

The clinical problem of Bell's palsy: is treatment with steroids effective?

I G WILLIAMSON

T R WHELAN

SUMMARY

A general practitioner can expect to see a case of Bell's palsy once every two years. Though uncommon, it has aroused controversy over its definition, its aetiology, and the best treatment. Although the majority of cases of this dramatic but usually self-limiting condition are seen in primary care, most of the literature comes from hospital studies. The evidence from four randomized controlled studies shows marginal benefit for steroids with a Mantel-Haenzel odd's ratio of 1.63 (95% CI 1.01 to 2.64); but, because of doubt about the methodology in some of the studies, this result must be interpreted with caution.

Keywords: Bell's palsy; steroids; randomized control trials.

Introduction

THE sudden onset of an isolated lower motor neurone facial weakness, though uncommon (20 per 100 000 per year^{1,2}), is often a dramatic event in a person's life and may produce considerable anxiety.³ Sir Charles Bell first drew attention to the distribution and function of the facial nerve in 1821, and since that time his name has been attached to acute lower motor neurone facial paralysis of unknown aetiology.⁴ The pragmatic definition of Bell's palsy used by Pietersen is probably the most appropriate for general practice.⁵ It is an acute peripheral monosymptomatic facial palsy without detectable causes. Numerous theories exist for the aetiology of Bell's palsy, but none are sufficiently convincing to explain the clinical features. Zulch believes that Bell's palsy is a syndrome with numerous aetiologies capable of triggering the same end mechanism: facial paralysis.⁶ While the causes are unknown it is impossible to develop rational treatments.

The relative rarity of Bell's palsy means there are few published randomized controlled trials (RCTs) of its treatment. The literature from primary care on Bell's palsy is also extremely sparse.⁷⁻¹²

Aims and methods

The aims of this review are twofold: first, to provide a useful overview of the clinical problem of Bell's palsy, to include its definition, aetiology, natural history and referral considerations; and, secondly, to carry out a focused and systematic review of the efficacy of steroids in its treatment.

For the general review, a hand search of the literature on facial paralysis was performed using the Index Medicus, covering the period 1970 to the end of 1995. In addition, for the focused systematic review, both authors independently searched for any past

trials of steroid treatments in Bell's palsy. Medline and BIDS searches were performed from 1985 to the end of 1995, overlapping with the systematic review of steroid treatment published by Stankiewicz in 1987.¹³ We used the mesh keywords; facial paralysis, Bell's palsy, steroids and drug therapy. Further searches under prednisolone, adrenocorticotrophic hormone (ACTH), cortisone and steroid treatment yielded minimal extra information. We also used the science citation index and, by two subsequent steps, searched for additional references. We wrote to six manufacturers of steroids to see if there were any unpublished studies on the effects of steroids, and (in accordance with guidelines for literature reviews) consulted five ear, nose and throat (ENT) specialists active in this field.¹⁴

We focused on facial nerve recovery as the principal outcome, and evaluated whether this was complete or incomplete using Pietersens criteria (0 = complete recovery, grades I-IV indicate varying degrees of incomplete recovery).⁵ The other main outcome studied was duration to complete recovery.

The main studies selected are those included in the meta-analysis; they were chosen only if they were RCTs of steroids which either used placebo controls or had an untreated control arm. All such RCTs were included, and a list of important study characteristics is provided with the results. Non-randomized comparative studies (notably prospective studies and the two largest retrospective studies) are also critiqued to provide further evidence on the efficacy of steroids. Studies where the outcome was difficult to categorize, or which were seriously flawed in their methodology, were excluded (six in total).

Is Bell's palsy a mono- or a polyneuropathy?

