Bell’s palsy: a summary of current evidence and referral algorithm

Graeme E Glassa,⁎ and Kallirroi Tzafettab

⁎Nuffield Department of Orthopaedics, Rheumatology and Musculoskeletal Sciences, University of Oxford, Oxford, UK and bSt. Andrews Centre for Plastic Surgery Broomfield Hospital, Chelmsford, UK.

⁎Correspondence to Graeme E. Glass, Nuffield Department of Orthopaedics, Rheumatology and Musculoskeletal Sciences, University of Oxford, Kennedy Institute of Rheumatology, Roosevelt Drive, Oxford OX 3 7FY, UK; E-mail: graeme.glass@ndorms.ox.ac.uk

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Abstract

Spontaneous idiopathic facial nerve (Bell’s) palsy leaves residual hemifacial weakness in 29% which is severe and disfiguring in over half of these cases. Acute medical management remains the best way to improve outcomes. Reconstructive surgery can improve long term disfigurement. However, acute and surgical options are time-dependent. As family practitioners see, on average, one case every 2 years, a summary of this condition based on common clinical questions may improve acute management and guide referral for those who need specialist input. We formulated a series of clinical questions likely to be of use to family practitioners on encountering this condition and sought evidence from the literature to answer them. The lifetime risk is 1 in 60, and is more common in pregnancy and diabetes mellitus. Patients often present with facial pain or paraesthesia, altered taste and intolerance to loud noise in addition to facial droop. It is probably caused by ischaemic compression of the facial nerve within the meatal segment of the facial canal probably as a result of viral inflammation. When given early, high dose corticosteroids can improve outcomes. Neither antiviral therapy nor other adjuvant therapies are supported by evidence. As the facial muscles remain viable re-innervation targets for up to 2 years, late referrals require more complex reconstructions. Early recognition, steroid therapy and early referral for facial reanimation (when the diagnosis is secure) are important features of good management when encountering these complex cases.

Key words: Bell’s palsy, corticosteroids, cross facial nerve graft, facial nerve palsy, facial nerve paralysis, Herpes simplex, idiopathic, meatal segment.

Introduction

Spontaneous, unilateral facial nerve paralysis in the absence of an identifiable cause, described by Charles Bell in 1831 (hence sometimes known as Bell’s palsy or BP) remains the most common cause of unilateral facial nerve paralysis (1). Clinical evidence of improvement occurs spontaneously within 3 weeks in 85% of cases and 71% eventually recover normal hemifacial function (2) while 13% exhibit mild residual hemifacial weakness while 16% exhibit moderate to severe deficits including disfiguring facial asymmetry, incompetence of the oral commissure (drooling), brow ptosis, incomplete eyelid closure, hemifacial spasms, mass movement contractions and gustatory lacrimation (2,3). These distressing sequelae have prompted the search for reliable therapeutic options to manage the acute presentation. Established facial asymmetry may be managed surgically, with the best results achieved using the patients’ own facial musculature as the re-innervation target (4,5). However, this option is critically time-dependent, hence it is imperative that family practitioners are familiar with both the acute management and the reconstructive options available, as an expeditious referral process is the key to improving outcomes among the 29% that do not recover normal function. As family practitioners are estimated to encounter, on average, one acute case every 2 years (6) a summary of best practice, from initial presentation to specialist referral is needed. This review seeks to summarize our
understanding of BP and facilitate the expeditious referral of established hemifacial paralysis.

Methods

Scope
The aspects of our current understanding of idiopathic facial nerve paralysis reviewed included epidemiology, clinical presentation, aetiology and pathophysiology, diagnosis (including differential), prognosis and management of both acute and established facial nerve paralysis. These subheadings were formulated as clinical questions to impart direct clinical relevance to the review. Components were considered with respect to the preclinical and clinical evidence available. Publications linking preclinical studies with clinical trials were also sought.

