K. J. Lee: Essential Otolaryngology and Head and Neck Surgery (IIIrd Ed)

Chapter 10: Facial Nerve Paralysis

Evaluation

Evaluation of patients with facial nerve paralysis must be a careful process including a detailed history, physical examination, audiometry with impedance testing, and appropriate x-rays. It is important to separate a central facial paralysis from a peripheral type before embarking on a diagnostic plan. A central unilateral facial paralysis usually will involve only the lower face as the innervations of the upper face are derived from crossed and uncrossed fibers. Paralysis of the peripheral type will involve the upper and lower face. The common causes of facial paralysis are found in Table 10-1. Diagnosis of lesions from a standpoint of level of impairment is found in Table 10-2. It is imperative not to label a facial paralysis as Bell's palsy prior to an exhaustive diagnostic workup to rule out a definitive etiology. By definition, Bell's palsy suggests idiopathic facial paralysis.

Table 10-1. Common Causes of Facial Paralysis

Congenital

Dystrophia myotonica, Möbius syndrome (facial diplegia associated with other cranial nerves), Bell Melkersson syndrome (furrowed tongue, faciolabial edema), familial.

Traumatic

Basal skull fracture, facial injuries, penetrating injury to middle ear, altitude paralysis, use of forceps at birth, molding trauma at birth.

Neurologic

Landry-Guillain-Barre syndrome (ascending paralysis), multiple sclerosis, myasthenia gravis, opercular syndrome (cortical lesion in facial motor area), Millard-Gubler syndrome (abducens palsy, with contralateral hemiplegia due to lesion in base of pons involving corticospinal tract).

Infectious

External otitis, otitis media, mastoiditis, chicken pox, herpes zoster (Ramsey Hunt syndrome), encephalitis, poliomyelitis (type 1), mumps, mononucleosis, leprosy, influenza, coxsackievirus, malaria, syphilis, sarcoidosis, Heerfordt's disease - uveoparotid fever.

Metabolic

Diabetes mellitus, hyperthyroidism.

Neoplastic

Cholesteatoma, VIII nerve tumor, glomus jugulare, leukemia, meningioma, hemangioblastoma, sarcoma, carcinoma (invading or metastatic), anomalous sigmoid sinus, hemangioma of tympanum, hydradenoma (external canal), osteopetrosis, facial nerve tumor (cylindroma), schwannoma, teratoma, Hand-Schüller-Christian disease.

Toxic

Thalidomide (Miehlke's syndrome of cranial nerve VI and VII with congenital malformed external ears and deafness), tetanus, diphtheria.

Iatrogenic

Mandibular block anesthesia, antitetanus serum, vaccine treatment for rabies, post polio immunization, parotid surgery, mastoid surgery, posttonsillectomy, and postadenoidectomy.

Table 10-2. Diagnosis of Lesions from Level of Impairment

Supranuclear: Good tone, intact upper face, presence of spontaneous smile, neurologic deficits.

Cerebrovascular accident, trauma.

Nuclear: Involvement of the sixth and seventh cranial nerves, corticospinal tract signs.

Vascular or neoplastic, poliomyelitis, multiple sclerosis, encephalitis.

Angle: Involvement of vestibular and cochlear portions of the eight cranial nerve; the facial nerve, particularly taste, lacrimation and salivation may be altered; the fifth and later ninth, tenth, and eleventh cranial nerves may become impaired.

Neurinoma, meningioma, fracture, cholesteatoma, arachnoid cyst.

Geniculate ganglion: Facial paralysis, hyperacusis, alteration of lacrimation, salivation, and taste.

Herpes zoster oticus, fracture, Bell's palsy, cholesteatoma, neurinoma, arteriovenous malformation, meningioma.

Tympanomastoid: Facial paralysis, alteration in salivation and taste; lacrimation intact.

Bell's palsy, cholesteatoma, fracture, infection.

Extracranial: Facial paralysis (usually a branch is spared), salivation and taste intact, deviation of jaw to normal side.

Trauma, tumor, parotid carcinoma, pharyngeal carcinoma.

