Introduction

Exophthalmos is a condition of altered thyroid metabolism that causes protein depositions within the extraocular muscles, increasing their bulk as much as tenfold. Graves’ disease is now recognized as a multisystem disorder characterized by one or more of the following: 1) hyperthyroidism associated with diffuse hyperplasia of the thyroid gland, 2) infiltrative ophthalmopathy leading to exophthalmos, and 3) infiltrative dermopathy with localized pretibial myxedema. Therapy is still primarily directed at the manifestations of the disease in a palliative fashion rather than at preventing the underlying destructive autoimmune process. We will focus on the evaluation and management of the ophthalmic manifestations of Graves’ disease.

Pathophysiology

The pathogenesis of endocrine orbitopathy is not well understood. Although thyroid dysfunction is an important part of Graves’ disease, it alone is not the cause of the orbital symptoms. Many patients with exophthalmos from Graves’ orbitopathy are euthyroid at the time that eye symptoms appear, although a thyroid releasing hormone stimulation test or T3 suppression test will usually reveal dysthyroidism in most of these cases. Treatment of the thyroid disease, either with blocking agents or by ablation with radioactive iodine or surgery, does not prevent later development of orbital manifestations or ameliorate eye symptoms already present. The prevailing theory is that Graves’ disease and its associated ophthalmopathy is an autoimmune disease. Antibodies against thyroglobulin, and T-cell lymphocytes sensitized against orbital tissues have been demonstrated in this patient population. Current theory involves autoreactive T cells, which arise through either an escape from clonal deletion, failure of suppressor T cells activity, or through molecular mimicry to become reactive to TSH receptors. As the autoimmune process amplifies, T lymphocytes are activated and humoral immunity produces antibodies to the TSH receptor that are stimulatory, resulting in hyperthyroidism.
The extraocular muscles are the site of the most clinically evident changes in patients with Graves’ ophthalmopathy. The muscles are enlarged and there is an associated intense proliferation of perimysial fibroblasts and dense lymphocytic infiltration. The retrobulbar fibroblast has been found to play a key role in the development of Graves’ ophthalmopathy. They secrete a range of glycosaminoglycans, the deposition of which is a hallmark of Graves’ ophthalmopathy and causes interstitial edema as a result of its intensely hydrophilic nature. These cells also can produce major histocompatibility complex class II molecules, heat shock proteins, and lymphocyte adhesion molecules, which allow them to act as target and effector cells in the ongoing immune process in those with Graves’ ophthalmopathy. In addition, autoantibodies against fibroblast antigens have been found in a majority of patients with Graves’ ophthalmopathy. The fibroblast antigen may be similar to all or part of the TSH receptor and therefore represents a shared thyroid-eye antigen. Such antigenic similarity would explain the immune crossreactivity between these two sites. Lymphocytes are also active in the ongoing immune process of Graves’ ophthalmopathy. Orbital lymphocyte infiltrates have been found to be primarily T cells. Cytokines released by T cells have been shown to induce fibroblast proliferation and collagen and glycosaminoglycan deposition. Thus, the T cell-fibroblast interaction may be responsible for the clinical manifestations of Graves’ ophthalmopathy.

**Ophthalmopathy Classification**

Because of the fixed volume of approximately 30 ml of the orbit, expansion of the cross-sectional diameter of the extraocular muscles is manifested as an outward movement of the globe, with anterior protrusion of the mobile orbital fat. For an increase in soft tissue volume of 5 cc the globe becomes approximately 4-5 mm proptotic. In addition to thickening of the muscles, fat herniation, and proptosis, there is also upper and lower eyelid retraction and divergence of gaze. This retraction is due to the fibrosis of the lid retractor muscles and leads to a widened palpebral fissure which accentuates the proptosis. Except in acute cases of exophthalmos, the eyelids can close sufficiently to protect the cornea. Although more than 50% of patients with Graves’ disease have eye symptoms, only about 5% are severe enough to warrant intervention. Other symptoms of importance are diplopia from extraocular muscle involvement, exposure keratopathy, glaucoma, and severe congestive changes. Lid retraction is the orbital symptom that is most likely to regress completely without treatment. Proptosis usually peaks 4 to 13 months after the onset of the disease, and regression in the range of 3 to 7 mm occurs in half of the patients over the ensuing 1 to 3 years.

