

VISUAL LOSS AS A COMPLICATION OF NON-OPHTHALMOLOGIC SURGERY: A REVIEW OF THE LITERATURE

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Decreased visual acuity and loss of visual ability are devastating anesthetic and surgical complications. The incidence is greater in patients with preexisting hypertension, diabetes, sickle cell anemia, renal failure, gastrointestinal ulcer, narrow-angle glaucoma, vascular occlusive disease, cardiac disease, arteriosclerosis, polycythemia vera, and collagen vascular disorders. Precipitating factors for ischemic optic neuropathy include prolonged hypotension, anemia, surgery, trauma, gastrointestinal bleeding, hemorrhage, shock, prone position, direct pressure on the globe, and long operative times.

Prone and Trendelenburg positions can lead to visual loss related to decreased venous return from the head. Visual impairment may result from increased intracranial pressure, which contributes to undue pressure on the optic

nerve. The prone position increases the risk of direct compression injury to the orbit and corneal abrasion.

Astute attention to positioning is imperative, especially with the prone position. At-risk patients should receive transfusion once the calculated allowable blood loss has been surpassed. Unacceptable hemoglobin and hematocrit values should be corrected preoperatively and levels monitored during the case to avoid intraoperative anemia in at-risk patients. The blood pressure of patients with predisposing diseases should be kept within normal limits. To avoid this devastating complication, it is imperative that anesthesia providers understand contributing factors and prevention strategies.

Key words: Anterior ischemic optic neuropathy, intraocular pressure, ischemic optic neuropathy, posterior ischemic optic neuropathy.

Anatomy and physiology

The optic nerve contains 1.2 million myelinated nerve fibers. These fibers arise from cell bodies in the ganglion cell layer of the retina. The optic nerve can be divided into 4 areas: the anterior or intraocular, posterior or intraorbital, intracanalicular, and intracranial. All of these areas have their own unique blood supply.¹ This segmental blood supply accounts for the various visual field sections affected by disease processes such as anterior optic neuropathy.² The blood supply to the optic nerve head is not standard and can vary between individuals.² The primary sources of blood to the optic nerve head are the posterior ciliary arteries.²

Interruption of the blood flow to any of the arteries supplying the optic nerve results in ischemia. This ischemia causes axonal swelling and can profoundly damage myelinated nerve fibers. Ischemia to the optic nerve results in decreased axoplasmic flow and subsequent vision loss.³

The retina and the optic nerve have different blood supplies. The retina derives its blood supply from more than one source. One source is the central retinal artery, which arises from the ophthalmic artery, which origi-

nates from the internal carotid artery. The central retinal artery splits into 4 tracts that feed the 4 quadrants of the retina.^{1,4} The second source arises from the choriocapillaris, which is the innermost area of the choroid.¹

Cause of visual impairment

Postoperative vision loss can be the result of several precipitating factors. Ischemia to the optic nerve leading to ischemic optic neuropathy (ION) is one such cause and can be classified as anterior or posterior, depending upon location of the lesion in the optic nerve.^{5,6} Other mechanisms identified in vision loss are retinal artery occlusion, cortical blindness, and ophthalmic venous obstruction.^{5,6} Ischemic eye injuries related to spinal stenosis repair are usually unilateral and caused by venous occlusion, central retinal artery occlusion, anterior or posterior ION, or infarction in the occipital lobe.⁷ A common thread throughout these scenarios is ischemia affecting at least one area of the visual pathway.⁵

Visual defects can vary from quadrantal defects to complete blindness.⁸ Visual loss as a result of optic nerve ischemia is classified as ION. Cortical blindness is generally the diagnosis if the visual loss is the result of optic radiation or occipital cortex involvement. Other sources of vision loss are central retinal artery

occlusion and increased venous pressure related to obstruction of outflow from the eye.¹

