Chapter 96 – Optic Nerve Decompression

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Optic nerve injury with loss of vision is a devastating event.[1–10] Optic nerve injury can be classified in various ways, including but not limited to the anatomic region and pathophysiologic mechanisms. Anatomic classification includes intraorbital, intracanalicular, and intracranial sites of injury. Pathophysiologic classification includes traumatic and nontraumatic mechanisms of injury. Traumatic optic nerve injury can be further subclassified as either direct or indirect injury. Direct injury includes penetrating injury to the orbit or optic nerve canal as a result of a foreign body or fracture of the lesser wing of the sphenoid. Indirect injury is generally the result of blunt head trauma. The exact mechanism of injury is not well understood, however, but may include hematoma, neural edema, and disruption of the microvascular circulation and axonal transport. Nontraumatic causes of optic nerve injury include compressive effects from benign or malignant tumors, inflammatory conditions such as Graves' disease, and fibro-osseous lesions. The most effective treatment of traumatic optic neuropathy (TON) remains controversial. Options for treatment include observation, intravenous corticosteroids, and surgical decompression. In 1999 the International Optic Nerve Trauma Study (IONTS) attempted to answer the question of which therapeutic modality was most efficacious in treating indirect TON. It attempted to randomize patients to "megadose" steroids alone or in conjunction with extracranial optic nerve decompression. Because of patient recruitment factors, however, this was abandoned and the investigators proceeded to a comparative nonrandomized interventional study with concurrent treatment groups. They concluded that there was no clear benefit of corticosteroid therapy over optic canal decompression and that treatment should be tailored to the individual patient. Subsequent to IONTS, several retrospective series have been published that both support and refute the use of optic nerve decompression in addition to corticosteroid use for the treatment of indirect optic neuropathy.

Despite the controversy regarding TON, optic nerve decompression to alleviate compressive effects is widely accepted. Its technique, indications, and complications are the focus of this chapter, although the concepts apply to most situations.

PATIENT SELECTION

Patient selection is dictated by the cause of the visual loss.[6,11–14] In the scenario of TON, a diagnosis must first be secured. The diagnosis of TON is a clinical diagnosis supported by a history of direct or indirect injury to the head or face. The level of consciousness of the patient can make the situation fairly straightforward or make it a challenge. Features of TON include an afferent pupillary defect, monocular or binocular involvement, impairment of color vision, visual field defects, loss of visual acuity (ranging from mild to no light perception), and delayed development of optic atrophy in the weeks after the injury. In the setting of compressive optic neuropathy secondary to benign or malignant tumors, chronic inflammatory processes, or fibro-osseus lesions, many of the same features will be present; however, their onset will be much more insidious, and careful and detailed ophthalmologic assessment will be required to quantify the visual deterioration. Once compressive neuropathy is ascertained, the underlying pathology and degree of compression will dictate the need for surgical decompression.

PREOPERATIVE PLANNING

Before embarking on endoscopic decompression of the optic nerve, imaging studies of the paranasal sinuses and skull base are critical (Figs. 96-1 to 96-4). Understanding the close relationship of the optic nerve to the paracavernous internal carotid artery is fundamental before proceeding to surgery (Figs. 96-5 and 96-6). In the setting of benign tumors and fibro-osseus lesions leading to compressive neuropathy, the sphenoid sinus and nasal anatomy may be distorted and almost impossible to predict (see Fig. 96-2). Image guidance becomes a valuable tool to assist the endoscopic surgeon in safely approaching the optic nerve and skull base. We prefer a fine-cut computed tomography (CT) scan of the sinuses and skull base with contrast enhancement before proceeding to optic nerve decompression. In situations in which there may be a soft tissue mass causing compression, magnetic resonance imaging (see Figs. 96-3 and 96-4) can be added and fused with the CT scan to give further assistance in preoperative planning and navigation. Thorough ophthalmologic assessment, including all the issues mentioned in the previous section, is a prerequisite.

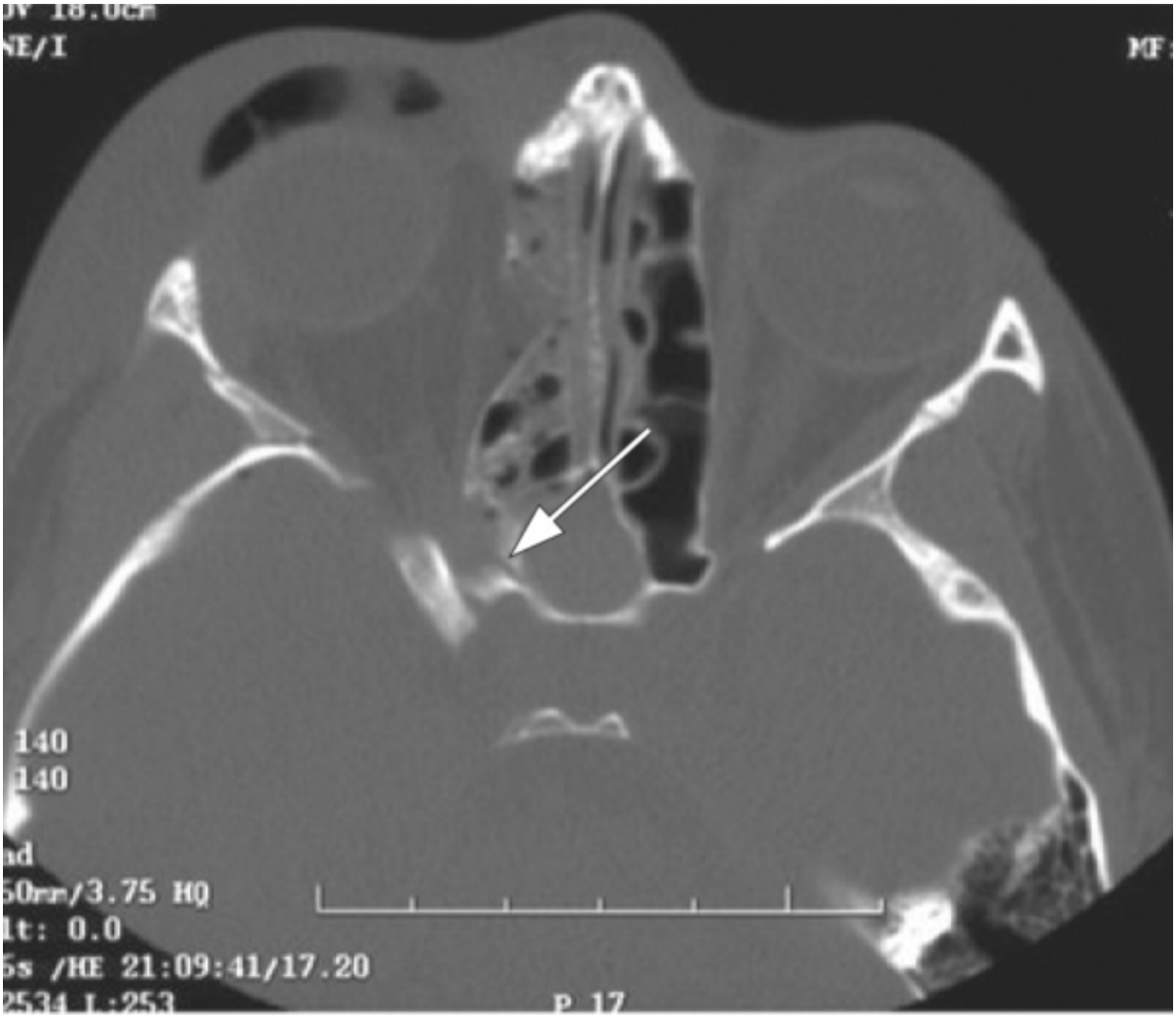

Figure 96-1 Axial computed tomography scan demonstrating a fracture of the right optic nerve canal *(arrow)* in a patient involved in a motor vehicle accident.

