Brazilian Journal of
ANESTHESIOLOGY

EDITORIAL

Comprehensive perioperative eye protection

Total postoperative visual loss is a rare complication that has been described mainly after spine surgeries,[1] and less frequently after heart surgery or other procedures.[2] Several types of ophthalmologic complications can increase morbidity and the risk of postoperative *delirium*, postponing rehabilitation and contributing to increase hospital costs, in addition to the potential decline in quality of life after hospital discharge. In addition to scarce studies on postoperative visual changes, there is no consensus on risk factors associated with those complications and their actual incidence. Similar to organic cardiovascular, pulmonary, or neurological protection, eye protection should be taken into account, especially in view of risk of visual loss in specific types of surgeries.

It is important to consider that the visual system has an elaborate compensation mechanism for homeostatic changes and that neuroadaptation is highly compensatory, making changes that occur and their consequences minimal. The first study on postoperative visual changes was published in 1987 as a prospective assessment of patients submitted to cardiovascular surgeries, in which postoperative neuro-ophthalmological changes were found and characterized by areas of retinal infarction or emboli, changes in visual field, decreased visual acuity, and Horner Syndrome.[3] Although retrospective studies with thousands of patients have been published, the actual incidence of postoperative visual loss is not known.[4,5] A Brazilian retrospective study that included 39,431 patients submitted to non-ocular surgeries reported nine cases of ocular events (2.3:10,000), one case of which of blurry vision, and no cases related to visual loss.[6]

Few prospective studies have assessed perioperative ocular or visual events. The limitations of intraoperative intraocular pressure (IOP) monitoring, measurement of the optic nerve sheath diameter, or even of measuring intracranial pressure (ICP) render possible preventive and protective actions for visual loss difficult. IOP is usually measured by applanation tonometry, which measures the amount of force required to flatten a certain area of the cornea, and values ranging between 10 and 20 mmHg are considered normal. However, the method has many limitations in terms of isolated measurements, and is subject to nychthemeral variation, variation in cornea width, type and quality of the equipment used, body positioning, and technical skills of who measures. Moreover, increased IOP is not necessarily related to optic nerve injury or visual impairment.

Albeit limitations, there is interest for prospective studies assessing perioperative ocular and visual events that add knowledge to the perioperative behavior of IOP, the diameter of the optical nerve sheath, and of ICP itself in different types of surgery. There is also interest in the correlation between the behavior of these data and the occurrence of *delirium*, cognitive dysfunction and other significant perioperative organic changes, and the possible impact of such changes on postoperative outcomes. The present edition of BJAN has articles related to the topic. One of the articles assessed perioperative visual acuity and IOP of patients submitted to major non-cardiac surgeries,[7] and another assessed patients submitted to heart surgery with and without cardiopulmonary bypass.[8] Two other observational studies[9,10] assessed the effect of the head-down tilt position during robotic prostatectomy on IOP, in that one of the studies correlated IOP with the optic nerve sheath diameter. Indirectly related to the topic, this edition also brings an original case report related to ICP and IOP behavior, with noninvasive monitoring during robotic prostatectomy.[11] Last, two studies assessed the role of regional eye anesthesia on IOP measurements and local perfusion. El Fawal et al assessed the minimum effective volume (MEV) for peribulbar blockade and possible association with ocular complications.[12] Cabral et al, in turn, assessed the effect of clonidine as adjuvant to anesthetic blockade on IOP and other eye perfusion parameters.[13]

Postoperative ocular or visual changes in non-ophthalmological surgeries can be classified into four groups:

1 External eye injury or cornea trauma, that can result in irritation, abrasion or laceration, prevented by care in positioning, and using lubricants and eyelid occlusion.[14,15]

2 Cortical blindness, generally accompanied by a parieto-occipital cerebrovascular accident. Cortical blindness may result from global or focal ischemia, hypoxemia, intracranial hypertension, hemorrhage, vascular occlusion, thrombosis, intracranial hemorrhage, vasospasm, embolism, and cardiac arrest.[16]

3 Retinal ischemia or occlusion of the central artery of the retina, that evolves with decreased blood supply to the entire retina, while occlusion of the retinal artery branch is a circumscribed lesion that only affects a portion of the retina. The lesion is unilateral in most cases and the most frequent cause is external eye compression, that leads to increase in IOP and decrease in the central artery flow of the retina.[17-19]

