CLINICAL REVIEW

Bell's palsy and its treatment

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Introduction

The recent introduction of new therapeutic regimens for patients with Bell's palsy has stimulated much interest in this disorder. However, Bell's palsy is already the subject of an extensive and often conflicting literature. During a controlled trial, as yet incomplete, of steroid therapy in this disorder, the opportunity was therefore taken to critically review some of the more important contributions to it.

Although patients with a peripheral facial palsy were described by Powell in 1813, it was Charles Bell who, shortly afterwards, first established the function of the seventh cranial nerve (Bell, 1821). His name was initially attached to all cases of facial palsy. However, it has now become customary to designate as Bell's palsy a lower motor neurone facial paresis, of acute onset, unaccompanied by evidence of aural or neurological disease and without other local cause.

Incidence

Although it is one of the commonest conditions seen in neurological practice (Peiris & Miles, 1965) there have been few studies of its frequency in the general population. Gregg (1961) estimated an average annual incidence of 0.16/thousand population in Belfast by an analysis of hospital records while Melotte (1961) found an annual incidence of 0.25–0.16/thousand at risk from a survey of selected general practitioners in England. It occurs in all age groups and has an equal sex incidence.

There are occasional reports of a familial incidence. Simmonds (1919) reported a 17-year-old girl who had had two attacks of Bell's palsy; her sister, maternal aunt, uncle and grandfather had previously been affected. Cawthorne & Haynes (1956) described a patient and her brother, both of whom had had recurrent Bell's palsies. Alter (1963) found that in thirty of 105 probands at least one other family member had been previously affected compared with four such relatives in a similar number of

control patients. He suggested that hereditary factors were important, probably as a single autosomal dominant trait with low penetrance rather than as a recessive trait since there was no increased consanguinity in the Bell's palsy group. There is a paucity of data from other sources on the incidence of Bell's palsy in family aggregates. However, of seventy patients with Bell's palsy seen by the author, in only three cases—in all of whom there was an uneventful past history—had another family member been previously affected. This was no higher than the incidence of Bell's palsy in the family members of seventy consecutive patients seen with other neurological disorders. It would seem, therefore, that while hereditary factors may be of importance in individual cases, their general significance remains uncertain.

There is a high recurrence rate in patients who have had a Bell's palsy and this is consistent with an hereditary predisposition to the disorder. Cawthorne & Haynes (1956) recorded a recurrence rate of 9.5% in their series and in the author's series there was a 10% recurrence rate.

The hereditary factor is unknown. The stylo-mastoid foramen (Leiner, 1919) and fallopian canal (Fowler, 1961) are variable in size and anatomical abnormalities may predispose to the condition. Cawthorne & Haynes (1956) found unusual cellularization of the mastoid bone in two patients with recurrent Bell's palsy and suggested that an increased susceptibility to temperature change might be a significant pathogenic factor. Similar radiological changes were not present in six personal cases of recurrent Bell's palsy nor in the cases of Alter (1963).

Aetiology

The aetiology of Bell's palsy is unknown and although a number of pathogenic factors have been proposed, their importance has not been convincingly demonstrated. Exposure to cold has been considered an important factor by many (Cobb &

Coggeshall, 1934; Merwarth, 1949). This implies a seasonal variation in the incidence of Bell's palsy which has not been confirmed (Adler, 1943; Winternitz, 1947; Park & Watkins, 1949; Kettel, 1959). However, Leibowitz (1966) found it to be more common in the cold season among patients below the age of 40 years. In a subsequent study he found that cases appeared in clusters suggesting that they were related to a common aetiological factor, possibly infective (Leibowitz, 1969).

Aitken & Brain (1933) showed by complement fixation tests that herpes zoster antibodies were present in four of twenty-two cases of Bell's palsy that were clinically indistinguishable from each other. It has since been shown that a facial palsy may also occur in association with mumps (Saunders & Lippy, 1959), rubella (Fowler, 1963), varicella (Wallace, 1960; Ravin, 1961) and infectious mononucleosis (Fowler, 1963; Saunders, 1963; Davidson & Salter, 1964). In the great majority of patients with Bell's palsy, virus isolation and antibody studies have, however, proved negative.

