Overview of Facial Nerve: Injury, Repair and Regeneration

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Abstract

Facial nerve injury is a common clinical entity; it may arise from congenital, infectious, idiopathic, traumatic, neoplastic, endocrine, neurologic, and systemic causes. It is also not uncommon for nerve to be deliberately sacrificed at time of surgery in order to completely remove malignancy**.** Consequences of this injury can be devastating and include exposure keratosis and blindness, facial asymmetry, poor nasal airflow, oral incompetence, psychological stress and synkinesis, in which miswiring of nerves results in synchronous involuntary muscle movement with expressions. The main drawback of nerve repair is its inability to guarantee complete functional recovery. For example, axonal misalignment can cause partially reversible neuronal atrophy, which can interfere with the production of neurotrophic factors for accelerated regeneration. Management of facial nerve injuries continues to be one of the most difficult issues faced by the surgeons. It follows a complicated algorithm depending on the mechanism and the location of the injury, the time course of the paralysis, the medical condition and prognosis of the patient. Although many techniques have been developed to rehabilitate patients with facial paralysis (FP), the outcome of all of these procedures lacks the symmetry or spontaneity of an intact facial nerve. Therefore, the aim of the present study was to review facial nerve injury, facial nerve repair and regeneration.

Keywords: Facial nerve injury; Repair; Management; Regeneration

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Introduction

The facial nerve is derived from the second branchial arch. The second branchial arch also produces the muscles of the face, the occipitofrontalis muscle, the platysma, the

stylohyoid muscle, the posterior belly of the digastric muscle, the stapedius muscle, and the auricular muscles (1).

The facial nerve innervates the muscles derived from the second branchial arch and carries sensory and parasympathetic fibers of the nervus intermedius (2).

Detailed and precise knowledge of the topographic relationships and fascicular organization of the facial nerve is necessary for anyone undertaking microsurgery for treatment of facial nerve injuries (3).

For full restoration of nerve function, the facial nerve must regenerate and expand from the facial motor nerve while maintaining its function, and ultimately must connect to the damaged terminal area (4).

If a neuronal cell body is destroyed, it can no longer survive. However, if a portion of the axon is amputated, the neuron can regenerate the axon; moreover, under appropriate conditions, the cell may also restore its synaptic function. The change in nerve fibers after nerve damage depends on the degree of damage, with local demyelination and remyelination occurring for minor damage such as neuropraxia and axonal degeneration, and regeneration for severe damage (5).

❖ **Facial nerve healing and regeneration:**

The Wallerian degeneration process usually begins 24–36 h after the initial injury, at which point axons start to disintegrate and Schwann cells at the distal end release growth factors. Thereafter, calcium influx occurs continuously throughout the cytoplasm and mitochondria via action of the distal pump. Influx of calcium triggers endogenous proteolysis and degeneration of the cytoskeleton. As the process continues, axons start to collapse and Schwann cells lose their myelin sheath (6).

Furthermore, after a few days, Schwann cells de-differentiate owing to their lost connection with axons, starting a vigorous proliferation. Galectin-3 is known to play a key role in activating myelin phagocytosis. In this process, macrophages and Schwann cells are promoted to degrade myelin, thus having a major importance in the degeneration process (7).

Many cells, including macrophages, are also recruited and serve to remove degenerated axons and myelin debris. A number of factors have been demonstrated to participate in this process, including pro-apoptosis factors, neurotrophic factors, and growth-associated protein (GAP)-43 (8).

Both types of Schwann cells, the pre-existent and the recently produced Schwann cells, align together to form the bands of Bungner, which are highly aligned fibers formed by the basal lamina of the Schwan cells. These bands are key topographical cues responsible for guiding the axon and their growth cones, from the proximal to the distal site, across the gap (9).

