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REVIEW ARTICLE

Perioperative visual loss after nonocular surgery

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Abstract Although rare, a change in visual acuity after surgery for nonocular procedures has devastating consequences. Increased recognition and discussion of this complication is reported in recent literature, most notably following spinal and cardiac surgery. Various pathologies may be responsible for perioperative visual loss (POVL), including ischemic optic neuropathy, retinal vascular occlusion, and cortical blindness. Here we review the incidence of the problem, the anatomy and physiology of the ocular circulation, variants of POVL, and proposed predisposing factors. Potential perioperative methods to prevent this complication are discussed, and suggested treatment modalities are presented.

Keywords Visual loss · Complications · Blood loss · Hypotension · Ischemic optic neuropathy · Retinal vascular occlusion

Introduction

Although rare, a change in visual acuity after surgery for nonocular procedures has devastating long-term consequences. The first report of perioperative visual loss was in 1948 by Slocum et al. [1] involving a patient in the prone position during spinal surgery. The suspected etiology was improper positioning of the head on a headrest with direct pressure on the globe. The incidence has varied significantly from study to study depending on the methods used for detection, the patient population involved, and surgical procedures [2–12] (Table 1). Although there is significant variation in the incidence of perioperative visual loss (0.056–1.3 %) reported in these studies, the highest risk is during spinal and cardiac surgery.

Various pathologic processes have been identified that may be responsible for POVL, including ischemic optic neuropathy (ION), central retinal artery occlusion (CRAO), and cortical blindness. This manuscript reviews the anatomy and physiology of the eye and visual pathways with an emphasis on the regulation of blood flow, presents the proposed etiologies of POVL, and discusses preventive and treatment strategies.

Anatomy and physiology of the eye and visual pathways

Anatomy

Light and visual inputs are translated into neural input by the rods and cones at the back of the retina. Axons of the optic nerve originate from the retinal ganglion cells. The optic nerve passes through the scleral canal and connects in the lateral geniculate nuclei. The majority of axons (optic radiations) that transmit visual signals terminate in the visual cortex of the brain, mostly in the occipital and parietal lobes [16]. Pathology affecting the structure of the eye itself or any of the components of the visual pathway may result in POVL. Central retinal arterial occlusion occurs at the optic disc in the retina. ION affects the optic nerves. Cortical blindness results from damage (ischemic or embolic) to the visual cortex or the optic radiations.

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Table 1 Studies investigating the incidence of perioperative visual loss

References	Type of surgery, cohort size, and study design	Incidence of postoperative visual loss	General findings
Roth et al. [3]	Retrospective review of 60,965 patients for nonocular surgery (single institution over 4.5 years)	Only one case of permanent visual loss. Ocular injuries noted in 34 (0.056 %) patients, the majority of which were corneal abrasions	Risk factors for ocular injury included long surgical procedures, lateral or prone positioning, head and neck procedures, general anesthesia, and surgery on a Monday. Specific cause identified in only 21 % of cases
Warner et al. [4]	Retrospective review of 501,342 anesthetics over a 12-year period	POVL lasting more than 30 days identified in 4 patients for an incidence of 0.0008 %.	405 cases of new-onset vision loss or visual changes in 410,189 patients who underwent 501,342 anesthetics and who survived at least 30 days. Two hundred sixteen patients regained full vision or acuity within 30 days. Of the 189 patients who developed vision deficits for more than 30 days, 185 underwent ophthalmologic or neurologic procedures in which ocular or cerebral tissues were surgically damaged or resected. The authors concluded that the incidence of POVL was very low.
Nuttall et al. [5].	Retrospective review of 27,915 patients undergoing cardiac surgery	ION identified in 17 (0.06 %) patients	Case-matched controls to evaluate risk factors. Risk factors identified included minimum postoperative hemoglobin value and the presence of atherosclerotic vascular disease. In ION patients, 13 of 17 (76.5 %) had a postoperative hemoglobin $\leq$8.5 g/dl compared with 14 of 34 controls (41.2 %)
Kalyani et al. [6]	Retrospective review of a 9-year period involving 9,701 patients for cardiac surgery	Visual disturbances, including ION, identified in 11 (0.11 %) patients	Risk factors included associated vascular disease and changes in hemoglobin levels. Hemoglobin changes included both absolute and relative decrease
Chang and Miller [7]	Retrospective review of 14.102 cases over a 20-year period	ION identified in 4 (0.028 %) patients	One patient spontaneously recovered. Risk factors included intraoperative hypotension and anemia
Lee et al. [8]	Review of cases from the ASA Postoperative Visual Loss Registry and Closed Claims Project over a 6-year period	POVL noted in 93 patients. Bilateral issues noted in 55 patients	Case-matched controls to evaluate risk factors. ION cases had significantly longer anesthetic duration, blood loss, and use of Mayfield pins. Bilateral involvement was more common in ION cases. Blood loss >1,000 ml and anesthetic duration >6 h was present in 96 % of cases
Patil et al. [9]	Retrospective review of a 9-year period involving 4,728,815 patients from the NIS	POVL incidence reported as 0.094 %	Highest incidence noted in spinal surgery for scoliosis (0.28 %) and posterior lumbar fusion (0.14 %). Patient age noted to be a factor, with an increased risk of 5.8 in patients <18 years of age and 3.2 in patients >84 years of age
Shen et al. [10]	Retrospective review of 10-year period involving 5.6 million patients from the NIS	POVL incidence of 0.09 % in cardiac surgery and 0.03 % in spinal fusion	Patients <18 years were reported to have the highest risk for POVL because of a higher risk for CB; patients >50 years were at greater risk of developing ION and CRAO. Other positive predictors for the occurrence of POVL were male gender, Charlson index, anemia, and blood transfusion

