POSTTRAUMATIC OPTIC NERVE DECOMPRESSION

YADRANKO DUCIC, M.D., F.R.C.S.(C)
KAMEL ITANI, M.D., F.A.C.S.

INTRODUCTION

In approximately one third of cases, patients with maxillofacial trauma sustain ocular injury, of which 10% involve blinding. Although the causal temporal relationship between traumatic head injury and loss of vision has been observed since the time of Hippocrates, the exact etiology of this phenomenon is not completely understood. Obviously, direct impingement upon the optic nerve may result from the displacement of fractured osseous elements at the level of the orbital apex. However, visual loss is frequently noted following blunt head injury. This may be a result of the disparity in the elastic time constants (hence, tissue oscillation frequency) between the bone of the optic canal and that of soft tissue elements, such as the optic nerve. The variation in the amplitude of vibration between the bones and the soft tissues at the level of the orbital apex will result in a local cavitation (as with missile injuries), with subsequent disruption of small vessels and hemorrhage. This is of paramount importance. It is realized that the optic nerve occupies the entire diameter of the optic canal, which varies in size from a mean width of 4.78 mm anteriorly to a mean width of 7.07 mm posteriorly. Acute intracanalicular hemorrhage or edema within the narrow confines of the osseous optic canal may lead to nerve compression, and hence, the potential for visual loss. As the optic nerve has no significant capacity for regeneration once axonal degeneration has occurred, both the recognition and the treatment of posttraumatic optic nerve compression must be addressed as expediently as possible.

SURGICAL DECOMPRESSION AND APPROACHES

There has been significant debate regarding the role and route of surgical decompression in this patient population. Although not definitively proven in a randomized prospective trial, there appears to be substantial evidence to support surgical intervention in selected circumstances. Numerous surgical approaches have been described to provide access to the optic canal and to facilitate optic nerve decompression.

Intracranial decompression via a frontotemporal craniotomy may be contraindicated in the (often noted) concomitant presence of cerebral concussion or contusion as a result of the prolonged retraction necessary to effect adequate decompression of the optic canal. We do not favor an extracranial lateral orbitotomy or an intracranial low temporal craniotomy approach due to the potential for traction injury to the oculomotor, trochlear, and abducens nerves traversing the superior orbital fissure and the ophthalmic artery in the inferolateral portion of the optic canal. Likewise, we avoid a transantral ethmoidectomy route to inferior optic canal decompression due to the need for mobilization of the vidian and maxillary nerves and the potential for avulsion of the ophthalmic artery. Visualization via this approach is quite limited.

We generally favor the extracranial external transethmoidal approach that is outlined below. The principles utilized in this technique are also readily applicable to the transnasal approaches that are often utilized by endoscopic sinus surgeons.

PERTINENT ANATOMY

In the average adult, the optic nerve (measuring 4 cm) is enveloped by the meninges and encircled by protective orbital fat except for the length of the osseous optic canal which spans a distance of 5 to 11 mm within the lesser wing of the sphenoid bone (Figures 1 and 2). The optic canal is occupied by the optic nerve in its superomedial portion and the ophthalmic artery (which, along with coaxial arcades from the pia mater) in its inferolateral portion (Figure 3). The bone of the optic canal is thickest at the level of the optic foramen (average 0.62 mm) and thinnest in its course along the superolateral wall of the sphenoid sinus (average 0.2 mm). In our experience, in approximately 5% of optic nerves, there is an incomplete osseous covering within the sphenoid sinus. This may not be apparent from preoperative radiographic examination. Sharing the lateral wall of the sphenoid sinus are (from superior to inferior) the optic nerve, the carotid artery, the second division of the trigeminal nerve and, on occasion, some posterior ethmoidal cells.

The transethmoidal approach to orbital nerve decompression involves the identification and removal of parts of the frontal process of the maxilla, the lacrimal bone, the ethmoidal air cell system, and the anterolateral wall of the sphenoid sinus to allow for improved access of the optic canal. If the frontoethmoidal suture line is followed within the orbit, the anterior ethmoidal artery, the posterior ethmoidal artery, and the optic nerve as it enters the optic canal are sequentially encountered (Figure 1). The mean distance between the posterior ethmoidal artery and the optic nerve varies between 4 and 7 mm (Figure 2). One should be aware that in 5% of individuals, the anterior ethmoidal artery either is not present or is vestigial.