Metabolic changes in the cell body also increase synthesis of mRNA, enzymes, and protein that support axon regeneration. Genes encoding proteins such as GAP-43 and cytoskeletal proteins are upregulated **(10).**

There is upregulation of glial derived neurotrophic factor and brain-derived neurotrophic factor which drive axonal regeneration **(11).**

Following higher grades of axonal injury, the proximal nerve cell bodies undergo several gene expression changes to adopt a regenerative phenotype. This includes the upregulation of actin, tubulin, and GAP43 proteins **(12).**

A growth cone is formed at the end of the proximal stump. Filopodia are formed at the growth cone and function to sample the surrounding milieu as the growth cone proceeds towards the distal nerve stump. Growth cones are directed through the stroma and surrounding tissue by a variety of chemo-attractants and repellants **(13).**

Schwann cells also produce factors that guide the growth of the regenerating axon through the bands of Bunger towards the distal stump target. Regeneration of axons into wrong endoneurial tubes and end organs leads to worse functional outcomes **(Figure 1).** Once the regenerated axon has reached its target, the Schwann cell reverts to supportive phenotype and assists with re-myelination of the axon **(14).**

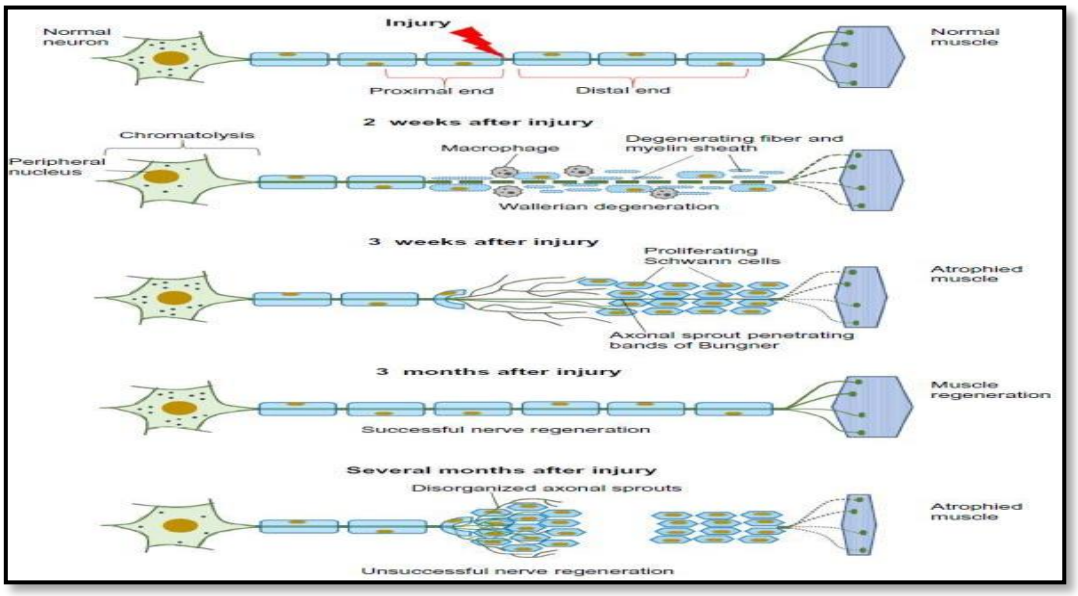

Fig. (1): Cellular responses to nerve injury: degeneration and regeneration [14].

Overall, Schwann cells affect peripheral nerve regeneration in three distinct manners: (i) proliferation, (ii) development of bands of Bungner, and (iii) secretion of adequate growth factors **(15).**

If axons do not regenerate, the Bungner band contracts, Schwann cells decrease in number, and the damaged area is uniformly filled by connective tissue **(16).**

Many observations regarding factors influencing successful nerve regeneration have been made. The distance of a nerve lesion from its targeted tissue has a significant impact on

recovery. The further the injury is from the target tissue the lower the chance of meaningful regeneration. Studies of irradiated nerves demonstrate a slowed but capable regenerative capability. As previously mentioned, the degree of nerve injury has a major impact on success of nerve regeneration **(12).**

