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Endoscopic orbital decompression

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Lateral wall decompression

The effectiveness of orbital decompression has been enhanced by the use of endoscopes in orbital decompressive surgery. In patients with severe proptosis and visual compromise, surgical intervention provides definitive therapy. Endoscopes have allowed for more complete removal of the bone posteriorly to the orbital apex under direct visualization, which has led to a decrease in morbidity. Two-wall decompression is now the minimum procedure that successfully achieves release of contents at the orbital apex. Whether the endoscope is used as an adjunct surgically or postoperatively to facilitate debridement, it has become an important tool in the armamentarium for the successful management of proptosis. The endoscopic view of posterior ethmoidal sinuses and the sphenoid sinuses enables the surgeon to successfully carry out a posterior decompression, and provides the opportunity to decompress the optic nerve if the need arises.
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The goal of orbital decompressive surgery is to restore the normal volume relationship between the orbital soft tissues and the surrounding orbital bone volume. Orbital decompression reduces proptosis, intraorbital pressure, and subsequently improves orbital congestion, allows for improvement in corneal exposure, and can also address compressive optic neuropathy. Various methods of orbital decompression have been described, such as removal of orbital fat (orbital fat decompression), and one or a combination of the 4 orbital walls with or without the use of endoscopic visualization.

Proptosis can lead to both cosmetic deformity and functional problems. Proptosis can present from various etiologies, such as infectious, noninfectious, traumatic, vascular, and neoplastic processes and congenital abnormalities. However, the most common cause of proptosis is thyroid eye disease (TED; Graves ophthalmopathy, thyroid orbitopathy). If the condition is left untreated, it can lead to exposure keratopathy, lagophthalmos, diplopia, and subsequent visual compromise.[1-3]

Pathogenesis

TED is associated with Graves disease in more than 80% of cases and is an autoimmune disorder characterized by inflammation and expansion of the orbital fat and extraocular muscles. Although it has been identified in all age groups, it primarily affects adults in the fourth and fifth decades of life. TED can profoundly impair a patient's ability to work and perform activities of daily living.

The pathophysiology of TED is not completely understood; there is evidence for both humoral and cell-mediated immune processes.[1-4] TED has both an active phase and an inactive, or chronic, phase. Active-phase TED results from lymphocytic infiltration of the orbital and periorbital fat and muscles. The active phase generally persists for 6 months to 3 years and is typically longer in smokers and those with prolonged hypothyroidism.[5,6] Nevertheless, the duration and severity of dis-

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ease varies within each individual and can often be unpredictable. After the inflammatory process ends, fibrosis and the associated disabling symptoms persist in the stable, inactive phase.

Immunomodulatory agents are believed to affect the activity of orbital lymphocytes and fibroblasts.[7,8] The most commonly used immunomodulators include corticosteroids with or without adjunctive external beam orbital radiation (XRT) in moderate to severe cases of TED. Corticosteroids are typically administered orally or intravenously. Local injections of corticosteroids into the orbit have failed to provide an effect in improving orbitopathy.[9,10]

Treatment

The goals of medical therapy are to shorten the duration and minimize the severity of the active phase; however, disease management varies widely.[11,12] The goal is to reduce the chronic-phase disfigurement and disability produced by irreversible fibrosis. In the active phase, orbital decompression is reserved for those few patients with optic neuropathy unresponsive to medical therapy or in whom medical treatment is contraindicated, as for those with insulin-dependent diabetes.[12]

XRT was first used empirically to treat TED. Although the mechanism for its action is not fully understood, radiation (XRT, typical total dose = 20 Gy) is biologically active against infiltrating lymphocytes, tissue-bound monocytes, and fibroblasts to alter the local cellular matrix and interrupt the inflammatory process in a more permanent fashion than can be achieved with corticosteroids.[9,13] One recent prospective, double-masked, sham-controlled clinical trial produced more debate than consensus regarding the efficacy of XRT therapy for TED.[14,15]

As an alternative to corticosteroids and XRT, other immunomodulatory agents, such as azathioprine, cyclosporine, rituximab, intravenous immunoglobulin, and plasmapheresis, have been used, but they play a more limited role.[16-21] Efficacy studies have not been well modeled, and are mainly small, retrospective, or uncontrolled.[22-26] Nevertheless, the use of rituximab has shown promise in treating those particular patients with severe TED that are corticosteroid-resistant.[27]

Stable-phase TED

The goal of stable-phase orbital decompression is to restore the globe to its normal position within the orbit, which results in an improvement in proptosis, exposure keratopathy, and orbital congestion/pain. Patients who have been clinically stable and have documented a lack of change in clinical measures for an interval of 6 months are candidates for orbital decompressive surgery. The patient should be stable endocrinologically, not have any recent change in thyroid therapy, and be medically cleared for approximately 2 hours of surgery requiring general anesthesia.