Traditionally, Bell's palsy has been defined as a clinically isolated facial neuropathy. Support for this convention comes from pathology reports^{15,16} and from ENT surgeons undertaking decompression of the facial nerve in the petrous temporal bone.¹⁷ However, evidence for a subclinical cranial and even a peripheral polyneuropathy has been gained from other studies.¹⁷⁻²⁶

Aetiology

There are four basic aetiologies proposed for Bell's palsy: genetic, where hereditary factors have been shown to be important,²⁷⁻²⁹ vascular, where the arteries involved in supplying the facial nerve produce oedema and compression;³⁰⁻³¹ infective causes, notably viruses but also some bacteria;³²⁻³⁶ and an immunological cause essentially involving an auto-immune process.³⁷⁻³⁹ Negative viral studies have been reported.⁴⁰⁻⁴² Numerous theories elaborate on the mechanism of nerve damage. The cause or causes of Bell's palsy remain unproven despite many claims to the contrary in the literature.

Natural history

Bell's palsy is the commonest type of facial palsy. It often begins with pain behind the ear, which is a bad prognostic sign according to some studies,⁴³ or with impairment of taste. The paralysis usually reaches a peak at two days, but may continue to worsen over 10 days.⁴⁴ Numbness of the face is often reported. However, many authorities state that it is never present in sensory testing⁴⁵

I G Williamson, MD, MRCP, FRCS, senior lecturer, Primary Medical Care Group, School of Medicine, Southampton University; and T R Whelan, MRCP, general practitioner, Newport, Isle of Wight. Submitted: 22 November 1995; accepted: 22 July 1996.

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whereas others have described it in 48% of cases.²² Loud sounds may cause the patient discomfort. Any other neurological signs should cast doubt on the diagnosis of Bell's palsy. Table 1 shows the signs and causes of a lower motor neurone facial palsy. Some patients may complain of epiphora (watery eyes), which is usually due to weakness of the facial muscles, or of a dry eye; food may collect in the affected cheek. Bilateral Bell's palsy is uncommon (0.7–3.3%) and is reviewed by Yanagihara.⁴⁶ Bell's palsy is noted in children as young as five, and increases in incidence with age.^{47,48}

The true natural history of Bell's palsy is difficult to establish from most studies because of selection bias. In hospital-selected series, the overall complete recovery rate varies from 57–85% for untreated Bell's palsy.^{5,43,48–52} It is likely that, in general practice, recovery rates will be higher than in hospital studies.

For all patients who are followed up and who never develop a complete paralysis, full recovery is highly likely. For most patients, recovery usually commences within three weeks, with a median time to complete recovery of six weeks. Complete recovery will generally not occur after the first four months. The only absolutely bad prognostic sign is a failure to recover any kind of movement in the face within four weeks of onset of the palsy.⁴⁸

Mathews found a marked difference in full recovery with age, which was noted in 69% of those aged under 40, but only in 44% of those aged over 40.⁴⁸ Other authors have also noted that increasing age affects prognosis.^{5,53,54}

Pietersen has noted a poor outcome in diabetics and in pregnancy.⁵ Bell's palsy appears more common in the third trimester of pregnancy and in the puerperium.^{11,55,56}

Referral considerations

On presentation, 30–70% of patients have no demonstrable facial movements on the affected side (complete palsy) and require immediate referral for further investigation and eye care.^{5,9,45} Any patient who shows continuous progression of the paralysis over many days or weeks, or who has unusual neurological or other clinical features, such as a sudden deterioration in hearing, should be referred because of the possibilities of tumour or other causes. However, hospital-based studies have revealed that lower motor neurone lesions of the face due to neoplasms are uncommon and constitute only about 0.5% of all causes. Other causes of acute facial paralysis include trauma (7%), herpes zoster oticus or Ramsay Hunt syndrome (2%), acute otitis media (1%), middle ear surgery (1%), and cholesteatoma (0.6%).⁵⁷

It is undesirable to refer all cases of uncomplicated and incomplete idiopathic facial palsy. Half of patients with Bell's palsy attending hospital have been observed to exhibit a considerable

degree of psychological distress.³ For some, there may be unnecessary anxiety about brain cancer. The general practitioner has to consider the other costs of the referral, which include actual costs and side-effects from over-enthusiastic investigation.

