International Journal of Research in Health and Allied Sciences

Journal home page: www.ijrhas.com

Official Publication of "Society for Scientific Research and Studies" [Regd.]

ISSN: 2455-7803

Review Article

PROSTHODONTIC IMPLICATIONS OF BELL'S PALSY- A REVIEW

[1]Dr. Renu Gupta, [2]Dr. Saloni Sharma, [3]Dr. Bhavya Aggarwal, [4]Dr. Nikhil Kapila, [5]Dr. Minal Kanwar

[1]Head of Department of prosthodontics crown and bridge, H. P Government Dental College and Hospital, HP
[2,4,5]Post-graduate student, Department of prosthodontics crown and bridge, H. P Government Dental College and Hospital, HP
[3]MBBS Graduate student, India

ABSTRACT:
Prosthodontics is concerned with the diagnosis, treatment planning rehabilitation and maintenance of oral function, comfort appearance and health of patients with clinical conditions associated with missing or deficient teeth and oral and maxillofacial tissues using biocompatible substitutes. To achieve success in this technique exacting & demanding field, there must be meticulous attention to every detail – from the initial patient interview & diagnosis, through the active treatment phase, & to a planned schedule to follow up care. Neuromuscular conditions need to be evaluated before panning for prosthetic treatment.
Keywords: Bell's Palsy, Prosthodontics

Received: 10 July, 2023 Accepted: 12 August, 2023

Corresponding Author: Dr. Renu Gupta, Head of Department of prosthodontics crown and bridge, H. P Government Dental College and Hospital, HP

This article may be cited as: Gupta R, Sharma S, Aggarwal B, Kapila N, Kanwar M. Prosthodontic Implications of Bell's Palsy- A Review. Int J Res Health Allied Sci 2023; 9(3):105- 115

INTRODUCTION

Prosthodontics is concerned with the diagnosis, treatment planning rehabilitation and maintenance of oral function, comfort appearance and health of patients with clinical conditions associated with missing or deficient teeth and oral and maxillofacial tissues using biocompatible substitutes. To achieve success in this technique exacting & demanding field, there must be meticulous attention to every detail – from the initial patient interview & diagnosis, through the active treatment phase, & to a planned schedule to follow up care. Neuromuscular conditions need to be evaluated before panning for prosthetic treatment.[1]

Facial paralysis is a condition that involves loss of control of facial muscles on the affected side and is generally sudden in onset. The nerve involved is the VII cranial nerve.[2]

Based on the site of the neurons affected they are classified as upper motor neuron (UMN) and lower motor neuron (LMN) paralysis.[2]

In an upper motor neuron lesion, example- stroke, the forehead is spared since this region is bilaterally represented in the cortex. Looking for 'forehead sparing' is thus a way of differentiating between upper and lower motor neuron causes of facial weakness.[3]

Based on the location of the causal pathology, facial paralysis can be categorized as central or peripheral. Central facial paralysis results from disorders of the neural system above the facial nucleus, while peripheral facial paralysis is caused by damages to the facial nucleus or facial nerve. Bell's palsy is the most common peripheral facial paralysis.[4]

Idiopathic facial nerve paralysis, commonly known as Bell's palsy (BP), is a cranial nerve VII condition leading to facial weakness (paresis) or paralysis. This acute unilateral paralysis or paresis has no identifiable cause and occurs over <72 h. The name comes from Sir Charles Bell, a surgeon, who took great interest in sensory (trigeminal nerve) and motor (facial nerve) innervations of the face.[5]

Its cause is mostly idiopathic, but exposure to cold and several other contributory factors have been named such as rheumatic hypothesis, ischemic hypothesis, immunological hypothesis, viral hypothesis. The combination of

facial paralysis and herpes infection is called Ramsay Hunt syndrome. The incidence being 15-40 cases per 100,000 population with female predilection affecting middle age group having equal chances of involvement of right and left nerves.[6]

Signs and symptoms of Bell's Palsy include ipsilateral drooping of the eyelid, dry eye, excessive tearing, drooping of the corner of the mouth, post auricular pain, loss of taste sensation in the anterior 2 thirds of the tongue, difficulty eating, dry mouth, slavering, altered sensation and hyperacusis. The most pathognomonic sign of Bell's Palsy is the Bells's Phenomenon, characterized by upward rolling of the eye when attempting to close the eyelid.[5] Eyeball rolls upward so that the pupil is covered and only the white sclera is visible.[6]

Patients with Bell's palsy experience sudden weakness or paralysis on affected side of the face with abrupt loss of muscular control. They also face difficulty in wrinkling the forehead, closing the eye, whistling, raising the eyebrow on the affected side. The corner of the mouth droops causing drooling of saliva. There is obliteration of the nasolabial fold. Since the buccinator muscle weakens, food is retained in maxillary and mandibular buccal and labial vestibules. Involvement of chorda tympani nerve leads to loss of taste in the anterior two-third of the tongue and reduced salivation. The expression of the face changes drastically resulting in a mask-like appearance to the facial features.[6]

