IJARETY

International Journal of Advanced Research in Education and TechnologY (IJARETY)

Volume 11, Issue 6, November-December 2024

Impact Factor: 7.394

www.ijarety.in editor.ijarety@gmail.com

International Journal of Advanced Research in Education and TechnologY(IJARETY)

| ISSN: 2394-2975 | www.ijarety.in| | Impact Factor: 7.394| A Bi-Monthly, Double-Blind Peer Reviewed & Referred Journal |

|| Volume 11, Issue 6, November-December 2024 ||

DOI:10.15680/IJARETY.2024.1106106

'Facial Nerve' and Applied Anatomy- A Narrative Review

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ABSTRACT: A complicated cranial nerve with a variety of motor, sensory, and autonomic activities is the face nerve (cranial nerve VII). It is a crucial factor in many medical and surgical specialties because of its complex physical relationship with important head and neck tissues, extending from the brainstem to its terminal branches. Clinically, there are several difficulties in diagnosing and treating diseases including Bell's palsy, trauma, and malignancies because of the facial nerve's involvement. Its use in reconstructive and cosmetic surgery also emphasizes the need of having accurate anatomical knowledge. The care of face nerves has been transformed by developments in imaging technologies and surgical methods, such as nerve grafting and functional rehabilitation. In order to maximize patient care and results, this review highlights how crucial it is to comprehend the applied features of the facial nerve.

I. INTRODUCTION

The seventh cranial nerve is responsible for much of what makes us individual--the facial expression worn by each individual.[1] Facial nerve anatomy is categorized in terms of its relationship to the cranium and temporal bone (intracranial, infratemporal, and extratemporal) or its four distinct segments (branchial motor, visceral motor, general sensory, and special sensory) The facial nerve nucleus comprises of motor, sensory and parasympathetic motor component. The sensory root is composed of both a sensory component and a parasympathetic motor component. The sensory root provides taste function to the anterior two thirds of the tongue, sensory control of lacrimation, and controls the stapedial reflex. Associated with this functionality it controls the output of the sublingual and submandibular glands.[1] The sensory nuclei of the facial nerve are located in the upper medulla and lower pons.

GUSTATORY (SOLITARY) NUCLEUS

The solitary nucleus is located in the rostral and lateral part of the medulla oblongata. Taste fibers from the anterior 2/3 of the tongue reach the geniculate ganglion via the chorda tympani nerve and from there travel to the nucleus of the tractus solitaries. The nucleus is shared among the facial nerve (anterior two-thirds of the tongue and palate), the glossopharyngeal nerve (posterior third of the tongue and vallate papillae) and the vagus nerve (epiglottis).[2]

PARASYMPATHETIC (SUPERIOR SALIVATORY) MOTOR NUCLEUS OF THE FACIAL NERVE

The superior salivatory and lacrimal nucleus is just slightly superior to the gustatory nucleus, at the inferior pons. These preganglionic fibers travel to the submandibular ganglion via the chorda tympani nerve to innervate the submandibular and sublingual glands, and to the sphenopalatine ganglion via the greater superficial petrosal nerve to innervate the lacrimal, nasal, and palatine glands. The superior salivatory nucleus is activated by olfaction, corneal and nasal irritation, and stimulation of taste buds in the oral mucosa.[3]

MOTOR NUCLEUS -The motor nucleus of the facial nerve lies deep within the pons where it receives input from the precentral gyrus of the motor cortex, which innervates the ipsilateral and contralateral forehead. The cerebral cortical tracts also innervate the contralateral portion of the remaining face. This accounts for the sparing of the forehead motion in supranuclear lesions of the facial nerve. It is also known as the branchiomotor nucleus (special visceral motor nucleus) as its efferent fibres supply the muscles which develop from the second pharyngeal (branchial) arch. These

| ISSN: 2394-2975 | www.ijarety.in| | Impact Factor: 7.394| A Bi-Monthly, Double-Blind Peer Reviewed & Referred Journal |

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include muscles of the scalp, facial expression and the auricle, the buccinator, platysma, posterior belly of digastric, stylohyoid and stapedius.[2]