PATIENT EVALUATION

The initial step in the management of a patient with traumatic optic neuropathy requires a specific diagnosis of optic nerve injury, after due consideration is given to other causes of decreased vision. This may be a complicated task in the multitrauma patient with a decreased level of consciousness and other concomitant globe or orbital injuries. For example, a hyphema may mask the underlying presence of traumatic optic neuropathy or retinal edema.

Testing of visual acuity is of paramount importance in any patient who has sustained significant facial trauma. Confrontation or computerized visual field testing may reveal deficits prior to any apparent visual acuity abnormalities. A diligent and specific search must be undertaken to rule out an afferent pupillary defect (Figure 4). The diagnosis of traumatic optic neuropathy may be made at the bedside by documenting a decrease in visual function (i.e., visual acuity or visual fields) in the presence of an afferent pupillary defect and in the absence of major ocular injury.

Visual evoked potentials are a reliable indicator of the integrity of the visual pathway and may be a good predictor of visual recovery in posttraumatic blindness. Approximately 90% of patients with positive visual evoked potentials, whether normal or abnormal, will have complete or partial visual recovery. Only 20% of patients with initially absent potentials will show evidence of any recovery of vision. Although prognostically helpful, the inability to predict with absolute certainty the visual outcome in any given patient has limited the widespread acceptance of visual evoked potentials in most centers.

To rule out the presence of fracture segment displacement through the optic canal, computed tomography (CT) of the orbit, performed in the horizontal and sagittal planes, is an essential diagnostic modality that should be considered in any patient suspected of having an orbital apex injury. Plain radiographs, although occasionally useful as a diagnostic modality, generally cannot provide the specific detail available with CT. Ophthalmological consultation should also be sought.

Once the diagnosis of traumatic optic neuropathy has been made, one needs to elicit any previous or ongoing history of medical problems that would contraindicate the possible usage of high-dose steroids (e.g., a significantly immunocompromised state, poorly controlled diabetes mellitus, or a bleeding peptic ulcer). The presence of any of these conditions may compel the surgeon to bypass initial medical therapy in favor of immediate, direct surgical intervention.

INITIAL TREATMENT

If visual acuity and/or visual fields are compromised and there is an afferent pupillary defect but no fracture displacement or hematoma at the level of the orbital apex evident on CT, a megadose steroid protocol is followed. We utilize a protocol that has been proven therapeutic in spinal cord injury studies (Figure 5). In the absence of an active documented cerebrospinal fluid (CSF) leak, we initiate the protocol with a loading dose of Solu-Medrol (30 mg/kg), followed by 15 mg/kg in 2 hours, and then continued every 6 hours for 72 hours. If there is no subjective or objective improvement in the patient's visual status within 48 hours, steroid therapy is discontinued.
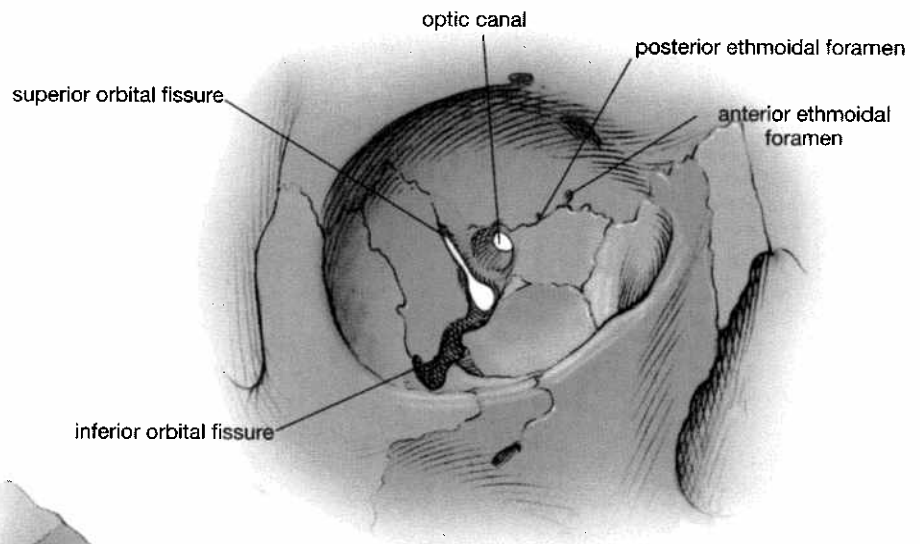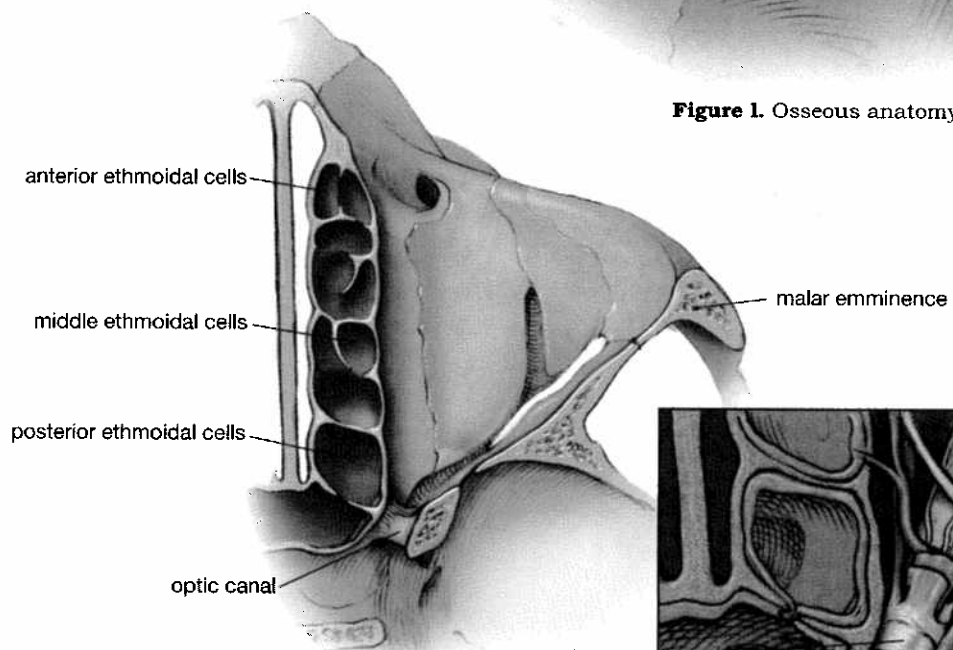
Figure 1. Osseous anatomy of the orbit.

