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

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Article abstract—Objective: To determine the effectiveness of steroids, acyclovir, and surgical facial nerve decompression in Bell's palsy. Methods: The authors identified articles by searching MEDLINE and selected those that prospectively compared outcomes in patients treated with steroids, acyclovir, or surgery with patients not receiving these modalities. The authors graded the quality of each study (class I to IV) using a standard classification-of-evidence scheme. They compared the proportion of patients recovering facial function in the treated group to the proportion of patients recovering facial function in the control group. Results: The authors identified no adequately powered class I studies for any treatment modality. The pooled results of two class I and two class II studies showed significantly better facial outcomes in steroid-treated patients compared with non-steroid-treated patients (relative rate good outcome 1.16, 95% CI 1.05 to 1.29). One class II study demonstrated a significant benefit from acyclovir in combination with prednisone compared with prednisone alone (relative rate good outcome 1.22, 95% CI 1.02 to 1.45). All studies describing outcomes in patients treated with facial nerve decompression were graded as class IV. Conclusion: For patients with Bell's palsy, a benefit from steroids, acyclovir, or facial nerve decompression has not been definitively established. However, available evidence suggests that steroids are probably effective and acyclovir (combined with prednisone) is possibly effective in improving facial functional outcomes. There is insufficient evidence to make recommendations regarding surgical facial nerve decompression for Bell's palsy. Well-designed studies of the effectiveness of treatments for Bell's palsy are still needed.

The Quality Standards Subcommittee of the American Academy of Neurology is charged with developing practice parameters for neurologists for diagnostic procedures, treatment modalities, and clinical disorders. The selection of topics for which practice parameters are developed is based on prevalence, frequency of use, economic impact, membership involvement, controversy, urgency, external constraints, and resources required. This report addresses the effectiveness of controversial therapies for Bell's palsy.

Bell's palsy is an acute, peripheral facial paresis of unknown cause.1 Usually, the diagnosis is established without difficulty in patients presenting with unexplained unilateral isolated facial weakness.2 Most patients with Bell's palsy recover without treatment—71% achieve complete recovery, 84% achieve near normal function.3 The disease is common, with an annual incidence of 20 per 100,000. Thus, despite its good prognosis, Bell's palsy leaves more than 8,000 people in the United States each year with permanent, potentially disfiguring facial weakness.

Commonly employed, noncontroversial treatment modalities for Bell's palsy include eye patching and lubrication to protect the cornea.4 Controversy remains regarding the effectiveness of commonly used pharmacologic therapies—steroids and acyclovir—as well as surgical facial nerve decompression.

Although the etiology of Bell's palsy remains unclear, there are reasons to believe steroids, acyclovir, or facial nerve decompression might improve outcomes in patients with this disorder. Bell's palsy may result from inflammation and subsequent me-
chanical compression of the facial nerve in the temporal bone, possibly initiated by the herpes simplex virus. Steroids might reduce facial nerve inflammation, and surgery might relieve facial nerve compression, whereas acyclovir might treat the putative inciting infection.

To determine if steroids, acyclovir, and surgical facial nerve decompression are effective in improving facial functional outcomes in Bell’s palsy, we performed a systematic review and analysis of the literature. Based on this review, we propose recommendations for the use of these therapies.

**Process.** Identification and selection of studies. We searched the National Library of Medicine’s MEDLINE database from 1966 to June 2000. Three searches were performed in which we combined the term “facial paralysis or Bell’s palsy” with “prednisone or prednisolone or hydrocortisone,” “acyclovir,” and “surgery.” We subsequently screened the resultant articles and their references for those studies that compared outcomes in prospectively assembled Bell’s palsy patients treated with steroids, acyclovir, or surgery to concurrent patients not treated with these modalities.

Study characteristics. The following study design characteristics were extracted from the identified articles:

- Cohort size and study setting.
- Treatment allocation method.
- Age, sex, severity of palsy, and duration of palsy before treatment.
- Medication regimen used or decompression procedure performed.
- Length of follow-up.
- Percentage of patients completing the study.
- Method of facial function outcome assessment, including the use of masking.