Eye involvement in those with Graves’ disease is bilateral in the majority of patients although 5% to 14% of patients will have unilateral disease depending on the method of detection. Major asymmetry of eye involvement is common and Graves’ ophthalmopathy remains the most common etiology of unilateral proptosis in adults. A clinical classification system for eye involvement by Graves’ disease was adopted by the American Thyroid Association (ATA). This classification is strictly clinical and has been helpful for reporting purposes. The disease does not always progress systematically and may skip over some of the classes. It also does not consider the disease activity which is
important in making patient treatment decisions. There have been other classification systems proposed, but the ATA system is still used for educational purposes and clinical evaluation. ATA class I disease is the mildest form of the disease and involves lid lag and the appearance of a “stare”. This is thought to occur initially from an increase in sympathetic sensitivity to catecholamines. As the disease progresses there is lymphocytic inflammatory infiltrate into the extraocular muscles and orbital fat. The fibroblasts proliferate and deposit glycosaminoglycans. The resulting muscle and fat enlargement combines with interstitial edema to cause an increase in intraocular pressure. Over time, increases in intraocular pressure also produce conjunctival chemosis, excessive lacrimation, periorbital edema, and photophobia which characterized ATA class II disease. As enlargement of orbital muscle and fat progresses, the volume of the orbital contents increases. An increase in 4 ml in the volume of the orbital contents will result in 6 mm of proptosis (ATA class III disease). As the extraocular muscles become increasingly enlarged by edema and infiltration, they also become dysfunctional resulting in decreased ocular mobility and diplopia (ATA class IV disease). Over time this progresses to a permanent fibrotic, restrictive ophthalmoplegia. Progressive proptosis also dramatically interferes with the eye’s protective mechanism of the cornea, causing exposure, desiccation, irritation, and ultimately ulceration (ATA class V disease). Corneal ulceration becomes a vision threatening problem, with a risk of permanent corneal scarring, and requires immediate attention. ATA class VI disease is the most severe form of Graves’ ophthalmopathy and involves damage to the optic nerve leading to impairment of vision. Optic nerve involvement typically presents as a painless gradual loss of visual acuity or visual field. The damage comes from compression and crowding of the optic nerve at the orbital apex by the enlarged extraocular muscles. Impairment of visual fields or color vision may be found in patients with normal visual acuity.

**Patient Evaluation**

Most patients with Graves’ disease are initially evaluated by a medical specialist. A full endocrinology workup is essential in the diagnosis and management of Graves’ disease. Some patients complain of the symptoms of hyperthyroidism, including heat and cold intolerance, weight loss, and emotionally and physically hyperactive states. For other patient, exophthalmos is the only presenting symptom. Any patient with unilateral or bilateral exophthalmos should be presumed to have thyroid disease. Most of the tests are repeated in the preoperative assessment for stability of the disease and for comparison with baseline tests results. In the acute stages, the values are characteristically increased for total triiodothyronine and free T3, total thyroxine and free T4, reverse T3 thyroid uptake, clearance and release of I¹³¹, and serum assays of thyrotropin-releasing hormone and thyroid-stimulating immunoglobulin. In some apparently euthyroid patients, more detailed testing of thyroid function may be required to uncover thyroid dysfunction. These studies include the suppression of radioiodine uptake with T3 to assess for non-TSH mediated thyroid stimulation, the thyrotropin-releasing hormone stimulation test to determine the presence of low grade suppression of the hypothalamic-pituitary axis, and TSH stimulation of thyroid reserve. Overall most if not all patients with euthyroid ophthalmopathy can be shown to have some degree of thyroid dysfunction.
The physical examination can confirm the upper and lower eyelid retraction, proptosis, and other physical signs of hyperthyroidism. The physician may notice tachycardia, sweating, hyperactivity, and anxiety during the examination. A particularly revealing sign is a hyperemia over the lateral rectus muscles. This is pathognomonic for thyroidal eye disease. A complete ophthalmologic examination and a head and neck evaluation should be performed, giving particular attention to the thyroid status. A thorough examination by a skilled ophthalmologist is critical for the diagnosis and management of Graves’ ophthalmopathy. Serial eye exams are required to monitor disease activity, progression, and response to therapy. Eye examination should include attention to soft tissue changes, including lid edema and retraction, chemosis, scleral injection, documentation of proptosis, and intraocular pressure in primary and upward gaze, limitation of ocular motility, strabismus and visual function in the form of acuity, color vision, and visual fields.

