Section 10 – ORBITAL SURGERY

Chapter 95 – Orbital Decompression

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Decompression of the orbit may be considered for the treatment of space-occupying lesions causing compromise of function as a result of expansion of the orbital contents or a decrease in the rigid orbital confines. The volume of the orbit averages about 30 mL, and only anteriorly is it not bound by bone. By far the most common indication for decompression is Graves orbitopathy, but similar surgical techniques are applied to infectious, traumatic, neoplastic, and iatrogenic complications (e.g., orbital hematoma after sinus surgery) (Figs. 95-1 and 95-2).

The incidence of Graves disease in the general population is about 1% to 3%. Females are affected about five times as often as men, but older men who experience orbitopathy often have a more fulminant course. About a third of patients with Graves disease will have orbitopathy, often asymmetrical and usually mild or moderate. Approximately 85% of patients evaluated primarily for orbitopathy will be found to have a dysthyroid state. In adults, Graves disease is the most common cause of unilateral and bilateral proptosis. The first report of decompression for this condition occurred in 1911.[1]

The pathogenesis of Graves orbitopathy remains only partially understood. Serum levels of antibodies to the thyroid-stimulating hormone (TSH) receptor and collagen XIII seem to correlate with orbital disease activity.[2] TSH receptors are found on orbital fibroblasts, which may differentiate into adipocytes, and are more sensitive to TSH receptor antibodies than dermal fibrocytes are. The proptosis is due to an increase in retrobulbar glycosaminoglycan deposition and edema, as well as to a later increase in retrobulbar fat. Smoking is thought to affect this either by creating increased oxygen-reactive species or by causing local tissue hypoxia, both of which lead to increased chemokines recruiting T cells from the bloodstream. TH_2 cells are the predominant type involved in the orbital disease.[3]

The acute phase of the disease, which consists of orbital congestion, inflammatory cell infiltrate, and forward movement of the globe, generally lasts between 6 and 24 months. After this phase, the disease is characterized pathologically by increased fibrosis of the tissues. A second acute phase may occasionally affect the patient later in life. Asymmetrical involvement of the orbits is frequent.

The only external factor proved to affect Graves orbitopathy is cigarette smoking. Current smoking outweighs previous smoking as a predictor of disease severity. Patients in whom orbital disease develops are four times as likely to smoke, and discontinuation of smoking appears to lessen the severity, as well as protect patients from the development of further symptoms.[4] Pretibial myxedema, acropachy, and female sex are also associated with more severe orbital disease.[5]

Symptoms of Graves orbitopathy include eyelid retraction, proptosis, diplopia, dryness of the eyes, a sensation of pressure behind one or both eyes, and swelling of the lids and conjunctiva (chemosis). Signs consists of eyelid retraction more than 1 mm from the limbus, eyelid lag on downward gaze (failure of the upper eyelid to descend properly when the patient looks down), unilateral or bilateral proptosis, restriction of extraocular movements (particularly upward gaze and abduction), hyperemia over the insertions of the horizontal rectus muscles, and corneal staining with fluorescein observed with a cobalt filter (Figs. 95-3 and 95-4).

The course of the orbital disease and the course of any associated thyroid disease in Graves disease are independent. Euthyroid patients may have severe orbitopathy causing optic neuropathy or corneal perforation, whereas severely hyperthyroid patients may have no signs or symptoms. Treating the thyroid abnormalities is essential to the patient's general health, but it is unlikely to help the eye disease. Exceptions are removal of a large goiter, which may lessen the orbital symptoms, and treatment with radioactive iodine, which sometimes acutely worsens the condition of the orbits (ameliorated by concurrent treatment with oral steroid).[6]

Orbital decompression is sometimes undertaken for other causes of proptosis: infection with abscess, tumor, trauma to the optic nerve, or hematoma (such as might occur after endoscopic sinus surgery). Abscesses located along the lamina papyracea in the orbit ormedially along the roof or floor are most amenable to endoscopic drainage. In the setting of head trauma and a new optic neuropathy, traumatic optic neuropathy is assumed. If the optic canal is fractured, the fragments may be removed endoscopically, but authors disagree about the benefits of

this procedure.[7–9] Occasionally, an orbital apex tumor causing compressive optic neuropathy may be successfully treated by decompression when resection of the tumor is inadvisable.[10]

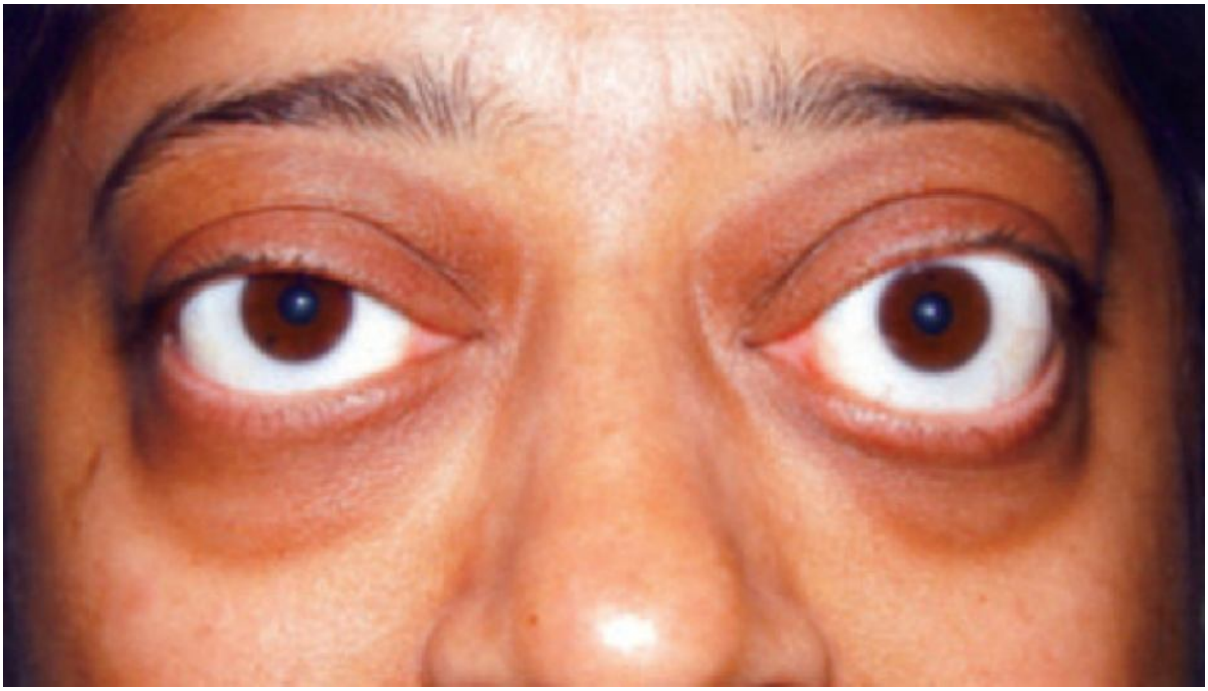

Figure 95-1 Patient with Graves orbitopathy preoperatively.

