ANESTHESIOLOGY

Perioperative Care of the Patient with Eye Pathologies Undergoing Nonocular Surgery

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ABSTRACT

The authors reviewed perioperative ocular complications and implications of ocular diseases during nonocular surgeries. Exposure keratopathy, the most common perioperative eye injury, is preventable. Ischemic optic neuropathy, the leading cause of perioperative blindness, has well-defined risk factors. The incidence of ischemic optic neuropathy after spine fusion, but not cardiac surgery, has been decreasing. Central retinal artery occlusion during spine fusion surgery can be prevented by protecting eyes from compression. Perioperative acute angle closure glaucoma is a vision-threatening emergency that can be successfully treated by rapid reduction of elevated intraocular pressure. Differential diagnoses of visual dysfunction in the perioperative period and treatments are detailed. Although glaucoma is increasingly prevalent and often questions arise concerning perioperative anesthetic management, evidence-based recommendations to guide safe anesthesia care in patients with glaucoma are currently lacking. Patients with low vision present challenges to the anesthesia provider that are becoming more common as the population ages.

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Perioperative injury to the eye ranges from corneal injuries producing pain and reversible blurred vision to serious disorders of the retina or optic nerve causing permanent blindness. The eye is the most important sensory organ, with up to 50% of cerebral cortical neurons serving visual function.[1] Low vision or blindness are major disabilities accompanied by significant emotional suffering and high cost to the healthcare system.[2] Accordingly, prevention of vision loss is of paramount concern, and it is critically important that anesthesia providers be competent in prevention, diagnosis, and treatment of vision-impacting complications of nonocular surgery and the important considerations in delivering anesthesia to patients with chronic ophthalmic disease.

Corneal injury from exposure keratopathy is the most common perioperative eye injury, presenting with pain, foreign body sensation in the eye, blurry vision, and photophobia. Its incidence has been dramatically reduced by educating providers in best preventive measures, from 1.20 per 1,000 to 0.09 per 1,000 in one quality improvement study.[3] A full-thickness corneal transplant is associated with a risk for 1 yr of incision dehiscence, leading to catastrophic loss of eye contents.[4] The anesthesia provider needs to protect the eye of such individuals undergoing anesthesia and surgery from even minor trauma or compression.

The leading cause of perioperative blindness is ischemic optic neuropathy. There have been significant advances in determining the perioperative risk factors, publication of multispecialty-driven evidence-based advisories, and encouragingly, a dramatic decrease in its incidence in spinal fusion surgery,[5,6] where the estimated national U.S. incidence was 1.63 per 10,000 spine fusion surgeries in 1998 to 2000 and 0.6 per 10,000 in 2010 to 2012.[5,7] While some patients have regained vision spontaneously or *via* various treatment modalities, a major remaining challenge is the lack of any proven treatment. Central retinal artery occlusion and cerebral blindness can produce overlapping signs and symptoms; hence, accurate diagnosis of these entities is essential to drive appropriate therapies.

Idiopathic intracranial hypertension is an elevation in cerebrospinal fluid pressure caused by decreased cerebrospinal fluid absorption or elevated cerebral venous sinus pressures.[8,9] Its main permanent morbidity is to the visual system, characterized by papilledema associated with vision loss, and sixth nerve palsy causing diplopia.[10] These patients often present for labor analgesia, and commonly questioned is the safety of placing epidural catheters.[11] There is, however, currently no evidence to justify the withholding of epidural or spinal analgesia.

Less certain are the anesthetic implications of patients with primary open angle glaucoma.[12] To date there are only limited studies on the impact of elevated intraocular pressure from patient positioning for surgery on the functioning of the optic nerve and outcomes. On the other hand, acute angle closure glaucoma in the postoperative period is a medical emergency usually triggered by specific drugs in susceptible individuals that requires immediate reduction of intraocular pressure to prevent permanent damage to the optic nerve.[13]

With the increasingly aging population and rising prevalence of chronic degenerative diseases including cataract, glaucoma, diabetic retinopathy, and age-related macular degeneration, the anesthesia provider is likely to encounter patients with existing vision impairment. Low vision or blindness currently affects about 2.5% of the population of the United States.[2] Circadian rhythm disorders, altered alertness, and mood changes may be present.[14] The lack of visual cues may complicate communication, including difficulties in discussing the anesthetic plan and the obtaining of informed consent.[15]

This review provides a best evidence-based approach to the anesthetic implications of diseases of the eye, and the prevention of perioperative eye complications. The review is organized anatomically, beginning with diseases of the outer covering of the eye, the cornea, followed by the posterior eye, the retina, and the optic nerve.

Cornea
Exposure Keratopathy
Epidemiology

Exposure keratopathy is a defect in the corneal surface epithelium and the most common cause of postoperative ocular complaints. The incidence is 0.9 to 3.3 per 1,000 general anesthetics.[16,17] The majority completely heal; however, in the American Society of Anesthesiologists (Schaumburg, Illinois) Closed Claims Study of cases from 1974 to 1987, 16% resulted in permanent ocular damage, although the authors did not report the nature of the residual injury.[18] Since the corneal epithelium is heavily innervated by the trigeminal nerve, eye pain, foreign body sensation, photophobia, and erythema are common presenting complaints. Blurry vision can also occur.[19] Insufficient closure of the eyelid and/or insufficient tear production, and inadvertent contact with the eye by the patient or provider (*e.g.*, a finger upon awakening, or hanging name tag, respectively), are the most common causes.[16,17] Pain control and follow-up with an eye provider for monitoring of proper healing and prevention of infection are the focus of treatment.[20]

Perioperative Evaluation of Patients at Risk of Exposure Keratopathy

Several independent risk factors are associated with perioperative exposure keratopathy.[16,21–23] These include patient-specific characteristics, and factors related to surgery and anesthesia. Impaired tear production during general anesthesia decreases the lubrication and proper nourishment of the corneal surface.[24] Zernii *et al.* demonstrated in a rabbit model that general anesthesia decreased tear film stability. The tear break-up time test demonstrated short-term tear film destabilization. Total antioxidant activity of tears declined for up to 1 h after 3 h of general anesthesia.[25] The resulting increase in reactive oxygen species was responsible for the damage to the corneal epithelium.[26] Thyroid eye disease is associated with proptosis, and lagophthalmos, which can prevent full closure of the eye, increasing the likelihood of exposure keratopathy.[27,28] Prone, lateral, or Trendelenburg positioning of the patient for surgery may result in the cornea contacting items or surfaces.[16] Extreme head down positioning, *e.g.*, for robotic surgery, is another risk factor, possibly because of periorbital swelling and partial opening of the eyelids during anesthesia.[29]

Prevention

Prevention is centered on proper taping of the eyes, and verifying that eyelids are completely closed soon after induction of general anesthesia, preferably even before airway management.[30] Taping alone provides protection equivalent or superior to ointment, goggles, hydrophilic contact lenses, Geliperm (Geistlich Pharma North America, USA) dressings, and bio-occlusive dressings.[31] Disadvantages of these adjuvants should be considered, *e.g.*, flammability of petroleum gel, sloughing of the corneal epithelium and conjunctival hyperemia from preservative-based ointments, foreign body sensation and rubbing of the eyes upon awakening from ointments and gels, and risk of compression of the eye from improperly placed goggles.[32–35]

Some patients present with elaborate and expensive glued false eyelashes or eyelash extensions, and they may be reluctant or even unable to easily remove them. False eyelashes are best removed if possible before anesthesia and surgery.[36] We find that the best strategy is sensitivity to the patient's concerns by counseling them that the eyelashes will be carefully protected, although we do warn them of the heightened risk of corneal injury from contact of the false lashes with the corneal surface. We suggest gentle closure of the eyes with cotton eye pads, then covering the pads with a layer of bio-occlusive dressing. This effectively covers the eyes and prevents the tape or bio-occlusive dressing from pulling out the eyelashes. The anesthesia provider should frequently examine the face during surgery to verify that there is no pressure on the eyes and that the eyes are protected from exposure.

Successful Quality Improvement Decreased Anesthesia-related Exposure Keratopathy

Large-scale quality improvement programs have significantly reduced the incidence of perioperative exposure keratopathy. Vetter *et al.* utilized a "Plan-Do-Check-Act" cycle and a standardized eye protection protocol consisting of eye

lubrication with an aqueous–based gel and clear occlusive dressings to cover the eyelids and surrounding skin.[3] The intraoperative injury rate decreased significantly from 1.20 per 1,000 to 0.09 per 1,000 after implementation, and was sustained for the entire 45–month follow–up period. This study again demonstrated that most perioperative incidents of exposure keratopathy are preventable and is an excellent example of the success of provider education in reducing an anesthesia morbidity affecting the eye.

