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Neuro-ophthalmology Update

Neuro-ophthalmological approach to facial nerve palsy

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Abstract

Facial nerve palsy is associated with significant morbidity and can have different etiologies. The most common causes are Bell's palsy, Ramsay–Hunt syndrome and trauma, including surgical trauma. Incidence varies between 17 and 35 cases per 100,000. Initial evaluation should include accurate clinical history, followed by a comprehensive investigation of the head and neck, including ophthalmological, otological, oral and neurological examination, to exclude secondary causes. Routine laboratory testing and diagnostic imaging is not indicated in patients with new-onset Bell's palsy, but should be performed in patients with risk factors, atypical cases or in any case without resolution within 4 months. Many factors are involved in determining the appropriate treatment of these patients: the underlying cause, expected duration of nerve dysfunction, anatomical manifestations, severity of symptoms and objective clinical findings. Systemic steroids should be offered to patients with new-onset Bell's palsy to increase the chance of facial nerve recovery and reduce synkinesis. Ophthalmologists play a pivotal role in the multidisciplinary team involved in the evaluation and rehabilitation of these patients. In the acute phase, the main priority should be to ensure adequate corneal protection. Treatment depends on the degree of nerve lesion and on the risk of the corneal damage based on the amount of lagophthalmos, the quality of Bell's phenomenon, the presence or absence of corneal sensitivity and the degree of lid retraction. The main therapy is intensive lubrication. Other treatments include: taping the eyelid overnight, botulinum toxin injection, tarsorrhaphy, eyelid weight implants, scleral contact lenses and palpebral spring. Once the cornea is protected, longer term planning for eyelid and facial rehabilitation may take place. Spontaneous complete recovery of Bell's palsy occurs in up to 70% of cases. Long-term complications include aberrant regeneration with synkinesis. FNP after acoustic neuroma surgery remains the most common indication for FN rehabilitation.

Keywords: Facial nerve palsy, Bell's palsy, Lagophthalmos, Acoustic neuroma, Neuro-ophthalmology

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Introduction

Facial nerve palsy (FNP) can have many different causes. It spans across all races and ages and has significant functional, psychological and social consequences.

Appropriate management is complicated by the wide spectrum of clinical presentation and disease severity. This article reviews the anatomy, the main causes and discusses acute management as well as the long-term options for long-standing FNP. The ophthalmologist plays a pivotal role in the multi-disciplinary team involved in the evaluation and rehabilitation of these patients.

Anatomy

The facial nerve (FN) may become dysfunctional anywhere along its course. The knowledge of its anatomy and origin of its branches may help the clinicians to localize the lesion.

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It is both a motor and sensory nerve with 3 nuclei:

(1) The main motor nucleus controls the muscles of facial expression. It lies deep in the lower part of the pons. Voluntary facial movements originate in the precentral gyrus. White matter tracts pass through the internal capsule and cerebral peduncles along with other corticobulbar fibers. The portion of the nucleus that supplies the muscles of the upper part of the face receives corticonuclear fibers from both cerebral hemispheres and that of the lower half of the face receives fibres only from the contralateral cortex. Therefore, with a lesion involving the upper motor neurons, only the contralateral lower part of the face will be paralyzed (central palsy). However, with a lesion of the main motor nucleus or FN itself (lower motor neuron lesion), all the affected ipsilateral side will be paralyzed (peripheral palsy). Another separate involuntary pathway exists (extrapyramidal pathways) controlling mimetic or emotional changes in facial expression and is largely responsible for involuntary blinking.
(2) The parasympathetic nuclei are the superior salivatory nucleus, which sends fibers for salivary secretion and the lacrimal nucleus, which supplies the lacrimal gland. It receives afferent fibers from the hypothalamus for emotional responses and from the trigeminal sensory nuclei for reflex lacrimation secondary to irritation of the cornea and conjunctiva.
(3) The sensory nucleus receives taste fibers from the anterior two-thirds of the tongue.

