

Peripheral Facial Palsy: Anatomy And Physiology. An Update

C Pereira, E Santos, J Monteiro, A de Araújo Morais, C Tôrres Santos

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Abstract

The peripheral facial palsy attacks about 20-60 persons in 100000 per year. The course, in many times, is benign. It is a review article that exposes the principal pathological entities that cause facial paralysis with emphasis in accurate diagnosis beginning from the medical history, and physical examination.

INTRODUCTION

Peripheral facial paralysis is one of the commonest mononeuropathies. The frequency of idiopathic peripheral paralysis or Bell's palsy varies between 62% and 93% of all cases. The annual incidence of Bell's palsy is about 20 per 100,000, with an increasing incidence seen with increasing age. The topographic diagnosis of the lesion is based on symptoms associated with the paralysis. The facial nerve is a mixed nerve with special visceral efferent, general visceral efferent, special visceral afferent, and general somatic afferent functions. The objective of this review is to discuss the anatomy and physiology and correlates with clinical findings in lesions of the facial nerve.

FACIAL NERVE ANATOMY

A synopsis of facial nerve anatomy is important in understanding its pathophysiology ^{1,2,3,4,5,6,7}

The facial nerve is a mixed nerve with special visceral efferent, general visceral efferent, special visceral afferent, and general somatic afferent functions ^{3,5}. The facial nerve emerges from the brainstem along with a smaller nerve (nervus intermedius of Wrisberg) and courses laterally to the internal auditory canal (IAC). The facial nerve enters with the cochleovestibular nerve into IAC ^{3,5}. The intracranial portion of the nerve is able to withstand slow stretching from a tumor but is quite susceptible to trauma. The facial nerve, along with the intermediate nerve of Wrisberg and the auditory nerve, passes through the internal auditory canal. As the nerve travels farther laterally in the IAC, it enters the narrow fallopian canal, becoming encased in periosteum and epineurium. Of significance, the narrowest portion is at the

labyrinthine section, which contains the geniculate ganglion. The facial nerve occupies 83% of the available space compared with 23% in the mastoid portion. It is the labyrinthine portion where it is most likely that a vicious cycle of edema from infection, vascular compromise, and ischemia, thought to be the pathophysiologic process in Bell's palsy, can occur. Four branches of the facial nerve occur within the fallopian canal (the greater and lesser superficial petrosal nerves, the nerve to the stapedius muscle, and the chorda tympani nerve). The stylomastoid foramen opens at the base of the petrosa between the mastoid process and the styloid. The facial nerve runs anteriorly in the substance of the parotid gland, crosses the external carotid artery, and divides at the posterior border of the ramus of the mandible into two primary branches: the superior (temporofacial) and the inferior (cervicofacial), from which numerous offsets, in a plexiform arrangement called the parotid plexus. These nerves are responsible for facial expression, eye closure, and assistance with mastication and speech. The branches of the facial nerve in the parotid space are: 1. the ansa of Haller (inconstant); 2. the posterior auricular branch; 3. the stylohyoid branch; 4. the posterior belly of the digastric branch; 5. the lingual branch (inconstant).

The facial nerve derives both an intrinsic and an extrinsic blood supply. The extrinsic vascular supply comes from three primary sources (the stylomastoid artery, the petrosal artery, and the internal auditory artery) and derives the intrinsic system. The narrow labyrinthine portion of the nerve is a transition watershed zone between blood supply from the vertebral and carotid artery systems. This

vasculature courses between the epineurium and the bony periosteum and is jeopardized by trauma, swelling from infection, and surgical injury. During these times, survival of the nerve depends on the tenuous supply of the intrinsic system.

PHYSIOLOGY OF THE FACIAL NERVE

The facial nerve carries three types of nerve fibers: branchial motor, visceral motor and visceral sensory ^{2,6,7,8,9,10,11}. The site of lesion determinates the clinical findings (Table 1).

Figure 1

Table 1: Clinico-Anatomical Correlation of Disorders of Facial Cranial Nerve.

