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

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Abstract
Peripheral facial paralysis is a diagnostic challenge. Acute facial palsy is 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.

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

apparent relationship between toxemia, primiparity, and hyper
tension. 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 sig-
nificant and implies a direct relationship between diabetes and
Bell’s palsy. Preservation of taste was significantly more com-
mon 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. Dys-
acusis; failure to dampen the vibrating ear ossicles, as deter-
dined by middle ear function studies, loss of taste of the an-
terior two thirds of the tongue, and decreased sublingual and
submandibular salivary secretion are most suggestive of a le-
sion in the tympanomastoid portion of the facial nerve [27].

Depending upon the extent of palsy, the prognosis for re-
covery 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, mas-
sage, 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 (Acylovir, Valacy-
lovir, 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 trans-
ducer of the scan probe creates pulses with frequencies >2 MHz
using Piezoelectric crystals or chips for ultrasonic imaging. When
the 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 por-
tion 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. Ro-
bust 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 proce-
dure for ultrasound scanning is fairly easy. A coupling agent is
placed between the ultrasound probe and/or the skin at the lo-
cation 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 ultra-
sound on, the transducer is maneuvered across the measuring
site with continues 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 with-
in which the A-scan uses echo reflectivity to calculate nerve tis-
sue properties, and kinetic echography during which the physi-
cal pressure of the probe is employed to characterize the nature
of tissues in question [22].

Time gain compensation (TGC) is a setting applied in diag-
nostic 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 loga-
rithmic 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 ap-
proach 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 re-
turns 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 var-
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 pal-
sy [28]. Accumulating evidence supports a viral inflammatory-
immune mechanism. In about 60% of cases, Bell’s palsy is asso-
ciated 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, examina-
tion of the spinal fluid reveals aplacocytosis 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 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 lobe 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 hyperechoic 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.

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Classification System for Reporting Results of Recovery

<table>
<thead>
<tr>
<th>Degree of Injury</th>
<th>Grade</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal (1°)</td>
<td>I</td>
<td>Normal symmetric function in all areas</td>
</tr>
<tr>
<td>Mild dysfunction (barely noticeable) (1° or 2°)</td>
<td>II</td>
<td>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</td>
</tr>
<tr>
<td>Moderate dysfunction (obvious difference) (2° or 3°)</td>
<td>III</td>
<td>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</td>
</tr>
<tr>
<td>Moderately severe dysfunction (3°)</td>
<td>IV</td>
<td>Obvious disfiguring weakness; inability to lift brow; incomplete eye closure and asymmetry of mouth with maximal effort; severe synkinesis, mass movement, spasm</td>
</tr>
<tr>
<td>Severe dysfunction (3° to 4°)</td>
<td>V</td>
<td>Motion barely perceptible; incomplete eye closure, slight movement of corner of mouth; synkinesis, contracture, and spasm usually absent</td>
</tr>
<tr>
<td>Total paralysis</td>
<td>VI</td>
<td>No movement, loss of tone, no synkinesis, contracture, or spasm</td>
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Supplement: Facial ultrasonography instructional diagram
(Created with BioRender.com)

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


