https://doi.org/10.48047/AFJBS.6.2.2024.2664-2672

African Journal of Biological Sciences

Journal homepage: http://www.afjbs.com

Research Paper Open Access

Electrodiagnosis and Ultrasonographic Assessment of Bell's Palsy

Ghada S. Nageeb [1], Abeer M. Elshafey [2], Mahmoud M. Gabal [3], Alaa Othman Mohamed [4]

1 Ghada Sanad Nageeb, Professor of Rheumatology and Rehabilitation, Faculty of Medicine, Zagazig University, Egypt.

2 Abeer Mohamed Elshafey, Professor of Rheumatology and Rehabilitation, Faculty of Medicine, Zagazig University, Egypt.

3 Mahmoud Mohamed Ibrahim Gabal, Lecturer of Radiodiagnoses, Faculty of Medicine, Zagazig University, Egypt.

4 * Corresponding author: Alaa Othman Mohamed Fathy, Department of Rheumatology and Rehabilitation, Kenayat Hospital, Kenayat city, Egypt.

Corresponding author: Alaa Othman Mohamed

Email: dr.alaaothmanrhm@gmail.com

Article History

Volume 6, Issue 2, Apr-Aug 2024

Received: 10 August 2024

Accepted: 15 August 2024

Published: 15 August 2024

doi: 10.48047/AFJBS.6.2.2024.2664-2672

Abstract: Background: The facial nerve, the seventh cranial nerve, is responsible for innervating the head and neck region. Bell's palsy is the most common cranial neuropathy, causing facial nerve paralysis, which is typically unilateral. It accounts for 60-75% of cases of facial palsy. Bell's palsy is acute idiopathic unilateral lower motor neuron facial nerve weakness or paralysis, with no other neurologic or systemic manifestations. Most cases recover within months, with 85-90% of patients recovering completely within one month. However, 15% progress to complete degeneration and may develop complications like residual paresis, contracture, and synkinesis. Electrodiagnostic tests used to evaluate facial nerve function include nerve conduction studies, electromyography (EMG), and blink reflex. Neuromuscular ultrasound (NMUS) is becoming a standard element in evaluating peripheral nerve and muscle disease. Recent studies suggest an additional role for neuromuscular ultrasonography as a measure of disease severity and distribution. Ultrasound has been used to predict facial nerve outcomes in Bell's palsy. Facial nerve diameter is measured proximally, distally, and midway, and the average diameter is calculated using these measurements.

Keywords: *Ultrasonographic Assessment, Bell's Palsy, Electrodiagnosis*

Introduction

The facial nerve is the seventh cranial nerve (CN VII). It arises from the brain stem and extends posteriorly to the abducent nerve and anteriorly to the vestibulocochlear nerve. It courses through the facial canal in the temporal bone and exits through the stylomastoid foramen after which it divides into terminal branches at the posterior edge of the parotid gland [1].

The facial nerve is consisting of the motor, sensory, and parasympathetic (secretomotor) nerve fibers which provide innervation to many areas of the head and neck region. The facial nerve is comprised of three nuclei [2].

- The main motor nucleus,
- The parasympathetic nuclei,
- The sensory nucleus.

The facial nerve emerges from the junction of the pons and medulla as two divisions: the primary root and the intermediate nerve. The larger primary root (facial nerve proper) innervates the muscles of facial expression, and the smaller intermediate nerve (L. nervus intermedius) carries taste, parasympathetic, and somatic sensor fibers [3].

The most important factor in the differential diagnosis of facial palsy is whether it is an LMN or UMN lesion, Due to bilateral cortical innervation of the upper facial muscles, only LMN lesions result in complete facial paralysis. Therefore, the most clinically useful assessment of UMN and LMN facial paralysis is to raise the eyebrows to assess the frontalis and to contract the eye to assess orbicularis oculi muscles. Lower motor neuron pathologies include Bell's palsy, Ramsay-Hunt syndrome, etc. Upper motor neuron lesions of facial nerve include stroke, multiple sclerosis, subdural hemorrhage, and intracranial tumors [4].

Facial nerve paralysis is the most common cranial neuropathy. Several studies have presented conflicting results regarding its epidemiology. Most of the studies reported equal incidence in females and males. Some studies stated higher incidence in young adults, but others found an increase in incidence with aging [5].

Epidemiological studies have been done in different governorates in Egypt where BP had an incidence rate between (98.9 – 107) /100000 population, higher among male patients than among female patients and peak age between 18 and 60 years, with low incidence at extremes of age [6].