The FN has both a motor root and a sensory/parasympathetic root (the intermediate nerve). They emerge between the pons and the medulla oblongata. They pass laterally in the posterior cranial fossa and in the cerebellopontine angle, with the vestibulocochlear nerve, and enter the internal acoustic meatus of the temporal bone, where it traverses the fallopian canal. The fallopian canal has 3 portions: the labyrinthine, the tympanic and the mastoidal. The geniculate ganglion is located between the labyrinthine and the tympanic portion. It is an important anatomical landmark since the great superficial petrosal nerve (responsible for lacrimal secretion) and the small petrosal nerve (which carries secretory fibers to the parotid gland) emerge from it. Therefore, FN lesions above the geniculate ganglion classically cause more severe ophthalmic symptoms because lacrimal secretion and orbicularis closure are involved. On the other hand, the nerve to the stapedius muscle and the chorda tympani (responsible for taste sensation from the anterior two-thirds of the tongue and salivary secretion) branch out at the mastoid segment. The main branch of the FN exists through the stylomastoid foramen. It runs through the parotid gland to innervate the facial musculature through five terminal branches: temporal, zygomatic, buccal, mandibular and cervical.

Epidemiology

Incidence of FNP varies between 17 and 35 cases per 100,000.[1,2] There is no sexual preponderance.[1]

The incidence in neonates varies from 0.6 to 1.8 per 1000 live births, 91% due to forceps delivery.[1]

Bell's palsy (BP) is the most common disorder and affects 11–40 persons per 100,000 each year, with peak incidence between 15 and 50 years old.[3] In pregnancy, especially during the third trimester and early postpartum, there is a 3-times greater incidence.[1,3]

Ramsay–Hunt syndrome (RHS), one main cause of FNP, presents in only 0.2% of all Varicella Zoster Virus (VZV) cases.[4]

Etiology (Table 1)

Etiology of FNP varies according to the published series. As reported by Rahman (2007), the most common causes include BP (51%), trauma (22%) and RHS (7%).[1] Peitersen (2002) found 38 different etiologies of peripheral FNP (1701 cases of BP, 116 RHS, 76 diabetic, 46 pregnant and 169 neonates).[5] According to Hohman (2014), BP accounted for 38% of cases, acoustic neuroma resections 10%, cancer 7%, iatrogenic injuries 7%, RHS 7%, benign lesions 5%, congenital palsy 5%, Lyme disease 4%, and other causes 17%.[6] One analysis of 40 pediatric patients with peripheral FNP found 65% of BP, 37.5% infection, 2.5% tumor lesion and 2.5% suspected chemotherapy toxicity.[7]

FNP can occur with supranuclear, nuclear or infranuclear lesions and may be grouped into idiopathic (1), infectious (2), traumatic (3) and neoplastic (4) (Table 1).[8,9]

Supranuclear lesions may be caused by a lesion in the motor cortex, the subcortex or corticobulbar tracts. Commonly, the etiology is vascular, but may be demyelinating or tumoral.

Lower motor neuron lesions can be categorized anatomically[10]:

(1) Nuclear: Tumoral, inflammatory or ischemic pathology. It is usually associated with ipsilateral 6th nerve palsy and may also affect the descending corticospinal tracts causing contralateral limb weakness (*Millard–Gubler* syndrome).
(2) Cerebellopontine angle: Its contents include the CN V superiorly, the CN IX and X inferiorly and the CN VII and VIII in between. One of the first signs of this syndrome is the loss of corneal reflex on the ipsilateral side. Usually caused by an acoustic neuroma, it can also be caused by meningiomas, metastases, cholesteatomas or aneurysms. It is suspected in the case of impairment associated with CN VIII (deafness, vertigo, hyperacusis, tinnitus).
(3) Facial canal: The proximal part of the canal is particularly prone to ischemia and compression. BP, fractures of the temporal bone, malignant otitis externa or suppurative otitis media, RHS and neoplastic processes can affect the FN here.
(4) Parotid: A parotid mass with FNP is in general malignant. Other etiologies include inflammatory parotitis from infection or granulomatous conditions (sarcoidosis).

Pathophysiology

Idiopathic

BP is an acute paralysis of one side of the face of unknown etiology, which remains a diagnosis of exclusion.[11]

Table 1. Etiologies of facial nerve palsy.

Etiology	
Idiopathic	Bell's palsy
Infectious	Ramsay–Hunt Syndrome (Varicella Zoster Virus), Lyme disease, tuberculous chronic middle ear infections, dengue fever, leprosy, mumps, Epstein–Barr virus, cytomegalovirus, HIV, HTLV-1, polio, tetanus and diphtheria
Traumatic/ iatrogenic	Fractures of the temporal bone, post surgery of tumours in the cerebellopontine angle, oral and maxillofacial surgical procedures, otologic procedures, cosmetic procedures and forceps delivery
Neoplastic/ infiltrative	Acoustic neuroma, parotid tumors, facial nerve schwannomas, malignant tumors of the external meatus, nasopharyngeal carcinomas, lymphomas. Sarcoidosis, leukemia, collagenosis and amyloidosis
Miscellaneous	Neurologic causes: Multiple sclerosis, myasthenia gravis, Guillan–Barre syndrome, hereditary hypertrophic neuropathy, Melkersson–Rosenthal syndrome, Moebius syndrome, cerebrovascular accident Systemic/metabolic causes: Diabetes Mellitus, hyperthyroidism, hypertension, pregnancy, acute porphyria, carbon monoxide toxicity, vitamin A deficiency, ethylene glycol ingestion

