Original Article

Facial nerve paralysis: Diagnosis, evaluation, and management

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Abstract

Problem Statement: Charles Bell showed that the seventh nerve was responsible for motor innervation of the face. Since that time, many etiologies of facial paresis or paralysis have been identified; controversy still exists, however, over the etiology, diagnostic methods, and treatment of acute facial paralysis. The facial nerve begins developing around week 3 of gestation with a collection of neural crest cells called the acousticofacial primordium that forms the acoustic and facial nerves. The facial nerve develops within the second branchial arch. By week 8, the terminus branches have developed, and the facial muscles develop by week 12. The facial nerve fully develops by age 4. **Conclusion:** Facial paralysis can be devastating to the patient. Prompt recognition of the etiology and intervention are vital. Steroid and antiviral therapy are the hallmarks of initial treatment. Controversies exist regarding the degree of ancillary testing needed and the type of surgical intervention. The American Academy of Otolaryngology-Head and Neck Surgery Foundation is currently working on a consensus statement regarding the management of acute facial paralysis.
Key Words: Facial nerve paralysis, Bell\'s palsy, Surgery management.

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INTRODUCTION

The facial nucleus fibers extend to the precentral gyrus (motor cortex) of the cerebral cortex. The facial nucleus is in the ventrolateral aspect of the pons. The fibers of the facial nucleus extend dorsally around the sixth (abducens) cranial nerve nucleus. They emerge from the lower pons between the olive and the restiform body. The facial nerve is divided into five segments: the intracranial, intracanalicular, labyrinthine, tympanic, and mastoid. The nerve receives its blood supply from the anterior inferior cerebellar artery, the middle meningeal artery, and the stylomastoid branch of the postauricular artery. The facial nerve contains four types of fibers:

1. Branchial or somatic motor (special visceral) efferents innervate the muscles of facial expression, stylohyoid, posterior belly of the digastric, and stapedius.
2. Visceral motor (general visceral) efferent's innervate the lacrimal, nasal, submandibular, and sublingual glands as well as the mucous membrane of the nose and hard and soft palate.
3. Special sensory afferent fibers innervate the anterior two thirds of the tongue via the chorda tympani nerve.
4. General (somatic) sensory afferent fibers innervate the posterior ear canal and conchum cavum.

Incidence

Acute facial paresis/paralysis is one of the most common neuropathies, with an incidence of 15 to 40 per 100,000. This condition is usually diagnosed and managed by multiple specialists. Facial paresis can be due to infectious, ischemic, neoplastic, or autoimmune processes. Acute facial paresis of unknown etiology is termed Bell palsy and is thought to be viral in origin. It is uncommon in young patients, but the incidence increases with age. There is some thought that diabetes mellitus and pregnancy can be aggravating factors in the development of Bell palsy.

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Pathogenesis

As already noted, it is important to realize that there are a multitude of etiologies for acute facial paralysis. Etiologies include congenital, traumatic, infectious, neoplastic, genetic, neurological, vascular, idiopathic, toxic, and iatrogenic. Congenital causes include Mobius syndrome and myotonic dystrophy. Because the facial nerve is exposed near the stylomastoid foramen because the mastoid tip is not developed at birth, delivery trauma, such as forceps use, can be associated with facial paralysis. Trauma to the skull base can lead to facial paralysis. Transverse fractures of the temporal bone, which usually involve the labyrinth, have a high incidence of facial nerve impingement, injury, or - laceration. Penetrating injuries of the face, middle ear, or cranium can cause facial nerve injury. Barotrauma can also lead to facial paralysis. Infections can lead to an inflammatory response around the nerve. Given that roughly 50% of patients have natural dehiscence of the fallopian canal, this loss of bone can lead to perineural involvement of an inflammatory process. Infections of the bony ear canal, mastoid, middle ear, or parotid are common causes. Multiple viral infections, including varicella, Herpesviridae, human immunodeficiency virus, coxsackievirus, influenza, polio, and mononucleosis can lead to facial paresis/paralysis as well. Encephalitis and meningitis can also lead to facial paralysis. It is important to review for any patient history of tuberculosis, Lyme disease, or botulism. Skull base osteomyelitis (malignant otitis externa) is a bony infection of the temporal bone usually caused by *Pseudomonas aeruginosa* and seen in diabetics and immunocompromised patients. Patients present with severe otalgia, often worse at night, otorrhea, and granulation tissue along the floor of the ear canal at the bony-cartilaginous junction. Treatment centers around local debridement, intravenous and topical antibiotics directed toward *Pseudomonas* (fluoroquinolone), and diabetic control. Milder infections can occasionally be treated with oral antibiotics. Neoplastic causes include facial nerve neuromas, hemangiomas, or schwannomas. Tumors of the cerebellopontine angle, such as acoustic neuroma and glomus tumors, can also lead to facial paralysis, although facial paresis in the setting of acoustic neuroma is rare. Malignant brainstem tumors or metastatic carcinomas can affect facial nerve function as well. Unusual and idiopathic causes such as Melkersson-Rosenthal syndrome, thrombotic thrombocytopenic purpura, Cuillain-Barre syndrome, multiple sclerosis, and myasthenia gravis need to be considered. Uncommon illnesses such as sarcoidosis, Wegener granulomatosis, and eosinophilic granuloma are also potential etiologies. Iatrogenic· causes most commonly occur during otologic surgery in the tympanic segment. Injury to the mastoid segment is the next most common site. Iatrogenic injuries most commonly occur when the nerve has not been clearly identified. Before any intervention is done on a patient with an immediate postoperative facial paralysis, the patient should be observed to make sure that this is not a lidocaine induced paralysis. If the paralysis persists, intervention should be initiated immediately. Facial nerve. monitoring during surgery may help with early rec-ognition of injury, and stimulation may enhance the ability to identify the nerve. However, this is not a substitute for surgical skill and knowledge of the anatomy and its variations. Pathophysiology of facial nerve injury has been classified under two systems-Seddon's (1943) and Sunderland's (1951)-based on site of lesion. These systems do have prognostic value when considering outcomes of facial nerve injury (Table 69.1).

