Published in final edited form as:
Am J Ophthalmol. 2008 April ; 145(4): 604–610. doi:10.1016/j.ajo.2007.09.016.

Perioperative Visual Loss After Nonocular Surgeries

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Patients assume a certain risk of vision loss when undergoing ophthalmic surgery, but awaking blind after elective nonocular surgery is a catastrophic event for the patient, the surgeon and the anesthesiologist. Although relatively rare, perioperative visual loss has warranted a substantial number of publications in the medical literature and has become an important medical-legal issue.

How often will the ophthalmologist see this?

The actual incidence of perioperative visual loss remains elusive. Only one patient suffered persistent post-operative visual loss not related to corneal injury among 60,965 consecutive patients undergoing nonocular surgery at the University of Chicago between 1988 and 1992 (1). In an even larger retrospective study of 501,342 noncardiac surgeries at the Mayo Clinic, only 4 patients (0.0008%) developed vision loss for longer than 30 days without direct surgical trauma to optic or cerebral tissues (2 ischemic optic neuropathies and 2 occipital infarctions) (2). However, the Mayo Clinic study specifically excluded cardiac procedures because of their perceived high risk of multiple causes of perioperative visual loss. Indeed, in a different retrospective study of 27,915 cardiopulmonary bypass (CABG) procedures from the Mayo Clinic, 17 (0.06%) patients were identified with perioperative ischemic optic neuropathy (3), a similar incidence to two other studies of optic neuropathy complicating CABG in which 0.113% (4) and 0.09% (5) of patients were affected. If one specifically looks at the occurrence of visual complications after spine surgeries, the incidence may even be greater. Among a series of 3450 spine surgeries at 3 medical centers from 1985 to 1994, Stevens et al (6) identified seven cases of ophthalmic complications with visual loss, including 2 patients with occipital infarctions (one bilateral), 4 patients with ischemic optic neuropathy, and one patient with a central retinal vein occlusion, for an overall incidence of 0.2%. Similarly, in a study of nearly 225,000 surgeries over a 15-year period, the incidence of perioperative visual loss after spinal surgery was 3 of 3,351 patients (0.09%), a 50-fold higher rate compared with all other nonocular procedures (7). In another retrospective review of 14,102 spine surgeries over 20 years at a single institution (Johns Hopkins), perioperative ischemic optic neuropathy was identified in 4 cases (0.028%) (8).

Where is the injury?

When called to see the patient who awakens from general anesthesia with visual loss, it is paramount first to determine the location of the lesion causing the visual problem, as the etiologies will differ dramatically depending on the site of injury. For example, chiasmal visual

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This work was presented in part at the 2008 Annual Meeting of the North American Neuro-Ophthalmology Society, March 10, 2008.

B. Financial Disclosure: Dr. Newman has received compensation as a medical-legal expert on the topic of perioperative visual loss from both plaintiff and defense attorneys.

loss would most likely be a result of compression by a rapidly expanding pituitary mass, as can be seen with pituitary apoplexy (9), while in other locations, the mechanism is more likely to be ischemic or anoxic.

Anterior Segment

The most commonly reported location of ocular injury during anesthesia is the cornea, most frequently the result of corneal abrasions and or exposure (1). This is usually not a cause of permanent visual loss.

Retina

The most commonly reported retinal cause of visual loss during general anesthesia for nonocular surgery is a central retinal artery occlusion. Central retinal artery occlusion can result from systemic or local arterial embolism, the likely cause when this complication occurs in the setting of cardiac and other vascular surgeries. For example, in one prospective study of 312 patients who underwent CABG, retinal ischemia (cotton wool spots) occurred in 17.3% of patients and retinal emboli in 2.6% (10). However, the mechanism for central retinal artery occlusion in prone position spine surgeries is more often external compression of the globe (11,12). This typically occurs when the head is malpositioned on a headrest. Central retinal artery occlusion, and even ophthalmic artery occlusion, in this setting is almost always unilateral and is often accompanied by other signs of external compression of the periorbital tissues and/or the globe. In the American Society of Anesthesiologists Postoperative Visual Loss Registry's analysis of 93 spine surgery cases with postoperative visual loss (13), 10 patients had central retinal artery occlusions, all unilateral, 7 of whom had additional evidence of peri-ocular trauma, including ipsilateral decreased supraorbital sensation, ptosis, erythema, corneal abrasion, and ophthalmoplegia.

