Bell’s Palsy

Diagnostic and Treatment Considerations

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Department of Otolaryngology
Grand Rounds Presentation
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Outline

- Anatomy
- Pathophysiology
- Diagnostics
- Treatment
- Conclusions

Before Bell’s Palsy

After Bell’s Palsy
Facial Nerve Anatomy

- Contains 7,000-10,000 fibers
- Nuclei
  - Somatic – Motor
  - Taste – Tractus solitarius
  - Secretomotor – Superior salivatory
- Segments
  - Intracranial (cisternal)
  - Meatal
  - Labyrinthine
  - Tympanic
  - Mastoid
  - Extratemporal

(J Neurol Neurosurg Psychiatry 2001;71:149-154)
Facial Nerve Segments

- Intracranial
- Meatal
- Labyrinthine
- Tympanic
- Mastoid
- Extratemporal
Facial Nerve Segments

- Intracranial
- Meatal
- Labyrinthine
- Tympanic
- Mastoid
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(radiopaedia.org)
(info.med.yale.edu)
Facial Nerve Segments

- Intracranial
- Meatal
- Labyrinthine
- Tympanic
- Mastoid
- Extratemporal

- Internal auditory canal (IAC)
- 8mm
- Zero branches

(Lalwani AK, ed. Current Diagnosis and Treatment: Otolaryngology Head and Neck Surgery. 2nd Ed.)
Facial Nerve Segments

- Intracranial
- Meatal
- Labyrinthine
- Tympanic
- Mastoid
- Extratemporal

- IAC to geniculate ganglion
- 3-4mm
- Three branches from geniculate ganglion

(Lalwani AK, ed. Current Diagnosis and Treatment: Otolaryngology Head and Neck Surgery, 2nd Ed.)
Facial Nerve Segments

- Intracranial
- Meatal
- Labyrinthine
- Tympanic
- Mastoid
- Extratemporal

- Geniculate ganglion to pyramidal eminence
- 8-11mm
- Zero branches

(Lalwani AK, ed. Current Diagnosis and Treatment: Otolaryngology Head and Neck Surgery. 2nd Ed.)
Facial Nerve
Segments

- Intracranial
- Meatal
- Labyrinthine
- Tympanic
- Mastoid
- Extratemporal

- Pyramidal eminence to stylomastoid foramen
- 8-14mm
- Three branches
Facial Nerve Segments

- Intracranial
- Meatal
- Labyrinthine
- Tympanic
- Mastoid
- Extratemporal

- Stylomastoid foramen to major branches
- 15-20mm

(www.facialparalysisinstitute.com)
## House-Brackmann Scale

(House 1985)

<table>
<thead>
<tr>
<th>Grade</th>
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<tbody>
<tr>
<td>I</td>
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<tr>
<td>II</td>
<td>slight weakness</td>
<td>moderate to good movement</td>
<td>complete closure minimal</td>
<td>slight asymmetry</td>
</tr>
<tr>
<td></td>
<td>non-disfiguring weakness</td>
<td>slight to moderate movement</td>
<td>complete closure maximal</td>
<td>slight weakness maximal effort</td>
</tr>
<tr>
<td>III</td>
<td>disfiguring weakness</td>
<td>none</td>
<td>incomplete closure</td>
<td>asymmetric with maximal effort</td>
</tr>
<tr>
<td>IV</td>
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<tr>
<td>Grade</td>
<td>Appearance</td>
<td>Synkinesis</td>
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<td>normal</td>
<td>normal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>slight weakness</td>
<td>synkinesis barely noticeable</td>
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<tr>
<td></td>
<td>normal resting tone</td>
<td>contracture or spasm absent</td>
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<td></td>
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<tr>
<td>III</td>
<td>non-disfiguring weakness</td>
<td>obvious but not disfiguring synkinesis</td>
<td></td>
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<tr>
<td></td>
<td>normal resting tone</td>
<td>mass movement or spasm present</td>
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<tr>
<td>IV</td>
<td>disfiguring weakness</td>
<td>severe synkinesis, mass movement, or spasm</td>
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<tr>
<td></td>
<td>normal resting tone</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>V</td>
<td>minimal movement</td>
<td>synkinesis, contracture, and spasm usually absent</td>
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<td></td>
</tr>
<tr>
<td></td>
<td>asymmetric resting tone</td>
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<td></td>
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<td>no synkinesis, contracture, or spasm</td>
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Bell’s Palsy

