# Optic Nerve Sheath Fenestration—Indications and Techniques

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## Abstract

The optic nerve is a white matter tract of the brain and its sheath is composed of pia mater, arachnoid mater, and dura mater. The subarachnoid space (SAS) of the optic nerve sheath, which contains cerebrospinal fluid, is contiguous with the SAS of the brain. Elevated intracranial pressure is transmitted to the optic nerve head through the SAS resulting in papilledema. Optic nerve sheath fenestration (ONSF) can be an effective surgical technique to preserve vision in patients with progressive optic neuropathy due to papilledema caused by idiopathic intracranial hypertension (IIH); however, a dreaded complication of ONSF is visual loss. This article will discuss the rationale, indications, and complications of ONSF. A variety of surgical techniques can be employed to perform an ONSF. In particular, the medial transconjuctival approach will be highlighted.

## Keywords

Papilledema, optic nerve, idiopathic intracranial hypertension, optic nerve sheath fenestration

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Optic nerve sheath fenestration (ONSF) is a surgical procedure used by ophthalmologists—particularly neuro-ophthalmologists and orbital specialists—to reduce the pressure within the subarachnoid space (SAS) of the optic nerve. The first notation of ONSF dates back to 1872 when it was described as a technique to relieve optic nerve head (ONH) edema, but at that time there was not a clear understanding of the pathogenesis of ONH edema.[1]

Subsequent landmark experiments by Hayreh validated the scientific use of ONSF in cases of ONH edema secondary to elevated intracranial pressure (ICP), also known as papilledema.[2–8] Modern day studies have shown that ONSF can improve visual function in numerous conditions that produce ONH edema—especially in cases of visual loss secondary to papilledema caused by idiopathic intracranial hypertension (IIH). Unfortunately, much confusion arises from terminology used in the early literature that loosely applied the term papilledema to ONH edema due to a variety of pathologies. The appropriate use of papilledema is in the scenario of ONH edema due to elevated ICP. It is important to differentiate papilledema from ONH edema caused by local or systemic processes in the absence of elevated ICP (see *Table 1*).[9,10] Additionally, the clinician should recognize pseudopapilledema, a condition that can mimic papilledema, is associated with elevation and an indistinct optic disc margin (see *Figure 1*).

This article's purpose is to review the regional neuroanatomy of the optic nerve and the relationship of the SAS between the optic nerve and brain. In addition, we will discuss the rationale, indications, limitations, complications, and surgical techniques of ONSF. Finally, we will detail our preferred method for performing an ONSF.

## Anatomy

Prior to undertaking a discussion of ONSF, we will outline the anatomy of the optic nerve, the optic nerve sheath, and the relationship of the SAS between the optic nerve and brain.

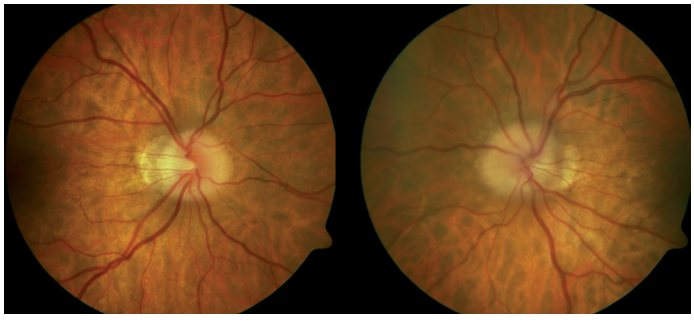
### Optic Nerve

The optic nerve is anatomically divided into four distinct segments as it courses from the eye to the optic chiasm (see *Figure 2*). The optic nerve sheath refers to the three meningeal membranes—dura mater, arachnoid mater, and pia mater—that cover the optic nerve.

