

A Review of 250 Cases of Bell's Palsy

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SUMMARY

Two hundred and fifty cases of Bell's palsy seen at the ENT Department of Groote Schuur Hospital are reviewed. The most important findings which emerge are the greater incidence in women, many of whom are in the third trimester of pregnancy; pain in or around the ear, and depression or loss of electrical response in the affected muscles—which are both of bad prognostic significance; and permanent sequelae, many of only minor degree, which remain in about one-third of all complete palsies, unless prednisolone is administered within a week of onset in which case the incidence of sequelae is greatly reduced.

An earlier plea for the referral of such a case as an emergency to a centre especially interested in the diagnosis and treatment of this condition, is repeated.

S. Afr. Med. J., 48, 593 (1974).

Bell's palsy poses several unanswered questions of considerable practical importance in relation to diagnosis, prognosis and treatment. The causation of this relatively common condition and the nature and location of the lesion are still a matter of debate. The general practitioner may only see a case at infrequent intervals and may have the good fortune to observe complete recovery in every case. From this the mistaken idea is still prevalent that the prognosis is always favourable, an erroneous concept that figures drawn from the present study dispel. The plea made earlier is repeated that cases of Bell's palsy be referred as emergencies, for specialist assessment and treatment without which other causes of a 7th nerve palsy may be overlooked, with most serious consequences.

Partly as a result of the publication of an article in the *South African Medical Journal* in June 1969¹ an encouraging number of early cases of Bell's palsy were referred to our ENT Department. This made possible a trial of steroid therapy which others have advocated as being effective if given within a few days after the onset of the palsy. Earlier in the series the records were not as complete as is desirable, but a routine approach to diagnosis and treatment has gradually evolved and is now standardised. These earlier incomplete records prevented a full evaluation of all aspects through the whole series, but interesting facts and figures nevertheless emerged. Failure of patients to report back as requested made follow-up information incomplete.

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Date received: 11 October 1973.

PATIENTS

Females predominated in the ratio of 151 to 104. The right ear was affected 140 times and the left 119, 4 cases being bilateral. The age distribution is shown in Fig. 1, the peak incidence being in the third decade.

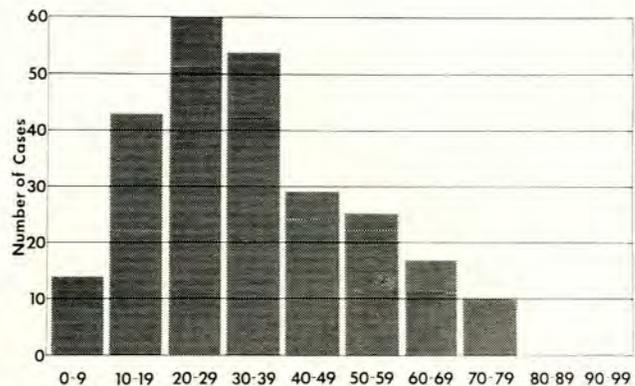


Fig. 1. Age distribution in 250 cases of Bell's palsy.

Recurrent palsy occurred in 15 cases. In one the opposite side was affected 4 days after the first, in another there was an interval of 2 weeks between the two sides, and a child of 4½ years had had an attack on the same side 9 months previously, each attack reported as following immediately after a fright. Of the 12 cases where the side of the previous attack was known, it was the same in 6 and opposite in 6. Sixteen palsies developed during pregnancy or in the puerperium and one after a miscarriage. Six examples of the Ramsay-Hunt syndrome were encountered but not included among the statistics of the Bell's palsies.

CONCOMITANT SYMPTOMS

There are three symptoms commonly noted by the patient in addition to the facial palsy. Epiphora due to lack of tone in the lower eyelid and consequent failure of the punctum to make contact with the globe of the eye is often present. Pain is a frequent complaint, and may be in the ear, spread more widely over the head, down the neck, or into the eye. It is usually present for only a few days and may precede the palsy for up to 72 hours; but occasionally it comes on several days after the palsy and may be severe and persistent. Tenderness over the stylo-mastoid foramen may be present.

Pain is of bad prognostic significance and its presence or absence was specifically asked for in 205 cases. Pain was present in 101 cases and was followed by permanent sequelae in 31 of these. In a further 24 it was present, but follow-up to determine permanent defects was not possible. Pain was stated to be absent in 65 patients, of whom only 4 developed sequelae, while follow-up was not possible in another 14.

Patients were asked about subjective taste changes. Of 71 who reported this, 12 developed sequelae, while 54 who denied the symptom also produced 12 permanent defects. It would appear that this symptom has no prognostic significance.

INVESTIGATIONS

Objective taste testing by electrogustometry was performed on a number of patients, but the answers in the type of patients seen were not very reliable, and it was not done as a routine.