Medical treatments of Bell's palsy

Randomized controlled trials of steroids and results of meta analysis

Burgess, in a useful statistical critique, appreciated that complete paralysis and partial paralysis carried different prognoses.⁵⁸ He stated that these groups must be considered separately in any trial of steroid versus placebo. In determining the sample size required he assumed a 60% spontaneous recovery rate. In order to show a 25% improvement from steroid therapy, he used a sample size of at least 194 patients in each of four trial sub-groups (complete, incomplete, steroid, and placebo). With smaller sample sizes, significant improvements due to treatment could be missed. None of the RCTs that were identified from those using placebo controls contain anything like this number of patients. The study characteristics are shown in Table 2. All were hospital-based RCTs with placebo or untreated control arms; there were no studies from primary care.

The first documented randomized study was that of Taverner in 1954, who treated 13 patients with oral cortisone (200 mg on the first day, decreasing over the next seven days), starting within ten days of the onset of palsy.⁶⁰ He had eleven controls. No statistically significant difference in recovery was found between the steroid and the control group. Retrospectively, he admitted the deficiencies of this trial, namely that the numbers were too small, and that the steroid dose was too low and started too late.⁵³ May claimed no difference in recovery rates between prednisolone and placebo, but his results are also invalidated by too small a sample size — only 25 patients and 26 controls.⁶¹ He does not give the age range for his study groups. Similarly, Wolf claimed no difference in recovery rates between 107 patients on prednisolone and 132 on placebo.⁵⁴ His study was not blinded and had an untreated rather than a placebo control arm. The numbers are too small to draw conclusions about the effectiveness of steroids. However, he did find a statistically significant reduction in autonomic synkinesis ('crocodile tears') in the steroid group. Austin's study also failed to show a difference in facial recovery at six months, but again the numbers were inadequate: 35 patients and 41 controls.⁶² All four studies contain insufficient information to judge the effectiveness of randomization.

Summative information for the four selected RCTs is shown in Table 3. The chi-square test for heterogeneity of the samples for

Table 1. Signs and causes associated with various sites for lower motor neurone facial palsy.

Site	Signs	Causes
Brainstem	Gaze palsy, sixth nerve palsy, nystagmus, long tract signs, taste spared	Multiple sclerosis, pontine glioma, stroke
Cerebellopontine angle	Deafness, absent corneal reflex, ipsilateral ataxia	Acoustic neuroma, meningioma, epidermoid, glomus tumour, granuloma
Cranial polyneuropathy	Bilateral seventh nerve palsy, palsy of other cranial nerves	Sarcoid, meningeal cancer, Lyme disease, Guillain-Barré syndrome, tuberculosis
Facial canal	Taste ±, hyperacusis ±; cause determined by other symptoms and signs	Bell's palsy, herpes zoster, trauma, middle ear disease, petrous temporal cancer
Peripheral nerve	Taste and hearing unaffected	Parotid tumour, trauma
Muscle	Muscular or neuromuscular disorder may simulate bilateral facial weakness; other signs will be present	Facioscapulohumeral dystrophy, myasthenia, myotonic dystrophy

Table 2. Study characteristics. All hospital-based randomized controlled trials with placebo or untreated control arms.

	Taverner (1954)	May (1976)	Wolf (1978)	Austin (1993)
Numbers of subjects	26	51	239	76
Age profile	17–65 years	Not stated	5–70 years	18–70 (mean 36.8) years
Complete or incomplete palsy	Both	Both	Both	Both
Time before starting treatment	<10 days	<2 days	<5 days	<5 days
Treatment regime	Cortisone 200 mg decreasing	Prednisolone (total 410 mg)	Prednisolone 60 mg decreasing (total 760 mg)	Prednisolone 30 mg decreasing (total 205 mg)
Duration of follow up	157 days	6 months	1 year	6 months
Placebo controlled	Yes	Yes	No	Yes
Outcome assessment blinded	Yes	Yes	No	Yes
Single blinded	–	–	–	–
Double blinded	Yes	Yes	–	Yes
Open study	–	–	–	–

Table 3. Clinical outcome: percentage of sample with complete recovery from facial paralysis. All hospital-based randomized controlled trials with placebo or untreated control arms.

