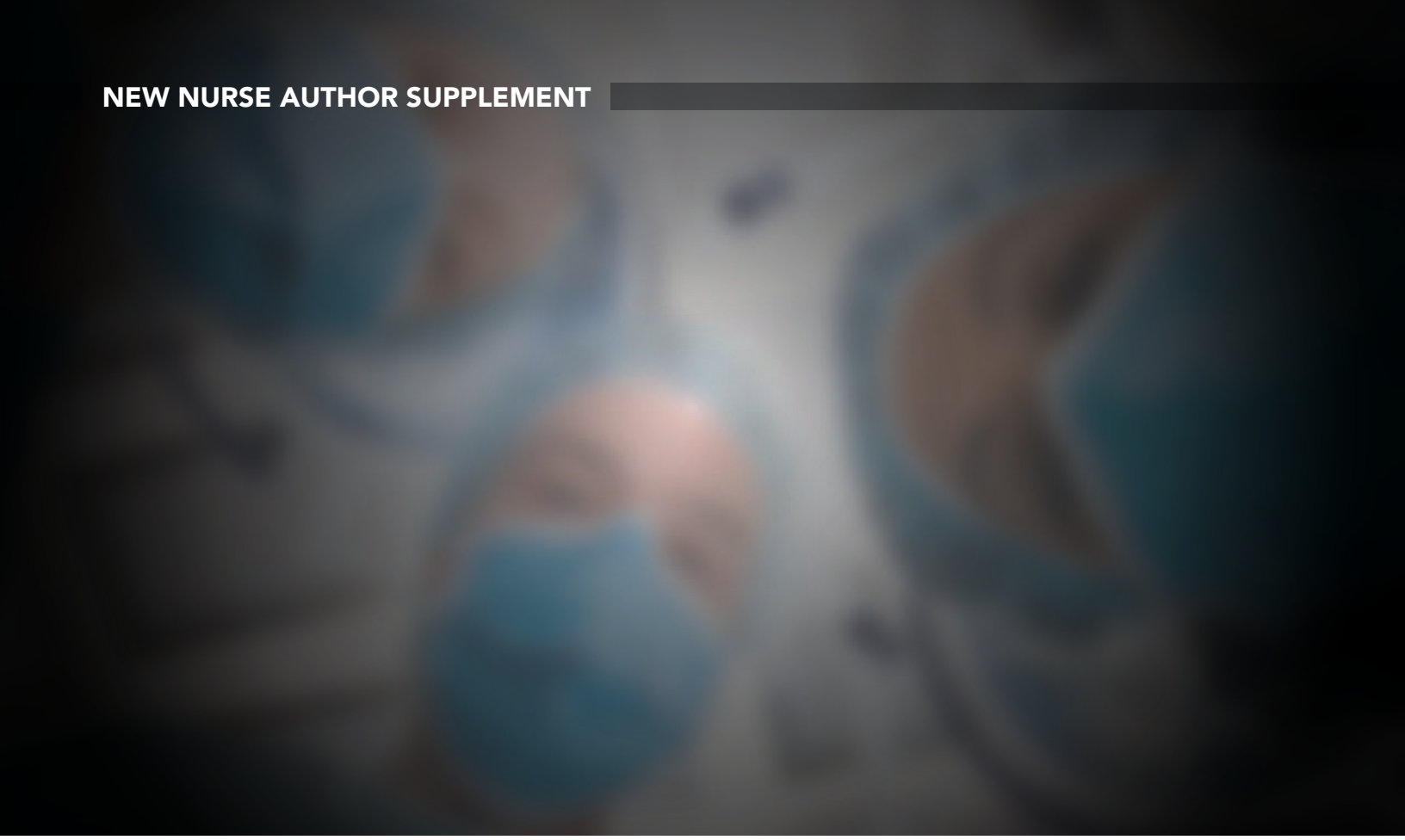

Suddenly Blind: Postoperative Vision Loss

Sandra Krug, CRNA, APRN, RRT

Keywords: POVL, postoperative vision loss, postoperative complications, surgical positioning, Trendelenburg

Postoperative vision loss (POVL) is an uncommon but devastating complication caused by retinal and optic nerve ischemia from prolonged intraocular pressure, related to head-down, prone positioning during anesthesia. LNCs should be aware of this, risk factors, optimal monitoring, and potential treatments when reviewing a case of postoperative blindness.

