

Clinical Practice Guideline: Bell's Palsy

Reginald F. Baugh, MD¹, Gregory J. Basura, MD, PhD²,
Lisa E. Ishii, MD, MHS³, Seth R. Schwartz, MD, MPH⁴,
Caitlin Murray Drumheller⁵, Rebecca Burkholder, JD⁶,
Nathan A. Deckard, MD⁷, Cindy Dawson, MSN, RN⁸,
Colin Driscoll, MD⁹, M. Boyd Gillespie, MD, MSc¹⁰,
Richard K. Gurgel, MD¹¹, John Halperin, MD¹²,
Ayesha N. Khalid, MD^{13,14}, Kaparaboyna Ashok
Kumar, MD, FRCS¹⁵, Alan Micco, MD¹⁶,
Debra Munsell, DHSc, PA-C¹⁷,
Steven Rosenbaum, MD¹⁸, and
William Vaughan¹⁹

Otolaryngology-
Head and Neck Surgery
149(3S) S1-S27
© American Academy of
Otolaryngology-Head and Neck
Surgery Foundation 2013
Reprints and permission:
sagepub.com/journalsPermissions.nav
DOI: 10.1177/0194599813505967
<http://otojournal.org>



Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

Abstract

Objective. Bell's palsy, named after the Scottish anatomist, Sir Charles Bell, is the most common acute mono-neuropathy, or disorder affecting a single nerve, and is the most common diagnosis associated with facial nerve weakness/paralysis. Bell's palsy is a rapid unilateral facial nerve paresis (weakness) or paralysis (complete loss of movement) of unknown cause. The condition leads to the partial or complete inability to voluntarily move facial muscles on the affected side of the face. Although typically self-limited, the facial paresis/paralysis that occurs in Bell's palsy may cause significant temporary oral incompetence and an inability to close the eyelid, leading to potential eye injury. Additional long-term poor outcomes do occur and can be devastating to the patient. Treatments are generally designed to improve facial function and facilitate recovery. There are myriad treatment options for Bell's palsy, and some controversy exists regarding the effectiveness of several of these options, and there are consequent variations in care. In addition, numerous diagnostic tests available are used in the evaluation of patients with Bell's palsy. Many of these tests are of questionable benefit in Bell's palsy. Furthermore, while patients with Bell's palsy enter the health care system with facial paresis/paralysis as a primary complaint, not all patients with facial paresis/paralysis have Bell's palsy. It is a concern that patients with alternative underlying etiologies may be misdiagnosed or have unnecessary delay in diagnosis. All of these quality concerns provide an important opportunity for improvement in the diagnosis and management of patients with Bell's palsy.

Purpose. The primary purpose of this guideline is to improve the accuracy of diagnosis for Bell's palsy, to improve the quality of care and outcomes for patients with Bell's palsy, and to decrease harmful variations in the evaluation and management of Bell's palsy. This guideline addresses these needs by encouraging accurate and efficient diagnosis and treatment and, when applicable, facilitating patient follow-up to address the management of long-term sequelae or evaluation of new or worsening symptoms not indicative of Bell's palsy. The guideline is intended for all clinicians in any setting who are likely to diagnose and manage patients with Bell's palsy. The target population is inclusive of both adults and children presenting with Bell's palsy.

Action Statements. The development group made a *strong recommendation* that (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, and (d) clinicians should implement eye protection for Bell's palsy patients with impaired eye closure. The panel made *recommendations* that (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, and (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. The development group provided the following *options*: (a) clinicians may offer oral antiviral therapy in addition to oral steroids within 72 hours of symptom onset for patients with Bell's palsy, and (b) clinicians may offer electrodiagnostic testing to Bell's palsy patients with complete facial paralysis. The development group offered the following *no recommendations*: (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, and (c) no recommendation can be made regarding the effect of physical therapy in patients with Bell's palsy.

Keywords

Bell's palsy, facial nerve disorder, facial nerve pathophysiology, idiopathic facial nerve paralysis, idiopathic facial nerve paresis, otolaryngology

Received July 16, 2013; revised August 26, 2013; accepted August 30, 2013.

Introduction

Bell's palsy, named after the Scottish anatomist, Sir Charles Bell, is the most common acute mono-neuropathy, or disorder affecting a single nerve, and is the most common diagnosis associated with facial nerve weakness/paralysis.¹ Bell's palsy is a rapid unilateral facial nerve paresis (weakness) or paralysis (complete loss of movement) of unknown cause. The condition leads to the partial or complete inability to voluntarily move facial muscles on the affected side of the face. Although typically self-limited, the facial paresis/paralysis that occurs in Bell's palsy may cause significant temporary oral incompetence and an inability to close the eyelid, leading to potential eye injury. Additional long-term poor outcomes do occur and can be devastating to the patient. Treatments are generally designed to improve facial function and facilitate recovery.

The myriad treatment options for Bell's palsy include medical therapy (steroids and antivirals, alone and in combination),²⁻⁴ surgical decompression,⁵⁻⁸ and complementary and alternative therapies such as acupuncture. Some controversy exists regarding the effectiveness of several of these options, and there are consequent variations in care. In addition,

numerous diagnostic tests available are used in the evaluation of patients with Bell's palsy. Many of these tests are of questionable benefit in Bell's palsy, including laboratory testing,^{9,10} diagnostic imaging studies, and electrodiagnostic tests.¹⁰⁻¹² Furthermore, while patients with Bell's palsy enter the health care system with facial paresis/paralysis as a primary complaint, not all patients with facial paresis/paralysis have Bell's palsy. It is a concern that patients with alternative underlying etiologies may be misdiagnosed or have unnecessary delay in diagnosis. All of these quality concerns provide an important opportunity for improvement in the diagnosis and management of patients with Bell's palsy.

When evaluating a patient with facial weakness/paralysis for Bell's palsy, the following should be considered:

- Bell's palsy is rapid in onset (<72 hours).
- Bell's palsy is diagnosed when no other medical etiology is identified as a cause of the facial weakness.
- Bilateral Bell's palsy is rare.^{13,14}
- Currently, no cause for Bell's palsy has been identified.
- Other conditions may cause facial paralysis, including stroke, brain tumors, tumors of the parotid gland or infratemporal fossa, cancer involving the facial nerve, and systemic and infectious diseases, including zoster, sarcoidosis, and Lyme disease.^{1,15-17}
- Bell's palsy is typically self-limited.
- Bell's palsy may occur in men, women, and children but is more common in those 15 to 45 years old; those with diabetes, upper respiratory ailments, or compromised immune systems; or during pregnancy.^{1,6,18}

The guideline development group (GDG) recognizes that Bell's palsy is a diagnosis of exclusion requiring the careful elimination of other causes of facial paresis or paralysis. Although the literature is silent on the precise definition of what constitutes acute onset in facial paralysis, the GDG accepted the definition of "acute" or "rapid onset" to mean that the occurrence of paresis/paralysis typically progresses to its maximum severity within 72 hours of onset of the paresis/paralysis (**Table 1**). This guideline

¹University of Toledo Medical Center, Toledo, Ohio, USA; ²University of Michigan, Ann Arbor, Michigan, USA; ³Johns Hopkins University, Baltimore, Maryland, USA; ⁴Virginia Mason Medical Center, Seattle, Washington, USA; ⁵Department of Research and Quality Improvement, American Academy of Otolaryngology—Head and Neck Surgery Foundation, Alexandria, Virginia, USA; ⁶National Consumers League, Washington, DC, USA; ⁷Cooper University, Camden, New Jersey, USA; ⁸University of Iowa, Iowa City, Iowa, USA; ⁹Mayo Clinic, Rochester, Minnesota, USA; ¹⁰Medical University of South Carolina, Charleston, South Carolina, USA; ¹¹University of Utah, Salt Lake City, Utah, USA; ¹²Overlook Medical Center, Summit, New Jersey, USA; ¹³Emerson Hospital, Concord, Massachusetts, USA; ¹⁴Harvard Medical School, Boston, Massachusetts, USA; ¹⁵University of Texas Health Science Center, San Antonio, Texas, USA; ¹⁶Northwestern University Feinberg School of Medicine, Chicago, Illinois, USA; ¹⁷Louisiana State University Health Sciences Center New Orleans, New Orleans, Louisiana, USA; ¹⁸HackensackUMC Mountainside Hospital, Montclair, NJ; ¹⁹National Committee to Preserve Social Security and Medicare, Falls Church, Virginia, USA.

Corresponding Author:

Reginald F. Baugh, MD, Division of Otolaryngology, University of Toledo Medical Center, 3000 Arlington Ave, Toledo, OH 43614, USA.
Email: reginald.baugh@utoledo.edu

Table 1. Abbreviations and definitions of common terms.

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

does not focus on facial paresis/paralysis due to neoplasms, trauma, congenital or syndromic problems, specific infectious agents, or postsurgical facial paresis or paralysis, nor does it address recurrent facial paresis/paralysis. For the purposes of this guideline, Bell's palsy is defined as follows: acute unilateral facial nerve paresis or paralysis with onset in less than 72 hours and without an identifiable cause (**Table 1**).

Literature cited throughout this guideline often uses the House-Brackmann facial nerve grading scale. This commonly used scale, designed to systematically quantify facial nerve functional recovery after surgery that puts the facial nerve at risk, has been used to assess recovery after trauma to the facial nerve or Bell's palsy.¹⁹ It was not designed to assess initial facial nerve paresis or paralysis of Bell's palsy. The House-Brackmann facial nerve grading system is described in **Table 2**.²⁰

While a viral etiology is suspected, the exact mechanism of Bell's palsy is currently unknown.²¹ 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. The facial nerve carries nerve impulses to muscles of the face and also to the lacrimal glands, salivary glands, stapedius muscle, taste fibers from the anterior tongue, and general sensory fibers from the tympanic membrane and posterior ear canal. Accordingly, patients with Bell's palsy may experience dryness of the eye or mouth, taste disturbance or loss, hyperacusis, and sagging of the eyelid or corner of the mouth.^{13,18} Ipsilateral pain around the ear or face is not an infrequent presenting symptom.^{21,22}

Numerous diagnostic tests have been used to evaluate patients with acute facial paresis/paralysis for identifiable causes or aid in predicting long-term outcomes. Many of these tests were considered in the development of this guideline, including the following:

- Imaging: computed tomography (CT) or magnetic resonance imaging (MRI) to identify infection,

inflammation, tumor, fractures, or other potential causes for facial nerve involvement

- Electrodiagnostic testing to stimulate the facial nerve to assess the level of facial nerve insult
- Serologic studies to test for infectious causes
- Hearing testing to determine if the cochlear nerve or inner ear has been affected
- Vestibular testing to determine if the vestibular nerve is involved
- Schirmer tear testing to measure the eye's ability to produce tears

Most patients with Bell's palsy show some recovery without intervention within 2 to 3 weeks after onset of symptoms and completely recover within 3 to 4 months.¹ Moreover, even without treatment, facial function is completely restored in approximately 70% of Bell's palsy patients with complete paralysis within 6 months and as high as 94% of patients with incomplete paralysis; accordingly, as many as 30% of patients do not recover completely.²³ Given the dramatic effect of facial paralysis on patient appearance, quality of life, and psychological well-being, treatment is often initiated in an attempt to decrease the likelihood of incomplete recovery. Corticosteroids and antiviral medications are the most commonly used medical therapies. New trials have explored the benefit of these medications. The benefit of surgical decompression of the facial nerve remains relatively controversial.²⁴

There are both short- and long-term sequelae of Bell's palsy, including an inability to close the eye, drying and corneal ulceration of the eye, and vision loss. These can be prevented with appropriate eye care. The short-term sequelae, such as inability to close the eye and drying of the eye, warrant careful management, but treatment results can be favorable. Long term, the disfigurement of the face due to incomplete recovery of the facial nerve can have devastating effects on psychological well-being and quality of life. With diminished facial movement and marked facial asymmetry, patients with facial paralysis can have impaired interpersonal

Table 2. House-Brackmann facial nerve grading system.¹⁹

Grade	Defined by	
1	Normal	Normal facial function in all areas.
2	Mild dysfunction	Slight weakness noticeable only on close inspection. At rest: normal symmetry of forehead, ability to close eye with minimal effort and slight asymmetry, ability to move corners of mouth with maximal effort and slight asymmetry. No synkinesis, contracture, or hemifacial spasm.
3	Moderate dysfunction	Obvious, but not disfiguring difference between 2 sides, no functional impairment; noticeable but not severe synkinesis, contracture, and/or hemifacial spasm. At rest: normal symmetry and tone. Motion: slight to no movement of forehead, ability to close eye with maximal effort and obvious asymmetry, ability to move corners of mouth with maximal effort and obvious asymmetry. Patients who have obvious but no disfiguring synkinesis, contracture, and/or hemifacial spasm are grade III regardless of degree of motor activity.
4	Moderately severe dysfunction	Obvious weakness and/or disfiguring asymmetry. At rest: normal symmetry and tone. Motion: no movement of forehead; inability to close eye completely with maximal effort. Patients with synkinesis, mass action, and/or hemifacial spasm severe enough to interfere with function are grade IV regardless of motor activity.
5	Severe dysfunction	Only barely perceptible motion. At rest: possible asymmetry with droop of corner of mouth and decreased or absence of nasal labial fold. Motion: no movement of forehead, incomplete closure of eye and only slight movement of lid with maximal effort, slight movement of corner of mouth. Synkinesis, contracture, and hemifacial spasm usually absent.
6	Total paralysis	Loss of tone; asymmetry; no motion; no synkinesis, contracture, or hemifacial spasm.

relationships and may experience profound social distress, depression, and social alienation.²⁵ There are a number of rehabilitative procedures to normalize facial appearance, including eyelid weights or springs, muscle transfers and nerve substitutions, static and dynamic facial slings, and botulinum toxin injections to eliminate facial spasm/synkinesis.²⁶⁻³⁰ This guideline, however, focuses more on the acute management of Bell's palsy and will not address these interventions in detail.

Guideline Purpose

The primary purpose of this guideline is to improve the accuracy of diagnosis for Bell's palsy, to improve the quality of care and outcomes for patients with Bell's palsy, and to decrease harmful variations in the evaluation and management of Bell's palsy. This guideline addresses these needs by encouraging accurate and efficient diagnosis and treatment and, when applicable, facilitating patient follow-up to address the management of long-term sequelae or evaluation of new or worsening symptoms not indicative of Bell's palsy. The guideline is intended for all clinicians in any setting who are likely to diagnose and manage patients with Bell's palsy. The target population is inclusive of both adults and children presenting with Bell's palsy.

This guideline is intended to focus on a limited number of quality improvement opportunities deemed most important by the GDG and is not intended to be a comprehensive

guide for diagnosing and managing Bell's palsy. A comprehensive list of the topics and issues considered by the GDG is available in **Table 3**. The recommendations outlined in this guideline are not intended to represent the standard of care for patient management, nor are the recommendations intended to limit treatment or care provided to individual patients. The guideline is not intended to replace clinical judgment for individualized patient care. Our goal is to create a multidisciplinary guideline with a specific set of focused recommendations based on an established and transparent process that considers levels of evidence, harm-benefit balance, and expert consensus to resolve gaps in evidence. These specific recommendations are designed to improve quality of care and may be used to develop performance measures.