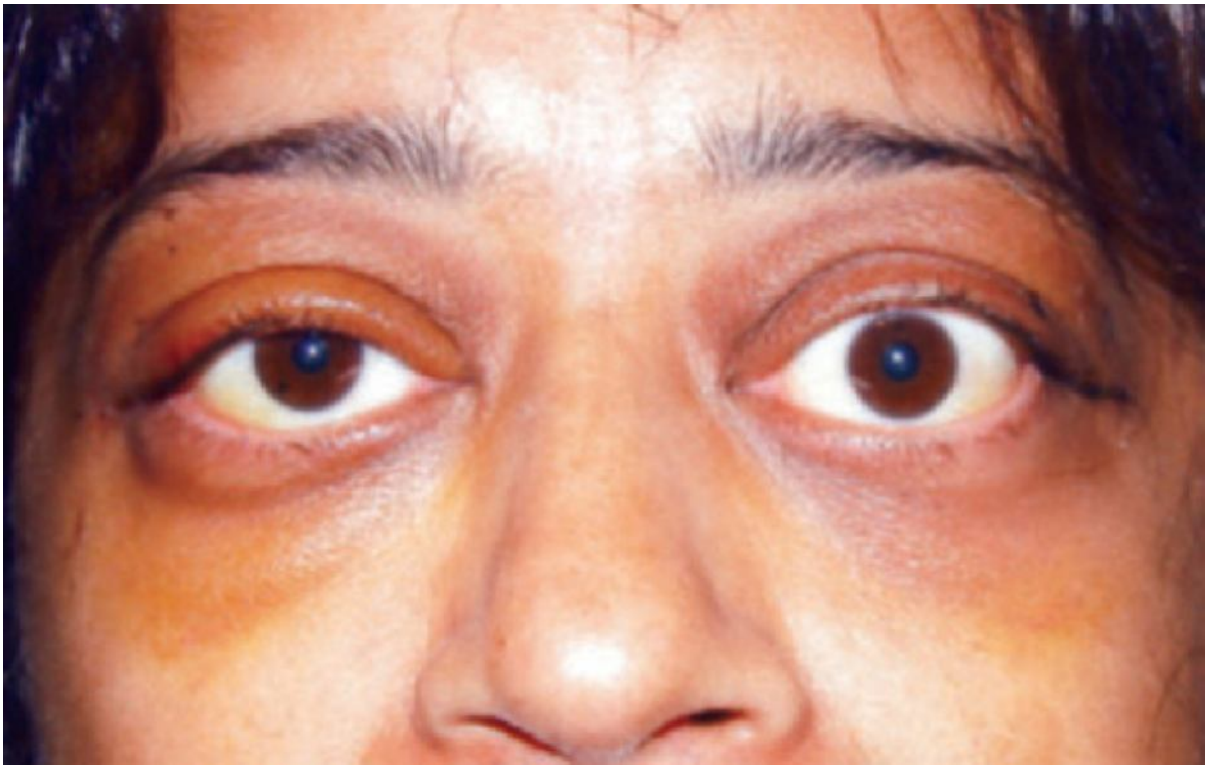

Figure 95-2 Patient with Graves orbitopathy 6 days after balanced decompression of both eyes.

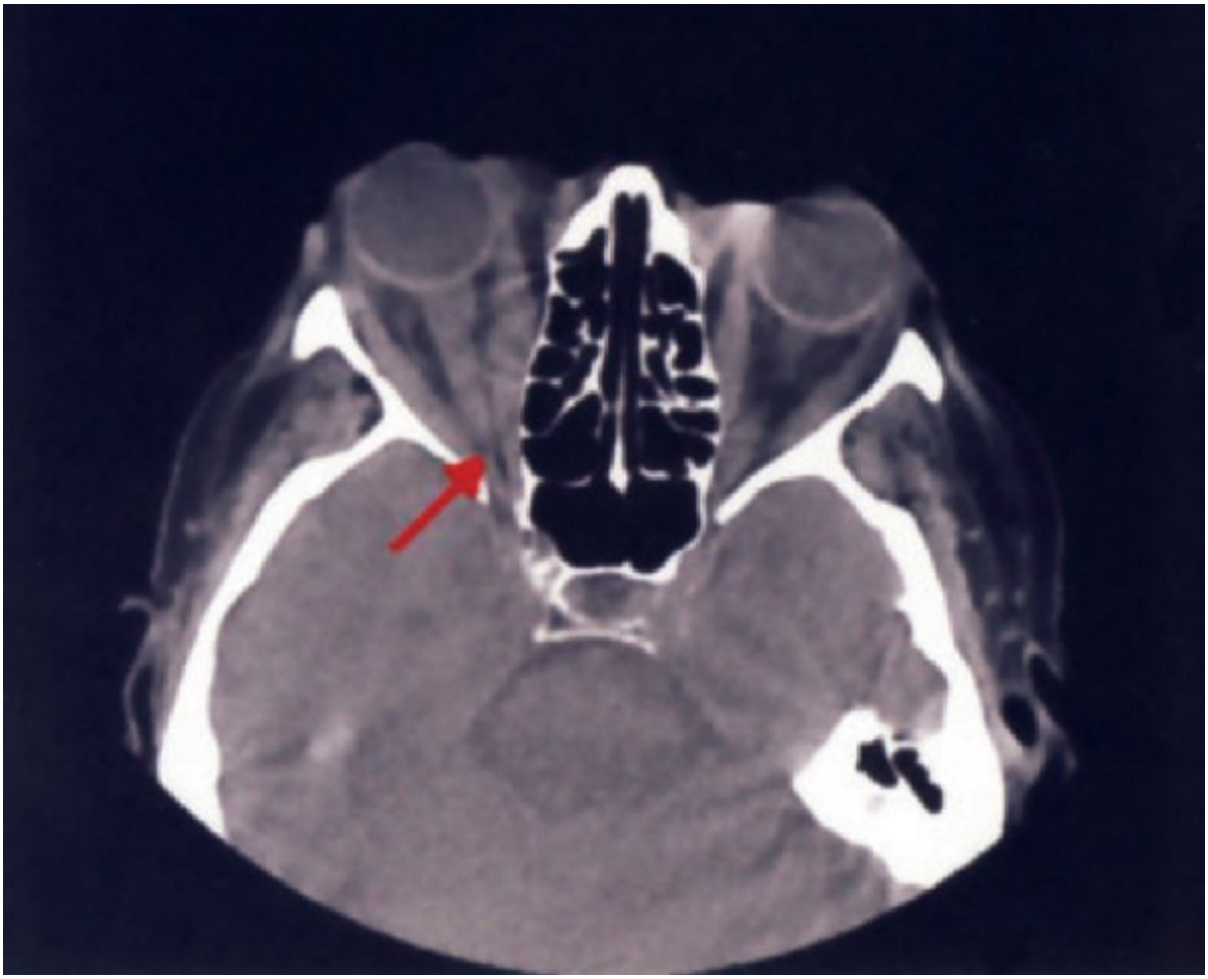

Figure 95-3 Patient with Graves orbitopathy, optic neuropathy in the right eye, visual acuity of 20/50 in the right eye, and Ishihara color vision of 1.5 of 11 plates. Note the apical crowding.