INTRODUCTION

The plaintiff attorney interviews a woman who wants to sue her surgeon. The patient underwent a posterior cervical disc fusion with instrumentation and was in the operating room for 6 hours. Instrumentation means that metal hardware was attached to the spine. The patient woke up from surgery unable to see. "Does she have a case?" the attorney asks you.

POSTOPERATIVE VISION LOSS

The Centers for Disease Control and Prevention reports vision loss as among the top ten causes of disability in the United States. It is a condition feared by many. People with vision loss are more likely to report depression, diabetes, hearing impairment, stroke, falls, cognitive decline, and premature death. Vision loss often leads to an inability to drive, read, keep accounts, or travel in unfamiliar places, substantially compromising the quality of life. Postoperative vision loss (POVL) not only has profound

implications for emotional well-being and return to preoperative function level but also increases the length of stay postoperatively and leads to higher medical bills. Patients who experience POVL incur an average of 8.6 hospital days versus 4.1 days for those unaffected. The average cost of stay more than doubles: $49,532 compared with $22,697 for those without POVL.[1]

The first documented report of POVL was in 1948 and was attributed to improper head positioning during a procedure with the patient in the prone position.[2] In 2006 an anesthesiologist, Dr. Anthony D. Lehner, experienced POVL following prolonged (7.5 hours) lumbar spine surgery in prone Trendelenburg position. (Figure 1) He stated that he was not able to open his eyes until noon the day after surgery due to edema. His vision loss was permanent, ending his career. Dr. Lehner then advocated for patient education, highlighting the potential risk of POVL. He recommended disclosing the risk of POVL to each patient undergoing a procedure at potential risk at the time of informed consent. Clearly stating the risk of POVL also helps patients and their family members understand this is a complication of positioning, not of the anesthetic itself.[3] *(Ed. note: See also N. Radoslovich, OR positioning for the LNC, JLNC Spring 2019, p. 24)*

INTRAOCULAR PRESSURE (IOP)

Increased intraocular pressure (IOP) of the aqueous humor (fluid) inside the eye can have serious effects. Studies show steep Trendelenburg and prone positions, as well as excessive fluid replacement, influence IOP, and contribute to POVL.[4] The first controlled study of IOP in prone anesthetized patients was by Cheng et al. in 2001. IOP initially decreased at anesthesia induction. However, when the patient was prone versus supine positions under general anesthesia, Cheng et al. found IOP doubled. There was a direct correlation between time spent prone and severity of IOP. They suggest a linear relationship between time spent in the prone position and IOP. Increased IOP associated with periorbital edema, venous hypertension, and abnormal eye fluid mechanics contributes to orbit hypoperfusion by decreasing the pressure gradient below a critical level at the optic nerve and retina[5] resulting in hpoperfusion and anoxic injury to these delicate structures.

When surgery requires steep Trendelenburg or prone position, venous pressure rises from increased intraabdominal and intrathoracic pressure, creating increased IOP. Think how you would feel lying on your face or standing on

Figure 1. Surgical positioning

your head for long periods of time. As venous pressure increases in the head and neck, higher hydrostatic pressure causes capillary leakage. This forces fluid to accumulate in the interstitial space, further constricting venous return and limiting perfusion to the optic nerve. Most cases of ischemic optic neuropathy (ION) occur in the posterior region of the optic nerve, where collateral branches of the pial plexus arteries supply flow. The optic nerve travels through the optic canal and an enclosed space constructed of collagen and bone in the sclera called the lamina cribrosa (Figure 2). Edema in the area compresses the optic nerve, leading to the ischemia.[6]

Ischemic optic neuropathy can lead to permanent optic nerve atrophy. However, vision may be preserved with timely correction of underlying causes (such as perioperative anemia and hypotension), and suitable treatment.

ISCHEMIC OPTIC NEUROPATHY (ION)

Blood supply to the optic nerve originates from the internal carotid artery, which branches into the ophthalmic artery and thence to the central retinal artery. The most common cause for POVL is ION, encompassing approximately 89% of documented POVL cases. As blood supply becomes disrupted in either the anterior or posterior segment, it results in anterior ischemic optic neuropathy (AION) or posterior ischemic optic neuropathy (PION), respectively.[8]

AION is characterized by sudden, painless, bilateral visual deficit ranging from a minor decrease in visual acuity to blindness, unilaterally, or bilaterally.[7] PION in the postoperative period presents as a sudden painless loss of visual acuity, and it may continue to degenerate but typically does not improve.[8]

Unilateral optic nerve dysfunction, visual field deficits, and absence of other causes of decreased vision are indicative of afferent pupillary defect and are diagnostic indicators of ION.

