Appendix 1 (as supplied by the authors): Complete clinical practice guideline for the management of Bell palsy

John R. de Almeida, MD, MSc, FRCSC

Gordon H. Guyatt, MD, MSc, FRCPC

Sachin Sud, MD, MSc, FRCSC

Joanne Dorion, PT, BScPT

Michael D. Hill, MD, FRCPC

Michael R.Kolber, MD, MSc, CCFP

Jane Lea, MD, FRCSC

Sylvia Loong, Reg PT

Balvinder K. Somogyi, BSW

Brian D. Westerberg, MD, FRCSC

Chris White, MD, FRCPC

Joseph M. Chen, MD, FRCSC

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

Introduction

Bell's palsy is an acute onset idiopathic weakness or paralysis of the face of peripheral nerve origin. Other peripheral causes of facial weakness or paralysis include inflammation of the ear or temporal bone (otitis media, mastoiditis, cholesteatoma), viral infections (herpes zoster oticus or Ramsay Hunt syndrome, Lyme disease), granulomatous diseases (sarcoidosis, Melkersson-Rosenthal syndrome), and neoplasms of the cerebellopontine angle, temporal bone, or parotid gland.

Bell's palsy affects 20-30 persons per 100,000 on an annual basis and 1 in 60 individuals will be affected over the course of their lifetime.[1,2] The condition is responsible for almost three-quarters of all acute facial palsies.[1] There is no gender predilection, although it has been associated with pregnancy.[1] Studies have also suggested an association between Bell's palsy and diabetes.[3,4] Certain vaccinations have also been associated with the development of this condition including an intranasal influenza vaccine[5] and a Swedish influenza H1N1 vaccine.[6] To date, there has been no association with North American vaccines.[7,8]

Roughly 75% of patients with Bell's palsy believe they are having a stroke.[9] For the clinician, it is important to differentiate peripheral from central causes for facial weakness. Patients with peripheral (lower motor neuron) facial nerve palsies demonstrate weakness of both the upper and lower half of the face. However, due to upper motor neuron innervation from both cerebral hemispheres, central facial nerve palsies

demonstrate paresis or paralysis only on the lower quadrant of the face with sparing of eye closure and forehead wrinkle movements.[1,3,10]

The major etiology of Bell's palsy is believed to be a viral infection of the facial nerve by the herpes simplex virus.[11-13] In one study, herpes virus DNA was identified in the endoneurial fluid at the time of surgical decompression of the nerve.[14] As a result of this viral infection the facial nerve swells and is compressed in its canal as it courses through the temporal bone causing an ischemic injury to the nerve.

In development of these guidelines, a number of prognostic factors were considered to be relevant. Classifying Bell's palsy according to the severity of weakness provides prognostic information that may guide treatment. The majority of Bell's palsy patients (70%) have a complete facial paralysis, while 30% have partial weakness or paresis.[15] There are several instruments available to quantify the severity of Bell's palsy.[15] The House-Brackmann (**Table 1**) and Sunnybrook scales are the most commonly used instruments.[16,17] These validated instruments objectively grade the extent of facial muscle paresis allow treating clinicians to monitor patients and to counsel them regarding the likelihood of recovery. Patients with mild to moderate paresis (the equivalent of House-Brackmann grades II-IV) have better rates of recovery than those with severe to complete paresis (House-Brackmann grades V-VI).[18] In a large single-institution cohort study, recovery rates of 61% and 94% were documented in patients with complete and incomplete paralysis respectively.[19]

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

Electroneuronography (ENoG) may also provide additional prognostic information. This electrical stimulation test compares the affected or paralyzed side to the unaffected side. In one study, only 42% of patients with a greater than 90% degeneration of the facial nerve function on affected side had a good recovery (House-Brackmann grade 1 or 2).[20] Historically, ENoG has been used to select patients with a poor prognosis who may be candidates for surgical decompression of the facial nerve.

Other important clinical outcomes exist in Bell's palsy. Up to 16% will have residual involuntary movements and/or synkinesis,[15] while others may have abnormal lacrimation with salivation (crocodile tears). Failure to protect the cornea among patients who are unable to blink adequately may result in corneal ulceration and permanent visual impairment. Those with residual deficits may have a long-term reduced quality of life and psychological distress.[4,21,22]

Previous guidelines have made recommendations for the treatment of Bell's palsy. A Quality Standards Subcommittee of the American Academy of Neurology issued a practice parameter in 2001 concluding that there was not sufficient evidence to support the use of corticosteroids, antiviral agents, or surgical decompression for Bell's palsy.[23] A recent update of this guideline, however, advocated the use of cortisteroids and concluded that antivirals may be of modest beneft.[24] Of two other treatment guidelines, one concluded that there was insufficient evidence to give corticosteroids in children[25] and the other suggested use of corticosteroids based on a narrative review of the available evidence.[26] Recent systematic reviews for exercise and electrostimulation physiotherapy[27] as well as surgical decompression[23] have provided further insight. This

growing body of literature was the impetus for developing guidelines for the treatment of Bell's palsy. This guideline will review the evidence for the medical treatment of Bell's palsy with corticosteroids and antivirals, facial exercise and electrostimulation physiotherapy, decompression surgery, the need for eye protective measures, and the need for further investigation and specialist referral in persisting and progressive cases. Recommendations were made using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) system for the development of clinical practice guideline recommendations.[28-31] Because of the important prognostic implications of initial severity of paresis/paralysis, this guideline panel made separate recommendations based the degree of facial weakness at presentation (mild to moderate paresis versus severe to complete paresis). This guideline is aimed at all health care providers but primarily at frontline or primary care physicians who treat Bell's palsy.

Methods

Selection and Organization of the Panel

A working group of eleven members was assembled under the auspices of Canadian Society of Otolaryngology and Canadian Neurological Sciences Federation. The group consisted of three methodologists (JDA, SS, GG), three Otolaryngology - Head and Neck Surgeons (JDA, JC, BDW), two Neurologists (MDH, CW), one Family Physician (MK), two Facial Nerve Therapists (JD, SL), and one patient (BKS). An

additional methodologist (JL) was involved in the review process, but not the guideline recommendations. Panelists were selected based on a demonstrated academic interest in Bell's palsy and/or by recommendation of relevant national societies. The patient panel member was selected by invitation from one of the panelists. Guidelines were developed through a series of four conference calls and multiple email correspondences between January, 2010 and June, 2012. GG chaired the panel and JDA was in charge of organizational aspects of the process.

Defining the Clinical Questions and Important Outcomes

Content experts in the panel including the Otolaryngology-Head and Neck Surgeons, Neurologists, Family Physician, and Facial Therapists were involved in drafting a list of clinical questions and subject topics to be addressed by the guideline (**Table 2**). The working group identified outcomes deemed important to patients at the outset of the guideline development process (**Table 3)**. Each outcome was rated by each panel member on a scale from 1-9 as suggested by the GRADE working group.[29] In making ratings, panelists were instructed to, on the basis of their personal or clinical experience, to take the perspective of a patient who has Bell's palsy. Scores from 1-3 were classified as not important, scores from 4-6 were classified as important but not critical, scores from 7-9 were classified as critical. Evidence for outcomes were evaluated by systematic review and then used in making recommendations. The primary outcome

for this analysis is unsatisfactory facial motor recovery (House-Brackmann score of grade 3-6 or equivalent). Although other facial outcomes such as synkinesis and autonomic dysfunction may be considered unsatisfactory recovery, we have defined these as separate outcomes from the primary outcome.

Data Collection, Synthesis, and Meta-analysis

Relevant systematic reviews were identified for each clinical question by searching on PubMed/MEDLINE through June 2011. All identified systematic reviews were presented to the group in summary format including number of included studies, number of patients, outcomes considered, relevant findings and methodologic limitations. One systematic review for each subject area was selected. Reviews were selected based on methodologic quality, number of studies included, and most recently published. For subject areas in which no systematic review was available, new reviews were conducted. Updated reviews were performed for reviews published more than 2 years prior to the start of the guideline development process.

Previous systematic reviews were chosen by the group for the use of corticosteroid, antiviral agent, and combined treatment (recommendations 1-6);[32] the use of exercise physiotherapy and electrostimulation physiotherapy (recommendations 7-9);[27] and the use of surgical decompresssion in the treatment of Bell's palsy (recommendation 10).[23] Updates to these systematic reviews were performed using the same search strategy and data extraction and synthesis methods as defined in the primary review and

included studies up to June 2011. New systematic reviews of randomized controlled trials or observational studies were conducted for eye protective measures (recommendation 11), specialist referral (recommendation 12), investigation for malignancy (recommendation 13), values and preferences, and cost-resource use.

The searches for new studies included MEDLINE, EMBASE, CENTRAL, PsychInfo, CINAHL, and clinicaltrials.gov through January 2011. Bibliographies of relevant articles were also searched. Included studies for all reviews were RCT's with the exception of the surgical decompression review, as RCT's for surgical decompression were unavailable. For recommendations 11-13 we did not identify any randomized controlled studies or observational studies. In the absence of evidence from randomized controlled trials or observational studies, recommendations were based on panelists' personal experience.

For new or updated reviews, two reviewers (2 of JDA, CW, JL, SS) screened all studies for eligibility and extracted data. Disagreements were resolved by consensus. For each review, the setting, patient eligibility, number of patients, treatment in each arm, and outcomes considered in each study was recorded, and data was gathered for each of the chosen outcomes. Risk of bias was assessed using Cochrane systematic review guidelines, which assess adequacy of random sequence generation, allocation concealment, blinding, loss to follow-up, selective reporting, or other biases.[33] Meta-analyses were conducted using RevMan Software (version 5.0). Heterogeneity was assessed using the I^2 statistic.

For each treatment or intervention, we created summary tables summarizing the available evidence as suggested by the GRADE working group.[29] These summary tables included information for all identified desirable and undesirable outcomes. For each outcome, we recorded the number of studies, number of patients, evidence ratings, relative risk with treatment, baseline risk, intervention group risk, number needed to treat, quality of evidence, and outcome importance.

Evaluating Confidence in Effect Estimates (Quality of Evidence)

Confidence in effect estimates was assessed based on guidelines suggested by the GRADE working group.[29] In short, the GRADE group suggests that the confidence in effect estimates for any outcome can be given one of four grades (very low, low, moderate, or high). Factors that can decrease the confidence in effect estimate include risk of bias, inconsistency of results, indirectness of evidence, imprecision, and publication bias. Factors that increase the confidence in an effect estimate include a large magnitude of effect, plausible confounding that would reduce a demonstrated effect, and a dose-response gradient. The overall confidence for any given recommendation is based on evaluating the confidence in effect estimates for all the outcomes of interest. We rated the overall confidence across all of these outcomes as the lowest confidence of effect estimate for any critical outcome.

Making Recommendations (GRADE)

Recommendations were made using the GRADE system [28-31] which categorizes recommendations as strong or weak. The strength of recommendations are based on four factors:[28] the balance of desirable and undesirable consequences, the confidence in effect estimate for each of the critical and important outcomes,[29] variability in patient values and preferences and resource use.[31]

Recommendations were made based on nominal group techniques whereby the group members met by conference call, voiced their opinions, and reflected on the opinions of others. At the end of the discussion and after considering the above four factors, the group made a consensus recommendation—either strong or weak in favour or against or no recommendation. When consensus was elusive (recommendations 5 and 7), the recommendations were submitted to a blind vote and recommendations were based on a process previously described.[34] Each working group member voted in one of five categories (strong in favor, weak in favor, no recommendation, weak against, strong against). For a recommendation to be in favour at least 50% of group members were required to vote in favour, with no more than 20% voting against. For a recommendation to be strong recommendation, at least 70% of the group were required to endorse it as a strong recommendation.

Conflicts of Interest

Group members disclosed financial and intellectual conflicts of interest. Each potential conflict of interest (COI) was evaluated to determine whether the COI was acceptable or unacceptable. The following criteria were determined a priori and served as exclusion criteria from panel involvement.

- *Significant equity holding in company related to the subject matter of the guidelines*
- *Significant income originating from a company related to the subject matter of the guidelines*
- *Refusal to avoid financial involvement in industries tied to therapies for which recommendations will be made for a period of a year following the development of the guidelines.*

Four panel members were determined to have important but acceptable COI's. Three members (JDA, GG, JC) had an intellectual COI with the recommendations for antiviral and corticosteroid therapy as they had previously published on this topic. One member (SL) had a financial COI on the topic of exercise physiotherapy as she is currently involved in the delivery of this service in private practice. These individuals were permitted to participate in collecting and interpreting evidence, but were not involved in the deliberation for recommendations for which they were conflicted.[35]

Implementation

Implementation of these guidelines will begin with publication of the completed manuscript, which will facilitate wide distribution. Endorsing organizations will be asked to make a copy of the guidelines available to physician and other health-care members. To aid in utilization of these guidelines, all pertinent health-care stakeholders have been involved in the guideline making process and relevant professional organizations have been invited to review and/or endorse the guideline.