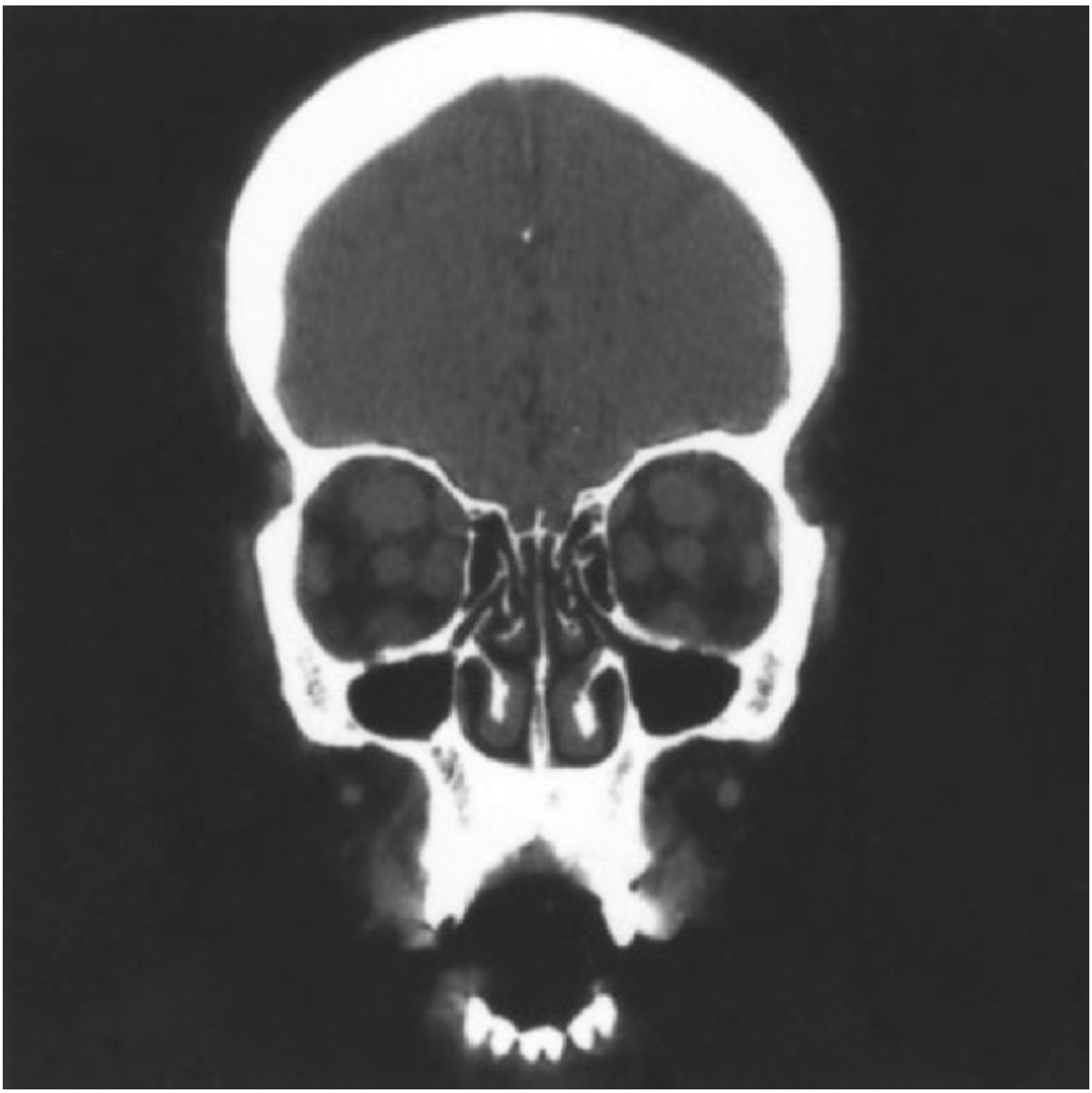

Figure 95-4 Coronal view of the patient from Figure 95-3.

ANATOMY

The orbit is bounded by four walls: superior, medial, inferior, and lateral. The medial wall of the orbit extends from the ethmoidal roof superiorly to its junction with the inferior wall inferiorly. Posteriorly, it is continuous with the medial surface of the optic canal. Anteriorly, it is bounded by the thicker bone of the lacrimal fossa and ascending process of the maxilla. The inferior wall of the orbit is thicker than the medial wall and is divided by the infraorbital neurovascular bundle. The bulge of the canal is often visible, and the vessels can be visualized through the thinned bone. The lateral wall of the orbit is bounded laterally by the temporalis muscle (Figs. 95-5 and 95-6).