The motor nucleus of the facial nerve provides the efferent component of refexes initiated by stimulation of other sensory cranial nerves. These refexes include blinking in the corneal refex, forcible closure of the eyelids in sneezing, contraction of the orbicularis oculi narrowing the palpebral fissure in response to bright light, and contraction of the stapedius in response to loud sound.[2]

INTRA-TEMPORAL PART OF THE FACIAL NERVE (Fig.1)

The facial nerve courses through the temporal bone and may be further sub-divided into 4 segments: the meatal, the labyrinthine, the tympanic, and the mastoid segments.[4] The facial nerve and nervus intermedius exit the brain stem at the pontomedullary junction and travel laterally with the eight cranial nerve to enter the internal acoustic meatus. Within the internal acoustic meatus, the meatel segment of facial nerve runs laterally. The meatal foramen, the narrowest portion of the fallopian canal is about 0.68 mm. It has been implicated in the etiology of Bell palsy, as the narrow diameter and bony confines leave little room for expansion due to edema or inflammation.[5] The labyrinthine segment shortest of the infratemporal segments is approximately 4 mm and extends perpendicular to the temporal bone and terminates at the geniculate ganglion. The geniculate ganglion gives rise to the first branch of the facial nerve— the greater petrosal nerve—which carries visceral motor parasympathetic fibers to the lacrimal gland. The external petrosal nerve is the second branch that is occasionally present and provides sympathetic innervation to the middle meningeal artery. The lesser petrosal nerve is the third branch from the geniculate ganglion. This branch typically carries parasympathetic fibers associated with the glossopharyngeal nerve (ninth cranial nerve) to the parotid gland. The junction of the labrinthine and tympanic segments of the facial nerve is characterized by a relatively acute angle. This turn in the nerve is referred to as the external, anterior, or first genu of the facial nerve. Compression of the facial nerve within the labyrinthine segment is common given the canal's narrow dimensions.[6]

The tympanic segment begins just distal to the geniculate ganglion extends approximately 1 cm and runs in a horizontal manner. The termination of the tympanic segment of the facial nerve is marked by another abrupt change in direction of the nerve where it connects with the mastoid segment The change to a vertical course is known as the posterior or second genu of the facial nerve.

The longest intratemporal segment of the facial nerve is the mastoid segment, which descends just anterior to the mastoid process. There are 2 important branches of the mastoid segment of the facial nerve. The first is the nerve to the stapedius muscle, which originates from the junction of the superior one third and inferior two thirds of the segment, to innervates the stapedius muscle. Paralysis of the stapedial nerve due to nerve damage leads to hyperacusis, intensification of sounds in the related ear. Because the cell bodies of this motor nerve are not located in the facial nuclei, patients with congenital facial palsies such as Mobius syndrome retain innervation to the stapes when the other facial mimetics are paralyzed.[7]

The second branch is the chorda tympani nerve is the terminal extension of the nervus intermedius, which arises from the inferior one third of the mastoid segment just above the stylomastoid foramen. It joins the lingual branch of the trigeminal nerve to provide parasympathetic innervation to the submandibular and sublingual glands. Two types of fibres are contained in the chorda tympani nerve: parasympathetic preganglionic fibres from the superior salivatory nucleus, and special visceral taste fibres from the anterior two-thirds of the tongue. The chorda tympani exits the temporal bone through the petrotympanic fissure.[1]

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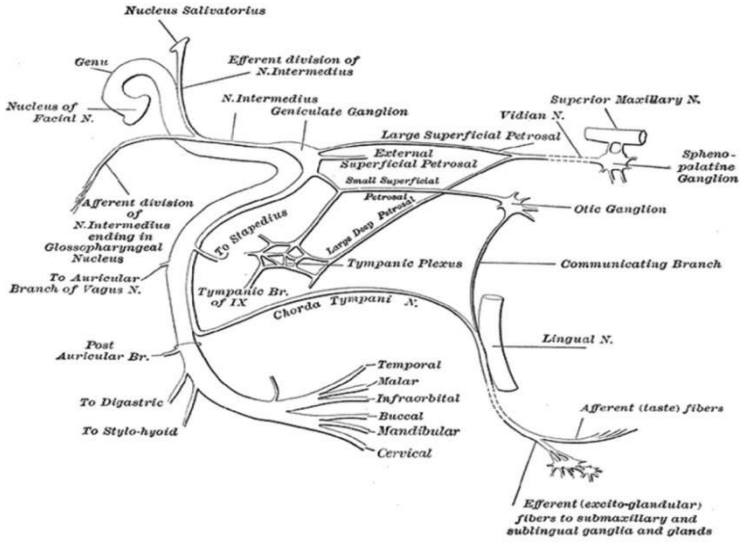