Herpes simplex virus (HSV) reactivation in the geniculate ganglion is thought as the major cause of BP. Murakami isolated HSV-1 DNA from the endoneurial fluid of the FN by PCR during the acute phase of BP.[12] Histopathology shows the entire FN infiltrated by inflammatory cells, with edema, axonal changes and myelin breakdown suggesting viral neuritis.[1]

Another etiology has been postulated: perineural edema from retention of fluid and mechanical compression within the bony course of the FN. The increased incidence during pregnancy supports this theory.[8] Murai demonstrated that the mean cross-sectional area of the labyrinthine and horizontal segments of the canal were significantly smaller on the affected side of patients with BP compared with the contralateral side on computed tomography.[13,14]

Infectious

RHS is a severe complication of VZV reactivation in the geniculate ganglion and is the most common confirmed infective cause for FNP. The classic triad consists of otalgia, vesicles in the auditory canal and ipsilateral FNP.[14]

Borrelia burgdorferi, is a known cause of FNP in endemic regions.[1] Fifty percent of cases of BP in children younger than 10 years old are due to Lyme disease.[3] Presumptive diagnosis should be made in patients presenting with FNP associated with induration and erythema of the face, particularly in the summer.

FNP can be the first sign of AIDS, but is generally described in chronic HIV infection.[9]

Traumatic/Iatrogenic

Both blunt and penetrating cranio-facial trauma may cause FN injuries.[8] Road traffic accidents are the leading cause of temporal bone's fractures and in 31% of cases originates injury to the FN.[2] The FN may be mobilized, manipulated or even sacrificed in craniofacial tumor removal, such as acoustic neuromas. Rinaldi (2012) reported a 38.7% incidence of postoperative facial nerve deficit after acoustic neuroma surgery, besides anatomical preservation of the FN.[15] Total ipsilateral facial weakness, decreased tearing, hyperacusis, associated defects with V, VI, VIII, and Horner syndrome occur classically post-surgery of tumors in the cerebellopontine angle.[8] According to Hohman (2014), the most common operation resulting in FN injury was temporomandibular joint replacement.[6]

Neoplastic

FNP may result from tumors themselves, either through direct compression, significant stretching or infiltration of the nerve. Cerebellopontine angle lesions may cause multiple cranial nerve defects or affect the CN VII in isolation. Matthies and Samii found that up to 17% of patients with acoustic neuroma had signs of FN dysfunction prior to tumor resection.[1,16]

FNP is the most frequent neurological presentation of sarcoidosis. There is usually a bilateral, asymmetric involvement of the parotid gland.[17,18]

Clinical presentation (Table 1)

Idiopathic

BP typically presents with sudden and rapid onset of unilateral facial weakness, often within a few hours. Sixty percent of patients report preceding viral illness. The motor deficit is almost always unilateral, with both the upper and lower parts of the face affected.[3] There is often drooping of the eyebrow, corner of the mouth and loss of the ipsilateral nasolabial fold. It is an isolated mononeuropathy and the association with other CN palsies should alert the examiner to other causes.

Bell's phenomenon or the upward movement of the eye on attempted closure of the lid due to weakness of the orbicularis oculi is usually present.[3]

Maximal weakness presents after 2 days and resolution in 3–4 weeks. Patients may also complain of ipsilateral earache, numbness of the face, tongue and ear.[3] Cases of hyperacusis, tinnitus, taste disturbances and decreased lacrimation have also been reported.[3]

Ramsay–Hunt syndrome

RHS is a clinical diagnosis based on unilateral facial weakness plus vesicular lesions in the ipsilateral ear, hard palate or anterior 2/3 of the tongue. Otalgia or vertigo complete the triad. Lesions are not required for diagnosis and around 2–35% of unilateral FNP without vesicles are actually *herpes zoster sine herpete*.[4] Murakami observed that auricular vesicles appeared before (19.3%), during (46.5%), or after (34.2%) the onset of FNP.[19]

Zoster patients (88%) have a high incidence of complete paralysis and have a more severe painful paralysis associated with vestibulocochlear symptoms at onset.