Before the advent of high-resolution computed tomography (CT), the choice of orbital wall to be removed was driven by the familiarity of the surgeon with one particular surgical approach rather than an understanding of the orbital pathology. In 1911, Dollinger first described orbital decompression by modifying Kronlein's lateral orbital approach to removing orbital tumors.[28] His approach decompressed orbital contents into the temporal fossa, but decompression was limited by the temporalis muscle, and there was a visible scar at the incision site. In 1931, Naffziger performed decompression through a fronto-temporal craniotomy.[22] The success of this procedure was limited by the morbidity associated with transmitted brain pulsations to the eye, as well as the increased risk of meningitis and cerebrospinal fluid leaks. Sewall[22] used a medial orbital wall approach to achieve decompression via an external ethmoidectomy approach. Hirsch performed decompression by removal of the floor via the Caldwell-Luc approach. In 1957, Walsh and Ogura[29] improved this approach further by extending the decompression to the medial wall.

The high-incidence rates of infraorbital nerve hypoesthesia (up to 66%) lead surgeons to look for surgical options with less morbidity.[23,30] As a result, there was a return to the direct orbital approaches in the 1980s, which included the eyelid approach to the lateral wall, floor, and medial wall, which reduced the complication rates substantially.[16-18,24] Furthermore, with the advent of endoscopic sinus surgery, sinus surgeons have introduced endoscopic medial wall decompressions as another arsenal in orbital decompression. This has led to a complete medial wall decompression, which is often difficult through a direct transcaruncular approach.[19-21,25]

The etiology of proptosis lies in the discrepancy between the volume of orbital contents and the accommodative capacity of the orbit. The bony orbit is made of several bones that constitute a fixed space. It follows that proptosis can be reduced by measures that either decrease the orbital contents (ie, steroids, irradiation) or increase the capacity of the orbit (ie, orbital decompression). Surgical decompression involves the removal of medial, superior, or inferior walls. The indications for surgical intervention are as follows:

- progressive proptosis
- optic neuropathy
- optic nerve compression
- orbital inflammation or pain refractory to medical management
- exposure keratopathy; and
- cosmetic deformity

The type of decompression applied depends on the needs of the patient, the degree of proptosis, and the technical skills of the surgeon. The selection of a surgical procedure varies on the basis of a complete clinical examination, CT or magnetic resonance imaging interpretation, and premorbid facial photographs. The clinical examination includes Hertel exophthalmometry (objective measurement of the amount of proptosis), upper and lower lid retraction, lagophthalmos, ocular motility, and corneal exposure.[26] Reviewing premorbid photos provides the surgeon the amount of proptosis reduction required to return the patient as close to their premorbid state as possible. Patients with preoperative motility impairment with or without diplopia are more likely to experience worsened motility or diplopia after medial wall decompression. These particular patients should be counseled about possible postoperative stra-

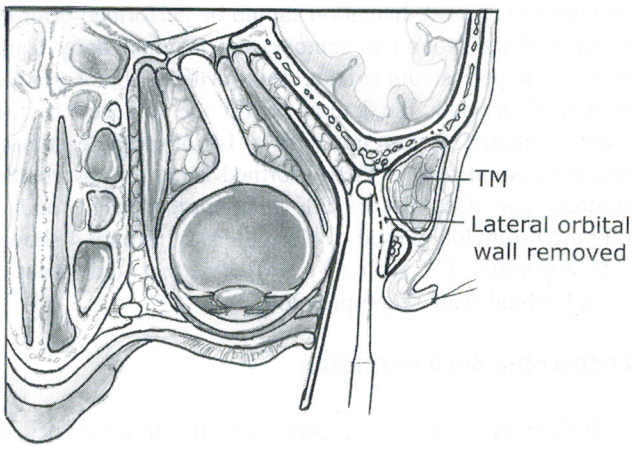

Figure 1 A malleable retractor is used to retract the orbital contents away from the lateral wall in this figure after undergoing a lateral wall decompression. Laterally, the bone is resected until the temporalis muscle becomes exposed. TM, temporalis muscle.

bismus surgery. The risk of postoperative diplopia is reduced in patients with patients with normal ocular motility or if they are to undergo fat or lateral wall decompression.