- Sir Charles Bell first described facial paralysis in 1818
- Acute but limited facial paralysis
  - Rapid onset
  - Few associated symptoms
  - Spontaneous recovery
- Most common diagnosis for facial nerve palsy
- Diagnosis of exclusion
  - Historically thought to be idiopathic
  - Herpes simplex virus (HSV) reactivation

(BMJ 2004; 329(7465):553–557.)
Bell’s Palsy

Demographics

- Incidence of 30 per 100,000
  - Pregnant females (3.3 times greater)
  - Diabetics (4-5 times greater)
- Equal gender distribution in middle age
  - Females, 10-19 years (twice as common)
  - Males, > 40 years (1.5 times greater)
- Equal unilaterality
- Bilateral involvement in less than 1%
- Recurrence rate of 10%
- Positive family history in 10%
Bell’s Palsy
Natural History

- Outcomes of 1011 untreated patients (Peiterson 1982)
- Mean age between 40-44 years
- Less common before 15 years and after 60 years
- No gender predilection
- Recurrence in 6-9%
Bell’s Palsy

Natural History

- Outcomes of 1011 untreated patients (Peiterson 1982)
  - Paresis alone
    - Occurred in 31%
    - Complete recovery in 95%
  - Complete unilateral paralysis in 69%
    - Some recovery by 3 weeks (85%)
    - House-Brackmann 1 in 71%
    - House-Brackmann 2 in 13%
    - House-Brackmann 3-5 in 16%
Bell’s Palsy
Natural History

- Outcomes of 1011 untreated patients (Peiterson 1982)
- Complete recovery by one month in 85%
- Progression to complete degeneration in 15%
  - Signs of recovery after 3-6 months
  - Sequelae associated with longer recovery
    - Diminished function
    - Contracture with movement
    - Tearing
Bell’s Palsy
Associated Symptomatology

- Outcomes of 1011 untreated patients (Peiterson 1982)
- Reduced stapedial reflex
- Postauricular pain
- Dysgeusia
- Decreased lacrimation
- Phonophobia
Bell’s Palsy

Pathophysiology

- Historically thought to be idiopathic
- Two theories
  - Vascular congestion
  - Viral polycranioneuropathy
Pathophysiology
Vascular Congestion

- Autonomic vascular instability (Mcgovern 1955)
- Spasm of nutrient arterioles
  - Secondary ischemia
  - Nerve edema
  - Compression within fallopian canal
- Possible triggers
  - Cold temperature
  - Psychosomatic
Pathophysiology

Infectious

- Acute infectious polyneuritis cerebralis acusticofacialis by Antoni in 1919 (Freidman 2000)
- Facial nerve edema from viral inflammatory response
- HSV proposed etiology in 1972 (McCormick)
Pathophysiology

Infectious

- Burgess (1994)
- Surgita (1995)
- Murakami (1996)
- Furuta (1998)
Pathophysiology

Infectious

- Burgess (1994)
- Surgita (1995)
- Murakami (1996)
- Furuta (1998)

- Patient who died six days after developing Bell’s palsy
- HSV type 1 (HSV-1) DNA in temporal bone section
Infectious

- Burgess (1994)
- Surgita (1995)
- Murakami (1996)
- Furuta (1998)

- Inoculation of mice with HSV-1 DNA
  - Auricle in 104
  - Tongue in 30

- Transient facial paresis
  - Began 6-9 days after inoculation
  - Spontaneous recovery after 3-7 days