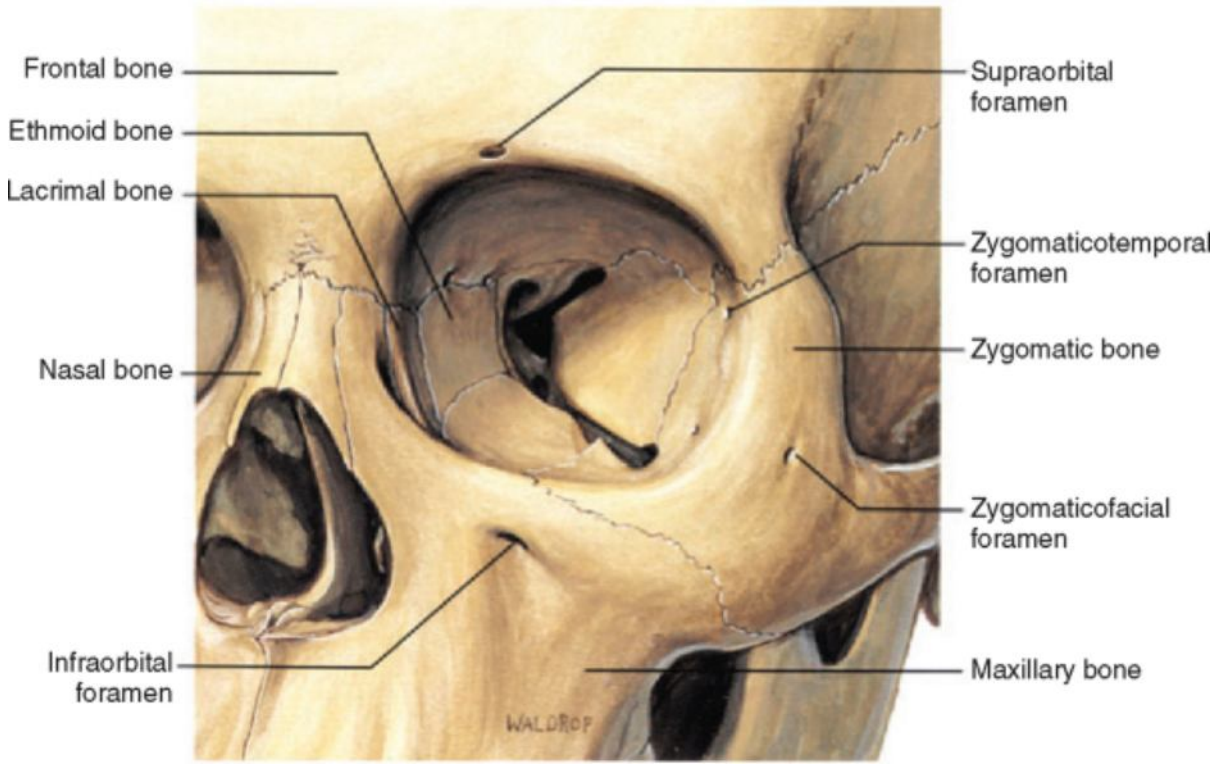

Figure 95-5 Orbital bones, frontal view.
(From Dutton JJ: Atlas of Clinical and Surgical Orbital Anatomy. Philadelphia, WB Saunders, 1994.)

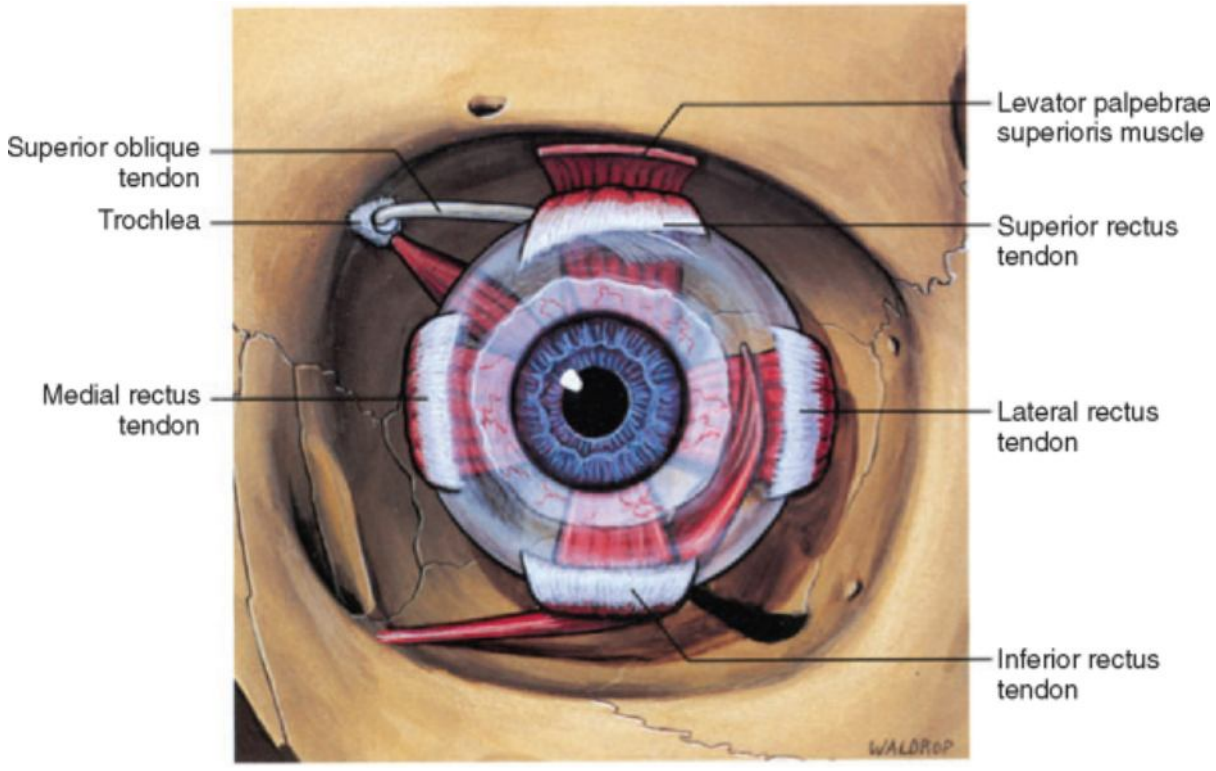

Figure 95-6 Extraocular muscles, frontal composite view.
(From Dutton JJ: Atlas of Clinical and Surgical Orbital Anatomy. Philadelphia, WB Saunders, 1994.)

PATIENT SELECTION

By the time that a patient with Graves disease has reached the otolaryngologist for consultation, the patient will most likely have been evaluated by an ophthalmologist. A frank conversation between the two physicians will

establish the urgency with which surgery should proceed: a patient with severe exposure keratitis may require decompression within the week, whereas another patient with inactive disease and strictly cosmetic concerns may be scheduled when convenient for surgeon and patient. If the patient has not undergone a complete ophthalmologic examination, including evaluation of optic nerve function, extraocular movement, and ocular surface, one must be conducted before planning surgery. Documentation of preoperative visual function is critical.

Occasionally, other disease processes will necessitate surgical decompression of the orbits, including infection, tumor, hemorrhage, and trauma. Usually, these cases will develop more rapidly than dysthyroid orbitopathy, and the decision regarding timing and the approach for surgery will be more urgent. Surgical approaches are the same; however, each of these cases will have its attendant challenges. In all these situations, the anatomy will be obscured and great care must be taken to avoid harm to vital structures that the surgeon seeks to protect.

In the case of an orbital abscess causing proptosis, particularly in children younger than 9 years, it is often prudent to wait 24 to 48 hours for a response to intravenous antibiotics. In adults, particularly when the abscess is associated with concurrent sinus disease, the decision to proceed with endoscopic drainage of the cavity (when located medially) or external drainage (when located superiorly or laterally) should be made earlier (Fig. 95-7). In the case of optic neuropathy, surgery should proceed as soon as safely possible.