According to Aditya, [7] pregnant women are 2 - 4 times more prone to the development of facial palsy compared to non-pregnant women. Ragupathy and Emovon, [8] reported an incidence of Facial palsy in 45.1 per 100 000 pregnant women, with the highest frequency occurring during the third trimester of pregnancy.

The higher incidence of Bell’s palsy in pregnancy may stem from hypercoagulability, which can cause microcirculatory insult [9].

Most of the cases of facial nerve palsy are typically unilateral, although bilateral cases can occur. The most common cause is the Bell's palsy which represents 60-75 % of the cases. Traumatic facial palsy is the second most frequent cause, and Herpes zoster oticus (Ramsay-Hunt Syndrome) is the third most common cause [10].

Bell’s palsy (BP) is acute idiopathic unilateral lower motor neuron facial nerve weakness or paralysis with no other neurologic or systemic manifestation and most cases shows recovery of function within months [11].

Susceptibility of recurrence of Bell’s palsy record (12%) of cases had a recurrent facial paresis or palsy with 1-6 episodes of paralysis with more likely to occur in the 1st two years from the onset [12].

The prognosis for Bell's palsy is generally good with 85 to 90% of patients recovering completely within one month. The remaining 15% of the cases progress to complete degeneration and will not usually show signs of recovery for three to six months and may develop complications such as residual paresis, contracture and synkinesis [13].

Theories of Bell’s palsy

Many theories have been proposed to explain the origin of Bell’s palsy like ischemia, immunological reactions, and viral infections. Ischemia (due to disturbed circulation in the vasa nervorum) leads to the nerve injury in Bell’s palsy. Vascular spasm causes a swelling of the nerve in the Facial canal. This theory is the back ground for the surgical decompression in the treatment of the disease. An immunological hypothesis was introduced by Mc-Govern and his co-workers, based on their experimental work on animals [14].

Greco et al. [15] demonstrated abnormal lymphocyte transformation in patients with Bell’s palsy and suggested that it may results from cell-mediated immunity against peripheral nerve antigens. This hypothesis encouraged the research on the utilization of steroid and other immunotherapies in the treatment of facial palsy.

Eviston et al., [16] suggested herpes simplex virus (HSV) as a cause of Bell's palsy. The theory was supported by the results in the serological study of HSV antibodies by Adour and coworkers in 1975. The viral theory has gained most of the interest, especially the neurotropic herpes viruses, which are known to become latent after primary infection, and are considered important candidates in disease development.

Love and Colleges tested the endo-neurial fluid from the facial nerve and biopsies from the posterior auricular muscle for HSV-1 DNA and varicella zoster virus (VZV) DNA using polymerase chain reaction (PCR) in 14 Bell's palsy patients undergoing decompression surgery. HSV-1 genomes were detected in 11 out of 14 patients, and VZV in none. They concluded that HSV-1 is the major etiologic agent in Bell's palsy [17].

These results were further supported by Rhaif et al. [18] who found significantly increased levels of HSV DNA in the saliva from 47 patients with Bell's palsy. Despite these findings, studies have failed to support this theory, and the role of HSV in the disease is still unclear [19].

Clinical picture of Bell's palsy

Because Bell's palsy is a diagnosis of exclusion, it must be based on thorough history and physical examination, as well as the use of diagnostic testing when necessary. Clinical features of the disorder that may help to distinguish it from other causes of facial paralysis include a sudden onset of unilateral facial paralysis which reaches the peak in less than 48 hours, and the absence of signs and symptoms of CNS, ear, and cerebellopontine angle disease [20].

Bell's palsy is a condition characterized by a droopy appearance on one side of the face and difficulty opening or closing the eye on the affected side. It can develop 1 to 2 weeks after a cold, ear infection, or eye infection. Symptoms include facial weakness, droopy mouth, difficulty making facial expressions, difficulty pronouncing words, dry eyes and mouth, altered taste, drooling, sensitivity to sound, difficulty eating and drinking, muscle twitches, eye irritation, and headache. In rare cases, it may affect both sides of the face [21].

