**TITLE: Facial Nerve Paralysis: Ocular Management**
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Ocular complications from facial nerve paralysis can be quite devastating. Facial nerve paralysis results in cosmetic as well as functional problems. With facial nerve paralysis the upper lid fails to drop down and close. The lower lid may become lax and evert, developing a lower lid ectropion. Paralysis of the upper lid leads to lagopthlamos, which results in incomplete closure of the lid over the cornea. This can cause the cornea to dry, disrupting the tear film and results in pain, corneal ulceration, infection and even corneal perforation. Pre-injury factors that lead to increased risk of complications are the lack of a good Bell phenomenon, corneal anesthesia and pre-injury dry eye.

Normal eye closure is facilitated by the action of the obicularis oculi. Contraction of this muscle results in lowering of the upper lid. The lower eye lid has a minimal contribution to eye closure. The eyelid functions to protect the eye from light, foreign material, injury and desiccation. It also functions in the distribution of tears and maintenance of the tear film. The skin of the eyelids is very thin, with the upper lid being thicker than the lower lid. There is little subcutaneous fat and the skin is adherent to the tarsal plate.

The normal horizontal distance of the eye is 28-30mm. The distance from the upper lid margin to the supratarsal crease is 10mm. The margin reflex distance, or the distance from the corneal reflex to the lid margin, is approximately 4-5mm for the upper lid and 5mm for the lower lid. The lid margin of the lower lid rests at the limbus of the eye. The upper lid margin rests approximately 2mm below the limbus.

The tarsus, or tarsal plate, is a dense fibrous tissue that provides contour and a fibrous skeleton to the lids. The tarsus is 25mm in length and 1mm thick. The tarsus contains the Meibomian glands. The height of the upper tarsus is 10mm and the lower tarsus is about 4mm.

The main protractor of the eye is the obicularis and is innervated by the facial nerve. The main retractors of the eye are the levator and Mullers muscle, innervated by the oculomotor nerve. The obicularis has three components: the orbital part, the pretarsal and the preseptal parts. The pretarsal and preseptal components are responsible the eye closure. The preseptal and pretarsal components extend medially to form the medial canthal tendon. The same areas extend

laterally to form the lateral canthal tendon.  The lateral canthal tendon attached to Whitnalls tubercle on the medial aspect of the lateral orbital wall.  The lateral canthus attachment is slightly higher than the medial canthal attachment.  The medial canthal complex is important for normal lacrimal functioning.  The lacrimal sac is situated between the anterior limb and posterior limb of the medial canthal tendon.  When blinking occurs, the tears are forced to the lacrimal sac through the canaliculus.  When the eye opens, the sac is compressed and the tears are forced out of the sac to the inferior meatus.

When eye paralysis occurs with impaired facial nerve function treatment should begin immediately.  Initial treatment consists of using ophthalmic drops and ointments, taping the eye, soft contact lenses, scleral shields or moisture chambers.  Also of utility is the tarsorrhaphy suture.  Despite the initial management of the eye, the majority of patients will require definitive surgical treatment to prevent permanent damage to the cornea.  Surgical treatments include the temporalis muscle transfer, encircling the eye with fascia lata or silicone, palpebral springs, tarsorrhaphy, lower lid shortening, lid loading or combinations of these procedures.

Palpebral springs are placed under the skin of the upper lid.  They act to force the eye closed with relaxation of the levator and Mullers muscle.  Palpebral springs are less visible, but are technically more difficult to place and have a higher rate of extrusion.

Tarsorrhaphy works well for lower lid ectropion.  However, it is cosmetically less appealing and often results in loss of peripheral vision.

Lower lid shortening procedures consist of wedge excision with lateral canthopexy.  This procedure works well for lower lid ectropion and as an adjunct to upper lid loading.

The early technique of upper lid loading consisted of making an incision in the pretarsal crease and forming a pocket in the subcutaneous tissue.  A weight was then placed in the pocket and the skin closed.  This technique resulted in frequent mobility of the weight and extrusion.  The earlier weights were stainless steel.  These weights had a higher profile, were migratory and had a high rate of extrusion.  Later, the gold weight was used.  It has a higher density so a heavier weight of the same size can be used.  It is also malleable, so it can be conformed to the contour of the globe.  It was also mobile and a high rate of extrusion.

The later technique of upper lid loading consist of making an incision in the upper pretarsal crease and incising through the levator aponeurosis.  The tarsal plate is exposed and the weight is placed directly on the tarsal plate and fixed with sutures.  The levator aponeurosis, muscle and skin are then closed in layers.  This provides better coverage of the weight and prevents extrusion and migration and the plate is less visible.  The technique is straight forward and consistent.  Though gold remains the implant material of choice, recently a titanium chain has been developed.

Kinney et al described an algorithm for the management of lid paralysis.  Surgical techniques described included auricular cartilage grafting, tarsorrhaphy, lateral canthotomy, elevation of the subobicularis oculi fat and brow lifting.  Ultimately, gold weight implantation was necessary.

Snyder et al investigated the timing of gold weight implantation.  They evaluated the

outcomes and complications of early (<30 days) and late (>30 days) gold weight implantation. They found that 89.2% achieved satisfactory eye closure. The early and late groups were statistically similar in eye closure and complication rates.

Foda investigated upper lid gold weight implantation and lower lid shortening. He found that this combination resulted in complete correction of lagophthalmos and ectropion. Pre op symptoms resolved in 92.5% of the patients studied.

Harrisberg reported a series of 103 patients with upper lid gold weight implants. Forty six patients had their weights removed. Of these, 78% were removed because of facial nerve recovery. The remaining 22% were removed because of cosmetic dissatisfaction, implant becoming too superficial, migration and partial extrusion. The majority of these implants were placed using the earlier method.

In a series 16 patients, Chepeha reported improvement in lagophthalmos, corneal coverage and a high patient satisfaction. The lagophthalmos improved from an average of 7.5mm to 0.5mm. Corneal coverage improved from 73% to 100% coverage. There were no extrusions in this series.

In conclusion, upper lid loading with gold weights is safe and effective. They may be implanted early and are reversible. When used in combination with lower lid shortening, upper lid gold weights provide excellent cosmetic and functional results in the paralyzed eye.

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