



Ultrasonographic Characteristics of the Facial Nerve in Patient with Bell's Palsy

Hadi Khazaei*; Danesh Khazaei; Davin Ashraf; Shravani Mikkilineni; John D Ng

Casey Eye Institute, Oregon Health & Science University, Portland, Oregon, 97239, USA.

*Corresponding Author: Hadi Khazaei

Casey Eye Institute, Oregon Health & Science University,
Portland, Oregon, 97239, USA.

Tel: 503-494-3010, Fax: 503-494-3011;

Email: khazaei@ohsu.edu

Abstract

Peripheral facial paralysis is a diagnostic challenge. Acute facial palsies are mostly "idiopathic" (Bell's palsy) but it is a diagnosis of exclusion, and therefore cases of acute, acquired, isolated peripheral facial paralysis should be investigated thoroughly. Comprehensive efforts should be made to diagnose the cause, because some percentages of the patients referred with a diagnosis of Bell's palsy were found to have a treatable, progressive, or life-threatening lesion.

Although imaging can rule out some dangerous etiologies including compressive lesions, the findings are otherwise frequently non-specific and may only reveal patterns and locations of tissue involvement which may statistically be more common in certain disease entities. Imaging is often not specific enough to verify exact disease entities or obviate a biopsy.

High-resolution ultrasonography has emerged as a complementary tool assessment of neuromuscular disorders, it assesses nerve anatomy, detects changes in nerve size in response to different pathologies, and identifies extrinsic compressive lesions.

The purpose of this article is to explain the principles of the ultrasound techniques, outline the procedures, measurements and interpretation of the results, evaluate the reliability and validity of this method and to highlight the advantages and limitations of ultrasonography in patients with Bell's palsy.

Received: Jun 05, 2022

Accepted: Jun 23, 2022

Published Online: Jun 25, 2022

Journal: Annals of Ophthalmology and Visual Sciences

Publisher: MedDocs Publishers LLC

Online edition: <http://meddocsonline.org/>

Copyright: © Khazaei H (2022). *This Article is distributed under the terms of Creative Commons Attribution 4.0 International License*

Keywords: Idiopathic (Bell's) Facial Palsy; Facial Nerve Ultrasonography.

Introduction

The facial nerve can be affected by various disorders, including Bell's palsy, Guillain-Barre syndrome [1,2] Ramsay-Hunt syndrome, otitis media, sarcoidosis, tumors of the maxillary sinus and parotid glands, Lyme disease, and chronic inflammatory demyelinating polyneuropathy [3,4]. The reported frequency of a positive family history for idiopathic palsy has ranged from 2.4% to 28.6% [33-35].

A family history of facial palsies is noted in 14% of patients, and the syndrome is recurrent in 12%. Of those with a history of recurrence, the same side is involved in 36%. Bell's palsy appears to have a higher incidence during pregnancy. In one study, the calculated frequency in pregnant women was 45.1/100.000 births, compared with 17.4/100.000 per year in nonpregnant women of the same age group. Over 75% of the palsies occurred in the third trimester of pregnancy, and there was no



Cite this article: Khazaei H, Khazaei D, Ashraf D, Mikkilineni S, Ng JD. Ultrasonographic Characteristics of the Facial Nerve in Patient with Bell's Palsy. *Ann Ophthalmol Vis Sci.* 2022; 5(1): 1029.

apparent relationship between toxemia, primiparity, and hypertension. Finally, there appears to be a genetic predisposition to Bell's palsy.

In a case-control study 24.8% of patients with Bell's palsy had diabetes, compared with an age-matched control group who had a 13.1% incidence of diabetes. This difference is highly significant and implies a direct relationship between diabetes and Bell's palsy. Preservation of taste was significantly more common in patients with diabetes than in nondiabetics with Bell's palsy. This finding in diabetic patients is in accordance with previously reported studies and suggests a lesion distal to the chorda tympani branch of the facial nerve [30-32].

Subjective complaints include pain around the ear, facial numbness, changes in taste and numbness of the tongue. Dysacusis; failure to dampen the vibrating ear ossicles, as determined by middle ear function studies, loss of taste of the anterior two thirds of the tongue, and decreased sublingual and submandibular salivary secretion are most suggestive of a lesion in the tympanomastoid portion of the facial nerve [27].

