

Revision Facial Nerve Surgery

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Transection of the facial nerve can result from blunt or penetrating trauma to the face or temporal bone. It can also occur accidentally during surgery, or as a planned surgical procedure carried out in the interest of eradicating disease. If transection is recognized at surgery, direct anastomosis or cable grafting is the procedure of choice. Immediate nerve repair offers the best chance for recovery, which usually begins within 9 to 12 months [1]. What if at the end of this period there is neither clinical nor electrical evidence of recovery? A report by May and colleagues [2] discussed the investigation and management of such a case; however, this specific problem has not been reviewed in the English language since then. This article presents two similar cases with poor outcomes. The authors review current understanding of the immediate and long-term changes that occur in the neuron, axon, and muscle after injury to the nerve, and the underlying pathology that led to graft failure. They also evaluate the applicable surgical options described in the literature and the diagnostic test results that help in selecting the most appropriate surgical procedure in these cases.

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Materials and methods

Patient # 1

A 20-year-old man sustained a blow to the left side of his head, and, as a result, he lost consciousness for several hours. In the emergency room he was noted to have a left infranuclear facial paralysis and bleeding from the ipsilateral ear. The patient was confined to the intensive care unit for an extended period because of other serious body injuries. He sought consultation for his left facial paralysis and hearing loss 2 months after the original injury. Physical examination of the head and neck showed no abnormality, except for a complete left infranuclear facial paralysis. The Rinne was negative on the left and the Weber lateralized to the left. An audiometric evaluation confirmed a conductive hearing loss on the left side. A CT scan of the temporal bone showed an otic capsule sparing longitudinal fracture of the left temporal bone. In an effort to understand the nature of the lesion causing the paralysis, in the face of a CT-scan-confirmed intact fallopian canal, MRI with contrast of the brain and facial nerve were obtained. The MRI was read by the neuroradiologist as showing an intraneural hematoma at the geniculate ganglion. The facial nerve was decompressed by way of a transmastoid-subtemporal approach. The incus was found to be dislocated and was therefore removed. To access the geniculate ganglion, the head of the malleus was amputated. Examination of the fallopian canal from the meatal foramen to the second genu failed to show spicules or a fracture line traversing the canal. The fallopian canal was opened from just distal to the meatal foramen to the second genu, and, in view of the findings noted on the MRI scan, this segment of the nerve was excised. A great auricular nerve graft was approximated to the two ends of the transected facial nerve. No sutures were used to stabilize the anastomoses, and no glues were used at the anastomotic sites. A malleus-stapes assembly was placed through the facial recess and the incision closed. Two months later, an audiogram showed closure of the air-bone gap to within 15 dB of bone scores. Histologic examination of the nerve specimen showed dense fibrosis in the geniculate ganglion.

When the patient returned 6 months later, there was no clinical or electrophysiologic evidence of recovery. His cornea was reported normal by the ophthalmologist, and with eye closure a good Bell's phenomenon was noted. The patient returned for a follow-up examination 3 years later and an evaluation of facial nerve function showed a complete paralysis on the left side. He did not wish to undergo any electromyography (EMG) studies on this visit; nor did he agree to any further surgery.

Patient # 2

Over the course of 4 years, an 18-year-old woman developed a progressive right-sided facial paralysis. She did not admit to any loss of hearing in the right ear, or to any loss of taste, or tearing. During the course of a pregnancy

6 months earlier, she had noted a more rapid progression of the facial paralysis. Examination of the head and neck showed a complete right infra-nuclear facial paralysis but no other abnormalities. Tuning fork tests showed positive Rinne on both sides and the Weber lateralized to the right. A CT scan of the temporal bone (Fig. 1A,C) and an MRI brain scan (Fig. 1B,D) confirmed a tumor in the region of the geniculate ganglion, thought to be a facial nerve schwannoma. An EMG showed fibrillation potentials in all areas of the face. In May 2002, the tumor was resected via a right transmastoid-subtemporal approach with primary cable grafting using the great auricular nerve. No sutures were used to anchor the graft. The extent of the tumor mass was judged visually, rather than with frozen

