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Traumatic Optic Neuropathies

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Traumatic Optic Neuropathy as a Complication of Head Injury

Epidemiology

Traumatic optic neuropathy may be a result of severe head trauma or may be associated with little or no evidence of head injury. Traumatic optic neuropathy occurs in approximately 0.5% to 5% of closed head injuries[1] and in 2.5% of patients with maxillofacial trauma and midface fractures.[2] Loss of consciousness is associated with traumatic optic neuropathy in 40% to 70% of cases.[3,4] In closed head injuries, the site of injury causing blindness is often the forehead or supraorbital ridge, less commonly the temporal region. Patients from 20 to 40 years of age represent the major trauma population who experience traumatic optic neuropathy.[5]

The prevalence of severe initial visual loss ranges from about 43% to 56%. Visual loss may present with no light perception to 20/20 with an associated visual field defect. More severe visual loss is usually associated with optic canal fracture. In some studies, 43% (6/14)[6,7] to 56% (13/23)[8] of patients presented with light perception or no light perception (NLP) following traumatic optic neuropathy.

Deceleration injury directed to the ipsilateral forehead or to the midface region from motor vehicle and bicycle accidents is the most common cause of traumatic optic neuropathy in 17% to 63% of cases.[9] The second most common cause of traumatic optic neuropathy is motorcycle accidents followed by falls in as many as 50% of cases.[9] Other situations that may cause traumatic optic neuropathy include assault, gunshot wounds, falling objects, skateboarding, and even very minor head injuries. Iatrogenic injury may occur during endoscopic sinus surgery and orbital surgery.[9]

Basic Anatomy of the Optic Nerve

To better understand the location and mechanisms of optic nerve injury, the anatomic relationships are reviewed.

Optic Nerve Head

The axons of the retinal ganglion cells converge on the posterior pole of the globe at the optic disc. The intraocular portion of the optic nerve is approximately 1 mm long and is the shortest portion of the nerve. It can be divided into prelaminar and laminar segments. In the prelaminar portion, the optic disc, which is oval shaped and approximately 1.5 mm horizontally × 1.75 mm vertically, consists of unmyelinated axons of the retinal ganglion cells, astrocytes, capillary-associated cells, and fibroblasts. The central retinal artery and vein traverse centrally from the disc. These millions of axons emerge from the globe as fascicles and pass through the lamina cribrosa, 200 to 300 fenestrations through the choroids, and sclera in the laminar portion of the optic nerve head.[10]

The size of the scleral canal and the angle of exit of the canal from the eye may cause variations in the appearance of the optic disc.

A larger scleral canal leads to a larger physiological cup size. A smaller scleral canal causes a small or absent physiological cup and gives the appearance of a crowded optic nerve head.[10]

The ophthalmic artery arises from the ophthalmic branch of the internal carotid artery. It passes anteriorly through the optic canal alongside the optic nerve, which is covered with dura. In the orbit, the ophthalmic artery gives rise to the central retinal artery, which enters the optic nerve sheath approximately 10 mm behind the globe and extends anteriorly to emerge from the center of the optic disc. The central retinal artery does not directly supply blood to the optic disc. Much of it is derived from the choroidal feeder vessels, short posterior arteries, and some from the pial arterial network, which all contribute to the circle of Zinn–Haller, a perineural arteriolar anastomosis that encircles the optic nerve head. The retrolaminar portion of the optic nerve is supplied by anastomosing branches of the central retinal artery and the pial arteries. The laminar and prelaminar portions of the nerve are mainly supplied by branches of the posterior ciliary arteries. Only a small fraction of the blood supply to the optic nerve head comes from choroidal branches of the posterior ciliary arteries that extend to the optic nerve head.[10]

The central retinal vein drains most of the optic nerve head. During chronic compression of the intraorbital optic nerve or after central retinal vein occlusion, optociliary shunt vessels (preexisting anastomosis between superficial disc veins and choroidal veins) may enlarge and shunt venous blood from the retina to the choroids. Eventually, it drains into the vortex veins leading to the superior and inferior ophthalmic veins.[10]

Orbital Optic Nerve

From the posterior aspect of the globe, the orbital segment of the optic nerve extends to the orbital apex. It is approximately 25 mm long and has a sinuous course that allows free movement of the globe and protects the nerve from injury when there is orbital proptosis.[1] The width of the orbital optic nerve is about 3 mm to 4 mm in diameter, twice as wide, mainly because of the myelin produced by oligodendrocytes and its encasement with meninges. Myelination of the axons of retinal ganglion cells extends from the point where they exit the globe to the point where they synapse in the lateral geniculate nucleus. The myelinated optic nerve is encased with all three layers of the meninges (the dura, arachnoid, and pia mater). The outermost layer is the dura, which is composed of collagen and is continuous with the sclera. At the orbital apex, the dura fuses with the periosteum and with the annulus of Zinn. Underneath the dura is the arachnoid. Arachnoid trabeculae connect this layer with the dura and the underlying pia mater, where capillaries traverse as they enter into the substance of the optic nerve. The subarachnoid space, filled with cerebrospinal fluid (CSF), is continuous with the intracranial portion.[10]

As the optic nerve passes posteriorly toward the optic canal, it is surrounded by orbital fascia, fat, nerves, and vessels. Most of the blood supply to the orbital optic nerve derives from capillaries from the surrounding pial plexus. In the posterior orbit, the optic nerve is crossed superolaterally by the nasociliary branch of the trigeminal nerve, ophthalmic artery, superior ophthalmic vein, and superior division of the oculomotor nerve. The superior rectus, levator palpebrae muscles, trochlear nerve, and frontal branches of the trigeminal nerve are located superior to the optic nerve in the roof of the orbit. Inferior to the optic nerve at the floor of the orbit lie the inferior and medial recti muscles and the inferior division of the oculomotor nerve. Between the optic nerve and the lateral wall of the orbit are the lateral rectus muscle and abducens nerve. Between the lateral rectus muscle and the optic nerve is the ciliary ganglion, parasympathetic postganglionic neurons innervating the constrictor pupillae and ciliary muscles.[10]

Regarding the blood supply of the optic nerve, the anterior orbital optic nerve is surrounded by four posterior ciliary arteries that are branches of the ophthalmic artery. In the middle of the orbit, the ophthalmic artery traverses inferolaterally to the optic nerve until it crosses under (or occasionally over) it. In 6%

of cases, the ophthalmic artery can lie medially to the optic nerve as they both reach the orbital apex and pass through the annulus of Zinn, a tendon from the origin of insertion of the four recti muscles.[11] The medial location of the ophthalmic artery is predisposed to injury during a trans-sinus approach for optic nerve decompression.[11]