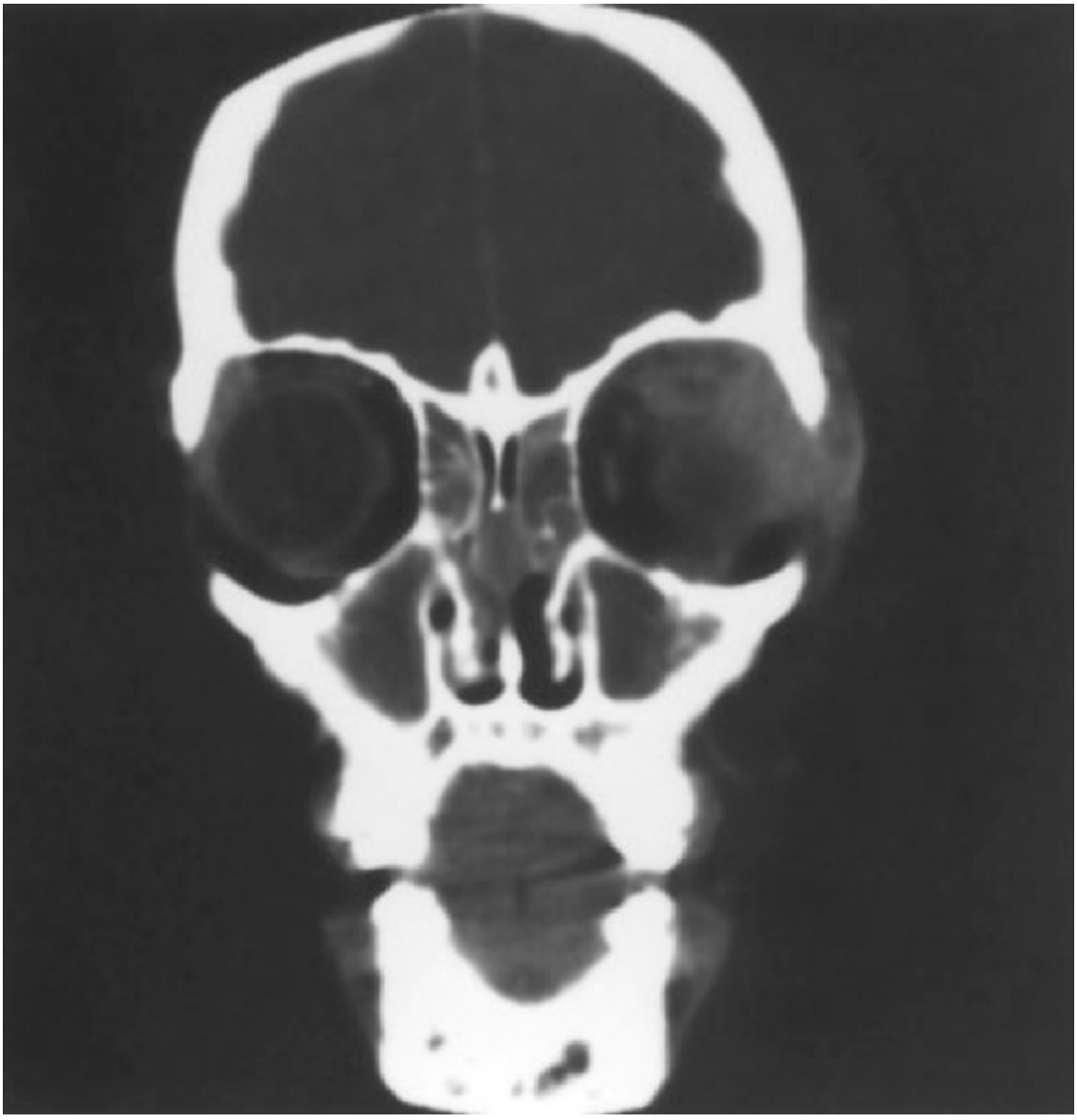

Figure 95-7 Twelve-year-old patient with a superior subperiosteal orbital abscess that improved after endoscopic ethmoidectomy and percutaneous drainage of the abscess.

Orbital hematoma, whether traumatic, iatrogenic, or rarely, spontaneous, should also be approached with urgency. If optic neuropathy is present and lateral canthotomy/cantholysis has failed, decompression of the orbit is necessary. Endoscopic decompression of the medial wall is particularly helpful because a fairly posterior

decompression of the optic nerve is possible.

Frequently in cases of trauma with fracture of the orbit, the orbit will have decompressed itself. In some instances, however, bone fragments may impinge on orbital structures and require urgent removal or reduction. This occurs again in the presence of optic neuropathy or with an intractable increase in intraocular pressure (Fig. 95-8).

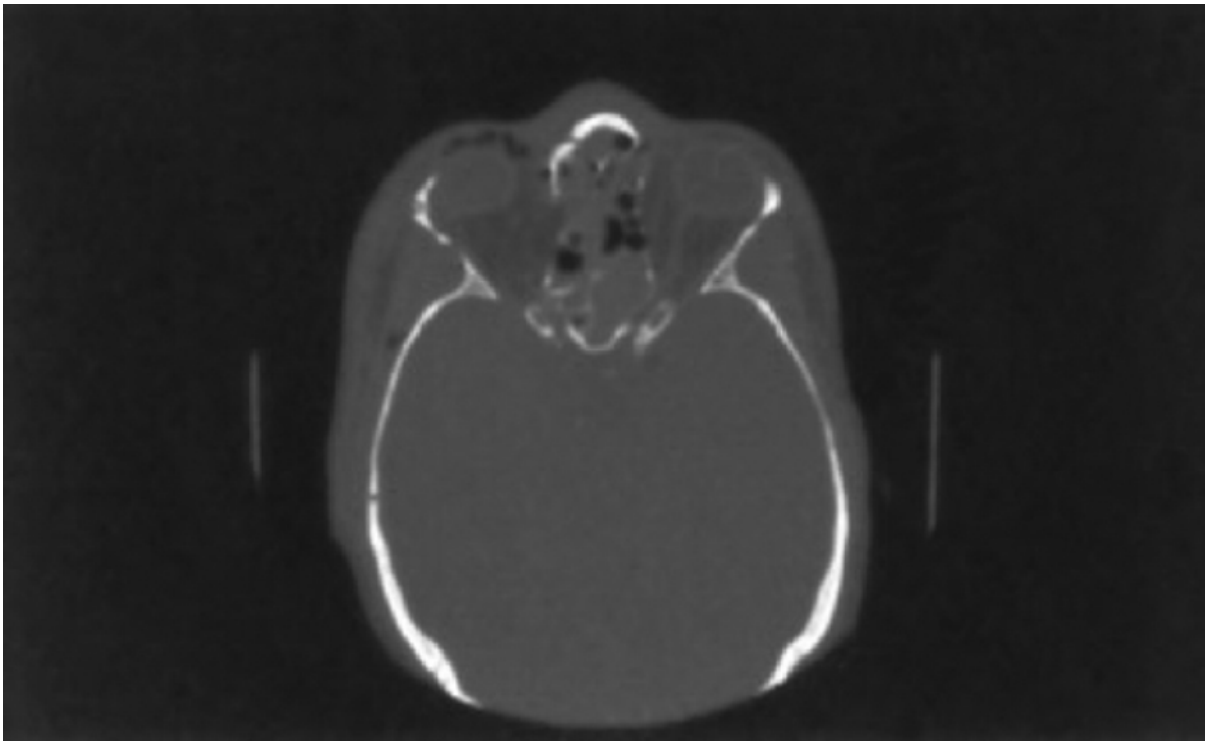

Figure 95-8 Patient with bone fragments in the optic canal and right optic neuropathy after closed head injury.