Corneal Transplant (Penetrating Keratoplasty)

The anesthesia provider is likely to encounter a patient with a recent penetrating keratoplasty.[37] Approximately 48,000 penetrating keratoplasties, also known as full-thickness corneal transplants, were performed in the United States in 2021. Traumatic globe rupture or wound dehiscence after even minimal trauma is an important concern in a patient for nonocular surgery after penetrating keratoplasty. This devastating complication (fig. 1) can result in choroidal hemorrhage (bleeding from the choroidal circulation that supplies blood to the outer retina), and loss of vitreous and retina through the wound opening followed by profound vision loss.[38] The highest period of traumatic rupture is in the month after corneal surgery because wound strength is almost entirely dependent upon sutures. It takes 6 to 12 months after suture removal for the corneal wound strength to typically reach its maximum.[38,39] To minimize the risk of rupture within this time period, a conservative and do–no–harm recommendation is to apply an eye shield over the taped eyelids, providing two layers of protection from direct pressure or unintended trauma.[40] Anesthesia providers should be particularly cautious to avoid pressure at any time on an eye with a penetrating keratoplasty when positioning the patient in the prone or lateral positions for surgery.[4,41,42]

Open Globe Injury

It may be necessary to anesthetize a patient with an open globe injury who must first undergo a more urgent surgical procedure. The major risk is loss of eye contents through the wound from increased intraocular pressure. Use of succinylcholine in open globe injury is a long-standing controversy. The history of this controversy and reasons for the common recommendations not to use succinylcholine have been well considered elsewhere.[43] Many of
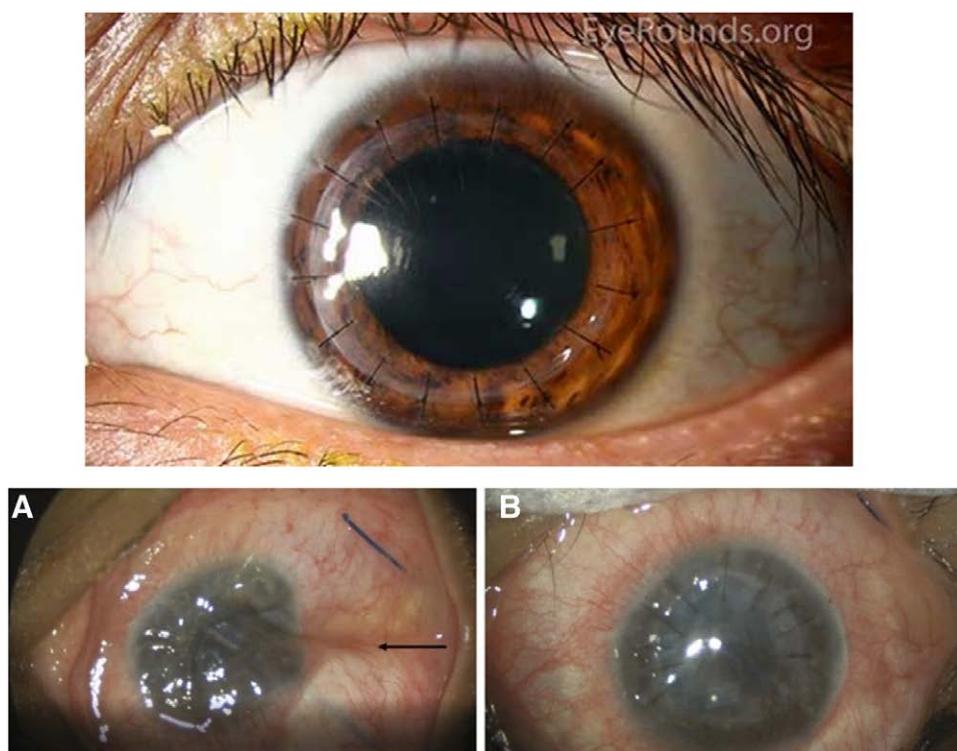
Fig. 1. Penetrating keratoplasty. (*Top*) Grafts for penetrating keratoplasty with 16 interrupted sutures. Reproduced with permission from the Department of Ophthalmology at the University of Iowa, and available at https://www.EyeRounds.org. Accessed March 24, 2022. (*Bottom*) Intraoperative photographs from a different case of dehisced penetrating keratoplasty. (*A*) Note the deflated globe with indentation of the sclera (*black arrow*) and corneal edema. (*B*) After repair, the return of the globe contour and wound closure with 10 interrupted sutures can be seen. Reproduced with permission from the publisher. Davies E, Yonekawa Y. Case 6: Dehiscence of penetrating keratoplasty from blunt trauma in Management of Open Globe Injuries. Edited by Grob S, Kloek C. Switzerland, Springer, 2018. https://doi.org/10.1007/978-3-319-72410-2_11.

the recommendations not to use succinylcholine are based upon anecdotal reports. In a feline open globe injury model, succinylcholine did not result in loss of intraocular components, significant because the cat eye closely resembles that of humans.[44] A sensible strategy in addition to protecting the eye from further trauma is to use alternative nondepolarizing neuromuscular blocking agents, in conjunction with induction and other agents that lower intraocular pressure, but where indicated appropriately, succinylcholine may be used safely.[43]

Contact Lenses

Contact lenses are in widespread use, and a common question is whether they should be removed before surgery and anesthesia. Contact lenses have been associated with conjunctival epithelial defects, defects of the lid wiper epithelium, and diminished tear film breakup time.[45] Contact lens wearers have an increased risk of corneal injuries compared to those who do not wear contact lenses.[46] Although there are no studies examining intraoperative contact lens use as an independent risk factor for corneal injury, we recommend a conservative and do–no–harm approach of removing them before induction of anesthesia.

Diseases of the Retina

Patients Who Have Undergone Vitrectomy

Vitrectomy is a common eye surgical procedure performed for proliferative diabetic eye disease, retinal detachment, and retinal tumors.[47] Tamponade agents provide surface tension in the vitreous across retinal breaks and prevent fluid flow into the subretinal space until the surgical repair becomes complete. Gases may be used,[48] including sulfur hexafluoride, which dissipates in 10 to 14 days, and perfluoropropane, which lasts 55 to 65 days.[49] Most centers provide a brightly colored wrist band indicating the type of gas used.[50] Anesthesia providers must be aware of the presence and type of vitreous gas bubble to prevent inadvertent vision-threatening increases in intraocular pressure, which can lead directly to central retinal artery occlusion in patients with recent retinal detachment surgeries or vitrectomies.

The major risk is from the administration of nitrous oxide, which diffuses into the gas bubble faster than it can exit,[51] dramatically increasing intraocular pressures. Case reports documented devastating vision loss from the sudden onset of profoundly increased intraocular pressure and resulting central retinal artery occlusion in patients undergoing surgery after recent vitrectomy with use of nitrous oxide for anesthesia.[52–57] Accordingly, nitrous oxide should be avoided for at least 2 weeks in a patient with sulfur hexafluoride, and 2 months in a patient with the longer-lasting perfluoropropane.[58]

Retinal Artery Occlusion

Retinal artery occlusion can be central or branch retinal artery occlusion. Perioperatively, it most often occurs after cardiac surgery, or in spinal fusion surgery with the patient positioned prone.[59] It also may occur with a vascular injury in sinus or nasal surgery, or from emboli during interventional radiological procedures involving the head and face.[60]

Signs and symptoms (table 1) are a sudden onset of painless visual loss, abnormal pupils, retinal whitening, and narrowed retinal arterioles.[61] Pallor of the ischemic retina renders the underlying choroidal circulation visible, causing the classic cherry-red spot seen on ocular fundus examination. Perioperative central retinal artery occlusion in the setting of spine surgery is usually due to external compression of the eye, and in cardiac surgery, it is usually due to emboli.[62]

Retinal Artery Occlusion in Spine Surgery

Square or circular foam headrests or devices with mirrors to view the eyes, or securing the head into a frame using pins, should prevent ocular compression in the patient positioned prone for spinal fusion surgery.[63] Anesthesia providers should check the head and the eyes intermittently to ensure there is no compression of the eye, which could lead to increased intraocular pressure and retinal damage. The eyes should also be checked after any movement of the body in which where the head position may have become altered. As human studies are obviously unethical or impractical, no "safe" or accepted period of time for these eye inspections has been established. Rodents have similar ocular circulation and retinal structure to humans. Their retinal ganglion cells were damaged within a minimum of 20 to 30 min of elevated intraocular pressure. In monkeys, occluding the central retinal artery occlusion at its site of entry into the dural sheath of the optic nerve could be tolerated for a longer period, requiring at least 105 min before damage was detectable.[64] However, the difference is that in monkeys, the central retinal artery was specifically occluded without increasing intraocular pressure. In the rodents, the increased intraocular pressure produced by elevating pressure in the anterior chamber of the eye resulted in an occlusion of both the central retinal artery to the inner retina and the choroidal circulation to the outer retina.[65] The latter is more comparable experimentally to external compression of the human eye. Therefore, besides proper precautions to prevent external compression, a conservative and do–no–harm recommendation is to check the eyes for compression every 20 min in the patient positioned prone for surgery.

