days after the onset of paralysis but we have no firm evidence on this point. Treatment should begin as soon as possible unless there are specific contraindications or the condition is mild. Side-effects are not common and it is submitted that the risks are justified by the reduction in the amount of permanent sequelæ.

Acknowledgment: I am indebted to the Editor of the *British Medical Journal* for permission to reproduce material already published.

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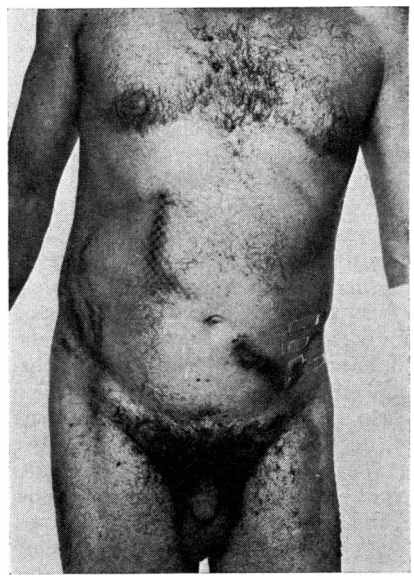

Fig 1 **Case 4** *Burns on trunk with imprint of vest*

Mr Anthony Richards
(St Mary's Hospital, London W2)

Traumatic Facial Palsy

Four cases of traumatic facial palsy presented different problems of management:

Case 1
Partial palsy of delayed onset following blunt trauma. A young woman was struck on the side of the head with a beer mug and knocked unconscious for five minutes. She had transient deafness and vertigo, but only presented when a partial left facial palsy developed after two weeks; nerve excitability tests showed depression on the injured side. X-rays showed no fracture. In this case of delayed palsy with little likelihood of fracture, there was no indication to explore the nerve, and an ACTH regime was given (Taverner 1966). Clinical improvement occurred at five days, and recovery was complete by two weeks, with normal excitability tests.

Case 2
Subtotal palsy of delayed onset with suspected fracture. A young woman received a flat blow to the side of the head as she fell over on to a concrete floor; X-rays showed no fracture, and the initial diagnosis, as there was blood in the meatus and a conductive deafness, was ruptured tympanic membrane. The facial palsy began on the fourth day, and on the next day she presented with subtotal paralysis, absent taste sensation, normal lacrimation, a blue immobile tympanic membrane, and 60 dB conductive deafness; vertigo had been present for two days. The decision to operate was based on the suspicion of fracture, and ideal timing – just as the palsy was becoming complete. A fracture was found to run across the mastoid process, down the meatus, across to the medial wall, and on to the tegmen, where there was a loose fragment of bone. The nerve was explored and decompressed, and a spicule of bone which was embedded in the sheath, damaging a few fibres half way down the descending part in the line of the fracture, was removed. Some recovery had occurred in two weeks, and full movement in four weeks.

This accords with the conclusions of McHugh (1963) that a delayed palsy does not rule out disrupted anatomy.

Case 3
A sharp injury divided the nerve within the parotid; careful suturing produced a good result. This youth received a gash on the side of the face through which parotid tissue bulged out; facial movement was present only in the mandibular region. At operation a large piece of glass was removed from the wound. The nerve trunk was found using a standard parotidectomy incision, and the branches traced in the usual way; the injury was at the second branching, and spared only the mandibular and cervical subdivisions. The peripheral ends were found by retrograde dissection from the front of the gland, and continuity restored by direct suture of the four branches with human hair, using a Bunnell double right-angled stitch. At one month the upper fibres were denervated, and there was no movement, but function was returning by 6 months, and recovery was complete by 10 months.

