# The possible contributing factors for the success of steroid therapy in Bell's Palsy: a clinical and electrophysiological study

Tarek S. Shafshak, M.D., Aymen Y. Essa, M.D.*, Fathy A. Bakey, M.D.†

## Abstract

Incomplete recovery from Bell's palsy was observed in some patients even after the intake of corticosteroids. This prospective study was performed on 160 patients with unilaterial nonrecurrent Bell's palsy in order to investigate the role of prednisolone on the prognosis of Bell's palsy. Ninety-three patients were given prednisolone tablets (1 mg/kg body wt/day up to 70 mg) for six successive days, then the dose was reduced gradually over the next four days. The remaining 67 patients were not given prednisolone (control group). Facial nerve recovery was assessed clinically and electrophysiologically for up to one year. The results of this study suggested that the most probable contributing factor for the success of prednisolone in improving the prognosis of Bell's palsy was its early intake (within the first 24 hours following onset).

**Key words: Facial paralysis; Adrenal cortex hormones; Prednisolone**

## Introduction

Mononeuritis of the facial nerve is quite a common neurological problem in Alexandria as well as in many parts of the Middle East (El-Ebiary, 1971). The underlying causes are numerous, but clinical experience indicates that the majority of unilateral acute facial palsies are of the Bell's (idiopathic) type (El-Ebiary, 1971; Moore, 1990). The currently accepted theory for Bell's palsy suggests that oedema and entrapment of the facial nerve, most probably secondary to viral infection, might be the cause of facial paralysis (Bance and Rutka, 1990; Petruzzelli and Hirsch, 1991). In the management of patients with facial paralysis. Bell's palsy is still considered as the diagnosis of exclusion (Petruzzelli and Hirsch, 1991).

Although the majority of nondiabetic patients with Bell's palsy return to normal facial function, some patients are left with residual facial weakness, synkinesis, and/or contracture (El-Ebiary, 1971; Adour *et al.*, 1974). The percentage of incomplete recovery among nondiabetic Bell's palsy patients ranges from 16 to 30 per cent (Adour *et al.*, 1974; Peitersen, 1982; Moore, 1990). The latter group of patients usually suffers from permanent disfigurement and impaired facial function. This could lead to psychological problems especially in women El-Ebiary, 1971).

Steroid therapy and surgical decompression of the facial nerve have been tried in order to improve the prognosis of Bell's palsy and to decrease the chance of developing complications. However, data suggest that surgery does not improve the outcome of Bell's palsy (Huizing *et al.*, 1981; May *et al.*, 1985). The treatment of Bell's palsy with systemic corticosteroids also remains controversial (Burgess *et al.*, 1984; May *et al.*, 1985; Moore, 1990). Furthermore, Moore (1990) and Burgess *et al.* (1984) suggested that a prospective study showing a statistically significant benefit from the use of corticosteroids is still lacking. Therefore, this prospective study is aimed at investigating the effect of prednisolone on the prognosis of Bell's palsy.

## Methods

### Patients

This study was carried out on 160 patients with acute nonrecurrent unilateral Bell's palsy of no more than six days duration. Patients were selected, by a doctor who was blind to the patients' grouping, to have complete or nearly complete facial paralysis of grade V and VI according to the facial nerve grading system of House and Brackmann (1985). Patients were selected after careful clinical examination to exclude any other cause of facial palsy. Those with severe hypertension, glaucoma, peptic ulcer, and manifest cardiac disease were not included. Pregnant and diabetic patients were also excluded from this study. The diagnosis of diabetes mellitus was made according to World Health Organization criteria (WHO Report, 1985).

## Procedures

### First assessment of patient

On the first day the patient was seen, the extent (according to the facial nerve grading system of House and Brackmann, 1985) and the duration of the facial muscle

paralysis were both recorded. Data concerning the patient's age, sex, and history of heartburn or any associated medical problem were recorded. Also, the patient's blood pressure and body weight were measured.

### Corticosteroid therapy

Ninety-three patients were given prednisolone tablets (steroid group). Patients were instructed not to take any other drug. The prednisolone was given on a full stomach immediately after the diagnosis had been made. The daily dose was calculated as 1 mg/kg of body wt; as suggested by Moore (1990) and Petruzzelli and Hirsch 1991), and because prednisolone has the same potency as prednisone (Castles, 1985). It was given in divided doses after meals for six successive days. Then, the dose was reduced gradually over the next four days. In order to avoid serious drug side effects, a maximum daily dose of 70 mg was not exceeded. It should be noted that patients who sought medical advice after the sixth day of the onset of Bell's palsy did not participate in this study because it has been observed by Adour et al. (1978) and Brown (1982) that corticosteroids are only useful if started within the first week following onset.

