ORIGINAL PAPERS

Surgical Therapeutic Algorithm in Facial Paralysis

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Abstract

Facial nerve paralysis is a debilitating condition with multiple etiologies, with aesthetic, functional, psychological and social impact. Given the complex multitude of causes that may generate such condition, a therapeutic algorithm is mandatory when attempting reconstruction. Severity, timing, patient adherence to a rehabilitation program, status of ipsilateral and contralateral facial nerves and particularities of each patient are all criteria which should be accounted when choosing a treatment option. After initial assessment, a variable treatment panel is available based on condition type include medicamentous therapy, rehabilitation program, dynamic and static procedures surgical procedures, having as primary aim functional restoration achieving aesthetic balanced facial features. This paper summarizes current knowledge in facial paralysis reconstruction and presents an algorithmic approach that eases decision making and therapeutic strategy.

Keywords: facial paralysis, algorithm, reconstruction, nerve transfer, free muscle transfer.

Rezumat

Paralizia facială, de diverse etiologii, reprezintă o patologie invalidantă determinând afectare atât functională cât si estetică cu consecințe psiho-sociale semnificative. Tinând cont de multitudinea etiologiilor asociate paraliziei faciale, este necesară elaborarea unui algoritm terapeutic ce ghidează planul reconstructiv. Severitatea leziunii, timpul scurs de la instalarea paraliziei, statusul inervației regionale cât și al nervului facial contralateral, complianța terapeutică a pacientului reprezintă elemente cheie ce trebuie luate în considerare în ghidarea deciziei terapeutice. După evaluarea amănunțită a leziunii, sunt luate în discuție o serie de procedee terapeutice în funcție de natura și severitatea paraliziei faciale incluzând: tratament conservator medicamentos, recuperare medicala, cât și tehnici chirurgicale dinamice și statice având drept obiectiv reconstrucția funcțională adecvată cât și redobândirea esteticii faciale. Acest articol face o trecere în revistă a opțiunilor terapeutice actuale utilizate pentru reanimarea facială și de asemenea prezintă un algoritm terapeutic integrativ ce vizează ghidarea deciziei reconstructive în paralizia facială pentru obținerea unor rezultate optime funcționale și estetice.

Cuvinte cheie: paralizie facială, algoritm, reconstrucție, transfer de nerv, transfer muscular.

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BACKGROUND

Etiopathogenesis

Facial paralysis can be caused by congenital disorders, acquired illnesses, trauma, or malignancy, determining substantial functional and emotional impact on patients regardless of cause. Impaired functional abilities include trouble generating facial expressions, speaking, eating, and drinking.[1] Facial deformity causes psychological distress, anxiety, depression, maladaptive behaviors and impaired emotional well-being, suggesting that psychological adjustment to the facial impairment is a critical predictor of overall recovery.[2,3]

The most prevalent cause of facial nerve paralysis is Bell palsy, which accounts for almost 70% of all facial nerve paralysis.[4] It often manifests as a lesion of the lower motor neurons with complete unilateral paralysis. Although the vast majority of cases are idiopathic, any clinician must rule out a cerebrovascular stroke or other possible underlying condition.[5] Trauma is the second most common cause of facial nerve palsies, accounting for 10 to 23% of all cases. Viral infection, which causes facial nerve palsy, accounts for 4.5 to 7%, whereas neoplasia accounts for 2.2 to 5%.[6]

There is no convincing evidence to suggest that facial nerve palsies are more common in any gender or ethnicity, and people of all ages might be afflicted. However, it is well recognized that idiopathic facial nerve palsies more typically affect people aged 15 to 45.[4,7]

Bell's palsy is an idiopathic condition, however immunological, ischemia, and genetic variables are correlated with its pathogenesis. Recent studies have linked the reactivation of latent herpes virus in the geniculate ganglion and its migration to the facial nerve course[8,9]. The most prevalent neurotropic alpha herpes viruses are herpes zoster virus (HZV) and herpes simplex virus (HSV)[10]. These may stay dormant in the ganglia life-long. HZV's nerve-spreading satellite cells make it more aggressive.[11]

Primary, secondary, or tertiary causes of vascular ischemia have been reported. Diabetes mellitus increases the risk of primary ischemic neuropathy, which exacerbates neural inflammation[8,12]. Vasospasms can reduce blood flow and induce acute inflammation, resulting in rare primary ischemic neuritis[13]. Secondary ischemia increases capillary permeability, producing fluid buildup, edema, and nerve compression[14]. Hereditary fallopian canal narrowing affects 4-14% of people. This hereditary component is generally autosomal dominant and puts the nerve at risk of early compression even with mild edema[11,15].