Anatomical Site of Damage	CN VII Finding	Other Neurological and Medical Findings	Common Etiologies	Comments
Motor cortex	Palsy of lower face (upper face spared), tone and emotional movements intact, tearing, salivation, taste intact	Hemiparesis (ipsilateral) Tongue weakness (ipsilateral) Frontal lobe signs	Vascular lesion of motor cortex or internal capsule on the side contralateral to the weakness	Facial weakness rarely seen as sole finding. Spasticity and hyperreflexia are found
Posttine nucleus	Facial palsy on same side as the lesion (upper and lower), tearing, salivation, taste intact	Hemiparesis (contralateral) Aldoacus palsy (ipsilateral)	Infarction (Millard-Gubler and Foville syndromes), pontine glioma, multiple sclerosis, thalamic tonecity, Mobius syndrome	The lesion is below the facial decussation and above the corticospinal decussation. If congenital, may see anomalies of the ear or jaw
Cerebellopontine angle	Facial palsy on same side as the lesion (upper and lower), tearing, salivation, taste may be abnormal	Decreased hearing, increased tinnitus, vestibular impairment (CN VIII), facial numbness (CN V), ataxia incoordination (cerebellum)	Acoustic neuroma, meningioma facial nerve neuroma, cholesteatoma	CN VIII is usually involved, and its symptoms are usually the chief complaint, especially unilateral hearing loss. As lesions enlarge, other structures are involved
Internal auditory meatus (neatal segment)	Facial palsy on same side as the lesion (upper and lower), tearing, salivation, taste probably involved	Decreased hearing, increased tinnitus, vestibular impairment (CN VIII)	Intracanalicular neuroma (acoustic or facial)	Signs and symptoms from CN VII and CN VIII only
Facial canal labyrinthine segment	Facial palsy on same side as the lesion (upper and lower), tearing, salivation, taste probably involved	Decreased hearing, increased tinnitus, vestibular impairment (cochlea or vestibule involvement)	Bells palsy, Ramsay Hunt syndrome, temporal bone fracture, hemangioma	When the facial canal is involved, the cochlea and/or the vestibular organs may be involved. Hearing loss can be conductive or sensorineural
Facial canal tympanic segment	Facial palsy on same side as the lesion (upper and lower), salivation, taste probably involved (tearing spared)	Decreased hearing, increased tinnitus, vestibular impairment	Bells palsy, otitis media, cholesteatoma, temporal bone fracture	If there was trauma, check for CSF otorrhea, blood in ear canal, Battle s sign, impaired consciousness
Facial canal mastoid segment	Facial palsy on same side as the lesion (upper and lower), salivation, taste may be involved (tearing spared)	Decreased hearing, increased tinnitus, vestibular impairment	Bells palsy, otitis media, cholesteatoma, glioma tumor	Lesions before the chorda tympani have impaired salivation and taste
Styloaastoid foramen	Facial palsy on same side as the lesion (upper and lower), tearing, salivation, taste not involved	Evidence of facial trauma, parotid or pharyngeal mass, uvulitis	Facial laceration, fracture, or surgery, parotid tumors, parotidosis	Sparing of peripheral branches may be seen

The facial expression depends on the 7000 motor fibers of the facial nerve firing in unison to bring about contraction ¹². The motor nucleus of the facial nerve provides branchial motor fibers to all the muscles of the face except the levator of the upper eyelid. Over and above that, the motor fibers also supply the stapedius muscle, the postauricular muscles, the posterior belly of digastric and the platysma. Visceral motor preganglionic, parasympathetic fibers from the lacrimal nucleus pass by way of the intermediary nerve through the geniculate ganglion and greater superficial nerve. Visceral motor fibers from the superior salivatory nucleus provide preganglionic, parasympathetic fibers to the sphenopalatine ganglion, where postganglionic fibers traverse the palatine nerves to palatal glands and nasal nerve to nasal glands. Other visceral motor preganglionic,

parasympathetic fibers from the superior salivatory nucleus leave the facial nerve through the chorda tympani nerve to the lingual nerve, then supply the submandibular and sublingual glands. Visceral sensory fibers have their cell bodies in the geniculate ganglion and receive impulses from the taste buds of the anterior two thirds of the tongue.