Evaluating face muscles by House-Brackman Classification of Facial Function and giving a grade for each patient [22]:

Grade	Characteristics
I. Normal	Normal facial function in all areas
II. Mild dysfunction	Gross Slight weakness noticeable on close inspection May have slight synkinesis At rest, normal symmetry and tone Motion Forehead - Moderate to good function Eye - Complete closure with minimal effort Mouth - Slight asymmetry
III. Moderate dysfunction	Gross Obvious but not disfiguring difference between sides Noticeable (but not severe) synkinesis, contracture, or hemifacial spasm At rest, normal symmetry and tone Motion Forehead - Slight to moderate movement Eye - Complete closure with effort Mouth - Slightly weak with maximum effort
IV. Moderately severe dysfunction	Gross Obvious weakness and/or disfiguring asymmetry at rest, normal symmetry and tone Motion Forehead - None Eye - Incomplete closure Mouth - Asymmetrical with maximum effort
V. Severe dysfunction	Gross Only barely perceptible motion at rest, asymmetry Motion Forehead - None Eye - Incomplete closure Mouth - Slight movement
VI. Total paralysis	No movement

Complications of Bell's palsy

A number of complications can occur as a result of Bell's palsy, depending on the extent of nerve damage. Most of the patients fully recover from Bell's palsy within four months. However, about 2 in every 10 people experience long-term complication resulting from Bell's palsy [23].

Irreversible damage to the facial nerve may occur in complete palsy and loss of voluntary movement. It affects 20-30% of patients and can cause synkinesis, corneal ulceration, speech problems, facial contracture, and loss of taste. It can cause eye closure, infection, blindness, speech problems, and facial contracture, leading to disfigurement and a deeper line between the nose and mouth. Damage to the taste fibers of the facial nerve can also result in loss or reduced taste [24].

Management of facial nerve palsy

Corticosteroid treatment

The report of the Quality Standards Subcommittee of the American Academy of Neurology (AAN) [25]; released guidelines stating that steroids are highly likely to be effective and increase the likelihood of recovery of facial nerve function in new-onset Bell's palsy. The four trials showed significantly better facial outcomes in steroid-treated patients compared with non-steroid-treated patients.

The recommended dose of prednisone for the treatment of bell's palsy is 60 mg/day for 6 days, followed by a taper, for a total of 10 days. Caution should be used in patients with any of the following: tuberculosis, immunocompromise, pregnancy, active infection, sarcoidosis, sepsis, peptic ulcer disease, diabetes mellitus, renal or hepatic dysfunction and malignant hypertension [26].

Antiviral treatment

The combination of antivirals and corticosteroids may not significantly impact incomplete recovery rates in Bell's palsy, but the results are unclear. Corticosteroids alone are likely more effective than antivirals alone, and antivirals "valacyclovir 1,000 mg daily for 5 days" plus corticosteroids "prednisolone 60 mg daily for 5 days" then tapering for a total treatment of 10 days" with placebo plus prednisolone" are more effective than placebo or no treatment. There is no clear benefit from antivirals alone over placebo. The combination of antivirals and corticosteroids likely reduces late sequelae and fewer episodes of long-term sequelae in corticosteroid-treated participants [27].

Local eye treatment

Regardless of treatment given, all patients must be counseled regarding proper eye care to prevent exposure keratitis. Patients should use natural tears during the day and should put lubrical ointment in the eye at night. Patients should avoid fans and dust, and should consider wearing eye protection when they are outdoors in the wind [28].

Physical rehabilitation

A. Electrotherapy

The electrotherapy modalities reviewed included: electrical Stimulation (ES) and short-wave diathermy (SWD). No evidence supports electrical stimulation benefit for acute facial paralysis but it's effective for chronic condition in improving the facial motor control, the excursion of movement and in decreasing synkinesis. No evidence supports the benefit of using continuous mode of short- wave diathermy, while pulse mode can facilitate healing process in acute condition. Utilization of laser in acute or chronic conditions has not been supported by studies [29].

B. Neuromuscular retraining

Neuromuscular retraining involves selective motor training to control symmetrical movement and prevent gross motor activity (synkinesis). Patient reeducation is crucial in the treatment process, using EMG biofeedback and mirror exercises. The patient learns muscle action in the mirror and performs small symmetrical movements on the sound side to identify correct responses. Movements should be initiated slowly and gradually, allowing the patient to observe the angle, strength, and speed of each movement. Rapid movements are not recommended as they don't help control abnormal movement [30].

Biofeedback (BF) is a technique that combines electromyography (EMG) with biofeedback to record muscle electrical activity. It has been proven effective in restoring muscle control and rehabilitating movement

patterns after pathologies like facial muscles, muscle atrophy, stroke, or sports injuries. It provides information that is difficult to access naturally, facilitating voluntary muscle activity and improving involuntary regulation [31].

