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REVIEW ARTICLE

Facial Nerve Palsy: Anatomy, Etiology, Evaluation, and Management

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ABSTRACT The ophthalmologist may be the first clinician to see a patient who presents with acute facial nerve palsy. Under such circumstances the ophthalmologist should make every effort to establish the underlying cause of the facial palsy and ensure that the patient's cornea is adequately protected. This article reviews the anatomy of the facial nerve, the varied disorders that may cause a facial palsy, a detailed evaluation of such a patient, and the various medical and surgical treatments available.

KEYWORDS Facial nerve palsy; lagophthalmos; gold weight implants; brow lift; paralytic ectropion

INTRODUCTION

Normal facial function plays a critical role in a person's physical, psychological, and emotional makeup. Facial disfigurement can affect all these components and can result in social and vocational handicap. The ophthalmologist plays a pivotal role in the multidisciplinary team involved in the evaluation and rehabilitation of patients with facial nerve palsy (Lee et al., 2004).

ANATOMY

There are four brainstem nuclei to cranial nerve VII. The facial motor nucleus, which controls muscles of facial expression; the superior salivatory nucleus, which sends fibers for lacrimal gland secretion and salivary secretion; the nucleus solitarius, which receives fibers of taste for the anterior two-thirds of the tongue; and the trigeminal sensory nucleus, which receives sensory fibers for a small portion of the external ear.

The facial nerve leaves the cerebellopontine angle caudal to the trigeminal nerve adjacent to the nervus intermedius (tearing, salivation, taste) and then enters the internal auditory canal of the temporal bone along with the 8th cranial nerve. Large lesions of cranial nerve VII or VIII may cause loss of corneal sensation from pressure on the trigeminal nerve. The facial nerve proceeds with a 30-mm course through the temporal bone, the longest interosseous course of any cranial nerve, which makes the facial nerve vulnerable to swelling.

Three branches leave the facial nerve within the temporal bone. The first, greater superficial petrosal nerve, arises at the geniculate ganglion. It carries

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lacrimal and palatine secretory fibers to the pterygopalatine ganglion. Postganglionic fibers for tear secretion then follow the infraorbital nerve and branch off with frontozygomatic branches that innervate the lacrimal gland. The other two branches of the facial nerve include a small branch to the stapedius muscle within the middle ear and a branch, chorda tympani, receiving fibers of taste from the tongue and sending fibers to innervate the salivary glands.

The facial motor fibers exit at the stylomastoid foramen to supply the muscles of facial expression. The facial nerve runs through the parotid gland to innervate the facial musculature through five main branches: the temporal, zygomatic, buccal, mandibular, and cervical branches.

Facial nerve lesions above the geniculate ganglion classically cause more severe ophthalmic symptoms because lacrimal secretion and orbicularis closure are involved. Central lesions can cause crocodile tears when regenerating fibers of the chorda tympani grow down the lacrimal secretory neural pathway.

ETIOLOGY

The causes of 7th nerve palsy are myriad, but can be broadly divided into idiopathic, traumatic, infectious, and neoplastic.

Idiopathic

Bell's palsy is defined as an idiopathic paralysis of the facial nerve and is a diagnosis of exclusion. It is the most common cause of facial weakness, accounting for approximately 49–51% of all cases (May and Klein, 1991). It is typically unilateral, with a sudden onset, and generally spontaneously resolves within 6 months. Many etiologies have been proposed, including a viral inflammatory mechanism (May and Klein, 1991), and systemic steroids and/or acyclovir have been recommended as treatment. The current available evidence has shown that early treatment with prednisolone significantly improves the chances of complete recovery at 3 and 9 months (Sullivan et al., 2007). However, there is no evidence of a benefit of acyclovir given alone or an additional benefit of acyclovir in combination with prednisolone (Sullivan et al., 2007).

Traumatic

Traumatic injury is the second most common etiology of facial nerve paralysis, comprising 8–22% of cases (May and Klein, 1991). A significant proportion of these injuries occur during delivery either due to birth canal trauma or forceps delivery (May et al., 1981; Smith et al., 1981). Other causes of traumatic paralysis include surgical trauma, penetrating parotid or middle ear trauma, barotrauma, facial fractures, and temporal bone fractures (Marenda and Olsson, 1997). High-resolution computed tomography is used for localization of nerve injury in suspected cases of temporal bone trauma. In the absence of gross radiographic abnormalities, electrophysiologic testing helps predict the likelihood of spontaneous recovery. Primary end-to-end neurorrhaphy is the preferred management for transection injuries, while facial nerve decompression may benefit other forms of high-grade nerve trauma. Secondary facial reanimation procedures are useful adjuncts when initial facial nerve repair is unsuccessful or impossible (Lee et al., 2004).

