Perioperative vision loss (POVL) associated with routine surgical procedures is a rare but catastrophic event. Although no clear direct cause of POVL has been determined, it is theorized that decreased ocular perfusion pressure of the optic nerve is responsible for POVL. This article will investigate theories as to why POVL occurs, risk factors associated with POVL, anatomy related to POVL, preventive measures that may help to reduce risk of POVL, and interventions that the anesthesia provider can implement.

Keywords: Perioperative visual loss, postoperative vision loss, prone surgery, steep Trendelenburg.

Objectives
At the completion of this article, the reader should be able to:
1. Compare normal blood flow to and from the optic nerve, with compromised blood flow as characterized in anterior ischemic optic neuropathy, posterior ischemic optic neuropathy, and central retinal arterial occlusion.
2. Discuss the relationship between ocular perfusion pressure, intraocular pressure (IOP), and mean arterial pressure.
3. Identify surgical procedures that are associated with perioperative vision loss (POVL).
4. Identify specific surgical and patient factors that contribute to POVL.
5. Discuss interventions that can reduce IOP in the prone or steep Trendelenburg positions.

Introduction
The first report of perioperative vision loss (POVL) was documented in 1948, and this condition was attributed to improper head positioning during a procedure performed with the patient in prone position. In a retrospective analysis of 541,485 patients who had spinal surgery between 1996 to 2005, the overall incidence of POVL was documented as 1.9 events per 10,000 cases. Although rare, the frequently catastrophic nature of this complication increases the importance of its prevention.

Vision loss after surgery not only has profound implications for emotional well-being and return to presurgical functioning but also increases the length of stay after surgery and subsequently incurs greater costs. Patients who experienced vision loss spent an average of 8.6 days in the hospital vs 4.1 days for those unaffected. The average cost of stay was $49,532 compared with $22,697 for those without vision changes.

Anatomy and Physiology of the Eye
The eye is a complex optical system, similar in function to a camera; it comprises an aperture system (pupil), lens, and film (retina). The quantity of light that is allowed to pass through the pupil is controlled by the iris; the ciliary muscle surrounding the iris either constricts or relaxes, adjusting the amount of light that is permitted into the eye. Depending on the pupillary aperture, the quantity of light allowed to enter the eye can change by 30 times. Light rays become focused through the lens and then pass through vitreous humor, which gives the eye its form and shape. The lens focuses the image onto the retina, which is reversed and inverted in respect to the object being perceived. The brain is trained to consider the inverted image as normal and perceives the object in the upright position.

The retina is composed of about 3 million cones, which are responsible for color vision, and 100 million rods, responsible for perceiving black and white and for...
night vision. Stimulation of the cones and rods by light forms an action potential through the retinal neurons to the optic nerve. The optic nerves travel through the optic chiasm, synapsing onto the dorsal lateral geniculate nucleus of the thalamus and then to the primary visual cortex of the medial occipital lobe.³

Blood supply to the optic nerve originates from the internal carotid artery, which branches into the ophthalmic artery, and finally to the central retinal artery.⁴ Blood supply to the optic nerve can be divided into 2 distinct regions, the anterior optic nerve, or optic nerve head, and the posterior optic nerve. As demonstrated in Figure 1, the anterior portion of the optic nerve receives a greater supply of blood from the central retinal artery and posterior ciliary arteries,⁴ whereas the posterior segment of the optic nerve is supplied solely by penetrating pial arteries and their collateral branches.⁴ Venous return drains initially into the central retinal vein, and subsequently the superior and inferior ophthalmic veins.³

The eye maintains its shape with intraocular fluid, which is divided into 2 sections.³ Aqueous humor fills the anterior chamber of the eye, a free-flowing fluid that is continuously being formed and reabsorbed at a rate of 2 to 3 µL/min.³ All the aqueous humor is produced by the ciliary processes, linear folds that are extensions of the ciliary body, and exuded into the space between the lens and the iris (Figure 2).³ Aqueous humor is reabsorbed as it enters the canal of Schlemm, a venous blood vessel that surrounds the eye, and then drains into extracellular veins.³