Retinal Artery Occlusion in Cardiac Surgery

In the U.S. Nationwide Inpatient Sample, there were 5.8 million cardiac operative discharges from 1998 to 2013, and

Table 1. Typical Eye Examination Findings and Differential Diagnosis in Perioperative Cornea, Retinal, Optic Nerve, or Visual Cortex Injury

	Exposure Keratopathy	Acute Angle Closure Glaucoma	Anterior Ischemic Optic Neuropathy	Posterior Ischemic Optic Neuropathy	Cerebral Blindness	Central Retinal Artery Occlusion
Visual acuity	May be blurry	Cloudy or blurry; excessive tearing; redness; seeing halos	Altitudinal defect; scotoma; less commonly, may have no light perception	Variable, ranging from visual field loss to complete blindness in affected eye(s)	Hemianopia	Varies from visual field loss to no light perception
Pain	May be severe; may have photophobia	May be severe	None	None	None	May be present in cases of external compression of the eye
Other non-eye symptoms	—	Headache, nausea, vomiting	—	—	Signs and symptoms of stroke	
Intraocular pressure	Normal	Very high (> 60 mmHg)	Normal	Normal	Normal	Normal
Optic disk	Normal	Optic nerve head edema	Optic nerve head edema, later optic atrophy	Normal, later optic atrophy	Normal	Normal, later optic atrophy
Retina	Normal	Normal	May have attenuated retinal arterioles	May have attenuated retinal arterioles	Normal	Normal in the hyperacute and chronic stages; in acute stage, cherry-red spot (macula), pallor, narrowed retinal arteries
Pupillary light reflex	Normal	Dilated pupil	Absent or afferent pupil defect	Absent or afferent pupil defect	Normal	Absent or afferent pupil defect
Ocular muscle function	Normal	Normal	Normal	Normal	Normal	May be impaired if the eye has been externally compressed leading to central retinal artery occlusion
Gonioscopy	—	Narrow angle, corneal edema	—	—	—	—

4,564 cases of retinal artery occlusion, or 7.8 per 10,000. Systemic risk factors were a history of giant cell arteritis, transient ischemic attack, carotid artery stenosis, embolic stroke, hypercoagulability, myxoma, diabetes mellitus with ophthalmic complications, and aortic insufficiency. Operative risk factors were bleeding, and aortic, mitral valve, and septal surgery.[62] Conditions that increased the risk of embolization to the retinal circulation, namely, carotid disease, opening the heart, and a pre–existing abnormal retina, were predictors of the complication in cardiac surgery. Although an exceptionally large study, it is limited by its retrospective nature, reliance upon and inability to confirm diagnostic codes, and not distinguishing between branch and central retinal artery occlusion.

Prevention of retinal artery occlusion in cardiac surgery is a poorly studied area in need of more research. There are some recommendations for prevention of stroke that appear relevant. Calcific emboli in the central retinal artery and its branches with accompanying visual field deficits from opening of the left ventricle, cardiopulmonary bypass, and manipulating a calcified aorta have been described after coronary artery bypass grafting.[66] Little can be done about opening the left ventricle and cardiopulmonary bypass as they are necessary accompaniments to the surgery, but epiaortic ultrasound can detect atheromatous regions and guide the location of aortic cannulation to decrease emboli.[67–69] Its efficacy to decrease retinal microemboli in particular has not yet been documented, but it is currently recommended for coronary artery bypass grafting as class IIA evidence to decrease stroke by the American College of Cardiology and the American Heart Association.[70]

Treatment

Central retinal artery occlusion is a medical emergency with only a limited time window before retinal neurons die and severe vision loss occurs.[71] Early recognition (table 1) is essential in order to enable treatment within the time window and the opportunity to achieve at least some recovery of vision. Thrombolysis with tissue plasminogen activator is the most commonly offered treatment when a stroke center is accessible within 4 to 5 h of suspected central retinal artery occlusion. The efficacy of thrombolysis has not yet been demonstrated in randomized clinical trials, but nonrandomized studies and meta–analyses strongly suggest it improves visual outcome.[72–74] Some groups have also reported success with thrombolysis combined with other therapies.[75–77]

An important concern in the postoperative patient is the risk of bleeding from thrombolytic agents.[71] Due to these risks, where there is a delayed diagnosis, or where rapid access to a stroke center is not available, how to proceed must be decided on a case-by-case basis in consultation with a neuro-ophthalmologist. Without thrombolysis, inhaling carbogen (95% oxygen, 5% CO_2), anterior chamber paracentesis, hyperbaric oxygen delivery, and acetazolamide have been used with some reported successes.[77–82]

Optic Nerve

Idiopathic Intracranial Hypertension and Vision

Definition and Pathophysiology

Idiopathic intracranial hypertension is elevated intracranial pressure caused by decreased cerebrospinal fluid absorption or elevated cerebral venous sinus pressures.[8,9] Its major permanent morbidity is vision loss due to optic neuropathy.[8,10] Visual signs and symptoms are optic nerve swelling (papilledema), diplopia ("double vision"), and vision loss.[10] The modified Dandy criteria that are used for diagnosis include (1) signs and symptoms of increased intracranial pressure, (2) absence of localizing or focal neurologic signs other than cranial nerve VI paresis, (3) elevated cerebrospinal fluid pressures, and (4) no etiology for increased intracranial pressure on neuroimaging or analysis of cerebrospinal fluid.[8,10] The incidence is 0.9 per 100,000 per year, higher in obese women of childbearing age.[10] Anesthesiologists are most likely to encounter idiopathic intracranial hypertension in pregnant women requesting labor analgesia.

Preoperative Evaluation

Medications to lower intracranial pressure should not be stopped. A neuro-ophthalmologist should be consulted if there any concerns about intracranial pressure control or evidence of worsening visual function. Acetazolamide, commonly used to treat idiopathic intracranial hypertension, inhibits carbonic anhydrase, increasing renal bicarbonate excretion, which may result in metabolic acidosis. Caution is recommended in the anesthetized patient who also has chronic obstructive pulmonary disease, as they may be unable to increase ventilation in response to the metabolic acidosis, leading to prolonged postoperative mechanical ventilation.[83]

Anesthetic Management

The most common perioperative concern is the safety of neuraxial anesthesia or analgesia. Spinal or epidural anesthesia has been used effectively and uneventfully for labor analgesia and cesarean delivery, including in those with cerebrospinal fluid diversion devices.[84] Since there is no obstruction to cerebrospinal fluid flow in idiopathic intracranial hypertension, a rapid decline in cerebrospinal fluid pressure from a lumbar puncture is quickly balanced by caudal cerebrospinal fluid flow and does not result in uncal herniation or visual changes. However, there are some controversies and concerns about the use of epidural catheters. Some have postulated that large volumes of epidural local anesthetics may further increase intracranial pressures. For example, Hilt *et al.* reported an exaggerated increase in intracranial pressure in a patient with idiopathic intracranial hypertension after a lumbar epidural injection of bupivacaine.[85] A review examined four case reports of epidural analgesia for labor in patients with idiopathic intracranial hypertension. One was complicated by an inadvertent dural puncture headache. There are four additional case reports concerning five patients, of whom three received a combined epidural–spinal or a spinal catheter, with no complications reported.[86–89] Although the literature is sparse, there does not seem to be enough justification to withhold or alter the normal procedures for labor analgesia in patients with idiopathic intracranial hypertension.

Ischemic Optic Neuropathy

Epidemiology

Ischemic optic neuropathy is the leading cause of sudden vision loss in those older than 50 yr, typically occurring spontaneously and without previous warning signs. Anterior ischemic optic neuropathy affects the optic nerve head and always causes optic nerve swelling in the acute setting. Posterior ischemic optic neuropathy impacts the portion of the optic nerve behind the eye that is not visible by examination of the ocular fundus. Optic disc swelling upon symptom onset clinically differentiates ischemic optic neuropathy into, respectively, anterior ischemic optic neuropathy and posterior ischemic optic neuropathy (table 1).[90] Ischemic optic neuropathy is also classified as nonarteritic or arteritic, the latter associated with vasculitis. Nonarteritic anterior ischemic optic neuropathy has an estimated annual incidence of 2.3 to 10.2 per 100,000 in the United States.[91] Arteritic anterior ischemic optic neuropathy, caused by mechanical vascular occlusion of the short posterior ciliary arteries from giant cell or temporal arteritis, generally occurs in those older than 60 yr, has a female predilection, and responds to steroids.[92] Nonarteritic posterior ischemic optic neuropathy is rare outside of the perioperative setting. In contrast, posterior ischemic optic neuropathy is a common presentation of arteritic ischemic optic neuropathy.[93] Ischemic optic neuropathy is an important consideration in anesthesia planning because perioperative ischemic optic neuropathy is a devastating complication seen most commonly in association with spine or cardiac surgery.