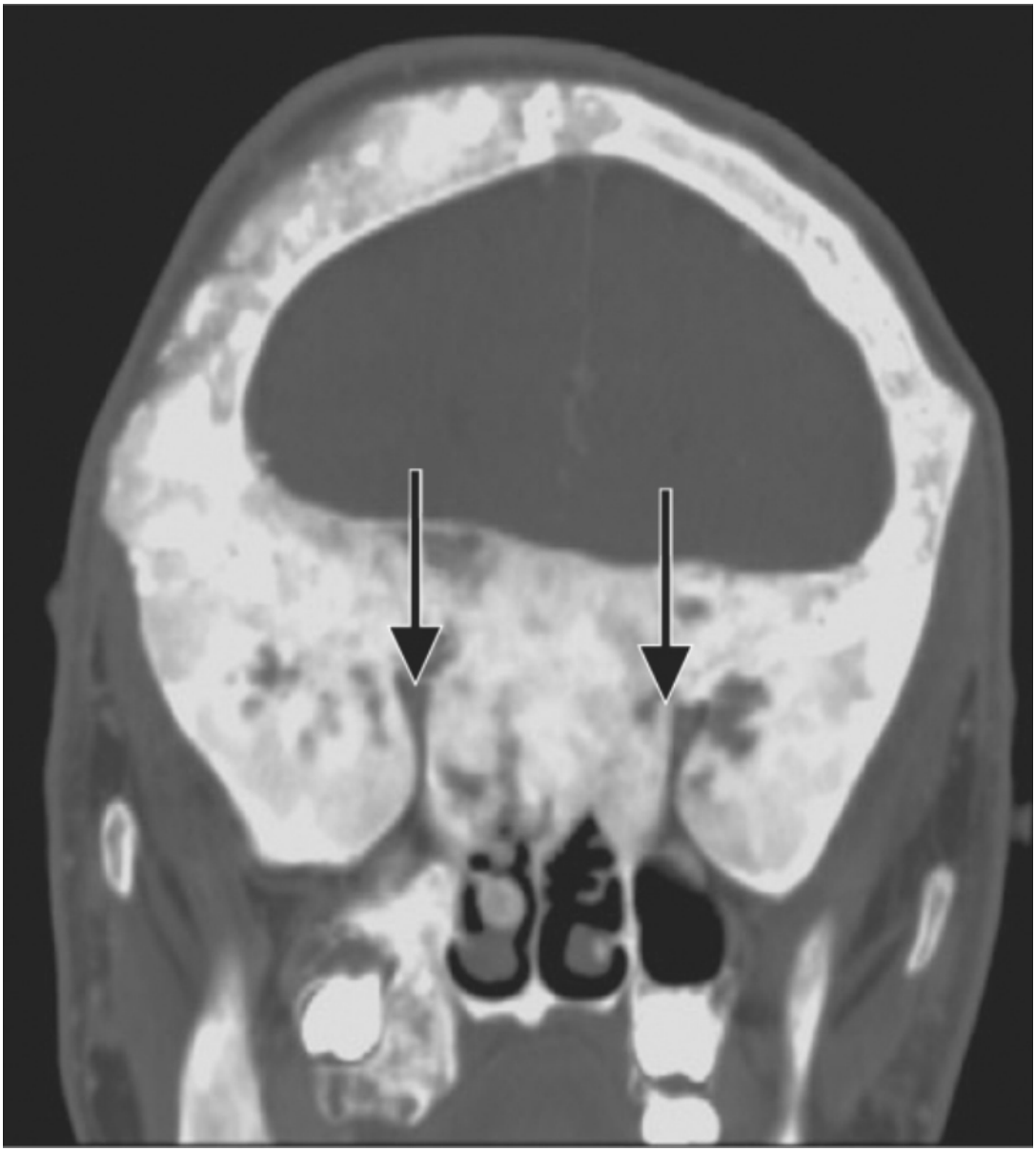

Figure 96-2 Coronal computed tomography scan demonstrating severe fibrous dysplasia with bilateral compression of the optic nerves *(arrows)*.