Retrochiasmal Visual Pathways

Visual loss from injury to the retrochiasmal visual pathways will cause either homonymous hemianopia (if unilateral) or cerebral/cortical visual impairment (if bilateral). Pupillary reactions will be normal. Injuries in this region in the perioperative period are almost always ischemic in origin. The most common mechanism is embolic cerebral infarction, usually to the posterior cerebral arteries. Other mechanisms include watershed ischemia at the borderzones of the posterior and middle cerebral arteries, cortical necrosis from global anoxia, and cervical artery dissection from neck surgery or neck trauma during intubation. The overall risk of stroke after general, noncardiac procedures is very low (0.08–0.7%), but resection of head and neck tumors and cardiac and vascular surgeries are associated with higher risks (for example, the risk after isolated CABG is 1.4–3.8%, after carotid endarterectomy 5.5–6.1%, after combined CABG and valve surgery 7.4%, and after valve surgery 4.8–8.8%) (14). Most perioperative strokes are infarctions and are embolic in origin (14). Although the general proportion of embolic cerebral infarctions that affect the posterior cerebral artery territory and would give visual field defects is estimated at 9%, the specific relative incidence of visual loss from retrochiasmal infarction in the perioperative period is unknown.

Optic Nerve

The most common site of permanent injury to the visual pathways in the setting of general anesthesia for nonocular surgery is the optic nerves, and the most often presumed mechanism of injury in this location is ischemia. Anterior ischemic optic neuropathy (AION) will manifest disc edema acutely, while posterior ischemic optic neuropathy (PION) patients will have a normal acute ocular fundoscopic appearance. However, in both settings, pupillary reactivity will be abnormal. Ischemic optic neuropathies are now the most frequently reported conditions associated with permanent perioperative visual loss, and the most common surgical setting in

which they occur is spine surgery in the prone position (15,16). Amongst cardiac procedures, AION appears to be more common than PION (3,5). Amongst spinal surgeries and radical neck dissections, PION predominates, especially among prolonged spine surgeries performed in the prone position (13,15–17). Reports of ischemic optic neuropathy in this setting appear to be increasing (13).

Ischemic optic neuropathies are most common

The literature on ischemic optic neuropathy in the perioperative period consists of primarily case reports, small case series and reviews (3–8,10,18–27), one retrospective case-control study (28), 2 recent important systematic reviews (15,16), one interim report from a large multi-institutional database (13), and a critical review and evaluation of the availability and strength of evidence (17).

In 1999, in response to concerns that perioperative visual loss seemed to be increasing, particularly for spine surgery, the American Society of Anesthesiologists (ASA) established the ASA Postoperative Visual Loss (POVL) Registry to collect detailed information on cases of POVL occurring after nonocular surgery. The purpose of the database is to identify the risk factors associated with perioperative visual loss, ideally in order to develop preventative measures. The database consists of voluntary, anonymously reported cases of patients with visual loss within 7 days of non-ocular surgery, using a standardized form which includes detailed demographic, historical, examination, and intraoperative information (see www.asaclosedclaims.org). As of June 2005, 131 cases of POVL had been reported to the registry, 95 of them spine cases, 12 cardiac cases, 6 major vascular cases, 5 orthopedic cases, and 13 miscellaneous cases (13). An interim report of 93 cases of POVL associated with spine surgery was published in 2006 (13), including 83 cases of ischemic optic neuropathy and 10 cases of central retinal artery occlusion. Among the 83 optic neuropathy cases, 67% were PION, 23% AION, and 10% unspecified ischemic optic neuropathy.