The main features of the Bell's palsy are:

A. Facial droop and difficulty making facial expressions, such as closing eye or smiling;

B. Increased sensitivity of sound on the paralyzed side;

C. Pain through the jaw on the affected side;

D. A decreased ability to eat, chew, talk.[7]

Diagnosis of facial paralysis is primarily based on clinical presentation, including weak eyebrow lifting, incomplete eye closure, drooping mouth corner, dry eye, loss of taste sensitivity, hyperacusis and ear pain.[4]

Tests such as the blink test, facial expressions, stapedial reflex checking, Schirmer test, and taste testing in the anterior two-thirds of the tongue support the diagnosis.[2]

The etiology and degree of facial paralysis are quite variable and so are its treatment and treatment outcomes at this time.[4]

Although the etiology of BP is unclear, most cases resolve without treatment. Short term consequences of BP include the inability to close the eye, potential cornea injuries and eventual eye dryness, which can be managed clinically with a favourable prognosis. Long term, incomplete recovery from BP can result in facial asymmetry, disfigurement, reduced facial movement and many other complications that greatly reduced the patient's quality of life. Although various modalities are available to improve quality of life, patients with BP often experience depression, reduced quality of interpersonal relations, anxiety, decreased self- esteem and social isolation.[5]

The medical management of Bell's palsy includes massive doses of steroid administered as a bolus dose, tapered off over the next few weeks followed by physiotherapy (galvanism, massage and facial exercises). Surgical Intervention may necessitate nerve decompression (internal and external), nerve grafting, nerve anastomosis-reanimation (cross facial nerve grafts, nerve transfers and free muscle transplantation).[6]

In prosthodontics it appears indispensable to transfer the view from the mouth to the casts. Sometimes mistakes occur and collaboration between dental technician and a dentist interrupts, wherefore appropriate treatment cannot be accomplished. Accordingly, one of the most important factors how to maintain this professional communication is taking into account the correct determination of custom tray borders, border molding and impressions.[7]

The prosthodontic management of these patients requires a systematic approach as the clinical features of Bell's palsy may interfere with most of the steps such as impression making, jaw relation, denture retention, and stability.[6]

This review article aims to discuss the current evidence for the etiology, diagnosis and treatment of Bell's palsy and its implications in the field of Prosthodontics.

EPIDEMIOLOGY

Bell's Palsy is the most common cranial mononeuropathy with a reported incidence rate of 11.5–40.2 per 100000.2,13 It is seen equally in males and females and occurs in all ages with a propensity to affect people in mid to later life.14 No race predilection exists, and both sides of the face are equally affected, although bilateral Bell's Palsy is rare (0.3% of cases).[5]

Various epidemiological studies have established a seasonal trend, with higher incidence in colder months of the year. Even without treatment, 70% of affected people will have complete resolution, while approximately 30% will experience partial or incomplete recovery. The recurrence rate of Bell;s Palsy is estimated to be 7% and does not correlate with the prognosis. Recurrence should alert the clinician to alternative causes of facial paralysis.14 Possible risk factors for Bell;s Palsy include hypertension, severe preeclampsia, psychological concerns, pregnancy, radiation exposure, diabetes, obesity, upper respiratory infection and migraine. Numerous cases of Bell's Palsy have been documented following the administration of influenza vaccines as well as many others. Similarly, recent studies have identified Bell;s Palsy as a side effect of the new mRNA SARS-CoV-2 vaccine.[5]

CRANIAL NERVE VII ANATOMY

Cranial nerve VII is a multifunctional nerve with significant motor, sensory and parasympathetic activities. Its nerve fibres are associated with 3 nuclei in the medulla oblongata and the pons: solitary (sensory), facial (motor) and superior salivatory (parasympathetic). The central motor nucleus of the facial nerve is located in the precentral gyrus of the motor cortex. Fibres from the central motor nucleus travel down in the corticobulbar tracts and supply the contralateral facial nucleus.[5]

The facial nerve is divided into 6 segments: cisternal, meatal, labyrinthine, tympanic, mastoid and extracranial. The cisternal segment includes the motor root and the nerve of Wrisberg, which advance to the internal acoustic meatus. At the meatus, these nerve segments unite to form the meatal portion of the facial nerve. Throughout the cisternal and meatal paths, CN VII travels with the vestibulocochlear cranial nerve (VIII).[5]

Further, the labyrinthine portion extends from the cochlea to the vestibule of the inner ear and joins the geniculate ganglion. The tympanic portion runs inferior to the lateral semicircular canal and gives rise to the mastoid segment of the facial nerve. The mastoid segment exits the skull through the stylomastoid foramen and becomes the extracranial portion of the nerve. This portion gives rise to the digastric and posterior auricular nerves and advances through the parotid gland to give rise to the terminal branches.[5]

Each segment gives rise to various branches (Table 1). Understanding the function of these branches is extremely helpful in determining the location of the pathology along the facial nerve. Figure 2 depicts structures innervated by the facial nerve, as well as the superficial extracranial branches.[5]