Health Care Burden

Bell's palsy is a relatively uncommon condition, but one that affects people across the age and sex spectrum, with incidence ranging from 11.5 to 53.3 per 100,000 person years in different populations.³¹⁻³⁵ Notably, Bell's palsy is seen in the pediatric population, with 1 study citing an incidence of approximately 6.1 in 100,000 in children 1 to 15 years of age.³⁶ In 1 integrated health system, the incidence of Bell's palsy in children 18 years or younger was 18.8 per 100,000 person years in a 5-year study.³⁷ In that study, the incidence rate increased by age and was higher in females than in

Table 3. Topics and issues considered in Bell's palsy guideline development.^a

Alternative/Complementary Medicine	Pain Management
Combination therapy vs monotherapy	Patient presentation
Comorbidities	Patient referral
Dental hygiene	Patient support
Differential diagnosis	Physical therapy
Electrodiagnostic testing	Physiologic testing
Eye care	Predictors of return of nerve function
House-Brackmann scale	Prognostic indicators
Hyperbaric oxygen therapy	Reconstructive options
Imaging	Steroid/antiviral therapy
Management of synkinesis	Surgical decompression

^aThis list was created by the guideline development group to refine content and prioritize action statements; not all items listed were ultimately included or discussed in the guideline.

males across all age strata. Although Bell's palsy is seen in patients across a large age spectrum, the incidence was noted to be highest in the 15- to 45-year-old age group.¹

There are several known risk factors for Bell's palsy, including pregnancy. In a study of pregnant women, of 242,000 deliveries, 0.17% of expectant mothers were diagnosed with Bell's palsy.³⁸ Obesity, chronic hypertension, and severe preeclampsia also increase the risk. Diabetes is also a risk factor, and hypertension may be independently associated with an increased risk of Bell's palsy.³⁹ Risk factors for Bell's palsy include the following:

- Pregnancy
- Severe preeclampsia
- Obesity
- Hypertension and chronic hypertension
- Diabetes
- Upper respiratory ailments

The psychological burden of facial paralysis can be tremendous. Facial expression is fundamental to one's sense of well-being and ability to integrate into a social network.⁴⁰ 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.²⁵ 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.⁴¹ There are links between diminished attractiveness and depression, and these data may suggest that patients with paralyzed faces are at risk for depression, which can lead to decreased productivity and increased health care expenses.

Costs associated with Bell's palsy include those attributed to visits to the emergency room or urgent care, primary

care, and laboratory and imaging studies. The evaluations described are myriad and may include audiometric, vestibular, electrical, and serologic tests, as well as CT imaging or MRI studies. A "gold standard" treatment has yet to be defined, and as such, medical and surgical intervention is variable. No explicit cost estimates for the diagnosis and management of Bell's palsy are available, but with 35,000 to 100,000 cases annually in the United States, based on aforementioned estimates of incidence, the cost of addressing Bell's palsy is undoubtedly significant.

Methods

This guideline was developed using an explicit and transparent a priori protocol for creating actionable statements based on supporting evidence and the associated balance of benefit and harm.⁴² The GDG followed the protocol through all stages of the development process. The GDG consisted of 17 members representing otolaryngology-head and neck surgery, neurology, facial plastic and reconstructive surgery, neurotology, emergency medicine, primary care, otology, nursing, physician assistants, and consumer advocacy.

Literature Search

All literature searches were performed by an information specialist through July 2012. Two initial searches were performed to identify clinical practice guidelines, systematic reviews, and randomized controlled trials (RCTs). The searches were performed in multiple databases, including the National Guidelines Clearinghouse (NGC) (www.guideline.gov), The Cochrane Library (Cochrane Database of Systematic Reviews, DARE, HTA Database, NHS EED), the Cumulative Index to Nursing and Allied Health Literature (CINAHL), EMBASE, PubMed, CMA Infobase, NHS Evidence ENT and Audiology, National Library of Guidelines, National Institute of Clinical Excellence (NICE), Scottish Intercollegiate Guidelines Network (SIGN), New Zealand Guidelines Group (NZGG), Australian National Health and Medical Research Council (ANHMRC), the Guidelines International Network (G-I-N), Allied and Complementary Medicine (AMED), Agency for Healthcare Research and Quality (AHRQ), Health Services/Technology Assessment Texts (HSTAT), and the TRIP database.

1. Clinical practice guidelines were identified by a National Guideline Clearinghouse, CMA Infobase, NHS Evidence ENT & Audiology, National Library of Guidelines, NICE, SIGN, NZGG, ANHMRC, TRIP database, G-I-N, and PubMed search using *guideline* as a publication type or title word. The search identified 1 guideline after removing duplicates, clearly irrelevant references, and non-English-language articles.
2. Systematic reviews were identified through NHS Evidence ENT & Audiology, Cochrane Library (Cochrane Database of Systematic Reviews, DARE, HTA Database, NHS EED), PubMed, EMBASE, CINAHL, AMED, AHRQ, HSTAT, and the TRIP

database. The final data set included 30 systematic reviews or meta-analyses that were distributed to the GDG members. Articles were excluded if they were not available in English and did not meet the GDG's quality criteria (ie, the review had a clear objective and method, an explicit search strategy, and a valid method of data extraction).

3. RCTs were identified through MEDLINE, EMBASE, CINAHL, and CENTRAL and totaled 49 trials.

The following search parameters were used for both literature searches:

- Scope: Acute onset of facial nerve paresis or paralysis (Bell's palsy)
- Population: Adults, children
- Exclusions: None
- Keywords: Bell's palsy, Bell palsy, acute facial paralysis, unilateral facial nerve paralysis/palsy, acute facial nerve paralysis/palsy, idiopathic facial nerve paralysis/palsy, bilateral facial nerve paralysis/palsy, recurrent facial nerve paralysis/palsy, acute facial paralysis and steroid use, acute facial paralysis, facial paresis and antiviral use, surgical management of Bell's palsy, surgical management of acute facial nerve paralysis, facial paralysis, and pregnancy

Results of all literature searches were distributed to GDG members, including electronic listings with abstracts (if available) of the searches for clinical guidelines, RCTs, systematic reviews, and other studies. This material was supplemented, as needed, with targeted searches to address specific needs identified in writing the guideline through February 2013.

In a series of conference calls, the working group defined the scope and objectives of the proposed guideline. During the 10 months devoted to guideline development ending in February 2013, the GDG met twice, with in-person meetings following the format previously described, using electronic decision-support (BRIDGE-Wiz, Yale Center for Medical Informatics, CT) software to facilitate creating actionable recommendations and evidence profiles.⁴² Internal electronic review and feedback on each guideline draft were used to ensure accuracy of content and consistency with standardized criteria for reporting clinical practice guidelines.⁴³

American Academy of Otolaryngology—Head and Neck Surgery Foundation (AAO-HNSF) staff used the Guideline Implementability Appraisal and Extractor (GLIA) to appraise adherence of the draft guideline to methodological standards, to improve clarity of recommendations, and to predict potential obstacles to implementation.⁴⁴ The GDG members received summary appraisals in February 2013 and modified an advanced draft of the guideline.

The final guideline draft underwent extensive external peer review. Comments were compiled and reviewed by the chair of the GDG, and a modified version of the guideline was distributed and approved by the full GDG. The recommendations contained in the guideline are based on the best available data published through February 2013. Where data were lacking, a combination of clinical experience and expert consensus was used. A scheduled review process will occur at 5 years from publication or sooner if new compelling evidence warrants earlier consideration.

Classification of Evidence-Based Statements

Guidelines are intended to produce optimal health outcomes for patients, to minimize harms, and to reduce inappropriate variations in clinical care. The evidence-based approach to guideline development requires the evidence supporting a policy be identified, appraised, and summarized and that an explicit link between evidence and statements be defined. Evidence-based statements reflect both the quality of evidence and the balance of benefit and harm that is anticipated when the statement is followed. The definitions for evidence-based statements are listed in **Table 4**^{45,46} and **Table 5**.

Guidelines are not intended to supersede professional judgment but rather may be viewed as a relative constraint on individual clinician discretion in a particular clinical circumstance. Less frequent variation in practice is expected for a "strong recommendation" than might be expected with a "recommendation." "Options" offer the most opportunity for practice variability.⁴⁶ Clinicians should always act and decide in a way that they believe will best serve their patients' interests and needs, regardless of guideline recommendations. Clinicians must also operate within their scope of practice and according to their training. Guidelines represent the best judgment of a team of experienced clinicians and methodologists addressing the scientific evidence for a particular topic.

Making recommendations about health practices involves value judgments on the desirability of various outcomes associated with management options. Values applied by the GDG sought to minimize harm and diminish unnecessary and inappropriate therapy. A significant goal of the GDG was to be transparent and explicit about how values were applied and to document the process.

Financial Disclosure and Conflicts of Interest

The cost of developing this guideline, including travel expenses of all GDG members, was covered in full by the AAO-HNSF. While the AAO-HNSF sponsored the guideline development process and facilitated development through staff support and travel expenses, it did not directly influence the scope or content of the guideline. Potential conflicts of interest for all GDG members in the past 2 years were compiled and distributed before the first conference call. After review and discussion of these disclosures,⁴⁷ the GDG concluded that individuals with potential conflicts could remain on the GDG if they (1) reminded the GDG of potential conflicts before any related discussion, (2) recused

Table 4. Guideline definitions for evidence-based statements.

Statement	Definition	Implication
Strong recommendation	A strong recommendation means the benefits of the recommended approach clearly exceed the harms (or that the harms clearly exceed the benefits in the case of a strong negative recommendation) and that the quality of the supporting evidence is excellent (Grade A or B). ^a In some clearly identified circumstances, strong recommendations may be made based on lesser evidence when high-quality evidence is impossible to obtain and the anticipated benefits strongly outweigh the harms.	Clinicians should follow a strong recommendation unless a clear and compelling rationale for an alternative approach is present.
Recommendation	A recommendation means the benefits exceed the harms (or that the harms exceed the benefits in the case of a negative recommendation), but the quality of evidence is not as strong (Grade B or C). ^a In some clearly identified circumstances, recommendations may be made based on lesser evidence when high-quality evidence is impossible to obtain and the anticipated benefits outweigh the harms.	Clinicians should also generally follow a recommendation but should remain alert to new information and sensitive to patient preferences.
Option	An option means that either the quality of evidence that exists is suspect (Grade D) ^a or that well-done studies (Grade A, B, or C) ^a show little clear advantage to one approach vs another.	Clinicians should be flexible in their decision making regarding appropriate practice, although they may set bounds on alternatives; patient preference should have a substantial influencing role.
No recommendation	No recommendation means there is both a lack of pertinent evidence (Grade D) ^a and an unclear balance between benefits and harms.	Clinicians should feel little constraint in their decision making and be alert to new published evidence that clarifies the balance of benefit vs harm; patient preference should have a substantial influencing role.

^aSee **Table 5** for definition of evidence grades.

Table 5. Evidence levels for grades of evidence.^a

Grade	Treatment and Harm	Diagnosis
A	Well-designed randomized controlled trials performed on a population similar to the guideline's target population	Systematic review of cross-sectional studies with consistently applied reference standard and blinding
B	Randomized controlled trials; overwhelmingly consistent evidence from observational studies	Individual cross-sectional studies with consistently applied reference standard and blinding
C	Observational studies (case control and cohort design)	Nonconsecutive studies, case-control studies, or studies with poor, nonindependent, or inconsistently applied reference standards
D	Mechanism-based reasoning or case reports	
X	Exceptional situations where validating studies cannot be performed and there is a clear preponderance of benefit over harm	

^aAmerican Academy of Pediatrics (AAP) classification scheme⁴⁷ updated for consistency with current level of evidence definitions.⁴⁸

themselves from a related discussion if asked by the GDG, and (3) agreed not to discuss any aspect of the guideline with industry before publication. Last, GDG members were reminded that conflicts of interest extend beyond financial relationships and may include personal experiences, how a member earns a living, and the member's previously established "stake" in an issue.⁴⁸

Guideline Key Action Statements

Each evidence-based statement is organized in a similar fashion: an evidence-based key action statement in bold, followed by the strength of the recommendation in italics. Each key action statement is followed by an "action statement profile" of aggregate evidence quality, level of confidence in the evidence, benefit-harm assessment, and

Table 6. Summary of guideline action statements.

Statement	Action	Strength
1. Patient history and physical examination	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.	Strong recommendation
2. Laboratory testing	Clinicians should not obtain routine laboratory testing in patients with new-onset Bell's palsy.	Recommendation (against)
3. Diagnostic imaging	Clinicians should not routinely perform diagnostic imaging for patients with new-onset Bell's palsy.	Recommendation (against)
4. Oral steroids	Clinicians should prescribe oral steroids within 72 hours of symptom onset for Bell's palsy patients 16 years and older.	Strong recommendation
5A. Antiviral monotherapy	Clinicians should not prescribe oral antiviral therapy alone for patients with new-onset Bell's palsy.	Strong recommendation (against)
5B. Combination antiviral therapy	Clinicians may offer oral antiviral therapy in addition to oral steroids within 72 hours of symptom onset for patients with Bell's palsy.	Option
6. Eye care	Clinicians should implement eye protection for Bell's palsy patients with impaired eye closure.	Strong recommendation
7A. Electrodiagnostic testing with incomplete paralysis	Clinicians should not perform electrodiagnostic testing in Bell's palsy patients with incomplete facial paralysis.	Recommendation (against)
7B. Electrodiagnostic testing with complete paralysis	Clinicians may offer electrodiagnostic testing to Bell's palsy patients with complete facial paralysis.	Option
8. Surgical decompression	No recommendation can be made regarding surgical decompression for Bell's palsy patients.	No recommendation
9. Acupuncture	No recommendation can be made regarding the effect of acupuncture in Bell's palsy patients.	No recommendation
10. Physical therapy	No recommendation can be made regarding the effect of physical therapy in Bell's palsy patients.	No recommendation
11. Patient follow-up	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.	Recommendation

statement of costs. In addition, there is an explicit statement of any value judgments, the role of patient preferences, clarification of any intentional vagueness by the GDG, exceptions to the statement, any differences of opinion, and a repeat statement of the strength of the recommendation. Several paragraphs subsequently discuss the evidence base supporting the statement. An overview of each evidence-based statement in this guideline can be found in **Table 6**.

The role of patient preference in making decisions deserves further clarification. For statements in which the evidence base demonstrates clear benefit, although the role of patient preference for a range of treatments may not be relevant (such as with intraoperative decision making), clinicians should provide patients with clear and comprehensible information on the benefits to facilitate patient understanding and shared decision making, which in turn leads to better patient adherence and outcomes. In cases where

evidence is weak or benefits are unclear, the practice of shared decision making—again, where the management decision is made by a collaborative effort between the clinician and an informed patient—is extremely useful. Factors related to patient preference include (but are not limited to) absolute benefits (numbers needed to treat), adverse effects (number needed to harm), cost of drugs or procedures, and frequency and duration of treatment.