Perioperative ischemic optic neuropathy has been reported after a wide variety of nonocular surgeries, including spinal surgery, cardiac surgery, radical neck dissection, and vascular, abdominal and orthopedic procedures (13,16). In the ASA Registry, 73% of cases occurred in the setting of spine surgery. In a review of only perioperative PION cases, (16), 54.2% of procedures were spine surgeries, and 13.3% were radical neck dissections.

The mean age of perioperative ischemic optic neuropathy patients is approximately 50 (13, 15,16), with a wide range of 5 to 81 years (8,13,15,16,23,24). Men predominate among the spine cases, even though spinal fusion procedures are performed fairly equivalently on men and women (13).). Additionally, spine patients are in general younger and more healthy than their CABG counterparts. Indeed, although at least one vascular risk factor, including obesity, was noted in 82% of the patients in the ASA Registry, 64% of these patients were relatively healthy, with ASA physical status I or II (13). Similarly, in a review of perioperative ischemic optic neuropathy among spine surgery patients, nearly half of the patients had no vascular risk factors (15). In a case-control study of patients with ischemic optic neuropathy after CABG (3), there was no difference in the number of atherosclerotic risk factors in patients who developed optic neuropathy versus those who did not. Although, intuitively, the presence of vascular risk factors would seem to be a logical predisposing risk factor for perioperative ischemic optic neuropathy, the occurrence of this complication in children, teenagers and in relatively healthy young adults weakens this proposed association, at least in some cases.

Almost all patients with perioperative ischemic optic neuropathy have vision loss upon gaining sufficient consciousness after the surgical procedure (13,15,16). Most of the cases in which there is a “delay” in visual loss likely represent a delay in either recognition or reporting of the vision loss, rather than a true window of normal vision after surgery. True exceptions with a

definite onset of visual loss days after the surgery are nearly always AION cases, particularly after CABG, reminiscent of the delay in AION onset which has been classically reported in patients with massive blood loss (29). The role of optic disc morphology and cup-to-disc ratio in these cases remains unknown.

Most cases of perioperative ischemic optic neuropathy have bilateral simultaneous involvement, usually with very poor visual function (13,16,27). Count fingers vision or worse was documented in 76% of eyes in a review of perioperative PION (16), and no light perception was reported in 54% of eyes in that series and in 46% of eyes of spine-related ischemic optic neuropathy cases from the ASA Registry (13). Some degree of visual recovery occurred in 40% of patients in the PION review (16), although this was usually in patients with better visual function initially. Hand motion or worse final vision still persisted in 55% of patients.

Imaging usually is not very helpful in these patients, except to rule out the occasional surprising alternative diagnosis such as pituitary apoplexy or bilateral occipital infarctions. There are two reports of patients with PION after CABG in whom magnetic resonance imaging demonstrated changes within the optic nerves themselves (30,31). It is certainly possible that these findings would be more frequent if designated orbital views with diffusion-weighted sequences were performed acutely.

Spine surgeries, especially in the prone position, figure prominently among the settings in which perioperative ischemic optic neuropathy occurs. In one review of the literature of ischemic optic neuropathy cases following prone spine surgery (15), most procedures were lumbar fusions, although both cervical and thoracic procedures have been reported. Mean operative time exceeded 450 minutes. In the ASA Registry (13), 89% of the spine cases with ischemic optic neuropathy involved fusion and/or instrumentation on more than one vertebral level in the thoracic, lumbar or sacral spine, with 39% of patients having had previous spine surgery. All of the ASA Registry patients but two were positioned prone for at least a portion of the procedure. The mean anesthetic duration for these patients was 9.8 hours, with a range of 3.9 to >14 hours, and 94% of cases lasted 6 hours or longer. The mean prone position duration was 7.7 hours.

