The Facial Nerve

Facial Nerve Disorders, Facial Nerve Tests

Mike Puricelli, MD
David Chang, MD
Overview

• Anatomy
• Case
• Facial Nerve Injury
• Natural History of Bell’s Palsy
• Risk Stratification for Recovery
• AAO-HNS Recommendations for Bell’s Palsy
• Literature Update on “No recommendation”
Background

• Facial nerve contains 10,000 fibers
  – 7,000 myelinated and innervate muscles of facial expression
  – 3,000 innervate salivary and lacrimal glands, sensory fibers to posterior EAC, and sensory fibers for taste

Embryology

- Facial nerve appears near the end of the first month of gestation: acousticofacial primordium
- Sixth week of gestation, the motor division of the facial nerve establishes its position in middle ear and extratemporal development begins
- By the end of the third gestational month, a majority of the facial musculature is identifiable and functional

Embryology

• Fallopian canal ossification starts by the fifth/sixth gestational month
  – Not complete until several years after birth
    • Leads to facial nerve dehiscence
• At birth, the facial nerve is located just beneath the skin near the mastoid tip, as it emerges from the temporal bone
  – At risk when a postauricular incision is made in a young child, as is often done for ear surgery
• As the mastoid tip grows, the facial nerve assumes a more medial position
• Myelination continues until the age of 4 years
Meatal Segment

- Facial nerve exits brainstem ventrolaterally as rootlets that coalesce prior to entering IAC
- Blood flow from labyrinthine artery (anterior inferior cerebellar artery)
- Easily injured by manipulation at this region

Labyrinthine Segment

- Runs from medial to lateral, from the fundus to the geniculate ganglion
  - Facial nerve is anterosuperior corner of porus acousticus
- Thinnest and shortest segment of the FN
  - 2.5-6 mm in length
- Diameter of the fallopian canal is smallest in the labyrinthine segment
- Thin plate of bone separating the nerve from the middle cranial fossa dura superiorly (absent 10-15%)
- First branch is greater superficial petrosal nerve, anterior surface of the geniculate ganglion
  - 93% of facial nerve injuries from longitudinal temporal bone fractures occur in this area
    - GSPN may be a traction force for certain closed head injuries

Tympanic Segment

• After the geniculate ganglion, the facial nerve curves posteriorly at an angle ranging between 60 and 90°
• When the nerve reaches the level of the oval window, it starts to curve inferiorly, forming the second genu
• Dehiscence
  – In some series has been reported to be up to 50%

Mastoid Segment

- Extends from the second genu to the stylomastoid foramen
- Facial nerve at the posteroinferior quadrant of the annulus may cross lateral to the plane of the annulus, making it susceptible to injury
- Epineurium is thickest in distal mastoid segment

Extratemporal Segment

- Traverses substance of the parotid gland
- Extensive and variable branching at pes anserinus
- Enters deep surface of facial muscles

Microscopic Anatomy

• Motor cell bodies are located in the brainstem
• Endoneurium covers schwann cells over individual axons
• Multiple axons are gathered into fascicles and covered by perineurium
  – Variable arrangement and compactness
  – Susceptible to injury at brainstem
• Epineurium protects the groups of fascicles
  – Houses the venules and arterioles

Microscopic Anatomy

Fig. 3—Multifascicular nerve. Note the epineurial nerve sheath, as well as interfascicular epineurium with venules and arterioles. Condensation of the perineurium surrounds each fascicle. No vessels larger than capillaries are found within the fascicle.
Fig. 4—Facial nerve fascicular anatomy. Notice the monofascicular pattern that is maintained from the internal auditory canal to the upper mastoid segment. In the distal mastoid portion, the nerve breaks up into two, three, or four distinct fascicles.
Question

• What segment of the fallopian canal is the most susceptible to compression?
  – Meatal
  – Labyrinthine
  – Tympanic
  – Mastoid
Question

• What segment of the fallopian canal is the most narrow?
  – Meatal
  – Labyrinthine
  – Tympanic
  – Mastoid

• “Fisch measured the diameter of the fallopian canal throughout the temporal bone and found that the narrowest point was at the junction of the internal auditory canal and the labyrinthine portion, which he called the meatal foramen. The meatal foramen averaged 0.68 mm in diameter, whereas the remainder of the fallopian canal measured 1.02 to 1.53 mm”

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Case

• A 51 year old presents to the ED facial nerve paralysis
Clinical Evaluation

- Onset
- Timing
  - Slow vs Rapid
- Degree
- Associated signs/symptoms
- Recurrence

Clinical Evaluation

• Two days ago
• Sudden
• Complete paralysis
• No facial spasms, vesicles on the face
• Denies neurologic deficits
• No prior episodes
Physical Examination