CT or magnetic resonance imaging is used to evaluate the relative contribution of the enlargement of the extraocular muscles to the observed proptosis.[31,32] Patients can demonstrate markedly enlarged extraocular muscles (EOMs) that fill virtually all the orbital volume. Others demonstrate normal extraocular muscles with expansion of the fat compartment, resulting in proptosis. However, the most common scenario is a balanced expansion of both the EOMs and orbital fat soft-tissue volume, resulting in proptosis.[33,34]

Furthermore, the para-orbital sinuses should be evaluated because they vary considerably and have an impact on the relative success of decompression in this area. When one considers a lateral wall decompression, the size of the lateral wall and sphenoid bone marrow should also be evaluated.

Surgical options: orbital decompression

When expansion of the orbital fat compartments mainly contributes to the proptosis, patients will benefit from fat

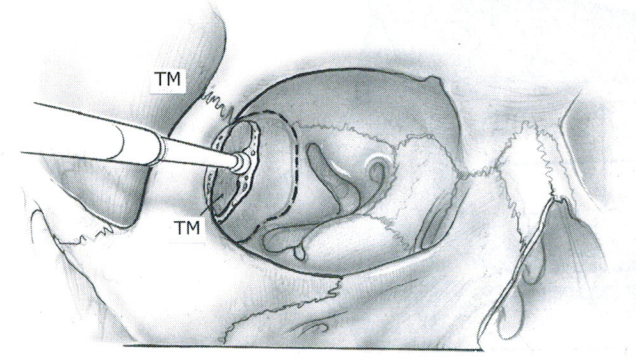

Figure 2 The left lateral orbital rim is thinned with a cutting burr. The superior extent of bone resection is at the level of the lacrimal fossa and the inferior extent is just lateral to the infraorbital canal. TM, temporalis muscle.

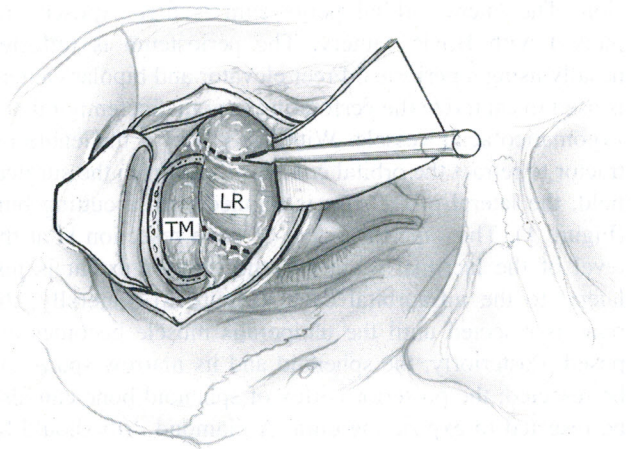

Figure 3 Once an adequate decompression is performed, the periosteum is incised in a parallel fashion above and below the lateral rectus muscle to allow the orbital contents to prolapse into the expanded bony space. LR, lateral rectus muscle; TM, temporalis muscle.

decompression alone, but it often presents as a mixed mechanism. Patients should be made aware that later soft-tissue eyelid surgery may be of value to better improve the esthetic appearance. An adequate fat decompression can provide a reduction of proptosis on average of 3-4 mm.

Patients with predominately enlarged EOMs would most benefit from a bone decompression. On the basis of published data, one should expect 2-3 mm of proptosis reduction with each wall of bone removed. In general, the number of bones to be decompressed is based on the desired decompressive effect. Usually, we perform an endoscopically mediated medial wall decompression in combination with a lateral wall decompression. If further decompression is required, the nasal floor of the orbit is also decompressed.