Important Point in Diagnosis

1. Bilateral simultaneous facial paralysis is a sign of central or generalized disease and should not be confused with Bell's palsy.

2. Patients with slowly progressive facial paralysis, whether partial or total, with no evidence of recovery after 6 months, should be suspected of a neoplasm involving the facial nerve.

3. Facial paralysis associated with pain, sensorineural hearing loss, vertigo, and a red pinna with vesicles have herpes zoster oticus or Ramsey Hunt syndrome, the vesicles being present in the area of sensory distribution of the facial nerve.

4. Hitzelberger's sign: Decreased sensitivity in the concha corresponding to the sensory distribution of the seventh nerve is suggestive of a space-occupying lesion in the internal auditory canal.

5. The incidence of severe degeneration in Bell's palsy approximates 15%. However, in herpes zoster the incidence approximates 40%.

6. Ten percent of the patient with Bell's palsy have a positive family history. Recurrent facial paralysis recurs in 10% of the patients with Bell's palsy, and is much more common on the ipsilateral and contralateral sides.

7. Thirty percent of the patients with recurrent facial paralysis on the same side have been found to have tumors.

Steps in the Evaluating a Bell's Palsy

Is it partial or total?

Partial

No treatment. Close follow-up.

Total

1. Determine the level of involvement:

a. Taste impairment?

b. Presence of stapedial reflex?

c. Schirmer's test. It is important to note that this is a gross test and it is of no significance unless the normal eye tears at least 30% more than the involved eye.

2. One or more of the following tests can be performed:

a. Nerve excitability test

b. Conduction latency test

c. Strength-duration studies

d. Electromyography (EMG)

e. Maximal stimulation test

f. Salivary flow.

When increasing difficulty in eliciting responses in these tests is noted, one should consider that thde nerve is showing signs of denervation. If one believes in decompression of the nerve, it should be done as soon as it is feasible.

Nerve Excitability Test

This test uses a once per second square-wave pulse, 1 msec in duration. This test has no clinical use in a partial paralysis or within 3 days of total paralysis. After the third day of total paralysis, the normal side is first tested to obtain the threshold needed to elicit the slightest flicker of facial muscle movement. The electrode is placed percutaneously along the stylomastoid foramen and then along the main branches of the facial nerve. After recording the thresholds for the normal side, the electrode is placed at the same locations on the diseased side. The respective thresholds are then compared. A greater than 3-4 mamp difference is considered significant, suggesting denervation. For those who believe in decompression of the facial nerve, the nerve excitability test should be performed daily after the third day of total paralysis. As soon as a consistent 3-4 mamp or more difference in threshold between the normal and the abnormal sides appears, decompression is performed. In the nerve excitability test, one must avoid stimulating the muscle directly so that a false threshold is not obtained.

Conduction Latency Test

The conduction latency test also uses a once per second square-wave pulse, 1 msec in duration. A secon electrode is placed in a distal facial muscle. The time taken by the impulse to reach the distal electrode is recorded as conduction latency. The normal conduction time from the angle of the mandible to the facial muscle in the midline is about 4 msec. Like the nerve excitability test, it does not demonstrate prolonged conduction times until 72 hours after denervation. After 72 hours, a completely transected nerve shows increasing conduction time until no excitability is demonstrable. A lengthening of conduction time also may imply partial denervation.

Unlike the nerve excitability test, the conduction latency test is harder to perform both for the doctor and the patient. Hence it is not used clinically in the office.

Strength-Duration Studies

A particular muscle is selected for this test. A square-wave pulse of varying duration and intensity is applied until a just visible twitch is noted. As one goes from a longer pulse duration to a shorter pulse duration, the threshold needed to elicit a just visible twitch is recorded. The intensities for various pulse durations are recorded for the normal side. A denervated nerve will show considerably higher thresholds. The strength-duration curve is not altered in neuropraxia and is not altered till 7 days after denervation.

Rheobase: The strength of current just strong enough to depolarize (mamp).