• Full head and neck examination
• Face/EAC lesions
• Other neurologic losses
• Severity grading
  – Particular attention to eye closure and presence of Bell’s phenomenon
<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal</td>
<td>Normal facial function in all areas</td>
</tr>
</tbody>
</table>
| II      | Mild dysfunction       | Gross: slight weakness noticeable on close inspection; may have very slight synkinesis  
At rest: normal symmetry and tone 
Motion—Forehead: moderate to good function 
Motion—Eye: complete closure with minimum effort 
Motion—Mouth: slight asymmetry |
| III     | Moderate dysfunction   | Gross: obvious but not disfiguring difference between two sides; noticeable but not severe synkinesis, contracture, or hemifacial spasm  
At rest: normal symmetry and tone 
Motion—Forehead: slight to moderate movement 
Motion—Eye: complete closure with effort 
Motion—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 
Motion—Eye: incomplete closure  
Motion—Mouth: asymmetric with maximum effort |
| V       | Severe dysfunction     | Gross: only barely perceptible motion  
At rest: asymmetry  
Motion—Forehead: none 
Motion—Eye: incomplete closure 
Motion—Mouth: slight movement |
| VI      | Total paralysis        | No movement                                                                        |
Bell’s Palsy - Definition

• Minimum diagnostic criteria
  – Paralysis or paresis of all muscle groups of one side of the face
  – Sudden onset
  – Absence of signs of central nervous system (CNS) disease
  – Absence of signs of ear or cerebellopontine angle disease
• Bell’s palsy is diagnosed when no other medical etiology is identified as a cause of the facial weakness.

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
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<tbody>
<tr>
<td>Acute</td>
<td>Occurring in less than 72 hours</td>
</tr>
<tr>
<td>Bell’s palsy</td>
<td>Acute unilateral facial nerve paresis or paralysis with onset in less than</td>
</tr>
<tr>
<td></td>
<td>72 hours and without identifiable cause</td>
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<tr>
<td>Electromyography (EMG) testing</td>
<td>A test in which a needle electrode is inserted into affected muscles to</td>
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<tr>
<td></td>
<td>record both spontaneous depolarizations and the responses to voluntary</td>
</tr>
<tr>
<td></td>
<td>muscle contraction</td>
</tr>
<tr>
<td>Electroneuronography (ENoG) testing</td>
<td>A test used to examine the integrity of the facial nerve, in which</td>
</tr>
<tr>
<td></td>
<td>surface electrodes record the electrical depolarization of facial</td>
</tr>
<tr>
<td></td>
<td>muscles following electrical stimulation of the facial nerve</td>
</tr>
<tr>
<td>Facial paralysis</td>
<td>Complete inability to move the face</td>
</tr>
<tr>
<td>Facial paresis</td>
<td>Incomplete ability to move the face</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>Without identifiable cause</td>
</tr>
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</table>

Baugh, RF. Basura, GJ. Ishii, LE. Et al. Clinical Practice Guideline: Bell’s Palsy. Otolaryngology Head and Neck Surgery. 149(3S) S1–S27
Bell’s Palsy - Pathophysiology

• Proposed causes
  – Microcirculatory failure, viral infection, ischemic neuropathy, and autoimmune reactions
• “While a viral etiology is suspected, the exact mechanism of Bell’s palsy is currently unknown. Facial paresis or paralysis is thought to result from facial nerve inflammation and edema. As the facial nerve travels in a narrow canal within the temporal bone, swelling may lead to nerve compression and result in temporary or permanent nerve damage.”

Baugh, RF. Basura, GJ. Ishii, LE. Et al. Clinical Practice Guideline: Bell’s Palsy. Otolaryngology Head and Neck Surgery. 149(3S) S1–S27
Question

• Which of the following must be ruled out prior to establishing the diagnosis of Bell’s palsy
  – Stroke
  – Ear disease
  – Cerebellopontine angle disease
  – Stroke and CPA disease
  – All of the above
Question

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  – Stroke
  – Ear disease
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  – Stroke and CPA disease
  – All of the above

Key to acute evaluation is ruling out these three critical pathologies. Corticobulbar tract through the internal capsule through the pyramidal tracts within the basal pons. In the caudal portion of the pons, most of the facial nerve fibers cross the midbrain to reach the contralateral facial nucleus. Small number of facial nerve fibers innervate the ipsilateral facial nucleus, a majority of which are destined for the temporal branch of the nerve. Central lesions spare the forehead muscle since they receive input from both cerebral cortices, whereas peripheral lesions will involve all branches of the facial nerve.