Technique

Lateral wall decompression

Lateral wall decompression may be approached through a lateral canthal incision or an extended eyelid crease inci-

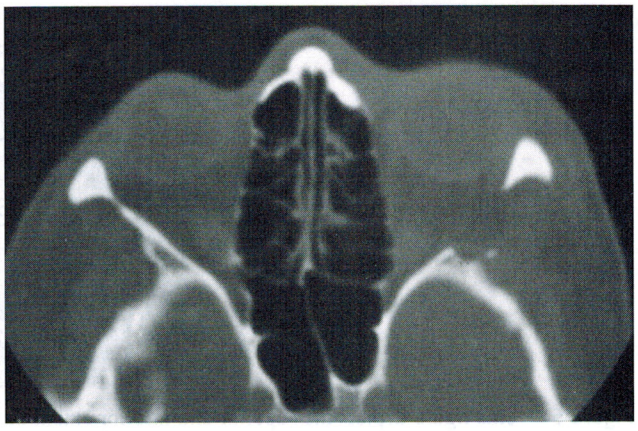

Figure 4 Postoperative axial CT of a patient who underwent a left lateral orbital wall decompression. (Color version of figure is available online.)

sion. The lateral orbital periosteum is first exposed and incised with Bovie cautery. The periosteum is reflected nasally using a periosteal Freer elevator and bipolar cautery is used to cauterize the perforating zygomaticotemporal and zygomaticofacial vessels. With the use of a malleable retractor to retract the orbital contents away from the surgical field, the lateral orbital rim is thinned with a cutting burr (Figure 1). The superior extent of bone resection is at the level of the lacrimal fossa and the inferior extent is just lateral to the infraorbital canal (Figure 2). Laterally, the bone is resected until the temporalis muscle becomes exposed. Posteriorly, the sphenoid and its marrow space can be resected; the posterior cortex of sphenoid bone can also be resected to expose the dura. A diamond drill should be used to avoid injury to the dura. Once an adequate decompression is performed, the periosteum is incised in a parallel fashion above and below the lateral rectus muscle to allow the orbital contents to prolapse into the expanded bony space (Figure 3). At this stage, the prolapsing orbital fat can be removed as well. Irrigation should be performed prior to closure of the wound to remove any residual bony fragments. The periosteum is then closed with a 4-0 absorbable suture. If divided, the lateral canthi are attached to the reapproximated periosteum with a 4-0 absorbable suture, and the commissure is then reformed using an absorbable sutures. The deep tissues are next approximated, and a running 6-0 nylon is used to close the skin. Figure 4 shows a postoperative CT scan of a left orbit that underwent a lateral orbital wall decompression.

Endoscopic decompression

Before the start of the procedure the nasal cavity is decongested with topical application of 4% cocaine on cotton pledgets. The nasal cavity on the side to be decompressed is inspected with a 0-degree rigid endoscope. A Freer elevator is used to gently medialize the middle turbinate and a cottonoid pledget is placed into the mid-