STATEMENT 1. PATIENT HISTORY AND PHYSICAL EXAMINATION: 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. *Strong recommendation based on observational studies of alternative causes of facial paralysis and reasoning from first principles, with a preponderance of benefit over harm.*

Action Statement Profile

- Aggregate evidence quality: Grade C
- Level of confidence in evidence: High
- Benefit: Identification of other causes of facial paresis/paralysis, enabling accurate diagnosis; avoidance of unnecessary testing and treatment; identification of patients for whom other testing or treatment is indicated; opportunity for appropriate patient counseling
- Risks, harms, costs: None
- Benefit-harm assessment: Preponderance of benefit
- Value judgments: The GDG felt that assessment of patients cannot be performed without a history and physical examination and that it would not be possible to find stronger evidence, as studies excluding these steps cannot ethically be performed. Other causes of facial paresis/paralysis may go unidentified; a thorough history and physical examination will help avoid missed diagnoses or diagnostic delay.
- Intentional vagueness: None
- Role of patient preferences: None
- Exceptions: None
- Policy level: Strong recommendation
- Differences of opinion: None

Supporting Text

The purpose of this statement is to emphasize that a minority of cases of facial paresis or paralysis do have an identifiable cause and therefore are not Bell's palsy and should not be treated as such. Many of these potential etiologies can be readily identified by appropriately assessing patient history and conducting a thorough physical examination. In addition, the acute onset of symptoms is a cardinal feature of Bell's palsy, and the rate of symptom onset must be confirmed by history to make the diagnosis. The sudden onset of unilateral facial paresis/paralysis and the absence of signs indicative of another underlying cause allow for the diagnosis of Bell's palsy. In addition to determining whether the patient's face is paretic or paralyzed, the history and physical examination should be directed at detecting neurologic, otologic, oncologic, inflammatory, or infectious causes; cerebello-pontine angle pathology; or vascular insufficiencies. While not all-inclusive, **Table 7** outlines many other causes of facial paralysis and some of their distinguishing features.

Approximately 70% of facial nerve palsies are considered Bell's palsy.^{1,49} This statistic indicates that 30% of patients presenting with facial paresis/paralysis have other underlying causes. To decrease the likelihood of misdiagnosis, a comprehensive history should seek symptoms such as dizziness, dysphagia, or diplopia, which suggest diagnoses other than Bell's palsy. The clinician should document function of all other cranial nerves and should inquire about a viral prodrome or hyperacusis. The clinician should inquire about underlying medical problems that could predispose the patient to facial paralysis, such as prior stroke, brain

tumors, skin cancers on the head or face, parotid tumors, facial/head trauma, or recent infections (**Table 7**). The timing of onset of symptoms remains important. Symptoms associated with neoplastic or infectious causes of facial paralysis often progress gradually, relative to the sudden-onset characteristic of Bell's palsy.

Symptoms suggestive of Bell's palsy may include pain in the ear and postauricular region; weakness of facial musculature, including the inability to chew food without difficulty; poor/ineffectual eye closure; alteration of taste, occasionally accompanied by numbness or tingling of the cheek/mouth; ocular pain and tearing¹³; or a family history of Bell's palsy. Bell's palsy presents disproportionately among pregnant women and people with diabetes, influenza, a cold, or other upper respiratory illness.^{6,18}

After a careful history, a comprehensive physical examination may confirm a suspected etiology or reveal an as-yet unidentified cause of the paresis/paralysis. Careful inspection of the ear canal, tympanic membrane, parotid gland, and skin of the head face and cheek is essential. Ear infection, cholesteatoma, and vesicular rashes (indicative of zoster infection) must be ruled out. The presence of ulcerative lesions on the skin suggestive of skin cancer or masses of the cheek should be noted. Along with characterization of the overall movement of the face, all cranial nerves should be assessed, paying specific attention to the extent of facial weakness and whether all nerve branches are involved. This information could help identify the sparing of forehead movement suggestive of a central pathology, such as stroke,^{13,50} or could point to a more peripheral lesion affecting only a single branch of the nerve.⁵¹

Signs and symptoms atypical for Bell's palsy, including bilateral facial nerve paresis or paralysis, may warrant additional specialized and more extensive laboratory testing. Clinicians should consider infrequent causes of facial paralysis, including Lyme disease in endemic areas, sarcoidosis, Guillain-Barré syndrome, Sjögren's syndrome, and leprosy.^{1,15-17} Those patients whose history and physical examination indicate an identifiable cause of facial paresis/paralysis should be managed accordingly and are excluded from the remainder of this guideline.

The emotional impact of this condition should not be underestimated. Patients often struggle with the facial disfigurement caused by the facial paresis/paralysis and its social ramifications. Clinicians can provide welcome assistance to their patients by providing reasonable expectations about recovery and duration of symptoms.

A comprehensive discussion of all causes of facial paresis/paralysis is beyond the scope of this guideline, but it is the responsibility of the evaluating clinician to conduct an appropriate patient history and to examine the patient with the specific intent of finding an underlying cause. Bell's palsy is, by definition, a diagnosis of exclusion. **Table 7** is not a comprehensive list of etiologies but captures the general categories and distinguishing features of potential causes of facial paresis/paralysis of which the evaluating clinician should be aware.

Table 7. Etiologies and clinical features of facial paralysis.

	Condition	Etiologic Agent	Distinguishing Factors
Autoimmune	Guillain-Barré	Autoimmune/infectious	Acute polyneuropathy; ascending paralysis; weakness of hands, feet progressing to the trunk
	Melkersson-Rosenthal syndrome	Unknown	Recurrent facial paralysis, swelling of face/lips, and fissures or folds in tongue
	Multiple sclerosis	Unknown	Abnormal neurologic examination with intermittent symptoms
	Sarcoidosis	Unknown	May be bilateral; laboratory abnormalities including angiotensin-converting enzyme (ACE) level
Congenital	Mobius syndrome	Possibly viral	Age (young), bilateral in nature, unable to move face or eyes laterally
Endocrine	Diabetes	Microvascular disease	Other signs and symptoms of diabetes, laboratory testing
Idiopathic	Acute facial nerve paresis/paralysis	Unknown	Classic Bell's palsy with other etiologies excluded
Infectious	Encephalitis/ meningitis	Fungal, viral, or bacterial	Headache, stiff neck, cerebrospinal fluid abnormalities
	Herpes simplex	Herpes simplex virus along axons of nerve residing in the geniculate ganglion	Fever, malaise
	Human immunodeficiency virus (HIV)	Human immunodeficiency virus (HIV)	Fever, malaise, CD4 count
	Lyme disease	Spirochete <i>Borrelia burgdorferi</i>	May be bilateral, rash, arthralgias
	Mononucleosis	Epstein-Barr virus	Malaise, difficult to distinguish
	Otitis media	Bacterial pathogens	Gradual onset, ear pain, fever, hearing loss
	Ramsay Hunt syndrome	Herpes zoster virus	Pronounced prodrome of pain, vesicular eruption in ear canal or pharynx
Inherited	Syphilis	<i>Treponema pallidum</i>	Other neurologic and cutaneous manifestations
	Heritable disorders	Autosomal dominant inheritance	Family history as high as 4%, may have other neurologic disorders
Neoplastic	Facial nerve tumor, skin cancer, parotid tumors	Multiple carcinomas of the head and neck	May involve only select branches of the facial nerve or other cranial nerves and present as multiple cranial neuropathies
Neurovascular	Stroke	Ischemia, hemorrhage	Forehead sparing most often, extremities often involved
Traumatic	Injury to facial nerve	Trauma, including forceps delivery	Timing of injury coincides with trauma

STATEMENT 2. LABORATORY TESTING: Clinicians should not obtain routine laboratory testing in patients with new-onset Bell's palsy. *Recommendation (against) based on observational studies and expert opinion with a preponderance of benefit over harm.*

Action Statement Profile

- Aggregate evidence quality: Grade C
- Level of confidence in evidence: High
- Benefit: Avoidance of unnecessary testing and/or treatment, avoidance of pursuing false positives, cost savings
- Risks, harms, costs: Potential missed diagnosis
- Benefit-harm assessment: Preponderance of benefit
- Value judgments: While the GDG felt that there are circumstances where specific testing is indicated in at-risk patients (such as Lyme disease serology in endemic areas), these patients can usually be identified by history.

- Intentional vagueness: We used the word *routine* to specify that under certain circumstances, laboratory testing may be indicated.
- Role of patient preferences: Small (there is an opportunity for patient education)
- Exceptions: None
- Policy level: Recommendation (against)
- Differences of opinion: None

Supporting Text

The purpose of this statement is to reduce unnecessary laboratory testing in patients with Bell's palsy. While laboratory testing may be indicated in selected patients with identifiable risk factors or atypical presentation of sudden-onset unilateral facial paresis/paralysis, laboratory testing is not indicated when history and physical examination do not suggest an alternative cause. The GDG identified no literature indicating a role for laboratory testing in the absence of suggestive history.

Assessing for Lyme Disease

The use of targeted laboratory testing to assess for specific diagnoses of concern (based on history or examination findings) can be fruitful. In endemic areas, Lyme disease can be the cause of facial paralysis in up to 25% of cases.⁵² An interactive Lyme disease map and Lyme disease cases and incidence by state for 2002-2011 are available from the Centers for Disease Control and Prevention.⁵³ For patients in endemic areas (or patients who have recently traveled to endemic areas), Lyme disease serology should be drawn, particularly when a patient's history is suggestive of an exposure.⁵⁰

Currently, there are 3 antibody tests available to aid in the diagnosis of Lyme disease, typically performed using a 2-step process. If screening tests such as enzyme-linked immunosorbent assay (ELISA) and indirect fluorescent antibody (IFA) are positive or borderline, the more specific Western blot is performed to confirm the result. ELISA is considered a more reliable and accurate test than IFA, but IFA may be used if ELISA is not available. While not addressed in this guideline, bilateral facial nerve paresis or paralysis is atypical of Bell's palsy and may warrant additional specialized and more extensive laboratory testing.

Although laboratory testing itself carries minimal risk for patients, many of the tests ordered to look for unusual etiologies are costly. The routine use of a battery of laboratory tests that are highly unlikely to alter the diagnosis is not cost-effective. Avoiding routine laboratory testing can prevent patient anxiety and costly workups on false-positive test results. Facial paralysis can result from conditions such as human immunodeficiency virus (HIV)/AIDS, Guillain-Barré syndrome, multiple idiopathic cranial nerve neuropathies, brainstem encephalitis, syphilis, leukemia, sarcoidosis, Melkersson-Rosenthal syndrome, or bacterial meningitis. As Lyme disease becomes endemic in new areas, the likelihood that the condition needs to be considered grows each year.

Testing for these conditions is warranted when sufficient clinical suspicion exists.

STATEMENT 3. DIAGNOSTIC IMAGING: Clinicians should not routinely perform diagnostic imaging for patients with new-onset Bell's palsy. *Recommendation (against) based on observational studies with a preponderance of benefit over harm.*

Action Statement Profile

- Aggregate evidence quality: Grade C
- Level of confidence in evidence: High
- Benefit: Avoidance of unnecessary radiation exposure, avoidance of incidental findings, avoidance of contrast reactions, cost savings
- Risks, harms, costs: Risk of missing other cause of facial paresis/paralysis
- Benefit-harm assessment: Preponderance of benefit
- Value judgments: None
- Intentional vagueness: The word *routine* was used to indicate there may be some clinical findings that would warrant imaging.
- Role of patient preferences: Small, but there is an opportunity for patient education/counseling
- Exceptions: None
- Policy level: Recommendation (against)
- Differences of opinion: None

Supporting Text

The purpose of this statement is to discourage the routine use of diagnostic imaging for patients with new-onset Bell's palsy. History and physical examination are the most vital aspects of making the diagnosis of Bell's palsy. Acute facial paresis/paralysis in the absence of explanatory history or physical findings is idiopathic in the vast majority of cases.⁵⁴ The routine use of diagnostic imaging is not recommended at the time of initial presentation of these patients. While MRI studies of Bell's palsy may commonly show enhancement along the involved (ipsilateral) facial nerve—especially around the area of the geniculate ganglion—this finding does not influence the course of therapy. In fact, this enhancement may be confused with another process such as a small tumor of the facial nerve or other incidental findings, leading to further unnecessary testing.⁵⁵ In addition, imaging is costly and associated with potential risk.⁵⁶⁻⁵⁹

Magnetic resonance imaging and CT scans do have some risks as well as considerable cost.⁵⁶ Computed tomography contrast has the higher risk of induced allergic reactions,⁵⁷ although when contrast is used with either scan, there is the risk of adverse reactions, such as allergic contrast reaction, nephropathy, or nephrogenic systemic fibrosis. The CT scans also expose patients to ionizing radiation, which can increase the future risk of malignancy.⁵⁸ The American College of Radiology (ACR) recognizes that studies have demonstrated increased cancer risk, even with low levels of

radiation exposure, especially in children.⁵⁹ Accordingly, the ACR advises against imaging unless there is a clear medical benefit outweighing any associated risk.⁵⁹ While there are no studies evaluating the cost of imaging in patients with Bell's palsy, these studies can range from hundreds to thousands of dollars.

While the use of routine imaging at the time of initial diagnosis is discouraged, the GDG recognizes that there is a distinct role for imaging in the event of suggestive history or physical findings (ie, trauma to the temporal bone or history of tumor) or if the paralysis fails to recover in the expected time frame or worsens. The literature strongly supports that any presentation of facial paresis/paralysis inconsistent with Bell's palsy should be further evaluated by imaging.⁶⁰ Features atypical of Bell's palsy include a second paralysis on the same side, paralysis of isolated branches of the facial nerve, paralysis associated with other cranial nerve involvement, or no sign of recovery after 3 months. Magnetic resonance imaging of the entire course of the facial nerve, with and without contrast, is the imaging test of choice for patients in these circumstances. Imaging should include both the internal auditory canal (IAC) and face, to image the whole course of the facial nerve. If an MRI is contraindicated, a contrast-enhanced CT can be used.

STATEMENT 4. ORAL STEROIDS: Clinicians should prescribe oral steroids within 72 hours of symptom onset for Bell's palsy patients 16 years and older. *Strong recommendation based on high-quality randomized controlled trials with a preponderance of benefit over harm.*

Action Statement Profile

- Aggregate evidence quality: Grade A
- Level of confidence in evidence: High
- Benefit: Improvement in facial nerve function, faster recovery
- Risks, harms, costs: Steroid side effects, cost of therapy
- Benefit-harm assessment: Preponderance of benefit
- Value judgments: None
- Intentional vagueness: None
- Role of patient preferences: Small
- Exceptions: Diabetes, morbid obesity, previous steroid intolerance, and psychiatric disorders. Pregnant women should be treated on an individualized basis.
- Policy level: Strong recommendation
- Differences of opinion: None

Supporting Text

The purpose of this statement is to encourage the use of oral corticosteroids for patients 16 years and older with new-onset Bell's palsy. Goals of treatment for Bell's palsy patients include decreasing recovery time and improving facial nerve functional recovery.

Inflammation and edema causing compression of the facial nerve as it travels through the fallopian (facial) canal is the leading posited mechanism of Bell's palsy. Potent anti-inflammatory agents, such as oral corticosteroids, target the inflammatory process, presumably decreasing nerve edema and thereby facilitating the return of facial nerve function.

An evidence-based practice parameter developed by the American Academy of Neurology (AAN) recently evaluated the efficacy of oral corticosteroids and acyclovir in patients with Bell's palsy.⁶¹ Based on the results of 2 randomized clinical trials with objective outcomes,^{23,62} the AAN concluded that "steroids are highly likely to be effective and should be offered to increase the probability of recovery of facial nerve function (risk difference 12.8 percent–15 percent)."⁶¹

The study by Sullivan et al,⁶² a double-blind, placebo-controlled, randomized, factorial trial involving 551 patients, reported significant improvement of facial nerve function in patients treated with prednisolone within 72 hours of onset. Participants in the study were randomly assigned to groups treated with prednisolone, acyclovir, placebo, or both active agents. All participants were treated for 10 days, and all patients were 16 years of age and older. Sullivan et al reported that 83% of the participants randomized to prednisolone had recovered facial nerve function 3 months after treatment compared with 63.6% of those randomized to placebo ($P < .001$). Evaluation 9 months post-treatment revealed 94.4% recovery in the prednisolone group and 81.6% recovery in the placebo group.