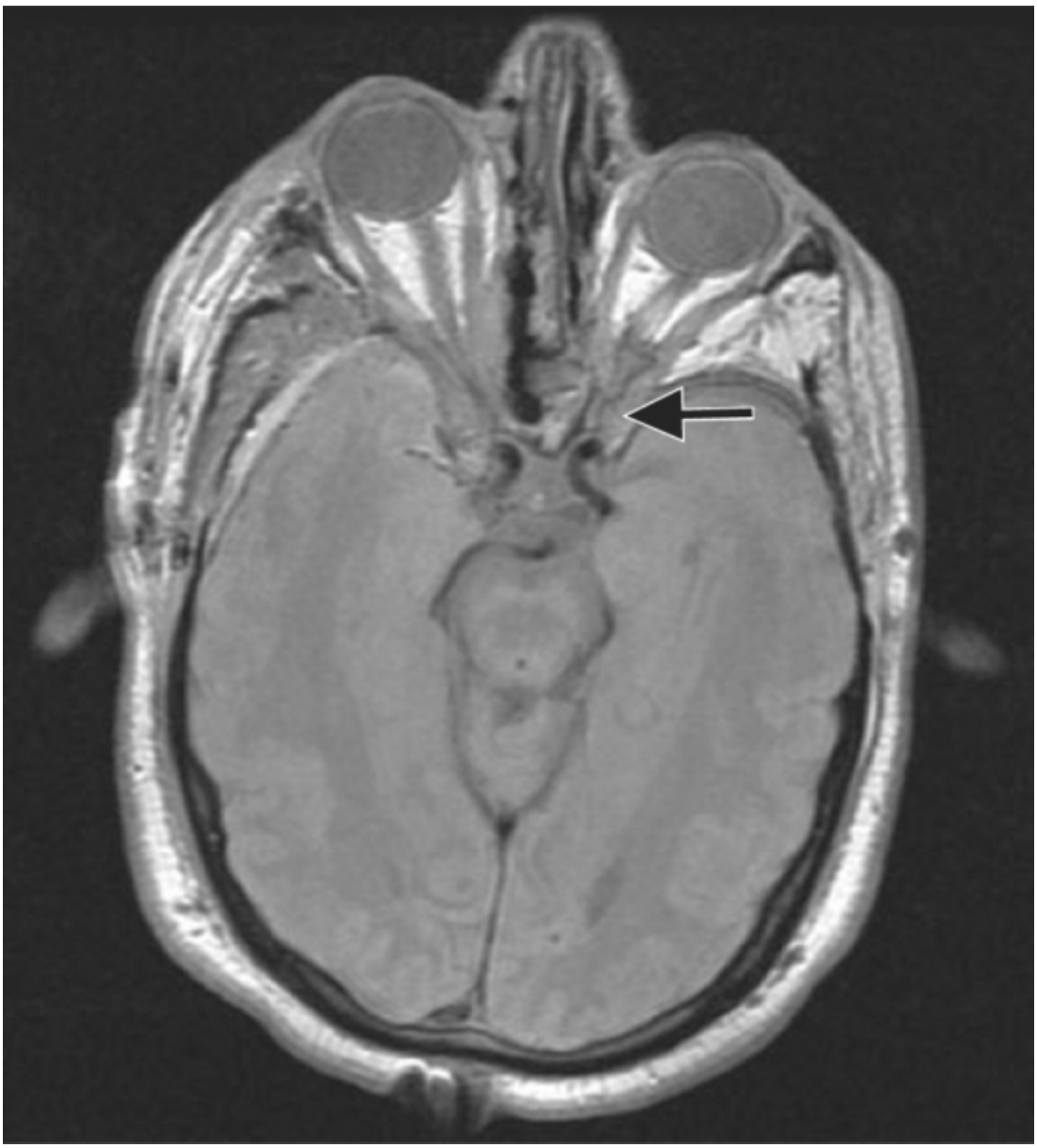

Figure 96-3 Axial magnetic resonance image demonstrating perineural extension of adenoid cystic carcinoma *(arrow)* along the left optic nerve.

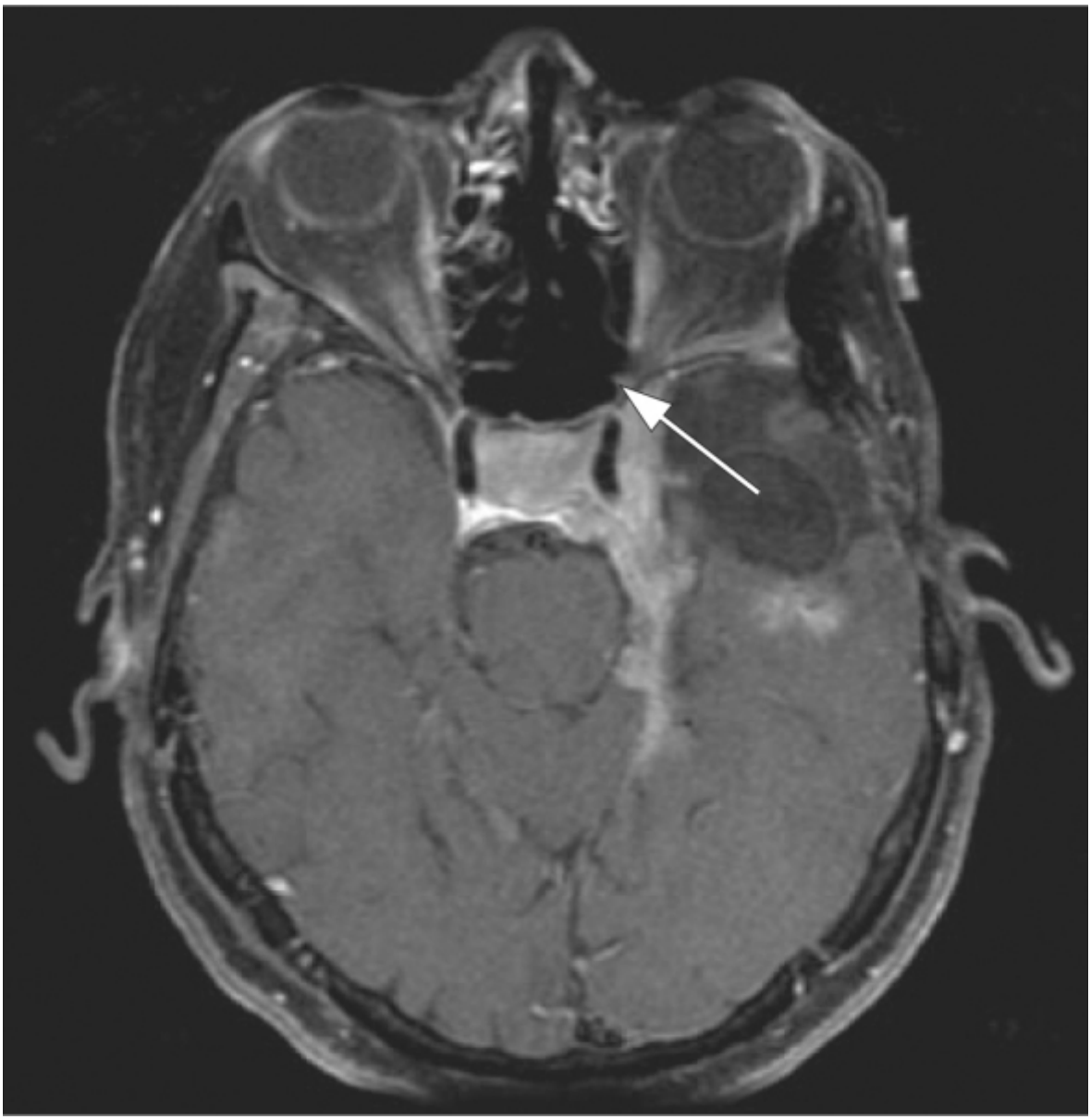

Figure 96-4 Axial magnetic resonance image demonstrating a recurrent meningioma *(arrow)* compressing the optic nerve within its canal and intracranially.