Normally the autoregulation of the optic nerve head provides adequate circulation to the eye during periods of varying perfusion pressures.² The *perfusion pressure of the eye* is defined by Hayreh² as the difference between the systemic mean arterial pressure (MAP) and the intraocular pressure (IOP). Perfusion pressure will decrease with unopposed decreases in the mean arterial pressure or increases in IOP.⁵ If there is a dramatic increase or decrease in the perfusion pressure of the eye, autoregulation becomes ineffective.² The optic nerve head depends on circulation from the posterior ciliary arteries. The posterior ciliary arteries need to be in equilibrium with IOP to ensure adequate blood flow.⁹ If the IOP increases or the perfusion pressure decreases, blood flow to the optic nerve will be compromised.⁹

Changes to the arteries that perfuse the optic nerve head by disease processes such as arteriosclerosis, atherosclerosis, vasculitis, drug-induced vasoconstriction or dilation, and other cardiovascular diseases will directly affect perfusion of the optic nerve.²

Ischemic optic neuropathy

After ischemia occurs, the optic nerve becomes edematous, which can further exacerbate the perfusion problem.⁹ Damage to the optic nerve can create scotomata, which are blind spots in the field of vision unlike the blind spot in the optic disc area of the eye.^{10(p595)} Histopathologic studies performed on affected optic nerves reveal interstitial edema, a loss of myelin, astrocytes, and diapedesis of the erythrocytes.⁸ Ischemic optic neuropathy is manifested clinically by acute or subacute painless loss of acuity of the visual field or loss of vision.^{11,12} It is characterized by pallid edema of the optic nerve¹² and often is associated with an afferent pupillary defect.^{11,13}

Ischemic optic neuropathy is classified as anterior or posterior.¹³ These classifications are defined by the symptomatology and the source of the blood supply. Dunker et al³ found that anterior ION occurs more frequently than posterior ION. Both can be attributed to hypotension. Advanced age, diabetes, and hypertension seem to be associated with anterior ION.³

• *Anterior ION.* Anterior ION is one of the most widespread and visually devastating diseases in the middle-aged and elderly populations.² Anterior ION is seen most commonly during the fifth and sixth decades of life and is manifested as a sudden onset of painless visual loss.¹² This disease process occurs when the IOP exceeds that of the perfusion pressure in the arteries that supply blood to the optic nerve head.¹⁴

Anterior ION frequently is caused by infarctions in the watershed zones. A *watershed zone* is a capillary bed that lies distal to the branches of the short posterior ciliary arteries in the choriocapillaris.¹ This area is characterized by areas without adequate vascularity.² There are differences in the vascular anatomy between individuals and, possibly, between eyes in the same individual. This can result in differences in the severity of injury between individuals.^{1,2} The importance of the location of the watershed zones is that these regions represent areas of the optic nerve with limited perfusion and are at most risk for ischemia.²

Ischemia can be classified by the degree that occurs: mild, moderate, or severe. *Mild* ischemia interferes with axoplasmic flow, although it does not alter transmission of the visual impulse. *Moderate* ischemia decreases axoplasmic flow and visual conduction impulses, and *severe* ischemia results in instantaneous, irreparable damage.¹

Anterior ION is further classified into 2 groups: nonarteritic and arteritic.^{1,2} The most common group is nonarteritic, which occurs often during the postoperative period and is of most interest to the anesthesia provider.¹ This type generally is due to a transitory decrease in oxygenation to the choroid surrounding the optic disc, causing swelling of the optic disc.^{1,11} This swelling can be the result of "decreased perfusion pressure, increased resistance to blood flow or decreased oxygen carrying capacity."^{1(p1021)}

The origin of arteritic ION is inflammatory. It often is associated with systemic disease processes, for example, giant cell arteritis, lupus erythematosus, hypertension, diabetes, or atherosclerosis.^{2,10} Frequently it is caused by thrombotic or embolic lesions of the arteries and arterioles nourishing the optic nerve head. The posterior ciliary arteries become occluded, and there is decreased perfusion to the anterior optic nerve, which these arteries supply.²