Salivary flow testing was performed on 36 of these patients and the results were reported by Potgieter.² He did not find that this test gave better prognostic information than the electrical tests, and it was more time-consuming and unpleasant for the patient.

The stapedius reflex is usually lost in the ear on the affected side, and this was studied by means of a Madsen impedance meter, model ZO 61 in the earlier cases, and later with a ZO 70. Recently a special booster amplifier has been used to give up to a maximum of 120 db for the stimulus tone. This examination was undertaken in 120 patients, and a further 11 are excluded from analysis as no reflex was obtainable even from the normal side. Apart from its value as an aid in localising the point of the lesion, it was hoped that it might have some prognostic value. This was not realised, as the recovery of the reflex was extremely variable and unrelated to the progress of recovery in the facial muscles. In some patients it remained absent even when the face had almost entirely returned to normal, while in others it returned, but with a diminished amplitude of the impedance change. In a few cases a reflex was obtained, but this rapidly fatigued. The reflex was not lost in 29 patients, in 14 of whom the palsy remained partial. In 4 it was reduced but not entirely lost at any stage. The time of return was noted to be before a 50% recovery of function in the face in 21 patients, and after 50% function in 35 patients. In 31 patients the time of return could not be determined for various reasons.

The facial palsy was diagnosed as being of the Bell's type when it was a lower motor neurone defect, and all other causes such as ear infection, trauma, herpes or any other neurological diseases had been excluded. In small children it is difficult to test facial muscle functions critically, and in some such patients it was not easy to decide if the palsy was complete or partial, or to exclude an upper neurone lesion, which, however, was unlikely at this age.

Nerve excitability tests were performed on all cases in the later part of the series, using a Medelec model 402

stimulator. Two positions on each side of the face were used as a routine, and in some patients stimulation was also given over the main trunk of the nerve at its exit from the stylomastoid foramen. The two most sensitive points were found to be over the lateral orbital margin and about 1.5 cm below the angle of the mouth. The exact point of maximum sensitivity varied from patient to patient and was searched for with a supra-threshold stimulus. The threshold in milli-amperes at this location was then determined, the point below the mouth usually requiring about 1 mA more than the one at the outer side of the eye. Tests were not done at intensities above 10 mA, as overflow to the muscles of mastication usually occurred then. The normal threshold varied from 1 to 4 mA. Some workers have used the criterion of 3.5 mA difference between the two sides as an indication of axonotmesis, but 4 mA was chosen in this series, as the variations found even on the normal side from day to day made fractions of a milli-ampere seem insignificant. With very few exceptions the tests were clearly well above or below the 4 mA criterion, so that the exact level used would have made little or no difference to the result.

Considering only cases with complete palsies, in 107 the difference between the two sides did not ever exceed 4 mA; of these 83 recovered with no sequelae, 7 developed some permanent defect, 14 were lost to follow-up and 3 are too recent for assessment. Where the difference did exceed 4 mA, 27 out of 50 cases developed sequelae, 4 are lost to follow-up and 7 are too recent for assessment. Only 13 are therefore definitely known to have recovered with no defect. From these figures the 4 mA level seems to be a useful guide to prognosis.

Schirmer's test of lacrimal secretion was performed on 83 patients. It is felt that this is a rather crude test and only a very marked difference between the two sides was taken as evidence of involvement of the great petrosal nerve. From experience in testing of patients who have been subjected to Vidian neurectomy, the denervated side usually produces only about one-third of the secretion of that on the normal side. Accordingly the test was only considered to be indicative of a lesion proximal to the geniculate ganglion if the secretion was less than half of that on the normal side. One problem in Bell's palsy is that epiphora is usually present from a pool of secretion lying in the lower fornix as has been mentioned above. This pool must be taken into account when assessing the response to the inhalation of ammonia in the nostrils. Usually this pool causes the paper strip to show an immediate wetting on the palsied side 1-2 cm in extent. Secretion starts later in the normal eye, often after a few minutes' delay, and overtakes the extent of wetting on the palsied side if secretion there is reduced. If the affected side is secreting normally both sides advance together.

Fisch and Esslen³ reported that in some cases secretion on the palsied side was more than on the normal side, a finding also encountered in the present series. Using the criteria given above, secretion was normal in 49 cases, reduced in 23 cases, and increased in 11. Reduced secretion suggests that the lesion is in the labyrinthine segment of the facial nerve, an area that Fisch explores as a routine by the middle fossa approach to the internal auditory

meatus. Exposure of this region had not yet become practicable at Groote Schuur Hospital during the period covered by this series.

OTHER CONDITIONS

In 17 cases Bell's palsy developed during pregnancy or in the postpartum period. Ten of these occurred in the third trimester, 3 in the earlier part of the pregnancy, 3 within a month after delivery, and one followed 2 weeks after a miscarriage.