Funding

All organizational and publication costs were internally funded by the panel members from their respective institutions.

Recommendations and Rationale

1) Corticosteroid treatment for acute Bell's palsy of any severity.

In patients with acute Bell's palsy of any severity, we recommend the use of corticosteroids. ***(Strong Recommendation: Moderate Confidence in Effect Estimate)***

Systematic review of the literature yielded ten studies comparing corticosteroids to placebo including 1285 patients (**Table 4, 5, 6**).[36-46] We had moderate confidence in

effect estimate because of imprecision in effect estimates. Meta-analysis of these studies demonstrated a relative risk (RR) of 0.69 (95% CI, 0.55-0.87) of unsatisfactory facial recovery.[32] In the previously published meta-analysis,[32] a subgroup analysis was presented for mild to moderate paresis (3 studies)[36,41,42] versus severe to complete (4 studies)[36,41,42,43] paralysis. Statistical analysis did not show a statistically significant steroid subgroup effect (i.e. no difference in relative risk in the two groups), either due to a subgroup effect not being present, or due to insufficient power to show an effect.[32] We therefore applied the same RR (0.69; 95% CI, 0.55 – 0.87) for both mild to moderate paresis and severe to complete paralysis at presentation.

The estimated risk of incomplete recovery without treatment for patients with mild to moderate paresis was 6 per 100[19] and a corresponding risk with treatment of 4 per 100 (95% CI, 3-5) resulting in an absolute risk reduction of 2% (95% CI, 1- 3%) and a number needed to treat (NNT) of 50 (95% CI, 33-100). In patients with severe to complete paresis, the baseline risk for incomplete recovery is 39 per 100.[19] the risk of unsatisfactory recovery after treatment with corticosteroids is 27 out of 100 (95% CI, 21 –34) resulting in an absolute risk reduction of 12% in this group (95% CI, 5–18%) corresponding to an NNT of 8 (95% CI, 6 – 20)(**Table 6**). Three studies with 671 patients showed a significant reduction in synkinesis and autonomic dysfunction (RR = 0.56; 95% CI, 0.41-0.76), [36,40,45] with a similar number needed to treat of 8 (95% CI, (6 – 17).

Although corticosteroids have potential complications, seven studies involving 1155 patients showed no increase risk of major or minor side effects in those receiving

short term treatment versus control subjects.[36,37,39,40,41,44,45] There were no reported cases of avascular necrosis of the hip in patients treated with corticosteroids. One study reported 4 episodes of gastric ulceration in patients treated with corticosteroids given in combination with antivirals and no episodes in control patients.[47]

The dosing regimens for corticosteroids were highly variable in the systematic review. All studies used either prednisone or prednisolone derivatives. All but one study[36] used a tapered regimen. The duration of treatment ranged from 6 to 17 days,[40,45] with 6 studies using a ten day course.[36,37,39,41,43,44] Subgroup analysis in the previously published meta-analysis identified a significantly better effect with a total dose of 450 mg or higher compared to less than 450 mg.[32] A reasonable regimen suggested by four of the included studies involves a five day course of 60 mg per day followed by a five day taper, reducing the previous day's dose by 10 mg per day.[37,39,41,44]

The window of opportunity for treatment with corticosteroids is unclear. Some recent trials only included patients treated within 72 hours of symptom onset.[18, 37] We performed subgroup analyses to see if patients treated outside of this window had poorer recovery and were unable to show a difference.[32] However, a recent subgroup analysis of the large Swedish randomized controlled trial, suggested that steroids are only beneficial if started within the first 48 hours ($p=0.5$).[48]

The treating clinician should explore for relative contraindications to corticosteroid therapy such as diabetes, peptic ulcer disease and a remote history of

tuberculosis. If present, a discussion with the patient, explaining the potential benefit of corticosteroids in treating Bell's palsy and the potential risks should ensue.

Using corticosteroids for the treatment of Bell's palsy is also cost-effective. Based on resource use data from a recent UK cost-effectiveness analysis, corticosteroids treatment dominated all other treatment options including placebo, combined corticosteroids and antiviral therapy, and antiviral alone treatment.[49] Patients on steroids required fewer outpatient specialist visits and fewer visits to the primary care physician than all other treatment arms.

2) Antivirals without corticosteroids for acute Bell's palsy of any severity.

In patients presenting with acute Bell's Palsy of any severity, we recommend <u>against</u> antiviral treatment alone. (*Strong Recommendation: Moderate Confidence in Effect Estimate)*

Systematic review of the literature yielded two studies comparing antivirals plus placebo to double placebo including 658 patients (**Table 4, 5, 7**).[36,37,46] There were no significant study limitations or publication bias, the results were consistent and direct. However, there was some imprecision in the effect estimates. Meta-analysis of these studies demonstrated no benefit in the relative risk of unsatisfactory recovery (RR = 1.14; 95% CI 0.8-1.62) for patients treated with antivirals compared to placebo. Subgroup

analysis failed to detect a difference in effect between patients with mild to moderate paresis and those with severe to complete paralysis.

Major and minor side effects associated with antiviral therapy were infrequent. One episode of recurrent atrial fibrillation was described in a patient who received antiviral agents. Meta-analysis of two studies (N=653) comparing antiviral therapy to placebo did not show an increase risk of major or minor side effects, although wide confidence intervals due to small numbers of trials and outcome events resulted in considerable imprecision.[36,37]

There was no significant benefit of antiviral therapy for synkinesis and autonomic dysfunction (RR = 1.04; 95% CI, 0.75 – 1.43, one trial, N=373); pain at 9 months (Adjusted Odds Ratio (OR) = 0.05, 95% CI -0.91 – 1.01, one trial, N=496);[36] or health-related quality of life at 9 months (Adjusted OR) = -0.02, 95% CI -0.05-0.01, one trial, N=496).[36]

In a study of cost-utility, administration of antivirals was shown to be more costly and less effective than no therapy suggesting that antiviral treatment was dominated by no treatment, corticosteroid alone, and combined treatment.[49] Patients treated with antivirals alone required more visits to their general practitioners and more specialist outpatient appointments than all other treatment arms and than the placebo group.

3) Combined Corticosteroid/Antiviral therapy for Acute Bell's Palsy with mild to moderate paresis

For patients presenting with acute Bell's palsy with mild to moderate paresis, we suggest <u>against</u> the addition of antivirals to corticosteroids for patients (*Weak recommendation: Moderate Confidence in Effect Estimate)*.

Systematic review of the literature yielded 8 studies comparing combined corticosteroids and antiviral therapy to corticosteroids alone that included 1298 patients (**Table 8**).[36,37,38,46,50-54] The confidence in effect estimate was rated as moderate due to some imprecision in effect estimates. Meta-analysis of these studies demonstrated a relative risk of unsatisfactory recovery of 0.75 (95% CI, 0.56-1.00) for patients treated with combined therapy compared to corticosteroids alone. Assuming a risk of unsatisfactory facial recovery in patients with mild or moderate paresis with corticosteroid only treatment of 4 per 100, the corresponding risk with combined treatment is 3 per 100 (95% CI, 2-4) resulting in an absolute risk reduction of 1% (95% CI, 0 – 2%).

Major and minor side effects associated with adding an antiviral to corticosteroid therapy were infrequent. Meta-analysis of four studies (N=941) comparing antiviral therapy to placebo did not show an increase risk of major (RR 1.33, 95% CI 0.26 – 6.82) or minor side effects (RR 1.16 0.81 - 1.62), although wide confidence intervals due to small numbers of trials and outcome events resulted in considerable imprecision.[36,37,47,50]

In three studies with 511 patients, there was a reduction in the risk of synkinesis and autonomic dysfunction when an antiviral was added to corticosteroid (RR 0.59; 95% CI 0.39-0.89).[36,40,45] Assuming a risk of synkinesis of 15 per 100 in patients treated only with corticosteroids,[19] the corresponding risk with the addition of an antiviral is 9 per 100, with a NNT of 17 (95% CI 20-50).

The cost-effectiveness analysis cited above also addressed the addition of antiviral therapy and found that corticosteroid monotherapy was less costly and more effective than combined therapy.[49] In our meta-analysis of several studies including the trial on which this cost-effectiveness study was based, we found the contrary; that the addition of antivirals to corticosteroids is more effective than corticosteroid monotherapy. This difference is likely due to increased statistical power as a result of pooling studies.

The weak recommendation against the use of antivirals for patients with mild to moderate paresis places relatively high value on the low absolute reduction (and high number needed to treat) in the risk of unsatisfactory recovery and relatively low value on the benefit of reduced synkinesis. Patients with mild to moderate paresis who place higher value on avoiding synkinesis might consider antivirals in addition to corticosteroids.

4) Combined Corticosteroid/antiviral for acute Bell's Palsy with severe paresis to complete paralysis

For patients with acute Bell's palsy with severe to complete paresis, we suggest the combined use of antivirals and corticosteroids. (*Weak Recommendation: Moderate Confidence in Effect Estimate)*

As in the previous recommendation, meta-analysis demonstrated a relative risk of unsatisfactory recovery of 0.75 (95% CI 0.56-1.00) for patients treated with combined therapy compared to corticosteroids alone.[32] However, in the severe paresis to complete paralysis group, the risk of unsatisfactory recovery with corticosteroid alone is 27 per 100 and the corresponding risk with antivirals and corticosteroids is 20 per 100 (95% CI; 15 – 27) resulting in an absolute risk reduction of 7% (95% CI, 0 – 12 %).

The dosing regimen for antivirals was heterogenous across the included studies. Five studies used valacyclovir,[37,38,50,51,52] while three used acyclovir.[36,53,54] No studies used a tapered regimen. Treatment duration varied between 5 to 10 days—three studies used a 5 day regimen;[50,51,53] 3 studies used a 7 day regimen;[37,38,52] and 2 studies used a 10 day regimen.[36,54] All studies prescribing acyclovir used a minimum dose of 400 mg given five times daily. Two studies prescribing valacyclovir gave three times daily dosing,[37,38] while three studies gave twice daily dosing.[50,51,52] All except one study[50] recommended using at least 1 gram doses of valacyclovir.

Treating physicians must consider the additional cost of the medication, the side-effect profile of adding an antiviral medication and the values and preferences of the patient in making this decision. Our recommendation places a relatively high value on an

uncertain benefit with the addition of antiviral agents (borderline statistical significance). Patients who put a high value on avoiding the inconvenience, cost, and rare adverse effects associated with antiviral agents are likely to choose against their use.

5) Exercise physiotherapy for acute Bell's Palsy of any severity

For patients with acute Bell's palsy of any severity, we make no recommendation regarding the use of exercise physiotherapy. (*No recommendation: Very Low Confidence in Effect Estimate)*

During the course of reinnervation, spontaneous facial movement will occur naturally when eating, drinking, speaking, and with nonverbal facial communication. Exercise is unlikely to expedite or improve the reinnervation process that occurs in the acute setting. However, it may assist in the initial strengthening process after reinnervation. In the early stages of Bell's palsy, patients are likely to value education regarding the process and timeframe for nerve recovery.[55] As many patients have a strong desire to exercise affected facial musculature, they should be cautioned against maximal effort, nonspecific exercises in the presence of complete paralysis, as only the muscles on the unaffected side of the face are responding. Maximal effort exercises are ineffective and potentially lead to hyperactivity of the uninvolved musculature. Patients are likely to

value knowing that movements will naturally occur with reinnervation and that exercises are unnecessary at this stage.[56]

Three studies were included in a systematic review evaluating the use of exercise physiotherapy in the management of Bell's palsy **(Tables 9, 10, 11)**. Confidence in effect estimates was rated as very low due to serious risk of bias, indirectness, and imprecision of results. Meta-analysis of these studies was not carried out due to heterogeneity of interventions, outcomes, and follow-up. The first of these studies, which compared Kabat Physiotherapy (n=9), a proprioceptive neuromuscular rehabilitation technique, to a non-physiotherapy group (n=11) treated with corticosteroids and antivirals, showed[57] no difference in facial recovery at 15 days after symptom onset using the House-Brackmann scale.

A second study compared facial exercises in combination with a control treatment (medical therapy, acupuncture) (n=43) in comparison to control treatment alone (n=31).[58] This group was able to demonstrate an improvement in facial recovery at one month but no longer term follow-up were demonstrated.