Intracanalicular Optic Nerve

The optic nerve, ophthalmic artery, and sympathetic fibers from the carotid plexus all enter the optic foramen of the optic canal in the apex of the orbital roof. The ophthalmic artery enters the optic canal inferior and lateral to the optic nerve. The length of the optic canal is about 10 mm. It is formed by the two lesser wings of the sphenoid bone. Its thinner medial wall separates the optic nerve from the sphenoid and posterior ethmoid sinuses. In about 4% of patients the optic nerve may have areas covered only by the nerve sheath and sinus mucosa, without any bony covering between the intracanalicular optic nerve and the adjacent paranasal sinus. These sinuses may eventually enlarge into the optic canals producing pneumosinus dilatans. This finding is often seen with an adjacent optic nerve sheath meningioma.[10]

Because the dura of the optic nerve is fused with the periosteum of the optic canal, impact forces that deform bone may be more easily transmitted to the intracanalicular portion of the optic nerve. The intracanalicular optic nerve is also susceptible to injury under the fixed edge of the falciform dural fold at the near edge of the optic canal. The tightly fixed optic nerve within the optic canal is also predisposed to compression from small lesions, arising within the optic canal or at either of its openings, that may be difficult to visualize on thin-section computed tomography (CT) or magnetic resonance imaging (MRI) scanning.[10]

Intracranial Optic Nerve

The intracranial optic nerve is covered by a firm fold of dura as it exits the optic canal. The distance between the two optic nerves at this point is about 13 mm, and they extend posteriorly, superiorly, and medially to join at the optic chiasm. The length of the intracranial optic nerve varies from 3 mm to as long as 16 mm, but is usually about 10 mm. If the intracranial optic nerve is shorter than about 12 mm, the optic chiasm is prefixed, in which it is located more anteriorly and superiorly to the sella turcica. If the intracranial optic nerve is longer than 18 mm, the optic chiasm is postfixed, in which it is located more posteriorly to the dorsum sellae. The variation in the length of the optic nerve affects the types of visual field defects caused by tumor in the suprasellar region.[10]

Dorsal to the optic nerve is the olfactory tract at the ventral surface of the frontal lobes. Ventral to the optic nerve are the anterior cerebral and anterior communicating arteries. The internal carotid artery from the cavernous sinus may sometimes emerge laterally to the optic nerve. The optic nerve is also adjacent to the internal carotid artery where it bifurcates into the anterior cerebral and middle cerebral arteries and to the proximal portion of the posterior communicating artery. The intracranial optic nerve is supplied by the ophthalmic artery from the internal carotid artery. These anatomic relationships predispose the optic nerve to be injured by traumatic aneurysms of the internal carotid, ophthalmic, and anterior cerebral arteries.[10]

Localization of Direct Optic Nerve Injuries from Head Trauma

Direct injury to the optic nerve needs to be distinguished from indirect injury. Direct injury arises from penetrating trauma, such as from orbital fractures associated with midfacial fractures. The most common optic nerve injuries involve posterior indirect injuries, followed by chiasmal, and direct injuries. Direct traumatic optic neuropathy is less common because the laxity of the intraorbital optic nerve allows for both absorption and deflection of the penetrating object. The resilience of the dura to penetration also offers further protection.

If an object penetrates into the orbit, the optic nerve may be directly injured by complete or partial transaction of the nerve, contusion of the nerve, or by compression from hemorrhage or foreign-body impingement.[12] Optic nerve

transection occurs as a complication of midfacial trauma and orbital fracture. Visual loss is NLP caused by transection of the optic nerve, perhaps from a bony fragment seen on CT scan.

If orbital hemorrhage is present, an orbital compartment syndrome may occur. An enlarged optic nerve sheath may also be seen on CT. Orbital hemorrhage may be diffuse or localized in the orbit. It is often accompanied by proptosis and ophthalmoplegia. Increased orbital pressure causes injury to the optic nerve, which may be decreased by elevating the head and administering acetazolamide to lower intraocular pressure. If not, lateral canthotomy and drainage of the orbital hemorrhage may be necessary.[12]

Orbital emphysema can also occur in the setting of paranasal sinus injury. Thin fractures of the bone lining the orbital wall may produce a ball-valve effect so that air accumulates in the orbit to cause proptosis and compression of the optic nerve. Drainage of air by insertion of a needle into the retro-orbital space may resolve this condition.[12]

Optic nerve avulsion is often caused by sudden rotation or anterior displacement of the globe with a finger or object to result in optic nerve injury at the lamina cribosa.[12] Funduscopic findings are commonly seen and include peripapillary vitreous hemorrhage, partial or complete optic nerve head avulsion, optic disc swelling, venous congestion, central retinal artery nonperfusion, and retinal edema. In partial and complete avulsion of the optic nerve head, a ring of hemorrhage is formed around the optic disc. The site of avulsion is seen as a dark crescentic area over the disc. If injury occurs at the orbital optic nerve anterior to the point at which the central retinal artery enters and the central retinal vein exits, arterial and venous obstruction and disc swelling may be seen.

Optic nerve swelling without retinal changes can also occur from hemorrhages in the optic nerve sheath posterior to the origin of the central retinal vessels. Prompt treatment of an optic nerve sheath hematoma may lead to visual recovery. An expanded nerve sheath causing proptosis and a central retinal artery or vein occlusion could indicate the presence of an optic nerve sheath hematoma, especially in the setting of a progressive optic neuropathy. Drainage by sheath fenestration usually restores vision.[13,14]

Localization of Indirect Optic Nerve Injuries from Head Injury

The most common optic nerve injuries involve posterior indirect injuries, followed by chiasmal, and direct injuries (Table 5.1).[15]

Posterior indirect is the most common type of traumatic optic neuropathy and is usually a result of a frontal or midfacial trauma that also may be trivial. The intracranial portion of the nerve is relatively fixed within the bony canal. The orbital bone transfers force from the forehead and brow to the orbital apex.[16,17] The intracanalicular portion of the optic nerve is the most common site of indirect optic nerve injury.[18] Visual loss is usually immediate, and less often delayed or progressive, with variable visual field defects associated with an afferent papillary defect and/or dyschromatopsia. Often no ophthalmoscopic signs of injury are seen initially. Injury to the distal optic nerve in the orbit, optic canal, or intracranial cavity usually leads to disc atrophy and pallor after 3 to 5 weeks. If head trauma with loss of consciousness produces increased intracranial pressure, papilledema may be seen. Optic canal fracture on CT often does not correlate with the severity of the optic neuropathy. It is also imperative to distinguish a preexisting optic neuropathy, which can be observed as optic atrophy in a patient with acute head trauma.[9]