### Optic Nerve Head

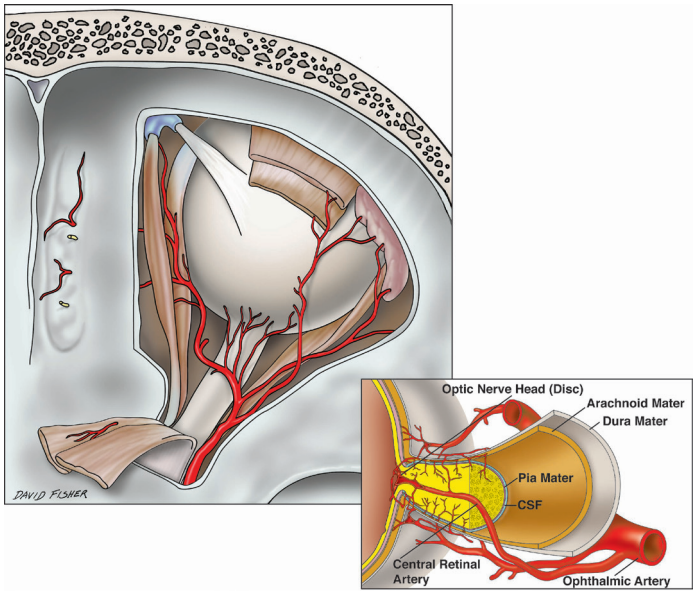
The first segment of the optic nerve is the ONH (or optic disc) located at the insertion of the nerve into the eye. The ONH represents the convergence of approximately 1.2 million axons of the retinal ganglion cells (RGCs). The ONH, which measures 1 mm in length and 1.5 mm in diameter, is represented by the physiologic blind spot on perimetry testing and is located approximately 4 mm nasal from the center of the macula (i.e. fovea). The ONH receives its blood supply from the circle of Zinn-Haller and the posterior ciliary arteries, which are branches of the ophthalmic artery.

## Figure 1: Fundus Photograph of a Patient with Myopic Tilted Optic Discs Giving the Appearance of Pseudopapilledema



*Note that although the margins of the optic nerve head are indistinct, there is no obscuration of the overlying blood vessels. This patient was seen for a routine eye examination and had no visual or neurologic complaints. Other causes of pseudopapilledema include congenital optic disc anomaly, optic disc drusen, and a hypoplastic optic disc.*

## Figure 2: Superior view of the Intraorbital Optic Nerve




*A window is created through the orbital roof. The superior rectus muscle is reflected to allow visualization of the optic nerve. Inset shows the detailed anatomy of the optic nerve sheath. Illustration by David Fisher CMI. CSF = cerebrospinal fluid.*

### Intraorbital Optic Nerve

The second segment of the optic nerve is the intraorbital optic nerve. At the ONH, the unmyelinated axons of the retinal nerve fiber layer (RNFL) make a 90° turn to exit the eye. The lamina cribrosa, a distinct region of the sclera consisting of stacks of fenestrated sheets of elastic fibers and connective tissue, allows the passage of the optic nerve axons from the eye into the retrobulbar orbital space. After passing through the lamina cribrosa, the axons become covered by myelin derived from oligodendrocytes. The presence of myelin increases the diameter of the intraorbital optic nerve to approximately 3 mm. Posterior to and continuous with the sclera, the optic nerve procures a dural sheath, in addition to the arachnoid mater and pia mater. A unique anatomical feature of the intraorbital optic nerve is the fact that its length (28 mm) is nearly double the distance from the back of the eye to the orbital apex (15 mm). This configuration allows for the globe to freely rotate within the orbit and to compensate for any pathologic axial shifts within the orbit without causing visual dysfunction. The blood supply of the intraorbital optic nerve is derived from the pial network of vessels from the ophthalmic artery.[9]

## Table 1: Causes of Optic Disc Edema (Non-papilledema)

| |
|---|
| **Ischemic** |
| • Nonarteritic anterior ischemic optic neuropathy |
| • Arteritic anterior ischemic optic neuropathy |
| **Inflammatory** |
| • Optic neuritis, optic perineuritis |
| • Thyroid-related optic neuropathy |
| • Uveitis |
| • Sarcoidosis |
| **Infectious** |
| • Tuberculosis |
| • Neuroretinitis due to cat scratch disease |
| • Syphilis |
| **Neoplastic** |
| • Infiltrative optic neuropathy from tumor cells (leukemia, metastases) |
| • Orbital tumor (capillary hemangioma, meningioma) |
| • Optic nerve tumor (optic nerve sheath meningioma, optic nerve glioma) |
| • Compressive optic neuropathy from intracranial mass lesion |
| **Traumatic** |
| • Traumatic optic neuropathy |
| **Hereditary** |
| • Leber hereditary optic neuropathy |
| **Toxic/Metabolic** |
| • Amiodarone |
| • Methanol, ethylene glycol |
| • Disulfiram |
| • Cyclosporin, tacrolimus |