Multiple intraoperative factors have been proposed as “causal”, “risk factors”, or “associated” with perioperative ischemic optic neuropathy. They include hypotension, relative hypotension, anemia, blood loss, hypoxia, hemodilution, hypovolemia, infusion of large amounts of crystalloid, use of vasoconstricting agents, head position either above or below the heart, elevated venous pressure, elevated intraocular pressure, ocular globe compression, and an individual susceptibility (perhaps anatomically or physiologically related to the blood supply to or from the optic nerves). All of these factors could theoretically play a role in the development of ischemic optic neuropathy, with the exception of ocular globe compression and elevated intraocular pressure; globe compression and elevated intraocular pressure could be possible causes of central retinal artery occlusion, unlikely causes of AION and most certainly not the cause of PION. However, there are many difficulties in determining the exact cause of perioperative ischemic optic neuropathy. First of all, the causes and contributing factors, and their relative importance, may vary depending on the procedure (for example, the causes in patients undergoing CABG versus those undergoing spine procedures very likely differ). Similarly, the risk factors in any given patient may be different. It is likely that multiple factors need to be present in combination for visual loss to occur. Finally, many of these factors may be epiphenomena of the procedures themselves and not necessarily causal. For example, post-operative periorbital facial edema is common in patients undergoing prolonged procedures in the prone position, but there is no pathophysiologic mechanism by which facial edema can cause perioperative ischemic optic neuropathy (17).

Some degree of perioperative anemia and hypotension have been reported in most, but not all, cases of perioperative ischemic optic neuropathy in the literature. However, as Buono and Foroozan point out (16), anemia and hypotension are common in the perioperative course of many surgical procedures, especially CABG and spinal surgeries, and very few patients develop perioperative ischemic optic neuropathy, strongly arguing against their exclusive role. Indeed, in the only case-control study of this topic (28), perioperative hematocrit and blood pressure were no different between patients who experienced postoperative visual loss after spine surgery and control patients who did not. Among the ASA registry spine optic neuropathy cases, the lowest mean hematocrit was 26 ± 5%, with 17% of cases having a nadir hematocrit of 30% or better (13). Furthermore, among these patients, blood pressure varied widely, with 33% of cases in which the lowest systolic blood pressures were greater than 90 mm, and only 20% of cases in which the lowest systolic blood pressure was 80 mm or less. Thirty-four percent of cases had the lowest mean arterial pressure or systolic blood pressure 40% or more below baseline.

The case-control study by Myers and colleagues (28) retrospectively compared patients with perioperative visual loss after spine surgery with control spine surgery patients without visual loss. Although this study also included patients with visual loss from retinal vascular occlusions and cortical blindness, ischemic optic neuropathy patients predominated. The only differences between the POVL patients and controls were a longer duration of surgery in the prone position and a larger estimated blood loss. In the ASA Registry spine patients (13), an estimated blood loss of 1,000 ml or greater occurred in 82% of cases, and anesthetic duration of 6 hours or longer was present in 94% of cases. One of these 2 factors was present in all but three ischemic optic neuropathy cases.

One notable clinical fact is that perioperative ischemic optic neuropathy almost without exception occurs without any other accompanying evidence of vascular injury in other critical organs, including the brain, even in patients with preexisting atherosclerosis, diabetes and hypertension. This again emphasizes that the optic nerve of some patients, and particularly aspects of its vasculature, may be uniquely vulnerable to hemodynamic perturbations in the prone position.

The literature contains only three histopathologic studies of perioperative ischemic optic neuropathy (32–34), notably none of which were performed in patients who had been in the prone position or who underwent spine surgery. Johnson and colleagues (32) reported a clinical-pathological case of a 59 year old woman with chronic anemia who developed bilateral PION after an exploratory laparotomy (in the supine position) complicated by severe intraoperative hemorrhage and hypotension requiring vasopressors. Nine days later she died of sepsis, and neuropathologic examination revealed bilateral symmetric fusiform swelling and infarction with central intraparenchymal hemorrhage of the intraorbital portion of the optic nerves, with no other central nervous system abnormalities. Similarly, in a 67 year old man who suffered PION after radical neck dissection “complicated by intraoperative hypotension and anemia” who died from sepsis 14 days after the procedure, there was hemorrhagic infarction of the distal and proximal ends of the intraorbital optic nerves, involving the central portion of the nerves, but sparing the peripheral fibers (33). Conversely, in a 48 year old man with bilateral PION after bilateral neck dissection, autopsy one year after visual loss revealed complete loss of axons of the most severely affected optic nerve and loss of the peripheral axons with sparing of the central axons of the optic nerve on the side of partially recovered vision and a constricted visual field (34).