Depending upon the extent of palsy, the prognosis for recovery of facial function can be predicted with a high degree of accuracy, with 90% of patients having a satisfactory recovery. Treatment for Bell's palsy is supportive, involving heat, massage, and facial biofeedback exercises. Decompressive surgery has not been shown to alter the natural history of Bell's palsy, and the use of steroids is controversial. At present, the decision to use steroids should be individualized. Considerations should include the patient's age, their general medical condition, the duration and the completeness of the palsy, and the presence of pain [15].

The routine uses of Antiviral medications (Acyclovir, Valacyclovir, etc.) in the treatment of Bell's palsy is becoming more widely accepted. A recent, double-blind study of Bell's palsy supports the combination of acyclovir and prednisone over prednisone alone. Further studies are necessary to determine whether acyclovir should be used alone in Bell's palsy [16-19].

The objectives of this study are to describe the sonographic characteristics of the facial nerve in healthy individual vs patients with Bell's palsy and to establish average values of its diameter.

Methodology

The basic concept of ultrasonography is the reflection of sound waves from tissues in the path of the beam. The transducer of the scan probe creates pulses with frequencies >2 MHz using Piezoelectric crystals or chips for ultrasonic imaging. When the wave pulses come in contact with a tissue interface (e.g., skin-subcutaneous fat, fat-muscle, and muscle-bone), they are partially reflected back to the transducer, which detects these reflections as the echo signal. Dense or rigid structures do not allow the waves to pass through and therefore a greater portion reflect back to the transducer. Such structures will create a strong echo which appears bright white on the screen, and the position of the dot represents the depth from which the echo was received, which are then combined to form an image [20].

The relative strength, or amplitude, of echoes is depicted by the brightness of the image on the computer screen. Robust reflections appear white, weaker reflections appear gray and regions free of reflections are black. This produces a two-dimensional grey-scale image with white borders for the skin-

subcutaneous fat and muscle-bone interfaces and an obvious, but less distinct border for the fat-muscle interface. The procedure for ultrasound scanning is fairly easy. A coupling agent is placed between the ultrasound probe and/or the skin at the location to be measured. This creates a bond between the probe and skin, thereby reducing echogenic interference and making it easier to maneuver the probe over the skin. With the ultrasound on, the transducer is maneuvered across the measuring site with continuous contact with the skin. Thickness of tissues is measured with the help of electronic calipers. Identification and placement of the 2 caliper points delineates the boundaries of the structure to be measured which improves the accuracy of the measurement. The scanned images on the monitor are saved for further interpretation and analysis [21].

Targeted exams include: Topographical echography in which a mass may be detected on the B-scan and its dimensions are measured utilizing the A-scan, sagittal plane scanning to gauge the facial nerve versus inflammation, quantitative analysis within which the A-scan uses echo reflectivity to calculate nerve tissue properties, and kinetic echography during which the physical pressure of the probe is employed to characterize the nature of tissues in question [22].

Time gain compensation (TGC) is a setting applied in diagnostic ultrasound imaging to account for tissue attenuation. By increasing the received signal intensity with depth, the artifacts in the uniformity of a B-mode image intensity are reduced. The purpose of TGC is to normalize the signal amplitude with time, compensating for depth.

When the image is displayed, similar materials should have similar brightness, regardless of depth; this is achieved by "Linear-in-dB" Gain, which means the decibel gain is a linear function of the control voltage. Gain is expressed in dB, a logarithmic ratio of the output power relative to the input power. Gain can be calculated by subtracting the input from the output levels when both are expressed in dBm, which is power relative to 1 milliwatt.

The TGC creates uniformity in the brightness of the echoes when used in conjunction with the overall gain. The best approach is to center all the TGC settings before adjusting the overall gain. After adjusting the overall gain, the TGC can then be adjusted to compensate for attenuation at specific depth. Gain is a uniform amplification of the ultrasonic signal that returns to the transducer after it travels through the tissue.

Discussion

Bell's palsy is a term reserved to designate an acute peripheral facial palsy of unknown cause. The disorder is self-limiting, non-progressive, not life-threatening, and spontaneously recovers; presently, it can be neither prevented nor cured. Incidence varies between 15 and 40 per 100,000 population annually [25-26].

The facial palsy is not in itself diagnostic. Tumors, similar to Bell's palsy, may present with incomplete, complete, sudden, slowly progressive, or recurrent ipsilateral peripheral facial palsy [28]. Accumulating evidence supports a viral inflammatory-immune mechanism. In about 60% of cases, Bell's palsy is associated with a viral prodrome. However, when a facial nerve palsy progresses for more than 3 weeks, a tumor must be excluded. In some cases of otherwise uncomplicated Bell's palsy, examination of the spinal fluid reveals aplacytosis and an increase in protein, without a micro-organism being disclosed [29].