- Histopathology
  - Neural edema
  - Inflammatory cell infiltration
  - Vacuolar degeneration

- HSV antigens
  - Beginning 6-20 days after inoculation
  - Facial nerve, geniculate ganglion, and facial nerve nucleus
Pathophysiology

Infectious

- Burgess (1994)
- Surgita (1995)
- Murakami (1996)
- Furuta (1998)

- Transmastoid decompression during active phase of disease
- HSV-1 in endoneural fluid of 11 out of 14 with Bell’s palsy
  - No varicella-zoster virus (VZV)
  - No Epstein Barr
- Ramsay Hunt
  - VZV present
  - No HSV-1
- Trauma or neoplasm
  - No HSV-1
  - No VZV
Pathophysiology

Infectious

- Burgess (1994)
- Surgita (1995)
- Murakami (1996)
- Furuta (1998)

- Polymerase chain reaction of saliva
  - Bell’s palsy in 47
  - Ramsay Hunt in 24
  - Healthy, HSV-positive in 16 (control)

- HSV-1
  - In 50% with Bell’s palsy
  - In 19% of controls

- Testing within 7 days
  - HSV-1 in 40% of Bell’s palsy
  - HSV-1 in 7% of Ramsay Hunt

- HSV-1 usually undetectable by second week
- **McKeever (1987)**
  - Lymphocytic infiltrate
  - Myelin degeneration
  - Most pronounced at labyrinthine segment
- **And perineural edema** (Donoghue 1983; Podvinec 1984)
- **Facial nerve entrapped at meatal foramen** (Fisch 1983)
  - Conductive block at this site (Gantz 1982)
  - Ischemia with increased or prolonged constriction
  - Wallerian degeneration results
    - Axonotmesis
    - Neurotmesis

(Lalwani AK, ed. *Current Diagnosis and Treatment: Otolaryngology Head and Neck Surgery, 2nd Ed.*)
Bell’s Palsy

Diagnostics

- History
- Physical examination
- Radiology
- Topography
- Audiology
- Electrophysiology
Hearing loss or vertigo

Timing
- Sudden onset
- Evolution over 2-3 weeks

Presence of ear disease

Vesicular eruption

Bilateral

Recurrence
Diagnostics

History and Physical Examination

- Hearing loss or vertigo
  - Timing
    - Sudden onset
    - Evolution over 2-3 weeks
  - Presence of ear disease
  - Vesicular eruption
  - Bilateral
  - Recurrence

- Symmetric audiological function
- Absent ipsilateral acoustic reflex
- Bell’s palsy questioned if vertiginous
- Clinical threshold for cerebrovascular accident

Diagnostics

History and Physical Examination

- Hearing loss or vertigo
  - Timing
    - Sudden onset
    - Evolution over 2-3 weeks
  - Presence of ear disease
  - Vesicular eruption
  - Bilateral
  - Recurrence

- Symmetric audiological function
- Absent ipsilateral acoustic reflex
- Bell’s palsy questioned if vertiginous
- Clinical threshold for cerebrovascular accident
Diagnoses

History and Physical Examination

- Hearing loss or vertigo
- **Timing**
  - Sudden onset
  - Evolution over 2-3 weeks
- Presence of ear disease
- Vesicular eruption
- Bilateral
- Recurrence

- Occurs over 24-48 hours
- Can progress to complete paralysis over 1-7 days
- Rule out neoplasm if evolution past 3 weeks
Diagnostics

History and Physical Examination

- Hearing loss or vertigo
- Timing
  - Sudden onset
  - Evolution over 2-3 weeks
- Presence of ear disease
- Vesicular eruption
- Bilateral
- Recurrence

- Chronic otitis media
- Cholesteatoma
Diagnostics

History and Physical Examination

- Hearing loss or vertigo
- Timing
  - Sudden onset
  - Evolution over 2-3 weeks
- Presence of ear disease
- Vesicular eruption
- Bilateral
- Recurrence