Infectious

Infection is the next most common etiology of facial paralysis (May and Klein, 1991). Ramsay Hunt syndrome caused by herpes zoster is classically associated with zoster vesicles on the ear, in the external auditory canal or tympanic membrane, with vestibulo-auditory symptoms due to the proximity of the 8th cranial nerve in this area (Sweeney and Gilden, 2001). Lyme disease (Borrelia Burgdorferi) is a known infectious cause of facial palsy and should be considered in the differential diagnosis of any patient who has visited endemic areas (Peltomaa et al., 2002). Tuberculous otitis media should be considered in the presence of chronic middle ear disease. Facial palsy can be the first presenting sign of AIDS, but is generally described in chronic HIV infection (Schot et al., 1994). Other infections include polio (Moses et al., 1985), mumps (Endo et al., 2001), cytomegalovirus (Strauss, 1981), mononucleosis (Johnson and Avery, 1991), leprosy (Lubbers et al., 1994), cat scratch fever (Chiu et al., 2001), and dengue fever (Patey et al., 1993).

Neoplastic

Acoustic neuroma of the adjacent nerve VIII or other cerebellopontine angle tumors, such as a meningioma

or a tumor of the glomus jugulare, are usually associated with facial nerve weakness after surgery, as opposed to a preoperative facial palsy. Magnetic resonance of the cerebellopontine angle usually establishes the diagnosis of a tumor. Recovery will occur in cases where the nerve has been bruised or stretched during tumor removal, but is less likely to occur where a large segment of the nerve had to be removed, with or without an interpositional nerve graft.

Malignant tumors of the external auditory canal, such as a squamous cell carcinoma or an adenoid cystic carcinoma, can extend into the temporal bone and cause proximal facial nerve palsy. Malignant parotid tumors (e.g., mucoepidermoid carcinoma, adenoid cystic carcinoma) and facial nerve schwannomas may all cause facial nerve palsies. Metastatic lesions from the orbit, lung, breast, or kidney can, on rare occasions, affect the facial nerve. Malignant facial skin lesions (e.g., basal cell carcinoma, squamous cell carcinoma) may cause peripheral facial palsy. In particular, perineural spread of cutaneous squamous cell carcinoma is a common cause of slowly progressive facial palsy.

Miscellaneous

Neurologic causes of facial nerve paralysis include multiple sclerosis, myasthenia gravis, Guillain-Barre syndrome, hereditary hypertrophic neuropathy, Melkersson-Rosenthal syndrome, Moebius syndrome, and cerebrovascular accident (Marenda and Olsson, 1997).

Systemic and metabolic disorders implicated in facial nerve paralysis include diabetes mellitus, hyperthyroidism, hypertension, pregnancy, acute porphyria, autoimmune syndromes, sarcoidosis, amyloidosis, carbon monoxide toxicity, tetanus, diphtheria, vitamin A deficiency, ethylene glycol ingestion, and alcoholism (Marenda and Olsson, 1997).

HISTORY OF DISEASE

Obtaining an accurate history of the onset, progress, and associated symptoms of newly acquired facial nerve palsy is extremely helpful in determining the potential cause of the palsy. More importantly, it serves as a guide for prognosis and timing of any necessary surgical intervention.

Acute versus Chronic Facial Nerve Palsy

The type of onset is not diagnostic but can be prognostic. Slowly progressive onset is suggestive of tumor compression (Jackson and von Doersten, 1999).

Complete versus Incomplete Facial Nerve Palsy and Recovery

If the idiopathic palsy remains incomplete, a complete or satisfactory recovery is the rule. Whether the palsy is complete or incomplete, excellent recovery occurs, with few exceptions, when recovery begins in the first 3 weeks. If recovery begins between 21 days and 2 months, most patients will have a satisfactory recovery of function. However, if recovery does not begin until 2 to 4 months after onset of palsy, recovery will be unsatisfactory in most patients (May and Schaitkin, 2000).