There currently exists no consistently effective treatment for patients with anterior ION, and in most cases, vision does not improve.^{12,15} However, some sources state that systemic corticosteroids may minimize edema to the optic nerve head.⁹

• *Posterior ION.* Posterior ION generally involves the area of the optic nerve between the optic foramen at the orbital apex and the area of entry of the central retinal artery.^{1,13} Posterior ION does not involve the retina as other disease processes do.³ It can be caused by thrombotic or embolic situations affecting the vessels that supply blood to the optic nerve head.⁵ The most common cause is decreased partial pressure affecting this blood supply, suggesting that decreased mean arterial pressure or increased IOP are significant

risk factors.⁵ The optic nerve becomes ischemic and edematous. Visual loss occurs from axonal compression.³ On examination, the optic disc usually exhibits pallid edema.^{1,12,13}

- *Central retinal artery occlusion.* Central retinal artery occlusion can be caused by extraocular pressure on the orbit of the eye itself, resulting in elevated IOP.⁵ Wolfe et al⁴ noted that unexplained intraoperative bradyarrhythmias or conduction abnormalities may indicate an increase in intraorbital pressure while in the prone position.

Incidence of eye injury and visual loss

The reported incidence of eye injury and visual loss varies greatly. Table 1 summarizes the data related to visual loss. Roth et al¹⁶ performed a retrospective analysis of patients who underwent anesthesia for nonocular surgeries during an approximately 4.5-year period. Of the 60,965 patients, 34 (0.06%) were identified as having sustained eye injuries with 1 (<0.001%) having irreversible visual loss. This analysis found that patients who were of advanced age (mean ± SD, 51.2 ± 16.6 years) had a higher incidence of visual impairment. Other factors identified included longer operative times, procedures involving the head and neck, and lateral positioning. The most common injury noted was corneal abrasion.

Stevens et al¹⁷ identified 7 (0.2%) of 3,450 cases during a 9-year period (1985-1994) in which vision loss occurred after spinal surgery. Ischemic optic neuropathy was the most common diagnosis.

The incidence seems to be higher in patients undergoing open heart surgery and cardiopulmonary bypass.¹ Shaw et al¹⁸ noted out of 312 patients having coronary artery bypass graft surgery, 80 patients (25.6%) developed some neuro-ophthalmological complication. Shapira et al¹² reported an incidence of 8 (1.3%) of 602 cases in this patient population. In contrast, Sweeney et al¹⁹ studied 7,685 patients who had cardiac bypass surgery and found that 7 (<0.1%) developed ION.

A retrospective study by Warner et al²⁰ reported that 405 (0.1%) of 410,189 surgical patients reported some degree of postoperative vision loss. Of these, 216 regained full vision within 30 days. One patient (<0.001%) of 118,782 (0.0008%) developed irreversible vision loss. This study did not include cardiovascular surgeries.

Data from closed insurance claims analysis are summarized in Table 2. The American Association of Nurse Anesthetists Foundation Closed Claims Study analyzed 241 cases of which 8 (3.3%) involved eye injuries.²¹ Of these, 4 involved corneal abrasions that

Table 1. The incidence of visual loss as a complication of anesthesia and surgery

Researcher	Incidence*	Type of surgery
Roth et al ¹⁶	1/60,965 (<0.06)	Nonocular general surgery
Stevens et al ¹⁷	7/3,450 (0.2)	Spinal surgery
Shaw et al ¹⁸	80/320 (25.6)	Cardiac surgery
Shapira et al ¹²	8/602 (1.3)	Cardiac surgery
Sweeney et al ¹⁹	7/7,685 (<0.1)	Cardiac surgery
Warner et al ²⁰	1/118,782 (<0.1)	Nonocular, non-cardiac general surgery

* Numbers in parentheses are percentages.