- Ramsay-Hunt syndrome
Hearing loss or vertigo

Timing
  - Sudden onset
  - Evolution over 2-3 weeks

Presence of ear disease

Vesicular eruption

Bilateral

Recurrence

- Guillain-Barre syndrome
- Lyme disease
- Intracranial neoplasm
Diagnostics

History and Physical Examination

- Hearing loss or vertigo
- Timing
  - Sudden onset
  - Evolution over 2-3 weeks
- Presence of ear disease
- Vesicular eruption
- Bilateral
- Recurrence

Diagnostics

History and Physical Examination

- Usually excludes Bell’s palsy
- Melkersson-Rosenthal syndrome

(Rev Bras Otorrinolaringol. 2002; 68(5): 755-760)
- Localize lesion
- Computed tomography
  - Trauma
  - Mastoiditis
  - Cholesteatoma
- Magnetic resonance imaging (MRI)
  - Nerve enhancement
  - No correlation with site or degree of enhancement
  - Exclude neoplasm
Diagnostics

Topography

- Schirmer test → greater superficial petrosal
- Stapedial reflex → stapedial branch
- Electrogustometry → chorda tympani
- Salivary flow → chorda tympani
- Unable to predict location or outcome
Diagnostics

Audiology

- Evaluate for pathology of eighth cranial nerve
- Bell’s palsy
  - Symmetric audiological function
  - Absent ipsilateral acoustic reflex
- Retrocochlear pathology
  - Asymmetrical thresholds
  - Acoustic reflex decay
Diagnostics
Electrophysiology

- Provides prognostic information
  - Not used for paresis only
  - Initiated 3 days after progression to complete paralysis

- Tests
  - Nerve excitability test (NET)
  - Maximum stimulation test (MST)
  - Electroneuronography (ENoG)
  - Electromyography (EMG)
Diagnostics

Electrophysiology

- Nerve injury
  - Neuropraxia: conduction block but with axonal continuity
  - Axonotmesis: axoplasmic disruption but endoneural sheath preservation
  - Neurotmesis: disruption of axonal and supportive cells

- Test results
  - Neuropraxia
  - Axonotmesis
  - Neurotmesis
Nerve injury

- Neuropraxia: conduction block but with axonal continuity
- Axonotmesis: axoplasmic disruption but endoneural sheath preservation
- Neurotmesis: disruption of axonal and supportive cells

Test results

- Neuropraxia
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Nerve injury
- Neuropraxia: conduction block but with axonal continuity
- Axonotmesis: axoplasmic disruption but endoneural sheath preservation
- Neurotmesis: disruption of axonal and supportive cells

Test results
- Neuropraxia
- Axonotmesis
- Neurotmesis
  - NET, MST, and ENoG normal
  - No voluntary motor action potentials on EMG
Nerve injury
- Neuropraxia: conduction block but with axonal continuity
- Axonotmesis: axoplasmic disruption but endoneural sheath preservation
- Neurotmesis: disruption of axonal and supportive cells

Test results
- Neuropraxia
- Axonotmesis
- Neurotmesis
  - NET, MST, and ENoG with rapid and complete degeneration
  - EMG
    - No voluntary motor action potentials
    - Myogenic fibrillation potentials after 10-14 days
Nerve injury
- Neuropraxia: conduction block but with axonal continuity
- Axonotmesis: axoplasmic disruption but endoneural sheath preservation
- Neurotmesis: disruption of axonal and supportive cells

Test results
- Neuropraxia
- Axonotmesis
- Neurotmesis
  - Similar results as axonotmesis
  - Less predictable outcome
  - Cannot differentiate between the two
Described by Hilger in 1964

Compare thresholds for minimal muscle contraction
- Normal side
- Paralyzed side

Difference of 3.5mA
- Severe degeneration
- Higher likelihood of poorer outcome

Inaccurate within first 3 days of Bell’s palsy onset

Subjective comparison
Electrophysiology

Maximum Stimulation Test

- Compare facial movement with maximum stimulation
- Greater degree of weakness with worsening degeneration
- Inaccurate within first 3 days of Bell’s palsy onset
- Subjective comparison
Electrophysiology