Recurrence

Recurrence of facial nerve palsy occurs in Bell's palsy, tumors, and Melkerrson-Rosenthal syndrome. Alternating side of recurrence is seen more with Bell's palsy. Ipsilateral recurrence implies tumor until proven otherwise. Bilateral acute palsy may suggest Lyme disease, Guillain Barre, or acute leukemia.

Facial Nerve Grading Systems

The gold standard for grading facial nerve function is the House-Brackmann grading scale (House and Brackmann, 1985) (Table 1). Due to the limitations and subjectivity of this scale, several new scales of various degrees of objectivity and ease of use have been introduced (Kang et al., 2002). These include the Nottingham system, the Sunnybrook scale, the Yanagihara, and the Sydney system, all with their advantages and disadvantages (Berg et al., 2004; Coulson et al., 2005).

CLINICAL EVALUATION

Upper eyelid: Evaluate upper eyelid retraction. Upper eyelid retraction contributes to lagophthalmos due to the unopposed action and tone of the levator and Muller's muscles.

Blink reflex: It is often missing. Instead, there is only a slight flutter.

Eyelid closure: Evaluate lagophthalmos on gentle and forced closure. The extent of lagophthalmos will often

TABLE 1 **The House-Brackmann Grading Scale**

Grade	Description	Characteristics
1	Normal	Normal facial function in all areas
2	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
3	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
4	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
5	Severe dysfunction	Gross: only barely perceptible motion At rest: asymmetry Motion: forehead—none eye—incomplete closure mouth—slight movement
6	Total paralysis	No movement

dictate the extent and timing of medical and surgical intervention to protect the eye.

Brow: Evaluate eyebrow position and range of elevation. Severe brow ptosis can cause secondary eyelid ptosis, interfering with visual field. In the early stages of weakness, this may be helpful in protecting the globe.

Lower eyelid: Evaluate paralytic ectropion. Pay particular attention to medial canthal tendon laxity.

Midface: Evaluate midface position, as this can have a significant mechanical effect on the lower eyelid. Evaluate nasolabial fold, cheek tone and elevation.

Mouth: Evaluate mouth symmetry, ability to drink, eat, and whistle.

Neck: Evaluate platysma muscle strength.

Hearing: It can be grossly tested by gentle finger rubbing to compare hearing on each side to detect severe loss.

Corneal sensation: It should be carefully tested and compared to the normal side. Acute loss of corneal sensation indicates a severely guarded prognosis for patients with facial palsy and demands aggressive treatment.

Bell's phenomenon: It should be evaluated because patients with good Bell's phenomenon may tolerate poor closure much better than those with poor Bell's.

Tear function: A Schirmer's test is performed to determine tear production. Tearing may be decreased with facial nerve palsy if the salivatory nucleus or branches to the lacrimal gland have been affected. On the other hand, tearing may be increased with aberrant regeneration or reflex tearing from ocular irritation secondary to exposure and drying of the ocular surfaces.

Synkinesis: Spontaneous twitching or cross innervation due to aberrant regeneration may occur in longstanding or recovering facial nerve palsy. The most noticeable areas of synkinesis involve the orbicularis oculi, nasolabial fold area, and mouth.

MANAGEMENT

Corneal Exposure and Lagophthalmos

Treatment directed at protecting the cornea depends on the predicted prognosis of return of nerve function and the degree of risk to the cornea based on the amount of lagophthalmos, the quality of Bell's phenomenon, and the presence or absence of paralytic ectropion.

Estimating the likelihood of recovery requires good communication between all those involved in the patient's care (Lee et al., 2004).

Temporary Treatment

Where there is low corneal risk and a good prognosis for recovery, the following should be considered.

Artificial tears are the mainstay of treatment in facial nerve palsy patients. Frequency of applications is based on patient symptoms and objective clinical findings of ocular surface drying. Choice of lubricant viscosity is based on the same criteria, also taking into account the patient's tolerance for visual blurring caused by more viscous products. Preservative-free lubricants are indicated when frequent use is required to decrease the risk of surface toxicity.

Bland lubricating *eye ointment* is very helpful for keeping the eye moist at night in the setting of paralytic lagophthalmos. It may also be used during the day if artificial tears are insufficient from keeping the eye moist.