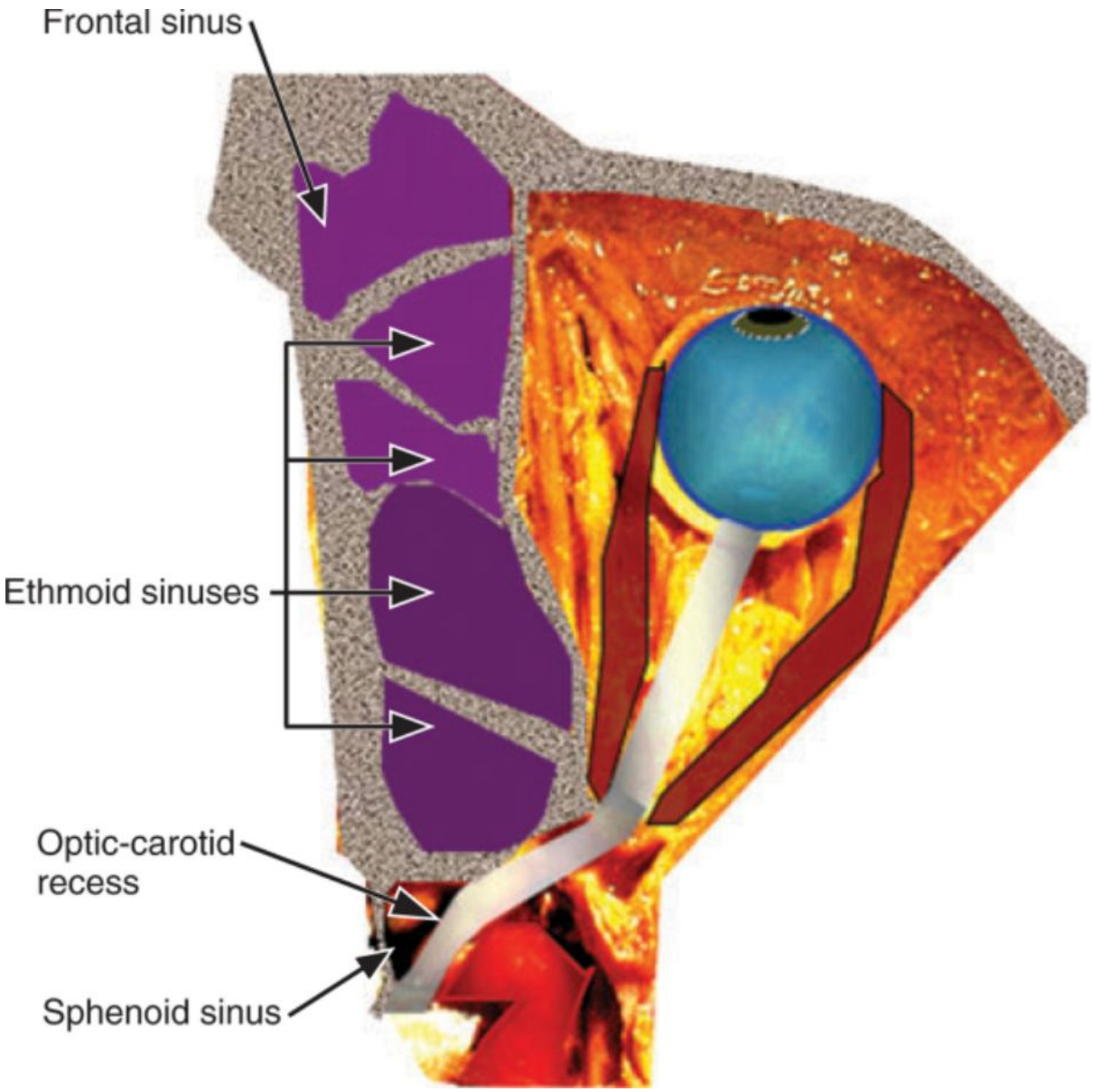

Figure 96-5 Diagrammatic representation in an axial plane demonstrating the lateral-to-medial direction of the optic nerve as it is followed in an anteroposterior direction and the relationship of the optic canal and internal carotid artery.

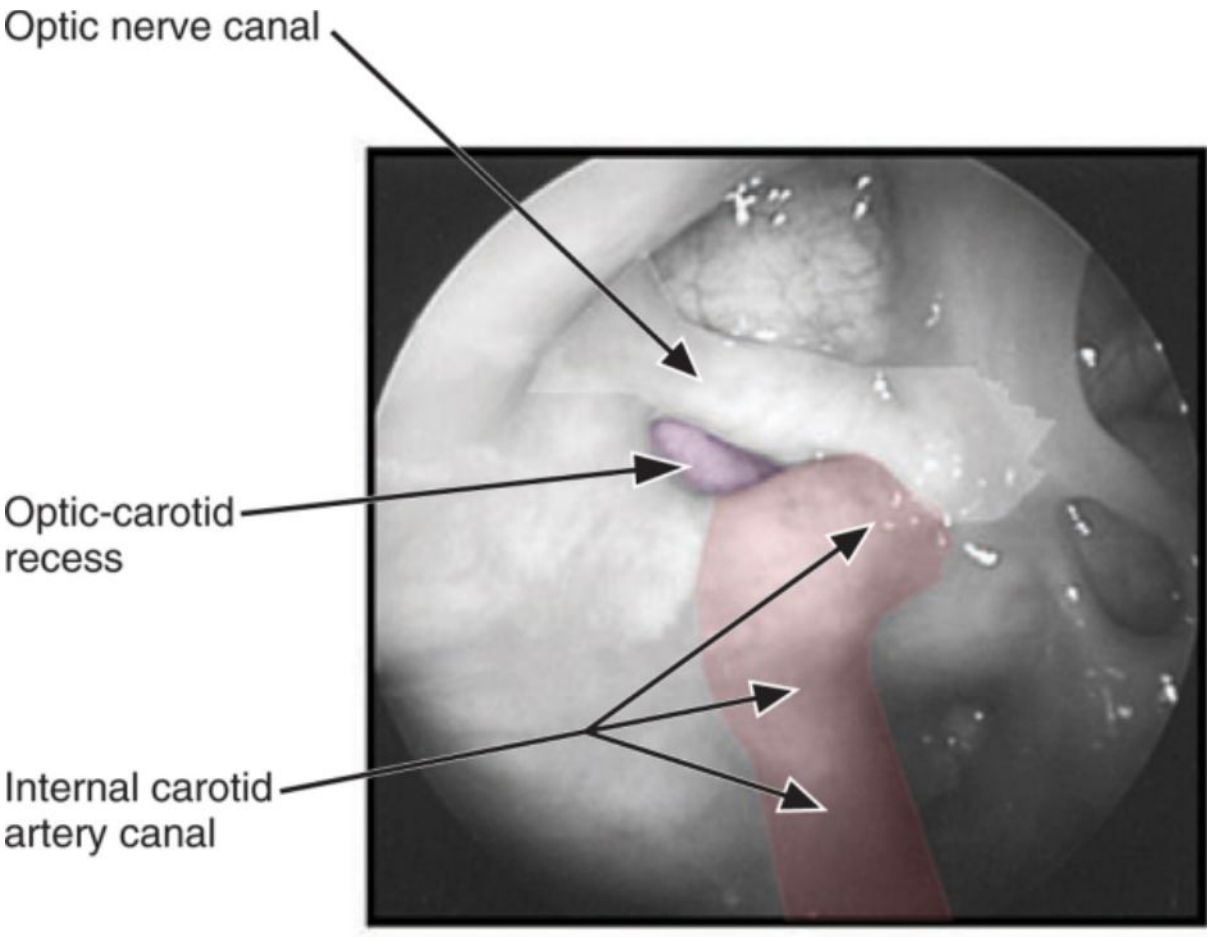

Figure 96-6 Digitally enhanced (highlighted structures) endoscopic view of the right sphenoid demonstrating the relationship of the optic nerve canal and the internal carotid artery. In this patient, pneumatization of the optic strut resulted in a deep optic carotid recess. Pneumatization of the anterior clinoid forms the recess superior to the optic canal.