An ischaemic aetiology of Bell's palsy has been proposed by others (Cawthorne & Haynes, 1956; Cohen, 1960; Blunt, 1962; Korkis, 1963). Arteriolar spasm (Hilger, 1949) perhaps as a consequence to cold exposure (Sullivan & Smith, 1950), is presumed to cause ischaemia of the facial nerve near the stylomastoid foramen and thereby lead to a failure of conduction. Secondary venous stasis leads to swelling of the nerve which is consequently compressed in the facial canal (Kettel, 1947, 1954). Such a theory remains speculative although it provides the theoretical basis for steroid therapy in this disorder. There is a well-documented but uncommon association of facial palsy with malignant hypertension (Lloyd, Jowitt & Still, 1966) and this has been attributed in some cases to arteriolar disease, causing infarction and swelling of the facial nerve in its bony canal (*British Medical Journal*, 1966). However, it is more often due to haemorrhage in the facial canal of the petrous bone (Moxon, 1869; Redwood, 1926). An association of facial palsy with benign hypertension has not been convincingly demonstrated.

In a series of patients with Bell's palsy, the frequency of diabetes mellitus was recently reported as 66% by Korczyn (1971a) who related the mononeuropathy to diabetic angiopathy. However, Aminoff & Miller (1972) reported a 6% frequency of impaired glucose tolerance in their series and, since this is no higher than in the general population (Butterfield, 1964), concluded that diabetes mellitus was not a common aetiological factor in the development of Bell's palsy in this country.

Korczyn (1971b) has also reported that in a series of eighty-five women with Bell's palsy, seven

presented during the third trimester of pregnancy. He suggested that this association was more frequent than would be expected by chance and speculatively related it to fluid retention. However, these conclusions lack adequate validation. Specialist referral is probably more likely for pregnant women, especially if they are regularly attending a hospital antenatal department, and there is no published evidence of an increased incidence of Bell's palsy in pregnancy.

Prognosis

Perhaps as a consequence of its uncertain aetiology many different therapeutic regimens have been advocated in this condition. Their validity cannot be adequately assessed without an appreciation of the factors affecting the prognosis in untreated cases. On clinical assessment, a poor prognosis is indicated by the presence of initial pain (Tumarkin, 1936; Cawthorne, 1952; Kettel, 1959; Taverner, 1959). Thus, Dalton (1960) found that of forty-four patients with Bell's palsy and accompanying pain only 59% made a complete recovery while of forty-two patients without pain, 74% recovered completely. Advancing age (Matthews, 1961) and a complete paresis (Matthews, 1961; Taverner, 1959) are also of poor prognostic significance. Thus, in a series of patients seen within 2 weeks of onset, 80% of those with a partial palsy recovered completely compared with 35% of those with a clinically complete palsy (Matthews, 1961). A clinically and electromyographically complete facial paresis occurred in 28% of patients in Taverner's (1955) series and in 26% of the series by Aminoff & Miller (1972).

Early onset of recovery is of favourable prognostic significance (James & Russell, 1951); in cases of complete recovery improvement usually begins within 1 month of onset. The rate of complete recovery is usually given as 85% (Cawthorne & Haynes, 1956). However, a complete recovery rate of only 68% was obtained by Matthews (1961) in patients seen within 6 days of onset and he was only able to obtain a higher figure by the most favourable assessment of patients seen in the first 2 days. Moreover, he showed that a complete palsy in the second week after onset had a significantly lower chance of complete recovery than such a palsy in the first week. In the formulation of clinical trials of different therapeutic regimens patients must, therefore, be appropriately matched with regard to these factors.

The prognosis in Bell's palsy depends on whether physiological (reversible) block has occurred or whether the facial nerve degenerates. Patients with physiological block will recover completely without sequelae, and require no specific treatment. In

patients with denervation, recovery is incomplete and long-term sequelae may be troublesome. A contraction of the affected side may develop leading to deepening of the nasolabial fold and narrowing of the palpebral fissure, but this often reduces any facial asymmetry at rest. An involuntary contraction of one part of the face may occur when another part is moved. Such associated movements were found by Taverner (1955) to occur in all patients with denervation as also did spontaneous twitching movements, often very slight, which occurred about the mouth on blinking. There was no relationship between the degree of recovery in these patients and the presence of associated movements. These sequelae have been attributed to branching and misrouting of regenerating axons so that a motor neurone discharge excites two distinct areas in the face (Lipschitz, 1906) and animal studies have provided support for this view (Howe, Tower & Duel, 1937). Unilateral lachrymation on eating (Kaminsky, 1929) may also develop and occurred in 21% of patients with denervation in the series of James & Russell (1951) and in 12% of Taverner's (1955) series. It has similarly been attributed to misrouting of regenerating nerve fibres. Other concepts of the pathogenesis of these phenomena have been critically reviewed by Taverner (1955) and will receive no further consideration here.