The eyelids can be *taped* together with a stiff tape to close the eye at night. However, it is essential to ensure that the tape crossing the palpebral fissure does not touch the cornea or conjunctiva, thus preventing further trauma. On the other hand, the lower eyelid can be taped upward toward the lateral canthus to improve closure.

Moisture chambers act as barriers to evaporation. There are numerous types on the market. They are usually translucent. They may have adhesive or be held in place with an elastic strap. Alternatively, if a commercial moisture chamber is not available, one can apply petroleum jelly around the orbital rim and place a circular piece of clear cellophane or plastic wrap on top to create a moisture seal.

The degree of lagophthalmos and the amount of lubricants needed may be reduced with temporary eyelid loading using *external eyelid weights* (MedDev Corporation, Los Angeles, CA, USA). They are made of pure tantalum and are similar in design to those for implantation, but lack fixation holes. The weights are fixed to the pretarsal skin surface with double-sided hypoallergenic adhesive tape. The weights' anterior surfaces are available in a variety of skin tones. Such weights are useful for a temporary facial palsy but can also be used for a trial period before subjecting a patient to an upper eyelid gold weight implant (Seiff et al., 1995; Mavrikakis and Malhotra, 2006).

Botulinum toxin, injected either transcutaneously through the skin crease or subconjunctivally at the upper border of the tarsus, will produce complete ptosis and afford corneal protection (Ellis and Daniell, 2001). The cornea may, however, still be at risk, where there is a poor Bell's phenomenon coupled with marked laxity of the lower lid. This procedure also has the disadvantage of affecting the patient's vision and may provide less than adequate protection as the levator function returns.

Another means of closure is *temporary tarsorrhaphy* (central or lateral). This can be achieved with a simple suture or cyanoacrylate glue (Donnenfeld et al., 1991) as a temporizing measure. As a general rule, however, the results of permanent tarsorrhaphy are functionally and aesthetically poor. A tarsorrhaphy often fails to achieve adequate corneal coverage and allows segmental deterioration over time. In addition, examination of the cornea can be difficult, and the field of vision is limited for the patient. Complications of the procedure, which may occur after lysis of the intermarginal eyelid adhesions, include notching of the eyelid margin, entropion, and trichiasis.

In patients who cannot be satisfactorily managed with topical lubricants alone or who have decreased tear production, *punctal occlusion* is beneficial. This can be achieved temporarily with the use of silicone punctal plugs. If these are tolerated without secondary epiphora, surgical punctal occlusion can be performed under local anesthesia. A simple disposable cautery device is used with a brief application to the puncta.

Permanent Treatment

Where no functional improvement of the nerve is anticipated, the long-term protection of the cornea is more complex and depends on the degree and manner in which the upper and lower eyelids are affected.

Upper eyelid. In the upper eyelid, good passive closure and an improvement in the quality of the blink can be achieved with *gold weight* insertion. It also serves to lower the retracted upper eyelid. This is the most commonly performed surgery for facial nerve palsy and should be emphasized that this procedure does not restore normal blink reflex. It is equally effective in the early as the later stages (Snyder et al., 2001), with the advantage that, if nerve function improves, the weight is easy to remove. Gold is preferred as a material for the weight because of its high density, malleability, minimal tissue reactivity,

and color compatibility with skin (Pickford et al., 1992).

A preoperative trial is important in determining the weight required for satisfactory eyelid closure and eyelid position in primary gaze. The patient is placed in a sitting position, and the weight is taped onto the skin surface of the upper eyelid over the tarsal plate. Progressively heavier weights are placed until good eyelid closure, with ptosis of not more than 1–2 mm is obtained (Jobe, 1974; Gilbard and Daspit, 1991).

Although upper eyelid loading is safe, predictable, and reversible, there is no consensus on technique (Mavrikakis and Malhotra, 2006). Many techniques have evolved with their own advantages and disadvantages (Mavrikakis and Malhotra, 2006). The most popular approach currently appears to be pretarsal fixation due to its simplicity; however, our preferred approach combines high pretarsal fixation with direct levator aponeurosis coverage (Caesar et al., 2004; Mavrikakis and Malhotra, 2006). Complications of infection, allergic reaction (Doyle et al., 2005), astigmatism (Mavrikakis et al., 2006; Saleh et al., 2007), migration, and extrusion can occur, but are infrequent. Implantation with a platinum weight (John Weiss International) should be considered in patients with a personal and/or family history of gold allergy (Doyle et al., 2005).