CT scans of the orbit can be helpful in the diagnosis of Graves’ ophthalmopathy in the euthyroid patient and are essential if surgical intervention is being considered. Typical CT findings include a twofold to eightfold enlargement of the extraocular muscle bodies, sparing the tendinous portions. The changes are bilateral in 90% of patients although asymmetry is very common. The medial and inferior rectus muscles are most commonly involved. The orbital and extraocular muscle volume may be estimated using CT images. Ultrasound of the orbit can be helpful to confirm the suspicion of orbital thyroid disease which is detected on physical examination. This test can demonstrate a thickening of all of the extraocular muscles. Although not as beneficial in the initial diagnosis and surgical planning as the CT scan, it is proposed as an inexpensive, noninvasive method for monitoring response to therapy (steroids or radiation). MRI of the orbits has proven excellent for the soft tissues of the orbit. Recent studies have suggested that T2 weighted MRI images may provide a sensitive measure of active inflammation in the orbit. Unfortunately, MRI provides little detail of the bony anatomy of the orbit, which is required if the possibility of surgical intervention is being entertained. The scans are required to rule out any other pathologic condition of the orbit, especially in the unilateral case of exophthalmos. If considering decompression, the scans should include the paranasal sinuses to help in operative planning. Infectious, allergic, or inflammatory conditions of the sinuses may disallow surgery unless adequately resolved preoperatively.

Graves’ ophthalmopathy presents a spectrum of clinical manifestations that are reminiscent of other clinical entities. The most common differential diagnosis to consider in bilateral proptosis is pseudotumor cerebri. In this disease there is diffuse edema of the soft tissues seen on imaging studies. The high prevalence of asymmetric eye involvement also may lead to the suspicion of a unilateral disease process rather than a systemic one. Lymphoma of the orbit can produce proptosis. With lymphoma there is a more localized mass seen on the scan, usually near the apex, and the proptosis may be ascentric. Thyroid exophthalmopathy is usually centric. Other space occupying lesions, such as metastatic tumor, vascular anomaly, neurofibroma, and retinoblastoma, can cause unilateral proptosis. Congenital shallowness of the orbits causes an obvious unilateral or bilateral exophthalmos. Although the differential diagnosis for proptosis is extensive, most other disease entities have only superficial similarities to Graves’ ophthalmopathy.
and can be quickly ruled out. Most importantly, the clinician should maintain a high
degree of suspicion if the diagnosis of Graves’ ophthalmopathy is to be made in a timely
fashion.

Management

A multispecialty team approach for the patients with Graves’ disease and Graves’
ophthalmopathy is recommended because of the multiple organ systems involved and the
variety of diagnostic and therapeutic modalities needed to provide optimal care. The
team should include an endocrinologist, radiologist, nuclear medicine physician,
radiotherapist, ophthalmologist, otolaryngologist, and neurosurgeon.

