A General Approach to Facial Palsy

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KEYWORDS

• Facial palsy • Facial reanimation • Facial nerve • Synkinesis

KEY POINTS

- Facial palsy is a devastating condition encompassing a spectrum of movement disorders that range from flaccid paralysis to postparalytic facial hyperactivity.
- Timing and selection of diagnostic and therapeutic interventions in facial palsy are critical.
- Therapeutic management may comprise medical therapy, surgical decompression, physical therapy, injectable fillers, and surgical reanimation procedures.

INTRODUCTION

Facial palsy (FP) is a devastating condition with functional and esthetic sequelae resulting in profound quality-of-life (QOL) impairment.[1,2] When acquired, the inciting insult typically results in acute flaccid facial palsy (FFP). Depending on the degree of neural injury, ultimate outcomes range from persistent and complete FFP to full return of normal function. In between these extremes exist zonal permutations of hypoactivity and hyperactivity and synkinesis, often with symptomatic gustatory epiphora and facial discomfort, a condition known as postparalytic facial nerve syndrome[3,4] which arises from aberrant regeneration of the facial nerve.[5,6] For clarity, a summary of pertinent definitions is provided in **Table 1**. This article provides a diagnostic and therapeutic management approach to FP.

HISTORY AND PHYSICAL EXAMINATION

It is incumbent upon the treating clinician to establish a diagnosis for the underlying cause of the facial movement disorder. Causes of acute FP include Bell palsy, Ramsay-Hunt syndrome (varicella zoster virus), Lyme disease, otic infections and cholesteatomas, postsurgical insult (eg, following vestibular schwannoma extirpation), benign tumors (eg, facial nerve schwannomas or venous vascular malformations of

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Table 1
Relevant definitions

Term	Definition
Facial palsy	Term encompassing entire spectrum of facial movement disorders, including flaccid facial palsy, facial paresis, and postparalytic facial palsy
Flaccid facial palsy	Complete or near-complete absence of facial movement and tone, without synkinesis or hyperactivity
Facial synkinesis	Involuntary and abnormal facial muscle activation accompanying volitional or spontaneous expression
Postparalytic facial nerve syndrome	Syndrome comprising facial synkinesis, facial muscle rigidity, spasm, contracture, or pain. Gustatory epiphora (also known as Bogorad syndrome or crocodile tears) is often present. The syndrome is thought to result from aberrant axonal regeneration or ephaptic transmission following facial nerve insult
Postparalytic facial palsy	Facial movement disorder of postparalyic facial nerve syndrome, comprising varying degrees of zonal synkinesis, hypoactivity, and hyperactivity

the facial nerve), or malignant tumors (eg, parotid or hematogenous primaries, regional spread of cutaneous malignancies, or solid tumor metastases), congenital malformations, systemic infections (eg, human immunodeficiency virus, syphillis), autoimmune conditions (eg, antiphospholid antibody syndrome, sarcoidosis, systemic lupus erythematosus, Sjogen's), granulomatous diseases (eg, Melkersson-Rosenthal syndrome, sarcoidosis), and trauma.[7] Although rare, pontine infarcts or hemorrhages may present with isolated FP.[8]