### Intracanalicular Optic Nerve

The intracanalicular optic nerve is the third segment of the optic nerve, and begins at the point where the optic nerve enters the optic canal. At the orbital apex, the dura mater covering the optic nerve fuses with the periorbita of the orbit. It is also at this location that the optic nerve is encircled by the annulus of Zinn represented by the tendinous insertions of the four recti muscles. The intracanalicular portion of the optic nerve is anchored within the optic canal, which measures approximately 8–10 mm in length and 5–7 mm in width. The intracanalicular optic nerve represents a watershed zone because it has a dual vascular supply, anteriorly from branches of the ophthalmic artery and posteriorly from small vessels arising from the internal carotid artery and the superior hyphophyseal artery.[9]

### Intracranial Optic Nerve

The fourth and final segment of the optic nerve is the intracranial optic nerve. The optic nerve enters the cranial vault underneath the anterior clinoid process and over the ophthalmic artery. Upon exiting the optic canal, the dura of the optic nerve fuses with the periosteum of the middle cranial fossa. The nerve then travels a variable distance, ranging from 8–12 mm, before joining the optic chiasm. The intracranial optic nerve is supplied by branches from the internal carotid artery, the superior hypophyseal artery, anterior cerebral artery, and anterior communicating artery.[9]

## Optic Nerve Sheath and Subarachnoid Space

The optic nerve is a misnomer because it is not a peripheral nerve but rather a central nervous system (CNS) white matter tract. As a result of this common lineage between the optic nerve and the CNS, the SAS of the optic nerve is contiguous with the SAS of the brain. As mentioned above, just posterior to the sclera, the optic nerve is surrounded by the leptomeninges. The arachnoid membrane of the optic nerve functions to support and protect the underlying axons. The arachnoid membrane of the optic nerve is continuous with the arachnoid membrane of the subdural intracranial space and allows for the free circulation of cerebrospinal fluid (CSF) around the optic nerve and brain.

By virtue of the fact that the optic nerve sheath serves as a CSF conduit between the brain and the eye, pathology involving the contents of the cranium can lead to pathology of the ONH. Particularly, CNS pathology characterized by increased ICP, including intracranial masses, infectious diseases, inflammatory diseases, and IIH, can impact the ONH, both structurally and functionally. When raised ICP is transmitted to the SAS within the optic nerve sheath, ONH edema ensues. Studies in Rhesus monkeys have shown that papilledema is the first ophthalmoscopic sign of raised ICP.[2,4–8] Investigations examining the pathophysiology of papilledema have shown that axonal swelling at the ONH occurs due to a disturbance in the axoplasmic transport system (see *Figure 3*).[2,4–8,11] Nerve fiber dysfunction due to axonal swelling can result in loss of central vision, a decrease in peripheral vision, and, ultimately, optic atrophy.[11]

The exact relationship between the CSF dynamics of the brain SAS and optic nerve SAS has not yet been fully elucidated. Historically, it has been generally accepted that there is free bi-directional communication between these two spaces. Recently, studies in patients with optic nerve pathology have shown chemical and radiographic evidence of compartmentation of the SAS of the optic nerve, suggesting that the optic neuropathy due to increased ICP may occur as a result of CSF collection at the ONH.[12] Computed tomography-cisternography performed in patients with papilledema has shown reduced contrast-loaded CSF in the SAS of the optic nerve compared with the basal cistern, suggesting sequestration of CSF in the SAS of the optic nerve.[13] It has also been shown that patients with papilledema have a higher concentration of lipocalin-like prostaglandin D synthase (L-PGDS) in the SAS of the optic nerve compared with the lumbar CSF, further supporting the CSF sequestration theory.[13]

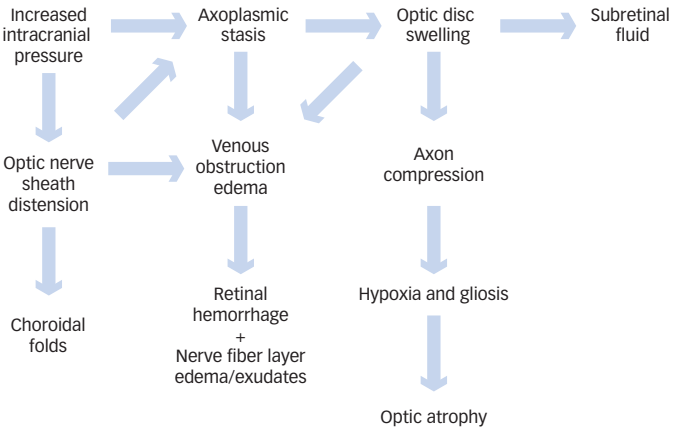
## Rationale for Optic Nerve Sheath Fenestration