History and Clinical Examination

When examining patients with acute facial palsy, it is of the utmost importance to obtain a complete medical history, paying special attention to the time course of onset, progression, recurrence, accompanying symptoms, oncological history, head trauma, subjective hearing loss, and recent travel.[16,17] Then, resting and moving facial nerve function will be assessed and compared to the contralateral side. First, the patient should attempt to raise their eyebrows to test the frontalis muscle. A central cause should be ruled out if the upper third of the face is spared. The examiner next instructs the patient to close their eyes lightly, then as hard as possible. Lagophthalmos is the inability to close the eye without effort. When prompted to close their eyes, clinicians may often see the eye rolling upward, also known as the Bell's phenomena. Severe lagophthalmos and the absence of Bell's phenomenon increase the risk of corneal exposure keratopathy, especially during sleep. To test zygomaticus major muscle function, the patient smiles without and with showing the teeth. Finally, the patient puckers and presses the lips together to test orbicularis oris muscle function and everts the lower lip to test lower lip depressors. Synkinesis movements, such as closing the eye with lip pucker, should be noted during these facial movements, as they play a pivotal role in evaluating aberrant reinnervation among neural fibers.[17,18] Patients may also experience changes in taste, sound sensitivity, otalgia, and tear and saliva production.[19]

The House-Brackmann Facial Nerve Grading System(displayed in table 1) may be used to evaluate facial nerve dysfunction. This grading scale ranges from I (no weakness) to VI (complete weakness).[17,20]

Table 1. House-Brackmann scale for facial paralysis

Grade	Severity	Features
I	Unaffected/Normal	Facial function is intact
II	Mild dysfunction	Notable muscular changes seen only during examination, with mild weakness, otherwise tone and symmetry are normal
III	Moderate dysfunction	Obvious dysfunction and barely perceptible asymmetry, associated with synkinesis movements, hemifacial spasms or contractures. Eye can be completely closed.
IV	Moderate-severe dysfunction	Asymmetry and weakness are easily noticed, cannot close eye, however symmetry and tone are maintained during rest.
V	Severe dysfunction	Barely perceptible movements. Asymmetry at rest
VI	Total paralysis	No movement is exhibited

It is useful for initial evaluations and decision-making strategies. However, it is not sensitive enough to identify subtle motor changes and cannot provide synkinesis-specific assessment. Ongoing clinical work aims to improve and standardize patients' evaluation through development of more precise grading systems assessing both static and dynamic activity, facial lesions distribution and occurrence of synkinesis.[17,21,22]

Diagnostic Workup

If the diagnosis is consistent with Bell's palsy, no lab or radiographic investigations are necessary. In the presence of any uncommon clinical pattern, individuals should be investigated for the underlying cause of their symptoms. To rule out neoplastic or infectious conditions, gadolinium-enhanced MRI of the whole parotid gland and the course of the facial nerve, and computed tomography of the temporal bone and neck are performed. MRI scanning is particularly beneficial for assessing the cerebellopontine angle and detecting intratemporal lesions that may be causing facial nerve compression. MRI scans may also reveal facial nerve enhancement surrounding the geniculate ganglion.[4,23,24]