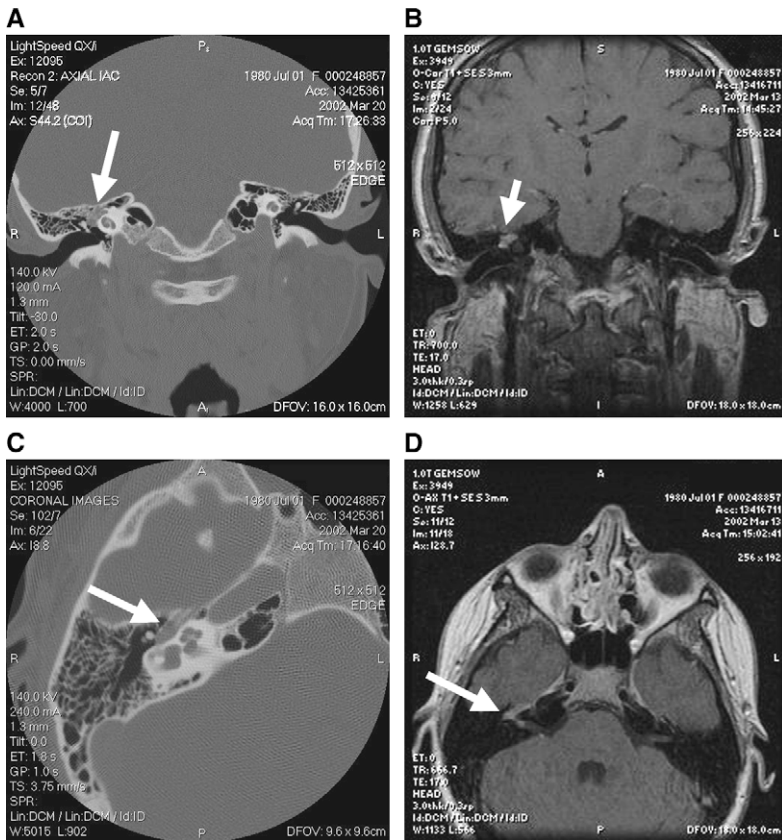


Fig. 1. (A) and (B) Preoperative coronal CT and MRI for patient #2. MRI image, T1WI with IV contrast demonstrates enlargement and homogeneous contrast enhancement of the geniculate ganglion of the right facial nerve (*small arrow*). Coronal noncontrast CT demonstrates a soft tissue mass expanding the bone surrounding the geniculate ganglion of the right facial nerve (*large arrow*). (C) and (D) Preoperative axial CT and MRI for patient #2 demonstrate enlargement of the region of the geniculate ganglion and tympanic segment of the right facial nerve canal (*arrows*).

section. The ossicular reconstruction was completed using a Total Ossicular Replacement Prosthesis (TORP). Her postoperative recovery was unremarkable, and an audiogram done 3 months later showed a 10 dB conductive hearing loss. The pathology report confirmed a diagnosis of “meningothelial and psammomatous meningioma.” This diagnosis was reached by two independent pathologists at two different institutions.

Three years later she still has a complete right infranuclear facial paralysis. In addition, there is blephroptosis on the right side, a wider palpebral fissure on the right, inadequate closure of the right eye, collapse of the nasal ala on the right, and inadequacy of the oral sphincter upon puffing the cheeks (Fig. 2). These observations correspond with the patient’s complaints of drooling liquids from the angle of the mouth, and excessive tearing. An ophthalmologic consultation reported no corneal ulcers and a good Bell’s



Fig. 2. Facial function 3 years postoperatively for patient #2.

phenomenon. An EMG study showed no motor unit action potentials, and “clear insertional activity, fibrillations and positive sharp waves.” An MRI scan showed a recurrent tumor in the right labyrinthine segment of the nerve (Fig. 3). A CT scan of the right temporal bone showed postsurgical changes and a “mild to moderate enlargement of the right facial canal, particularly in the labyrinthine segment with a focus of increased density... most likely hyperostotic changes compatible with the known recurrent perigenicular meningioma.”

Discussion

Acute changes in neuron, axon, and muscle after nerve injury

An understanding of the postinjury changes that occur in the facial nerve neuron, its axon, and the muscles innervated by it is important for understanding the rationale for the current rehabilitative algorithms. Within 1 to 2 days of injury to the nerve, its cell body swells, its nucleus moves peripherally, and there is loss of Nissl substance (chromatolysis); this activity reaches its peak in about 2 weeks (Fig. 4). Electron microscopic studies have shown that the number of cellular organelles also increased [3]. These neuronal changes are accompanied by changes in the microenvironment of the facial nucleus. Within 24 hours, microglia, which are phagocytic cells of the central nervous system, are stimulated, move into direct contact with injured neurons, and displace synaptic input. This phenomenon is known as *synaptic stripping* [3]. Astrocytes, which provide physical and metabolic support to cells of the central nervous system, show rapid induction of growth

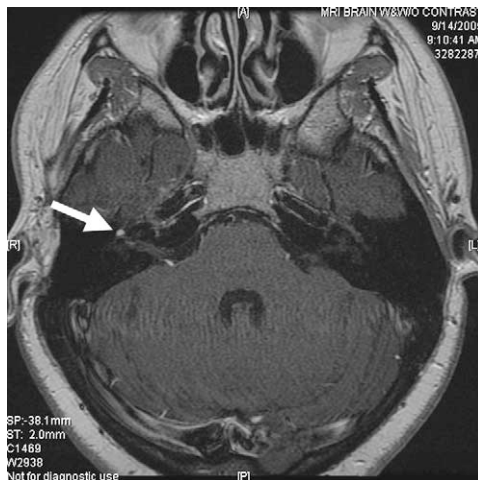


Fig. 3. Postoperative MRI for patient #2. Axial T1WI with contrast demonstrates punctuate contrast enhancement in the right geniculate ganglion, indicating residual meningioma (arrow).