A third study compared facial exercises and "conventional therapy" (n=85) to "conventional therapy" alone (n=60); "conventional therapy" was not described in any detail.[59] The study evaluated two outcomes including unsatisfactory recovery as well as synkinesis. The study showed no difference in unsatisfactory recovery but did demonstrate an improvement in synkinesis.

The group was unable to achieve consensus regarding exercise physiotherapy for acute Bell's Palsy of any severity and therefore no recommendation was made. In a blind vote, 4 panel members voted in for a recommendation in favor, 4 voted for a recommendation against, and 3 were neither in favor nor against.

6***) Exercise Physiotherapy for chronic Bell's palsy of any severity not demonstrating recovery***

For patients with persistent facial weakness of any severity after acute symptoms, we suggest exercise physiotherapy. ***(Weak Recommendation: Very Low Confidence in Effect Estimate)***

We identified only one study evaluating the use of exercise physiotherapy for patients not having a complete recovery of facial function (**Table 11**).[60] Confidence in effect estimate which began as low (observational study) was rated as very low due to risk of bias as well as imprecise results. This non-randomized study evaluated facial recovery outcomes using the House-Brackmann score of patients who underwent exercise therapy after at least 9 months of facial weakness from the development of symptoms. One group received exercise, stretch, and massage physiotherapy (n=24); another group (n=24) did not. The physiotherapy group demonstrated a significant improvement in

House-Brackmann scores (Mean Difference = 0.6, 95% CI,0.1-1.1) compared to the control group.

This recommendation implies a high value on an uncertain benefit, a high value on patients' experience of active participation in the recovery process, and a low value on the inconvenience and cost associated with physiotherapy. Patients who do not share these values are unlikely to choose physiotherapy.

7) Electrical stimulation (ES) in the management of acute Bell's Palsy of any severity

For patients with acute Bell's palsy of any severity, we suggest <u>against</u> the use of electrostimulation. (*Weak Recommendation: Very Low Confidence in Effect Estimate)*

Electrostimulation therapy evokes facial muscular responses by delivering an external electrical stimulus. The premise of this therapy is that induction of reinnervation by electrostimulation will minimize muscular atrophy for those patients who are destined to have incomplete or delayed recovery. Evoked responses require the use of relatively long pulse durations that will satisfy the prolonged chronaxies of denervated muscle fibers. Pulse durations of 1 to 40 milliseconds are commonly used, and duration of up to 200 milliseconds have been reported.[61-63]

A systematic review of the literature identified 4 studies (**Table 9, 10, 12**).[64-67] Included studies were heterogeneous in terms of outcomes measures, follow-up and interventions spanning 5 decades; therefore, no meta-analysis was performed. Mosforth et al randomly allocated patients with acute Bell's palsy into a group treated with electrostimulation and massage (n = 43) vs. massage alone (n = 40)[64] and found no significant differences in unsatisfactory recovery or synkinesis. Flores et al randomized patients into those treated with infrared and electrostimulation (n=77) and those treated with corticosteroid treatment (n=72)[65] and found no significant differences.

Manikandan et al. randomly assigned patients to a group (n=28) consisting of treatment with facial exercises, massage, and three times daily electrical stimulation and a second group (n=28) which consisted of facial neuromuscular re-education with patient-specific excercises.[66] The authors demonstrated significantly greater improvement in the re-education group compared to the electrostimulation, massage, and exercise group in facial recovery using the Sunnybrook grading system, but found no significant differences in synkinesis outcomes.

Alakram et al. compared heat, massage, exercises, and electrostimulation (n=8) to a group which received those same treatments without electrostimulation (n=8) in a non-randomized study.[67] The electrostimulation group showed a higher, although not statistically significant, recovery rate.

The available very-low quality evidence provides little support for electrostimulation in increasing the likelihood of satisfactory recovery. The safety profile

for ES at various levels of intensity and duration has not been established; further, the added costs and the repeated nature of the intervention are disincentives against its use.

8) ***Surgical Decompression for acute Bell's palsy with severe to complete paralysis.***

For patients with severe to complete paresis, we suggest <u>against</u> the routine use of surgical decompression. (*Weak Recommendation: Very Low Confidence in Effect Estimate)*

A systematic review of the literature identified six studies including 388 patients with complete or near complete facial paralysis (**Table 13, 14, 15**).[68-73] Confidence in effect estimate was rated as very low due to serious methodologic limitations, and imprecision and indirectness of results. In two studies, the labyrinthine segment (meatal foramen), the presumed site of lesion for Bell's palsy, was decompressed routinely through a middle cranial fossa surgical approach.[69,72] In another study, a middle cranial fossa approach was performed in 6 (7%) patients only.[68] The remaining studies evaluated a transmastoid approach, in which the labyrinthine segment would not have been decompressed.[68,70,71,73] No studies randomized patients. Treatment allocation was governed either by patient wishes[69,73] or was not reported.

One study available for middle fossa decompression of the meatal foramen prospectively evaluated 70 patients with total facial paralysis of two weeks duration or less, and greater than 90% degeneration on electroneuronography. [69] The patients were then offered surgery. Patients who chose surgery (n=34) had a 91% chance of a fair recovery (House-Brackmann score 1 or 2) compared to 42% in those treated medically (n=36). Of note, this was a multi-center study involving surgeons with a significant degree of experience performing middle cranial fossa approaches.

Surgical decompression of the facial nerve has potentially severe risks including: hearing loss (3-10% of patients),[68,69,72] either sensorineural (SNHL) or conductive (CHL) or mixed; a risk of further damaging the facial nerve (unknown prevalence); and CSF leaks (associated with a middle fossa craniotomy approach) [4%].[69] Interestingly, a recent survey of Otologists who perform this type of surgery showed that 35% of respondents feel that surgery does not improve outcomes, 34% have never performed this decompression surgery for Bell's palsy and only 5% have averaged more than one surgery per year indicating a relative lack of experience with this particular operation.[74]

Surgery may be an option in those patients with severe facial nerve degeneration on ENoG who are not improving with conservative management and who are willing to accept the surgical risks. However microsurgical decompression of the meatal/labyrinthine segment of the facial nerve is a highly technical procedure, performed in advanced treatment facilities only and may need to be performed within 14 days to be effective.[69]

9) Eye protective measures for patients with incomplete eye closure following Bell's palsy

For patients with Bell's palsy and incomplete eye closure, we recommend the routine use of eye protective measures. (*Strong Recommendation: very low confidence in estimates).*

No randomized comparative studies or observational studies have compared visual outcomes with or without eye protective measures for the management of incomplete eye closure in Bell's palsy. We rated our confidence in the effect estimate as very low due to the absence of any randomized trials or observational studies to support the benefit of eye protective treatments. However, inadequate lubrication/hydration of the cornea can lead to exposure keratitis, corneal ulceration, and eventually visual loss.

Conservative treatment for eye protection includes the use of artificial tears and ointment to maintain hydration of the cornea. To prevent accidental corneal injury during sleep, taping the eye at night is also recommended as long as eye closure is incomplete. Protective eye glasses or sunglasses could be helpful to protect the eye from debris with outside exposure. Botox injections into the levator palpebrae superioris muscle can be used to produce temporary ptosis and provide corneal protection with the anticipation that initiation of restoration of eye closure movement will occur upon the termination of the botox effect.[75] If performed, this should be done by an experienced practitioner.

Surgical options to protect the cornea include temporary tarsorrhaphy which is achieved with a suture to create eye closure which can be easily reversed if the facial nerve recovers.[76,77] In chronic cases, where eye closure is incomplete, a permanent gold/platinum weight may be surgically inserted in the affected upper eyelid to aid eye closure movement.[76-78]

This recommendation places a high value on an uncertain benefit of protective measures and a low value on the associated inconvenience. The rationale for the recommendation is the potentially catastrophic consequences of visual loss following keratitis and the minimal undesirable consequences associated with the intervention.

10) Referral to a specialist in Bell's palsy showing progressive deterioration of facial nerve function or failing to show any signs of recovery.

For patients with Bell's palsy showing either progressive deterioration of facial function over time or failure to show any recovery of nerve function, we recommend the referral to a specialist. (*Strong Recommendation: Very low confidence in estimates*)

We identified no randomized or observational studies comparing outcomes with Bell's palsy patients who were referred versus those not referred. Therefore, our confidence in the effect estimates was very low. It is likely that frontline primary care

physicians manage most cases of adults with Bell's palsy. This may be less true for children with Bell's palsy: one study found general practitioners referred 78% of patients in the acute setting either to Pediatricians (54%), Otolaryngology-Head and Neck Surgeons (22%), and other (General Medicine, Physiotherapist) (2%).[79] The majority of patients with Bell's palsy show signs of recovery within 3 weeks to 3 months and in those patients who are improving, referral to a specialist is unlikely to be helpful.

For patients who do not show improvement in facial nerve function or have a progression of their facial nerve paralysis, referral to an Otolaryngologist may be reasonable to confirm the diagnosis and exclude other conditions. Over thirteen years, a tertiary care center found eleven cases of malignant skull base neoplasms from 320 cases that were initially diagnosed as Bell's palsy.[80] The authors suggest that a delay in diagnosis and treatment may result in increased rates of morbidity and mortality although there was no comparative group in this study.

Boahene and colleagues reported on 11 patients diagnosed with Bell's palsy who proved to have occult malignancies.[81] It is unclear how many patients were seen during this study that were diagnosed with Bell's palsy that did not have a malignancy. All of these patients had normal imaging studies, and malignancies were diagnosed on surgical exploration. In this study the time to progression to total paralysis was 9 months. All of these malignancies were eventually treated with a combination of surgery, chemotherapy, and radiotherapy.

Other non-malignant causes may also masquerade as Bell's palsy. In a study of facial nerve schwannomas, 6 of 28 cases were originally diagnosed as Bell's palsy.[82] These tumors commonly present with either recurrent episodes of facial paralysis or delayed progression of facial paresis and can be managed either expectantly or surgically.[82,83]

The possibly catastrophic consequences of having a serious condition that mimics Bell's palsy, albeit rare, and the unlikely serious harm consequent on referral motivate this strong recommendation despite very low confidence in estimates of effect.

11) Investigation for neoplasms with imaging in Bell's palsy showing progressive deterioration of facial nerve function or failing to show any signs of recovery

For patients with Bell's palsy showing either progressive deterioration of facial function over time or failure to show any recovery of nerve function, we recommend investigation for neoplasms with imaging. (*Strong Recommendation: Very Low Confidence in Effect Estimate*)

We identified no randomized trials or observational studies investigating the role of imaging for neoplasms in patients with Bell's palsy, nor were there any randomized controlled trials investigating choice of imaging technique. As such our confidence in the

effect estimate was rated as very low. In a large series following the natural history of over one thousand patients with Bell's palsy, 85% of patients began to show some signs of recovery within three weeks of symptom onset.[15] In another study, 28% of patients in a trial of over 800 total patients experienced progression of symptoms with worsening of facial paralysis up to 17 days after symptom onset.[84] As such, it is unlikely that imaging prior to three weeks after symptom onset in patients with progressive facial paresis is of any benefit.

In patients who have not responded to initial treatment and show progressive facial paralysis, investigation for neoplasms along the course of the facial nerve should include imaging of the course of the facial nerve (brainstem/temporal bone/parotid gland) with either MRI or high resolution CT scan. Although there are no trials comparing various imaging techniques for progressive facial nerve dysfunction, each technique has its merit.[85] MRI may be better suited to evaluate the brainstem, cerebellopontine angle, and bone/soft tissue interfaces as well as parotid gland while high resolution CT scan may be more widely available and better suited for studying the intra-temporal segment of the nerve.[86]

Contrast enhanced MRI scans have been shown to be capable of detecting even small lesions within the temporal bone or parotid gland.[87] Treating clinicians should be mindful, however, that even if the initial diagnosis of Bell's palsy is correct, subsequent MRI imaging maybe abnormal. On post-contrast T1 weighted MRI, the facial nerve after Bell's palsy may show enhancement from the internal auditory canal out to the peripheral

nerve in the parotid gland.[88] In many cases, reversal of contrast enhancement of the nerve is noted after the acute inflammatory period is over.[87] However, even in chronic stages, MRI may show changes in the intensity of the facial nerve on the affected side on T2 weighted imaging,[89] although most resolve by two months after symptoms onset.[90]

The possibly catastrophic consequences of neglect in the rare instances of underlying malignancy, and the unlikely serious harm consequent on referral motivate this strong recommendation despite very low confidence in estimates of effect.