4 Ischemic optic neuropathy (ION), most frequently associated with postoperative spine surgeries,[3,20] can be inflammatory or secondary to an occlusive disorder or non-inflammatory condition of blood vessels. ION can lead to neuronal injury caused by apoptosis, and can be replicated *in vitro* by reducing oxygen supply.[21] Such studies demonstrate that optic nerve ischemia contributes to diffuse axonal destruction. When oxygen supply decreases, adenosine triphosphate is depleted, leading to membrane depolarization and Na^+ and Ca^{2+} inflow through specific voltage-dependent channels with reversal exchange of Na^+ and Ca^{2+} in the pump.[22] Cells become overloaded with Ca^{2+} and impair the activation of proteolytic enzymes and other metabolic processes.

Several predisposing factors have already been described for postoperative visual changes, such as personal history of atherosclerosis, diabetes mellitus, arterial hypertension, obesity, among others. Lack of evidence for identifying actual characteristics and incidence of postoperative visual changes are the rationale for more studies on the topic.[23] Among the several factors described as contributing to postoperative visual loss, are:

- *Prone position during spine surgery:* positioning the head below the heart may lead to increased IOP. This pressure increase mechanism was proven based on the assessment of patients in the head-down tilt position,[24] with higher pressure on episcleral vessels. Eye perfusion pressure results from the difference between mean arterial pressure and IOP. Whenever possible, it is recommended to position a patient's head above the level of the heart, or in a neutral position during spine surgery on a prone position patient, maintaining cardiac output and perfusion pressure of the optical nerve.[25,26]

- *External pressure over the eye:* it is a complication mainly observed during spine surgeries performed with a patient in prone position, and patients with face anatomy abnormality, *osteogenesis imperfecta*, and exophthalmia are more vulnerable to external compression to the eyes. Moreover, retinal ischemia can be related to systemic-related decreased blood perfusion, thrombosis, or local embolism, in addition to impaired retinal venous drainage.[17-19] In order to prevent ischemia, attention should be paid to patient positioning, avoiding eye globe compression or inadvertently leaning of equipment or of a surgical team member on patients' eyes. Observation should be ongoing even when using appropriate positioners, and using a mirror with indirect view of eye positioning is recommended.

- *Arterial hypotension:* brain hypo flow can contribute to a likely change in blood flow in vessels and to the optical nerve. The literature does not offer enough evidence to quantify the level of potentially dangerous hypotension.[16,24]

- *Hemodilution:* hemodilution associated with major anemia can be an additional factor in the impairment of tissue perfusion.[22,27,28]

- *Hypervolemia:* hyper-hydration can make tissue perfusion difficult, and has been described in cases of ION. Given the central retinal vein is near the optical nerve, the ''internal compartment syndrome'' can occur. Alternatively, accumulation of fluid in the proximity to the cribriform plate can compress axons.

- *Vasopressors:* a relationship between prolonged infusion of vasopressors and ION has been described in patients submitted to cardiac surgery[29] and spine surgery.[30] However, the role of vasopressors remains unclear. Hypertensive patients treated with ACE-inhibitors or angiotensin II receptor blockers, frequently along with betablockers or calcium channel blockers, are more prone to developing ION. These patients frequently become unstable during the intraoperative and can require vasopressors for hemodynamic stabilization.[31]

Several risk factors can be present on the same patient unpredictably, especially in those who present some level of arterial hypotension, hypovolemia, and/or anemia. Arterial hypotension, bleeding, prolonged surgery, and volume overload can occur frequently in several patients submitted to major surgery. A combination of these factors, possibly together with abnormal self-regulation in the posterior portion of the optic nerve, pro-thrombotic trends, and other specific patient factors, can lead to enough decreased oxygen supply to the optical nerve to cause ischemic injury.[18,24]

In this edition of BJAN, Badessa et al[7] performed a prospective observational study that included visual acuity and IOP assessment in the pre- and postoperative periods of elderly patients submitted to long major non-ophthalmological surgeries to assess the incidence and risk factors for postoperative visual changes. Among the 107 elderly patients assessed, 21 presented visual function impairment on the third postoperative (PO) day, and of those, 33% on the 21st PO, totaling 6.5% of total patients assessed. It was possible to identify that the changes were characterized by distance visual acuity reduction shown by the Snellen table with change in refraction on the 3rd and 21st PO, accompanied by increased IOP on the 3rd PO, with improvement on the 21st PO. Diabetes mellitus, duration of surgery, and arterial hypotension during induction were considered independent risk factors for postoperative visual dysfunction.