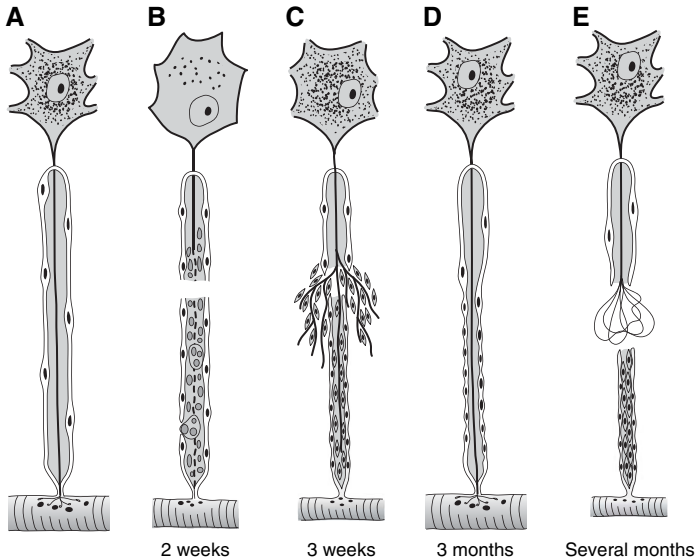


Fig. 4. Main changes that take place in an injured nerve fiber. (A) Normal nerve fiber, with its perikaryon and the effector cell (striated skeletal muscle). Note the position of the neuron nucleus and the number and distribution of Nissl bodies. (B) When the fiber is injured, the neuronal nucleus moves to the cell periphery, and the number of Nissl bodies is greatly reduced. The nerve fiber distal to the injury degenerates along with its myelin sheath. Debris is phagocytosed by macrophages. (C) The muscle fiber shows pronounced disuse atrophy. Schwann cells proliferate, forming a compact cord penetrated by the growing axon. The axon grows at a rate of 3mm/day. (D) In this example, the nerve fiber regeneration was successful. Note that the muscle fiber was also regenerated after receiving nerve stimuli. (E) When the axons do not penetrate the cord of Schwann cells, its growth is disorganized. (Adapted from Ross MH, Romrell LJ, Kaye GI. Response of neurons to injury. In: Ross MH, Romrell LJ, Kaye GI, editors. Histology: a text and atlas. Ross's 3rd edition. Baltimore, Maryland: Williams and Wilkins; 1995. p. 284.)

factors and gradually replace microglia on the neuronal surface. With successful nerve regeneration, the astrocytes retract and the neuronal surface is repopulated with synaptic terminals [3].

Although there is no measurable proliferation of capillaries, a functional activation of local vasculature occurs, so that regenerating motor neurons receive additional energy and building materials. These changes are thought to facilitate the production of new axoplasm, and, teleologically, this is sound and acceptable. However, the experimental observations of Stennert [4] provide a central cause for postrecovery synkinesis. In a cohort of 12 young female rats, six underwent a facio-facial anastomosis and the other six served as a control group. Twenty-seven months later, all animals underwent surgical exposure of the peripheral facial nerve branches for application of different colored retrograde tracers: Di I (red), FG (white), and FB (blue). One week later, all rats were perfused, their brains removed, and the brainstem sections examined under a fluorescent microscope, using

different UV filters. The unoperated rats showed normal somatotopic organization of the corresponding motor neurons inside the facial nucleus as red, white, and blue coherent clusters (Fig. 5). The operated rats showed loss of the clusterlike somatotopic organization and several motor neurons showed a mixture of tracer colors (Fig. 6).

During regeneration, there is an overall increase in transport of protein in the axon, and the increased flow of axoplasm leads to a considerable enlargement of the axon tip of the proximal nerve segment. An accumulation of signaling molecules at the distal end of the proximal segment is also evident; this is known as the *growth cone*. Within a week of injury, bundles of axonal sprouts (filopodia) grow into the distal segment of the transected nerve. This neurite outgrowth can be inhibited by several factors, including the persistence of myelin in the distal segment [3].

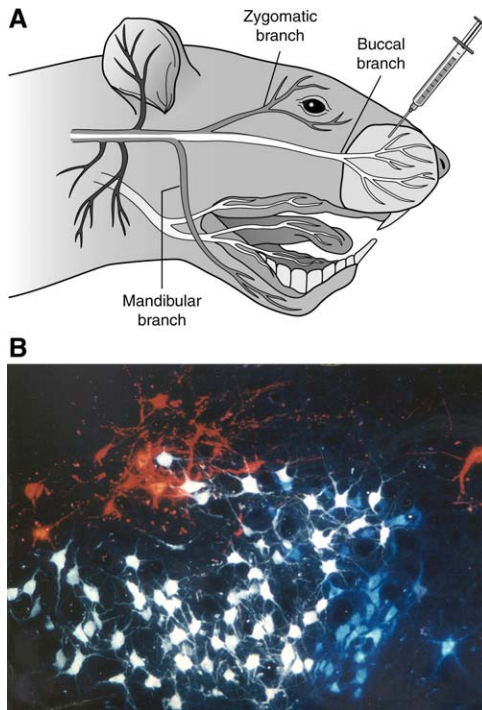


Fig. 5. (A) Injection of different retrograde tracers into the facial muscles. (Adapted from Stenert E. Synkineses and the autoparalytic syndrome. In: The facial palsies—complementary approaches. 1st edition. Utrecht, The Netherlands: Lemma Publishers; 2005. p. 117–133; with permission.) (B) Normal somatotopic clusterlike organization of the facial nucleus after retrograde tracing in nonoperated control. (Adapted from Dohm S, Streppel M, Guntinas-Lichius O, et al. Local application of extracellular matrix proteins fails to reduce the number of axonal branches after varying reconstructive surgery on rat facial nerve. *Restor Neurol Neurosci* 2000;16:117–26; with permission.)