Figure 1. Central FNP due to ischemic vascular stroke. Only the lower part of the left face is affected.

General evaluation

Initial evaluation should include accurate clinical history,[1] followed by a comprehensive examination,[2] establishing whether the FNP is acute or chronic, unilateral or bilateral, exclude secondary causes and differentiate between proximal and distal lesions.

Clinical history

The clinician should inquire about underlying medical problems that could predispose to FNP, such as prior stroke, brain tumors, skin cancers of the head or face, parotid tumors, facial/head trauma, or recent infections.

Symptoms such as dizziness, dysphagia, or diplopia suggest a diagnosis other than BP.

The timing of onset remains important. Symptoms associated with neoplastic or infectious causes of FNP often progress gradually.[20]

Recurrence of FNP occurs in BP, tumors and Melkersson–Rosenthal syndrome. Alternating side of the recurrence is seen more with BP and ipsilateral recurrence implies tumor until proven otherwise.[9]

Physical examination

A thorough examination of the head and neck, including ophthalmological, otological, oral and neurological examination should be performed.

Ophthalmologic examination

Baseline visual acuity, pupillary reactions, Schirmer's test (if pathological, it locates the impairment upstream from the origin of the great superficial petrosal nerve or along its path) and optic disc evaluation.[1]

Evaluation of sensitivity of the cornea: absence of ipsilateral corneal reflex is an early sign of cerebellopontine angle syndrome.

Otological examination (including otoscopy)

Parotid swelling may suggest a malignant or inflammatory mass. Vesicles around the ear suggest RHS. Inflammation or pus from the ear may indicate malignant otitis externa. Tenderness of the mastoid may suggest infection spreading from the middle ear.

Oral examination

The sensation of taste of the anterior two-thirds of the tongue and the function of salivary glands are not frequently tested. Ask the patient about it or place small amounts of sugar, salt, vinegar and quinine for the evaluation.

Neurologic examination

Assessment of all cranial nerves

Ocular movements: ipsilateral VI nerve palsy may suggest a pontine lesion.

Evaluation of FN includes inspection of the face at rest, during speech, voluntary, emotional and involuntary movements with characterization of the overall movement of the face, the extent of the facial weakness and the involvement of all nerve branches.

Inspection at rest

Evaluate the upper eyelid retraction, the blink reflex and the paralytic ectropion.

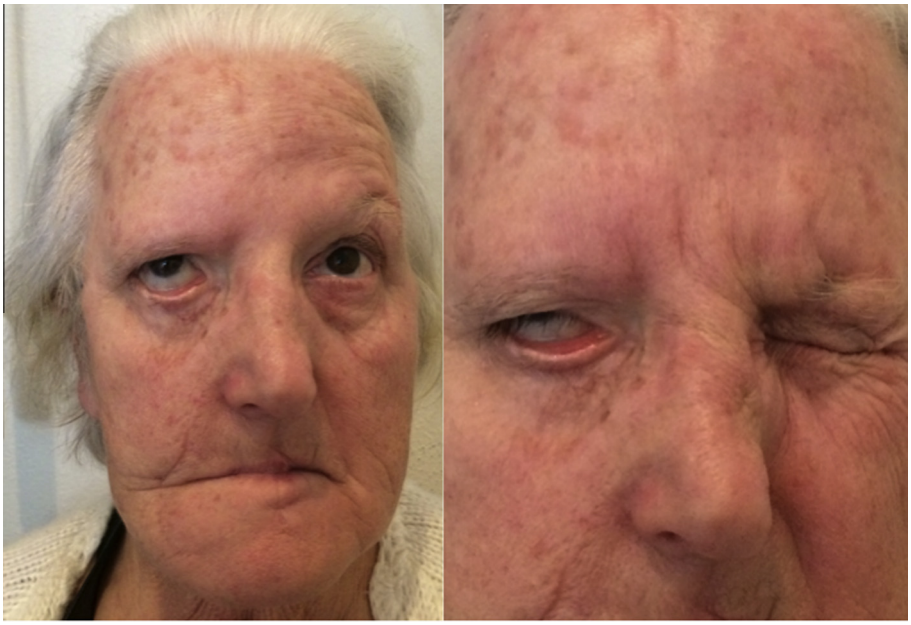

Figure 2. Peripheral right FNP with both upper (absence of frontal wrinkles) and lower parts of the face affected. The picture shows lagophthalmos with medial canthal paralytic ectropion. Bell's phenomenon is present.