Chronaxie: The length of duration needed to depolarize using an intensity two times the rheobase (msec).

Electromyography (EMG)

This test determines the activity of the muscle itself. A needle electrode is inserted into the muscle and recordings are made during rest and voluntary contraction. Fig. 10-1 illustrates a voluntary unit discharge, fibrillation potential, and polyphasic reinnervation potential. Degeneration of a lower motor nerve is followed in 14-21 days by spontaneous activity called fibrillation potential. Hence the EMG is not of diagnostic value until 2 weeks after denervation. The practical clinical usage of EMG is in the determination of reinnervation. Polyphasic reinnervation potentials are present 6-12 weeks before clinical return of facial function.

Maximum Stimulation Test

The maximum stimulation test is similar to the nerve excitability test except that it uses maximal rather than minimal stimulation. The main trunk as well as each major portion of the distal branches of the nerve (forehead, eye, nose, mouth, lower lip, and neck) on the normal and abnormal side are stimulated with an intensity that produces discomfort. The results of the test are expressed as a difference in facial muscle movement between the normal and the involved side. The finding of a difference is considered evidence of abnormality.

Salivary Flow Test

The salivary flow test is based on the fact that the preganglionic parasympathetic nerve fibers are on the outside of the VII nerve bundle; hence it is assumed that these fibers will be injured before the motor fibers. The proponents of this test further reason that the nerve excitability test examines the nerve distal to the injury while the salivary flow test checks the nerve at the site of injury.

After anesthetizing the anterior floor of the mouth, the test is performed by first dilating the Wharton's duct. A polyethylene tube is then cannulated into each duct. Lemon juice is next used to stimulate salivary flow of which the number of drops secreted per minute is counted for each side. A difference of 70% or greater is considered significant and warrants surgical decompression of the VII nerve.

Electroneuronography

Electroneuronography was popularized by Ugo Fisch. Similar in principle to the maximal stimulation test except that instead of visual observation of degree of response, there is a recording of the summation potential on an instrument similar to an EMG recording device. The normal side is compared with the abnormal side and the degree of degeneration estimated from the difference between the amplitude of the measured summation potentials on the two sides. Fisch recommends surgical decompression when the evoked summation potential is 10% or less than the normal side indicating, in his impression, 90% degeneration on the affected side. This should be done within 2 weeks of the onset of paralysis. Other investigators have felt that if the reduction was greater than 25%, decompression was indicated since by the time the level reached 10%, results were uniformly poor.

Treatment of Bell's Palsy

Before one can specifically advocate one mode of treatment over another, it is imperative to realize that the great majority of Bell's palsy patients have either partial paralysis or total paralysis without degeneration, i.e. maintaining the neuropraxia state. It is also fairly well recognized that, unless denervation has occurred, the patient more than likely will recover spontaneously with little synkinesis. Hence surgical treatment, if proposed, is reserved for those with total paralysis that have shown signs of denervation. There is no conclusive evidence to date that surgical decompression is of definite benefit. Some protocols treat Bell's palsy of all severities with steroids, others treat only cases of total facial palsy with steroids. Some clinicians believe that if the nerve is allowed to degenerate completely, the prognosis is poor and synkinesis is common.

A Guideline for the Management of Facial Nerve Paralysis

Bell's Palsy

A complete otologic, audiometric, and radiographic workup is needed.

Partial: No treatment.

Total:

1. Determine level of involvement.

2. Daily electrical test until:

a. Threshold of the involved side increases to 4 mamp.

b. There is evidence of some return of facial function.

If (a) is found, decompression of the facial nerve from the stylomastoid foramen to the level of blockage is performed. A "middle fossa" decompression should be done if the greater superficial petrosal nerve is involved.

Post Ear Surgery

Rule out effects of local anesthetics and too tight a mastoid packing.

1. Delayed onset (partial or complete): follow like Bell's palsy.

2. Immediate onset (partial or complete): explore the nerve before the "sun sets".

Traumatic (Head Injury)

1. Delayed onset (partial or complete): follow like Bell's palsy.

2. Immediate onset (partial or complete): explore the nerve when patient is stabilized.

Herpes Zoster Oticus

The most common motor nerve involved is the VII nerve, the next are III, IV, and VI. Treat like Bell's palsy.