Electroneuronography

- Compares compound action potential of both sides
  - Stimulate nerve at stylomatoid foramen
  - Measure muscular response near nasolabial groove
- Less intact motor axons with Wallerian degeneration
- Worse prognosis with rapid degeneration
- Inaccurate within first 3 days of Bell’s palsy onset
- Quantitative analysis, observer independent

(Am J Otol 1992; 13:127–133.)
Electrophysiology

Electroneuronography

- **Esslen (1977)**
  - Full recovery in 88% if < 90% degeneration
  - Full recovery in 30% if 90-95% degeneration
  - No full recovery if 100% degeneration

- **Fisch (1981)**
  - Satisfactory spontaneous recovery if < 90% degeneration within 3 weeks of onset
  - High likelihood of 95% degeneration if reach 90% degeneration
  - Permanent unsatisfactory result in 50% with 95-100% degeneration within 2 weeks of onset
Electrophysiology

Electromyography

- Measure action potentials with volitional movement
- Silence
  - Resting state
  - Muscle atrophy or fibrosis
  - Early acute paralysis
- Diphasic or triphasic with normal contraction
- Fibrillation indicates degeneration
- Polyphasic indicates reinnervation
Electrophysiology

Electromyography

- Quantitative analysis, observer independent
- Complementary test with ENoG
  - Regenerating nerve fibers do not complete a summation potential on ENoG
  - Degeneration if myogenic fibrillation potentials but no voluntary motor units on EMG
  - Regeneration if both defibrillation potentials and motor units on EMG
- No fibrillation potentials until 10-14 days after onset
- Unable to distinguish between total neuropraxic injury and regenerating nerve in acute phase
Treatment

- Observation
  - Monitor progression
  - Eye care

- Medical
  - Steroids
  - Antivirals

- Surgical decompression
**Treatment**

**Steroids Beneficial**

- Typically start prednisone 1mg/kg/d up to 70-80mg
  - Usually taper after 5-7 days
  - May extend therapy if no improvement

- **Some benefit with steroids** (Adour 1972; Katusic 1986)
  - If combined with antivirals (de Almeida 2009)
  - Optimal effect with early intervention (Brown 1982; Williamson 1996)
  - Prednisolone within 24 hours (Shafshak 1994)

- **Prednisone** (Austin 1993)
  - Randomized, double-blind, placebo-controlled
  - Improved recovery with prednisone
  - Statistically insignificant trend for denervation prevention
Ramsey (2000)  
- Meta-analysis of 27 prospective and 20 retrospective trials  
  - Three met inclusion criteria (1975-1994)  
  - Prospective, controlled trials  
  - Prednisone (≥400mg) started within 7 days of onset  
  - Steroids improved complete recovery by 17%  
- Generally positive benefit from excluded trials  
  - Complete recovery 49-97% with steroids  
  - Complete recovery 23-64% without

Cochrane Review: steroids increase frequency of complete recovery (Salinas, 2010)
Treatment

Steroids **Not** Beneficial

- No evidence of benefit  (May 1976; Stankiewitz 1987)
- Literature review  (Grogan 2001)
  - Nine studies compared steroids to placebo (1954-1999)
    - No difference in recovery or synkinesis
    - Most studies underpowered
    - Beneficial trend in some studies
  - *Probable* benefit with steroids
Treatment

Steroids **Not Beneficial**

- No benefit in children  (Prescott 1987)
- Pediatric literature review  (Salman 2001)
  - Eight trials and one review (1966-1998)
    - Five randomized
    - Prednisone or corticotropin
  - Only one exclusively studied children
  - Benefit reported in four trials
  - No statistical sub-analysis in all trials
  - Heterogeneity precluded meta-analysis or recommendation
Treatment
Antivirals