Voluntary movements evaluation

Ask the patient to wrinkle the eyebrows. Evaluate the eyebrow position and elevation. In central palsy the frontalis is spared, whereas in peripheral palsy, upper and lower portions of the face are affected (Figs. 1 and 2).

Ask the patient to show the teeth. If a lesion of the FN is present on one side, the mouth is distorted. A greater area of the teeth is revealed on the side of the intact nerve (Fig. 1).

Raise the patient's eyelids while asking to close the eyes. If the orbicularis oculi is paralyzed, the eyelid on that side is easily raised. Look for Bell's phenomenon (Fig. 3).

Evaluate the lagophthalmos on gentle and forced closure (Fig. 2).

The House–Brackmann (HS) grading system (Table 2) helps to document the degree of FNP and to predict recovery. It is the most widely used and accepted.

Figure 3. Testing for Bell's phenomenon in a patient with FNP after acoustic neuroma resection.

Laboratory testing

Clinicians should not obtain routine laboratory testing in patients with new-onset BP. However, it may be indicated in patients with identifiable risk factors or atypical presentation. In endemic areas, Lyme disease serology should be considered.[20]

Diagnostic imaging

Imaging is not systematic with peripheral FNP, although it is with central FNP.[10,19] Almost all patients with BP regain some function within 3 months of onset. Any case without resolution within 4 months should undergo contrast-enhanced imaging of the parotid gland, temporal bone and brain. Repeat imaging is indicated if symptoms persist at 7 months without a readily identifiable cause. Biopsy of the affected tissue adjacent to the FN may be considered if imaging is negative at 7 months.[3] Imaging is needed if there is progression or recurrence of BP or if there is association with other CN palsies.[10]

Management

Many factors are involved in the treatment decision-making process of patients with FNP: the underlying cause, expected duration of nerve dysfunction, anatomical manifestations, severity of symptoms and objective clinical findings.[14]

Steroids and acyclovir

Nerve conduction does not become abnormal until 3 days after the onset of paresis when nerve degeneration develops. The goal of medical therapy is to treat patients within this 3-day window.[1]

Updated Guidelines of the American Academy of Neurology (AAN) state that systemic steroids should be offered to

Table 2. House–Brackmann grading system.

Grade	Description	Characteristics
I	Normal	Normal facial function
II	Mild dysfunction	Gross: slight weakness noticeable on close inspection, may have very slight synkinesis. At rest: normal symmetry and tone. Motion: forehead- moderate to good function, eye-complete closure with minimum effort, mouth-slight asymmetry
III	Moderate dysfunction	Gross: obvious but not disfiguring difference between the two sides; contracture and/or hemifacial spasm. At rest: normal asymmetry and tone. Motion: forehead- slight to moderate movement; eye- complete closure with effort; mouth- slightly weak with maximum effort
IV	Moderately severe dysfunction	Gross: obvious weakness and/or disfiguring asymmetry. At rest: normal asymmetry and tone. Motion: forehead-none; eye- incomplete closure; mouth: asymmetric with maximum effort
V	Severe dysfunction	Gross: only barely perceptible motion. At rest: asymmetry. Motion: forehead-none; eye- incomplete closure; mouth- slight movement
VI	Total paralysis	No movement

patients with new-onset BP to increase the chance of FN recovery (Level A recommendation).[21] A recent Cochrane review found that only 23% of patients treated with corticosteroids had incomplete recovery of facial motor function at 6 months, compared to 33% of patients treated with placebo.[22] In addition, patients receiving corticosteroids had a significant reduction in motor synkinesis.[3,22]

HSV infection is believed to be the main cause of BP; it would then be reasonable to include acyclovir in its treatment.[1] However, a Cochrane systematic review concluded that antivirals provide no significant benefit over placebo in generating complete recovery from BP.[23] According to the AAN, patients with new-onset BP, might be offered antivirals (in addition to steroids) (Level C), because of the possibility of modest increase in recovery.[21]

In RHS, a Cochrane review of the sole randomized controlled trial comparing combined treatment to corticosteroids alone showed no significant difference in outcomes.[24] The largest RHS treatment study was a retrospective analysis of 80 cases. Patients treated with acyclovir–prednisone within 72-h of symptoms onset had a complete recovery rate of 75% *vs* 30% of patients treated after 7 days. Early administration also reduced nerve degeneration.[4,25] Acyclovir 800 mg 5 times daily or 1 g valacyclovir TID for 7–10 days plus prednisone 1 mg/kg for 5 days and taper was used in published trials.[4]