Search strategy
A database search for full text articles published in English was performed. Databases used included Pubmed, MEDLINE (1956-October 2013), EMBASE (1990-October 2013), the Cochrane database of systematic reviews and the Cochrane controlled trials register. The search was performed using medical subject heading (MeSH) term ‘Bell palsy’ and associated entry terms ‘idiopathic (OR inflammatory) AND facial neuropathy OR facial paralysis’. References listed in the accepted articles were checked for further articles of relevance. All study types were included. Additionally, the online trial registers ClinicalTrials.gov and the national research register (NRR) were scrutinized for completed, discontinued and ongoing trials relating to facial nerve palsy (FNP). The search strategy was performed in accordance with the Cochrane Highly Sensitive Search Strategy guideline in the Cochrane Handbook for Systematic reviews of Interventions. The review of acute management is reported in line with the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) statement. The reviews of observational components are reported in line with the strengthening the reporting of observational studies in epidemiology (STROBE) statement.

Inclusion criteria
Studies were included if they reported on the any aspect of the epidemiology, clinical presentation, aetiology, pathophysiology, diagnosis, prognosis and management of idiopathic FNP and were of acceptable quality as defined by the statements above. The exception to this was that all aetiological papers and alternative management papers were included in order that the lack of sound data was exposed. Both clinical and pre-clinical studies of relevance were sought.

Exclusion criteria
Studies were excluded if they reported on upper motor neurone FNP or lower motor neurone FNP with an alternative attributable cause (i.e. not, by definition, Bell’s palsy). Articles not available as full-text articles in English were excluded. Conference abstracts were not considered.

Outcome measures
Quantitative data were sought for epidemiology, clinical presentation, management and prognosis, with qualitative data sought for aetiology, pathophysiology, diagnosis and management.

Study selection and analysis
The first author performed an abstract review of 1236 articles identified, after exclusion of duplicates. Full text review was undertaken for 132 studies that met the inclusion criteria and additional studies that possibly met the inclusion criteria. Discrepancies were resolved by consensus with the senior author (KT). Data were summarized under the clinical question headings as described above. Meta-analysis for the use of corticosteroid +/- antiviral in the acute management of Bell’s palsy was reported here but not repeated by us. In the final section we have used the evidence available and lessons from our own experience to propose a referral algorithm.

How common is it?
BP may be defined as an acute, peripheral unilateral (or rarely bilateral) neuropathy of the facial nerve thus representing 49–75% of all facial nerve palsies (7–9). The estimated annual incidence is between 13 and 34 per 100 000 (10,11) implying a lifetime risk of 1 in 60 (1) with the left and right side equally affected (7,12,13). Around 0.4% of acute, peripheral FNP is bilateral in presentation (14) although a definitive (non Bell’s) diagnosis is made in most of these cases. BP is three times more common in pregnant women (15) and also more common in those with diabetes mellitus (16–18). BP is less common in children (19). Each family practitioner is estimated to encounter one new case every 2 years (6).

Is it just facial droop?
Ipsilateral facial weakness as a result of paralysis of the muscles of facial expression is the most commonly recognized feature of BP. However, it may be accompanied by a number of additional features including altered sensation in the distribution of the sensory glossopharyngeal and trigeminal nerves (face, ear, pharynx and posterior tongue) in up to 80%, facial and retroocular pain in 60%, altered taste in 57%, intolerance to loud noise (hyperacusis) in 30%, reduced sensation in distribution of the C2 dermatome (neck and occiput) in 20%, vagal weakness in 20% and trigeminal motor weakness (chewing) in 3% (7). Dry eyes (xerophthalmia), dry mouth (xerostomia) and lacrimation
induced by a salivary stimulus (‘crocodile tears’) are features in up to 17% (7). A diagram of the facial nerve and associated anatomy helps to make sense of these complex patterns of presentation (Fig. 1).