Although electrophysiological tests are appropriate for prognosis, they are costly, time-consuming, and only beneficial for a short period of time (less than three weeks after symptom onset)[4,25]. Electroneuronography (ENoG) and electromyography (EMG) are useful methods for determining the reversibility of paralysis. EMG is usually used to rule out permanent atrophy, which can take up to 18 months, whereas ENoG predicts the progression of synkinesis[26]. Electroneurography (ENOG) is the most precise method for assessing the level of palsy, measuring the amplitude of muscular action potentials. After 2 weeks of paralysis, ENoG is no longer recommended since patients who fail to reach a degeneration threshold of >90% in the first 2 weeks have a favorable prognosis for recovery.[17,27] Electromyography (EMG) - detects facial muscle activation by identification of fibrillation potentials, signs of muscular denervation.[4] In patients with incomplete paralysis or early recovery, electrodiagnostic testing is not indicated.[17] Reinnervation surgery might be considered before complete muscle and nerve motor unit atrophy, in individuals who have failed to recover functionally within 6 months of clinical and electrophysiologic testing.[26]

Blood testing are recommended in recurring instances where autoimmune or infectious etiologies, such as HIV, are suspected. Serology testing are indicated in Lyme disease endemic locations.[4,17]

TREATMENT

General measures

In the acute phase of Bell's palsy, the goals of treatment include conservative measures for accelerating recovery and preventing corneal complications. Eye care includes eye patches and lubrication; lubricating drops should be applied frequently throughout the day, and an eye ointment should be applied at night.[4, 28]

Medical treatment

Because spontaneous recovery is common in Bell's palsy, medical treatment is still debatable, but certain medications can alleviate symptoms and improve recovery. A review of the literature comparing oral steroids to placebo reveals substantial evidence for the use of oral steroids in early facial palsy[29]. Oral corticosteroids should be administered within 72 hours after symptoms onset. Corticosteroids lower the possibility of an inadequate facial recovery. However, after fourteen days from symptom onset, no therapy will affect outcomes.[17,29,30]

There is significant evidence that antiviral monotherapy has no impact in Bell's palsy[31]. Nevertheless, a Cochrane analysis revealed that the combination of steroids and antivirals was superior than steroid monotherapy. The efficacy of antivirals after 72 hours from the onset of symptoms is debatable, except in immunocompromised patients, for whom it is advised.[30-32]

Nonpharmacologic treatments for Bell's palsy include physiotherapy, facial exercises, neuromuscular retraining, or acupuncture.[28] There is no evidence to support the use of physiotherapy or electrical stimulation in the early stages of facial palsy management.[33] It has no proven benefit; on the contrary, it may promote abnormal reinnervation and excessive motor unit recruitment, favoring abnormal movement patterns or synkinesis. Given that a proportion of facial paralysis patients develop facial muscle weakness and synkinesis, long term rehabilitation must focus on voluntary movement control and synkinesis limitation.[28,33-35]

Surgical treatment

Sir Harold Gillies noted in 1919 that the purpose of surgical reconstruction is to restore normal shape; this is done initially to regain function and later to address aesthetics. Concerning facial paralysis, the critical challenge is that there is currently no feasible method to recover the 17 facial expression muscles.[36] The primary surgical treatment objectives are: normal facial appearance at rest, symmetry with voluntary movements, corneal protection, symmetrical dynamic smile, restauration of oral, nasal and ocular sphincter control, emotional and volitional facial movement control and avoiding secondary morbidity.[37-38]

The treatment technique for facial paralysis generally differs depending on the severity of the paralysis. Numerous factors influence the decision to perform face reanimation, including the origin of nerve injury, the magnitude of the injury, the duration of paralysis, the patient's age and comorbidities, and expectations.[39] Establishing the mechanism (congenital vs. acquired) and timing–early, subacute and late is mandatory for surgical planning.[4,40] Static therapy techniques are beneficial for eye protection and preserving resting symmetry. Dynamic procedures, on the other hand, allow for functional reanimation and should be pursued wherever possible to allow for facial movements and psychosocial interactions.[41-43]

In most cases, Bell's facial palsy is temporary, with progressive improvement after two weeks and usually full functional recovery within three months.[19,44]

Acute facial paralysis, installed for a few weeks, is usually treated by facial nerve decompression or facial nerve repair. Nerve transfer procedures are used to treat intermediate length paralysis, which can last up to two years. Meanwhile, prolonged facial paralysis that lasts more than two years usually necessitates regional or free muscle transfer. Free functional muscle flap (FFMF) transfer has lately become the standard of care for persistent facial paralysis, with the gracilis being the most routinely used.[45,46]