Risk Factors

Although the exact cause of nonarteritic anterior ischemic optic neuropathy remains elusive, most affected patients have a small optic cup-to-disc ratio (fig. 2A).[94] As a result, axons subjected to ischemia are compressed at the lamina cribrosa, the exit point of the optic nerve from the eye (fig. 2B).[95] Other risk factors for anterior ischemic optic neuropathy are hypertension, diabetes, smoking, hyperlipidemia, hypercoagulable states, obstructive sleep apnea, and migraine.[96,97]

Pathophysiology

It has been hypothesized that nonarteritic anterior ischemic optic neuropathy is the result of vascular dysregulation, as well as anatomical and physiologic variations in the blood supply to the optic nerve.[97] In a small study, 20% of normal subjects demonstrated abnormal autoregulation of blood flow in the optic nerve head.[98] Due to the constricted space of the lamina cribrosa, edema resulting from axonal ischemia leads to a vicious circle of vascular compression and ischemia of the optic nerve axons.[99] Rodent models have demonstrated the presence of a significant neuroinflammatory component with high levels of proinflammatory prostaglandins and cytokines.[100] In later stages, there is death of the axons in the optic nerve and apoptotic death of the retinal ganglion cells.[101] The latter results in the optic disc pallor or atrophy that is visible in the ocular fundus in the late stages of ischemic optic neuropathy. People who have spontaneous nonarteritic ischemic optic neuropathy in one eye are at increased risk of nonarteritic ischemic optic neuropathy in the fellow eye. To date, treatment of sleep apnea is the only intervention that has been shown to be associated with reduced risk of fellow eye involvement.[102]

In contrast, posterior ischemic optic neuropathy affects the part of the optic nerve between the lamina cribrosa and the optic canal, a region with a blood supply (fig. 3) that results in watershed zones. A peripheral centripetal vascular system is from recurrent branches of the peripapillary choroid and the circle of Zinn, which are anastomoses of branches of the posterior ciliary arteries. Pial branches from the central retinal artery and ophthalmic arteries, and posterior ciliary arteries are added contributors. An axial centrifugal vascular system formed by branches from the intraneural central retinal artery contributes but is not present in every eye. These differences in blood supply in the posterior optic nerve may render some individuals more susceptible to posterior ischemic optic neuropathy; however, there is no current method for detecting such differences in humans.[103,104]

Patients at Increased Risk for Perioperative Ischemic Optic Neuropathy

Epidemiology

Ischemic optic neuropathy is the most common cause of perioperative vision loss. Anterior ischemic optic neuropathy occurs more frequently in patients after cardiac surgery and with a history of coronary artery disease.[66] Posterior ischemic optic neuropathy is more common in those undergoing spine fusion surgery, especially on the lumbar or sacral spine, and is usually bilateral.[105] Posterior ischemic optic neuropathy is also associated with intraoperative
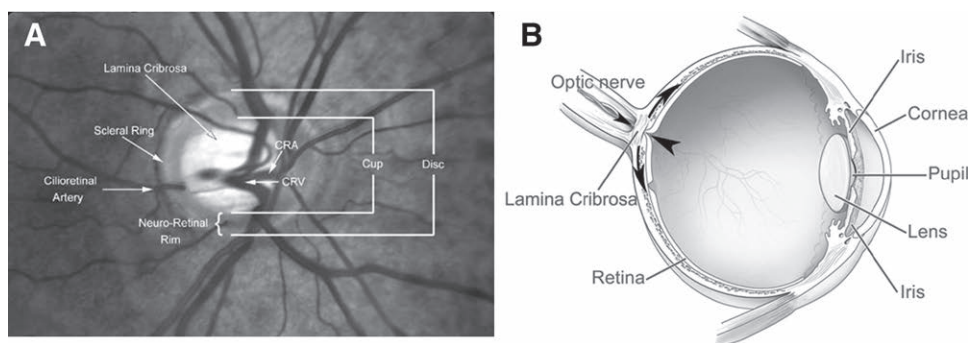
Fig. 2. (*A*) The components of the cup and disc in the fundus image of the eye. In a low cup-to-disc ratio, the axons that constitute the optic nerve are at risk of compression as they exit the eye in the lamina cribrosa. In a high cup-to-disc ratio, there is a higher risk of glaucoma. Reproduced with permission from https://novel.utah.edu and Dr. Kathleen Digre, the copyright holder. A link to the figure is available at https://collections.lib.utah.edu/ark:/87278/s6d24vxw. Accessed March 24, 2022. CRA, central retinal artery; CRV, central retinal vein. (*B*) Cross-section of the eye illustrating locations of retina, optic nerve, and the lamina cribrosa. The *large arrowhead* shows the pressure exerted by the intraocular pressure, and the *small arrowhead* the retrobulbar cerebrospinal fluid pressure. The intraocular pressure also generates a pressure load to the inner surface of the eye wall, as shown by the curved arrows. Reprinted with permission from the publisher from Imaging of the lamina cribrosa and its role in glaucoma: A review. Clinical & Experimental Ophthalmology, Cheng C-Y, Girard MJA, Koh V, *et al.* John Wiley and Sons, 2018.
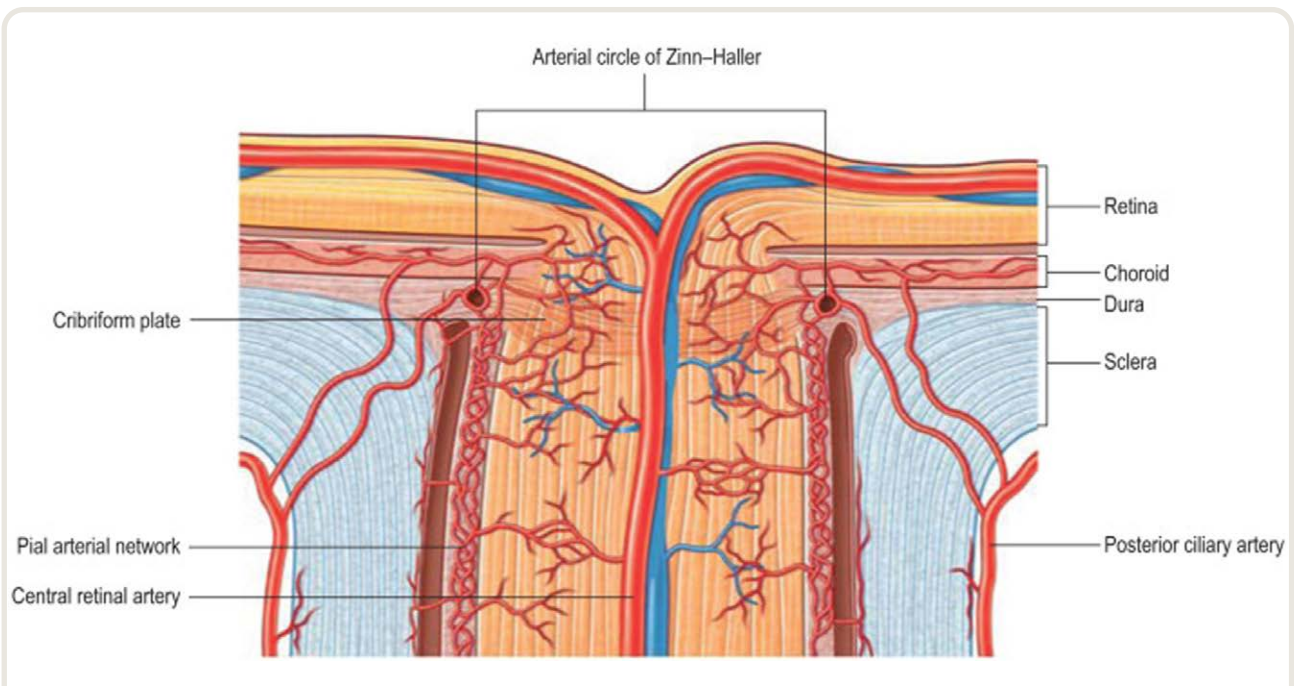
Fig. 3. The circulation of the optic nerve head and posterior portion of the optic nerve. The blood supply of the optic nerve head (*top*) is derived primarily from the arteriolar anastomotic circle of Zinn–Haller, which is supplied by the posterior ciliary arteries, the pial arteriole plexus, and the peripapillary choroid. The posterior optic nerve is supplied by pial branches from the central retinal and ophthalmic arteries, and posterior ciliary arteries. An axial centrifugal vascular system formed by branches from the intraneural central retinal artery contributes but is not present in every eye. Reprinted with permission from the publisher from Pineles SL, Balcer LJ. Visual loss: Optic neuropathies in Neuro-ophthalmology: Diagnosis and Management, 3rd edition. Edited by Liu GT, Volpe NJ, Galette S. New York, Elsevier, 2019, pp. 5, 101–196.