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Facial Nerve Injury - Classification

<table>
<thead>
<tr>
<th>SEDDON</th>
<th>NEUROPRAXIA FIRST</th>
<th>AXONOTMESIS SECOND</th>
<th>NEUROTMESIS</th>
<th>THIRD</th>
<th>FOURTH</th>
<th>FIFTH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Injury</td>
<td></td>
<td></td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Myelin</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Axon</td>
<td></td>
<td></td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Endoneurium</td>
<td></td>
<td></td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Perineurium</td>
<td></td>
<td></td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Epineurium</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mechanism</td>
<td>Compression Segmental demyelination</td>
<td>Crush Wallerian degeneration</td>
<td>Cut Wallerian degeneration</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Consequence</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>
Facial Nerve Injury – First Degree

• “Neuropraxia”
• Conduction is blocked but axoplasmic transport continues
• Nerve distal to the lesion retains normal electrical stimulation though voluntary motor function is abnormal
• Rapid recovery after insult removed

Facial Nerve Injury – First Degree

**FIRST DEGREE INJURY**
(Conduction Block)

Fig. 5—First-degree injury. Nerve fiber is twisted or compressed in such a way as to distort intraneural anatomy, yet allows axoplasmic flow in both directions. Note the myelin layer is also maintained in these injuries.

Facial Nerve Injury – Second Degree

• “Axonotmesis”
• Axonal continuity is lost without loss of surrounding structures
• Wallerian degeneration occurs distally
• Proximal axon retains normal histologic conformation
• Connective tissue elements remain viable permitting return to nervous original destination

Facial Nerve Injury – Second Degree

Fig. 6—Second-degree injury. With continued pressure, torsion, or both, axoplasmic continuity is disrupted. The distal axon’s trophic influence on the myelin layer is lost, resulting in distal degeneration of the myelin layer. The Schwann cell and endoneurium, which together constitute the endoneurial tube, remain intact.
Facial Nerve Injury – Third Degree

• “Neurotmesis”

• Endoneurial tube is disrupted in addition to axon and myelin
  – Allows for aberrant regeneration
    • Synkinesis

Facial Nerve Injury – Third Degree

Fig. 7—Third-degree injury. With increasing torsion, pressure, or some sharp injuries, all components of nerve fiber are disrupted. The distal axon and myelin layer degenerate, while the endoneurial tube forms a receptive conduit to attract regenerating axon sprouts.
Facial Nerve Injury – Fourth Degree

• “Neurotmesis”
• Includes perineurium injury
• Axons from one fascicle are free to enter another fascicle
• Common in blunt crush injuries
• Intraneural scarring may prevent some axons from reaching their destination
  – Worsened synkinesis
  – Incomplete motor recovery

Facial Nerve Injury – Fourth Degree

Fig. 8—Fourth-degree injury. (In this diagram, the entire nerve is shown.) Note that intraneural disruption has occurred, and perineurium surrounding each fascicle is absent. This allows regeneration of axon sprouts outside the fascicle, as shown.
Facial Nerve Injury – Fifth Degree

• “Neurotmesis”
• All components of peripheral nerve are transected
• Regenerative axons escape the confines of the nerve sheath
  – Neuroma formation
• Low probability for useful regeneration

Facial Nerve Injury – Fifth Degree

Fig. 9—Fifth-degree injury. Here, injury is similar to fourth-degree injury, except that the epineurial nerve sheath is also torn. This allows extraneural axonal regeneration and neuroma formation outside the sheath.
Facial Nerve Injury – Sixth Degree

- Trunk injury that is not uniform across the nerve
- Arguably the most common facial nerve lesion

Summary of Sunderland Classification

Table I. Classification of nerve injuries after Sunderland (19, 20)

<table>
<thead>
<tr>
<th>Degree</th>
<th>Pathology</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Neuropraxia</td>
<td>Complete</td>
</tr>
<tr>
<td>2</td>
<td>Axonotmesis</td>
<td>Complete</td>
</tr>
<tr>
<td>3</td>
<td>Neurotmesis</td>
<td>Incomplete</td>
</tr>
<tr>
<td>4</td>
<td>Perineurium disruption</td>
<td>Non-functional</td>
</tr>
<tr>
<td>5</td>
<td>Complete disruption</td>
<td>None</td>
</tr>
</tbody>
</table>

Facial Nerve Injury

• Cessation of retrograde axonal transport stimulates gradual cessation of neuroeffector substance production and protein synthesis
  – If injury is too severe: glial scarring
• Second to fifth degree injuries the distal segment undergoes Wallerian degeneration
  – Axoplasm and myelin “dissolve” (macrophages) to the motor endplate
  – Not rapid
    • Leads to “stimulability” for 72 hours following injuries
  – Essential process due to inhibiting factors in myelin
  – Brungers bands (myelin sheath and endoneurium) attract regenerating axons
• Synaptic clefts can fill with collagen

Facial Musculature

• Fiber size reduces and histologic evidence of denervation is present within days (enlarged nucleoli, reduction in myofibrils, loss of mitochondria, loss of sarcoplasmic reticulum)
  – Eventually become replaced by fat cells and connective tissue

• Hyperactive function of the facial nerve may be noted following injury (twitching)
  – Alteration of the distance between nodes of Ranvier
  – Thinner myelin than normal axon
  – New cell body-motor unit arrangement may not correspond with the previous arrangement
  – Spontaneous discharges at the site of injury

• Precise duration of viability remains a matter of debate

A patient has complete facial nerve paralysis HB 6/6 that subsequently recovers to HB 3/6. What is the lowest Seddon Sunderland classification of nerve injury that could have been present?