Table 1 continued

References	Type of surgery, cohort size, and study design	Incidence of postoperative visual loss	General findings
Holy et al. [11]	Retrospective review of a 7-year period involving 126,666 cases at a single institution	POVL incidence of 17 of 126,666 (0.013 %) cases	Case-matched controls to evaluate risk factors. No difference noted in perioperative variables among the two groups. In particular, no difference noted in hemodynamic variables
The Postoperative Visual Loss Study Group [12]	Case-matched control of 80 patients from the ASA POVL registry	80 patients with POVL vs. 315 without	After multivariate analysis, risk factors for ION after spinal fusion surgery included male sex (OR 2.53, 95 % CI 1.35–4.91, $P = 0.005$), obesity (OR 2.83, 95 % CI 1.52–5.39, $P = 0.001$), Wilson frame use (OR 4.30, 95 % CI 2.13–8.75, $P < 0.001$), anesthesia duration (OR per 1 h = 1.39, 95 % CI 1.22–1.58, $P < 0.001$), estimated blood loss (OR per 1 L = 1.34, 95 % CI 1.13–1.61, $P = 0.001$), and colloid as percent of nonblood replacement (OR per 5 % = 0.67, 95 % CI 0.52–0.82, $P < 0.001$). After cross-validation, AUC = 0.85, sensitivity = 0.79, and specificity = 0.82

CPB cardiopulmonary bypass, *ION* ischemic optic neuropathy, *POVL* perioperative visual loss, *NIS* National Inpatient Sample, *CB* cortical blindness, *CRAO* central retinal artery occlusion, *ASA* American Society of Anesthesiologists, *OR* odds ratio, *CI* confidence interval, *AUC* area under the curve