We graded the quality of the evidence provided by each study (class I, II, III, IV) using the classification-of-evidence scheme in Appendix 1. In this scheme, class I studies are judged to have a low risk of bias and class IV studies are judged to have a high risk of bias. Studies were graded independently by each author. Differences were resolved after discussion.

Measures of therapeutic effect. For each study, using two-by-two tables, we compared the proportion of patients recovering good facial function in the treated group to the proportion of patients recovering good facial function in the control group by calculating the relative rate (RR) by means of the following formula:

\[
RR = \frac{A/(A + C)}{B/(B + D)}.
\]

<table>
<thead>
<tr>
<th>Recovery</th>
<th>Good</th>
<th>Poor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treated</td>
<td>A</td>
<td>C</td>
</tr>
<tr>
<td>Control</td>
<td>B</td>
<td>D</td>
</tr>
</tbody>
</table>

In separate analyses, we calculated the RR at which patients in the treated group recovered complete facial function. We also calculated the 95% CI of the RR.

In studies using the House and Brackmann facial function scoring system, we considered an outcome of grade I or II a good recovery. When comparing the proportion of patients recovering complete facial function, we considered an outcome of grade I a complete recovery. In studies using the Adour/Swanson grading scale, we considered a facial paralysis recovery profile (FPRP) of greater than seven and a recovery index (FPRI) of greater than five a good recovery. We considered an FPRP of 10 and an FPRI of 10 a complete recovery.

When necessary to improve the precision of the measured RR, we pooled the results from different studies using general variance-based meta-analytic techniques. To minimize the risk of bias in the resulting summary estimate of effect, we pooled studies with the lowest risk of bias first, adding studies with a higher risk of bias only when necessary to further increase precision.

Recommendations. Only studies receiving a grade of class III or better were considered in the formulation of the recommendations. We formulated practice recommendations after considering the estimated effect sizes, the significance of the effect, and the consistency of the effect between studies.

To account for the quality of evidence, we determined a strength-of-recommendation level for each recommendation using the scheme in Appendix 2. We determined the strength of recommendation based on the number and quality of studies available to derive the estimate of effect. Thus, for example, an intervention demonstrating a consistent and significant benefit in two class I studies would earn a level “A” recommendation. We planned to recommend such an intervention as established as effective. An intervention demonstrating a consistent and significant benefit in two class II studies would earn a level “B” recommendation and would be recommended as probably effective. Similarly, an intervention demonstrating a consistent and significant benefit in two class III studies would earn a level “C” recommendation. We planned to recommend such an intervention as possibly effective.

**Analysis.** In patients with Bell’s palsy, do steroids improve facial functional outcomes? Our search identified 230 articles that described steroid use for the treatment of Bell’s palsy. Nine8,10-17 of these studies prospectively compared outcomes in patients treated with oral steroids to concurrent patients who were not treated with steroids. The characteristics of these studies are listed in table 1.

Study characteristics. In these nine studies, patients meeting standard diagnostic criteria for Bell’s palsy were allocated to treatment with steroids or placebo. Most studies limited enrollment to adults. One study17 enrolled children only. The proportion of patients with severe facial weakness in each study varied considerably (0 to 91%). The time allowed
from symptom onset to treatment allocation also varied widely between studies (2 to 14 days).

With the exception of one study that used hydrocortisone, authors used oral prednisone or prednisolone. Authors from another study did not specify the corticosteroid used, but we assumed it was prednisone because the dosage was similar to other studies using this medication. Multiple dosage regimens of oral steroids were used. The most commonly reported regimen was 1 mg/kg of oral prednisone, up to 70 mg per day, split into twice-daily dosing. The starting dose was continued for 6 days, then tapered off over a subsequent 4 days. Outcomes were assessed in a masked fashion.