blood loss, and is more likely to cause complete blindness compared to anterior ischemic optic neuropathy.[106]

The estimated incidence of perioperative ischemic optic neuropathy is 1 in 60,000 to 125,000 anesthetics,[16,107] mostly in spine, cardiac, vascular, orthopedic joint replacement, and head and neck surgery.[59,108,109] In the U.S. Nationwide Inpatient Sample, spine and cardiac surgery had the highest rates of perioperative vision loss, at 3.09 per 10,000 and 8.64 per 10,000, respectively.[59] There has been little change in incidence of ischemic optic neuropathy after cardiac surgery. An encouraging trend is that the incidence of ischemic optic neuropathy after spinal fusion surgery has been declining, from an estimated 1.63 per 10,000 spine fusion surgeries in 1998 to 2000 to 0.6 per 10,000 in 2010 to 2012.[5,7]

Clinical Presentation and Treatment

The onset of perioperative ischemic optic neuropathy is usually within 24 to 48 h after surgery; often the patient recognizes a change in vision upon awakening. There is an afferent pupil defect or nonreactive pupil(s), and vision loss ranging from visual field deficits to no light perception. Color vision is often diminished.[105] An altitudinal deficit, *i.e.*, loss of visual sensation in the horizontal half of the visual field, is more typical of anterior than posterior

ischemic optic neuropathy.[97] Cases that follow spinal fusion surgery are more commonly posterior ischemic optic neuropathy, and are bilateral more than 50% of the time.[105] When a patient complains of loss of vision after surgery or ischemic optic neuropathy is suspected, expeditious ophthalmological consultation is paramount for diagnosis[110] and rapid initiation of treatment (table 1).

Unfortunately, there are no evidence-based treatments for perioperative ischemic optic neuropathy.[111–115] The disease often produces vision loss that will profoundly and unexpectedly change the patient's life; therefore, any non-harmful treatment that has some chance of restoring vision should be considered. Case reports have documented some improvement using the following treatments: (1) restoring hemoglobin and systemic blood pressures to or close to baseline,[116,117] (2) elevating the head to decrease the presumed increase in venous pressure if there is significant facial edema,[118] (3) high-dose steroids,[93,119,120] and (4) hyperbaric oxygen.[111] The treatment should be chosen in consultation with a neuro-ophthalmologist.

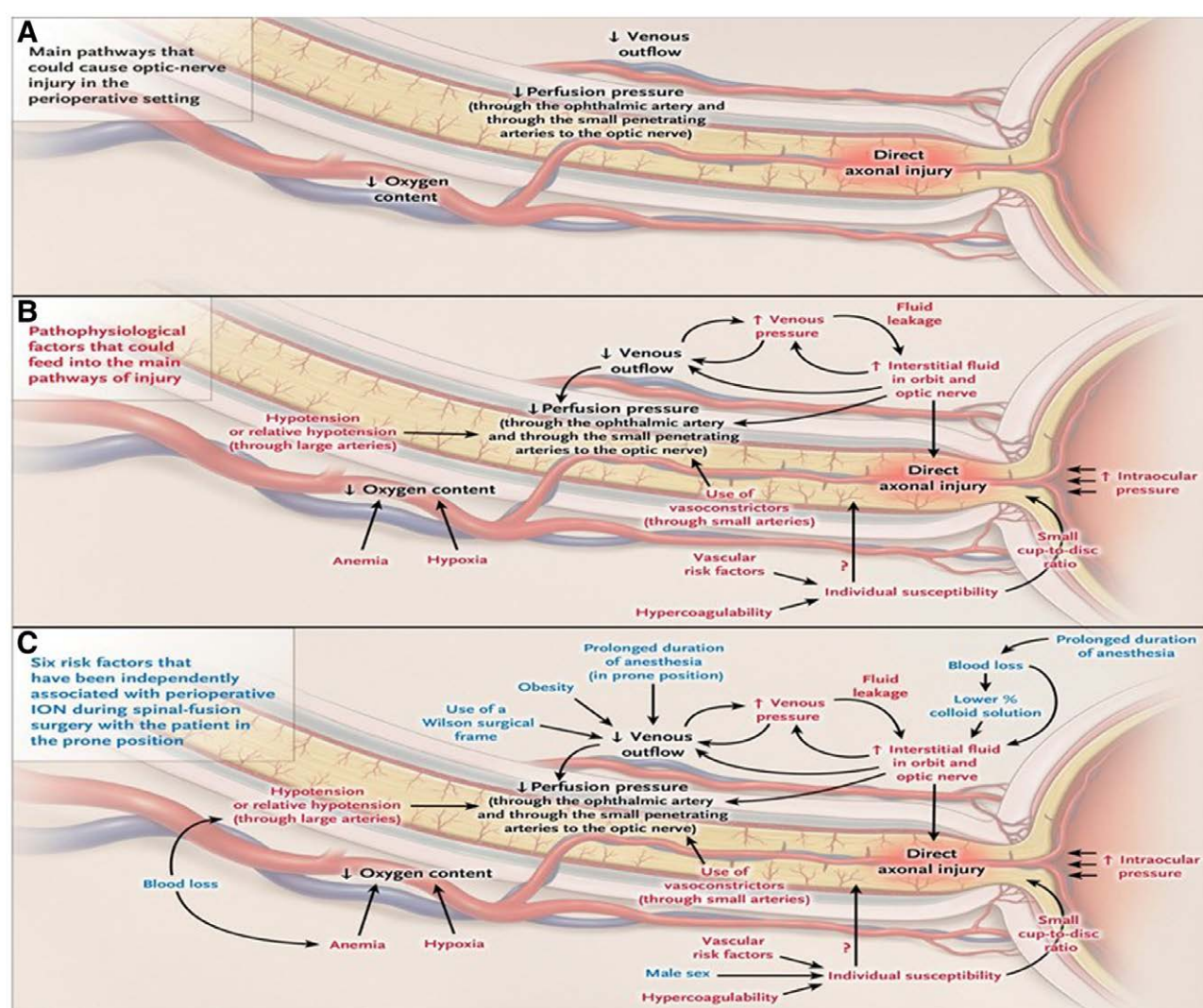### Ischemic Optic Neuropathy and Spinal Fusion Surgery

Prone positioning, large estimated blood losses, lower colloid compared to crystalloid fluid administration, lengthier anesthesia duration, Wilson frame use, obesity, and

male sex were significantly and independently associated with ischemic optic neuropathy after spinal fusion surgery.[105] These factors reduce oxygen delivery to the optic nerve (fig. 4), leading to axonal degeneration, which is evident as optic nerve pallor or atrophy within weeks to months after the event.[121–123] Although not proven, if fluids administered perioperatively are comprised of a lower percentage of colloid *versus* crystalloid solution, there may be accumulation of fluid within the optic nerve. There have been no studies of differences with specific types of colloids. Prone position can increase venous pressure in the optic nerve, an effect potentiated by use of the Wilson

surgical frame (where the head is placed lower than the heart) and obesity.[90] Increased interstitial fluid accumulation and decreased perfusion pressures may contribute to the pathophysiology of ischemic optic neuropathy.

Animal models were developed to try to determine the mechanisms of perioperative ischemic optic neuropathy. Lee *et al.* showed that a hematocrit decreased to 15%, jugular venous occlusion, and hypotension (50 mmHg) combined in adult pigs significantly decreased blood flow to the optic nerve. But limitations were lack of histology of the optic nerve or optic nerve function, and pig eye circulation differing significantly from human.[123] Roth *et al.*

Fig. 4. Possible factors contributing to perioperative ischemic optic neuropathy. (*A*) Decreased perfusion pressure, decreased oxygen delivery, increased venous pressure, and direct axonal injury as components of axonal damage. (*B*) The multiple proposed mechanisms of perioperative ischemic optic neuropathy. (*C*) In *blue*, the impact of the six independent risk factors for ischemic optic neuropathy after spinal fusion surgery with the patient positioned prone for surgery: male sex, obesity, Wilson frame, longer anesthesia, greater estimated blood loss, and lower percent colloid solution. Reprinted with permission from the publisher from Biousse V, Newman NJ. Ischemic optic neuropathies. N Engl J Med 2015; 372:2428–36.

found that hemodilution and 70-degree head-down tilt in rats significantly altered visual evoked potentials, electrical activity specifically originating in retinal ganglion cells (scotopic threshold response), and increased glial reactivity in the optic nerve.[122] However, this study lacked histological measurements of axonal damage in the optic nerve to correlate with the functional changes. Updated animal models may be useful in determining mechanisms of perioperative ischemic optic neuropathy and new treatment strategies. Regeneration of the optic nerve and connection to the visual cortex remain ongoing challenges.[124] There are exciting new experimental studies in rodents suggesting that regeneration of the optic nerve may be possible using cell-based therapies, but there are still few clinical trials in humans.[125]