C. Manual Massage

Massage can be performed in conjunction with other treatment options. It can be done to improve perceptual awareness. Other methods of treatments include; Apply moist heat to the paralyzed area to help reduce pain, try drinking with straw and eat on the side of your mouth that feels most comfortable [32].

D. Kabat Rehabilitation

Kabat rehabilitation is a type of motor control rehabilitation technique based on proprioceptive neuromuscular facilitation (PNF). During Kabat procedure, the therapist facilitates the voluntary contraction of the impaired muscle by applying a global stretching then resistance to the entire muscular section and motivates action by verbal input and manual contact. Prior to Kabat, ice stimulation is performed to a specific muscular group, in order to increase its contractile power [33].

Surgical treatment

Surgical decompression of the facial nerve is a potential treatment option for patients at risk of poor recovery. However, there is no consensus on the optimal timing and approach. Some authors suggest that the most favorable outcomes occur within 14 days of symptom onset, while others suggest delayed surgery between 3 weeks and 4 months. The middle fossa approach offers good access to the labyrinthine segment of the facial nerve with an acceptable complication risk, while the transmastoid approach is effective with a relatively low complication rate [34].

Other surgical options include the following, [35]:

- Subocularis oculi fat (SOOF) lift.
- Implantable devices (gold weights) placed into the eyelid.
- Tarsorrhaphy.
- Transposition of the temporalis muscle.
- Facial nerve grafting.
- Direct brow lift.

The electrodiagnostic tests

The electrodiagnostic tests that are usually utilized to evaluate the function of the facial nerve are nerve conduction studies, electromyography (EMG) and blink reflex. These tests may aid in assessing the outcome of a patient who has persistent and severe palsy. An important point to consider is that most of the electromyographic/nerve conduction studies usually do not show an abnormality during the first 3 weeks following nerve injury [36].

1-The nerve excitability test:

Nerve excitability test (NET) is the oldest and best-known electrophysiological test with well-established clinical efficacy. During this test, the lowest current eliciting a facial twitch is defined as the threshold of excitation and the difference in thresholds between the two sides is calculated [37].

NET is less painful due to weak stimulation with just enough current to meet the threshold, and the required equipment is compact and inexpensive to procure. However, it is impossible to calculate the percentage of degenerated nerve fibers, and NET is inferior to ENoG in terms of accurate prognostic prediction for facial palsy. The appropriate timing NET is 7 to 10 days after the onset [38].

2- Electroneuronography (ENoG)

Electroneuronography (ENoG) is an electrophysiological test performed to evaluate the functional integrity and the degree of degeneration of the facial nerve [39]. ENoG may be interfered by possible collateral nerves which are regenerated after 2 weeks post-onset. Clinicians should keep in mind that ENoG should be performed for the first time at about 72 hours post-onset and again at 3 to 5 day intervals until confirmation can be determined [37].

Electroneurography (ENoG) reliably predicts the prognosis of facial palsy. However, the results of ENoG are dependent on the location, where the wave is detected, as a compound muscle action potential (CMAP) arising from the facial muscles. [40].

ENoG responses were measured on the affected and unaffected sides increasing current intensity to evoke compound muscle action potential. The compound muscle action potential (CMAP) was obtained from the frontalis and mentalis muscles to measure amplitude degeneration ratio, then from nasalis muscle to measure degeneration index using this equation: [100 − (ENoG amplitude affected/unaffected side) × 100] [41].

Blink reflexes

Corneal and blink reflexes are crucial for assessing the integrity of trigeminal and facial cranial nerves, which comprise the reflex arc. They result in excitation of orbicularis oculi motor units and lid closure. The afferent innervation originates from the nasociliary and supraorbital branches of the trigeminal nerve, while the efferent motor response is interceded via the facial nerve to the orbicularis oculi muscle [42].

The blink reflex (BR) test, which assesses damage to facial nerves by recording the action potential of the orbicularis oculi muscles in response to electrical stimulation of the supraorbital nerves, has been shown useful in estimating facial palsy. Although BR test results have been found prognostic in patients with facial palsy, uniform standards for factors prognostic of paralysis have not been established [43].

Electrical stimulation of the supraorbital nerve elicits two or more temporally separate responses of the orbicularis oculi muscles, an ipsilateral early component (R1) via a short latency pontine pathway and bilateral late component (R2) through a complex route. Of the two, R1 serves as a more reliable measure of nerve conduction along the reflex pathway, whereas R2 helps localize the lesion to the afferent or efferent reflex arc [44].