Supranuclear pathology, more commonly known as upper motor neuron (UMN) palsy, results when neuronal fibres above the facial nucleus are disrupted. UMN palsy affects the contralateral half of the lower face, sparing the forehead and the eyebrow muscles. A UMN lesion of the cortex and/or corticobulbar tract spares the lacrimal and salivary secretions, and the taste function is preserved.[5]

Infranuclear pathology or lower motor neuron (LMN) palsy results from lesions at the facial nucleus or the exiting fascicles. These lesions affect the ipsilateral upper and lower face. Therefore, involvement of the forehead muscle (i.e., frontalis) and the eyebrow muscle (i.e., corrugator supercilii) provides insight into the nature of the condition. The sparing of the forehead and eyebrow muscles is attributed to the bilateral innervation of the upper third of the face and contralateral innervation of the lower 2 thirds.26 Precise location of an LMN palsy can be discerned via functional assessment of other neighbouring structures. LMN palsy involving the facial nucleus or the fascicles within the brainstem may exhibit contralateral hemiparesis, ataxia, nystagmus and CN III–CN VI palsy, as well as ophthalmoparesis.[5]

Conversely, LMN palsy involving the facial nerve after it exits from the brainstem may display complete ipsilateral hemifacial weakness, dysgeusia and a decrease in lacrimation and salivation.[5]

Facial nerve segments	Branches	Function
Cisternal		
Meatal		
Labyrinthine (including geniculate ganglion)	Greater superficial petrosal	• Parasympathetic supply to nose, palate and lacrimal gland • Forms nerve of pterygoid canal with deep petrosal nerve
	Lesser petrosal	• Secretory fibres to the parotid gland
	External petrosal	• Sympathetic fibres to middle meningeal artery
Tympanic		
Mastoid	Stapedius	• Motor supply to stapedius • Allows tolerance of loud noises
	Chorda tympani	• Sensory taste fibres to anterior 2 thirds of tongue • Parasympathetic innervation of the submandibular and sublingual glands
	Nerve from auricular branch of vagus	• Sensory fibres traveling with facial nerve; headed back into the skull
Extracranial	Posterior auricular	• Motor supply to posterior and superior auricular muscles and the occipital belly of occipito-frontalis muscle
	Digastric	• Motor supply to posterior belly of digastric muscle
	Stylohyoid	• Motor supply to stylohyoid muscle
	Temporal	• Supply anterior and superior auricular muscles, muscles of the forehead (frontalis) and superior portion orbicularis oculi
	Zygomatic	• Supply orbicularis oculi, muscles of nasal aperture (e.g., nasalis) and elevator muscles of the lip (e.g., zygomaticus)
	Buccal	• Supply musculature surrounding upper and lower lip and buccinator
	Marginal mandibular	• Supply musculature of the lower lip, depressors of the lip and mentalis
	Cervical	• Supply platysma

Table 1 Branches arising from segments of the facial nerve and their functions.

Grade	Symptoms
I	• Normal facial function
II	• Slight weakness at rest (only noticeable on close inspection) • No synkinesis, contracture or spasms • Normal symmetry and tone motion at rest
III	• Obvious but not disfiguring difference between sides • Noticeable synkinesis, contracture and spasms • Complete eye closure with effort • Normal symmetry and tone motion at rest
IV	• Obvious weakness and disfiguration of affected side • Normal symmetry and tone motion at rest • Incomplete eye closure
V	• Barely perceptible motions observed • Complete asymmetry
VI	• Complete paralysis, no movement

Table 2- House-Brackmann facial nerve grading system.

Differential diagnosis	Conditions
Parenchymal lesions	• Multiple sclerosis, stroke, abscess, encephalitis, neoplasms
Trauma	• Iatrogenic, basilar skull fracture, temporal bone fracture
Neoplasms	• Schwannoma, neuroma, meningioma, metastatic, cholesteatoma, parotid perineural invasion, skin cancers
Congenital	• Mobius syndrome, forceps trauma
Meningitis	• Bacterial, viral (HIV, Zika, Epstein-Barr virus, polio), fungal, mycobacteria (tuberculosis, leprosy), spirochete (Lyme disease, syphilis), sarcoidosis, neoplastic
Infection	• Ramsay Hunt syndrome, Guillain-Barre syndrome, chronic inflammatory demyelinating polyradiculoneuropathy, osteomyelitis of skull base, otitis media, parotitis, mastoiditis, amyloidosis, granulomatosis with polyangiitis, polyarteritis nodosa, Sjogren syndrome, mononucleosis
Neuromuscular junction disorders/myopathy	• Oculopharyngeal dystrophy, myotonic dystrophy, myasthenia gravis, facioscapulohumeral muscular dystrophy
Endocrine	• Diabetes mellitus
Neurovascular	• Stroke
Other	• Melkersson-Rosenthal syndrome, Paget's disease, ethylene glycol ingestion, idiopathic intracranial hypertension, hereditary neuropathy, Wernicke encephalopathy, acute lymphatic leukemia, neck carotid artery dissection

Table 3- Differential diagnosis for facial nerve palsy.

