An Evidence-Based Approach to Facial Reanimation

Nate Jowett, MD*, Tessa A. Hadlock, MD

KEYWORDS

• Facial palsy • Facial paralysis • Facial reanimation • Evidence-based medicine • Synkinesis

KEY POINTS

- Management of facial palsy (FP) is dictated by the pattern and time course of dysfunction.
- Therapeutic options include pharmaceutical agents, corneal protective measures, physical therapy (PT), chemodenervation agents, fillers, and a myriad of surgical procedures.
- Good evidence from well-designed studies supports the use of glucocorticoids and antivirals in the setting of idiopathic and acute viral FP and botulinum toxin (BTX) and PT in the setting of synkinesis.
- A plethora of surgical techniques and their respective outcomes have been described in the literature, but few use controls, blinded assessment, and validated scales to reduce bias.
- Outcomes research in facial paralysis should comprise standardized subjective quality-of-life (QOL) and objective functional measures.

INTRODUCTION/OVERVIEW

Whether congenital or acquired, FP is a devastating condition with functional and aesthetic sequelae resulting in profound psychosocial and QOL impairment.[1,2] When acquired, the inciting insult typically results in acute flaccid facial palsy (FFP). Long-term functional outcomes range from full return of normal function to persistent and complete FFP. In between these extremes exist zonal permutations of hypoactivity and hyperactivity and synkinesis; such patterns of dysfunction may collectively be referred to as nonflaccid facial palsy (NFFP).[3] To reduce ambiguity,[4] a summary of pertinent definitions is provided in **Table 1**.

When severe, FFP results in loss of static and dynamic facial symmetry, brow ptosis that obscures vision, paralytic lagophthalmos resulting in exposure keratitis, collapse of the external nasal valve (ENV) impairing nasal breathing, oral incompetence, and articulation impairment. Management is focused on eye protection, restoration of symmetry at rest, and dynamic reanimation. Synkinesis-related symptoms predominate in NFFP, with periocular synkinesis resulting in a narrowed palpebral fissure width that impairs peripheral vision, midfacial synkinesis restricting meaningful smile, and platysmal synkinesis resulting in neck discomfort and facial fatigue. Efforts are concentrated on improving dynamic symmetry.

THERAPEUTIC OPTIONS AND SURGICAL TECHNIQUES

Therapeutic options for FP are dictated by the pattern and time course of dysfunction and may include pharmaceutical agents, corneal protective measures, PT, chemodenervation agents, fillers, and a myriad of surgical procedures. Patients may be classified into 1 of 5 domains: acute FFP, FFP with potential for spontaneous recovery (PSR), persistent FFP with viable or nonviable facial musculature, and NFFP. **Fig. 1** summarizes

Division of Facial Plastic and Reconstructive Surgery, Department of Otolaryngology, Facial Nerve Center, Massachusetts Eye and Ear Infirmary, Harvard Medical School, 243 Charles Street, Boston, MA 02114, USA
* Corresponding author.
E-mail address: nathan_jowett@meei.harvard.edu

Facial Plast Surg Clin N Am 23 (2015) 313–334
http://dx.doi.org/10.1016/j.fsc.2015.04.005

Table 1
Relevant definitions

Term	Definition
Facial palsy	Term encompassing entire spectrum of facial movement disorders including facial paralysis, flaccid facial palsy, and nonflaccid facial palsy
Facial paralysis	Complete absence of facial movement and tone
Flaccid facial palsy	Absence or weakness of facial movement and tone, without synkinesis or hyperactivity
Nonflaccid facial palsy	A postparetic state whereby aberrant nerve regeneration has occurred, consisting of varying degrees of zonal synkinesis and hypoactivity and hyperactivity
Facial synkinesis	Involuntary and abnormal facial muscle activation accompanying volitional or spontaneous expression

the therapeutic options by zone and side for FFP and NFFP.

The acute setting comprises the first few weeks following onset of FFP whereby medical therapy (immunosuppressants, antivirals, and/or antibiotics), surgical decompression, or neuroplasty may be indicated. Eye lubrication and taping of the eye closed at night is indicated if paralytic lagophthalmos is present to prevent exposure keratopathy, in addition to PT for patient education and upper eyelid stretching to aid passive closure. Correction of paralytic lagophthalmos may be achieved by tarsorrhaphy or by placement of an eyelid spring or weight. Indications include poor prognosis for rapid recovery, inadequate Bell phenomenon, and absent recovery at 3 months.[5] Where nerve continuity is believed to be intact in the setting of FFP, for example, following resection of a vestibular schwannoma (VS) where FN stimulation was achieved before closure, a PSR exists with a plateau expected by 9 to 12 months.[6] Other than corneal protective measures, observation during this period is warranted.

When paralysis persists after nerve insult, native facial musculature is believed to remain receptive to reinnervation for up to 2 years after denervation. During this period, dynamic reanimation procedures using native facial musculature are possible, such as direct repair or interposition grafting of FN stumps (in the setting of an accessible FN discontinuity) or nerve transfer to the distal FN stump or specific branches (where the proximal FN is inaccessible or nonviable). Coaptation of a portion of the ipsilateral hypoglossal nerve to the entire distal FN stump restores resting tone to the face, whereas targeted nerve transfers aim to restore specific volitional facial movements such as smile or blink by coaptation of the donor nerve to the specific distal FN branch controlling the muscle of interest. Common donor nerves for targeted transfer include branches of the contralateral FN (cross-face nerve grafting [CFNG]) or ipsilateral branches of the trigeminal nerve.

When reinnervation of the facial musculature is not possible, therapeutic options in long-standing FFP, in addition to PT and corneal protective measures, include static zonal suspensions, lower eyelid tightening, and dynamic smile reanimation. Targeted suspensions of the brow, lower eyelid, midface, nasal valve, nasolabial fold (NLF), and oral commissure may be achieved using sutures, fascia lata, and bioabsorbable or permanent implants. Tightening of the lower lid may be achieved by the lateral tarsal strip (LTS) procedure[7] with or without medical canthal tendon plication. Dynamic smile reanimation may be achieved through antidromic[8] or orthodromic[9] temporalis muscle transfer or free muscle transfer[10,11] with motor innervation provided through cranial nerve transfer. Such procedures may be paired with weakening of the normal-side brow or lip depressors or the use of fillers to efface the healthy NLF. Options for dynamic reanimation of the lower lip include anterior digastric muscle transfer,[12] CFNG or split hypoglossal neurotization of the depressor muscles or transferred digastric muscle,[13] and inlay of a T-shaped fascia graft.[14]

NFFP is by definition a chronic condition with intact yet aberrantly reinnervated facial musculature. Lagophthalmos in NFFP is exceedingly rare. PT is first-line treatment; a comprehensive program includes patient education, soft-tissue mobilization, mirror and electromyography (EMG) biofeedback, and neuromuscular retraining.[15] 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. In cases with severe restriction of oral commissure excursion, regional