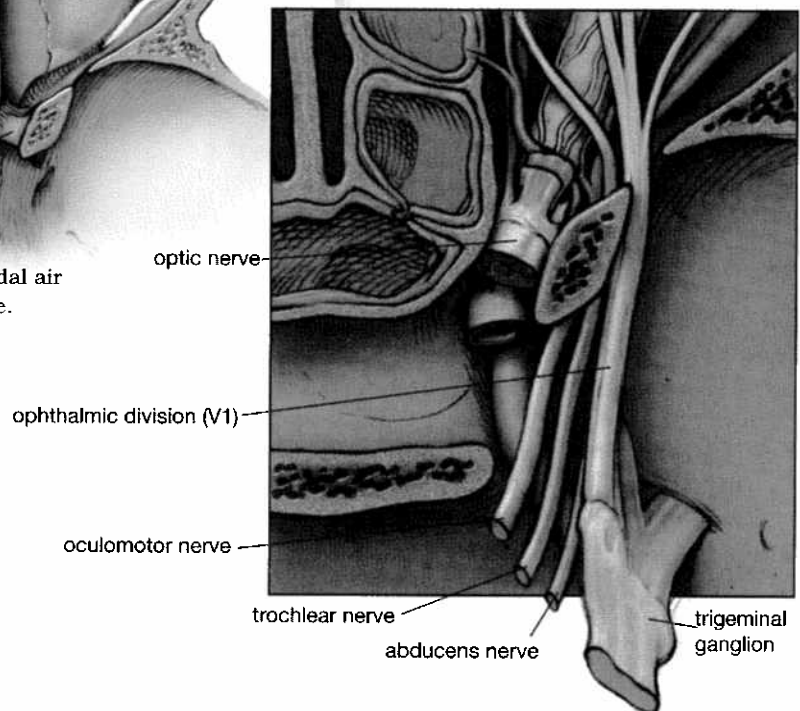**Figure 2.** Relationship of ethmoidal air cell system to the optic nerve.