Condition	Defining characteristics
Cerebrovascular event (e.g., stroke)	• Forehead muscles are spared, headache, ipsilateral limb weakness
Guillain-Barre syndrome	• Ascending weakness, reflexes absent
Diabetes mellitus	• Increased blood glucose
Infection (e.g., meningitis and encephalitis)	• Meningeal signs, fever, pain, abnormal cerebrospinal fluid (CSF)
Lyme disease	• Rash, malaise, arthralgia, bilateral facial palsy
Ramsay Hunt syndrome	• Pain, vesicular eruption and/or scabs in or around the ear canal
Sarcoidosis	• Bilateral facial palsy, elevated angiotensin-converting enzyme
Neoplasms (e.g., parotid tumour)	• Insidious onset, palpable mass, partial involvement of facial nerve branches

Table 4- Defining characteristics of conditions associated with facial paralysis and/or paresis.

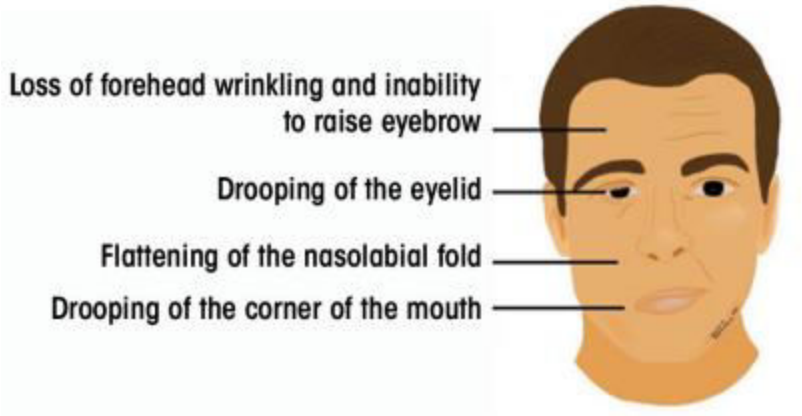


Figure 1- Signs of acute unilateral Bell's palsy

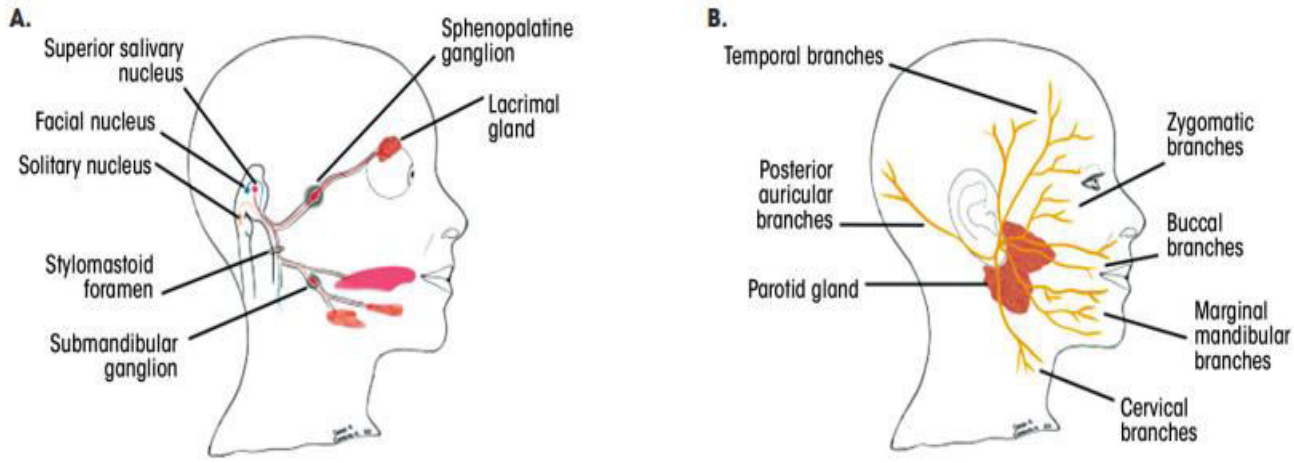


Figure 2- A. Structures innervated by the facial nerve. The red (parasympathetic), blue (motor) and orange (sensory) lines demonstrate the multifunctionality of the facial nerve. B. Superficial branches of the extracranial portion of the facial nerve.