Table 2. The incidence of visual loss as a complication of anesthesia and surgery as reported through closed insurance claims analysis

Researcher	Incidence*	Type of surgery
AANA Foundation closed claims ²¹	4/241 (1.7)	Nonocular
Gild et al, ²² ASA closed claims	43/2,046 (2.10)	Nonocular

* Numbers in parentheses are percentages.

resolved and 4 resulted in permanent visual impairment or loss. In 3 cases that resulted in loss of vision, the patients were in the prone position for spinal procedures lasting 5.45 to 12.5 hours. Periods of hypotension and anemia were present in 2 of these 3 claims. The fourth claim involved episodes of severe hypotension and hypertension during vaginal delivery, resulting in visual impairment in one eye.²¹

Gild et al²² reviewed 2,046 closed claims between 1974 and 1987 for the American Society of Anesthesiologists' Closed Claims project. Of these claims, 71 (3.47%) were related to some type of eye injury. The most common injury was corneal abrasion, followed by hemorrhage and vitreous loss. Patient movement, described as "bucking," coughing, or restlessness was implicated in the majority (21%) of these claims. Other methods of injury included chemical irritation from the prep solutions (9 claims), direct trauma to the eye during repositioning (6 claims), pressure from the anesthetic mask (2 claims), placement in the prone position (1 claim), and 1 traumatic injury resulting from a laryngoscope falling into the eye. Two

claims denoted increased ocular pressure as the causative factor, and 1 was related to a hypoxic event following cardiac arrest. Permanent visual impairment occurred in 61% of the aforementioned cases.²²

Of the claims, 43 (2.10%) were filed because of visual loss. The associated injuries included direct damage to the eye, its structures, blood supply, and the optic nerve. Excluded were injuries to the nerves innervating the extraocular muscles and occipital lobe damage.²²

Onset of symptoms

Often patients note visual changes immediately in the recovery room but tend not to report them because they assume the changes are normal postoperative sequelae.⁸ Shapira et al¹² described the interval to the onset of symptoms as anywhere from postextubation to the 10th postoperative day. Katzman et al⁹ reported that although the onset of visual loss is usually immediate, it may be delayed for several days. Brown et al⁸ stated that if the patients do not report vision loss immediately, they will report the visual loss within 1 week of surgery.

Preexisting and predisposing factors

Although many cases have been reported in patients who exhibited no predisposing risk factors, conditions have been cited in the literature that may contribute to visual loss or have been noted to contribute to decreased visual acuity. Predisposing factors include giant cell arteritis,^{17,19} atherosclerosis,¹¹ hypertension,^{8,11,17,23-27} temporal arteritis,^{24,26} smoking,^{23,27} diabetes,^{1,5,11,23-25} hematologic disorders,²⁶ sickle cell disease,⁵ narrow-angle glaucoma,²⁴ atherosclerotic vascular occlusive disease,^{5,23,24,27} emboli,²⁶ renal failure, hemodialysis,⁸ cardiac disease,^{8,27} and collagen vascular disorders.^{1,26} Certain disease states, peripheral vascular disease,¹⁷ arteriosclerosis,^{1,5,26,27} and vasculitis^{5,26} in particular, have been noted because they can cause increased resistance to blood flow, potentially causing ischemia.⁵

Multiple risk factors exist for the development of visual complications and ION. Throughout the literature, the most commonly reported perioperative factors related to ION are deliberate hypotension,^{11,17} gastrointestinal bleeding²⁸ or anemia^{1,5,9,11,29} or both, hypotension,^{1,8,9,17,23-29} hemorrhage,^{5,9} shock,¹⁹ hypothermia,^{5,19} and long operative times.^{17,23} Other factors associated with an increased risk of ION are large intraoperative blood loss, direct compression on the globe,⁵ and prolonged duration of prone positioning.^{5,23,29}