Blink reflex isn't a useful test for evaluating prognosis of facial paralysis within the first week of onset, and less useful than ENoG and nerve excitability test within the second week and similar to ENoG and nerve excitability test within the third week of onset [36].

Facial nerve conduction velocity (NCV)

Facial nerve conduction velocity (NCV): NCV is clinically used in determining the type and extent of nerve damage in a variety of peripheral nerve disorders. [45].

The facial nerve is stimulated at two points. First the nerve trunk is stimulated at the stylomastoid foramen; and second the marginal mandibular branch of the facial nerve is stimulated at the angle of the mandible. The recording electrodes are placed bilaterally on the mentalis muscle. One on the opposite side served as a reference [46].

Analysis and interpretation of Facial nerve conduction velocity ion velocity the nerve conduction velocity in m/s is calculated by dividing the distance between the two stimulation points (in mm) by the difference in latencies (in ms). The normal value is 48±5 m/s. Incomplete recovery was common in BP patients when the nerve conduction velocity was below 30 m/s [41].

Electromyography

EMG is most valuable in the time frame of 2–3 weeks to 3 months after the onset of a facial nerve injury. EMG is helpful in monitoring for regeneration if reinnervation occurs. EMG should be used and interpreted in combination with clinical examinations [47].

EMG is the only electrophysiological test useful after nerve excitability loss and degeneration. After 2-3 weeks, electrical stimulation tests like NETand ENoG become ineffective. Fibrillation potentials or positive waves on EMG can confirm facial nerve degeneration, and polyphasic reinnervation potentials can be seen as early as 4-6 weeks after paralysis onset [37].

Neuromuscular ultrasound

Neuromuscular ultrasound (NMUS) is becoming a standard element in the evaluation of peripheral nerve and muscle disease. When obtained simultaneously to electrodiagnostic studies, it provides dynamic, structural information that can refine a diagnosis or identify a structural etiology. NMUS can improve patient care for those with mononeuropathies, polyneuropathy, motor neuron disease and muscle disorders [48].

Imaging has been described as a sensitive method for distinguishing among etiologies of unilateral facial paralysis. Specifically, gadolinium-enhanced magnetic resonance imaging (MRI) is the modality of choice for lesions located within the parotid gland, cerebellopontine angle, and internal auditory canal (IAC), whereas high-resolution computed tomography (CT) is preferred for temporal bone pathology [49].

Recent studies have suggested an additional role for neuromuscular ultrasonography as a measure of disease severity and distribution. By including ultrasound assessment in ongoing and future clinical trials of therapeutic interventions in neuromuscular disease, it will be possible to determine how the technique can be used to facilitate clinical trials and the evaluation of new promising therapies [50].

Neuromuscular ultrasound is the scanning of nerve and muscles using high- resolution ultrasound for the purpose of assessment of neuromuscular disorders. To obtain high-resolution images for the nerves, high frequency transducer is needed (at least 12 MHz).

Nerves are cable-like structures and have a distinct architecture consisting of fascicles and surrounding epineurium [51].

Normal nerve appearance in the cross-sectional (axial) view is described as “honey-comb” due to the alternating, relatively hyperechoic (bright) epineurium and perineurium of fascicles. In the sagittal view, this may be seen as hyperechoic streaks parallel to the epineurium. Nerves can also be distinguished by their low anisotropy, meaning their appearance does not change significantly with tilting of the transducer, compared to structures with high anisotropy such as tendons and muscle [48].

Any nerve should be scanned in two views; transverse and longitudinal view. In transverse view, the nerve has a distinct perineurial border surrounding a relatively hypoechoic matrix (nerve fascicles give a honey comb appearance, and are less densely fibrillar than a tendon). In longitudinal view, it appears roughly as parallel linear strands consistent with endoneurial fibrous tissue (hyperechoic fibrillar cords). It is also very important to keep the transducer perpendicular to the nerve at all times during scanning especially on measuring the cross-sectional area (CSA) and the diameter [51].

Minimal pressure should be applied by the transducer during scanning to avoid artificial deformation of the nerve which may cause errors in measurement. Any nerve is typically assessed for size, shape, echogenicity, vascularity and mobility. For every nerve, there is a preferred patient position, but in general, the position should be comfortable to the patient first and should be comfortable to the examiner as well [50].

Gupta et al., [52] mentioned that, ultrasound has been utilized to predict facial nerve outcomes in Bell’s palsy. In this prospective, controlled study, patients with Bell’s palsy, ultrasound was performed 2–7 days after the onset of paralysis. Facial nerve diameter was measured proximally at the stylomastoid foramen, distally just proximal to the pes anserinus, and midway between these two points. The average diameter of the facial nerve was calculated using these three measurements and then compared with blink reflex studies and nerve conduction studies.