The study by Engstrom et al²³ was a similarly randomized, double-blind, placebo-controlled, multicenter trial involving 829 patients (ages 18-75 years). This trial compared the short- and long-term effects of prednisolone and valacyclovir in facial nerve recovery attributed to Bell's palsy. Individuals within 72 hours of initial diagnosis were randomized to placebo-plus-placebo, prednisolone-plus-placebo, valacyclovir-plus-placebo, or prednisolone-plus-valacyclovir groups. Statistically significant shorter times to recovery were noted in the 416 patients treated with prednisolone compared with the 413 patients who did not receive prednisolone.

Treatment of Bell's palsy with oral corticosteroids is not without risk. Known side effects of oral corticosteroid use include gastrointestinal disturbances, reactivation of peptic ulcer disease, loss of control of glucose levels, elevated blood pressure, peripheral edema, and mood swings or episodes of acute psychosis. Although rare, avascular necrosis of the femoral head has been reported. Pregnant patients and patients with diabetes were routinely excluded from randomized trials. Accordingly, these patients should be handled on an individualized basis.^{54,63,64}

Both of the randomized clinical trials with objective outcomes above used prednisolone for a 10-day course. One used prednisolone 25 mg twice daily for 10 days, and the other used 60 mg per day for 5 days, then tapered over 5 days.^{23,62} Based on these studies, the GDG recommends a

10-day course of oral steroids with at least 5 days at a high dose (either prednisolone 50 mg for 10 days or prednisone 60 mg for 5 days with a 5-day taper) initiated within 72 hours of symptom onset. The benefit of treatment after 72 hours is less clear.

Use of Steroids in Children with Bell's Palsy

Information is limited, as children were excluded from most treatment trials of new-onset Bell's palsy. Several studies indicate that the prognosis of untreated Bell's palsy in children is better and that children show higher rates of spontaneous recovery than do adults; therefore, the potential benefit of corticosteroid treatment is inconclusive.⁶⁵ Compelling data are lacking for steroid use in children, and the need for steroid treatment is unclear.^{54,66} The GDG identified 2 systematic reviews/meta-analyses that specifically addressed oral corticosteroid use in children.^{67,68}

Pitaro and Daniel⁶⁷ performed a systematic review of children diagnosed with new-onset Bell's palsy treated with steroids and found no controlled trials on the subject. Studies identified for inclusion in the systematic review had low evidence levels and concluded that the evidence for the use of steroids in children is inconclusive.

The systematic review by Salman and MacGregor⁶⁸ sought to include pediatric trials, including patients with Bell's palsy younger than 16 years who were treated with steroids. Identified studies exhibited flawed randomization methodologies and reported both children and adults together. Only 1 included study exclusively involved children, and it was not placebo controlled. The remainder of the identified trials did not perform separate analyses for the pediatric group. The systematic review concluded that there was no firm evidence for the routine use of steroids in children with new-onset Bell's palsy.

Despite the absence of quality trials supporting steroid use in children, given the presumed similar disease process of Bell's palsy in adults and children, as well as the generally favorable benefit-harm ratio of steroid therapy, oral steroids may be considered in pediatric patients with a large role for caregiver involvement in the decision-making process.

STATEMENT 5A. ANTIVIRAL MONOTHERAPY: Clinicians should not prescribe oral antiviral therapy alone for patients with new-onset Bell's palsy. *Strong recommendation (against)* based on high-quality randomized controlled trials with a preponderance of benefit over harm.

Action Statement Profile

- Aggregate evidence quality: Grade A
- Level of confidence in evidence: High
- Benefit: Avoidance of medication side effects, cost savings
- Risks, harms, costs: None
- Benefit-harm assessment: Preponderance of benefit
- Value judgments: None
- Intentional vagueness: None

- Role of patient preferences: Small
- Exceptions: None
- Policy level: Strong recommendation (against)
- Differences of opinion: None

Supporting Text

The purpose of this statement is to discourage the use of antiviral monotherapy for patients with new-onset Bell's palsy. Although Bell's palsy is a disorder of unknown cause, there is evidence that infection with or reactivation of a virus within the facial nerve ganglion may be a cause of the disorder. Viral reactivation occurs in a minority of patients with Bell's palsy but does not appear to change the outcome or response to therapy.⁶⁹ Still, the theory that Bell's palsy has a viral etiology has served as rationale for numerous trials investigating whether antiviral therapy has a primary role in the treatment of Bell's palsy. The most extensively studied antivirals to date include acyclovir and valacyclovir.^{2,23,62}

The evidence is clear that antiviral therapy alone is no better than placebo with regard to facial nerve recovery in Bell's palsy. The most comprehensive and well-designed randomized controlled trial to date failed to find an improved rate of facial nerve recovery in 207 patients treated with valacyclovir alone, compared with 209 patients treated with placebo alone.²³ Meta-analyses of numerous randomized controlled trials investigating antiviral therapy for Bell's palsy are consistent in their finding that antiviral therapy alone is no better than placebo and is inferior to steroid therapy with regard to facial nerve recovery rate. The meta-analyses do have heterogeneity due to differences in antiviral drugs used, drug dosing, and timing of therapy initiation but are nevertheless consistent and unequivocal in their finding of no benefit from antiviral therapy as a single-modality treatment for Bell's palsy.^{2,70-74}

The recommendation against antiviral monotherapy offers the benefit of avoiding the cost and side effects of ineffective antiviral therapy for Bell's palsy. The most commonly observed side effects of antiviral therapy are gastrointestinal related and include nausea, vomiting, and diarrhea, with rare severe reactions, including hives, bronchospasm, angioedema, and hepatic or renal failure. Adverse events from antiviral therapy were rarely reported in clinical trials of patients with Bell's palsy and were limited to gastrointestinal upset.⁷¹ Accordingly, no serious adverse events from antiviral therapy were noted in the Bell's palsy literature.^{2,23,54,62}

Pediatric Bell's palsy patients were not included in the antiviral trials, and therefore there is no evidence supporting the use of antiviral therapy alone in pediatric patients with Bell's palsy. Antiviral therapy may also carry an increased risk for pregnant patients. In summary, antiviral therapy alone (acyclovir or valacyclovir) is not recommended in the treatment of Bell's palsy due to lack of effectiveness of currently available drugs, unnecessary cost, and the potential for drug-related complications. Although this may well be a class effect for this group of drugs, it is theoretically

possible that other antivirals presently available or developed in the future may be shown to be effective.

STATEMENT 5B. COMBINATION ANTIVIRAL THERAPY: Clinicians may offer oral antiviral therapy in addition to oral steroids within 72 hours of symptom onset for patients with Bell's palsy. *Option based on randomized controlled trials with minor limitations and observational studies with equilibrium of benefit and harm.*

Action Statement Profile

- Aggregate evidence quality: Grade B
- Level of confidence in evidence: Medium, because the studies cannot exclude a small effect
- Benefit: Small potential improvement in facial nerve function
- Risks, harms, costs: Treatment side effects, cost of treatment
- Benefit-harm assessment: Equilibrium of benefit and harm
- Value judgments: Although the data were weak, the risks of combination therapy were small.
- Intentional vagueness: None
- Role of patient preferences: Large; significant role for shared decision making
- Exceptions: Diabetes, morbid obesity, and previous steroid intolerance. Pregnant women should be treated on an individualized basis.
- Policy level: Option
- Differences of opinion: None

Supporting Text

The purpose of this statement is to address the use of oral antiviral therapy in combination with oral steroids for patients with new-onset Bell's palsy. Antiviral therapy combined with oral steroids was not statistically significantly superior to oral steroids alone when given within 72 hours of onset of facial paralysis in the 2 highest quality, randomized controlled trials published to date.^{23,62} One study found no difference in complete facial nerve recovery time between 210 patients treated with valacyclovir and prednisolone and 213 patients treated with prednisolone and placebo, whereas the other study found that 83% (105/127) of patients had full nerve recovery at 3 months when treated with prednisolone and placebo, compared with 79.7% (99/124) of patients treated with prednisolone and acyclovir.^{23,62} Based on these studies, antiviral agents alone provide no benefit; however, in combination with steroids, these studies could not conclusively rule out a small yet nonstatistically significant benefit.⁶¹

An additional large trial of 829 patients with Bell's palsy treated within 72 hours of onset has recently been published, confirming these findings.⁷⁵ Several meta-analyses analyzing various groupings of randomized controlled trials have also found no benefit in facial nerve recovery from combination antiviral and steroid therapy compared with steroid therapy alone.^{1,76-78}

Several trials with smaller sample sizes and lower methodological quality found limited improvements in long-term facial nerve recovery in patients with Bell's palsy treated with combination antiviral therapy and steroids compared with steroid therapy alone. One trial of 221 patients randomized to valacyclovir and prednisolone vs placebo and prednisolone found a significantly higher rate of full facial nerve recovery at 6 months in the combination group (96.5%) compared with the steroid and placebo group (89.7%).⁷⁹ Another trial of 34 patients treated with famciclovir and prednisolone compared with 34 patients treated with prednisolone alone found significantly higher rates of full nerve recovery at 1 and 3 months in the combination group.⁸⁰ These results are suspect as significantly more patients in the steroid-only group had complete facial paralysis at presentation.⁸⁰ A meta-analysis that analyzed 18 trials found that the combination of antiviral therapy with steroids resulted in a 25% reduced risk of incomplete nerve recovery of borderline significance compared with steroid therapy alone (relative risk [RR], 0.75; 95% confidence interval [CI], 0.56-1.00).⁷³ It is estimated that 26 patients would require treatment with the combination therapy to achieve 1 better facial nerve outcome than with steroid therapy alone.⁷⁸

In summary, antiviral therapy in addition to steroid therapy has not been proven to be of benefit in the treatment of Bell's palsy in large, high-quality clinical trials, although a small benefit cannot be completely excluded. Due to the potential of a small benefit in facial nerve functional recovery and the relatively low risk of antiviral therapy, the GDG concluded that patients may be offered combination therapy if treated within 72 hours of onset of Bell's palsy, with a large role for shared decision making.

STATEMENT 6. EYE CARE: Clinicians should implement eye protection for Bell's palsy patients with impaired eye closure. *Strong recommendation based on expert opinion and a strong clinical rationale with a preponderance of benefit over harm.*

Action Statement Profile

- Aggregate evidence quality: Grade X
- Level of confidence in evidence: High. Eye protection has been the standard of care, and comparative studies with a no-treatment arm are unethical.
- Benefit: Prevention of eye complications
- Risks, harms, costs: Cost of eye protection implementation, potential side effects of eye medication
- Benefit-harm assessment: Preponderance of benefit over harm
- Value judgments: None
- Intentional vagueness: None
- Role of patient preferences: Small
- Exceptions: None
- Policy level: Strong recommendation
- Differences of opinion: None

Supporting Text

The purpose of this statement is to encourage the use of prophylactic eye care in those patients with Bell's palsy with incomplete eye closure. Bell's palsy is a condition that predisposes the eye to injury due to incomplete closure of the eyelid (lagophthalmos) from upper eyelid retraction or lower lid ectropion, as well as failure of the lacrimal pump mechanism, decreased blink and tear production, and loss of the corneal "squeegee effect" on the side affected by facial palsy. Incomplete closure of the eyelid may lead to deposition of foreign particles in the eye, corneal abrasions, exposure keratitis and/or corneal ulcerations.^{26,81-83} Clinicians should be aware of symptoms such as burning, itching, eye irritation, changes in vision, and pain.

The GDG found no studies to either support or refute the use of prophylactic eye care in patients with Bell's palsy, although most authors recommend prophylactic eye care.^{27,36,84-92} Studies in patients with eye problems similar to those found in patients with Bell's palsy—such as intensive care unit (ICU) patients in a coma where eyelid muscle function is absent—yielded a number of articles recommending treatment.⁸² None of the studies found statistical benefit or harm from any of the following treatment modalities for the prevention of corneal damage:

- Use of sunglasses⁹³
- Frequent administration of lubricating ophthalmic drops^{27,82,89,91,93}
- Frequent administration of ophthalmic ointments^{27,82,89,91}
- Use of a moisture chamber using a polyethylene cover⁸²
- Eye patching or taping^{27,91,93}
- Combination of the above treatments⁸²

Supportive care consisting of ocular surface hydration is advisable for all Bell's palsy patients with incomplete eye closure. Ophthalmic drops add some hydration, prevent loss of moisture, and do not tend to blur vision but do require repeated instillation. Ointments generally require less frequent administration and are more effective in preventing loss of moisture but tend to blur vision. Moisture chambers and eye patching or taping are particularly effective when used at night. Some authors have not recommended or have discouraged eye patching^{89,94,95} due to the risk of corneal damage from poor patient execution of the procedure. Despite these concerns, many authors have reported successful use of both interventions. Clinicians who recommend either eye taping or patching should ensure that patients have been carefully instructed in proper execution.

Other recommended eye treatments in those patients who fail supportive eye care or patients with severe, persistent lagophthalmos should have a detailed ophthalmologic evaluation. Other considerations may ultimately include the use of botulinum toxin injections, or temporary or permanent tarsorrhaphy or surgery to weight the upper eyelid.^{26-28,96}

Botulinum injections may improve eyelid closure for a period of months, whereas surgical options improve lid closure permanently, thereby mitigating the ongoing risk for ocular complications.^{27,29}

In summary, although there have been no direct comparisons of various protective methods, based on the corneal abrasion risk, ICU literature, and expert opinion, the GDG feels it is critical to recommend supportive eye care for all Bell's palsy patients with incomplete eye closure. Initially, lubricating drops and/or ointment should be used in patients with incomplete eye closure. The presence of ocular symptoms such as pain, irritation, or itching should prompt an expeditious referral to an eye specialist to prevent corneal damage.

STATEMENT 7A. ELECTRODIAGNOSTIC TESTING WITH INCOMPLETE PARALYSIS: Clinicians should not perform electrodiagnostic testing in Bell's palsy patients with incomplete facial paralysis. *Recommendation (against) based on observational studies with a preponderance of benefit over harm.*

Action Statement Profile

- Aggregate evidence quality: Grade C
- Level of confidence in evidence: High
- Benefit: Avoidance of unnecessary testing, cost savings
- Risks, harms costs: None
- Benefit-harm assessment: Preponderance of benefit over harm
- Value judgments: None
- Intentional vagueness: None
- Role of patient preferences: None
- Exceptions: None
- Policy Level: Recommendation (against)
- Differences of opinion: None

Supporting Text

The purpose of this statement is to discourage the use of electrodiagnostic testing in Bell's palsy patients with incomplete facial paralysis. Electrodiagnostic testing procedures, such as electro-neurophysiologic (ENoG) testing and facial electromyography (EMG), have been used to quantify the extent of damage to the facial nerve. In ENoG testing procedures, surface electrodes record the electrical depolarization of facial muscles following electrical stimulation of the facial nerve, whereas facial EMG is performed by inserting a needle electrode into affected muscles and recording spontaneous depolarizations and the responses to voluntary muscle contraction. The cost, inconvenience, and discomfort of invasive EMG testing are outweighed by the likelihood of full recovery in most patients.