**What causes it?**

Fisch and Esslen observed that compression of the facial nerve occurred at the level of the geniculate ganglion within the meatal segment; the narrowest part of the entire course of the facial nerve (20,21). In a report of two cases Sheehy et al. (22) observed that the nerve was markedly swollen in the labyrinthine segment of the facial canal. In 1972, McCormick (23) attributed BP to reactivation of herpes simplex virus (HSV) within the geniculate ganglion. However, his theory was circumstantial and subsequent attempts to confirm a causal relationship have proved challenging (24). Reasons for this include the observation that BP usually occurs on only one occasion, while reactivated HSV-1 in the sacral and trigeminal ganglia cause recurrent episodes of genital and oral herpes, respectively. Additionally, serological evidence for HSV-1 infection is difficult to obtain and does not provide irrefutable proof of a causal relationship. In a murine model of auricular HSV-1 infection, Murakami et al. (25) established that while all mice mounted an antibody response, only mice that exhibited FNP expressed HSV-1 DNA within the nerve and in the brainstem. Clinical evidence of HSV-1 infection within the geniculate ganglion remains elusive, as confirmation of reactivated HSV requires a tissue specimen obtained during an acute episode. Furuta et al. (26) and Takasu et al. (27) observed the presence of HSV-1 in 11 of 15 and 15 of 17 cadaveric geniculate ganglia, respectively and also confirmed latency, by the detection of latency-specific transcription proteins. Murakami et al.

![Figure 1](image-url)

**Figure 1.** The diagrammatic course and associated anatomy of the facial nerve as it traverses the facial canal within the petrous temporal bone, emerging through the stylomastoid foramen to arborise into its terminal branches.
evaluated endoneurial fluid collected from 14 patients with BP treated by surgical decompression. They identified HSV DNA in 11 of 14 cases. These studies present circumstantial evidence for a causal relationship yet are the key data on which the HSV argument is based. A summary of the aetiological theories, and the evidence for them, is presented in Table 1.

What are the most likely differential diagnoses?
Alternative diagnoses depend on the identification of an attributed cause. The surgical sieve may help here. Congenital facial nerve palsy (such as Moebius syndrome) are out with the scope of this review and, in any case, are not likely to be confused with BP. Assuming that the cause is acquired, one must first exclude upper motor neurone lesions by history and clinical examination (3). Lower motor neurone lesions are commonly idiopathic, traumatic, neoplastic or inflammatory in nature. Again, the history and examination yields vital clues, particularly in excluding traumatic or neoplastic causes. An inflammatory facial nerve paralysis caused by reactivation of varicella zoster virus (VZV) within the geniculate ganglion often mimics BP (45). This is known as Ramsay Hunt syndrome when associated with a vesicular eruption within the auditory canal, and ‘zoster sine herpete’ when the vesicular eruption is absent (46,47). Zoster sine herpete may account for 8–34% of all BP diagnoses (43,46) and possibly more in children (48) but is associated with a more severe symptomatic profile and poorer prognosis (45). The many infectious agents implicated in BP (and the wide variations in observational data in support of each) suggest a final common pathway of compressive and ischaemic nerve injury as a result of infection by one or more of several implicated agents (49).

How do I make the diagnosis?
BP remains a clinical diagnosis. A central (upper motor neurone) lesion exists above the facial motor nucleus in the pons. Peripheral lesions may be present along the nerve represented in Figure 1. Cells of the facial motor nucleus innervating the lower face receive corticobulbar fibres from the contralateral cerebral hemisphere. Cells of the facial motor nucleus innervating the upper face receive corticobulbar fibres from both cerebral hemispheres. Hence, an upper motor neurone lesion manifests as facial motor weakness with sparing of the upper face (preserved brow lift). By contrast lower motor neurone lesions (including BP) do not spare the upper face. Evoked EMG/electromyography studies may be used to define the severity of nerve palsy and quantify improvement. Reduced amplitude suggests axonal degeneration while prolonged latency suggests demyelination. The test must be performed sufficiently long after onset (3 or more days) to permit time for axonal degeneration (3) but becomes unreliable after about 2 weeks. The role of Lumbar puncture and MRI is unclear.

