Orbital and optic nerve decompression

O.C Okonkwo1 MBBS MRCS FRCS ORL-HNS and S K Ahmed1 MBBS PhD FRCS ORL-HNS
1 Department of ENT and Skullbase Surgery, Queen Elizabeth Hospital, University Hospitals Birmingham, Birmingham, B12 2GW

Correspondence:
O.C Okonkwo, Department of ENT and Skullbase Surgery
Queen Elizabeth Hospital, University Hospitals Birmingham, Birmingham, B12 2GW
E-mail: o.okonkwo@nhs.net

Abstract
Background: The concept of orbital decompression was first described in 1890 by Julius Dollinger. However, in recent decades the endoscopic approach to the orbit and optic nerve has evolved.

The underlying pathologies that lead to increased intraorbital pressure secondary to extrinsic compression of the globe or that lead to optic nerve compression are varied, but they are linked as they all impact on optic nerve perfusion pressure and can cause optic neuropathy.

The indications for orbital decompression can be acute or chronic. However, optic nerve decompression should only be considered if there is evidence of compressive optic neuropathy with deteriorating visual acuity, unresponsive to medical treatment, in the presence of an intact optic nerve.

Prior to surgery thorough work up in conjunction with an ophthalmologist is essential.

We discuss developments in surgical techniques for orbital and optic nerve decompression.


Key words
Orbit, optic nerve, decompression, endoscopic

Anatomy
The orbit is a rigid anatomical structure made up of 7 bones, bound by 4 bony walls and bound anteriorly by the eyelids and orbital septa.

The posterior aspect of the orbit narrows to a confluence forming the orbital apex where the optic canal, superior orbital fissure and inferior orbital fissure transmit nerves and vessels into the orbit.

The rigid boundaries of the orbit create a fixed capacity of approximately 30ml and there is little room to accommodate any significant increase in volume of the globe or extra ocular soft tissue. As a consequence, changes in volume are associated with extrinsic compression of the globe and sequelae such as proptosis, diplopia and visual loss.

Likewise, the optic canal which runs through the lesser wing of sphenoid and transmits the optic nerve and ophthalmic artery, is a fixed capacity bony canal and any change in volume of its contents can result in compressive optic neuropathy.

This often manifests as slowly progressive decrease in visual acuity, with dyschromatopsia; a relative afferent pupillary defect; visual field defect; and optic atrophy or oedema.

Background
The concept of orbital decompression was first described in 1890 by Julius Dollinger. The traditional external approaches are well established i.e. trans conjunctival, transcranial and lateral orbitotomy. However, in recent decades the endoscopic approach to the orbit and optic nerve has evolved, initially endonasally, as pioneered by Kennedy et al in 1990 and more recently via endoscopic transorbital neuroendoscopic surgery (TONES). These approaches are minimally invasive and allow good access and visualization of ocular structures.

Aetiology
The underlying pathologies that lead to increased intraorbital pressure secondary to extrinsic compression of the globe or that lead to optic nerve compression are varied, but they are linked as they all impact on optic nerve perfusion pressure and can cause optic neuropathy (see table 1). The most commonly encountered of all these pathologies is thyroid eye disease.

Indications
The indications for orbital decompression can be acute or chronic. (See table 2) The indications for optic nerve decompression are, however, more tenuous and the literature is inconclusive. There have been studies to show
that it is of no benefit in traumatic optic neuropathy due to
the high rate of spontaneous resolution in visual acuity. However, the
literature suggests that optic nerve decompression should be considered if there is evidence
of compressive optic neuropathy with deteriorating visual
acuity, unresponsive to medical treatment, in the presence
of an intact optic nerve.

Work up
Ophthalmological assessment is essential prior to the
procedure. This includes measurement of visual acuity,
assessment of colour vision, evaluation of proptosis and
assessment of eye movements and diplopia.

A CT scan evaluating the orbits and sinuses is required to
review relevant anatomy and the surgeon should identify
the middle turbinate attachment, location and course of the
anterior ethmoid artery, and the presence of an Onodi cell
as the optic nerve may course through the lateral aspect of
the cell.

Furthermore, MRI imaging of the orbit and brain is the
imaging modality of choice when assessing the optic
nerve and soft tissue within the orbit.

Procedure
The goals of surgery are in optic nerve decompression are
to reduce or reverse vision loss. Orbital decompression has
the additional goals to prevent ocular surface damage;
relieve orbital pain and congestion; reduce proptosis,
diplopia, lid retraction, chemosis, lid oedema, and fat
prolapse.

There are 4 areas in which orbital decompression can be
achieved (See table 3). Each has unique considerations
and can impact on subsequent rehabilitative ocular surgery
i.e. eyelid and strabismus procedures, therefore we feel
these patients should be managed in conjunction with
ophthalmology colleagues. In this article we will focus on
transnasal orbital decompression.

Technique
Endoscopic visualization via the trans nasal route allows
access to the medial aspect of the orbit, orbital floor and
orbital apex making it an ideal approach for accessing
both intra and extra conal structures located in the medial
and posterior aspect of the orbit. It has the advantage that
it is low morbidity when compared to other techniques
and does not leave a scar. However, it can be associated
with new onset or worsening of pre-existing strabismus,
double vision and globe dystopia, therefore patients must
be counselled on the possible need for subsequent
strabismus surgery.

Orbital Decompression
After adequate decongestion of the nose the surgeon first
performs a large middle meatal antrostomy with removal
of the uncinate process anteriorly and bony removal
extending posteriorly to the posterior maxillary wall. The
superior limit is the orbital floor and inferior limit is the
superior margin of the inferior turbinate.