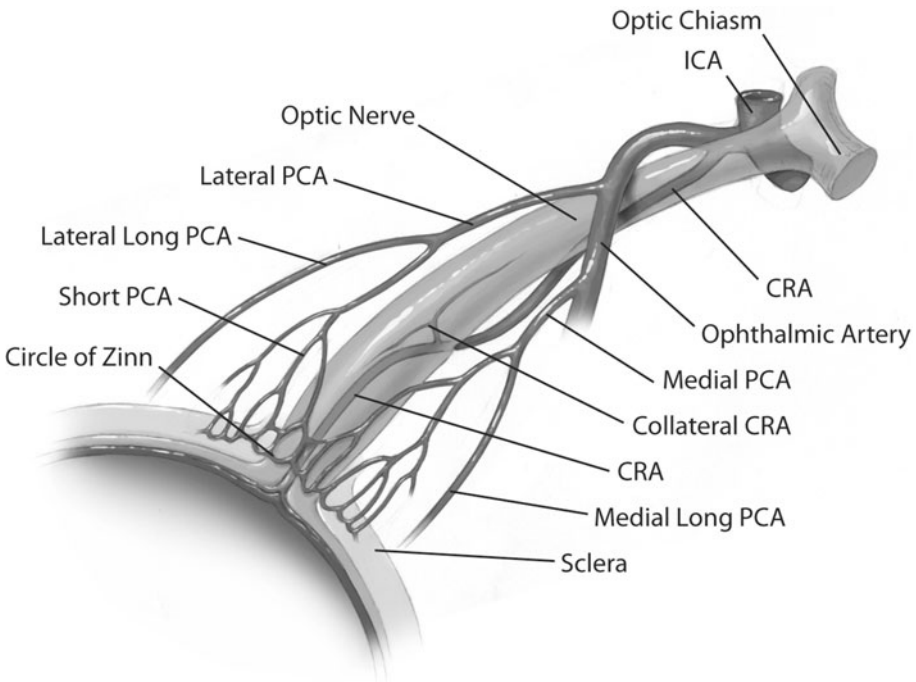

Fig. 1 Blood flow to the eye. The majority of the blood supply is provided by the ophthalmic artery, a branch of the internal carotid artery (ICA). *PCA* posterior ciliary artery, *CRA* central retinal artery

Vascular supply

Of prime importance in discussing POVL is to understand the blood supply to the eye and visual pathways (Fig. 1) [13–15]. The ophthalmic artery, which originates from the internal carotid artery, provides the majority of blood supply to the eye and the associated visual pathways, including retina, globe, and optic nerves. The central retinal artery, a branch of the ophthalmic artery, perfuses the inner layers of the retina. Venous drainage is provided by veins that correspond to and course with the arterial supply outlined above. Although POVL is generally the result of

pathology on the arterial side, various pathologies may affect venous drainage, resulting in thrombosis of the single central retinal vein. As the globe is a closed space, in a similar fashion as the brain, perfusion of the eye can be thought of as the difference between mean arterial pressure (MAP) and intraocular pressure (IOP), the difference being known as retinal perfusion pressure [13, 14].

Physiology of the eye, ocular blood flow, and intraocular pressure

There are limited data regarding the compensatory physiologic responses of this vasculature and vascular bed to changes in perfusion pressure, arterial blood gas tensions, including partial pressure of carbon dioxide in arterial blood ($PaCO_2$), and hemoglobin. The occurrence of blindness related to ION without damage to the central nervous system (CNS) suggests that different mechanisms may regulate cerebral (CBF) and optic nerve (ONBF) blood flow [16]. Although the compensatory mechanisms that control ONBF are similar to those of CBF, they are less efficient in maintaining flow, thereby placing the optic nerve at risk of ischemia during periods of hypotension or anemia.

In addition to changes in MAP, CBF can be altered by changes in $PaCO_2$. Hyperventilation with a decrease in $PaCO_2$ decreases CBF. Although used as a therapeutic maneuver to transiently decrease ICP in patients with intracranial hypertension, prolonged hyperventilation is detrimental both to the brain and the eye [17, 18]. An additional factor that may impact ONBF and oxygen delivery is an alteration in IOP [19, 20]. IOP is higher in the patient in the prone position compared to the sitting position. It increases when moving from the reverse Trendelenburg position to horizontal and then to the Trendelenburg position [19, 20].

The functions required for normal vision comprise a complex process requiring optimum IOP, adequate blood and oxygen supply, with efficient venous blood drainage combined with normal functioning of the cornea, pupil, lens, retina, optic nerve, and cerebral cortex [21, 22]. Although the CNS generally maintains CBF during periods of hypotension and anemia, the same compensatory mechanisms do not appear to be in place to regulate ONBF.

Etiology of POVL

The primary etiologic events responsible for POVL include: (1) direct injury to the visual apparatus, (2) occlusion of the retinal vessels resulting in interruption of the arterial supply, (3) ION, and (4) cortical blindness.

