Preliminary Communications

Prevention of Delayed Traumatic Facial Palsy

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Taverner *et al.* (1966) showed that treatment with intramuscular A.C.T.H. gel increased the recovery rate from Bell's palsy and reduced the incidence of denervation. They claimed that even better results would be obtained if all patients were treated within 24 hours of the onset of paralysis. It is possible that the mechanism of delayed traumatic facial weakness is similar to that of Bell's palsy and that in both conditions there is an inflammatory reaction in or around, or a swelling of the facial nerve in its canal, and that this leads to ischaemia and in some cases to denervation. If this is so, and if one could identify those patients particularly liable to develop this complication of head injury, it might be expected that prophylactic treatment with A.C.T.H. would be effective.

The results we have obtained so far are sufficiently suggestive of a protective value of A.C.T.H. against late traumatic facial weakness that we feel justified in publishing this preliminary communication—particularly since, with Taverner *et al.*, we have found the treatment itself to be harmless, provided a correct dosage is observed.

Selection of Patients

In cases of head injury, damage to the facial nerve in its canal is nearly always associated with a fractured petrous bone, though it may sometimes be difficult, even with specially projected views, to demonstrate radiologically the fracture and its precise relation to the facial canal. A common clinical sign of this type of fracture is bleeding from the ear on the same side. In a series of 2,712 consecutive and unselected cases of head injury admitted to the Radcliffe Infirmary, Oxford, some degree of facial nerve involvement was detected in approximately 50% of patients showing unequivocal radiological evidence of a fractured petrous bone whether or not this was accompanied by a bleeding ear (Potter, 1964).

Two varieties of facial weakness are usually recognized: *immediate,* where the nerve has been directly concussed, contused, or lacerated at the moment of injury; and *delayed,* where the weakness results from some reaction occurring after an interval, usually during the first week or occasionally even later. In addition, a third, or *mixed,* group of cases can be recognized, where there has been an immediate weakness, which may then either recover and be followed by delayed weakness or progress later to a more severe weakness or complete paralysis (Potter, 1964). It is not our policy to operate on patients in the first group, and the present paper is concerned with *delayed facial weakness only.* The prognosis in this variety is normally good, but denervation and perverted reinnervation may occur and result in some permanent disfigurement. Prevention of the complication should therefore be attempted if a simple and safe method is available, and this appears to be provided by A.C.T.H.

In choosing patients for prophylactic treatment we felt it desirable that criteria should be used which could be readily recognized in casualty departments without the need to employ sophisticated techniques of investigation. It was also necessary that we should be able to discover, from an untreated control group, the expected incidence of facial weakness in the series to be treated.

During the past year we therefore selected all patients who had a bleeding ear and who were shown also to have radiological evidence of a petrous temporal fracture on the same side. Those with blood behind an intact drum were not included. In an earlier series of 5,000 consecutive and unselected cases of head injury admitted to this unit between 5 January 1962 and 9 May 1965 we found that there had been 70 patients with the same combination of a fractured petrous bone and a bleeding ear and that 27 of these had shown some degree of delayed facial weakness—an expected incidence for this complication, therefore, of 38.5%.

Results

In the past year there have been 16 patients showing the combination of a fractured petrous bone and bleeding ear, and all have received prophylactic A.C.T.H. soon after the injury. Without treatment we should have expected six of these patients to develop some delayed facial weakness; but only two did so. One developed rapidly a complete facial palsy on the second day after injury; denervation ensued despite treatment, and there has been no recovery during the month following the injury. In the second case there was only slight and transient facial weakness lasting for some 10 hours on the second day after injury. The ages of the 16 patients ranged from 2 to 77.

We have the impression—but it is no more than a clinical impression—that these patients receiving A.C.T.H. have recovered unusually smoothly from the other effects of their head injuries.

Method

Corticotrophin (A.C.T.H.) gelatin injection *B.P.* was given daily by intramuscular injection for eight days. Since, in the great majority of cases, delayed traumatic facial weakness appears during the week following injury (Turner, 1944; Potter, 1964) and because our object was prophylaxis, we did not feel it necessary to employ the full 10-day therapeutic course adopted by Taverner *et al.* for patients with Bell's palsy, except in the case of a single patient who developed the complete paralysis between the second and third doses.

The first injection was given within a few hours of the injury. The dosage was as follows: 80 units daily for three days, 60 units on the fourth day, 40 units on the fifth day, 20 units on the sixth day, and 10 units on both the seventh and eighth days. For children the dosage was reduced according to weight and age, but this reduction was apparently insufficient in a 3½-year-old who showed marked oedema of the lower abdominal wall and genitalia for five days at the end of his course of treatment.

Though one might suspect that the plasma cortisol level is relevant to this problem, we know of no certain evidence yet that this is so, and Taverner (1954) had already found cortisone ineffective in the treatment of Bell's palsy. Nevertheless, after the first dose we gave the A.C.T.H. in the afternoons, because it is known that the plasma cortisol is normally at a relatively low level at that time—as also is the level of A.C.T.H. (Ney *et al.*, 1963). (If, indeed, a high plasma cortisol level contributes to the protection of a patient from delayed traumatic facial weakness we would expect this complication to occur less often in the more severely injured patients. This possibility is at present under investigation.)