Fig.1. The Intratemporal and Extratemporal course of the facial nerve. (Source: Gray H. Gray's anatomy: Anatomy of the human body. 20th edition [3])

EXTRA TEMPORAL BRANCHES OF THE FACIAL NERVE: (Fig 2)

The extratemporal component of the facial nerve begins when the facial nerve exits the stylomastoid foramen. In the adult, it is protected laterally by the mastoid tip, tympanic ring, and mandibular ramus and in children younger than 2 years it is relatively superficial. Postauricular incisions in children must be carefully planned because the trunk of the facial nerve is a subcutaneous at this level. As the facial nerve exits the stylomastoid foramen, immediately posterior to the styloid process gives off branches before entering the parotid gland, motor branches to the posterior belly of digastric, stylohyoid, superior auricular, posterior auricular(Haller's ansa), and occipitalis muscles. Before the facial nerve innervates the parotid gland, it typically divides into 2 major divisions, a temporofacial and cervicofacial branch. There are multiple branches that enter the parotid gland, and are connected by a complex series of communicating branches.[8] The facial nerve trunk is usually identified approximately 1 cm deep and just inferior and medial to the tragal point. The parotid and superficial musculoaponeurotic system (SMAS) can then be carefully divided to expose the facial nerve for facial nerve reconstruction.[9]

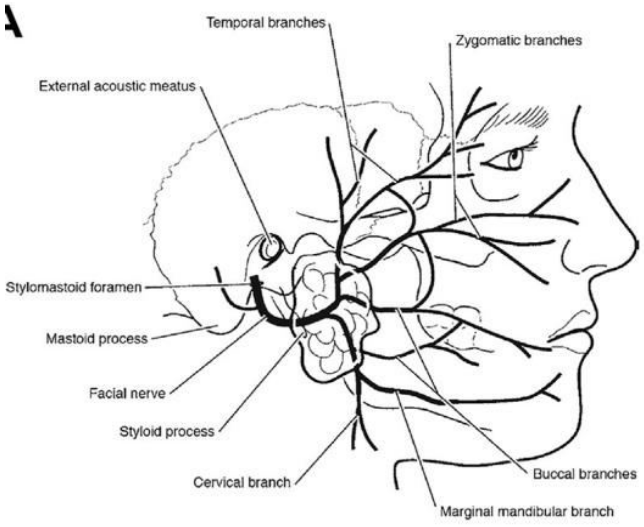

Fig 2- The Extra temporal branches or the facial nerve. [Source: Kochhar A, Larian B, Azizzadeh B. Facial Nerve and Parotid Gland Anatomy. Otolaryngol Clin North Am. 2016 Apr;49(2):273-84. [4]]

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The main trunk of the facial nerve, the pes anserinus, divides into 2 trunks: an upper trunk that gives rise to the frontal, zygomatic, and buccal branches and a lower trunk that terminates in the marginal mandibular and cervical branches.

The frontal branch runs parallel to the superficial temporal vessels and runs across the central portion of the zygoma to supply the frontal belly of the occipitofrontalis, the orbicularis oculi, the corrugator supercilii, and the anterior and superior auricular muscles. The zygomatic branch over the periosteum of the zygomatic arch to innervate the zygomatic, orbital, and infraorbital muscles. The buccal branch travels with the Stensen duct anteriorly over the masseter muscle to supply the buccinator, upper lip, and nostrils. The marginal mandibular branch runs along the inferior border of the parotid gland to innervate the lower lip and chin muscles. The cervical branch innervates the platysma . All muscles of facial expression are innervated on their deep surface except for the mentalis, levator anguli oris, and buccinator muscles.[10] Interconnections exist between the branches of zygomatic and buccal branches hence there is higher rate of recovery of function in distal injuries in this region, as well as the high rate of synkinesis that accompanies recovery of a proximal injury.[11]