In a series of 257 patients with Bell's palsy seen within 2 weeks of onset, Taverner (1959) reported a 40% incidence of denervation; 25% of patients with denervation were dissatisfied with the final outcome. Attempts at treatment have, therefore, been directed at reducing this incidence.

In determining whether or not denervation has occurred, the clinical application of electrodiagnostic techniques has proved of considerable value. Electrodiagnostic methods for assessing prognosis were first applied to patients with facial palsy by Duchenne 100 years ago. He stimulated the facial nerve with an electrode in the external auditory meatus and in patients who subsequently recovered incompletely was unable to evoke a response after about 1 week from the onset of the disorder. Taverner (1955) assessed prognosis correctly in ninety-three of ninety-six cases by using an electromyographic technique to detect fibrillation potentials. These indicate that denervation has occurred but their appearance may be delayed for some 2 or 3 weeks after the onset of facial weakness. Gilliatt & Taylor (1959) stimulated the facial nerve percutaneously in front of the ear and measured conduction time to the orbicularis oculi in three patients before and after the nerve was sectioned. There was little, if any, change in latency of the muscle response until it became unobtainable; moreover, although the visible muscle twitch disappeared within 3 or 4 days

a response was recorded electromyographically for a further 2 or 3 days. They emphasized that electromyographic sampling may reveal surviving motor unit responses to nerve stimulation in the absence of clinical correlates and stressed its importance in determining whether denervation after Bell's palsy was partial or complete.

Campbell *et al.* (1962) tested facial nerve excitability by percutaneous stimulation, comparing the intensity of current required to produce minimal visible contraction of facial muscles on the normal and affected sides. All patients had a complete facial palsy and were assessed as soon after the third day as possible. If the affected side responded fully at the same current intensity as the normal side, the lesion was attributed to physiological conduction block proximal to the site of stimulation; 90% of these patients subsequently made a full recovery. The affected facial nerve became inexcitable in some patients due presumably to complete denervation and the rate of complete recovery in this group was reduced to 20%. Since responses to nerve stimulation were not recorded electromyographically, it is possible that some patients with partial denervation were included in this group and the recovery rate would probably have been lower if these had been excluded. In the remaining patients, the facial nerve was excitable but at a higher intensity than normal; this was attributed to partial denervation and only 49% subsequently recovered completely. The poor prognostic significance of an inexcitable facial nerve was confirmed by Langworth & Taverner (1963) who reported that conduction in the facial nerve was retained in patients with partial denervation although it was often delayed.

Peiris & Miles (1965) assessed the prognosis in patients with Bell's palsy using the method of electro-gustometry. They found that an increase in the threshold to anodal galvanic stimulation of the tongue on the affected side was associated with evidence of denervation in twenty of twenty-one cases seen within 14 days of onset of the weakness. In twenty-one patients with no evidence of denervation the galvanic threshold was normal. The method enabled a correct prognosis to be made before facial nerve conduction was affected. Thus, in nine of their twenty-one patients with denervation facial nerve conduction was normal within 6 days of onset of the weakness when the response to galvanic stimulation of the tongue was already abnormal. However, after 14 days from onset, the correlation of an altered taste threshold with denervation is less complete, presumably because taste recovers early. The value of electro-gustometry was confirmed by Taverner, Kemble & Cohen (1967) who used it in a series of patients they studied within five days of onset of facial paresis. They were able thereby to

predict which patients would recover completely with an accuracy of 93%. Curiously, subjective impairment of taste is not a good prognostic indicator (Taverner, 1959) and does not correlate with the results of electrogustometry (Taverner *et al.*, 1967).