Discussion

The guidelines presented herein present the best available evidence to date for the management of patients afflicted with Bell's palsy or idiopathic facial paralysis. These guidelines include a treatment algorithm based on systematic and comprehensive review of the literature with incorporation of the best available evidence (**Figure 1**). The members of the guideline working group were selected to represent a variety of stakeholders involved in the management of Bell's palsy including Otolaryngology-Head and Neck Surgeons, Neurologists, Family Physicians, Facial Therapists, guideline methodologists, and patients. The use of the GRADE system was adopted to promote transparency to users in the group's decision making.

A recent concurrent guideline was published through the American Academy of Otolaryngology-Head and Neck Surgery.[91] This recent guideline had a similar scope,

and some similarities in their recommendations, however, there were some notable differences. Their target audience included all treating providers, however, our guideline is aimed primarily at primary care physicians. Their guideline also focused on diagnostic work-up of Bell's palsy, whereas, ours was mainly focused on treatment issues. We suggested combined corticosteroid and antiviral therapy only in patients with severe to complete paralysis (NNT = 14) and suggested against its use patients with mild-moderate paresis because we felt that the benefit was not as clear in this subgroup (NNT = 100), whereas they suggested combined treatment as an option in all cases and specific subgroups were not considered independently for these recommendations. We also suggested that facial physiotherapy may be indicated in patients who with long-standing paresis and whom experience no improvement, whereas their guideline did not consider this specific subpopulation but did not make a recommendation for physiotherapy in acute cases. This difference may be attributable also to differences in the panel composition with our panel consisting of facial physiotherapists and a patient and their panel consisting mainly of medical personnel. Lastly, our group suggested against the use of routine surgical decompression in patients with severe paresis to complete paralysis whereas their group did not make a recommendation. We felt that given the balance of beneficial and harmful outcomes and the available evidence that this should not be routinely done.

Our recommendations have limitations that should be considered in their interpretation and application. First of all, the natural rates of recovery without treatment were based on a single-author observational study of over 2500 patients.[15] We considered

another alternative for estimating the natural recovery rate using the placebo group recovery rates from all randomized trials. However, this does not provide an accurate estimate of the natural recovery rate, as trials tend to be a homogenous population with exclusion of many co-morbid or elderly individuals. The importance of this estimate is that the absolute benefit of any treatment depends on the estimate of the natural recovery rate.

The estimates of the effect of many of these treatments in particular subgroups of patients is also unclear. There is some evidence to suggest that patients with diabetes, hypertension or pregnant patients are predisposed to Bell's palsy.[1] However, the effect of medical and surgical treatment has not been studied in these subgroups as they are often excluded from trials. Therefore, the group decided not to make specific subgroup recommendations for these populations. Furthermore, subgroup analysis of patients with mild to moderate paresis versus severe paresis to complete paralysis in a previous meta-analysis suggested that there is a non-significant trend to increased benefits of corticosteroids in patients with mild-moderate paresis than in those with severe paresis or complete paralysis.[32] However, due to the absence of statistical significance and inconsistency across studies we decided that it was unlikely a subgroup effect exists. Further study confirming or refuting a differential effect of steroids in mild-moderate versus severe paresis to complete paralysis may require an update to the present guideline.

Lastly, gaps in the available data have implications for the current recommendations. While moderate quality evidence exists for medical treatment with corticosteroids and antiviral agents, confidence in estimates for all relevant patient-important outcomes for surgery and physiotherapy are very low. Using the GRADE approach, recommendations are also based on evidence of values and preferences of patients with the disease condition as well as cost and resource allocation information. We identified no studies for Bell's palsy investigating the values and preferences of patients and only one study that examined costs of corticosteroids and antivirals for Bell's palsy.[49] Further research in these areas are needed.

References

1. Holland NJ, Weiner GM. Recent developments in Bell's palsy. BMJ. 2004;329(7465):553-557.
2. Hauser WA, Karnes WE, Annis J, Kurland LT. Incidence and prognosis of Bell's palsy in the population of Rochester, Minnesota. Mayo Clin Proc. 1971;46(4):258-264.
3. Gilden DH. Bell's Palsy. N Engl J Med 2004;351:1323-31
4. Bosco D, Plastino M, Bosco F, Consoli A, Labate A, Pirritano D, Consoli D, Fava A. Bell's palsy: a manifestation of prediabetes. Acta Neurol Scand 2011: 123: 68–72

5. Mutsch M, Zhou W, Rhodes P, Bopp M, Chen RT, Linder T, Spyr C, Steffen R. Use of the Inactivated Intranasal Influenza Vaccine and the Risk of Bell's Palsy in Switzerland. N Engl J Med 2004;350:896-903.

6. Bardage C, Persson I, O□rtqvist A, Bergman U, Ludvigsson JF, Granath F. Neurological and autoimmune disorders after vaccination against pandemic influenza A (H1N1) with a monovalent adjuvanted vaccine: population based cohort study in Stockholm, Sweden. BMJ 2011;343:d5956 doi: 10.1136

7. Lee GM, Greene SK, Weintraub ES, Baggs J, Kulldorff M, Fireman BH, Baxter R, Jacobsen SJ, Irving S, Daley MF, Yin R, Naleway A, Nordin JD, Li L, McCarthy N, Vellozzi C, DeStefano F, Lieu TA, on behalf of the Vaccine Safety Datalink Project. H1N1 and Seasonal Influenza Vaccine Safety in the Vaccine Safety Datalink Project Am J Prev Med 2011;41(2):121–128

8. Izurieta HS, Haber P, Wise RP, Iskander J, Pratt D, Mink C, Chang S, Braun MM, Ball R.Adverse Events Reported Following Live, Cold-Adapted, Intranasal Influenza Vaccine. JAMA. 2005;294:2720-2725

9. Holland J, Bernstein J. Bell's palsy. Clinical Evidence 2011;03:1204
10. Tiemstra JD, Khatkhate N. Bell's Palsy: Diagnosis and Management. Am Fam Physician 2007;76:997-1002

11. Adour KK, Bell DN, Hilsinger RL Jr. Herpes simplex virus in idiopathic facial paralysis (Bell palsy). JAMA. 1975;233(6):527-530.
12. Theil D, Arbusow V, Derfuss T, et al. Prevalence of HSV-1 LAT in human trigeminal, geniculate, and vestibular ganglia and its implication for cranial nerve syndromes. Brain Pathol. 2001;11(4):408-413.
13. Furuta Y, Fukuda S, Suzuki S, et al. Detection of varicella-zoster virus DNA in patients with acute peripheral facial palsy by the polymerase chain reaction, and its use for early diagnosis of zoster sine herpete. J Med Virol. 1997;52(3):316-319.

14. Fisch U, Esslen E. Total intratemporal exposure of the facial nerve. Arch Otolaryngol. 1972;95(4):335-341.
15. Peitersen E. Bell's Palsy: The Spontaneous Course of 2,500 Peripheral Facial Nerve Palsies of Different Etiologies. Acta Otolaryngol 2002; Suppl 549: 4–30
16. House JW, Brackmann DE. Facial nerve grading system. Otolaryngology Head Neck Surg. 1985;93(2):146-7.
17. Ross BG, Fradet G, Nedzelski JM. Development of a sensitive clinical facial grading system. Otolaryngol Head Neck Surg. 1996;114(3):380-6.
18. Sullivan F, Swan I, Daly F. Prenisolone or Acyclovir in Bell's palsy. N Engl J Med. 2008;358(3):306-7.
19. Peiterson E. The natural history of Bell's palsy. Am J Otol. 1982;4(2):107-111.
20. Gantz BJ, Rubinstein JT, Gidley P, Woodworth GG. Surgicalmanagement of Bell's palsy. Laryngoscope 1999;109:1177–1188.
21. Weir AM, Pentland B, Murray J, Crosswaite A, Mountain R. Bell's Palsy: the effect on self-image, mood state and social activity. Clinical Rehab 1993; 7: 88
22. Validation of the Synkinesis Assessment Questionnaire. Ritvik P. Mehta, MD; Mara WernickRobinson, PT, MS, NCS; Tessa A. Hadlock, MD. Laryngoscope, 117:923–926, 2007.
23. 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(7):830-836.
24. Gronseth GS, Paduga R. Evidence-based guideline update: steroids and antivirals for Bell palsy. Neurology. 2012;79:1-5.
25. Lunan R, Nagarajan L. Bell's palsy: A guideline proposal following a review of practice. J Ped Child Health. 2008;44:219-220.
26. Murthy JM, Saxena AB. Bell's palsy: Treatment guidelines. Ann Indian Acad Neurol. 2011. 14(Suppl 1):S70-2.
27. Teixeira LJ, Valbuzo JS, Prado GF. Physical therapy for Bell's palsy (idiopathic facial paralysis). Cochrane Database Syst Rev. 2011;Dec 7;(12):CD006283.
28. Guyatt GH, Oxman AD, Vist GE, Kunz R, Falck-Ytter Y, Alonso-Coello P, Schunemann HJ. GRADE: an emerging consensus on rating quality of evidence and strength of recommendations. BMJ. 2008;336:924-26.
29. Guyatt GH, Oxman AD, Vist GE, Kunz R, Falck-Ytter Y, Schunemann HJ. GRADE: what is "quality of evidence" and why is it important to clinicians? BMJ. 2008;336:995-998.
30. Guyatt GH, Oxman AD, Kunz R, Falck-Ytter Y, Vist GE, Liberati A, Schunemann HJ. GRADE: going from evidence to recommendations. BMJ. 2008;336:1049-1051.
31. Guyatt GH, Oxman AD, Kunz R, Jaeschke R, Helfand M, Liberati A, Vist GE, Schunemann HJ. GRADE: Incorporating considerations of resources use into grading recommendations. BMJ. 2008;336:1170-1173.

32. de Almeida JR, Al Khaboi M, Guyatt GH, et al. Combined corticosteroid and antiviral treatment for Bell Palsy: a systematic review and meta-analysis. JAMA. 2009;302(9):985-93.
33. Higgins J, Altman DG. Chapter 8: Assessing risk of bias in included studies. In: Higgins J, Green S, eds. *Cochrane Handbook for Systematic Reviews of Interventions 5.0.1.* Oxford, UK: The Cochrane Collaboration; September, 2008
34. Jaeschke R, Guyatt GH, Dellinger P, et al. Use of GRADE grid to reach decision on clinical practice guidelines when consensus is elusive. BMJ. 2008;337:a744.
35. Guyatt G, Akl EA, Hirsh J, et al. The vexing problem of guidelines and conflict of interest: a potential solution. Ann Intern Med. 2010;152(11):738-741.
36. Sullivan FM, Swan IR, Donnan PT, et al. Early treatment with prednisolone or acyclovir in Bell's palsy. N Engl J Med. 2007;357(16):1598-1607.
37. Engstrom M, Berg T, Stjernquist-Desatnik A, et al. Prednisolone and valaciclovir in Bell's palsy: a randomized, double blind, placebo-controlled, multicentre trial. Lancet Neurol. 2008;7(11):993-1000.
38. Antunes ML, Fukuda Y, Testa JRG. Clinical treatment of Bell's palsy: comparative study among valaciclovir plus deflazacort, deflazacort and placebo. *Acta AWHO*. 2000;19(2):68-75.
39. Austin JR, Peskind SP, Austin SG, et al. Idiopathic facial nerve paralysis: a randomized double blind controlled study of placebo versus prednisone. *Laryngoscope*. 1993;103:1326-33.
40. Lagalla G, Logullo F, Di Bella P, et al. Influence of early high-dose steroid treatment on Bell's palsy evolution. Neurol Sci. 2002;23:117-122.
41. Martinez GC, Abarca B, Alvarado CL, et al. Paralisis de Bell: evaluacion del tratamiento steroidal. Bol Hosp San Juan de Dios. 1990;37(1):13-17.
42. May M, Wette R, Hardin WB, et al. The use of steroids in Bell's palsy: a prospective controlled study. Laryngoscope. 1976;86(8):1111-1122.
43. Tekle-Haimanot R. Idiopathic facial paralysis (Bell's palsy) in 167 Ethiopians, with a controlled therapeutic trial in 59 patients. *Ethiop Med J*. 1987;25(1):23-7.
44. Unuvar E, Oguz F, Sidal M, et al. Corticosteroid treatment of childhood Bell's palsy. Ped Neurol. 1999;21(5):814-16.
45. Wolf SM, Wagner JH, Davidson S, et al. Treatment of Bell palsy with prednisone: a prospective randomized study. Neurol. 1978;28:158-61.
46. Madhok V, Falk G, Fahey T, Sullivan FM. Prescribe prednisolone alone for Bell's palsy within 72 hours of symptom inset. BMJ. 2009;338:b255.
47. Roy A, Jose J, Karnath V, et al. Efficacy of acyclovir and methylprednisolone versus methylprednisolone alone in treatment of Bell's palsy. J Neurol sci. 2005;238(suppl 1):S207.
48. Axelsson S, Berg T, Jonsson L, Engstrom M, Kanerva M, Pitkaranta A, Stjerquist-Desatnik A. Prednisolone in Bell's palsy related to treatment start and age. Otol Neurotol. 2011:32(1):141-6.
49. Hernandez RA, Sullivan F, Donnan P, Swan I, Vale L. Economic evaluation of early administration of prednisolone and/or acyclovir for the treatment of Bell's palsy.