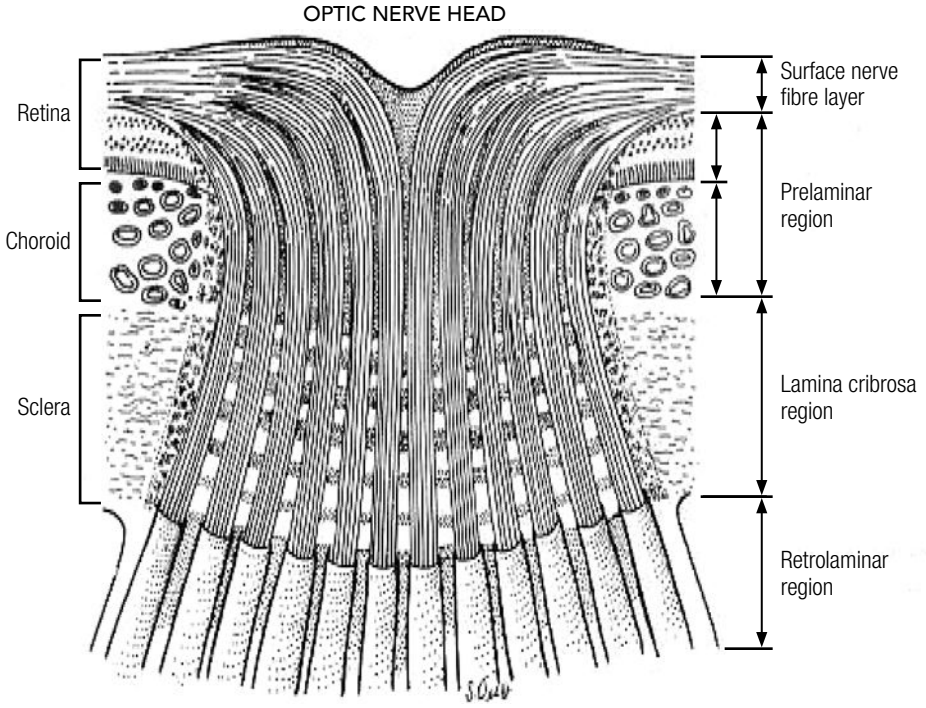

Figure 2

Blood supply determines the extent of the visual loss of the optic nerve. In anemic patients, whose blood has decreased oxygen-carrying capacity, hypotension may lead to an infarction of the optic nerve head where blood supply is vulnerable to compression.

Some treatments for ION include retrobulbar steroid injections, antiplatelet therapy, anticoagulants, phenytoin, norepinephrine, and blood replacement. Other preventative measures include avoidance of prolonged reduction of oxygen delivery to the eye from hypotension or anemia and minimizing the time a patient is prone.[9] A literature review by Buono and Foroozan reveals the correction of hemodynamic derangements as the only proven valuable treatment modality.[10]

ION can lead to permanent optic nerve atrophy. However, vision may be preserved with timely correction of underlying causes (such as perioperative anemia and hypotension), and initiation of suitable treatment.[11] Although there are no accurate statistics, underlying disease factors contributing to POVL are diabetes, vascular disease, and glaucoma. Lee et al. theorize that hypotensive ION occurs from optic nerve compartment syndrome, suggesting high venous pressure and interstitial tissue edema can compromise blood flow to the optic nerve. They cite increased blood loss and prolonged anesthetic time as factors associated with POVL.

The American Society of Anesthesiologist (ASA) POVL Registry cites

increased blood loss and hemodilution to be prominent in POVL. This Registry provides a mean surgical time of 5.5 hours in reviewed cases of POVL. Members of the ASA POVL Task Force also advocate frequent eye assessments along with proper documentation. Since avoiding pressure on the eyes is essential in preventing central retinal artery occlusion, ocular pressure must be assessed frequently by anesthesia providers in cases with patients in the prone position.[13] In a study by Lee and colleagues, anesthesia providers documented eye checks in only 51% of cases of POVL.[12]

Once established that the patient is experiencing POVL, an ophthalmologist should be contacted urgently for a consultation to determine the cause and to create an interventional plan.[15] However, there is no established treatment, so prevention is key. Pre-existing conditions include cardiovascular disease, long-standing or poorly controlled hypertension, known visual disorders such as glaucoma or end-organ ocular damage. In these patients, it would be practical to sustain systemic blood pressure as close to baseline values as possible and to avoid extended declines in ocular perfusion pressure (OPP).[14]

Fluid management is complicated in a procedure that is associated with substantial blood loss and prolonged anesthesia time; however, the ASA Practice Advisory recommended in 2006 that anesthesia providers should incorporate colloids along with crystalloid in fluid administration. Central venous pressure monitoring may also be useful in high-risk patients.