Upper eyelid retraction is a common sequela of facial paralysis. There are numerous procedures available for lowering the upper lid (*retractor recession*), and the choice will depend on the amount of retraction. Mullerectomy (Hassan et al., 2005) is sufficient to treat small degrees (1–3 mm) of retraction, but with larger amounts retractor recession transconjunctivally or with an anterior approach may be required. An alternative to the gold weight implant is the less frequently used *palpebral spring* (Morel-Fatio and Lalardrie, 1964). This is the most commonly used method of dynamic eyelid animation. This approach uses a custom made stainless steel spring, which is surgically 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, being an active implant, allows for a more rapid eyelid excursion and complete closure compared to gold weights. However, the spring's disadvantages may outweigh its benefits, particularly in inexperienced hands, because it is more prone to exposure and extrusion, is technically difficult, and often requires multiple surgical adjustments. Other dynamic procedures that have been described to effectively close the eyelid include the Arion sling (Arion, 1972) and temporalis muscle transfer (Masters et al., 1965). The *Arion sling* (silicone encircling band) consists of implanting a rod of silicone around the upper and lower eyelids and securing with tension medially and laterally. When the upper eyelid opens, tension is placed on the cerclage. When the patient relaxes the levator muscle, the stretched cerclage causes closure of the eyelid. The Arion sling offers the advantage of reanimating the upper and lower eyelid. These can work quite well for a period of time but then tend to weaken. Tightening can be attempted, but the silicone can migrate in the eyelid tissues, making these efforts futile.

With the *temporalis muscle transfer*, the force of closure is generated by a cerclage around the eyelid of a strip of temporalis fascia attached to the temporalis muscle. When the patient clenches the jaw, tension is placed on the eyelid, causing it to close. Consequently, the patient still experiences lagophthalmos at night as the patient's temporalis muscle relaxes. The palpebral spring, Arion sling, and temporalis muscle transfer are procedures that are not advocated or used by the author.

Lower eyelid. When addressing problems of the lower eyelid, the choice of surgical procedure will depend on the degree of laxity or ectropion and on the state of the medial and lateral canthal tendons. Also, because of the progressive laxity and stretching of permanently paralyzed tissues, a series of different procedures may be required over time.

Lower eyelid and canthal resuspension help protect the cornea, improve blink coverage, enhance the lacrimal pump, and improve paralytic ectropion. The *lateral tarsal strip* (Anderson and Gordy, 1979) has been the most commonly performed procedure for this purpose and frequently works well. However, it tends to weaken over time and provides minimal medial elevation. This persistent medial canthal tendon laxity can be difficult to correct. *Medial canthal tendon plication* can be performed, and a variety of procedures have been described; however, a good solution has not been identified. The *suborbicularis oculi fat (SOOF) lift* appears to have much promise in the management of paralytic lower eyelid retraction (Alford, 2000; Olver 2000; Horlock et al., 2002). Functionally, the lower eyelid extends from the lash line to the upper lip. The constant action of gravity on the paralyzed midface structures causes them to sag and drag the lower eyelid margin down with

them. Repositioning and supporting the midface structures with a SOOF elevation provides good support for the lower eyelid. Where there is significant lower lid retraction, this can be combined with insertion of a *spacer* (tarsus, hard palate, auricular cartilage). In cases of marked tissue atrophy, an *autogenous fascial sling* can be threaded hammock-like through the entire length of the eyelid, anchored by fixation to the medial canthal tendon and the lateral orbital periosteum, to achieve adequate lower eyelid elevation (Lee et al., 2004).

Epiphora

Reflex tearing from ocular exposure. This could be improved by reduction of the palpebral aperture and improvement of eyelid closure.

Lacrimal pump failure. A paralyzed orbicularis impairs lacrimal pump function. An increase in eyelid laxity exacerbates the epiphora. An augmented lateral tarsal strip (Chang and Olver, 2006) can be undertaken to facilitate tear drainage. Jones tube insertion may be appropriate to decrease the resistance to tear outflow.

Aberrant innervation of lacrimal gland. Crocodile tears/gustatory lacrimation results from the misrouting of postganglionic parasympathetic secretomotor fibers. This hypersecretion can be successfully treated with botulinum toxin injections (Keegan et al., 2002).