50. Hato N, Yamada H, Kohno H, et al. Valacyclovir and prednisolone treatment for Bell's palsy: a multicenter, randomized, placebo-controlled study. Otology Neurotol. 2007;28:408-413.
51. 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.
52. Vazquez MC, Sanchez N, Calvo J. Eficacia de los antivirales en la paralysis de Bell. *Rev Med Urug*. 2008;24(3):1-8
53. Yeo SG, Lee YC, Park DC, et al. Acyclovir plus steroid vs steroid alone in the treatment of Bell's palsy. *Am J Otolaryngol*. 2008;29:163-6.
54. Adour KK, Ruboyianes JM, Von Doersten PG, et al. Bell's palsy treatment with acyclovir and prednisone compared with prednisone alone: a double-blind, randomized, controlled trial. *Ann Otol Rhinol Laryngol*. 1996;105:371-377.
55. Coulson SE, Croxon GR. Facial nerve rehabilitation – the role of physiotherapy. Aust J Otolaryng. 1994;1:418-421
56. Diels, H.J. (1995). New concepts in nonsurgical facial nerve rehabilitation. In E. Myers & C. Bluestone (Eds.), Advances in otolaryngology head and neck surgery, 9, (pp. 289-315). Chicago, Mosby-Year Book, Inc.
57. Barbara M, Antonini G, Vestri A, Volpini L, Monini S. Role of Kabat physical rehabilitation in Bell's palsy: a randomized trial. Acta Oto-Laryngologica.2010;130:167-172.
58. Wang XH, Zhang LM, Han M, Zhang KQ. Clinical application of functional exercise and staged therapy in treatment of facial nerve paralysis. Zhonghua Linchuang Kangfu Zazhi [Chinese Journal of Experimental and Clinical Virology]. 2004;8(4)616-617.
59. Wen CM, Zhang BC. Effect of rehabilitation training at different degree in the treatment of idiopathic facial palsy: a randomized controlled comparison. Zhongguo Linchuang Kangfu. 2004;8(13):2446-7.
60. Beurskens CHG, 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.
61. Cole J, Zimmerman S, Gerson S: Nonsurgical neuromuscular rehabilitation of facial muscle paresis, in Rubin LR (ed): The Paralyzed Face. St. Louis, Mosby-Year Book, Inc., 1991, pp 107-112.
62. Chusid JG: Correlative Neuroanatomy and Functional Neurology. Los Altos, Lange Medical, 1982, pp 99-101.
63. Farragher DJ: Electrical stimulation: A method of treatment for facial paralysis, in Rose FC, Jones R, Vrbova G (eds): Neuromuscular Stimulation: Basic Concepts and Clinical Implications. New York, Demos, 1989, vol 3, pp 303-306.
64. Mosforth J, Taverner D. Physiotherapy for Bell's palsy. British Medical Journal. 1958;2(5097):675-7.
65. Flores PF, Medina RZ, Haro LG. Idiopathic peripheral facial paralysis treatment physic therapy versus prednisone [tratamiento de la paralisis facial periferica

idiopatica: terapia fisica versus prednisone]. Revista medica del Instituto Mexicano del Seguro Social. 1998;36(3):217-221.
66. Manikandan N. Effet of facial neuromuscular re-education on facial symmetry in patients with Bells palsy: arandomized controlled trial. Clinical Rehabilitation. 2007;21:338-343.
67. Alakram P, Puckree T. Effects of electrical stimulation on house-brackmann scores in early bells palsy. Physiotherapy Theory and Practice. 2010;26(3):160-166.
68. Brown JS. Bell's palsy: a 5-year review of 174 consecutive cases: an attempted double blind study. Laryngoscope 1982;92:1369–1373.
69. Gantz BJ, Rubinstein JT, Gidley P, Woodworth GG. Surgicalmanagement of Bell's palsy. Laryngoscope 1999;109:1177–1188.
70. May MM, Taylor FH. Bell's palsy: surgery based upon prognosticindicators and results. Laryngoscope 1981;91:2092–2105.
71. May MM, Klein SR, Taylor FH. Idiopathic (Bell's) facial palsy:natural history defies steroid or surgical treatment. Laryngoscope 1985;95:406–409.
72. Fisch U. Surgery for Bell's palsy. Arch Otolaryngol 1981;107:1–11.
73. Yanagihara N, Hato N, Murakami S, Honda N. Transmastoid decompression as a treatment of Bell's palsy. Otolaryngol Head Neck Surg. 2001;124(3):282-6.
74. Smouha E, Toh E, Schaitkin BM. Surgical treatment of Bell's Palsy: Current Attitudes. Laryngoscope. 2011;121:1965-1970.
75. Prell J, Rampp S, Rachinger J, et al. Botulinum toxin for temporary corneal protection after surgery for vestibular schwannoma. J Neurosurg. 2011;114:426–431.
76. Lee V, Currie Z, Collin JRO. Ophthalmic management of facial nerve palsy. Eye. 2004;18:1225–1234.
77. Smith MFW, Goode RL. Eye Protection in the paralyzed face. Laryngoscope. 1979;89(3):435-442.
78. Henstrom DK, Lindsay RW, Cheney ML, Hadlock TA. Surgical Treatment of the Periocular Complex and Improvement of Quality of Life in Patients With Facial Paralysis. Arch Facial Plast Surg. 2011;13(2):125-8.
79. El-Hawrani AS, Eng CY, Ahmend SK, Clarke J, Dhiwakar M. General practitioners' referral pattern for children with acute facial paralysis. J Laryngol Otol. 2005;119:540-542.
80. Marzo SJ, Leonetti JP, Petruzzelli G. Facial paralysis caused by malignant skull base neoplasm's. Neurosurg Focus. 2002;12(5):e2.
81. Boahene DO, Olsen KD, Driscoll C, Lewis JE, McDonald TJ. Facial nerve paralysis secondary to occult malignant neoplasm's. Otolaryngol Head Neck Surg. 2004;130:459-465.
82. Zhagn R, Liu JP, Dai C. Misdiagnosis of facial nerve tumor. Zhonghua Er Bi Yan Hou Tou Jing Wai Ke Za Zhi. 2007;42(11):817-20.
83. Perez R, Chen JM, Nedzelski JM. Intratemporal facial nerve schwannoma: a management dilemma. Otol Neurotol. 2005;26(1):121-6.

84. Marsk E, Hammarstedt L, Berg T, Engstrom M, Jonsson L, Hultcrantz M. Early deterioration in Bell's palsy: prognosis and effect of prednisolone. Otol Neurotol. 2010;31(9):1503-7.
85. Raghavan P, Mukherjee S, Phillips CD. Imaging of the facial nerve. Neuroimaging Clin N Am. 2009;19(3):407-25.
86. Schwaber MK, Zealear D, Netterville JL, Seshul M, Ossoff RH. The use of magnetic resonance imaging with high-resolution CT in the evaluation of facial paralysis. Otolaryngol Head Neck Surg. 1989;101(4):449-58.
87. Girard N, Poncet M, Chays A, et al. MRI exploration of the intrapetrous facial nerve. J Neuroradiol. 1993;20(4):226-38.
88. Song MH, Kim J, Jeon JH, Cho CI, Yoo EH, Lee WS, Lee HK. Clinical significance of quantitative analysis of facial nerve enhancement on MRI in Bell's palsy. Acta Otolaryngol. 2008;128(11):1259-65.
89. Kinoshita T, Ishii K, Okitsu T, Ogawa T, Okudera T. High-intensity facial nerve lesions on T2-weighted images in chronic persistent facial nerve palsy. Neuroradiology. 2001;43(5):388-92.
90. Engstrom M, Thuomas KA, Naeser P, Stalberg E, Jonsson L. Facial nerve enhancement in Bell's palsy demonstrated by different gadolinium-enhanced magnetic resonance imaging techniques. Arch Otolaryngol Head Neck Surg. 1993;119(2):221-5.
91. Baugh RF, Basura GJ, Ishii LE, et al. Clinical practice guideline: Bell's palsy. Otolaryngol Head Neck Surg. 2013;149(3 Suppl):S1-27.

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

Figure 1 – Clinical Decision Making Algorithm for Acute Bell's Palsy

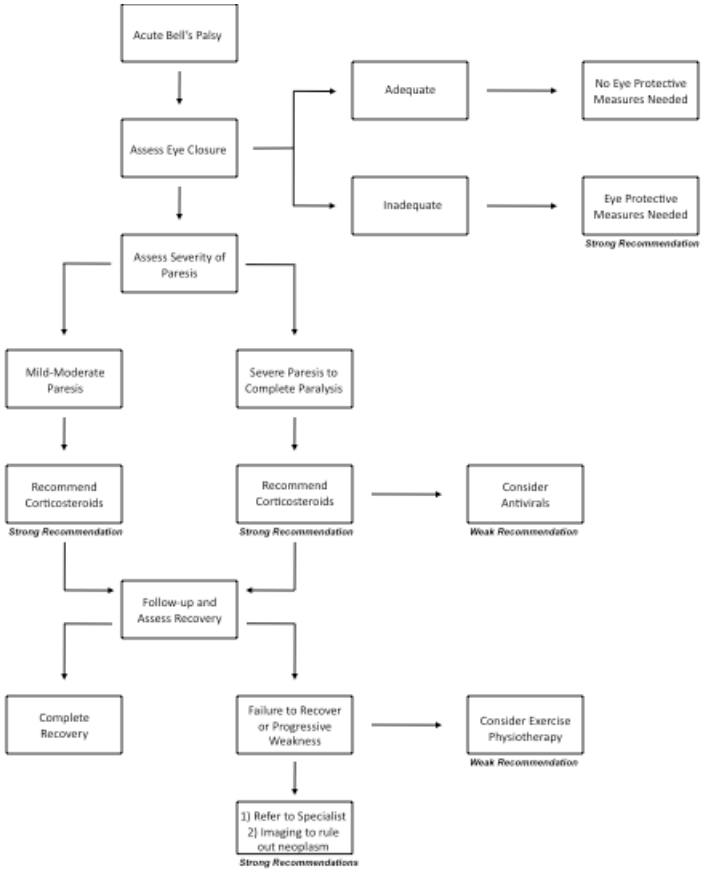

Table 1. House-Brackmann Facial Grading System (adapted from 16)

Grade	**Characteristics**
I - Normal	**Normal facial function in all areas**
II – Mild Dysfunction	**Gross** • **Slight weakness noticeable on close inspection** • **May have very slight synkinesis** • **At rest normal symmetry and tone** **Motor** • **Forehead: moderate to good function** • **Eye: complete closure with minimal effort** • **Mouth: slight asymmetry**
III – Moderate Dysfunction	**Gross** • **Obvious but not disfiguring difference between two sides** • **Noticeable but not severe synkinesis, contracture, or hemifacial spasm** • **At rest normal symmetry and tone** **Motor** • **Forehead: slight to moderate movement** • **Eye: complete closure with effort** • **Mouth: slightly weak with maximal effort**
IV – Moderately Severe Dysfunction	**Gross** • **Obvious weakness and/or disfiguring asymmetry** • **At rest normal symmetry and tone** **Motor** • **Forehead: none** • **Eye: incomplete closure** • **Mouth: Asymmetric with maximum effort**
V – Severe Dysfunction	**Gross** • **Only barely perceptible motion** • **At rest asymmetry** **Motor** • **Forehead: none** • **Eye: incomplete closure** **Mouth: movement**
VI – Total Paralysis	**No movement**