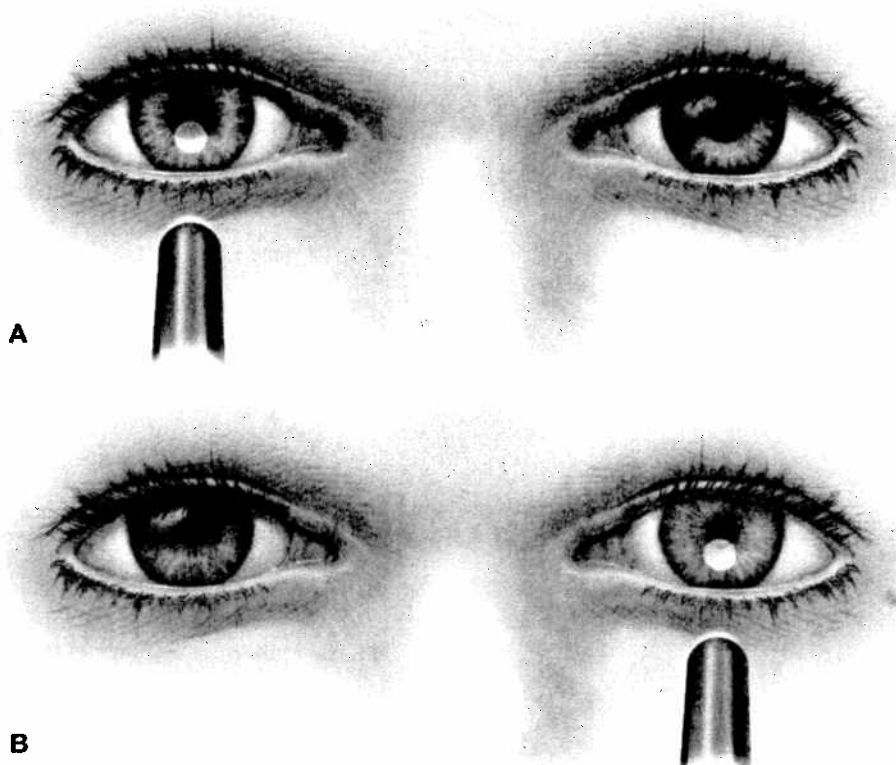**Figure 3.** Superficial anatomy of the orbit, superior view.

Figure 4. Patient with afferent pupillary defect in the right eye. **A,** light directed at the injured right eye; both pupils paradoxically dilate. **B,** light directed at the normal left eye; both pupils constrict.

If there is significant visual improvement, steroids are continued for an additional 5 days, followed by a taper in the dosage over the subsequent 5 days. We also routinely institute treatment with a blood-brain barrier penetrating antibiotic (e.g., 2 gm cefotaxime intravenously every 8 hours).

It has been our observation that patients with worsening visual acuity following trauma tend to do better than those with a similar degree of immediately stable visual loss. We proceed with optic nerve decompression if the patient has the following: an ongoing CSF leak; deterioration in visual status while receiving high-dose steroid therapy or after the discontinuation of steroid therapy; or an apparent impaction of an osseous fragment into the optic canal at the initial radiographic examination. Unless

a frontotemporal craniotomy is required for the treatment of concomitant intracranial injury, our preferred approach to optic nerve decompression is via an external transethmoidal access. We believe that this represents the safest, simplest, and most direct route to the optic canal.

In our experience, the absence of a documented orbital apex fracture coupled with a lack of improvement in visual status after an initial 48-hour trial of megadose steroid therapy indicates a poor outcome in terms of recovery of function in patients with optic nerve decompression. Thus, in this scenario, it is generally not offered to the patient.