ONSF is a surgical technique to reduce the hydrostatic pressure on the ONH. From a purely mechanical perspective it is believed that an opening within the optic nerve sheath will allow for a sudden and sustained drop in the SAS pressure and relief of the compartment syndrome on the ONH. However, the exact mechanism by which ONSF can relieve ONH edema is not entirely clear. It has been theorized that ONSF works by creating a CSF filter from the SAS of the optic nerve into the surrounding orbital tissue, thereby reducing the CSF volume and pressure surrounding the ONH. Magnetic resonance imaging conducted on patients in the early postoperative period after ONSF have demonstrated a fluid collection adjacent to the fenestration site that disappeared in the late postoperative period.[14] Some patients who have undergone ONSF developed a cyst-like structure adjacent to the fenestration site, further implying a CSF filtration process.[15] Additional support to the CSF filtration theory was achieved by creating a model using Bernoulli's equation of fluid dynamics. According to Bernoulli's equation, increased velocity of fluid leads to a decrease in pressure of that fluid. Based on this model, ONSF is thought to increase the velocity of CSF in the optic nerve sheath and thereby decrease the CSF pressure transmitted to ONH.[16] The theory of CSF filtration induced by ONSF may help explain the clinical finding of decreased ONH edema in the fellow unoperated eye after unilateral ONSF despite persistent elevated ICP.[16] An alternative theory for the relief of ONH edema based on histopathologic and radiologic findings suggests that ONSF promotes fibrous tissue proliferation at the incisional site, thereby preventing the transmission of elevated CSF pressure to the ONH.[17,18]

## Indications for Optic Nerve Sheath Fenestration

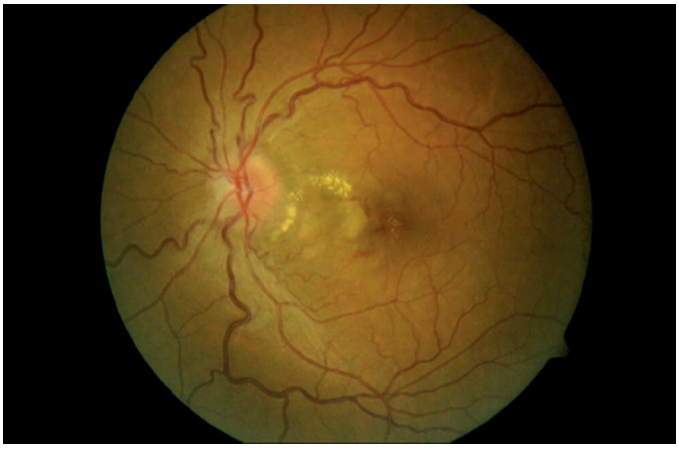
ONSF has been tried in the setting of many optic neuropathies and has been found to improve visual function in some cases and worsen vision

### Figure 3. Schematic Diagram of the Pathogenesis of Papilledema




*Reproduced with permission from Schirmer and Hedges.[11]*

### Figure 4: Photograph of a Patient with Idiopathic Intracranial Hypertension who Noted Sudden Visual Loss in the Left Eye



*There are hard exudates and hemorrhage in the macula consistent with a subretinal neovascular membrane. Other causes of vision loss due to papilledema include papilledema-induced optic neuropathy, macular edema/exudates, central retinal vein occlusion, and ischemic optic neuropathy.*

**Table 2: Clinical Criteria for Diagnosing Idiopathic Intracranial Hypertension**

- Symptoms and signs attributed to increased ICP
- Documented elevated ICP during lumbar puncture measured in the lateral decubitus position with legs and head in a straight and relaxed position
- Normal cerebrospinal fluid composition (normal cell count, normal glucose, normal protein)
- No evidence of ventriculomegaly, mass, structural, or vascular lesion on magnetic resonance imaging or contrast-enhanced computer tomography, normal magnetic resonance venography imaging
- Normal neurologic exam, with the exception that patient may have a sixth nerve palsy

*ICP = intracranial pressure. Adapted from Friedman and Jacobson.[30]*

in others. To date, there has not been a randomized, prospective trial evaluating the efficacy of ONSF in papilledema. However, ONSF is most commonly indicated for optic neuropathy in the setting of raised ICP, particularly in the setting of medically resistant progressive visual loss due to IIH.