We graded the evidence from two studies as class I, II, and III studies were mixed. The two class I studies and one class II study did not show significantly better outcomes in steroid-treated patients. However, these studies were insufficiently powered to exclude a clinically important effect from steroids. One class II and one class III study demonstrated significantly better outcomes in the steroid-treated patients. In these studies, patients with Bell’s palsy were 1.2 times as likely to attain good facial functional recovery as untreated patients. No study showed significantly worse facial functional outcomes in patients treated with steroids. Four of five of the studies demonstrated a trend for better outcomes in the steroid-treated patients.

No study showed a significant difference in the time to recovery between steroid-treated patients and controls. One class I study reported a median

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Cohort size</th>
<th>Age, y (range)</th>
<th>Female sex, %</th>
<th>Rx steroid dose</th>
<th>Duration, d</th>
<th>Follow-up, mo</th>
<th>Completion rate, %</th>
<th>Blind</th>
<th>Class</th>
<th>NH, %</th>
<th>RR good recovery (CI)</th>
<th>RR complete recovery (CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>May8</td>
<td>1976</td>
<td>51</td>
<td>53% &gt;30</td>
<td>45</td>
<td>Prednisone 410 mg, 10 d</td>
<td>47</td>
<td>2</td>
<td>6</td>
<td>100</td>
<td>Yes</td>
<td>81</td>
<td>0.99 (0.76–1.30)</td>
<td>0.92 (0.60–1.44)</td>
</tr>
<tr>
<td>Taverner9</td>
<td>1954</td>
<td>26</td>
<td>Mean 40 (12–76)</td>
<td>50</td>
<td>Hydrocortisone 1 g, 8 d</td>
<td>23</td>
<td>9</td>
<td>NS</td>
<td>100</td>
<td>Yes</td>
<td>67</td>
<td>1.07 (0.64–1.80)</td>
<td>—</td>
</tr>
<tr>
<td>Brown10</td>
<td>1982</td>
<td>82</td>
<td>—</td>
<td>—</td>
<td>Named 400 mg, 10 d</td>
<td>0</td>
<td>3</td>
<td>12</td>
<td>100</td>
<td>Yes</td>
<td>73</td>
<td>1.20 (0.97–1.50)</td>
<td>1.20 (0.97–1.49)</td>
</tr>
<tr>
<td>Austin11</td>
<td>1993</td>
<td>76</td>
<td>Mean 37 (18–71)</td>
<td>49</td>
<td>Prednisone 405 mg, 10 d</td>
<td>22</td>
<td>5</td>
<td>6</td>
<td>71</td>
<td>Yes</td>
<td>83</td>
<td>1.21 (1.05–1.39)</td>
<td>1.71 (1.00–2.95)</td>
</tr>
<tr>
<td>Shafshak12</td>
<td>1994</td>
<td>160</td>
<td>(17–60)</td>
<td>19</td>
<td>Prednisolone 420 mg, 10 d</td>
<td>91</td>
<td>6</td>
<td>12</td>
<td>100</td>
<td>Yes</td>
<td>69</td>
<td>1.24 (1.03–1.49)</td>
<td>1.76 (1.08–2.87)</td>
</tr>
<tr>
<td>Wolf13</td>
<td>1978</td>
<td>239</td>
<td>Median 31–40 (5–70)</td>
<td>—</td>
<td>Prednisone 760 mg, 17 d</td>
<td>31</td>
<td>5</td>
<td>12</td>
<td>100</td>
<td>No</td>
<td>98</td>
<td>1.02 (0.99–1.06)</td>
<td>1.09 (0.98–1.22)</td>
</tr>
<tr>
<td>Adour14</td>
<td>1972</td>
<td>304</td>
<td>Median 20–39</td>
<td>53</td>
<td>Prednisone 216 mg, 12 d</td>
<td>NS</td>
<td>14</td>
<td>1</td>
<td>85</td>
<td>No</td>
<td>64</td>
<td>1.39 (1.20–1.62)</td>
<td>1.58 (1.25–2.00)</td>
</tr>
<tr>
<td>Abraham–Inpijn15</td>
<td>1987</td>
<td>200</td>
<td>Mean 41</td>
<td>50</td>
<td>Prednisone 570 mg, 15 d</td>
<td>51</td>
<td>&lt;11</td>
<td>89%</td>
<td>12</td>
<td>100</td>
<td>62</td>
<td>—</td>
<td>1.38 (0.97–2.00)</td>
</tr>
<tr>
<td>Unuvar16</td>
<td>1999</td>
<td>22</td>
<td>Mean 4.4</td>
<td>50</td>
<td>Prednisolone 1 mg/ kg, 10 d</td>
<td>100</td>
<td>3</td>
<td>12</td>
<td>100</td>
<td>No</td>
<td>100</td>
<td>1.00</td>
<td>1.00</td>
</tr>
</tbody>
</table>