	No. with complete recovery in steroid group (%)	No. with complete recovery in control group (%)	Observed difference in proportions	95% confidence intervals
Taverner (1954)	10/13 (77)	8/11 (73)	0.04	-0.31 to 0.39
May (1976)	15/25 (60)	17/26 (65)	-0.05	-0.32 to 0.21
Wolf (1978)	94/107 (88)	105/132 (80)	0.08	-0.01 to 0.18
Austin (1993)	19/35 (54)	13/41 (33)	0.2	0.01 to 0.44
Total	138/180 (77)	134/210 (64)	0.12	0.03 to 0.21

3 degrees of freedom is 2.09 (not significant). Using confidence interval analysis, odds ratios and 95% confidence intervals (CIs) were determined for each of the studies. The pooled odds ratio with 95% CI was determined by the Mantel-Haenzel method and can be considered as a weighted odds ratio. For all four studies the Mantel-Haenzel odds ratio is 1.63, with a 95% CI of 1.01 to 2.64. If Wolf's non-blinded non-placebo controlled study is excluded, then the odds ratio based on a smaller sample size is 1.46, with a 95% CI of 0.76 to 2.79.

In 1971, Taverner published the results of a further RCT of two alternative types of steroid therapy — oral prednisolone versus injected ACTH.⁵³ This was a single-blinded study. The prednisolone group received 80 mg daily for five days (equivalent to 80 units ACTH), decreasing over the next four days. The ACTH group received only 60 units per day for five days, decreasing over the next four days. Only patients with complete palsy were selected; those at risk of side-effects from steroids (for example, hypertensives, diabetics and dyspeptics) were excluded. A total of 186 patients completed the trial but 293 were not admitted, mostly because they were seen too late (after day 4) or were outside the age range. Although the design is statistically flawed and there is a question mark about the equivalence of the doses used, it claimed a highly significant advantage of prednisolone over ACTH ($P < 0.005$) in preventing some degree of denervation.

Ramos Macias conducted a randomized trial of steroids alone versus steroids plus acyclovir in 45 patients with facial palsy.⁶³ There was no difference in recovery of function in the Bell's palsy sub-group, whereas benefit was observed in the Ramsay-Hunt sub-group.

The time to recovery was not significantly different in the four RCTs giving information on this outcome.^{54,60–62} The mean time to complete recovery ranged from 51.4–63 days in the treated group and from 69–69.3 days in the untreated group. May

observed no difference between the groups in his study,⁶¹ and Wolf quoted a median time to complete recovery of 2 months for both groups.⁵⁴ In Pietersen's natural history study (untreated), the majority of patients obtained normal function of facial muscles from three weeks to two months after the onset of the palsy.⁵

Non-randomized comparative studies

Sychev used ACTH for three days, followed by oral prednisolone. In the untreated group, 12 showed severe sequelae, but none did in the treated group.⁶⁴ We were unable to obtain this Russian paper cited by Stankiewicz¹³ so cannot assess the considerable advantage claimed for steroid therapy.

Ekstrand used ACTH to treat patients with a poor prognosis, judged by sialometry and stapedial reflex.⁶⁵ He claimed a statistically significant improvement in the steroid group ($n = 30$), but the controls were few ($n = 12$) and did not receive placebo. As with most other studies the effects of age were not assessed.

Hyden treated 63 patients with prednisolone and compared the results with 74 patients with good prognosis who received no treatment.⁶⁶ He failed to show any advantage for cortisone therapy and believed that his earlier studies contained a systematic error in that they included undiagnosed *Borrelia*-induced palsies.⁶⁷

Shafshak claimed a significant benefit of prednisolone over no treatment ($\chi^2 = 7.88$, $P < 0.01$) when treatment commenced within 24 hours of the onset of the palsy.⁶⁸ However, the treatment and control groups contained about four times as many men in the samples (most incidence studies describe an equal distribution). He concludes that delay in initiating treatment beyond 24 hours is critical in determining outcome.

Adour's non-blinded RCT was curtailed at an early stage for ethical reasons because his treatment group reported significant benefit from steroid therapy in terms of reduced pain. This led to

the use of retrospective controls.⁶⁹ A large retrospective study was described by Prescott, who analysed the records of 879 Bell's palsy patients from the first 10 years of his facial paralysis clinic.⁵⁷ All were intended to receive a full course of prednisolone (80 mg decreasing), but for various reasons only 446 (51%) did so. Facial recovery did not appear to be influenced by treatment with steroids.