Vitreous humor forms the posterior chamber of the eye between the lens and the retina, and maintains the shape and consistency of the eye. Vitreous humor, also referred to as the vitreous body, is a gelatinous mass composed of a fibrillar network of elongated proteoglycan molecules. Water and other small substances are able to diffuse through the vitreous humor; however, the fluid does not flow and behave as the aqueous humor.³

**Types of Postoperative Vision Loss**

The most prevalent cause for POVL is ischemic optic neuropathy (approximately 89% of POVL documented cases).⁴ The optic nerve can be divided into anterior and posterior segments, based on blood supply as described earlier. As the blood supply becomes disrupted in either the anterior or posterior segment, it results in anterior ischemic optic neuropathy or posterior ischemic optic neuropathy, respectively.⁴ Although there are no conclusive data as to why ischemic optic neuropathy occurs,⁵ anterior ischemic optic neuropathy is more frequently associated with cardiac surgery, and posterior ischemic optic neuropathy with spine surgery.⁶

Anterior ischemic optic neuropathy is characterized by sudden, painless, bilateral visual deficit ranging from a minor decrease in visual acuity to blindness, unilaterally or bilaterally.⁴ Diagnosis of anterior ischemic optic neuropathy is made by fundoscopic examination revealing optic disc edema, with or without peripapillary flame-shaped hemorrhages, and reduced pupillary light reflex.⁴,⁵ Posterior ischemic optic neuropathy presents with an acute loss of vision and visual field deficits, either unilaterally or bilaterally. Although initial physical examination findings reveal a normal optic disc, over subsequent weeks, optic atrophy and pallor develops.⁵,⁶ The clinical reality is that there is marked individual variability in blood supply to the optic nerve, and so the pattern of vision loss can be variable between individuals.⁶

Postoperative vision loss could also arise from central retinal arterial occlusion—most often associated with improper patient positioning, external compression to the eye, or emboli—which results in a reduction in blood supply to the entire retina (approximately 11% of POVL documented events).⁵,⁶ Visual field deficits initially can be severe, but, unlike ischemic optic neuropathy, can improve with time.⁴ Results of ophthalmoscopic examination demonstrate a pale edematous retina and a cherry-red spot on the macula.⁴,⁵ The use of horseshoe headrests during spine surgery can lead to unilateral vision loss...
involving periorbital eyelid edema, chemosis (swelling of the conjunctiva), ptosis, and corneal abrasion.6

Ocular Perfusion Pressure
Direct perfusion of the optic nerve is described by the relationship between intraocular pressure (IOP) and mean arterial pressure (MAP).3 Ocular perfusion pressure (OPP) is defined as the difference between MAP and IOP (OPP = MAP – IOP).7 Normal IOP ranges between 12 and 20 mm Hg, averaging about 15 mm Hg; it is regulated to remain constant in the healthy eye, usually ± 2 mm Hg of its baseline.3 The pressure is determined by resistance to outflow of aqueous humor from the anterior chamber as it is reabsorbed in the canal of Schlemm, and then into venous circulation.3 In the healthy eye, autoregulation occurs so that as pressure increases in the eye, the rate of flow into the canal of Schlemm increases, thus maintaining constant blood flow during changes in perfusion pressure.3

However, in certain situations, there can be an increased resistance to outflow in the canal of Schlemm, which results in increasing IOP. As IOP becomes pathologically high, it is no longer possible to maintain consistent blood flow, because autoregulation is disrupted. Once the IOP reaches 45 to 55 mm Hg, study findings have demonstrated a linear relationship between increasing IOP and declining blood flow.8,9 Boltz et al9 found that OPP is determined primarily by IOP, not MAP as previously believed; changes in arterial pressure are autoregulated more effectively than are increases in IOP.