Facial Synkinesis

Synkinetic movements secondary to aberrant regeneration may occur. Typical movements include blinking with movements of the mouth and spasms of the face with eyelid closure. Some patients experience complete eyelid closure when using the perioral muscles. These synkinetic movements respond well to botulinum toxin, with only very low doses required due to denervation hypersensitivity.

NEUROMUSCULAR FACIAL RETRAINING

The basic aim of facial rehabilitation is facial symmetry at rest and when facial expressions are being performed. The patient practices appropriate timing and extent of muscle contraction on the affected side to match the expression of the unaffected side of the face (Lee et al., 2004). These exercises may be enhanced using surface EMG biofeedback and may be particularly useful for disfigurement from facial synkinesis (Cronin and Steenerson, 2003).

SOFT TISSUE REPOSITIONING

Soft tissue repositioning includes management of brow and midface ptosis. Correction of *brow ptosis* can be achieved either via a direct approach (excision of an ellipse of skin, orbicularis, and frontalis muscle, with fixation of deep tissues with nonabsorbable sutures to the periosteum just above the brow), an internal browpexy (where the brow tissues are anchored to the brow periosteum usually via an upper lid blepharoplasty approach), or an endoscopic approach (with a subperiosteal plane of dissection with elevation of the soft tissues on its periosteal pedicle anchored with bone fixation) (Lee et al., 2004).

Midface ptosis may be addressed with a static sling using autogenous fascia lata to pull the lip and face upward toward the zygomatic arch. The fascial strips are threaded through the muscles of the mouth with a Wright's fascia needle and tied tightly on the zygomatic arch.

FACIAL REANIMATION-DYNAMIC PROCEDURES

If there is little likelihood of spontaneous recovery of facial function, the patient should be counseled regarding general facial reanimation. Cooperation between ophthalmology, neurosurgery, head and neck surgery, and plastic surgery is needed to provide the patient with optimal care. The procedures available may provide some degree of facial tone or voluntary facial movement but typically do not generate good voluntary blink.

Temporalis Muscle Transfer

Temporalis muscle flaps reflected from the temporalis fossa to the face can be used to animate the midface, including the upper lip and the angle of the mouth (Croxson et al., 2000). This procedure can be used in patients with chronic facial nerve palsy who are not candidates for facial nerve-transfer procedures.

Free Nerve-Muscle Grafts

Free grafts composed of nerve and innervated muscle can be transferred into the paralytic face for reinnervation. The most commonly used free grafts are gracilis, latissmus dorsi, and inferior rectus abdominis. This procedure is useful in the absence of distal facial nerve

fibers or motor endplate function, with a large soft tissue defect in the cheek, and where other dynamic reanimations have not been successful. The neurovascular pedicle is anastomosed either with the ipsilateral functioning proximal end of the facial nerve or with the contralateral nerve that supplies the normal unaffected side of the face.

Ipsilateral Nerve Transposition

An ipsilateral motor nerve can be anastomosed to innervate the distal facial nerve. The patient must then train themselves to stimulate the substituted nerve in order to move the reinnervated area of the face. There is a significant postoperative recovery period. This also results in sacrifice of an adjacent, otherwise normal area or organ of the face. Examples of nerve substitutions include:

1. Hypoglossal-facial anastomosis. A variation called the jump graft only uses a partial incision of the 12th nerve rather than a complete transection, thus preserving some of the tongue function. Reanimation is generally achieved more successfully in the lower face, and many patients achieve functional adaptation of not needing to think about moving their tongue to animate their face. Lacrimal function is not restored.
2. Spinal accessory nerve-facial anastomosis. Movement of the shoulder causes facial contraction.

Facial nerve-cross face anastomosis. The contralateral facial nerve can be used to innervate the paralytic side. Use of the contralateral facial nerve allows for a more symmetric facial animation when compared to muscle transfers or nerve substitutions with other cranial nerves.

1. Partial facial anastomosis. A partial anastomosis from the redundant area of the zygomatic and buccal branches can be used to innervate the paralytic side with less risk of inducing complete paralysis of the donor side.
2. Free nerve grafts. Free nerve grafts can be used to connect the non-paralytic with the paralytic side. Free nerve grafts take a very long time to attain maximal effectiveness.

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