Facial palsy in closed head injuries : the prognosis / J.W. Aldren Turner.

Reprinted from THE LANCET, *June* 10, 1944, *p.* 756.

FACIAL PALSY
IN CLOSED HEAD INJURIES
THE PROGNOSIS

J. W. ALDREN TURNER, D M OXFD, M R C P
MAJOR RAMC; NEUROLOGICAL SPECIALIST

From a Military Hospital for Head Injuries

THE purpose of this paper is to determine the prognosis of peripheral facial palsy occurring as a complication of closed head injuries. Cases of incomplete recovery are apt to obtrude themselves and give a wrong idea of the prognosis. A series of 70 consecutive cases with a traumatic facial palsy admitted to a military hospital for head injuries were studied for this purpose. The types of injury were :

Motor cycle accidents ..	21	Cycle accidents	5
Car accidents	17	Air-raid casualties	4
Falls	12	Crush injuries	2
Injury to pedestrians by vehicles	7	Miscellaneous	2

The facial nerve was the second most commonly injured of the cranial nerves in a series of 1550 consecutive head injuries studied in another connexion (Aldren Turner 1943), damage to it being exceeded in frequency only by damage to the olfactory nerve.

Post-traumatic amnesia is often used as a criterion of the severity of head injury, though at times it is open to fallacy (Symonds and Russell 1943). The length of post-traumatic amnesia in these 70 cases was as follows :

No loss of consciousness (including 1 crush injury) ..	6	1–24 hours	16
Under 1 hour	9	1–7 days	30
		Over 7 days	9

There was no constant position of impact to the head as judged by the site of abrasions or bruises in the early stages after the injury : this is in contrast to cases of

injury to the optic nerve where the position of impact is almost always in the frontal region ; and to olfactory nerve injuries where frontal or occipital impacts are usual (Leigh 1943).

Traumatic facial palsies are of two types : those occurring at the time of the injury, and those delayed in onset. In patients comatose for some days after an injury there may be some difficulty in determining the time of the onset of the facial palsy. In this series there were 36 immediate palsies, and 34 cases of delayed palsy among which was 1 case with a bilateral traumatic facial palsy.

Delayed facial palsy developed at any time between the 2nd and 8th day after the injury, as follows :

Day after injury	Cases	Day after injury	Cases
2nd	1	6th	2
3rd	5	7th	5
4th	5	8th	2
5th	3		

The bilateral case will be considered separately. In one case in which the facial palsy developed on the 8th day, only the lower part of the face was affected on that day, and the signs resembled in some ways an upper motor neurone lesion ; but by the next day the upper part of the face was affected as well.

In 22 of these delayed cases the facial palsy was only partial, in 11 it was complete. In no less than 19 cases recovery started within a few days of the onset of the palsy and progressed to complete recovery within less than 3 weeks. Another 6 cases began to recover within 3 weeks of the palsy appearing, and in these recovery was complete within a further 4 weeks, except for one case which did not clear up completely for 12 weeks after the injury. Of the remaining 8 cases, 5 are known to have recovered completely within 4 months, but it is not certain exactly how long recovery took, and 1 had been posted abroad and could not be followed up.

The other 2 cases were less satisfactory. One showed incomplete recovery with associated facial movements in 6–8 months ; this sequel is commoner in the immediate palsies and will be discussed later. The last case developed an acute otitis media 5 days after his injury, which had been accompanied by bleeding from both ears ; this man's facial palsy developed on the same side

as, and at the same time as, the acute otitis media, and 6 months later there was no clinical evidence of any recovery in the facial nerve. A mastoid operation had been done in the interval, but the facial nerve had not been inspected.

Immediate facial palsy.—Of these cases, 17 were only partial, the other 19 complete. As in the delayed palsies, a group (9 cases) started to recover in a few days and were normal within about 3 weeks, in another group (15 cases) recovery started within 3 weeks and was complete within 6–8 weeks. There was, however, a considerably larger proportion of slow and incomplete recoveries in the immediate than in the delayed palsies. Of the remaining 12 cases of immediate palsy, 3 began to recover within 6–8 weeks and had recovered completely within 3 months: 6 showed no improvement for 3 months after injury and then slow recovery started and progressed for some months. In these cases normal facial movements did not return; for though eventually there was fairly good voluntary power some contracture of the facial muscles remained with deepening of the nasolabial and other grooves on the face. The most disfiguring feature, however, was the development of associated facial movements which interfered with the normal free play of facial expression; when the eyebrow was raised the corner of the lip on the same side would retract and on smiling there might be involuntary wrinkling of the forehead. In addition, in most of these cases some impairment of voluntary power persisted so that there was facial asymmetry when the patient was smiling or talking.

In this group of cases, where delayed and imperfect recovery takes place and associated movements develop, there has been actual regeneration of the facial nerve; while in the cases showing quicker and complete recovery the neural lesion has been a physiological block without actual nerve degeneration. The physiology of the associated movements has been given an anatomical basis by the experimental work of Howe, Tower and Duel (1937), who studied the facial recovery in monkeys after freezing, alcohol injection, section, and suture of the nerve. They found that as voluntary power in the face returned mass movements similar to those seen in man appeared and that as the paralysis became less these movements spread over a progressively wider area. They made histological studies of the regenerating nerves in

3 monkeys in which there were definite associated movements, and demonstrated branching of the regenerating axons which gives one axis cylinder connexions over a wide area of the facial musculature.

