

# Ischemic Optic Neuropathy After Spine Surgery

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*Visual loss is a traumatic occurrence that has been reported after prone spine surgical procedures performed under general anesthesia. The most common cause of postoperative visual loss is ischemic optic neuropathy. Although the incidence of postoperative visual loss is rare, this devastating injury has been reported more frequently.*

*Several factors increase the risk for the development of ischemic optic neuropathy. Results from several case studies have attributed ischemic neu-*

*ropathy with vision loss after general anesthesia to perioperative anemia, blood loss, hypotension, and prolonged operative times.*

*Ischemic optic neuropathy usually presents with painless visual loss and visual field deficits during the immediate postoperative period. There is no definitive treatment. Prevention is the key.*

**Keywords:** Blood loss, hypotension, ischemic optic neuropathy, visual loss.

**L**oss of vision after spine surgery is an uncommon but devastating postoperative occurrence. Partial or complete, unilateral or bilateral visual loss may occur and is typically painless, with no specific treatment. Although reports of postoperative visual loss have been documented in supine patients, most reported cases have followed general anesthesia for spine procedures in the prone position.<sup>1</sup>

Impaired vision after prone spine procedures performed under general anesthesia is usually due to changes in blood flow to the eyeball or optic nerve, related to either decreased perfusion or embolism. Direct pressure on the periorbital area or globe may also contribute to visual impairment, but this etiology is less frequent. Visual impairment is usually irreparable despite the mechanism.<sup>2</sup>

The first case of blindness after a prone spine procedure was reported by Slocum et al<sup>3</sup> in 1948. Improper positioning of the head on a Bailey headrest was the cause of blindness. In 1954, Hollenhorst et al<sup>4</sup> reported a case of blindness that resulted from prone positioning using the Mayfield horseshoe headrest. The result of these studies established a link between increased intraocular pressure (IOP) and hypoperfusion to vision loss.<sup>2</sup>

Ischemic optic neuropathy (ION) is the most common cause of postoperative visual loss in nonophthalmologic surgery. Ischemic optic neuropathy is classified as being either anterior or posterior, depending on the condition of the optic disc. With anterior ischemic optic neuropathy (AION), the optic disc is pale and swollen, whereas with posterior ischemic optic neuropathy (PION), the optic disc is not affected.<sup>1</sup> The incidence of AION, PION, and central retinal artery occlusion after spine surgery is less than 0.2%.<sup>5</sup>

## Anatomy of the Visual Pathway

Normal vision is a multifaceted process with the follow-

ing requirements: (1) tear film; (2) the lens and cornea, necessary for focusing; (3) the retina with its photoreceptors; (4) the optic nerve; (5) the cerebral cortex; and (6) intact blood supply. Loss of visual acuity or blindness may arise if a disturbance in 1 or more of these regions occur. The orbit protects the globe, or eyeball. Two-thirds of the refractory power in the eye is supplied by the cornea, which is a lucid protective layer that covers the front of the eye. The eyelid offers protection for the eye, and lubrication for the eye is offered by the lacrimal and sebaceous glands. Light is refracted and focused onto the photoreceptors of the retina by the lens.<sup>2</sup>

Axons of the optic nerve originate from the retinal ganglion. The intraocular portion of the optic nerve is encompassed by the optic disc.<sup>6</sup> After the optic nerve passes through the scleral canal, it goes toward the diencephalon, where it attaches in the lateral geniculate nuclei. The neurons terminate in the parietal lobe and the occipital lobe of the brain, which processes the visual image.<sup>2</sup>

The ophthalmic artery, which originates from the internal carotid artery, is the major blood supply to the eye. The retina, globe, and optic nerve receive blood supply from the ophthalmic artery. The central retinal artery, which is a division of the ophthalmic artery, provides blood to the inner retina. The difference between the IOP and the mean arterial pressure in the retinal artery characterizes retinal perfusion. The middle and posterior cerebral arteries are the primary source of blood supply to the occipital lobe.<sup>2</sup>

The central retinal artery and the posterior ciliary artery (PCA) trunks, which divide into the main PCAs, compose the ocular branches of the ophthalmic artery. The short PCAs are a division of the main PCAs and supply the posterior choroid and the anterior portion of the optic nerve. Normally 2 to 3 PCA trunks divide into the medial and lateral PCAs.