Clinically, direct measurement of IOP is accomplished with the use of an applanation tonometer.1,3,7 In many studies described in this article, the Tono-Pen XL applanation tonometer (Reichert) is used to measure IOP.7

To measure IOP, the tonometer footplate is placed on the anesthetized cornea of the eye.3 When the central plunger is activated, it causes a plunger to be displaced inward toward the cornea.3 The tonometer records the amount of displacement, and then converts it into IOP.3

Although the use of a tonometer can accurately measure IOP, very few operating rooms have a tonometer available. Also, in certain positions, such as prone, it would be difficult for the practitioner to gain access to the eyes to use the tonometer. Visual assessment of the conjunctiva using the Molloy/Bridgeport Anesthesia Associates Observation Scale allows the practitioner to determine when IOP is elevated (Table 1).10 According to the scale, certain visual clues can indicate to the anesthesia provider that there is increased IOP. The presence of ocular edema signals that IOP has increased to 2.5 times the baseline pressure. Conjunctival swelling, or chemosis, indicates that the IOP is 3.4 times higher than the baseline pressure, and ecchymosis signals IOP is 4.3 times higher than baseline.10 Chemosis is detected as the white outer coating rises above the iris, appearing gelatinous (Figure 3).10 The presence of chemosis is a clue for the anesthesia provider; once chemosis is observed, IOP is approaching a critical level, allowing time for the provider to implement a beneficial intervention.10 Although multiple theories involve OPP to describe the mechanism by which POVL occurs, there are no conclusive data at this time to completely support it.

Potential Causes of Postoperative Vision Loss
Although historically ischemic optic neuropathy was frequently explained in relation to systemic factors that affect MAP, more recent work has focused on other possibilities such as acute venous congestion and com-
partment syndrome of the optic nerve. When surgical conditions require the patient to be positioned in either steep Trendelenburg or prone, the venous pressure rises because of increased intra-abdominal and intrathoracic pressure, creating increased IOP. As venous pressure rises in the head and neck, the higher hydrostatic pressure in the capillary beds causes capillaries to leak, forcing interstitial fluid to accumulate, constricting venous return, and eventually limiting perfusion to the optic nerve because of increased IOP. In addition, most cases of ischemic optic neuropathy occur in the posterior region of the optic nerve, in which flow is supplied by the collateral branches of the penetrating pial arteries; thus, it is already vulnerable to reduction in blood flow.

Building on the theory that increased venous pressure and congestion can create POVL, other studies have suggested that compartment syndrome of the optic nerve contributes to POVL. High venous pressure and interstitial edema accumulation compromises blood flow to the optic nerve. The optic nerve travels through the optic canal and lamina cribrosa, an enclosed space constructed of collagen and bone; as the area becomes more edematous, compression could occur to the optic nerve, leading to ischemia. Although the direct etiology of POVL has not been agreed on, there are certain surgical, patient, and positioning characteristics that have been determined to be correlated factors in the development of POVL.

- **Surgical Procedures Associated With Postoperative Visual Loss.** In a study analyzing more than 5.6 million patients in the Nationwide Inpatient Sample, it was 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). Reassuringly, within the 10 years that the study was conducted, between 1996 and 2005, the overall incidence of POVL has remained constant or has declined. In a more recent survey of POVL correlating to spinal surgery, the overall rate had decreased to 1.9 events per 10,000 cases. It should be noted, however, that true incidence is impossible to obtain and document. Many cases of POVL go without being reported at all, or if the incident is investigated in closed claims court, then it would not be reported until the case is completely closed.

Other surgical procedures that have been associated with POVL include head and neck surgery, sinus surgery, vascular surgery, cesarean delivery, gynecologic surgery, vascular surgery, liposuction, and orthopedic surgery. In surgical procedures involving the nose and sinuses, blindness was reported after direct surgical damage to the optic nerve. Although there are many reports and studies evaluating POVL, overwhelming evidence has demonstrated that spine and cardiac procedures have higher incidence of POVL than any other surgical procedure.

- **Factors Associated With Postoperative Visual Loss.** Determining the contributory factors and etiology of POVL has been a source of extensive debate and study in recent years. In a multi-institutional case-control study evaluating POVL after spine surgery, the following risk factors for ischemic optic neuropathy were identified: male gender, use of the Wilson frame, high estimated blood loss, obesity, anesthetic duration, and decreased percentage colloid administration of total fluids. However, this study was completed retrospectively with an anonymous submission to the registry; limitations due to bias and inaccuracy could have influenced study outcomes. Other studies have cited steep Trendelenburg position, prone position, and excessive fluid replacement to influence IOP and contribute to POVL. Table 2 summarizes factors associated with POVL.