- Prednisone & acyclovir (Adour 1996)
  - Double-blind with prednisone and acyclovir or placebo
  - Therapy within 3 days of onset
  - Prednisone & acyclovir
    - Less facial weakness on MST
    - Less unsatisfactory recovery

- Prednisone alone better than acyclovir alone (De Diego 1998)

- Literature review (Grogan 2001)
  - Acyclovir vs prednisone; acyclovir & prednisone vs prednisone
  - Lack of studies to establish benefit
  - Possible benefit with adding acyclovir to prednisone
Treatment

Antivirals

- Prednisone & valacyclovir vs no treatment \( (Axelsson\ 2003)\)
  - Improved complete recovery (87.5% vs 68%)
  - Less House-Brackmann IV or worse (1.8% vs 18%)
  - Complete recovery in >60 years (100% vs 42%)

- Prednisolone & valacyclovir vs placebo \( (Hato\ 2007)\)
  - Prospective, randomized placebo-controlled
    - Six academic tertiary care centers
    - 222 patients
  - Improved recovery rate with valacyclovir (96.5% vs 89.7%)

- Cochrane Review \( (Lockhart\ 2009)\)
  - Antivirals plus steroids beneficial over placebo alone
  - Antivirals alone not beneficial over steroids or placebo alone
Treatment

Surgical

- First described in 1932 by Balance & Duel
  - Stylomastoid foramen in 1930’s
  - Tympanic segment in 1960’s
- Decompression beneficial  
  [Giancarlo 1970]
- No benefit with decompression from geniculate ganglion to stylomastoid foramen  
  [McNeill 1974]
- Transmastoid
  - Decompression may be beneficial  
    [May 1979]
    - From geniculate to labyrinthine segment
    - Meatal foramen was not decompressed
  - No benefit from transmastoid approach within 14 days  
    [May 1984]
  - No benefit with decompressing mastoid segment alone  
    [May 1985]
Treatment

Surgical

- Fisch (1972)
  - Total nerve decompression via middle cranial fossa and transmastoid approach
  - Conduction block proximal to geniculate ganglion

- ENoG with 90% degeneration
  - Decompress meatal foramen within 3 weeks (Fisch 1981)
  - Decompression within 2 weeks (Gantz 1999)
    - Steroids if ENoG with <90% degeneration, no antivirals
    - Decompress if ENoG with >90% degeneration & no EMG activity by 2 weeks
**Treatment**

**Surgical (Gantz 1999)**

- Multicenter study, surgery vs steroids
- Middle cranial fossa
  - Decompress internal auditory canal through tympanic segment
  - Surgical control if decompress *after* 2 weeks of paralysis
- Improved outcomes if decompress within 2 weeks
  - House-Brackmann recovery I/II (91% vs 42% steroids) by 7 months
  - House-Brackmann recovery III/IV (9% vs 58% steroids) by 7 months
  - Similar results between surgical control and steroid groups
- House-Brackmann recovery I/II in all with ENoG <90% degeneration
Treatment Algorithm

**Acute paresis**

- Days 0–14: Prednisone + valacyclovir
  - Follow-up 5 days
  - Paresis: Follow-up 1 month
  - Paralysis: ENoG Follow paralysis protocol

- Days >14: Observation follow-up 6 months

**Acute paralysis**

- Days 0–3: Prednisone + Valacyclovir
  - Follow-up 3 days
  - <90% Degeneration: Prednisone + valacyclovir
    - Follow-up depends on ENoG up to 14 days
  - >90% Degeneration: Recommend MCF decompression

- Days 3–14

- Days >14: Follow-up 6 months

Prednisone = 1 mg/kg x 7 days, Valacyclovir = 500 mg tid x 10 days

(Brackmann 2010)
Conclusion

- Most common diagnosis of facial paralysis
- Diagnosis of exclusion
- Prognostic information with electrophysiology
- Medical therapy
  - Steroids
  - Antivirals
- Surgical decompression
  - ENoG with >90% degeneration
  - No voluntary EMG activity within 14 days of paralysis
References


References