## Optic Nerve Sheath Fenestration for Various Optic Neuropathies

The utility of ONSF in improving visual outcome of optic neuropathies from a variety of etiologies has been reported in small case series and case reports. In a case series of three patients with progressive visual loss due to radiation-induced optic neuropathy, ONSF improved visual function in all the patients.[19] ONSF was found to be effective in patients with postdecompression blindness, a syndrome characterized by visual loss due to a 'compartment syndrome' in the intraorbital optic nerve after removal of a CNS lesion.[20,21] Additionally, sporadic case reports have described improvement in visual function after ONSF in patients with osteopetrosis, optic nerve glioma, and chronic inflammatory demyelinating polyneuropathy.[22–24]

ONSF has also been utilized successfully in traumatic optic neuropathy. Guy et al. demonstrated reversal of progressive visual loss after ONSF in two patients with traumatic optic neuropathy. One of the patients had an optic nerve hematoma and the other developed delayed visual loss from an arachnoid cyst of the optic nerve sheath.[25] Another case series described improvement in visual function after ONSF in a patient with traumatic optic neuropathy and radiographic evidence of an optic nerve sheath hematoma.[26] This same case series showed improvement in visual function in three patients with traumatic optic neuropathy who underwent ONSF combined with optic canal decompression.[26]

Based on retrospective case series and case reports, there was enthusiasm for ONSF as an effective treatment for nonarteritic anterior ischemic optic neuropathy (NAION), the most common cause of an acute optic neuropathy in patients older than 55 years of age.[27] The National Eye Institute (NEI) funded a prospective multicentered study to assess the efficacy of ONSF in acute NAION. The Ischemic Optic Neuropathy Decompression Trial (IONDT) was a randomized, placebo-controlled trial that found ONSF was not more effective than observation in the final visual outcome of patients with NAION. In addition those patients who underwent ONSF had a greater risk of losing vision compared with the observation group.[28,29]

## Optic Nerve Sheath Fenestration for Elevated Intracranial Pressure

Papilledema can lead to visual loss from optic nerve or retinal (macular) disease (see *Figure 4*). ONSF is indicated in some cases of papilledema, most commonly for visual loss due to IIH.

IIH is typically encountered in obese women in their second, third, and fourth decades of life; and is characterized by normal neuroimaging and elevated CSF pressure with normal CSF studies (see *Table 2*). In most cases, IIH can be effectively treated medically with the use of an ICP lowering agent such as acetazolamide and a weight-reduction program.[30] Surgical treatment of IIH is achieved through ONSF, a CSF diversion procedure (i.e. lumboperitoneal or ventriculoperitoneal shunt), or venous sinus stenting and is reserved for those patients who cannot tolerate medical therapy or develop progressive symptoms despite maximal medical treatment.[30] ONSF is warranted in patients with IIH who are receiving maximal medical therapy and yet have progressive visual loss or impending visual loss with minimal or tolerable headaches. By contrast, a CSF-diversion procedure is preferred over ONSF for those patients with medially resistant, unremitting and disabling headaches with or without visual loss. Recently, several small case series have demonstrated the efficacy of venous sinus stenting in some cases of IIH; however, this therapeutic modality requires further exploration.[31–37]

In a meta-analysis based on retrospective data of ONSF performed on 423 eyes in patients with IIH, visual acuity improved in 50 % of eyes and visual field improved in 72 % of eyes.[38] In the same meta-analysis, worsening of the visual field or visual acuity occurred in only 11 % of cases. In a recent study, 62 IIH patients with bilateral papilledema who underwent unilateral ONSF were found to have a decrease in the median grade of papilledema in both the operated and the nonoperated eye. The median grade of papilledema in the operated eye decreased from grade 3 preoperatively to grade 0.5 by 12 months. The median grade of papilledema in the nonoperated eye decreased from grade 2 before surgery to a grade 1 12 months postoperatively[39] (see *Table 3*). Studies have shown that ONSF is safe and effective in pediatric patients as well.[40,41] A retrospective chart review of 17 pediatric eyes with IIH showed decreased ONH edema with stabilization or improvement in visual function in all eyes after ONSF.[41]

ONSF is not an entirely benign procedure and can be associated with both minor and profound ocular complications (see *Table 4*). In a review of the published IIH literature, the complication rate of ONSF was found to range broadly between 4.8–45 % with a mean of 12.9 %.[30] In the same review of 317 cases of ONSF, 13 % of cases were deemed a failure, which was defined as progressive visual loss despite the surgery or need for reoperation.[30] In addition, case reports have described patients with progressive visual loss after ONSF due to sustained elevated ICP.[42] A recent study using an experimental rat model found optic atrophy and degeneration of RGCs after ONSF, suggesting that in humans RGC neurodegeneration may be a consequence of ONSF.[43]