The circle of Zinn-Haller around the optic nerve is structured by the medial and lateral short PCAs. The pial branches, choroidal branches, and other vessels originate from the circle of Zinn-Haller to supply the optic nerve. This circle is complete in 77% of postmortem human specimens and has narrowed segments in 43%.<sup>6</sup> In 75% of postmortem eyes, the medial PCA is the main source of blood supply to the optic nerve. Since the PCAs are end arteries without anastomoses, this allows for variations in the location of the watershed zones in the optic nerve. The various locations of the watershed zones may be somewhere between the fovea and the nasal border of the optic disc. Their locations can be temporal to the optic nerve head, pass through portions of the optic nerve head, or comprise the entire optic nerve head. Whether or not portions of the optic nerve head are vulnerable to ischemia is determined by the locality of the watershed zone. The etiology of ION has been linked to the anatomical modification in the PCA circulation.<sup>6</sup>

### Definition and Types of Ischemic Optic Neuropathy

Unilateral optic nerve dysfunction, visual field defect, and an absence of other causes of decreased vision are indicative of the presence of an afferent pupillary defect and are diagnostic indicators of ION.<sup>7</sup> Ischemic optic neuropathy usually occurs suddenly after surgery or nonsurgical bleeding without forewarning signs and is the primary origin of sudden visual loss in patients 50 years or older. Ischemic optic neuropathy can be categorized as either anterior or posterior, with anterior being more common. This categorization is determined by whether the insult occurs in the anterior or posterior portion of the optic nerve. Both AION and PION can be further classified as being arteritic or nonarteritic.<sup>6</sup>

- *Anterior Ischemic Optic Neuropathy.* Anterior ION is usually painless and irreversible with 2 distinguishing characteristics: (1) existence of visual field deficits and/or alterations in visual acuity and (2) edema of the optic disc with ensuing atrophy. The initial symptom of AION is optic disc edema. Splinter hemorrhages around the optic disc may also occur. Infarction of the short PCAs in the choriocapillaris in the watershed areas is the source of visual loss with AION; the inferior half of the visual field being the most prevalent area for the visual field defect. The extensiveness of visual loss is determined by blood supply of the optic nerve.<sup>7</sup>

The most severe type of AION is arteritic. Arteritic AION is a systemic disease that usually occurs in patients 60 years or older and has a female prevalence, with its etiology being temporal arteritis. Arteritic AION is emergent and if left untreated can lead to permanent blindness.<sup>6</sup>

- *Posterior Ischemic Optic Neuropathy.* Posterior ION manifests itself as acute loss of vision with visual field deficiencies and is usually not related to occlusive vascular disease.<sup>7</sup> Ischemia of the retrolaminar region of the optic

nerve is a characteristic of PION. With PION the lesion is located further posteriorly than in AION, and no optic disc anomaly is evident with initial funduscopic examination. Its occurrence is associated with giant cell arteritis, systemic lupus erythematosus, sickle cell disease, and fungal infections as well as after surgical procedures.<sup>6</sup>

### Symptoms and Time of Onset of Ischemic Optic Neuropathy

The most frequently reported visual loss secondary to prone positioning during spine surgery is PION. This visual impairment is typically noticed by the patient upon waking from general anesthesia or very soon afterward. It may take 1 or more days after the procedure for the patient to notice changes in vision. Patients may experience a momentary blurring of vision that seems to be predominantly in the left eye. Initially with the funduscopic examination, the optic nerve appears to be normal, but gradually over several days the optic nerve at the optic disc becomes pale. The optic nerve atrophies over a period of several weeks.<sup>2</sup>

### Risk Factors for Ischemic Optic Neuropathy

Numerous factors have been proposed as risk factors for postoperative ION. Some of these factors include decreased systemic blood pressure, blood loss, anemia or hemodilution, an increase in intraocular or orbital venous pressure, anomalous autoregulation in the optic nerve circulation and/or anatomical deviation in the way blood is supplied to the optic nerve, and vasopressor use. Hypertension, diabetes, atherosclerosis, and retrobulbar hemorrhage have also been identified as contributing factors to ION. Even though hypotension and anemia are factors that have been suggested through case reports to cause ION, ION occurs in patients without these factors and also in patients with blood pressures and hematocrits that anesthesiologists consider to be in acceptable ranges.<sup>8</sup>

Specific risk factors have been reported to be associated with ION after spine surgery. These associated risk factors encompass lengthy operative procedures in the prone position, which involve hypotension, hemodilution or anemia, blood loss, and infusion of large amounts of intravenous fluids.<sup>8</sup> Major blood loss has been reported to be a risk factor for ION after prone spine surgery. It has not been determined whether or not the anemia or the decline of perfusion pressure, which is attributable to hypotension related to blood loss, is the leading cause. Reports of visual loss related to ION have been made in patients without hypotension or anemia.<sup>1</sup>