ANATOMICAL LANDMARKS TO IDENTIFY DISTAL FACIAL NERVE BRANCHES

BRANCH	LOCATION
Frontal	Found within a triangle bordered laterally by a line drawn from 0.5 cm below the tragus to 2 cm above the lateral brow, inferiorly by the zygomatic arch, and medially by the margin of the lateral orbital rim.[12]
Zygomatic/ buccal	Identified at the midway point on a line drawn from the root of the helix and the lateral commissure of the mouth .[13]
Marginal mandibular	Posterior to the facial artery, the marginal mandibular nerve is located above the inferior border of the mandible in 80% but almost all of the time when anterior to the facial artery.[10]

SUBDIVISIONS AND FUNCTION OF THE FACIAL NERVE[14]

SUBDIVISION	FUNCTION
Branchial (special visceral efferent)	Motor innervation of muscles of facial expression, stylohyoid, stapedius, posterior belly of digastric
Visceral motor (general visceral efferent)	Preganglionic parasympathetic innervation of sublingual/submandibular gland, lacrimal gland, nasal mucosa/mucous membrane
Special sensory (special afferent)	Provides taste from the anterior two-thirds of the tongue via the chorda tympani nerve
General sensory (general somatic afferent)	Provides sensory input from the auricular concha, portions of external auditory canal, and tympanic membrane

CLINICAL PROBLEMS AFECTING THE FACIAL NERVE

Facial nerve deficits may occur affecting the nervous system at different levels of the facial nerve pathway.

Stroke: Stroke or cerebrovascular accident often involves the internal capsule of the brain. The corticonuclear and cortcospinal tracts are known as upper motor neurons, while motor cranial nerve nuclei and anterior horn cells of the spinal cord are lower motor neurons. Upper motor neuron lesions (above the facial nucleus in the pons) produce paralysis of muscles of lower half of the face only because the upper part of the facial motor nucleus receives input from the motor cortex of both sides of the brain. The patient is able to wrinkle the forehead and close the eye. As the lower part of the facial motor nucleus receives input from the contralateral motor cortex only, the lower facial muscles contralateral to the brain lesion will show paralysis. Thus, sparing of the upper facial muscles is diagnostic of an upper motor neuron lesion.

LOWER MOTOR NEURON LESIONS:

These include lesions of the facial nerve nucleus or distal to it and the site can be determined by testing the functions of the branches of facial nerve. These branches are concerned with ipsilateral lacrimation, the stapedial reflex, ipsilateral submandibular salivary gland secretion, and taste function from the ipsilatral anterior 2/3rd of the tongue.

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	SITE OF LESION	CHARECTERISTICS
1.	Geniculate ganglion	Diminished lacrimation
2.	Tympanic region	Decreased stapedial reflex, submandibular gland secretion and taste sensation from the anterior 2/3rd of the tongue
3.	Mastoid region	Reduction in taste sensation from the anterior 2/3rd of the tongue and ipsilateral submandibular salivary gland secretion.
4.	At the stylomastoid foramen	All the branches will reflect normal function on testing
5.	Distal to stylomastoid foramen	Extent of involvement of the facial nerve branches is determined by asking the patient to raise the eyebrows, close the eye ,flare the nostrils and smile.

GENERAL CHARACTERISTICS

UPPER MOTOR NUERON LESIONS	LOWER MOTOR NUERON LESIONS
AFFECTS MAINLY MUSCLES OF THE LOWER PART OF THE FACE.	WHOLE FACE IS AFFECTED. COMPLETE PALSY
PALSY IS RARELY ISOLATED	PARALYSIS ALWAYS ISOLATED
LOSS OF EMOTIONAL MOVEMENT	EMOTIONAL MOVEMENT PRESERVED
NO MUSCLE CONTRACTURE	MARKED MUSCLE CONTRACTURE
NO REACTION OF DEGENERATION	REACTION OF DEGENERATION
EMG & NERVE CONDUCTION NORMAL	EVIDENCE OF LOWER MOTOR NUERON LESION ON EMG

