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Review Article

Reanimation following facial palsy: present and future directions

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Abstract

Background: An understanding of the management of chronic facial palsy is vital for otolaryngologists, due to its common presentation to ENT surgeons. There is currently a lack of consensus on the optimum management of this condition. This article reviews the existing literature and offers a perspective on current management, as well as an insight into future treatments.

Methods: A literature search was performed, using the Medline, Embase and Cochrane databases from 1966 to the present, using the keywords listed below. Articles were reviewed. Selection was limited to English language articles on human subjects.

Results and conclusion: A tailored, multidisciplinary approach using combinatorial therapy should be used for reanimation of the face following facial palsy. Advances in surgical and non-surgical techniques, and the exchange of information from centres of excellence via global databases, will enable objective appraisal of results and the development of an evidence-based approach to facial reanimation.

Key words: Facial Reanimation; Facial Palsy; Facial Weakness; Face Transplant; Dynamic Reanimation; Static Reanimation; Synkinesis

Introduction

Facial nerve palsy is a life-altering condition with profound aesthetic, functional and psychosocial implications. The optimum management is fraught with debate, due to the lack of objective, quantitative measures of facial weakness and reliable prognostic indicators for spontaneous recovery. Added to this is the inconsistency of rehabilitative outcomes in the existing literature.

It is important to fully understand the complexity of the involved pathological mechanisms, and the available interventions, in order to achieve the best possible outcome. This article presents a synopsis of management options for patients with facial palsy, and also summarises the clinical and scientific research that will shape the future direction of this evolving field.

Aetiology

Table I summarises the myriad conditions which may result in facial palsy. Bell's palsy is the commonest cause in adults. In neonates, birth trauma is the commonest cause and complete recovery occurs in the majority of cases.[2]

Clinical assessment and evaluation

Thorough clinical history-taking and examination should be undertaken to identify possible aetiological factors for facial palsy. A head and neck examination and neurological examination should be performed. Facial function should be evaluated in different regions of the face, both at rest and with active movement. Forehead sparing may suggest a central aetiology. Any synkinesis and dyskinesis should be recorded. Muscle weakness is graded using the House–Brackmann scale, due to its simplicity and fair inter-observer agreement[3] (although a variety of other regional and global grading systems exist).

Assessment of functional impairment is critical. Ocular sequelae are noted, including lagophthalmos and subsequent corneal exposure, lid retraction, and ectropion. Nasal obstruction from nasal valve collapse, speech deficit and oral incompetence relating to orbicularis oris weakness are recorded. The state of the facial musculature and location of the severed nerve stumps provide important information guiding rehabilitative options (e.g. donor nerve and muscle grafts).

Finally, the patient's expectations should be explored and possible rehabilitative options discussed.

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TABLE I

AETIOLOGY OF FACIAL PALSY

Idiopathic
Bell's palsy
Melkersson–Rosenthal syndrome
Autoimmune syndrome
Traumatic
Temporal bone fracture
Facial injuries
Barotrauma
Infective
Malignant otitis externa
Acute otitis media
Cholesteatoma
Ramsay–Hunt syndrome
Lyme disease
Leprosy
Mumps, encephalitis
Neoplastic
Acoustic neuroma
Cerebellopontine angle tumours
Glomus jugulare tumour
Parotid tumours
Iatrogenic
Surgery: temporal bone, parotid, facial
Post-immunisation
Mandibular block anaesthesia
Embolisation
Congenital & genetic
Forceps delivery
Mobius syndrome
Dystrophy myotonica
Toxic
Thalidomide
Alcoholism
Arsenic
Carbon monoxide
Metabolic
Diabetes mellitus
Hyperthyroidism
Hypertension
Acute porphyria
Vitamin A deficiency
Pregnancy
Nervous system
Opercular syndrome*
Millard–Gubler syndrome
Motor neuron disease
Guillain–Barré syndrome
Granulomatous
Sarcoidosis

Adapted with permission.[1,2] *Cortical lesion in facial motor area.

Commonly performed investigations are summarised in Table II.