Medical Management

All patients with dysthyroid ophthalmopathy require complete endocrinologic evaluation
and management of their hyperthyroidism. The medical management of Graves’ disease
usually centers on the suppression of the thyroid activity through subtotal thyroidectomy,
$^{131}$I ablation, or exogenous thyroid hormone. After euthyroid status is achieved for 6
months, the orbital status usually stabilizes. However, 1% to 2% of patients develop an
acute deterioration of their orbital status, usually in the form of decreased visual acuity or
field defects. The treatment of choice is administration of 80 to 120 mg of prednisone
daily for as long as 14 days. If the visual dysfunction does not improve or prolonged
steroids is required to maintain visual acuity, surgical decompression is indicated. The
multiple side effects of steroid therapy are well known and include glucose intolerance,
weight gain, psychosis, peptic ulcer disease, and osteoporosis with vertebral fracture.
Thus, corticosteroid therapy should be considered temporizing while awaiting either
regression or stabilization of the disease or definitive therapy. Adjunctive treatment for
exposure keratitis and conjunctivitis includes the use of ocular lubricants and artificial
tears, moisture chambers, and taping retracted eyelids if possible. Low-dose radiation
therapy has been used successfully, but there is risk to the lens and the optic nerve with
this technique. Usually 20 Gy is delivered in 10 fractions over 2 weeks. The fractions
are delivered in a field behind the lateral canthus to spare the cornea and lens. There has
been reported a good to excellent response overall in 35% to 92% of patient and
improvement of impaired visual acuity in 33% to 85% of patients treated with orbital
radiation. Patients treated early in the course of the disease with pronounced soft tissue
involvement are most likely to benefit. Proptosis, ophthalmoplegia, and optic neuropathy
are less responsive, and patients with long standing stable disease are not likely to benefit
from radiation.

Surgical Management

Preoperative counseling is done to explain the surgical procedure and potential risks to
the procedure. The major counseling issues center on risks of vision motility disorders
and failure to achieve a satisfactory result. Optic nerve or retinal injury can occur from
undue or prolonged globe retraction during orbital decompression. Retrobulbar
hematoma from bleeding can lead to blindness. The procedures can lead to epistaxis,
damage to infraorbital nerve, scarring of skin incisions, and infection. In general, the
more advanced the exophthalmos, the more extensive is the surgery required to gain even modest improvement, and very few patients are completely satisfied with the initial procedure.

Surgical management is considered for two stages of dysthyroid exophthalmos. In the acute or subacute stage, steroids are employed to resolve or improve visual disturbances. If the patient fails to regain visual acuity or if the steroids are required for long term maintenance, surgical decompression is indicated. In the late stage, when palpebral retraction, exophthalmos, or ocular involvement is seen, cosmetic decompression is indicated. The usual functional indications for surgical decompression are decreasing visual acuity, visual field defects, abnormal visual-evoked potentials, and disc edema. Corneal exposure with keratitis not responsive to conservative medical management is another indication for decompression. Improvement of the cosmetic aspect of this disorder is becoming recognized as a valid indication for orbital decompression, as long as the patient understands the inherent risks to vision.

Superior orbital decompression involves unroofing the entire superior orbital wall by a frontal craniotomy. The advantage is the large amount of bone that can be removed by this technique. It is limited however by its disadvantages. These include having to perform a craniotomy, the delicate sparing of cranial nerves, and the transmission of vascular pulsations from the brain postoperatively. The procedure is performed in conjunction with neurosurgery who exposes the orbit by a frontal craniotomy. After the optic nerve has been adequately visualized, the bony roof of the orbit is carefully removed from just anterior to the optic foramen to the anterosuperior orbital rim. After the entire superior periosteum has been uncovered, it can be carefully incised in an H-shaped fashion, allowing the orbital fat to herniate into the cranial vault. A section of titanium mesh and a pericranial flap are used to close the defect. Because of the morbidity associated with this approach, superior decompression is considered only for severe cases of contracted orbit associated with proptosis and decompression associated with orbital trauma.