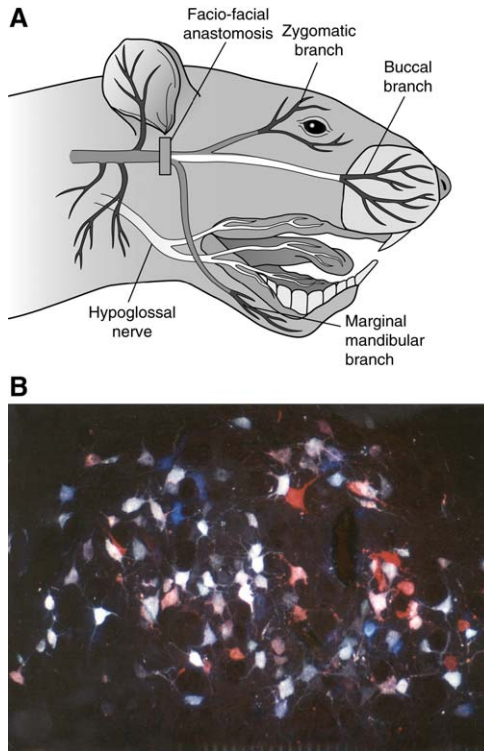


Fig. 6. (A) Injection of different retrograde tracers into the facial muscles after facio-facial anastomosis. (Adapted from Stennert E. Synkineses and the autoparalytic syndrome. In: The facial palsies—complementary approaches. 1st edition. Utrecht, The Netherlands: Lemma Publishers; 2005. p. 117–133; with permission.) (B) Facial nucleus 8 weeks after facio-facial anastomosis and 10 days after identical triple retrograde labeling as in Fig. 5 (B). Note the complete loss of somatotopic organization of the facial nucleus; motor neurons are scattered throughout the whole nucleus. (Adapted from Dohm, S, Streppel M, Guntinas-Lichius O, et al. Local application of extracellular matrix proteins fails to reduce the number of axonal branches after varying reconstructive surgery on rat facial nerve. *Restor Neurol Neurosci* 2000;16:117–26; with permission.)

Nerve fibers disconnected from their neuron begin to degenerate within hours of the injury. However, nerve conductivity and axonal transport in the distal segment are preserved for several days, and this is the basis for electrical tests such as electroneurography. Within the first 2 to 3 days, there is axolysis and proliferation of Schwann cells (wallerian degeneration). The myelin sheath also fragments and there is a breakdown of the blood-nerve barrier. Thus, circulating monocytes enter the nerve parenchyma, transform into tissue macrophages, and, together with Schwann cells, phagocytose axonal and myelin fragments. Proliferating Schwann cells form tubular structures within the endoneurial tubes (Bands of Büngner) and these strongly support axonal regeneration. The breakdown of the blood-brain barrier

allows serum growth factors, such as transferrin, to enter the nerve parenchyma and facilitate axonal regeneration (Fig. 7).

The morphologic change at the myoneural junction is that the primary synaptic cleft becomes shallower and the secondary synaptic clefts become shallower and wider (Fig. 8). The basal lamina of the target muscle persists; this structure is known to be a vital factor in the formation of new synaptic connections during reinnervation.

A number of morphologic changes also occur in a denervated muscle. The one most widely recognized, both in terms of number and types of