Although ONSF is performed most commonly in the setting of papilledema due to IIH, ONSF is also indicated in cases of papilledema with impending or progressive visual loss due to an unresectable CNS mass, an arteriovascular malformation of the vein of Galen, venous

**Table 3: Summary of Articles on Optic Nerve Sheath Fenestration for the Treatment of Idiopathic Intracranial Hypertension**

| | | | | Surgery | | | | | Outcome | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Authors/Year | No. of pts | No. of eyes | Mean Age (Years) | M | F | Bilateral | Unilateral | Primary | Mean FU in Months (Range) | AI or Same | AI | FI | Field and/or Acuity Worse | Subsequent Procedures |
| Corbett et al., 1988 | 28 | 40 | 30 (14–62) | 8 | 20 | 12 | 16 | 26 | 26.9 (0–90) | 34 | 12 | 21 | 13 | 2 CSF diversions |
| Sergott et al., 1988 | 23 | 29 | 38.1 (18–63) | 1 | 22 | 6 | 17 | 17 | 21.5 (3–45) | 23 | 21 | 29 | 1 | 2 repeat ONSFs |
| Spoor et al., 1991 | 35 | 69 | 32.3 (6–72) | 4 | 31 | 21 | 14 | 33 | 18.1 (2–48) | 69 | 44 | 68 | 0 | Repeat ONSF (16 in 13 eyes) acute cases |
| Acheson et al., 1994 | 11 | 15 | 37.1 (23–53) | 3 | 8 | 4 | 7 | 7 | 24 (12–84) | 4 | 9 | 8 | 2 | 4 CSF diversions; 1 subtemporal decompression |
| Goh et al., 1997 | 19 | 29 | 33.1 (16–52) | 6 | 13 | 10 | 9 | 19 | 15.7 (1–50) | 15 | | 9 | 2 | 4 repeat ONSFs |
| Banta and Faris, 2000 | 86 | 158 | 32.1 (NA) | 13 | 73 | 72 | 14 | 86 | 20 (1–108) | 148 | | 71 | 16 | 4 repeat ONSFs and CSF diversions; 2 CSF diversions only; 2 ONSFs only |
| Chandrasekaran et al., 2006 | 32 | 51 | 33.4 (17–65) | 3 | 29 | 18 | 14 | 25 | 27.6 (0–121) | 32[1] | 13[1] | 13[2] | 8[3] | 11 CSF diversions |
| Summary | 252 | 423 | 33.6 (6–72) | 43 (17 %) | 209 (83 %) | 148 (59 %) | 104 (41 %) | 226 (90 %) | 21.1 (0–121) | 357 (87 %) | 113 (50 %) | 226 (72 %) | 42 (11 %) | 26 repeat ONSFs (6 %); 19 CSF diversions (4 %); 1 subtemporal decompression (<1 %); 4 ONSFs and CSF diversions (1 %); Total: 12 % reoperation rate |

*AI = acuity improvement; CSF = cerebrospinal fluid; FI = field improvement; FU = follow up; pts = patients; ONSF = optic nerve sheath fenestration; NA = not available. 1. Based on 29 eyes (limited follow-up data available); 2. Based on 31 eyes; 3. Based on 39 eyes for acuity and 31 eyes for visual field. Adapted from Feldon.[38]*

sinus thrombosis, and obstruction of the cerebral venous system from a compressive lesion.[20] In addition, case reports have described patients with cryptococcal meningitis and visual loss caused by papilledema who have been successfully treated with ONSF.[44–46]

## Surgical Technique

The three main surgical approaches for ONSF are superior eyelid, lateral orbital, and medial transconjunctival.

In the following section we will provide a general overview of each approach with a special emphasis on the medial transconjunctival approach, which is the preferred technique of the authors.