The facial palsy is typically assessed with Electrodiagnostic (EDX) testing, which may include nerve conduction studies [5], blink reflex recording [6], electromyography [7,8] and magnetic resonance imaging [18]. The severity of the facial palsy has no relationship to the findings on MRI and the unaffected facial nerve may also show pathologic enhancement [14].

High-resolution ultrasonography has emerged as a complementary tool to EDX testing in assessment of neuromuscular disorders [14,17]; it assesses nerve anatomy [10], detects changes in nerve size in response to different pathologies [11,12] and identifies extrinsic compressive lesions [13].

The use of the M-mode or time-dependent intensity modulated ultrasound technique for ophthalmologic investigations was described by Coleman and Richard Weininger [37]. This technique provides the investigator with a means for monitoring structural changes in the eye during physiologic or pharmacologic experimental conditions, or a combination of both, and is particularly useful in studying optically inaccessible structures [36-37]. M-mode is a one-dimensional ("icepick") analysis of the tissue being evaluated. In an M-mode evaluation, echoes from underneath the icepick are displayed across the screen from left to right, creating a distance/time graph with time on the horizontal axis and tissue depth on the vertical axis.

In a pilot study conducted amongst 12 healthy individual vs 12 Bell's patient, mean facial nerve diameter was $.8 \pm .2$ mm among controls and $1.1 \pm .3$ mm among the palsy group. The facial nerve diameter was significantly larger in patients than controls with a significant side-to-side difference in patients as well [23].

In a recent study, 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 using a 10 MHz linear array transducer [24]. 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 [24].

We are conducting a similar study, measuring diameter of the facial nerve in patients with recent history of Bell's palsy comparing with contralateral side as control. In this study, the extra-cranial part of the facial nerve is scanned bilaterally along its longitudinal axis inside the parotid gland using a chip embedded 10 MHz linear array transducer probe (Butterfly IQ+).

The facial nerve diameter is measured at its thickest part immediately inside its hyperechoic border. Measurement calipers are extended to span between the inner borders of the hyperechoic edges of the nerve. We have recently published these finding in our "Hemifacial lipoatrophy" study [38].

The subjects were asked to lie in the lateral decubitus position on the side opposite the scanned site. For facial nerve scanning, the probe was placed just under the ear lobule with the mark on the probe directed toward the examiner's left side to image the nerve along its longitudinal course inside the parotid gland, after its exit from the stylomastoid foramen. A cross-sectional view of the facial nerve is difficult to obtain because of the small caliber of the facial nerve. The probe was

kept perpendicular to the skin at all times with minimal pressure by the probe, to ensure accurate measurements. The facial nerve diameter was measured at the site of maximum thickness with measurement marks placed on the border of the hyperechoic edges of the nerve. One patient had facial synkinesis and was additionally scanned using ultrasound M-mode, where the probe is positioned on the orbicularis oculi muscle and the patient was asked to blink the ipsilateral eye.

Facial nerve sonographic features were consistent in all controls (Opposite side), it appeared as a thin tubular-like structure with a hypoechoic center and hyperechoic rim. In affected side (Bell's palsy side), facial nerve echogenicity was hyperechoic with blurred nerve's outer rim. No increase in Doppler signal of the facial nerve was noted in controls or affected side. M-Mode scan was normal in facial synkinesis patient.

Limitations

Ultrasound use in the diagnosis, prognosis, and monitoring of facial nerve disorders is not without its shortcomings. Moreover, the user-dependent nature of sonographic imaging may be detrimental to study consistency and reproducibility. Facial nerve cross-sections are oval rather than round, with their maximum coronal volume occurring over a fixed area, making it difficult for a radiologist or ultrasound technician to reproduce the same angle and location for it. In our recent stimulation cadaveric study of facial nerve, we could successfully identify all branches of facial nerve and its surrounding structures using a high frequency 3D ultrasound with 10MHz linear array transducer probe with Bi-plane preset. This will help to delineate the facial structures and perform intraoperative ultrasound guided procedures like fine-needle biopsy or injections. New ultrasound devices and accompanying software designed might help to minimize these limitations. Another limitation is that only the peripheral portion of the facial nerve is accessible for imaging. Pathology at brainstem to stylomastoid foramen cannot be visualized so ultrasound cannot obviate the need for MRI in cases where that is indicated.