Erol et al.[8] studied 45 patients submitted to cardiac surgery, comparing IOP in procedures with pulsatile and non-pulsatile cardiopulmonary bypass, and surgeries without cardiopulmonary bypass. IOP was assessed before, during, and after surgery, and during cardiopulmonary bypass, it

decreased more prominently in patients submitted to non-pulsatile cardiopulmonary bypass. During head-down tilt positioning for surgeries without cardiopulmonary bypass, some patients presented transient increase in IOP.

Robotic technology for prostatectomy is increasingly frequent. Such procedures, as for robotic surgery for other pelvic procedures, require steep head-down tilt body positioning, with transient increase in IOP, and it is a relative counterindication for patients with open angle glaucoma, aimed at avoiding additional damage to the optical nerve.[32] The presence of ION has been reported for surgeries with over five-hour duration (example: radical laparoscopic prostatectomy), with values of IOP above 40 mmHg.[16,33] This edition of BJAN is publishing two articles related to IOP assessment in robotic prostatectomy. Balkan et al.[9] investigated the effect of the Trendelenburg position (35º to 45º) and abdominal carbonic gas insufflation on the diameter of the optical nerve sheath, IOP and hemodynamics during robot-assisted laparoscopy for prostatectomy in 34 patients. The authors observed increase in IOP during head-down tilt position, without significant correlation with the optical nerve sheath diameter. In another study, Kondo et al.[10] assessed 21 patients to quantify changes in IOP in time in patients put on the Trendelenburg position during robot-assisted laparoscopy for prostatectomy. In addition to confirming increased IOP during Trendelenburg positioning, the authors concluded that the increase was moderate 90 minutes after positioning, with a mean IOP value 7 mmHg above baseline, resuming to the baseline value roughly 30 minutes after returning to the supine position.

Little is known on the intraoperative correlation between ICP and IOP. The new Brain4care™ monitor registers noninvasive intracranial compliance measurements, based on small cranial volume variations, using a tension measurer positioned on temporal bone skin. The monitor analyzes intracranial pressure waves to determine if brain compliance is preserved or not.[34,35] In this edition of BJAN, Saba et al describe the case of an elderly patient submitted to robotic prostatectomy with noninvasive ICP monitoring with Brain4care™.[11] Such monitoring revealed normal intracranial compliance during anesthesia induction, with fast increase after head-down tilt positioning, despite normal vital signs, low pulmonary pressure, and appropriate anesthesia depth. In addition to such monitoring allowing early detection of changes and treating increased ICP, the changes observed coincide with the increase observed in other studies on IOP during robotic surgery head-down tilt position. Future studies can assess the correlation between IOP and noninvasive ICP measurements during robotic surgeries performed in steep head-down tilt position.

Further in this edition, El Fawal et al. assessed MEV for peribulbar blockade.[12] By using an up-and-down method, the authors showed that MEV was inversely proportional to the axial length of the eye globe. Cabral et al, in turn, concluded that adding 1 mcg. kg^{-1} of clonidine to the subtenion blockade for cataract surgeries reduced IOP and ocular pulse amplitude, without affecting eye perfusion pressure.[13] Both studies provide evidence on the correlation between ocular pressure and perfusion measurements, and volume of anesthetics in ophthalmological blocks and adding adjuvants, which is extremely important to avoid complications.

High-risk patients should be identified in the preoperative and the likelihood of postoperative visual impairment should be discussed in detail, along with prevention strategies,[16,24,36] and specialized ophthalmologic assessment may be indicated in some cases. Careful positioning, hemodynamics, hematocrit levels and arterial oxygenation optimization contribute to the prevention of postoperative visual changes.[37] Although ocular perfusion can be normal during normotension, transient increases in IOP should be considered to occur, for example during tracheal extubation, and transiently impair such perfusion.[24,33]