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

Table 2. Summary of Recommendations

Treatment	Recommendation	Strength	Confidence in Effect Estimate
Corticoseroids (any severity)	We recommend the use of corticosteroids for patients presenting with acute Bell's palsy of any severity	Strong	Moderate
Antivirals (any severity)	We recommend **against** the use of antivirals alone for patients presenting with acute Bell's Palsy of any severity	Strong	Moderate
Corticosteroids+Antivirals (mild-moderate paresis)	We suggest **against** the combined use of antivirals and corticosteroids for patients presenting with acute Bell's Palsy with mild to moderate paresis.	Weak	Moderate
Corticosteroids+Antivirals (severe-complete paresis)	We suggest the combined use of antivirals and corticosteroids for patients presenting with acute Bell's Palsy with severe paresis.	Weak	Moderate
Exercise physiotherapy (Acute; any severity)	We make **no recommendation** regarding the use of exercise physiotherapy for acute Bell's palsy of any severity given the relative lack of good quality data.	NA	Very Low
Exercise physiotherapy (Chronic)	We suggest exercise physiotherapy for patients with persistent facial weakness after acute onset symptoms.	Weak	Low
Electrostimulation (Acute; any severity)	We suggest **against** the use of electrostimulation in the acute management of Bell's palsy of any severity	Weak	Very Low
Surgical Decompression (severe-complete paresis)	We suggest **against** the routine use of surgical decompression for Bell's palsy with severe to complete paresis given the poor quality of evidence and the potential for harmful outcomes.	Weak	Very Low
Eye Protective Measures for Incomplete Eye Closure	We recommend the routine use of eye protective measures in patients with Bell's palsy and incomplete eye closure.	Strong	Very Low
Referral to Specialist for Progressive Cases	We recommend a referral to a specialist for Bell's palsy cases that fails to recover or shows signs of progression of facial paresis.	Strong	Very Low
Work-up for Neoplasm in Progressive Cases	We recommend a work-up for neoplasm with imaging for Bell's palsy cases that fails to recover or shows signs of progression of facial paresis.	Strong	Very Low

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

Table 3: Clinically Important Outcomes

Desirable Outcomes

Minimizing Unsatisfactory Facial Recovery (primary outcome)
Reduction in Synkinesis and Facial Spasm
Reduction in Autonomic Dysfunction (crocodile tears)
Reduction in Pain
Quality of Life
Visual loss

Undesirable Outcomes

Minor Adverse Effects
- Drug Therapy: GI upset, flushing, rash
- Physiotherapy**:** pain
- Surgical Therapy: wound infection, minor hemorrhage

Major Adverse Effects
- Drug Therapy: anaphylaxis, avascular necrosis of the hip (cortiocosteroids), peptic ulcer exacerbation or gastrointestinal bleed, exacerbation or development of a life-threatening infection (corticosteroids)
- Physiotherapy: worsening of facial nerve grade
- Surgical Therapy: Meningitis, cerebritis, intracranial abscess, seizure, death, hearing loss, worsening of facial function.

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

Table 4. Study Characteristics of Randomized Controlled Trials for Corticosteroids and Antivirals for Treatment of Bell's Palsy.

	Treatment Studied	Randomization Groups	Number of Patients Randomized	Time to Treatment	Duration of Follow Up	Facial Nerve Grading Scale (max score)	Definitions of Recovery and Initial Severity (based on facial nerve grading scale)	Outcome Results (treatment vs. control)
Adour et al,[54] 1996	Antiviral Agents	Placebo fives times daily x 10d Prednisone 30 mg twice daily x 5 d and taper (10 mg/d) x 5 d	46	Within 5 d	4 mo	FPRI (10)	UR: < 10 MP: n/a SP: 0	<u>Outcome:</u> Mean FPRI score <u>Results:</u> Mean difference in FRPI 9.57 ± 1.22 vs. 8.80 ± 2.19 (p =0.04)
		Acyclovir 400 mg fives times daily x 10 d Prednisone (as above)	53					

Antunes et al,[38] 2000	Antiviral Agents	Placebo three times daily x 7 d Placebo daily x 7 d	17	Within 5 d	Minimum 4 mo	HB (6)	UR: > 1 MP: n/a SP: n/a	<u>Outcome:</u> Number of incomplete recoveries <u>Results:</u> 2 incomplete recoveries in the first two arms (no statistics used)
		Placebo three times daily x 7d Deflazacort 60 mg daily x 2d and taper (15 mg/2d) x 6 d	14					
		Valacyclovir 500 mg three times daily x 7 d Deflazacort (as above)	15					
Austin et al,[39] 1993	Cortico-steroids	Placebo twice daily x 10 d	41	Within 5 d	Minimum 6 mo	HB (6)	UR: > 1 MP: 2-4 SP: 5-6	<u>Outcome:</u> Time to recovery <u>Results:</u> Mean time to recovery 51.4 d vs 69.3 (NS)
		Prednisone 30 mg twice daily x 5 d and taper (10 mg/d) x 5 d	35					

Engstrom et al,[37] 2008	Antiviral Agents and Cortico-steroids	Placebo three times daily x 7 d Placebo daily x 10 d	209	Within 3 d	1 year	Sunnybrook [a] (100), HB (6)	UR: > 1[a] MP: n/a SP: n/a	Outcome: Time to recovery Results: Prednisolone vs no prednisolone (HR 1.4; 95% CI, 1.18-1.64; p < 0.0001) Valacyclovir vs. no Valacyclovir (HR 1.01; 95% CI, 0.85-1.19; p = 0.9)
		Placebo three times daily x 7 d Prednisolone 60 mg daily x 5 d and taper (10 mg/d) x 5 d	213					
		Valacyclovir 1000 mg three times daily x 7 d Placebo daily x 10 d	207					
		Valacyclovir (as above) Prednisolone (as above)	210					
Hato et al,[50] 2007	Antiviral Agents	Placebo twice daily x 5 d Prednisone 60 mg daily x 5 d, 30 mg daily for 3 d and 10 mg daily for 2 d	107	Within 7 d	6 mo	Yanagihara scoring system (40)	UR: < 36 MP: > 20 SP: ≤ 20	Outcome: Number of satisfactory recoveries Results:

		Valacyclovir 500 mg twice daily x 5 d Prednisone (as above)	114					110 of 114 (96.5%) vs. 96 of 107 (89.7%) ($p < 0.05$)
Kawa-guchi et al,[51] 2007	Antiviral Agents	Placebo twice daily x 5 d Prednisolone 20 mg three times daily x 5 d, 10 mg three times daily x 3, 10 mg daily x 2 d	66	Within 7 d	6 mo	Yanagihara (40)	UR: < 36 MP: ≥ 10 SP: < 10	Outcome: Number of satisfactory recoveries Results: 69 of 84 (82.1%) vs. 46 of 66 (69.7%) (NS)
		Valacyclovir 1000 mg twice daily x 5 d Prednisolone as above	84					
Lagalla et al,[40] 2002	Cortico-steroids	Placebo solution intravenously daily x 6 d Polyvitamin therapy (2500 μg vitamin B12, 12 mg nicotinamide, 150 mg vitamin C and 0.70 mg folic acid) intramuscularly daily x 15 d	28	Within 72 hrs	12 mo	HB (6)	UR: > 2 MP: n/a SP: n/a	Outcome: Number of satisfactory recoveries Results: 26 of 30 (83.3%) vs. 21 of 28 (75.0%)

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

		Prednisone 1 g intravenously daily x 3 d then 0.5 g intravenously daily x 3d Polyvitamin (as above)	30					(no p value given)
Martinez et al,[41] 1990	Cortico-steroids	No placebo in control arm	45	Within 10d	120 d	Modified Adour Mechelse (5)	UR: < 4 MP: ≥ 2 SP: ≤ 1	<u>Outcome:</u> Number of satisfactory recoveries <u>Results:</u> 41 of 42 (97.6%) vs. 41 of 45 (91.1%) (NS)
		Prednisone 1mg/kg/d daily x 5 d and taper (5 mg/d)	42					
May et al,[42] 1976	Cortico-steroids	Vitamins (dosing regimen unclear)	26	Within 2 d	6 mo	May (function	UR: fair, poor MP:	<u>Outcome:</u> Number of

		Vitamins Prednisone 410 mg total dose (dosing regimen unclear)	25			either complete, fair, poor)	incomplete SP: complete	satisfactory recoveries <u>Results:</u> 15 of 25 (60.0%) vs. 17 of 26 (65.4%) (NS)
Roy et al,[47] 2005	Antiviral Agents	No placebo in control arm Methylprednisolone 5mg/ kg/ d intravenously x 3 d	32	Within 5 d	3 mo	FPRP (10)	UR: < 10 MP: n/a SP: n/a	<u>Outcome:</u> Number of complete recoveries <u>Results:</u> 28 of 32 (87.5%) vs. 28 of 32 (87.5%) (NS)
		Acyclovir 400 mg five times daily x 7 d Methylprednisolone (as above)	32					
Sullivan et al,[35] 2007	Antiviral Agents and Cortico-steroids	Placebo five times daily x 10 d Placebo twice daily x 10 d	141	Within 72 hrs	9 mo	HB (6)	UR: > 1 MP: 2-4 SP: 5-6	<u>Outcome:</u> Number of complete recoveries

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

		Placebo five times daily x 10 d Prednisolone 25 mg twice daily x 10 d	138					<u>Results:</u> Prednisolone vs no prednisolone 237 of 251 (94.4%) vs 200 of 245 (81.6%) ($p < 0.001$) Acyclovir vs no Acyclovir 211 of 247 (85.4%) vs 226 of 249 (90.8%) ($p = 0.10$)
		Acyclovir 400 mg five times daily x 10 d Placebo twice daily x 10 d	138					
		Acyclovir (as above) Prednisolone (as above)	134					
Tekle-Haimanot et al,[43] 1987	Cortico-steroids	Vitamins (B1, B6, B12) (dosing regimen unclear)	29	Within 96 hrs	5 mo	May grading system (function either complete, fair, poor)	UR: fair, poor MP: n/a SP: n/a	<u>Outcome:</u> Number of satisfactory recoveries <u>Results:</u> 19 of 30 (63.3%) vs 18 of 29 (62.1%) (NS)
		Prednisone 40 mg daily x 5 d and taper (5mg/d) (dosing regimen unclear)	30					

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

Unuvar et al,[44] 1999	Cortico-steroids	No placebo given in control arm	21	Within 72 hrs	12 mo	HB (6)	UR: > 2 MP: n/a SP: 4-5	Outcome: Number of satisfactory recoveries Results: 21 of 21 (100%) vs 21 of 21 (100%) (NS)
		Methylprednisolone 1 mg/kg/d daily x 5 d and taper x 5 d	21					
Vazquez et al,[52] 2008	Antiviral Agents	Placebo twice daily x 7 d Prednisone 1 mg/kg daily x 7 d and taper (10 mg/3d) x 14 d	19	Within 72 hrs	6 mo	Sunnybrook (100)	UR: < 90 MP: n/a SP: n/a	Outcome: Number of satisfactory recoveries Results: 18 of 22 (86.4%) vs 17 of 19 (89.5%) (NS)
		Valacyclovir 1g twice daily x 7d Prednisone (as above)	22					
Wolf et al,[45] 1978	Cortico-steroids	No placebo given in control arm	132	Within 5 d	1 yr	Undescribed grading	UR: weakness	Outcome: Number of

		Prednisone 60 mg daily x 10d then 40 mg daily x 2 d then 30 mg daily x 2 d then 10 mg daily x 1 d then 5 mg daily x 2d	107			system (facial weakness graded as none, mild, moderate, severe)	MP: n/a SP: n/a	satisfactory recoveries <u>Results:</u> 94 of 107 (87.6%) vs 106 of 132 (80.3%) (NS)
Yeo et al,[53] 2007	Antiviral Agents	No placebo given in control arm Prednisone 40 mg daily x 4 d and taper (20 mg/2d) x 6 d	47	no limit	6 mo	HB (6)	UR: > 2 MP: n/a SP: n/a	<u>Outcome:</u> Number of satisfactory recoveries <u>Results:</u> 41 of 44 (93.1%) vs 40 of 47 (85.1%) (NS)
		Acyclovir 2400 mg daily x 5 d Prednisone (as above)	44					

Abbreviations: FPRP/FPRI – Facial Paralysis Recovery Profile/Facial Paralysis Recovery Index, HB – House-Brackmann Scale, UR – Unsatisfactory Recovery, MP- Moderate Paresis, SP – Severe Paresis, NS - non-significant

[a] Results for meta-analysis based on House-Brackmann Scale used when multiple scales used in a given study

Table 5. Assessment of Study Quality for Corticosteroids and Antivirals for Treatment of Bell's Palsy.