The time course of palsy onset, progression, prior therapies, resultant symptoms, and their impact on facial function and QOL are documented. A thorough history is invaluable in establishing the diagnosis; the clinician may inquire as to the presence of otovestibular symptoms (hearing loss, hyperacusis, vertigo, imbalance, otorrhea, otalgia), other focal neurologic deficits (eg, diplopia, facial anesthesia), constitutional symptoms (fever, chills, fatigue, malaise, sweats, weight loss), meningitic (headache, nuchal rigidity), and Lyme-specific symptoms (recent tick bite or exposure, erythema migrans rash, arthralgias, myalgias, low back pain), and inflammatory symptoms (eg, orofacial swelling or parotitis, uveitis) or known autoimmune conditions. In the setting of acute idiopathic FP, red flags suggesting a diagnosis other than Bell palsy include bilateral paralysis, slow onset of facial weakness (weakness in Bell palsy fully evolves over 24–72 hours), asymmetric weakness across facial zones at onset, constitutional symptoms (fever, lethargy, malaise, myalgias), headache (other than retroauricular pain and otalgia, which occur frequently in Bell palsy), presence of other focal neurologic deficits (diplopia, hearing loss, vertigo), and absence of recovery of facial tone within 4 months of palsy onset. Facial symptoms vary according to the timing of presentation and degree of recovery. FFP results in paralytic lagophthalmos and ocular irritation, loss of facial symmetry at rest, collapse of the external nasal valve, and oral incompetence. Postparalytic facial palsy (PFP) presents with facial synkinesis, muscle hyperactivity, contracture, and epiphora. Platysmal synkinesis results in neck discomfort and facial fatigue. Periocular synkinesis results in a narrowed palpebral fissure width. Lack of meaningful smile occurs in severe cases.

A thorough head and neck examination, including otoscopy and detailed cranial nerve examination, is performed. Zonal assessment of facial function at rest and with movement is crucial (**Fig. 1**). The brow position together with its effect on the

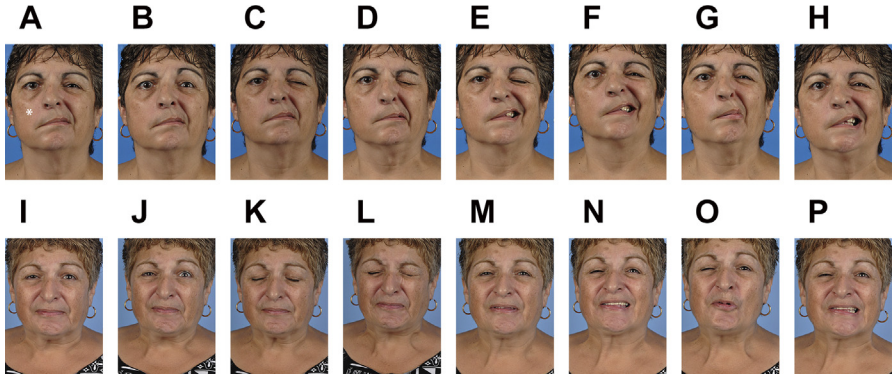

Fig. 1. Acute FFP (*top*) and subsequent PFP (*bottom*) in Ramsay-Hunt syndrome (varicella-zoster viral FP). Complete flaccid paralysis on the affected side (*asterisk*) is demonstrated at rest (*A*), and with brow elevation (*B*), gentle eye closure (*C*), full-effort eye closure (*D*), gentle smile (*E*), full-effort smile (*F*), lip pucker (*G*), and lower lip depression (*H*). The patient lacks Bell phenomenon (*C*, *D*). At 1 year following symptom onset, the affected brow remains depressed, while hyperactivity has developed in the orbicularis oculi, mentalis, and platysma muscles at rest (*I*). Volitional brow elevation remains impaired (*J*), while marked brow synkinesis is present with eye closure (*K*, *L*). As is usual in PFP, eye closure is adequate (*K*, *L*). Smile symmetry is improved with light effort (*M*); commissure restriction is noted with full-effort smile (*N*). Near normal return to function of the orbicularis oris muscle is noted (*O*). Lip depressor function remains weak on the affected side (*P*). Periocular, mentalis, and platysmal synkinesis is worsened by smile, pucker, and lip depression (*N*–*P*).