Management of the paralysed face

Ideally, the management of facial palsy should be undertaken at a centre of excellence where multidisciplinary expertise is available. Any treatment of facial palsy must address the underlying cause. A combination of surgical, medical and physical therapy approaches are commonly used to reanimate the face. The functional goals are to protect the eye, restore the nasal airway, and re-establish oral competence. Cosmetic goals include creating improved balance and symmetry of the face at rest, and achieving coordinated movement of facial musculature.

In cases in which the nerve is transected, early surgical nerve repair is recommended. If the facial nerve is anatomically intact, one must weigh the chances of potential spontaneous recovery against the risk that delayed innervation will result in a poorer outcome. It remains difficult to accurately predict which patients with an intact but paralysed nerve will recover function spontaneously.

The medical and surgical management of Bell's palsy, one of the commonest causes of facial weakness, has been debated for decades. Recently, Sullivan *et al.*[6] and Engstrom *et al.*[7] have demonstrated that oral prednisolone improves Bell's palsy outcomes when administered early, whilst antivirals are ineffective. The role of facial nerve decompression in Bell's palsy remains contentious, and is discussed below.

Management of the paralysed face: surgical reanimation

The surgical options for the management of facial nerve palsy can be divided into dynamic and static procedures (see Figure 1). Dynamic reconstruction aims to restore muscle movement, whilst static techniques stabilise the soft tissue structures. A combination of methods is generally employed because no single procedure is ideal in achieving all the desired goals.

The timing of facial nerve palsy repair may be divided into three phases: acute (i.e. less than three weeks), intermediate (three weeks to two years) and late (more than two years). For acute paralysis, the mainstay is facial nerve decompression and facial nerve repair. Intermediate and chronic paralysis usually require nerve transfer procedures and muscle transfer (free and regional), respectively. Static reanimation (described below) is a useful adjunct during all phases of nerve palsy (Figure 1).

Dynamic reanimation

Facial nerve decompression. Facial nerve decompression for traumatic as well as Bell's palsy has been well described, and has been recommended by Fisch when there is greater than 90 per cent degeneration of the facial nerve compared with the contralateral side.[4,5] A recent study showed that surgical decompression based on poor electroneuronography profiles improved the chances of near-normal recovery of facial function, if carried out within two weeks of onset of paralysis.[8]

In traumatic cases, immediate surgical decompression is useful, e.g. evacuation of haematomas and excision of impinging fractured bone surrounding the facial nerve.[9,10] In their large series, Darrouzet *et al.*[11] demonstrated operative management to be most successful in patients with total or immediate facial nerve paralysis as well as those with poor prognostic electromyography (EMG) patterns.

Electrophysiological test results should be used with caution when estimating the prognosis for return of facial function, as illustrated by Brodsky *et al.*[12] This study also illustrates the role of delayed

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TABLE II

COMMON INVESTIGATIONS FOR FACIAL PALSY

Investigation	Possible diagnoses
Haematological	Lyme disease, syphilis, HIV
Radiological	MRI: cerebellopontine angle lesion
	CT: temporal bone fracture, cholesteatoma
Audiography & vestibular testing	SNHL may indicate retrocochlear pathology
	Conductive hearing loss may indicate middle-ear lesion
Schirmer's lacrimation test & taste test	Topognostic test to localise lesion
Electrophysiological	(1) To estimate degree of nerve injury during acute phase
	(2) To provide prognostic indicator for spontaneous recovery
– Electroneuronography	Measures evoked CMAP, corresponding to number of electrically excitable nerve fibres
	Useful 3–21 days post-injury (not performed earlier as Wallerian degeneration not yet present and function may be overestimated)
	CMAP compared with unaffected side of face
	In Bell's palsy, degeneration to less than 10% function (compared with contralateral side) is considered significant and marks threshold for possible surgical exploration[4,5]
– Electromyography	Measures volitional muscle response
	Useful 3 wks post-paralysis to assess denervation (fibrillation potentials), reinnervation (polyphasic potentials) and viability of facial musculature for reinnervation procedures
Muscle biopsy	Assesses muscle viability
Photos, videos	Pre- & post-operative documentation of facial nerve recovery

HIV = human immunodeficiency virus; MRI = magnetic resonance imaging; CT = computed tomography; SNHL = sensorineural hearing loss; CMAP = compound muscle action potential; wks = weeks

nerve decompression in the management of facial palsy, contradicting previous reports.[13]

Facial nerve repair. In cases of facial nerve transection, immediate restoration of continuity provides best results. The key to success is achieving a tension-free anastomosis and careful epineural repair.