Inadvertent anaesthesia of the facial nerve

During administration of Inferior alveolar nerve block when the needle is inserted too deeply about 30-35 mm leads to transient facial palsy. To minimize the chances of anaesthetizing the facial nerve, it is important that the needle tip touches bone prior to injecting anaesthetic solution. The symptoms include transient loss of motor function to some or all of the muscles of facial expression on the ipsilateral side. The duration of paralysis is similar to the duration of local anesthesia. The patient should be from reassured that the effects will be transitory, no other specific treatment is needed but the patient should be monitored until the muscle activity begins to return. The eye should be covered to prevent the cornea from drying out.

II. FACIAL PALSY

Trauma

Trauma is the second most common cause of facial nerve paralysis. Fracture of the temporal bone causes facial nerve injury. Gunshot wounds of the temporal bone cause facial paralysis in over 50% of cases. The facial nerve can also be injured during middle ear and mastoid surgery. [3]

Herpes Zoster Oticus

Herpes zoster oticus (Ramsay-Hunt Syndrome) is the third most common cause of facial nerve paralysis. This is a manifestation of a dormant varicella zoster virus reactivating in cranial nerve ganglia during decreased immunity.Symptoms are otalgia, facial paralysis, facial numbness, and a vesicular eruption on the concha, external auditory canal, and palate. The prognosis is poorer than Bells palsy. Treatment includes steroids, Acyclovir 1 gm TID and eye care

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Otitis Media

In acute otitis media ,when there is dehiscence in the fallopian canal, bacteria directly invades along the nerve.. Facial paralysis may begin within a few days of onset of an acute otitis media and is usually incomplete. The facial palsy associated with acute otitis media generally resolves with aggressive management of the infection.

Tumors

About 5% of cases of facial nerve paralysis are caused by tumors. The most common benign tumor causing facial nerve paralysis is a facial nerve schwanomma. The most common malignant tumors causing facial paralysis are mucoepidermoid carcinoma and adenoid cystic carcinoma of the parotid gland. The management of facial nerve paralysis caused by tumors depends upon the lesions location, size, and malignant potential and may include transposition of the nerve, division and reanastomosis, interposition grafting, and cranial nerve crossover.

Melkerson-Rosenthal Syndrome

Melkerson-Rosenthal syndrome is a rare disease that consists of a triad of symptoms: recurrent oro-facial edema, recurrent facial palsy, and lingua plicata (fissured tongue). Facial paralysis and lingua plicata occur in half of the patients. The complete triad is only present in 25% of these patients. This condition usually starts in the second decade of life, and the manifestations usually occur sequentially. Facial paralysis occurs in 50 to 90% of these patients and tends to be abrupt. A history of bilateral sequential paralysis and relapse after initial recovery is common. The site of the paralysis usually corresponds to the facial swelling. Facial nerve decompression is indicated if episodes of facial paralysis are frequent and progressive.

Congenital Facial Paralysis

Congenital facial paralysis incidence is about 0.23% of live births. The most common cause is birth trauma (80%), which is usually evident by other signs of injury. These include a history of a difficult delivery with or without forceps, facial swelling, bruising over the mastoid or extratemporal course of the nerve, and hemotympanum. The mechanism of injury is thought to be from direct pressure during forceps use or from pressure on the infants face by the sacral prominence during delivery.

Mobius' syndrome consists of a broad spectrum of clinical findings which can range from an isolated unilateral facial paralysis to bilateral absence of facial and abducens nerve function. In this syndrome, the facial nerve forms but consists of only a fibrotic tract. The muscles of facial expression may form in some cases, but degeneration to fibrosis generally occurs rapidly. Many other cranial nerves may be involved (III, IV, IX, X, XII) and skeletal abnormalities may be present. Treatment for these cases of newborn facial paralysis is generally delayed until late childhood and usually requires static slings and muscle transfers.

A rare cause of isolated newborn facial paralysis is dysgenesis of the intra temporal facial nerve. The nerve is usually very thin and fibrotic in the distal part of the mastoid. Facial function is not usually present. The prognosis for trauma related facial nerve paralysis at birth is usually excellent. Surgical decompression should not be considered until the nerve has had a chance to recover or until >90% degeneration has occurred.