Medial orbital decompression can be approached through the standard external ethmoidectomy incision or through a coronal forehead approach. With the standard ethmoidectomy approach, the medial canthal tendon is displaced laterally by elevating the periosteum over the anterior lacrimal crest. The lacrimal sac is elevated out of its fossa and retracted laterally with a retractor. The anterior and posterior arteries are identified and ligated with surgical clips. The anterior artery is divided for exposure, but the posterior artery is left intact for orientation. The ethmoidectomy is carried posteriorly to the posterior ethmoid artery. A complete ethmoidectomy is performed and all of the mucosa bearing septa are removed. The posterior ethmoid cells are removed as far back as posterior ethmoid plate, but care is taken to avoid any injury to the optic nerve in this region. When the coronal incision is employed, the medial canthal tendon is left intact, and the ethmoidectomy is carried out from above. This approach carries a greater risk of injury to the lacrimal sac and insertion of the trochlea because of the need for wider periosteal undermining to achieve adequate exposure. After the ethmoidectomy is complete, the medial orbital periosteum is incised longitudinally with the axis of the orbit.
or in an H-shaped outline, allowing the orbital fat to herniate through the periosteal defect into the ethmoidectomy cavity. The incisions are closes in the standard fashion, usually with Penrose drains for short term operative site drainage.

Inferior orbital decompression creates a large inferior orbital floor blow out fracture while sparing injury to the infraorbital nerve. The procedure can be done through a Caldwell-Luc approach, but some authors prefer a subciliary or transconjunctival eyelid incision combined with a Caldwell-Luc maxillary antrostomy. This combined approach allows safe visualization of the floor through the orbital exposure while removing the bone through the antrostomy. A skin-muscle flap is elevated in the lower eyelid and the orbital rim is visualized. The periosteum over the rim is incised and elevated from the orbital floor for approximately 4 cm. A Caldwell-Luc incision is made sublabially and a wide antrostomy is made. The mucoperiosteum is removed from the roof of the maxillary sinus. Now the course of the infraorbital nerve can be visualized. Using a periosteal elevator or small osteotome, the bone medial and lateral to the nerve is carefully fractured. Through the antrostomy the remainder of the floor can be removed with Takahashi forceps and a back-biting rongeur. By removing this bone under direct visualization, inadvertent injury of the globe, muscles, or optic nerve is avoided. In general, a 3 cm anteroposterior range for bone removal is safe. The thin, easily removable bone becomes thicker and denser as the posterior extent of the orbit is reached. Medially the bone can be removed to the lacrimal fossa and laterally it can be removed to the thick bone of the zygoma. Once the defect is as large as it can be, the periorbita is incised longitudinally to allow the orbital fat to herniate into the defect and into the maxillary sinus. The number of incisions is determined intraoperatively by assessing the degree of residual proptosis after each incision. Some authors recommend operating on the most severe eye first with planned incomplete recession because an additional 1 to 2 mm of recession develops during the first 3 months after surgery. The less severe eye is then decompressed to match the position of the first eye. Four to six incisions in the periorbita usually will be adequate. Gentle spreading of the incisions with a hemostat and teasing the fat out through these defects can also be performed. The fat herniates into the two defects on either side of the infraorbital nerve, which limits somewhat the amount of herniation that can take place. Next the middle meatal ostium is enlarged to provide an adequate drainage and ventilation orifice to the maxillary sinus. The sinus is then irrigated free of blood and debris before closing the Caldwell-Luc incision and a Penrose drain can be placed into the sinus and brought out the opening in the nose. The eyelid incision is closed in two layers – the orbital rim periosteum and the eyelid skin. By leaving out the intervening soft tissue sutures the risk of ectropion is decreased. As with all decompression procedures associated with the paranasal sinuses, perioperative antibiotic prophylaxis is used. Immediately after surgery all patients, unless medically contraindicated, are maintained on high-dose corticosteroids with a slow taper. Combined antral and ethmoid decompression has been shown to produce over 5 mm of mean reduction of proptosis. For those with optic neuropathy, this decompression was effective in improving vision in 92% of patients. Major reduction in intraocular pressure seen in 100% of patients, improvement in extraocular motility seen in 36%, and improvement in strabismus seen in 47% are other benefits of decompression. Utilizing
an inferior decompression through a transconjunctival incision alone, one could expect approximately 3.5 mm mean reduction of proptosis.