In cases of Graves orbitopathy, communication between the ophthalmologist, otolaryngologist, and patient will determine how best to serve the patient. Patients may be divided into two groups: those who require urgent surgery during an active phase of disease and those who seek cosmetic and functional improvement later in the course of the disease. Whenever possible, it is best to defer patients from the first group into the second. If the patient has active orbitopathy characterized by inflammatory signs and evolution and can be treated by nonsurgical means, this should be pursued first. Temporization may often be accomplished with the use of oral (1 mg/kg/day) or intravenous pulse prednisone or by radiation treatment of the orbits (up to 2000 cGy, below the threshold for causing optic neuropathy). Retinopathy rarely occurs late after radiation treatment, but nearly always in diabetic patients, and cataract formation appears only as frequently as it does when patients are treated with steroids.[11] Surgery is both more technically challenging and more likely to be complicated by excessive bleeding in the setting of active inflammation. However, some patients will require immediate surgery, particularly when an impending corneal perforation or severe optic neuropathy is present.

Patients who have passed the active stage and entered the cicatricial phase are good candidates for decompression, either for improvement in appearance or for maximization of residual function. Endoscopic decompression of the medial wall combined with removal of the medial floor and lateral wall offers the most significant decrease in proptosis, up to 6 to 10 mm. The degree of improvement depends partly on the increase in volume, but also on the rigidity of the tissues (periorbital tissues that are more densely fibrotic relax poorly into the newly created space). Decompression of the orbital roof is seldom performed. Unilateral or bilateral orbital decompression may be performed at the same time.

PREOPERATIVE EVALUATION

All patients undergoing orbital decompression require a comprehensive preoperative ophthalmologic examination. A computed tomography (CT) scan of the orbit is helpful to identify any anatomic abnormalities before surgery.

It is helpful for the otolaryngologist to see the patient preoperatively to obtain a history of sinonasal problems and evaluate the anatomy of the nasal cavity. The CT scan should be reviewed to assess for occult disease and anatomic variations that may pose problems: low cribriform plate, Onodi cells, Haller cells, or nonpneumatization of sinuses. Active sinusitis should be treated and completely resolved before proceeding with elective orbital decompression. Severe deviations of the nasal septum can interfere with surgical access and may need to be

corrected at the same time. Coexistent sinus problems such as chronic sinus obstruction or nasal polyposis can also be addressed. Informed consent includes the usual discussion of risks from endoscopic sinus surgery: visual loss, diplopia, cerebrospinal fluid (CSF) leak, hemorrhage, sinusitis, loss of olfaction, and intranasal synechiae.

PREOPERATIVE PLANNING

Preoperative counseling is particularly important so that the patient understands that this procedure, when performed for Graves disease, is likely to be the first in a series of surgeries. New-onset or worsened postoperative diplopia will occur in about a third of patients undergoing decompression of three walls,[12] but it has been observed in retrospective series that a “balanced decompression” consisting of only the medial and lateral walls is associated with only about a 10% incidence of new-onset diplopia.[13] Strabismus surgery may be performed for unresolved diplopia, and eyelid surgery (either correction of retraction or blepharoplasty) may be required after that.

SURGICAL APPROACHES

Over the years a number of different operations have been described for decompression of the orbit, which may include anywhere from one to four walls. The amount of decompression that can be achieved increases with the number of walls removed. Removal of the superior wall is not usually performed because of its minimal additional benefit and the added morbidity. The medial wall may be decompressed from an external ethmoidectomy approach, a transantral approach, or a transnasal approach. The inferior wall may be decompressed from a transconjunctival approach, a transantral approach, or a transnasal approach. With the advent of endoscopic sinus surgery techniques, most surgeons prefer a transnasal endoscopic approach for decompression of the medial and inferior orbital walls. Removal of bone lateral to the infraorbital nerve provides minimal additional decompression and risks injury to the infraorbital nerve. The lateral orbital wall is decompressed from an external approach.

Medial Wall

The nose is decongested with pledgets soaked in 0.5% oxymetazoline. If a septoplasty is necessary for access, it is performed first. The inferior aspect of the middle turbinate is resected to provide greater access and room for the orbital tissues to expand. The uncinate process is removed and the antrostomy is maximally enlarged posteriorly and superiorly. The bulla ethmoidalis is opened for removal of ethmoid air cells in an anterior-to-posterior direction. The sphenoid sinus is opened and the sphenoidotomy is maximally enlarged with Kerrison rongeurs. Residual septations are then removed in a posterior-to-anterior direction along the skull base. The nasofrontal recess is exposed, but further dissection of the frontal sinus is unnecessary.

The medial wall of the orbit is palpated and the thin bone of the lamina papyracea is fractured anteriorly. Bone fragments are carefully elevated from the underlying periorbita with a Cottle elevator or ball-tipped probe. Removal of bone continues posteriorly to the anterior face of the sphenoid sinus. Superiorly, bone is removed to within 2 mm of the skull base while being careful to not fracture the skull base. Removal of bone continues inferiorly to the junction of the medial and inferior orbital walls. Examination of the maxillary sinus with an angled endoscope usually reveals the neurovascular bundle of the infraorbital nerve running across the roof of the maxilla. The floor of the orbit is then removed with angled Kerrison rongeurs to the medial aspect of the neurovascular bundle. Removal of additional bone does not contribute to decompression and risks injury to the nerve.

If the decompression is being done for proptosis, decompression of the orbital apex beyond the anterior wall of the sphenoid sinus is not necessary.[14] If the patient is undergoing decompression for visual loss, additional bone is removed posteriorly. Landmarks within the sphenoid sinus are first identified: the carotid canal, optic canal, and optic-carotid recess.[15] The bone is carefully thinned with a diamond drill over the optic canal until it can be fractured and elevated with a small elevator. Drilling of bone should be along the axis of the optic canal to avoid injury to the carotid artery.

A sickle blade is then used to incise the periorbita of the medial orbit, starting posteriorly and superiorly and proceeding anteriorly. Multiple parallel incisions are made from the ethmoid roof to the floor of the orbit. The medial rectus muscle is often infiltrated and enlarged and is susceptible to injury if the incisions are too deep. Intervening strands of periorbita are transected to allow complete herniation of the orbital contents. Gentle external pressure on the orbit with the hand facilitates herniation of orbital fat into the ethmoid defect (see Video 95-1).

Silastic nasal splints are placed to prevent intranasal synechiae. Nasal packing is not used.