House classification system assesses the degree of voluntary movement present to document the grade of facial paralysis.[15]

Grade	Degree	Description
I	Normal	Normal facial movements; No synkinesis
II	Slight	Mild deformity, mild synkinesis, good forehead function, slight asymmetry
III	Moderate	Obvious facial weakness, forehead motion present, good eye closure, asymmetry, Bell's phenomenon present
IV	Moderately	Obvious weakness, increasing synkinesis; no forehead motion
V	Severe	Very obvious facial paralysis, some tone present, cannot close eye
VI	Total	Complete facial paralysis, absent tone

It is also important to determine if the paralysis is central or peripheral. Supranuclear (central) lesions produce contralateral voluntary lower facial paralysis. The frontalis muscle is spared because of the bilateral innervation. Emotional response (facial motion on laughing or crying) may also be preserved with central lesions. Presence of **Bell's**

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phenomenon (upward outward turning of the eyeball as the patient attempts to close the eyelids) indicates a peripheral lesion.

Topognostic Testing

The principle behind topognostic testing is that lesions distal to the site of a particular branch of the facial nerve will spare the function of that branch. Moving distally from the brainstem, these tests include: the schirmer test for lacrimation, the stapedial reflex test (stapedial branch), taste testing (chorda tympani nerve), salivary flow rates and pH (chorda tympani).

Electrophysiologic Tests

These tests are useful for patients with complete paralysis for determining prognosis for return of facial function and the endpoint of degeneration by serial testing. They are not useful in patients with incomplete paralysis.

III. BELL'S PALSY

Bell's palsy is the most common cause of facial paralysis and accounts for more than half of all cases. Traditionally, this was considered to be a diagnosis of exclusion after ruling out all other possible causes. However, it has recently been considered a positive diagnosis if the following are present: unilateral weakness of all facial muscles of sudden onset, possibly associated with a viral prodrome, no evidence of central nervous system pathology, no evidence of a Cerebellopontine angle(CPA) lesion, no history of otologic disease. Patients may exhibit evidence of concomitant sensory cranial polyneuritis with otalgia or postauricular pain, dysacousis or hyperacusis, dysgeusia, decreased tearing or epiphora, and facial hypesthesias/dysesthesias of V or IX .

Definition

Bell's palsy is an idiopathic, acute, sudden onset, isolated lower motor neuron facial palsy.
Bell's palsy is named after Sir Charles Bell (1774-1842), who first described the syndrome along with the anatomy and function of the facial nerve.[16]

Etiology

Herpes simplex virus- Although the exact etiology of Bell's palsy is still unclear it is believed that herpes simplex infection is the most likely agent. This belief is supported by an increased incidence of HSV antibodies in patients with Bell's palsy. Associated known clinical conditions are diabetes, severe hypertension, last trimester of pregnancy, dental anaesthesia, exposure to cold Other suspected causes are otitis media, herpes zoster infection, head injury, multiple sclerosis.

Site of lesion in Bell's palsy is the meatal foramen (junction of the internal auditory canal portion of the nerve and the labyrinthine segment of the nerve), which is considered to be the narrowest portion of the fallopian canal. As the edema within the nerve increases, axonal flow and circulation are inhibited resulting in varying degrees of nerve injury (first, second, and third degree). Patients who are most severely affected develop a high level of third degree injury which can result in the loss of endoneural tubules and misdirected axonal regeneration.

Bell's palsy is sudden in onset and maximum paralysis occurs in 24 hrs. The striking feature is the paralysis of the whole side of the face, with inability to close the eyelids, 'ironing' of the forehead skin creases, and drooping of the angle of the mouth.[16]

Patients may initially complain of retro-auricular pain, due to involvement of the somatic sensory fibres in the facial nerve. In addition to the motor facial defcit, loss of taste in the anterior two-thirds of the tongue may be detected, indicating compression of the chorda tympani. If the compression affects the nerve higher in the facial canal, hyperacusis may be a symptom due to involvement of the stapedial nerve.