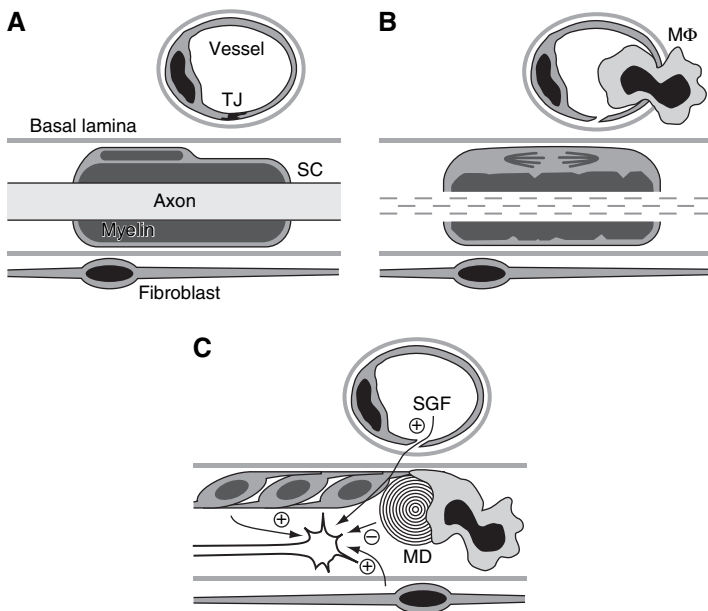


Fig. 7. Cellular changes in the injured and regenerating peripheral nerve. (A) Longitudinal section through a normal neural tube with myelinated motor axons and associated Schwann cells (SC) surrounded by basal lamina. The tight junctions (TJ) of the adjacent endoneurial vessels form the structural basis of the blood-nerve barrier in the normal, uninjured peripheral nerve. (B) Axonal injury causes a rapid degeneration of the distal, disconnected axon, followed by a destabilization of the associated myelin and Schwann cell proliferation. Axonal degeneration also leads to a breakdown of the blood-nerve barrier and adhesion of circulating monocytes, which transform into tissue macrophages (MO) and later invade the endoneurial tubes. (C) Proliferating Schwann cells form endoneurial Bungner bands that strongly support axonal regeneration. They are assisted by adjacent fibroblasts that provide neurotrophic factors and nutritional material for axonal growth. The absence of the blood-nerve barrier also provides access for serum growth factors (SGF), such as transferrin, to aid ongoing axonal regeneration. In contrast, the myelin debris (MD) contains a number of inhibitory molecules that block neurite outgrowth. The removal of myelin by macrophages plays a central role in promoting nerve regeneration. (Adapted from May M, Gantz B, Hughes G. Management of failed nerve graft following facial nerve resection for facial nerve neurofibroma. *Head Neck Surg* 1987;9:184-7; with permission.)

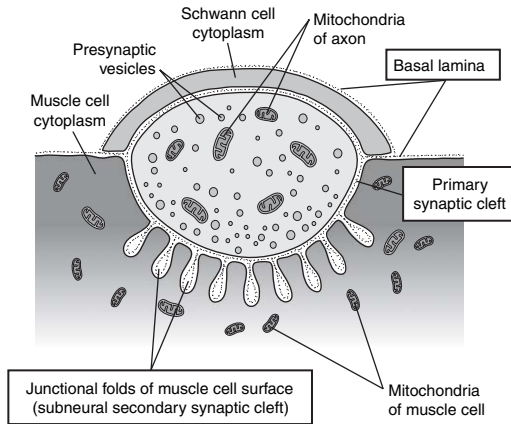


Fig. 8. The neuromuscular junction. An axon is shown making contact with the muscle cell. Note how the junctional folds of the muscle cell augment the surface area within the synaptic cleft. The basal lamina extends throughout the cleft area. The cytoplasm of the Schwann cell is shown covering the axon terminal. (Adapted from Ross MH, Romrell LJ, Kaye GI. Response of neurons to injury. In: Ross MH, Romrell LJ, Kaye GI, editors. Histology: a text and atlas. Ross's. 3rd edition. Baltimore, Maryland: Williams and Wilkins; 1995. p. 284.)

fibers, is pronounced muscle atrophy. Muscle atrophy of 20% to 90% of baseline has been reported [5]. Histologically, there is a well-documented increase in the number of satellite cells (Fig. 9) [6]. These satellite cells are myogenic precursors and could be involved in the restoration of muscle mass after reinnervation. Acetylcholine receptors are spread over the surface of the denervated muscle. Ultimately, many denervated muscle fibers are replaced by fat cells and connective tissue. Neuromuscular changes following nerve transection are summarized in Table 1. All this information is, for the most part, derived from observations made on limb musculature in small animals, and, in fact, very little research has been done on muscles innervated by the facial nerve [6].

Chronic changes in the facial nerve, nucleus, and muscle after long-term injury

Because the problem the authors are confronted with is the management of long-term facial paralysis, it is pertinent to discuss the effects of long-term denervation on the neuron, proximal segment, distal segment, myoneural junction, and muscle. Information on this is sparse in the literature, particularly with reference to the facial nerve. The experimental work of Stennert [4] provides an understanding of changes that occur at the nuclear level of a transected nerve, as described earlier.

Limited knowledge exists of the long-term effects of nerve transection on the proximal segment of the injured nerve. The hitherto unknown effects of stretch injury on the proximal segment in humans have been described by