Conclusion

Bell's palsy is acute idiopathic unilateral lower motor neuron facial nerve weakness or paralysis, with no other neurologic or systemic manifestations; which affects 60-75% of cases. Electrodiagnostic tests used to evaluate facial nerve function include nerve conduction studies, electromyography (EMG), and blink reflex. Recent studies suggest an additional role for neuromuscular ultrasonography as a measure of disease severity and distribution. Ultrasound has been used to predict facial nerve outcomes in Bell's palsy.

References

1. Dulak D, Naqvi IA. Neuroanatomy, Cranial Nerve 7 (Facial) [Updated 2023 Jul 24]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK526119/
2. Patel BC, Seneviratne SO. Facial nerve anatomy and clinical applications. Treasure Island (FL): StatPearls Publishing; 2022. Available from: https://www.ncbi.nlm.nih.gov/books/NBK554569/. Accessed March 29, 2023.

3. Moore, K. L., Dalley. A. F., & Agur, A. M. R. (2014). Clinically Oriented Anatomy (7th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.
4. Walker NR, Mistry RK, Mazzoni T. Facial Nerve Palsy. [Updated 2023 Jul 4]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK549815/
5. Galnares-Olalde, J. A., López-Hernández, J. C., García-Grimshaw, M., et al., (2022). Guillain-Barré Syndrome in Mexico: An Updated Review Amid the Coronavirus Disease 2019 ERA. Rev Investig Clínica, 8319.
6. El-Tallawy H, Farghaly W, Shehata G, Badri R, Hassan M, Hamed M et al (2016) Incidence and clinical predictors of outcome of Bell's palsy, Al-Quseir City, Red Sea governorate, Egypt, the Egyptian journal of neurology. Psychiatry Neurosurg 53(2):70–73
7. Aditya V. LMN Facial Palsy in Pregnancy: An Opportunity to Predict Preeclampsia-Report and Review. Case Rep Obstet Gynecol. 2014;2014:626871. doi: 10.1155/2014/626871. Epub 2014 Mar 17. PMID: 24772359; PMCID: PMC3977111.
8. Ragupathy, K., Emovon, E. Bell's palsy in pregnancy. Arch Gynecol Obstet 287, 177–178 (2013). https://doi.org/10.1007/s00404-012-2506-0
9. Phillips, Katie & Heiser, Alyssa & Gaudin, Robert & Hadlock, Tessa & Jowett, Nate. (2017). Onset of bell's palsy in late pregnancy and early puerperium is associated with worse long-term outcomes. The Laryngoscope. 127. 10.1002/lary.26569.
10. Sathirapanya, P., Fujitnirun, C., Setthawatcharawanich, S., et al., (2018). Peripheral facial paralysis associated with HIV infection: a case series and literature review. Clinical Neurology and Neurosurgery, 172, 124-129.
11. Geddes, Alan & Barry, J. & Thomas, C.. (2018). Increased incidence of Bell's palsy with worse outcomes in pregnancy. British Journal of Oral and Maxillofacial Surgery. 56. 10.1016/j.bjoms.2018.06.016.
12. Cirpaciu D, Goanta CM, Cirpaciu MD. Recurrences of Bell's palsy. J Med Life. 2014;7 Spec No. 3(Spec Iss 3):68-77. PMID: 25870699; PMCID: PMC4391412.
13. Cannon, R. B., Gurgel, R. K., Warren, F. M., et al., (2015). Facial nerve outcomes after middle fossa decompression for Bell's palsy. Otology & Neurotology, 36(3), 513-518.
14. Danesh, A., & Ouanounou, A. (2022). Bell's Palsy: Etiology, Management and Dental Implications. J Can Dent Assoc, 88(m8), 1488-2159.