Postoperative ocular or visual impairment should be assessed early by a specialist. In the case of central artery occlusion of the retina, treatment of patients without a history of glaucoma can include eye massage to reduce IOP or attempt to free retinal flow. Intravenous acetazolamide and oxygen inhalation have been attempted to improve dilation and increase oxygen supply from retinal vessels and the choroid, and the indication for thrombolysis can be discussed with an expert, albeit a procedure frequently contraindicated in the immediate postoperative period.[37,38]

More studies are required for assessing risk factors, prevention, monitoring, diagnosis. and treatment of perioperative visual impairment, especially in patients who present transient blurry vision. Comprehensive eye protection in the perioperative should be encouraged, especially for patients submitted to robot-assisted procedures and in Trendelenburg position, during heart or spine surgery, as well as in those with preoperative risk factors and submitted to major surgical procedures. The literature does not present consensus on IOP monitoring or optical nerve sheath diameter measurements during the intraoperative. The possibility of optimizing intraoperative neurological monitoring with noninvasive intracranial pressure measurements can contribute to eye protection and prevention of postoperative complications, especially for high-risk patients.

Conflicts of interest

The authors declare no conflicts of interest.

References

1. Kla KM, Lee LA. Perioperative visual loss. Best Pract Res Clin Anaesthesiol. 2016;30:69–77.
2. Shen Y, Drum M, Roth S. The prevalence of perioperative visual loss in the United States: a 10-year study from 1996 to 2005 of spinal, orthopedic, cardiac, and general surgery. Anesth Analg. 2009;109:1534–45.
3. Shaw PJ, Bates D, Cartlidge NE, et al. Neuro-ophthalmological complications of coronary artery bypass graft surgery. Acta Neurol Scand. 1987;76:1–7.
4. Warner ME, Warner MA, Garrity JA, et al. The frequency of perioperative vision loss. Anesth Analg. 2001;93:1417–21, table of contents.
5. Roth S, Thisted RA, Erickson JP, et al. Eye injuries after nonocular surgery. A study of 60,965 anesthetics from 1988 to 1992. Anesthesiology. 1996;85:1020–7.
6. Kara-Junior N, Espindola RF, Valverde Filho J, et al. Ocular risk management in patients undergoing general anesthesia: an analysis of 39,431 surgeries. Clinics (Sao Paulo). 2015;70:541–3.
7. Badessa GG, Almeida JP, Fukushima JT. Incidence and risk factors of postoperative visual function impairment in elderly

patients undergoing nonocular surgery: a prospective cohort study. Braz J Anesthesiol. 2021;71:599–606.

8. Erol G, Doganci S, Tumer NB, et al. Changes in intraocular pressure during coronary artery bypass graft surgery: an observational study. Braz J Anesthesiol. 2021;71:612–7.

9. Balkan B, Emir NS, Demirayak B, et al. The effect of robotic surgery on intraocular pressure and optic nerve sheath diameter: a prospective study. Braz J Anesthesiol. 2021;71:607–11.

10. Kondo Y, Echigo N, Mihara T, et al. Intraocular pressure during robotic-assisted laparoscopic prostatectomy: a prospective observational study. Braz J Anesthesiol. 2021;71:618–22.

11. Saba GT, Quintao V, Zefeirno S, et al. Noninvasive intracranial pressure real-time waveform analysis monitor during prostatectomy robotic surgery and Trendelenburg position: case report. Braz J Anesthesiol. 2021;71:656–9.

12. El Fawal SM, Nofal WH, Sabek EAS, et al. Minimum effective volume of local anesthetic in peribulbar block: does it differ with the eyeball axial length? Braz J Anesthesiol. 2021;71:635–41.

13. Cabral S, Carraretto AR, Sousa A, et al. Effect of adding clonidine to lidocaine on ocular hemodynamics durign sub-Tenon's anesthesia: randomized double-blind study. Braz J Anesthesiol. 2021;71:628–34.

14. Stambough JL, Dolan D, Werner R, et al. Ophthalmologic complications associated with prone positioning in spine surgery. J Am Acad Orthop Surg. 2007;15:156–65.

15. Grover VK, Kumar KV, Sharma S, et al. Comparison of methods of eye protection under general anaesthesia. Can J Anaesth. 1998;45:575–7.

16. Patil CG, Lad EM, Lad SP, et al. Visual loss after spine surgery: a population-based study. Spine (Phila Pa 1976). 2008;33:1491–6.