CLINICAL SIGNIFICANCE OF FACIAL NERVE

Facial nerve paralysis may occur in neurological lesions influencing the nervous system at various stages of the facial nerve distribution. It is of two types: central facial paralysis and peripheral facial paralysis.

a. Central facial paralysis

Upper motor neuron lesion

The motor fibres of facial nerve traverses at different areas in the central nervous system, hence damage to either the upper brainstem or cerebral cortex brings about paresis of the lower portion of the face inverse to the side of the neurological lesion in nervous system. But these lesions will spare the forehead muscles, because the upper part of face has innervation from both the side of cerebral hemisphere.[8]

Lower motor neuron lesion

In pons, the lower motor lesion affecting the facial nerve motor nucleus results in homolateral paralysis of whole side of the face. These lesion occurs due to various pathologies like inflammation, stroke and neoplasia.[8]

b. Peripheral facial paralysis

Lower motor neuron lesion after the nerve exits from the brainstem (Bell's Palsy)

If the neurological lesion occurs at termination of facial canal, where the facial nerve leaves the skull it results in paralysis of muscles of facial expression. The symptoms on the affected side includes loss of facial skin folds and lines, drooping of corner of the mouth, absence of wrinkles in the forehead, widened palpebral fissure and inability to close the eyelids withloss of tears. But the taste sensation is spared because it is supplied by chorda tympani which branches from facial nerve before stylomastoid foramen.The causes of lower motor neuron lesions beyond the brainstem includes viral infections, inflammatory conditions, vascular conditions and neoplasms.[8]

ETIOLOGY

The precise etiology of Bell's palsy is unknown, and its diagnosis is based on exclusion. Consequently, it is essential to eliminate all other potential etiologies of facial paralysis and paresis before diagnosing BP. Currently, there are numerous theories regarding the cause of Bell's Palsy.

ANATOMY

The anatomy of the facial nerve and its long intra-bony path has been considered as potential factors in the etiology of Bell's palsy. The fallopian canal is a narrow Z-shaped canal in the temporal bone. Although it provides protection to the facial nerve, it predisposes the nerve to entrapment neuropathies following inflammation or edema. Ozan and Arslan found increased cross-sectional area of the facial nerve and decreased cross-sectional area of the internal acoustic meatus associated with Bell's palsy. Therefore, compression of the facial nerve caused by inflammation or edema along its narrow intrabony path may cause facial nerve abnormalities.[5]

VIRA INFECTION

Viral infection is hypothesized to be another etiological factor in Bell's palsy. Varicella zoster virus (VZV) and herpes simplex virus (HSV) are DNA viruses that remain dormant in nervous ganglia subsequent to primary infection. They can reactivate anytime throughout the host's lifespan and cause recurrent infections. Reinfection by VZV is also possible and more severe than HSV-1 because of its spread via satellite cells. A possible

biomolecular mechanism of HSV-mediated neuronal dysfunction involves abnormal expression of p53 upregulated modulator of apoptosis (PUMA) and the innate immune signaling molecule, SARM1.[5]

ISCHEMIA

The outer layer of the facial nerve consists of a periosteal membrane overlying a vascular plexus coating the epineurium of the facial nerve. Primary ischemia can result from vasospasms, leading to facial nerve neuropathy. Although this type of ischemia is rare, it can be appreciated in cases of diabetes mellitus and embolization of the middle meningeal artery. Quickly following acute ischemia, inflammation of the nerve begins with recruitment and activation of macrophages. Primary ischemia is also observed in animal models, with facial nerve paralysis occurring 5–15 minutes after blockage of the vascular network.[5]

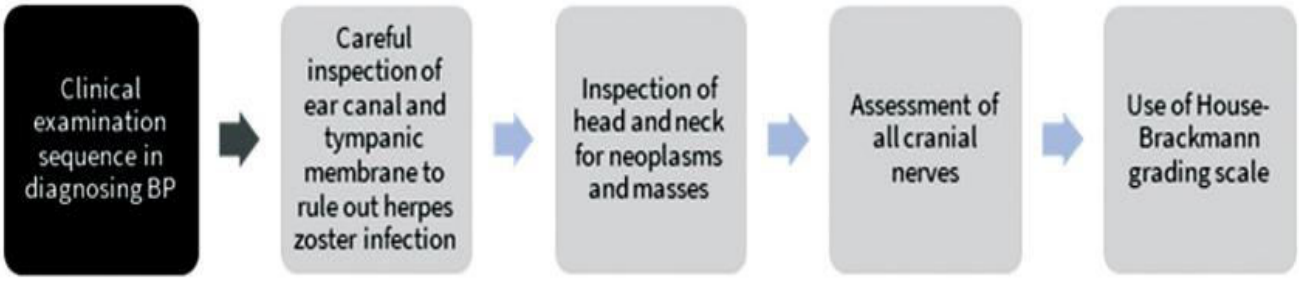
Secondary ischemia is a consequence of primary ischemia, with initial constriction of the arterioles and subsequent dilation of the capillaries leading to production of a transudate. This transudate can compress the lymphatic capillaries, further increasing transudate production and inducing ischemia. Secondary ischemia can progress to tertiary ischemia, where perivasculitis and endarteritis are evident. Thickening or fibrosis of the facial nerve sheath are often observed at this stage and may require surgical decompression to prevent permanent facial paralysis.[5]