The intracranial portion of the optic nerve is least likely to have traumatic damage. Chiasmal injury is uncommon and is usually the result of severe closed head injury or an abrupt traction on the globe. The tethering of the optic nerve within the optic canal may prevent transmission of force to the chiasm. According to a review of 18 cases of autoenucleation,[19,20] 33% of the optic nerve transections occurred at the anterior chiasm, whereas 55% of them occurred at the orbital apex. This review and other studies[21,22] suggest that strong and abrupt tractional forces on the globe are required to cause tears in the

Table 5.1. Types of traumatic optic neuropathy from head injury (adapted from Lessell[15])

Type	Pathogenesis	Clinical findings	Management
Direct	Penetrating object causing direct injury to the optic nerve by complete or partial transection of nerve or contusion of nerve; hemorrhage or foreign body compressing the optic nerve.	Initial variable level of vision that often worsens. Orbital hemorrhage may cause orbital compartment syndrome. Enlarged optic nerve sheath may be seen on CT scan.	Removal of foreign body impinging on optic nerve. Lateral canthotomy and cantholysis; drainage of subperiosteal hematoma if present. Optic nerve sheath fenestration if nerve sheath hematoma or edema is seen on CT scan, and if hematoma is confirmed to be subdural on oblique view on MRI scan.
Anterior indirect	Sudden rotation of anterior displacement of globe with object causing injury to the anterior segment of the optic nerve, often at the lamina cribrosa. Rare type of traumatic optic neuropathy.	Prepapillary vitreous hemorrhage. Partial or complete optic nerve head avulsion. Papilledema, venous congestion, central retinal artery occlusion, retinal edema.	Treatment for central retinal artery occlusion, if present. Look for occult rupture of the globe or extraocular muscle avulsion.
Posterior indirect	Frontal or midfacial trauma or trauma that may appear trivial causing indirect optic nerve injury. Most common type of traumatic optic neuropathy.	No ophthalmoscopic signs of injury. Afferent pupillary defect and/or dyschromatopsia. Immediate visual loss is common. Delayed or progressive visual loss occurs in a few cases. Loss of consciousness and midfacial fractures are common. Variable visual field defects. Optic canal fracture on CT does not correlate with severity of optic neuropathy.	(See Management section.)
Chiasmal	Severe closed head injuries or an abrupt traction on the globe may cause chiasmal injury.	Variable visual field defects. Central visual acuity may be normal. Anosmia, diabetes insipidus, or other endocrinopathies; skull base fractures and other neurological deficits may be present.	No treatment for optic nerve injury. Neurosurgical consultation may be needed for associated intracranial injuries.

optic nerve with chiasmal injury. Clinical findings may include normal central visual acuity, variable visual field defects, such as bitemporal hemianopsia and defects from unilateral lesions of von Willebrand's knee. Anosmia, diabetes insipidus, or other endocrinological disorders, fractures of the skull base, cerebrospinal leakage, meningitis, thalamic injury, and other neurological deficits may also be seen. No treatment is yet available.[19]

Diagnostic Tests

Traumatic optic neuropathy is a clinical diagnosis. It usually occurs after head trauma with or without loss of consciousness. Decreased best

corrected visual acuity and an RAPD, without other ocular pathology that could account for the visual loss, would support the diagnosis of traumatic optic neuropathy affecting the posterior orbital, intracanalicular, or intracranial portion of the optic nerve. These patients usually have 20/400 or less in the affected eye.[15] More subtle optic nerve injury, which is thought to occur in less than 10% of cases,[4] may present as delayed visual loss.

Examination of the ocular adnexa is important to identify orbital rim fractures and periorbital swelling, which can mimic proptosis. Resistance to retropulsion of the globe and increased intraocular pressure measured by tonometry can help detect retro-orbital hemorrhage. Retraction of the swollen eyelids is needed to look for evidence of penetrating ocular injury. Blunt injury to the iris can cause hyphema, angle recession, and even lens dislocation.[15]

On funduscopy, a ring of hemorrhage at the site of injury is indicative of partial or complete avulsion of the optic nerve head.[23] Injury between the globe and where the central retinal vessels enter the optic nerve can cause venous obstruction and traumatic anterior ischemic optic neuropathy.[13,24] Hemorrhage in the optic nerve sheath posterior to the origin of the central retinal vessels may produce only optic disc swelling.[25] Papilledema from increased intracranial pressure may even be superimposed on traumatic optic neuropathy.[26] Decreased visual acuity with an afferent papillary defect without intraocular pathology is usually indicative of intracanalicular or intracranial optic nerve injury.

If the patient is unconscious or if the RAPD is absent in bilateral cases, visual evoked potentials (VEP) may help in confirming the suspicion of traumatic optic neuropathy, especially in comatose patients. In unilateral traumatic optic neuropathy, flash VEP amplitudes that are at least 50% of the normal eye are critical for a good visual outcome.[27] An absent VEP response indicates that visual loss is complete, and recovery of vision is unlikely.[28] An absent electroretinogram (ERG) is associated with a poor potential for visual recovery.[29]

Localization of injury by visual field testing is limited. There is no pathognomonic visual field loss diagnostic of optic nerve injury. Altitudinal visual field defects, central, paracentral, and centrocecal scotomas, and generalized field constriction have been reported.[3,30–32] Humphrey visual field testing or confrontational testing at the bedside is useful in documenting degree of visual recovery. Optical coherence tomography (OCT) is able to assess and monitor axonal loss after traumatic optic neuropathy.[33] Based upon earlier work by Lundstrom and Frisen,[34] serial fundus photography showed that trauma to the intracranial optic nerve caused gradual disappearance of the retinal nerve fiber layer (RNFL) during weeks 4 to 8. Similar RNFL changes can be seen with the use of OCT.

Early transient increase followed by progressive loss of the retinal nerve fiber layer in traumatic optic neuropathy can be documented by the GDx NFL scanning laser polarimeter nerve fiber analyzer (Laser Diagnostic Technologies, San Diego, CA, USA). In a study by Miyahara et al.,[35] the early increase in RNFL represented transient edema of the nerve fibers. Nerve fiber atrophy was completed by day 90 following the injury and was correlated with enlargement of the optic disc. A scanning laser polarimeter nerve fiber analyzer may be used to quantitate the severity of optic nerve damage and the effectiveness of therapy in traumatic optic neuropathy.