Chronic Otitis Media

Partial or complete: mastoidectomy and facial nerve decompression; ? tympanoplasty.

Acute Otitis Media

1. ? Treat like Bell's palsy.

2. ? Simple mastoidectomy.

3. ? Myringotomy.

Acute Mastoiditis with Facial Paralysis

Treatment includes simple mastoidectomy and decompression of the facial nerve and myringotomy or simple mastoidectomy and myringotomy.

Another concept for the management of facial nerve paralysis was advocated by Sunderland. His classification of facial nerve injury and recommended treatment is outlined in Table 10-3.

Table 10-3. Classification and Treatment of Facial Nerve Injury

First-degree (neurapraxia). Onset to third day. Damming of proximal and distal flow of axoplasm. Lacrimation > 70%. Salivation > 70%. Maximal stimulation equal. ENG equal. Treatment: Eye care. Sedation. Analgesic. Reassurance. Reevaluate incomplete paralysis in 1 week. If paralysis becomes complete reevaluate immediately. Complete paralysis should be reevaluated and retested every other day until recovery or placement in second-degree. Natural history: Complete recovery beginning in 1-3 week. Surgical results: No surgery necessary.

First-to-second degree (neurapraxia-axonotmesis). Third to fifth day. Disruption of axons. Lacrimation < 25%. Salivation < 25%. Maximal stimulation lowered. ENG < 50%. Treatment: Supportive care as for first-degree. Surgical decompression to labyrinthine segment. Natural history: Fair recovery. Minimal cosmetic and functional impairment beginning in 3 weeks to 2 months. Surgical results: Complete recovery beginning 7-10 days.

Second-to-third-degree (axonotmesis-neurotmesis). Fifth to fourteenth day. Disruption of axons and myelin tubes. Lacrimation - dry eye. Salivation < 25%. Maximal stimulation - lowered to absent. ENG = 25%. Treatment: Same as second-degree. Natural history: Poor recovery. Obvious incomlete recovery with marked complications of faulty regeneration: tic, spasms, synkinesis; beginning 2-4 months. Surgical results: Fair recovery beginning 3 weeks to 2 months.

Third-to-fourth-degree (neurotmesis). Disruption of perineurium, myelin tubes, and axons. Lacrimation - absent if lesion at or proximal to geniculate ganglion. Salivation < 25%. Maximal stimulation - response lost 2 or 3 days. ENG - response lost 3-5 days. Treatment: Ideally, explore and repair immediately or within 30 days. Natural history: If nerve is completely severed some spontaneous recovery may occur in rare instances. If incompletely severed, recovery may occur but results in marked facial weakness with no recovery of parts of face or mass movement. Surgical results: Repair within 30 days: 90%. Excellent (symmetry, eye and mouth movement), or good symmetry and movement beginning 4-10 months.

Fifth-degree. Disruption of epineurium, perineurium, myelin, and axons. Lacrimation - same. Salivation - same. Maximal stimulation - same. ENG - same. Treatment: Same. Natural history: Same. Surgical results: Same.

Miscellaneous

1. Neuropraxia: Blockage due to localized pressure without axonal degeneration or nerve sheath interruption. ? Chemical basis.

2. Axonotmesis: Blockage of replenishment of axoplasm to distal segment. Degeneration of myelin sheath without disruption of neurolemmal sheath.

3. Neurotmesis: Disruption of nerve trunk.

4. Synkinesis: A single axon innervating widely separated facial muscles. It also has been postulated that unmyelinated nerve regeneration gives rise to more synkinesis and as more myelin is laid down, less synkinesis is noted.

5. Möbius syndrome: Facial paralysis in the newborn due to central nerve lesion or agenesis of facial muscles.

6. Melkersson-Rosenthal syndrome: Recurrent unilateral or bilateral facial palsy associated with chronic or recurrent edema of face and with fissured tongue. Unknown etiology. Peak age is 20s; histologically, dilated lymphatic channels, giant cells, and inflammatory cells are seen.