15. Greco, A., Gallo, A., Fusconi, M., et al., (2012). Bell's palsy and autoimmunity. Autoimmunity reviews, 12(2), 323-328.
16. Eviston, T. J., Croxson, G. R., Kennedy, P. G., et al., (2015). Bell's palsy: aetiology, clinical features and multidisciplinary care. Journal of Neurology, Neurosurgery & Psychiatry, 86(12), 1356-1361.
17. Love, S., Perry, A., Ironside, J., et al., (2018). Viral infections. In Greenfield's Neuropathology-Two Volume Set (pp. 1111-1215). CRC Press.
18. Rhaif, R. H., Al-Obaidi, A. B., & Al-Khazrajee, A. K. K. (2020). The Potential Role of Human Herpes Virus-6 in Idiopathic Facial Nerve Paralysis. Iraqi Journal of Medical Sciences, 18(1).
19. Chang, K. V., Wu, W. T., & Özçakar, L. (2020). Ultrasound imaging and guidance in peripheral nerve entrapment: hydrodissection highlighted. Pain Management, 10(2), 97-106.
20. Zimmermann, J., Jesse, S., Kassubek, J., et al., (2019). Differential diagnosis of peripheral facial nerve palsy: a retrospective clinical, MRI and CSF-based study. Journal of Neurology, 266(10), 2488-2494.
21. Kahn April and Jennifer Larson (2023): "What is Bells Palsy" https://www.healthline.com/health/bells-palsy.
22. House JW, Brackmann DE. Facial nerve grading system. Otolaryngol Head Neck Surg. 1985 Apr. 93(2):146-7. [QxMD MEDLINE Link].
23. Alayat, M. S., Elsodany, A. M., AlMatrafi, N. A., et al., (2022). Effectiveness of multiwave locked system laser on the treatment of patients with idiopathic Bell's palsy: a randomized double-blind placebo controlled trial. Lasers in Medical Science, 1-8.
24. Timorkhan, M. A., & Safey, Z. Z. (2022). The diagnosis and treatment of Bell's palsy. Middle East Journal of Family Medicine, 20(7).
25. Babl, F. E., Herd, D., Borland, M., et al., (2022). Efficacy of Prednisolone for Bell Palsy in Children: A Randomized, Double-Blind, Placebo-Controlled, Multicenter Trial. Neurology.
26. Jalali, M. M., Soleimani, R., Soltanipour, S., et al., (2021). Pharmacological treatments of Bell's palsy in adults: a systematic review and network meta-analysis. The Laryngoscope, 131(7), 1615-1625.
27. Gagyor I, Madhok VB, Daly F, Sullivan F. Antiviral treatment for Bell's palsy (idiopathic facial paralysis). Cochrane Database Syst Rev. 2019 Sep 5;9(9):CD001869. doi: 10.1002/14651858.CD001869.pub9. PMID: 31486071; PMCID: PMC6726970.
28. Gronseth GS and Paduga R, (2012): American Academy of Neurology. Steroids and antivirals for Bellpalsy: report of the Guideline Development Subcommittee of the American Academy of Neurology. Neurology, 79 (22): 2209–13.
29. Diels, J. (2019). 62 Nonoperative Facial Rehabilitation in Vestibular Schwannoma: Facial Neuromuscular Retraining. Comprehensive Management of Vestibular Schwannoma. New York, NY: Thieme, 418-421.
30. Kaur, A. (2018). Physiotherapy For Facial Palsy: The Facial Rehabilitation Guide. FSP Media Publications.
31. Morales-Sánchez V, Falcó C, Hernández-Mendo A, Reigal RE. Efficacy of Electromyographic Biofeedback in Muscle Recovery after Meniscectomy in Soccer Players. Sensors (Basel). 2022 May 26;22(11):4024. doi: 10.3390/s22114024. PMID: 35684645; PMCID: PMC9185253.
32. Krishnan, A., & Kogilavani, S. (2016). Bell's Palsy. Journal of Indian Academy of Neurology, 19(2), 164-174.