Corneal abrasion and scleral injury

Direct damage to the ocular apparatus (cornea, sclera and/or globe) remains the most common and, fortunately, generally treatable and reversible cause of POVL [23, 24]. In the majority of cases, the key is prevention by using effective lubrication and taping of the eyelid to ensure it completely covers the cornea. Additional causes of corneal damage during the perioperative period include inadvertent and direct contact of the eye with cleaning liquids when anesthetic masks are washed and reused or agents used for surgical skin preparation.

Interruption of the retinal arterial supply

This process results most commonly from increased IOP due to external pressure during positioning. IOP can also increase from processes within the globe, including retrobulbar hemorrhage. Thrombotic or embolic processes may result in occlusive disease of the retinal vasculature, including the ophthalmic and central retinal artery. Retinal emboli occur most commonly during open cardiac procedures, the incidence being dependent upon various factors, including cardiopulmonary bypass technique and oxygenator type [25, 26]. Hypotension is an uncommon cause of this type of POVL. Although involvement is more commonly the result of pathology on the arterial side, various pathologies may also affect venous drainage, resulting in thrombosis of the single central retinal vein and subsequent blindness.

When considering central retinal artery occlusion, the process may involve the entire artery after it braches from the ophthalmic artery or a branch from the retinal artery (branch retinal artery occlusion). Central retinal artery occlusion results in ischemia of the entire retina, resulting in complete and generally irreversible loss of vision. Signs and symptoms of central retinal artery occlusion include unilateral loss of vision, the lack of light perception, an afferent pupillary defect, and edema formation of the periorbital area, eyelid, and sclera (chemosis) [27]. If there is sparing of the cilioretinal artery, there may be some sparing of visual function. Imaging studies show only proptosis and edema, with swelling of the extraocular muscles. Funduscopic examination reveals macular and retinal edema, a cherry-red spot, and vasculature attenuation. The classically described cherry-red spot results from the white appearance of retinal ganglion cells that swell due to ischemia, with the foveal center (center of the macula) appearing red because there are no ganglion cells to obscure the underlying retinal pigment epithelium. The presentation and clinical symptomatology of branch retinal artery occlusion are similar, although visual loss may be partial depending on the areas of the retina that are

affected. Etiologic mechanisms include primarily embolic phenomenon or vasospasm. The right eye is affected more commonly than the left eye, which probably reflects the greater possibility of emboli traveling to the right carotid artery.

In general, treatment regimens for central retinal artery occlusion and branch retinal artery occlusion have been ineffective. Ischemic changes followed by visual loss occur in as few as 20 min in rodents and approximately 100 min in primates [28, 29]. The potential for recovery is poor, with approximately 60 % of patients suffering permanent blindness and <25 % recovering useful vision [30]. Potential therapeutic interventions aimed at lowering IOP include ocular massage and administration of acetazolamide or osmotic diuretic agents [30]. Other potential therapeutic maneuvers include induced hypercarbia, topical hypothermia, locally applied thrombolytic agents, and hyperbaric oxygen (HBO) therapy [31].

Ischemic optic neuropathy

Ischemic optic neuropathy represents the most frequently reported condition associated with permanent POVL [8]. It is the result of any process that leads to inadequate delivery of oxygen to the optic nerves, including decreased MAP, vasculature vasoconstriction (related to hypocarbia), or factors affecting oxygen delivery (decreased cardiac output, anemia, low oxygen saturation) [32]. When compared with central retinal artery occlusion or branch retinal artery occlusion, in which the pathology is located in the retina, ION pathology is isolated to the optic nerve. ION can be broadly divided into anterior ischemic optic neuropathy (AION) and posterior ischemic optic neuropathy (PION), involving the damage to the optic nerve head or the remainder of the optic nerve, respectively [32]. With AION, funduscopic examination reveals edema of the optic disk, whereas the disk appears normal in PION. Clinical signs and symptoms include painless visual loss or decreased acuity, an afferent pupillary defect (loss of light reflex), and no light perception.

