Endoscopic Orbital and Optic Nerve Decompression

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Soon after its introduction by Walsh and Ogura in the 1950s\textsuperscript{[1]}, transantral decompression of the orbit became a popular technique to treat patients with severe proptosis from Graves’ disease. This procedure enabled otolaryngologists to remove the orbital floor and medial wall through the familiar Caldwell-Luc approach, allowing for decompression of the enlarged orbital muscles and fat into the maxillary and ethmoid sinuses. Soon after the introduction of endoscopic instrumentation for the performance of sinus surgery in the mid-1980s, however, surgeons began to experiment with an entirely transnasal approach to treat diseases of the orbit.

Kennedy et al\textsuperscript{[1]} and Michel et al\textsuperscript{[2]} first described endoscopic orbital decompression in the early 1990s. Because high-resolution endoscopes provided improved visualization in key anatomic regions, including the medial orbital wall and skull base, this technique soon gained widespread acceptance for the treatment of patients with Graves’ disease, largely replacing the transantral approach. In addition to providing enhanced visualization, endoscopic instrumentation allowed for complete and safe bone removal, particularly along the orbital apex and optic canal. Although the use of endoscopic orbital decompression for the orbital manifestations of Graves’ disease has been well established, the indications and applications for optic nerve decompression remain in evolution.

Graves’ orbitopathy (dysthyroid orbitopathy)

Graves’ disease is an autoimmune disorder that primarily affects the thyroid and the orbit. Thyroid manifestations are characterized by the
production of autoantibodies to the thyrotropin receptor and subsequent hyperstimulation with resultant hyperthyroidism. The thyroid manifestations of Graves’ disease generally are treated with medications, radiation (iodine 131), or surgery.

The orbital manifestations, known as dysthyroid orbitopathy, also represent an autoimmune process, although the exact antibody target is unclear. Inflammation with infiltration of T cells and increased glycosaminoglycan deposition results in enlargement of orbital fat and extraocular muscles. The increased orbital contents result in increased pressure within the bony orbit and resultant proptosis, compression of the optic nerve, or both. The degree of proptosis does not correlate with the overall severity of disease because patients with poor compliance of the orbital septum may not experience significant proptosis, but can have severe compression at the orbital apex and optic neuropathy. The orbital and thyroid manifestations of Graves’ disease follow distinct and independent clinical courses.

Clinical manifestations of dysthyroid orbitopathy range from mild findings, such as tearing, photophobia, and conjunctival injection, to significant proptosis, diplopia, exposure keratopathy, and visual loss from optic neuropathy. The clinical course of Graves’ orbitopathy can be divided into acute and chronic phases, with the acute phase characterized by active inflammation and lasting 6 to 18 months. The chronic phase is characterized by fibrosis and represents the preferred phase for surgical intervention.

**Medical treatment of dysthyroid orbitopathy**

Local measures, such as lubrication, eyelid taping, and patching, for patients with dryness and diplopia represent initial conservative treatment approaches. More aggressive treatments include the use of orbital radiation and systemic corticosteroids. Both treatments seem to be most effective during the acute phase of the disease. The use of orbital radiation is controversial, and its efficacy has been challenged by two more recent randomized prospective trials [3,4]. Systemic corticosteroid treatment shows symptomatic improvement, but symptoms generally recur after steroid treatment. Because of the deleterious side effects of long-term corticosteroid use, steroid treatment often is used as a temporizing measure or in conjunction with surgical decompression.

**Endoscopic orbital decompression**

The endoscopic technique allows for unmatched visualization of critical anatomic regions, such as the skull base and orbital apex, and avoids external or sublabial incisions. The entire medial orbital wall and the medial portion of the orbital floor is removed with endoscopic decompression (Fig. 1).
Technique

The patient is positioned in the supine position, and topical vasoconstriction is achieved with 4% cocaine pledgets. Draping is the same as with standard endoscopic sinus procedures, and the eyes are exposed in the surgical field, but protected with scleral shields. Image guidance systems may be used at the surgeon’s discretion. Lidocaine 1% with 1:100,000 epinephrine is injected along the lateral nasal wall in the region of the maxillary line (a bony eminence that extends from the anterior attachment of the middle turbinate to the root of the inferior turbinate).