INDICATIONS

There are numerous indications for optic nerve decompression, including but not limited to optic neuropathy secondary to a compressive effect from either benign or malignant tumors, inflammatory processes such as Graves' disease, or fibro-osseous lesions such as fibrous dysplasia. Direct or indirect TON that has not responded to conservative or medical management is likewise an indication.

SURGICAL TECHNIQUE

The patient is positioned supine on the operating table and an orotracheal airway is secured. We insert cottonoids soaked in 0.05% oxymetazoline as a topical decongestant. Image guidance or intraoperative navigation systems are useful, if not critical for the surgery. If an optical tracking navigation system is available, we fix the head in a slight turn toward the surgeon's side with a three-pin head holder. We then obtain appropriate image guidance registration via surface fiducial markers (placed before imaging), a light-emitting diode–based face mask, or a laser surface registration device. For electromagnetic navigation systems, the helmet is placed and a straight probe is used for registration. Once this has been completed, the middle turbinate, lateral nasal wall, and posterior nasal septum are then sequentially infiltrated, in a posterior-to-anterior direction, with 0.5% lidocaine (Xylocaine) with 1:100,000 epinephrine. Resection of the right middle turbinate may be necessary, albeit rarely, to improve visualization and widen the space for instrumentation. In addition, outfracturing of the inferior turbinates widens the nasal corridor.

We use a 0-degree rod-lens endoscope to provide visualization throughout most of the procedure and reserve the use of an angled endoscope for visualization of the superior ethmoid sinuses or skull base. An uncinectomy is completed with back-biting rongeurs and a straight-blade microdébrider. Its superior aspect is spared because the frontonasal recess does not need to be exposed. We then complete anterior and posterior ethmoidectomies with a microdébrider or Tru-cut rongeurs (Fig. 96-7). This exposes the skull base (fovea ethmoidalis) and lamina papyracea. In patients who suffer trauma to the optic canal and orbit, the surgeon should be attentive to the possibility of exposed periorbita or orbital fat, or both. Via a transmeatal approach we remove the inferior aspect

of the superior turbinate. The superior turbinate forms the medial aspect of the posterior ethmoids, and removal of it exposes the natural ostium of the sphenoid sinus. The ostium is enlarged in an inferior and medial direction until the endoscope can be inserted inside the sphenoid sinus for close visual inspection. Herniation of intracranial structures or an exposed internal carotid artery may be associated with trauma to the skull base. After these possibilities are discarded, the rest of the rostrum of the sphenoid is removed with up- and down-biting 2-mm Kerrison rongeurs. The rostrum must be removed until the roof and lateral wall of the sphenoid sinus are in plane with the roof of the posterior ethmoid sinuses and the lamina papyracea, respectively. Inferiorly, the posterolateral septal artery runs 1 cm above the posterior choana, and it is best to avoid injury to it. It can easily be controlled with bipolar or suction electrocautery. Use of suction electrocautery posterior to the rostrum or over the lamina papyracea is best avoided. Next, the posterior aspect of the lamina papyracea is removed to expose the periorbita covering the orbital apex (Fig. 96-8). Any injury to the periorbita will result in herniation of orbital fat, thereby impeding visualization. Cauterizing the fat with bipolar electrocautery may control limited herniation of this fat. The position of the optic nerve is at the top of the orbital apex, and it follows a lateral-to-medial and caudal-to-cephalad direction. The posterior ethmoidal artery is 4 to 6 mm anterior to the nerve.

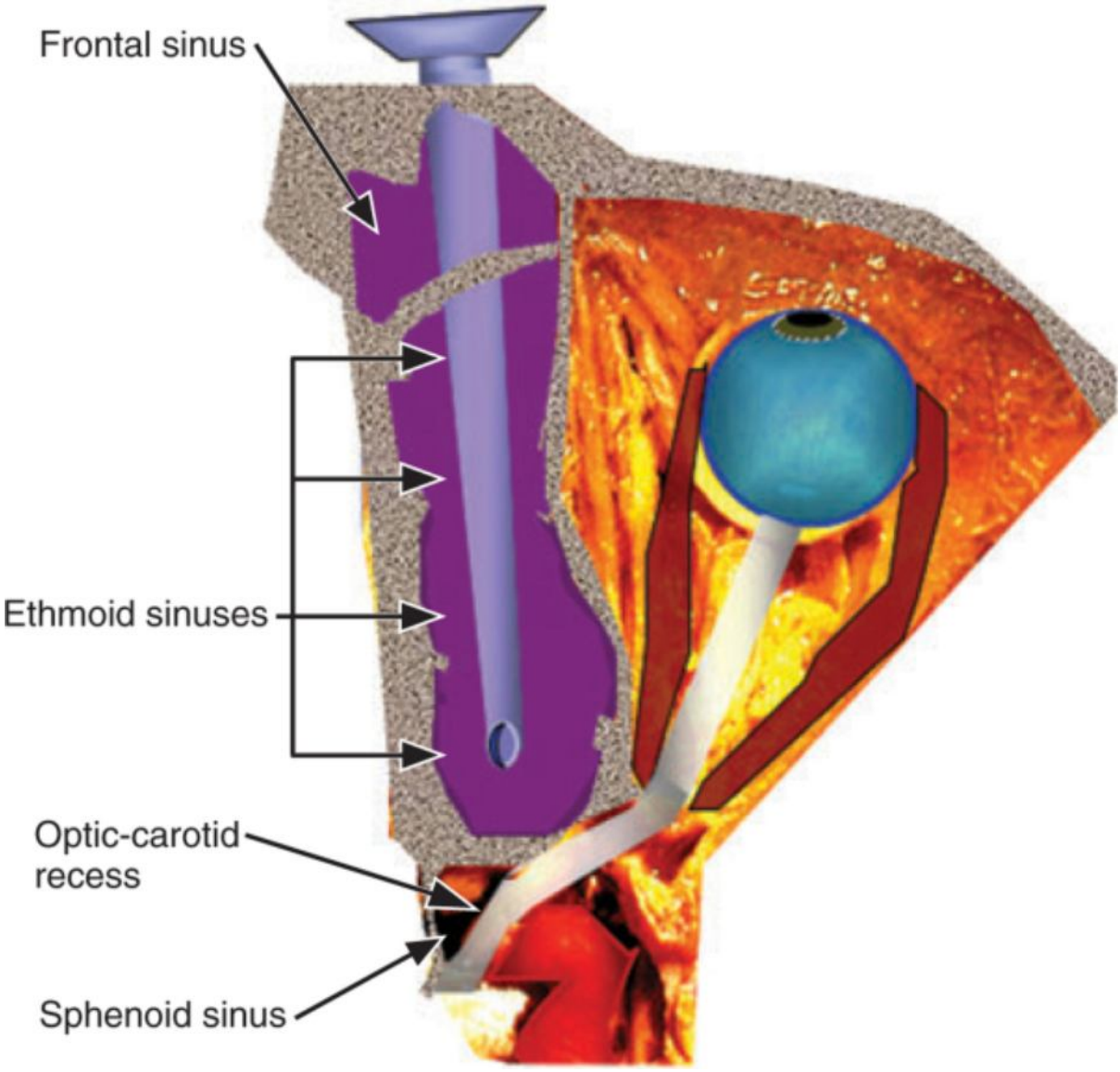

Figure 96-7 Diagrammatic representation in an axial plane demonstrating complete ethmoidectomy and sphenoidotomy to expose the lamina papyracea and skull base.