Of the 3 remaining cases in my series, 1 has been followed up for over two years and there is no return of voluntary power; it has proved impossible for me to examine the other 2, but in 1 case the patient's doctor reported that there had been no recovery after 18 months, and the third patient reports that there has been no improvement in two years. It is noteworthy that in 2 of these 3 bad results there was an acute otitis media on the side of the facial palsy after the injury; it will be recalled that there was also an acute otitis media in the only case among the delayed palsies where there had been no recovery after 6 months. These findings, though perhaps not conclusive, suggest that a middle-ear infection complicating a traumatic facial palsy is of bad import.

In the early stages of a complete palsy it is impossible to tell whether rapid recovery will follow or whether regeneration of the nerve will have to be waited for. After three weeks the electrical reactions are of some help; if a faradic response is still obtainable rapid recovery will follow in most cases. In some of the later cases of the series electromyographic records were made by Captain Graham Weddell, RAMC, and it is hoped that further work of this type will make it possible to tell at earlier stages whether the nerve has actually degenerated.

Bilateral facial palsy.—There was one case in the series of a bilateral traumatic facial palsy in which both sides recovered completely in the course of 8 weeks—this case was admitted to hospital about a fortnight after his injury; the previous records were incomplete, but it appeared that both facial palsies had been delayed in onset. There are a number of records of bilateral cases (Keiper 1923) and the prognosis does not seem to differ from the unilateral cases.

ÆTIOLOGY

In at least 51 of the 70 cases there was bleeding from the ear on the side of the facial palsy immediately after the injury, and it is likely that the figure is really higher

than this. In 5 cases there was leakage of cerebrospinal fluid from the ear which stopped in a few days and did not recur. Meningitis never developed, though in 1 case there was an ærocele ; in this case thorough X-ray studies were made of the frontal sinuses and cribriform plate and no fracture could be demonstrated, so it was presumed that the intracranial air entered through a tear in the dura overlying the petrous part of the temporal bone, an event of considerable rarity. A similar case is described by Davis (1943).

Deafness on the same side is a common accompaniment of a traumatic facial palsy ; there is no connexion between the degree of deafness and the prospect of quick or slow facial recovery. Complete perception deafness may be present with no facial palsy ; and in a case not included in this series, where a severe degree of facial palsy had persisted for 26 years after a head injury, I was unable to demonstrate any deafness to the watch or whispered voice.

It is probable that a traumatic facial palsy is always associated with a fracture in the petrous part of the temporal bone, though this is not always demonstrable in X-ray studies, especially when the fracture line is in the longitudinal axis of the bone. The exact cause of the facial palsy is not certainly known, but it may be either a transient block or a lesion in continuity. In the immediate cases the lesion is probably either laceration of the nerve or an intraneural vascular accident, while in delayed cases pressure on the nerve by blood in the fallopian aqueduct is the likely cause.

TREATMENT

In the large proportion of rapidly recovering cases no treatment is needed ; and in the slowly recovering ones no treatment will hasten regeneration of the nerve : all that can be done is to try to keep the facial muscles in good condition till the nerve has regenerated. A wire splint hooking round the corner of the mouth and the back of the pinna should be worn to prevent the muscles sagging, and massage, given by the patient himself working upwards and outwards from the angle of the mouth, should be applied several times a day. Regular treatment with galvanic current is probably worth while, though the experimental evidence of its value is

still meagre. When recovery starts it is advisable for the patient to practise facial movements in front of a mirror, especially where associated facial movements are occurring, in an endeavour to move the various parts of the face separately. Unfortunately, if Duel's view of the mechanism of production of associated movements is correct, this constant practice is unlikely to yield much in the way of results.

As regards operative treatment, no series of results has been published. My series provides no argument for operating unless there has been no recovery for at least 6 months after the injury, in which case exploration of the nerve in the facial canal might possibly be advisable. Electromyographic records should be made first to determine whether any motor units are getting through to the muscles (Weddell et al. 1943).

Could early decompression of the facial nerve in the canal, in the cases which are going to recover slowly by regeneration, hasten the recovery and prevent the development of associated movements and facial contracture? The answer to this is unknown, and if a series of cases treated in this way should be published in the future, it will be instructive to compare it with the present series, in which no decompression operation has been performed.

SUMMARY

A series of 70 consecutive cases of facial palsy complicating a closed head injury have been followed up.

Of the 34 cases of delayed palsy good recovery occurred in all except 2.

Of the 36 cases of immediate palsy, 6 showed incomplete recovery with development of associated movements and 3 showed no recovery at all.

Of the 4 cases (3 immediate and 1 delayed) in which there was no recovery, 3 had had an acute otitis media on the side of the facial palsy.

My thanks are due to the Officer Commanding, Military Hospital (Head Injuries), for permission to study these cases; to Brigadier Hugh Cairns, and Captain Graham Weddell, RAMC, for help in the preparation of the paper; and to the Medical Research Council, which sponsored the head injury follow-up scheme.

REFERENCES

Davis, D. O. (1943) *Brit. J. Surg.* **30**, 237.
Howe, H. A., Tower, S. S. and Duel, A. B. (1937) *Arch. Neurol. Psychiat.* **38**, 1190.
Keiper, G. F. (1923) *Ann. Otol., &c., St. Louis*, **32**, 236.
Leigh, A. D. (1943) *Lancet*, i, 38.
Symonds, C. P. and Russell, W. R. (1943) *Ibid*, p. 7.
Turner, J. W. A. (1943) *Brain*, **66**, 140.
Weddell, G., Feinstein, B. and Pattle, R. E. (1943) *Lancet*, i, 236.

The Lancet Office,
7, Adam Street, Adelphi, London, W.C.2.