periocular complex is noted. The degree of paralytic lagophthalmos, presence or absence of Bell phenomenon, width of the palpebral fissure, and position of the lower lid are noted; laxity is assessed using distraction and snap-back tests. The depth and orientation of the nasolabial fold (NLF), position of the oral commissure, and presence and degree of brow, ocular, midfacial, depressor, mentalis, and platysmal synkinesis are evaluated. Attention is paid to the contralateral hemi-face with regard to whether weakening of a given paired muscle group, such as the hemi-brow or depressor labii inferioris, is likely to result in improved symmetry. Photography and videography to document appearance of face at rest and with 7 volitional facial movements (brow elevation, light- and full-effort eye closure, and smile, lip pucker, lower lip depression) on presentation and follow-up are essential (see **Fig. 1**). Spontaneous smile may be assessed by elicitation using humorous video clips.[9]

INVESTIGATIONS

When the history and physical examination are consistent with Bell palsy, further investigation is not required except in Lyme endemic areas, where serology is always prudent.[10–12] Imaging studies (such as a fine-cut computed tomography of the temporal bone without contrast, and/or gadolinium-enhanced MRI of the temporal bones and parotid gland) are indicated to rule out benign or malignant tumors affecting the facial nerve and should be ordered in the setting of abnormal otoscopy or tuning fork findings, palpable parotid or neck mass, slow- or asymmetric-onset or FP, slowly progressive FP, unilateral recurrent FP, or recent FP demonstrating absent recovery at 4 months. Blood work (such as complete blood count, erythrocyte sedimentation rate, C-reactive protein, rheumatoid factor, antinuclear antibody, antineutrophil cytoplasmic antibody, antiphospholipid antibodies, angiotensin converting enzyme) is

indicated in recurrent cases, or where autoimmune conditions are suspected. Electroneuronography (ENoG) is indicated between 3 and 14 days of symptom onset in patients who present with delayed traumatic or idiopathic FFP who demonstrate complete absence of hemi-facial movement on examination, to assess candidacy for acute facial nerve decompression.

THERAPEUTIC MANAGEMENT

Given the breadth of therapeutic options, management of FP can be daunting. It is useful to classify patients with FP into 1 of 5 management domains, based on timing of presentation, and status of the facial nerve and facial musculature (Fig. 2): acute FFP, FFP with potential for spontaneous recovery, FFP with viable facial musculature with low potential for spontaneous recovery, FFP without viable facial musculature, and PFP. Therapeutic strategies may include pharmaceutical agents, corneal protective measures, physical therapy (PT), chemodenervation agents, fillers, and a myriad of surgical procedures. Organization of potential interventions by type and side of FP and facial zone is valuable for developing a therapeutic plan (Fig. 3, Table 2).

Acute Flaccid Facial Palsy (Intact Facial Nerve)

This domain encompasses the first 72 hours to 2 weeks following onset of acute facial nerve injury. The role of the clinician is to establish a diagnosis, initiate appropriate medical therapy (such as immunosuppressant, antiviral, or antibiotic), manage exposure keratopathy risk, and determine candidacy for acute surgical intervention. In the setting of Bell palsy, administration of high-dose corticosteroids within 72 hours of symptom onset shortens recovery time.[13] Combined use of antivirals and corticosteroids in Bell palsy may be of additional clinical benefit, especially for those with severe to complete paralysis,[14,15] and good evidence supports combination therapy in VZV.[16] Delayed onset or incomplete FP following trauma or iatrogenic insult warrants corticosteroids and observation. Iatrogenic injury resulting in immediate and complete paralysis of one or more FN branches warrants urgent surgical exploration. Patients with complete idiopathic or posttraumatic paralysis with an ENoG response demonstrating greater than 90% degeneration, and absent voluntary motor units on electromyography (EMG) are referred for neurotology consultation for consideration of surgical decompression within 14 days of symptom onset.[14,17] Lyme disease–associated FP is treated with a prolonged course of oral doxycycline or intravenous ceftriaxone.[18] Although adjuvant corticosteroid therapy is commonly prescribed, its role in Lyme is unclear.[12,19,20] Otitis media–associated FP is treated with wide myringotomy with or without mastoidectomy, corticosteroids, topical and parenteral antibiotics.[21] Eye lubrication with nighttime taping of the eye closed is typically indicated to prevent exposure keratopathy. PT for education and instruction on upper eyelid stretching to aid passive closure may be of benefit. Correction of paralytic lagophthalmos may be achieved by temporary tarsorrhaphy or upper eyelid weighting; indications include poor prognosis for rapid recovery, inability to work due to ocular symptoms, inadequate Bell phenomenon, and absent recovery at 4 months.[22]