Surgery begins with an incision just posterior to the maxillary line and through the uncinate process. The uncinate process is medialized and removed; this allows visualization of the natural ostium of the maxillary sinus. With orbital decompression, it is necessary to open the maxillary sinus widely to achieve good access to the orbital floor and prevent blockage of the ostium from orbital fat, which protrudes after decompression. The ostium can be opened to the floor of the orbit superiorly, the wall of the maxillary sinus posteriorly, the thick bone of the frontal process of the maxilla anteriorly, and the inferior turbinate inferiorly. If the antrostomy is extended beyond the frontal process of the maxilla anteriorly, there is risk of damage to the nasolacrimal duct. Using a 30° endoscope, the wide antrostomy should allow easy visualization of the infraorbital nerve as it courses along the floor of the orbit.

An endoscopic sphenoidotomy is performed in the standard fashion. After sphenoidotomy, the anterior and posterior ethmoid arteries can be identified as they course along the skull base. We advocate removal of the middle turbinate during orbital decompression to optimize exposure of the medial orbital wall and facilitate postoperative cleanings.

Fig. 1. Bone removed during endoscopic orbital decompression includes the entire medial wall of the orbit and the medial portion of the orbital floor with the lateral limit of decompression at the infraorbital nerve.
An image guidance system may be used at this point to confirm removal of all ethmoid cells along the medial orbital wall.

When complete exposure of the medial orbital wall is obtained, a spoon curet is used to penetrate the thin bone of the lamina papyracea carefully (Fig. 2). This bone is elevated while preserving the underlying periorbita. Bone removal proceeds superiorly toward the ethmoid roof, inferiorly to the orbital floor, and anteriorly to the maxillary line. Bone in the region of the frontal recess is left intact; if bone is removed from this region, herniated fat may obstruct drainage of the frontal sinus.

As dissection proceeds posteriorly, thick bone is encountered in the region of the orbital apex within 2 mm of the sphenoid face. This bone corresponds to the anulus of Zinn, from which the extraocular muscles originate and through which the optic nerve passes; this represents the posterior limit of a standard decompression. For patients with optic neuropathy, experienced surgeons may consider continuing the decompression posteriorly into the sphenoid sinus. The benefits of incorporating an optic nerve decompression into standard orbital decompression are unclear and may lead to inadvertent injury to the nerve. Anteriorly, fragments of bone are removed where the lamina papyracea joins the lacrimal bone. The thick white fascia of the lacrimal sac may be uncovered, but should not be opened. Thick bone anterior to the maxillary line covers much of the lacrimal sac and should not be removed.

Removal of the orbital floor may be the most technically challenging portion of the procedure. Only the portion of the floor medial to the infraorbital nerve is removed. A spoon curet is used to engage the orbital floor at its medial extent and down-fracture the bone (Fig. 3). The bone of the orbital floor
is thicker than that of the medial orbital wall, and significant force is required for this maneuver. The spoon curet may not be sturdy enough for this portion of the procedure, and a more robust mastoid curet may be used. The bone may fracture in one large piece, typically with a natural cleavage plane at the canal of the infraorbital nerve. If it fractures into several small pieces, a $30^\circ$ endoscope and curved instrumentation may facilitate bone removal, while preserving the infraorbital canal as the lateral limit of dissection.

When the lamina papyracea and medial orbital floor have been removed, the periorbita is fully exposed. A sickle knife may be used to open the periorbita (Fig. 4). Care must be taken to avoid “burying” the tip of the sickle knife and potentially injuring the underlying orbital contents, such as the medial rectus muscle. The use of a Steri-strip to cover all but the distal 2 mm of the knife may prevent deeper penetration. The periorbital incision should be initiated at the posterior limit of decompression (just anterior to the sphenoid face) and brought anteriorly so that prolapsing fat does not obscure visualization. Parallel incisions are performed along the ethmoid roof and orbital floor. To minimize the risk of postoperative diplopia, a sling of fascia overlying the medial rectus muscle may be preserved, while the remainder of the periorbita is removed using angled Blakesley forceps (Fig. 5) [5]. In patients with optic neuropathy, the fascial sling technique is not used to allow maximal decompression. A ball-tipped probe and sickle knife may be used to identify and incise remaining fibrous bands, which often course superficially between lobules of orbital fat. On completion of the procedure, a generous prolapse of fat into the opened ethmoid and maxillary cavities should be observed. The globe may be palpated to confirm a decrease in retropulsion.
Depending on the clinical scenario and desired degree of decompression, a lateral decompression may be performed concurrently. When performed immediately after medial decompression, the orbital contents are retracted easily in a medial direction allowing for excellent exposure of the lateral bony wall. Concurrent excision of intraconal fat also may be performed if

Fig. 4. A sickle knife is used to incise the periosteum. Care is taken to control the tip of the knife and prevent injury to the deeper orbital structures.