SURGICAL TECHNIQUE
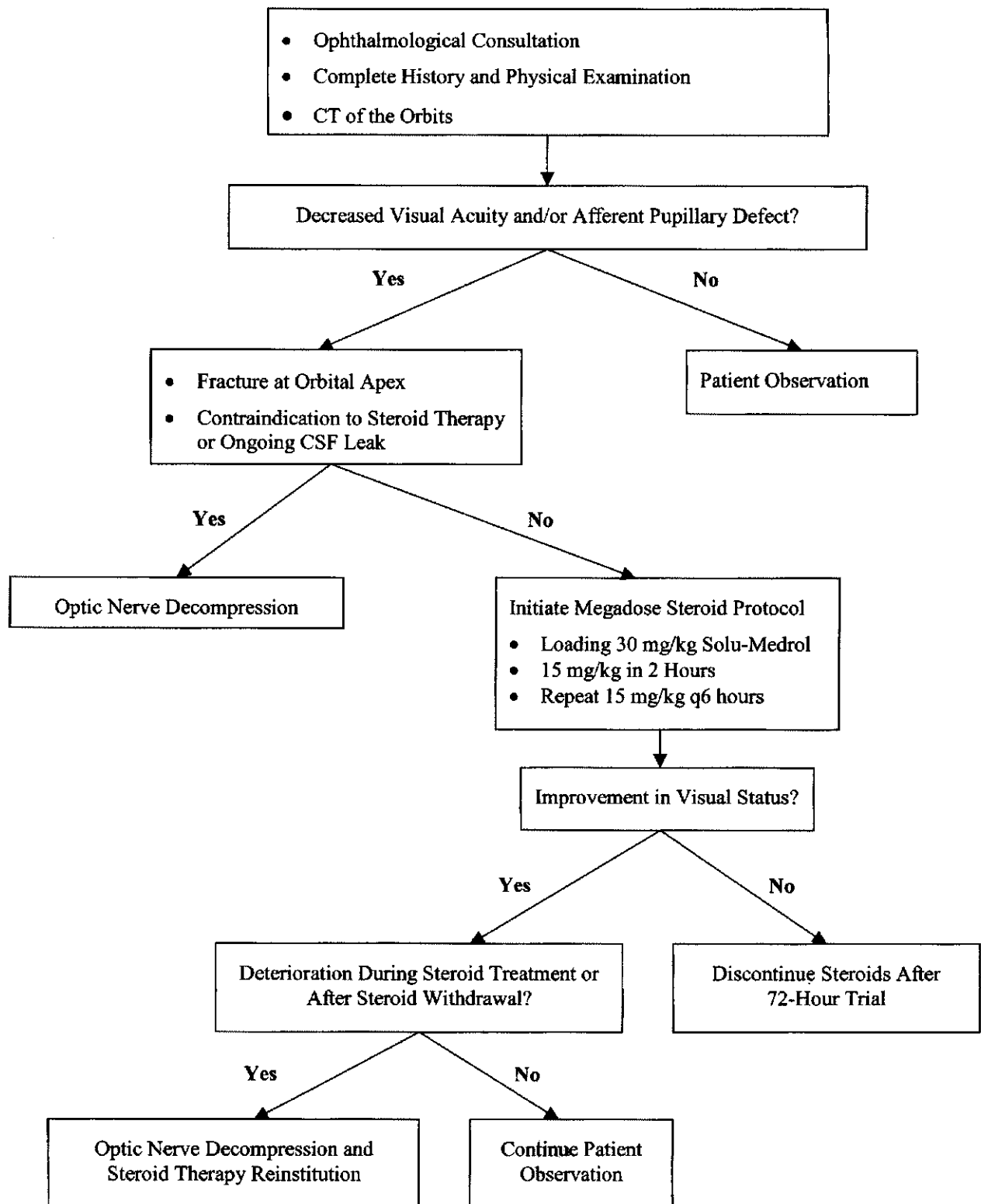The patient is placed supine onto the operating table

Figure 5. Algorithm for treatment of a patient with possible posttraumatic optic nerve decompression.

with the head resting securely on a headrest. The face is prepped with a Betadine surgical scrub and the upper two thirds of the face is draped in a sterile manner. The nasal cavity ipsilateral to the optic nerve injury is infiltrated with 1% lidocaine with 1:100,000 epinephrine solution followed by the topical application of 4% cocaine solution impregnated on 1-inch cottonoid patties. The medial canthal region is likewise infiltrated with 1% lidocaine with 1:100,000 epinephrine solution to assist in effecting a bloodless field. A temporary tarsorrhaphy suture of 5-0 silk is utilized to facilitate intraoperative corneal protection. It is removed at the completion of the procedure.

At this point, a #15 scalpel blade is used to fashion an incision extending for a length of approximately 3 cm and coursing along midway between the nasal dorsum and the medial canthal region (Figure 6A). Care is taken to preserve the supratrochlear neurovascular bundle. The angular vessel branches at the margin of the incision should be controlled using electrocautery. The medial canthal ligament is detached and marked with a silk suture for later reattachment at the conclusion of the procedure (Figure 6B). Next, by maintaining dissection in the subperiosteal plane, one will necessarily elevate the lacrimal sac laterally from the lacrimal fossa, exposing the anterior and posterior lacrimal crests. Visualization is increased by gently retracting the globe laterally with a malleable retractor. We routinely release pressure on the orbital contents secondary to retraction by removing all retractors at least every 10 minutes. The anterior and posterior ethmoidal arteries are controlled with bipolar cautery and/or hemoclip application as they are seen emerging from likenamed foramina along the frontoethmoidal suture line in the medial wall of the orbit.