The facial nerve can be repaired directly (if tension-free coaptation can be achieved) or using a cable nerve graft (commonly using the sural nerve or great auricular nerve). Spector *et al.*[14] have shown that cable nerve grafting can lead to poorer functional recovery, i.e incomplete reinnervation of facial divisions, decreased voluntary contractions and more severe synkinesis, compared with direct end-to-end anastomoses. Despite this, the cable graft results of this study[14] were encouraging, with 92–95 per cent of patients experiencing some return of facial function, and 72–79 per cent achieving good functional results.

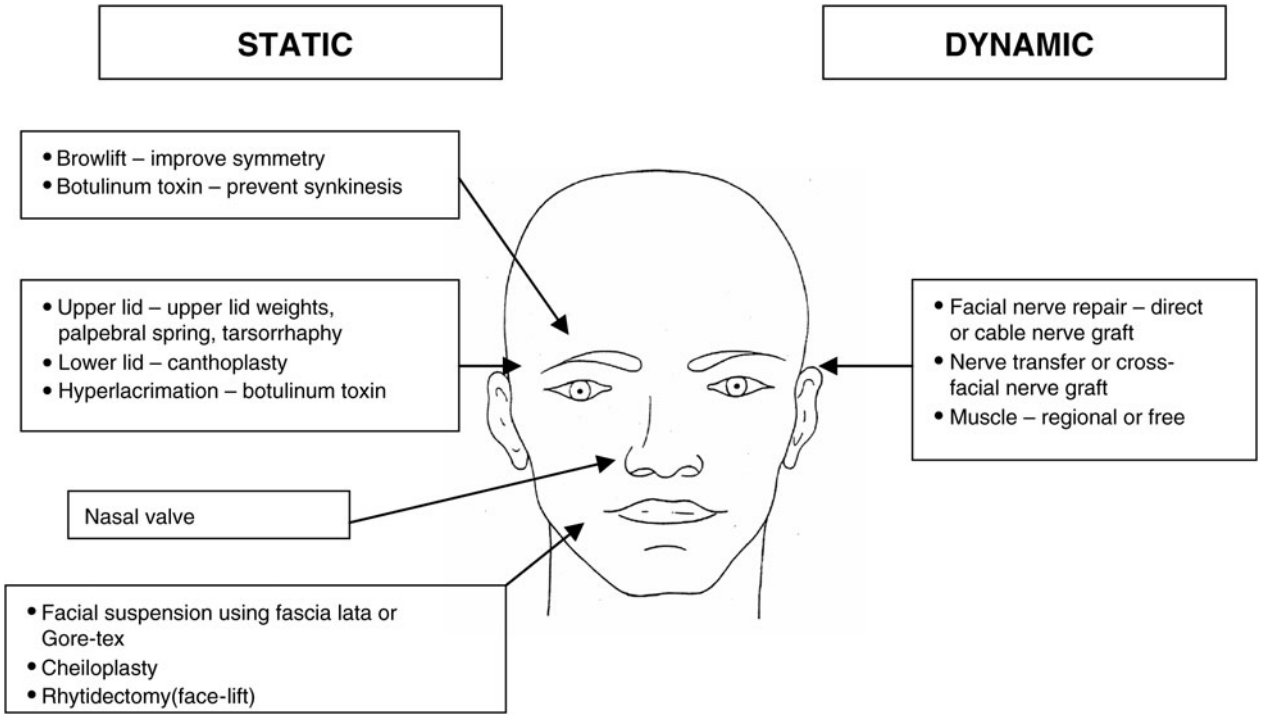

FIG. 1

Summary of facial reanimation options.