It is unclear how low the hemoglobin level must drop or the length of time the hemoglobin level must remain low to result in intraocular neuropathy.[16] A literature review by Gilbert, a neuro-ophthalmologist, concludes that any interruption to blood flow autoregulation can lead to POVL.[17] Even more confusing is though case reports suggest hypotension and anemia are factors that cause ION, it can occur in patients without these factors and in patients with blood pressures and hematocrit that anesthesiologists considered to be in acceptable ranges. We do know that after spine surgery risks factors for ION include the lengthy operative procedures in the prone position, hypotension, hemodilution or anemia, blood loss, and infusion of large amounts of intravenous fluids.[18]

IOP AND ION

Remember the two types of ION: AION, anterior ischemic optic neuropathy due to ischemia of the optic disc, and PION, posterior ischemic optic neuropathy due to ischemia of the retrobulbar optic nerve.[19] In AION the optic disc is acutely edematous, whereas in PION the optic disc is acutely healthy but atrophies weeks to months after the event.[20] Hayreh published an in-depth review of retinal blood flow and autoregulation also noting that optic nerve perfusion pressure (OPP) equals carotid arterial pressure or mean blood pressure minus IOP in all position[30] when IOP exceeds jugular venous pressure (JVP). As you might expect, studies show that peak IOP is the principal determinant of functional loss.[21]

Normal IOP ranges between 12 and 20 mmHg, averaging about 15 mmHg; it is regulated to remain constant in the healthy eye, usually +/- 2 mmHg of its baseline. Pressure can be determined by resistance to outflow of aqueous humor from the anterior chamber while reabsorption in the canal of Schlemm occurs, and then goes into the venous circulation. In healthy eyes, autoregulation occurs so that as pressure increases in the eye, the rate of flow into the canal of Schlemm increases, maintaining constant blood flow during changes in perfusion pressure.[22]

Increases in IOP may also be a source of postoperative ION. There are reports of visual loss related to ION in patients without hypertension or anemia. However, patients with chronic hypertension may be intolerant to hypotension since it contributes to a shift in the autoregulatory curve of optic nerve blood flow.

There are also reports of POVL documented in supine patients, but most reported cases follow general anesthesia for spine procedures in the prone position.[23] According to Sappington et al., during surgical procedures with anesthesia, especially those requiring extreme positioning, autoregulatory processes do not respond to increases in IOP by decreasing the production of aqueous humor.[24]

CONSIDERATIONS TO LIMIT EFFECTS OF IOP

A proposed intervention is to administer a topical anhydrase inhibitor and beta-adrenergic receptor blocking agent ophthalmic solution once IOP reaches 40 mmHg. A hand-held tonometer can make contact with the eye and be used to measure IOP in any surgical position. If IOP increases, second doses can be given.[25] These medications reduce IOP by decreasing aqueous humor production through inhibition of carbonic anhydrase II in the ciliary processes and direct action on beta2-adrenergic receptors in the ciliary processes.[26]

A study by Grover et al. of beta-adrenergic blocker eyedrop administration in patients under general anesthesia showed that this protects against increased IOP in patients following tracheal intubation.[27] According to Shemesh et al., large-scale clinical studies demonstrate the importance of early IOP reduction to prevent optic nerve damage and visual loss. Their studies mention that the second and even a third dose of beta-adrenergic eye drops administered per day is shown to lower IOP 25.9% over time.[28]

As shown in Figure 3, chemosis is detected as the white outer coating rises above the iris, appearing gelatinous. The presence of chemosis should alert the anesthesia provider that inflammation is occurring in the eye. Once chemosis is visible, IOP is approaching a critical level, allowing time for the provider to intervene. Because it is not possible in all operating rooms to measure IOP, detecting chemosis is an indicated clinical endpoint at which topical anhydrase inhibitor and beta-adrenergic receptor blocking agent ophthalmic solution would be valuable.[29]