A 4-mm osteotome is then used to perforate the junction of the lacrimal bone with the nasal processes of the frontal and maxillary bones, as well as passing through the lamina papyracea (Figure 7A). This provides the surgeon with a direct view into the ethmoidal sinus. A small rongeur and/or curette is utilized to remove the septations between the individual air cells, thereby completing an external ethmoidectomy, from anterior to posterior. The use of a Killian long-bladed self-retaining nasal speculum aids in allowing the surgeon unimpeded two-handed access to facilitate subsequent decompression (Figure 7B).

Magnifying loupes or a microscope fitted with a 300-mm lens will greatly increase the visualization afforded to the surgeon at this point. Once the ethmoidal cells have been exenterated, the superolateral wall of the ethmoid and the adjacent sphenoid are inspected to allow for identification of the characteristic bony bulge of the optic canal (Figure 8A). Any displaced or impacted osseous fragments at this level are gently dissected free with small periosteal elevators and removed from the operative field. Mucosa is stripped from the lateral wall of the sphenoid sinus (Figure 8B). Caution should be exerted while dissecting the mucosa covering the cavernous portion of the carotid artery that courses just inferolateral to the optic canal. Hemostasis may be facilitated by the application of oxymetazoline or 4% cocaine-soaked cottonoid patties.

Beyond simple removal of impacted osseous fragments, further decompression will rely upon positive identification of the optic tubercle, which initially appears as the bulge of the optic canal into the ethmoidal air cell system. This is the thickest part of the optic canal that will be encountered. It should be drilled away using a long-shaft small diamond burr on a Stryker drill under constant suction-irrigation (Figure 8C). The remainder of the medial wall of the optic canal may also be removed with the drill (as for the optic tubercle) or, alternatively, with microcurettes. This latter approach is usually made possible by the presence of the significantly thinner optic canal that is found posterior to the tubercle. The entire medial wall of the optic canal needs to be removed in order to allow for complete and adequate optic nerve decompression. We do not believe that it is necessary to routinely open the sheath of the optic nerve and, in fact, believe that this may be harmful. Hemostasis should be meticulous. We often place a small piece of Gelfoam over the decompressed optic nerve to facilitate this process without the need for bipolar cautery at this level.

The medial canthal ligament should be reattached at the conclusion of the procedure. We prefer to reattach it to the medial orbital wall with the use of a pre-threaded Mitek microscrew (Figure 9). This allows for the most precise and stable long-term fixation of the medial canthal ligament. If this is not available, 5-0 Tebdec or 5-0 nylon suture may be used to affix the ligament to either the periosteum (of the medial orbital wall) or to a drill hole made by the surgeon (using a 1.7-mm drill bit) in the medial orbital wall bone. Closure of the initial incision is performed in two layers (5-0 Vicryl sutures in a buried interrupted fashion for the subcutaneous layer, and 5-0 Prolene sutures for the skin incision). Closure is accomplished over a small rubber drain. The long-term esthetic result of this access incision is generally quite well camouflaged and most often results in favorable scar formation.

