesia on intraocular pressure in lumbar disc surgery in the prone position: A randomized, controlled clinical trial. J Clin Anesth 2018;46:54-8.

How to cite this article: Upadhyay P, Ichhpujani P, Bhartiya S. Ups and Downs of Intraocular Pressure under Anesthesia. Cli Exp Vis Eye Res J 2021;4(1):17-24.