Neuroimaging may also help in localizing the site of optic nerve injury. CT scan with 1.5-mm axial sections allows good reformation along any axis and allows sufficient resolution to image optic nerve position orbital hematoma, orbital edema, intrasheath hematoma, nonorganic foreign bodies, and bony fractures. Optic canal fractures are seen on CT scans in approximately 36% to 67% of cases.[36] The force from trauma is transferred to the sphenoid and then to the optic nerve as it traverses the optic canal.[37] After metallic foreign bodies are ruled out by CT scan, MRI is more sensitive for detecting chiasmal injury and subtle intraneural or intrasheath hemorrhage, distinguishing it from epidural hemorrhage.[4,38] MRI of the orbit may reveal focal edema of the optic nerve or optic nerve sheath enhancement with gadolinium. On T_2-weighted images, the

hyperintense signal from CSF surrounding the injured optic nerve may be absent when compared with the normal nerve. MRI may distinguish intrasheath from intraneural hemorrhage. MRI is also superior to CT in delineating chiasmal injury.[39–41]

Color Doppler imaging may help to differentiate extrinsic optic nerve compression caused by orbital hemorrhage from other causes of optic neuropathy. The B-scan portion of this imaging technique may also help identify optic nerve sheath hematoma.[42,43] The color Doppler portion may help in evaluating perfusion to the optic nerve head.[44,45]

Visual Prognosis

In the natural history of indirect posterior optic nerve injuries, recovery is never complete. Subtle visual field, color vision, and papillary defects persist despite complete recovery of visual acuity by Snellen measurements. Most patients develop optic atrophy. Spontaneous improvement from case series ranged from 20% to 71%.[6,46,47] In Lessell's series,[6] the extent of visual loss did not correlate with the potential for spontaneous recovery. The variation in extent and rate of recovery and response to treatment could be related to the pathogenesis of traumatic optic neuropathy in various clinical circumstances. For direct optic nerve injuries, the possibility of visual recovery is much less, but recovery of vision has occurred in such cases.

In indirect traumatic optic neuropathy, four features were significant in predicting no recovery of visual acuity: (1) the presence of blood in the posterior ethmoidal cells; (2) loss of consciousness associated with traumatic optic neuropathy; (3) absence of recovery after 48 h of corticosteroid treatment; and (4) age of patient over 40 years.[48] Patients who have the foregoing four poor prognostic factors could be considered for optic canal decompression. In this study, 87% of patients who had improved visual outcome experienced visual recovery within 48 h of the initiation of corticosteroid treatment. Another sign of favorable recovery is optic nerve swelling after blunt trauma has been associated with a favorable prognosis for visual recovery. Brodsky et al.[49] reported three patients who had partial recovery of vision.

Pathology

Based on 174 postmortem examinations by Crompton[18] on patients who died after closed head trauma, optic nerve dural sheath hemorrhages was found in 83% of patients. Interstitial optic nerve hemorrhages occurred in 36% of these patients; two-thirds had the hemorrhage within the optic canal. Tears and ischemic lesions occurred in 44% of patients; in 81%, these involved the intracanalicular optic nerve, and in 54% these affected the intracranial optic nerves.

From a case series of patients who had blunt head trauma, more than 50% of patients who had traumatic optic neuropathy were found to have sphenoid bone fractures on CT scan. Laser interferometry studies done by Anderson et al.[17] showed that forces applied to the frontal bone during a deceleration injury are transmitted to and concentrated in the optic canal. Elastic deformation of the sphenoid bone allows transfer of the force into the intracanalicular portion of the optic nerve. The firm attachment of the dural sheath to the optic nerve in the optic canal is thought to predispose it to shearing forces, resulting in tearing of axons and vessels that leads to contusion necrosis. The development and location of a fracture depends upon the elasticity of the bone, in that thicker bone is more inelastic and more likely to fracture. Direct injury to the optic nerve from displaced bony fragments in the optic canal is uncommon.

Shearing forces from blunt head trauma can displace the intracranial optic nerve upward against the falciform dural fold that overlies the intracranial end of the optic canal, resulting in direct or indirect injury. A frontal blow is transmitted posteriorly along the orbital walls to the sphenoid bone and the optic canal. A deceleration injury would allow the globe and the majority of the intraorbital contents to continue forward, whereas the intracanalicular optic nerve would remain immobile because of its tethering at the orbital apex and optic canal. The deceleration would be a shearing force to

the optic nerve. Direct optic nerve injury from partial or complete avulsion from the globe usually does not sever the nerve and often leads to permanent injury to just a portion of the nerve.[17]

Although optic canal decompression is performed based upon the hypothesis that edema inside the bony canal may lead to more swelling and ischemia of the optic nerve, there is not much evidence that optic nerve edema within the optic canal plays a significant role in causing traumatic posterior optic neuropathy. Vascular changes in and around the optic nerve may play a more important role than just the swelling of the nerve itself. Decreased perfusion pressure to the optic nerve within the optic canal during increased intracranial pressure can also decrease blood flow to the optic nerve causing ischemia.[5]

Pathogenesis

Forces from shearing injury cause tears to the microvasculature that are seen as hemorrhage in the optic nerve and its sheaths on pathology. Indirect trauma to the axons may also cause a focal area of impaired axonal transport. This functional separation of the nerve into a proximal and distal segment usually occurs within 6 to 24 h of injury.[50] The distal segment that is separated from the soma undergoes Wallerian degeneration. The proximal segment that is connected with the soma swells to produce a retraction ball. The soma then may undergo apoptosis, as shown in studies of optic nerves after ischemic optic neuropathy, with experimental glaucoma, and after trauma.[51–53]

Apoptosis is programmed cell death involving active cellular processes through final common pathways. Injured retinal ganglion cells release extracellular glutamate that induces excitotoxicity. High glutamate concentrations activate *N*-methyl-D-aspartate (NMDA) receptors that allow entry of excessive calcium into the cell. It has been shown that optic nerve crush leads to an increase in extracellular vitreal glutamate, but the steps by which axotomy induces excitotoxic damage to ganglion cells is still being studied.[54] This abnormally high concentration of calcium leads to inappropriate activation of cascades of proteases, nucleases, and lipases that attack cellular constituents, leading to the generation of highly reactive free radicals. The final stage of apoptosis, execution, occurs through the activation and function of caspases, aspartate-specific cysteine proteins. There are at least 10 homologues of the initially described caspase, interleukin-1-beta-converting enzyme (ICE).[55] The predominant caspase involved in cell death appears to be CPP32 (caspase-3). Caspase inhibitors may be a possible therapeutic target (see Management section).