SUMMARY

Sixteen patients were given prophylactic A.C.T.H. after they had sustained head injuries. Each had shown radiological evidence of a fractured petrous bone and had had bleeding from the ipsilateral ear.

The expected incidence in an untreated group of the same size would have been six patients showing delayed facial weakness.

Only two patients developed delayed facial weakness. In both this occurred on the second day. In one it was mild and lasted only 10 hours; in the other it was complete and denervation occurred despite treatment.

It seems that A.C.T.H. offers some protection against this complication of head injury.

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Medical Memoranda

Sarcoid Myopathy

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Only 12 cases (Hinterbuchner and Hinterbuchner, 1964) have been found in the literature where sarcoidosis presented as a pure myopathy without evidence of other systemic involvement. I present another case which has responded dramatically to corticosteroid therapy.

CASE HISTORY

A 55-year-old Englishwoman presented in March 1966 with a three-week history of calf stiffness alleviated by exercise. She had no other symptoms, was on no drugs, and had no relevant family or social history.

In April she was admitted to hospital, and examination showed severe weakness in her legs, more so on the left, with slight thigh wasting. Weakness was also present in the elbows and wrists, but without wasting. The muscles were non-tender and felt normal. Her gait was stiff and wide-based, and she tired easily. No other significant abnormality was found and no definitive diagnosis made. Physiotherapy produced some improvement, and she was discharged after one month. By August wasting and weakness had developed in all proximal limb muscles. Walking was difficult without support, resulting in several falls.

She was readmitted to hospital in January 1967 with severe weakness in her hands, hardly able to walk even with support, and unable to sit up unaided or do her hair. She had an occasional dry cough but no other complaints. Examination showed obvious muscle wasting of all the limbs, whose tone was slightly reduced. The muscles felt rubbery but were not tender, fasciculating, or showing involuntary movements. Movement was full, but with severe generalized weakness, including the sternomastoids and face. The other systems showed no abnormality.

Investigations.—A skeletogram showed mild osteoporosis; W.B.C. 3,900–7,000/cu. mm. (monocytes 8.5–12.5%); serum proteins 6.5–7.2 mg./100 ml. (albumin 3.0–4.4 mg., globulin 2.8–3.5 mg.); normal electrophoresis; latex fixation test weakly positive (Putkonen *et al.*, 1965); urine creatine 460 mg./24 hours (normal 0–50 mg.). Muscle biopsy showed no necrosis or acid-fast bacilli but the characteristics of sarcoidosis—a granulomatous process with small foci of epithelioid cells surrounded by small mononucleated cells and some multinucleated giant cells.

The following investigations showed no abnormality. Blood: haemoglobin, platelets, P.C.V., M.C.H.C., film, L.E. cells, E.S.R., urea, electrolytes, cholesterol, alkaline phosphatase, calcium, phosphorus, liver function, antistreptolysin titre, aldolase, lactic acid dehydrogenase, creatine, creatine phosphokinase, creatinine, lead, culture. Urine: routine analysis, bile derivatives, porphobilinogen, uroporphyrins, Bence Jones protein, calcium, creatinine, renal steroid leucocyte excretion, intravenous pyelogram. Throat swab, culture for tubercle, 1:10 tuberculin test, Wassermann reaction, V.D.R.L., cerebrospinal fluid, electrocardiogram, Kveim test, radioactive iodine uptake.

Treatment was begun in February with prednisone, 15 mg. daily, reducing to 10 mg. daily in June, the only available therapy of value (Crompton and MacDermot, 1961; Silverstein *et al.*, 1965). After three weeks she could sit up unaided, and by April felt much improved and could walk 30 yards (27 metres) with support. Power had improved considerably in her legs and was normal in her arms apart from slight weakness in her right fingers. In June she was walking up to 100 yards (90 metres) with slight support.

Though remissions occur in the sarcoid process, I thought that, since the condition had progressed inexorably for 15 months, corticosteroid therapy had caused rapid improvement.

COMMENT

The skin lesions of sarcoidosis were first described by Boeck (1899), and literature has amassed covering involvement of nearly all the tissues in the process (Mayock *et al.*, 1963). The diagnosis of sarcoidosis should always be confirmed by histology (Mayock *et al.*, 1963), usually accompanied by an intermittent monocytosis, eosinophilia, leucopenia, raised serum calcium and globulin, and occasionally a raised alkaline phosphatase. A positive Kveim reaction occurs in 60% of patients, with false-negative results in long-standing or inactive disease (Siltzbach, 1964) and during corticosteroid therapy.

The outcome of sarcoidosis is usually spontaneous remission, but irreversible lung or myocardial fibrosis may cause death. Treatment with A.C.T.H. or corticosteroids may produce remission (Silverstein *et al.*, 1965), but 60% of patients do not respond (Crompton and MacDermot, 1961).

Since Sundelin's (1925) original report several cases of muscular involvement in generalized sarcoidosis have been described, taking several forms—asymptomatic microscopic granulomata, palpable nodules, or myopathic. A purely myopathic presentation is very rare, only 12 cases have been found in the literature (McConkey, 1958; Hinterbuchner and Hinterbuchner, 1964). Muscle biopsy shows characteristic sarcoid histology—interstitial infiltration with giant cells, epithelioid cells, and lymphocytes, with tissue replacement by a pallisade-like and reticular collagen fibre network, leading to fibrosis. Occasionally widespread muscle fibre degeneration