	Adequate Sequence Generation?	Allocation Conceal-ment?	Blinding?	Incomplete Outcome Data Addressed?	Free of Selective Reporting?	Free of Other Bias? • Description of Other Biases
Adour et al,[54] 1996	Yes	Yes	Yes	Unclear	No	No • Use of *per treatment* analysis
Antunes et al,[38] 2000	Unclear	Unclear	Yes	Yes	No	Yes
Austin et al,[39] 1993	Yes	Yes	Yes	No	No	No • Prognostically imbalanced groups – more severe pareses in control group
Engstrom et al,[37] 2008	Yes	Yes	Yes	No	Yes	No • Premature trial termination • Modified intention to treat analysis • Industry funding

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

Hato et al,[50] 2007	Yes	Yes	No	No	Yes	No • Post randomization exclusions of varicella zoster positive patients
Kawaguchi et al,[51] 2007	Yes	Yes	No	Yes	Yes	No • Post randomization exclusions of varicella zoster positive patients
Lagalla et al,[40] 2002	Yes	Yes	Yes	Yes	No	No • Post randomization exclusions of varicella zoster positive patients
Martinez et al,[41] 1990	Unclear	Unclear	No	No	Yes	Yes
May et al,[42] 1976	Yes	Yes	Yes	Yes	No	Yes
Roy et al,[47] 2005	Yes	Yes	No	Yes	Yes	Yes
Sullivan et al,[35]	Yes	Yes	Yes	Yes	Yes	Yes

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

2007						
Tekle-Haimanot et al,[43] 1987	Unclear	Unclear	No	Yes	Yes	No • Poorly described statistical methods
Unuvar et al,[44] 1999	Yes	Unclear	No	Yes	Yes	Yes
Vazquez et al,[52] 2008	Yes	Yes	Yes	Yes	No	No • Modified intention to treat analysis
Wolf et al,[49] 1978	Unclear	Unclear	No	Unclear	Yes	Yes
Yeo et al,[53] 2007	Unclear	Unclear	Yes	Yes	Yes	Yes

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

Table 6 : Evidence Profile for Corticosteroids for Mild to Moderate and Severe to Complete Paresis (Recommendations 1)

Evidence Profile				**Summary of Findings**					
Outcome	Number of Studies	Number of Patients	Evidence Ratings (Methodologic Limitations, Consistency, Directness, Precision, Publication Bias)	Relative Risk (95% CI)	Assumed Risk without treatment	Corres-ponding Risk with treatment (95% CI)	Number Needed to Treat Benefit (95% CI)	Confidence in Effect Estimate	Outcome Importance
Unsatis-factory Recovery (Mild - Moderate Paresis)	10	1285	No Serious Limitations Consistent Direct Precise Not Detected	0.69 (0.55 – 0.87)	6 per 100[a]	4 per 100 (3 – 5)	50 (33 - 100)	++++ High	Critical
Unsatis-factory Recovery (Severe – Complete Paresis)	10	1285	No Serious Limitations Consistent Direct Precise Not Detected	0.69 (0.55 – 0.87)	39 per 100[a]	27 per 100 (21 – 34)	8 (6 – 20)	++++ High	Critical

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

Major Side Effects	7	1155	No Serious Limitations Consistent Direct Imprecise[b] Not Detected	0.19 (0.01 – 3.96)	3 per 1000[c]	1 per 1000 (0 – 12)	NNTB 500 (NNTB 333 - ∞ - NNTH 111)	+++ Moderate	Critical
Minor Side Effects	7	1155	No Serious Limitations Consistent Direct Imprecise[b] Not Detected	1.20 (0.67 – 2.15)	7 per 100[c]	8 per 100 (5 – 15)	NNTH 100 (NNTB 50 - ∞ - NNTH 13)	+++ Moderate	Important
Synkinesis and Autonomic Dysfunction	3	671	No Serious Limitations Consistent Direct Precise Not Detected	0.56 (0.41 - 0.76)	27 per 100[c]	15 per 100 (11 – 21)	8 (6 – 17)	++++ High	Critical
Pain	1	496	No Serious Limitations Consistency (n/a) Direct Imprecise Not Detected	Mean Difference - 0.08 (-1.10 – 0.94)	n/a	n/a	n/a	+++ Moderate	Important
Quality of Life	1	496	No Serious Limitations Consistency (n/a) Direct Precise Not Detected	Mean Difference -0.06 (-0.09 – -0.03)	n/a	n/a	n/a	+++ Moderate	Critical
Visual Loss	0	0	n/a	n/a	n/a	n/a	n/a	n/a	Critical

Abbreviations: CI – Confidence Interval, NNTB – number of patients needed to treat for one patient to benefit, NNTH – number of patients needed to treat for one patient to be harmed

[a] Assumed risk is derived from a large observational study describing the natural history of untreated BP patients. Unsatisfactory recovery is reported in this study as 29% (overall) and stratified by initial severity of paresis.[3]

[b]Quality of evidence downgraded due to a large confidence interval with the possibility of harm.

[c]Assumed risk was derived from the mean control group event rate for each secondary outcome.

[d]Serious limitations for pain and quality of life outcomes because they are taken from one study and outcomes include combined effect of corticosteroids vs. placebo and corticosteroids and antivirals vs. antivirals, without adjusting for interaction.

Table 7: Evidence Profile for Antiviral Agents for Mild to Moderate and Severe Paresis (Recommendations 2)

Evidence Profile					**Summary of Findings**				
Outcome	Number of Studies	Number of Patients	Evidence Ratings (Methodologic Limitations, Consistency, Directness, Precision, Publication Bias)	Relative Risk (95% CI)	Assumed Risk without treatment	Corres-ponding Risk with treatment (95% CI)	Number Needed to Treat Benefit (95% CI)	Confidence in Effect Estimate	Outcome Importance
Unsatis-factory Recovery (Mild-Moderate Paresis)	2	658	No Serious Limitations Consistent Direct Imprecise[a] Not Detected	1.14 (0.8 -1.62)	6 per 100[b]	7 per 100 (5 – 10)	NNTH 100 (NNTB 100 - ∞ -NNTH 25)	+++ Moderate	Critical

Unsatis-factory Recovery (Severe-Complete Paresis)	2	658	No Serious Limitations Consistent Direct Imprecise[a] Not Detected	1.14 (0.8 -1.62)	39 per 100[b]	44 per 100 (31 – 63)	NNTH 20 (NNTB 13 - ∞ - NNTH 4)	+++ Moderate	Critical
Major Side Effects	2	653	No Serious Limitations Consistent Direct Imprecise[a] Not Detected	0.50 (0.05 -5.40)	3 per 1000[c]	2 per 1000 (0 – 16)	NNTB 1000 (NNTB 333 - ∞- NNTH 77)	+++ Moderate	Critical
Minor Side Effects	2	653	No Serious Limitations Consistent Direct Imprecise[a] Not Detected	0.85 (0.56 - 1.29)	7 per 100[c]	6 per 100 (4 – 9)	NNTB 100 (NNTB 33 - ∞ - NNTH 50)	+++ Moderate	Important
Synkinesis and Autonomic Dysfunction	1	373	No Serious Limitations Consistency (n/a) Direct Imprecise[a] Not Detected	1.04 (0.75 - 1.43)	27 per 100[c]	26 per 100 (19 – 36)	NNTH 100 (NNTB 17 - ∞ - NNTH 9)	+++ Moderate	Critical
Pain (N = 1, n = 496)	1	496	No Serious Limitations Consistency (n/a) Direct Imprecise[a] Not Detected	0.05 (-0.91 – 1.01)	n/a	n/a	n/a	++ Moderate	Important
Quality of Life (N = 1,	1	496	No Serious Limitations Consistency (n/a) Direct	-0.02 (-0.05 – 0.01)	n/a	n/a	n/a	+++ Moderate	Critical

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

n = 496)			Imprecise[a] Not Detected						
Visual Loss	0	0	n/a	n/a	n/a	n/a	n/a	n/a	Critical

Abbreviations: CI – Confidence Interval, NNTB – number of patients needed to treat for one patient to benefit, NNTH – number of patients needed to treat for one patient to be harmed

[a]Quality of evidence downgraded due to a large confidence interval with the possibility of harm.

[b] Assumed risk is derived from a large observational study describing the natural history of untreated BP patients. Unsatisfactory recovery is reported in this study as 29% (overall) and stratified by initial severity of paresis.

[c]Assumed risk was derived from the mean control group event rate for each outcome for the steroid trials listed in the previous table.

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

Table 8: Evidence Profile for Combined Corticosteroids and Antiviral Agents for Mild to Moderate and Severe to Complete Paresis in comparison to Corticosteroid Alone Treatment (Recommendations 3,4)

Evidence Profile					**Summary of Findings**				
Outcome	Number of Studies	Number of Patients	Evidence Ratings (Methodologic Limitations, Consistency, Directness, Precision, Publication Bias)	Relative Risk (compared to steroid treatment alone) (95% CI)	Assumed Risk (steroid treatment alone)	Corres-ponding Risk with combined treatment (95% CI)	Number Needed to Treat Benefit (compared to steroids alone) (95% CI)	Confidence in Effect Estimate	Outcome Importance
Unsatis-factory Recovery (Mild - Moderate Paresis)	8	1298	No Serious Limitations Consistent Direct Imprecise[a] Not Detected	0.75 (0.56 – 1.00)	4 per 100[b]	3 per 100 (2 – 4)	100 (50 - ∞)	+++ Moderate	Critical
Unsatis-factory Recovery (Severe – Complete Paresis)	8	1298	No Serious Limitations Consistent Direct Imprecise[a] Not Detected	0.75 (0.56 – 1.00)	27 per 100[b]	20 per 100 (15 – 27)	14 (8 - ∞)	+++ Moderate	Critical
Major Side Effects	4	941	No Serious Limitations Consistent Direct Imprecise[a] Not Detected	1.33 (0.26 – 6.82)	1 per 1000[c]	1 per 1000 (0 – 7)	∞ (NNTB 1000 - ∞ - NNTH 167)	+++ Moderate	Critical

Minor Side Effects	4	941	No Serious Limitations Consistent Direct Imprecise[c] Not Detected	1.16 (0.82 - 1.62)	8 per 100[b]	9 (7 – 13)	NNTH 100 (NNTB 100 - ∞ - NNTH 20)	+++ Moderate	Important
Synkinesis and Autonomic Dysfunction	3	511	No Serious Limitations Consistent Direct Precise Not Detected	0.59 (0.39 - 0.89)	15 per 100[b]	9 per 100 (4 – 13)	17 (20 – 50)	++++ High	Critical
Pain	0	0	n/a	n/a	n/a	n/a	n/a	n/a	Important
Quality of Life	0	0	n/a	n/a	n/a	n/a	n/a	n/a	Critical
Visual Loss	0	0	n/a	n/a	n/a	n/a	n/a	n/a	Critical

Abbreviations: CI – Confidence Interval, NNTB – number of patients needed to treat for one patient to benefit, NNTH – number of patients needed to treat for one patient to be harmed

[a]Quality of evidence downgraded due variations in treatment effect.

[b]Assumed risk is the risk for that particular outcome in the corticosteroid group (see previous table)

[c]Quality of evidence downgraded fro large confidence intervals with possibility of harm.

[d]Only two studies describe rigorous monitoring of minor adverse-effects.

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

Table 9. Study Characteristics of Studies for Physiotherapy for Treatment of Bell's Palsy

Study	Treatment Studied	Randomization Groups	Number of Patients Randomized	Time to Treatment	Duration of Follow Up	Facial Nerve Grading Scale (max score)	Definitions of Recovery and Initial Severity (based on facial nerve grading scale)	Outcome Results (treatment vs. control)
Barbara et al.,[57] 2010	Exercise/Massage (Kabat Physiotherapy: propriective neuromuscular rehabilitation)	Kabat physiotherapy (daily x 15 d) + control medications (acyclovir 400 mg three times/d x 15d + prednisone 40 mg/d x10 d then 5 d taper)	9	4 d	15 d	HB (6)	Improvement to HB 1 or 2 at 15 d	5/9 improved vs. 3/11
		control medications (as above)	11					