Surgical procedures commonly identified in the literature as potentially contributing to postopera-

tive visual problems include cardiopulmonary bypass,^{12,18,19,24} coronary artery bypass grafting,⁸ procedures involving the head and neck,^{10,16,24} oral surgery,⁸ and orthopedic procedures.⁸ Postoperative vision loss most commonly is associated with spinal surgeries performed with the patient in the prone position.^{1,7,17,23}

Deliberate hypotension, a technique often used during surgery to decrease blood loss, may lead to a decrease in the perfusion pressure of the eye.^{11,17} Whether this alone can cause ischemia to the eye is a question that needs to be evaluated further because research is limited in this area. Cheng et al⁵ found that of 24 patients with visual loss, a deliberate hypotensive technique had been used for only 2. Both patients also had a large blood loss of 2,000 mL. The authors concluded that the hypotensive technique alone could not be considered the sole contributing factor.⁵ Katz et al²⁷ reported 4 cases of visual complications in which deliberate hypotension was used during lumbar spine surgery.

Cardiopulmonary bypass surgery uses a hypothermic technique to promote neuroprotection and reduce oxygen consumption. In some cases, this may contribute to ischemia of the eye.^{5,12,19} The blood flow decreases due to increasing viscosity with decreasing temperature, and this may create ischemia in the watershed areas of the optic nerve.^{12,19}

- *Patient positioning.* Positioning is cited frequently as a contributing factor related to vision loss. Roth et al¹⁶ concluded that positioning in the lateral position contributed to 6 of 34 demonstrated eye injuries. Other studies demonstrated evidence suggesting prone or Trendelenburg positioning as the origin of vision loss.^{4,6,7,14,29-32} The prone position presents unique challenges in protecting the orbits of the eye. For many spinal surgeries, the patient is placed in a prone, cerebral-dependent position. This position can decrease venous return from the head, leading to capillary bed stasis and decreased perfusion to the optic nerve. Obstructive venous outflow leading to ION has been documented in the literature.²⁹ Normally the venous drainage would exit through the superior ophthalmic vein into the cavernous sinus. If this pathway is altered in any way, it can lead to increased intracranial pressure and IOP, which can in turn lead to an increase in episcleral venous pressure. Elevated episcleral pressure can result in a decrease in choroidal blood flow in the eye, which normally acts as the venous outflow for the aqueous humour.²⁹ The resultant consequence to the eye is ION.

The Relton-Hall and the Wilson frames often are used for prone positioning of patients during surgery. They are designed so that the position of the head is

dependent compared with the rest of the body,⁶ decreasing venous outflow from the head and increasing IOP. This, as well as the prone position itself, can increase IOP. Malposition in a horseshoe-type headrest also has been cited as contributing to visual loss.^{4,22} The Mayfield headrest, another positioning tool, may not be appropriate if a patient has exophthalmos or a flattened nasal bridge as this may allow pressure on the orbits of the eye.⁴ If these devices are to be used, caution must be taken to avoid venous obstruction from the head.

Review of the literature

There are numerous case reports but few authentic research studies in the literature regarding the topic of ION. Seven studies were reviewed; only 1 was a prospective, controlled study. Of the other 6 retrospective studies, only 1 compared data with data for a control group.²³

Myers et al²³ reviewed 37 cases in which patients experienced visual loss after spinal surgery. Of the 37 cases, 27 reports were obtained from physicians via a survey, and 10 were obtained from existing literature. Documentation was complete for 28 cases, which were compared with a control group of 1,736 people who had spinal fusions.