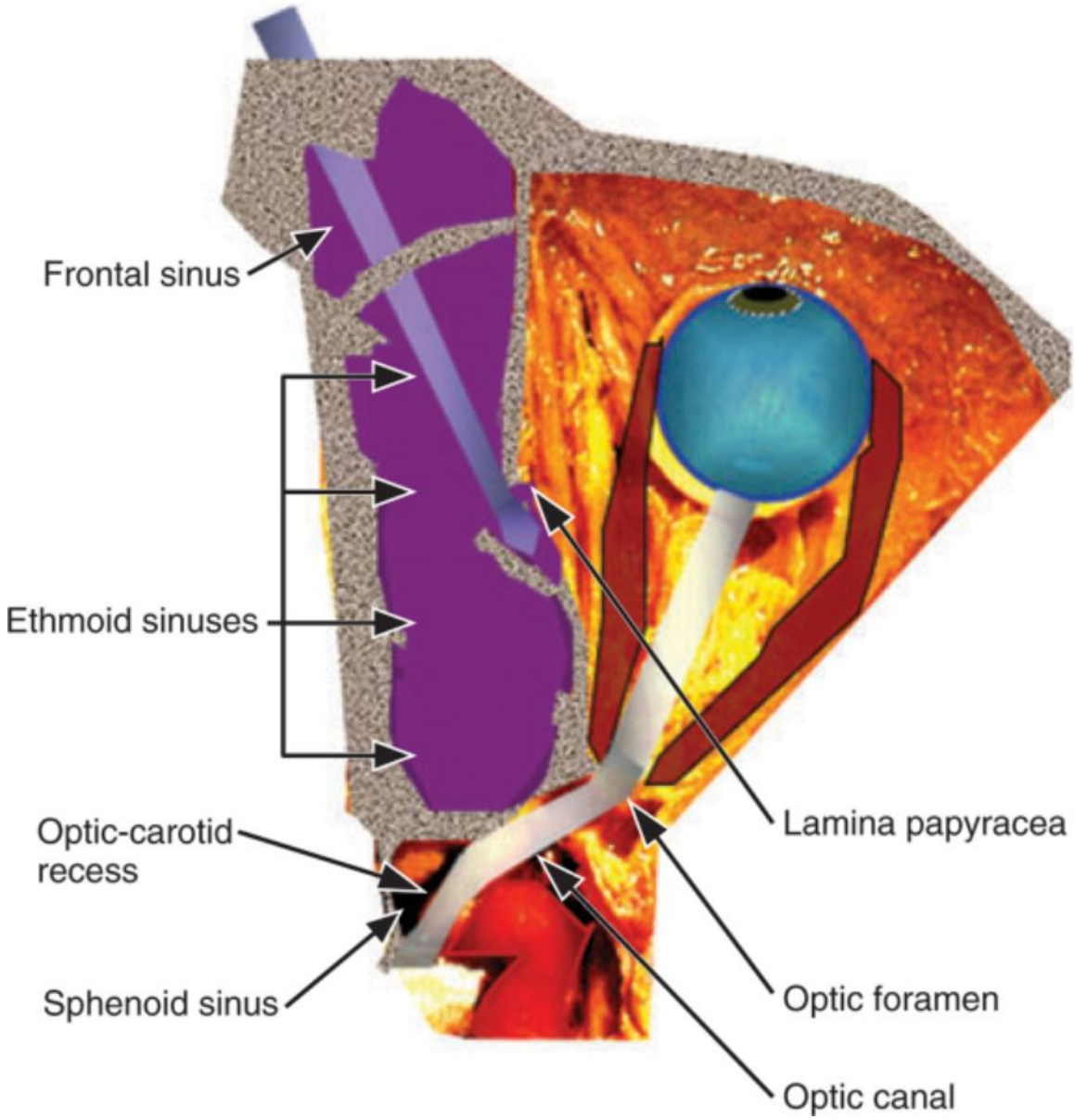

Figure 96-8 Diagrammatic representation in an axial plane demonstrating removal of the posterior aspect of the lamina papyracea. The periorbita is preserved to avoid herniation of orbital fat.

If the foramen and optic canal are fractured, they can often be removed with a small Cottle elevator or 1- to 2-mm bone curette to displace the fractured fragments medially. Otherwise, the medial aspect of the foramen and canal has to be thinned with a 3-mm burr mounted on a high-speed drill (Fig. 96-9). It is critical to provide continuous irrigation to avoid thermal injury to the nerve or ophthalmic artery. The canal is decompressed back to the optic carotid recess because this marks the optic strut and the point where the nerve is intradural (Figs. 96-10 and 96-11). The decompressionshould include the medial 120 degrees. More extensive decompression may be achieved if the optic strut (below the nerve) and anterior clinoid (above the nerve) are pneumatized (see Fig. 96-6). The optic nerve sheath is not opened because this does not contribute to the decompression. Furthermore, opening the sheath may cause leakage of cerebrospinal fluid (CSF) by opening the meningeal sleeve that often accompanies the optic nerve extracranially.