There are multiple published studies that concluded patient position is a significant factor that increases IOP, specifically prone and steep Trendelenburg posi-
The occurrence of bilateral POVL was reported in a patient undergoing laparoscopic prostatectomy in the steep Trendelenburg position, without presence of intraoperative hypotension, hemodilution, extreme blood loss, or perceived metabolic disturbance; however, the patient remained in 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 a 3-year period. The IOP was measured using a tonometer at designated times during the surgery (Table 4). Molloy found a direct correlation between the duration of time in steep Trendelenburg position during surgery and increasing IOP. No study participants had any adverse events, and the study itself prompted further investigation into interventions the anesthesia provider can implement in order to reduce IOP.

After data review of 541,485 patient cases from the Nationwide Inpatient Sample database from 2002 to 2009, the highest accounts of POVL occurred in patients undergoing thoracic fusion for correction of deformity. Total count of POVL during the 8-year survey was 105 cases; of those, 53 occurred during surgery for spinal deformity correction. Also, average age was younger in the group that experienced vision loss (37.6 years) compared with the group that did not experience POVL.

Approximately 69% of ischemic optic neuropathy cases after spine surgery occur in male patients, even though there are equal numbers of each sex undergoing spine surgery. Although there are no anatomical differences between male and female spines, there is some evidence that estrogen offers a protective effect to retinal ganglion cells and perhaps prevents vision loss from occurring.

A pivotal publication by Lee et al cited increased blood loss and prolonged anesthetic time to be associated factors of POVL. Estimated blood loss of 1 L or greater occurred in 82% of cases of vision loss after spine surgery, and anesthesia lasted more than 6 hours in 94% of cases. Fluid shifts, capillary leak, and interstitial edema formation are known complications of increased estimated blood loss. Longer anesthetic time allows time for more fluids to be administered, and increases time the patient is vulnerable to blood loss.

Obese patients have increased abdominal girth, which when positioned in either steep Trendelenburg or prone, can increase intra-abdominal, intrathoracic, and central venous pressure leading to raised venous pressure in the head. In a similar manner, the Wilson frame also increases pressure in the head, since the head is lower than the heart (Figure 4). In both situations, obesity and use of the Wilson frame, prolonged increased venous pressure in the head and the eye’s orbit leads to interstitial fluid accumulation and reduction of OPP by increased IOP.

Some of these factors are, unfortunately, unavoidable during extensive surgical procedures. However, identification of these risk factors has led researchers to develop interventions the anesthesia provider can implement during prolonged surgical procedures performed in patients in the prone or steep Trendelenburg position. It is important to note that although risk factors have been determined by an increasing number of large database studies, a causal relationship between patient positioning, increased IOP, and the occurrence of POVL has yet to be established, predominantly because of the low incidence of vision loss that occurs in the surgical period.

### Table 3. Intraocular Pressure (IOP) and Time in Prone Position

(Data from Cheng et al, n = 20.)

<table>
<thead>
<tr>
<th>Position</th>
<th>Average IOP (mm Hg)</th>
<th>Timing of measurement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>19 ± 1</td>
<td>Before induction</td>
</tr>
<tr>
<td>Supine 1</td>
<td>13 ± 1</td>
<td>10 min after induction</td>
</tr>
<tr>
<td>Prone 1</td>
<td>27 ± 2</td>
<td>Before incision</td>
</tr>
<tr>
<td>Prone 2</td>
<td>40 ± 2</td>
<td>At conclusion of surgery</td>
</tr>
<tr>
<td>Supine 2</td>
<td>31 ± 2</td>
<td>Before reversal and emergence</td>
</tr>
</tbody>
</table>

- **Noncontributory Factors.** The POVL Study Group did not find patient characteristics such as older age, hypertension, atherosclerosis, smoking, or diabetes to be statistically significant in development of ischemic
optic neuropathy after spine surgery, contrary to other studies published. Based on newer data, the POVL Study Group suggests that the etiology of POVL may be related to intraoperative physiologic changes rather than by preexisting disease. In addition, presence of intraoperative hypotension and anemia were not found by the POVL Study Group to be associated with the development of vision loss after a surgical procedure. In the most recent practice advisory produced by the American Society of Anesthesiologists Task Force on POVL, deliberate hypotension was not shown to be associated with the development of POVL. The Task Force concluded that the use of deliberate hypotension could therefore be considered on a case-by-case for patients during spine surgery. There is no documented minimum hemoglobin or hematocrit value that has been associated with the development of POVL. For that reason, attempts to determine a transfusion threshold that would eliminate the risk of POVL related to anemia are inconclusive.