Conclusion

Ultrasound may show an increase in facial nerve diameter and side-to-side difference in diameter in patients with facial nerve palsy compared to controls. The diameter of the affected side may be significantly larger than that of the healthy side in patients with Bell's palsy. Ultrasound also may be helpful in the diagnosis of other causes of facial nerve palsy. Serial ultrasonographic scanning of the nerve from disease onset until recovery would also help advance this promising technique.

Declaration of helsinki

This review is adhered to the ethical principles outlined in the Declaration of Helsinki as amended in 2013.

Funding: None

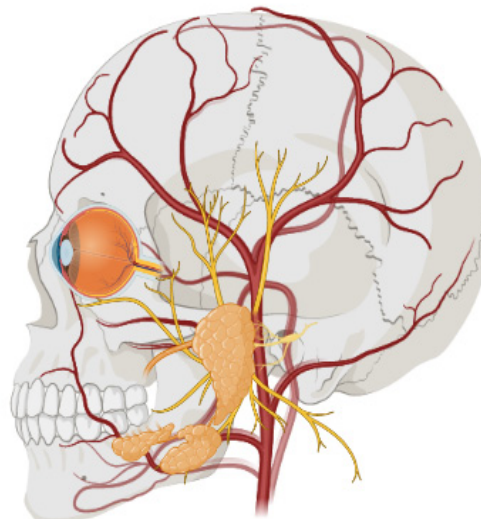
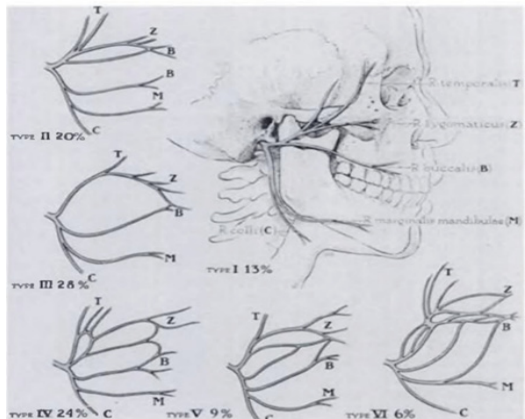
Conflict of interest: None

Authors' contribution Acknowledgement:

All the concerned authors jointly edited and approved the final manuscript. The authors thank Dr. Kaneez Abbas for her critical feedback and assistance in developing the search strategy and for proof reading.

Supplement: Facial ultrasonography instructional diagram
(Created with BioRender.com)

Patterns of peripheral distribution of facial nerve. (Modified from Davis RA, Anson BJ, Puddinger JM, Kurth RE: Surgical anatomy of the facial nerve and parotid gland based upon a study of 350 cervical facial halves. Surg Gynecol Obstet 102:385, 1956)