Although blood flow to the optic nerve is controlled via autoregulation, as many as 20% of healthy individuals have anomalous autoregulatory function of the circulation to their anterior optic nerve. There is a small range of perfusion pressures that operate autoregulation, and numerous processes, such as aging, diabetes mellitus, and evident arterial hypotension, may interrupt autoreg-

ulation. Patients with chronic hypertension may be intolerant to hypotension since it contributes to a shift in the autoregulatory curve of optic nerve blood flow.<sup>1</sup>

It has been reported that anesthetized prone patients experience a rise in IOP. Reduction in the perfusion of the retina and optic nerve may result from increases in IOP. Administration of large amounts of intravenous fluid may lead to orbital congestion that contributes to increases in IOP. Increases in IOP may also be a source of perioperative ION.<sup>1</sup>

## Patient Management

With increased reports of perioperative visual loss (POVL) associated with spine procedures performed under general anesthesia, a practice advisory was developed by the American Society of Anesthesiologists (ASA) Task Force on Perioperative Blindness. This task force consisted of 12 members appointed by the ASA. The job of the task force was to examine and evaluate scientific literature, acquire professional agreement and public opinion, and to evolve a practice advisory related to perioperative management of prone patients undergoing spine procedures under general anesthesia. The perioperative period was designated as the time frame from the immediate preoperative assessment through discharge from the acute healthcare facility. Posterior ION, AION, and central retinal artery occlusion were the areas of concentration by the task force.

The advisory body's intention was to augment awareness of POVL and to decrease its occurrence. The task force defined POVL as "permanent impairment or total loss of sight associated with a spine procedure during which general anesthesia is administered."<sup>5</sup>

• *Preoperative Management.* The ASA Task Force on Perioperative Blindness agrees that the susceptibility of patients to perioperative ION increases with the existence of identifiable preoperative risk factors. These risk factors include (1) vascular risk factors such as hypertension, glaucoma, carotid artery disease, smoking, obesity, and diabetes; (2) prolonged procedures, defined as exceeding an average of 6.5 hours in duration; (3) preoperative presence of anemia; (4) substantial blood loss, defined as loss that reaches an average of 44.7% of estimated blood volume; and (5) prolonged procedures in combination with substantial blood loss. The task force is in agreement that there are identifiable preoperative risk factors. However, specific preoperative patient characteristics that might predispose patients to perioperative ION have not yet been identified. It is the belief of the task force that a preoperative ophthalmic or neuro-ophthalmic evaluation would not be beneficial in the identification of patients at risk for POVL. Another recommendation of the task force is to inform patients of the small, unpredictable risk of POVL associated with prolonged procedures and substantial blood loss. Since the occurrence of visual loss after spine procedures of short duration is low, the decision to educate the pa-

tients who are not at high risk should be decided on a case-by-case basis.<sup>5</sup>

• *Intraoperative Management.* Multiple intraoperative factors are linked to POVL in patients who underwent spine surgery. Among these contributing factors are hypotension, blood loss, anemia, hypovolemia, hypoxia, hemodilution, edema of the face, use of vasopressors, infusion of large amounts of fluid, pressure on the eye, prone and head-down positions, increased venous pressures, and extended duration of surgery. The common factors of lengthened surgical duration and substantial blood loss are present in most patients who have been affected by POVL.<sup>5</sup>

Although intraoperative hypotension has been reported to be an associated contributing factor to POVL, induced intraoperative hypotension has been linked to few complications in most patients with POVL. These data imply that hypotension independent of other risk factors is not likely to be the direct contributing cause in POVL. The amount of time the patient is exposed to hypotension may be more of a contributor versus the degree of hypotension. Anemic patients who experience hypotension may have an occurrence of infarction of the optic nerve head, where blood supply is vulnerable to compression from edema.<sup>7</sup>

Preoperative presence of chronic hypertension, cardiac problems, and renal and vascular disease influences the intraoperative management of blood pressure in high-risk patients. Blood pressure management is also affected by fluid administration, rate of blood loss, deliberate hypotension, and the use of vasopressors. Postoperative visual loss has been reported after procedures in which substantial blood loss and hypotension has occurred.<sup>5</sup>

The ASA Task Force on Perioperative Blindness emphasizes vigilant monitoring of systemic blood pressure in high-risk patients. The task force agrees that the use of deliberate hypotension is not a contributing factor to POVL, and its use should be evaluated on a case-by-case basis. Their recommendation for the use of deliberate hypotension in patients without preoperative chronic hypertension is that blood pressure maintenance should be on average within 24% of the estimated mean arterial pressure or with a minimum systolic blood pressure of 84 mm Hg, ranging between 50 and 120 mm Hg.<sup>5</sup>