My patient has BP and wants to know when it will resolve. What do I tell her?
An objective, standardized, quantifiable assessment of facial nerve function serves as the basis for evaluating the clinical course. The most commonly used system is the House–Brackmann score (HBS, see supplemental table). Overall, 71% of patients with a diagnosis of BP will recover completely (HBS I), including 61% with a complete palsy (HBS VI) and 94% with a partial palsy (HBS II–V). Eighty-four percent will have a complete or near complete recovery (HBS I–II). Hence, 29% will have a residual deficit that will be moderate to severe in 16% (2,3). Acute treatment options target this group. Poorer recovery is anticipated with increasing age [50], although not with diabetes mellitus (51). Children with partial facial nerve palsies usually recover (52) but among those with complete palsies, the outcome is as poor as for adults (12).

I have made a diagnosis of acute BP. What do I do now?
Steroids form the mainstay of acute medical management of BP (53). The regimen proposed by Adour et al. (54), to be commenced within 72 hours of symptom onset, is 1mg/kg per day in two divided doses tapered from Day 5 to 10. Variations in steroid regimen may be confounding variable in systematic reviews of steroid efficacy (55). The addition of antiviral therapy is based on presumed viral aetiology. The antiviral most commonly used in therapeutic trials is acyclovir, however, with a bioavailability of between 15% and 30% (56), alternatives with improved pharmacokinetic profiles such as vanacyclovir and famcyclovir have been offered as alternatives. A summary of the major literature for the use of steroids +/- antivirals is shown in Table 2. The systematic reviews/metanalyses and larger, robustly designed randomized controlled trials favour the use of steroids alone. The evidence suggests that, of the 29% expected not to recover fully, one-third to one half of these will attain a full recovery with steroid therapy (58,66,69). Overall, up to 1 in 10 treated will avoid the need for surgery as a result of treatment.

Studies supporting a cumulative benefit from the addition of antiviral therapies include two of the smaller studies, both of which are methodologically flawed (71,72). The other, Hato et al. (68) was conducted in tertiary referral centre. All patients commenced therapy within 72 hours of symptom onset and the investigators were not blinded to the therapeutic regime, necessitating a cautious approach to their conclusions.