Intracellular calcium also activates inducible nitric oxide synthase (NOS) to cause increased production of nitric oxide, a highly reactive free radical used for the signaling and regulation of various physiological processes that also induces apoptosis. Free radicals from various sources cause intracellular degeneration and activate the early steps of the apoptotic cascade.[56–59] Retinal ganglion cells do not have NOS.[60] NOS-mediated excitotoxic cell damage relies on the abundant amounts of inducible NOS expressed in reactive astrocytes. In situ hybridization shows intense nitric oxide synthase mRNA signals in the ganglion cell layer and inner nuclear layer, indicative of neuronal NOS proteins being transported through axons into the terminals in the inner nuclear layer. Neuronal NOS appears to play a role in retinal ganglion cell excitotoxicity mediated via the NMDA receptor.[61]

In addition, the excess calcium can also directly cause mitochondrial failure, causing depletion of energy and the generation of more free radicals. Partial ischemia and reperfusion of transiently ischemic areas may generate further oxygen free radicals.[62] The release of these oxygen free radicals leads to peroxidation of lipids in the retinal ganglion cell membrane.[63]

Bradykinin and kallidin initiate the release of arachidonic acid from neurons. Through a series of steps, arachidonic acid is transformed into various types of prostaglandins and oxygen free radicals are released. Peroxidation of lipids in the cell membrane may lead to decreased vascular autoregulation and increasing cellular/tissue edema. This type of edema within the optic canal may then produce a compartment

syndrome causing more ischemia to the optic nerve. Loss of regulation of calcium homeostasis leads to shifting of extracellular calcium to the intracellular space by voltage-gated and receptor-gated calcium channels. The excess intracellular calcium leads to cell death.[63]

Besides ischemia, inflammation contributes to further neural damage. Mediators of inflammation are released to attract polymorphonuclear lymphocytes and macrophages. Within the first 2 days after injury, polymorphonuclear lymphocytes predominate to cause immediate tissue damage. They are then replaced by macrophages by about 7 days after injury. These macrophages are thought to contribute to delayed tissue damage, as in delayed posttraumatic demyelination. Macrophages release glial promoting factors. This astroglial response after spinal cord injury may inhibit axonal regeneration processes. Inhibition of macrophage responses have been shown to decrease reactive gliosis, as shown in spinal cord injury studies.[62,63]

Management

Currently, the use of systemic corticosteroids in traumatic optic neuropathy is accepted as some form of treatment that is better than none at all. The beneficial effects of this medication are extrapolated from those shown in the treatment of acute spinal cord injury in the second National Acute Spinal Cord Injury Study (NASCIS-2).[64] In NASCIS 2,[64] a multicenter, randomized, double-blind, placebo-controlled study in patients with acute spinal cord injury, patients were randomly assigned to receive placebo, naloxone, or methylprednisolone within 12 h of spinal injury. Methylprednisolone was given as an initial dose of 30 mg/kg followed by a continuous infusion of 5.4 mg/kg/h. Compared with placebo, treatment with methylprednisolone within 8 h of injury resulted in a significant improvement in motor and sensory function. These effects of methylprednisolone in the treatment of spinal cord trauma do not seem to extend to the treatment of optic nerve trauma, however. The International Optic Nerve Trauma Study in 1999[65] showed that neither corticosteroid treatment nor optic canal decompression changed the visual outcome of patients with traumatic optic neuropathy. It was clinically reasonable to consider treatment on an individual patient basis. In this prospective observational study, visual outcomes were compared with patients following observation alone, high-dose steroids given within 7 days of the injury, and optic canal decompression with or without corticosteroids and performed within 7 days of the injury. The initial visual acuity of NLP predicted a poor outcome in all groups. No clear benefit was demonstrated for patients undergoing high-dose steroid therapy, or canal decompression surgery compared to observation alone. The 57% improvement of 3 Snellen lines or more in the untreated group suggested that spontaneous visual recovery also played a role in visual outcome. Some studies[66,67] have even shown that methylprednisolone exacerbated axonal loss after optic nerve crush injury in rodent models. In a more recent study by Ohlsson et al.,[68] however, methylprednisolone showed no effect on retinal ganglion cell survival, macrophage activity at the site of injury, axonal degeneration/regeneration, or visual function. These results could explain the lack of efficacy demonstrated in the International Optic Nerve Trauma Study, in which there was no clear benefit for either corticosteroids or optic canal decompression. No randomized, double-blind clinical studies to date provide evidence that methylprednisolone is more effective than observation in the treatment of optic nerve trauma.

Surgical decompression of the optic canal for intracanalicular traumatic optic neuropathy has a limited role in the management of traumatic optic neuropathy. This treatment is based on the hypothesis that swelling in the optic canal may lead to a compartment syndrome. The increasing edema would decrease tissue perfusion to cause more postinjury ischemia to the optic nerve. This procedure is thought to decrease edematous pressure in the optic canal to reverse ischemia and axonal conduction block, which can result in irreversible axonal degeneration.[69,70] In the International Optic Nerve Trauma Study in 1999,[65] no clear benefit was demonstrated for patients who were given high-dose corticosteroids within 7 days of injury

compared to those who underwent optic canal decompression within 7 days of injury with or without corticosteroids. About 57% of the untreated group experienced a spontaneous improvement of visual acuity of 3 lines or more. The initial visual acuity of NLP predicted a poor prognosis. According to a review by McCann and Seiff,[71] 28% of patients with traumatic optic neuropathy have some spontaneous improvement in vision. Based on combined unmatched uncontrolled human studies, vision improves in approximately 50% of patients treated with corticosteroids, 57% of patients treated with optic nerve decompression, and 62% of patients treated with corticosteroids and optic nerve decompression.[71] Most patients with a response to corticosteroids will have improved 1 week after initiating treatment.[72] Of patients treated with corticosteroids who did not improve after 3 weeks of observation, 51% still benefit from surgery.[71,72] The final visual outcome was not correlated with the interval between injury and surgical intervention.[5,71–74] Therefore, optic canal decompression surgery has a limited role in the management of traumatic optic neuropathy, it is appropriate for conscious patients without injuries to the globe who also have progressive visual deterioration.

