FACIAL NERVE PALSY

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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. 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 lacrimal and palatine secretory fibers to the pterygopala- tine 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 in- clude 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 fora-men 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 be- cause lacrimal secretion and orbicularis closure are in- volved. 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.

1. **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. 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, and systemic steroids and/or acyclovir have been recommended as treatment.

2. **Traumatic**

Traumatic injury is the second most common etiology of facial nerve paralysis, comprising 8–22% of cases. A significant proportion of these injuries occur during delivery either due to birth canal trauma or forceps delivery. Other causes of traumatic paralysis include surgical trauma, penetrating parotid or middle ear trauma, barotrauma, facial fractures, and temporal bone fractures.

3. **Infection**

Infection is the next most common etiology of facial paralysis. Ramsay Hunt syn-drome 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. 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. 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. Other infections include polio, mumps, cytomegalovirus, mononucleosis, leprosy, cat scratch fever, and dengue fever.

4. **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.

5. 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.
Systemic and metabolic disorders implicated in facial nerve paralysis include diabetes mellitus, hyperthyroidism, hypertension, pregnancy, acute porphyria, autoimmune syndromes, sarcoidosis, amyloidosis, car bon monoxide toxicity, tetanus, diphtheria, vitamin A deficiency, ethylene glycol ingestion, and alcoholism.

- 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
- 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 System

The gold standard for grading facial nerve function is the House-Brackmann grading scale (House and Brack-mann, 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.

These include the Nottingham system, the Sunnybrook scale, the Yanagihara, and the Syd-ney system, all with their advantages and disadvantages.

Grade	Description	Characteristics
I	Normal	Normal facial function in all areas
II	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
III	Moderate dysfunction	Gross: obvious but not disfiguring difference between two sides; noticeable but not severe synkinesis, contracture, and/or hemifacial spasm At rest: normal symmetry and tone Motion: Forehead – slight-to-moderate movement Eye – complete closure with effort Mouth – slightly weak with maximum effort
IV	Moderately severe dysfunction	Gross: obvious weakness and/or disfiguring asymmetry At rest: normal symmetry and tone Motion: Forehead – none Eye – incomplete closure Mouth – asymmetric with maximum effort
V	Severe dysfunction	Gross: only barely perceptible motion At rest: asymmetry Motion: Forehead – none Eye – incomplete closure Mouth – slight movement
VI	Total paralysis	No movement

Clinical Evaluation

Upper eyelid: Evaluate upper eyelid retraction. Up- per 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 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 deter- mine 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 long- standing or recovering facial nerve palsy. The most noticeable areas of synkinesis involve the orbicularis oculi, nasolabial fold area, and mouth.

Management:

Medication, surgery, rehabilitation and lately botulinum toxin type A (BTX-A) for treatment of FNP have been recommended. When conservative treatments do not have positive effects, surgery, including neurolysis, facial nerve graft, cut of tendon, or muscle graft to face is the treatment choice. In some cases for removing synkinesis, surgery is recommended. Selective neurolysis may relief synkinesis temporarily; however, synkinesis frequently recurs, sometimes more severe compared to status before the intervention

Physical Therapy Management:

- Anti-inflammatory effect by ultrasound therapy, it prevents inflammation and denervation
- Electrical stimulation (faradic and pulse galvanic) in peripheral facial nerve injuries reported antithesis
- Short duration stimulation (Faradic or FES) could not induce contraction in denervated or injured fibers and if any contraction observed, it is related to innervated and sound muscle fibers.
- Therefore, long duration stimulation (pulse galvanic) should be used.
- First of all, long duration stimulation is annoying for patients; second, these stimulations could neither prevent atrophy nor regenerate the nerve. Third, they could create mass movement and increase synkinesis.
- EMG biofeedback
- Mirror biofeedback
- Mime therapy (combination of thermotherapy, massage, and neuromuscular reeducation)
- Active, active-assistive, and resistive exercise
- Massage and light stretch with the aim of improving blood flow of soft tissue and damaged muscle.