POSTOPERATIVE CARE

Visual acuity and pupillary reflexes should be quantified postoperatively and followed serially. Any deterioration in visual function should be noted, and treatable causes identified and addressed. An orbital hemorrhage is suspected if there is increasing prop-
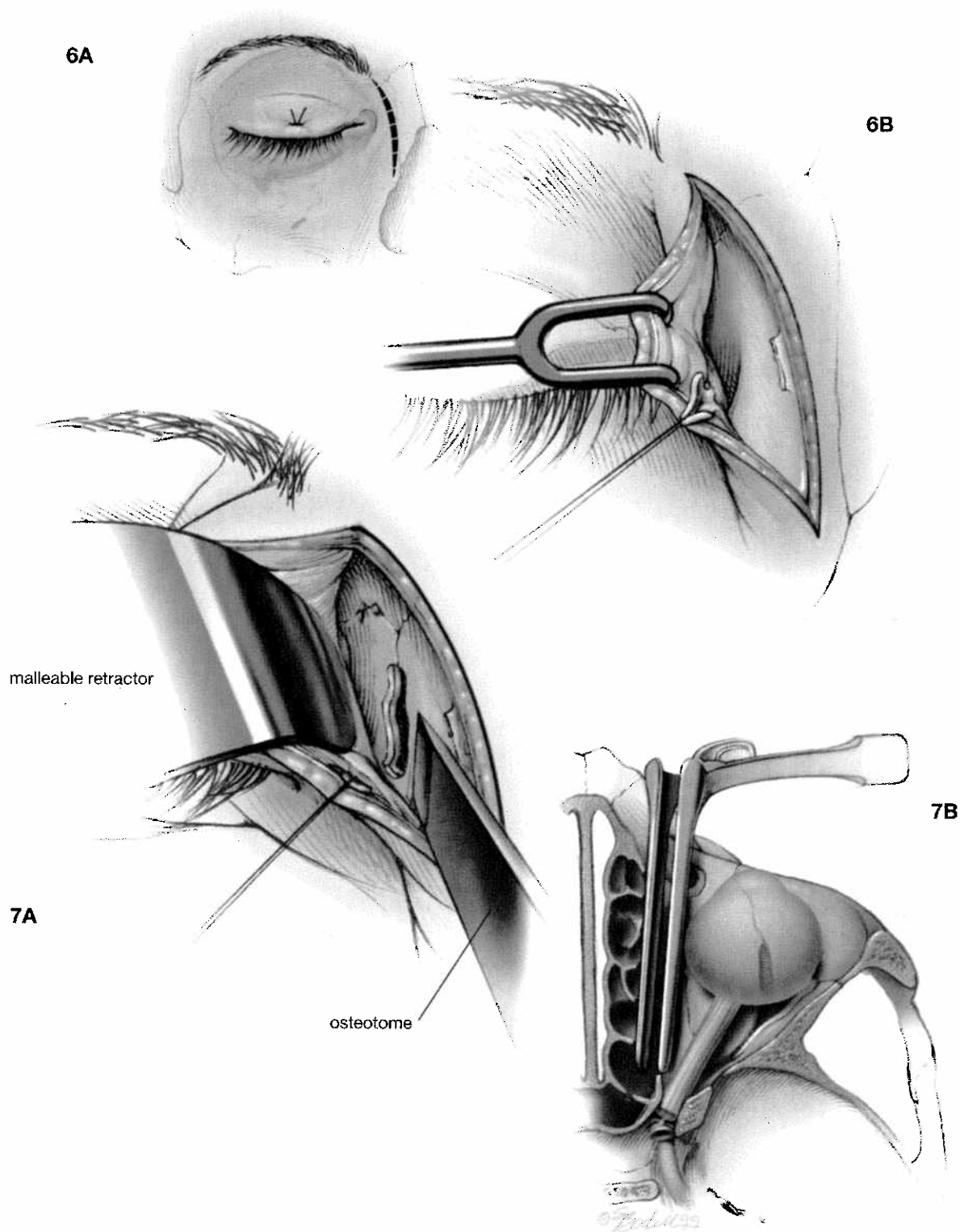
6A

6B

malleable retractor

7B

7A

osteotome

Figure 6. A, access incision. Note the tarsorrhaphy suture for globe protection. **B,** the medial canthal tendon is detached as dissection proceeds along the medial orbit in a subperiosteal plane.