Management guidelines for direct and indirect traumatic optic neuropathy[15,75–77] should be individualized according to the patient's situation. In the review by Bilyk and Joseph,[12] it is recommended that an eye examination must rule out other etiologies of decreased vision and exclude occult ruptured globe. If a subperiosteal hematoma is present, then a canthotomy and cantholysis should be performed for drainage. If there is no contraindication to corticosteroids and it is within 8 h of injury, then methylprednisolone at 30 mg/kg/h IV is immediately given as a loading dose, and then 4.0 mg/kg/h continuous IV infusion for 24 h, or an additional 15 mg/kg 2 h later and 15 mg/kg every 6 h for up to 72 h. According to most clinicians, if the patient is evaluated beyond 24 h of injury, then no corticosteroids are indicated. High-resolution CT scan of the orbit and sinuses with 1.5-mm axial and coronal sections with bone windows should be performed to include the optic canal and cavernous sinus. If bony fragments are seen to impinge on the intracanalicular optic nerve, or if no fracture or a fracture without obvious impingement is seen on CT scan, then an optic canal decompression should be considered. Visual function needs to be monitored every 2 to 4 h for the first 12 h. If vision improves on high-dose corticosteroids, then an oral prednisone taper is recommended after 72 h of IV steroids. If vision does not improve during the first 12 to 24 h of high dose corticosteroids, then an optic canal decompression can be offered to the patient. Patients who are unconscious and who have injuries to the globe should not undergo surgery. If the optic nerve injury is more than 7 days old, then surgery is also not an option.

According to Sofferman,[78] adequate decompression of the optic canal requires (1) removal of at least 50% of the circumference of the osseous canal; (2) removal of bone along the length of the canal; and (3) total longitudinal incision of the dural sheath including the annulus of Zinn. In cases where visual loss is progressive and delayed with the evolution of an intrasheath hematoma, surgical decompression of the intracanalicular nerve has been proposed.[14,76,77]

There are several possible approaches to the decompression of the optic canal. (1) Goldberg and Steinsapir[79] recommend a transethmoidal/transorbital approach, which allows removal of more than 180° of the bone. Some recent reports have demonstrated that the endoscopic ethmoidectomy technique may offer another surgical option.[80] In a report of 31 patients[81] with indirect traumatic optic neuropathy who received methylprednisolone injections and endoscopic optic nerve decompression, 70% of 23 patients who started treatment before 7 days after injury experienced visual improvement.

Lateral canthotomy and cantholysis are necessary for orbital hemorrhage/edema causing a compressive orbitopathy and optic neuropathy. Orbital CT should then be performed to rule out a subperiosteal hemorrhage or other ocular pathology that could account for the visual loss. If vision does not improve, orbital decompression may need to be considered to allow expansion of orbital soft tissues.[79]

In children and adolescents, traumatic optic neuropathy is caused by mechanisms similar to those that cause it in adults. In a retrospective review of 40 children,[82] treatment did not improve visual outcome. The severity of visual loss and rate and degree of improvement are also similar. The most common causes were motor vehicle accidents (62%) and sports injuries (22%). Trauma was blunt in 78% of cases and penetrating in 22%. Improvement was more likely when vision was 20/200 or better at presentation, regardless of treatment. Patients with NLP acuity at presentation rarely experienced significant visual improvement despite treatment. Severe initial visual loss with baseline NLP and the presence of a fracture in the optic canal on CT scan were poor prognostic signs, predictive of poor visual outcome. Three patients in this series had an improvement of at least 2 Snellen lines from a baseline of NLP after treatment.

New Perspectives in the Protection, Repair, and Restoration of the Injured Optic Nerve

Strategies for neuroprotection, defined as intervention to produce enduring benefits by favorable influencing underlying etiology or pathogenesis and thereby forestalling onset of illness or clinical decline,[83] have been investigated to prevent apoptosis in the management of traumatic optic neuropathy. It has recently been shown that the innate adaptive T-cell-mediated immune response directed against self-antigens located at the site of damage can be neuroprotective after optic nerve or injury. This protective autoimmune response is spontaneously evoked in some individuals, but not strongly enough to significantly affect recovery. By augmenting this response in individuals who spontaneously manifest it and by inducing this autoimmune response in those incapable of manifesting it, optimal neurological functional recovery was attained.[84–86] Protective autoimmunity is defined as a benign autoimmune response that contributes to the maintenance and protection of injured neurons and the promotion of recovery after traumatic injury to the neuron.[84,87] The mechanism of T-cell-mediated neuroprotection is not yet known. Vaccination of rats and mice with the synthetic copolymer Cop-1 after optic nerve crush injury leads to a significantly increased survival rate of retinal ganglion cells.[84,88] Compared to the untreated group, rats treated with antimyelin basic protein T cells had higher visual evoked potentials at days 1, 5, 7, and 15 after crush injury to the optic nerve.[89,90] Vaccination did not induce autoimmune disease based on repeated passive transfer experiments using T cells directed against myelin basic protein itself compared to the nonencephalitogenic cryptic epitope.[91] Similar neuroprotective efficacy was observed with both sets of T cells, suggesting that autoimmune disease was not an inevitable side effect of treatment with T cells that recognize myelin basic protein.

The benefits from T-cell-mediated neuroprotection is manifested by a reduction of secondary degeneration,[92] which may be induced by increased concentrations of glutamate, nitric oxide, or other mediators of toxicity. Augmenting or inducing the body's own mechanism for protective intervention in neural injury allows for more physiological mediated benefit with minimal risk.

NMDA receptor blockers, such as memantine and dizocilpine (MK801), and AMPA and kainite receptor blockers, such as NBQX and DBQX, have been shown to protect retinal ganglion cells after experimental optic nerve injury in animals.[62,63,93,94] To control nitric oxide synthase-mediated damage, *N*-nitro-l-arginase, nipradilol, and aminoguanidine can reduce nitric oxide or inhibit NOS for neuroprotection.[95]

Alpha-2-adrenergic receptors, such as brimonidine, have a neuroprotective effect on the experimentally injured optic nerve when brimonidine is injected intraperitoneally.[96,97]

Caspase inhibitors block apoptosis and may be important therapeutic molecules for the treatment of traumatic optic neuropathy and other neurodegenerative diseases.[98] Intraocular injection of various caspase inhibitors has been shown to salvage up to 34% of retinal ganglion cells from cell death after optic nerve transection.[99]