- First
- Second
- Third
- Fourth
- Fifth
- Sixth
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Development of synkinesis requires at minimum violation of the multiple endoneurial tubes.
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Peitersen’s Landmark Study

• Copenhagen Facial Nerve Study
  • Followed spontaneous course of idiopathic peripheral facial nerve palsy without any treatment
  • 2,570 cases of peripheral facial nerve palsy studied during a period of 25 years
    • 1,701 cases of Bell's palsy

Peitersen’s Landmark Study

Fig. 6. Distribution of time of complete recovery (cumulative) after the onset of paresis.

Peitersen’s Landmark Study

Table VII. Distribution of patients with initial incomplete and complete paresis who make a full recovery from Bell’s palsy

<table>
<thead>
<tr>
<th>Paresis with full recovery</th>
<th>Initial</th>
<th>Final</th>
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<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
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<tr>
<td>Incomplete</td>
<td>512</td>
<td>30</td>
</tr>
<tr>
<td>Complete</td>
<td>1189</td>
<td>70</td>
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Question

• What percent of patients with complete facial nerve paralysis will experience a full recovery?
  – 6%
  – 13%
  – 39%
  – 61%
  – 87%
  – 94%
Question

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Bell’s Palsy - Complications

• Short term
  – Corneal abrasions

• Long term
  – Dysarthria, facial contracture, synkinesis, and the social-psychological challenges of facial asymmetry
  – “The psychological burden of facial paralysis can be tremendous. Facial expression is fundamental to one’s sense of wellbeing and ability to integrate into a social network. With diminished facial movement and marked facial asymmetry, patients with facial paralysis can have impaired interpersonal relationships and experience profound social distress, depression, and social alienation. Recent data show that patients with facial paralysis are perceived by casual observers as emoting negatively compared with individuals without paralyzed faces and are considered significantly less attractive.”
• How does one choose those who will improve versus those who will not improve?
Topognostic Testing

• Abnormalities predict poorer recovery

• Schirmer’s test
  – Sterile filter paper placed into conjunctival fornix of each eye and compares the rate of tear production

• Stapedius reflex
  – Absent reflex or less than ½ contralateral is considered abnormal

• Taste
  – Common for patients to have losses, reducing value of testing

• Salivary flow
  – Canulation of submandibular duct and measurement of flow

• Salivary pH
  – Less than 6.1

Question

What is the sequence of branches from the facial nerve?

- Nerve to stapedius
- Greater superficial petrosal nerve
- Chorda tympani
Question
Electrodiagnostic Testing

• Differentiate class I vs classes II-V
• Short term
  – Useful only if decompression is considered
• Long-term
  – Useful to determine reconstructive options
Electrodiagnostic Testing

- Nerve excitability test
- Maximum stimulation test
- Electroneuronography
- Electromyography

Nerve Excitability Test

• Stimulating electrode over stylomastoid foramen or peripheral branch and return electrode taped to forearm
  – Current is delivered at increasing levels until twitch is noted
  – Lowest current causing a twitch is noted and compared to contralateral side
• Lags at least 3-4 days behind the biologic event but can evolve over 1-2 weeks in Bell’s palsy
  – Difference of 3.5 milliamperes may be used to indicate decompression
  – 150% increase in threshold may be used as an indication for surgery
• No role after complete loss is present and confirmed because clinical improvement precedes testing improvement

Maximum Excitability Test

• Stimuli are used to evaluate facial movement until maximum movement on the unaffected side is achieved

• Same current is applied to the other side of the face and the percentage of facial movement is estimated
  – Estimate of number of intact axons

Electroneuronography

• Facial nerve is stimulated transcutaneously at the stylomastoid foramen
  – Responses to maximal stimulation are measured by evoked compound muscle action potential in the nasolabial groove
• Peak to peak amplitude is averaged and compared
  – There is test-retest variability of up to 20%
• No role for testing in recovery phase