Primary nerve repair

Primary neurorrhaphy provides the best functional outcome.[47] The indication for facial nerve repair is direct injury to facial nerve axons hence facial paralysis or near-complete facial paralysis (grade V or VI).[46] If the surgeon aims for direct repair, there are some principles that need to be kept in mind. Firstly, if the facial nerve is injured medially to the lateral canthus, there is no need for repair as the nerve arborization can compensate any functional deficits. The repair needs to be performed in the first 72 hours so the surgeon can properly identify the distal branches using nerve stimulation. However, if the soft tissues are severely damaged that it contraindicates the primary repair in the first 72 hours, the surgeon must mark the nerve stumps so they can be repaired in a delayed fashion. The frontal or the marginal mandibular branches of the facial nerve must be repaired all the time, because injury to these branches leaves the patient with severe muscle weakness with low chances of spontaneous recovery. Thus, direct repair is best performed at the time of injury and the results are most favorable when it is performed at least in the first year after the initial trauma.[26,48,49] When assessing the recovery after the procedure, the surgeon must keep in mind that nerve regeneration takes place with a rate of 1 mm/day.[50,51]

Facial nerve interposition grafts

An interposition non-vascularized nerve graft (greater auricular nerve, sural nerve, medial or lateral antebrachial cutaneous nerve, thoracodorsal nerve and the superficial radial nerve) is recommended when both stumps are available but they cannot be approximated for direct repair.[52] Gaps more than 1 cm require an interposition graft to provide a tension-free nerve suture.[43] The ansa cervicalis has also been evaluated as a donor nerve, and there is some evidence that motor nerve grafts are superior to sensory nerve grafts[53].

Some studies have advocated for the use of vascularized nerve grafts in circumstances when nerve regeneration is impeded by a large nerve gap, limited soft-tissue covering, or prior wound irradiation[54]. In the event of a short (8 mm) nerve gap repair, polyglycolic acid conduits and collagen tubes may offer an alternative to conventional nerve grafts.[26] The greatest probable outcome for either primary nerve repair or cable grafting is House-Brackmann Grade III facial function.[45]

Nerve transfers

Nerve transfer restores function to the recipient end-organ (skin or muscle) by coaptating a functioning donor nerve to a denervated recipient nerve.[55] Nerve transfers are indicated for patients with intact distal facial nerve branches and viable mimic muscles with permanently damaged intracranial and/or intratemporal segments. Nerve transfers should be conducted within two years of paralysis since beyond this period the nerve scar might obstruct conduction rendering the transfer useless. End-to-side coaptation determines less donor site morbidity than end-to-end transfers. Surgeons mostly use the hypoglossal and masseteric nerves[55]. Other donor nerves for cross-nerve anastomosis, such as the accessory nerve, and sections of the cervical plexus and ansa nervi hypoglossi, create greater donor site morbidity and yield poorer outcomes.[37, 56]

Hypoglossal nerve transfer is useful for immediate reconstruction, when the proximal stump of the facial nerve is lost through tumor resection. Its advantages are the good muscle tone, a normal looking aspect in the resting state, eye protection and the possibility of voluntary facial movements. However, it does not allow for spontaneous facial expression and, as expected, the ipsilateral side of the tongue will become paralyzed and atrophied. Even though the consequences on the ipsilateral side of the tongue are usually well tolerated, this rests on the presumption that the other inferior ipsilateral cranial nerves (IX, X, XI) are functional. In such cases with dysfunctional inferior ipsilateral cranial nerves or when the patient is not willing to accept ipsilateral tongue paralysis, **the hypoglossal jump-graft** is a suitable solution. The procedure involves end-to-side neurorrhaphy between the hypoglossal nerve and a donor nerve graft (e.g. the great auricular nerve) that serves as a jump graft to the facial nerve's main trunk. The hypoglossal-facial jump nerve anastomosis involving only a section of the hypoglossal nerve prevents tongue atrophy, and its success rate is equivalent to that of the traditional procedure[37,45,56,57]. Terzis established a novel approach in face reanimation with the **"babysitter" procedure**. Coaptation of 40 percent of the ipsilateral hypoglossal to the facial nerve on the afflicted side is done simultaneously with cross-facial nerve grafting and subsequent micro-coaptations after 8 to 15 months. The Baby-Sitter procedure allows for motor plaque preservation on the paralyzed side until the cross-face graft reinnervation.[57]