Treatment

The high spontaneous recovery rate in any large series of patients makes mandatory the careful scrutiny of any proposed treatment. Controlled trials of treatment with oral cortisone (Taverner, 1954), and cervical sympathetic blockade (Fearnley *et al.*, 1964) have provided no evidence of benefit. Galvanic stimulation of the facial musculature is similarly without benefit (Mosforth & Taverner, 1958).

Treatment with ACTH for patients seen within 5 days of onset of their facial weakness has been alleged to reduce the failure (denervation) rate to 13% from a predicted 40% (Taverner *et al.*, 1966; Taverner *et al.*, 1967). Several cogent criticisms of these studies have, however, been made (Groves, 1968; Campbell, 1968). Patients referred to hospital must always be selected (Matthews, 1961) and it is not clear whether local practitioners were asked to refer all patients or whether the study was performed on patients who happened to have been referred. This is of some importance for Melotte (1961) found that only 35% of patients seen by their general practitioner were referred for specialist opinion and these may well have constituted the more severe cases. Moreover, in their initial controlled trial, Taverner and his colleagues failed to show any significant benefit of therapy and it was only by the use of retrospective controls that their claim could be justified. A failure-rate of 40% was quoted for these controlled patients, but Taverner (1959) earlier reported this as the failure-rate for patients seen within 14 (not 5) days. Since the failure-rate increases with time from onset of Bell's palsy, it is clearly unjustified to compare patients treated within the first 5 days to untreated patients first seen within 2 weeks (Matthew, 1961). The denervation-rate in patients seen within five days has recently been reported as 11% (Groves, 1968) and 13% (Campbell, 1968) which is similar to the failure-rate reported in patients treated with ACTH.

It may well be that the early treatment with ACTH of patients with Bell's palsy will successfully reduce the denervation-rate, but it must be concluded that this has not yet been adequately demonstrated.

Taverner, Cohen & Hutchinson (1971) have recently compared ACTH with prednisolone therapy in this disorder. Patients were assessed clinically and electromyographically, and only those with a presumed bad prognosis were entered in the trial.

Unfortunately, no control group of untreated patients was studied so that the denervation-rate without treatment in the group of patients selected for study is unknown. Moreover, the dose of ACTH used was lower than that which had previously been employed and for which benefit has been claimed. Ninety-four patients received ACTH and thirty-two developed denervation while only thirteen of ninety-two treated with prednisolone developed denervation on clinical assessment. The exact significance of these results is unclear in the absence of an adequate control group. However, since patients were selected to have a poor prognosis, it can be inferred that a failure rate of only 14% in the prednisolone-treated group probably implies a favourable therapeutic response.

Surgical treatment of Bell's palsy has been advocated by some aural surgeons. Direct exposure of the facial nerve trunk in cases of paralysis was first described by Alt in 1908. The subsequent work of Ballance & Duel (1932) led to the introduction of surgical decompression of the facial nerve by opening the fallopian canal from the stylo-mastoid foramen to the lateral semicircular canal in cases of severe facial paralysis. However, there is no published evidence to show that surgical treatment favourably influences the prognosis in Bell's palsy since most reports detail uncontrolled observations made in selected cases (e.g. Kettel, 1947; Cawthorne, 1952; Sullivan & Smith, 1959). The only controlled trial is that of Mechelse and his colleagues (1971) who found that surgical decompression in the second and third weeks did not influence recovery. On the evidence currently available, the conclusion is therefore unavoidable that surgical decompression has no place in the management of patients with this disorder. This is supported by recent experimental studies. The facial nerve of the monkey has a similar course to that in man; by ultrasonic irradiation, a well-defined vascular lesion can be produced in its intratemporal portion simulating that which is presumed to occur in patients with Bell's palsy and similarly leading to facial paralysis. Facial nerve decompression, two or seven days after irradiation, did not affect the rate of recovery of facial function which was usually complete after about 8 weeks (Boyle, 1972).

Bell's palsy is an enigma. Its pathogenesis is uncertain and this is reflected in the multiplicity of therapeutic regimens which have been advocated for it in the past. The introduction of electrodiagnostic methods, particularly electrogustometry, has allowed the early recognition of patients with a poor prognosis and it is these patients alone who require treatment. Recent studies indicate that steroid therapy may well be helpful in these circumstances but this now awaits more adequate validation.

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