17. Sys J, Michielsen J, Mertens E, et al. Central retinal artery occlusion after spinal surgery. Eur Spine J. 1996;5:74–5.

18. Hollenhorst RW, Svien HJ, Benoit CF. Unilateral blindness occurring during anesthesia for neurosurgical operations. AMA Arch Ophthalmol. 1954;52:819–30.

19. Bradish CF, Flowers M. Central retinal artery occlusion in association with osteogenesis imperfecta. Spine (Phila Pa 1976). 1987;12:193–4.

20. Chang SH, Miller NR. The incidence of vision loss due to perioperative ischemic optic neuropathy associated with spine surgery: the Johns Hopkins Hospital Experience. Spine (Phila Pa 1976). 2005;30:1299–302.

21. Levin LA, Louhab A. Apoptosis of retinal ganglion cells in anterior ischemic optic neuropathy. Arch Ophthalmol. 1996;114:488–91.

22. Ho VT, Newman NJ, Song S, et al. Ischemic optic neuropathy following spine surgery. J Neurosurg Anesthesiol. 2005;17:38–44.

23. Kelly DJ, Farrell SM. Physiology and role of intraocular pressure in contemporary anesthesia. Anesth Analg. 2018;126:1551–62.

24. Shahian DM, Speert PK. Symptomatic visual deficits after open heart operations. Ann Thorac Surg. 1989;48:275–9.

25. Roth S, Barach P. Postoperative visual loss: still no answers-yet. Anesthesiology. 2001;95:575–7.

26. Hoski JJ, Eismont FJ, Green BA. Blindness as a complication of intraoperative positioning. A case report. J Bone Joint Surg Am. 1993;75:1231–2.

27. Cheng MA, Sigurdson W, Tempelhoff R, et al. Visual loss after spine surgery: a survey. Neurosurgery. 2000;46:625–30, discussion 30–31.

28. Lee LA, Deem S, Glenny RW, et al. Effects of anemia and hypotension on porcine optic nerve blood flow and oxygen delivery. Anesthesiology. 2008;108:864–72.

29. Shapira OM, Kimmel WA, Lindsey PS, et al. Anterior ischemic optic neuropathy after open heart operations. Ann Thorac Surg. 1996;61:660–6.

30. Lee LA, Lam AM. Unilateral blindness after prone lumbar spine surgery. Anesthesiology. 2001;95:793–5.

31. Lee AG. Ischemic optic neuropathy following lumbar spine surgery. Case report. J Neurosurg. 1995;83:348–9.

32. Awad H, Malik OS, Cloud AR, et al. Robotic surgeries in patients with advanced glaucoma. Anesthesiology. 2013;119:954.

33. Roth S. Perioperative visual loss: what do we know, what can we do? Br J Anaesth. 2009;103 Suppl 1:i31–40.

34. Mascarenhas S, Vilela GH, Carlotti C, et al. The new ICP minimally invasive method shows that the Monro-Kellie doctrine is not valid. Acta Neurochir Suppl. 2012;114:117–20.

35. Frigieri G, Andrade RAP, Dias C, et al. Analysis of a non-invasive intracranial pressure monitoring method in patients with traumatic brain injury. Acta Neurochir Suppl. 2018;126:107–10.

36. Taugher PJ. Visual loss after cardiopulmonary bypass. Am J Ophthalmol. 1976;81:280–8.

37. American Society of Anesthesiologists Task Force on Perioperative Visual Loss. Practice advisory for perioperative visual loss associated with spine surgery: an updated report by the American Society of Anesthesiologists Task Force on Perioperative Visual Loss. Anesthesiology. 2012;116:274–85.

38. Mehta N, Marco RD, Goldhardt R, et al. Central retinal artery occlusion: acute management and treatment. Curr Ophthalmol Rep. 2017;5:149–59.

Maria José Carvalho Carmona [ID] [a,*],
Vinícius Caldeira Quintão [ID] [b]
[a] Universidade de São Paulo (USP), Faculdade de Medicina, Disciplina de Anestesiologia, São Paulo, SP, Brazil
[b] Universidade de São Paulo (USP), Faculdade de Medicina, Hospital das Clínicas, São Paulo, SP, Brazil

*Corresponding author.
E-mail: maria.carmona@fm.usp.br (M.J. Carmona).

27 September 2021