Masseteric nerve transfer from the trigeminal nerve is useful for immediate reconstruction when the proximal stump of the facial nerve cannot be used. Its popularity has recently increased because it provides consistent anatomy, enough axons for good excursion of smile and has low morbidity. Patients can grin after roughly six months by activating the masseteric nerve when biting down. After years of rehabilitation and retraining, clinical findings indicate that masseteric nerve transfer can result in a spontaneous smile. Hypothetically, this is due to neural plasticity as a result of the extensive cortical overlap between the representations of facial and masseter muscles.[58]

Cross-Facial Nerve Graft (CFNG)

When the proximal nerve stump is unavailable, the contralateral unaffected nerve stump might be utilized as a donor nerve, supplying motor axons to the afflicted side. Most frequently, sural nerve grafts are used to connect the healthy peripheric nerve branches to the those corresponding to different muscle groups on the paralyzed side. According to Terzis et al.[56] the best results from cross facial nerve grafting occur when the time of denervation is less than six months. The surgical process consists of two stages. A modified pre-auricular facelift incision is made on the normal, functional side of the face during the first stage. After elevating a skin flap anteriorly to the level of the lateral canthus, the superficial muscular aponeurotic system (SMAS) layer is pierced anterior to the parotid gland, and a nerve stimulator is used to identify the facial nerve branches. Depending on the desired function and the mapping of innervation targets for each branch, nerve branches to be sacrificed are chosen with care. The proximal end of the inverted sural nerve graft is thereafter coapted to the donor facial nerve branches under magnification. The distal end of the CFNG is tunneled subcutaneously from the donor side of the face to the paralyzed side of the face via a incision in the upper lip. When the regenerated axons reach the distal end of the cable graft, the second stage is performed. In adults, the average time between the two stages of a surgery is between 8 and 12 months. Cross facial nerve grafting may be combined with free muscle transfer for smile reanimation if the duration of denervation exceeds two years.[45,59,60]

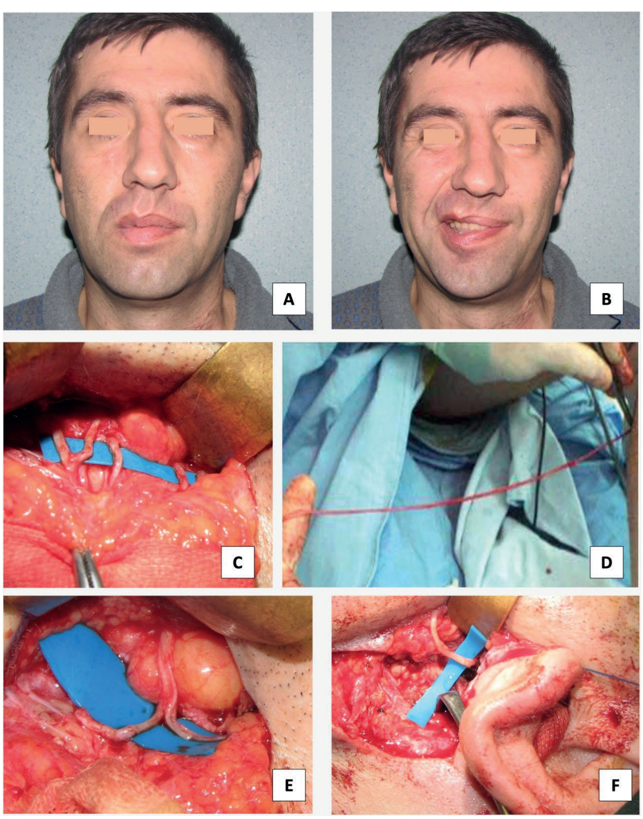

Figure 1. Facial paralysis reanimation using cross-nerve technique: A-B: preoperative clinical aspect; C: Healthy contralateral donor site D: Sural nerve graft harvest E-F: nerve graft coaptation