Lateral Wall

The lateral wall of the orbit is approached via a lateral canthotomy incision. The incision created is approximately 2 cm in length and extends posteriorly from the lateral canthal angle in a crow's foot crease. Stevens or similar scissors are used to cut the lateral canthal ligament and deepen the incision to bone. Bleeding is controlled with electrocautery. The periosteum is incised vertically along the apex of the rim with a scalpel or needle-tipped electrocautery. A Cottle elevator is then used to clear the periosteum from the lateral orbital wall while keeping the periorbita intact as much as possible. The periosteum must be elevated from both the interior and exterior surfaces of the lateral wall of the orbit. The bone should be identified as far superiorly as the fossa of the lacrimal sac and inferiorly to just above the level of the zygomatic arch. Careful elevation along the exterior surface proceeds in a posterior direction and then turns medially in the temporal fossa.

Once the periosteum has been elevated from the bone, a wide ribbon retractor is inserted between the periorbita and bone by an assistant. A Desmarres vein retractor is placed over the cut surface of the skin. An oscillating bone saw is used to cut through the lateral wall, perpendicular to the bone. Frequent pauses to assess position and adequacy of protection of the orbital contents are essential. It is only necessary to make an osteotomy posteriorly through the vertical zygomaticofrontal buttress. A parallel osteotomy is made inferiorly above the zygomatic arch, again through the triangular thick bone of the rim.

A rongeur is now used to grasp the vertical strut, which is rocked medially and laterally until the bone breaks free posteriorly. It will be necessary to elevate the remainder of the temporalis muscle from the posterolateral surface of the bone with electrocautery. Bone wax is applied as needed, and any remaining small fragments of bone are removed with the same instrument. The sharp surfaces of the remaining zygomatic and frontal bones are smoothed with a burr or rongeur.

A short-bladed knife, such as a sickle knife, is now applied to the periorbita in a very superficial manner, directed posteriorly to anteriorly, to slit the fibrous septa and allow the orbital contents to prolapse into the temporal fossa. Care is taken to place no traction on the orbital fat and to remain superficial so that the lacrimal gland and lateral rectus muscle are not damaged. Bipolar cautery is used to obtain hemostasis.

Careful closure of the wound is accomplished by precise realignment of the upper and lower gray lines of the eyelid with a single horizontal stitch of 6-0 absorbable material, such as polyglactin, buried laterally. The deep tissues are reapproximated with inverted 6-0 suture and the skin closed carefully with running 7-0 nylon or interrupted chromic gut suture. Before complete closure, a longitudinal strip of 0.25-inch Penrosedrain is placed into the wound to emerge between suture bites. A patch is placed loosely over the eye to absorb drainage. The patient is admitted for observation overnight, both for pain control and to monitor for any hemorrhage (Figs. 95-9 and 95-10).

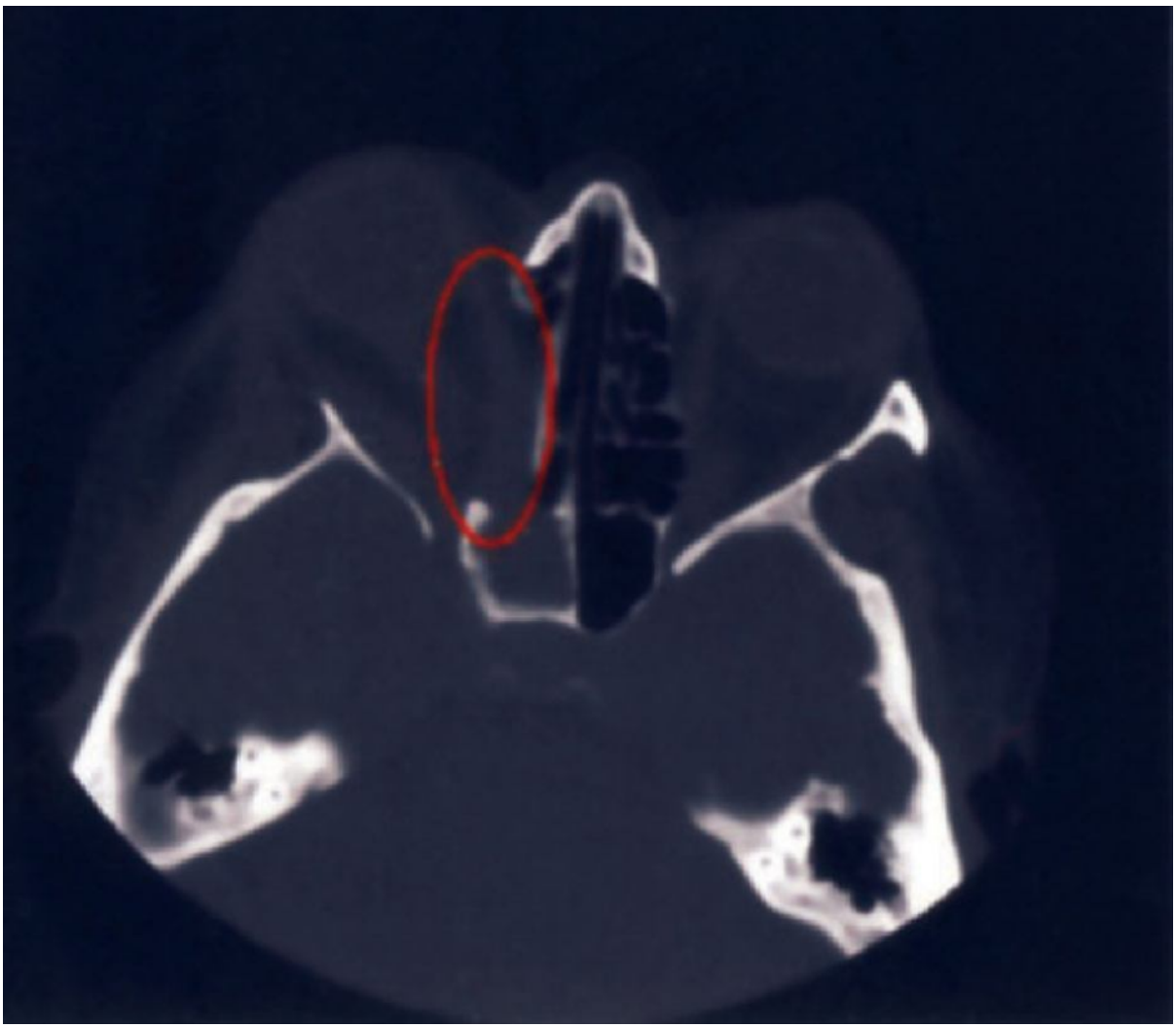

Figure 95-9 Postoperative axial view of the patient from Figure 95-3 with right optic neuropathy secondary to Graves orbitopathy. One week after balanced decompression, visual acuity in the right eye was 20/25 and Ishihara color vision was normal (17/18 plates). Note the herniation of the enlarged medial rectus into the bony defect.