POSITIONING

Pinkney et al. reviewed the relationship of patient positioning and IOP across all surgical specialties and concluded that rising IOP is time-dependent.[31] Porciatti and Nagaraju note benefit in reverse Trendelenburg positioning and illustrate decreased IOP and improved retinal ganglion cell function with this intervention.[32] Linder et al. also suggest that elevation of the head above the level of the heart reverses the effects of gravity-induced orthostatic venous pressure gradient resulting in decreased IOP.[33] However, the need for steep Trendelenburg positioning during lower abdominal robotic and laparoscopic procedures has dramatically increased over the past decade; more than 853 US hospitals were using the system as of 2010, and internationally the numbers doubled from 200 to 400.[34]

Figure 3. Chemosis.

There was a case report of bilateral POVL in a patient undergoing laparoscopic prostatectomy in steep Trendelenburg position, without intraoperative hypertension, hemodilution, extreme blood loss, or perceived metabolic disturbance. But he remained in a steep Trendelenburg position for 7.5 hours. This sentinel event prompted researchers in the institution where it occurred to develop a study investigating the relationship between IOP and steep Trendelenburg position. The study included 36 patients undergoing laparoscopic surgery in the steep Trendelenburg position over three years. IOP was measured using a tonometer at designated times during the surgery. Malloy found a direct correlation between the duration of time in steep Trendelenburg position during surgery and increased IOP.[35]

The surgical and anesthesia team can reduce IOP by unlocking the laparoscopic equipment and leveling the table for 5 minutes after the patient has been in the steep Trendelenburg position for four hours.[36] In a comparison of IOP between patients in a continuous steep Trendelenburg position compared with those who are placed in the supine (flat) position for 5 minutes after four hours of surgery, the patients who received the 5-minute supine rest had decreased IOP immediately following the rest period. They also had a faster return to baseline IOP after completion of the surgery than did patients who did not have a change in position intraoperatively.[37]

The 2003 Anesthesiology Update titled "Preventing blindness" stated that one in every ten neurosurgeons reported having had patients with POVL following lengthy back procedures.[38] It is important to initiate protocols during procedures requiring prolonged steep Trendelenburg or prone positions, in pursuit of providing an optimal level of ophthalmic safety for this patient population.[39]

Baig et al. performed a 2007 literature review citing bypass procedures as having a 4.5% incidence of postoperative vision loss and spine surgery a 2% incidence. Intraoperative factors were hypertension, blood loss, anemia, excessive fluid replacement, and duration of the surgical case.

Baig and colleagues reviewed POVL after spine surgery and suggest that the posterior optic nerve may be susceptible to decreased perfusion caused by increased venous pressure because of the nature of these small end vessels. They note that small pial branches supply the midorbital optic nerve, which is at risk in posterior ischemic optic neuropathy.[40] A 10-year study (between 1996 and 2005) analyzing more than 5.6 million patients in the Nationwide Inpatient Sample, determined that the highest rates of POVL involved cardiac surgery (8.4 events of 10,000 cases) and spinal fusion (3.09 events of 10,000 cases).[41]

AION is often associated with cardiac surgery; PION with spine surgery. There is individual variability in blood supply to optic nerves, so patterns of vision loss vary. Using horseshoe headrests in spine surgery can lead to unilateral vision loss involving periorbital edema, chemosis, ptosis, and corneal abrasion. In nose and sinus procedures, blindness can arise from direct surgical damage to the optic nerve. Using square or circular foam headrests with eye cutouts and a mirror to view the eyes helps prevent central retinal arterial occlusion from direct pressure on the eyes. There are reports

There is no established treatment, so prevention is key. Pre-existing conditions include cardiovascular disease, long-standing or poorly controlled hypertension, known visual disorders such as glaucoma or end-organ ocular damage.

of goggles dislodging and causing direct ocular pressure, resulting in unilateral central retinal arterial occlusion.

Roth suggests ensuring eyes are properly positioned behind eye cutouts on headrests and checking every 20 minutes, by palpation or visualization, to ensure there is no direct external compression to the eyes.[42]

Treatment options for the initial management of vision loss after spine surgery associated with PION consist of correcting volume depletion, correcting blood loss, restoring the blood pressure to normal, and possibly administering corticosteroids intravenously. The worst prognosis for perioperative blindness is with central retinal artery occlusion and PION. The beginning point for prevention is an increased awareness of the possibility of complications in addition to the correction of hypoperfusion to the eyes, particularly in patients with vaso-occlusive conditions, such as chronic essential hypertension and diabetes mellitus.[43]

SUMMARY

In our case study, the plaintiff experienced POVL. What should the LNC look for in chart analysis?