For most patients presenting with Bell's palsy, the chances of complete recovery are very high, with rates ranging from approximately 70% with no treatment to 94% with steroids.^{21,62} The small percentage of patients who do not completely recover can have the sequelae of permanent paresis or paralysis, however. Patients presenting with incomplete paralysis have a very

high likelihood of complete return of function.^{19,97} Accordingly, there is no meaningful role for electrodiagnostic tests. While electrodiagnostic tests may show reduced function in the setting of incomplete paralysis, there is no strong evidence that testing can successfully predict which patients are more or less likely to recover complete function.^{19,98}

The relationship between the degree of clinical weakness and electrodiagnostic findings has not been exhaustively studied. Engstrom et al¹⁹ demonstrated that only patients with House-Brackmann scores of III or greater have been associated with ENoG response amplitudes reduced to less than 25% of normal (**Table 2**). Other studies have simply excluded patients with incomplete paralysis.⁹⁸ General electrodiagnostic experience suggests that only patients with severe weakness will have a decrease in motor response amplitude to <10% of the normal side. It then follows that if a patient has clinically discernible voluntary contraction of the facial muscles at presentation, recovery is highly likely and electrodiagnostic testing provides no further insight. Therefore, unless complete facial paralysis is present, electrodiagnostic testing provides no clinical benefit.

STATEMENT 7B. ELECTRODIAGNOSTIC TESTING WITH COMPLETE PARALYSIS: Clinicians may offer electrodiagnostic testing to Bell's palsy patients with complete facial paralysis. *Option based on observational trials with equilibrium of benefit and harm.*

Action Statement Profile

- Aggregate evidence quality: Grade C
- Level of confidence in evidence: Medium due to variations in patient selection, study design, and heterogeneous results
- Benefit: Provide prognostic information for the clinician and patient, identification of potential surgical candidates
- Risks, harms, costs: Patient discomfort, inconvenience to undergo repeated electrical testing, cost of testing
- Benefit-harm assessment: Equilibrium of benefit and harm
- Value judgments: None
- Intentional vagueness: None
- Role of patient preferences: Large role for shared decision making, as electrodiagnostic testing may provide only prognostic information for the patient
- Exceptions: None
- Policy level: Option
- Differences of opinion: None

Supporting Text

The purpose of this statement is to assert that electrodiagnostic testing may offer prognostic information in patients with complete facial paralysis (ie, no discernible movement of the face). In patients who present with complete facial nerve

paralysis, the risk of incomplete recovery is higher than for those with mere paresis (ie, facial weakness).¹³ Electrodiagnostic testing has been shown in retrospective studies to identify patients with as much as a 50% chance of incomplete recovery.⁹⁷⁻⁹⁹ The results of electrodiagnostic testing in patients with complete facial paralysis may provide meaningful prognostic information, insofar as the results could alert the physician and patient to a heightened likelihood of incomplete recovery.¹⁰⁰

Electrodiagnostic testing can quantify the extent of nerve damage. In nerve lesions such as those occurring in Bell's palsy, nerve responses to electrical stimulation distal to the site of damage change over time. Initially, the nerve remains normally excitable, but within about 3 days, Wallerian degeneration (ie, degeneration of the axon distal to the site of an injury that has disconnected it from its neuronal cell body or origin) starts to result in a progressive decrease in the amplitude of the response, reflecting a functional loss of axons. With complete paralysis, electrophysiologic testing results become stable, and therefore informative, approximately 7 days following symptom onset.^{101,102} Testing can be performed earlier; however it can be misleading, as Wallerian degeneration progresses up to that point. Testing beyond 14 to 21 days may be less reliable.

- Electroneuronography: Surface electrodes are placed over selected facial muscles and the main trunk of the facial nerve is stimulated electrically. The amplitude of the maximal response (mV) is recorded and compared with the unaffected side.
- Electromyography: Needle electrodes are inserted into the facial muscles and depolarizations are recorded at rest and following voluntary attempts to contract the facial muscles.

In patients who present with a complete facial paralysis, ENoG testing may be used. In this procedure, surface electrodes record the electrical depolarization of facial muscles following electrical stimulation of the facial nerve, typically with stimulation in front of the ear overlying the main trunk of the facial nerve, which is distal to the presumed site of nerve damage in Bell's palsy. If the response amplitude on the damaged side exceeds 10% of the amplitude on the contralateral (intact) side, most patients recover normal or near-normal facial movement. If the amplitude is less than 10% of the normal side, patients may still achieve normal or near-normal function, although a higher percentage of patients do not.^{19,97-99} EMG testing may offer additional information in patients with complete paralysis and ENoG showing less than 10% function on the affected side.^{8,103}

Facial EMG, performed by inserting a needle electrode into affected muscles and recording both spontaneous depolarizations and the responses to voluntary muscle contraction, can provide information complementary to that obtained following electrical stimulation of the facial nerve. Individuals in whom the nerve is not electrically excitable and voluntary muscle contraction is not clinically visible may

still have preserved axons. Voluntary muscle contraction can trigger muscle depolarizations that are too small to see but can be recorded electrophysiologically. Demonstration of such potentials proves the presence of intact axons, predicting possible recovery; however, no systematic studies yet relate such findings to the likelihood of recovery.

While patients with Bell's palsy generally have excellent return of facial movement, electrodiagnostic testing in patients with complete paralysis can help to define a small subset of patients with poorer prognosis who may be counseled appropriately regarding potential reconstructive options or who, at the discretion of the clinician, may consider surgical decompression of the facial nerve (see Statement 8).

STATEMENT 8. SURGICAL DECOMPRESSION: No recommendation can be made regarding surgical decompression for Bell's palsy patients. *No recommendation based on low-quality, nonrandomized trials and equilibrium of benefit and harm.*

Action Statement Profile

- Aggregate evidence quality: Grade D
- Level of confidence in evidence: Low due to insufficient number of patients and poor quality of studies. Low confidence in the evidence led to a downgrade of the aggregate evidence quality from C to D.
- Benefit: Improved facial nerve functional recovery
- Risks, harms, costs: Surgical risks and complications, anesthetic risks, direct and indirect costs of surgery
- Benefit-harm assessment: Equilibrium of benefit and harm
- Value judgments: Although the data supporting surgical decompression are not strong, there may be a significant benefit for a small subset of patients who meet eligibility criteria and desire surgical management.
- Intentional vagueness: None
- Role of patient preferences: Large. The psychological impact of facial paralysis is significant but varies among patients. Concern about the facial deformity may make some patients willing to pursue a major operation for a small increase in the chance of complete recovery, while others may be more willing to accept the chance of poorer outcome to avoid surgery.
- Exceptions: None
- Policy level: No recommendation
- Differences of opinion: Major. The group was divided as to whether the evidence supported no recommendation or an option for surgery. This difference of opinion derived from controversy regarding the strength of evidence (C level evidence vs D level evidence).

Supporting Text

Surgical decompression of the facial nerve in patients with Bell's palsy has been used in select cases as an option to improve the chances of recovery of facial nerve function. There has been some controversy about the effectiveness of surgery to improve outcomes in Bell's palsy. This controversy stems from the good outcomes for patients with incomplete paralysis, the lack of large trials, the diversity of patients in existing trials (eg, degree and duration of paralysis), the variable surgical approaches that have been reported, and the lack of effectiveness in some trials relative to natural history. Previous systematic reviews have grouped these diverse trials and concluded a lack of definitive benefit of surgery.^{104,105} A few select trials have studied patients early after onset of symptoms and have surgically decompressed the facial nerve in the area most likely to be affected in Bell's palsy. Although small in size, these trials have demonstrated effectiveness at improving the percentage of patients recovering to normal or near-normal facial function. Based on the flawed nature of much of the literature and the significant risks and cost of surgical decompression of the facial nerve, despite a potential small benefit of surgical decompression, the GDG could make no recommendation for or against its use.

Most patients are not surgical candidates due to the excellent overall rates of recovery, ranging from approximately 70% with no treatment to 94% with steroids.^{21,62} Electrodiagnostic testing in patients with complete paralysis can identify a subset of patients with an increased likelihood of poor recovery²¹ (see Statement 7B). Specifically, patients with complete paralysis, greater than 90% reduction in amplitude on ENoG testing relative to the unaffected side of the face, and absent volitional nerve activity on EMG are less likely to recover spontaneously or with medical treatment alone.^{8,106,107} In this small group of patients, observation or medical treatment alone leads to relatively poor recovery rates, and therefore, given the significant morbidity of permanent facial paralysis, more aggressive intervention could be considered.

Early animal studies showed that decompression of the facial nerve within 12 days of injury improved regeneration of the nerve.¹⁰⁸ The same rationale has been applied to patients with Bell's palsy as the underlying pathophysiology is likely related to neural edema that compresses the nerve in an anatomically confined bony canal. In most cases of Bell's palsy, the site of constriction is thought to be at the most narrow portion of the facial nerve canal: the labyrinthine segment starting at the meatal foramen.^{106,107,109} The meatal foramen is at the lateral internal auditory canal where the labyrinthine segment of the facial nerve exits the internal auditory canal and rises to the geniculate ganglion.

Most studies reporting on decompression of the mastoid segment of the facial nerve (distal to the presumed site of injury in the labyrinthine segment) have failed to show any benefit of surgery. A Cochrane review by McAllister et al¹⁰⁵ searched for all randomized or quasi-randomized controlled

trials involving any surgical intervention for Bell's palsy. Only 2 studies met inclusion criteria for this meta-analysis.^{24,110} The pooled data from these studies did not show improved facial nerve outcomes for patients with surgical decompression of the facial nerve, although both studies used transmastoid decompression only.¹⁰⁵ May et al¹¹¹ and Aoyagi et al¹¹² also failed to show benefit of facial nerve decompression via a transmastoid approach. Yanagihara et al¹¹³ reported improvement with transmastoid decompression, but the geniculate and lateral labyrinth segment were also decompressed. This extended access via the transmastoid route required disarticulation of the incus, which risks creating conductive hearing loss.

In 1981, Fisch⁵ reported his experience with decompression of the labyrinthine segment via a middle fossa approach. The middle fossa approach requires a craniotomy but allows for removal of the bone surrounding the facial nerve at the meatal foramen and fallopian canal without affecting the structures of hearing. The decompressed group had a better return of function (79% for patients with 95%-100% reduction in amplitude, 93% for those with 90%-94% reduction in amplitude), while only 64% of control patients who were not decompressed regained normal function.

A subsequent study by Gantz et al⁸ provides the most compelling evidence in support of middle fossa decompression of the facial nerve in Bell's palsy. This was a multicentered, case-control study. Inclusion criteria included ENoG testing with >90% reduction in amplitude on the affected side and no voluntary EMG activity. Testing and treatment were done at least 3 but no more than 14 days after the onset of acute facial paralysis. In the surgical group, 31 of 34 (91%) patients achieved a House-Brackmann facial nerve outcome of I/II compared with only 15 of 36 (42%) of patients in the steroid-only control group ($P = .0002$).

No high-level evidence is available to confirm the findings of Gantz et al.⁸ Kim et al¹¹⁴ reported on a case series in which 11 of 13 patients who underwent middle fossa decompression achieved a House-Brackmann score of I or II. All patients in this study had ENoG reduced to <10% of the contralateral value, although the average time to surgery was 37 days (range, 8-75 days) after symptom onset.¹¹⁴ Another case series by Bodenez et al¹¹⁵ reported that 13 of 13 patients with ENoG reduced to <10% of the contralateral nerve recovered to House-Brackmann III after a subpetrosal approach, which also allows for bony removal at the meatal foramen and geniculate ganglion. Nerve decompression procedures took place a mean of 5 weeks after onset of Bell's palsy.

Middle fossa decompression of the facial nerve does have substantial costs and rare but serious risks.¹¹⁶ These include conductive or sensorineural hearing loss; injury to the facial nerve; risk of cerebrospinal fluid leak; infection; risks of temporal lobe retraction such as temporary or permanent aphasia, seizures, and stroke; and nonspecific risks with general anesthesia.¹¹⁷⁻¹¹⁹ A hospital stay including at least 1 day in the intensive care setting is generally necessary with the attendant costs.

There are logistical complexities in the expeditious evaluation of patients who may qualify for surgery. Patients with complete facial paralysis need to have electrodiagnostic testing (ENoG and EMG) performed after day 7 but before 14 days after onset of paralysis. If patients meet the electrodiagnostic criteria spelled out in the Gantz et al⁸ trial, surgery must be performed within 14 days of symptom onset for optimal effectiveness. Therefore, it is the responsibility of clinicians who adopt surgical decompression as part of their practice to develop facilitated pathways of referral to permit the proper evaluation and management of patients within this restricted timeframe.⁸

In addition, there are currently few clinicians able to perform this procedure. Due to the technical difficulty and paucity of patients who meet criteria for surgical candidacy, few centers in the United States perform a high volume of middle fossa surgery for Bell's palsy. In a 2011 survey of active members of the American Neurotology and Otolological Societies, only 22% of respondents reported performing more than 5 middle fossa decompression surgeries for Bell's palsy in the prior 10 years of practice, and only 4% had performed more than 10 in the same period.¹²⁰ Despite the infrequent performance of the surgery, most middle cranial fossa surgeons have the expertise to perform decompression of the facial nerve.

The literature is clear that transmastoid decompression of the facial nerve alone is not appropriate, yet there are limited data supporting surgical decompression of the meatal segment of the facial nerve in patients with complete paralysis, with electrodiagnostic testing demonstrating severe denervation within 2 weeks of onset of paralysis. While the panel could make no recommendation regarding surgical decompression for Bell's palsy, based on the limited data supporting its effectiveness, the cost and risks of the intervention, and the logistical complexities of identifying proper surgical candidates and arranging for surgery in the narrow window during which it may be effective, the dramatic psychological consequences of facial paralysis to some patients may outweigh these concerns. Facial nerve decompression is not recommended in children, as most recover with nonsurgical management.¹²¹

STATEMENT 9. ACUPUNCTURE: No recommendation can be made regarding the effect of acupuncture in Bell's palsy patients. *No recommendation based on poor-quality trials and an indeterminate ratio of benefit and harm.*

Action Statement Profile

- Aggregate evidence quality: Grade B
- Level of confidence in evidence: Low, due to significant methodological flaws in available evidence
- Benefit: Acupuncture may provide a potential small improvement in facial nerve function and pain

- Risks, harms, costs: Cost of acupuncture therapy, time required for therapy, therapy side effects, and delay in instituting steroid therapy
- Benefit-harm assessment: Unknown
- Value judgments: Due to the poor quality of the data and the inability to determine the harm-to-benefit ratio, the GDG could not make a recommendation.
- Intentional vagueness: None
- Role of patient preferences: Large
- Exceptions: None
- Policy level: No recommendation
- Differences of opinion: Major. The GDG was divided regarding whether to recommend against acupuncture or to make no recommendation.

Supporting Text

The purpose of this statement is to highlight the uncertainty regarding the use of acupuncture in treating patients with Bell's palsy. The GDG recognizes that a significant number of patients seek alternative/complementary medicine, including acupuncture, as a part of their medical care. Patient counseling regarding the potential benefits and complications from acupuncture therapy is often limited due to a relative paucity of high-quality evidence.