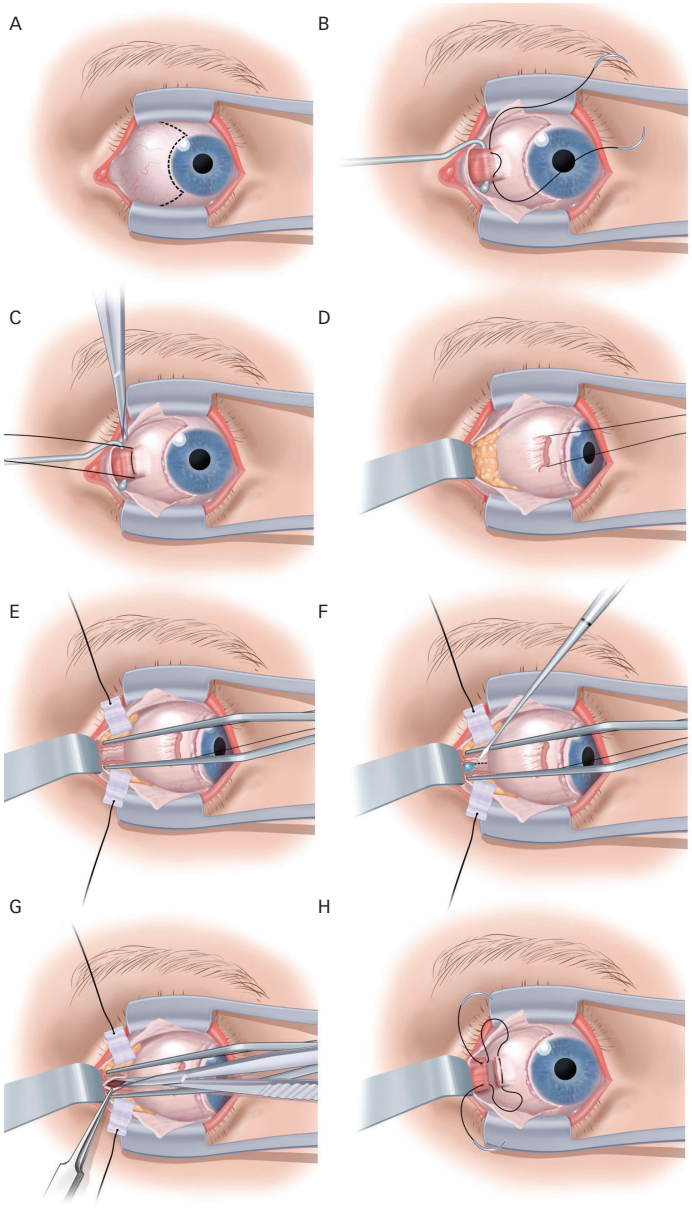
### Superior Eyelid Approach

Approaching the superior and lateral orbit via the anterior superior eyelid crease has been a surgical method preferred by orbital surgeons for removal of intraorbital lesions. In recent years, the anterior superior eyelid crease approach has been adopted by neurosurgery because of the excellent surgical exposure it provides to the anterior skull base.[47] A particular advantage of this approach is the excellent cosmetic outcome because the incision can be hidden within the natural superior eyelid crease.[48] For the purposes of performing an ONSF, the medial intraconal space is accessed through a superomedial eyelid crease incision.[49] The orbital septum is opened and the medial horn of the levator aponeurosis pushed laterally. With blunt dissection, a plane is created between the medial rectus muscle and the superior oblique tendon to access the posterior orbit avoiding the superior ophthalmic vein and vortex veins. With further posterior dissection, the optic nerve comes into view and a slit or rectangular window is created within the optic nerve sheath. Advantages of the superiomedial eyelid approach for an ONSF include decreased traction on the globe, decreased operating time, decreased bleeding, limited sharp dissection of normal tissue, and absence of extraocular muscle traction or dissection. Limitations of this approach include an increased distance from incision site to the optic nerve and an external (skin) incision compared with the medial transconjunctival approach (see below).

### Lateral Orbital Approach

The approach to the intraconal orbital space for ONSF through the lateral conjunctiva was first described in 1872.[1] Subsequently, the lateral approach was modified by a number of surgeons to improve access to the optic nerve. In 1988, Tse and colleagues described a lateral orbitotomy approach for ONSF.[50] The procedure begins with an en bloc removal of the lateral orbital wall. The periorbita is incised in a T-shaped fashion and blunt dissection of the perimuscular fasical sheaths is performed until the lateral rectus muscle is identified. A traction suture is placed under the insertion of the lateral rectus muscle and the suture is anchored medially, adducting the eye in order to move the optic nerve laterally. Dissection with specially designed orbital-neurosurgical brain retractors is used to gain access to the optic nerve. Once the retrobulbar portion of the optic nerve is adequately exposed, an operating microscope is used to assist in a window incision of the optic nerve sheath. The periorbita is closed with interrupted sutures and the bone fragment is re-approximated to the lateral orbital wall using nonabsorbable suture. Advantages of the lateral approach to ONSF include exposure of a longer segment of the optic nerve. Limitations of this approach include longer operating time, an