Flaccid Facial Palsy with Potential for Spontaneous Recovery

Where nerve continuity is thought intact in the setting of FFP, for example, following resection of a vestibular schwannoma where FN stimulation was noted before closure, a potential for spontaneous recovery exists, whereby return of facial tone and movement are expected within 6 to 12 months. Patients may benefit from PT, corneal protective measures, static periocular reanimation, and temporary chemodenervation of

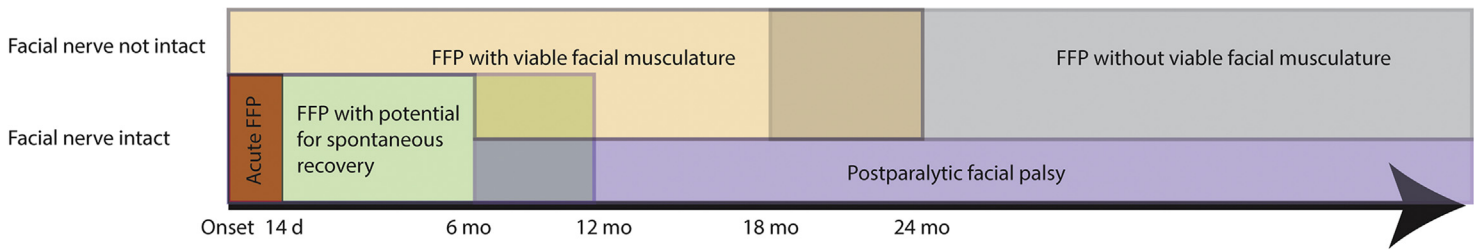

Fig. 2. FP management domains. Patients may be categorized according to timing of presentation from palsy onset, and status of the facial nerve and facial musculature. This conceptual framework is helpful in selecting appropriate therapeutic interventions. Medical therapy is often indicated in acute FFP. Close observation is indicated for a period of several months in FFP whereby there exists potential for spontaneous recovery (for example, following extirpation of a vestibular schwannoma with facial nerve preservation). Where the facial nerve is discontinuous, nerve repair or transfers (such as hypoglossal-to-facial and/or nerve-to-masseter to branches controlling smile) are immediately indicated; such transfers are also indicated in the case of persistent FFP following 6 to 12 months of observation because native facial musculature remains viable (ie, receptive to reinnervation) for a period of approximately 2 years. Where facial musculature is no longer viable (ie, absent or unreceptive to reinnervation), muscle transfers are indicated for smile reanimation. Patients with PFP (comprising synkinesis and varying degrees of zonal hypoactivity and hyperactivity) are typically managed with PT and chemodenervation; surgical reanimation is appropriate in severe cases.