Completion rate: percentage of subjects followed to study completion; severity: percentage of patients with complete palsy; duration: maximum duration of palsy before starting steroids.

Rx = medication; CI = 95% confidence interval; NH = natural history, percentage of non–steroid-treated patients attaining a good outcome; RR = relative rate of steroid-treated patients attaining outcome compared to non–steroid-treated patients; NS = not stated.
time to recovery of 45 days in both steroid-treated and untreated patients. One class II13 and one class I study11 reported a trend for steroid-treated patients to recover faster than did control patients, but the differences were not significant. The average number of days to recovery were 51 vs 69 and 63 vs 69, respectively.

None of the class I, II, or III studies described a significant decrease in the frequency of autonomic synkinesis (e.g., “crocodile tears”) in patients treated with steroids.

Some authors have suggested that steroids work best in patients with Bell’s palsy if started early.18 The articles reviewed provided little evidence to support or refute this assertion. Most of the patients enrolled in these studies were treated within 1 week of onset of facial paralysis. The class III study14 showed a nonsignificant trend of more benefit in patients who received steroids early (by day 1: RR 1.25; by day 2: RR 1.19; by day 3: RR 1.12).

Patient subgroups. Few articles provided information regarding the response of Bell’s palsy patient subgroups, such as patients with diabetes mellitus, hypertension, or recurrent facial palsy. Thus, we were unable to determine if the association between steroid treatment and facial outcomes was different in these patient populations.

Bell’s palsy patients with incomplete facial paralysis have excellent outcomes regardless of therapy.3 Thus, some have suggested that patients with complete facial palsy benefit most from steroids.15 The studies reviewed here provided little evidence relative to this issue. In a two-way analysis of variance of time to recovery, one class II study13 found no interaction between treatment and facial outcomes was different in these patient populations.

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Complications. Three studies8,11,13 discussed steroid side effects. Side effects occurred in 1 to 4% of treated patients. These side effects, in descending order of frequency, were dyspepsia, loss of blood sugar control, recurrent duodenal ulcers, mood swings, and acute psychosis. All effects resolved when treatment was stopped.

Combining evidence. The rates of facial recovery in steroid-treated patients relative to non–steroid-treated patients extracted from each study are plotted in the figure. The measured RR are ordered, left to right, by class of evidence. The 95% CI are represented by vertical lines.

None of the studies reviewed were conclusive. The RR with the lowest risk of bias came from the class I studies,10,11 Both employed random, masked methodologies. Although at low risk for bias, the measured RR from these class I studies were the least precise. This is indicated in the figure by the tall CI. These class I studies enrolled too few patients to definitively exclude an important effect (either benefit or harm) from steroids.

A more precise measure of the effect of steroids came from the single class III study.14 In this study, authors enrolled the largest number of patients.

However, the RR derived from this class III study was also the most prone to bias. The nonrandom treatment allocation employed in this study resulted in prognostically important differences between steroid-treated and non–steroid-treated patients. These confounding differences may have resulted in a spurious association between steroids and improved facial outcomes.