Ischemic Optic Neuropathy and Cardiac Surgery

There are only a few studies on perioperative ischemic optic neuropathy in cardiac surgery.[66,126] Two single-institution studies retrospectively examined risk factors in cardiac surgery. In a study of 600 patients, factors differing in those with ischemic optic neuropathy *versus* those unaffected were longer cardiopulmonary bypass time, lower minimum hematocrit, greater mean 24-hour postoperative weight gain, and greater vasoactive drug requirements to maintain adequate blood pressures.[127] In 28,000 cardiac surgeries, ischemic optic neuropathy occurred in 0.06%. Low minimum postoperative hemoglobin, severe vascular disease, preoperative coronary angiography within 48 h before surgery, longer cardiopulmonary bypass, and blood transfusions were associated with ischemic optic neuropathy.[128] This study had no correction for multiple comparisons. Rubin *et al.* studied 5,559,395 discharges after cardiac surgery in the U.S. Nationwide Inpatient Sample. Carotid artery stenosis, stroke, diabetic or hypertensive retinopathy, macular degeneration, glaucoma, and cataract were associated with increased risk of ischemic optic neuropathy.[7] This was the first study demonstrating a correlation between pre-existing eye disease and perioperative ischemic optic neuropathy, but further studies are necessary to assess the significance of this novel finding. Findings are limited by inability to confirm discharge diagnoses, and possible reporting bias.[129] Treatment considerations for ischemic optic neuropathy after cardiac surgery are similar to those after spine surgery.

Anesthetic Management in Patients at Elevated Risk for Perioperative Ischemic Optic Neuropathy

In 2019, the American Society of Anesthesiologists Task Force on Peri-operative Visual Loss published a practice advisory specific to ischemic optic neuropathy in lumbar and sacral spinal fusion surgery.[130] The task force recommended that patients in whom prolonged procedures, substantial blood loss, or both are anticipated should be informed of the

increased risk of visual loss. These recommendations were based upon evidence from the literature, and consensus of a group of expert neuroanesthesiologists, spine surgeons, and neuro-ophthalmologists. The advisory recommended maintaining arterial pressure at appropriately elevated levels in hypertensive patients, avoiding deliberate hypotension in high-risk patients unless the anesthesiologist and surgeon agree its use is essential, and treating prolonged significant decreases in blood pressure. Monitoring hemoglobin or hematocrit values was recommended in high-risk patients with substantial blood loss and keeping the head level with or higher than the rest of the body when possible. Consideration to stage complex procedures on a case-by-case basis was recommended. In theory, staging should result in less blood loss and fluid administration, but its impact on perioperative outcomes including ischemic optic neuropathy still has not been well described.[131]

Perioperative Ischemic Optic Neuropathy, Malpractice Litigation, and Informed Consent

Considering the serious nature of perioperative vision loss, malpractice litigation and large monetary payments are likely. In nine cases of perioperative vision loss in one U.S. insurer's database (Controlled Risk Insurance Company, Boston, Massachusetts), the mean indemnity payment was $906,000.[132] An informed consent discussion that includes a rare but life-changing complication like ischemic optic neuropathy can be challenging for the anesthesia provider. More than 80% of patients preferred full disclosure of risk of vision loss in spine surgery by the surgeon before the day of surgery.[133] Informing the patient about the heightened risk of ischemic optic neuropathy in advance allows more time for discussion and can decrease the considerable anxiety and possible delay of surgery.

About half of U.S. states apply the "reasonable patient standard," where the physician is expected to disclose all information that would influence any reasonable person to decide whether or not to undergo a procedure.[134] Vision loss, while rare, is life-changing and serious enough to constitute a material risk to a reasonable patient.[135] Although controversial, it has been suggested that the reasonable patient standard be applied uniformly.[134] Even in states where such informed consent is not required, the anesthesia provider should consider informing the patient of the risk of vision loss as such informed consent is consistent with modern concepts of patient autonomy.[136]

Cerebral Visual Loss

Epidemiology

Cerebral vision loss is a heterogeneous disorder encompassing multiple different causes of brain parenchymal dysfunction leading to visual impairment. There are few studies of perioperative cerebral visual loss. Understanding the differential diagnoses is critical because this entity may

be confused with posterior ischemic optic neuropathy and retinal artery occlusion since both have normal retinal and optic nerve examinations at the time of presentation (table 1). Among 808 coronary artery bypass grafting operations, there were 10 cases, but the responsible cerebral injury was confirmed by computerized tomography of the brain in just 5.[137] In 700 coronary artery bypass grafting operations and valve replacements by a single surgeon, there were 2 unilateral occipital cortex infarctions.[138] Shaw *et al.* found a 5% incidence of cerebral vision loss in 312 patients after coronary bypass grafting in a prospective study.[139]

Cerebral vision loss has also been reported after spine fusion surgery, but as with cardiac, there are mostly case series reporting very few cases and little information to discern cause.[140–142] Shen *et al.* examined more than 5.6 million discharges in the Nationwide Inpatient Sample with a procedure code for spine, cardiac, orthopedic, or abdominal (as control) surgery. A cerebral visual loss *International Classification of Diseases* code was in 0.38 per 10,000 discharges.[59] The highest risk to develop cerebral visual loss was in cardiac and spine surgery. A surprising finding was that those younger than 18 yr were the highest–risk age group. Subsequently, this was confirmed in the Nationwide Inpatient Sample for discharges specifically younger than 18 yr between 2002 and 2011 with a discharge diagnosis of scoliosis and a procedure code for spinal fusion surgery. Postoperative visual loss was in 0.16%; all were due to cerebral vision loss. Fusion of more than eight spinal levels was associated with an odds ratio of 2.4 for developing cerebral vision loss.[143] The cause of this increased risk of cerebral vision loss in spinal fusion in those younger than 18 yr is still unexplained.

Clinical Features

Symptoms differ depending upon the location of cerebral dysfunction, ranging from isolated homonymous (same in both eyes) unilateral visual field defects from unilateral occipital lobe pathology, to homonymous visual field loss with other neurologic symptoms due to unilateral parietal or temporal lobe pathology, to complete blindness with denial of symptoms due to bilateral occipital lobe pathology, and to visual processing disturbances with intact visual acuity and visual fields due to bilateral parietal lobe pathology. Perioperative stroke involving the internal carotid, or middle, or posterior cerebral arteries territories can result in cerebral vision loss. Bilateral occipital cortex (area V1) damage produces binocular bilateral visual loss, and unilateral damage presents as binocular unilateral visual loss in the form of homonymous hemianopia. If central vision is present and only V1 is infarcted, responses to threat and optokinetic nystagmus are intact. Pupil reaction, eye movements, and ocular fundus are normal. Agnosia (lack of awareness of deficit) and confabulation may be present in complete blindness due to cerebral injury, which is known as Anton syndrome (table 1).[144]

Mechanism and Pathophysiology

Cerebral vision loss is generally caused by emboli to the posterior cerebral arteries supplying area V1, or a watershed infarct in the parieto–occipital area that may be associated with hypotension.[145] The occipital pole, with its foveal representation in the retina, receives a dual blood supply from the middle cerebral and posterior cerebral arteries; therefore, central vision is often spared in posterior cerebral artery infarction.[146] The pathophysiology of cerebral vision loss after spine fusion and coronary artery bypass grafting remains incompletely understood. Embolism as fat and atheroma or microemboli of lipid and fibrin–platelet aggregates occurs during open heart sugery;[147] those with aortic atherosclerosis appear to be at increased risk of embolic phenomena.[148] The watershed zone between the middle and posterior cerebral arteries, worsened by existing cerebrovascular disease, can increase the susceptibility of a patient to decreased cerebral perfusion such as with systemic hypotension.[149]

Prevention and Treatment of Cerebral Vision Loss

Generally there is incomplete resolution of visual field defects. There are no specific treatments apart from prioritizing prevention of the progression of acute stroke. A stroke specialist should be consulted. If eligible for reperfusion therapy, treatment must be initiated rapidly, but thrombolysis may be contraindicated in postoperative patients. Mechanical thrombectomy without anticoagulation may be an option.[150] There are no randomized studies on prevention of cerebral vision loss in cardiac surgery, but adequate removal of air and particulate matter from the heart may decrease the risk of embolism.[151,152] Maintenance of adequate systemic perfusion pressure may prevent episodes of hypoperfusion in patients with known cerebrovascular disease, but no controlled studies have associated visual loss and perfusion pressure in open heart surgery; therefore, it is not possible to provide any evidence-based recommendations to prevent cerebral vision loss in this setting.