Increased IOP, periorbital edema, and double vision have resulted from the use of large volumes of crystalloids, but studies examining the effect of intravascular volumes on the incidence of POVL among patients who underwent spine surgery have received little attention in the literature.<sup>5</sup> The task force advises the use of colloids along with crystalloids for fluid resuscitation and replacement in patients with substantial blood loss, to lessen the incidence of POVL. Central venous pressure monitoring may also be useful in high-risk patients.<sup>5</sup>

Continuous extensive blood loss and decreased hemoglobin concentration intraoperatively have been reported as contributing factors to perioperative ION. The National Institutes of Health Consensus Panel on Blood Trans-

fusion and the ASA practice guidelines specify in routine clinical practice that hemoglobin levels higher than 8.0 g/dL do not necessitate blood transfusion. When blood volume is not maintained in the presence of uncontrolled hemorrhage, oxygen delivery to the optic nerve may be decreased, leading to either anterior or posterior ION. How low the hemoglobin level must be or the length of time the hemoglobin level is required to decrease to result in this complication has not been reported.<sup>4</sup>

Patients who experience substantial blood loss should have hemoglobin and hematocrit levels monitored at regular intervals to identify anemia. Since it is the belief of the task force that no documented lower limit of hemoglobin concentration has been linked to POVL, it has not established a transfusion threshold for the reduction of POVL.<sup>5</sup>

Local constriction of small blood vessels is an effect of vasopressors, which are used to elevate the blood pressure. The blood vessels supplying the eye are also vasoconstricted, resulting in lower perfusion pressure to the optic nerve.<sup>9</sup> A case report published in *Anesthesiology* described an unfortunate patient that developed ION following lumbar spinal fusion in which phenylephrine was used for perioperative blood pressure maintenance.<sup>10</sup> The task force believes that insufficient data exist for guiding the use of  $\alpha$ -adrenergic agonists during spine surgery for high-risk patients. Their use should be evaluated on a case-by-case basis.<sup>5</sup>

Data from case reports suggest that acute-onset ION or central retinal artery occlusion in patients who underwent spine surgery may result from direct pressure to the eyes while using a sheet roll or headrest during patient positioning. Facial edema is a common occurrence in the prone-positioned patient, but it is the opinion of the task force that no pathophysiologic method links facial edema as the cause of perioperative ION. They also believe that no scientific evidence exists where ocular compression allegedly is the causative agent for isolated cases of perioperative AION and PION. The task force advises the anesthesia provider to use careful head positioning, where the head is level with or higher than the heart when possible and maintained in a neutral forward position. Task force members also advocate frequent eye assessments along with proper documentation.<sup>5</sup>

Perioperative ION can occur in patients who have undergone prolonged spine surgical procedures while positioned prone and who have experienced substantial blood loss. Although no studies reporting the effect of surgical staging on reducing the incidence of POVL have been documented, the task force believes that staged spine surgery procedures should be considered in high-risk patients. Although staged spine surgery procedures may be associated with additional costs as well as risks that may include infection, neurologic injury or thromboembolism, the task force believes that using the staged procedure may reduce these patient

risks along with reducing the risk of POVL.<sup>5</sup>

• *Postoperative Management.* The use of magnetic resonance imaging to evaluate the degree of visual loss after spine surgery in patients with PION has not proved to be valuable. The ASA Task Force on Perioperative Blindness agrees that the vision of the high-risk patient should be assessed after the patient becomes alert. It also advocates an urgent ophthalmologic consultation if a patient reports any visual deficits.<sup>5</sup>

## Treatment of Ischemic Optic Neuropathy

There is controversy surrounding treatment options for ischemia and hypoperfusion in the posterior aspect of the eye. An ophthalmologic consultation is appropriate in all circumstances, and patient management should be guided by an ophthalmologist.<sup>2</sup> Numerous treatment modalities have been attempted, with little success in treating AION. Some of these treatments include retrobulbar steroid injections, antiplatelet therapy, anticoagulants, phenytoin, norepinephrine, and blood replacement.<sup>7</sup>

An ophthalmologic consultation should be requested immediately with the occurrence of vision-related complications. Treatment options for the initial management of vision loss after spine surgery associated with PION consist of correcting volume depletion, correcting blood loss, restoring the blood pressure to normal, and possibly administering corticosteroids intravenously.<sup>2</sup>