33. Dhanya, S. (2019). Proprioceptive Neuromuscular Facilitation (PNF) in Stroke Rehabilitation: A Review. Journal of International Academic Research for Multidisciplinary, 7(4), 1-10.
34. Kim BH, Yang H, Yoo MC. Usefulness of the Blink Reflex in Diagnosing Isolated Infraorbital Neuropathy Following Midface Augmentation with AlloPlastic Facial Implants: A Case Report. Life (Basel). 2022 Jul 26;12(8):1122. doi: 10.3390/life12081122. PMID: 35892924; PMCID: PMC9394420.
35. Goel, A. (2019). Facial Nerve Palsy (Bell's Palsy). StatPearls Publishing.
36. Guntinas-Lichius O, Volk GF, Olsen KD, Mäkitie AA, Silver CE, Zafereo ME, et al. Facial nerve electrodiagnostics for patients with facial palsy: a clinical practice guideline. Eur Arch Otorhinolaryngol. 2020 Jul;277(7):1855-1874. doi: 10.1007/s00405-020-05949-1. Epub 2020 Apr 8. PMID: 32270328; PMCID: PMC7286870.
37. Lee DH. Clinical Efficacy of Electroneurography in Acute Facial Paralysis. J Audiol Otol. 2016 Apr;20(1):8-12. doi: 10.7874/jao.2016.20.1.8. Epub 2016 Apr 21. PMID: 27144227; PMCID: PMC4853888.
38. Haginomori SI. An electrophysiological prognostic diagnosis for facial palsy. Auris Nasus Larynx. 2023 Apr;50(2):180-186. doi: 10.1016/j.anl.2022.08.010. Epub 2022 Aug 31. PMID: 36057466.
39. Skilbeck, Christopher & Mackeith, Samuel. (2022). The Facial Nerve. 10.1201/9781003175995-21.
40. Ayani Y, Haginomori SI, Wada SI, Nakano H, Hamada M, Ichihara T, et al. Latency shift in compound muscle action potentials during electroneurography in facial palsy. Eur Arch Otorhinolaryngol. 2019 Dec;276(12):3281-3286. doi: 10.1007/s00405-019-05634-y. Epub 2019 Sep 13. PMID: 31520164.
41. Abdelal, I.T., Eliwa, E.A., Ebaid, A.M. et al. Usefulness of electrophysiology in the prediction of outcome of Bell's palsy patients. Egypt Rheumatol Rehabil 47, 24 (2020). https://doi.org/10.1186/s43166-020-00022-5.
42. Khan, M. Z. (2021). Physiology, Blink Reflex.
43. Young Min Hah, Sang Hoon Kim, Junyang Jung, Sung Su Kim, Jae Yong Byun, et al. Prognostic value of the blink reflex test in Bell's palsy and Ramsay-Hunt syndrome. Auris Nasus Larynx. 2018;45(5):966-970. doi: 10.1016/j.anl.2018.01.007.
44. Kimura, Jun, 'Studies of the Facial Nerve and the Blink Reflex', Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice, 4 edn (2013; online edn, Oxford Academic, 1 Sept. 2013), https://doi.org/10.1093/med/9780199738687.003.0008, accessed 15 Dec. 2023.
45. Zhou J, Zhong Z, Li J, Fu W. Motor nerve conduction velocity is affected in segmental vitiligo lesional limbs. Int J Dermatol .. . 2016 Jun;55(6):700-5. doi: 10.1111/ijd.13171. Epub 2016 Feb 24. PMID: 26916936.
46. Emma Graf, Guy J. Petruzzelli,Identification and dissection of the facial nerve during parotidectomy,Operative Techniques in Otolaryngology-Head and Neck Surgery,Volume 29, Issue 3,2018,Pages 144-149,ISSN 1043-1810,https://doi.org/10.1016/j.otot.2018.09.005.
47. Urban, Elisabeth & Volk, Gerd & Geißler, Katharina & Thielker, Jovanna & Dittberner, Andreas & Klingner, Carsten & Witte, Otto & Guntinas-Lichius, Orlando. (2020). Prognostic factors for the outcome of Bells' palsy: A cohort register-based study. Clinical Otolaryngology. 45. 10.1111/coa.13571.
48. Gonzalez NL, Hobson-Webb LD. Neuromuscular ultrasound in clinical practice: A review. Clin Neurophysiol Pract. 2019 Jul 12;4:148-163. doi: 10.1016/j.cnp.2019.04.006. PMID: 31886438; PMCID: PMC6921231.
49. Su, B.M., Kuan, E.C. and St. John, M.A. (2018), What is the role of imaging in the evaluation of the patient presenting with unilateral facial paralysis?. The Laryngoscope, 128: 297-298.
50. Baek S-H, Kim YH, Kwon Y-J, et al. The Utility of Facial Nerve Ultrasonography in Bell's Palsy. Otolaryngology–Head and Neck Surgery. 2020;162(2):186-192. doi:10.1177/0194599819896298
51. Cartwright MS, Walker FO. Neuromuscular ultrasound in common entrapment neuropathies. Muscle Nerve. 2013 Nov;48(5):696-704. doi: 10.1002/mus.23900. Epub 2013 Sep 2. PMID: 23681885.
52. Gupta S, Mends F, Hagiwara M, Fatterpekar G, Roehm PC. Imaging the facial nerve: a contemporary review. Radiol Res Pract. 2013;2013:248039. doi: 10.1155/2013/248039. Epub 2013 May 23. PMID: 23766904; PMCID: PMC3676972.