Electromyography

- Recording of spontaneous and voluntary muscle potentials using needles in the muscle
- If voluntary active facial motor units functioning, there is excellent prognosis for spontaneous recovery
  - Spontaneous fibrillation potentials indicate denervation and axonal degeneration
  - Polyphasic potentials suggests reinnervation
  - Electrical silence suggests neuromuscular degeneration without recoverable stimulability or reinnervation
- Role in longstanding paralysis along with muscle biopsy to determine muscle receptivity

## Electrodiagnostic Testing Criteria

<table>
<thead>
<tr>
<th>Test</th>
<th>Criteria to Consider Facial Nerve Decompression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nerve excitability test (NET)</td>
<td>&gt;3.5-mA threshold difference between sides</td>
</tr>
<tr>
<td>Maximal stimulation test (MST)</td>
<td>No response on injured side at maximal stimulation</td>
</tr>
<tr>
<td>Electroneurography (EnoG)</td>
<td>&gt;90% degeneration within 14 days</td>
</tr>
</tbody>
</table>
Prognosis

• ENoG has excellent prognostic qualities concerning Bell’s palsy outcomes
  – Less than or equal to 90% degeneration at 3 weeks had an 80% to 100% chance of good recovery
  – Maximal degeneration of greater than or equal to 95% had a 60% to 70% chance of persistent facial dysfunction

• No strong data comparing multiple testing modalities though ENoG seems to be the favored testing approach
Prognosis

- Prospective observational study
- Patients with acute facial paralysis of House-Brackmann (HB) grade IV or greater diagnosed with Bell’s palsy or Ramsay Hunt Syndrome from August 2007 to July 2011
- After treatment with oral corticosteroid, antiviral agent, and protective eye care, patients were followed up until recovery or 12 months from onset.
- 66 patients with Bell’s palsy
- Logistic regression analysis showed 90% chance of recovery within 6 months, expected with ENoG values of 69.2% degeneration (Bell’s palsy)
- Receiver operating characteristics (ROC) curves showed ENoG values of 82.5% (Bell’s palsy as a critical cutoff value of nonrecovery until 1 year, with the best sensitivity and specificity

Prognosis

• 142 consecutive patients with Bell's palsy treated with steroid plus antiviral agents
• Multivariate analysis was used to identify if Yanagihara grading score and ENoG predict recovery
• Receiver operating characteristic (ROC) curves were constructed for ENoG and grading score
  – Area under the ROC curve for ENoG was broader than those for grading score, indicating that ENoG was superior to grading score in terms of accuracy for prognosis prediction
  – The ROC curve revealed that more than 85% degeneration on ENoG had the best specificity (77.8%) and sensitivity (71.4%) to predict nonrecovery

Bell’s Palsy - Management

Figure 170-1. Facial paralysis: management algorithm.

Question

• What receiver operating characteristic (ROC) curve cutoff for degeneration offers the best sensitivity and specificity for non-recovery?
  – 69.2%
  – 85%
  – 90%
  – 95%
Question

• What receiver operating characteristic (ROC) curve cutoff for degeneration offers the best sensitivity and specificity for non-recovery?
  – 70%
  – 85%
  – 90%
  – 95%

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Takemoto: The ROC curve revealed that more than 85% degeneration on ENoG had the best specificity (77.8%) and sensitivity (71.4%) to predict nonrecovery.
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Strong recommendation
- (a) clinicians should assess the patient using history and physical examination to exclude identifiable causes of facial paresis or paralysis in patients presenting with acute-onset unilateral facial paresis or paralysis
- (b) clinicians should prescribe oral steroids within 72 hours of symptom onset for Bell’s palsy patients 16 years and older
- (c) clinicians should not prescribe oral antiviral therapy alone for patients with new-onset Bell’s palsy
- (d) clinicians should implement eye protection for Bell’s palsy patients with impaired eye closure

Recommendation
- (a) clinicians should not obtain routine laboratory testing in patients with new-onset Bell’s palsy
- (b) clinicians should not routinely perform diagnostic imaging for patients with new-onset Bell’s palsy
- (c) clinicians should not perform electrodiagnostic testing in Bell’s palsy patients with incomplete facial paralysis
- (d) clinicians should reassess or refer to a facial nerve specialist those Bell’s palsy patients with (1) new or worsening neurologic findings at any point, (2) ocular symptoms developing at any point, or (3) incomplete facial recovery 3 months after initial symptom onset

Option
- (a) clinicians may offer oral antiviral therapy in addition to oral steroids within 72 hours of symptom onset for patients with Bell’s palsy
- (b) clinicians may offer electrodiagnostic testing to Bell’s palsy patients with complete facial paralysis

No recommendation
- (a) no recommendation can be made regarding surgical decompression for patients with Bell’s palsy
- (b) no recommendation can be made regarding the effect of acupuncture in patients with Bell’s palsy
- (c) no recommendation can be made regarding the effect of physical therapy in patients with Bell’s palsy
Surgical Decompression