Then a complete sphenoid and ethmoidectomy is performed to
expose the medial wall of the orbit from the skull base
superiorly to the roof of the maxillary sinus inferiorly; and
anteriorly from the maxillary line to the face of the

Table 1: Underlying Aetiology of Globe and Optic Nerve Compression

<table>
<thead>
<tr>
<th>Inflammatory disorders</th>
<th>Trauma</th>
<th>Neoplasia</th>
<th>Benign masses</th>
<th>Infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thyroid eye disease</td>
<td>Bony displacement</td>
<td>Sella/parasellar masses</td>
<td>Mucocoele</td>
<td>Abscess</td>
</tr>
<tr>
<td>Ocular myositis</td>
<td>Hematoma</td>
<td>Orbital/orbital apex masses</td>
<td>Meningiomas</td>
<td>Post septal cellulitis</td>
</tr>
<tr>
<td>Systemic inflammatory disease</td>
<td>Oedema</td>
<td>Nasal/paranasal masses</td>
<td>Fibrodysplasia</td>
<td></td>
</tr>
</tbody>
</table>

Table 2: Indications for orbital decompression

<table>
<thead>
<tr>
<th>Acute</th>
<th>Chronic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute optic neuropathy</td>
<td>Disfiguring proptosis</td>
</tr>
<tr>
<td>orbital compartment syndrome</td>
<td>chronic pain/discomfort</td>
</tr>
<tr>
<td>corneal decompensation</td>
<td>congestion</td>
</tr>
<tr>
<td>acute globe subluxation</td>
<td>corneal exposure/ulceration</td>
</tr>
<tr>
<td>Severe orbital inflammation</td>
<td>Progressive orbitopathy not responding to other measures</td>
</tr>
</tbody>
</table>
sphenoid sinus posteriorly (Figure 1). During this exposure one should be careful not to penetrate the skull base; traumatise the anterior or posterior ethmoid arteries; or inadvertently enter the orbit or sphenoid sinus.

The lamina papyracea is carefully removed, commonly using a Cottles elevator, with care taken to avoid trauma to the periorbita (Figure 2). Fragments are removed using the Blakesley forceps.

An inferiormedial orbital strut is preserved (Figure 3) to keep the eyeball in the same axial position reducing the risk of double vision.

If an inferior decompression is required, the medial aspect of the orbital floor is often thicker than the lamina papyracea and is thinned carefully with a high speed diamond burr with the inferior orbital nerve used as the posterior lateral limit. The periorbita is carefully lifted off the floor of the orbit and the floor is then carefully removed with a j shaped curette or sickle knife.

The periorbita is then incised and septations broken down with gentle blunt dissection so that fat prolapses into the nasal cavity. (Figure 4).

Be aware that posteriorly there is less extraconal fat, so the medial rectus is very quickly encountered and can sometime be seen through the periorbita.

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**Table 3: Areas of Orbital Decompression and Surgical Access**

<table>
<thead>
<tr>
<th>Fat Compartment</th>
<th>Transcutaneous</th>
<th>Transconjunctival</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orbital floor</td>
<td>Transorbital</td>
<td>Transcutaneous</td>
</tr>
<tr>
<td></td>
<td>Transantral</td>
<td>Transnasal</td>
</tr>
<tr>
<td>Medial Wall of Orbit</td>
<td>Transorbital</td>
<td>Transantral</td>
</tr>
<tr>
<td></td>
<td>Transnasal</td>
<td></td>
</tr>
<tr>
<td>Lateral Wall of Orbit</td>
<td>Tranorbital</td>
<td>Transconjunctival</td>
</tr>
</tbody>
</table>

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**Figure 1:** Image of left skull base after complete ethmoidectomy (white arrow)

**Figure 2:** Left lamina papyracea being removed with cottles elevator.

**Figure 3:** Coronal CT scan showing inferiomedial strut of bone that is preserved between the medial orbital wall and inferior orbital walls.
When decompression surgery is performed, expected benefits include reduction in exophthalmos, periorbital puffiness (swelling and fat prolapse) and lid retraction. Results are generally proportional to the extent of bone removal and fat herniation into the sinuses.⁷

Other positive effects of decompression include a decrease in intraocular tension and relief of pain, improvement in pre-existing strabismus and cure of postural visual obscuration in patients with orbital and optic nerve microvasculopathy.⁸

**Optic Nerve Decompression**

Decompression of the optic nerve employs similar techniques to orbital decompression.

A complete spheno-ethmoidectomy is performed, and lamina papyracea is elevated posteriorly to the orbital apex and the optic tubercle (the thicker bridge of bone between the ethmoid and sphenoid).

The optic nerve is identified in the sphenoid sinus and the optic tubercle is drilled to eggshell thickness and removed to expose the optic nerve sheath (Figure 5).

Using this technique, it is possible to decompress 180-270 degrees around nerve.

Some authors advocate incision of the optic nerve sheath along the optic nerve and through the Annulus of Zinn in order to relieve idiopathic intracranial hypertension. Place the incision(s) at the superomedial quadrant, as the ophthalmic artery is located in the inferomedial quadrant of the optic canal.

However, opening the optic nerve sheath is controversial and exposes the patient to CSF leakage as well as the ophthalmic artery to injury and should therefore be reserved for very specific cases.⁹

**Summary**

Transnasal surgery gives good access to the medial orbital compartment and optic nerve

The indications for orbital and optic nerve decompression must be carefully considered

Decision making must be done in conjunction with an ophthalmologist

**References**