Other medical treatments

Kawai treated 109 patients with a modification of Stennert's regime. This consists of a steroid, adenosine triphosphate (ATP), vitamin B12, and oral pentoxifylline, and is considered to enhance the microcirculation of the facial nerve.^{70,71} The results were compared using a historical control group of 224 patients of varying age and of both sexes. Controls had received intravenous ATP and vitamin B12 ± intravenous steroid ± stellate ganglion block. A statistical advantage is claimed but the trial design was inadequate. This is also true of Kinishi's study, which used a lower-dose regime.⁷² Mezzina conducted a double-blind placebo-controlled trial to test the therapeutic efficacy of acetyl-carnitine, a physiological derivative of acetylcholine, in promoting neuronal recovery in Bell's palsy in patients aged 11 to 67 years.⁷³ Acetyl-carnitine or placebo was given in addition to 50 mg of prednisolone for 14 days. Functional improvement occurred earlier in the acetyl-carnitine group. Acupuncture is much used in China as a treatment for Bell's palsy.⁷⁴

Reviews of treatment

The best reviews of treatment of Bell's palsy to date are those of Stankiewicz¹³ and Austin.⁶² Of those reviewing wider aspects of treatment, Hughes wrote a good general review; although he did not attempt to draw statistical inferences from his own experience of 63 patients over eight years, his recommendations about eye care are helpful.⁷⁵ Mountain reviewed three years' work at the Edinburgh facial paralysis clinic and emphasized the importance of rehabilitation and continuing emotional support to alleviate the psychosocial problems reported by half their patients.³ Steroids were not routinely given 'as there is little scientific evidence of their benefit'. Repeated botulinum toxin injections were given to 25 out of 210 cases, for synkinesis or associated movements. Tarsorrhaphy, brow-lifts and eyelid gold-weight implantations have been used for chronic facial rehabilitation problems. Hypoglossal-facial nerve anastomosis was their preferred technique for re-innervating the face when the proximal nerve showed no sign of recovery.

Conclusion

None of the few RCTs conducted so far have had the power independently to resolve whether treatment of Bell's palsy with steroids is effective. From these studies and others, including natural history studies, there is as yet no conclusive evidence that treatment with steroids is effective when looking at the outcome of facial nerve recovery. The uncommon nature of the condition, the high spontaneous recovery rate, selection bias, and disagreement about the cause or causes of Bell's palsy make the interpretation of most studies difficult or impossible, although the view that steroids are beneficial appears well entrenched in the literature.

A patient with a partial Bell's palsy does not require steroid treatment. Incomplete palsies should probably be reviewed again in the first fortnight to exclude progression. The patient should be asked to return promptly if a complete palsy develops, and we would recommend referral to exclude other diagnoses wherever

this is in doubt. The combined evidence from four published RCTs, which can be considered as homogeneous, shows a significant benefit from steroid treatment, with a Mantel-Haenszel odds ratio of 1.63 and a 95% CI of 1.01 to 2.64. However, there were some methodological problems with the studies used in this meta-analysis (which included one non-blinded non-placebo-controlled study); the above conclusion should therefore be interpreted with caution.

Increasing age carries a poorer prognosis for spontaneous recovery and it is notable that the elderly have largely been excluded from past trials. If a general practitioner is persuaded by the evidence to prescribe steroids to a patient with a complete weakness, then a regime of prednisolone 1mg/kg/day, to a maximum of 80 mg for 10 days, would be appropriate. Dangerous side-effects from a short course of steroids have been rare. If treatment is considered beneficial it must be instituted as soon as possible to achieve maximum effect and preferably within 24 hours. The definitive trial of steroid treatment, however, remains to be done and to minimize selection bias it should be done in primary care.

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Address for correspondence

IG Williamson, Primary Medical Care Group, School of Medicine, Southampton University, Southampton SO16 5ST.

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