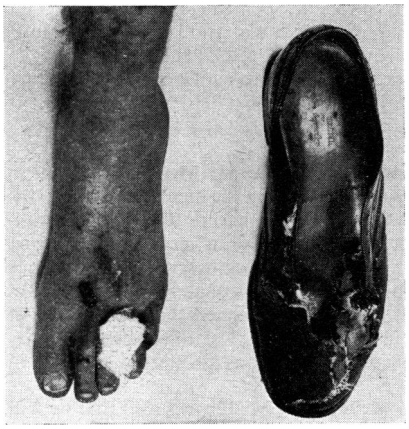

Fig 2 **Case 4** *Left foot and boot*

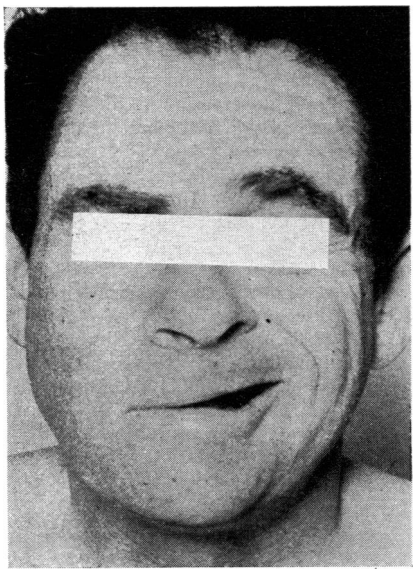

Fig 3 **Case 4** *Right facial palsy caused by lightning strike*

Case 4

A man aged 47 was sheltering under a tree during a storm, holding an umbrella in his right hand, when he was struck by lightning; this caused superficial burns to his arms, trunk, and legs, imprinting the pattern of his vest on to his abdomen (Fig 1), and was of sufficient force to blow the boot off his left foot (Fig 2), and to tear a ten shilling note in his hip pocket along the metal strip. He was unconscious for two hours. Next day he complained of deafness and tinnitus, and on the fourth day developed a right-sided facial palsy (Fig 3). There was a subtotal central perforation of the right drum with charred ragged edges. Audiography showed a conductive deafness on the right, with a 20 dB air-bone gap, and a C5 dip. The palsy began improving in one week, and completely recovered in two. The perforation healed spontaneously in eight weeks. He is none the worse for the experience, the only residual defect being a permanent loss on the audiogram at 4000 Hz.

Facial palsy after a lightning strike is rare; Bertelsen (1958) recorded a case (also of delayed onset), but no other cases could be found in the literature.

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DISCUSSION

Mr Ian P Munro (*Renfrewshire*) said that in a series of over 100 cases of Bell's palsy treated within twelve days of onset, most with prednisolone, some with ACTH, experience had been that degeneration, if it occurred, was slight in those aged under 40. Older patients, particularly if hypertension was present, seemed to suffer more severe degeneration, and some of these patients did not receive prednisolone.

Mr Munro asked Mr Groves and Dr Taverner whether age and hypertension affected the degree of degeneration in their series.

Mr William S Lund (*Oxford*) said that in view of the finding by Fisch & Esslen (1972) of a markedly swollen and congested facial nerve at the beginning of the fallopian canal i.e. proximal to the geniculate ganglion, there might be an indication in certain cases of total VII nerve palsy (Bell's type) for surgical decompression of the nerve from the lateral end of the internal auditory meatus outwards, via the middle cranial fossa approach. Further justification for this 'central decompression' was afforded by Professor Fisch's results of direct stimulation of the facial nerve, which showed a complete obstruction to electrical impulses at the beginning of the fallopian canal corresponding to the site of the swollen nerve.

REFERENCE
Fisch U & Esslen E
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Mr Colin M Johnston (*Faringdon, Berkshire*) said he had found a test described by Mr Charles Gledhill (personal communication) of value.

During mastoid surgery, particularly for cholesteatoma, the facial nerve was occasionally at risk from bruising. At the end of the operation it was possible to check the function: the anæsthetist was asked to lighten the plane of anæsthesia and the endotracheal tube was then slowly jerked in and out, producing a movement of the alæ nasi; if this move-

ment was present equally on both sides it gave reassurance that there was no damage to the nerve.

A second test, which could be used in the clinic, would determine whether full return of facial nerve function had occurred in the eyelid muscles. If the surgeon flicked his hand towards the patient's face as if to touch him a reflex closing of the eyelids would occur. Until full recovery of function had taken place there would be a slight time lag in the closing of the affected eye.

Mr J W Dixon (*Torquay*) said that he strongly supported Professor Miehlke's belief in early exploration when facial paralysis followed middle ear surgery. This was particularly necessary when there was doubt as to the state of the facial nerve at the end of the original operation.

Before completion of any ear operation, Mr Dixon now deliberately exposed and inspected any possibly damaged section of the facial nerve. In certain cases it was thus possible to decide at the first operation that the nerve was intact though perhaps bruised, and that should facial palsy occur postoperatively the correct treatment was conservative. Thus the need for and worry about further exploration might sometimes be avoided.

Mr A S Shalom (*St Peter's Hospital, Chertsey, Surrey*) asked for views on the value of stellate ganglion block in the treatment of Bell's palsy. On three occasions he had observed a response of either a twitch or a movement in the face within thirty minutes after stellate ganglion block was performed. In two of these patients the palsy was total. In a large number of other cases of facial palsy there was equivocal response from stellate ganglion block.

Mr H W H Shepperd (*Belfast*) asked whether Dr Taverner could offer any explanation why prednisolone should be more effective than ACTH.

Mr Shepperd reported a case of greenstick fracture involving the facial nerve.

Mr John Groves, in reply to Mr Munro, said that in his own series of Bell's palsies the incidence of denervation in the older age-groups had not been very significantly greater. It was, however, a very well recognized correlation which had been observed in numerous other published series. Unfortunately as far as selection for treatment was concerned the greater risk of denervation in older patients was offset by the higher incidence of contraindications to steroid therapy in this group.

In reply to Mr Shalom, Mr Groves said that he had not employed stellate ganglion block for Bell's palsy for many years, there being no satisfactory evidence that it could favourably influence the outcome to justify the significant hazards of the procedure.

Dr Taverner, in reply to Mr Munro, said that the frequency and severity of denervation in Bell's Palsy were directly related to the age of the patient at onset. Denervation was rare below the age of 10 years and became increasingly common with age, especially over 40–50 years.

He had no reliable evidence about the effect of hypertension.

In reply to Mr Shepperd he said that the result of the trial was surprising since it had been designed to show the opposite effect. Presumably the results of ACTH therapy were less satisfactory because of the variable response to stimulation of different adrenal glands and the relative delay in achieving adequate steroid levels.