The remaining 67 patients were not given prednisolone or any other medication, apart from occasional paracetamol tablets for pain. These patients were considered to be a control group. Patients' grouping was carried out by only one doctor in order to have nearly equal distribution of patients in both the control and steroid groups according to the extent of the paralysis, sex and age of the patient. The control group included mainly those patients who had refused to take prednisolone, because they were afraid of developing complications, and those with a relative contraindication to corticosteroids (e.g. heartburn, or moderate hypertension) because according to the ethical standards of the area where this research was conducted it was considered unethical to withhold corticosteroids from some patients with no contraindication to corticosteroids in order to establish a randomized treatment schedule.

### Electrophysiological examinations

(i) Facial nerve excitability test: percutaneous facial nerve stimulation (PFNS) for both the involved and the uninvolved sides, as the nerve emerges from the stylomastoid foramen (behind the neck of the mandible), was performed 10 days after the onset of the disease using 0.5 ms square pulses (of up to 20 mA) in all patients (modified after Adour et al., 1974; and Devi et al., 1978). The threshold intensity (the minimal excitability value) and the response (no response in the ipsilateral facial muscles, elicited response in all facial muscles, or elicited response in only some of the facial muscles) were recorded in each patient.

(ii) Needle electromyography (EMG); EMG was carried out for only three muscles, the orbicularis oris, orbicularis oculi, and frontal belly of occipitofrontalis on the involved side. EMG examination was done in: (a) all patients who had abnormal response (partial or no response) to PFNS; and (b) those with a normal response to PFNS, but who had incomplete clinical recovery two months after the disease onset. This is because many patients with a normal response to PFNS recovered com-

pletely in a few weeks and there was no need for an EMG examination. In patients with abnormal response to PFNS the EMG was performed two to three weeks after the onset of Bell's palsy. It was performed according to the technique described by Johnson (1988) using a coaxial needle recording electrode. Needle EMG was performed to detect signs of denervation which are definite indicators of the presence of axonal degeneration (Johnson, 1988).

(iii) Assessment: complete denervation was defined as loss of the facial nerve excitability by stimuli up to 20 mA, with no EMG motor unit action potentials, but in the presence of positive sharp waves and/or fibrillation potentials in all muscles examined by EMG. The category of partial denervation included patients with elicited response in some of the facial muscles on PFNS, but with the presence of positive sharp waves and fibrillation potentials in addition to only a few motor unit action potentials on EMG. Absence of denervation was considered when there was a normal response to PFNS (in the absence of fibrillation potentials and positive sharp waves on EMG examination among patients with incomplete recovery within the first two months). Minimal denervation was considered in the presence of a normal response to PFNS in addition to many EMG motor unit action potentials, but in the presence of few positive sharp waves in fibrillation potentials in only some of the examined facial muscles.

### Physiotherapy

All patients received superficial muscle heating, massage, and electrical muscle stimulation for all the paralytic facial muscles. Three sessions were given per week for up to six months or until complete recovery occurred. This physiotherapy programme was given to retard muscle atrophy, and to maintain muscle tone, without inducing contracture (Rusk, 1977; Sandler, 1982). This programme is known to have no effect on facial nerve recovery.

### Evaluation of recovery

Patients who had no evidence of facial muscle denervation and showed complete clinical recovery (with no residual facial asymmetry) within six months without any complication were considered to have an **excellent recovery.** Those who had minimal or no facial muscle denervation, but had negligible residual facial asymmetry only on close inspection during maximal effort in smiling, whistling, elevating the eye brow, or closing the eyes within six months of the disease onset were considered to have a **good recovery.** Patients with complete facial nerve denervation and incomplete clinical recovery (obvious weakness and/or disfigurement) in the involved facial muscles in spite of proper physical therapy for one year after the disease onset were considered to have a **poor recovery.** Those with partial facial muscle denervation and mild residual facial muscle weakness on maximal effort, with or without minimal contracture or synkinesis, within one year after the disease onset, were considered to have a **fair recovery.** Patients of both the steroid and control groups were assessed blindly by one doctor.