Did the healthcare providers:

- Consider risk factors for POVL, potential degree of permanency, and the immediate treatment options?
- Discuss with patients preoperatively about the risk of perioperative vision loss occurring during informed consent?
- Assess and document that the eyes are free of pressure throughout prone and Trendelenburg procedures?
- Incorporate colloids with fluid administration?
- Use 5 to 10° reverse Trendelenburg position during spine procedures performed with the patient in the prone position?
- During steep Trendelenburg procedures, use a 5-minute supine rest stop at the four-hour timeframe. Rest stops require undocking of laparoscopic equipment?
- Use soft foam headrest with cutouts for eyes to prevent direct external compression and a mirror for viewing the eyes?
- Consult an experienced ophthalmologist at first sign that the patient has altered vision after surgical procedure?

REFERENCES:

1. Nandyala SV, Marquez-Lara A, Fineberg SJ, Singh R, Singh K. Incidents and risk factors for perioperative visual loss after spinal fusion. Spine J. 2014;14(9):1866 - 872. https://reference.medscape.com/medline/abstract/24216394
2. Slocum HC, O'Neal KC, Allen CR. Neurovascular complications from malposition on the operating table. Surg Gynecol Obstet. 1948;86(6):729 - 734. https://www.ncbi.nlm.nih.gov/pubmed/18915944
3. Lechner AD. If my spine surgery went fine, why can't I see?: Postoperative visual loss and informed consent. APSF Newslett. 2008;23(1):1 – 3. https://www.apsf.org/article/if-my-spine-surgery-went-fine-why-cant-i-see-postoperative-visual-loss-and-informed-consent/
4. Evans LS. Increased intraocular pressure in severely burned patients. Am J Ophthalmolog. 1991;111(1):56 – 58. https://doi.org/10.1016/S0002-9394(14)76897-7
5. Cheng MA, Todorov A, Tempelhoff R, McHugh T, Crowder CM, Lauryssen C. The effect of prone positioning on intraocular pressure in anesthetized patients. Anesthesiology. 2001;95(6):1351 - 1355. https://anesthesiology.pubs.asahq.org/article.aspx?articleid=1944231
6. Postoperative Visual Loss Study Group. Risk factors associated with ischemic optic neuropathy after spinal fusion surgery. Anesthesiology. 2012;116(1):15 - 24. https://anesthesiology.pubs.asahq.org/article.aspx?articleid=1933604
7. Hayreh SS. Management for ischemic optic neuropathies. Indian J Ophthalmol. 2011;59(2):12 - 36. http://www.ijo.in/article.asp?issn=0301-4738;year=2011;volume=59;issue=2;spage=123;epage=136;aulast=Hayreh
8. Sadda SR, Nee M, Miller NR, Biousse V, Newman NJ, Kouzis A. Clinical spectrum of posterior ischemic optic neuropathy. Am J Ophthalmolog. 2001;132(5):743 - 750. https://jhu.pure.elsevier.com/en/publications/clinical-spectrum-of-posterior-ischemic-optic-neuropathy-4
9. Gill B, Heavner JF. Postoperative visual loss associated with spine surgery. Eur Spine

J. 2006;15(4):479 - 484. https://link.springer.com/article/10.1007/s00586-005-0914-6

10. Buono LM, Foroozan R. Perioperative posterior ischemic optic neuropathy: review of the literature. Surv Ophthalmol. 2005; 50(1):15 - 26. https://www.deepdyve.com/lp/elsevier/perioperative-posterior-ischemic-optic-neuropathy-review-of-the-Vzc0MrAibK

11. Abraham M, Sakhuja N, Sinha S, Rastogi S. Unilateral visual loss after cervical spine surgery. J Neurosurg Anesthesiol. 2003;15(4):319 - 322. https://journals.lww.com/jnsa/Abstract/2003/10000/Unilateral_Visual_Loss_After_Cervical_Spine.5.aspx

12. Lee LA, Roth S, Posner KL, et al. The American Society of Anesthesiologists Postoperative Visual Loss Registry: analysis of 93 spine cases with postoperative visual loss. Anesthesiology. 2006;105(4):652-665. https://www.ncbi.nlm.nih.gov/pubmed/17006060

13. American Society of anesthesiologist Task Force on Perioperative Blindness. Practice advisory for perioperative visual loss associated with spine surgery: an updated report by the American Society of Anesthesiologist Task Force on Perioperative Visual Loss. Anesthesiology. 2012;116(2):274 - 285. https://www.ncbi.nlm.nih.gov/pubmed/22227790

14. Roth S, Perioperative visual loss. In: Miller RD, ed. Miller's Anesthesia. 6th ed. Philadelphia, PA: Elsevier Churchill Livingstone; 2005:2991 - 3020.