Patients may complain of 'dry eye', which may be due to inability to blink regularly, although reduced lacrimation on that side may suggest involvement of the greater petrosal nerve. Inability to close the eyelids completely, particularly during sleep, may lead to corneal desiccation, and ulceration of the cornea is the most serious complication. Hence prescription of lubricating eye drops and taping the eye.[2]

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Many conditions can produce isolated facial nerve palsy identical to Bell's palsy. They can be distinguished from Bell's palsy as they have additional features.[16]

Differential Diagnosis for Facial Nerve Palsy [16]

	Disease	Cause	Distinguishing factors
Nuclear (peripheral)	Lyme disease	Spirochete Borrelia burgdorferi	History of tick exposure, rash, or arthralgias; exposure to areas where Lyme disease is endemic
	Otitis media	Bacterial pathogens	Gradual onset; ear pain, fever, and conductive hearing loss
	Ramsay Hunt syndrome	Herpes zoster virus	Pronounced prodrome of pain; vesicular eruption in ear canal or pharynx
	Sarcoidosis or GuillainBarré syndrome	Autoimmune response	More often bilateral
	Tumor	Cholesteatoma, parotid gland	Gradual onset
Supranuclear (central)	Forehead spared		
	Multiple sclerosis	Demyelination	Additional neurologic symptoms
	Stroke	Ischemia, hemorrhage	Extremities on affected side often involved
	Tumor	Metastases, primary brain	Gradual onset; mental status changes; history of cancer

MANAGEMENT

The management of facial paralysis is a multidisciplinary approach consists of a combination of pharmacologic therapy, physical therapy for facial neuromuscular retraining, and surgical intervention via dynamic and static techniques for facial reanimation.

LOCAL HEAT

Infra-red irradiation or moist heat over face or parotid region

LOCAL TREATMENT OF MUSCLES

Massage the facial muscles with bland oil twice daily for 5 minutes
The massaging movements should start from chin and upwards.
Prevention of facial sagging can be down done by application of strips of adhesive tape.
The tape is attached to the temple and extends down in a V shaped fashion to the upper and lower lips.

PROTECTION OF EYE

Eyes to be protected with dark glasses or eye patch.
Eyes to be washed with zinc-boric solution to prevent conjunctivitis

CORTICOSTERIODS

Oral corticosteroids have traditionally prescribed to reduce facial nerve inflammation in patients with Bell's palsy. Prednisone is typically prescribed in a 10-day tapering course starting at 60 mg per day.This helps in reducing secondary oedema.

ANTIVIRALS

Because of the possible role of HSV-1 in the etiology of Bell's palsy, the antiviral drugs acyclovir and Valacyclovir are prescribed. Either Acyclovir 400 mg can be given five times a day for seven days or Valacyclovir 1 g can be given three times per day for seven days.

GALVANISM

can be tried after 2 weeks of onset

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thrice a week
should not be given if face is tender

SURGERY
Surgical decompression of facial nerve within three weeks of onset has been recommended for patients who have persistent loss of function (greater than 90 percent loss on electroneurography) at two weeks. Overweighing the significant complications and the paucity of data supporting benefit, the American Academy of Neurology does not currently recommend surgical decompression for Bell's palsy.[17]
Anastomosis of facial nerve with accessory or hypoglossal can be considered.
Plastic surgery can also be tried.

SURGICAL MANAGEMENT OF ACUTE FACIAL PARALYSIS (<3 WEEKS)[18]
Management of acute facial paralysis may involve facial nerve decompression surgery in cases of virally-induced facial paralysis (Bell's palsy, Ramsay-Hunt syndrome) or primary facial nerve repair/grafting in cases of resection or transection of the facial nerve.

Facial nerve decompression
Transmastoid approach- When the injury is at the tympanic or mastoid segments of the facial nerve
Middle fossa approach- When the injury is at the labyrinthine segment of the facial nerve.
Translabyrinthine approach- This approach is done when the decompression of the entire intratemporal course of the facial nerve is indicated.
Facial nerve repair
Primary nerve repair: Primary neurroraphy provides the best return of facial nerve function but the repair should be tension free.
Cable grafting: Cable nerve grafts are done when a tension-free primary nerve repair is not possible. The best choice for donor nerve grafts are great auricular nerve, sural nerve, and the medial and ansa cervicalis.