To increase the precision of the measured RR while minimizing the risk of bias, we statistically pooled the rates from the two class I studies. The pooled result from these studies10,11 did not demonstrate a significant benefit from steroids (RR 1.01). However, the 95% CI of the combined RR was still too wide (0.80 to 1.27). The pooled result was insufficiently precise to be conclusive.

To further increase precision, we combined the RR of good facial recovery from the class I and class II studies. While increasing the precision of the derived RR of recovery, including the class II studies increased the risk of bias in the summary estimate of effect. The pooled RR from the two class I and two class II studies demonstrated a significant association between steroids and good outcomes (RR 1.16, 95% CI 1.05 to 1.29, vertical diamond in the figure). Thus, assuming 80% of patients with Bell’s palsy attain good facial outcomes without steroid treatment, an additional 14% might attain good outcomes if treated with steroids. The pooled effect from the class I and II studies was homogenous (p = 0.59) with overlapping CI. This suggests that the differences in the study results were potentially related to chance alone (sampling error).

Conclusion. Because of the absence of sufficiently powered class I studies, we conclude that a benefit of steroids in Bell’s palsy has not been definitively established. However, the available evidence supports a level “B” recommendation using the scheme in Appendix 2. Thus, based on the pooled result of class I and class II studies and a relatively benign side effect profile, we conclude that steroids
are safe and probably effective in improving facial functional outcomes in patients with Bell’s palsy.

In patients with Bell’s palsy, does acyclovir improve facial functional outcomes? Our search strategy identified 92 articles that described acyclovir use for the treatment of Bell’s palsy. Three19-21 of these studies prospectively compared outcomes in treated patients with those not treated with acyclovir. Study characteristics and outcomes of these studies are listed in table 2.

Study characteristics. In all of these studies, patients meeting standard diagnostic criteria for Bell’s palsy were allocated to treatment with acyclovir or prednisone. Two studies19,20 compared the effect of a combination of acyclovir and prednisone vs prednisone alone. One study20 compared acyclovir alone to prednisone alone. The dose of acyclovir varied between studies from 1,000 mg a day for 5 days to 2,400 mg a day for 10 days. Outcomes were measured after 3 to 12 months of follow-up.

One study19 employed randomized treatment allocation and masked outcome assessments. However, 17% of enrolled patients were lost to follow-up. For this reason, we graded evidence from this study as class II. Because of unmasked, nonindependent outcome assessments, as well as other methodologic flaws, the evidence from the two remaining studies20,21 was graded as class IV.

Therapeutic effect. Table 2 lists the rates of good or complete recovery in acyclovir-treated patients relative to patients treated with prednisone alone.

The single class II study19 demonstrated a significant benefit of acyclovir. In this study, patients treated with acyclovir and prednisone were 1.22 times more likely to attain good outcomes than patients treated with prednisone alone (95% CI 1.02 to 1.45). Thus, assuming 80% of patients with Bell’s palsy attain good outcomes on steroids alone, an additional 18% might attain good outcomes if treated with acyclovir and steroids.

Complications. The reported frequencies and nature of side effects in the acyclovir trials were similar to those with steroids.18-21 It was impossible to determine if the side effects reported were secondary to acyclovir or prednisone.

Conclusion. Because of the absence of class I studies, we conclude that a benefit of acyclovir in Bell’s palsy has not been definitively established. However, the available evidence supports a level “C” recommendation using the scheme in Appendix 2. Thus, based on the result of a single class II study and a relatively benign side effect profile, we conclude that acyclovir (combined with prednisone) is safe and possibly effective in improving facial functional outcomes in patients with Bell’s palsy.

In patients with Bell’s palsy, does facial nerve decompression improve facial functional outcomes? We found 104 articles describing surgical facial nerve decompression in patients with Bell’s palsy. Four12,22-25 of these studies prospectively compared outcomes in patients treated with surgery to those not treated. The characteristics and outcomes of these studies are listed in table 3.