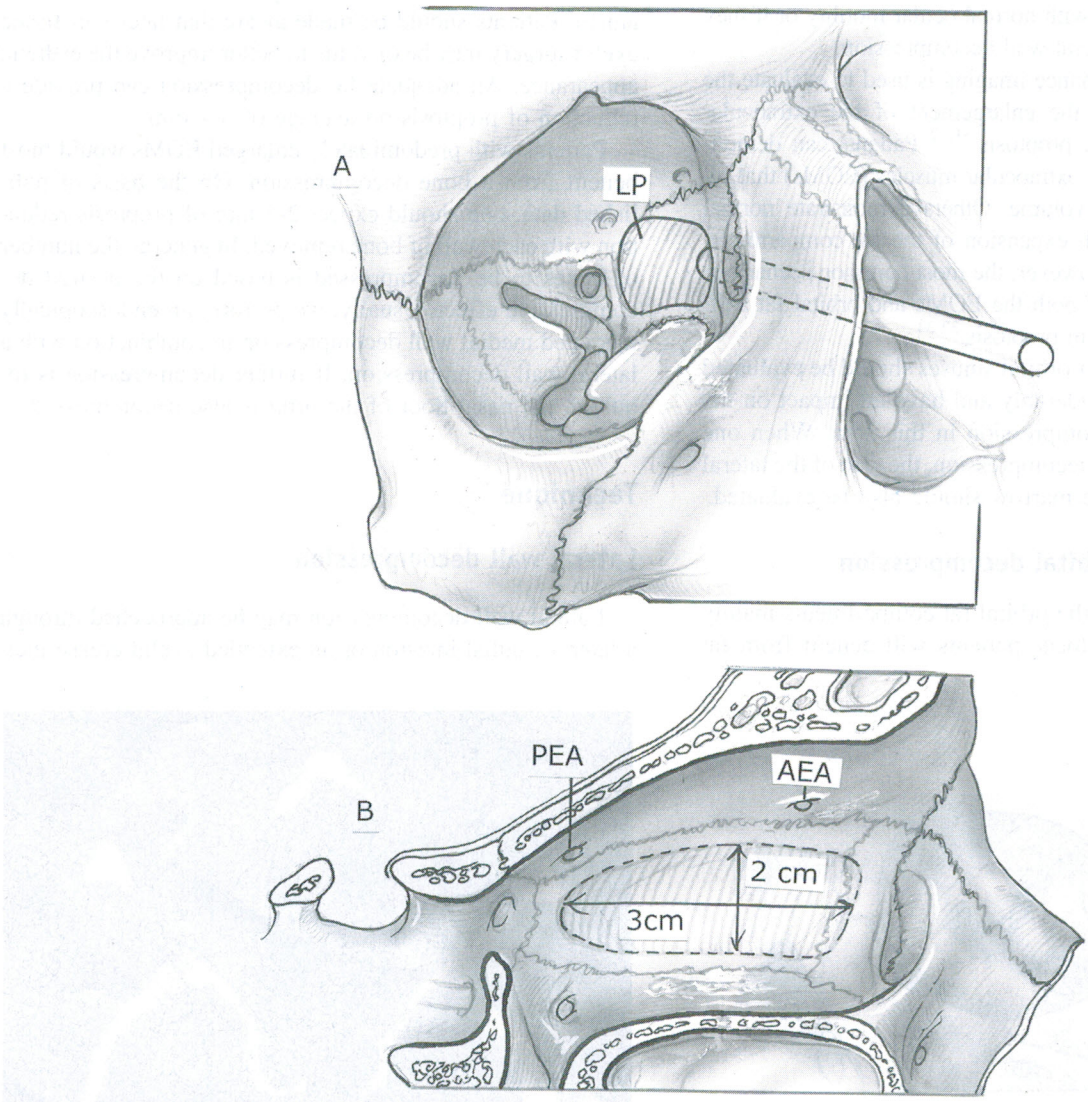

Figure 5 This figure depicts the superior and inferior extent of the medial wall decompression. A, The top figure is an anterior–posterior view of the orbit; B demonstrates a sagittal view of the medial wall. Intraoperative image-guidance is useful to determine the boundaries of the ethmoid sinus and confirm the identity of the lamina papyracea. AEA, anterior ethmoidal artery; LP, lamina papyracea; PEA, posterior ethmoidal artery.

dle meatus for additional decongestion. The vertical attachment of the middle turbinate and the face of the ethmoid bulla are injected locally with 1% lidocaine with 1:100,000 epinephrine. Intranasal injection of the sphenopalatine foramen provides additional hemostasis and analgesia. Topical hemostasis during the surgery is achieved using pledgets soaked with 20,000 thrombin and 1:1000 epinephrine.

Anterior ethmoidectomy is performed using either powered instrumentation or various bone punches; the preferred direction of bone removal is medial-to-lateral. Uncinectomy and infundibulotomy are not included in this technique to ensure that the ostiomeatal complex remains functional. It may be necessary to perform uncinectomy and infundibulotomy in certain cases where the room to accommodate the orbital contents in the middle meatus is not adequate. The ground lamella is encountered and opened to expose the posterior ethmoid cells. Intraoperative image-guidance is useful to determine the boundaries of the ethmoid sinus and confirm the identity of the lamina papyracea (Figure 5).

Once the lamina papyracea has been exposed the site to be decompressed is selected. This site should be posterior to the anterior ethmoidal artery, which consistently lies at the posterior border of the frontal sinus outflow tract, and anterior to the posterior ethmoidal artery, which emerges at the sphenoid rostrum. The bone of the lamina papyracea is entered by tapping it with an angled bone curette (Figure 6). The fractured bone is elevated away with the use of a Freer elevator and removed. Enlargement of the decompression