MUSCLE TRANSPOSITION

Regional muscle transfer may be used when the mimic muscles have been atrophied for such a long time that they no longer have any reinnervation potential whatsoever. Temporalis and masseter muscles are most commonly employed for facial reanimation because their blood supply and innervation give ample arcs of rotation without significant risk of necrosis or denervation. In order to create coordinated motions, retraining and biofeedback are required. Nevertheless, muscle transposition is an efficient technique that yields rapid and satisfying results.[55]

Masseter muscle transfer is suitable for reanimating the inferior half of the face. The technique uses 3 muscle fascicles, which are then sutured to the upper lip, the labial commissure and the inferior lip, respectively. When radical parotidectomy is performed, masseter muscle transposition with interdigitations in the recently denervated mimic muscles provides maximal myoneurotization.[61,62]

Temporal muscle transfer is more frequently used than masseter muscle transfer, because it provides a larger range of movement and it can more easily adapt to the orbit. The temporal muscle and its fascial extensions can be sutured to the eyelids, the nasal alae, the buccal commissure and the upper and lower lips. The Labbe technique can be employed in order to provide a smiling function using a lengthening myoplasty of the temporalis muscle as a dynamic procedure.[26,63]

Free Muscle Transfer

With the introduction of microvascular free tissue transfer, the field of face reanimation has evolved tremendously. Free muscle transfer may be used if the native facial musculature has been resected or denervated, when trigeminal nerve dysfunction precludes the use of regional muscle transfer and as the sole method of producing involuntary smile reanimation when combined with cross-face nerve grafting. Usually, the donor muscles are the gracilis or the extensor digitorum brevis, but the surgeon can opt for the latissimus dorsi, the last 3-4 digitations of the serratus anterior, the pectoralis minor, the tensor fasciae latae or the rectus abdominis. Gracilis muscle remains the workhorse of free muscle transfer for face reanimation. It is a thin, long muscle located in the middle of the thigh which enables a two-team approach, a facile harvest and owns an ideal neurovascular pedicle.[64,65]

Several donor nerves have been documented in the literature, including the cross facial nerve graft (CFNG), the masseteric nerve, the hypoglossal nerve, and the spinal accessory nerve, as well as their combinations. The CFNG has the capacity to induce a spontaneous and synchronized smile. However, the oral commissure excursion created is inferior to those of other donor nerves. The masseteric nerve has a larger axonal load and a more dependable oral commissure excursion, but at the sacrifice of spontaneity.[59,66-68]

Typically, the gracilis muscle transfer for unilateral facial paralysis is completed in two stages using a cross face nerve graft as a neural input. On the healthy side, the proximal ends of the donor facial nerve branches are sutured to the sural nerve graft under the surgical microscope. The distal end of the nerve graft is anchored near the recipient vessels with a colored, non-resorbable suture, which can be easily identified in the second surgery. Vascular anastomoses are performed to the facial vessels or to the superficial temporal vessels. The obturator nerve to the gracilis muscle will be coapted to the distal end of the sural nerve graft (Figure 2)

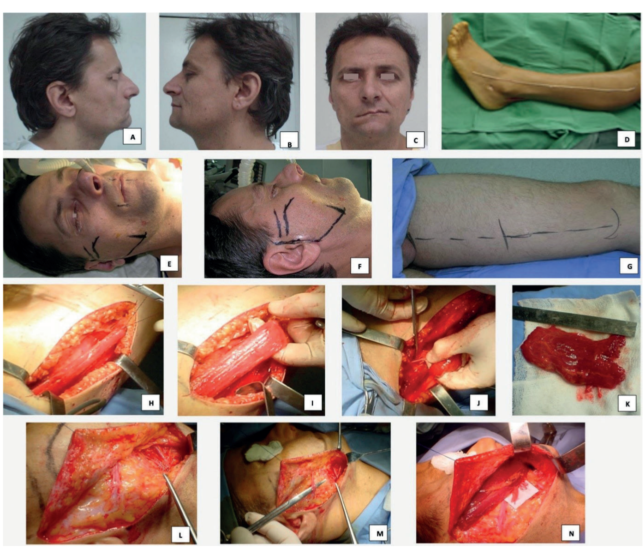

Figure 2. Surgical technique of facial reanimation using gracilis muscle free flap transfer