Because a lack of neurotrophins can lead to apoptosis, supplying the damaged neurons with neurotrophins may promote neuronal survival. These neurotrophins can be delivered directly to the injured neurons, or their genes can be transferred to the neurons by viral vectors. Fibroblast growth factor injected into the spinal cord of adult rats after injury prevents ventral horn neurons from death with improved respiratory function.[100] In experimental optic nerve crush injuries in cats, intravitreal injection of either brain-derived growth factor (BDGF) or glial-derived neurotrophic factor (GDNF) has been shown to increase the survival of retinal ganglion cells.[101–103] Several neurotrophins have been shown to increase glutamate transporter expression in culture and possibly to reduce excitotoxicity. Therefore, both caspase inhibitors and neurotrophins may delay retinal ganglion cell death in the setting of axonal injury.[101–103]

Because of the inherent axonal regeneration inhibitory molecules, such as Nogo, MAG, and OMgp, in the myelin and oligodendrocytes of the central nervous system (CNS), antibodies against these various molecules have allowed the optic nerve to regenerate in animals.[104] Antibodies against central myelin proteins added to crushed optic nerves in mice have resulted in axonal sprouting.[105] Blocking the Rho inhibitory pathway with enzyme C3 in mice has lead to axonal regeneration after transection and reconnection of the optic nerve in vivo.[106] Exogenous growth factors, such as BDNF, neurotrophin-3, and neurotrophin-4, can enhance further axonal repair and regeneration in the optic nerve and spinal cord.[100,107] Optic nerve transection has been shown to also lead to an increased level of GDNF and its receptors as an innate response to injury. Both endogenous as well as exogenous GDNF can support axotomized retinal ganglion cells in neuroprotection and neuroregeneration.[108] Optic nerve regeneration can also be induced by intraocular injection of dibutyryl cAMP. Dibutyryl cAMP has been shown to induce significant axonal regeneration through the crush site of injury of the optic nerve in mice.[109]

Gene therapy is still experimental and may offer limited restoration of vision in injured optic nerves, because this treatment slows the rate of ganglion cell loss after injury and does not prevent a significant proportion of injured cells from dying. A treatment that can stimulate the regeneration of axotomized retinal ganglion cells would be needed.[110]

Furthermore, Schwann cells and fibroblasts isolated from the peripheral nerve can promote retinal ganglion cell survival after optic nerve transection, possibly by secreting neurotrophic factors. In adult rats with transected optic nerves, Schwann cells and fibroblasts from peripheral nerve have been transplanted intravitreally to promote intraretinal axonal sprouting.[111] In a study by Vidal-Sanz et al.,[112] up to 10% of the retinal ganglion cells grew an axon into the peripheral nerve graft connecting the severed optic nerve with the superior colliculus in the rat. Anatomic synapses were demonstrated.

Substances that guide axonal growth towards the target neurons, such as integrins, are inherently present for the appropriate synaptic connections to be established after optic nerve injury. Retinal ganglion cells can navigate their way to synapse correctly to form retinotopic connections, which has been shown histologically in animal models following optic nerve transection and peripheral nerve grafting.[113,114] In a study by Thanos et al.,[115] peripheral nerve grafting between both severed optic nerves and the optic tract allowed axons to reinnervate the major visual targets in the midbrain and thalamus. Restoration of visual function was confirmed by the animal's ability to discriminate spatial patterns and by the presence of visual evoked cortical potentials.

Stem cell implantation offers more hope for the restoration of vision. These cells are pluripotent and differentiate into any neural cell type to integrate with host cells. Stem cells from the retina and the ciliary body of human embryos can be induced to differentiate into retinal ganglion cells.[116] Another possible and still experimental technique is to harvest cells from the host's ciliary body that have the potential to develop into retinal neuronal and glial cells.[117] Stems cells from the ependymal zone of the hippocampus[118] can be placed in the subretinal space to allow engraftment into the retina and

growth of axons toward the host optic nerve. In humans, a sural nerve graft may need to be placed in the opening of the sclera, choroids, and retina on the nasal side of the optic disc with the other end of the graft in contact with the ipsilateral geniculate body.[119]

Gene therapy can help slow the rate of retinal ganglion cell loss after axotomy. Cheng et al.[120] showed that adeno-associated virus-mediated gene transfer to increase the expression of the trkB receptor by retinal ganglion cells to stimulate the receptors with intravitreally delivered BDNF would then increase neuronal survival after optic nerve transection. With this technique, 76% of retinal ganglion cells remained alive at 2 weeks after axotomy, compared to less than 10% of the neurons without treatment. Similar slowing of retinal ganglion cells has been shown with adenoviral delivery of X-chromosome-linked inhibitor of apoptosis (Ad. XIAP) to the optic nerve stump. After adding intravitreal adenovirus encoding glial cell line-derived neurotrophic factor (Ad.GDNF), 47.3% of retinal ganglion cells were rescued from apoptosis 2 weeks after transection.[121] Although these animal models yield promising therapy, the slowing of neuronal death after axonal injury is unlikely to be sufficient for visual function in humans. Treatment that can either prevent a significant proportion of injured neurons from apoptosis or which can stimulate the regeneration of axotomized retinal ganglion cells is needed.

Traumatic Optic Neuropathy as a Complication of Ocular Surgery

Surgical procedures in and around the optic nerve are becoming important causes of direct and indirect optic nerve trauma.

Optic Nerve Injury Related to Periorbital Injections

Anesthetic injections at the orbital apex may cause direct optic nerve injury.[122–125] Katsev et al.[126] recommended that the needle introduced beyond the orbital rim for both intraconal and periconal injections be no longer than 31 mm to avoid damage to the optic nerve. MRI of the orbit often reveals localized edema of the optic nerve or optic nerve sheath enhancement with gadolinium. The hyperintense signal from CSF around the injured optic nerve may be absent on T_2-weighted imaging when compared with the normal nerve. Optic nerve injury has been reported with other procedures with a blunt cannula and in sub-Tenon's injection.[125]

An MRI of the orbits with T_1 fat saturation and gadolinium is recommended for any optic neuropathy occurring within the first 24 h of periocular injection. If findings of needle injury are present, then a trial of high-dose corticosteroids should be considered, although corticosteroids have not been proven to be efficacious. In treatment with corticosteroids, in three of four patients[122–124] only one patient had partial visual recovery.[5–7]

Optic Nerve Injury after Cataract Surgery

Cataract surgery is one of the most common ocular surgeries, and visual loss is a rare complication. Nonarteritic ischemic optic neuropathy (NAION) following uncomplicated cataract surgery with either periocular anesthesia or general anesthesia has been reported.[127–131] In a retrospective study by McCulley et al.,[132] 2 of 5787 patients developed ischemic optic neuropathy within 6 weeks of cataract surgery, but 1 had previous NAION in the other eye 21 months earlier. In another study by McCulley et al.,[133] all 18 cases of NAION in the 17 patients occurred within 6 months of surgery. These data help confirm that intraocular lens surgery is associated with the occurrence of NAION.