Surgery for FN decompression

Surgical decompression remains highly controversial due to its risks and should be considered in refractory cases.[3]

There is some evidence in favor of surgical decompression with documented loss of >90% of axonal fibers on the electroneurography prior to day 14 of weakness onset.[1,26] Hato et al. decompressed the tympanic and mastoid segments of the canal with concurrent placement of basic fibroblast growth factor impregnated biodegradable gelatin hydrogel around the nerve. The rate of complete recovery was 75% compared to 44.8% by the conventional decompression method and 23.3% on the steroids alone.[14]

Corneal exposure and lagophthalmos

On FNP patients, the cornea is especially at risk because of improper lid closure due to lagophthalmos and paralytic ectropion and due to decreased tear production and distribution of the tear film. Some works also demonstrated increased meibomian gland dysfunction.[27,28]

Treatment directed at protecting the cornea depends on the degree of nerve lesion and of the risk of corneal damage based on the amount of lagophthalmos, the quality of Bell's phenomenon and the presence or absence of paralytic ectropion.[8,9] Furthermore, corneal sensation testing should be performed, as patients with both V and VII palsies are at increased risk of developing corneal decompensation.[29] Neurotrophic corneal epithelium is more prone to injury, heals poorly and patients may be unaware of the corneal damage.

The goals of therapy are to protect the cornea and to restore the blink response using minimal intervention and maintaining good visual acuity.[1]

Temporary treatment

If recovery is expected, less invasive techniques should be employed.

When there is low corneal risk and good prognosis for recovery, intensive lubricants and taping the lid with a stiff tape overnight will usually be enough.[8] Preservative-free teardrops during the day and a more viscous ointment overnight may be used. Moisture chambers act as barriers to evaporation.[9]

Scleral contact lenses were recently described as a valid alternative to tarsorrhaphy for patients with corneal exposure and anesthesia, providing effective protection in an esthetically acceptable way and optimizing visual function.[30,31]

Botulinum toxin injection induces ptosis by temporarily paralyzing the levator palpebrae superioris and thus protecting the cornea. It is an excellent low-risk temporary (effect sustained for a mean of 46 days) alternative for postoperative high grade FNP, when the FN is anatomically intact.[32] In 16 of 21 patients further surgical intervention was avoided and corneal healing was obtained. Significant improvement in corneal symptoms and decreased use of artificial tears was also reported.[14] However, it affects patients' vision and may provide less than adequate protection as the levator function returns.[8,9]

Temporary tarsorrhaphy can be achieved with a simple suture or cyanoacrylate glue. The classic central tarsorrhaphy is cosmetically and visually poor, but gives good protection. Lateral tarsorrhaphy may not adequately close the eye, particularly if there is significant lower lid ectropion.[8]

External eyelid weights have similar design to those for implantation and are fixed to the pretarsal skin surface with double-sided hypoallergenic adhesive tape.[9]

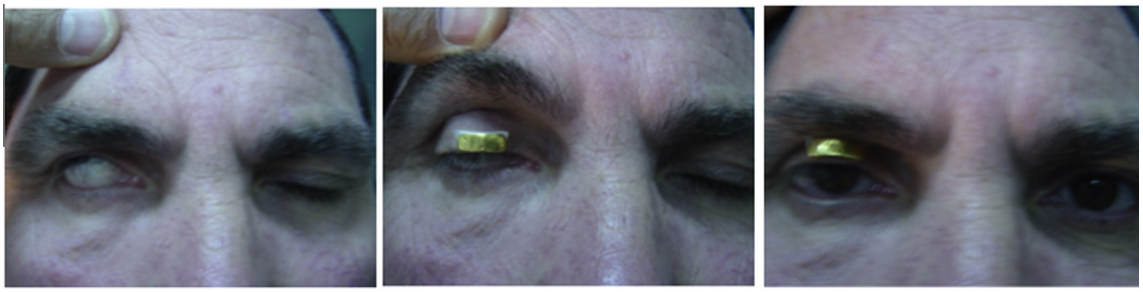

Figure 4. Gold weight trial procedure. Gold weight reduction of the lagophthalmos, without inducing significant upper eyelid ptosis in the primary position.