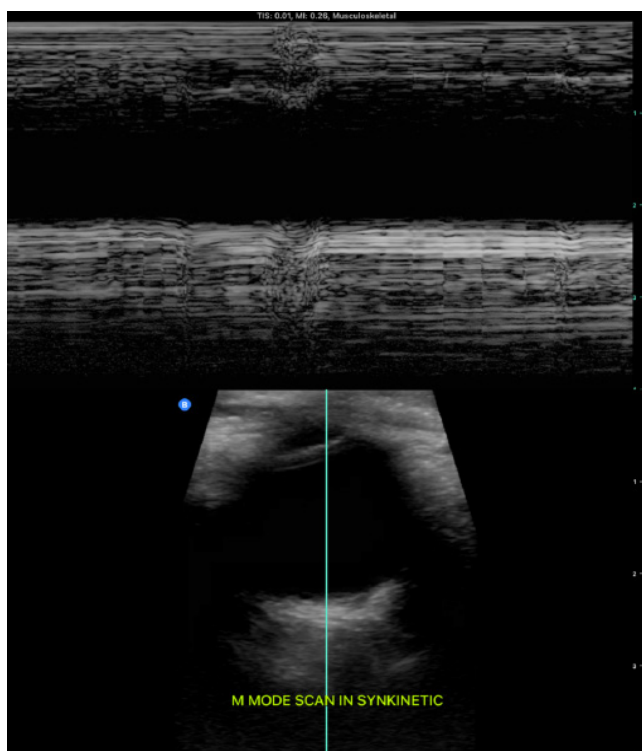


Created in BioRender.com

Classification System for Reporting Results of Recovery

Degree of Injury	Grade	Definition
Normal (1°)	I	Normal symmetric function in all areas
Mild dysfunction (barely noticeable) (1° or 2°)	II	Slight weakness noticeable only on close inspection; complete eye closure with minimal effort; slight asymmetry of smile with maximal effort; synkinesis barely noticeable, contracture, or spasm absent
Moderate dysfunction (obvious difference) (2° or 3°)	III	Obvious weakness, but not disfiguring; may not be able to lift eye-brow; complete eye closure and strong but asymmetric mouth movement with maximal effort; obvious, but not disfiguring, synkinesis, mass movement, or spasm
Moderately severe dysfunction (3°)	IV	Obvious disfiguring weakness; inability to lift brow; incomplete eye closure and asymmetry of mouth with maximal effort; severe synkinesis, mass movement, spasm
Severe dysfunction (3° to 4°)	V	Motion barely perceptible; incomplete eye closure, slight movement of corner of mouth; synkinesis, contracture, and spasm usually absent
Total paralysis	VI	No movement, loss of tone, no synkinesis, contracture, or spasm

(House JW: Facial nerve grading system. Laryngoscope 93:1056, 1983).



Normal facial nerve in a healthy volunteer: The nerve appears in as thin tubular structure with hypoechoic center and hyperechoic outer border (arrow) inside the homogenous parotid gland. The diameter measured .8 mm. M-Mode scan was normal in facial synkinesis patient.