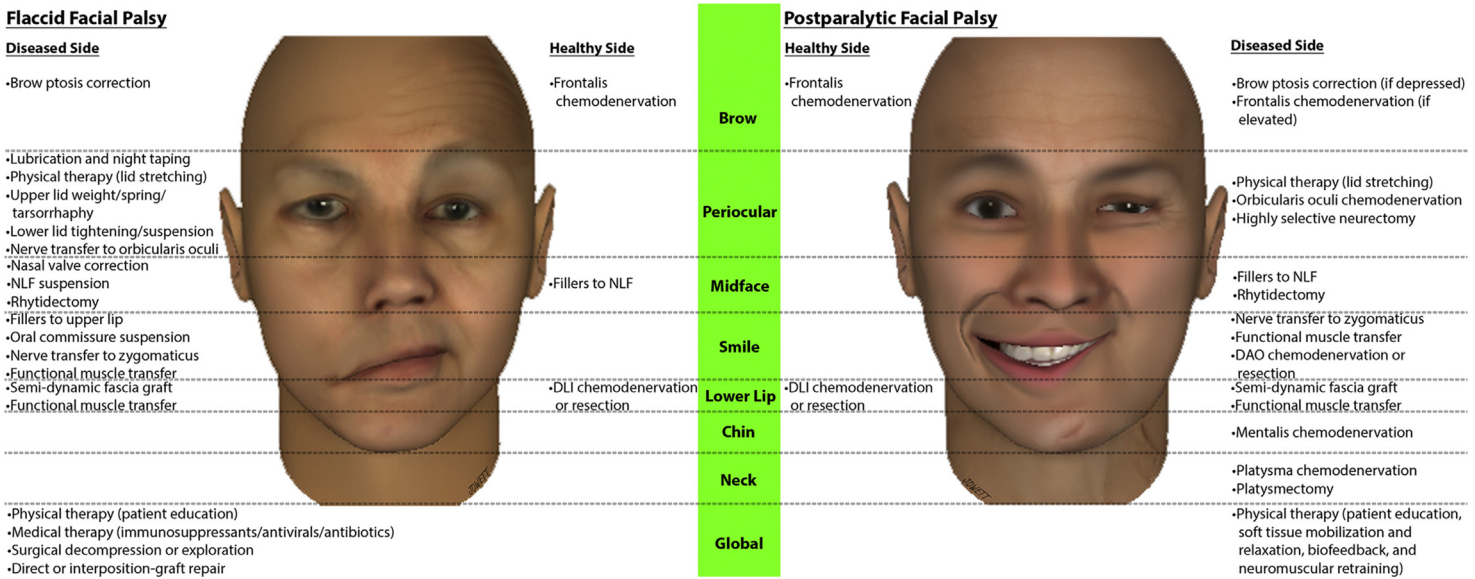

Fig. 3. Therapeutic options in FFP and PFP, by facial zone and side. A plethora of targeted therapeutic interventions may be used to restore balance and symmetry in hemi-FP.

Table 2
Therapeutic options in flaccid facial palsy and postparalytic facial palsy

Setting	Medical Management	PT	Injections	Surgical Management
Acute FFP (intact facial nerve)	• Corticosteroids: idiopathic (Bell), varicella zoster (VZV/Ramsay-Hunt), acute otitis associated, delayed traumatic, delayed iatrogenic • Antivirals: VZV, consider for idiopathic • Antibiotics (targeted): indicated for Lyme disease or acute otitis • Eye protection is always indicated ○ Daytime lubricating eye drops ○ Night time lubricating ointment, eyelid taping	• Patient education • Eyelid stretching	• None indicated	Adjunctive • Facial nerve decompression: indicated for idiopathic and posttraumatic complete FFP with ENoG response <90% and absent voluntary motor units on EMG between 3 and 14 d of symptom onset • Wide myringotomy ± tube placement ± mastoidectomy: indicated for acute otitis Static reanimation • Eyelid weight (reversible if recovery ensues)
FFP with potential for spontaneous recovery	• Corneal protection is always indicated ○ Daytime lubricating eye drops ○ Night time lubricating ointment, eyelid taping	• Patient education • Eyelid stretching	• Botulinum toxin ○ Contralateral brow ○ Contralateral depressor labii inferioris (DLI)	Static reanimation • Eyelid weight • Consider lower lid tightening in elderly patients
FFP with viable facial musculature and low potential for spontaneous recovery	• Corneal protection is always indicated ○ Daytime lubricating eye drops • Night time lubricating ointment, eyelid taping	• Patient education • Eyelid stretching • Targeted PT following dynamic reanimation	• Botulinum toxin ○ Contralateral brow ○ Contralateral DLI • Volumizing fillers ○ Contralateral NLF ○ Ipsilateral lips	Static reanimation • Brow ptosis correction • Eyelid weight • Lower lid tightening • External nasal valve correction • NLF suspension • Oral commissure suspension Dynamic reanimation • Direct end-to-end repair or interposition grafting (for facial nerve transections/ resections) • XII–VII for facial tone • Cross-facial nerve grafting or V–VII for targeted reanimation of expression (blink, smile)