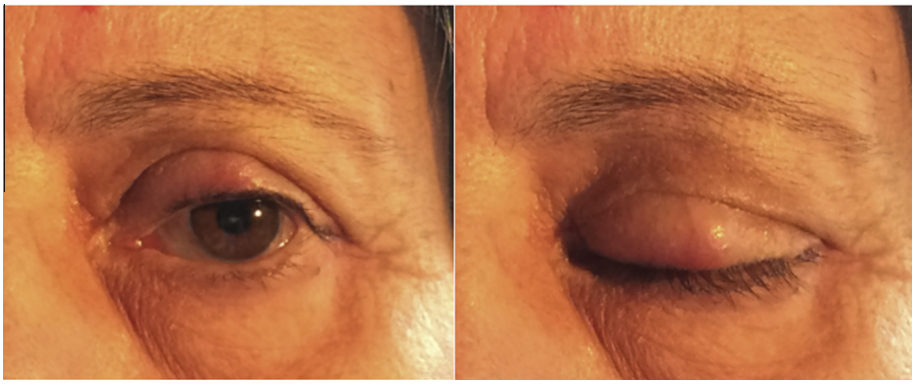

Figure 5. Upper eyelid load gold weight combined with lateral tarsorrhaphy. Complete eyelid closure was achieved, without inducing significant ptosis.

Silicone punctal plugs can be used in patients who cannot be satisfactorily managed with lubricants or have decreased tear production.[9]

Permanent treatment

When no recover of nerve function is expected, the long-term protection of the cornea is more complex and depends on the degree and manner in which the upper and lower lids are affected. The palpebral aperture can be closed in four ways: (1) by lowering the upper eyelid, (2) raising the lower eyelid, (3) medial and (4) lateral closure.

(1) An upper eyelid load weight provides passive lid closure and increased blink response, along with lowering of the retracted upper eyelid. It has been shown to significantly reduce lagophthalmos, improve corneal coverage and decrease lubricant-dependence. Lid loading with 99.9% pure gold is the most commonly performed surgery for FNP of any etiology.[8] It is quite simple to place, has a very low complication rate,[29] is equally effective in early and later stages and if the nerve function improves it is easy to remove.[8] Advantages over tarsorrhaphy are better cosmesis and maintenance of binocular visual field. The standard gold weights range from 0.6 g to 1.6 g in 0.2 g increments. The success of gold weight implantation depends on accurate prediction of the ideal gold weight for a given patient. Serial increments of trial gold weights are pasted to the pretarsal upper eyelid skin to assess the expected postoperative outcome (Fig. 4). An ideal weight would be one that achieves adequate reduction of the lagophthalmos without inducing significant ptosis. Hontanilla (2001) suggested a correction factor of 0.2 g to be added to the final weight before lid loading.[33] Aggarwal (2007) assessed the accuracy of the gold weight trial procedure in predicting the postoperative eyelid closure and concluded that it led to a 30% undercorrection and that a higher correction factor should be considered for patients with preoperative lagophthalmos higher than 8 mm.[34] Thin-profile platinum weights can be an alternative to the gold.[35] (Fig. 5).

An alternative to the gold weight implant is the less frequently used palpebral spring. This is the most commonly used method of dynamic eyelid animation. A custom-made stainless steel spring is implanted and secured to the superior orbital rim and pretarsal area. When the levator relaxes as the opposite eye closes, the spring actively pushes the eyelid down. The spring allows for a more rapid eyelid excursion and complete closure compared to gold weights and helps to restore corneal squeegee effect. However, erosion is a common problem that may require frequent adjustments.[36]

Upper eyelid retraction is common sequelae of FNP due to the unopposed action of the levator and to thixotropy (crossbridge formations between actin and myosin filaments causing stiffness of the levator muscle).[8] This should be addressed prior to considering lid loading. The choice of the procedure depends on the amount of retraction: mullerectomy is sufficient to treat 1–3 mm of retraction, but with larger amounts retractor recession transconjunctivally or with an anterior approach with or without a spacer material may be required.[8]

(2) When addressing problems of the lower eyelid, the choice of surgical procedure will depend on the degree of laxity or ectropion and state of the medial and lateral canthal tendons. Increased support to raise the lower lid can be achieved by combining medial and lateral canthoplasties.

When there is significant lower lid retraction, this can be combined with insertion of a spacer. In cases of marked tissue atrophy, an autogenous fascial sling can be threaded hammock-like through the entire length of the lid, anchored by fixation to the medial canthal tendon and lateral orbital periosteum.[8].