Alakram et al,[67] 2010	Electrostimulation	Electrostimulation (30 min/wk) + control physiotherapy (heat x 5 min/wk, massage x 10 min/wk, exercise x10 reps/wk, home exercise 10 rep 3x/d) + control medication (prednisolone 2mg/kg/d x 14d taper)	8	Within 30 d	3 mo or improvement of 80% on HB scale	HB (6)	Percentage rate of recovery (calculated by difference in HB score divided by time to improvement in weeks)	37.6 ± 18.1 vs. 29.6% ± 12.5 vs. (p=0.36)
		Control physiotherapy (as above) and Control medication (as above	8					
Manikan-dan et al,[66] 2007	Electrostimulation	Electrostimulation with galvanic and faradic currents 3x/d for 14 d, facial exercises with mirror for 3	28	n/a	3 mo	Sunny-brook Facial Grading Score	N/a	Difference in scores post-treatment 16.5 vs. 27.5

		mo, massage				(100)		(p<0.01)
		9Individualized exercises with mirror (facial neuro-muscular re-education, 5-10 reps 3x/d) for 3 mo and massage	28					
Beurskens et al, [60] 2006	Facial exercise	Facial exercises with mirror feedback, Stretch exercises, Massage (10 min/d),	24	9 mo	3 mo	Sunny-brook Facial grading Score (100)	Difference in SB Score or HB Score	Difference in SB 21.5 (95% CI, 17.9-21.5) Difference in HB 0.6 (0.1, 1.1)
		Wait List	24					
Wang et al.,[58] 2004	Facial Exercises	Functional Exercises and Control treatment (cortisone, mexobalamin,	43	n/a	1 mo	Pottman Score	Facial Function	MD = 8.47, 95% CI, 7.05 – 9.89

		vitamin B12, physical therapy, massage and acupuncture (details not described)						
		Control treatment (as above)	31					
Wen et al,[59] 2004	Facial Exercises	Facial rehabilitation exercises and "conventional therapy" (not described)	85	n/a	3 mo	n/a	Unsatis-factory Recovery	92.9% vs 88.3% recovery (RR = 0.61, 0.21 – 1.71)
		Conventional therapy (as above)	60					
Flores et al.,[65]1998	Electrostimulation	Electrostimulation and infrared	77	Within 3 d	6 mo	May Scale	Time to reccovery	10/77 (13.0%) vs 11/72 (15.3%) [RR = 0.85, 0.38 -1.88)]
		Prednisone for up to 14 d	72					
Mosforth et al.,[64]	Electrostimulation	Electrostimulation (infrared and interrupted	43	Within 2 wk	6 mo	unclear	Unsatis-factory	14/43 vs 9/40

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

1958		galvanic currents daily until contractions resumed, then 3x/wk) and massage daily for 2-6 mo.					Recovery	
		Massage daily	40					

Abbreviations: HB- House-Brackmann facial grading system

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

Table 10. Assessment of Study Quality for Physiotherapy for Treatment of Bell's Palsy

	Adequate Sequence Generation	Allocation Concealment	Blinding	Incomplete Outcome Addressed	Free of Selective Reporting	Free of Other Bias
Barbara et al.,[57] 2010	Unclear	Unclear	Unclear	No[a]	Yes	Yes
Alakram et al.,[67] 2010	No[b]	No	No	No[a]	Yes	Yes
Manikandan et al.,[66] 2007	Yes	Yes	No	No[a]	Yes	Yes
Beurskens et al.,[60] 2006	No[c]	No	Yes	No[a]	Yes	Yes
Wang et al.,[58] 2004	Yes	Yes	Yes	No[a]	Yes	Yes
Wen et al.,[59]	Unclear	Unclear	No	No[a]	Yes	Yes

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

2004						
Flores et al.,[65] 1998	Unclear	Unclear	No	No[a]	Yes	Yes
Mosforth et al.,[64] 1958	Yes	Yes	No	No[a]	Yes	Yes

[a] No reporting of adverse outcomes and other patient important outcomes

[b] Patients not truly randomized, alternating patients received experimental or control treatment.

[c] Coin flip

Table 11: Evidence Profile for Exercise Physiotherapy (Recommendation 5,6)

Evidence Profile				**Summary of Findings**				
Outcome	Number of Patients	Evidence Ratings (Methodologic Limitations, Consistency, Directness, Precision, Publication Bias)	Relative Risk (95% CI)	Assumed Risk without treatment	Corres-ponding Risk with treatment (95% CI)	Number Needed to Treat Benefit (NNTB) (95% CI)	Confidence in Effect Estimate	Outcome Importance
Barbara 2010								
Unsatis-factory Recovery (15 days)	20	Serious Limitations[a] n/a Indirect Imprecise Not Detected	0.61 (0.27 – 1.38)	73 per 100[b]	44 per 100 (20 – 100)	3 (NNTB 2 - ∞ - NNTH 4)	+ Very Low	Critical
Beurskens 2006								
Improve-ment in Facial Function for chronic paralysis	48	Serious Limitations[a] n/a Direct Imprecise Not Detected	MD = 0.6[c] (0.1 – 1.1)	n/a	n/a	n/a	++ Low	Critical
Wang 2004								

Improve-ment in Facial Function	74	No Serious Limitations n/a Indirect Imprecise Not Detected	MD = 8.47[d] (7.05 – 9.89)	n/a	n/a	n/a	++ Low	Critical
Wen 2004								
Unsatis-factory Recovery	145	Serious Limitations[a] n/a Indirect Imprecise Not Detected	0.61 (0.21 – 1.71)	29 per 100	18 per 100 (6 – 50)	9 (NNTB 4 - ∞ - NNTH 5)	+ Very Low	Critical
Synkinesis	145	Serious Limitations[a] n/a Indirect Precise Not Detected	0.24 (0.08 – 0.69)	27 per 100	6 per 100 (2 – 19)	5 (4 – 13)	++ Low	Critical

MD – mean difference; Abbreviations: CI – Confidence Interval, NNTB – number of patients needed to treat for one patient to benefit, NNTH – number of patients needed to treat for one patient to be harmed

[a]no description of randomization, allocation, blinding.

[b] assumed risk refers to the unsatisfactory recovery rate at 15 days after onset of symptoms with treatment using steroids and antivirals. The rate is derived from the control group unsatisfactory recovery rate.

[c] Mean difference in House-Brackmann score between experimental and control groups after three months using pre-test score as a covariate using ANCOVA.

[d] Mean difference in Pottman score between experimental and control groups after one month.

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

Table 12: Evidence Profile for Electrostimulation Physiotherapy (Recommendation 7)

Evidence Profile				**Summary of Findings**				
Outcome	Number of Patients	Evidence Ratings (Methodologic Limitations, Consistency, Directness, Precision, Publication Bias)	Relative Risk (95% CI)	Assumed Risk without treatment	Corres-ponding Risk with treatment (95% CI)	Number Needed to Treat Benefit (NNTB) (95% CI)	Confidence in effect estimate	Outcome Importance
Alakram 2010								
Percentage rate of Recovery	16	Serious Limitations[a] n/a Indirect Imprecise Not Detected	37.6 (18.1) vs. 29.6 (12.5) (p = 0.36)[b]	n/a	n/a	n/a	+ Very Low	Critical
Manikandan 2007								
Improve-ment in Facial Function	56	Serious Limitations[a] n/a Indirect Imprecise Not Detected	MD = 12.00 (1.26 - 22.74) {favours control}[c]	n/a	n/a	n/a	+ Very Low	Critical

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

Synkinesis	56	Serious limitations[a] n/a Indirect Imprecise Not Detected	0.20 (0.01 – 3.99) {favours control}	n/a	n/a	n/a	+ Very Low	Critical
Flores 1998								
Unsatis-factory Recovery	149	Serious Limitations[a] n/a Indirect Imprecise Not Detected	0.85 (0.38 – 1.88)	20 per 100	17 per 100 (8 – 38)	33 (NNTB 8 - ∞ - NNTH 5)	+ Very Low	Critical
Mosforth 1958								
Unsatis-factory Recovery	83	Serious Limitations[a] n/a Indirect Imprecise Not Detected	1.45 (0.71 – 2.97)	29 per 100	42 per 100 (21 – 86)	NNTH 8 (NNTB 13 - ∞ - NNTH 2)	+ Very Low	Critical
Synkinesis	83	Serious limitations n/a Indirect Imprecise Not Detected	1.25 (0.56 – 2.79)	27 per 100	34 per 100 (15 – 75)	NNTH 14 (NNTB 8 - ∞ - NNTH 2)	+ Very Low	Critical

MD – mean difference; Abbreviations: CI – Confidence Interval, NNTB – number of patients needed to treat for one patient to benefit, NNTH – number of patients needed to treat for one patient to be harmed

[a]Serious deficiencies in methodology including at least one of the following no blinding, no allocation concealment, incomplete outcomes assessed and inadequate random sequence generation.

[b]Measure of effect listed here is a formula described by the author as percentage rate of recovery which is listed as number of grades of improvement in House-Brackman score divided by number of weeks.

[c]Mean Difference in Sunnybrook score.

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

Table 13. Study Characteristics of Studies for Surgical Decompression for Treatment of Bell's Palsy

Study, Year	Cohort Size (n, Surgery; n, Medical or Conservative Therapy)	Age Mean (range)	Severity of Bell Palsy	Duration of Bell palsy (days)	Follow-up (mo.)	Surgical Approach	Control group therapy	Outcome Scale	Definition of Recovery	Masked Outcome Assessment	RR, fair recovery (95% CI)	RR, complete recovery (95% CI)	Complications of Surgery
Brown et al.,[68] 1982	92 (41, 51)	NR	complete palsy	≤14	12	Vertical horizontal (56%), stylomastoid (37%), mid cranial fossa (7%)	Supportive therapy (10/51); Cortisone (41/51)	Complete, Fair, Poor†	Complete or Fair recovery assessed by blinded opthalmologist	Yes	1.09 (0.90–1.32)	1.30 (0.89–1.90)	Conductive deafness (10%); perceptive deafness in (5%); "giddiness" (2%)
Gantz et al,[69] 1999	70 (34,36)	(20-66)	complete palsy + poor prognosis based on ENOG and EEMG	≤14	7	Meatal foramen and labyrinthine decompression through mid cranial fossa; 9% had combined mastoid and mid cranial fossa approach	Steroids	H-B	HB 1 or 2	No	2.19 (1.47–3.27)	2.96 (1.20–7.34)	Hearing loss (4%) and CSF leak (4%)
May et al,[70] 1981‡	60 (42, 18)	NR	Complete palsy with poor prognosis based on LT, ST, MST, EEMG (55/60); Incomplete palsy wih poor prognosis (5/50)	≤14	6	Decompression of stylomastoid to labyrinthine segement using transmastoid approach (36/42); vertical mid-horizontal (6/42)	Steroids (6/18); supportive therapy (12/18)	Same as Brown 1982	Complete or Fair recovery assessed by 10 blinded physicians who examined photographs	Yes	1.14 (0.79 - 1.65)	6.43 (0.92-45.07)	NR
May et al,[71] 1985‡	38 (25,13)	NR	complete palsy + poor prognosis	≤14	6	Transmastoid, Extralabyrinthine,	Supportive or steroids	Modified H-B	HB 1 or 2	Yes	0.87 (0.24–	0/25 vs. 1/13	NR

			based on EEMG			Subtemporal					3.07)		
Fisch et al,[72] 1981	27 (14,13)	42 (25-72)	complete palsy + poor prognosis based on EEMG	≤21	12-36	Mid cranial fossa (93%) or Combined transmastoid and mid cranial fossa (7%)	No surgery; other interventions not described	Composite score based on objective and subjective global evaluation (0-100%); and detailed evaluation of facial symmetry scale (0-100)	Investigators did not analyse for partial or complete recovery. For the purposes of the review, complete recovery defined as score of 100 for DEFS	Yes	-	3.30 (0.82 – 12.90)	Hearing loss (7%)
Yanagihara et al,[73] 2001	2001	49 (16-84)	HB Grade V or VI, despite steroid therapy x 2 weeks	>14	12	Transmastoid approach; decompression of distal labyrinthine to stylomastoid	No surgery; all patients received steroids	H-B	H=B 1 or 2	No	1.17 (0.97 - 1.57)	1.93 (1.04-3.56)	No complications

†Complete recovery (symmetric voluntary and spontaneous facial movements); Fair (slight to moderate facial paralysis and synkinesis); Poor (severe facial paralysis and synkinesis);

‡some patients were enrolled in both studies H-B = House-Brackmann; EEMG = evoked EMG; ENoG = electroneuronography; MST=maximal stimulation test; LT = lacrimation test; ST = salivary test

Trial (Author, year of publication)	Adequate sequence generation	Adequate allocation concealment	Blinding	Complete outcomes reporting[a]	Complete outcomes data	Free of Other Bias	Overall risk of bias

Appendix to: de Almeida JR, Guyatt GH, Sud S, et al. Management of Bell palsy: clinical practice guideline. *CMAJ* 2014. DOI 10.1503/cmaj.131801.

Brown et al.,[68] 1982	No	No	No[b]	Yes	Yes	Yes	High
Gantz et al.,[69] 1999	No	No	No	Yes	No[c]	Yes	High
May et al.,[70] 1981	No	No	No[b]	No[a]	Yes	Yes	High
May et al.,[71] 1985	No	No	No[b]	No[a]	Yes	Yes	High
Fisch et al.,[72] 1981	No	No	No[b]	No[a]	Yes	Yes	High
Yanagihara et al.,[73] 2001	No	No	No	Yes	Yes	Yes	High

Table 14. Assessment of Study Quality for Surgical Decompression for Treatment of Bell's Palsy

[a]surgical complications not reported

[b]Outcome assessors were blinded to treatment received; patients and treating physicians were not blinded in any study

[c]3 patients who underwent surgical decompression were lost to follow-up and not included in the analysis