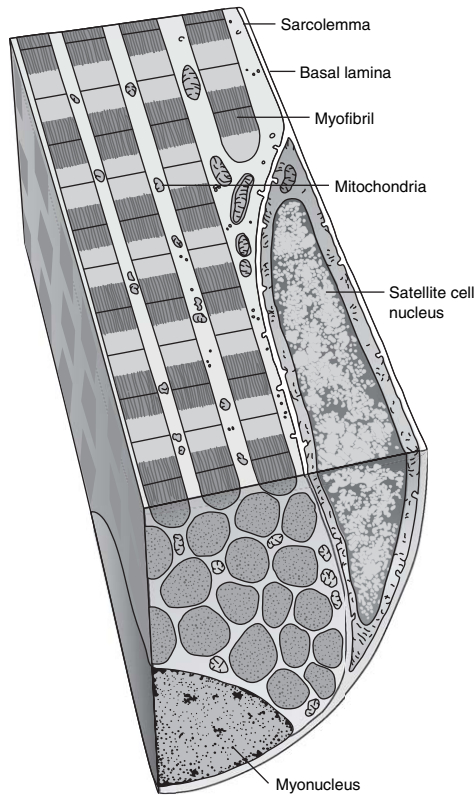


Fig. 9. A muscle-fiber–satellite-cell complex. Note that the satellite cell and the muscle fiber are enclosed within a common basal lamina. (*Adapted from* Carlson BM. *Skeletal muscle-denerivation, reinnervation, and muscle transplantation*. In: *The facial nerve*. May's 2nd edition. New York: Thieme; 2000. p. 81–94.)

Felix and colleagues [7]. They noted that the proximal segment showed dense retrograde fibrosis for varying distances along the length of the nerve.

Ylikoski and colleagues [8] have described long-term changes in the distal segment of the human facial nerve. They studied the distal segment of five human facial nerves that had been transected 17 days earlier in one subject, 3 months earlier in two subjects, 7 months earlier in one subject, and 30 months earlier in one subject.

All the pathologic specimens were studied by light and electron microscopy. All five subjects underwent XII-VII anastomosis. The histopathologic findings, follow-up periods, and results of the anastomosis are summarized in Table 2. The most striking findings were seen in subject # 4, in whom the result was poor and the distal segment showed total fibrosis, even though the nerve anastomosis was done just 7 months after transection. This result contrasts with subject # 5, in whom there was only slight endoneurial fibrosis 30 months after transection, and whose functional result was “much

Table 1
Summary of the changes that occur in the neuromuscular complex

Structure	Pathologic changes	Time course
Cell body	Cell body swells and nucleus marginalizes. There is chromatolysis, hyperplasia of cellular organelles, and loss of somatotopic organization.	1 to 2 days
Microglia	Microglia contact affected neurons and cause synaptic stripping. They are replaced by astrocytes that promote growth factors and metabolic support.	Within 24 hours
Proximal axon	Axoplasm increases and proximal tip engorges (growth cone). Filopodia sprout and seek out endoneurial tubules.	Hours to days
Distal axon	Axonal transport is preserved for 48–72 hours. Axolysis and Schwann cells proliferate, myelin sheaths fragment and are phagocytosed by microglia. Bands of Büngner form.	Days
Myoneural junction	Primary and secondary synaptic clefts become shallow. Basal lamina is scaffold for muscle regeneration and reorganization of myoneural junctions.	Days to weeks
Muscle	Muscle atrophy and hypoplasia ensue within months. Satellite cells multiply and differentiate to restore muscle mass. Acetylcholine receptors spread out then regroup to accept incoming regenerated nerve.	Weeks to months

improved” 6 months after anastomosis. It appears, therefore, that endoneurial fibrosis is not a function of time, but is certainly an impediment to regeneration, as shown by this study and the study by Felix and colleagues [7]. Nevertheless, Carlson [6] is of the opinion that “a significant factor that determines the level of success of reinnervation of a denervated muscle is the condition of the distal pathways through which the regenerating axons must pass.” He drew particular attention to the state of the basal lamina at the myoneural junction (see Fig. 8). If this structure is filled with deposits of collagen, the outgrowth of regenerating axons is inhibited.

Data on long-term changes at the myoneural junction and in human facial muscles are scanty. As for the histopathology of denervated muscle, Belal [9] provided one of the first reports on the structure of human muscle in facial paralysis. He studied the postauricular and stapedius muscles of 14 subjects whose facial nerves had been injured. The paralysis occurred during the course of surgery ($n = 8$), or the subjects developed Bell’s palsy ($n = 3$), or herpes zoster ($n = 1$), or tumors ($n = 2$), or malignant external otitis ($n = 1$). The duration of the paralysis ranged from 10 days to 6 years (Table 3). The pathologic changes were correlated neither to the underlying cause nor to any EMG studies that may have been done. Without such a correlation, these data, though valuable in themselves, do not help in the management decisions of long-term paralysis.

Bardosi [10] reported subsequently on the ultrastructure of normal and denervated human facial muscle. Facial muscle biopsies were obtained from 25 subjects ranging in age from 14 to 89 years. In this cohort, 17

Table 2
Summary of clinical and histopathologic data of five subjects who underwent hypoglossal-facial anastomosis

Case	Age	Sex	Diagnosis	Postop. time from facial nerve severance	Histopathologic findings in distal stump of facial nerve	Clinical results of re-innervation surgery	Follow-up time
1	32	F	Acoustic neuroma	17 days	Wallerian degeneration in progress, all stages present; sensory fibers intact	Slightly improved function	6 months
2	31	M	Meningioma	3 months	Major portion degenerated with scattered surviving fibers; increased endoneurial fibrosis; bands of Büngner; intact sensory bundle	Much improved function	18 months
3	47	F	Meningioma	3 months	Degeneration of all nerve fibers; increased endoneurial fibrosis; bands of Büngner	Much improved function	4 years
4	28	F	Meningioma	7 months	Total fibrotic change	No improvement	3 years
5	32	F	Middle ear cholesteatoma with intracranial extension	30 months	Nerve trunk of multifascicular type; degeneration of myelinated fibers except a few (vagus branch); slight endoneurial fibrosis; bands of Büngner	Much improved function	6 months

Data from Ylikoski J, Hitselberger WE, House WF, et al. Degenerative changes in the distal stump of the severed human facial nerve. Acta Otolaryngol 1981; 92:239–248.