Visual loss may present with optic disc edema as an anterior ischemic optic neuropathy or with a normal disc as a posterior ischemic optic neuropathy.[128,129] NAION after cataract surgery may occur within several hours to 4 to 6 weeks postoperatively. It has been shown that if ischemic optic neuropathy occurs in one eye after cataract surgery, the risk of recurrence in the other eye may be as high as 30% to 50% with subsequent surgery.[127,131] It has been postulated that an increase in intraocular pressure during

the postoperative period may contribute to the development of NAION. In a study of 11 patients with NAION after cataract extraction, Hayreh[128] postulated that the typical increase in intraocular pressure after surgery along with a decrease in systemic blood pressure during general anesthesia lead to decreased perfusion of the optic nerve head and ischemia. The type of anesthesia during surgery was not specified in this case series. There are also other studies that document affected patients with normal intraocular pressures in the perioperative period.[127,134]

Most patients experience spontaneous improvement in visual acuity. Corticosteroids have not been shown to be effective. Most clinicians monitor the intraocular pressure in the perioperative period to help prevent the first ischemic event. Because the risk of NAION to the other eye is high, subsequent contralateral cataract extraction is not recommended.[128]

Optic Nerve Injury after Vitrectomy

Ischemic optic neuropathy after vitrectomy to relieve vitreous traction in macular holes is related to surgical manipulation and to local anesthesia. After a pars plana vitrectomy, the cortical vitreous is peeled off the retina, at least around the hole. Aspiration of the cortical vitreous around the optic nerve head may occasionally extend to the equator. This suctioning process of the posterior hyaloid may shear peripapillary axons. Near the nasal edge of the disc, an air–fluid exchange is performed that can cause direct pressure to the optic nerve. At the end of the procedure, the eyes are usually filled with long-acting gas, such as perfluoropropane or sulfur hexafluoride. Patients are then required to position themselves face-down for at least 20 h per day for about 2 weeks. Because the intraocular pressure may rise in the immediate postoperative period to cause optic nerve head ischemia, topical medications or oral acetazolamide are given to patients to help prevent this complication.[135–140]

Postoperatively, patients may develop visual field defects. In a study by Melberg and Thomas,[139] 3 of 157 patients had temporal visual field defects after vitrectomy. In 2 of 3 patients the visual field defects were beyond the central 30° of fixation and could only be detected by Goldmann perimetry. These absolute defects could not be attributed to retinal detachment or schisis. As all patients underwent general anesthesia, injury to the optic nerve was unlikely. It was hypothesized that direct trauma to the optic nerve during aspiration of the air–fluid exchange procedure caused these field defects. In another study,[138] 8 patients experienced visual loss after vitrectomy and all had retrobulbar injections; 7 of 8 patients had fluid–air exchange with long-acting gas; 4 of 8 patients developed afferent pupillary defects, and 4 of 8 developed inferotemporal field defects. Five of 8 had optic disc pallor without associated disc edema. No branch retinal vein or branch retinal artery occlusions occurred. The few retinal detachments did not explain the visual field defects. It was postulated that direct trauma to the optic nerve occurred, either by mechanical pressure from the suction catheter tip during air–fluid exchange or by injury from the needle during retrobulbar injection. Indirect trauma secondary to suction on the posterior hyaloid or shearing of peripapillary axons was another possibility. Although increased intraocular pressure may cause ischemic optic neuropathy, no increase in intraocular pressure was noted during the surgery. In another study, Boldt et al.[135] suggested that direct compression from the gas bubble itself could cause enough pressure to damage the nerve fiber layer. The retina may also have toxic injury from the gas bubble itself. Other studies confirm the common finding of temporal field defects with vitrectomies for macular holes.[136,137] The incidence of inferotemporal or temporal visual field defects ranges from 1.9% to 20.5%. In the study by Paques et al.,[140] 10% of patients had arcuate defects.

Optic Nerve Injury after Trabeculectomy

Visual loss after trabeculectomy is not common. Patients who seem to be at highest risk for significant visual loss after trabeculectomy are those who have advanced glaucoma with preexisting severe visual loss. These high-risk patients often have a field defect that splits fixation or extends within 5° of fixation. Other patients with postoperative hypotony may be

predisposed to ischemic optic neuropathy. In a study of 508 eyes of 440 patients,[141] only 4 cases (less than 1%) of visual loss were observed. These 4 patients had preoperative visual field defects that split fixation, a finding consistent with a study by Kolker.[142] Three of these patients also had postoperative hypotony in which the intraocular pressures ranged from 0 mmHg to 2 mmHg on postoperative day 1. The low pressures persisted for 1 week in 2 eyes and for 1 month in 1 eye. Disc edema was not observed, but another study by Kawasaki and Purvin[143] described 2 patients who developed unilateral optic disc edema after trabeculectomy with intraocular pressures in the low-normal range. Because they did not have severe glaucoma, they did not experience visual field defects. Therefore, intraocular pressures that are too low might predispose to optic nerve injury.

Optic Nerve Injury after Blepharoplasty

Optic nerve injury is uncommon after blepharoplasty. Blindness is estimated at 0.04%.[144] Visual loss is most likely caused by compression of the optic nerve or of the central retinal artery by a retrobulbar hematoma. The visual loss is reversible with prompt treatment. In a report by Kelly and May,[145] a patient developed unilateral blindness from a retrobulbar hemorrhage after lower eyelid blepharoplasty. No blood flow was seen in the retinal arterioles, but after immediate lateral cantholysis and drainage, the patient regained retinal perfusion and recovered normal vision within 72 h. Other similar cases of optic nerve dysfunction secondary to compression by a retrobulbar hematoma after blepharoplasty have been reported with documented abnormal visual evoked potentials and normal electroretinogram.[146,147]

Optic Nerve Injury after Endoscopic Sinus Surgery

PION following intranasal anesthetic injection has been thought to be related to submucosal injection of an anesthetic with epinephrine, causing vasospasm.[148]

Traumatic optic neuropathy can rarely be seen after endoscopic sinus surgery. The lack of orientation to surgical landmarks predisposes the surgeon to this complication.[149] The optic nerve canal indents the lateral wall of the sphenoid sinus, an important surgical landmark. Onodi cells usually cover the sphenoid sinus, but can also surround the optic nerve to cause confusion in surgical anatomy. CT scan cannot reliably identify these cells to help prevent this problem.[150]

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