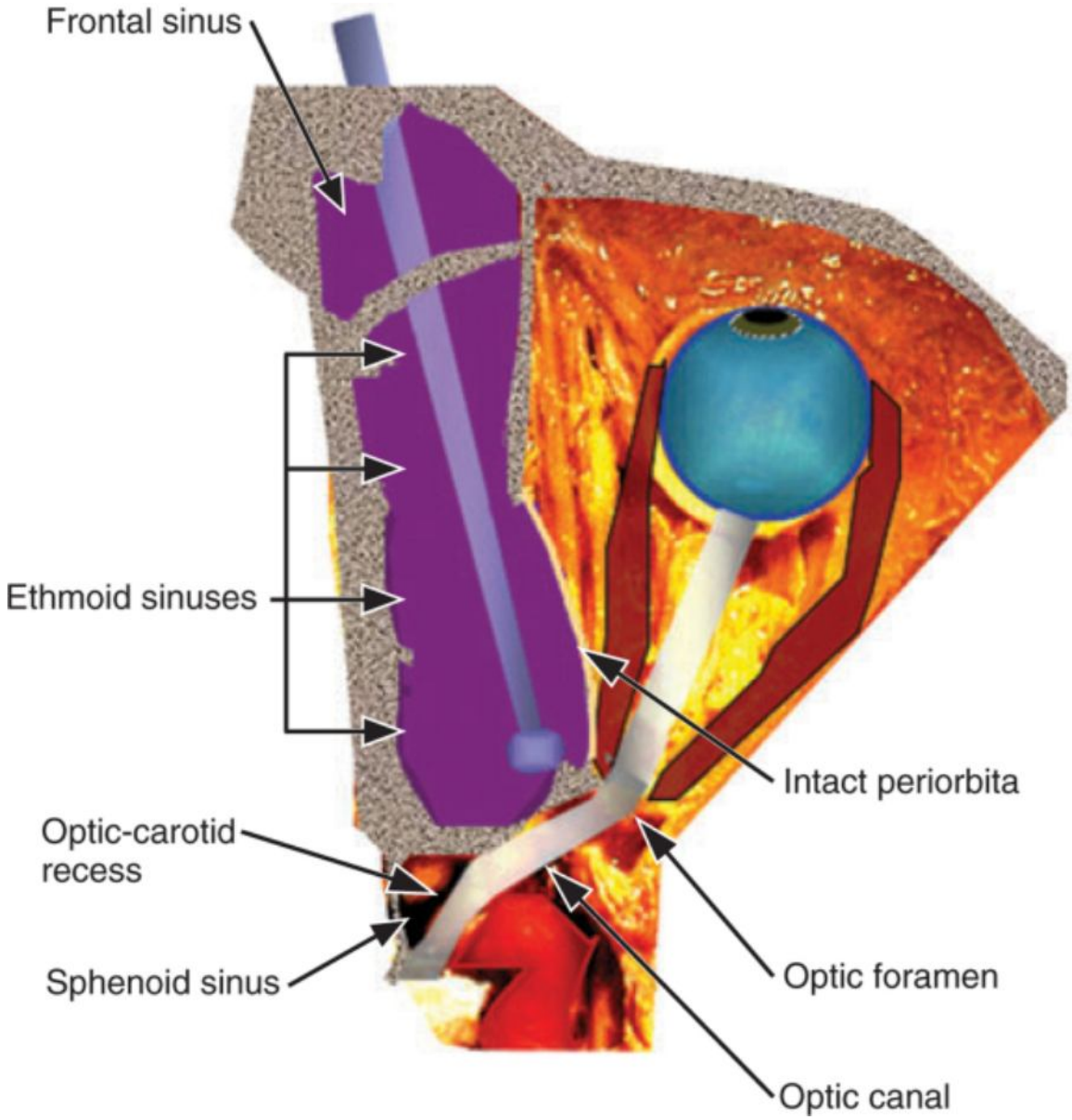

Figure 96-9 Diagrammatic representation in an axial plane demonstrating drilling of the optic foramen (pterygoid process).

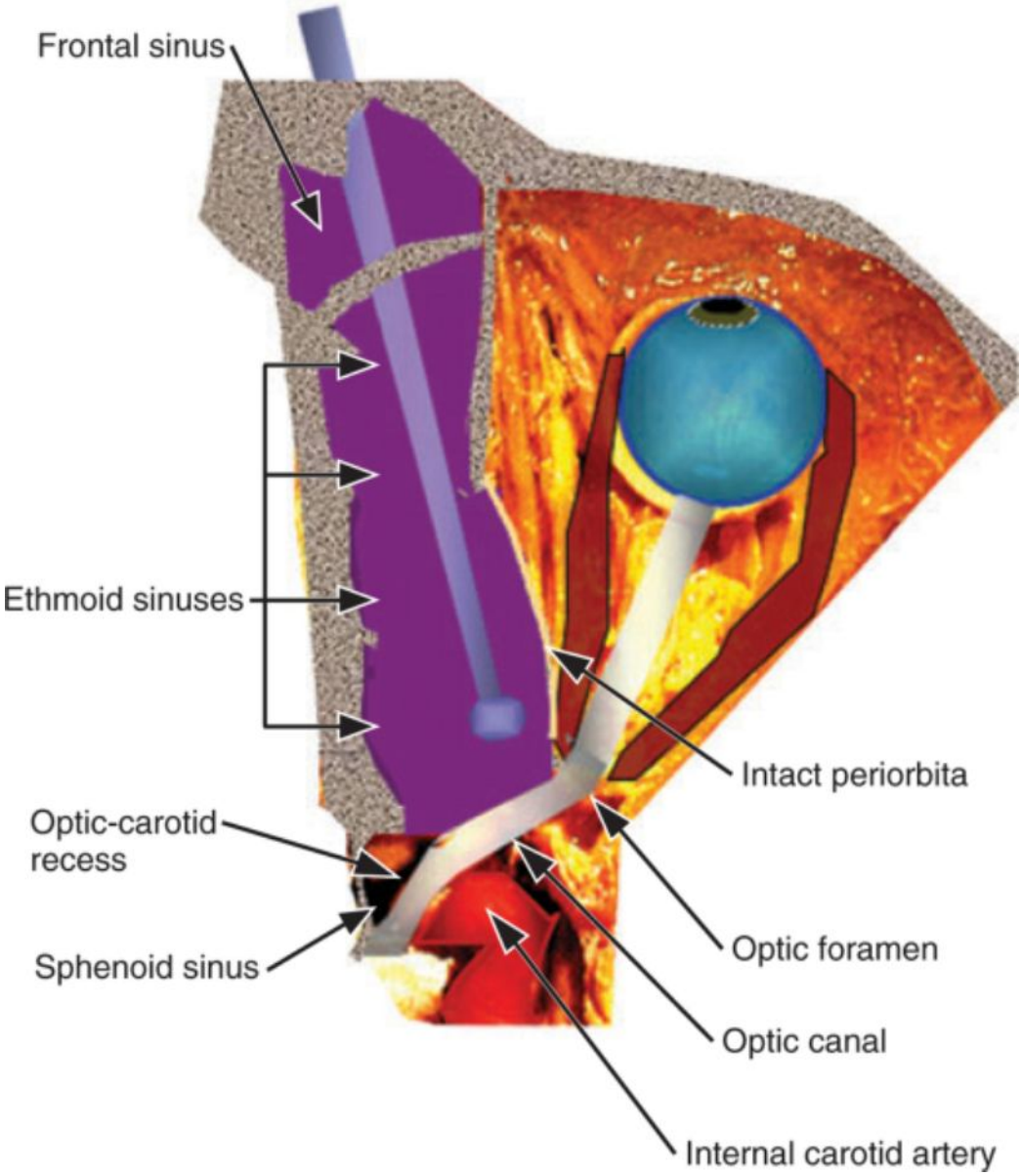

Figure 96-10 Diagrammatic representation in an axial plane demonstrating complete decompression of the optic canal.