EVALUATION

Important details of the history that should be inves-tigated include the time of onset of paresis, the speed of progression, any precipitating factors, and any associated symptoms, such as pain, hearing loss, taste disturbance, tinnitus, otorrhea, or systemic illnesses. These details are important in helping to develop a differential diagnosis. Examination involves careful observation of the face. It is important to describe the face at rest, noting if there is any asymmetry at rest. Also, one should observe for any signs of synkinesis, or mass movement (e.g., when the patient is asked to close the eyes tightly, the corner of the mouth draws up). There should be a careful examination of the ears, looking for infections, granulation tissue, cholesteatoma, or masses that might lead to a paralysis. It is also important to note any signs of vesicular lesions that are around the auricle or on the face, consistent with Ramsay Hunt syndrome. The House-Brackmann scale is used to describe the degree of facial weakness and the level of recovery. This grading system uses a scale of I to VI with the grades described briefly as follows:

- I: Normal movement
- II: Minimal weakness with symmetry at rest, possibly with mild synkinesis
- III: Mild to moderate weakness, with eye closure and symmetry at rest
- IV: Symmetry at rest with some movement and incomplete eye closure
- V: No asymmetry or mild asymmetry at rest with no movement
- VI: No movement and gross asymmetry at rest

Special Investigations

Nearly 70% of facial paresis can be attributed to Bell palsy. In patients where there is a suspicion of other possible etiologies based on history and physical exam, additional testing may be warranted. Further.

Table 1: Classification of facial nerve injury

Seddon Classification	Name	Site of lesion	Characteristics
Class I	Neuropraxia	conduction block	Intact nerve; temporary; no wallerian degeneration; full recovery.
Class II	Axonotmesis	Axon/myelin sheath	Preservation of endo-, peri-, and epineurium; Wallerian degeneration occurs; recovery overall good but may be incomplete.
Class III	Neurotmesis	Transection	Total disruption; Wallerian degeneration; recovery poor without surgery.
Sunderland Classification			
Class I(1st degree)	Neuropraxia	conduction block	Intact nerve; temporary; no wallerian degeneration; full recovery.
Class II(2nd degree)	Axonotmesis	Axon/myelin sheath	Preservation of endo-, peri-, and epineurium; Wallerian degeneration occurs; recovery overall good but may be incomplete.
Class II(3rd degree)	Neurotmesis	Transection	Peri-and epineurium intect; recovery possible, may be incomplete.
Class II(4th degree)	Axonotmesis	Axon/endo- and perineurium	Epineurium intact; recovery incomplete; surgery advice
Class III(5th degree)	Neurotmesis	Transection	Recovery poor without surgery.

Investigation may include an audiogram. It is important to document any type of hearing loss. It is helpful to note if the stapedius reflex is intact. Patients with Lyme disease may present with recurrent or bilateral paralysis. There are several blood tests that may be helpful if there is a possibility of Lyme disease exposure, including a serum Lyme titer, a complete blood count, sedimentation rate, glucose testing, and angiotensin-converting enzyme level testing. Imaging can play an important role in the workup of facial paralysis. The test of choice is magnetic resonance imaging with gadolinium. It is important that this test is protocoled to encompass all segments of the facial nerve, including the parotid gland. Imaging is not recommended in the acute presentation of facial paresis/paralysis. Imaging should be considered when a Bell palsy does not show any signs of recovery after 4 to 6 months, or if there is segmental paralysis. If there is a history of trauma, then a computed tomographic scan without contrast is the exam of choice to evaluate the bony anatomy. One of the most helpful tests in facial nerve paralysis is electrical testing. Electromyography (EMG) and evoked EMG can evaluate motor unit potentials. This test is helpful if the patient is first being seen 10 to 14 days after the onset of the paralysis. The presence of motor unit potentials shows that the nerve is still innervating the muscle. Multiphasic action potentials indicate that the nerve is beginning to reinnervate the muscle. another good prognostic sign. Fasciculations are a poor prognostic sign for recovery. EMG is not necessary for patients with incomplete paresis (House-Brackmann score I-V) because studies show these patients will enjoy good recovery of function. Electroneuronography (ENoG) is very important in the early stages of a complete paralysis. The test is very helpful to determine the degree of degeneration of the nerve compared with the contralateral, normal side. This test is ideally performed within the first 2 weeks of the onset of a paralysis and can be performed 72 hours after the onset. If the test suggests> 90% weakness compared with the normal side, then surgical decompression should be considered.