• Retrospective review
  – 91 Bell’s palsy patients

• Facial paralysis patients not treated by surgery, those whose ENoG amplitudes did not degenerate beyond 90% regained normal (HB grade I or II) function

• Patients with greater than or equal to 90% within the first 2 weeks after onset were offered surgical decompression, provided no voluntary motor potentials were recorded with subsequent standard monopolar electromyography (EMG)
  – Surgical intervention was beneficial in restoring facial function (44% vs. 66% return to HB I or II)

Surgical Decompression

- Multicenter prospective trial
- Patients
  - Total paralysis (Bell’s)
  - >90% degeneration on ENOG
  - No voluntary motor unit EMG potentials
  - Within 14 days of onset
- Method
  - Surgical decompression of the facial nerve through a middle cranial fossa: tympanic segment, geniculate ganglion, labyrinthine segment, and meatal foramen

<table>
<thead>
<tr>
<th>H-B Grade</th>
<th>MCF Decompression &lt;14 Days</th>
<th>Steroid Only Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>14</td>
<td>5</td>
</tr>
<tr>
<td>II</td>
<td>17</td>
<td>10</td>
</tr>
<tr>
<td>III</td>
<td>2</td>
<td>19</td>
</tr>
<tr>
<td>IV</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

**Summary:**
- I/II 31 (91%)
- III/IV 3 (9%)

**Total:**
- 34
- 36

H-B = House-Brackmann.
Surgical Decompression

Decompression: Cochrane Review

- Randomized or quasi-randomized controlled trials involving any surgical intervention for Bell's palsy
- Search date: 29 October 2012
- Two trials
  - Total of 69 participants
  - Surgeons in both studies decompressed the nerves of all the surgical group participants using a retroauricular approach
  - The first study showed that the operated group and the non-operated group (who received oral prednisolone) had comparable facial nerve recovery at nine months
    - One operated participant had 20 dB sensorineural hearing loss and persistent vertigo
  - The second study reported no statistically significant differences between the operated and control (no treatment) groups
- There is only very low quality evidence from randomized controlled trials and this is insufficient to decide whether surgical intervention is beneficial or harmful in the management of Bell's palsy.
Surgical Decompression

• “Early enthusiasm for transmastoid decompression of the tympanic and mastoid segments of the facial nerve has waned, and the procedure has been abandoned, because randomized trials showed no benefit and because of evidence that the site of the lesion is in the proximal labyrinthine portion of the facial nerve, which is inaccessible through the mastoid”
Expert Opinion

- Survey
- Members of the American Otological Society and the American Neurotology Society
- Eighty-six neurotologists responded out of 334 surveys (26%)

Fig. 3. Responses to question 5 of the survey. ENoG = electoneurography; AP = action potential; EMG = electromyography.

Physical Therapy

- Assess the efficacy of early physical therapy in association with standard drug administration versus pharmacological therapy only
- June 2008 to May 2010
  - 87 patients were randomly assigned to the experimental group (prednisone and valacyclovir plus physical therapy, n = 39) or the control group (pharmacological therapy, n = 48) within 10 days of onset
  - Intention-to-treat analyses
- No significant differences were found between the study and control groups for outcome of synkinesis

| Table 2. Differences in Proportion of Patients Reaching HB Grade II at the End of the 6-Month Follow-up Between the 2 Treatment Groups A and B, as Calculated by Propensity Score Inverse-Weighting-Adjusted Multivariate Logistic Regression Analyses. a |
|-----------------|-----------------|-----------------|
|                 | Group A          | Group B          | Adjusted P Value |
| Whole study sample | 36/48 (75%)     | 33/39 (86%)     | .27              |
| HB grade V/VI    | 11/23 (48%)     | 17/23 (74%)     | .038             |
| HB grade IV      | 25/25 (100%)    | 16/16 (100%)    | 1.00             |

Abbreviation: HB, House-Brackmann Facial Grading Scale.

a A, pharmacological treatment; B, pharmacological treatment plus physical therapy. P values significant at a level of significance \( \alpha = .05 \) (2-sided) are in bold.

Figure 3. Subgroup of patients with House-Brackmann (HB) grade V or VI at study entry (n = 38): Kaplan-Meier curves showing time to reach primary outcome (ie, HB grade II) according to treatment group. Group A, pharmacological treatment; group B, pharmacological treatment plus physical therapy. *p value by log-rank test.