Fig. 5. A sling of periorbita overlying the medial rectus muscle may be preserved to minimize the risk of postoperative diplopia. This sling technique is not used in cases of optic neuropathy where decompression of the orbital apex is desired.
deemed necessary. Bilateral decompressions may be performed concurrently or in a staged procedure.

Nasal packing is avoided to ensure maximal decompression and avoid compression of exposed orbital contents. The patient is discharged the morning after surgery with a prescription for oral antibiotics and instructions to begin twice-daily nasal saline irrigations. At the first postoperative visit, 1 week after surgery, crusting is cleaned from the surgical site under endoscopic guidance.

For patients with severe comorbidities, with a strong preference for local anesthesia, or in whom surgery is being performed on an only seeing eye, decompression may be accomplished under local anesthesia with sedation [6]. This approach allows the surgeon to monitor the patient’s vision throughout the procedure. Sedation may be achieved with an intravenous bolus of propofol (0.4–0.8 mg/kg) before injection of local anesthesia, followed by an infusion of 75 to 95 µg/kg during the procedure. Local anesthesia is administered initially with 4% cocaine pledgets followed by injection of lidocaine 1% with 1:100,000 epinephrine as described for patients undergoing general anesthesia. Patients often report discomfort during removal of the lamina papyracea. This sensation may be relieved by infiltration of a small amount of additional anesthetic solution into the periorbita.

Results

The goals of orbital decompression vary depending on the indication for the procedure. In patients with compressive optic neuropathy, restoration of visual deficits is the key outcome, whereas in patients with corneal exposure or severe proptosis, ocular recession may be the primary end point. The rate of improvement after endoscopic orbital decompression for Graves’ orbitopathy ranges from 22% to 89% [1,7,8]. This wide variation in results reflects the diverse patient populations and definitions of improvement. Postoperative deterioration of visual acuity occurs in less than 5% of patients [2,7,8]. Ocular recession as a result of endoscopic decompression alone averages 3.5 mm (range 2–12 mm). The addition of concurrent lateral decompression to the endoscopic procedure provides an additional 2 mm of globe recession [8].

Complications

Diplopia is a frequent complication of orbital decompression with 15% to 63% of postoperative patients reporting new-onset diplopia or worsening of preexisting symptoms [2,6,8–11]. This complication is believed to be a result of a change in the vector pull of the extraocular muscles. Decompressive surgery rarely alleviates preexisting diplopia. Patients who have diplopia after decompressive surgery frequently require strabismus surgery for correction. All patients should be informed of the possibility of postoperative double vision and the potential for further surgical intervention.
Several methods to decrease postoperative diplopia have been reported. Multiple authors have described the preservation of a strut of inferomedial bone between the decompressed floor and medial wall [7,12]. When this strut is maintained, however, it is technically difficult to remove any of the orbital floor through a purely endoscopic technique. The maintenance of a facial sling in the region of the medial rectus has been shown to decrease postoperative diplopia [5]. This technique provides similar support as the medial strut technique, but allows for endoscopic access to decompress the medial orbital floor. The concept of a balanced decompression (concurrent medial and lateral decompression) also has been suggested as a means to decrease postoperative diplopia [9,13,14]. When operating for compressive optic neuropathy, techniques designed to limit diplopia also may limit the extent of decompression, and postoperative diplopia often is accepted as a concession to improved visual acuity.

Postoperative bleeding after decompression is best approached through endoscopic identification and direct cauterization of the bleeding site. Nasal packing generally is not used to avoid pressure on the exposed orbital apex and optic nerve. Postoperative infection is minimized through the use of postoperative antibiotics with staphylococcal coverage. A large maxillary antrostomy and limited bone removal in the frontal recess region minimize the risk of developing postoperative sinusitis. Epiphora may develop if the maxillary antrostomy is extended too far anteriorly with transaction of the nasolacrimal duct. This complication may be treated readily with an endoscopic dacryocystorhinostomy. Leakage of cerebrospinal fluid (CSF) and blindness are rare complications that have been reported primarily after nonendoscopic decompression techniques.

