# Ophthalmology<sup>®</sup>

# **Oculofacial Plastic Surgery:** Thinking Outside the Socket

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The term "oculofacial" represents the expanded scope of practice of oculoplastic surgery that includes surgery of the forehead, midface, and lower face. There is an overlap between the practice focuses of craniofacial plastic surgeons, head and neck surgeons, oral maxillofacial surgeons, and dermatological surgeons in facial surgery. However, the ophthalmologist provides a unique skill set because of advanced knowledge and comfort with periocular anatomy and function. This issue of *Ophthalmology Rounds* presents the role of the ophthalmologist in the management of facial disorders: the subspecialty of oculofacial plastic surgery.

Historically, the specialties of ophthalmology and otolaryngology were collectively termed "EENT"; ie, eyes, ears, nose, and throat. The American Academy of Ophthalmology and Otolaryngology was founded in 1903. Over the ensuing decades, the 2 specialties separated into their respective fields. The American Board of Ophthalmology was the first specialist board in North America, established in 1916. Although ophthalmic plastic surgical procedures were being performed by ophthalmologists at this time, there was an increasing trend towards further specialization. Watershed anatomical regions that neither ophthalmologist nor otolaryngologist independently managed included the nasolacrimal system, periocular region, and the orbit. By the beginning of World War II, oculoplastic surgery was recognized as a distinct subspecialty of ophthalmology.<sup>1,2</sup> This provided the missing link between ophthalmology and the disciplines of neurosurgery, otolaryngology, oral surgery, and general plastic surgery.

In 1969, the American Society of Ophthalmic Plastic & Reconstructive Surgery (ASOPRS) was founded. This organization now represents over 600 members internationally and has coined the term "oculofacial plastic surgery" to better describe the expanded scope of practice for oculoplastic surgery. ASOPRS is currently working towards attaining board certification for oculofacial plastic surgery by the American Board of Medical Specialties. The Canadian Society of Oculoplastic Surgeons (CSOPS) was founded in 1981.

#### **Oculofacial Evaluation**

After a patient enters the examination room, ophthalmology training typically conditions the practitioner to narrow focus and scan ocular structures, moving systematically from eyelids to anterior segment to posterior segment. This ability to "zoom in" and detect subtle discrepancies in normal patterns of microanatomy develops keen observation skills in ophthalmologists over time. However, this approach can lead to "missing the forest for the trees" in certain clinical scenarios. It is helpful to routinely check the entire face for symmetry, skin quality, and for the presence of abnormal lesions. For certain patients, specific testing of extraocular motility, trigeminal sensation, facial nerve function, globe retropulsion, or lymph node palpation is also necessary.

The goal of this paper is to provide clinically relevant information from the field of oculofacial plastic surgery for the comprehensive ophthalmologist. Clinical cases are used to highlight the value of examining the entire facial profile of a patient presenting to an ophthalmology practice. The cases will help review pertinent anatomical subunits of the face, formulate differential diagnoses, and consider surgical and non-surgical management options for various clinical entities. Take-home messages are highlighted at the end of every case.

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#### Case 1

A 64-year-old male presents with right lower eyelid ectropion that has worsened over the past 2 years (Figures 1A,B). His past medical history is significant for basal cell carcinoma (BCC) resected from the right cheek 7 years ago.

**Figures 1A,B:** Right lower eyelid ectropion (frontal [A] and right three-quarter [B] views).



The differential diagnosis includes involutional, paralytic, mechanical, or cicatricial etiology. The periocular examination is significant for moderate-to-severe eyelid laxity and right lagophthalmos (1 mm). Extraocular motility is full and pupillary responses are normal. Slitlamp evaluation is normal. Management options include observation, conservative therapy with artificial tears, or surgical repair by lateral canthal tightening.

Although this appears to be a straightforward case of right lower eyelid involutional ectropion, an examination of facial nerve function demonstrates decreased frontalis (brow) function and weaker orbicularis oculi tone on the ipsilateral side. Using the House-Brackmann scale,<sup>3</sup> facial nerve function is II-III/VI on the right side compared with I/VI on the left. Lymph node palpation of the head and neck region reveals a firm, nontender, parotid lesion. The mass effect is causing impaired right facial nerve function, contributing to the eyelid laxity and ectropion. This patient eventually underwent surgical resection of the parotid mass by the head and neck service.

The histopathological diagnosis was metastatic BCC.