The site of the ischemia between the two entities may also vary. AION involves the region supplied by the branches of the short posterior ciliary arteries near the globe itself. PION involves the vascular supply of the optic nerve between the globe and the midpoint of the orbital portion of the optic nerve at the entry point of the central retinal artery. There may be slight variations in etiologic factors of these two entities, as PION is more commonly seen following spinal surgery, whereas AION is reported more commonly following cardiac surgery [33, 34]. In addition to spine and cardiac surgery, ION has been reported after a variety of other surgical procedures, including major vascular procedures, nonspine orthopedic procedures, radical neck dissection, sinus surgery, liposuction, prostatectomy, and obstetric and Cesarean section. Both surgery- and patient-related factors are assumed to be potential risks for ION. Surgery-related conditions include the prone position, procedure duration, amount of blood lost, and the need for intraoperative blood pressure support with vasoactive agents. Pre-existing patient-related comorbid conditions that may predispose to ION include conditions that predispose to atherosclerotic disease, including hypertension, diabetes, hyperlipidemia, tobacco use, and obesity. Given the rare occurrence in a small subset of patients without predisposing factors, it has been suggested that specific anatomic variations of the optic nerve and its vascular supply—such as abnormal ONBF autoregulation, variations in optic nerve abnormal blood supply anatomy, and a low cup-to-disc ratio—may also exist [35, 36].

As with other causes of POVL, the lack of prospective data hinders the ability to identify specific risk factors, preventative maneuvers, and treatment interventions. In response to such issues, the Postoperative Visual Loss (POVL) registry was established in 1999 by the American Society of Anesthesiologists (ASA), with an initial report published in 2006 [8]. Of the 93 cases, the majority (83) were attributed to ION, with the remaining ten assumed to be due to central retinal artery occlusion. All except two patients had been positioned prone for some part of the surgical procedure. Blood loss >1,000 ml or anesthetic duration >6 h was present in 96 % of these cases.

Using a case–control study design with 315 patients matched to 80 ION patients, the POVL Study Group attempted to determine additional factors that might be associated with ION [12]. The percent of colloid used for nonblood replacement was inversely related to the odds ratio (OR) for ION. Physiologic tenets regarding fluid resuscitation combined with the findings of the above-mentioned study have led the Practice Advisory on Perioperative Blindness of the ASA to recommend the use of colloids during prolonged spinal surgery [37]. The amount of fluid administered is suggested as a possible causative factor in ION. Excessive fluid resuscitation using crystalloid is suggested as a causative factor in the 2006 publication from the ASA's POVL registry, with an average of 9.7 L of crystalloid being administered intraoperatively to patients who developed ION [8]. Excessive volume resuscitation resulting in hypervolemia may alter the venous outflow parameters of the orbit, resulting in orbital compartment syndrome. These concerns have led to the suggestion for central venous pressure monitoring for prolonged procedures in which large-volume resuscitation may be required (see below) [37].

Another potential causative factor identified for ION is intraoperative hypotension [38–40]. However, given the

duration of the cases involved, blood loss that may occur, resuscitation required, and presence of comorbid patient conditions, it may be impossible to separate hemodynamic instability from other etiologic factors. Myers et al. [41] noted no difference in the degree of hypotension and hemoglobin values between case-matched control patients and those who developed ION following spinal fusion. The 2006 report from the ASAs' POVL registry failed to find any definitive causal link between hypotension and ION, although many patients did experience intraoperative hypotension [8].

Given the relationship between oxygen delivery and hemoglobin values, there may obviously be a causal relationship between anemia, ION, and POVL. Although Myers et al. [41] reported greater intraoperative blood loss in patients with POVL after spine surgery, there was no difference in the lowest recorded hematocrit values between groups. Given increasing concerns regarding the impact of allogeneic blood use on postoperative outcome, clinical practice guidelines for surgery suggest that a hemoglobin $\geq$8 g/dl is acceptable for the majority of patients [42]. The ASA Practice Advisory Group contends that there is no transfusion threshold that could be expected to eliminate the development of POVL.

Cortical blindness

Cortical blindness results from a stroke in the parietal–occipital areas of the cortex responsible for reception and integration of visual input. This results from hypoxic–ischemic events, including hypoperfusion injuries, and thrombotic and embolic events. In rare cases, these events have been noted in patients with congenital heart disease, including patent foramen ovale, allowing for right-to-left shunting with paradoxical emboli. Clinical examination of the patient reveals the painless loss of visual acuity with an intact pupillary response to light—the latter indicating that the lesion is distal to the optic chiasm and the normal pathways of the light reflex. Funduscopic examination is unremarkable. Radiologic examination of the CNS with computed tomography (CT) or magnetic resonance imaging (MRI) reveals the area of infarct.