Glaucoma

Epidemiology

Glaucoma is a group of progressive age-related and intraocular pressure-dependent optic neuropathies causing slow, progressive retinal ganglion cell degeneration, eventually resulting in irreversible visual field loss.[153–155] It is the second leading cause of blindness in the world, affecting more than 3 million in the United States and more than 70 million worldwide, expected to reach 112 million worldwide by 2040.[156] The elderly are disproportionately affected.[157,158] With an increasingly aging population, anesthesia providers are very likely to encounter patients with glaucoma.

There are three main types of glaucoma. *(1) High tension glaucoma,* characterized by increased resistance to or blockage of aqueous humor drainage, accounts for more than 80% of glaucoma in the United States.[12] Patients are usually asymptomatic during the early stages. Risk factors are a high degree of near-sightedness, family history, Black race, increasing age, cup-to-disc ratio greater than 0.7 (fig. 2A), optic disc asymmetry, and intraocular pressure 22 mmHg or greater.[159] *(2) Normal tension glaucoma* is glaucoma occurring despite normal intraocular pressure.[160] Hypotheses concerning its origin include retinal ganglion cell or axonal hypersensitivity, restricted glymphatic flow,[161] activated microglia,[162] vasculopathy, or intermittent hypotension.[163] Alternatively, low intracranial pressure causes a pressure gradient across the lamina cribrosa.[164,165] The lamina cribrosa displaces posteriorly (fig. 2B), deforming its pores and compressing the axonal fibers and blood vessels of the optic nerve.[166] *(3) Angle-closure glaucoma* is an emergent form of glaucoma characterized by acute reduction in the angle between the iris and cornea, narrowing the trabecular meshwork, and rapidly elevating the intraocular pressure (fig. 5).

Pharmacologic and Surgical Treatment

Reducing intraocular pressure is the only proven method to treat glaucoma.[167] β–Adrenergic receptor antagonism or carbonic anhydrase inhibition decrease aqueous humor production. Prostaglandin analogues increase aqueous humor outflow *via* the uveoscleral pathway. α-Adrenergic agonists decrease aqueous humor production and increase uveoscleral outflow.[168] Surgical interventions include minimally invasive surgery, trabeculectomy, and drainage devices.[169]

Preoperative Evaluation and Anesthetic Care

The main perioperative concern in glaucoma is a degradation in visual acuity or worsening of visual field deficits due to increased intraocular pressure; however, their occurrence has, to date, not been well documented. Many factors in the perioperative period are associated with increased intraocular pressure, including but not limited to head-down, prone, and lateral positioning; insufflation of gas into the abdomen for laparoscopic and robotic surgery; endotracheal intubation; coughing; succinylcholine; and volume of intravenous fluids.[170,171] Conversely, inhalation anesthetics and induction agents including propofol lower intraocular pressure.[172] Most of these alterations are short-lived with the exception of the more prolonged increases from surgical positioning, particularly with the patient positioned head-down or prone. The impact of prolonged prone or head-down patient positioning on visual outcomes in those with glaucoma has not been well described. Most studies are small and limited, measuring intraocular pressure mostly in nonglaucomatous subjects.[173–180]

Head-down Positioning in Subjects with Glaucoma: Nonsurgical. Moderate head-down tilting can be used as a provocative test to identify patients who progress to development of glaucoma. However, these findings are derived from small studies. After 8 min of 10-degree head-down tilt, there were greater changes in the pattern electroretinogram, a specific test of retinal ganglion cell function, in those previously identified with early glaucoma.[181,182]
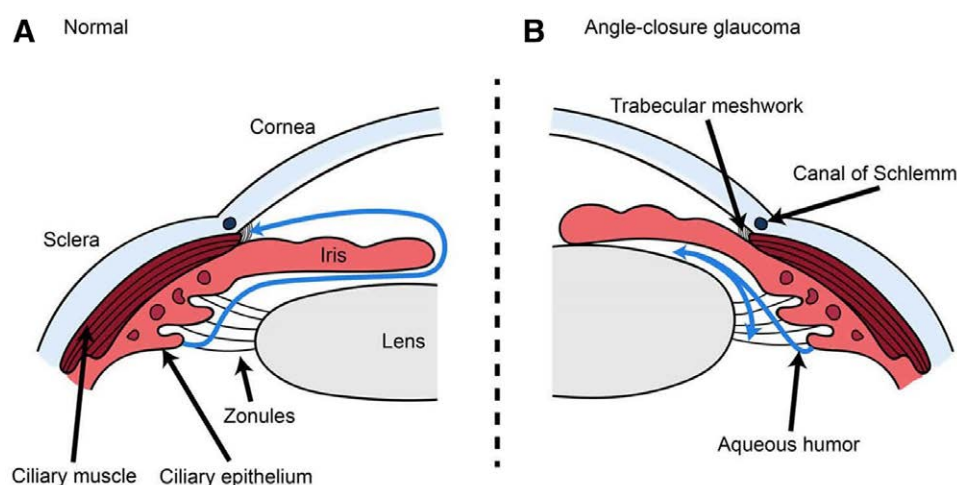
Fig. 5. (*A*) In a normal eye, aqueous humor flows out of the eye into draining veins through the canal of Schlemm as shown with a *blue curving line* on the *left*. (*B*) In acute angle closure glaucoma, forward bowing of the iris obstructs the normal aqueous humor flow around the lens and iris, and there is no access to the canal of Schlemm (*blue curving lines*), resulting in an acute increase in intraocular pressure. Reproduced with permission from https://www.healthjade.com. Accessed March 24, 2022.

Prone or Head-down Positioning and the Surgical Patient with Glaucoma. Numerous studies documented increased intraocular pressure in patients positioned prone for surgery, mostly related to alterations in chest wall mechanisms and intrathoracic pressure.[183–191] Head-up tilting and eye drops lowered intraocular pressure.[186,187] The degree and duration of intraocular pressure increases that can adversely affect vision, and the specific impact upon glaucoma patients, of prone positioning have not been determined. Robotic-assisted laparoscopic prostatectomy or hysterectomy with steep Trendelenburg positioning (25- to 45-degree head-down) increases intraocular pressure due to increased episcleral venous pressure,[192] predictable from the Goldmann equation,[193] intraocular pressure = (F/C) + P, where F represents aqueous flow rate, C represents aqueous outflow, and P is episcleral venous pressure. Most studies of steep Trendelenburg positioning and robotic-assisted prostatectomy or hysterectomy have small sample sizes and are in patients without glaucoma.[179,194–197] The biggest study included 51 nonglaucomatous subjects who underwent robotic-assisted laparoscopic prostatectomy or hysterectomy while positioned in steep Trendelenburg. No changes were found at 3 months in retinal nerve fiber layer or ganglion cell complex thickness, foveal threshold, mean deviation, or pattern standard deviation.[194] One small study examined visual outcomes in 10 glaucoma patients who underwent robotic-assisted laparoscopic prostatectomy in steep Trendelenburg. Two subjects developed progressive thinning of the retinal nerve fiber layer 2 months after surgery. The visual fields were not described.[175]

Conclusions on Anesthesia for Patients with Glaucoma or at Risk for Glaucoma

Most studies have focused solely on intraoperative changes in intraocular pressure. There have been very few studies of patients with glaucoma having anesthesia and surgery; clearly there is a need for further research with measurement of visual outcomes. The authors are each typically asked a few times a year what should be done with the glaucoma patient who requires surgery while placed in the prone or head-down position. There is currently no evidence-based answer to this question. Rather than intraocular pressure, the more meaningful parameter in surgical patients positioned head-down or prone is likely mean ocular perfusion pressure. This can be inferred from long-term follow-up studies of glaucoma patients, where ocular perfusion pressure variability appears to a risk factor for progression of visual field deficits. The 24-h mean ocular perfusion pressure variability correlated with faster paracentral visual field loss progression.[198,199] However, these findings were in patients studied during a much longer time than would be experienced by a surgical patient. Since ocular perfusion pressure is approximately the difference between

mean arterial blood pressure and intraocular pressure, a reasonable do-no-harm recommendation based upon this known physiology is that systemic blood pressures should be maintained close to preoperative baseline in subjects with glaucoma when positioned prone or head-down for surgeries. Intraocular pressure–lowering drugs should be continued in patients with glaucoma until the time of surgery.

