Bell's Palsy – A Restrospective Study

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Introduction

BELL'S PALSY is a lower motor neurone paralysis of the facial nerve, characterized by its acute onset, its lone involvement, and its tendency towards spontaneous recovery. Though much is known about the illness, little is known about its aetiology. Cawthorne and Wilson (1963), and later Langworth and Taverner (1963), postulated that Bell's Palsy was due to compression of the facial nerve in the facial canal, especially at the stylo-mastoid foramen, as a result of swelling and oedema in the fibrous sheath of the facial nerve, caused by an inflammatory process. Ballance and Duel (1963, 1964) reported confirmation of this in their decompression operations. However, later workers did not find any swelling or oedema of the facial nerve, and Drachman (1969) considered that ischaemic facial palsy due to compression of the nerve only occurred in exceptional cases.

Whilst many conflicting views are held by various workers and researchers on the pathogenesis of Bell's Palsy, even more so are the views held by the medical profession on the treatment of the condition. Many advocate masterly inactivity as the best course of treatment as the majority of them (about 80-90%) will recover spontaneously, within 6 weeks to 6 months. The only measures needed are reassurance and attention to the affected eye. Some would include massage and electrical stimulation of the affected facial muscles. But these do not show to have any beneficial effects. In fact, they may even be harmful as they may aggravate the development of contractures. In the earlier days, the commando-type decompression operations were popular, but fortunately this drastic measure is now almost abandoned in the treatment of this benign and almost self-curing disease. Of late, more and more convincing reports are coming forward on the successes in the use of corticosteroids in the treatment of Bell's Palsy. Taverner and his colleagues (1966, 1967) showed that the adminstration of corticotrophin within the first 4 days of the development of Bell's Palsy could reduce the overall incidence of denervation by two-thirds in selected cases and that of severe denervation by 90%. In their later studies (Taverner et al, 1971), it was shown that prednisolone was even more superior than corticotrophin in the treatment of Bell's Palsy.

In this paper, the author made some valuable observations with regard to the aetiology and the role of prednisolone in the treatment of Bell's Palsy, in a retrospective study of a small endemic outbreak of Bell's Palsy in the District of Sitiawan in Perak.

Material and Methods

In the months between May and July 1973, 8 cases of Bell's Palsy presented to the author's clinic for consultation and treatment. Out of the 8 cases, 5 presented within 2 to 4 days of the onset of the illness, I presented on the 8th day, 1 on the 3rd week (about 21 days), and another came about 2 months after the onset of the illness. Diagnosis was made from history, clinical examination and the exclusion of other causes of facial palsy. When it was made certain that the patients were not having an underlying systemic disease, like diabetes mellitus or tuberculosis, all of them were put on a short 10-day course of oral prednisolone, starting with

an initial dose of 30 mg a day for the first 4 days, and then slowly tailing off the dose at the rate of 5 mg a day, Patients were followed up second – daily for one week, then weekly for one month, then monthly until complete recovery occurred. If recovery was slow and not in sight after 4 days, the original starting dose of 30 mg per day was continued for a further 4 days, and then tailed off. If fullness or tenseness in the ears, or heaviness of the head were complained of, the dose was immediately reduced to about 20 mg per day and maintained there until signs of recovery were noted and then the dose was slowly tailed off. Response was assessed clinically. During each follow-up each patient was weighed, his BP checked, and the functions of the affected facial nerve was tested.

Observations and Discussion

Though the study involves only 8 cases, some valuable observations are worth noting.

Out of the 8 cases, 2 are brothers living in the same house, 2 are an uncle and his neice living in close proximity in the same area. All are from two villages in the Sitiawan District about 5 miles apart, and all presented in the short three-month period between May and July 1973. Also, during this period there was an influenza-like epidemic in the region, and one of the patients had an influenza-like illness one week prior to the development of the Bell's Palsy. These observations strongly implicated an infective agent, possibly and probably a virus, as the causal agent in this small endemic outbreak of Bell's Palsy. However, it is regretable that a viralogical study was not done.

Table 1
Made of Presentation

Presenting Complaints	Number of Patients
Crooked mouth	8
Numbness or 'thickish' sensation of the affected side of the face	4
Hyperacusis	1
Loss of taste sensation of anterior 2/3 of tongue	1
Pain behind ear of the affected side	2
Dizziness and vertigo	1

The above summarizes the mode of presentation by the cases studied. All the 8 cases pointed to the crooked mouth as their main complaint. Out of these, two pointed to the non-paralysed side as the side involved because the functioning muscles pulled the angle of the mouth of this side upwards and towards it giving the impression of the mouth being 'crooked' upwards on this side. One case was particularly severely affected presenting with all the above complaints. His dizziness and vertigo persisted and became more severe even after the ceasation of prednisolone therapy and complete recovery of his facial nerve, and necessitated hospitalisation for one week.

Table 2
Age Sex incidence

	Age in years				Total
	15-20	20-30	30-40	40-50	
Number of Patients	1	5	1	1	8
Sex	F	M	M	M	

Prednisolone Therapy

The response to prednisolone therapy was remarkable. Those who presented within one week of the onset of the illness for treatment showed signs of improvement within one week of treatment, and complete recovery occurred within two to three weeks. One case who presented on the second day of the disease had complete recovery within a week. Another case who presented at the third week took about one month for complete recovery. And one case, an 18-year-old school girl, who presented at about two months, did not show any improvement one month after treatment; she subsequently absconded.

It is learned from here that the earlier prednisolone therapy is started the faster is full recovery. Also, prednisolone therapy does not only prevent denervation but it definitely hastens recovery. Taverner and his colleagues (1971) recommended a higher dose, starting at 20 mg tid for 4 days, then reducing by 20 mg per day for 4 days, and ending on the final day with 10 mg. The author used a much smaller dose. It is the author's belief that the sodium and water retention effect of prednisolone may aggravate the already swollen and oedematous facial nerve thus causing more compression especially in the first few days of the treatment. In this study, 2 patients complained of fullness or sensation of tension in the ears two days after the treatment, and five put on 2-3 lbs in the first week of treatment, and one even complained of worsening of the condition until the dose was reduced. This showed that the harmful sodium and water retention effect of prednisolone could offset and even surpass its beneficial anti-inflammatory effect. This is especially so when

the dose is large and in the first few days of the treatment. Observation in the study showed that the sodium and water retention effect of prednisolone usually lessens as the therapy is continued even if the dose is not reduced. In the initial treatment of Bell's Palsy, the sodium and water retention effect of prednisolone is most undesirable as it will cause more oedema and swelling and hence more compression. The main aim of the treatment is to prevent denervation as a result of ischaemia caused by the compression. Therefore, especially for our Asians, who on the whole tolerate smaller drug dose than our European counterparts, it is the author's opinion that the smaller dose should be used instead of that recommended by Taverner and his colleagues in order to prevent more oedema and hence more compression. Also adjustment should be made for individual cases when necessary.

Conclusion

An opportunity was taken to make a retrospective study of a small endemic outbreak of Bell's Palsy in the District of Sitiawan. Though the study involved only 8 cases, some valuable observations were made. A viral agent was strongly implicated as the causal agent in the endemic, and the study showed that prednisolone has difinitely a place in the treatment of Bell's Palsy. It hastens recovery and prevents denervation. Taverner and his colleagues used prednisolone only for selected cases and did not recommend the treatment for teenage patients as most would recover spontaneously. It would seem, however, that these young patients would be denied the benefit of the treatment, if they are not given at least a trial course of the treatment. It is the author's opinion that all patients with Bell's Palsy presenting within one month of the onset of the illness should be given a trial of the treatment.

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