Regrettably, there is no established treatment of ION.<sup>6</sup> Intraocular pressure can be lowered by acetazolamide in an effort to improve blood flow to the optic nerve head and retina. Mannitol and furosemide are diuretics that can be used to minimize edema. Corticosteroids may be helpful in reducing axonal swelling in the acute phase, but in the postoperative phase they may be a contributing factor to increasing the patient's risk for wound infection. When ION occurs along with considerable reductions in blood pressure and hemoglobin concentration, attention should be given to increasing ocular perfusion pressure or the hemoglobin concentration. If it is suspected that increased ocular venous pressure is the problem, positioning the patient in a head-up position may be helpful. With documentation of increased IOP in cases of ION, attempts should be made to lower IOP.<sup>6</sup>

Few data are available that document treatment options for ION. A literature review by Buono and Foroozan<sup>11</sup> revealed that in numerous cases of ION, no treatment attempts were made. However, in the cases where treatment was attempted, the therapies used were correction of hemodynamic instabilities, systemic corticosteroids, antiplatelet therapy, and actions to lower intraocular and cerebrospinal fluid pressures. Correction of hemodynamic derangements was the only treatment modality that proved to be valuable.<sup>11</sup>

## Outcomes of Ischemic Optic Neuropathy

There have been some reported instances of recovery of

vision in patients with ION, but this does not always occur. Ischemic optic neuropathy can lead to permanent loss of vision related to optic nerve atrophy.<sup>12</sup> The result of ION is usually irreversible unilateral or bilateral loss of vision or blindness. There have been some instances when central retinal artery occlusion and ION have improved, but full recovery of vision in the affected eye is uncommon. The worst prognosis for perioperative blindness is with central retinal artery occlusion and PION.<sup>2</sup>

In one study it was reported that a patient experienced complete recovery of vision after suffering perioperative PION following the correction of anemia and hypotension.<sup>11</sup> Blood transfusions were given to maintain the hematocrit to a level above 35%, and blood pressure maintenance was above 140/80 mm Hg. During the postoperative period the patient's vision was 20/70 in the right eye (OD) and 20/200 in the left eye (OS). The vision was 20/40 OD and 20/30 OS within the 48-hour period after the blood transfusion. The patient's vision had improved to 20/20 in both eyes at a 7-month follow-up examination.<sup>11</sup> In some circumstances, the patient's vision may be saved if the underlying causes such as perioperative anemia and hypotension are corrected without delay and suitable treatment is initiated without unnecessary delay.<sup>12</sup>

### Preventive Measures

Based on results of a preoperative ophthalmologic assessment, there is no patient profile that unmistakably identifies a high-risk patient for ION. Prevention is therefore vital. The beginning point for prevention is an increased awareness of the possibility for complications in addition to the connection of hypoperfusion to the eyes, particularly in patients with vaso-occlusive conditions, such as chronic essential hypertension and diabetes mellitus.<sup>2</sup>

Because the preoperative condition of the patient's optic nerve circulation is unknown and no effective means are available to monitor the optic nerve intraoperatively, these factors are hindrances for recommended strategies for the prevention of ION. Nevertheless, some generalized recommendations can be made. For patients with known preexisting conditions such as cardiovascular disease, long-standing or poorly controlled hypertension, known visual disorders such as glaucoma or end-organ ocular damage, it would be practical to sustain systemic blood pressure as close to baseline values as possible and to avoid extended declines in ocular perfusion pressure.<sup>6</sup>

When planning patient care, the anesthesia care provider should encourage patients to stop smoking before surgery, document visual disorders before surgery, take care that no direct pressure is applied to the patient's eyes, and use sensible drugs and anesthetics in patients with glaucoma. Some other preventive measures include avoidance of prolonged reduction of oxygen delivery to the eye that results from hypotension or anemia and possibly minimizing the time a patient is positioned prone.<sup>7</sup>

### Summary

Visual loss after anesthesia is a rare but distressing occurrence. It has been reported in patients that are positioned supine during general anesthesia but occurs more frequently in patients positioned prone for spine surgery procedures.

Ischemic optic neuropathy is the most common cause of visual loss in nonocular procedures. It usually occurs suddenly after surgery or nonsurgical bleeding without forewarning. Ischemic optic neuropathy is categorized as either anterior or posterior. Categorization depends on the condition of the optic disc, with anterior being more common than posterior.

Several risks factors have been documented as contributing factors to ION. Hypotension, lengthy surgical time and excessive blood loss are the main contributing risk factors to ION. Regrettably, there is no established treatment of ION. Because treatment options for loss of vision associated with prone spine surgery and general anesthesia are restricted, prevention is vital.

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