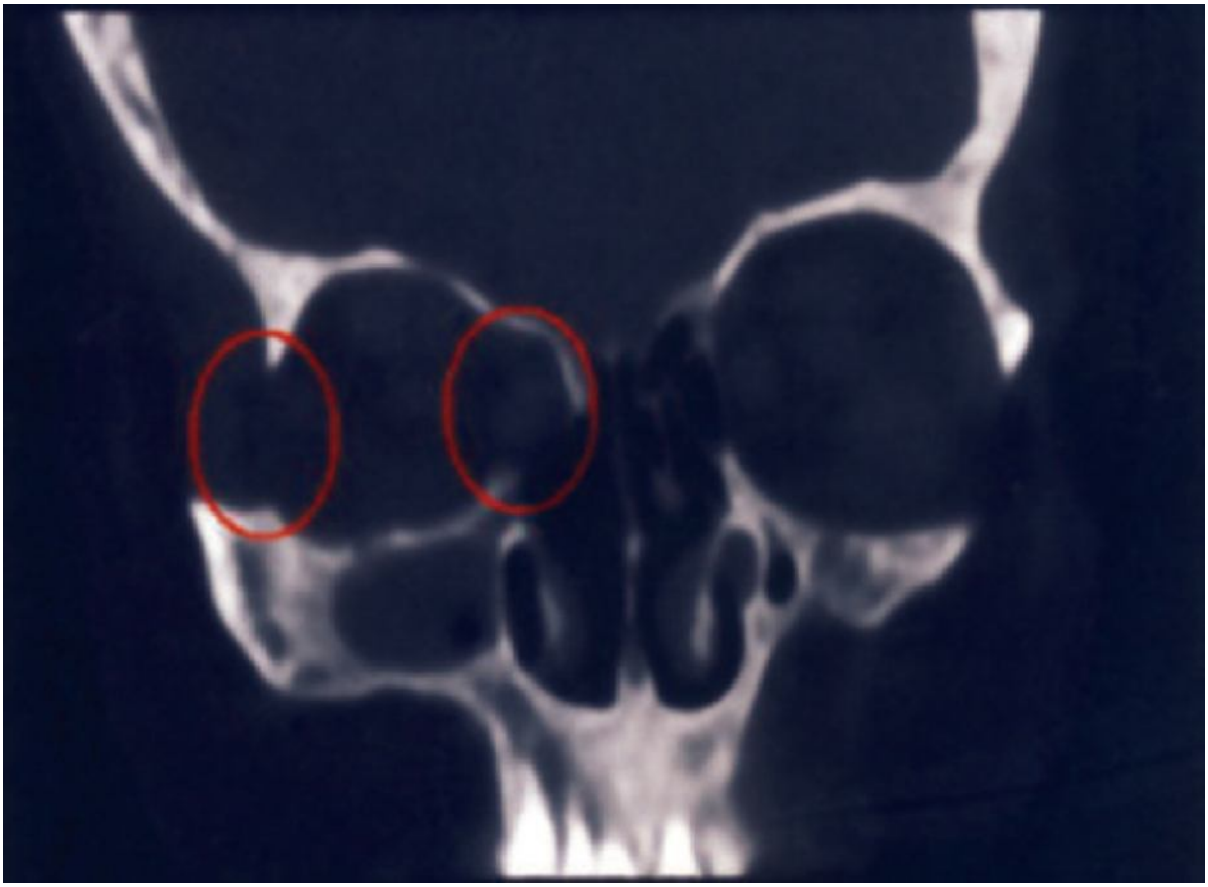

Figure 95-10 Postoperative coronal view of the patient from Figure 95-3. Note the bony defects with herniation of the orbital contents.

POSTOPERATIVE CARE

Patients are administered a steroid pulse intravenously while undergoing surgery and observation (8 to 12 mg of dexamethasone during the procedure, and 4 to 8 mg every 8 hours thereafter while in the hospital). A rapid taper of oral steroid medication is adequate for most patients; those decompressed in an acute setting require a much more aggressive regimen and longer taper.

Patients are instructed to not blow their nose for 1 month after the procedure, and a steroid/antibiotic ointment is applied to the sutures twice a day for a week. Nonabsorbable sutures are removed in 5 to 7 days. Intranasal splints are removed at 1 week, and a saline spray is used as necessary to minimize crusting. Mucosalization of the orbital tissues is usually complete by 2 months. Patients are counseled to expect some initial diplopia and that the final appearance is often not reached for at least 3 months.

Patients are observed for at least 6 months after surgery before consideration of extraocular muscle or eyelid surgery.

COMPLICATIONS

The risks associated with endoscopic medial and inferior orbital decompression are similar to those after any endoscopic sinus surgery. Excessive or aggressive removal of bone superiorly can fracture the skull base and cause a CSF leak. Injury to the ethmoidal arteries can occur and result in a retro-orbital hematoma. Posteriorly, the optic nerve and carotid artery are at risk for injury. The medial rectus muscle is often enlarged and is susceptible to injury when the periorbita is incised. Postoperatively, the exposed orbital fat is at greater risk for infection, but this is a rare occurrence. Positive pressure ventilation after extubation or nose blowing by the patient postoperatively can force air into the orbital tissues and result in subcutaneous emphysema. Injury to the infraorbital nerve can lead to loss of sensation of the midface.

The lateral rectus muscle is susceptible to injury during decompression of the lateral orbital wall. Ecchymosis of the periorbital tissues is common but may be associated with a hematoma.

As discussed previously, unbalanced decompression of the orbit can result in significant diplopia. The diplopia is often transient but may require corrective surgery if it persists beyond 6 months.

SUMMARY

Decompression of the orbit is considered for the treatment of Graves ophthalmopathy and other mass lesions of the orbit (abscess, hematoma, and neoplasm). Maximal decompression can be achieved with endoscopic decompression of the medial and inferior orbital walls and external decompression of the lateral wall. Balanced decompression minimizes the risk for postoperative diplopia.

PEARLS

- Balanced decompression (medial and lateral orbital walls) minimizes the likelihood of postoperative diplopia.
- Careful realignment of the lateral commissure of the eyelids is necessary for an optimal cosmetic result.
- In patients with compressive optic neuropathy, it is vital to perform posterior medial wall decompression to relieve pressure on the optic nerve in the orbital apex.
- When decompressing the optic canal, the optic nerve sheath should be opened cautiously in the superonasal quadrant to minimize the risk of injury to the ophthalmic artery.
- Any removal of intraconal fat must be done with a minimum of traction and from the inferotemporal quadrant (between the lateral and inferior rectus muscles) to decrease the risk of retrobulbar hematoma and injury to orbital structures.

PITFALLS

- The medial and lateral rectus muscles are often enlarged and just deep to the periorbita and may be injured by aggressive sharp dissection.
- Bony decompression without adequate division of the periorbital septa is ineffective.
- Aggressive endoscopic removal of bone superiorly may be associated with fractures of the skull base and CSF leak.
- Unrecognized injury to the ethmoid arteries may cause retro-orbital hematoma, a risk that is increased when the patient coughs or struggles when emerging from anesthesia.
- Monopolar cautery should be avoided near the apex of the orbit.