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Primary repair and nerve grafting proximal to the stylomastoid foramen are not generally recommended, as they can lead to significant synkinesis and dyskinesis due to the poor fascicular topography.[15]

Nerve transfers and cross-facial nerve grafting. Nerve transfers and cross-facial nerve grafting have a role when the proximal stump of the facial nerve is unavailable and the peripheral system is still viable, e.g. following skull base surgery or resection of an acoustic neuroma.

For nerve transfers, a variety of donor nerves (e.g. spinal accessory nerve, masseteric branch of the trigeminal nerve and motor branches of the cervical plexus) can be coapted to the distal stump of the facial nerve; however, the hypoglossal–facial nerve (XII–VII) anastomosis, popularised by Conley and Baker,[16] remains the most popular technique.

There are two options for nerve transfer: either complete or partial transection of the hypoglossal nerve. Classically, complete transection of XII and coaptation to VII resulted in significant speech and swallowing difficulties.[17] Incising the XIIth nerve distal to the descendens hypoglossi and bridging the gap between the XIIth and VIth nerves with an interpositional jump graft have been shown to preserve tongue function in 87 per cent of patients. However, a weaker facial response and a longer recovery time (up to two years) were observed due to axonal loss and fibrosis resulting from multiple anastomosis.[18] In a further modification to preserve tongue function, the XIIth nerve was split longitudinally and approximated to the VIIth nerve.[19] Recovery of facial function following the use of half of the hypoglossal nerve is reportedly equivalent to that expected after routine hypoglossal–facial anastomosis.

Unlike the above methods, cross-facial nerve grafts can enable more symmetrical movement. Originally developed by Scaramella[20] and Smith,[21] this technique involves anastomosing the redundant facial branches on the normal side to branches on the paralysed side, often employing an interposition graft (e.g. involving the sural nerve or great auricular nerve). This approach is usually utilised in combination with other techniques such as free muscle transfers, with input to the transplanted muscles from the contralateral facial nerve ensuring symmetrical movement.

Muscle transfer. As muscle atrophy sets in, in cases of longstanding paralysis, the focus of reanimation shifts from reinnervation of native muscles to the use of alternative muscles for reanimation, for example via regional or free muscle transfer.

In the former approach, the temporalis muscle (and to a lesser extent the masseter and digastric muscles) is commonly employed to achieve dynamic reanimation of the lower face, due to its favourable smile vector pull.[22] Not only is recovery of facial movement almost immediate, this method is easier to perform compared with facial nerve repair. This may be highly desirable in select patients, e.g. those in poor health, patients with a poor prognosis or those who want immediate results. Various modifications have been introduced (e.g. fascial extensions (to the upper and lower lip)[23] and temporalis tendon transfer)[24] to limit the depression left in the temporal fossa as well as the bulge resulting from the transposed muscle. The masseter muscle is not usually employed to reanimate the lower face, due to the inferior smile created by its lateral vector of pull.

For individuals with paralysis of the depressors of the lower lip, owing to isolated palsy of the marginal mandibular nerve, digastric muscle transposition has been shown to improve symmetry at rest and on attempted smile. Conley *et al.*[25] reported that 33 of 36 patients had satisfactory results with this technique.

Dramatic advances in the field of facial reanimation were made with the introduction in 1976 of microneurovascular free muscle transfer, by Harii *et al.*[26] Free muscle transfer not only provides soft tissue coverage of a defect, but also offers the opportunity to achieve involuntary, mimetic facial movements.

Free muscle transfer is usually performed as a two-stage procedure, the first stage involving a cross-facial nerve graft. The second stage, six to nine months later, involves microneurovascular transfer of the free flap (usually the gracilis muscle, and less often the serratus anterior, latissimi dorsi and rectus abdominis). Movement is typically detected by six months, but may take one year or longer. In cases of bilateral facial paralysis (e.g. Mobius syndrome) where there is no cross-facial nerve grafting available, the gracilis muscle transfer is performed in one stage and the masseteric branch of the trigeminal nerve is used to drive the gracilis muscle.[27]

Static reanimation

The regions of the face affected by facial palsy are commonly divided into upper (i.e. forehead, eyebrow and eyelids), middle (nose) and lower (cheek, lip) zones. Static reanimation procedures provide both cosmetic and functional benefits.