Acupuncture involves the placement of fine needles into specific points on the skin.¹²² There are several suggested theories regarding the physiologic mechanism of acupuncture, including increasing the excitability of the nerve and promoting regeneration of the nerve fibers and formation of collateral branches, enhancing muscle contraction and blood circulation to the nerves.¹²² However, there is little confirmatory evidence to support these hypotheses. Suggested acupoints are often chosen along Yang meridians, thought by practitioners to correspond to the facial nerve trunk and the facial musculature.^{123,124} In 1 systematic review, combinations of manual or electro-acupuncture with moxibustion (a traditional Chinese method involving burning herbal preparations containing mugwort) are recommended with a total course of 20 to 40 daily treatments, although frequency and duration of treatment are often individualized.¹²⁴

A systematic review of acupuncture treatment of Bell's palsy by Chen et al¹²² included 6 RCTs with a total of 537 participants. Five RCTs used acupuncture, while 1 RCT used acupuncture combined with medication. Four RCTs compared acupuncture with medication, while 2 compared acupuncture with manipulation and physical therapy, respectively. Collectively, the RCTs suggested that there was a benefit effect of improved facial nerve recovery following acupuncture in treating Bell's palsy. However, the GDG notes methodological flaws, including a paucity of information regarding randomization, blinding, and completeness of outcome data, as well as a high risk of bias. In addition, outcome assessment varied between trials and prevented the data from being combined in a meta-analysis. Therefore, these trials do not permit reliable conclusions.

A more recent systematic review of 8 RCTs conducted by Kim et al¹²⁵ identified several important factors, including the stage or severity of the paralysis and when acupuncture treatment began, that were not considered in the previously conducted Chen et al¹²² review. A meta-analysis of 4 trials showed a 7% higher recovery rate on various scales of facial nerve function of acupuncture alone compared with medical therapy alone.¹²⁵ A meta-analysis of 6 trials also showed an 11% higher recovery rate on various scales of facial function with acupuncture plus medical therapy compared with medical therapy alone.¹²⁵ However, the GDG concurs with Chen et al, noting that due to poor randomization, insufficient blinding methods, and high risk of bias, the number and quality of trials are inadequate to draw firm conclusions despite the efforts of Chen et al to combine studies for a meta-analysis. Moreover, the GDG notes that in accordance with the work of Vickers et al,¹²⁶ most of the RCTs may reflect a selective publishing bias that clouds the generalizability of the results.¹²⁷

Acupuncture practiced by well-educated, trained, and experienced practitioners appears relatively safe and of minimal risk.¹²⁸ While acupuncture is popular and most widely practiced in China, most reports on the safety of acupuncture have been published elsewhere.¹²⁸ Prospective studies of European origin estimate the incidence of mild acupuncture-related adverse events at 6.7% to 15%,¹²⁹⁻¹³³ while 2 observational studies estimate the incidence of serious adverse events to be between 0.024% and 2.2%.^{129,130} One systematic review of Chinese acupuncture literature cited a total of 479 cases of adverse events reported in the 115 articles included in the review, but the first authors of the included studies were members of medical departments, court jurisdictions, and police departments, and a minority of the study authors were the acupuncturists who performed the procedure that led to the adverse event.¹²⁸ Whether the variation in reporting of adverse events is a reflection of the relative safety of acupuncture, the underreporting of adverse events, or the limited use of acupuncture remains unclear. Although age has been associated with a poorer response to acupuncture by some, it does not appear to be an independent risk factor for adverse outcomes.¹³⁴ Unsupervised self-treatment is strongly discouraged, as improper technique is often the source of adverse outcomes.¹³⁵ Persistent pleas for increasing safety standards and standardization of clinical acupuncture practices over the past decade in Europe and China (where acupuncture is more widely practiced) suggest acupuncture care delivery can be of variable quality,^{132,134,136,137} despite the very low reported incidence of serious adverse events: just 0.024% in 1 prospective observational study.¹²⁹

In summary, while RCTs are available supporting the use of acupuncture, the GDG downgraded the value of these trials due to the poor quality of the studies. As a result, the GDG was unable to determine the ratio of benefit to harm and therefore could make no recommendation regarding the use of acupuncture. There is an opportunity to share with the patient the uncertainty about this treatment. There is a

need for future, more rigorous RCTs that may overcome the limitations of the current evidence.

STATEMENT 10. PHYSICAL THERAPY: No recommendation can be made regarding the effect of physical therapy in Bell's palsy patients. *No recommendation based on case series and equilibrium of benefit and harm.*

Action Statement Profile

- Aggregate evidence quality: Grade D
- Level of confidence in evidence: Low, due to significant flaws in existing trials
- Benefit: Potential functional and psychological benefit
- Risks, harms, costs: Cost of therapy, time required for therapy
- Benefit-harm assessment: Equilibrium of benefit and harm
- Value judgments: Patients may benefit psychologically from engaging in physical therapy exercises
- Intentional vagueness: None
- Role of patient preferences: Large role for shared decision making
- Exceptions: None
- Policy level: No recommendation
- Differences of opinion: None

Supporting Text

The purpose of this statement is to highlight the uncertainty regarding the role of physical therapy in Bell's palsy. No accepted and consistent definition of physical therapy for Bell's palsy was found throughout the literature. Several therapy modalities were discussed, including thermal treatment, electrotherapy, massage, facial exercise, and biofeedback,^{54,138,139} and most studies combined more than 1 treatment method.¹⁴⁰⁻¹⁴³ Mime therapy, the most common form of physical therapy offered, was defined as including massage, relaxation exercises, inhibition of synkinesis, and emotional expressive exercise.¹⁴⁰⁻¹⁴³ Several small studies suggested physical therapy resulted in some level of improvement in patients experiencing prolonged symptoms.^{54,140-147} Unfortunately, the therapeutic impact of physical therapy in these case reviews is impossible to separate from spontaneous recovery.¹⁴⁶

There was no significant difference in any measure of clinical outcome found between the different rehabilitation techniques described in the literature.¹³⁸ A Cochrane systematic review regarding the incorporation of physical therapy for Bell's palsy concluded that no treatment produced significantly more improvement than was observed in untreated control groups.^{138,139} Risks associated with physical therapy were not identified in the literature reviewed. There is no evidence to support the benefit or harm of physical therapy as a treatment for Bell's palsy.¹³⁹ In addition, the lack of standardized therapy modalities and protocols

hampers the ability of the GDG to make a recommendation regarding the use of physical therapy for patients with Bell's palsy.

STATEMENT 11. PATIENT FOLLOW-UP: 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. *Recommendation based on observational studies with a preponderance of benefit over harm.*

Action Statement Profile

- Aggregate evidence quality: Grade C
- Level of confidence in evidence: High
- Benefit: Reevaluation for alternate diagnoses of facial paralysis, discussion of therapeutic/reconstructive options, psychological support of patient
- Risks, harms, costs: Cost of visit, time dedicated to visit
- Benefit-harm assessment: Preponderance of benefit over harm
- Value judgments: The GDG sought to address the importance of identifying alternate diagnoses in the absence of recovery and potential assessment for rehabilitative options. The GDG recognized a lack of established time for patient follow-up; however, based on the natural history of Bell's palsy, most patients will show complete recovery 3 months after onset.
- Intentional vagueness: Several specialties have the expertise to reevaluate these patients; therefore, the term *facial nerve specialist* is used to indicate the clinician who could most appropriately assess new or worsening symptoms in these patients.
- Role of patient preferences: Small
- Exceptions: None
- Policy level: Recommendation
- Differences of opinion: None

Supporting Text

The purpose of this statement is to guide the management of patients with Bell's palsy who experience (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.¹ A Bell's palsy case in which the patient fails to recover completely within 3 months warrants reevaluation to reconsider the initial diagnosis given the lack of resolution. Reconstructive options may also be considered. The GDG appreciated the possibility of other causes of facial nerve paralysis, such as a cutaneous malignancy, for example, and felt that the 3-month timeframe would be an appropriate window of opportunity to identify other causes. For patients developing new neurologic symptoms or progression of paresis/

paralysis (particularly if there is a history of head and neck cancer), immediate reevaluation is warranted, as a condition other than Bell's palsy could be the source of the paresis/paralysis.

The clinician making the initial diagnosis of Bell's palsy may or may not be the provider best suited to further evaluate the patient. For new neurologic findings, a referral to a neurologist would be appropriate. Alternatively, in the event of concern regarding a head and neck malignancy, a referral to a head and neck specialist would be appropriate. If an otolaryngologist with expertise in facial nerve paralysis were the initial provider, then a return visit for reassessment to the same clinician would be reasonable.

Bell's palsy patients with incomplete facial nerve recovery are at risk for functional and psychological impairment. Long-term functional impairment includes incomplete eye closure, lacrimal dysfunction, nasal airway obstruction, and oral incompetence. To prevent serious ocular complications in severe cases of facial paresis/paralysis with incomplete eye closure, the patient should be referred to an ophthalmologist or eye specialist for evaluation and treatment. In the interim, patients should be educated about strategies for eye closure (ie, taping) and moisturization (ie, eye ointment, artificial tears, humidified eye chambers).

From a psychological standpoint, patients with facial paralysis experience psychosocial dysfunction and diminished quality of life as a result of their appearance. Patients may have difficulty expressing emotion, which may impair their ability to interact in society. As a result of their appearance, patients may be stigmatized. There are a multitude of reconstructive procedures available to improve function and appearance for paralyzed faces. These procedures include eyelid weights, brow lifts, and static and dynamic facial slings, to name a few. The timing of these reconstructive techniques should be discussed with a facial plastic and reconstructive surgeon if the patient has not shown meaningful functional recovery.

Patients may also benefit from some form of support or counseling to deal with the emotional and physical consequences of persistent facial paresis/paralysis. Depression and, rarely, chronic pain should be managed by referral to the appropriate specialist. By encouraging follow-up in a directed manner for patients with incomplete recovery, patients may be able to take advantage of these options.^{25,40,41,148}

This recommendation represents the minimum follow-up required. The GDG recognized there may be value in additional early follow-up for all patients with Bell's palsy for support, monitoring of recovery, and identifying the development of complications or new neurologic findings.

Implementation Considerations

The clinical practice guideline is published as a supplement to *Otolaryngology–Head and Neck Surgery*, which will facilitate reference and distribution. A full-text version of the guideline will be accessible, free of charge, at <http://www.entnet.org>. In addition, all AAO-HNSF guidelines are

now available via the *Otolaryngology–Head and Neck Surgery* application for smartphones and tablets. The guideline will be presented to AAO-HNSF members as a miniseminar at the AAO-HNSF Annual Meeting & OTO EXPO. Existing brochures and publication by the AAO-HNSF will be updated to reflect the guideline's recommendations.

As a supplement to clinicians, an algorithm of the guideline's action statements has been provided (**Figure 1**). The algorithm allows for a more rapid understanding of the guideline's action statements and can be adopted as a quick reference guide to support the implementation of these recommendations.

Research Needs

This guideline was based on the current body of evidence regarding treatment of Bell's palsy. As determined by the GDG's review of the literature, assessment of current clinical practices, and determination of evidence gaps, research needs were determined as follows:

1. Consolidated Standards of Reporting Trials (CONSORT) recommendations should be followed in all RCTs, ensuring valid outcomes measures are employed.¹³⁹ The evaluation of patients prior to treatment, during treatment, immediately following treatment, and again months later, as well as collection of quality-of-life data prior to and following treatment, are all key components for a comprehensive RCT.
2. The timing and outcomes for patients with and without surgical facial nerve decompression need to be compared. More specifically, the surgical approach (ie, middle fossa), age (young vs old), and time to improvement from onset of paresis/paralysis to recovery (with levels of recovery) need to be compared with traditional medical management groups (ie, steroids and/or antivirals).
3. Future clinical trials should compare eye care tactics for patients with Bell's palsy to provide the basis for stronger recommendations (eg, comparison of the frequency of treatment or content of ointment).
4. The benefits and risks of steroid use in children with Bell's palsy need to be better elucidated.
5. Further study is needed to more comprehensively compare the concurrent use of combined steroids and antiviral therapy on Bell's palsy outcomes. Future trials may benefit from designs that allow subgroup analysis to help identify which patients are more likely to benefit from combination therapy.
6. More rigorous randomized controlled trials are needed to address the effectiveness of acupuncture.
7. The efficacy of different physical therapy treatment modalities on asymmetry, symmetry of voluntary movement, and synkinesis in patients with Bell's palsy needs to be elucidated.

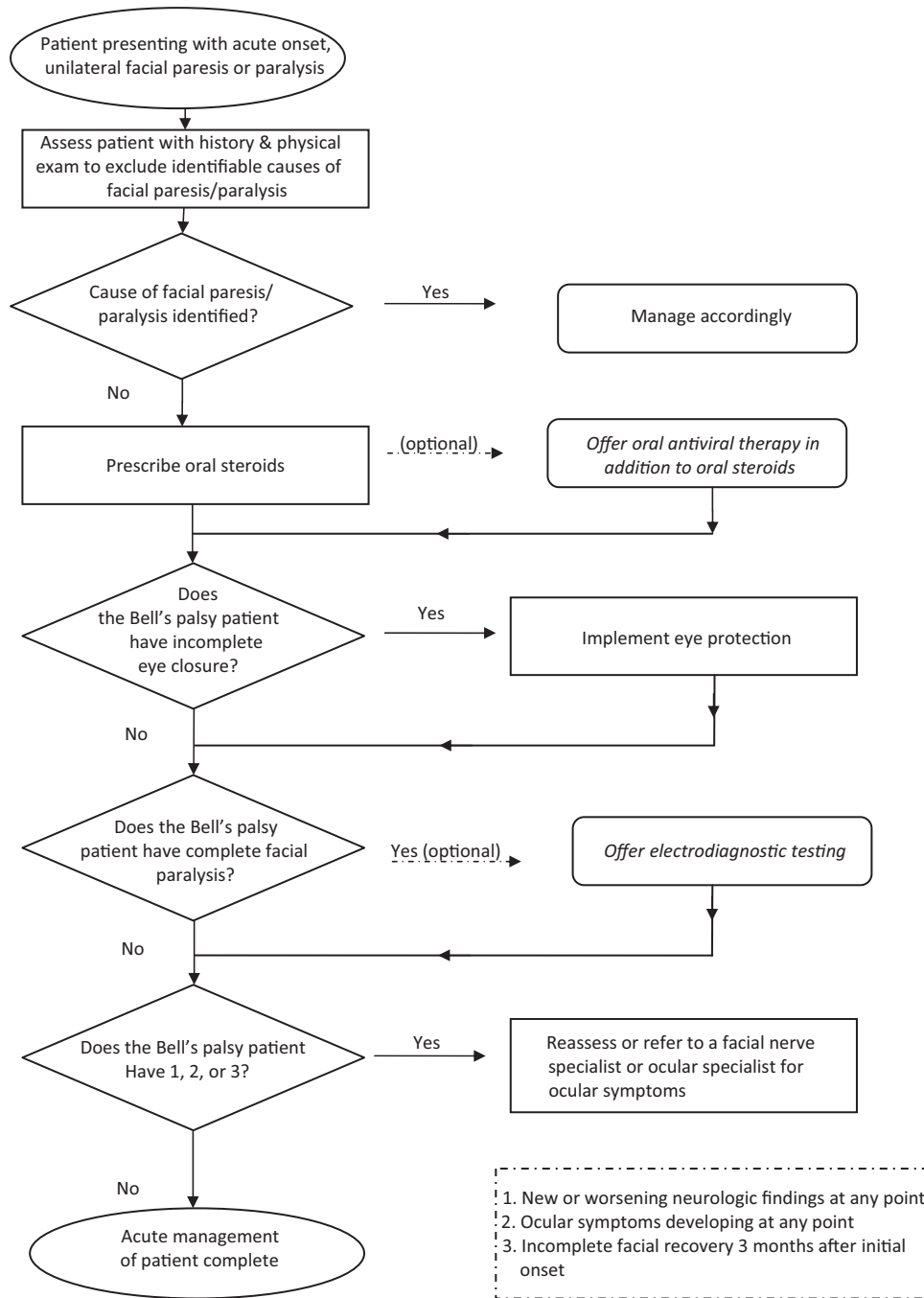


Figure 1. Algorithm of guideline key action statements.