The majority of the cases that preceded visual loss were instrumented posterior spinal fusions. Blood loss and surgery times were greater in patients who experienced visual loss, despite the fact that blood pressure and hematocrit values were similar to those in the control group. Myers et al²³ indicated that this accounted for the large number of cases in which a hypotensive technique was implemented without subsequent ophthalmologic complications. It is possible that the effect of blood loss on visual complications may be separate from that of decreased blood pressure in some cases. Of the 37 patients, 26 had unilateral effects and 11 had bilateral problems. Fifteen had complete blindness. Visual field loss was noted in 18 patients, and loss of acuity was documented in 5 patients. The most common diagnosis was ION (22 cases), central retinal occlusions were noted in 9 cases, and cortical ischemia was responsible in 3 cases. The remaining cases did not indicate a definitive diagnosis.²³ Direct pressure on the eye from improper positioning was the causative factor for visual loss in some cases. Anemia due to large blood loss and hypotensive events in which the systolic blood pressure decreased below 90 mm Hg were listed as other contributing factors despite the fact that generally this hypotension is tolerated among patients.²³

Follow-up evaluations showed that visual acuity improved in 8 cases and the visual field improved in 5 cases. Although no measures of this improvement were discussed in this article, the authors quantified improvement as small. Twenty-five patients who had no light perception initially showed no improvement.²³

Spinal surgeries also were found to be significant in the studies by Dunker et al³ and Myers et al.²³ Seven cases were reported during a 9-year period in which 71.4% developed posterior ION after spinal surgeries.³ Similarly, Katz et al²⁷ noted spinal surgeries in their article as a common connection to visual impairment.

Another category of surgeries frequently stated in the literature as having a high incidence of postoperative vision loss was cardiac procedures, in particular coronary artery bypass surgeries.^{12,18} Shapira et al¹² documented that 8 (1.3%) of 602 patients who had coronary artery bypass surgery developed anterior ION.

Shaw et al¹⁸ reviewed data for 312 patients who had coronary artery bypass grafting. Of these, 80 developed neuro-ophthalmic complications. Interestingly, 40 of those patients had preexisting ophthalmologic abnormalities.

Dunker et al³ described preexisting conditions in patients who had postoperative eye complications. These precipitating conditions included high blood pressure and diabetes (57.1%). Sweeney et al¹⁹ also noted hypertension and diabetes as preexisting conditions. Katz et al²⁷ examined 4 cases in which 3 patients had a history of coronary artery disease and diabetes and were smokers. Cerebrovascular accidents, peripheral vascular disease, and cardiac failure were noted by Shaw et al.¹⁸

Strong correlations were found related to anemia and hypotension as causative factors for vision complications. Brown et al,⁸ Dunker et al,³ and Katz et al²⁷ found that patients with vision loss had significant episodes of hypotension and anemia. Shapira et al¹² noted that the patients who developed anterior ION had lower hematocrit values than a control group. In addition, causative hypotensive events were noted in studies done by Sweeney et al¹⁹ and Shaw et al.¹⁸ Deliberate hypotension was connected to vision loss in the research done by Katz et al.²⁷ Blood loss was a significant factor in the study by Myers et al.²³ Katzman et al⁹ reported a blood loss of 16,000 mL in a patient reporting postoperative visual loss. Finally, Sundaram et al³³ noted a case in which a patient developed blindness in the right eye following a cardiopulmonary arrest.

Various clinical diagnoses were made, including the most common, ION,^{3,10,28} as well as retinal infar-

tion, retinal emboli, cerebral visual field defects, and Horner syndrome.¹⁸

Review of surgical case reports

Ischemic optic neuropathy was the diagnosis in the majority of cases described in the case reports (12/20 [60%]).^{6,7,11,13,24,29,32,34-38} Central retinal artery occlusion was noted in 15% (3/20),^{4,14,29} and 1 case involved retrobulbar optic neuritis in 5% (1/20).³⁷