(3) The medial palpebral aperture closure depends on the laxity of the medial canthal tendon. A punctual ectropion can be treated with a medial canthoplasty or a medial tarsal strip. Where there is significant tendon laxity, a deep periosteal Royce-Johnston suture or medial wedge excision could be used.

(4) Permanent lateral tarsorraphy has largely been superseded by the lateral canthal sling.[8]. It has the advantage that it can be augmented by inserting the strip higher on the rim to assist in tear drainage or combining it with a small lateral tarsorraphy if the horizontal aperture needs to be shortened.[8]

Dynamic correction of paralytic lagophthalmos frequently involves transfer of the temporalis muscle, which is effective and can provide strong eyelid closure over and for an extended period of time. Reanimation of paralyzed muscles using adjacent motor nerves has also been attempted: Hayashi performed an hypoglossal-facial nerve anastomosis with excellent success rates[37]; Corrales used hypoglossal-facial nerve anastomosis to treat patients with central FNP with comparable success with those for peripheral dysfunction[38] and the masseter nerve can also be utilized.[13]

The use of permanent tarsorrhaphy has decreased since other rehabilitation procedures arise. It is most suited to cases with corneal sensory deficits. Despite being cosmetically and visually poor, for patients in whom medical therapy is difficult and lacrimal gland function is lost, it remains an important treatment option.

Other modalities of therapy

No evidence supports significant benefit from physical therapy or acupuncture for BP. Hyperbaric oxygen therapy might be effective on moderate to severe BP.[39–41]

Prognosis

Spontaneous, complete recovery of BP occurs in up to 70% of cases. Usually remission begins within 3–4 weeks, with complete recovery within 6 months. Peitersen found that, in 2570 cases of peripheral FNP, 85% of patients' function was returned within 3 weeks and in the remaining 15% after 3–5 months. In 71% normal mimical function was obtained, sequelae were slight in 12%, mild in 13% and severe in 4% of patients. Contracture and synkinesis was found in 17% and 16% of patients respectively.

Patients presenting with incomplete paresis show 93–98% of spontaneous complete recovery. Some factors that influence prognostic outcome in BP are shown in (Table 3). The recurrence rate of BP is about 12% of cases but multiple recurrences are rare.[3]

RHS has a less favorable recovery profile than BP: 21% return to normal function and 79% develop sequelae, 54% with poor recovery.

FNP after surgery: Pelaz (2008) evaluated the recovery to normal function after complete FNP secondary to acoustic neuroma surgery and found that only 16.6% achieved HB grade I. The majority presented HB grade III (33.3%) or IV (26.6%). Poor recovery was associated with a tumor size bigger than 2 cm, males, age >65 years and lesions resected by the translabyrinthine approach.[42] Rinaldi (2012) reported a long-term facial deficit after surgery of 37.1%.[15]

Table 3. Negative prognostic factors that influence outcome in Bell's palsy.

Prognostic factor	Value
Pain or altered taste	No evidence
Complete paralysis	Strong evidence
Age >60 years-old	Strong evidence
Minimal recovery by 3-weeks	Strong evidence
Pregnancy	Strong evidence (complete recovery 52%)
Nerve degeneration (electrophysiological testing)	Strong evidence
Diabetes	Some evidence
Hypertension	Some evidence

Complications, sequelae, synkinesis

Long-term complications can develop from BP. Yamamoto observed sequelae in 9.1%[43]; Kawai described an incidence of 19%[44] and Peitersen reported 29%.[1,5] It is more common in complete or nearly complete FNP.

When nerve fibers are damaged they may aberrantly regenerate. During regeneration, excessive collateral branching of the axons occur, not only at the site of the lesion but also along the entire course of the nerve. These extra-axons may contribute to the abnormal location of the regenerated axon in the facial nucleus and results in synkinesis. Hyperexcitability and ephaptic phenomenon were also implicated.[1] This can result in lacrimation while eating or crocodile tears (aberrant connections with the lacrimal ducts instead of the salivary glands) or involuntary uncoordinated muscle movement associated with voluntary movement of the muscle when regenerating motor neurons innervate inappropriate muscles.[3] Rarely, aberrant innervation may result between two adjacent cranial nerves such as facial-trigeminal and facial-oculomotor synkinesis.[1]

These abnormal movements can be more distressing than the FNP itself.[1] Botulinum toxin injection and facial reanimation are among the proposed methods of treatment.

Conflict of interest

The authors declared that there is no conflict of interest.

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