15. American Society of Anesthesiologist Task Force on Perioperative Blindness. Practice advisory for perioperative visual loss associated with spine surgery: a report by the American Society of Anesthesiologist Task Force on Perioperative Blindness. Anesthesiology. 2006;104(6):1319 - 1328. https://anesthesiology.pubs.asahq.org/article.aspx?articleid=1923154

16. Hollenhorst RW, Svien HJ, Benoit CF. Unilateral blindness occurring during anesthesia for neurological operations. Arch Ophthalmol. 1954;52(6):819 - 830. https://www.ncbi.nlm.nih.gov/pubmed/13217529

17. Gilbert ME. Postoperative visual loss: a review of the current literature. J Neur Ophthalmol. 2008;32(4):194 - 199. https://www.tandfonline.com/doi/abs/10.1080/01658100802114646

18. Ho VT, Newman NJ, Song S, Ksiazek S, Roth S. Ischemic optic neuropathy following spine surgery. J Neurosurg Anesthesiol. 2005;17(1):38 - 44. https://www.deepdyve.com/lp/elsevier/ischemic-optic-neuropathy-following-spine-surgery-055LkuMYbQ

19. Fandino W. Strategies to prevent ischemic optic neuropathy following major spine surgery: a narrative review. J Clin Anesth. 2016;125(3):445 – 464. https://doi.org/10.1016/j.jclinane.2017.09.009

20. Atkins JH. Neuroendocrine physiology: fundamentals and common syndromes.In: Brambrink A, Kirsch J, eds. Essentials of Neurosurgical Anesthesia & Critical Care. New York, NY: Springer; 2012:21 - 37.

21. Bui BV, Edmunds B, Cioffi GA, Fortune B. The gradient of retinal functional changes during acute intraocular pressure elevation. Invest Ophthalmol Vis Sci. 2005;46(1):202-213. https://iovs.arvojournals.org/article.aspx?articleid=2163431

22. Guyton AC, Hall JE. Textbook of Medical Physiology. 12th ed. Philadelphia, PA: Elsevier Saunders; 2011.

23. Hunt K, Bajekal R, Calder I, Meacher R, Eliahoo J, Acheson JF. Changes in intraocular pressure in anesthetized prone patients. J Neurosurg Anesthesiol. 2004;16(4):287 - 290. https://www.ncbi.nlm.nih.gov/pubmed/15557832

24. Sappington RM, Sidarova T, Long DJ, Calkins DJ. TRPV1: contribution to retinal ganglion cell apoptosis and increased intracellular Ca2+ with exposure to hydrostatic pressure. Invest Ophthalmol Vis Sci. 2009;50(2):717 - 728. http://www.psy.vanderbilt.edu/faculty/sappingtonlab/21trpv1contribution

25. Molloy B, Cong X. Perioperative dorzolamide - timolol intervention for rising intraocular pressure during steep Trendelenburg position surgery. AANA J. 2014;82(3):203 – 211. https://www.ncbi.nlm.nih.gov/pubmed/25109158

26. Yeh J, Kravitz D, Francis B. Rational use of the fixed combination of dorzolamide - timolol in the management of raised intraocular pressure and glaucoma. Clin Ophthalmol. 2008;2(2):389 - 399. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2693974/

27. Grover VK, Sharna S, Kakkar RK, Grewal S, Gupta A. Topical timolol prevents intraocular pressure response to succinylcholine and tracheal intubation. Bahrain Med Bull. 1995;17(4):1 - 4. http://www.bahrainmedicalbulletin.com/december_1995/topical_timolol.pdf

28. Shemesh G, Moissoiev E, Lazar M, Kurtz S. Intraocular pressure reduction of fixed combination timolol maleate 0.5% and dorzolamide 2% (Cosopt) administered three times a day. Clin Ophthalmol. 2012;6:283 - 287. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3292411/