References

1. Narayanan RP, James N, Ramachandran K, Jaramillo MJ. Guillain-Barre syndrome presenting with bilateral facial nerve paralysis: a case report. *Cases J.* 2008; 1: 379.
2. D'Amore A, Viglianesi A, Cavallaro T, Chiaramonte R, Muscoso EG, et al. Guillain-Barre syndrome associated with acute onset bilateral facial nerve palsies. A case report and literature review. *Neuroradiol J.* 2012; 25: 665-670.
3. Kokubun N, Hirata K. Neurophysiological evaluation of trigeminal and facial nerves in patients with chronic inflammatory demyelinating polyneuropathy. *Muscle Nerve.* 2007; 35: 203-207.
4. Varela H, Rubin DI. Facial and trigeminal neuropathies as the initial manifestation of chronic inflammatory demyelinating polyradiculopathy. *J Clin Neuromuscul Dis.* 2009; 10: 194-198.
5. Lydiatt WM, Sobba-Higley A, Morrow P, Moore GF. Use of electroneuronography in monitoring facial nerve paralysis. *Nebr Med J.* 1992; 77: 231-234.
6. Kimura J. Electrodiagnosis of the cranial nerves. *Acta Neurol Taiwan.* 2006; 15: 2-12.
7. Batra SP, Sinha A, Singh NN, Abrol BM. Electro-diagnosis in peripheral facial nerve paralysis. *Indian J Otolaryngol.* 1973; 25: 76-86.
8. Grosheva M, Guntinas-Lichius O. Significance of electromyography to predict and evaluate facial function outcome after acute peripheral facial palsy. *Eur Arch Otorhinolaryngol.* 2007; 264: 1491-1495.
9. Cartwright MS, Brown ME, Eulitt P, Walker FO, Lawson VH, et al. Diagnostic nerve ultrasound in Charcot-Marie-Tooth disease type 1B. *Muscle Nerve.* 2009; 40: 98-102.
10. Meng S, Tinhofer I, Weninger WJ, Grisold W. Anatomical and ultrasound correlation of the superficial branch of the radial nerve. *Muscle Nerve.* 2014; 50: 939-942.
11. Nakamichi KI, Tachibana S. Enlarged median nerve in idiopathic carpal tunnel syndrome. *Muscle Nerve.* 2000; 23: 1713-1718.
12. Schreiber S, Abdulla S, Debska-Vielhaber G, Machts J, Dannhardt-Stieger V, et al. Peripheral nerve ultrasound in ALS phenotypes. *Muscle Nerve.* 2014.
13. Lai LP, Chen B, Kumar S, Desai R, Mendoza J, et al. Ganglion cyst at the fibular head causing common peroneal neuropathy diagnosed with ultrasound and electrodiagnostic examination: A case report. *Am J Phys Med Rehabil.* 2014; 93: 824-827.
14. Kohsyu H, Aoyagi M, Tojima H: Facial nerve enhancement in Gd-MRI in patients with Bell's palsy. *Acta Otolaryngol.* 1994; 511: 165.
15. Knox G. Treatment controversies in Bell's palsy. *Arch Otolaryngol Head Neck Surg* 124:821, 1998
16. May M, Klein SR, Taylor FH. Idiopathic (Bell's) facial palsy: Natural history defies steroid or surgical treatment. *Laryngoscope.* 1985; 95: 406.
17. Stankiewicz JA. A review of the published data on steroids and idiopathic facial paralysis. *Otolaryngol Head Neck Surg.* 1987; 97: 481.
18. Grogan PM, Gronseth GS. Practice parameter: Steroids, acyclovir, and surgery for Bell's palsy (an evidence-based review). Report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology.* 2001; 56: 830.
19. Adour KK, Ruboyianes JM, Von Doersten PG, et al. Bell's treatment with acyclovir and prednisone compared with prednisone alone: A double-blind randomized controlled trial. *Ann Otol Rhinol Laryngol.* 1996; 105: 37.
20. Khazaei H, Khazaei D, Ashraf D, Mikkilineni S, Ng JD. Overview of Orbital Ultrasonography. *Ann Ophthalmol Vis Sci.* 2022; 5: 1028.
21. JP Noce. "Fundamentals of diagnostic ultrasonography," *Biomedical Instrumentation and Technology.* 1990; 24: 456-459.
22. Lo YL, Fook-Chong S, Leoh TH, Dan YF, Lee MP, et al. High-resolution ultrasound in the evaluation and prognosis of Bell's palsy. *Eur J Neurol.* 2010; 17: 885-889.
23. Tawfik EA, Walker FO, Cartwright MS. "A Pilot Study of Diagnostic Neuromuscular Ultrasound in Bell's Palsy". *J Neuroimaging.* 2015; 25: 564-570.
24. Gupta S, Mends F, Hagiwara M, Fatterpekar G, Roehm PC. "Imaging the facial nerve: A contemporary review". *Radiol Res Pract.* 2013; 2013: 248039.
25. Hauser WA, Karnes WE, Annis J. Incidence and prognosis of Bell's palsy in the population of Rochester, MN: *Mayo Clin Proc.* 1971; 46: 258.
26. Adour KK, Byl FM, Hilsinger RL. The true nature of Bell's palsy: Analysis of 1000 consecutive patients. *Laryngoscope.* 1978; 88: 787.
27. May M, Hardin WB. Facial palsy: Interpretation of neurologic findings. *Trans Am Acad Ophthalmol Otolaryngol.* 1977; 84: 710.
28. Peitersen E: The natural history of Bell's palsy. *Am J Otol.* 1982; 4: 107.
29. Katusic SK, Beard CM, Wiederholt WC. Incidence, clinical features and prognosis in Bell's palsy. *Ann Neurol.* 1986; 20: 622.
30. Kohler A, Chofflon M, Sztajzel R, et al. Cerebrospinal fluid in acute peripheral facial palsy. *J Neurol.* 1999; 246: 165.
31. Park HW, Watkins AL. Facial paralysis: Analysis of 500 cases. *Arch Phys Med.* 1949; 30: 749.
32. Paolino E, Granieri E, Tola MR. Predisposing factor in Bell's palsy: *J Neurol.* 1985; 232: 363.
33. Pechet P, Schattner A. Concurrent Bell's palsy and diabetes mellitus: A diabetic mononeuropathy? *J Neurol.* 1985; 232: 363.
34. Hilsinger RL, Adour KK, Doty HE. Idiopathic facial paralysis, pregnancy and the menstrual cycle. *Ann Otol Rhinol Laryngol.* 1975; 84: 433.
35. Takahash A, Fujiwara R. Familial Bell's palsy. Report of seven families. *Clin Neurol (Tokyo).* 1971; 11: 454.
36. Coleman DJ, Carlin B. A New System for Visual Axis Measurements in the Human Eye Using Ultrasound. *Arch Ophthalmol.* 1967; 77: 124-127.
37. Coleman DJ, Konig WF, Katz L. A Hand-Operated, Ultrasound Scan System for Ophthalmic Evaluation. *Amer J Ophthalmol.* 1969; 68: 256.
38. Khazaei H, Khazaei D, Brundage D, Mikkilineni S, Dailey RA. Facial Ultrasonography in acquired facial lipoatrophy. *Inter J. Research and Scientific Innovation (IJRSI).* 2022; 9: 48-51.