Younger age is a known independent factor in improved outcome. This can be summarized by decrease and delay in Wallerian degeneration, decreased neurotrophic factor production and effects, increased transport time along the axon, and decreased substrate production for axonal regrowth as age increases. The result is a smaller, slower, less functional nerve in older patients **(17).**

❖ **Facial nerve injury and repair**

Nerve injuries can be classified into five degrees. First-degree injury refers to an undisrupted nerve with neurapraxia; second-degree injury involves Wallerian degeneration of the nerve axons but leaves the surrounding membranes intact; and the third- through fifth-degrees of injury reflect partial or complete transection of the nerve with the loss of endoneurial, perineurial, and epineurial tubes, respectively **(18).**

Non degenerative neuropraxia from blunt trauma will not need surgical reconstruction, whereas disruption leading to degenerative neurotmesis likely does require surgical treatment **(19).**

Early cases are typically managed in the first year after injury through the restoration of neural inputs to the same existing facial muscles with acceptable results **(20).**

Patients with acquired paralysis who underwent serial clinical and/or electrophysiologic testing and who failed to demonstrate any functional recovery by 6 months can be considered for a reinnervation procedure before complete muscle and motor end plate atrophy **(21).**

It is well known that a longer denervation time and advancing patient age impede nerve regeneration. Generally, there is no clearly defined, universally applied cutoff limit in regard to denervation time **(22).**

▪ ***Options for nerve reconstruction:***

Three options exist for nerve reconstruction: (1) primary tension-free coaptation of the nerve segments, (2) Cable grafting between the proximal and distal nerve stumps, and (3) Nerve transfer, which is indicated when the proximal segment of the facial nerve is not available **(23).**

• **Direct repair:**

End-to-end repair is the simplest form of nerve repair and can be accomplished by suturing the severed end of a nerve back together. This technique is appropriate when the

ends of the nerve can be reapproximated in a tension-free manner. If needed, the nerve can be mobilized to allow for a tension-free closure **(24).**

Primary tension-free coaptation of the nerve segments is possible within 72 h from the onset of the lesion and is known to bring the best results possible **(25).**

Attachment of nerve endings can be accomplished with microsurgical sutures or using fibrin adhesives which allow for a shorter procedure and potentially similar outcomes when compared with suturing **(26).**

- **Nerve grafts:**

If the nerve gap is larger than 5-mm, then primary repair may be suboptimal, as excess tension reduces axonal diameter, places ischemic stress on the nerve, and is associated with worse functional outcomes **(27).**

Nerve grafting involves harvesting a portion of a nerve from a distant site to bridge a gap between 2 nerve endings. A nerve graft provides an ideal environment for axon regeneration. The ends of the nerve graft can be sutured to the native nerve endings or attached with a tissue adhesive **(28).**

An alternative to nerve grafting is the use of biologic (e.g., veins) or synthetic conduits (made of polyglycolic acid or collagen). Like a nerve graft, the aim of these conduits is to provide a protected environment for axon regeneration **(29).**

Nerve grafting may be an appropriate technique if nerve damage or resection results in a substantial gap between the 2 ends of the nerve, precluding a tension-free end-to-end repair. It is also appropriate to perform multiple separate grafts when multiple branches of the facial nerve are sacrificed **(28).**

Historically, surgeons utilized a variety of donor sites to obtain donor nerves for grafting. Common options include the sural nerve, greater auricular nerve, ansa cervicalis nerve, medial antebrachial cutaneous nerve, and nerve to the vastus lateralis **(26).**

While the graft should have similar cross-sectional area to the recipient nerve, additional length or altered geometry of the autograft does not appear to have an influence. Reversing polarity of the cable graft has been theorized to influence nerve regeneration, however, this has not borne out in animal models through functional, histologic, or electrophysiologic parameters **(30).**

Current considerations for selecting an autograft include donor site morbidity (loss of sensation, pain, paresthesia, or loss of motor function arising from removal of a nerve for use in grafting), branching pattern, length of nerve required to reconstruct the defect, caliber-match of the nerve, ease of harvest, and need for incisions **(31).**