29. Molloy B. A preventative intervention for rising intraocular pressure: development of the Molloy/Bridgeport Anesthesia Associates Observation Scale. AANA J. 2012;80(3):213 - 222. https://www.aana.com/docs/default-source/aana-journal-web-documents-1/preventive-intervention-0612-p213-222.pdf?sfvrsn=f17048b1_6

30. Hayreh SS. Ischemic optic neuropathy. Prog Retin Eye Res. 2009;28(1):34 - 62. https://doi.org/10.1016/j.preteyeres.2008.11.002

31. Pinkney TD, King AJ, Walter C, Wilson TR, Maxwell-Armstrong C, Acheson AG. Raised intraocular pressure (IOP) and perioperative visual loss in laparoscopic colorectal surgery: a catastrophe waiting to happen? A systematic review of evidence from other surgical specialties. Tech Colorectal. 2012;16(5):331 - 335. https://link.springer.com/article/10.1007/s10151-012-0879-5

32. Porciatti V. Nagaraju M. Head – tilt lowers IOP and improves RGC dysfunction and glaucomatous DBA/2J mice. Exp Eye Res. 2010;90(3):452 - 460. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2824077/

33. Linder Bj, Trick GL, Wolf ML. Altering body position affects intraocular pressure and visual function. Invest Ophthalmolog Vis Sci. 1988;29(10):1492 - 1497. https://iovs.arvojournals.org/article.aspx?articleid=2177869

34. Barbash GI, Glied SA, New technology and health care costs: the case of robot - assisted surgery. N Engl J Med. 2010;363(8):701 - 704. https://www.nejm.org/doi/full/10.1056/NEJMp1006602

35. Molloy BL. Implications for postoperative visual loss: steep Trendelenburg position and effects on intraocular pressure. AANA J. 2011;79(2):115 - 121. https://www.aana.com/docs/default-source/aana-journal-web-documents-1/

implications_0411_p115-121.pdf?sfvrsn=d27c5ab1_6

36. Freshcoln M, Diehl MR. Repositioning during robotic procedures to prevent postoperative visual loss. OR Nurse. 2014;8(4):36 - 41. https://www.nursingcenter.com/journalarticle?Article_ID=2504350

37. Molloy BL. A comparative assessment of intraocular pressure and prolonged steep Trendelenburg position vs. supine position intervention. Poster presented at the Postgraduate Assembly in Anesthesiology; December 11, 2010; New York, NY. https://www.researchgate.net/publication/235609105_A_Comparative_Assessment_of_Intraocular_Pressure_in_Prolonged_Steep_Trendelenburg_Position_Versus_Level_Supine_Position_Intervention

38. Benumof JL. Preventing blindness after prone cases. Anesthesiology Update. San Diego, CA: University of California School of Medicine. 2003;45(9):1.

39. Awad H, Santilli S, Ohr M, et al. The effects of steep Trendelenburg positioning on intraocular pressure during robotic radical prostatectomy. Anesth Analg. 2009;109(2):473 - 478. https://journals.lww.com/anesthesia-analgesia/Fulltext/2009/08000/The_Effects_of_Steep_Trendelenburg_Positioning_on.30.aspx

40. Baig MN, Lubow M, Immesoete P, Bergese SD, Hamdy EA, Mandel E. Vision loss after spine surgery: review of the literature and recommendations. Neurosurger Focus. 2007;23(5):15 - 21. https://doi.org/10.3171/FOC-07/11/15

41. Shen Y, Drumm 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. Anesthesiology Analg. 2009;109(5):1535 - 1545. https://journals.lww.com/anesthesia-analgesia/fulltext/2009/11000/The_Prevalence_of_Perioperative_Visual_Loss_in_the.32.aspx

42. Roth S. Perioperative visual loss: what do we know, what can we do? Br J Anaesth. 2009;103(suppl I):i30-i40. https://academic.oup.com/bja/article/103/suppl_1/i31/230262/

43. Stambough JL, Dolan D, Werner R, Godfrey E. Ophthalmologic complications associated with prone positioning and spine surgery. J Am Acad Orthop Surg. 2007;15(3):156 – 165. https://journals.lww.com/jaaos/Abstract/2007/03000/Ophthalmologic_Complications_Associated_With_Prone.5.aspx

Sandra Krug, CRNA, APRN, RRT has worked in the health care industry for over 25 years as a Respiratory Therapist and Nurse. She is a clinically active Certified Registered Nurse Anesthetist in the state of Florida. Sandra also has an independent business as a Legal Nurse Consultant. info@LNCKRUG.com