Study characteristics. In all studies, patients meeting standard diagnostic criteria for Bell’s palsy were allocated to treatment with facial nerve decompression or medical therapy. The majority of patients in each study had complete facial paralysis and for this reason had poorer prognoses.1 Most had been treated with steroids. Authors reported varied surgical approaches. Outcomes were measured after 6 to 36 months of follow-up.

Patients were not randomly allocated to surgical and nonsurgical groups in any study. Additionally, no study described masked or independent assessment of facial functional outcomes. For these reasons, the evidence from all of these studies was graded as class IV.

Therapeutic effect. Table 3 lists the rates of good or complete recovery in patients undergoing facial nerve decompression relative to nonsurgical patients from each of the class IV studies. Only one study22 demonstrated a significant association between surgery and improved facial outcome.

Complications. Permanent unilateral deafness was the most common serious side effect from facial
The design of such studies should include the following: of the intervention relative to its potential benefit. Plating such studies should carefully weigh the risk modalities are still needed. Investigators contemplations. Definitive studies of the effectiveness of these prone methodologies that preclude definitive conclusions. Including insufficient statistical power and bias-roids, acyclovir, and facial nerve decompression for available evidence regarding the effectiveness of ste-preceding recommendations are based on the best evidence-based recommendations for the use of facial nerve decompression in patients with Bell’s palsy. All of the studies reviewed had flaws, including permanent hearing loss, were reported from surgical facial nerve decompression. For these reasons, we were unable to develop evidence-based recommendations for the use of facial nerve decompression in patients with Bell’s palsy. 

**Practice recommendations.** For patients presenting with Bell’s palsy:

- Early treatment with oral steroids is recommended as probably effective to improve facial functional outcomes (Level B).
- Early treatment with acyclovir in combination with prednisone is recommended as possibly effective to improve facial functional outcomes (Level C).

There is insufficient evidence to make recommendations regarding the use of facial nerve decompression to improve facial functional outcomes (Level U).

**Recommendations for future research.** The preceding recommendations are based on the best available evidence regarding the effectiveness of steroids, acyclovir, and facial nerve decompression for Bell’s palsy. All of the studies reviewed had flaws, including insufficient statistical power and bias-prone methodologies that preclude definitive conclusions. Definitive studies of the effectiveness of these modalities are still needed. Investigators contemplating such studies should carefully weigh the risk of the intervention relative to its potential benefit. The design of such studies should include the following:

- Random allocation to treatment groups.
- Complete follow-up of enrolled patients.

Masked, standardized outcome assessments, including time to maximum recovery.

Sufficient power to detect important differences between therapies.

Subgroup analyses to detect interactions between treatment, severity of paralysis, duration of palsy before initiation of therapy, and patient characteristics such as the diabetes mellitus, hypertension, and recurrent palsy.

**Disclaimer.** This statement is provided as an educational service of the American Academy of Neurology. It is based on an assessment of current scientific and clinical information. It is not intended to include all possible proper methods of care for a particular neurologic problem or all legitimate criteria for choosing to use a specific procedure. Neither is it intended to exclude any reasonable alternative methodologies. The AAN recognizes that specific care decisions are the prerogative of the patient and the physician caring for the patient, based on all of the circumstances involved.

**Acknowledgment**

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**Appendix 1**

**Definitions for classification of evidence**

**Class I.** Evidence provided by a randomized, controlled clinical trial (RCT) with masked outcome assessment in a representative population. The following are required: a) primary outcomes are clearly defined; b) exclusion and inclusion criteria are clearly stated; c) adequate accounting of dropouts and crossovers with numbers sufficiently low to have minimal potential for bias; and d) relevant baseline characteristics are substantially equivalent among treatment groups.