INFLAMMATION

Recently, more evidence has emerged regarding BP and inflammation-induced demyelination of the facial nerve. Numerous studies have shown the association of a potential new marker— neutrophil to lymphocyte ratio (NLR)—with various inflammatory diseases, such as systematic lupus erythematous and hepatitis B. Similarly, a meta-analysis concluded that the NLR for patients with BP was higher than in patients without BP, and a higher NLR coincided with a worse prognosis. This indicates a change in the peripheral subpopulation of white blood cells as in other inflammatory demyelinating diseases, such as multiple sclerosis and Guillian-Barre syndrome.[5]

- Acute onset (<72 h)
- No other diagnosed medical conditions causing facial palsy
- No bilateral facial palsy (rare)
- Self-limited episode
- No recurrence
- No prolonged palsy
- (4 + months)

Figure 3- Criteria for diagnosing Bell's Palsy



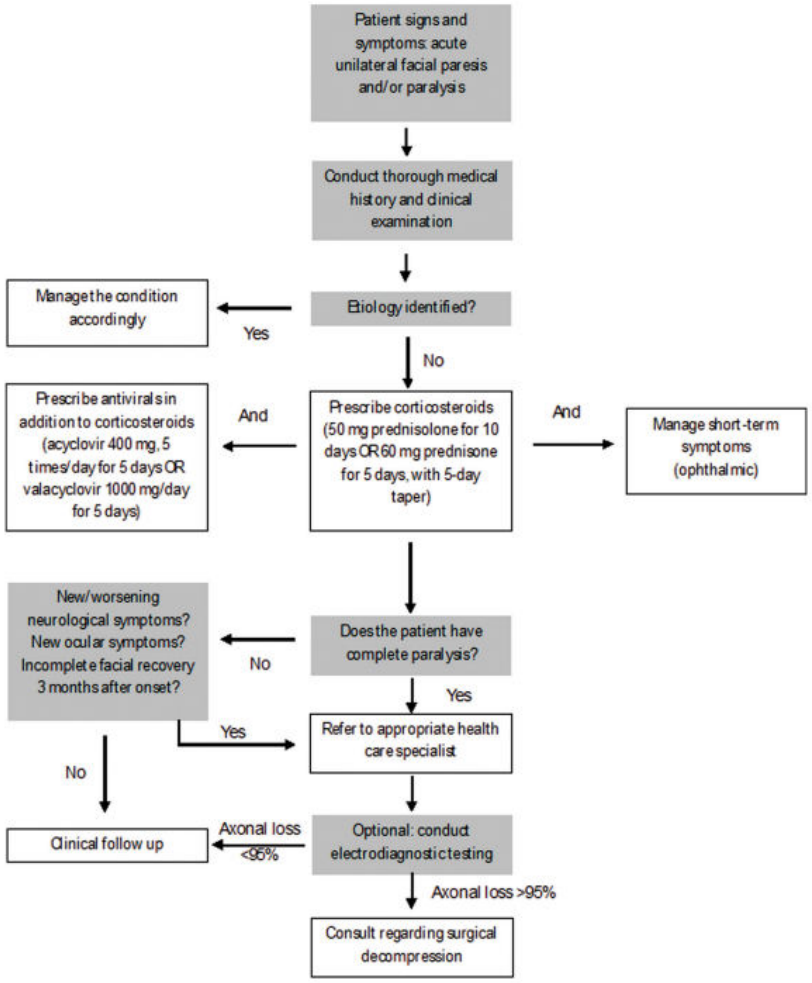


Figure 4- Algorithm for management of Bell's palsy.

PROSTHODONTIC SIGNIFICANCE

Objectives of prosthetic rehabilitation

1. To support weakened facial musculature.
2. So as to decrease the amount of surgical procedures in case patient refused to have another surgery.
3. To provide mainly comfort and esthetics to the patient with improved confidence and due to positive esthetic changes improve social interactions.[9]

In bells palsy patients, there are many symptoms that jeopardize prosthodontic treatment . Cheek biting, uncontrolled flow of saliva, having mask like expressionless appearance, unpredictable and erratic mandibular movement are few symptoms. During impression taking, jaw relation and other procedures, they may interfere.[9]

GENERAL PROSTHODONTIC CONSIDERATIONS

1. In patients with progressive neurological disorders, it is best to restore the oral cavity as early as possible, because the patient's cooperation decreases as functional and cognitive competence decline with time. Patient's physician should always be consulted as and when required.[10]
2. As most of these disorders are commonly associated with depression, the anxiety in such patients may be alleviated by relaxation and diversion methods, allowing caregiver to sit next to the patient as well as by scheduling a short appointment time (30–45 min), preferably early morning. While communicating, direct eye contact with a gentle smile and short simple sentences should be used. A continuous reassurance should be given along the course of treatment.[10]
3. For patients having poor neuromuscular control and irregular jaw movements, use of the most consistent measurement after repeated swallow and relax maneuver for measuring vertical dimension at rest (in upright position), and Dawson's bimanual manipulation technique for recording centric relation (in supine position), has been advocated. The various occlusal schemes suggested in these patients to aid in prosthetic stability have been mentioned in Table 5.[10]