PERIPHERAL FACIAL NERVE PATHOLOGY

The differential diagnosis of facial palsy is showed in Table 2. Pathology involving facial nerve in cerebellopontine angle often involves acoustic nerve (sensorineural hearing loss, tinnitus, vestibular weakness) ¹. Common etiologies include acoustic neuromas ¹⁴and meningiomas. In the labyrinthine segment, facial palsy is seen with ipsilateral hearing loss and vestibular weakness. Hyperacusis and a decrease in tearing and in salivation with loss of taste can be detected. With lesions of the tympanic segment that are distal to the greater superficial petrosal nerve, all of the above may be found, except that lacrimation is intact. If the mastoid segment is involved, as with tympanic lesions, hearing and balance may be affected, and lacrimation is preserved. Whether the lesion is proximal to the nerve branch for the stapedius or to the chorda tympani determines whether there is hyperacusis, decreased salivation, or loss of gustation. Hemangiomas, neuromas, cholesteatomas, temporal bone fractures, and the inflammation causing Bell's palsy are some disorders of the facial canal that can cause facial palsy ^{1,7,15}. A lesion after the nerve leaves the temporal bone results in only a lower motor neuron deficit of the mimetic muscles. Atrophy and fasciculations are found. Pathology of individual nerve branches can be seen with parotid tumors, facial or mandibular trauma, and after facial surgery.

Figure 2

Table 2: Causes of facial palsy identified in a review of medical literature (1900-1990).

Birth
Molding
Forceps delivery
Dystrophia myotonica
Mobius syndrome (facial diplegia associated with other cranial nerve deficits)
Trauma
Basal skull fractures
Facial injuries
Penetrating injury to middle ear
Altitude paralysis (barotrauma)
Scuba diving (barotrauma)
Lightning
Neurologic
Opercular syndrome (cortical lesion in facial motor area)
Millard-Gubler syndrome (abducens palsy with contralateral hemiplegia caused by lesion in base of pons involving corticospinal tract)
Infection
External otitis
Otitis media
Mastoiditis
Chickenpox
Herpes zoster ophthalmicus (Ramsay Hunt syndrome)
Encephalitis
Polioomyelitis (type I)
Mumps
Mononucleosis
Leprosy
Influenza
Coxsackievirus
Malaria
Syphilis
Scleroma
Tuberculosis
Botulism
Acute hemorrhagic conjunctivitis (enterovirus 70)
Gnathostomiasis
Mucormycosis
Lyme disease
Cat scratch
AIDS

Figure 3

Metabolic
Diabetes mellitus
Hypothyroidism
Pregnancy
Hypertension
Acute porphyria
Vitamin A deficiency
Neoplastic
Benign lesions of parotid
Cholesteatoma
Seventh nerve tumor
Olfactory jugular tumor
Leukemia
Meningioma
Hemangioblastoma
Sarcoma
Carcinoma (involving or metastatic)
Anomalous sigmoid sinus
Carotid artery aneurysm
Hemangioma of tympanum
Hydradenoma (external canal)
Facial nerve tumor (cylindroma)
Schwannoma
Teratoma
Hand-Schuller-Christian disease
Fibrous dysplasia
Neurofibromatosis II
Toxic
Thalidomide (Mishkitz syndrome, cranial nerves VI and VII with congenital malformed external ears and deafness)
Ethylene glycol
Alcoholism
Arsenic intoxication
Tetanus
Diphtheria
Carbon monoxide

Figure 4

Iatrogenic
Mandibular block anesthesia
Antitetanus serum
Vaccine treatment for rabies
Postimmunization
Parotid surgery
Mastoid surgery
Posttonsillectomy and adenoidectomy
Iontophoresis (local anesthesia)
Embolization
Dental
Idiopathic
Bell's, familial
Melkersson-Rosenthal syndrome (recurrent alternating facial palsy, furrowed tongue, facialial edema)
Hereditary hypostrophic neuropathy (Charcot-Marie-Tooth disease, Dejerine-Sottas disease)
Autoimmune syndrome
Amyloidosis
Temporal arteritis
Thrombotic thrombocytopenic purpura
Periarteritis nodosa
Lambert-Eaton-Ryden syndrome (ascending paralysis)
Multiple sclerosis
Myasthenia gravis
Sarcoidosis (Hersfordt syndrome-inoaparotid fever)
Osteopetrosis