Given the multitude of potential “causative” factors that have been proposed for perioperative ischemic optic neuropathy, it is not surprising that there have been many proposed theories on the pathogenesis of the disorder. One theory may not apply to all procedures and to all patients.

Most theories start from the reasonable assumption that the disorder is vascular in etiology, but the lack of histopathologic studies in relevant patients precludes our ability to even definitively make this statement, and certainly leaves open the question of whether (especially in PION cases) the primary vascular insult is arterial or venous. Ischemia can result from poor oxygen content within the circulating blood, reduced arterial perfusion pressure, or increased resistance to blood flow. Poor oxygen content could result from a reduction in hemoglobin levels, hemodilution, blood loss with crystalloid replacement, and hypoxia, but these perturbations would be expected to affect other organs and tissues throughout the body, a very rare occurrence in perioperative ischemic optic neuropathy. Reduced arterial perfusion pressure could occur with systemic hypotension, but this too would be expected to affect other tissues besides the optic nerves. Other causes of reduced arterial perfusion pressure to the optic nerve could include head-up position, local compression of the small pial arteries that supply the posterior optic nerve because of elevated orbital venous pressures related to the prone position or from fluid overload and resultant dependant orbital edema, or direct vasoconstriction of these small vessels by vasopressors (administered, ironically, to increase systemic blood pressure and thereby increase arterial perfusion pressure). Finally, the prone position and head-down positioning could increase venous pressure and resistance to local blood flow, as would ligation of the jugular veins as performed in radical neck dissections, causing venous infarction. In cases of perioperative AION, the pre-existing optic nerve morphology and presence of a “disc at risk” may predispose a patient to the disorder. This is one factor that could be assessed pre-operatively, but thousands of patients would need to be screened to potentially prevent one case of visual loss. For PION, although a similar individualized optic nerve vascular feature may exist in the posterior optic nerves of susceptible patients, preoperative identification of these patients is currently impossible. The nature of this patient-specific susceptibility is unknown, although both anatomic and physiologic mechanisms have been proposed, including a vascular watershed region in the posterior optic nerves and defective autoregulation (13,15,16,32).

There is no proven effective treatment for perioperative ischemic optic neuropathy. Proposed therapies have included correction of hemodynamic derangements, systemic corticosteroids, anti-platelet therapy, and lowering of the intraocular pressure (6,16,35). Ideally, prevention of perioperative ischemic optic neuropathy would be the goal. Unfortunately, we can not specifically predict preoperatively which patient characteristics make an individual patient susceptible to this injury. Furthermore, since the pathogenesis of this disorder remains unclear, preventive measures remain elusive. Given the rarity of the complication, global recommendations to alter hemodynamic parameters in all patients have the potential to cause more harm than good. For example, elevating the intraoperative blood pressure in spine procedures may increase bleeding into the operative site, thereby increasing the amount of blood loss and increasing the length of the procedure, the two factors found to be most significantly related to perioperative visual loss (13,28). Similarly, the use of vasopressors to elevate the blood pressure may cause local constriction of small blood vessels, including those supplying the optic nerves (36). Blood transfusion carries its own set of risks. The American Society of Anesthesiologists in general has recommended that transfusion is rarely indicated when the intraoperative hemoglobin is greater than 10, and almost always indicated when the hemoglobin is less than 6, but they caution that this should be individualized for each patient (37).