Figure 3. Surgical technique of facial reanimation using extensor digitorum brevis free flap transfer

To enhance oral commissure excursion and spontaneity, dual innervation of the gracilis with the masseteric nerve and contralateral facial nerve via a CFNG was later proposed.[69] Dual innervation with a single stage free gracilis muscle transfer is considered to be as efficient as the conventional two-stage CFNG free gracilis transfer, with the added benefit of enhanced dynamic smile symmetry.[70]

More recently, the gracilis flap was designed safely as a double paddle muscle flap for multivector face reanimation. The multivector gracilis flap design improves all components of the smile display and has the ability to produce periorbital wrinkling characteristic of a Duchenne smile. With a multi-vector flap design, attempts have been made to generate a genuine smile beyond the basic movement obtained by a single vector facial reanimation.[71]

STATIC PROCEDURES FOR FACIAL REANIMATION

There are substantial benefits to static procedures in facial reanimation that can give an alternative or enhance the outcomes of dynamic facial reanimation. Static techniques can be used in both chronic and temporary facial paralysis when nerve recovery is anticipated. The indications for static reconstruction are: elderly patients with significant comorbidities, extensive facial defects secondary to trauma or tumor resection, failed microsurgical facial reanimation, functional disabilities (for which the aims of the procedures are to protect the cornea, to improve the nasal airways and to prevent salivation) and symmetry improvement in the resting state.[72,73]

Correcting brow ptosis is an important component in treating patients with facial paralysis. Several procedures have been described: endoscopic brow lift, direct brow lift (coronal, mid-forehead, brow incision), or minimally invasive temporal brow lift. Oculoplastic management of the paralyzed eye is critical because exposure keratitis can result in permanent vision loss. Placement of an eyelid weight: lid loading with a gold or platinum weight is a very efficient procedure for correcting lagophthalmos. Palpebral spring technique (Morel-Fatio) is a technically demanding procedure that can be used in place of an eyelid weight. Upper eyelid blepharoplasty can be used in individuals with severe dermatochalasis.[45,74]

Lateral tarsorrhaphy implies mattress sutures used to coapt the lateral aspects of the upper and lower lid tarsal plates in order to conduct a "reversible" lateral tarsorrhaphy. Lateral tarsal strip procedure is an effective approach for treating paralytic lower lid ectropion. Static facial slings are often inserted from the zygomatic arch/temporalis fascia to the oral commissure and nasolabial fold to provide face support. A variety of materials, including fascia lata, Gore-Tex, and AlloDerm, have been proposed for use as sling materials. A fascia latae sling from the alar base to the zygoma/temporalis fascia was used to stent open the external nasal valve after nasal valve collapse.[45] Synkinetic lower lip depressor muscles can be treated by ipsilaterally weakening the depressor anguli oris muscle with botulinum toxin injections, marginal mandibular nerve neurectomy, or depressor labii inferioris myomectomy.[26]

Proposed Therapeutic algorithm in facial nerve paralysis

A step-by-step therapeutic algorithm was compiled in order to simplify and facilitate decision making process providing favorable functional and aesthetic outcomes for patients with facial paralysis, as presented in Figure 4.[4,26,37,45,75,76]