Table 1: Types of occlusal schemes suggested in neuromuscular deficit patients	
Authors	**Occlusal scheme suggested**
Rajapur *et al.*[30]	Lingualized balanced occlusion
Morita *et al.*[6]	Bilaterally balanced occlusion (with balancing ramps), using zero-degree teeth
Mootha *et al.*[8]	Balanced occlusion using upper cuspless and lower blade teeth (improved mastication)
Robbins[3]	Zero-degree teeth, with protrusive and laterotrusive freedom on excursion

Table 5- Types of occlusal schemes in neuromuscular deficit patients

PROSTHODONTIC MANAGEMENT

Removable Prosthesis

Problems: Symptoms of facial palsy that jeopardize prosthodontic treatment include cheek biting, drooping of saliva, mask like expressionless appearance, erratic mandibular movement, difficulty in pronounciation of labiodental (F,V) and labial plosive (P,B) sounds and difficulty in obtaining retention and stability of denture due to facial asymmetry.[8]

The major reason for poor mandibular movements is uncoordinated neuromuscular control. In edentulous patients due to restricted sensory impulse and proprioception, they are incapable to achieve sufficient functional movements of mandible. So a systemic approach is needed to enhance mandibular movements and to modify the conventional method of complete denture fabrication.[8]

The modifications that can be done during prosthetic rehabilitation with a complete denture in patients with facial nerve palsy have been summarized in Table 6.[8]

STAGES	MODIFICATIONS	FUNCTION
Impression technique	Neutral zone impression technique	To counteract erratic mandibular movements which affects the retention of the denture
Border moulding	Functional movements of patient (swallowing, pursing lip, speaking etc.)	To record anatomic limits of the patient accurately
Teeth arrangement	Lingualised occlusion	Better retention and stability
Prior to definite denture	Interim denture with flat occlusal table	To check the tapping movements of mandible on maxillary teeth and for neuromuscular training. After patient is devoid of any discomfort the interim denture is replaced by definite denture

Table 6- Modification of complete denture during fabrication.

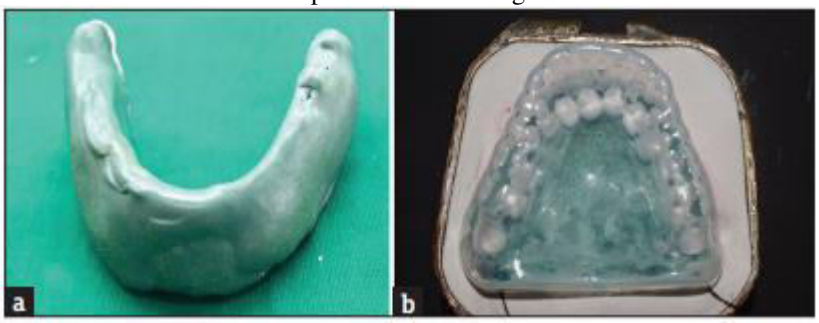


Figure 5- Prosthetic modifications for hemifacial palsy. (a) Neutral zone, (b) Hollow denture fabrication using thermoform beads.

Modifications of complete dentures after fabrication:

1. **Denture with detachable buccal or cheek plumper**

   Buccal or cheek plumper are cheek lifting devices placed on the contralateral side of denture to support the cheek and to decrease the depth of nasolabial fold. The male and female parts of attachment (press tich button, magnets, wires and buccal tube) are implanted in the outer surface of the buccal flange and inner surface of the plumber using self-cure acrylic resin then finishing and polishing are carried out. This detachable prosthesis provides good function and psychological support to the patient.[8]

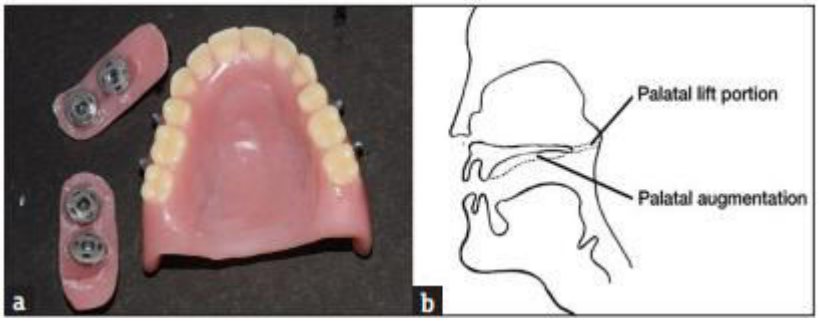


Figure 6- Prosthetic modifications for hemifacial palsy.(a) Cheek plumper prosthesis, (b) Combination palatal prosthesis.