Preexisting disease processes were present in many patients. Hypertension was noted in 8 of 20 (40%) patients.^{6,9,11,24,34,35,37} Diabetes was present in 5 of 20 (25%) patients as well.^{7,9,13,29,35} An even higher number had a history of smoking (7/20 [35%]).^{6,11,13,34,36} Alcohol abuse was noted in 3 of 20 (15%) cases.^{13,35,36} Obesity affected 3 of 20 (15%) individuals.^{9,29,35} Cardiac disease processes were present in 4 of 20 (20%) cases.^{6,34,35} Other conditions noted in the articles were Graves disease (1/20 [5%]),⁶ anemia (9/20 [45%]),^{4,9,13,34,34,38} hypercholesterolemia (1/20 [5%]),³⁵ atherosclerosis (1/20 [5%]),³⁸ and renal transplantation (1/20 [5%]).³⁴

Although not all case reports defined a specific relationship, predisposing factors were noted as follows: venous engorgement related to prone positioning,^{29,30} direct compression of the globe related to prone positioning,^{4,32} and eye edema related to the prone position.^{7,14} Large blood loss⁹ and significant decreases in hemoglobin levels, as well as deliberate hypotensive anesthesia, were prevalent in these case reports.^{9,11,37} There were many cases in which these episodes were thought to be potential contributing factors to postoperative vision loss and/or blindness.^{4,9,11,13,35,37,38} The 3 case reports by Jaben et al³⁴ concluded preexisting conditions of decreased oxygen carrying capacity were the cause of ION.

The recovery rates for the patients in these case reports were grim. Of the patients who developed ophthalmologic complications postoperatively, reported outcomes of 55% (10/18) never regained their sight,^{4,7,11,14,24,34-36,38} 44% (8/18) recovered to some degree,^{6,9,13,29,30,34,35,37} and the information regarding recovery was absent in the remaining cases.

Of the patients, 22% (4/18) cases reporting noticed visual loss immediately in the recovery period. All other patients remarked they had trouble seeing or could not see at all within 7 days of surgery. Rizzo and Lessell³⁵ described a patient who was being mechanically ventilated postoperatively and motioned often to her eyes. It was not until she was extubated the next day that she could tell the healthcare providers that she could not see at all. As is often the case, she never recovered vision and was completely blind.

Discussion

Centuries ago, vision loss was noted by Hippocrates to be associated with the massive hemorrhage experienced in the battlefields among soldiers who were seriously wounded.⁹ This observation continues to hold true as evidenced by contemporary research and case reports. After evaluating the information on ION, its incidence, pathophysiology, populations at risk, and contributing factors, it is clear that prevention is paramount.

Careful preoperative screening should be done to identify patients with disease processes or other risk factors that may predispose them to ophthalmic complications. Knowledge and identification of surgical procedures associated with a high incidence of ION, central retinal artery occlusion, or other ophthalmologic complications are imperative.

Williams et al¹ suggest that information pertaining to the potential for visual complications be part of the informed consent. However, this view is not held by all. Roth et al³⁹ state that the incidence of postoperative visual complications is so infrequent that the need to inform the patient needs to be balanced with the potential anxiety it could cause.

Certain surgeries are associated with large amounts of blood loss. This, along with the strong correlation between ION and concordant anemia and hypotensive episodes, makes it imperative that standards for administering transfusions to such patients be addressed. With the recent increased concern about transfusions and the risk of transmission of blood-borne illnesses, there has been an increasing reluctance to administer transfusions.^{11,40,41}

The National Institutes of Health Consensus Conference on Perioperative Blood Transfusion⁴⁰ recommends transfusion for individuals with a hemoglobin of less than 7 g/dL (70 g/L). Conference participants suggested assessing numerous factors before deciding to administer a transfusion. These factors include duration of anemia, duration of surgery, intravascular volume, potential for a large blood loss, and coexisting disease processes. The conference members also indicated the importance of realizing that concordant hypovolemia and anemia increase the patient's risk of mortality and morbidity. Recent changes in transfusion administration and lower acceptable hemoglobin levels may put patients at risk for ION.⁸ Prolonged periods of low hemoglobin levels and diminished oxygen-carrying capacity may result in inadequate blood and oxygen supplies for some patients. Blood replacement should be aggressive, using cell saver or autologous blood donated before surgery. Katz et al²⁷ also suggested that blood transfusions need to be used more liberally to avert visual loss or impairment. They

indicated that special consideration should be given to patients with a high risk of atherosclerosis.