Static reanimation is suitable in older, debilitated patients who cannot undergo more extensive procedures. It is also used to provide a temporary lift whilst waiting for nerve regeneration to occur, and in cases of chronic paralysis, either as an alternative to, or to enhance the results of, dynamic reanimation.

The various static techniques are described below.

Eye. Lagophthalmos (i.e. incomplete eye closure) and paralytic ectropion (in which the lower eyelid turns outwards) are complications of facial palsy. Inadequate management of these can lead to excessive corneal exposure, ulceration and blindness.

Lagopthalmos has been traditionally managed by tarsorrhaphy, but this leads to diminished visual fields and an unpleasant cosmetic result, and may fail to protect the cornea. Tarsorrhaphy has since been supplanted by upper eyelid loading procedures using gold or platinum weights. Thinner profile platinum weights are becoming increasingly popular, as they offer a better cosmetic outcome and a decreased incidence of allergy compared with gold implants.

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Lower lid ectropion may be corrected by medial and lateral canthoplasty and autogenous tissue implants to support the lower lid.[28]

Hyperlacrimation and synkinesis are also common sequelae of facial nerve palsy. A recent study by Boroojerdi *et al.*[29] found that injection of botulinum toxin into the lacrimal glands was highly effective in treating hyperlacrimation. Synkinesis can be reduced by local injection of botulinum toxin into the orbicularis oculi.[29]

Nose. Nasal collapse and obstruction results from paralysis of the dilator nasi. To remedy this, nasal lateralisation procedures can be performed either internally or externally.

External procedures involve suspending the lateral nasal ala from the cheek using strips of suspension material placed under the skin. The tension on the lateral nasal ala opens the internal valve, thus relieving airway obstruction. Paniello[30] and Friedman *et al.*[31] have shown an orbital suspension technique modified to correct collapsed nasal valves to be highly effective. The nasal cavity can also be widened or stiffened internally by placing cartilage in the nose framework; this prevents alar collapse during inspiration. The nasolabial fold may also be modified to correct effacement or overprominent folds, using a simple suture technique.[32]

Mouth. Rehabilitation of the mouth aims to create a competent oral commisure, through the use of a lower lip dam that prevents saliva leakage from the mouth. Drooping of the oral commisure, due to paralysis of the orbicularis oris as well as decreased lip tone, can result in asymmetry which is both aesthetically and functionally problematic.

Static suspension of the corner of the mouth can be achieved through the standard sling technique, which involves suspension of the orbicularis oris muscle to either the zygomatic arch or the orbital rim using strips of autologous or alloplastic material. Lip suspension can be achieved via cheiloplasty, which resects the redundant paralysed lip and replaces it with normal orbicular and lip tissue from the unaffected side. Wedge resection, lip flap transposition and Z-plasty tissue repositioning have all been employed. These methods (or a modification thereof) rotate innervated lip tissue from the contralateral side, resulting in partial reestablishment of a dynamic sphincter. Conley[33] performed this procedure with both dynamic and static techniques, and reported improvement in speech, eating, commissure competence and aesthetic appearance.

Aesthetic rehabilitation of the face. Aesthetic procedures such as rhytidectomy, blepharoplasty, brow lifts and facial slings are useful as adjuncts in patients who refuse, or are unsuitable candidates for, dynamic reanimation.

Brow lift is effective in treating asymmetry of the brows resulting from facial paralysis. In some patients, residual hyperactivity of the frontalis muscle on the normal side creates brow asymmetry which is only partially corrected by a brow lift. Injection of botulinum toxin into the non-paralysed side can reduce facial asymmetry by weakening the normal side. Botulinum toxin is most effective in improving symmetry when used in the forehead or the corner of the mouth. Botulinum toxin was initially introduced to treat patients with hemifacial spasms and hyperkinetic blepherospasm following Bell's palsy; its use has now been extended to selective medical neurolysis, as well as treatment of synkinesis and hyperlacrimation after Bell's palsy.[34]

A rhytidectomy (face-lift) can also improve facial asymmetry, especially at the corner of the mouth. More advanced face-lift techniques, such as deep plane face-lift, usually provide longer-lasting, more effective results.