Interventions

Once it is determined that the patient is experiencing vision loss after a surgical procedure, an ophthalmologist should be contacted urgently for a consultation to determine the cause and to create an interventional plan. However, the reality is that effective treatments after the development of ischemic optic neuropathy (the commonest cause of POVL) are limited. For this reason, attention is focused on interventions that may prevent the development of IOP and POVL (Table 55-7). Initial research in placing the patient in reverse Trendelenburg position during prone spinal procedures has shown beneficial reduction in IOP compared with patients in a neutral prone position or prone Trendelenburg position. The patient, placed in either a 5° or 10° reverse Trendelenburg position during a prone spinal procedure, had decreased IOP compared with neutral prone position. Elevating the head above the heart can improve venous drainage and limit increases in IOP. Limitations of this study include a lack of power in study size, duration of surgical procedures, and no collection of preoperative epidemiologic data for the patients enrolled in the study.

Steep Trendelenburg position is preferred during laparoscopic abdominal procedures, because it allows gravity to pull abdominal contents out of the surgical field. However, the change in position and insufflation of the abdomen both increase pressure in the abdominal cavity, which increases intrathoracic and central venous pressures. Over time, this will increase IOP, limiting venous return and promoting hypoperfusion to the optic nerve. An intervention the surgical and anesthesia team can implement to reduce IOP is to undock the laparoscopic equipment and level the table for a 5-minute period after the patient has been in the steep Trendelenburg position for 4 hours. In a comparison of IOP between patients in continuous steep Trendelenburg position compared with those who were placed in the supine position for a 5-minute period after 4 hours of surgery, the patients who received the 5-minute supine rest had decreased IOP immediately following the rest period and had a faster return to baseline IOP after completion of the surgery than did patients who did not have a changed position intraoperatively.

Presence of chemosis is an indicator to the anesthesia provider that IOP is approaching a critical level. A proposed intervention is to administer an ophthalmic solution of dorzolamide hydrochloride and timolol maleate (Cosopt) once IOP reaches 40 mm Hg. This ophthalmic solution contains a topical anhydrase inhibitor and a topical β-adrenergic receptor blocking agent; 1 mL of the medication contains 20 mg of dorzolamide and 5 mg of timolol.

### Table 4. Intraocular Pressure (IOP) and Time in Steep Trendelenburg (ST) Position

(Data from Molloy, n = 36.)

<table>
<thead>
<tr>
<th>IOP measurement (min)</th>
<th>Patient position</th>
<th>IOP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial</td>
<td>Flat (supine)</td>
<td>14 ± 4</td>
</tr>
<tr>
<td>30</td>
<td>ST</td>
<td>25 ± 8</td>
</tr>
<tr>
<td>60</td>
<td>ST</td>
<td>35 ± 10</td>
</tr>
<tr>
<td>90</td>
<td>ST</td>
<td>35 ± 8</td>
</tr>
<tr>
<td>120</td>
<td>ST</td>
<td>35 ± 10</td>
</tr>
<tr>
<td>Final</td>
<td>Flat</td>
<td>21 ± 6</td>
</tr>
</tbody>
</table>
The components of dorzolamide-timolol decrease elevated IOP by reducing aqueous humor production at the ciliary process. Although the medicine is administered topically, it is still absorbed systemically, and so careful consideration should be given to certain patient populations. The use of dorzolamide-timolol is not recommended in patients with reactive airway disease, asthma, severe chronic obstructive pulmonary disease, sulfa allergy (contains sulfonamide), or atrioventricular block. Caution should be taken in patients with diabetes or hyperthyroidism, as the potential systemic effects of a β-blocker could potentially mask hypoglycemia or thyrotoxicosis. Adverse reactions to dorzolamide-timolol include a bitter or sour taste, blurred vision, and ocular itching. Application of dorzolamide-timolol (Cosopt) during surgical procedures performed with the patient in steep Trendelenburg position has been shown to decrease IOP compared with a study group that did not receive dorzolamide-timolol for treatment of increasing IOP. Treatment with dorzolamide-timolol at any time during the surgical procedure was shown to stop escalating IOP. In the event that IOP starts to increase again, a second dose of dorzolamide-timolol can be given to stall increasing IOP. Because it is not possible in all operating rooms to measure IOP, detection of the presence of chemosis can be an indicative clinical endpoint at which dorzolamide-timolol would be a valuable intervention.