#### **Relevant anatomy**

The facial nerve (7<sup>th</sup> cranial nerve) exits the brainstem at the level between the pons and medulla. It has both intracranial and extracranial components. The intracranial branches provide parasympathetic innervation to the lacrimal and other glands, motor supply to the stapedius muscle of the inner ear, and taste sensation to the anterior tongue. The extracranial branches innervate all facial mimetic (expression) muscles. Five major extracranial branches exit distal to the stylomastoid foramen and innervate the frontalis muscle (temporal branch), orbicularis oculi, corrugator supraciliaris and procerus muscles (temporal and zygomatic branches), muscles to raise the lips and smile (buccal branch), muscles to depress the lips and frown (marginal mandibular branch), and neck muscles including the platysma (cervical branch). Minor extracranial branches include the posterior auricular nerve, which controls the movement of certain scalp and outer ear muscles, and branches to the digastric and stylohyoid neck muscles.

#### Take-home message

Any patient with brow asymmetry, eyelid laxity, ectropion, lagophthalmos, or an asymmetric smile may have subtle facial nerve paralysis. A patient can be screened quickly by asking them to perform the following movements:

- raise the eyebrows
- close both eyes as tightly as possible (pull the eyelids apart with your fingers to gauge muscle strength)
  smile while showing the teeth

Any abnormality or asymmetry detected should prompt additional work-up including palpation in the pre-parotid region (just anterior to the ear).

#### Case 2

A 60-year-old female presents with "droopy and heavy eyelids" that have been worsening over the past 5 years (Figure 2). This became particularly bothersome since her cataract extractions last year. She denies any double vision or eyelid fatigability and the past medical history is noncontributory.

**Figure 2:** "Droopy" eyelids. The margin-to-corneal light reflex distances of the upper eyelids are 0.5 mm for the right eye and 1.5 mm for the left eye.



When a patient presents with upper eyelid ptosis, 3 potentially fatal diagnoses should be immediately ruled out by history and clinical examination: oculomotor nerve (3<sup>rd</sup> cranial nerve) palsy, myasthenia gravis, and Horner syndrome. This is accomplished by testing extraocular motility, pupillary responses, and, when indicated, fatigability by measuring the upper eyelid position (margin-to-corneal light reflex distance of upper eyelid [MRD1]) at baseline and again after 2 minutes of sustained upgaze. Further investigation is required if any of these tests are abnormal. The differential diagnosis for upper eyelid ptosis also includes involutional ptosis (levator aponeurosis dehiscence), congenital ptosis (levator palpebrae superioris [LPS] weakness), chronic progressive external ophthalmoplegia, traumatic dehiscence, mechanical effect of tumour, and pseudo-ptosis (ipsilateral enophthalmos, contralateral eyelid retraction, or contralateral exophthalmos). A normal MRD1 measurement is 4.0 mm and any of the above causes for ptosis will decrease this distance. Our patient has an MRD1 of 0.5 mm for the right eye and 1.5 mm for the left eye (Figure 2).

The levator excursion should be tested for each eye. Excursion >15 mm indicates that the LPS muscle strength is normal and that a dehisced aponeurosis is the likely cause for ptosis. However, measurements <10 mm suggest weakness of the LPS muscle itself. In this patient, levator excursion was >20 mm in each eye. The presumptive diagnosis is levator aponeurotic dehiscence worsened after cataract surgery due to stretching of the tendon by intraoperative speculum use.

In addition to eyelid position, facial nerve function testing is indicated for this patient. The brow asymmetry demands that the frontalis and other facial mimetic muscles be tested to rule out a nerve paralysis. Patients with subtle, mild forms of Bell palsy may present with brow ptosis and no history of frank paralysis. In addition to frontalis function, the brows themselves should be tested to determine whether they rest in normal anatomical position, just anterior to the supraorbital rim. Brow position may be tested by simultaneously pressing both thumbs upon the forehead at the level of the eyebrows. The goals are first to palpate the supraorbital rim and then to pull the brows upward and determine whether or not the maneuver pulls excess eyelid skin away.

Dermatochalasis is the term used for excessive eyelid skin or prominent fat pads that may drape over the eyelid margin and/or create temporal hooding over the lateral canthus. Upper dermatochalasis may occur primarily with normal brow position or secondarily due to brow descent, collapsing eyelid skin into the interpalpebral zone. "True" ptosis implies that the MRD1 is <4.0 mm. However, patients with dermatochalasis commonly present with an appearance of ptosis despite normal MRD1. It is imperative to distinguish between true eyelid ptosis, simple dermatochalasis, and secondary dermatochalasis from brow descent because management options for these vary considerably.