The efficacy of corticosteroids in the management of children with BP was reviewed by Salman et al. (73) They found
<table>
<thead>
<tr>
<th>Theory</th>
<th>Key references</th>
<th>Summary of evidence</th>
</tr>
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<tbody>
<tr>
<td>HSV (HHV-1)</td>
<td>McCormick  (23)</td>
<td>Hypothesis only. No experimental evidence presented.</td>
</tr>
<tr>
<td></td>
<td>Adour et al.  (28)</td>
<td>Serological analysis of 41 BP vs. 41 matched controls.</td>
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<td></td>
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<td>41 of 41 (100%) versus 35 of 41 (85%) exhibited antibodies to HSV (P &lt; .05).</td>
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<td></td>
<td>Murakami et al.  (29)</td>
<td>Serological analysis and RT-PCR from endoneurial fluid of affected FN. versus controls.</td>
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<td></td>
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<td>HSV-1 AB titre positive in 12 of 13 (92%−BP), 4 of 9 (44%−Ramsay Hunt), 5 of 9 (56%−other controls);</td>
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<tr>
<td></td>
<td></td>
<td>HSV-1 DNA identified in FN of 11 of 14 (79%) with BP. VSV and EBV detected in 0 of 14 (0%).</td>
</tr>
<tr>
<td></td>
<td>Linder et al. (30)</td>
<td>RT-PCR for HSV-1, HSV-2 and VZV from orbicularis muscle biopsy of BP (n = 13) versus (control) cadaveric geniculate ganglion (n = 14).</td>
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<td></td>
<td></td>
<td>HSV-1 in 0 of 13 (0%) versus 12 of 14 (86%); VZV in 0% versus 43%.</td>
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<td>HHV-6 found in tear fluid of BP and control patients (review of evidence for VZV).</td>
</tr>
<tr>
<td>VZV (HHV-2)</td>
<td>Morgan and Nathwani  (31)</td>
<td>1726 patients with BP and 262 with Ramsay Hunt identified. Serological evidence for VZV in 9.3% and 84%, respectively.</td>
</tr>
<tr>
<td>EBV (HHV-4) Lyme borreliosis</td>
<td>Hyden et al. (32)</td>
<td>147 consecutive patients with BP. Paired CSF and serum samples (&lt;1 week after onset) for many viruses and Borrelia burgdorferi (Bb).</td>
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<tr>
<td></td>
<td></td>
<td>No evidence of any infection in 98 of 147 (67%). Evidence of viral infection in 33 of 147 (22%). EBV implicated in 19 (13%). Bb implicated in 16 of 147 (11%).</td>
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<td></td>
<td>Grose and Feorino (33)</td>
<td>Serological evidence of EBV AB in three cases of BP. One patient documented to be EBV negative prior to episode of BP. Proposed EBV BP as a variant of polynuineuritis with common aetiology.</td>
</tr>
<tr>
<td>CMV (HHV-5)</td>
<td>Mair and Traavik (34)</td>
<td>88 consecutive patients with BP. Serological analysis for AB to CMV. CMV AB detected in 64 of 88 (73%). Absence of adequate controls.</td>
</tr>
<tr>
<td>HHV-6</td>
<td>Pitkaranta et al. (35)</td>
<td>RT-PCR for HSV-1, HSV-2, HHV-6 and VZV from tear fluid of patients with BP and healthy controls. HHV-6 in 7 of 20 (35%) versus 1 of 20 (5%, P = 0.04).</td>
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<tr>
<td></td>
<td></td>
<td>VZV in 10% versus 0%. HSV and HHV-2 found in no samples.</td>
</tr>
<tr>
<td>Vascular ischaemia</td>
<td>Hilger (36)</td>
<td>Proposed BP as an ischaemic neuritis caused by arteriolar spasm with secondary oedema.</td>
</tr>
<tr>
<td></td>
<td>Kettel (37)</td>
<td>Proposed BP as a vicious circle of arteriolar spasm, transudation and oedema, nerve ischaemia, compression.</td>
</tr>
<tr>
<td>Autoimmune</td>
<td>McGovern et al. (38)</td>
<td>Dogs hypersensitised by IV injection of horse serum demonstrated FN paralysis after injection with Ringer’s lactate or horse serum into stylomastoid foramen. Complement-mediated damage to FN proposed.</td>
</tr>
<tr>
<td></td>
<td>Abramsky et al. (39)</td>
<td>Lymphocytes from patients with BP stimulated in presence of neuritogenic basic protein from myelin sheath.</td>
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<td></td>
<td>Hughes et al. (40)</td>
<td>Lymphocyte activation in 5 of 14 patients with BP versus 1 of 21 controls.</td>
</tr>
</tbody>
</table>
that while there was some evidence in support of their use, the evidence was lacking from the trials that focussed exclusively on children.

**My patient wants to explore alternative therapies. What is the evidence?**

The evidence does not support the use of surgical decompression as studies are hampered by small numbers, methodological variation and selection bias (74). Physical and occupational therapies such as electrical nerve stimulation (electrotherapy), thermal therapies such as heat/ice and exercise and massage therapy have long been used as adjunctive treatments for BP but no evidence of good methodological quality been found to support their use (75–77). Similarly, two systematic reviews of acupuncture as an adjunctive treatment found that the evidence for this therapy was similarly lacking (78,79).

**So the acute management didn’t work. What next?**

Prolonged hemifacial paralysis results in irreversible atrophy of the facial musculature. Management of chronic facial nerve paralysis includes: (i) temporising measures; (ii) static symmetrization; (iii) dynamic reanimation. Temporising measures manage distressing and socially embarrassing sequelae including hemifacial spasms, mass movement contractions (synkinesis) and gustatory lacrimation and mitigate eye problems caused by incomplete closure. Static symmetrization is used in patients for whom more complex, dynamic reanimation is not suitable (80).