- **Nerve Transfer:**

Nerve transfer entails recruiting an uninvolved, intact nerve, and attaching it to the distal branches of the damaged nerve **(Fig. 2)**. The goal is to provide alternate cortical input

to the mimetic musculature. For repair of the facial nerve, three nerves are most frequently recruited: the hypoglossal nerve, masseteric nerve, and the contralateral facial nerve **(32).**

There exists a critical time window after nerve injury during which the damaged facial nerve can be augmented through connection to another intact nerve. This allows for reinnervation of the native facial muscles but requires sacrifice of an alternate cranial nerve **(33).**

Sacrifice of an intact nerve to reinnervate the distal portion of the facial nerve may result in secondary motor deficits and functional deficits. Various techniques have been developed to reduce secondary morbidity by partial preservation of the hypoglossal nerve **(34).**

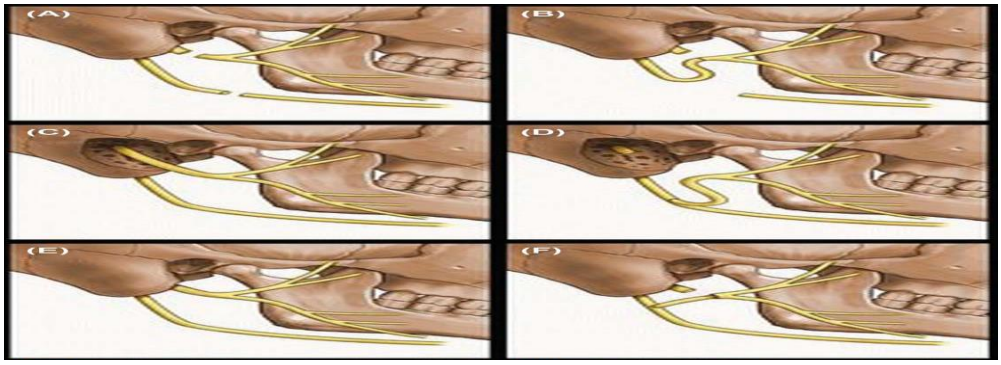

Fig. 2: Methods for facial reanimation with nerve transfers. Schematic drawing illustrating the lateral view of a classic hypoglossal-facial neurorrhaphy (A and B), hemihypoglossal to facial direct transfer (C and D), hemihypoglossal to facial nerve transfer with interposed jump graft (E and F) [32].

Masseteric nerve transfer offers many advantages including faster onset of movement, ease of dissection, and less morbidity without disturbance of masticatory muscle function. The mean onset of facial motion occurs approximately 6 months after surgery. The lack of donor site morbidity is attributed to the functional overlap of the muscles of mastication **(35).**

The contralateral facial nerve is also used for motor reinnervation. This technique is accomplished by using nerve grafts (usually the sural nerve), passing them across the face, and attaching them to branches of the damaged facial nerve **(36).**

The key advantage of this technique over other nerve transfer procedures is the ability of the contralateral facial nerve to allow for spontaneous mimetic motion and emotional expression. Limitations of the technique include in consistent results and the significant amount of time required for axons to traverse the length of the graft **(37).**

The denervated muscles are at risk of atrophy. Temporary "babysitter" nerve grafts may be used in conjunction with cross-facial nerve, providing motor input into the denervated nerve while the patient awaits reinnervation from the contralateral facial nerve. In the "babysitter procedure," a temporary anastomosis is created with other motor nerves (such

as the aforementioned hypoglossal or masseteric nerves) allowing for increased strength of contraction of the affected muscles **(38).**

- ***Territories of facial nerve injury and management options:***

During the evaluation of a patient for facial reanimation, it is paramount to determine the underlying cause, the exact extent of injury, time since onset, the viability of facial musculature,the presence and state of the facial nerve, associated cranial nerve deficits, the patient's overall health, and the patient's expectations and goals for rehabilitation **(39).**