Four of the patients were diabetics, all 50 years of age or more. Two of these developed sequelae, a third shows complete denervation and sequelae will probably develop, while the fourth appears likely to recover fully.

TREATMENT

Surgical Decompression

This is still a debatable procedure. Exposure of the tympanic and mastoid portion of the nerve was undertaken in 12 patients in the present series. One of these is lost to follow-up, but in all of the remaining 11 some degree of permanent sequelae remains, as is to be expected, since the operation was only performed when evidence of complete denervation was present. The indications were total loss of electrical responses to faradic stimulation and for the relief of severe pain. Once response was lost, an operation was performed as soon as possible. It was not advised in children under 10 years or during pregnancy.

Permanent Sequelae

These were observed in 46 patients but in 2 instances the patients attended only because of sequelae, and in one the treatment was in doubt, so these are excluded from the calculation of percentages. In many the defect was slight, causing no disability to the patient and observable only by the examiner. Where the palsy was rapidly resolving after a few weeks, it was assumed that there would be no permanent defect, and it was felt unjustifiable, and usually impracticable, to recall the patients for examination months later.

TABLE I. SEQUELAE OBSERVED IN 217 CASES

Incomplete recovery after 6 months	35
Synkinesis	34
Twitching of face (not definite facial spasm)	6
Crocodile tears	7
Chronic facial pain	1
Narrowed palpebral fissure	8
Contractures of face	2

The last two defects would appear to be due to a contracture of the previously paralysed muscles, most commonly the orbicularis oculi, and in the last-named the muscles of the mouth also.

Permanent defects were assessed at the end of 6 months, although in some cases they occurred earlier. Synkinesis was observed in one patient 3 months after the onset of the palsy. In 217 patients adequate follow-up has been possible and sequelae remain in 19%. If 62 partial cases are excluded, the figure becomes 27%, as no partial case developed sequelae. The defects observed are shown in Table I, some cases having more than one sequel.

Treatment of Early Cases with Steroids

This has been widely advocated, particularly by Taverner *et al.*,⁴ and was employed in many of our series, except where there was a delay of 7 days from the onset of the palsy. Children, women during the first three months of pregnancy, and patients with a history of diabetes, tuberculosis or peptic ulcer, were also excluded.

The rapidity of recovery was difficult to assess as most of our patients could not afford to attend frequently. Accordingly, the incidence of permanent sequelae as listed in Table I was used as the criterion by which to assess the benefit, if any, of steroid therapy. In 66 patients with complete palsies, in whom for various reasons steroids were not given, permanent sequelae were observed in 24; while of 46 treated by ACTH 14 had sequelae, and 5 sequelae developed in 43 cases treated by prednisolone, but were of slight degree in 3. Excluded from the comparison were 35 who had either not attended sufficiently for assessment to be made or are still under observation, 3 about whose adequate treatment there was some uncertainty, and 62 partial palsies, most of whom were not given steroids. Partial palsies of marked degree seen early were, however, sometimes treated with steroids because it was observed that some of these lesions progressed to a total loss of function after a few days. It was felt that the early use of steroids might prevent this progress. This remains a debatable point, but now that the benefit of prednisolone appears well authenticated it is felt that this treatment is justified in all but minor cases.

Summarising these 154 cases where follow-up was achieved, sequelae of some degree were observed in 35% of those not receiving steroids, in 30% of those given ACTH, and only in 12% of those treated with prednisolone. These percentages are very close to those found by Taverner *et al.*⁵ and reported in their second paper. In Taverner *et al.*'s first paper they considered ACTH to be more effective than oral cortisone, but the later trial⁷ suggested that prednisolone was even more effective. As this is given orally, avoiding the daily injections, it has been employed here in the more recent cases. These results in this small series are encouraging, although the numbers are too small to be conclusive, and the trial of prednisolone is continuing. The course consists of 80 mg daily for 5 days, followed by 60, 40, and 20 mg on the 3 succeeding days.

DISCUSSION

There is still a lot to be learnt about Bell's palsy. The cause is still a matter for dispute, various opinions being

held regarding the nature and site of the lesion; many different tests are employed in an attempt to find a reliable basis for prognosis; and treatment varies from one physician to another, and has often changed with further experience in the same worker. The literature on facial palsy is voluminous and only some will be mentioned here.

Most published series show no preponderance of side affected, and the difference noted in the present study of 7 right to 6 left is probably not significant.

The sex difference of almost 3 females to 2 males agrees with the findings of others. Robinson and Pou⁶ report a predominance of females of nearly 4 to 1. El-Ebiary⁷ from Egypt found 56% (under 30 years—61%) to be female, but over 50 years of age males predominated. On the other hand, Liebowitz⁸ from Jerusalem, and Cawthorne and Haynes⁹ from London, found equal sex incidence. The age distribution in this series differs from the Egyptian figures, where the greatest incidence was in the second decade, and mostly female. The Jerusalem figures peak in the third decade, as in Cape Town, and in the sixth decade.