### Data analysis

Patients with excellent and good recovery were con-

sidered to have a satisfactory outcome. This was because patients with a good recovery had nearly complete facial symmetry within six months and they were satisfied. Those with satisfactory recovery were equivalent to grade I and II according to the facial nerve grading system of House and Brackmann (1985). Patients with fair and poor recovery (equivalent to grade III–VI) were not satisfied. Therefore, they were considered to have an unsatisfactory outcome. It should be noted that patients with an unsatisfactory outcome had residual facial weakness, for at least one year after the onset of Bell's palsy, that was recognizable even by nonexperienced physicians. The percentages of patients with satisfactory and unsatisfactory outcomes in the control group were compared statistically to those in the steroid group using the chi-squared test (Hill, 1977).

## Results

There were 74 men and 19 women (aged 17 to 56 years) in the steroid group. The majority of them had complete paralysis (grade VI), but only eight patients had facial paralysis of grade V. Two patients with facial muscle weakness of grade V were seen during the first 24 h following the onset of Bell's palsy. Another two patients with grade V were first seen in the period 24–48 h following the onset. The patients' body weight ranged from 48 to 73 kg.

The control group included 60 patients with complete paralysis (grade VI) and seven patients with facial paralysis of grade V. Their body weight ranged from 51 to 77 kg and their age from 19 to 60 years. There were 55 men and 12 women.

The EMG examination was carried out in 75 patients of the steroid group and in 59 of the control group. Some volitional EMG activity (motor unit action potential of normal parameters) was recorded two to three weeks after the disease onset in six patients with complete paralysis (four in the steroid group and two in the control group). These six patients had a fair–excellent prognosis.

The recovery patterns among patients of both groups are displayed in Table I. In the steroid group, data were analysed with regard to the start of prednisolone intake following the onset of Bell's palsy. All patients who started prednisolone intake within the first 24 h from the onset of Bell's palsy showed satisfactory results compared with only 69 per cent of the control group (Table I).

In data collection, it was hard to determine the exact time of onset of Bell's palsy in five patients among those who were first seen three to five days after onset. These patients had usually discovered the paralysis on waking up. They were unable to decide whether Bell's palsy had developed before, during, or after several hours of sleep. In this condition, we have taken the onset of Bell's palsy from the time the patient discovered the paralysis, and therefore the last steroid subgroup included patients having a critical period of three to five days.

Patients of the steroid group had attained a significantly better recovery than patients of the control group ($\chi^2 = 5.35$; $p < 0.05$). Further statistical comparisons between the control group and the steroid subgroups revealed that patients who started prednisolone intake within the first 24 h had a significantly better recovery than those of the control group ($\chi^2 = 7.88$; $p < 0.01$). There was, however, no significant difference in the outcome between patients of the control group and those who started prednisolone intake either 24–48 h, two to three days, or three to five days after the onset of Bell's palsy ($p > 0.05$).

## Discussion

All patients who started prednisolone intake (1 mg/kg body wt) within 24 h from the onset of Bell's palsy had attained satisfactory results. The percentage of satisfactory recovery appeared to decline in relation to the delaying of the intake of prednisolone. However, patients who started prednisolone intake two to three days following the onset of Bell's palsy had attained a slightly less satisfactory result than those who started it three to five days after the onset. This slight difference could be overlooked because the number of patients who started prednisolone intake two to three days after the onset of Bell's palsy (only 17 patients) is the smallest subgroup. Although there was a significant difference in the outcome between the steroid group and control group, further statistical analysis attributed this significant difference to patients who started prednisolone intake within the first 24 h following the onset of Bell's palsy. Therefore, the period between the onset of Bell's palsy and the start of prednisolone intake appeared to be very important in determining the success of the prednisolone in improving the prognosis of Bell's palsy. Thus, one may call this period the 'critical period'.