Acupuncture

• The traditional Chinese theory of acupuncture emphasizes that the intensity of acupuncture must reach a threshold to generate de qi, which is necessary to achieve the best therapeutic effect.
  – “De qi is an internal compound sensation of soreness, tingling, fullness, aching, cool, warmth and heaviness, and a radiating sensation at and around the acupoints”
• Prospective multicentre randomized controlled trial involving patients with Bell palsy
  – Patients were randomly assigned to the de qi (n = 167) or control (n = 171) group
    • Both groups received acupuncture: in the de qi group, the needles were manipulated manually until de qi was reached, whereas in the control group, the needles were inserted without any manipulation
    • All patients received prednisone as a basic treatment
• Outcomes: facial nerve function at month 6, disability and quality of life 6 months after randomization

### Table 1: Baseline characteristics of the included patients

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>De qi group n = 167</th>
<th>Control group n = 171</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No. (%) of patients</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Women</td>
<td>69 (41.3)</td>
<td>76 (44.4)</td>
</tr>
<tr>
<td>Men</td>
<td>98 (58.7)</td>
<td>95 (55.6)</td>
</tr>
<tr>
<td>Age, yr, mean ± SD</td>
<td>38.12 ± 12.76</td>
<td>38.15 ± 12.75</td>
</tr>
<tr>
<td>Interval between onset of palsy and start of treatment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤ 72 h</td>
<td>108 (64.7)</td>
<td>98 (57.3)</td>
</tr>
<tr>
<td>&gt; 72 to ≤ 168 h</td>
<td>59 (35.3)</td>
<td>73 (42.7)</td>
</tr>
<tr>
<td>House–Brackmann score</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>8 (4.8)</td>
<td>10 (5.9)</td>
</tr>
<tr>
<td>3</td>
<td>88 (52.7)</td>
<td>93 (54.4)</td>
</tr>
<tr>
<td>4</td>
<td>54 (32.3)</td>
<td>57 (33.3)</td>
</tr>
<tr>
<td>5</td>
<td>17 (10.2)</td>
<td>11 (6.4)</td>
</tr>
<tr>
<td>Facial Disability Index score, mean ± SD</td>
<td>68.42 ± 14.13</td>
<td>67.84 ± 12.88</td>
</tr>
<tr>
<td>Physical function subscale</td>
<td>65.82 ± 14.82</td>
<td>67.58 ± 12.46</td>
</tr>
<tr>
<td>SocialWell-being subscale</td>
<td>63.17 ± 13.16</td>
<td>63.30 ± 12.29</td>
</tr>
<tr>
<td>Physical domain</td>
<td>63.17 ± 13.16</td>
<td>63.30 ± 12.29</td>
</tr>
<tr>
<td>Psychological domain</td>
<td>59.47 ± 10.98</td>
<td>60.24 ± 10.36</td>
</tr>
<tr>
<td>Social domain</td>
<td>65.87 ± 14.00</td>
<td>67.93 ± 11.82</td>
</tr>
<tr>
<td>Environment domain</td>
<td>60.78 ± 12.21</td>
<td>60.71 ± 12.16</td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Junior middle school</td>
<td>47 (28.1)</td>
<td>49 (28.7)</td>
</tr>
<tr>
<td>Senior middle school</td>
<td>41 (24.6)</td>
<td>41 (24.0)</td>
</tr>
<tr>
<td>Junior college</td>
<td>17 (10.2)</td>
<td>19 (11.1)</td>
</tr>
<tr>
<td>Bachelor</td>
<td>59 (35.3)</td>
<td>58 (33.9)</td>
</tr>
<tr>
<td>Postgraduate</td>
<td>3 (1.8)</td>
<td>4 (2.3)</td>
</tr>
<tr>
<td>Occupation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Civil servant</td>
<td>5 (3.0)</td>
<td>6 (3.5)</td>
</tr>
<tr>
<td>Technician</td>
<td>33 (19.8)</td>
<td>35 (20.5)</td>
</tr>
<tr>
<td>Clerk</td>
<td>27 (16.2)</td>
<td>30 (17.5)</td>
</tr>
<tr>
<td>Service</td>
<td>36 (21.6)</td>
<td>24 (14.0)</td>
</tr>
<tr>
<td>Worker</td>
<td>10 (6.0)</td>
<td>13 (7.6)</td>
</tr>
<tr>
<td>Student</td>
<td>32 (19.2)</td>
<td>33 (19.3)</td>
</tr>
<tr>
<td>Unemployed or retiree</td>
<td>24 (14.4)</td>
<td>30 (17.5)</td>
</tr>
<tr>
<td>Affected side</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td>85 (50.9)</td>
<td>84 (49.1)</td>
</tr>
<tr>
<td>Right</td>
<td>82 (49.1)</td>
<td>87 (50.9)</td>
</tr>
</tbody>
</table>

Note: SD = standard deviation, WHO = World Health Organization.

*Unless stated otherwise.
Used to assess facial-nerve function.