7. "Crocodile tears": Regenerating fibers innervate the lacrimal gland instead of the submaxillary gland.

8. Faradic current: Is a high frequency interrupted current that stimulates the nerve directly and elicits an all-or-none response.

9. Galvanic current: Is a constant direct current that stimulates the muscles directly.

10. Bell's phenomenon: The eyeball turns up and out during an attempt to close the eyes.

11. Facial paralysis of central origin is characterized by:

a. Intact frontalis and orbicularis oculi.

b. Intact mimetic function.

c. Absence of Bell's phenomenon.

12. Blood supply of the facial nerve (see Fig. 10-2):

a. ECA --> Posterior auricular artery --> Stylomastoid artery.

b. ECA --> Middle meningeal artery --> Greater superficial petrosal artery.

13. Distances:

Pons to IAM	= 23-24 mm
IAM	= 7-8 mm
Labyrinthine	= 3-4 mm
Tympanic	= 12-13 mm
Mastoid	= 15-20 mm
Parotid before branching	= 15-20 mm
Whole length	= 75-89 mm.

14. The chorda tympani branches off at about 5-7 mm before the stylomastoid foramen.

15. Facial nerve paralysis not involving the greater superficial petrosal nerve would give a "tearing" eye because of:

a. Paralysis of Horner's muscle that dilates the nasolacrimal duct orifice.

b. Ectropia and so produces malposition of the puncta.

c. Absenmce of winking, i.e. lack of the pumping action.

16. The most likely areas of compression in Bell's palsy have been noted to be in the

stylomastoid area and around the pyramidal eminence.

17. Korczyn reported that among 130 patients with Bell's palsy, 66% had either frank diabetes or an abnormal glucose tolerance test. It also has been stated that the percentage of denervation in Bell's palsy is higher in diabetics.

18. In parotid surgery, the facial nerve can be identified at 6-8 mm below the inferior "drop off" of the tympanomastoid fissure. This was described by H. G. Tabb.

19. Twenty-five percent of longitudinal fractures involve the facial nerve; 50% of transverse fractures involve the facial nerve.

20. The facial nerve regenerates at 3 mm/day.

21. Incapacitating facial spasm (particularly of the orbicularis oculi) can be treated by selective avulsion of the facial nerve branches through a parotidectomy approach.

22. Facial nerve innervates:

a. Stylohyoid muscle
b. Posterior belly of digastric muscle
c. Occipitofrontalis muscle - via posterior auricular branch
d. External auricular muscles - via posterior auricular branch
e. Orbicularis oculi - temporal division
f. Occipitofrontalis - temporal division
g. Anterior and superior auriculares - temporal division
h. Orbicularis oculi - upper zygomatic
i. Zygomatic muscles - lower zygomatic
j. Buccinator - buccal
k. Around nose and mouth - buccal
l. Depressor anguli oris - mandibular
m. Depressor labii inferioris - mandibular
n. Orbicularis oris - mandibular
o. Platysma - cervical.

23. Motor root from the motor facial nucleus at the level of pons gives muscular branches (see above) and stapedius nerve.

24. N. intermedius brings taste fibers from nucleus solitarius through chorda tympani to anterior two-thirds of the tongue.

25. N. intermedius brings parasympathetic fibers from superior salivary nucleus to the:

a. Lacrimal gland, palatine and nasal glands through the greater superficial petrosal nerve, vidian nerve, sphenopalatine ganglion, travelling across the maxillary nerve, with ophthalmic nerve and lacrimal nerve.

b. Parotid gland through the deep petrosal nerve, tympanic plexus, lesser superficial petrosal nerve, otic ganglion and then via auriculotemporal nerve.

c. Submaxillary and sublingual gland through chorda tympani that becomes attached to lingual nerve, to submandibular ganglion.

26. Tympanic plexus is formed by VII, IX and X and caroticotympanic nerve that carries sympathetic fibers from carotid plexus.