Conclusion, including treatment and preventative measures

Perioperative visual loss remains a devastating, albeit rare, complication following various surgical procedures. It is noted most frequently following cardiac and spinal surgery and related to one of three etiologies: ION, central/branch retinal artery occlusion, and cortical blindness. Although various etiologic factors related to the surgical procedure, the patient, and the patient's intraoperative course have been noted, identifying definitive risk factors remains problematic. Given the rarity of its occurrence and the lack of standardization in care, definitive suggestions regarding treatment are not possible.

Of significant importance is immediate recognition, suggesting that monitoring and evaluating visual acuity following high-risk procedures may be warranted. Immediate and emergent ophthalmological consultation is suggested in all cases. Treatment options that have failed to consistently prove effective include therapies aimed at decreasing inflammation or orbital swelling, including optic-sheath fenestration, systemic corticosteroids, and various pharmacologic agents, such as acetazolamide and diuretics including mannitol. Other therapies met with limited to no success include attempts to restore blood flow by modulating the coagulation cascade (anticoagulants, thrombolytic agents, and antiplatelet agents) and increasing perfusion pressure and oxygen delivery (increasing MAP and hemoglobin). However, anecdotal reports note the

Table 2 Perioperative recommendations for prevention and treatment

1. Preoperative questioning regarding visual acuity
   a. May need preoperative evaluation if there are questions regarding patient's preoperative status
2. Preoperative discussion of risk of POVL (0.1–0.2 %)
3. Identification of potential patient-related factors, including risk factors for atherosclerotic disease (hypertension, diabetes, obesity, hyperlipidemia), and paradoxical embolus risk factors (congenital heart disease or patent foramen ovale).
4. Preoperative discontinuation of erectile dysfunction medications, such as sildenafil.
5. Attention to intraoperative positioning with:
   a. Avoidance of direct ocular pressure
   b. Use of approved head-positioning devices for prone procedures
   c. Avoid prone positioning and Trendelenburg positioning; use slight head-up positioning, as feasible
   d. Maintain unobstructed access to patient's eyes to allow for repeated positioning of checks every 15–30 min
6. Intraoperative fluid management to include the use of colloid as feasible and clinically indicated; monitoring of central venous pressure during prolonged or complex procedures
7. Attention to intraoperative hemodynamic management with invasive arterial pressure monitoring
8. Frequent monitoring of hemoglobin during lengthy procedures and those associated with significant blood loss; no definitive transfusion trigger can be provided
9. Maintain normocarbia; avoid inadvertent intraoperative hyperventilation
10. Postoperative visual acuity evaluation following high-risk procedures
11. As far as clinically acceptable, maintain head-up position (30–45°) postoperatively to limit orbital edema

dramatic and prompt restoration of vision when hemoglobin and MAP were simultaneously increased. As noted above, hyperbaric oxygen therapy has also been suggested. Various strategies to prevent or limit the incidence of POVL have been suggested and developed (Table 2) [43]. Attention to optimizing intraoperative oxygen delivery may be useful, especially in patients with comorbid conditions. As noted, no definitive transfusion trigger can be offered, as the appropriate range remains 7–10 g/dl. Although intraoperative hypotension is a suggested risk factor, definitive recommendations regarding blood pressure management should be determined on a patient-to-patient basis. Controlled hypotension to limit the need for allogeneic transfusions remains an acceptable technique. Although the use of colloid to limit third-spacing and orbital edema has been suggested, its safety and efficacy must be balanced with recent concerns regarding the use of albumin or hydroxyethyl starch solutions. Finally, immediate postoperative evaluation of visual acuity is suggested to identify problems immediately. Emergent ophthalmological consultation should be obtained when questions arise, followed by funduscopic examination and CNS imaging to identify the cause of POVL. Although POVL remains a relatively rare perioperative complication, given its devastating nature, ongoing research is needed to further define its etiology, outline improved prevention methods, and investigate additional treatment options.

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