SURGICAL TREATMENT OF INTERMEDIATE DURATION FACIAL PARALYSIS (3 WEEKS to 2 YR)
This is in conditions where the nerve is intact but has stretch injury due to previous surgery and has not recovered well.
Nerve transfers and nerve crossover procedures are typically the treatment of choice in this type of facial palsy.
Nerve transfers and cross-facial nerve grafting
Cross-facial nerve grafting-In this procedure the intact and functional contralateral facial nerve is utilized.
The most commonly used procedure is the hypoglossal-facial nerve transfer.

SURGICAL TREATMENT OF CHRONIC FACIAL PARALYSIS (>2 YR)
In chronic facial palsy the muscles are atrophied and uses altnernate muscles for reanimation.
This includes Dynamic and Static techniques. Dynamic facial reanimation is done using free and regional muscle transfers. Static techniques for facial reanimation include oculoplastic procedures, eyelid weights, static facial suspension.
Regional muscle transfer- The workhorse for the regional muscle transfer for dynamic facial reanimation is temporalis muscle. Other regional muscle transfers include masseter muscle transfer for smile reanimation and the digastric muscle transfer for marginal mandibular nerve injuries.
Free muscle transfer- In cases where there is concomitant trigeminal dysfunction and regional flaps cannot be utilized gracilis, pectoralis minor, serratus anterior, latissimus dors are used as free muscle transfer.

STATIC TECHNIQUES FOR FACIAL REANIMATION
This technique can be used during any stage of facial palsy either chronic facial palsy or temporary facial palsy where the nerve recovery is expected.
Brow ptosis correction - Brow ptosiscorrection can be done by direct brow lift, endoscopic brow lift or temporal brow lift.
Upper eyelid management
Eyelid weight placement-To manage lagopthalmos gold or platinum weights are placed in the upper eyelid. Platinum is more ideal than gold as it is more inert. Eyelid weight placement enhances the blink reflex.
Palpebral spring procedure- A spring is placed between the superior orbital rim and a pocket at the superior aspect of the tarsus.

| ISSN: 2394-2975 | www.ijarety.in| | Impact Factor: 7.394| A Bi-Monthly, Double-Blind Peer Reviewed & Referred Journal |

|| Volume 11, Issue 6, November-December 2024 ||

DOI:10.15680/IJARETY.2024.1106106

Lateral tarsorrhaphy: This is a reversible technique where the lateral parts of upper and the lower tarsal plates are sutured together in case of keratitis.
All of these procedures are reversible if facial nerve recovery occurs.
Lower eyelid management
Lateral tarsal strip procedure-This technique addresses the paralytic lower lid ectropion. A lateral canthotomy is performed and the lower tarsus is trimmed and sutured directly to the lateral orbital rim periosteum.
Medial canthopexy: Medial ectropion of the lower eyelid is treated using a medial canthopexy technique in which the medial tarsus is sutured to the periosteum of the lamina papyracea.
Nasolabial fold modification-Nasolabial fold can be effaced or enhanced by suturing.
Static facial suspension- Slings like fascia lata are placed from the zygomatic arch/temporalis fascia to the oral commissure and nasolabial fold.
External nasal valve repair- External nasal valve collapse can be treated with a fascia lata sling from the alar base to the temporalis fascia to stent open the external nasal valve.

Prognosis:
The natural course of Bell palsy varies from early complete recovery to substantial nerve injury with permanent sequelae. Prognostically, patients fall into 3 groups with roughly equal numbers in each group.
Group 1 regains complete recovery of facial motor function without sequelae.
Group 2 experiences incomplete recovery of facial motor function, but no cosmetic defects are apparent to the untrained eye.
Group 3 experiences permanent neurologic sequelae that are cosmetically and clinically apparent.
Most patients develop an incomplete facial paralysis during asymmetry of facial muscles, while 5% experience severe deformities the acute phase. This group has an excellent prognosis for full recovery. Patients demonstrating complete paralysis are at higher risk for severe sequelae.
Of patients with Bell palsy, 85% achieve complete recovery.

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International Journal of Advanced Research in Education and Technology

ISSN: 2394-2975 Impact Factor: 7.394

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