**Table 3** Design characteristics and outcomes in controlled studies of patients with Bell’s palsy treated with facial nerve decompression

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Cohort size (range)</th>
<th>Mean age, y (range)</th>
<th>Female sex, %</th>
<th>Surgical approach</th>
<th>Severity, %</th>
<th>Duration, d</th>
<th>Follow-up, mo</th>
<th>Completion rate, %</th>
<th>Blind</th>
<th>Class</th>
<th>NH, %</th>
<th>RR good recovery (CI)</th>
<th>RR complete recovery (CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brown22</td>
<td>1982</td>
<td>92 — —</td>
<td>Vertical, stylo-mastoid, mid cranial fossa</td>
<td>100</td>
<td>14</td>
<td>12</td>
<td>100</td>
<td>No</td>
<td>IV</td>
<td>47</td>
<td>1.21 (0.97–1.5)</td>
<td>1.30 (0.89–1.90)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gantz22</td>
<td>1999</td>
<td>70 (20–66) —</td>
<td>Mid cranial fossa &amp; meatal foramen</td>
<td>100</td>
<td>14</td>
<td>7</td>
<td>100</td>
<td>No</td>
<td>IV</td>
<td>42</td>
<td>2.19 (1.47–3.27)</td>
<td>2.96 (1.20–7.34)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>May22</td>
<td>1985</td>
<td>38 — —</td>
<td>Transmastoid, extralabyrinthine, subtemporal</td>
<td>100</td>
<td>14</td>
<td>6</td>
<td>100</td>
<td>No</td>
<td>IV</td>
<td>23</td>
<td>0.87 (0.24–3.07)</td>
<td>—</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fisch22</td>
<td>1981</td>
<td>27 42 (25–72) 33</td>
<td>Mid cranial fossa &amp; meatal foramen</td>
<td>100</td>
<td>21</td>
<td>12–36</td>
<td>100</td>
<td>No</td>
<td>IV</td>
<td>15</td>
<td>3.30 (0.82–12.90)</td>
<td>—</td>
<td></td>
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</tr>
</tbody>
</table>

Completion rate: percentage of subjects followed to study completion; severity: percentage of patients with complete palsy; duration: maximum duration of palsy before starting steroids.

CI = 95% confidence interval; NH = natural history, percentage of nonsurgical patients attaining a good outcome; RR = relative rate of surgically treated patients attaining outcome compared to non–surgically treated patients; NS = not stated.
Class II. Evidence provided by a prospective matched group cohort study in a representative population with masked outcome assessment that meets at least one of the four criteria above or an RCT that lacks one criterion a through d.

Class III. All other controlled trials (including well-defined natural history controls or patients serving as their own controls) in a representative population where outcome assessment is independent of patient treatment.

Class IV. Evidence from studies not assessing outcomes independent of treatment, uncontrolled studies, case series, case reports, or expert opinion.

Appendix 2
Definitions for strength of recommendations

Level A. Established as effective, ineffective, or harmful for the given condition in the specified population. Usually, an “A” recommendation requires that the pooled result from two or more distinct class I studies demonstrates a consistent, significant, and important effect.

Level B. Probably effective, ineffective, or harmful for the given condition in the specified population. Usually, a “B” recommendation requires that a single class I study demonstrates a significant and important effect or the pooled result from two or more distinct class II studies demonstrates a consistent, significant, and important effect.

Level C. Possibly effective, ineffective, or harmful for the given condition in the specified population. Usually, a “C” recommendation requires that a single class II study demonstrates a significant and important effect or the pooled result of two or more distinct class III studies demonstrates a consistent, significant, and important effect.

Level U. Data are inadequate or conflicting. Given current knowledge, treatment is unproven and an evidence-based recommendation cannot be made.

Appendix 3
Quality Standards Subcommittee Members: Gary Franklin, MD, MPH (Co-Chair); Catherine Zahn, MD (Co-Chair); Milton Alter, MD, PhD; Stephen Ashwal, MD; John Calverley, MD; Richard M. Dubinsky, MD, Jacqueline French, MD, Michael Glantz, MD; Gary Gronseth, MD; Deborah Hirtz, MD; Robert G. Miller, MD; James Stevens, MD; and William Weiner, MD.

References