## Figure 5: Authors Preferred Technique for Performing an Optic Nerve Sheath Fenestration




*A. A medial limbal conjunctival peritotomy is performed with extension of the conjunctival incision superiorly and inferiorly; B. The medial rectus muscle is isolated with a muscle hook and a double-armed suture is secured to the insertional site (tendon) of the muscle; C. The muscle is detached from the globe using sharp scissors; D. A traction suture is placed through the muscle tendon and the globe is retracted laterally. The retrobulbar optic nerve is approached through the posterior reflection of tenon's capsule with the assistance of a malleable retractor; E. The orbital fat is retracted away from the optic nerve with small strips of cottonoids. The blades of a small angled forceps is used to straddle each side of the optic nerve to improve exposure to the optic nerve; F. A sharp microblade is used to incise the optic nerve sheath 2 mm posterior to the globe; G. The optic nerve sheath incision is extended posteriorly with microscissors to a total length of 3 to 5 mm; H. The traction suture is removed, the medial rectus is reattached to the globe, and the conjunctiva is closed using standard strabismus surgical techniques. Illustration by Rob Flewell CMI.*

external incision, and a more complex surgical procedure that requires removal of the orbital rim.[20]

## Medial Transconjunctival Approach

The medial approach to the retrobulbar optic nerve was first described by Galbraith and Sullivan in 1973.[51] The following description is the technique performed at the Duke Eye Center (see *Figure 5*).[41] After induction of general anesthesia, a medial limbal conjunctival peritomy is performed and the conjunctiva incision is extended superiorly and inferiorly. The medial rectus muscle is isolated and the tendon is secured with a double armed 6–0 vicryl suture. The muscle is detached from the globe using scissors, leaving a small remnant of muscle tendon attached to the globe. A 5–0 dacron traction suture is placed through the muscle tendon, and the globe is retracted laterally. The long posterior ciliary arteries are then identified between the superior and inferior poles of the insertion of the medial rectus muscle. With the aid of small malleable retractors the retrobulbar optic nerve is approached through the posterior reflection of tenon's capsule and retrobulbar orbital fat. The orbital fat is retracted away from the optic nerve with small strips of cottonoids. A small angled forceps is used to improve exposure of the optic nerve. With the assistance of the operating microscope a sharp blade on a long handle is used to incise the optic nerve sheath approximately 2 mm posterior to the globe with careful attention to avoid any blood vessels on the surface of the nerve. A fine toothed forceps is inserted into the incision site and extended posteriorly with microscissors to a total length of 3–5 mm. A tenotomy hook is sometimes inserted into the SAS and moved in the anterior–posterior direction to lyse any arachnoidal trabeculations and adhesions. On completion of the fenestration, the traction suture is removed, and the medial rectus is reattached to the globe using standard strabismus muscle technique. The conjunctiva is closed with 8–0 vicryl sutures. An antibiotic-steroid ointment is applied to the eye and a protective shield

## Table 4: Complications of Optic Nerve Sheath Fenestration

| |
|---|
| **Conjunctiva/Sclera** |
| • Conjunctival bleb |
| • Globe perforation |
| • Chemosis |
| • Subconjunctival Tenon's cyst |
| **Cornea** |
| • Dellen formation |
| • Corneal ulcer |
| **Anterior Chamber** |
| • Microhyphema |
| • Angle closure glaucoma |
| **Iris** |
| • Pupillary dysfunction causing sector/tonic pupil |
| **Retina/Choroid** |
| • Chorioretinal scar from excessive traction on the globe |
| • Branch retinal artery occlusion/central retinal artery occlusion |
| • Choroidal ischemia/infarction |
| **Optic Nerve** |
| • Traumatic optic neuropathy |
| • Myelinated nerve fibers |
| • Optic nerve cyst formation |
| **Orbit** |
| • Orbital apex syndrome |
| • Orbital infection |
| • Orbital hemorrhage |
| **Extraocular Muscles** |
| • Extraocular movement dysfunction (due to slipped/lost muscle, inaccurate reapproximation of muscle) |

is placed over the eye to prevent any direct external pressure. The advantages of the medial transconjunctival approach include increased width of the operating field, minimal distance from incision to the optic nerve, minimal bleeding, and lack of an external incision.

## Conclusion

ONSF is a relatively safe and effective surgical procedure in patients with visual loss due to papilledema. Although ONSF has been found to improve visual function in many diseases of the optic nerve, it has been best studied—although without the benefit of level I evidence—in cases of papilledema in the setting of IIH.

Timely ONSF in an IIH patient with visual loss may lead to stabilization and, sometimes, visual function improvement. There are three main approaches to ONSF, each offering its own advantages and limitations. ■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