Lateral orbital decompression can be approached through a variety of incisions, including a coronal, direct rim incision, or through an extended lateral canthotomy. The lateral canthotomy incision alters the shape of the lateral palpebral angle and the coronal incision is quite extensive and has the potential for injury to the frontal branch of the facial nerve. The direct lateral orbit incision can be made as an extension of the subciliary incision into a smile wrinkle line when the two types of decompression are used. After the incision of choice is made the periosteum over the lateral orbital rim is exposed. It is incised widely directly over the rim and elevated from the orbital side and infratemporal fossa side of the lateral orbit for approximately 3 to 3.5 cm posteriorly. The lateral orbital rim can be cut and mobilized leaving its attachment to the periosteum intact for later placement into its proper position. Much of the lateral orbital wall can be removed until the thick bone of the skull base is encountered (approximately 2.5 to 3.5 cm diameter circle of bone is removed). As with other sites of decompression, the periorbita is incised and the orbital fat gently teased out to protrude into the newly created space. The lateral extension of the orbital fat is limited by the position of the temporalis muscle. The orbital rim fragment, still attached to the periosteum, can be replaced and fixed into position with wire. The wound can be drained with a Penrose drain brought out through the incision.

Some surgeons are beginning to gain experience with decompression of the medial and medioinferior floors of the orbit through a transnasal approach. With this approach there is a lack of external incision and decreased potential risk of an oroantral fistula. The endoscopic sinus technique requires an experienced surgeon who can also decompress the orbit if there is retrobulbar bleeding. This technique can not decompress the orbital floor lateral to the infraorbital nerve and can not extensively open the periorbita for exposure and extrusion of orbital fat. This approach may require that a septoplasty be performed to allow good exposure to the middle meatal region. The uncinate process is taken down with a sickle knife or pediatric upbiting forceps. A large antrostomy is then created and enlarged superiorly to the level of the orbital floor and inferiorly to the roof of the inferior turbinate. A large antrostomy helps prevent postoperative obstruction of the opening by orbital tissue following decompression. The middle turbinate is routinely resected with angled scissors to provide additional room for the orbital tissues and to prevent postoperative adhesions. An endoscopic ethmoidectomy is carried out and the anterior and posterior ethmoid arteries are identified. The medial orbital wall is exposed from the fovea ethmodalis to the anterior face of the sphenoid sinus. As an alternative to performing a Caldwell-Luc approach, a puncture antrostomy of the anterior wall of the maxillary sinus is performed with a trocar. Insertion of the trocar allows placement of a 30 degree scope through the puncture antrostomy to provide visualization of the orbital floor while instrumentation is performed transnasally. The infraorbital nerve is identified and mucosa is elevated and removed from the roof of the maxillary sinus to expose the bone over the infraorbital nerve bundle. The bone of the lamina papyracea is carefully palpated and fractured with a cottle elevator. Care is taken to avoid laceration of the periorbita with herniation of orbital fat into the surgical field. The lamina papyracea is
removed to the level of the ethmoid arteries, but the lacrimal crest is not violated, in order to protect the sac and the canthal tendon. Removal of bone superiorly continues to within 2 mm of the fovea ethmodalis, posteriorly to the anterior face of the sphenoid sinus, and laterally to within several millimeters of the infraorbital nerve. At the juncture of the inferior and medial orbital walls, a buttress of bone is preserved anteriorly to avoid excessive inferior displacement of the globe and the development of hypoglobus. A sickle knife is then used to incise the orbital periosteum superiorly in a posterior to anterior direction. Care is taken to avoid excessive penetration with the sickle knife, since the medial rectus muscle is often enlarged, superficial, and at risk for injury. The orbital fat is allowed to protrude into the ethmoid cavity. Postoperative packing is not used. A silastic splint is placed intranasally and secured to the nasal septum with a single suture to prevent adhesions between the nasal septum and the lateral tissues. With endoscopic approach alone approximately 3 mm of exophthalmos can be corrected. Proptosis can be reduced by 5 mm if both endoscopic and lateral decompression are utilized.