In these cases, the duration of hemifacial paralysis is irrelevant. Dynamic reanimation is achieved by cross facial nerve grafting (CFNG). This involves co-apting peripheral motor branches of the contralateral facial nerve to the corresponding branches on the affected side via a donor conduit (usually sural nerve graft) tunnelled through the subcutaneous tissues of the face to the contralateral side. Success of this approach assumes the presence of viable muscle targets to re-innervate. The muscle targets remain viable for about 12–18 months (4). When denervation time exceeds 6–12 months but when the facial musculature is still salvageable, the ‘babysitter’ technique (co-apting the ipsilateral hypoglossal nerve to the facial nerve) buys time, preventing further atrophy while the cross facial nerve grafts neurotize (81). This technique may work up to 2 years following paralysis.

When irreversible atrophy of the facial musculature has occurred, cross facial nerve grafts lack muscle targets with which to innervate hence new target muscle must be imported, as a free tissue transfer. The gracilis (82) and pectoralis minor (83,84) and latissimus dorsi, are commonly used (85). The muscle is divided to achieve three motor objectives, including an ocular sphincter, a smile and depression of the lower lip, each of which requires a different vector of pull.

While satisfactory results are achieved they rarely match the result obtained by direct neurotization of the affected side and do so at the expense of a greater surgical burden. Hence, direct neurotization of the paralyzed facial musculature remains the gold standard in reconstructive facial symmetrization, but this option is critically dependent on prompt referral. With this aim in mind, a referral algorithm is proposed which guides referral

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**Table 1. Continued**

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<thead>
<tr>
<th>Theory</th>
<th>Key references</th>
<th>Summary of evidence</th>
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<tbody>
<tr>
<td>Adverse event following immunization (AEFI)</td>
<td>Rath <em>et al.</em> (41)</td>
<td>Review of data implicating vaccines (and adjuvants) in development of BP. Intra-nasal/enteric vaccines may precipitate BP in some cases. Trial data lacking standardised definition of BP.</td>
</tr>
<tr>
<td></td>
<td>Mutsch <em>et al.</em> (42)</td>
<td>Retrospective case-controlled study evaluating link between intranasal influenza vaccine and BP. 13 excess cases per 100000. Circumstantial evidence for aetiology in some cases.</td>
</tr>
<tr>
<td>Cold weather</td>
<td>Zeallear <em>et al.</em> (43)</td>
<td>Canine/feline model of continuous exposure of tympanic membrane to cold air. Reduction in compound action potential of 33–96% (n = 8). Histological findings of demyelination, axonal swelling and degeneration.</td>
</tr>
<tr>
<td></td>
<td>Danielidies <em>et al.</em> (44)</td>
<td>171 cases of BP analyzed with reference to eight meteorological parameters. NO correlation between BP and any parameter.</td>
</tr>
</tbody>
</table>

HSV, herpes simplex virus; HHV, human herpes virus; VZV, varicella zoster virus; EBV, Epstein–Barr virus; CMV, cytomegalovirus; BP, Bell’s palsy; FN, facial nerve; AB, antibody, RT-PCR, reverse transcriptase polymerase chain reaction.
Table 2. Summary of the major studies using steroids +/- antiviral therapy in the acute management of Bell's palsy