Disclaimer

This clinical practice guideline is provided for informational and educational purposes only. It is not intended as a sole source of guidance in managing Bell’s palsy. Rather, it is designed to assist clinicians by providing an evidence-based framework for decision-making strategies. The guideline is not intended to replace clinical judgment or establish a protocol for all individuals with this condition, and it may not provide the only appropriate approach to diagnosing and managing this program of care. As medical knowledge

expands and technology advances, clinical indicators and guidelines are promoted as conditional and provisional proposals of what is recommended under specific conditions, but they are not absolute. Guidelines are not mandates and do not and should not purport to be a legal standard of care. The responsible physician, in light of all the circumstances presented by the individual patient, must determine the appropriate treatment. Adherence to these guidelines will not ensure successful patient outcomes in every situation. The AAO-HNSF emphasizes that these clinical guidelines should not be deemed to include all proper treatment decisions or methods

of care, or to exclude other treatment decisions or methods of care reasonably directed to obtaining the same results.

© 2013 American Academy of Otolaryngology—Head and Neck Surgery Foundation. All rights reserved.

Author Contributions

Reginald F. Baugh, writer, chair; **Gregory J. Basura**, writer, assistant chair; **Lisa E. Ishii**, writer, assistant chair; **Seth R. Schwartz**, writer, consultant; **Caitlin Murray Drumheller**, writer; **Rebecca Burkholder**, writer; **Nathan A. Deckard**, writer; **Cindy Dawson**, writer; **Colin Driscoll**, writer; **M. Boyd Gillespie**, writer; **Richard K. Gurgel**, writer; **John Halperin**, writer; **Ayesha N. Khalid**, writer; **Kaparaboyana Ashok Kumar**, writer; **Alan Micco**, writer; **Debra Munsell**, writer; **Steven Rosenbaum**, writer; **William Vaughan**, writer.

Disclosures

Competing interests: Seth R. Schwartz received a research grant from the Cochlear Corporation. Colin Driscoll is a surgeon advisor and board member for the Cochlear Corporation, Advanced Bionics, and MED-EL Corporation. M. Boyd Gillespie has received grant support from and is a consultant for Gyrus-Olympus, Medtronic, and Inspire Medical. John Halperin is a stockholder for Abbott, Bristol Myers, Johnson & Johnson, and Merck; an expert witness in medical malpractice cases; and on the editorial board of *Neurology*. Ayesha N. Khalid has received an Acclarent fellowship research training grant. Kaparaboyana Ashok Kumar is a consultant for Southeast Fetal Alcohol Spectrum Disorders Training Center and Meharry Medical College. Alan Micco is on the speakers bureau for Alcon Labs. Steven Rosenbaum is a stockholder for Pfizer, Johnson & Johnson, GlaxoSmithKline, Sanofi, and Celgene Corporation.

Sponsorships: American Academy of Otolaryngology—Head and Neck Surgery Foundation.

Funding Source: American Academy of Otolaryngology—Head and Neck Surgery Foundation.

References

- Peitersen E. Bell's palsy: the spontaneous course of 2,500 peripheral facial nerve palsies of different etiologies. *Acta Otolaryngol Suppl*. 2002;(549):4-30.
- Lockhart P, Daly F, Pitkethly M, Comerford N, Sullivan F. Antiviral treatment for Bell's palsy (idiopathic facial paralysis). *Cochrane Database Syst Rev*. 2009;(4):CD001869.
- Salinas RA, Alvarez G, Daly F, Ferreira J. Corticosteroids for Bell's palsy (idiopathic facial paralysis). *Cochrane Database Syst Rev*. 2010;(3):CD001942.
- Linder TE, Abdelkafy W, Cavero-Vanek S. The management of peripheral facial nerve palsy: "paresis" versus "paralysis" and sources of ambiguity in study designs. *Otol Neurotol*. 2010;31:319-327.
- Fisch U. Surgery for Bell's palsy. *Arch Otolaryngol*. 1981;107:1-11.
- Adour K, Wingerd J, Doty HE. Prevalence of concurrent diabetes mellitus and idiopathic facial paralysis (Bell's palsy). *Diabetes*. 1975;24:449-451.
- Adour KK, Bell DN, Hilsinger RL Jr. Herpes simplex virus in idiopathic facial paralysis (Bell palsy). *JAMA*. 1975;233:527-530.
- Gantz BJ, Rubinstein JT, Gidley P, Woodworth GG. Surgical management of Bell's palsy. *Laryngoscope*. 1999;109:1177-1188.
- Piercy J. Bell's palsy. *BMJ*. 2005;330:1374.
- Ahmed A. When is facial paralysis Bell palsy? Current diagnosis and treatment. *Cleve Clin J Med*. 2005;72:398-401, 405.
- Song MH, Kim J, Jeon JH, et al. Clinical significance of quantitative analysis of facial nerve enhancement on MRI in Bell's palsy. *Acta Otolaryngol*. 2008;128:1259-1265.
- Sittel C, Stennert E. Prognostic value of electromyography in acute peripheral facial nerve palsy. *Otol Neurotol*. 2001;22:100-104.
- Gilden DH. Clinical practice. Bell's palsy. *N Engl J Med*. 2004;351:1323-1331.
- Kim YH, Choi IJ, Kim HM, Ban JH, Cho CH, Ahn JH. Bilateral simultaneous facial nerve palsy: clinical analysis in seven cases. *Otol Neurotol*. 2008;29:397-400.
- Liu J, Li Y, Yuan X, Lin Z. Bell's palsy may have relations to bacterial infection. *Med Hypotheses*. 2009;72:169-170.
- Morgan M, Moffat M, Ritchie L, Collacott I, Brown T. Is Bell's palsy a reactivation of varicella zoster virus? *J Infect*. 1995;30:29-36.
- Unlu Z, Aslan A, Ozbakkaloglu B, Tunger O, Surucuoglu S. Serologic examinations of hepatitis, cytomegalovirus, and rubella in patients with Bell's palsy. *Am J Phys Med Rehabil*. 2003;82:28-32.
- Adour KK, Byl FM, Hilsinger RL Jr, Kahn ZM, Sheldon MI. The true nature of Bell's palsy: analysis of 1,000 consecutive patients. *Laryngoscope*. 1978;88:787-801.
- Engstrom M, Jonsson L, Grindlund M, Stalberg E. House-Brackmann and Yanagihara grading scores in relation to electroneurographic results in the time course of Bell's palsy. *Acta Otolaryngol*. 1998;118:783-789.
- House JW, Brackmann DE. Facial nerve grading system. *Otolaryngol Head Neck Surg*. 1985;93:146-147.
- Peitersen E. Natural history of Bell's palsy. *Acta Otolaryngol Suppl*. 1992;492:122-124.
- Berg T, Axelsson S, Engstrom M, et al. The course of pain in Bell's palsy: treatment with prednisolone and valacyclovir. *Otol Neurotol*. 2009;30:842-846.
- Engstrom M, Berg T, Stjernquist-Desatnik A, et al. Prednisolone and valacyclovir in Bell's palsy: a randomised, double-blind, placebo-controlled, multicentre trial. *Lancet Neurol*. 2008;7:993-1000.
- Mechelse K, Goor G, Huizing EH, et al. Bell's palsy: prognostic criteria and evaluation of surgical decompression. *Lancet*. 1971;2:57-59.
- Valente SM. Visual disfigurement and depression. *Plast Surg Nurs*. 2004;24:140-148.
- Miwa H, Kondo T, Mizuno Y. Bell's palsy-induced blepharospasm. *J Neurol*. 2002;249:452-454.
- Bracewell RM. The treatment of Bell's palsy. *N Engl J Med*. 2007;357:1598-1607.
- Lyons CJ, McNab AA. Symptomatic nocturnal lagophthalmos. *Aust N Z J Ophthalmol*. 1990;18:393-396.
- Hayashi A, Maruyama Y, Okada E, Ogino A. Use of a suture anchor for correction of ectropion in facial paralysis. *Plast Reconstr Surg*. 2005;115:234-239.

30. Ramakrishnan Y, Alam S, Kotecha A, Gillett D, D'Souza A. Reanimation following facial palsy: present and future directions. *J Laryngol Otol*. 2010;124:1146-1152.
31. Brandenburg NA, Annegers JF. Incidence and risk factors for Bell's palsy in Laredo, Texas: 1974-1982. *Neuroepidemiology*. 1993;12:313-325.
32. Katusic SK, Beard CM, Wiederholt WC, Bergstralh EJ, Kurland LT. Incidence, clinical features, and prognosis in Bell's palsy, Rochester, Minnesota, 1968-1982. *Ann Neurol*. 1986;20:622-627.
33. Monini S, Lazzarino AI, Iacolucci C, Buffoni A, Barbara M. Epidemiology of Bell's palsy in an Italian Health District: incidence and case-control study. *Acta Otorhinolaryngol Ital*. 2010;30:198.
34. Rowlands S, Hooper R, Hughes R, Burney P. The epidemiology and treatment of Bell's palsy in the UK. *Eur J Neurol*. 2002;9:63-67.
35. Tsai HS, Chang LY, Lu CY, et al. Epidemiology and treatment of Bell's palsy in children in northern Taiwan. *J Microbiol Immunol Infect*. 2009;42:351-356.
36. Lunan R, Nagarajan L. Bell's palsy: a guideline proposal following a review of practice. *J Paediatr Child Health*. 2008;44:219-220.
37. Rowhani-Rahbar A, Baxter R, Rasgon B, et al. Epidemiologic and clinical features of Bell's palsy among children in Northern California. *Neuroepidemiology*. 2012;38:252-258.
38. Katz A, Sergienko R, Dior U, Wiznitzer A, Kaplan DM, Sheiner E. Bell's palsy during pregnancy: is it associated with adverse perinatal outcome? *Laryngoscope*. 2011;121:1395-1398.
39. Savadi-Oskouei D, Abedi A, Sadeghi-Bazargani H. Independent role of hypertension in Bell's palsy: a case-control study. *Eur Neurol*. 2008;60:253-257.
40. Byrne PJ. Importance of facial expression in facial nerve rehabilitation. *Curr Opin Otolaryngol Head Neck Surg*. 2004;12:332-335.
41. Ishii L, Godoy A, Encarnacion CO, Byrne PJ, Boahene KD, Ishii M. Not just another face in the crowd: society's perceptions of facial paralysis. *Laryngoscope*. 2012;122:533-538.
42. Rosenfeld RM, Shiffman RN, Robertson P. Clinical practice guideline development manual, third edition: a quality-driven approach for translating evidence into action. *Otolaryngol Head Neck Surg*. 2013;148(1)(suppl):S1-S55.
43. Shiffman RN, Shekelle P, Overhage JM, Slutsky J, Grimshaw J, Deshpande AM. Standardized reporting of clinical practice guidelines: a proposal from the Conference on Guideline Standardization. *Ann Intern Med*. 2003;139:493-498.
44. Shiffman RN, Dixon J, Brandt C, et al. The GuideLine Implementability Appraisal (GLIA): development of an instrument to identify obstacles to guideline implementation. *BMC Med Inform Decis Making*. 2005;5:23.
45. Classifying recommendations for clinical practice guidelines. *Pediatrics*. 2004;114:874-877.
46. Centre for Evidence Based Medicine. *The Oxford 2011 Levels of Evidence*. 2001. <http://www.cebm.net/index.aspx?o=5653>
47. Choudhry NK, Stelfox HT, Detsky AS. Relationships between authors of clinical practice guidelines and the pharmaceutical industry. *JAMA*. 2002;287:612-617.
48. Detsky AS. Sources of bias for authors of clinical practice guidelines. *CMAJ*. 2006;175:1033, 1035.
49. Yanagihara N. Incidence of Bell's palsy. *Ann Otol Rhinol Laryngol Suppl*. 1988;137:3-4.
50. Tiemstra JD, Khatkhate N. Bell's palsy: diagnosis and management. *Am Fam Physician*. 2007;76:997-1002.
51. May M, Schaitkin BM. Tumors involving the facial nerve. In: *The Facial Nerve*. 2nd ed. New York, NY: Thieme; 2000.
52. Halperin JJ, Golightly M. Lyme borreliosis in Bell's palsy. Long Island Neuroborreliosis Collaborative Study Group. *Neurology*. 1992;42:1268-1270.
53. Centers for Disease Control and Prevention. Lyme disease data. 2012. <http://www.cdc.gov/lyme/stats/index.html>. Accessed March 27, 2013.
54. Holland J. Bell's palsy. *Clin Evid (Online)*. 2008;2008:1204.
55. Kinoshita T, Ishii K, Okitsu T, Okudera T, Ogawa T. Facial nerve palsy: evaluation by contrast-enhanced MR imaging. *Clin Radiol*. 2001;56:926-932.
56. Mahoney E, Agarwal S, Li B, et al. Evidence-based guidelines are equivalent to a liberal computed tomography scan protocol for initial patient evaluation but are associated with decreased computed tomography scan use, cost, and radiation exposure. *J Trauma Acute Care Surg*. 2012;73:573-579.
57. Konen E, Konen O, Katz M, Levy Y, Rozenman J, Hertz M. Are referring clinicians aware of patients at risk from intravenous injection of iodinated contrast media? *Clin Radiol*. 2002;57:132-135.
58. Thrall JH. Radiation exposure in CT scanning and risk: where are we? *Radiology*. 2012;264:325-328.
59. American College of Radiology. ACR statement on recent studies regarding CT scans and increased cancer risk. http://gm.acr.org/MainMenuCategories/media_room/FeaturedCategories/PressReleases/StatementonRecentStudiesRegardingCTScans.aspx. Accessed March 5, 2013.
60. Bredon GE, Cody DT, Weiland LH. Facial palsy: unusual etiology. *Laryngoscope*. 1977;87:26-34.
61. Gronseth GS, Paduga R. Evidence-based guideline update: steroids and antivirals for Bell palsy: report of the Guideline Development Subcommittee of the American Academy of Neurology. *Neurology*. 2012;79:2209-2213.
62. Sullivan FM, Swan IR, Donnan PT, et al. Early treatment with prednisolone or acyclovir in Bell's palsy. *N Engl J Med*. 2007;357:1598-1607.
63. Katzung BG, Masters SB, Trevor AJ, eds. *Basic & Clinical Pharmacology*. 12th ed. New York, NY: McGraw-Hill.
64. Sweetman SC, ed. *Martindale: The Complete Drug Reference*. 34th ed. London, UK: Pharmaceutical Press; 2004.
65. Pitaro J, Waissbluth S, Daniel SJ. Do children with Bell's palsy benefit from steroid treatment? A systematic review. *Int J Pediatr Otorhinolaryngol*. 2012;76:921-926.
66. Atzema C, Goldman RD. Should we use steroids to treat children with Bell's palsy? *Can Fam Physician*. 2006;52:313-314.
67. Pitaro K, Waissbluth S, Daniel SJ. Steroids in pediatric Bell palsy: a systematic review. *Otolaryngol Head Neck Surg*. 2011;145:246.
68. Salman MS, MacGregor DL. Should children with Bell's palsy be treated with corticosteroids? A systematic review. *J Child Neurol*. 2001;16:565-568.