**Endoscopic optic nerve decompression**

Historically, the most frequent indication for optic nerve decompression has been traumatic optic neuropathy. More recent studies have questioned the role for decompression in these patients [15,16]. Currently, the most favorable indications for optic nerve decompression seem to be direct compression from fibro-osseous lesions or tumors [17–21]. Traditional surgical approaches for optic nerve decompression include transorbital, extranasal transethmoidal, transantral, intranasal microscopic, and craniotomy approaches. Endonasal endoscopic decompression of the optic nerve offers many advantages over these approaches, including excellent visualization, preservation of olfaction, rapid recovery time, lack of external scars, and less operative stress in patients who may have multisystem trauma.

**Surgical anatomy**

The optic nerve may be divided into three segments: intraorbital, intracanalicular, and intracranial. The goal of optic nerve decompression is to
relieve compressive forces within the intracanalicular portion of the nerve. The canal of the optic nerve is formed by the two struts of the lesser wing of the sphenoid and carries the optic nerve and the ophthalmic artery. Within the optic canal, the nerve is encased by three meningeal layers: pia, arachnoid, and dura mater. At the orbital apex is the fibrous anulus of Zinn. This thick, fibrous layer is the least expandable portion of the fibrous tissue around the optic nerve and has been suggested to be the most susceptible site for pathologic compression [22,23].

**Technique**

Patients are prepared for surgery in a similar manner to patients undergoing orbital decompression. A standard sphenoethmoidectomy is performed. The sphenoid face is opened widely, and the bulge of the optic nerve canal is identified along the lateral wall of the sphenoid sinus, superior to the carotid artery. In some patients, the optic canal may be identified initially in a posterior ethmoid or Onodi cell, which can be identified on pre-operative CT scan [24]. Identification and opening of the Onodi cell is important to provide adequate surgical exposure and allow full access to the optic canal.

After complete sphenoethmoidectomy, a spoon curet is used to fracture the lamina papyracea approximately 1 cm anterior to the optic canal. The lamina is removed carefully in a posterior direction to expose the annulus of Zinn. Care must be taken to avoid penetration of the periorbita because subsequent herniation of orbital fat obscures the surgical field. As the optic canal is approached, the thin lamina is replaced with the thick bone of the lesser wing of the sphenoid. This bone must be thinned before removal. A long-handled drill with a diamond burr is used methodically to thin the medial bone of the optic canal. While drilling, care must be taken to prevent contact of the drill bit with the prominence of the carotid artery, which is located just inferior and posterior to the optic nerve. After the bone is appropriately thinned, a microcuret is used to fracture the thinned bone in a medial direction, away from the optic nerve. Controversy exists as to the extent of bone that must be removed to achieve satisfactory decompression. For cases of traumatic optic neuropathy and dysthyroid orbitopathy, removal of bone for a distance of 1 cm posterior to the face of the sphenoid sinus is usually sufficient.

The authors generally do not recommend incision of the optic sheath in addition to bony decompression. Not only does this maneuver risk damage to the underlying nerve fibers and the ophthalmic artery, but also the risk of CSF leak and meningitis is increased. In select cases in which compression within the sheath is strongly suggested, such as with intrasheath hematoma or significant papilledema, opening of the optic nerve sheath may play a role.

When opening the optic nerve sheath, a sickle knife is used to incise the sheath just anterior to the anulus of Zinn. The sheath should be entered in
its superomedial quadrant to minimize risk to the ophthalmic artery. The incision in the sheath may be continued posteriorly with the sickle knife or with microscissors. Some authors suggest the application of fibrin glue to the incision site after decompression to minimize the risk of CSF leak [17].

**Results**

The efficacy of optic nerve decompression in traumatic optic neuropathy is unclear. With the significant rate of visual improvement with observation alone, well-designed prospective studies are necessary to comment definitively on the efficacy of this procedure. Despite significant efforts, such studies have not been performed, largely owing to the rarity of this condition. This lack of randomized prospective data is unlikely to change, and clinicians are left to draw conclusions from uncontrolled and often contradictory retrospective reports.

**Complications**

The risk of CSF leak, meningitis, and visual loss with optic nerve decompression seems to be significantly higher than with standard endoscopic sinus surgery or orbital decompression. Although several studies report no complications in study sizes ranging from 20 to 45 patients, several more recent studies have reported CSF leaks, some with associated meningitis and visual decompensation [15,25].

**Summary**

Endoscopic orbital and optic nerve decompression are advanced techniques that should be performed only by surgeons experienced in endoscopic nasal surgery. The use of a transnasal endoscopic approach for these procedures allows for excellent visualization of the critical anatomy and avoidance of external scars. Although the benefits of orbital decompression have been well established, the role of optic nerve decompression remains controversial.

**References**