The surgical management for simple dermatochalasis is upper blepharoplasty (Figures 3A,B) while that for secondary dermatochalasis is brow ptosis repair. Options include direct brow elevation by excision of a skin-muscle flap using a hyperbeveled trichophytic incision, minimally invasive external browpexy,<sup>4</sup> and endoscopic forehead and eyebrow elevation (Figures 4A,B). Many other brow-lifting techniques have also been described but are beyond the scope of this article.<sup>5-7</sup> Patients with true ptosis are treated by traditional techniques including levator advancement, conjunctiva-Müller muscle resection, and frontalis suspension with silicone slings (Figures 5A,B).

#### **Relevant anatomy**

The primary retractor of the eyelid is the levator palpebrae superioris muscle, innervated by the superior

**Figures 3A,B:** 3A: Simple upper and lower eyelid dermatochalasis (excess skin and prominent fat pads). 3B: 4 weeks after quad-lid blepharoplasty.



**Figures 4A,B:** 4A: Bilateral brow ptosis with secondary dermatochalasis and temporal hooding. 4B: 2 months post-operatively after endoscopic frontal advancement for brow elevation.



**Figures 5A,B:** 5A: Congenital left upper eyelid ptosis with no levator excursion and absent lid crease (left three-quarter view). 5B: 1 day post-operatively after frontalis sling.



division of the oculomotor (3<sup>rd</sup> cranial) nerve. Müller muscle is a secondary eyelid retractor innervated by the sympathetic nervous system and responsible for 2 mm of elevation. The primary retractor of the eyebrow is the frontalis muscle, innervated by the facial (7<sup>th</sup> cranial) nerve. The protractors of the eyelid and eyebrow include the orbicularis oculi, corrugator superciliaris, and procerus muscles, all of which are innervated by the facial nerve. These protractors are responsible for vertical glabellar lines and rhytids around the eyes.

#### Take-home message

A "droopy" eyelid may be secondary to true ptosis (MRD1 <4.0 mm), simple dermatochalasis (excess eyelid skin and fat), or secondary dermatochalasis (from brow descent). The surgical management for these entities is different, although some patients will require multiple approaches because of combined ptosis and dermatochalasis.

#### Case 3

An 85-year-old woman presents with a right lower eyelid lesion that has progressively enlarged over the past year (Figure 6). She has a history of regular cryotherapy treatments administered by her dermatologist for facial actinic keratoses.

**Figure 6:** Right lower eyelid nodular lesion with loss of eyelashes (madarosis), distortion of the lid margin architecture, and induration.



A useful approach to periocular "lumps and bumps" is to first determine whether features of malignancy are present and then to consider which anatomical structure is giving rise to the lesion (ie, epidermis, dermis, hair follicle, sweat gland, sebaceous gland, or other adnexal structure). Periocular cutaneous malignancies include BCC, squamous cell carcinoma, sebaceous cell carcinoma (SebCC), and cutaneous malignant melanoma. Between 5% and 10% of all skin carcinomas occur on the eyelid.<sup>8</sup> BCC is the most common skin cancer affecting the face and accounts for 90% of malignant eyelid tumours.<sup>8,9</sup> Although it carries a low metastatic potential, the carcinoma is locally invasive and destructive. Nonmelanocytic lesions (BCC, SCC) may demonstrate malignant features (Table 1). Melanocytic tumours (nevus, melanoma) may increase in size (vertical or radial growth), change in shape, or increase in pigmentation. Adnexal lesions (SebCC) may masquerade as chronic, unilateral blepharitis or recurrent chalazia. Any periocular cutaneous malignancy with orbital infiltration may cause vision loss, exophthalmos with or without double vision, impaired eye motility, or decreased trigeminal sensation.