Table 3
Natural history of denervation atrophy

Time course	Pathologic changes
10 days	Chains of subsarcolemmal nuclei Infoldings of nucleolar membrane No myofibrillar changes
17 days	Focal myofibrillar disruption Fragmentation of Z-line Subsarcolemmal mitochondrial hyperplasia Indentations of sarcolemma
6 weeks	Small-group atrophy of myofibrils Central migration of nuclei Disruption of some myofibrils
13 months	Large-group atrophy of myofibrils Degenerated myofibrils Increased collagen filaments
6 years	Large-group atrophy of myofibrils Dissociation of plasma and basement membranes Increased collagen filaments

Data from Kreutzberg GW, Raivich G. Neurobiology of regeneration and degeneration. In: The facial nerve. May's 2nd edition. New York: Thieme; 2000. p. 67–79.

subjects had undergone EMG tests and all showed evidence of denervation. The duration of facial paralysis in these 17 subjects ranged from 1 month to 36 years. However, no mention was made of the cause of the paralysis. The investigators found that the ultrastructure of normal human facial muscles did not differ in any respect from normal human striated muscle. The main difference between denervated and normal muscle was the presence of “a broad spectrum of inclusion bodies” in the former.

The success of reinnervation has been attributed for the most part to the condition of the muscle itself. Denervation muscle atrophy is well recognized clinically and histopathologically, and the principal cause is a reduction in the size and number of myofibrils within the muscle fiber [6]. Denervated muscles also undergo capillary reduction and connective tissue deposition (secondary atrophy); these changes could also add to the resistance to reinnervation [6].

Unfortunately, even with this background information, clinicians still have many questions:

- At what point after denervation does the facial musculature become incapable of reinnervation?
- Does the degree of muscle fiber atrophy influence its ability to reinnervate?
- Can regenerating fibers reach chronically denervated facial muscles?
- If regenerating nerve fibers can reach atrophic muscles, can functional neuromuscular junctions form?
- Can atrophic muscle fibers respond to the trophic influence of regenerating nerves?

The successful outcomes reported by Conley and colleagues [5] and Gagnon and Molina-Negro [11] in subjects with long-term paralysis suggest that although the above questions cannot be answered based on current clinical or experimental data, careful clinical observations can and should continue to be made, colleagues from radiology, neurology, and pathology should be closely involved, and patients should continue to be treated empirically. In most cases of long-term facial paralysis, it would be beneficial to know if a dynamic reinnervation is likely to succeed.

To this end, the authors would like to evaluate preoperatively the changes described above, in the neuron, the proximal nerve segment, the distal segment, the myoneural junction, and the denervated muscle. In practical terms, the only structure that can be studied preoperatively is the muscle, with EMG. On needle EMG, the first sign of axonal loss in the facial nerve is the development of abnormal spontaneous activity (ie, fibrillations and positive sharp waves that usually appear within 10 to 14 days of axonal transection) [12]. Fibrillation potentials are short duration (1–5 milliseconds) biphasic or triphasic discharges of 20 to 500 μ V amplitude. Positive sharp waves have an initial sharp positive deflection followed by a longer-lasting negative waveform; these can last up to 100 milliseconds and have amplitudes in the same range as fibrillation potentials. The fibrillation or positive sharp wave from each muscle fiber usually has a rhythmic discharge. However, the aggregate of fibrillations and positive sharp waves from multiple muscle fibers in the recording range of the needle electrode produce a sound over the amplifier that has been likened to that of “wrinkling tissue paper” [13]. Most investigators agree that fibrillations and positive sharp waves are a result of hyperexcitability in the denervated membrane of single muscle fibers [14]. Although the mechanisms underlying muscle and nerve membrane abnormalities after transection are not understood completely, there is increasing evidence that ion channel abnormalities are involved [15,16].

Fibrillations and positive sharp waves continue to be present in muscle on needle EMG until there is either reinnervation or atrophy with replacement by connective tissue [14]. Waveform size is not a universally accepted indicator of the amount of viable muscle or age of the lesion because of limitations associated with the distance of the recording needle electrode from the discharging muscle membrane, variation in fiber size, and sampling error. Measurement of the evoked compound muscle action potential amplitude on a nerve conduction study remains the standard method for assessing the amount of viable muscle. With progressive reinnervation (or progressive muscle atrophy), the number of positive sharp waves and fibrillation potentials decreases. Eventually, the size of the discharges diminishes; the decrease in size may be due to multiple factors, such as muscle atrophy or distance from the recording needle [17].