Recently several authors have begun to propose removal of orbital fat as an alternative procedure to orbital decompression. The approaches to these fat deposits are usually the standard ones, namely subciliary and upper lid crease. The orbital septum in both lids must be opened longitudinally and the fat compartments debulked. Caution must be rendered in achieving hemostasis with the bipolar cautery, avoiding the lacrimal gland in the upper lateral quadrant of the upper lid, and protecting the inferior oblique muscle. As much as 6 mm of proptosis reduction can be gained with this approach.

Complications

If Graves’ ophthalmopathy is allowed to progress unchecked, some patients develop progressive optic neuropathy, which can cause blindness. Others develop only the widely retracted eyelids, stiffness of orbital tissues, and exophthalmos. If there is significant eyelid retraction and exophthalmos, exposure of the cornea, ulcerations, and subsequent endophthalmitis can occur, and the globe can be lost to infection. The major complications of medical management is the failure to recognize when it is not sufficient treatment and to delay surgery. The vision may continue to deteriorated while on steroid therapy. The other complications from steroid therapy include gastric ulcer and perforation, irritable personality, failure to heal wounds, and reactivation of previously dormant infections. Radiation therapy with inadequate shielding can cause cataracts, pituitary suppression, and optic fibrosis. If surgery is performed after radiation, more bleeding and fibrosis is encountered and the optimal result is diminished because of the orbital stiffness.

Decompression surgery improves the function and cosmesis of approximately 75% of patients. Some degree of diplopia may be experienced by as many as 50% of these patients preoperatively and postoperatively. Postoperative muscle imbalance may be significantly reduced by complete removal of the inferior periorbita. It is also likely that taking great care in teasing the orbital fat into the decompressed area can help to lessen this possibility. Corneal abrasion after surgery can occur because the cornea is not
protected with a corneal protector during surgery. It is important to visualize the pupil periodically during the decompression because excessive retraction on the globe and optic nerve is heralded by a dilating pupil. The eyeball must be retracted for only short periods and then allowed to breathe to reduce the stresses on the vasculature and intraocular pressure. Retrobulbar hematoma formation from bleeding in the fat pad after incising the periorbita and teasing out the fat can also cause blindness. It is important to cauterize and irrigate after decompression surgery to identify any potential bleeding sites. Injury to the infraorbital nerve during inferior decompression procedure is not uncommon, and at least temporary neuropraxia occurs in most cases. After a lower eyelid subciliary incision, ectropion is usually transient and responds to time and gentle massage. Retinal hemorrhage is rarely observed and occurs only in diabetic patients. There is a risk of orbital cellulitis if significant sinusitis occurs due to the relatively unprotected orbital tissues. Early use of antibiotics for purulent rhinorrhea is prudent.

**Emergencies**

Retrobulbar hematoma, retinal vascular occlusion, and corneal ulcer are the major sight threatening emergencies associated with orbital decompression procedures. Retrobulbar hematoma is treated with opening of the skin incisions, evacuating the hematoma, irrigating the clots in the wound, bipolar cauterization of bleeding vessels, and adequate drainage of the wound. Retinal vascular occlusion and corneal ulceration are serious conditions and should be managed on an emergent basis by the ophthalmologist. Retinal vascular occlusion is usually related to increased intraocular pressure from edema of the orbit, excessive retraction, or retrobulbar hematoma. Blindness may result even with adequate treatment as the occlusion can not always be reversed. Late complications should be anticipated. The patient should be discharged with the appropriate lubricants, artificial tears, and instructions on taping the eye shut if necessary. The patient should be cautioned after discharge from the hospital to seek immediate care for increasing pain in the eye or for decreasing vision.
References