Perioperative Acute Angle-closure Glaucoma

Acute angle-closure glaucoma is a rare, vision-altering perioperative complication, first reported in 1957.[200] This ophthalmological emergency must be recognized and treated promptly to prevent optic nerve damage. The signs and symptoms are abrupt and severe eye pain, conjunctival redness, blurry vision, visual halos, a fixed mid-dilated pupil, corneal edema, headaches, and nausea or vomiting (table 1). Risk factors include a history of glaucoma, genetic predisposition, female sex, increased age, far-sightedness, shallow anterior chamber depth, increased lens thickness, small corneal diameter, and Asian race.[201] Most of what is known about perioperative acute angle-closure glaucoma is derived from case reports.[190,191,202–205]

Drugs used by anesthesia providers that may provoke acute angle-closure glaucoma in susceptible individuals include parasympatholytic or sympathomimetic drugs, such as atropine, scopolamine, ephedrine, or epinephrine, which cause pupil dilation. Psychologic stress causing sympathetic nervous system activation and mydriasis may also contribute.[206] It is imperative that a postoperative patient complaining of symptoms suggesting acute angle-closure glaucoma have a timely consultation with an ophthalmologist for immediate reduction of intraocular pressure and prevention of blindness. The diagnosis is confirmed by the findings of closed angles using gonioscopy. Smartphone and other portable devices enable these measurements to be conducted at the bedside without losing critical time transporting to the eye office or clinic.[207,208] A diagnosis can also be made rapidly using a penlight; with narrow angles, a shadow is visible in the nasal side of the iris.[209] Vision can be successfully preserved using medications to rapidly lower intraocular pressure, as well as *via* definitive therapy by a peripheral iridectomy, or a lens extraction.[210]

Patients with Low Vision

Anesthesia providers will likely encounter patients with low vision or blindness, present in approximately 2.5% of the population of the United States,[2] and expected to increase due to rising prevalence of diabetic retinopathy, age-related macular degeneration, glaucoma, and cataracts.[211]

Light perception *via* retinal photoreceptors, and a class of specialized intrinsically photosensitive retinal ganglion cells expressing melanopsin, is a major influence upon circadian rhythms and mood that is lacking in blind people.[212–214]

Disordered sleep and alertness and mood changes are common.[14] Non–24-h sleep–wake disorder is present in some blind subjects due to inability to perform the normal circadian pacemaker reset. Tasimelteon, a novel melatonin receptor 1 and 2 agonist, is currently the only Food and Drug Administration–approved treatment.[215] There have not been any human studies on this drug or on the impact of low vision or blindness on outcomes after anesthesia and surgery. In rodents, isoflurane-induced cognitive impairment was decreased by circadian rhythm resynchronization with melatonin, suggesting the importance of attempting to adjust circadian rhythm for higher brain function. The anesthesia provider should consider the possibility of delayed awakening or heightened emergence delirium in these subjects.[216]

The lack of visual cues that humans rely upon for routine interpersonal interactions complicates communication with the low-vision patient and may render it challenging to convey the anesthesia plan and to obtain informed consent.[15] Sensitivity to the special needs of low-vision subjects is an essential part of compassionate and appropriate care and our obligation to patients both legally and ethically. Appropriate accommodations for low-vision patients undergoing care in healthcare organizations are mandated in the United States by the Americans with Disability Act,[217] and by similar laws in other countries.[218] A suggestion for informed consent is a recorded audio consent.[219] However, this could be difficult to implement and may not be compatible with all electronic medical record systems. It may instead be necessary for the anesthesia provider to read the entire consent form to the patient, receive acknowledgment of understanding, and allow the opportunity for clarifications and answering questions. Then the patient can make "a mark" in place of the signature on the written or electronic consent form.

Summary

Ocular diseases have a profound impact on the quality of life of patients, and comprehensive, conscientious care of these patients is instrumental in mitigating the risk of preventable complications, ranging from corneal injury to blindness. Some of these injuries are easily preventable and treatable, such as exposure keratopathy, while others such as ischemic optic neuropathy currently have no recognized effective treatment, and the emphasis should be upon prevention. Tables 2 and 3 are a summary of risk factors for ocular complications (table 2) and commonly encountered ocular conditions (table 3), as well as anesthesia management recommendations.

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Dr. Roth dedicates this review to the memory of his parents, Yitzhak Yaakov and Miriam Hinda Roth.

Table 2. Ocular Complications in Nonocular Surgery, Risk Factors, Prevention, and Treatment

	Exposure Keratopathy	Loss of Eye Contents	Acute Angle Closure Glaucoma	Ischemic Optic Neuropathy	Central Retinal Artery Occlusion
Risk factors	General anesthesia Decreased tear production Patient positioning Abnormal eyelid closure	Corneal transplant Open globe injury	History of glaucoma Genetics Female Elderly Anatomical factors Asian race	Spine fusion Cardiac surgery Head and neck For spine surgery: Prone positioning, large estimated blood losses, lower colloid compared to crystalloid fluid administration, lengthier anesthesia duration, Wilson frame use, obesity, and male sex	Spine fusion Cardiac surgery Head and neck Use of nitrous oxide with recent vitrectomy and gas bubble For spine surgery: compression of the eye For cardiac surgery: embolism from heart or aorta
Prevention	Completely cover the eyes after induction of anesthesia Use of ointments in some cases Remove contact lenses Protect eye from false eyelashes	Protect eyes from trauma/increased intraocular pressure Eye shield, caution during positioning of patient	Medications to avoid in susceptible individuals: para-sympatholytic or sympathomimetic drugs	No established strategy but see section Anesthetic Management in Patients at Elevated Risk for Perioperative Ischemic Optic Neuropathy for ASA Task Force on Perioperative Visual Loss recommendations	Headrests and care to prevent eye compression in spine surgery with patient prone Decrease embolic phenomena in heart surgery Avoid nitrous oxide when vitreous gas bubble present
Treatment	Analgesia (local anesthetic eye drops not advisable) Antibiotics/eye cover in some cases Treatment/follow-up by an ophthalmologist	None; prevention is paramount to avoid profound visual loss	Immediate reduction of intraocular pressure by medications AND Peripheral iridectomy Treatment/follow-up by an ophthalmologist	Increase blood pressure and hemoglobin levels Elevate the head Steroids Hyperbaric oxygen None of these are proven effective In all cases, consultation on treatment with neuro-ophthalmologist	Thrombolysis, but may be risky in postoperative patient Consultation on treatment with neuro-ophthalmologist

ASA, American Society of Anesthesiologists.

Table 3. Commonly Encountered Ocular Conditions, Preoperative and Intraoperative Concerns, What Is Known/Not Known in Relation to Anesthesia, and Management Recommendations

	Idiopathic Intracranial Hypertension	Glaucoma	Low Vision
Preoperative eye concerns	Optic nerve disease: double vision, loss of vision, optic nerve swelling	Visual field loss, low vision, increased intraocular pressure	Difficulty with consent Communication issues Altered mood, alertness
Intraoperative concerns	Spinal and epidural analgesia Caution in COPD patients on acetazolamide	Head down or prone positioning and visual outcomes	None
What is known	Both spinal and epidural may be used safely Continue preoperative medications for the disease	Intraocular pressure increased by positioning prone or head-down	Many causes including glaucoma, diabetic retinopathy, macular degeneration, congenital
What is not known/perioperative management recommendations	—	Impact of further increases in intraocular pressure on vision after surgery/positioning prone or head-down is not known Advisable to keep systemic blood pressure at or near patient's baseline, as systemic blood pressure is a major influence on ocular perfusion pressure; evidence base still lacking Continue the patient's preoperative eye drops up until time of surgery	The impact of low vision with or without altered alertness on awakening after anesthesia is not known Anticipate the need for more time to obtain a preoperative anesthesia consent

Due to space constraints, not all conditions are shown.
COPD, chronic obstructive pulmonary disease.

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<div style="background:#b32025;color:white;">**ANESTHESIOLOGY REFLECTIONS FROM THE WOOD LIBRARY-MUSEUM**</div>

The Tale of Aspirin: A Weeping Willow to Soothe Your Sorrows

One summer day in Chipping Norton, England, the town's vicar, Reverend Edward Stone (1702 to 1768, *upper right plaque*), strolled pensively through his lush grounds, pausing to pluck a frond from a large willow tree that swayed gently in the breeze (*left*). He fiddled with the branch, then nibbled on it. Its "extraordinary bitterness" reminded him of cinchona bark, which was known for its antipyretic effect. Believing, a la Paracelsus, that the physical traits of plants hinted at their therapeutic properties, Stone theorized that the willow tree, which flourished amid moisture, could cure malarial fever, which also flared in humidity. He gathered a pound of willow branches, dried them "outside a baker's oven for over 3 months," then pulverized them into powder. The next time he fevered, Stone ingested the powder dissolved in liquid. He soon marveled at its power to soothe. After using willow bark to relieve the misery of 50 parishioners, he penned a letter in 1763 to the President of the Royal Society that publicly announced his discovery. The willow tree would later prove to be a source of salicylate, a potent anti-inflammatory. More than 100 years later, chemists at the Bayer Company would acetylate salicylate to make aspirin (1899, *right*). (Copyright © the American Society of Anesthesiologists' Wood Library-Museum of Anesthesiology. www.woodlibrarymuseum.org)

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