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Table 2
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Setting	Medical Management	PT	Injections	Surgical Management
FFP without viable facial musculature	• Corneal protection is always indicated ◦ Daytime lubricating eye drops ◦ Night time lubricating ointment, eyelid taping	• Patient education • Eyelid stretching	• Botulinum toxin ◦ Contralateral brow ◦ Contralateral DLI • Volumizing fillers ◦ Contralateral NLF ◦ Ipsilateral lips	Static reanimation • Brow ptosis correction • Eyelid weighting and lower lid tightening • Static facial sling: external nasal valve, NLF, oral commissure • Rhytidectomy • Contralateral DLI resection Dynamic reanimation • Smile reanimation ◦ Temporalis or free muscle transfer
PFP	• Corneal protection if blink inadequate (rare) ◦ Daytime lubricating eye drops ◦ Night time lubricating ointment, eyelid taping	• Patient education • Eyelid stretching • Biofeedback • Neuromuscular retraining • Targeted PT following dynamic reanimation	• Botulinum toxin ◦ Contralateral or bilateral brow ◦ Ipsilateral orbicularis oculi ◦ Contralateral DLI ◦ Ipsilateral depressor anguli oris (DAO) ◦ Ipsilateral mentalis ◦ Ipsilateral platysma • Volumizing fillers ◦ Ipsilateral or contralateral NLF ◦ Ipsilateral lips	Static reanimation • Brow ptosis correction • Highly selective neurectomy • Ipsilateral rhytidectomy • Contralateral DLI resection • Ipsilateral DAO resection • Platysmectomy Dynamic reanimation • Smile reanimation ◦ Temporalis transfer or free muscle transfer

the healthy-side depressor labii inferioris muscle to improve oral competence and articulation during this period. Close follow-up (every 3 months) is warranted to ensure recovery of function.

Flaccid Facial Palsy with Viable Facial Musculature and Low Potential for Spontaneous Recovery

In this clinical scenario, there exists discontinuity of the facial nerve or absent recovery of facial function noted within 6 to 12 months of FP onset. Native facial musculature is intact and likely receptive to reinnervation. Common clinical scenarios involve patients presenting with dense FFP resulting from temporal bone tumors (such as facial nerve schwannomas, venous vascular malformations, or cholesteatomas), cerebellopontine angle tumor extirpations, or pontine hemorrhage. Although no definitive criteria exist, evidence from case series suggests that facial musculature remains receptive to reinnervation for periods up to 24 months following denervation in adults,[23–26] and possibly longer periods in children. Within this period, nerve repair and transfers are indicated. Interposition graft repair should be contemplated in the setting of neural discontinuity; split-hypoglossal nerve transfer to the main trunk of the facial nerve is an alternative option where interposition graft repair is unfeasible or where no recovery is noted within 12 months. The goal of main trunk repairs and transfers is to restore facial tone and some form of blink; meaningful reanimation of expression is rarely achieved. Volitional expressions may be restored through targeted nerve transfers during this period, such as nerve-to-masseter transfer to lower zygomatic branches of the facial nerve for smile reanimation, or cross-face nerve grafting to upper zygomatic branches for blink restoration. Targeted nerve transfers should be considered in patients demonstrating minimal to no improvement in facial function 7 months following vestibular schwannoma resection with facial nerve preservation, as the probability of ultimate recovery of meaningful expression is less than 10%.[27] Static periocular reanimation (such as upper lid weighting and lateral tarsal strip procedure) is offered early in the course of palsy onset where recovery is likely to take several months.