During reinnervation, reliable and predictable changes occur in the morphology of motor unit action potentials evoked from the reinnervated

muscles. Larger, more complex motor unit action potential waveforms develop as a consequence of axonal sprouting and the increase in the number of muscle fibers receiving nerve supply from a single motor neuron. The number of functioning motor units in the recording territory of the needle EMG electrode depends on the degree of successful reinnervation. With more successful reinnervation, there are more functioning motor units. The term applied to the aggregate of motor units is “recruitment.” Recruitment is reduced when the number of motor units is less than would be seen in a normal muscle.

The causes of failure of return of function in the two cases discussed appear clear in hindsight. In the first case, the seminal observations of Felix [7] showed that in stretch injuries of the facial nerve, such as occur in longitudinal or otic capsule sparing fractures, there is extensive endoneurial fibrosis proximal and distal to the geniculate ganglion. This fibrosis prevents the growth of axonal sprouts into the Bands of Büngner in the nerve segment distal to the intervening band of fibrosis. These findings were unavailable at the time of the first patient’s surgery, and thus the nerve anastomosis was made proximally and distally to an area of the nerve that was fibrotic. Thus, failure of return of function was inevitable. Clearly, failure in the second case was due to the residual tumor at the proximal anastomotic site (see Fig. 3).

Management

Conley and May [18] classified facial restoration procedures into dynamic (Groups I-III) and static or adjunctive ones (Groups IV-VI). The details of individual procedures are summarized in Table 4. Most surgeons

Table 4
Classification of facial restoration procedures

Group	Type	Procedure
1	Dynamic	Facio-facial grafts: end-to-end, interposition, or double cable
2	Dynamic	Cranial nerve substitution: XII-VII, XII-VII jump, VII-VII (cross facial)
3	Dynamic	Muscle transposition: regional (temporalis, masseter, digastric) or free muscle neurovascular anastomosis (gracilis, and so forth)
4	Static	Suspension techniques (palmaris longus, fascia lata, gore-tex sling) or facial rejuvenation (face lift, brow lift, blepharoplasty)
5	Static	Eye care (gold weight, tarsorrhaphy, canthoplasty, dacryocystorhinostomy)
6	Static	Management of synkinesis (selective myectomy, neurectomy, Botox)

Data from Conley J, May M. Rehabilitation techniques for acute and long-standing facial paralysis. In: The facial nerve. May’s 2nd edition. New York: Thieme; 2000. p. 562–9; with permission.

would not attempt a Group I or II surgery (*vide infra*) if the duration of paralysis exceeds 2 years or more. This algorithm may have developed on the unproven assumption that with long-term denervation the target muscles for reinnervation are atrophied. In fact, the obstruction to reinnervation could be a fibrosed distal nerve segment [8] or deposition of collagen on the basal lamina [6]. Currently, no tests exist to evaluate these structures noninvasively. Thus, attempts at reinnervation become empiric. Conley and colleagues [5] have reported successful outcomes in very late cases. The ideal procedure is one that would allow mimetic facial motion without synkinesis or additional neurologic deficit. Theoretically, the procedures described in Groups I and II could achieve these objectives in the authors' two cases. In the first case, if the opportunity had existed to obtain EMG studies even 3 years after the original injury, and if fibrillation potentials were still recorded, reinnervation surgery could have been offered to this patient. Surgery success would depend on accurate frozen section study of the segments of nerve proximal and distal to the area of the geniculate ganglion. If the proximal fibrotic segment extended deep into the internal auditory canal, anastomosis with an interpositional graft would be difficult technically, and in such a case a XII-VII jump graft would be most appropriate. If, despite this surgery, there was failure of reinnervation, the patient could have been offered free muscle graft and cross-facial anastomosis, particularly as he was so young. Unfortunately, the patient refused further investigations or surgery.

The second patient presents two problems: the residual meningioma and the long-term facial paralysis. It could be argued that the meningioma can be followed conservatively with serial MRI scans. If this course is taken, the preferred Group I procedure would not be possible, but the patient could benefit from a cross-over procedure such as a XII-VII jump graft, distal to the tumor. Given the patient's young age, the authors recommend tumor excision under frozen section control and restoration of the anatomic continuity of the nerve with an interpositional graft. Again, as in the previous case, if the tumor extends deep into the internal auditory canal, it will be excised under frozen section control and a XII-VII jump graft will be offered. If this fails as well, muscle transposition will be recommended, as in the previous case.

Summary

The preoperative evaluation for either revision facial nerve surgery or long-standing facial paralysis is far from comprehensive and typically is limited to an EMG assessment. The EMG can determine if fibrillation potentials are present; however, these do not necessarily indicate the degree of muscle viability. To the best of the authors' knowledge, no scientific correlation exists between EMG fibrillation potentials and either the degree of muscle atrophy, or the potential for a successful outcome of dynamic

reanimation procedures. Clearly, controlled, double-blinded longitudinal study of chronic facial denervation and correlation with longitudinal EMG studies in the same cohort is needed.

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