Controlled hypotension is used in spinal surgeries in an attempt to minimize blood loss. The risks and benefits of deliberate hypotensive techniques and their effects need to be considered before using this technique. Prolonged hypotension may increase a patient's risk for ION. Patients with preexisting conditions will be at an even greater risk. Myers et al²³ suggested setting a minimum systolic pressure reading preoperatively and then keeping the blood pressure at or above that level. The anesthesia care team and the surgeon should consult preoperatively to discuss the risks and benefits and from there, develop an individualized plan of care for the patient.

The literature indicates the prone position^{4,6,7,14,29-31} and the lateral position¹⁶ as predisposing factors for eye injury. The eyes should be checked often and with any movement. Direct global compression must be avoided. Unexplained cardiac arrhythmias may signal compression on the orbits.⁴ Caution should be taken when positioning a patient in steep Trendelenburg due to the potential for decreased venous return, leading to decreased perfusion to the optic nerve. Facial and ocular compression must be avoided. Wolfe et al⁴ noted that physiologic differences in facial appearance may need to be considered. For example, a patient with a flattened nasal bridge or exophthalmos may be at higher risk if the prone position is used.⁴ Fluids should be administered carefully to decrease orbital swelling related to overhydration.¹²

Shapira et al¹² denoted specific interventions for cardiopulmonary bypass surgery. With the high incidence of postoperative blindness evident in the literature following cardiac surgery, these interventions seem pertinent and worth mentioning. They are as follows:

1. Brief duration of cardiopulmonary bypass.
2. Use of colloid prime in patients at high risk for anterior ION
3. Reduction of perioperative edema by judicious use of fluids and hemoconcentration.
4. Appropriate PCO₂ management to maintain cerebral autoregulation and decrease microembolization.
5. Avoidance of prolonged low perfusion states or hypertension.
6. Avoidance of profound systemic hypothermia except when specifically indicated.
7. Judicious use of exogenous inotropic and vasoconstricting agents.
8. Maintenance of adequate hematocrit and hemoglobin values in high-risk patients.
9. Intraoperative monitoring and adjustment of IOP in high-risk patients (eg, patients with glaucoma).

Immediate postoperative assessment of visual acuity seems prudent. Any findings that lead to concern in the postoperative assessment should trigger the provider to obtain an ophthalmology consult as soon as possible. Stevens et al¹⁷ indicated that a delay in diagnosis and treatment can lead to a poorer outcome.

Conclusion

The incidence of IONs and other ophthalmologic complications is highest in the middle-aged and elderly populations. Surgical procedures commonly associated with visual complications are spinal surgeries and cardiac surgeries, in particular, coronary artery bypass grafting. Long operative times and surgeries performed with the patient in the prone position lend themselves to complications as well. Some preexisting conditions that contribute to postoperative visual complications include hypertension, diabetes, coronary artery disease, peripheral vascular disease, and a history of smoking. A significant amount of evidence supports the strong correlation between anemia and hypotensive events with the development of IONs. Deliberate hypotension also puts patients at risk. Surgeries with large blood losses and coinciding drops in hemoglobin are further predisposing factors.

Consideration needs to be given concerning the limitation that of the articles reviewed; only one of the studies, that by Shaw et al,¹⁸ was a prospective study in which actual ophthalmologic examinations were performed preoperatively to definitively identify baseline data for each patient. Interestingly, Shaw et al¹⁸ found 40 of 312 patients with preexisting ophthalmologic abnormalities. It is likely that there were patients in other studies who were predisposed to complications but were not identified. Additional studies are needed to determine more risk factors and provide baseline information to researchers and anesthesia providers.

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