Amer's classification is a recent reliable system which classifies facial nerve injuries into 13 territories **(Table 1).** The facial nerve is divided into 3 segments; the main trunk (T), the divisions (D) and distal branches (B). The trunk zone is further subdivided into two groups: T- when no proximal trunk is available and T+ when proximal trunk is available. The distal branches are also subdivided into B+ and B-, based on the presence or absence of distal branches. Therefore, each explored facial nerve system is designated by two letters, the first indicates the availability of proximal healthy fascicles for repair and the second indicates the availability of healthy distal fascicles for repair **(40).**

Table 1: Amer's classification of territories of facial nerve injury and management options [(40)].

Territory	Availability of healthy fascicles for repair		Options of repair
	Proximally	Distally	
T-/T+	Not available	Main trunk	▪ Direct hypoglossal facial repair. ▪ Nerve transfer for smile reanimation and cross face nerve graft for eye closure.
T-/D	Not available	The divisions	▪ Direct hypoglossal nerve grafts to the two divisions. Either end to end or side to end. ▪ Graft from either the split hypoglossal nerve or Masseteric nerve for smile reanimation and cross face nerve graft to the eye.
T-/B+	Not available	The branches	▪ Grafts from the hypoglossal nerve to all branches. ▪ Nerve transfer for smile reanimation and cross face nerve graft for eye closure.
T-/B-	Not available	Not available	▪ Free flap for the smile and gold plate and tarsorrhaphy for the eye

T +/T+	Main trunk	Main trunk	▪ Direct repair. ▪ Nerve grafts if with gap.
T +/D	Main trunk	The divisions	▪ Direct repair. ▪ Nerve grafts if with gap.
T +/B+	Main trunk	The branches	▪ Nerve grafts
T +/B-	Main trunk	Not available	▪ Direct neurotization
D /D	The divisions	The divisions	▪ Direct repair. ▪ Nerve grafts if with gap.
D /B+	The divisions	The branches	▪ Nerve grafts
D /B-	The divisions	Not available	▪ Direct neurotization. ▪ Free flap for smile restoration
B +/B+	The branches	The branches	▪ Direct repair. ▪ Combined direct repair and grafts. ▪ Grafts only.
B +/B-	The branches	Not available	▪ Direct neurotization. ▪ Direct neurotization for eye closure and free flap for smile restoration

The selection of the best strategy for management and results of reconstruction of early cases vary dramatically due to various factors. The site and extent of facial nerve injury are two of the most important factors that determine the strategy used for repair and expected results. Therefore, knowledge of the exact territory of facial nerve injury is needed to design a proper strategic plane and choose the most appropriate technique for reconstruction **(40).**

- **Eye protection:**

Denervation of the orbicularis oculi muscle can lead to serious ocular complications involving dryness; irritation; a foreign body sensation; epiphora; and the long-term risk of corneal ulceration, infections, exposure keratitis, and possible vision loss **(41).**

In order to correct lagophthalmos, a thin-profile platinum weight can be safely placed under local anesthesia to improve eyelid closure without obstructing vision. Lower lid ectropion can be managed with a lateral or medial canthopexy and tarsal strip suspension. A minimally invasive brow lift allows one to permanently treat brow ptosis, which can obstruct vision and impede eye irritation **(42).**

Conclusion:

The regeneration of peripheral nerves is slow and usually incomplete.Therefore, peripheral nerve repair is less successful over long distances.

The process for peripheral nerve regrowth is complex and requires molecular, cellular, and microenvironment changes.

In general, the primary paradigm for managing damaged facial nerve has been early microsurgical repair and restoration of nerve continuity.

Facial nerve reconstruction is a unique and formidable challenge demanding an individual approach.

Reinnervation can provide satisfactory outcomes up to 12 to 18 months after denervation; however, the final functional and esthetic result is strongly dependent on the age and type of the procedure.

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Author contribution: Authors contributed equally in the study.

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