69. Kawaguchi K, Inamura H, Abe Y, et al. Reactivation of herpes simplex virus type 1 and varicella-zoster virus and therapeutic effects of combination therapy with prednisolone and valacyclovir in patients with Bell's palsy. *Laryngoscope*. 2007;117:147-156.
70. Alberton DL, Zed PJ. Bell's palsy: a review of treatment using antiviral agents. *Ann Pharmacother*. 2006;40:1838-1842.
71. Allen D, Dunn L. Aciclovir or valaciclovir for Bell's palsy (idiopathic facial paralysis). *Cochrane Database Syst Rev*. 2004;(3):CD001869.
72. Browning GG. Bell's palsy: a review of three systematic reviews of steroid and anti-viral therapy. *Clin Otolaryngol*. 2010;35:56-58.
73. de Almeida JR, Al Khabori M, Guyatt GH, et al. Combined corticosteroid and antiviral treatment for Bell palsy: a systematic review and meta-analysis. *JAMA*. 2009;302:985-993.
74. Yuen MC, Crawford I. Towards evidence based emergency medicine: best BETs from the Manchester Royal Infirmary. Bell's palsy and acyclovir. *Emerg Med J*. 2002;19:326-327.
75. Axelsson S, Berg T, Jonsson L, Engstrom M, Kanerva M, Stjernquist-Desatnik A. Bell's palsy—the effect of prednisolone and/or valaciclovir versus placebo in relation to baseline severity in a randomised controlled trial. *Clin Otolaryngol*. 2012;37:283-290.
76. Quant EC, Jeste SS, Muni RH, Cape AV, Bhussar MK, Peleg AY. The benefits of steroids versus steroids plus antivirals for treatment of Bell's palsy: a meta-analysis. *BMJ*. 2009;339:b3354.
77. Goudakos JK, Markou KD. Corticosteroids vs corticosteroids plus antiviral agents in the treatment of Bell palsy: a systematic review and meta-analysis. *Arch Otolaryngol Head Neck Surg*. 2009;135:558-564.
78. Numthavaj P, Thakkinstian A, Dejthevaporn C, Attia J. Corticosteroid and antiviral therapy for Bell's palsy: a network meta-analysis. *BMC Neurol*. 2011;11:1.
79. Hato N, Yamada H, Kohno H, et al. Valacyclovir and prednisolone treatment for Bell's palsy: a multicenter, randomized, placebo-controlled study. *Otol Neurotol*. 2007;28:408-413.
80. Shahidullah M, Haque A, Islam MR, et al. Comparative study between combination of famciclovir and prednisolone with prednisolone alone in acute Bell's palsy. *Mymensingh Med J*. 2011;20:605-613.
81. Bhatti MT, Schiffman JS, Pass AF, Tang RA. Neuro-ophthalmologic complications and manifestations of upper and lower motor neuron facial paresis. *Curr Neurol Neurosci Rep*. 2010;10:448-458.
82. Rosenberg JB, Eisen LA. Eye care in the intensive care unit: narrative review and meta-analysis. *Crit Care Med*. 2008;36:3151-3155.
83. Sturrock GD. Nocturnal lagophthalmos and recurrent erosion. *Br J Ophthalmol*. 1976;60:97-103.
84. Betharia SM, Sharma V. Inverse Bell's phenomenon observed following levator resection for blepharoptosis. *Graefes Arch Clin Exp Ophthalmol*. 2006;244:868-870.
85. Calder L, Balasubramanian S, Stiell I. Lack of consensus on corneal abrasion management: results of a national survey. *CJEM*. 2004;6:402-407.
86. Flynn CA, D'Amico F, Smith G. Should we patch corneal abrasions? A meta-analysis. *J Fam Pract*. 1998;47:264-270.
87. What to do about Bell's palsy: recovery takes time, patience, proper eye care, and maybe a medication. *Harv Womens Health Watch*. 2010;17:5.
88. Na KS, Yang SW. Two cases of inverse Bell's phenomenon following levator resection: a contemplation of the mechanism. *Eur J Ophthalmol*. 2009;19:285-287.
89. Lorch M, Teach SJ. Facial nerve palsy: etiology and approach to diagnosis and treatment. *Pediatr Emerg Care*. 2010;26:763-773.
90. Mitchell J. Ocular emergencies: Sec. 20 emergency medicine. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*. New York, NY: McGraw-Hill Education; 2004.
91. Murthy JM, Saxena AB. Bell's palsy: treatment guidelines. *Ann Indian Acad Neurol*. 2011;14(suppl 1):S70-S72.
92. Rodrigues de Araujo M. Management of Bell's palsy: a report of 2 cases. *J Can Dent Assoc*. 2008;74(9):823-837.
93. College of Optometrists. Facial palsy (including Bell's palsy). In: *Clinical Management Guidelines*. 2008. <http://www.college-optometrists.org/en/utilities/document-summary.cfm/docid/63AD5CC8-EB0F-41CB-89508DC2D43560FB>. Accessed February 13, 2013.
94. Turner A, Rabiou M. Patching for corneal abrasion. *Cochrane Database Syst Rev*. 2006;(2):CD004764.
95. Seiff SR, Chang JS Jr. The staged management of ophthalmic complications of facial nerve palsy. *Ophthal Plast Reconstr Surg*. 1993;9:241-249.
96. Zaidi FH, Gregory-Evans K, Acheson JF, Ferguson V. Familial Bell's palsy in females: a phenotype with a predilection for eyelids and lacrimal gland. *Orbit*. 2005;24:121-124.
97. Adour KK, Sheldon MI, Kahn ZM. Maximal nerve excitability testing versus neuromyography: prognostic value in patients with facial paralysis. *Laryngoscope*. 1980;90:1540-1547.
98. Baba S, Kondo K, Kanaya K, Ushio M, Tojima H, Yamasoba T. Bell's palsy in children: relationship between electroneurography findings and prognosis in comparison with adults. *Otol Neurotol*. 2011;32:1554-1558.
99. Sinha PK, Keith RW, Pensak ML. Predictability of recovery from Bell's palsy using evoked electromyography. *Am J Otol*. 1994;15:769-771.
100. Mantsopoulos K, Psillas G, Psychogios G, Brase C, Iro H, Constantinidis J. Predicting the long-term outcome after idiopathic facial nerve paralysis. *Otol Neurotol*. 2011;32:848-851.
101. Gilliatt RW, Taylor JC. Electrical changes following section of the facial nerve. *Proc R Soc Med*. 1959;53:1080-1083.
102. Tojima H, Aoyagi M, Inamura H, Koike Y. Clinical advantages of electroneurography in patients with Bell's palsy within two weeks after onset. *Acta Otolaryngol Suppl*. 1994; 511:147-149.
103. Sillman JS, Niparko JK, Lee SS, Kileny PR. Prognostic value of evoked and standard electromyography in acute facial paralysis. *Otolaryngol Head Neck Surg*. 1992;107:377-381.
104. 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;56:830-836.

105. McAllister K, Walker D, Donnan PT, Swan I. Surgical interventions for the early management of Bell's palsy. *Cochrane Database Syst Rev*. 2011;(2):CD007468.
106. Fisch U, Esslen E. Total intratemporal exposure of the facial nerve: pathologic findings in Bell's palsy. *Arch Otolaryngol*. 1972;95:335-341.
107. Gantz BJ, Gmur A, Fisch U. Intraoperative evoked electromyography in Bell's palsy. *Am J Otolaryngol*. 1982;3:273-278.
108. Yamamoto E, Fisch U. Experimentally induced facial nerve compression in cats. *Acta Otolaryngol*. 1975;79:390-395.
109. Ge XX, Spector GJ. Labyrinthine segment and geniculate ganglion of facial nerve in fetal and adult human temporal bones. *Ann Otol Rhinol Laryngol Suppl*. 1981;90:1-12.
110. Adour KK, Swanson PJ Jr. Facial paralysis in 403 consecutive patients: emphasis on treatment response in patients with Bell's palsy. *Trans Am Acad Ophthalmol Otolaryngol*. 1971;75:1284-1301.
111. May M, Klein SR, Taylor FH. Indications for surgery for Bell's palsy. *Am J Otol*. 1984;5:503-512.
112. Aoyagi M, Koike Y, Ichige A. Results of facial nerve decompression. *Acta Otolaryngol Suppl*. 1988;446:101-105.
113. Yanagihara N, Hato N, Murakami S, Honda N. Transmastoid decompression as a treatment of Bell palsy. *Otolaryngol Head Neck Surg*. 2001;124:282-286.
114. Kim IS, Shin SH, Kim J, Lee WS, Lee HK. Correlation between MRI and operative findings in Bell's palsy and Ramsay Hunt syndrome. *Yonsei Med J*. 2007;48:963-968.
115. Bodenez C, Bernat I, Willer JC, Barre P, Lamas G, Tankere F. Facial nerve decompression for idiopathic Bell's palsy: report of 13 cases and literature review. *J Laryngol Otol*. 2010;124:272-278.
116. House WF, Shelton C. Middle fossa approach for acoustic tumor removal. *Otolaryngol Clin North Am*. 1992;25:347-359.
117. Brackmann DE, House JR III, Hitselberger WE. Technical modifications to the middle fossa craniotomy approach in removal of acoustic neuromas. *Am J Otol*. 1994;15:614-619.
118. Glasscock ME, McKennan KX, Levine SC. Acoustic neuroma surgery: the results of hearing conservation surgery. *Laryngoscope*. 1987;97:785-789.
119. Shelton C, Brackmann DE, House WF, Hitselberger WE. Middle fossa acoustic tumor surgery: results in 106 cases. *Laryngoscope*. 1989;99:405-408.
120. Smouha E, Toh E, Schaitkin BM. Surgical treatment of Bell's palsy: current attitudes. *Laryngoscope*. 2011;121:1965-1970.
121. Barr JS, Katz KA, Hazen A. Surgical management of facial nerve paralysis in the pediatric population. *J Pediatr Surg*. 2011;46:2168-2176.
122. Chen N, Zhou M, He L, Zhou D, Li N. Acupuncture for Bell's palsy. *Cochrane Database Syst Rev*. 2010;(8):CD002914.
123. Cheng KJ. Neuroanatomical basis of acupuncture treatment for some common illnesses. *Acupunct Med*. 2009;27:61-64.
124. Zheng H, Li Y, Chen M. Evidence based acupuncture practice recommendations for peripheral facial paralysis. *Am J Chin Med*. 2009;37:35-43.
125. Kim JI, Lee MS, Choi TY, Lee H, Kwon HJ. Acupuncture for Bell's palsy: a systematic review and meta-analysis. *Chin J Integr Med*. 2012;18:48-55.
126. Vickers A, Goyal N, Harland R, Rees R. Do certain countries produce only positive results? A systematic review of controlled trials. *Controlled Clin Trials*. 1998;19:159-166.
127. Cumberworth A, Mabvuure NT, Norris JM, Watts S. Is acupuncture beneficial in the treatment of Bell's palsy? Best evidence topic (BET). *Int J Surg*. 2012;10:310-312.
128. Zhang J, Shang H, Gao X, Ernst E. Acupuncture-related adverse events: a systematic review of the Chinese literature. *Bull World Health Organ*. 2010;88:915C-921C.
129. Endres HG, Molsberger A, Lungenhausen M, Trampisch HJ. An internal standard for verifying the accuracy of serious adverse event reporting: the example of an acupuncture study of 190,924 patients. *Eur J Med Res*. 2004;9:545-551.
130. Witt CM, Pach D, Brinkhaus B, et al. Safety of acupuncture: results of a prospective observational study with 229,230 patients and introduction of a medical information and consent form. *Forsch Komplementmed*. 2009;16:91-97.
131. Norheim AJ. Adverse effects of acupuncture: a study of the literature for the years 1981-1994. *J Altern Complement Med*. 1996;2:291-297.
132. White A, Hayhoe S, Hart A, Ernst E. Adverse events following acupuncture: prospective survey of 32 000 consultations with doctors and physiotherapists. *BMJ*. 2001;323:485-486.
133. Macpherson H, Scullion A, Thomas KJ, Walters S. Patient reports of adverse events associated with acupuncture treatment: a prospective national survey. *Qual Saf Health Care*. 2004;13:349-355.
134. Park SU, Ko CN, Bae HS, et al. Short-term reactions to acupuncture treatment and adverse events following acupuncture: a cross-sectional survey of patient reports in Korea. *J Altern Complement Med*. 2009;15:1275-1283.
135. Yamashita H, Tsukayama H, White AR, Tanno Y, Sugishita C, Ernst E. Systematic review of adverse events following acupuncture: the Japanese literature. *Complement Ther Med*. 2001;9:98-104.
136. He W, Zhao X, Li Y, Xi Q, Guo Y. Adverse events following acupuncture: a systematic review of the Chinese literature for the years 1956-2010. *J Altern Complement Med*. 2012;18:892-901.
137. White A. A cumulative review of the range and incidence of significant adverse events associated with acupuncture. *Acupunct Med*. 2004;22:122-133.
138. Pereira LM, Obara K, Dias JM, Menacho MO, Lavado EL, Cardoso JR. Facial exercise therapy for facial palsy: systematic review and meta-analysis. *Clin Rehabil*. 2011;25:649-658.
139. Teixeira LJ, Valbuza JS, Prado GF. Physical therapy for Bell's palsy (idiopathic facial paralysis). *Cochrane Database Syst Rev*. 2011;(12):CD006283.
140. Beurskens CH, Heymans PG. Positive effects of mime therapy on sequelae of facial paralysis: stiffness, lip mobility, and social and physical aspects of facial disability. *Otol Neurotol*. 2003;24:677-681.
141. Beurskens CH, Heymans PG. Physiotherapy in patients with facial nerve paresis: description of outcomes. *Am J Otolaryngol*. 2004;25:394-400.

142. Beurskens CH, Heymans PG. Mime therapy improves facial symmetry in people with long-term facial nerve paresis: a randomized controlled trial. *Aust J Physiother.* 2006;52:177-183.
143. Beurskens CH, Heymans PG, Oostendorp RA. Stability of benefits of mime therapy in sequelae of facial nerve paresis during a 1-year period. *Otol Neurotol.* 2006;27:1037-1042.
144. Cardoso JR, Teixeira EC, Moreira MD, Favero FM, Fontes SV, Bulle de Oliveira AS. Effects of exercises on Bell's palsy: systematic review of randomized controlled trials. *Otol Neurotol.* 2008;29:557-560.
145. Barbara M, Antonini G, Vestri A, Volpini L, Monini S. Role of Kabat physical rehabilitation in Bell's palsy: a randomized trial. *Acta Otolaryngol.* 2010;130:167-172.
146. Manikandan N. Effect of facial neuromuscular re-education on facial symmetry in patients with Bell's palsy: a randomized controlled trial. *Clin Rehabil.* 2007;21:338-343.
147. Nakamura K, Toda N, Sakamaki K, Kashima K, Takeda N. Biofeedback rehabilitation for prevention of synkinesis after facial palsy. *Otolaryngol Head Neck Surg.* 2003;128:539-543.
148. Coulson SE, O'Dwyer N J, Adams RD, Croxson GR. Expression of emotion and quality of life after facial nerve paralysis. *Otol Neurotol.* 2004;25:1014-1019.