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>Papers included</th>
<th>Patient number</th>
<th>Features of study</th>
<th>Conclusions</th>
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<tr>
<td>Meta-analysis</td>
<td></td>
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<tr>
<td>Browning (57)</td>
<td>2010</td>
<td>16</td>
<td>unclear</td>
<td>Analysis of all combinations of corticosteroid, antivirals and placebo</td>
<td>Corticosteroids effective in acute management of BP. No significant benefit with use of antivirals alone or in combination with steroids compared with steroids alone.</td>
</tr>
<tr>
<td>Systematic reviews/meta-analyses</td>
<td></td>
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<tr>
<td>Salinas et al. (58)</td>
<td>2010</td>
<td>7</td>
<td>1507</td>
<td>Corticosteroids for BP (Cochrane review)</td>
<td>Incomplete recovery in 175/754 (23%) with corticosteroids versus 245/753 (33%) without. Corticosteroids effective in acute management of BP. No evidence that antivirals perform better than placebo. Antivirals found to perform significantly worse than corticosteroids.</td>
</tr>
<tr>
<td>Lockhart et al. (59)</td>
<td>2009</td>
<td>7</td>
<td>1987</td>
<td>Antiviral therapy for BP (Cochrane review)</td>
<td>No evidence of additional benefit from antiviral therapy</td>
</tr>
<tr>
<td>Goudakos and Markou (60)</td>
<td>2009</td>
<td>5</td>
<td>738</td>
<td>Corticosteroids + antivirals versus corticosteroids alone</td>
<td>No evidence of additional benefit from antiviral therapy</td>
</tr>
<tr>
<td>Quant et al. (61)</td>
<td>2009</td>
<td>6</td>
<td>1145</td>
<td>Corticosteroids + antivirals vs. corticosteroids alone</td>
<td>No evidence of additional benefit from antiviral therapy</td>
</tr>
<tr>
<td>de Almeida et al. (38)</td>
<td>2009</td>
<td>18</td>
<td>2786</td>
<td>Corticosteroids +/- antivirals vs. control; Antivirals +/- corticosteroids versus control</td>
<td>Corticosteroids effective in acute management of BP. Combination improved likelihood of avoiding unsatisfactory outcome (P = 0.05; &quot;borderline significance&quot;) therefore antivirals MAY provide additional benefit.</td>
</tr>
<tr>
<td>Allen and Dunn (62)</td>
<td>2004</td>
<td>3</td>
<td>246</td>
<td>Antiviral therapy for BP (Cochrane review)</td>
<td>Quality of evidence poor. Further, well designed studies needed</td>
</tr>
<tr>
<td>Grogen and Gronseth (63)</td>
<td>2001</td>
<td>12</td>
<td>1390</td>
<td>Efficacy of corticosteroids (n = 1160) +/- acyclovir (n = 230)</td>
<td>Steroids probably effective; acyclovir possibly effective. Insufficient evidence to recommend routine use. Patients with complete palsy treated with steroids were 17% more likely to have complete recovery than observation or placebo.</td>
</tr>
<tr>
<td>Ramsey et al. (64)</td>
<td>2000</td>
<td>2</td>
<td>206</td>
<td>Corticosteroids for complete facial nerve palsy</td>
<td>Quality of evidence justifying use of corticosteroids poor</td>
</tr>
<tr>
<td>Williamson and Whelan (65)</td>
<td>1996</td>
<td>4</td>
<td>392</td>
<td>Corticosteroids for BP</td>
<td></td>
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<tr>
<td>Randomised controlled trials</td>
<td></td>
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<tr>
<td>Engstrom et al. (66)</td>
<td>2008</td>
<td>839</td>
<td></td>
<td>Prednisolone + Valacyclovir vs. Prednisolone + Placebo versus Placebo + Valacyclovir versus Double placebo.</td>
<td>6-month complete recovery (HBS I) in 72%, 71%, 58%, 62%, respectively. Prednisolone effective in acute management of BP. No additional benefit from valacyclovir. Satisfactory recovery (HBS I-II) 93.1% (P + A) versus 85.1% (P) (P = NS). No additional benefit from acyclovir.</td>
</tr>
<tr>
<td>Yeo et al. (67)</td>
<td>2008</td>
<td>91</td>
<td></td>
<td>Prednisolone + Acyclovir versus Prednisolone alone</td>
<td></td>
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<tr>
<td>Study</td>
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<tr>
<td>Hato et al. (68)</td>
<td>2007</td>
<td>221</td>
<td></td>
<td>(Single blinded) Prednisolone + Valacyclovir versus Prednisolone + Placebo</td>
<td>6-month complete recovery (HBS I) in 96.5% versus 89.7%. Supports addition of valacyclovir in acute management of BP (assessors not blinded).</td>
</tr>
<tr>
<td>Sullivan et al. (69)</td>
<td>2007</td>
<td>496</td>
<td></td>
<td>Prednisolone + Acyclovir versus Prednisolone + Placebo versus Placebo + Acyclovir versus Double placebo.</td>
<td>9-month complete recovery (HBS I) in 94.4% with prednisolone versus 81.6% without; 85.4% with acyclovir versus 90.8% without. Early prednisolone is effective treatment in BP. No additional benefit from acyclovir.</td>
</tr>
<tr>
<td>De Diego et al. (70)</td>
<td>1998</td>
<td>101</td>
<td></td>
<td>Prednisone only (n = 47) versus Acyclovir only (n = 54)</td>
<td>3 month-satisfactory recovery (HBS I-II) 93.6% (P) versus 77.7% (A) (P &lt; 0.002). Prednisone more effective than acyclovir in acute management of BP.</td>
</tr>
<tr>
<td>Adour et al. (37)</td>
<td>1996</td>
<td>99</td>
<td></td>
<td>Prednisone + Acyclovir (n = 53) versus Prednisone + Placebo (n = 46)</td>
<td>4-month complete recovery (described as Facial Paralysis Recovery Profile (FPRP) 10, equal to HBS I) in 92% (P + A) versus 76% (P + Pl) (P = 0.02). Supports addition of acyclovir in acute management of BP.</td>
</tr>
<tr>
<td>Other trials</td>
<td></td>
<td>N/A</td>
<td>117</td>
<td>(Prospective, quasi-randomised) Prednisolone + Famcyclovir versus Prednisolone</td>
<td>29.4% (P + F) versus 11.9% (P) exhibited greater than or equal to 4 point increase in HBS. Supports addition of famcyclovir in acute management of BP.</td>
</tr>
<tr>
<td>Minnerop et al. (71)</td>
<td>2008</td>
<td>N/A</td>
<td>117</td>
<td>(Retrospective) Prednisolone + Acyclovir versus Prednisolone</td>
<td>Complete recovery (HBS-I) in 97% (P + A) versus 72% (P) (P &lt; 0.05). Supports addition of acyclovir in acute management of BP.</td>
</tr>
<tr>
<td>Tang et al. (72)</td>
<td>2009</td>
<td>N/A</td>
<td>84</td>
<td></td>
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within the window of opportunity to offer direct neurotization (Fig. 2).