In other words, it is very likely that the possible contributing factor for the success of a high dose of prednisolone (1 mg/kg body wt) in improving the prognosis of

TABLE I

RECOVERY IN THE STEROID GROUP (93 PATIENTS) COMPARED TO THE CONTROL GROUP (67 PATIENTS) IN RELATION TO THE START OF CORTICOSTEROID INTAKE FOLLOWING THE ONSET OF BELL'S PALSY*

| Facial nerve recovery | Time of corticosteroid intake in the steroid group | | | | Control group |
|---|---|---|---|---|---|
| | within 24 h n (%) | 24–48 h n (%) | 2–3 days n (%) | 3–5 days n (%) | n (%) |
| Excellent | 19 (83%) | 7 (35%) | 4 (23%) | 9 (27%) | 16 (24%) |
| Good | 4 (17%) | 10 (50%) | 9 (53%) | 17 (52%) | 30 (45%) |
| Fair | 0 | 3 (15%) | 3 (18%) | 5 (15%) | 17 (25%) |
| Poor | 0 | 0 | 1 (6%) | 2 (6%) | 4 (6%) |
| Total | 23 (100%) | 20 (100%) | 17 (100%) | 33 (100%) | 67 (100%) |

n = the number of patients in each group or subgroup.
*Note that satisfactory results (excellent and good recovery) were obtained in all patients who started corticosteroid intake within the first 24 h after onset of Bell's palsy. Satisfactory results occurred, however, in 85, 76 and 79 per cent respectively of patients in other steroid subgroups and in 69 per cent of the control group.

Bell's palsy is its early administration i.e. within 24 h from the onset of Bell's palsy.

The results of this study might explain the existing controversy about the role of corticosteroids in improving the prognosis of Bell's palsy. Adour *et al.* (1978) observed, in his retrospective study, a beneficial effect of prednisone when it was started within the first few days of Bell's palsy. Broadly speaking, his observation does not contradict the findings of the present study.

Brown (1982) reported complete recovery in 87 per cent of patients having incomplete paralysis, but in only 49 per cent of patients who had complete paralysis among his Bell's palsy patients who received cortisone. His patients were given 50 mg of cortisone for five days. The cortisone was started during the first three days following onset. He observed, however, that the best results were obtained when cortisone was started on the day of onset. It is possible that the low success rate or cortisone in Brown's series (1982) might have been due to the low dose of corticosteroids used as cortisone is less potent than prednisolone (Castles, 1985), in addition to the delay in giving it to some patients.

May *et al.* (1976) reported in their prospective study insignificant benefit from prednisone therapy in Bell's palsy. Although their patients started therapy within two days of the onset of the disease, they did not report the starting dose of prednisone or their observation among patients who received it within the first 24 h.

Wolf *et al.* (1978) reported insignificant benefit of prednisone in facial strength recovery among patients with Bell's palsy. Their patients received 60 mg of prednisone (irrespective of their body weight) for 10 days. They reported this finding in one group of patients who started prednisone intake within the first five days following the onset of Bell's palsy. There might have been a long 'critical period' in the majority of their patients.

Prescott (1988) reported failure of prednisolone (80 mg for five days) in improving the outcome of Bell's palsy. The intake of prednisolone began in the majority of his patients within four days following the onset of Bell's palsy. Approximately 25 per cent of his patients started therapy after the fourth day following the onset. He did not, however, mention the percentage of patients who started prednisolone on the day of the onset.

Although Moore (1990) reported that most cases of Bell's palsy recover spontaneously without treatment, unsatisfactory results were obtained in 31 per cent of our control patients. Peitersen (1982) reported, however, spontaneous complete recovery in only 71 per cent of his Bell's palsy patients without giving any treatment. Furthermore, Brown (1982) observed complete recovery, among his Bell's palsy patients without the use of corticosteroids, in 73 per cent of patients with incomplete paralysis and in only 40 per cent of patients with complete paralysis. The relatively high percentage of unsatisfactory recovery (31 per cent) among patients who did not receive corticosteroids (control group) in this study suggests that corticosteroids should be given to improve the prognosis of Bell's palsy. They should, however, be given as early as possible; and preferably in a high dose in order to get the best results. For rapid drug action, one might give the first dose of corticosteroids by the intravenous route.

The results of the present study suggest that the early administration of prednisolone, in a dose of 1 mg/kg body wt, could be useful in decreasing the probability of facial nerve degeneration, possibly through alleviating nerve oedema and compression. Therefore, a high dose of corticosteroids, but under strict medical supervision, might also be given to patients with a relative contraindication to corticosteroids, who are seen on the day of onset of Bell's palsy. This approach could decrease an unwarranted outcome.

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Address for correspondence:
Tarek S. Shafshak, M.D.,
Sidi Gaber, PO Box 41,
Alexandria 21311,
Egypt.