Management of the paralysed face: non-surgical measures

Corrective make-up

The effective use of cosmetics can change a person's appearance and create different impressions using optical illusion. Lack of forehead wrinkling, and the presence of a temporalis muscle bulge or depression, can be masked by an appropriate hairstyle, facial hair (for men) and/or make-up. Asymmetrical eyes can be hidden behind tinted glasses. Irregular or paralysed lips can be corrected with dental prosthesis or facial hair. In addition, the use of contrasting colours and items of jewellery can distract the eye from facial irregularities.

Physical therapy and biofeedback

The role of physiotherapy in neuromuscular re-education of the paralysed face is well established. Neuromuscular re-education is the process of relearning facial movements (to facilitate intended facial movement patterns whilst suppressing unwanted ones) using specific and accurate feedback. This technique may play several vital roles following dynamic muscle transfer: translating visible movement into a spontaneous, controlled smile; preventing synkinesis; and offering patients the opportunity to explore a conservative option for recovering facial movement.

The provision of feedback, via mirrors, surface EMG and video self-modelling, has been shown to result in improved overall symmetry and reduction in synkinesis.[35,36] A recent randomised, controlled trial of mime therapy (consisting of massage, relaxation, inhibition of synkinesis, and coordination and emotional expression exercises) showed improved facial symmetry and reduced severity of paresis in patients with facial nerve paresis.[37]

Future directions

Facial reanimation techniques are constantly being developed and refined. Of paramount importance is the establishment of an objective, quantitative facial function test or 'face-gram', which would allow comparison of different treatments, so that an

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evidence-based approach to facial reanimation can be adopted. A global database of these results (including facial palsy aetiology, face-gram scores, investigations and treatments) would allow robust appraisal of clinical management. Additionally, the emergence of centres of excellence that adopt a multidisciplinary approach, and improved cross-specialty communication, will also facilitate information exchange and thus help identify effective and ineffective therapies.

Modern surgical techniques focus on achieving involuntary mimetic motion, but also aim to reduce operative times, decrease scar length and improve the reliability of reanimation procedures. For example, the use of venous coupling for free tissue transfer has enabled reduced operative times[38] and also a reduced thrombosis incidence compared with traditional suture techniques, making this technique a useful tool in reanimation surgery.[38,39] Sural nerve harvesting has enabled a reduced scar length, through 'nerve stripping' and endoscopic techniques similar to those used for vein harvesting during coronary bypass grafting.[40,41]

The recent success of the allograft facial transplant represents another advance in the reconstructive process, especially for patients with disfigurement who cannot be helped by other methods of reconstructive surgery. In this technique, the skin, subcutaneous tissue and/or facial muscles are removed and replaced with similar tissue from a donor, with appropriate microneurovascular anastomosis. Apart from the surgical, ethical and psychological challenges of such a procedure, the need for post-operative immunosuppression (to prevent rejection of the new face) must be taken into account. Although this field is still in its infancy, it does open the door for facial transplants to be used for broader purposes. Chronic facial palsy with myofibrosis is one such case. The donor face can be anastomosed to the recipient's facial nerve (if viable) or transposed to adjacent nerves (e.g. the hypoglossal nerve), either directly or via a jump graft.

Laboratory research on neurotrophic factors and biological conduits will reveal more effective ways of bridging long neural gaps and accelerating facial nerve repair, whilst preventing axonal sprouting that may lead to synkinesis.

Conclusion

The key to successful facial reanimation is the use of a combination of methods to achieve facial symmetry, tone and motion. Unfortunately, surgery never restores the premorbid state, which is difficult for both patients and their clinicians. However, there has been tremendous progress in reanimation outcomes over recent decades, through continual improvement of existing techniques adapted from cosmetic surgery, and the use of emerging technologies. The future of facial reanimation will be shaped by advances in both clinical and basic laboratory research, leading to a better functional outcome for the individual patient.

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