Clinical Records

Delayed conductive deafness

By Ivor J. C. Frew (Newcastle upon Tyne)

The well recognized groups of traumatic deafness need little explanation. In Newcastle, patients showing a purely perceptive loss form the largest group. A long way behind come the group of mixed conductive and perceptive deafness. Finally, the purely conductive type is extremely uncommon.

At Newcastle General Hospital about 600 patients who have sustained head injuries are admitted each year to the Neuro-surgical department. A patient may be in the ward for only 24 hours, or, with a more severe injury, for a much longer period. The total for each year has been diminishing from a peak in 1964. Some of this is perhaps attributable to the decrease in heavy industry and coal mining, both unfortunately contributing quite heavily to the total number of head injuries (Table I).

TABLE I.
NEWCASTLE GENERAL HOSPITAL
HEAD INJURY ADMISSIONS.

1964–965
1965–703
1966–686
1967–578
1968–582

All patients have a careful examination of their ears on admission, and if any damage is apparent, the appropriate treatment is instituted. We try to ensure that all deaf patients have an audiogram performed before they leave hospital. At Newcastle General, over the past five years, an average of 17 per cent of patients admitted annually with head injuries complain of some deafness on leaving hospital.

I would like to add a fourth group to the three already described. These cases I have called a conductive deafness of delayed onset. A case record will illustrate best what I mean.

A girl, aged 18, was involved in a road accident in November 1964. She was unconscious on admission, with bleeding from the nose, but her ear-drums looked quite normal.

She regained consciousness in a few days, her only complaint, apart from a headache, being some deafness in the left ear. An audiogram demonstrated a perceptive deafness in this ear. Her recovery was uneventful, and she soon went home.

https://doi.org/10.1017/S0022215100072716 Published online by Cambridge University Press

An Out-Patient review by myself in October 1965, a year later, showed her hearing had returned to normal.

However, in July 1966, again almost a year later, she returned to Out-patients complaining of a return of the left-sided deafness. This had come on quite quickly over the past month or so She had not had any infective ear episode since her original injury, nor had she sustained any further trauma. An audiogram showed a left conductive deafness. I suggested to her that the correct procedure would be to look inside her ear to try and find the cause of the deafness, and perhaps improve her hearing. The patient was agreeable, and she was submitted to an exploratory tympanotomy in August 1966.

Once the middle ear had been opened, there was no difficulty in demonstrating the pathology. The terminal portion of the long process of the incus had been destroyed, although a thin fibrous strand still remained to show its original position. The capsule of the incudo-stapedial joint, containing the lenticular process, although thickened, appeared intact (Fig. 1).

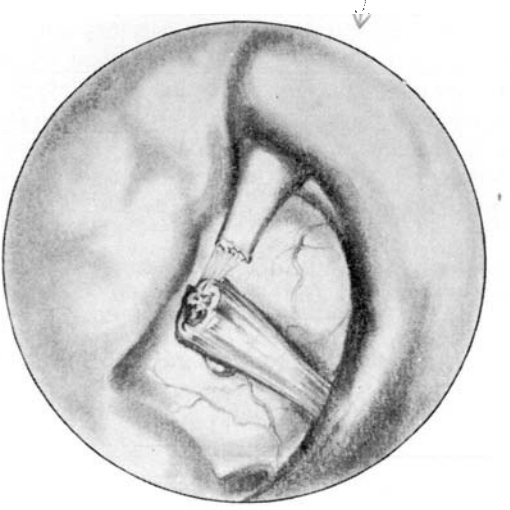


FIG. 1.

Continuity was re-established by trimming the end of the necrosed bone, cutting the tendon of the stapedius muscle and thus obtaining apposition of the incus and stapes. Some Gelfoam was placed around the new incudo-stapedial union, and the drum-head replaced. Her recovery was quite satisfactory, but her hearing only came up to within 15 decibels of normal, perhaps not a very good result, but at least some improvement.

Since then I have seen three more cases. These have been submitted to operation and identical ossicular disruption has been demonstrated in all cases. Adequate social hearing was obtained at about 15 decibels below threshold. A further case, which was discovered in the course of a medico-legal examination, refused operation.

All four cases had identical clinical histories, with about two years between the original head injury, and the onset of the conductive deafness.

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I do not see any reason why such cases of delayed conductive deafness should necessarily be associated with a preliminary period of perceptive loss. I would regard the latter as being purely a matter of chance. To my mind, the important factor must be the damage which occurs at the time of the accident, probably to the blood supply of the long process of the incus.

An obvious question must be—why so few cases in such a long time? I have been closely associated with the treatment of severe head injuries since 1951, but for many years the work was on the resuscitation of severe brain-stem injuries in association with my late friend and colleague, Mr. Ian McIver. Only since this problem has been somewhat improved, has time been available to give to a more detailed study of purely Ear, Nose and Throat damage to head injury patients. More staff, both neuro-surgical and Ear, Nose and Throat, has enabled us to provide some sort of service towards assessment of hearing, although even now it is not nearly as good as I would wish.

Continuous observation of these patients may be difficult. Many patients, especially those involved in road accidents may live in other parts of the country. Even some local inhabitants are unwilling to return regularly, accepting partial deafness as being of little consequence, unless their employment or social activities are threatened.

There is however, no doubt that any patient coming to an otologist, with a conductive hearing loss, would have every attempt made to determine and remedy the pathology. So, the question of any previous head injury might well not be relevant, provided the patient was seen and treated.

Surely, the danger possibly lies with those patients who have sustained a traumatic perceptive deafness which gradually clears up, not an uncommon story. Should some deafness return, the patient may well consider it to be a legacy of the original injury, and not seek advice. We now try to tell all patients to attend their own practitioner should any change in their hearing become apparent, in order that they can be referred to an Otologist.

From the few cases seen so far, it would seem reasonable to suppose that delayed conductive deafness, secondary to trauma, may appear about two years after the accident.

I would like to thank all my neuro-surgical colleagues at Newcastle General Hospital Regional Neuro-surgical centre for asking me to see their patients, and for allowing access to all their records.

The drawing is by Miss Mustart of the University of Newcastle upon Tyne.

E.N.T. Department
Newcastle General Hospital,
Newcastle-upon-Tyne.

https://doi.org/10.1017/S0022215100072716 Published online by Cambridge University Press

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