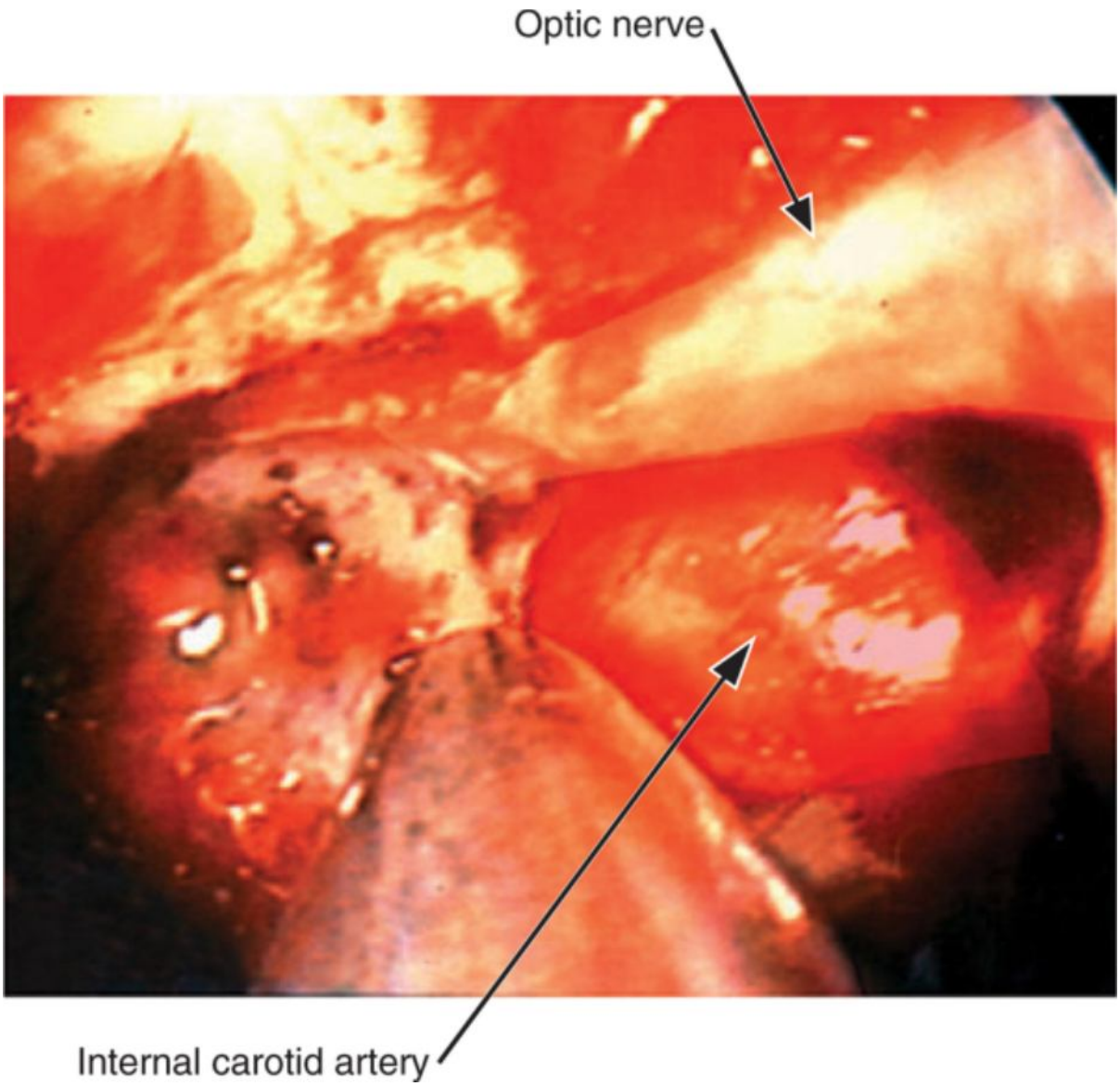

Figure 96-11 Digitally enhanced (highlighted structures) endoscopic view of a decompressed left optic nerve.

Subsequent to the decompression, copious irrigation is performed and Silastic splints are sutured to the nasal septum to prevent synechia formation. A small amount of packing can be used if hemostasis is an issue; however, one must be cautious because of the exposed optic nerve.

POSTOPERATIVE MANAGEMENT

Typically, patients are kept in the hospital overnight after optic nerve decompression. We routinely administer systemic steroids before, during, and after endoscopic decompression. Nasal saline sprays are started before discharge home. We routinely use silicone splints to prevent the formation of synechiae between the nasal septum and turbinate. Generally, they remain in place for 10 to 14 days. Nasal débridement is usually carried out biweekly for the first few postoperative visits, followed by monthly intervals. We recommend switching to a nasal saline douche after the first week to assist in remucosalization. We do not perform routine postoperative imaging after optic nerve decompression. Patients are monitored with serial visual examinations postoperatively, in addition to formal visual field testing in the first 24 hours.

COMPLICATIONS

Cerebrospinal Fluid Leak

In a patient with postoperative rhinorrhea, testing of the fluid for β2-transferrin or β-trace protein is prudent to confirm a CSF leak. Accumulation of irrigation fluid in the sinonasal tract, with subsequent drainage, can mimic a CSF leak in the immediate postoperative period.[15,16]

A postoperative CSF leak is best managed by immediate re-exploration and surgical repair.

Postoperative Sinonasal Bleeding

Significant postoperative nasal bleeding most commonly arises from a branch of the internal maxillary or anterior ethmoidal arteries, which are amenable to endoscopic control. Angiography with embolization is reserved for patients who are not surgical candidates. Care must be taken with this complication because blind nasal packing in the setting of a dehiscent, decompressed optic nerve may lead to a decrease in vision.

Subcutaneous Emphysema

Because of a dehiscence in the medial orbital wall and optic canal, vigorous nose blowing or sneezing can result in significant subcutaneous air collection and facial swelling. Patients should be forewarned about appropriate nasal precautions. Observation and potential use of antibiotics if any signs of infection are present are all that is required.

Visual Deterioration

Vision can worsen after optic nerve decompression. Such deterioration can be related to heat injury to the optic nerve as a result of inadequate irrigation during thinning of the optic canal or a vascular event affecting the ophthalmic artery. In addition, small bony fragments can lead to impingement of the optic nerve during outfracture of the optic canal with a curette.

PEARLS

- Thorough review of preoperative imaging is necessary to account for variations in anatomy such as Onodi cells.
- Image guidance is an extremely valuable tool in cases in which normal anatomy is distorted, such as by fibrous dysplasia and skull base neoplasms.
- Removal of the posterior 1 cm of the lamina papyracea facilitates identification of the optic foramen and optic canal decompression.
- Copious irrigation must be used during drilling over the optic canal to prevent thermal injury.
- One hundred twenty–degree medial decompression can be achieved to alleviate compressive symptoms.

PITFALLS

- Failure to bring all septations and air cells in plane with the lateral orbital wall will make decompression more difficult.
- Breach of the periorbita will lead to herniation of fat and poor visualization.
- Incision of the nerve sheath is contraindicated because it may produce a CSF leak.
- Care must be taken with postoperative packing to not cause iatrogenic compression of the exposed optic nerve.
- A high-speed drill shaft can cause alar burns if left in the same position for a prolonged period.