This patient has a right lower eyelid lesion with many features suggestive of malignancy arising from the deep epidermis. During the initial assessment, a complete ophthalmological examination is performed, including visual acuity, pupil reactivity, colour vision, slit-lamp evaluation, assessment of extraocular motility, periocular and facial skin assessment, retropulsion, exophthalmometry, dermatomal trigeminal sensation (V1 and V2), and head and neck lymph node palpation. Any evidence of orbital involvement requires orbital neuroimaging (computed tomography [CT] and magnetic resonance imaging with and without contrast). To confirm the diagnosis, an incisional or excisional biopsy can be performed for nonmelanocytic tumours. Pigmented or nonpigmented lesions suspicious for a melanocytic lesion (nevus or melanoma) are best treated by excisional biopsy with 2-4-mm margins and a "no-touch" technique to avoid spillage of tumour cells. Lesions suspicious for SebCC should be sent fresh so that fat stains such as oil-red-o can be used. Because these tumours can undergo pagetoid spread and commonly have

# Table 1: Malignant features of non-melanocytic lesions

- Loss of eyelashes (madarosis)
- Distortion of the eyelid margin architecture
- Ulceration
- Pearly borders
- Telangiectatic vessels
- Induration
- Lack of tenderness
- Bleeding
- Evidence of rapid lesion growth

multicentric involvement, map biopsies should be performed. The TNM (tumour/lymph nodes/metastasis) classification system for cutaneous malignancy distinguishes noneyelid and eyelid tumours.<sup>10</sup>

There are many surgical and nonsurgical management options for periocular cutaneous malignancies. Nonsurgical options include cryotherapy, radiation, photodynamic therapy, electrodissection and curettage, topical 5-fluorouracil, and topical immune modulators such as imiquimod. The problem with nonsurgical management, however, is that no pathological specimen is reviewed to ensure complete tumour eradication. Surgical resection of the lesion is the gold standard; options include primary excision with predetermined margins, frozen section-controlled excision, or Mohs micrographic surgery (MMS). The cure rates using these techniques for periocular BCC are 85%–100%.<sup>11-13</sup>

Our group has found that eyelid margins treated with full-thickness block resection of the primary lesion and en face frozen-section control of the medial and lateral margins results in a 5-year cure rate of 100%.<sup>14</sup> The advantage is that both tumour eradication and eyelid reconstruction are performed in a single operative setting. Larger lesions or those that involve the medial canthus or other facial areas may be more amenable for MMS to ensure complete tumour eradication with the benefit of maximal tissue preservation.<sup>11</sup> Disadvantages include lack of access to Mohs surgeons and the need for 2 separate operations. Defect reconstruction after MMS is typically performed by a general plastic, facial plastic, or oculoplastic surgeon. Because oculofacial plastic surgeons are comfortable with periocular eyelid and facial anatomy, they are uniquely trained to reconstruct large eyelid-cheek defects (Figure 7A-D).

Figure 7A-D. 7A: Right lower eyelid margin and midfacial defects after Mohs resection of basal cell carcinoma (right three-quarter view). 7B: Reconstruction by temporal rotation flap and large island-pedicle flap from the lower face. 7C: Left periocular-facial defect down to bone after necrotizing fasciitis debridement. 7D: Reconstruction by superficial musculoaponeurotic system (SMAS) lift with rotational midface flap and full-thickness skin graft from the contralateral upper eyelid.





#### Relevant anatomy

The eyelid margin consists of anterior lamellae (skin and orbicularis oculi muscle) and posterior lamellae (tarsal plate and conjunctiva). Lid margin reconstruction after tumour resection requires precise realignment of all 4 eyelid layers to optimize both function and cosmesis. This is why the tissue-preserving benefits of Mohs resection are not as useful for eyelid margin tumours. Preserved tissue will still need to be sacrificed in order to precisely realign the lid margin during reconstruction. The lower eyelid and upper cheek (to the level of the upper lip) should be considered as a single continuous anatomical unit. Resection and reconstruction in this region requires special care to avoid post-operative lid retraction, injury to branches of the facial and infraorbital nerves, and scarring caused by poorly oriented local flaps.

#### Take-home message

The goals for treating a patient with facial cutaneous malignancy are tumour eradication and reconstruction of the defect to optimize function and cosmesis. For eyelid margin tumours, these goals are best accomplished with full-thickness eyelid block resection using *en face* frozen section analysis and reconstruction, while larger lower eyelid-facial or medial canthal lesions may be more amenable to Mohs resection followed by secondary reconstruction.

#### Case 4

The emergency room calls you to assess a 57year-old male who was involved in a collision with a truck while riding his motorcycle. He complains of blurry vision on his left side. The brain CT is normal and the patient denies any focal neurological symptoms. On examination, his visual acuity is 20/25 OD and 20/400 OS. There is a moderate left relative afferent pupillary defect; however, slit-lamp, intraocular pressure, and dilated funduscopic examinations are normal. The periocular examination is significant for left enophthalmos (3 mm), hypoglobus (2 mm), and lateral canthal dystopia (Figure 8).