2. Denture with extended buccal flanges

In buccal flange of the dentures, spring loaded acrylic is used to provide support for cheek. It elevates the sagging cheek and decreases the vestibular food retention. But it increases the denture weight, hence hollow dentures are fabricated to diminish the heaviness of the prosthesis.[8]

3. Liquid supported complete denture

Liquid supported complete denture in maxillary edentulous arch and neutral zone technique in mandibular edentulous arch is very beneficial. The principle is that liquid denture adapts continuously with mucosa, acts as a reliner and produces optimal stress distribution. After the denture fabrication, insertion is done to the patient and they are asked to use it for 2 weeks. Later holes are made at maxillary tuberosity region and viscous liquid like glycerin is filled through the holes to act as liquid cushion.[8]

Supporting devices

1. Face lift devices

During the recovery stage of bell's palsy patients, face lifting devices are provided to restore phonetics, aesthetics and functions of facial muscles. It is a device that has the buccal plate extending from 1st premolar to 2nd molar on affected side and is retained by circumferential /universal clasp on maxillary molars.[8]

2. Larsen modifications of supporting prosthesis

Larsen et al modified removable prosthesis by placing a buccal attachment to support impression compound with acrylic resin which gives pressure in different directions. After desirable results in esthetics and speech is obtained, impression compound is replaced by cold cure resin.[8]

The various modifications are

1. Intraoral-extraoral approach using stainless steel wire loops (0.020) attached to the prosthesis.
2. Intraoral approach with no vestibular tension.
3. Intraoral approach with Distosuperior tension towards the posterior border of the zygomatic process of the maxilla.It is the most beneficial approach.
4. Intraoral approach with Mediosuperiortension towards the patient's midline and nasal ala.[8]

Maxillofacial prosthesis

To overcome the symptoms of bell's palsy like sagging of lower eyelids and inability to close the eyelids, lower eyelid supporting appliances are fabricated using stainless steel wire and are attached to nose pad of spectacles to overcome paralytic ectropion and provides facial symmetry.[8]

CONCLUSION

It is evident that Bell's palsy can significantly affect a patient's quality of life, as the human face is a vital structure in displaying emotions, communicating and carrying out essential functions. It is important for health care professionals to recognize this debilitating condition, diagnose it early and differentiate it from other potentially life threatening underlying causes.[5]

One side facial paralysis has always been a challenge not only for neurologists, otorhinolaryngologist and general doctors; it creates the same difficulties for prosthetic dentistry specialists. In some cases, the dental clinician may be the first to recognize symptoms of the disorders mostly affecting the geriatric patients, if early stage of its onset affects the oral environment or patient complaints of functional disturbances in old prosthesis. Therefore, apart from the treatment aspects, a trained prosthodontist must have a thorough understanding of the systemic conditions which may affect the manipulation of the prosthesis.

The ultimate treatment for any unrecovered facial paralysis will be a surgical intervention of the damaged nerve. When most of the cases are abandoned from surgery due to complications and other reasons, the oral prosthesis plays an important role in patient's well-being.[9]

The goal of the prosthetic treatment should be to support the weakened muscles and provide comfort and esthetics to the patient over a long period of time.

REFERNCES

1. Phukela SS, Malhotra T, Yadav B, Rewari A, Shekhawat K, Juneja M. Neuromuscular Disorder in Relation to Prosthodontics; A Comprehensive Prospective. Annals of R.S.C.B.2021;25(5):2361 – 2371.
2. Sagar P, Lahori M. Prosthodontic Rehabilitation of a Patient with Unilateral Facial Paralysis: A Rare Clinical Report. J Oral Health Comm Dent 2021;15(3):144–145.
3. Puri N, Thakkar S, Puri ST, Jin M, Vinayak V. Bells Palsy – A Review Of Idiopathic Facial Paralysis. J of Dent Sci and oral Rehab. 2011;44-45.
4. Zhao Y, Feng G, Gao Z. Advances in diagnosis and non-surgical treatment of Bell's palsy. Journal of otology.10(2015)7-12.
5. Danesh A, Quanounou A. Bell's Palsy: Etiology, Management and Dental Implications. J Can Dent Assoc.2022,88;m8.
6. Rajapur A, Mitra N, Prakash VJ, Rah SA, Thumar S. Prosthodontic rehabilitation of patients with Bell's palsy – Our experience. J Int Oral Health 2015;7(Suppl 2):77-81.
7. Alvydas Gleiznys, Zivile Zidonyte. Prosthodontic Treatment for Edentulous Patient with Bell's Palsy: A Case Report. Mod Res Dent. 5(2). MRD.000606. 2020.
8. Rathee M, Jain P, Santhanam D, Singh S. Prosthodontic implications of Nerves of orofacial region-A review. Asia Pacific DentaL Journal.2021;8(3):20-25.
9. Gupta R, Luthra RP, Abhishek, Aggarwal B. Bell's Palsy and Its Prosthodontic Significance. J of Applied Dent and Med Sci.2018;4(4):58-62.
10. Kaushik A, Bhatnagar A, Kaur T. Removable prosthodontic considerations for patients having neurologic and neuromuscular disorders. J Int Clin Dent Res Organ 2022;14:24-30.