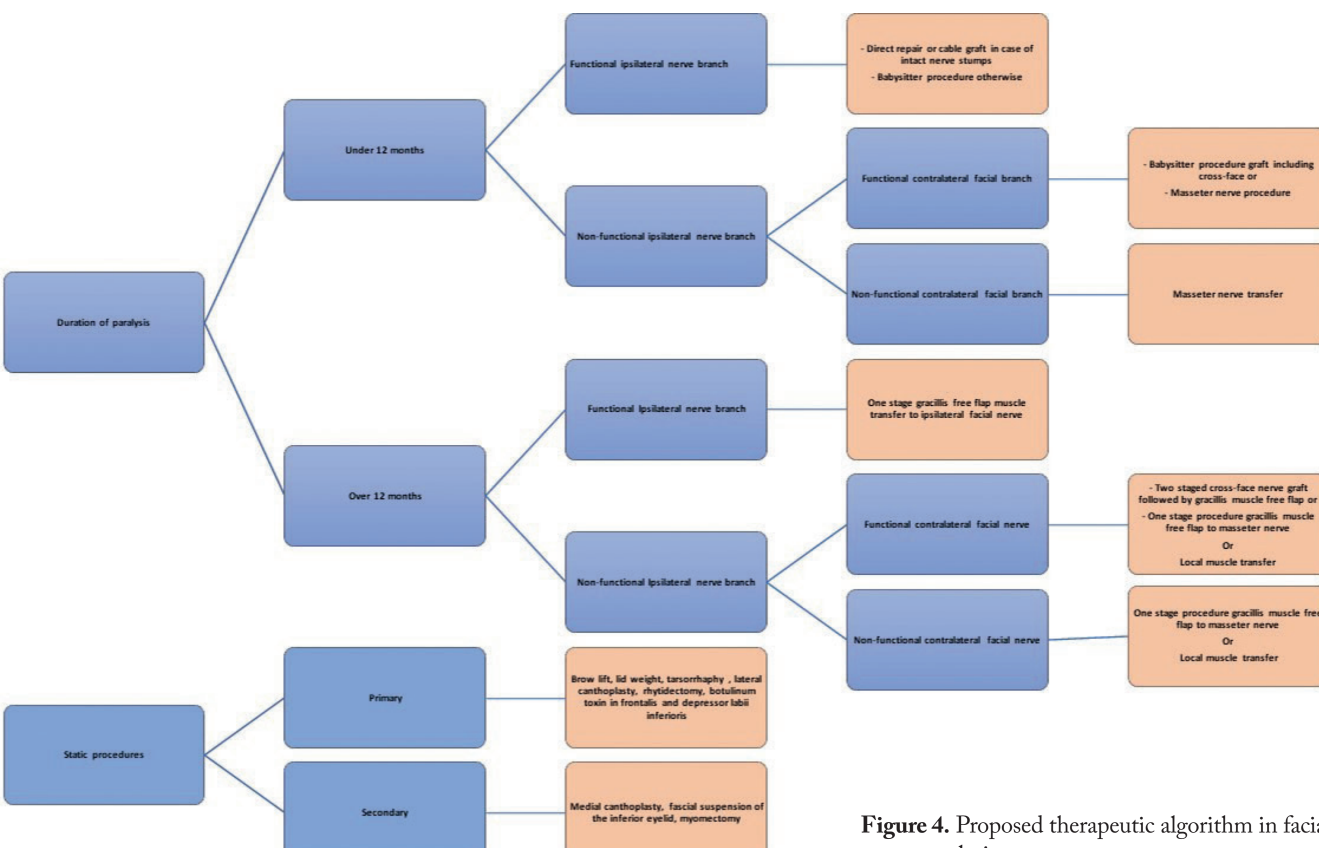

Figure 4. Proposed therapeutic algorithm in facial nerve paralysis

CONCLUSION

The facial nerve is a fundamental structure both for communication and abillity to express emotion, therefore, functional impairment can lead to a significant alteration in the quality of life.A thorough clinical examination coroborated with specific diagnostic tests ensure the correct assessment of the extent of the lesions and prognosis in facial paralysis. In the majority of cases, Bell's facial palsy is temporary, with progressive improvement and usually full functional recovery. The surgical treatment technique for facial paralysis generally differs depending on the severity of the paralysis. Numerous factors influence the decision to perform facial reanimation, including the mechanism of nerve injury, the extension of the injury, the duration of paralysis, the patient's age, comorbidities, and also patient 'wish. Establishing a correct therapeutic strategy based on severity and timing of the paralysis onset is mandatory for surgical planning. Static therapy techniques are beneficial for eye protection and preserving resting symmetry. Dynamic procedures, on the other hand, allow for functional reanimation and should be pursued wherever possible to regain the facial movements, restoring psychosocial interactions and quality of life.

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