From the study of published series it would appear that the prevalence, age and sex distribution of this condition varies considerably in different parts of the world.

Some authors note a seasonal incidence and have postulated an infective agent or climatic condition as being an influence. Liebowitz⁸ found that younger patients tended to develop Bell's palsy in the cold season while older patients have it in the warm weather. This reported trend was not apparent in our series, with peak incidence in July and a minimum in November and December, giving some support to a possible infective pathology.

The prevalence of Bell's palsy among women during the third trimester of pregnancy has been reported. Robinson and Pou⁶ found 20% of their female patients in the third trimester of pregnancy or in the puerperal period. The comparable figure in the present series is 10%, but out of 40 female patients in the 20-29-year age group, 11 were pregnant or in the puerperium. Edwards¹⁰ reports a similar experience. Pope and Kenan¹¹ report on 7 cases developing during pregnancy but do not think this incidence is unduly high, 5 occurring out of one series of 207 female cases. It is interesting that 4 of these developed in the third trimester and 6 had incomplete recovery, the seventh still recovering from surgical decompression. By contrast only 5 of the 17 cases in the present review developed permanent defects.

The value of electrical tests to distinguish between neuropraxia and axonotmesis is confirmed. Unfortunately the evidence of denervation is well known to be delayed up to 72 hours from the time of damage. It has been claimed that threshold excitability tests may still appear normal when only some axons remain viable, and the electromyographic response to a maximal stimulus has been advocated as giving earlier information. While the difference in threshold excitability between the two sides was employed as a

criterion in the present study, note should be taken of the work by Safman,¹² who presented evidence of the presence of bilateral pathology in many cases of Bell's palsy, suggesting that the apparently normal side may not be valid for comparison. Ideally, surgical decompression should be performed the moment axonotmesis is threatened, but before a significant number of axons have begun to degenerate; but, as is pointed out by Laumans and Jongkees,¹³ operation is worth doing even after evidence of denervation, in the hope of saving axons which are still in the stage of neuropraxia.

Electromyography gives clear evidence of denervation, but only 2 or 3 weeks after the onset, and so is of no help in deciding on emergency decompression in the early case. It was not employed in our patients.

It was noted that subjective taste changes appear to have no prognostic significance, a conclusion also reached by Laumans and Jongkees.¹³ Although opinions are divided, the consensus seems to be that such changes are without prognostic significance. The search for a test to give early evidence of threatened denervation continues, and its discovery will be a major contribution to the management of this disease.

Fisch and Esslen³ suggest that the lesion is often sited in the labyrinthine segment of the nerve, with the obvious deduction that surgical decompression, if undertaken, must include this relatively inaccessible area. This they do as a routine, and if their work is confirmed, this formidable and time-consuming technique must become part of the otologist's expertise.

The most rewarding outcome of this study has been the confirmation of the great benefit of treatment by prednisolone when given to early cases. It is unfortunate that with the population group from which these cases were drawn, follow-up is not always possible. In spite of this, it is felt that the figures adequately demonstrate the superiority of prednisolone over ACTH, and of both over treatment without steroids. The follow-up failure applies to all groups, so that the development of only 5 sequelae (minimal in 3) out of 43 cases is strong evidence of the value of treatment with prednisolone.

REFERENCES

1. Jarvis, J. F. and Maytham, D. V. (1969): *S. Afr. Med. J.*, **43**, 810.
2. Potgieter, M. G. (1973): *Ibid.*, **47**, 27, 100.
3. Fisch, V. and Esslen, E. (1972): *Arch. Otolaryng.*, **95**, 335.
4. Taverner, D., Fearnley, M. E., Kemble, F., Miles, D. W. and Peiris, O. A. (1966): *Brit. Med. J.*, **1**, 391.
5. Taverner, D., Cohen, S. B. and Hutchinson, B. C. (1971): *Ibid.*, **4**, 20.
6. Robinson, J. R. and Pou, J. W. (1972): *Arch. Otolaryng.*, **95**, 125.
7. El-Ebiary, H. M. (1971): *Rheum. Phys. Med.*, **11**, 100.
8. Liebowitz, V. (1966): *Neurology (Minneapolis)*, **11**, 100.
9. Cawthorne, T. and Haynes, O. R. (1956): *Brit. Med. J.*, **2**, 1197.
10. Edwards, C. E. (1964): *Amer. J. Obstet. Gynec.*, **89**, 274.
11. Pope, T. H. jun. and Kenan, P. D. (1969): *Arch. Otolaryng.*, **89**, 830.
12. Safman, B. L. (1971): *Ibid.*, **93**, 55.
13. Laumans, E. P. J. and Jongkees, L. B. W. (1963): *Ann. Otol.*, **72**, 621.