The ASA Perioperative Visual Loss Practice Advisory

In 2005, the American Society of Anesthesiologists appointed a Perioperative Visual Loss Task Force of 12 members to (1) review and assess the currently available scientific literature, (2) obtain expert consensus and public opinion, and (3) develop a practice advisory. The Task Force members consisted of anesthesiologists, neuro-ophthalmologists, spine surgeons (both

orthopedic and neurosurgical), and methodologists. In 2006, a Practice Advisory was generated on the topic (17). A "practice advisory" is a systematically developed report that is intended to assist decision making in areas of patient care, but it is not supported by the scientific literature to the same degree as a "standard" or "guideline" because of the lack of sufficient numbers of adequately controlled studies. Hence, a Practice Advisory is not intended to dictate a standard of care. In this situation, because of the rarity of the complication and the likely heterogeneous nature of the cases reported, the Task Force decided to focus on the perioperative management of patients undergoing spine procedures in the prone position under general anesthesia, the group at seemingly highest risk. The Task Force came to the following consensus conclusions regarding this group of patients:

1. There is a subset of patients who undergo spine procedures while they are positioned prone and receiving general anesthesia that has an increased risk for development of perioperative visual loss. This subset includes patients who are anticipated preoperatively to undergo procedures that are prolonged, have substantial blood loss, or both (so-called "high-risk patients").
2. Consider informing high-risk patients that there is a small, unpredictable risk of perioperative visual loss.
3. The use of deliberate hypotensive techniques during spine surgery has not been shown to be associated with the development of perioperative visual loss.
4. Colloids should be used along with crystalloids to maintain intravascular volume in patients who have substantial blood loss.
5. At this time, there is no apparent transfusion threshold that would eliminate the risk of perioperative visual loss related to anemia.
6. High-risk patients should be positioned so that their heads are level with or higher than the heart when possible. In addition, their heads should be maintained in a neutral forward position (e.g., without significant neck flexion, extension, lateral flexion, or rotation) when possible.
7. Consideration should be given to the use of staged spine procedures in high-risk patients.

Obviously, much further investigation needs to be done in order for us to fully understand the risk factors and mechanisms of optic nerve damage in the perioperative setting, especially in the prone-positioned patient. A major step would be the development of a valid animal model, particularly for PION in the setting of elevated orbital venous pressures, as likely occurs in radical neck dissection and in long prone procedures. A prospective case-control study would be unfeasible given the rarity of the complication, even among so-called "high risk" patients. However, a well-designed and well-performed retrospective case-control study using the standardized and systematically gathered patient information from the ASA Registry would be a logical next step in understanding this devastating visual complication of often routine elective surgery.

Approach to the patient with perioperative visual loss

So what is the ophthalmologist's role then in the management of the patient who awakens from general anesthesia complaing of visual loss? Clearly, there is no standard management that can be recommended for every patient, but the ASA Practice Advisory (17) suggests that in the high-risk spine surgery patient, vision should be assessed when the patient becomes alert (e.g., in the recovery room, intensive care unit, or nursing floor). If there is concern regarding potential visual loss, an urgent ophthalmologic consultation should be obtained to determine its cause. If an obvious ocular cause such as globe injury or central retinal artery occlusion is

not apparent, then urgent neuroimaging should be obtained, preferably MRI with gadolinium and stroke protocol techniques, to assess for intracranial pathology such as pituitary apoplexy or occipital infarction. If imaging is unrevealing, the likely cause of visual loss is ischemic optic neuropathy. Additional management may include optimizing hemoglobin levels, hemodynamic status, and arterial oxygenation. Careful documentation in the chart is essential and statements regarding causation should be made only when the evidence is clear.

Acknowledgments

A. Funding/Support: This study was supported in part by a departmental grant (Department of Ophthalmology) from Research to Prevent Blindness, Inc, New York, New York, and by core grant P30-EY06360 (Department of Ophthalmology) from the National Institute of Health, Bethesda, Maryland. Dr Newman is a recipient of a Research to Prevent Blindness Lew R. Wasserman Merit Award.

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