What are the challenges to the delivery of a ‘gold standard’ of care in Bell’s palsy?

If the lifetime risk of BP is 1 in 60 and 16% develop moderate to severe long term sequelae, then the lifetime risk of disfiguring facial asymmetry associated with BP is around 1 in 400 (1–3). Large met-analyses support commencement of steroid therapy within 72 hours of onset of symptoms (55,57,58). The reconstructive options are also time-critical, yet our experience is that patients are often referred years after the event. We advise that patients who exhibit obvious, asymmetric facial weakness after 4 weeks following onset of symptoms in the absence of another diagnosis must be considered for expeditious referral to ones regional or super-regional facial reanimation service. Management of BP within a multi-disciplinary team setting, with direct GP access is one potential model of care that merits evaluation.

Conclusions

Bell’s palsy may be defined as an acute, peripheral, unilateral, idiopathic, non-recurrent facial palsy and probably describes a heterogeneous clinical entity accounting for variations in presentation. Causality has remained difficult to establish, in part owing to variations in how BP is defined by the investigators, but reactivation of latent HHV (HSV-1, VZV, EBV and HHV-6) within the geniculate ganglion may precipitate a final common pathway of inflammation, oedema and ischemia of the intratemporal petrous portion of the facial nerve. Twenty-nine percent exhibit long-term sequelae which include moderate to severe disfigurement. Prompt medical management with oral corticosteroids improves likelihood of complete recovery (level 1 evidence). Expeditious tertiary referral when facial asymmetry

Figure 2. Primary care management and referral algorithm for Bell’s palsy.
is observed at 4 weeks post symptom onset, in the absence of an identifiable cause represents a paradigm shift in the management of facial palsy in primary care and may improve the salvage rate of the ipsilateral facial musculature.

**Supplementary material**

Supplementary material is available at *Family Practice* online.

**Declaration**

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Conflict of interest: none.

**References**