**Figure 8:** Left hypoglobus, enophthalmos, and lateral canthal dystopia after a motorcycle-truck collision.



Ophthalmology

First and foremost, the cause for traumatic vision loss with a pupillary defect needs to be addressed. Because the eye examination itself is normal, the site of injury is likely retrobulbar. The 3 most likely mechanisms to account for this include: • orbital compartment syndrome (secondary to

- retrobulbar hemorrhage, edema, or air)
- direct optic nerve injury (secondary to a bone fragment or foreign body)
- indirect traumatic optic neuropathy (TON)

Extraocular motility testing is necessary to determine whether eye muscle entrapment has occurred and whether an orbital mass or hemorrhage is present. Decreased retropulsion (ie, firm globe upon palpation over the closed eyelid) is suggestive of an orbital hemorrhage while normal or increased retropulsion is consistent with orbital volume expansion secondary to a large fracture. The infraorbital (V2) sensation is often decreased secondary to a blowout fracture. The orbital rims should be palpated for step-deformities. In addition to clinical testing for isolated orbital fractures, the patient should also be examined for possible zygomaticomaxillary complex (ZMC), naso-orbito-ethmoidal (NOE), and mandible fractures. Clinical signs of ZMC fracture include lateral canthal dystopia, malar eminence (cheek) flattening, posterior displacement and rotation of the lateral orbital wall, and pain while opening and closing the mouth. Clinical signs of NOE fracture include medial canthal dystopia, telecanthus, forehead numbness, and cerebrospinal fluid rhinorrhea. Clinical signs of a mandibular fracture include malocclusion (upper and lower teeth do not meet properly) and abnormal sensation in the teeth. A CT scan with 1-mm cuts through the axial and coronal planes of the orbit and facial bones should be ordered. The scans are reviewed for the presence of orbital, optic canal, or other facial fractures or of blood in the sphenoid sinus, which raises suspicion for an optic canal fracture.

This patient suffered left orbital floor and medial wall fractures in addition to a left ZMC fracture (Figure 9). He underwent transconjunctival orbital floor and medial wall fracture reduction using a porous polyethylene implant. The zygoma was repositioned through the same transconjunctival incision. Titanium plating was used to fixate the

**Figure 9:** Axial computed tomography image demonstrating left posterior maxillary wall fractures and a fracture through the left zygomatic arch.



zygoma in normal anatomical position. Transoral (ie, Caldwell-Luc) and transcutaneous approaches to the midfacial skeleton are also used but the transconjunctival approach provides excellent exposure and longterm cosmesis.

The cause of vision loss in the left eye was indirect TON. The patient was counseled about the relatively high rate of spontaneous recovery following indirect TON and the lack of evidence that high-dose corticosteroid therapy is more effective than observation alone.<sup>15</sup> The results of the Corticosteroid Randomization After Significant Head Injury (CRASH) trial<sup>16</sup> were also discussed, which showed higher mortality rates of patients treated with corticosteroids in the setting of acute traumatic brain injury.

#### **Relevant anatomy**

The zygomatic arch is a principal constituent of the midfacial skeleton. It overlies the temporalis muscle and is the origin of the masseter muscle. The zygoma provides normal cheek contour and lateral support for the globe. It has four bony attachments to the skull (maxillary bone, temporal bone, frontal bone, and sphenoid bone), which constitutes the zygomaticomaxillary complex (ZMC).

#### Take-home message

Patients with orbital fractures should also be examined clinically and radiologically for other facial fractures, including NOE, ZMC, and mandibular fractures. This can be accomplished by palpating over the nasal bone, examining the medial and lateral canthi, palpating over the cheek bones, asking the patient to open and close the mouth, and checking cheek and teeth sensation. Patients with indirect TON should be observed rather than treated with high-dose corticosteroid therapy.

#### Summary

Oculofacial plastic surgery is the field of eyelid, lacrimal, orbital, and facial surgery that is unique among all other surgical specialties that manage facial disorders. Examination of the facial profile of any patient presenting to an ophthalmology practice can typically be performed in under 1 minute and can help to establish accurate diagnoses and management plans for common clinical presentations.

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