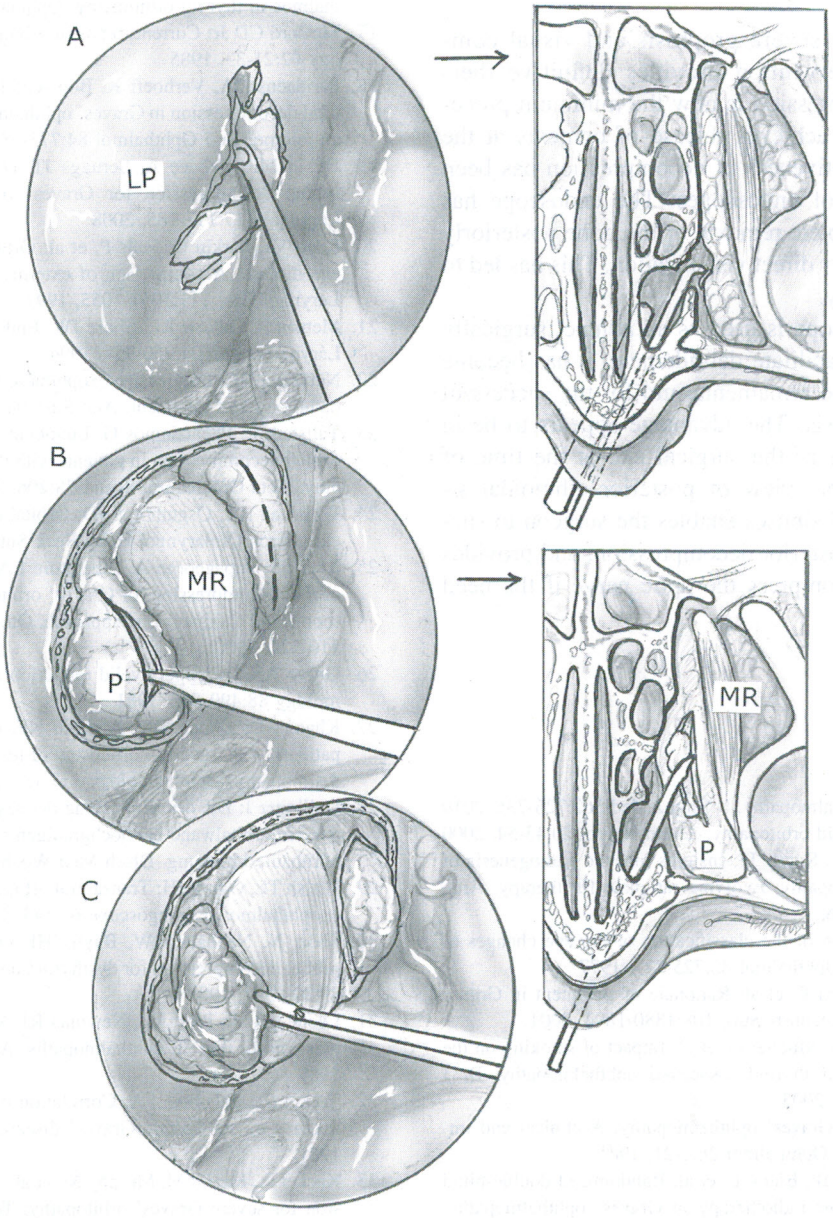

Figure 6 A, An angled bone curette is used to enter the bone of the lamina papyracea. The fractured bone is elevated away with a Freer elevator and removed. B, The exposed orbital periosteum is incised in a posterior-to-anterior direction using a sickle knife. The incision can be made above and below the medial rectus muscle to prevent direct injury to the muscle. C, Incised periorbital fascia and orbital fat allows for the herniation of the orbital contents into the ethmoidal sinus cavity. LP, lamina papyracea; MR, medial rectus; P, periosteum.

site by additional bone removal is accomplished with a Kerrison rongeur. The maxillary sinus natural ostium should be identified, which serves 2 purposes. First, the superior rim of the ostium represents the inferior limit of bone removal from the lamina papyracea. Second, it allows for preservation of a bony strut at the inferiomedial orbit to protect the inferior oblique muscle from prolapse or inadvertent injury. The exposed orbital periosteum is incised in a posterior-to-anterior direction using a sickle knife, which allows the orbital fat to herniate into the ethmoid sinus cavity (Figure 6). The procedure is concluded by application of a topical hemostatic agent such as FloSeal (Baxter, Deerfield, IL) and light nasal packing as needed.

Conclusions

In those patients with severe proptosis and visual compromise, surgical intervention provides definitive therapy. Two-wall decompression is now the minimum procedure that successfully achieves release of contents at the orbital apex. The effectiveness of decompression has been enhanced by the use of endoscopes. The endoscope has allowed for more complete removal of the bone posteriorly to the orbital apex under direct visualization. This has led to a decrease in morbidity.

Whether the endoscope is used as an adjunct surgically or postoperatively to facilitate debridement, it has become an important tool in the armamentarium for the successful management of proptosis. The advantage appears to be in the direct visualization of the surgical site at the time of surgery. The endoscopic view of posterior ethmoidal sinuses and the sphenoid sinuses enables the surgeon to successfully carry out a posterior decompression, and provides the opportunity to decompress the optic nerve if the need arises.

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