Use of square or circular foam headrests with eye cutouts and a mirror to view the eyes (eg, ProneView, Dupaco Inc) can help to prevent central retinal arterial occlusion associated with direct pressure to the eyes. There are documented reports, however, of goggles used to protect patient’s eyes becoming dislodged and causing direct ocular pressure, resulting in unilateral central retinal arterial occlusion. Roth suggests ensuring that the eyes are properly positioned behind the eye cutouts of the headrest and checked intermittently, every 20 minutes, by palpation or visualization, with subsequent documentation of action to ensure there is not direct external compression to the eyes. In a study by Lee et al., in only 51% of cases of POVL did the anesthesia provider document eye checks.

After an anesthesiologist experienced POVL for himself following a prolonged lumbar spine surgery in the prone Trendelenburg position, he advocated for patient education, highlighting the potential risk of this complication occurring. Lechner recommends disclosing the risk of POVL to each patient undergoing a procedure that has potential risk of POVL occurring. The Anesthesia Patient Safety Foundation highlights that the informed consent process should include discussing the remote risk of blindness due to ischemic optic neuropathy. By clearly stating the risk of vision loss, it also help patients and families understand this is a complication of positioning, and not of the anesthetic itself.

Management of fluids is complicated in a procedure that is associated with substantial blood loss and prolonged anesthetic duration; however, it is recommended by the American Society of Anesthesiologists 2006 Practice Advisory to incorporate colloids along with crystalloid in fluid administration. Use of colloids may help reduce edema formation; however, recent studies have illustrated that colloid administration in the critically ill patient is associated with increased mortality. Risk vs benefit should be analyzed on a case-by-case basis.

Excessive crystalloid infusion can contribute to many deleterious effects, including decreasing hematocrit value and promoting edema formation, and is associated with the adverse event of POVL. Fluid management is complex, and each anesthetic plan should accommodate for patient-specific requirements.

Multiple sources have cited length of anesthetic duration to be a highly correlated factor in the development of POVL. A strategy to reduce length of time a patient is in the prone position for a lengthy multilevel spinal operation would be to stage the procedure. This would require cooperation between the surgeon and the anesthesia team to develop a surgical plan if the dura-
tion of patient position would exceed 6 hours. There are risks that need to be taken into consideration if the decision to stage the procedure is made, including increased risk of infection and potentially creating an unstable spine.6,11

Conclusion
Best practice has not been determined to eliminate the risk of vision loss after surgery. Primary theories surrounding the adverse event include the accumulation of interstitial fluid with subsequent venous congestion and compartment syndrome in the optic canal. Recognizing patients who have an increased risk of POVL, such as surgical time greater than 6 hours, greater than 1 L of blood loss, use of prone or steep Trendelenburg position, excessive fluid replacement, use of the Wilson frame, male gender, or obesity, can help form an anesthetic plan to reduce the risk of vision loss. Interventions that can be implemented to reduce the venous congestion in the optic canal include placing the patient’s head higher than the heart, limiting the amount of crystalloid administration and incorporating colloids, preventing direct pressure to the eyes, a 5-minute supine rest during procedures requiring steep Trendelenburg position, and administration of dorzolamide-timolol to reduce increased IOP. Although the incidence of POVL has decreased in recent years, it is still important to discuss with patients the risk of vision loss after a surgical procedure when risk factors are present.

REFERENCES

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