Despite a thorough workup, 60 to 75 percent of all cases of facial paralysis are of unknown etiology and are called Bell's palsy^{1,7,15,16,17,18}. It is a diagnosis of exclusion: it is accurate only when no other cause for facial palsy can be found. Some characteristics are: it usually is acute in onset and unilateral, peripheral facial nerve dysfunction involving all distal branches, sudden onset with maximal facial weakness usually reached within several days, reduction in ipsilateral tearing or salivary flow in 10 percent, spontaneous improvement within 6 months, numbness or pain of the ear, face, neck or tongue is present in about 50 % of patients, 60 % of patients have a viral prodrome, a minority of patients have a recurrent facial palsy, a family history for Bell's palsy is present in about 14 % of cases, loss of ipsilateral tearing or submandibular salivary flow may be present in 10 %, about 90 % of cases have decreased or absent ipsilateral stapes reflex, and the chorda tympani nerve appeared red in 40 % of patients evaluated in the first 10 days after onset in whom this nerve could be seen^{1,7,13,15,18,19}. The annual incidence of Bell's palsy is about 20 per 100,000, with an increasing incidence seen with increasing age. The male-to-female and left-to-right ratio is approximately equal. High-risk patients include pregnant women and patients with diabetes mellitus or multiple sclerosis. The risk to pregnant women is 3.3 times greater than that for nonpregnant women^{15,20}. The risk of recurrence is 10%, and this recurrence may occur on either side. Of those who experience recurrence three times, the risk of a fourth recurrence is 50%^{15,21}. Diabetics are at higher risk²² (4.5 times more likely), and the risk of Bell's palsy increases with each decade of life²³.

Based on accumulated clinical and experimental evidence, the most commonly accepted cause of Bell's palsy is herpes simplex infection^{7,15,18}.

The Ramsay Hunt Syndrome is characterized by facial paralysis, herpetic vesicular eruptions, and vestibulocochlear dysfunction^{7,24}. Vesicular eruptions may occur over the ear, face, and neck down to the shoulder. There is more pain than in Bell's palsy, a greater likelihood of hearing loss, and a lower incidence of a complete recovery from facial paralysis (54%)¹. Pain was deep in the face, often radiating to the ear, and sometimes associated with lacrimation, nasal congestion, and salivation²⁴. A complete recovery of hearing loss occurs in roughly 45% of patients.

Lyme disease (*Borrelia burgdorferi*) is a multisystem infectious disease with prominent neurologic, cardiac, and dermatologic manifestations^{7,25,26}. Symptoms after a tick bite include headache, malaise, myalgias, chills and fever, nausea and vomiting, and neurologic sequelae. Facial paralysis may occur from an acute bacterial infection of the middle ear or mastoid, chronic otitis media, or necrotizing otitis externa^{1,7}. Cholesteatoma should be suspected if the onset of paralysis is gradual. This is contrasted to the 2- to 3-day onset of paralysis in acute otitis media. In immunocompromised hosts with otitis externa which spreads via vascular and fascial planes to cause cellulitis of the skull base and facial paralysis^{7,27,28}.

Intratemporal facial paralysis may be of a head trauma or may be iatrogenic in nature. Facial paralysis secondary to surgery of the middle ear and mastoid ranges in incidence from 0.2% to 1.4%^{7,29,30}. Temporal bone fractures are typically described in terms of the relationship of the fracture line to the long axis of the petrous pyramid. Longitudinal fractures are most common and constitute 90% of fractures. Injury commonly occurs in the region of the geniculate ganglion. Transverse fractures are less common but result in 38% to 50% of cases of facial paralysis³¹.

Melkersson-Rosenthal is characterized by facial paralysis, episodic facial swelling, and a fissured tongue (scrotal tongue or lingua plicata) and typically begins in adolescence and has been described as both sporadic and familial^{1,7,13,28,32}. The cause is unknown. With each episode of paralysis, facial edema worsens, and progressive disfigurement may occur.

CORRESPONDENCE TO

Prof. Dr. Carlos Umberto Pereira Av. Augusto Maynard,
245/404 49015-380 Aracaju – Sergipe e-mail:
umberto@infonet.com.br

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Author Information

Carlos Umberto Pereira

Department of Medicine, Federal University of Sergipe

Egmond Alves Silva Santos

Department of Neurosurgery, Conjunto Hospitalar do Mandaqui

João Tiago Silva Monteiro

Department of Medicine, Federal University of Sergipe

Aníbal de Araújo Moraes

Department of Physiotherapy, Tiradentes University

Carlos Michell Tôrres Santos

Department of Physiotherapy, Tiradentes University