MANAGEMENT

Treatment of acute facial paralysis always includes steroids unless there are contraindications, such as poorly controlled diabetes. Typical steroid dosing is 1 mg/kgrd of prednisone in a single dose on a full stomach in the morning tapered over 2 to 3 weeks. Other considerations for treatment include antiviral regimens (e.g., oral valacyclovir or famciclovir 500 mg three times a day). One very important consideration is eye care. The inability to close the eye can lead to corneal inflam mation and scarring. Patients should be instructed on the use of artificial tears and ophthalmic ointments to keep the eye moist. Taping or patching the eye at night may also help. Moisture chambers for the eye are also available if the patient needs protection during the day. Eyeglasses or sunglasses are strongly encouraged to keep airborne debris out of the eye, especially on windy days. Physical therapy may help with recovery. Patients are encouraged to try to move their face, even though they cannot see any movements. This allows the muscle to be stimulated with neurotransmitters to prevent atrophy. Electrical stimulation is also helpful in patients with a chronic or long-term paralysis and is thought to prevent muscle atrophy. These patients should continue their physical therapy.

Surgical Intervention

Surgical treatment of facial paralysis varies by etiology. It is widely accepted that iatrogenic injuries should be explored as soon as possible after allowing adequate time for any local anesthetic effect to wear off. In the case of acute trauma, exploration is usually performed in the setting of a complete (usually immediate-onset) paralysis. Surgical approach is generally through a combined trans mastoid and middle fossa craniotomy. Any impingements should be removed and the facial nerve canal decompressed in the labyrinthine and perigeniculate regions. If an injury has caused more than 50% of the nerve to be transected, a reanastomosis or graft should be performed. If a segment is missing, then a graft should be considered. Facial nerve monitoring will be helpful in identifying the damaged nerve or its transected ends (as long as there has been no Wallerian degeneration, which occurs 72 hours after the injury). Even with a grafting procedure, the expected best recovery is a House-Brackmann grade III. For patients with traumatic injuries, other common grafting procedures can be performed. The hypoglossal-facial transfer (XII-VII) is a very common procedure. The facial nerve is skeletonized and decompressed as medial as possible in the mastoid and is cut and transposed into the neck, where it is grafted to the hypoglossal nerve. Alternatively, the hypoglossal nerve can be partially transected and delivered up to the facial nerve trunk at the stylomastoid foramen. A cross-facial nerve (VII-VII) graft can also be performed. In acute idiopathic facial nerve paralysis (Bell palsy), surgical decompression may be considered when the ENoG shows> 90% degeneration compared with the normal side. This surgery should be undertaken within 14 to 21 days of the onset of complete paralysis. There is controversy over whether the nerve, including the internal acoustic canal portion, can be decompressed through a mastoid approach. Usually, a total decompression is performed through a combination of middle fossa and transmastoid approaches. Newer techniques are being developed to rehabilitate patients who regain little to no normal nerve function. Several procedures can be performed around the eye to aid in closure. A tarsorrhaphy can be performed to tighten the lower lid to prevent severe epiphora. The more common procedure performed around the eye is the placement of a gold weight in the eyelid to allow for closure of the lid when the third cranial nerve is relaxed. There are many procedures to improve the appearance of the face. Static slings, rhytidectomy, blepharoplasty, and browlift can improve the resting tone and symmetry of the face. Newer procedures allow for active motion. The first is a temporalis transfer, which rotates slings of the temporalis muscle and attaches them to different areas of the face. Free gracilis muscle transfer has also shown some promise. One of the unfortunate sequelae of recovery from a facial paralysis is synkinesis. When the peripheral fibers regenerate, their original directionality is lost. Therefore, fibers that should be innervating the perioral muscles may reinnervate the periorbital muscles. This commonly causes patients to wink their eye when they smile. Another sequela of recovery is spasm, which is typically seen after recovery of a facial paralysis after skull base surgery. In either circumstance, if synkinesis is severe, it can almost be as bothersome as the weakness itself. To lessen these side effects, botulinum toxin is usually targeted to the most affected muscle groups; however, overapplication of botulinum toxin may result in paresis.

CONCLUSION

Facial paralysis can be devastating to the patient. Prompt recognition of the etiology and intervention are vital. Steroid and antiviral therapy are the hallmarks of initial treatment. Controversies exist regarding the degree of ancillary testing needed and the type of surgical intervention. The American Academy of Otolaryngology-Head and Neck Surgery Foundation is currently working on a consensus statement regarding the management of acute facial paralysis.

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