### Table 2: Outcomes among patients in the de qi and control groups at 6 months

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (95% CI)*</th>
<th>Adjusted differences of least squares means (95% CI)*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intention-to-treat analysis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Complete recovery, no. (%)†</td>
<td>150 (89.8)</td>
<td>121 (70.8)</td>
</tr>
<tr>
<td>Facial Disability Index</td>
<td>191.8 (189.3–194.3)</td>
<td>182.0 (179.6–184.5)</td>
</tr>
<tr>
<td>Physical function</td>
<td>98.2 (96.8–99.5)</td>
<td>94.8 (93.5–96.1)</td>
</tr>
<tr>
<td>Social function</td>
<td>93.6 (92.2–95.1)</td>
<td>87.2 (85.8–88.7)</td>
</tr>
<tr>
<td>WHO Quality of Life (brief version) score</td>
<td>340.1 (334.8–345.4)</td>
<td>310.3 (305.0–315.5)</td>
</tr>
<tr>
<td><strong>Per-protocol analysis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Complete recovery, no. (%)†</td>
<td>150 (94.3)</td>
<td>121 (77.1)</td>
</tr>
<tr>
<td>Facial Disability Index</td>
<td>194.8 (193.4–196.1)</td>
<td>185.8 (184.5–187.2)</td>
</tr>
<tr>
<td>Physical function</td>
<td>99.8 (99.3–100.3)</td>
<td>97.0 (96.5–97.5)</td>
</tr>
<tr>
<td>Social function</td>
<td>95.0 (93.8–96.1)</td>
<td>88.9 (87.7–90.0)</td>
</tr>
<tr>
<td>WHO Quality of Life (brief version) score</td>
<td>345.1 (340.5–349.7)</td>
<td>315.0 (310.4–319.6)</td>
</tr>
</tbody>
</table>

Note: CI = confidence interval, OR = odds ratio, WHO = World Health Organization.

*Unless stated otherwise.
†Adjusted for age, sex, treatment centre, interval between onset of palsy and start of treatment, House–Brackmann score on day 1, and baseline score on the corresponding parameters (the scores on Facial Disability Index or its subscales, or the scores on World Health Organization Quality of Life, brief version or its subscales).
| Grade 1 on the House–Brackmann scale. |
| $Adjusted odds ratios (95% confidence intervals); adjusted for age, sex, treatment centre, interval between onset of palsy and start of treatment, and House–Brackmann score on day 1.
Question

• There RCT evidence for benefit of which of the following interventions?
  – Acupuncture
  – Physical therapy
  – Transmastoid decompression
  – Middle cranial fossa decompression
  – 1,2
  – 1,2,4
  – All of the above
Question

- There RCT evidence for benefit of which of the following interventions?
  - Acupuncture
  - Physical therapy
  - Transmastoid decompression
  - Middle cranial fossa decompression
  - 1,2
  - 1,2,4
  - All of the above
• **Strong recommendation**
  – (a) clinicians should assess the patient using history and physical examination to exclude identifiable causes of facial paresis or paralysis in patients presenting with acute-onset unilateral facial paresis or paralysis
  – (b) clinicians should prescribe oral steroids within 72 hours of symptom onset for Bell’s palsy patients 16 years and older
  – (c) clinicians should not prescribe oral antiviral therapy alone for patients with new-onset Bell’s palsy
  – (d) clinicians should implement eye protection for Bell’s palsy patients with impaired eye closure

• **Recommendation**
  – (a) clinicians should not obtain routine laboratory testing in patients with new-onset Bell’s palsy
  – (b) clinicians should not routinely perform diagnostic imaging for patients with new-onset Bell’s palsy
  – (c) clinicians should not perform electrodiagnostic testing in Bell’s palsy patients with incomplete facial paralysis
  – (d) clinicians should reassess or refer to a facial nerve specialist those Bell’s palsy patients with (1) new or worsening neurologic findings at any point, (2) ocular symptoms developing at any point, or (3) incomplete facial recovery 3 months after initial symptom onset

• **Option**
  – (a) clinicians may offer oral antiviral therapy in addition to oral steroids within 72 hours of symptom onset for patients with Bell’s palsy
  – (b) clinicians may offer electrodiagnostic testing to Bell’s palsy patients with complete facial paralysis

• **No recommendation**
  – (a) no recommendation can be made regarding surgical decompression for patients with Bell’s palsy
  – (b) no recommendation can be made regarding the effect of acupuncture in patients with Bell’s palsy
  – (c) no recommendation can be made regarding the effect of physical therapy in patients with Bell’s palsy

Baugh, RF, Basura, GJ, Ishii, LE. Et al. Clinical Practice Guideline: Bell’s Palsy. Otolaryngology Head and Neck Surgery. 149(3S) S1–S27