Figure 7. A, entrance is made into ethmoidal air cell system with an osteotome directed medially. **B,** a nasal speculum is inserted through the ethmoidectomy site.
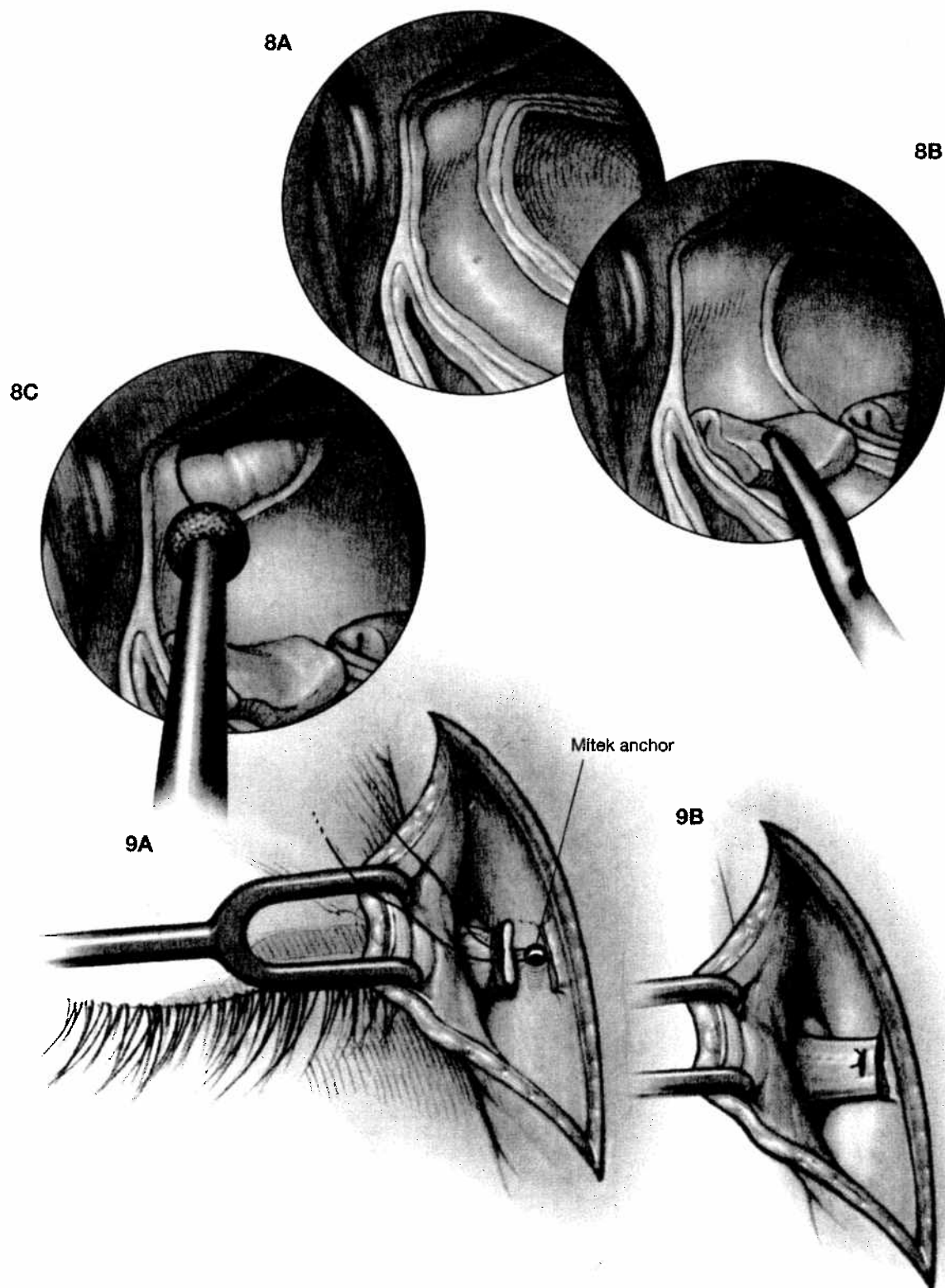
Figure 8. A, the osseous bulge of the optic canal is identified. **B,** mucosa is gently elevated to allow access to the optic canal. **C,** the optic canal is decompressed using a Stryker hand drill fitted with a round diamond burr.

Figure 9. A, the medial canthal tendon is reattached utilizing a Mitek anchor. **B,** the final position of the medial canthal tendon.

tosis, increased resistance to retropulsion, subconjunctival hemorrhage, or motility disturbances. An associated worsening of the optic neuropathy (e.g., visual loss or afferent pupillary defect) or the presence of central retinal artery occlusion or pulsation warrants an immediate lateral canthotomy and inferior cantholysis. Persistent central retinal artery occlusion or pulsation requires an anterior chamber tap and/or exploration of the orbit. The new onset of pupillary abnormalities with or without associated deterioration in the patient's visual acuity may herald the development of an orbital hematoma.

The rubber drain is removed on the first postoperative day, and the cutaneous sutures are removed on the fifth day. For a period of 2 weeks postoperatively, the patient should keep the head elevated even during the night. Nose blowing is forbidden for 1 month to avoid the development of orbital emphysema.

Prognostically, the patient may expect return of visual function to begin within 1 month of the decompression and continue to improve for up to 1 year postoperatively.

CONCLUSIONS

Although there is significant controversy in the surgical literature regarding the role, if any, of optic nerve decompression, the authors believe that with proper patient selection the results achieved in this patient population may be consistently rewarding. Prior to embarking on this surgery, it is necessary to have a thorough familiarity with the complex three-dimensional anatomy as well as with the surgical approach outlined. It is our belief that the safest and most direct route to decompression of the optic nerve is with the use of the external transethmoidal approach to the orbital apex.