Flaccid Facial Palsy Without Viable Facial Musculature

Where native facial musculature is absent (eg, following resection or congenital absence) or unlikely to be receptive to reinnervation (eg, long denervation period or marked distal perineural spread of a malignant tumor), nerve repair or transfers are no longer indicated. In addition to PT and targeted chemodenervation of healthy side lip depression and brow elevation, surgical interventions include static facial suspensions, static periocular reanimation, and muscle transfers. Targeted suspensions of the brow, lower eyelid, and midface, nasal valve, NLF, and oral commissure may be achieved using sutures, fascia lata, or bioabsorbable or permanent implants. Tightening of the lower lid may be achieved by the lateral tarsal strip procedure[28] with or without medical canthal tendon plication. Dynamic smile reanimation may be achieved through antidromic[29] or orthodromic[30] temporalis muscle transfer, or free muscle transfer[31,32] with motor innervation provided through cranial nerve transfer. Options for dynamic reanimation of the lower lip include anterior digastric muscle transfer[33] or inlay of a T-shaped fascia graft.[34]

Postparalytic Facial Palsy

PFP develops 6 to 18 months following severe facial nerve insult with spontaneous, yet aberrant, regeneration or following main trunk nerve grafting. Once present, it is permanent. Lagophthalmos is rare. PT is first-line treatment; a comprehensive

program includes patient education, soft tissue mobilization, mirror and EMG biofeedback, and neuromuscular retraining.[35] Blunting of hyperactivity through filler injection and weakening of hyperactive muscles through targeted chemodenervation, neurectomy, or resection in advanced disease is indicated in conjunction with PT. For many patients, targeted chemodenervation of diseased side orbicularis oculi, mentalis, and platysma offers significant improvements. Weakening of the diseased side depressor anguli oris muscle through chemodenervation or resection can result in dramatic improvement in smile dynamics in select cases.[36,37] In cases with severe restriction of oral commissure excursion, regional (eg, temporalis) or free (eg, gracilis) muscle transfer may be considered for dynamic smile reanimation. Targeted nerve transfers, such as nerve-to-masseter transfer to diseased-side zygomatic branches for smile reanimation, are largely ineffective in the setting of PFP.

CLINICAL OUTCOMES

Systematic tracking of therapeutic outcomes is a prerequisite to clinical excellence. Outcomes tracking in FP may entail patient-reported QOL measures, clinician-assessed grading of facial function, and objective measurement of facial displacements. QOL impact may be assessed using generalized patient-graded scales such as the SF-36.[38] The Facial Disability Index,[39] the Facial Clinimetric Evaluation,[1] and the Synkinesis Assessment Questionnaire[40] are patient-graded scales specifically designed and validated for use in FP to concurrently assess symptom severity and impact on QOL. Although global 5- or 6-point facial function scales exist (such as the House-Brackmann,[41,42] Fisch,[43] and others[44,45]), such scales lack the resolution necessary to capture meaningful changes in zonal function over time. The Yanagihara scale[46] provides Likert scale resolution of zonal appearance with movement, but not at rest, and lacks separate grading of synkinesis. The Sunnybrook Facial Grading System[47] provides weighted scores of zonal symmetry at rest and with motion in addition to synkinesis. A recently validated electronic facial paralysis assessment tool provides even higher resolution zonal data through use of continuous visual analogue scales to assess 5 static, 7 dynamic, and 4 synkinesis zonal parameters.[48,49] A computer vision-based facial landmark recognition algorithm has recently been used within a novel freeware application (Emotrics, Mass Eye and Ear Infirmary) for objective measurement of various facial displacements (eg, smile excursion) from clinical photographs.[50]

SUMMARY

Management of FP necessitates establishing a diagnosis and formulating a therapeutic plan according to the timing of presentation in flaccid cases, and specific pattern of facial dysfunction in patients presenting with aberrant neural regeneration. Therapeutic interventions include PT, injectables, and a plethora of surgical reanimation procedures.

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